On Evolution Under Sexual and Viability Selection: A Two-Locus Diploid Model

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Abstract.—A two-locus diploid model of sexual selection is presented in which the two loci govern, respectively, a trait limited in expression in one sex (generally male) and the mating preferences of the other sex (generally female). The viability of a male depends on its genotype at the trait locus. In contrast, all females are equally viable and all individuals are equally fertile with respect to the two loci. Near fixation at both loci, evolution at the mating locus is neutral and hence a new mating preference allele will increase only through random genetic drift or through a correlated response to the increase of a new advantageous trait allele. If, however, a polymorphism is already maintained at the trait locus through overdominance in fitness then the increase of a rare preference allele depends only on the recombination rate between the loci and not on the new preference scheme.

Key words.—Diploid model, disequilibrium, mate preference, polymorphism, sexual selection.

The importance of sexual selection as an evolutionary force is suggested by the ubiquity of secondary sexual characteristics and the remarkable nature of some of these traits such as the majestic feathers of the peacock or the ruby-red throat of the frigate. Starting with Darwin, many people have considered the evolutionary role of sexual selection whereby an advantage is conferred upon a phenotype not because it is more commonly chosen by "natural selection" but because it is more commonly chosen by potential mates [see, for example, Fisher (1958), O'Donald (1980), Lande (1981), Kirkpatrick (1982)]. Technically, sexual selection is not a counter-example to "survival of the fittest" because fitness measures reproductive output as well as viability, the former being a function of fertility, inclination to mate, and preferability.

Relationship between Different Selective Forces.—Were sexual selection to occur without affecting fecundity and on traits that do not appreciably contribute to viability, then different mating preferences would be neutral with respect to one another and the evolution of preferences would occur through the random action of genetic drift. However, in species with few potential mates, choosiness would almost inevitably lead to reduced fecundity. Furthermore, the very traits that are preferred often confer no viability advantage or may even be disadvantageous. For instance, individuals who produce pheromones can attract predators and parasites as well as prospective mates [e.g., Harris and Todd (1980)]. Hence, the fitness of an individual is often a function of fecundity and viability differences as well as the mating preferences within the population. Finally, as these mating preferences change from one generation to the next, so will the relative fitnesses of the individuals within a population. The question, then, is in what ways do these different selective forces interact to affect evolution. Mathematical models can help clarify the answer to this question.

Previous Models.—To study the simultaneous evolution of mating preferences and those traits under selection, models have been developed assuming either polygenic inheritance or inheritance based on relatively few loci. Quantitative genetic models in which both the preferences and the traits are polygenic have shown that sexual selection may be maladaptive, even to the point of the extinction of a species (Lande, 1980). When there is no direct viability or fertility selection on the preference locus, Lande (1981) noted that changes at the preference locus occur only as a "correlated response to selection on males" through associations between the loci (disequilibria). If an equilibrium is attained, it is on a line of neutral equilibria on which the forces of natural and sexual selection balance. The stability of this line depends only on the "mutability" of the genes and not on the linkage relationships between them (Lande, 1981). Finally,

in this theoretical framework, it is established that mating preferences need not be "adaptive" to evolve, that there is not a genetic "basis for expecting that females should generally be attracted to the most vigorous males in a population" (Heisler, 1985).

Assuming that sexual selection is based on the action of few loci, Kirkpatrick (1982) developed a haploid model to track the change in genotype frequencies at two loci, where one locus controls female mating preferences and the other locus controls a trait limited in expression to males. As in Lande's models, there is no direct viability or fertility selection on the preference locus and hence the preference locus evolves only through associations with the trait locus. Again as before, the equilibria of this system form a neutral curve, such that the final composition of a population is sensitive to both initial genotype frequencies and to perturbations in these frequencies. Finally, the rate of recombination did not alter the equilibrium curve nor its stability. Heisler and Curtsinger (1990) extended this model to the diploid case under the restrictions of free recombination ($r = 0.5$), an additive preference scheme, and directional viability selection (heterozygotes have intermediate viability). They also examined the important case of direct selection on the preference locus through the action of fertility selection, although I shall focus on their results for preferences that are selectively neutral. From simulations, they concluded that the diploid model differs from the haploid and polygenic models in that neutral curves of equilibria do not exist except in the case of complete dominance at the trait locus. Polymorphisms were rare, occurring in only 32 out of 9,920 simulations. Instead, evolutionary trajectories generally led to fixation at either the trait or preference loci. Movement along these trajectories, however, often occurred at very slow rates—"sufficiently slow that random genetic drift could easily overwhelm deterministic evolution in all but very large populations." Finally they noted that trajectories that led to the greatest change in the frequency of a preference allele occurred when heterozygotes were the most fit.

Recently, Gomulkiewicz and Hastings (1990) analyzed the stability properties of fixation on a trait allele to the introduction of a new trait allele when there was a polymorphism at the preference locus. They found that if the new trait allele had a higher fitness (calculated as the product of its viability and the harmonic mean of its mating advantage) then it would invade. This result along with the results presented within this paper give a complete picture of the stability properties of the two-locus two-allele model when at least one locus is initially fixed.

Present Investigation. — Herein I present results from a two-locus diploid model of sexual selection. I assume that there are viability differences between members of one sex and, for simplicity, no fertility differences between individuals. This model applies best to a lekking species, in which there are several potential mates, with a simple secondary sexual trait, such as a pheromone, produced by one sex and perceived by the other sex through simple means such as by a pheromone receptor [see, for example, Löfstedt et al. (1989)]. More generally, the model applies whenever one locus determines a trait for which viability selection is sex limited (we will assume this sex to be the male) and the other locus determines female preference. This study contributes to our knowledge of such models by examining the effects of genetic linkage between the two loci. It also looks at the introduction of a new preference scheme in a population polymorphic at the trait locus with interesting results. Both analytical and numerical results are presented.

THE METHOD

The Model and its Assumptions. — The organism in question is a diploid in a population large enough to ignore sampling error (genetic drift). The viability scheme is determined by the trait locus (T/t) such that all females have the same relative viability and all males of the same genotype (TT, Tt, or tt) have the same viability (V_{TT}, V_{Tt}, or V_{tt}). Females choose mates according to "fixed-relative preferences" [O'Donald (1980)]. The relative bias which a particular female has towards the different male genotypes depends on the second locus (P/p) as below:

Male genotype

		TT	Tt	tt
Female genotype	PP	α_0	α_1	α_2
	Pp	β_0	β_1	β_2
	pp	γ_0	γ_1	γ_2

[Given equal numbers of TT, Tt and tt males, the probability that a PP female will mate with a TT male is $\alpha_0/(\alpha_0 + \alpha_1 + \alpha_2)$, etc.] It is assumed that the female does not accrue a selective disadvantage by exercising a mating preference and that the P/p locus does not affect viability or male behavior. Hence each female has the same relative fitness and each male has a fitness that is a function of V_k, α_k, β_k, and γ_k. Note that the fitness of a particular male type changes over time as the genotypic frequencies evolve at the preference locus. Between the trait and preference loci, recombination occurs with a rate, r.

We record the genotypic frequencies following the birth of the offspring. The life cycle then proceeds through viability selection, mate choice, recombination, and new offspring production in that order. There are four possible haplotypes: TP (1), Tp (2), tP (3), and tp (4), but we must keep track of the diploid genotypes since mating is non-random. Denote the genotype frequencies by x_{ij} where i and j refer to the haplotypes inherited from each parent. Note that maternal and paternal haplotypes are not distinguished so that $x_{ij} = x_{ji}$, resulting in a total of 10 distinct genotypes.

As an example of how the recursions are developed, a female of genotype PP will mate with a male of a given trait genotype with the following probability:

Geno- type	Probability of mating
TT	$[V_{TT}\alpha_0(x_{11} + x_{12} + x_{22})]/u$
Tt	$[V_{Tt}\alpha_1(x_{13} + x_{14} + x_{23} + x_{24})]/u$
tt	$[V_{tt}\alpha_2(x_{33} + x_{34} + x_{44})]/u$

where u is a normalizing factor (the sum of the numerators), which ensures that the fertility of a female does not depend on her mating choice. These probabilities reflect viability and mating selection. The full recursions may be obtained from the author.

Stability Analysis: Fixation on TP

Consider first a population near fixation on the alleles T and P. This analysis will allow us to understand the conditions under which a new system of mating preferences and/or a preferred trait allele can increase when both are initially rare. Whether one or both of the rare mutant alleles will increase in a population can be determined by performing a local linear analysis on the recursions that track the change in genotypic frequencies given that TP/TP is the only common genotype. If the largest eigenvalue of this system is greater than one, invasion will occur. For the model at hand, there is always an eigenvalue equal to one and it is the largest eigenvalue unless:

$$\alpha_0 V_{TT} < \alpha_1 V_{Tt} \qquad [1]$$

in which case the largest eigenvalue exceeds unity. Thus a new trait allele will invade when the heterozygote (Tt) has a viability advantage and a relative mating advantage among the resident females (PP) or has an advantage that outweighs its disadvantage. Note also that [1] is simply the condition for initial increase of t when P is fixed and p is not introduced. If [1] fails to hold, the largest eigenvalue is always unity and the system is neutral with respect to the introduction of t and p, essentially because it is neutral to the introduction of p. Since both the new preference and the new trait allele are rare, associations cannot build up between the two loci which could aid in the initial increase of a disadvantageous trait allele. We conclude from this analysis that the fixation of *any* preference allele, including the one associated with random mating, is at best neutrally stable to the introduction of any other preference allele when the trait locus is also near fixation on one allele.

In a population fixed at both loci and undergoing random mating, a new allele that introduces preferential mating will increase only when rare through genetic drift or possibly in conjunction with the increase of a new advantageous trait allele (if the trait allele invades, associations may build up that increase the frequency of the preference

allele). Similarly, in a population in which the common male genotype is also the preferred type, a new more viable—but unattractive—male type can have a lower relative fitness and be lost from the population, a point made by Kirkpatrick (1982).

The results within this section are a special case of the results of Gomulkiewicz and Hastings (1990). They examined the case in which the P allele was not fixed but was instead at an intermediate frequency, as would be the case if genetic drift had swept the initially rare p allele into the population. They find a condition similar to [1], which states that invasion will occur if males of the new trait genotype (Tt) have a higher fitness than the common males (TT) among the current population of females.

Stability of an Overdominant Polymorphism in T/t with Fixation on P

Quite different results are obtained if one considers the initial increase of a new preference allele within a population that is already polymorphic for T/t and fixed at the preference locus. Since all females are initially PP, the fitness of a male in this population is simply the product of a female's preference for him and his viability:

Males

Geno-type	TT	Tt	tt
Fitness	$\alpha_0 V_{TT}$	$\alpha_1 V_{Tt}$	$\alpha_2 V_{tt}$
	$= W_{TT}(PP)$	$= W_{Tt}(PP)$	$= W_{tt}(PP)$

To maintain a polymorphism we assume that the heterozygotes have the highest fitness:

$$W_{TT}(PP) < W_{Tt}(PP) > W_{tt}(PP) \quad [2].$$

In this case, the following overdominant equilibrium is stable in a population of PP individuals:

$$x_{11} = \text{frequency}(TP\ TP)$$

$$= [W_{Tt}(PP) - W_{tt}(PP)]^2$$

$$\div [2W_{Tt}(PP) - W_{TT}(PP)$$

$$- W_{tt}(PP)]^2$$

$$x_{13} = \text{frequency}(TP\ tP)$$

$$= 2[W_{Tt}(PP) - W_{tt}(PP)]$$

$$\times [W_{Tt}(PP) - W_{TT}(PP)]$$

$$\div [2W_{Tt}(PP) - W_{TT}(PP)$$

$$- W_{tt}(PP)]^2$$

$$x_{33} = \text{frequency}(tP\ tP)$$

$$= [W_{Tt}(PP) - W_{TT}(PP)]^2$$

$$\div [2W_{Tt}(PP) - W_{TT}(PP)$$

$$- W_{tt}(PP)]^2 \quad [3].$$

Note that heterozygote advantage may result from overdominance in the viability or mating schemes or through the opposing action of viability and mating selection. For example, Semler (1971) studied threespine sticklebacks (*Gasterosteus aculeatus* L.) in which a balanced polymorphism maintains both red and nonred male breeding types presumably through the interaction of natural selection (predation by trout) against the red type and sexual selection favoring the red type [see O'Donald (1973) for a theoretical discussion of this polymorphism]. The condition for initial increase of p in a population that is originally polymorphic is more complex than the previous analysis near fixation on the TP haplotype and depends on the recombination fraction, r. The local linear analysis is presented in Appendix 1 along with the invasion criteria. Other than the limiting case discussed in Appendix 1, it was found that the leading eigenvalue is *always* greater than one (p will *always* invade) for $r < r^*$ where

$$r^* = \frac{[W_{Tt}(PP) - W_{tt}(PP)][W_{Tt}(PP) - W_{TT}(PP)]}{2W_{Tt}(PP)[2W_{Tt}(PP) - W_{TT}(PP) - W_{tt}(PP)]}.$$

This cutoff point depends only on the fitnesses $[W_i(PP)]$ before the introduction of p. The condition of heterosis [2] guarantees that r^* will be positive and below 0.25. For $r > r^*$, fixation on P is stable (all eigenvalues are less than unity): a loosely linked preference allele will never increase when rare from an overdominant equilibrium. If the V_i are chosen at random (uniformly) between 0 and 1, the α_i are chosen uniformly between 0 and 10, and r^* is calculated whenever there is heterozygote advantage, the

average value of r^* equals 0.14 (based on 100,000 samples), with r^* being greater than 0.2 in 22.7% of the samples but never greater than 0.25, as expected. Basically, r^* decreases as the fitness of the heterozygote approaches the fitness of one or the other homozygote. This indicates that the driving force for invasion is the advantage of the heterozygote; the smaller the advantage, the more restrictive are the conditions under which invasion will occur.

The fact that invasion depends on the recombination rate alone and not on the new mating preference scheme seems at first counterintuitive. While it must be true that invasion occurs through the maintenance of disequilibrium with sufficiently low recombination rates, the manner by which this works is not at first obvious. In fact, without a full analysis of the invasion criterion and an understanding of the development of disequilibria, one may be easily led to the wrong conclusions [see Koeslag (1990)]. In the following section, I describe how invasion occurs in both mathematical and intuitive terms.

Disequilibrium Analysis. — To clarify the role of disequilibrium, let us look at the changes in genetic associations that occur during the initial increase of the new (p) allele at an overdominant polymorphism. The analysis is presented in Appendix 2. There we show that, at $r = 0$, a positive association will always develop between the Tt genotype and one of the two rare haplotypes (Tp or tp). This positive association between the fittest genotype and a rare haplotype drives the initial increase of the rare allele.

As an example, consider the case in which the rare female (Pp) has a greater preference for TT males relative to the common females (PP). In this case, a Pp female will carry either the Tp haplotype or the tp haplotype and she will more likely mate with a TT male. If such a mating occurs, then the rare haplotype that she carried will be more often paired with a T allele in her children. If she carried the Tp chromosome, then this pairing leads to a positive association between the Tp haplotype and the homozygote (TT). If she carried the tp chromosome, then the increased preference for TT males would create a positive association between the tp

haplotype and the heterozygote (Tt). Since the heterozygote has the highest fitness in the original population, the tp haplotype will be carried by individuals who are more fit on average and so this haplotype will increase when rare. Initially, then, one haplotype decreases when rare while the other increases. At $r = 0$, one haplotype will invade and the other will not, but the invasion of one haplotype means that the p allele will also invade. Depending on the starting position, there may even be a slight initial decrease in the frequency of p reflecting the fact that the Tp haplotype is disappearing from the population, but eventually most of the p alleles will be in tp haplotypes that are increasing in frequency by the above argument. As long as there is enough linkage, the increase of one haplotype is sufficient to guarantee the invasion of the new allele. The rather surprising fact is that one of the haplotypes always develops a positive association with the Tt genotype, regardless of the new preference scheme, even if, as in this example, Pp females were to prefer Tt males less!

It is also worth having an "intuitive" understanding of why such a positive association develops when the Pp females prefer heterozygous males. I am grateful for the following heuristic supplied by an anonymous reviewer. Assume that TT males are more viable than tt males in the original population and for simplicity assume that mating is initially random. Also assume that the new females (Pp) greatly prefer heterozygous males over homozygous males ($\beta_1 \gg \beta_0 = \beta_2$). A Pp mother carrying the Tp haplotype will generally mate with a Tt male and so produce an equal mixture of TT and Tt genotypes among her offspring that receive the Tp haplotype. In contrast, a PP mother who passes the TP haplotype on to her offspring will produce homozygous TT offspring with a frequency equal to the original frequency of the T allele:

frequency (T) =

$$\frac{[W_{Tt}(PP) - W_{tt}(PP)]}{[2W_{Tt}(PP) - W_{TT}(PP) - W_{tt}(PP)]} \quad [4]$$

and Tt offspring the rest of the time. Since the TT genotype is by assumption more fit than the tt genotype, frequency (T) is greater

than 1/2. Hence the TP haplotype will be found in more TT offspring than Tt offspring whereas the Tp haplotype will be equally distributed between these two genotypes. A positive association will in this manner develop between the Tp haplotype and the heterozygous (Tt) genotype and this gives the Tp haplotype a fitness advantage. The opposite scenario applies to the new tp haplotype. We thus predict that for tight enough linkage the p allele will invade through the fitness advantage of the Tp haplotype. By similar intuitive arguments, one can in time convince oneself that one of the two new haplotypes will always become positively associated with the most fit genotype (Tt) and will thus rise in frequency.

To understand the role of recombination, first realize that recombination will only alter the gamete distribution of doubly heterozygous parents. With the first example described above, the following positive associations develop at $r = 0$:

Tp haplotype and TT genotype
tp haplotype and Tt genotype.

Hence double heterozygotes will tend to be TP/tp individuals, i.e., double heterozygotes tend to carry the favored haplotype. When recombination occurs in such a double heterozygote, there will be one fewer tp haplotype (and hence fewer $TPtp$ children) and one more Tp haplotype (and hence more $TPTp$ children). This increases the probability that the p allele will be carried by a homozygote (TT), decreases the probability that it will be carried by a heterozygote (Tt), and hence decreases the tendency for the p allele to invade. Generally, as recombination is increased, the positive association between one haplotype and the heterozygote decreases in magnitude while the positive association between the other haplotype and a homozygote increases in magnitude until these associations balance each other (at r^*), after which point invasion can no longer occur.

Characterizing an Internal Polymorphism—a Special Case

Besides understanding evolutionary trajectories near fixation points, we would also like to identify equilibria towards which such a system may go. Unfortunately most equilibria of this system cannot be found explicitly. Initial simulations of the model indicated that the following equilibria exist and can be stable at $r = 0$:

$x_{11} + x_{14} + x_{44} = 1$(only the TP and tp haplotypes are present)

$x_{22} + x_{23} + x_{33} = 1$(only the Tp and tP haplotypes are present).

In fact, with 100 different parameter sets started from the overdominant polymorphism described in the previous section, 65 converged upon one of these equilibria at $r = 0$. These are usually called high-complementarity equilibria (HCE) [Franklin and Lewontin (1970)]; clearly these equilibria are possible only in the absence of recombination. I was able to characterize a high-complementarity equilibrium in the case of a homozygous (tt) lethal trait and a dominant preference allele (p):

	Males		
Genotype	TT	Tt	tt
Viability	V_{TT}	V_{Tt}	0

		Male genotype		
		TT	Tt	tt
Female genotype	PP	α_0	α_1	α_2
	Pp	β_0	β_1	β_2
	pp	β_0	β_1	β_2

Under these conditions, the following HCE can be found analytically:

$$x^*_{22} = \beta_1(V_{Tt})(2\beta_1 V_{Tt} - \beta_0 V_{TT})$$
$$\times (\beta_1\alpha_1 V_{Tt} + \beta_1\alpha_0 V_{TT}$$
$$- \beta_0\alpha_1 V_{TT})/\text{Total}$$

$$x^*_{23} = 2(\beta_1 V_{Tt} - \beta_0 V_{TT})$$
$$\times (2\beta_1 V_{Tt} - \beta_0 V_{TT})$$
$$\times (\beta_1\alpha_1 V_{Tt} + \beta_1\alpha_0 V_{TT}$$
$$- \beta_0\alpha_1 V_{TT})/\text{Total}$$

$$x^*_{33} = (\beta_1 V_{Tt} - \beta_0 V_{TT})^2$$
$$\times (2\beta_1\alpha_1 V_{Tt} + \beta_1\alpha_0 V_{TT}$$
$$- 2\beta_0\alpha_1 V_{TT})/\text{Total}$$

where dividing by the total ensures that these three frequencies will sum to one. The complementary HCE involving the TP and tp haplotypes may be obtained by simply renaming the alleles at the P locus. All of the above three frequencies are positive when

$$\beta_1 V_{Tt} > \beta_0 V_{TT}.$$

A local stability analysis near this high complementarity equilibrium reveals that the polymorphism is locally stable whenever

$$\alpha_0 \beta_1 > \alpha_1 \beta_0.$$

This case is particularly interesting because it demonstrates that once the frequency of p is sufficiently high, sexual selection may maintain a homozygous lethal allele in a population under the simple condition that the heterozygote (Tt) has a mating advantage among Pp females, which is greater than its mating advantage among PP females ($\beta_1/\beta_0 > \alpha_1/\alpha_0$) and that this mating advantage outweighs whatever viability disadvantage the Tt genotype has relative to the TT homozygote ($\beta_1/\beta_0 > V_{TT}/V_{Tt}$).

NUMERICAL RESULTS

An extension of Heisler and Curtsinger's numerical analyses to include linkage reveals qualitatively different behavior and equilibria for a wide range of parameter values. One hundred ten viability vectors and preference matrices were chosen at random, with viability being uniformly distributed between 0 and 1 and preferences between 0 and 10. For each parameter set, 10 starting frequency vectors were randomly chosen. The recursions were iterated until the largest frequency change observed over a generation was less than 10^{-14}, at which point the population was said to be at equilibrium, or until a maximum of 100,000 generations had passed. Local stability of an equilibrium was confirmed by perturbing the frequencies slightly away from that equilibrium and reiterating. Recombination rates (r) were set at 0, 0.001, 0.2, and 0.5. Following Heisler and Curtsinger, I define:

$$W_{TT}(PP) = \alpha_0 V_{TT} \qquad W_{TT}(pp) = \gamma_0 V_{TT}$$

$$W_{Tt}(PP) = \alpha_1 V_{Tt} \qquad W_{Tt}(pp) = \gamma_1 V_{Tt}$$

$$W_{tt}(PP) = \alpha_2 V_{tt} \qquad W_{tt}(pp) = \gamma_2 V_{tt}$$

These values determine the fitnesses for TT, Tt, and tt males when the population is fixed on P in the first column and p in the second. I also define

$$W_{TT}(Pp) = \beta_0 V_{TT}$$

$$W_{Tt}(Pp) = \beta_1 V_{Tt}$$

$$W_{tt}(Pp) = \beta_2 V_{tt}$$

as the fitnesses of TT, Tt, and tt males in a hypothetical population composed solely of Pp females. The $W_i(Pp)$ give important information about possible equilibria. For instance, assume directional selection exists such that TT individuals are the most fit when within groups of either PP or pp females. In this case, a polymorphism is possible only if TT males are not the most fit in the presence of Pp females as well, because, if they were, the TT males would always have the highest fitness, irrespective of the composition of females in the population.

By considering the ordering of these fitness values, we can better understand the coevolution of traits and preferences. While there are 216 (6 × 6 × 6) orderings of $W_i(PP)$, $W_i(Pp)$, and $W_i(pp)$, many are made equivalent by simply changing the name given to an allele. These equivalence classes are shown in Table 1. In Appendix 3, I present the results in a manner that illustrates how an increase in the recombination rate changes the equilibrium towards which a certain population is attracted.

To emphasize some of the more interesting findings, only in 60 out of 110 parameter sets (cases) was fixation the only behavior observed (on T or t). As expected, when the T or t allele fixed, the equilibrium frequency of P depended on the starting conditions. Besides fixation, the most commonly observed behavior for complete linkage ($r = 0$) was the high complementarity equilibrium (HCE) that was found to exist and be stable in almost half of the cases (42/110). The only common criterion for the existence of the HCE was that the heterozygote (Tt) have the highest fitness among at least one genotypic group of females (PP, Pp or pp). As linkage was loosened to $r = 0.001$, almost all (37/42) of the HCE gave rise to a fully polymorphic, stable equilib-

TABLE 1. Classification system used to denote the direction of selection within different sectors of the female population.[a]

		Fitness ordering among pp females [$W_i(pp)$]					
Highest		TT	TT	Tt	Tt	tt	tt
Intermediate		Tt	tt	TT	tt	TT	Tt
Lowest		tt	Tt	tt	TT	Tt	TT
Class		d_1	u_1	o_1	o_2	u_2	d_2
Ordering (among PP females)	Class						
$TT > Tt > tt$	d_1	d_1,m,d_1*	d_1,m,u_1*	d_1,m,o_1*	d_1,m,o_2*	d_1,m,u_2*	d_1,m,d_2*
$TT > tt > Tt$	u_1	u_1,m,d_1	u_1,m,u_1*	u_1,m,o_1*	u_1,m,o_2*	u_1,m,u_2*	u_1,m,d_2
$Tt > TT > tt$	o_1	o_1,m,d_1	o_1,m,u_1	o_1,m,o_1*	o_1,m,o_2*	o_1,m,u_2	o_1,m,d_2
$Tt > tt > TT$	o_2	o_2,m,d_1	o_2,m,u_1	o_2,m,o_1	o_2,m,o_2	o_2,m,u_2	o_2,m,d_2
$tt > TT > Tt$	u_2	u_2,m,d_1	u_2,m,u_1	u_2,m,o_1	u_2,m,o_2	u_2,m,u_2	u_2,m,d_2
$tt > Tt > TT$	d_2	d_2,m,d_1	d_2,m,u_1	d_2,m,o_1	d_2,m,o_2	d_2,m,u_2	d_2,m,d_2

[a] The classification system is based on whether the fitness ordering is directional (d), underdominant (u), or overdominant (o). In the table, "m" refers to the fitness ordering among Pp females. There are six possible fitness orderings each for $W_i(PP)$, $W_i(Pp)$, and $W_i(pp)$, creating a total of 216 classes. However, certain classes are made equivalent simply by changing the label given to a particular allele. For instance, class (d_2,m,d_2) is equivalent to class (d_1,m,d_1); both have directional selection but for the opposite alleles. Similarly the class (o_2,m,u_1) is equivalent to the class (u_1,m,o_2) by switching P for p. Hence all classes can be converted to one of the asterisked (*) classes simply by changing the name given to one or both alleles.

rium point (the remaining 5 did not reach equilibrium within 100,000 generations). Such a polymorphism is expected sufficiently close to $r = 0$ by the small parameter theory [Karlin (1972)]. A further increase in recombination led to three main classes of behavior. The first, observed in 11 of the 42 cases, was an internal equilibrium point in linkage equilibrium ($D = x_1x_4 - x_2x_3 = 0$), which became stable at some value of recombination (r_0) and remained stable for all larger rates of recombination ($r > r_0$). In 15 of the 42 cases, the HCE gave rise to points slowly evolving along an internal curve (marked by dashed lines in Appendix 3) such that equilibrium was not reached within 100,000 generations even for completely unlinked genes ($r = 0.5$). With their parametric restrictions, Heisler and Curtsinger observed several of these quasi neutral curves, movement along which eventually terminated in fixation on P or p. Finally, in 16 of the 42 cases, increasing recombination eventually led to fixation on p (or P) with an overdominant polymorphism at the T locus, which was stable, as predicted above, for all r greater than $r*$.

Clearly, 10 starting points for each parameter set does not completely describe the complexity of the system. A more thorough analysis with 100 starting points demonstrated that rare but not unexpected behavior does occur as shown in Table 2. With a little thought, it becomes clear that certain equilibria should exist and be stable under

certain fitness orderings as described in Table 3. However, in the simulations, not all of these predictions were borne out as a consequence of the fact that the domain of attraction to such points need not be very large and that only ten trials per parameter set were run.

The role of recombination in the simulations is revealing. Let D denote the degree of linkage disequilibrium ($D = x_1x_4 - x_2x_3$). So far my results show that if $D = 0$ for some value of the recombination rate (r_D) then linkage equilibrium holds for all larger values of r ($r > r_D$). This is a general result for models in which the two double heterozygotes have the same fitness [Karlin and Liberman (1979); Christiansen (1990)]. Finally, it appears to be a general rule that increasing recombination decreases the possibility of stable polymorphisms.

TABLE 2. Rare but expected behavior is observed at equilibrium.

Fitness ordering[a]	Common behavior	Frequency	Uncommon behavior	Frequency
$u_1u_2u_2$[b]	Fixation on T	99/100	Fixation on t	1/100
$d_1d_1o_2$[c]	Fixation on T	99/100	HCE at $r = 0$ gives rise to fixation on p for larger r.	1/100

[a] See Table 1 for an explanation of the fitness orderings.

[b] The Tt heterozygote is never favored (underdominance) so that both fixation on T and t are stable. The domain of attraction to fixation on t is, however, smaller than that on T as indicated by the simulations.

[c] Both fixation on T and fixation on p (with T and t maintained in an overdominant polymorphism) are stable for recombination rates greater than $r*$ but again the domain of attraction to fixation on p is smaller.

TABLE 3. Equilibrium behavior may, in part, be deduced from the fitness orderings.

Fitness Ordering[a]	Stable equilibrium	
$d_1-, -d_1-, -d_1$	Fixation on T	(Directional selection)
$d_2-, -d_2-, -d_2$	Fixation on t	(Directional selection)
$u_1-, -u_1-, -u_1$	Fixation on T or t	(Underdominant selection)
$u_2-, -u_2-, -u_2$	Fixation on T or t	(Underdominant selection)
o_1-, o_2-	Overdominant polymorphism with P fixed (stable when $r > r^*$)	
$-o_1, -o_2$	Overdominant polymorphism with p fixed (stable when $r > r^*$)	

[a] See Table 1 for an explanation of the fitness orderings.

DISCUSSION

To explain the existence of secondary sexual traits that lower viability, we must understand how the genes that determine such traits and their mating preferences might initially increase. For general reviews of this subject, see Kirkpatrick (1987) and Pomiankowski (1988). For the specific case of the above two-locus model, preference genes increase when rare under one of the following three conditions. The first is that random genetic drift takes the frequency of the preference allele to a frequency high enough that the assumptions of the local linear analysis no longer apply (second order terms can no longer be ignored) [Kirkpatrick (1982)]. At this point the full recursions must be considered to determine the fate of the population. The above numerical analysis indicates that full polymorphisms are possible, especially when there is some degree of linkage between the loci.

The second scenario corresponds to Fisher's (1958) "runaway process." Here, an advantageous mutation (t) arises that increases when rare ($\alpha_0 V_{TT} < \alpha_1 V_{Tt}$) and a new preference allele (p) has the possibility of increasing as well through a "correlated response." This new preference allele may then give sufficient advantage to another trait mutation that lowers viability so that this disadvantageous mutation can also increase when rare. Using Fisher's example, an initial mutation that increases plumage size and produces a viability advantage through some means increases in frequency along with a preference for larger plumage. A secondary mutation that causes overly large plumage may then increase due to a mating advantage, despite the fact that the now burdensome plumage reduces viability. This "runaway process" would be checked only when the viability selection becomes severe enough to counter the mating selection.

Finally, if the secondary sexual trait is already polymorphic within the population through overdominance, any new preference allele can increase when rare if the trait and preference loci are sufficiently linked. This mechanism provides an alternative explanation for the initial rise in frequency of nonrandom preferences that can then further drive sexual selection.

After invasion, a polymorphic equilibrium may be stable, especially for low recombination rates. For instance, "simulations" that perturb a population away from an overdominant polymorphism indicate that the population dynamics often stabilize at a high-complementarity equilibrium for $r = 0$ and a fully polymorphic equilibrium for $0 < r < r^*$.

Although a two-locus model is strictly applicable to the most simple examples of sexual selection (e.g., stimulus—receptor systems), it does lend insight into the strength of sexual selection and the conditions under which preferences may evolve. Since a mutant preference will not significantly alter the fitness of a mutant male type, the initial increase of a preference allele in the absence of a polymorphism at the trait locus can proceed only by random genetic drift or as a "correlated response" to the increase of a new advantageous mutation. On the other hand, if there is already a polymorphism at the trait locus then a new preference allele can always invade given sufficient linkage between the two loci ($r < r^*$), even if the new preference allele increases the probability of mating with the least viable male. This fact demonstrates that models such as the one studied here can help us tune our intuition about evolutionary processes. For what we imagine to be adaptive and what actually is adaptive may be two very different things indeed.

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APPENDIX 1: *Local Linear Analysis*
(*Near Fixation on P and a Polymorphism in T/t*)

To determine whether or not p will increase when rare, we will examine the stability matrix that keeps track of the frequencies of the four rare genotypes: $TP/Tp(\epsilon_{x_{12}})$, $TP/tp(\epsilon_{x_{14}})$, $Tp/tP(\epsilon_{x_{23}})$, and $tP/tp(\epsilon_{x_{34}})$. The frequencies of Tp/Tp, Tp/tp, and tp/tp are very small $[O(\epsilon^2)]$ and are ignored.

$$\epsilon_{x_{12}}' = \epsilon_{x_{12}}[x_{11}V_{TT}(\alpha_0/u + \beta_0/v)/2$$
$$+ x_{13}(V_{TT}\alpha_0/u + V_{Tt}\beta_1/v)/4]$$
$$+ \epsilon_{x_{14}}[rx_{11}(V_{Tt}\alpha_1/u + V_{TT}\beta_0/v)/2$$
$$+ rx_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4]$$
$$+ \epsilon_{x_{23}}[(1 - r)x_{11}(V_{Tt}\alpha_1/u + V_{TT}\beta_0/v)/2$$
$$+ (1 - r)x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4]$$

$$\epsilon_{x_{14}}' = \epsilon_{x_{14}}[(1 - r)x_{11}(V_{Tt}\alpha_1/u + V_{TT}\beta_0/v)/2$$
$$+ (1 - r)x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4]$$
$$+ \epsilon_{x_{23}}[rx_{11}(V_{Tt}\alpha_1/u + V_{TT}\beta_0/v)/2$$
$$+ rx_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4]$$
$$+ \epsilon_{x_{34}}[x_{11}(V_{tt}\alpha_2/u + V_{TT}\beta_0/v)/2$$
$$+ x_{13}(V_{tt}\alpha_2/u + V_{Tt}\beta_1/v)/4]$$

$$\epsilon_{x_{23}}' = \epsilon_{x_{12}}[x_{13}(V_{Tt}\beta_1/v + V_{TT}\alpha_0/u)/4$$
$$+ x_{33}(V_{tt}\beta_2/v + V_{TT}\alpha_0/u)/2]$$
$$+ \epsilon_{x_{14}}[rx_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4$$
$$+ rx_{33}(V_{tt}\beta_2/v + V_{Tt}\alpha_1/u)/2]$$
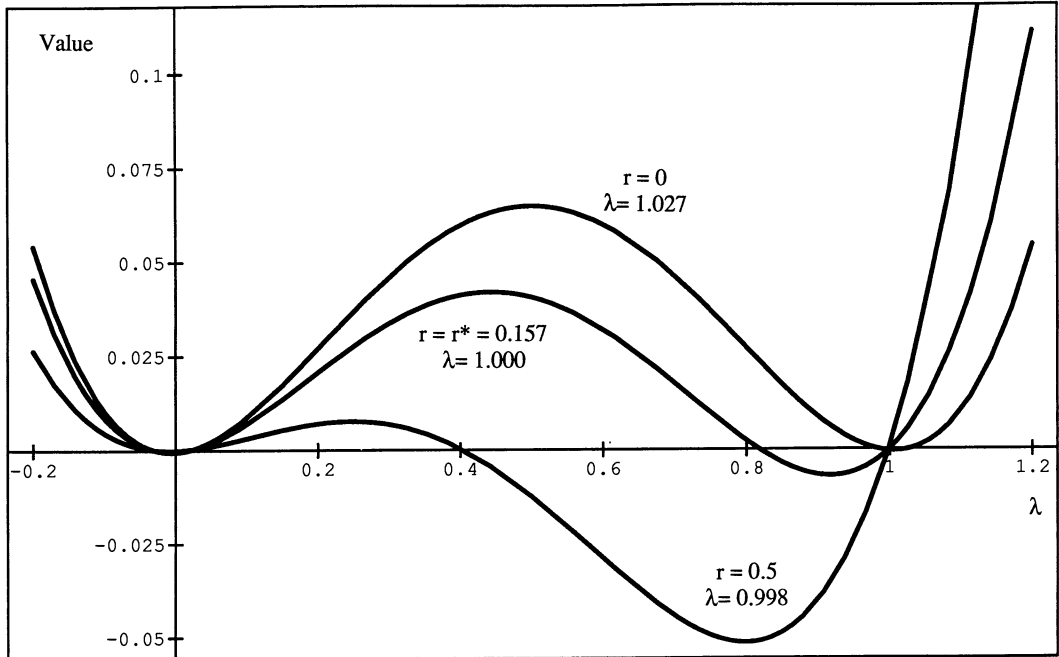$$+ \epsilon_{x_{23}}[(1 - r)x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4$$

FIG. 1. An illustration of the characteristic polynomial and its dependence on the rate of recombination, using the following parameters:

Male genotype:	Tt	Tt	tt
Viability (V_i):	1	2/3	1/3
Preference among			
PP females (α_i):	1	10	10
Fitness among			
PP females			
[$W_i(PP)$]:	1	20/3	10/3 ($\rightarrow$ overdominance)

Preference among
Pp females (β_i): 1 4 10
Fitness among
Pp females   ($\rightarrow$ directional
[$W_i(PP)$]:   1 8/3 10/3 selection)

which gives $r* = 0.157$. The three curves are for $r = 0$, $r = r*$, and $r = 0.5$. Notice that as recombination increases, the largest root decreases from $\lambda = 1.027$ to 1.000 to 0.998.

$$+ (1 - r)x_{33}(V_{tt}\beta_2/v + V_{Tt}\alpha_1/u)/2]$$

$$\epsilon_{x34}' = \epsilon_{x14}[(1 - r)x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4$$

$$+ (1 - r)x_{33}(V_{tt}\beta_2/v + V_{Tt}\alpha_1/u)/2]$$

$$+ \epsilon_{x23}[rx_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4$$

$$+ rx_{33}(V_{tt}\beta_2/v + V_{Tt}\alpha_1/u)/2]$$

$$+ \epsilon_{x34}[x_{13}(V_{Tt}\beta_1/v + V_{tt}\alpha_2/u)/4$$

$$+ x_{33}V_{tt}(\beta_2/v + \alpha_2/u)/2]$$

where

$$u \approx x_{11}V_{TT}\alpha_0 + x_{13}V_{Tt}\alpha_1 + x_{33}V_{tt}\alpha_2$$
$$v \approx x_{11}V_{TT}\beta_0 + x_{13}V_{Tt}\beta_1 + x_{33}V_{tt}\beta_2$$

and the frequencies (x_{11}, x_{13}, and x_{33}) are given by [3] in the text. The characteristic polynomial of the above stability matrix is a quartic function of α_i, β_i, V_i, and r. As an illustration, the shape of the characteristic polynomial is shown for various values of r in Figure 1.

When $r = 0$, the characteristic polynomial splits into two quadratics. The slope of these quadratics evaluated at $\lambda = 0$ is negative and at $\lambda = 1$ is positive. At $\lambda = 1$,

the value of one quadratic has the same sign as [A1] while the value for the other quadratic is opposite in sign to [A1], where [A1] equals:

$$\frac{[W_{Tt}(Pp) - W_{tt}(Pp)]}{[2W_{Tt}(Pp) - W_{TT}(Pp) - W_{tt}(Pp)]}$$

$$- \frac{[W_{Tt}(PP) - W_{tt}(PP)]}{[2W_{Tt}(PP) - W_{TT}(PP) - W_{tt}(PP)]}. \quad [A1]$$

Hence, one of the two quadratics has a leading eigenvalue greater than one and invasion will occur. In the limiting case when [A1] equals zero, the largest root of both quadratics equals unity and selection is expected to be neutral with respect to the new allele. Such is the case when the new allele does not change the expected frequency of the T allele in the population (I am indebted to Mark Kirkpatrick for pointing out this special case). We conclude that, except for some limiting cases, every new preference allele is able to invade when there is complete linkage.

When $r = 0.5$, the characteristic polynomial splits into a cubic and $\lambda = 0$. It can be shown that the leading term of the cubic is positive and the inflection point is between 0 and 1. At $\lambda = 1$, the polynomial is positive

with a positive slope. Hence the largest eigenvalue is strictly less than unity. No new preference allele can invade.

As claimed above, the value of the characteristic polynomial at $\lambda = 1$ is negative when $r = 0$ (unstable) and positive when $r = 0.5$ (stable). In fact, the value of the characteristic polynomial is linear in r and increases with increasing r at $\lambda = 1$. This demonstrates the existence of a value, r^*, below which the p allele will invade and above which fixation in P is stable. The value of r^* was determined by solving the characteristic polynomial for r when the largest root equalled one and found to be:

$$r^* = \frac{(V_{Tt}\alpha_1 - V_{tt}\alpha_2)(V_{Tt}\alpha_1 - V_{TT}\alpha_0)}{2V_{Tt}\alpha_1(2V_{Tt}\alpha_1 - V_{TT}\alpha_0 - V_{tt}\alpha_2)}$$

$$= \frac{[W_{Tt}(PP) - W_{tt}(PP)][W_{Tt}(PP) - W_{TT}(PP)]}{2W_{Tt}(PP)[2W_{Tt}(PP) - W_{TT}(PP) - W_{tt}(PP)]}.$$

APPENDIX 2: *The Development of Disequilibria*

In this appendix, we change the variables of interest from genotype frequencies (as in Appendix 1) to measures of association between the two loci during the initial introduction of the rare (p) allele at an overdominant polymorphism at the trait locus. This technique was introduced by Uyenoyama and Bengtsson (1989) and has proved useful in understanding the role of disequilibria in evolution [see, for instance, Uyenoyama, 1991].

Theory.—Consider a local stability matrix ($\mathbf{M}$) of dimension (n) such as the one considered in Appendix 1. To understand the initial development of disequilibria, we want to transform the basis of analysis from the rare genotype frequencies to a basis composed of the total frequency of the rare allele (p) and appropriately chosen disequilibria ($D_2 \ldots D_n$). These disequilibria measures must be linearly independent and should also be fairly easy to interpret. This basis change is most simply accomplished by performing the following matrix multiplication:

$$\mathbf{N} = \mathbf{AMA}^{-1} \qquad [A2]$$

where $\mathbf{A}$ is an appropriate transformation matrix, examples of which will follow. $\mathbf{N}$ is now the transformed linear stability matrix, i.e., the stability matrix in the new basis. Because $\mathbf{N}$ and $\mathbf{M}$ are "similar" ([A2] is the definition of similarity, see Gantmacher, Vol. 1, p. 68 (1960)), they have the same determinant, the same characteristic polynomial and the same invasion criteria. Hence, whenever the standard stability analysis predicts invasion, a stability analysis in the new basis will also predict invasion.

The disequilibria measured in the new basis may oscillate. In this procedure, we choose initial disequilibria so that they are locally invariant (let $_eD_i = D_i$ when $D_i' = D_i$), i.e., we set the generational change in the disequilibrium to zero in the neighborhood of the original equilibrium. This gives ($n - 1$) equations in (n) variables that are then solved to give the locally invariant disequilibria as functions of the total allele frequency (p). More precisely, we want to find a starting vector, $g = (p, _eD_2, \ldots, _eD_n)$, in the new basis such that the change in g over one generation is given by

$$(\mathbf{I} - \mathbf{N})g = g - \mathbf{N}g = \Delta, \qquad [A3]$$

where $\mathbf{I}$ is an n by n identity matrix and Δ is the column vector ($p - p'$, 0, $\ldots$, 0). To determine g, multiply both sides of [A3] by the inverse of $(\mathbf{I} - \mathbf{N})[(\mathbf{I} - \mathbf{N})^{-1}]$ and use the adjoint method for determining this inverse:

$$(\mathbf{I} - \mathbf{N})^{-1} = \text{adj}[(\mathbf{I} - \mathbf{N})]/\text{Det}(\mathbf{I} - \mathbf{N}), \qquad [A4]$$

where adj denotes the classical adjoint and Det denotes the determinant of a matrix. Combining [A3] and [A4] we get:

$$g = \text{adj}[(\mathbf{I} - \mathbf{N})]\Delta/\text{Det}(\mathbf{I} - \mathbf{N})$$
$$= \text{adj}[(\mathbf{I} - \mathbf{N})]_{.1} (p - p')/\text{Det}(\mathbf{I} - \mathbf{N}).$$

where $\text{adj}[(\mathbf{I} - \mathbf{N})]_{.1}$ is the first column of the adjoint of $(\mathbf{I} - \mathbf{N})$. We can now calculate the following relations:

$$p = \text{adj}[(\mathbf{I} - \mathbf{N})]_{11}(p - p')/\text{Det}(\mathbf{I} - \mathbf{N}) \qquad [A5]$$
$$_eD_i = \text{adj}[(\mathbf{I} - \mathbf{N})]_{i1} (p - p')/\text{Det}(\mathbf{I} - \mathbf{N})$$
$$\text{for } i \neq 1 \qquad [A6]$$
$$= \text{adj}[(\mathbf{I} - \mathbf{N})]_{i1}p/\text{adj}[(\mathbf{I} - \mathbf{N})]_{11},$$

where $\text{adj}[(\mathbf{I} - \mathbf{N})]_{i1}$ is the ith row in the first column of the adjoint of $(\mathbf{I} - \mathbf{N})$. At this point, we have all the elements of the starting vector, g, in terms of the initial allele frequency and the parameters of the model.

Let us assume that all the successive principal submatrices of $(\mathbf{I} - \mathbf{M})$ have positive determinants, where these submatrices are constructed by successively removing the first column and the first row. For a particular problem, this may be true in general or it may be true for weak modifiers of the trait of interest. Under this assumption, stability of the original equilibrium is ensured whenever

$$\text{Det}(\mathbf{I} - \mathbf{M}) > 0 \qquad [A7]$$

[Gantmacher, Vol. 2, p. 74 (1960)]. With positive submatrix determinants, condition [A7] is equivalent to the condition that all eigenvalues be less than unity in magnitude. We know that, for stability, [A7] must hold in the new basis as well [Det($\mathbf{I} - \mathbf{N}$) > 0], because of the similarity of $\mathbf{M}$ and $\mathbf{N}$. Using [A5], this constraint on the determinant of $(\mathbf{I} - \mathbf{N})$ may be rewritten as:

$$(p - p')\text{adj}[(\mathbf{I} - \mathbf{N})]_{11}/p > 0.$$

Note that the initial allele frequency (p) must always be positive. So long as the $\text{adj}[(\mathbf{I} - \mathbf{N})]_{11}$ is positive, we have the desired property that the original equilibrium is stable if the allele frequency decreases and is unstable if it increases. Note that if $\text{adj}[(\mathbf{I} - \mathbf{N})]_{11}$ is negative then invasion can occur despite a local decrease in the new allele frequency, presumably because disequilibrium builds up that will eventually drive the increase of the rare allele frequency. In summary, whenever all the determinants of the principle submatrices of $\mathbf{M}$ are positive and $\text{adj}[(\mathbf{I} - \mathbf{N})]_{11}$ is positive, we can rewrite the stability condition [A7] as:

$$(p - p') > 0,$$

and the criterion for invasion as:

$$(p' - p) > 0. \qquad [A8]$$

This particular method of deriving [A8] was shown to me by Shripad Tuljapurkar, to whom I am grateful.

In an application such as those that follow, [A8] is

a check on the original stability analysis, [A6] determines the signs of the locally invariant disequilibria and hence gives insight into the genetic associations which develop, and, finally, [A3] relates these genetic associations to the increase or decrease of the allele frequency and thus to the stability of the initial equilibrium.

The Case of Complete Linkage. — With the model at hand, when there is no recombination between the two loci ($r = 0$), the recursions of the local linear analysis presented in Appendix 1 can be split into two systems of equations which track the introduction of the two new haplotypes (Tp and tp) separately:

Introduction of Tp (Stability matrix, M_1)

$$\epsilon_{x12}' = \epsilon_{x12}[x_{11}V_{TT}(\alpha_0/u + \beta_0/v)/2$$
$$+ x_{13}(V_{TT}\alpha_0/u + V_{Tt}\beta_1/v)/4]$$
$$+ \epsilon_{x23}[x_{11}(V_{Tt}\alpha_1/u + V_{TT}\beta_0/v)/2$$
$$+ x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4]$$
$$\epsilon_{x23}' = \epsilon_{x12}[x_{13}(V_{Tt}\beta_1/v + V_{TT}\alpha_0/u)/4$$
$$+ x_{33}(V_{tt}\beta_2/v + V_{TT}\alpha_0/u)/2]$$
$$+ \epsilon_{x23}[x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4$$
$$+ x_{33}(V_{tt}\beta_2/v + V_{Tt}\alpha_1/u)/2]$$

Introduction of tp (Stability matrix, M_2)

$$\epsilon_{x14}' = \epsilon_{x14}[x_{11}(V_{Tt}\alpha_1/u + V_{TT}\beta_0/v)/2$$
$$+ x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4]$$
$$+ \epsilon_{x34}[x_{11}(V_{tt}\alpha_2/u + V_{TT}\beta_0/v)/2$$
$$+ x_{13}(V_{tt}\alpha_2/u + V_{Tt}\beta_1/v)/4]$$
$$\epsilon_{x34}' = \epsilon_{x14}[x_{13}V_{Tt}(\alpha_1/u + \beta_1/v)/4$$
$$+ x_{33}(V_{tt}\beta_2/v + V_{Tt}\alpha_1/u)/2]$$
$$+ \epsilon_{x34}[x_{13}(V_{Tt}\beta_1/v + V_{tt}\alpha_2/u)/4$$
$$+ x_{33}(V_{tt}(\beta_2/v + \alpha_2/u)/2]$$

For both 2×2 matrices, M_1 and M_2, we want to change the basis vector from genotype frequencies to a vector whose first element is the total frequency of the new haplotype (ρ_1 for Tp, ρ_2 for tp) and whose last element is an appropriately chosen disequilibrium. As long as the disequilibrium is not simply a multiple of the allele frequency (so long as the new basis elements are linearly independent), any disequilibrium measure could have been chosen but not all would be easy to interpret. A disequilibrium that is fairly easy to interpret in this system tracks the association of a haplotype (Tp or tp, respectively) with the T allele on the complementary chromosome. For the Tp haplotype, then, the new basis is:

$$\rho_1 = \text{frequency}(TP) = \epsilon_{x12} + \epsilon_{x23}$$
$$D_1 = \text{frequency}(TP/Tp)$$
$$- \text{frequency}(TP)\cdot\text{frequency}(Tp)$$
$$= \epsilon_{x12} - \text{frequency}(TP)(\epsilon_{x12} + \epsilon_{x23})$$

For the tp haplotype, the new basis is:

$$\rho_2 = \text{frequency}(tp) = \epsilon_{x14} + \epsilon_{x34}$$
$$D_2 = \text{frequency}(TP/tp)$$
$$- \text{frequency}(TP)\cdot\text{frequency}(tp)$$
$$= \epsilon_{x14} - \text{frequency}(TP)(\epsilon_{x14} + \epsilon_{x34})$$

Let A be the transformation matrix to the new basis. For both M_1 and M_2, the transformation matrix is:

$$A = \begin{pmatrix} 1 & 1 \\ 1 - \text{frequency}(TP) & -\text{frequency}(TP) \end{pmatrix}$$

Basically, the first row of A determines the total frequency of the new haplotype and the second row calculates the disequilibrium relevant to the particular haplotype. The transformation into the new basis is accomplished by performing the matrix multiplication:

$$N_1 = AM_1A^{-1} \qquad N_2 = AM_2A^{-1}$$

Using the technique outlined above, we find that the change in the Tp haplotype frequency, ($\rho_1' - \rho_1$), depends only on $_cD_1$. This follows intuitively from the fact that there is no direct selection on the preference types and any change in haplotype frequency must result from disequilibrium with the trait locus. In fact, the Tp haplotype increases in frequency whenever $_cD_1$ is negative (Tp associated with the Tt genotype) and decreases when $_cD_1$ is positive (Tp associated with the TT genotype) where the sign of $_cD_1$ is the same as the sign of:

$$\frac{[W_{Tt}(Pp) - W_{tt}(Pp)]}{[2W_{Tt}(Pp) - W_{TT}(Pp) - W_{tt}(Pp)]}$$
$$- \frac{[W_{Tt}(PP) - W_{tt}(PP)]}{[2W_{Tt}(PP) - W_{TT}(PP) - W_{tt}(PP)]}. \qquad [A9]$$

Repeating this analysis for N_2, we find that $_cD_2$ equals $_cD_1$, but this time the tp haplotype increases when $_cD_2$ is *positive* (tp associated with Tt) and decreases when $_cD_2$ is *negative* (tp associated with tt).

The above analysis confirms that the increase in allele frequency occurs only as a result of disequilibrium. Specifically, disequilibrium will always develop such that one and only one haplotype becomes positively associated with the Tt genotype. [A9] (also encountered in Appendix 1 as [A1]) is the critical measure that determines which haplotype will have this positive association and hence which haplotype drives the invasion of the new allele. Whenever [A9] ([A1]) equals zero, no associations develop between the new haplotypes and the Tt genotype and it is only in this limiting case that invasion will not occur.

With an Arbitrary Rate of Recombination. — For general r, M is the 4×4 matrix given in Appendix 1, which corresponds to an iteration of the general recursions over one generation when the (p) allele is rare. For the new basis, we retain the disequilibria, D_1 and D_2, from above and set:

$$\rho = \text{frequency}(p) = \epsilon_{x12} + \epsilon_{x14} + \epsilon_{x23} + \epsilon_{x34}$$
$$D_3 = \text{frequency}(Tp)$$
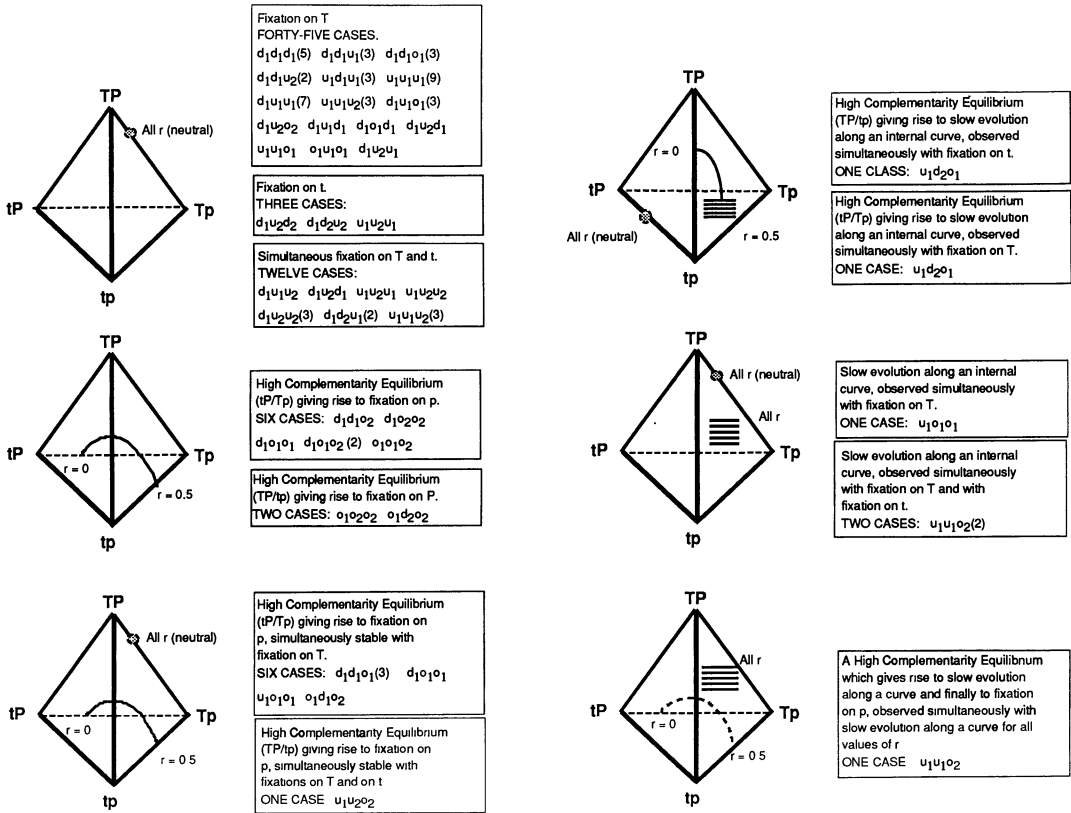$$- \text{frequency}(T)\cdot\text{frequency}(p)$$

FIG. A1. Equilibrium behavior as a function of recombination (see Appendix 3).

$$= (\epsilon_{x_{12}} + \epsilon_{x_{23}})$$
$$- \text{frequency}(T) \cdot (\epsilon_{x_{12}} + \epsilon_{x_{14}} + \epsilon_{x_{23}} + \epsilon_{x_{34}})$$

giving us the transformation matrix $(\mathbf{A})$ shown below: Define $\mathbf{N}$ by $\mathbf{N} = \mathbf{AMA}^{-1}$. The interpretation of $(\rho' - \rho)$ is difficult whenever $\text{adj}[(\mathbf{I} - \mathbf{N})]_{11}$ is negative. All such difficulties disappear in the vicinity of r^*, so I restrict my analysis to recombination rates which are sufficiently close to r^*. From this analysis, we can understand how the disequilibria change with increasing recombination such that, below r^*, invasion occurs but not above r^*. With this restriction we find that the change in the allele frequency $(\rho' - \rho)$ depends on the sign of:

$$-_e D_1 / \text{frequency}(T) + _e D_2 / [1 - \text{frequency}(T)]$$
$$[\text{A}10]$$

where both $_e D_1$ and $_e D_2$ have the same sign as before, i.e., the sign of [A9].

In the vicinity of r^*, increasing the rate of recombination increases $_e D_1$ (increasing the association be- tween the Tp haplotype and the TT homozygote) and decreases $_e D_2$ (increasing the association between the tp haplotype and the tt homozygote). Below r^*, the positive association between the Tt heterozygote and one of the haplotypes (tp if [A9] is positive and Tp if [A9] is negative) dominates [A10], which is therefore positive and invasion occurs. With increasing recombination, the haplotype which is positively associated with the heterozygote becomes less associated with the heterozygote whereas the haplotype that is positively associated with a homozygote becomes more associated with that homozygote. Both of these changes correspond to a lessening of the association between the p allele and the heterozygous (Tt) genotype. This occurs because of the fact that recombination will only affect double heterozygotes (Tt and Pp) and these types carry the 'favored' haplotype more often than expected. Whenever recombination occurs in such a double heterozygote, it will tend to break apart the 'favored' haplotype and create more of the 'disfavored' haplotype. At precisely r^*, the first and last terms of [A10] equal

$$\mathbf{A} = \begin{pmatrix} 1 & 1 & 1 & 1 \\ 1 - \text{frequency}(TP) & 0 & -\text{frequency}(TP) & 0 \\ 0 & 1 - \text{frequency}(TP) & 0 & -\text{frequency}(TP) \\ 1 - \text{frequency}(T) & -\text{frequency}(T) & 1 - \text{frequency}(T) & -\text{frequency}(T) \end{pmatrix}$$

TP — r = 0 (neutral) — tP — r = 0.2 - 0.5 — Tp — tp

Fully polymorphic equilibria (neutral, D=0) giving rise to an internal equilibrium point (D=0). FIVE CASES: $d_1o_1d_1(2)$ $d_1o_1o_1$ $d_1o_2d_1$ $d_1u_2o_1$

TP — r = 0 — tP — r = 0 — Tp — r = 0.2 - 0.5 — tp

High Complementarity Equilibrium (tP/Tp) observed as well as an internal equilibrium point (neutral, D=0) both giving rise to the same internal equilibrium point (D=0). ONE CASE: $o_1u_1o_2$

High Complementarity Equilibrium (TP/tp) observed as well as an internal equilibrium point (neutral, D=0) both giving rise to the same internal equilibrium point (D=0). ONE CASE: $o_1o_2o_1$

TP — tP — r = 0 — Tp — r = 0.2 - 0.5 — tp

High Complementarity Equilibrium (tP/Tp) giving rise to an internal equilibrium point (D=0). THREE CASES: $d_1o_1o_1$ $u_1o_2o_1$ $o_1o_1o_2$

High Complementarity Equilibrium (TP/tp) giving rise to an internal equilibrium point (D=0). THREE CASES: $o_1o_1o_1(2)$ $o_1o_2o_1$

TP — r = 0 — tP — r = 0 — Tp — r = 0.2 - 0.5 — tp

Two simultaneously stable High Complementarity Equilibrium both giving rise to slow evolution along an internal curve. TWO CASES: $o_1d_1o_1$ $o_1o_1o_1$

TP — tP — r = 0 — Tp — r = 0.2 - 0.5 — tp

High Complementarity Equilibrium giving rise to slow evolution along an internal curve. ELEVEN CASES: $d_1d_1o_2$ $d_1o_2o_2$ $d_1o_2d_2$ $u_1o_1o_2$ $u_1o_2o_2$ $o_1o_1o_2(2)$ $o_1o_1o_1$ $o_1o_2o_2$ $o_1u_2o_1$ $o_1d_2o_2$

TP — r = 0 — tP — r = 0 — Tp — r = 0.5 — tp

Two simultaneously stable High Complementarity Equilibria giving rise to an internal equilibrium point (D=0). THREE CASES: $d_1d_2o_2$ $o_1u_1o_1$ $o_1d_2o_2$
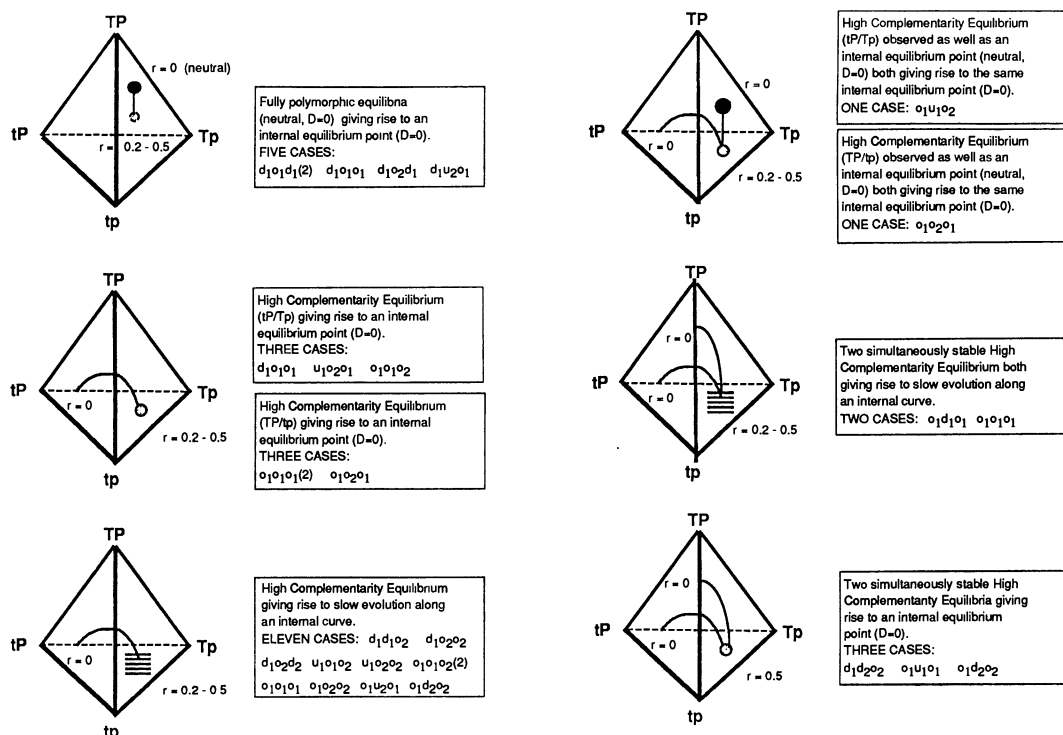
FIG. A2. Equilibrium behavior as a function of recombination (see Appendix 3).

one another in magnitude and there is no resulting change in allele frequency [neutrality; $(\rho' - \rho) = 0$]. Finally, above r^*, the positive association between one new haplotype and the heterozygote is not sufficiently strong to outweigh the positive association between the other new haplotype and the relevant homozygote and the rare allele disappears from the population.

APPENDIX 3: *Numerical Results*

One hundred ten parameter sets were randomly chosen such that preferences were uniformly distributed between 0 and 10 and all viabilities were uniformly distributed between 0 and 1. For each parameter set, initial genotype frequencies were randomly chosen and the dynamics iterated until an equilibrium was reached (all $\Delta_{ij} < 10^{-14}$) or 100,000 generations had passed. The same starting position was used for recombination rates of 0, 0.001, 0.2, and 0.5. Ten such trials were run for each parameter set in order to study the effect of initial position. Figures A1 and A2 pictorially represent the behavior observed at equilibrium in these simulations. Each tetrahedron represents the allele frequency space, with the outside edges corresponding to fixation at one of the loci and the corners corresponding to fixation at both loci. I focus on the change in equilibrium behavior as a function of the recombination rate (r). By drawing lines connecting the equilibria observed for different recombination frequencies, I mean to show only how the behavior changes starting from the same initial genotypic frequencies and do not mean to imply that a particular equilibrium moves along the given line. For instance, the overdominant equilibrium with P (or p) fixed exists for $r < r^*$ when it exists for $r > r^*$ but it is unstable. In the figures, I use the following symbols:

●

A fully polymorphic equilibrium point was reached within 100,000 generations, which was neutral with respect to small perturbations. Linkage disequilibrium $(D = x_1x_4 - x_2x_3)$ was not zero.

○

A fully polymorphic equilibrium point was reached within 100,000 generations which was stable with respect to small perturbations. Linkage disequilibrium $(D = x_1x_4 - x_2x_3)$ did equal zero.

▤

The dynamics did not reach equilibrium after 100,000 generations. The point reached (not an equilibrium) was then perturbed and again the dynamics were iterated. The point reached after this second run of 100,000 generations was different from the first but again was not an equilibrium.