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SEXUAL SELECTION AND THE EVOLUTION OF FEMALE CHOICE

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A paradox for evolutionists has been the development in one sex, usually females, of preferences for mates possessing characters that impair survival. Darwin (1871) extensively documented cases of sexual selection in animals and showed that many of the most dramatic examples are attributable to female mating preferences operating in polygynous populations. But while he was untroubled to explain the evolution of adaptations for male-male sexual competition, Darwin did not provide a hypothesis for the origin or maintenance of female mating preferences.

Fisher (1958, p. 150-153) provided a subtle solution to this puzzle. He reasoned that the initial evolution of a female mating preference would require "bionomic conditions in which such preference shall confer a reproductive advantage." He showed that evolution of the preferred male trait could then "proceed, by reason of the advantage gained in sexual selection, even after it has passed the point in development at which its advantage in Natural Selection has ceased." Because females with stronger preferences mate with males bearing more exaggerated traits, a genetic correlation between the preference and trait is maintained by sexual selection. If the more extreme males are increasing in frequency, the genetic correlation results in the evolution of stronger preferences and so causes further

selection for the extreme males. Fisher dubbed this a "runaway process." O'Donald (1967, 1980) used simulations to verify several essential features of Fisher's argument. He differed with Fisher, though, in his belief that females must respond to supernormal mating stimuli for a male trait to evolve to a point where it decreases viability. O'Donald (1977, 1980) pointed out that a small increase in the male trait might result in a small additional viability loss but a large gain in mating advantage by this mechanism.

The primary conclusion of the present paper is that the initial selective advantages for the female preference assumed by Fisher, O'Donald, and many later authors are not necessary for either the origin or subsequent elaboration of mating preferences for traits associated with reduced survivorship. Using a two-locus analytic model that follows evolution of both the preference and the trait in a polygynous population, I will show that such mating preferences are neither selected for nor against. A sufficiently strong mating preference can, however, maintain in the population a male trait that causes greatly reduced viability. The result can be a dramatic deterioration of the average survivorship, as Fisher and O'Donald concluded, but no particular assumptions about behavioral mechanisms (e.g., response to supernormal mating stimuli) are

necessary for this to happen. Because the frequency of the preference is not determined by the forces of sexual selection but yet is responsible for determining the frequency of the trait, weak forces such as genetic drift or pleiotropy may have a significant role in determining the direction, rate, and limits of evolution of the preference and trait.

These conclusions concur with results from models of sexual selection recently developed by Lande (1980, 1981). Lande's models, based on polygenic inheritance, show that female preferences can evolve simply as a correlated response to changes in the male character and that no positive selective advantage for the preference is required. He also found that the direction and extent of the evolution of sexual selection is highly indeterminate. The model described below demonstrates that these results can also be obtained from much simpler genetic assumptions.

I will examine the evolution of sexual selection in a polygynous population by using a two-locus model in which one locus codes for the preference and the second codes for the trait. The initial evolution of a mating preference and a male secondary sexual trait with reduced viability will be investigated by introducing alleles for these characters into a population lacking them. By finding the equilibria of this system, we will see that the frequency of the male trait is governed by the frequency of the preference, but that the frequency of the preference itself is not selected upwards or downwards. Next, further evolution of the system will be considered by studying the consequences of introducing a second, competing preference for a different male trait. This case includes the interesting situation where one of the mating preferences is for an "optimal" (high viability) male phenotype. Equilibria of this system again show there is no inherent tendency for one mating preference to replace another. This refutes the intuitive notion that selection will necessarily favor mating preferences for male genotypes that are superior under natural selection (e.g., Trivers, 1972; Zahavi, 1975). There are in fact situations where

if a mutant that decreases male viability appears, it will rapidly sweep through the population to fixation despite the presence of a stronger mating preference for a more viable alternative male phenotype. A final consideration will be the stability of the equilibria. Analysis will reveal some aspects about how sexual selection acts when a population is not at equilibrium, and show how rapid evolution might occur.

THE MODEL

The model supposes two major features of the natural history of the organism. First is that the polygynous males are contributing only gametes to the future generation. Second is that there is no direct relationship between a female's mating preference and her survivorship or fecundity. Two assumptions about the genetics are also made: the loci are autosomal and the system is haploid. The latter assumption is strictly for convenience of analysis, and simulations of diploid systems suggest that the qualitative conclusions drawn here still hold.

The two genetic loci of this system are a P locus that determines the female mating preference and a T locus that governs a trait expressed only in males. Each locus segregates for two alleles. The T_1 allele produces males without the secondary sexual characteristic, while the T_2 males bear a trait which reduces their viability to $1 - s$ (where $s > 0$) relative to the T_1 males. The P_1 females mate indiscriminately, while the P_2 females favor mating with T_2 males. The trait-bearing males are more conspicuous or attractive to P_2 females so that these females prefer to mate with them by a factor a_2 relative to T_1 males. (In a two-way choice experiment, a P_2 female would mate with a T_2 male a_2 times more frequently than with a T_1 male.) The P_2 females in the population therefore give a proportion $a_2 t_2' / (t_1' + a_2 t_2')$ of their matings to such males, where t_i' is the frequency of mature T_i males in the population. With P_2 females present a T_2 male mates more frequently than a T_1 male, and so this is a model of selective mating (Lewontin et al., 1968)

TABLE 1. *Frequencies of matings, where the P_1 allele exhibits no mating preference. The frequency of the T_1P_1 zygotes is denoted as x_1 , T_1P_2 as x_2 , T_2P_1 as x_3 , and T_2P_2 as x_4 . The x_i' are the frequencies of these genotypes in adult males following viability selection.*

		MALES				
		T_1P_1	T_1P_2	T_2P_1	T_2P_2	Total
FEMALES	T_1P_1	x_1x_1'	x_1x_2'	x_1x_3'	x_1x_4'	x_1
	T_1P_2	x_2x_1'/z	x_2x_2'/z	$a_2x_2x_3'/z$	$a_2x_2x_4'/z$	x_2
	T_2P_1	x_3x_1'	x_3x_2'	x_3x_3'	x_3x_4'	x_3
	T_2P_2	x_4x_1'/z	x_4x_2'/z	$a_2x_4x_3'/z$	$a_2x_4x_4'/z$	x_4
Total	$x_1'(\hat{p}_1 + \frac{\hat{p}_2}{z})$	$x_2'(\hat{p}_1 + \frac{\hat{p}_2}{z})$	$x_3'(\hat{p}_1 + \frac{a_2\hat{p}_2}{z})$	$x_4'(\hat{p}_1 + \frac{a_2\hat{p}_2}{z})$		
		$z = x_1' + x_2' + a_2x_3' + a_2x_4' = 1 + \frac{(1-s)(a_2-1)t_2}{1-st_2}$				
		$t_2 = x_3 + x_4, \quad \hat{p}_2 = x_2 + x_4$				

in which trait-bearing males receive a frequency dependent advantage.

We will assume that there is no direct selection on female choice by supposing that mating preferences affect only mating behavior and do not alter the survivorship or fecundity of the females. This assumption would be violated if preference alleles had other pleiotropic effects. In species which have male parental care, females mating with ornamented males might suffer decreased fecundity because of those males' decreased ability to contribute to the young. Another possibility is that females with extreme preferences might lose time and energy searching for mates. Any of these three effects would result in direct selection acting on the mating preferences, and would seriously affect the conclusions of the model.

A generation proceeds as follows. Frequencies of the genotypes are identical in the two sexes at birth because the loci are autosomal. Viability selection acts upon the males, and then mating occurs. The rules for mating given above produce the frequencies for the different types of matings shown in Table 1. Recombination between the gametes follows, and the next generation of zygotes is formed.

These steps lead to a set of recursion equations in the four genotype frequencies. These in turn can be translated into recursion equations in the allele frequen-

cies t_2 and \hat{p}_2 , and the linkage disequilibrium D . The linkage disequilibrium is a measure of nonrandom association between alleles at the T and P loci, and is defined as $D = x_1x_4 - x_2x_3$, where the x_i are the genotype frequencies as shown in Table 1. The variables t_2 , \hat{p}_2 , and D give an equivalent description of the evolution of the system but one which is more easily understood than the genotype frequencies. The equations can be written in terms of the changes in t_2 , \hat{p}_2 , and D , as given by equations 1a-c (next page).

The equilibria are found by setting to zero the lefthand side of equations 1 and solving. A surprising conclusion can be immediately deduced from equation 1(b): the frequency of the female mating preference allele changes only as a correlated response to changes in the male trait allele frequency. When there is nonrandom association of the T_2 and P_2 genes ($D \neq 0$), changes in the frequency of T_2 cause changes in the frequency of P_2 . But whenever the male trait is in equilibrium, the mating preference is also in equilibrium.

For any values of the mating preference strength (a_2), selection coefficient (s), and preference allele frequency (\hat{p}_2) there is a corresponding equilibrium frequency of the male trait (t_2). The assumptions of this model produce a set of equilibria which form the curve given by equation 2 (next page).

$$\Delta t_2 = \frac{1}{2}t_2(B - 1) \quad (1a)$$

$$\Delta p_2 = \Delta t_2 \left[\frac{D}{t_2(1 - t_2)} \right] \quad (1b)$$

$$\Delta D = \frac{1}{4}D[A + B + AB - 3] - \frac{1}{4}R[1 + A(1 - t_2) + Bt_2] \quad (1c)$$

where:

$$\begin{aligned} A &= \frac{1}{(1 - st_2)} \left[1 + p_2 \left(\frac{1}{z} - 1 \right) \right] \\ B &= \frac{(1 - s)}{(1 - st_2)} \left[1 + p_2 \left(\frac{a_2}{z} - 1 \right) \right] \\ R &= r \left(\frac{1}{1 - st_2} \right) \left\{ D^2 \left[\frac{1 - a_2(1 - s)}{z} - s \right] \right. \\ &\quad + D \left[1 + ((1 - t_2)(1 - p_2) + t_2 p_2) \left(\frac{1}{z} - s \right) \right. \\ &\quad \left. \left. + \frac{a_2}{z} (1 - s) (p_2(1 - t_2) + (1 - p_2)t_2) \right] \right. \\ &\quad \left. + t_2(1 - t_2)p_2(1 - p_2) \left[\frac{1 - a_2(1 - s)}{z} - s \right] \right\} \\ z &= 1 + \frac{(1 - s)(a_2 - 1)t_2}{1 - st_2}. \end{aligned}$$

This curve is shown as a heavy line in Figure 1. The example illustrated is for a male trait that reduces viability by 40% and a mating preference allele with strength $a_2 = 3$. For the trait allele to have any nonzero equilibrium frequencies it is required that $s < 1 - 1/a_2$, implying that the mating preference be sufficiently strong relative to the selection coefficient.

If this condition is met, there is always a frequency of the preference allele above which the trait allele is fixed.

Because each of the points on this curve is an equilibrium, if a population is displaced from one point on the curve to another point also on the curve, it will remain at this new position. The system is therefore neutrally stable with respect

$$t_2 = \begin{cases} 0 & \hat{p}_2 \leq \frac{s}{(a_2 - 1)(1 - s)} \\ \left[\frac{1}{s} + \frac{1}{a_2(1 - s) - 1} \right] \hat{p}_2 - \frac{1}{a_2(1 - s) - 1} & \frac{s}{(a_2 - 1)(1 - s)} < \hat{p}_2 < \frac{a_2 s}{a_2 - 1} \\ 1 & \frac{a_2 s}{a_2 - 1} \leq \hat{p}_2 \end{cases} \quad (2)$$

to perturbations which move the population along the curve. Once a population is on this curve, no forces arising from the assumptions of this model cause the mating preference (or the male trait) to increase or decrease in frequency. This leaves open the possibility that other factors—even the weak forces of genetic drift, mutation pressure, or pleiotropy—could determine the fate of the preference allele, dragging along with it the frequency of the male trait.

The finding that there is not a single point of equilibrium can be understood intuitively by separating the effects of natural selection and mating success. At any equilibrium it must be that the viability deficit which trait-bearing males suffer is exactly offset by the mating advantage they receive so that the two male phenotypes have identical fitness. The mating advantage is determined by both the strength and the frequency of the preference allele. Increasing the frequency of the preference allele increases the mating advantage. This does not necessarily result in fixation of the male trait allele, however, because the strength of the mating advantage decreases as the frequency of trait-bearing males increases. A new equilibrium can be reached at a higher frequency of both the P_2 preference allele and the T_2 male trait allele.

The equilibrium value of D , the linkage disequilibrium, can be found by noticing that when the gene frequencies are in equilibrium the terms in the first set of square brackets of equation 1(c) equal zero. This implies that $R = 0$, which specifies a quadratic for the equilibrium value of D with the two real roots:

$$\hat{D} = \frac{\hat{p}_2(1 - s\hat{t}_2)}{s} \pm \left\{ \left[\frac{\hat{p}_2(1 - s\hat{t}_2)}{s} \right]^2 - \hat{t}_2(1 - \hat{t}_2)\hat{p}_2(1 - \hat{p}_2) \right\}^{\frac{1}{2}}. \quad (3)$$

It can be shown that the larger root is always infeasible (requiring negative allele frequencies), while the smaller root is al-

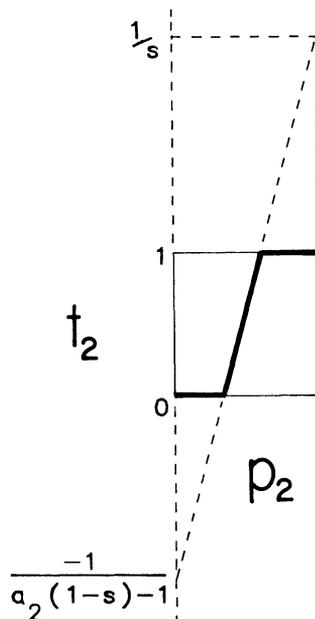


FIG. 1. Equilibria of gene frequencies (heavy curve) for the case in which P_2 females prefer mating with T_2 males, but P_1 females mate randomly. The example shown is for the parameter values $a_2 = 3$, $s = 0.4$. Dotted lines show a geometrical interpretation.

ways feasible and non-negative. Note that the equilibrium is independent of the recombination fraction, r . The value of D equilibrates at a level determined by the nonrandom mating rather than being a balance between selection and recombination as is found in many two-locus selection models (Ewens, 1969).

What if the deleterious trait is not limited to males? Interestingly, this does not prevent evolution of the trait or the mating preference. The equilibrium frequency of the trait is given by replacing \hat{p}_2 with $\hat{p}_2/2$ throughout equation 2. Thus female mating preference for a deleterious character that is not sexually dimorphic can also cause that trait to spread through the population. However, should such a process be initiated, modifiers causing the trait to be expressed only in males would be selected for.

If evolution of the trait and preference continues, a new mating preference might

$$\Delta t_2 = \frac{1}{2}t_2(H - 1) \quad (4a)$$

$$\Delta p_2 = \Delta t_2 \left[\frac{D}{t_2(1 - t_2)} \right] \quad (4b)$$

$$\Delta D = \frac{1}{4}D[G + H + GH - 3] - \frac{1}{4}S[1 + G(1 - t_2) + Ht_2] \quad (4c)$$

where

$$G = \frac{1}{1 - st_2} \left[\frac{a_1(1 - p_2)}{z_1} + \frac{p_2}{z_2} \right]$$

$$H = \frac{1 - s}{1 - st_2} \left[\frac{1 - p_2}{z_1} + \frac{a_2 p_2}{z_2} \right]$$

$$S = r \left(\frac{1}{1 - st_2} \right) \left\{ D^2 \left[\frac{1 - s - a_1}{z_1} - \frac{a_2(1 - s) - 1}{z_2} \right] \right. \\ \left. + D \left[((1 - t_2)(1 - p_2) + t_2 p_2) \left(\frac{1 - s}{z_1} + \frac{1}{z_2} \right) \right. \right. \\ \left. \left. + ((1 - t_2)p_2 + t_2(1 - p_2)) \left(\frac{a_1}{z_1} + \frac{a_2(1 - s)}{z_2} \right) \right] \right. \\ \left. + t_2(1 - t_2)p_2(1 - p_2) \left(\frac{1 - s - a_1}{z_1} - \frac{a_2(1 - s) - 1}{z_2} \right) \right\}$$

$$z_1 = a_1 - \frac{(1 - s)(a_1 - 1)t_2}{1 - st_2} \quad z_2 = 1 + \frac{(1 - s)(a_2 - 1)t_2}{1 - st_2}$$

The equilibria of gene frequencies are again a curve:

$$t_2 = \begin{cases} 0 & \hat{p}_2 \leq \frac{a_1 + s - 1}{(a_1 a_2 - 1)(1 - s)} \\ \frac{(a_1 a_2 - 1)(1 - s)}{(a_1 + s - 1)[a_2(1 - s) - 1]} \hat{p}_2 - \frac{1}{a_2(1 - s) - 1} & \frac{a_1 + s - 1}{(a_1 a_2 - 1)(1 - s)} < \hat{p}_2 < \frac{a_2(a_1 + s - 1)}{(a_1 a_2 - 1)} \\ 1 & \frac{a_2(a_1 + s - 1)}{(a_1 a_2 - 1)} \leq \hat{p}_2. \end{cases} \quad (5)$$

be introduced that would favor a different male phenotype. A particularly interesting case is that where the second preference is for an "optimal" male type favored by natural selection. Will such a preference sweep through the population, restoring the males to an "adapted" state?

Consider a population in which the P_1 females actively prefer to mate with T_1 males by a factor a_1 , just as P_2 females prefer mating with T_2 males by the factor a_2 . This produces a mating matrix somewhat more complex than that shown in

Table 1 because now both females mate nonrandomly. The T_2 males again have a survivorship of $1 - s$ relative to T_1 males.

The evolution of this system is described by equations 4a–c (above). The equilibria of the gene frequencies once again form a curve, given by equation 5 (above). This new equilibria curve is illustrated in Figure 2. Once again, there is no deterministic force which will cause the trait or preference frequencies to increase or decrease once the population is at equilibri-

um somewhere on the curve, but perturbations could move the population up or down the curve. For both trait alleles to be maintained in the population at some preference allele frequencies it is required that $1 - a_1 < s < 1 - 1/a_2$. If this condition is not met, one or the other of the trait alleles will always be fixed. This inequality could play an important role in limiting the evolutionary development of the preference and trait; preference mutations which discriminate strongly enough in favor of the more highly deleterious male traits may be rare.

The equilibrium value of D is given by the smaller of the two roots of the quadratic

$$\hat{D}^2 + I\hat{D} + \hat{t}_2(1 - \hat{t}_2)\hat{p}_2(1 - \hat{p}_2) = 0 \quad (6)$$

where

$$I = \hat{t}_2\hat{p}_2 + (1 - \hat{t}_2)(1 - \hat{p}_2) \frac{\{a_1[a_2(1 - s) + 1] + \hat{t}_2[a_2(1 - s)^2 - a_1]\}}{(1 - s)(a_1a_2 - 1)}.$$

(Note that all earlier results of the single preference system can be recovered as a special case by setting $a_1 = 1$.)

The implication of the neutral stability is that once the population lands somewhere on the equilibria curve there will be no deterministic force which will favor the more viable male phenotype. Even if T_1 males are more viable ($s > 0$) and the mating preference for them is stronger than that for the T_2 males ($a_1 > a_2$), there is no guarantee that the frequency of those males or the mating preference for them will increase in frequency. This is the case in the example of Figure 2, where the trait causes a 20% loss of viability. If sexual selection establishes a deleterious male type in a population, the introduction of a new preference for the ancestral or more viable male type will not necessarily restore the population to an "adapted" state. While equilibria of this model show that assumptions about female response to supernormal mating stimuli are not required for a less viable male trait to be maintained in the population, this behavioral

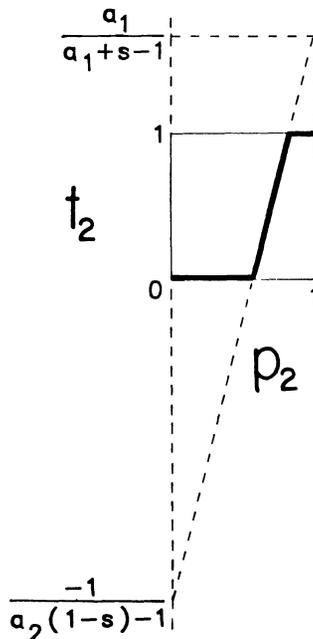


FIG. 2. Equilibria of gene frequencies (heavy curve) when both P_1 and P_2 females have mating preferences. The example shown is for the parameters $a_1 = 2.5$, $a_2 = 2.0$, and $s = 0.2$. Note that T_2 males are both less viable than T_1 males and receive a weaker mating preference from P_2 females than T_1 males do from P_1 females.

mechanism could facilitate the incorporation of still more extreme trait alleles (O'Donald, 1977).

What happens if the population starts far from the equilibria curve? Such a situation is biologically plausible and potentially very important. One can imagine a population in which there was genetic variation for the preference, but no variation at a relevant male trait locus that the preferences could discriminate. A preference allele for a less viable male type that was not yet present in the population could drift to high frequency, as the preference allele would be selectively neutral if females carrying it acted like other females in the absence of their preferred male phenotype. Following a mutation at a trait locus, very rapid evolution can occur. As the new trait allele increases in frequency, the preference allele also increases because of their positive association ($D >$

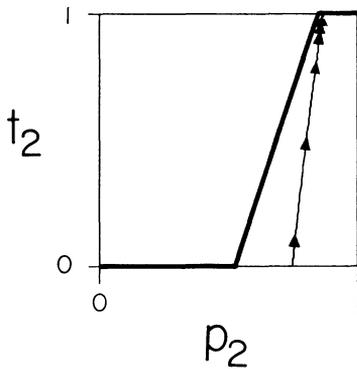


FIG. 3. Time course of sexual selection showing a less viable mutant male type being taken to fixation. Parameters are as in Figure 2: $a_1 = 2.5$, $a_2 = 2.0$, and $s = 0.2$. The initial conditions are $p_2 = 0.7$, $t_2 = 0.01$, and $D = 0$. Arrows are at 25 generation intervals. The heavy curve shows the equilibria. Note that as the T_2 allele increases in frequency it causes an increase in the P_2 frequency as a correlated response (Fisher's runaway process).

0) created by the nonrandom mating. The increases in the preference allele frequency result in further increases in the trait allele frequency. This is what Fisher (1958) described as a runaway process.

Figure 3 shows the runaway process carrying to fixation a male trait that confers a 20% decrease in male viability. In this example the population initially has only T_1 males, but there are both P_1 and P_2 females at the frequencies 0.3 and 0.7 respectively. The preference the P_1 females show for T_1 males is stronger than the preference P_2 females would show for the less viable T_2 males were they in the population: $a_1 = 2.5$ and $a_2 = 2.0$. Despite this, when the T_2 male allele is introduced at low frequency, it rapidly displaces the more viable T_1 male allele. The resulting decline in male viability is graphed in Figure 4. This example underscores the point that sexual selection will not necessarily favor either more viable male traits or mating preferences for them.

A final consideration is the stability of the equilibria curve itself. We have seen that when a population is displaced from one point on the curve to another point

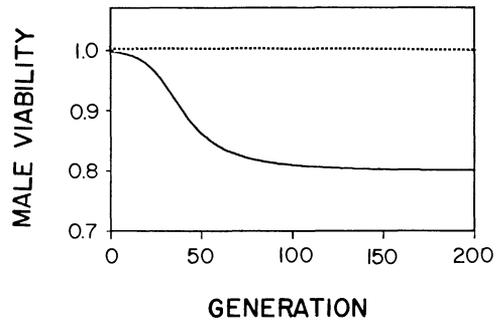


FIG. 4. Decline in the mean male viability following the introduction of the less viable T_2 trait allele for the case shown in Figure 3.

also on the curve, it will remain there (neutral stability). It remains to be shown what happens when a population is displaced to a point off the curve. An analysis of the equilibria described in the Appendix shows that the curve is always locally stable when the recombination rate r is positive. Thus if a population is moved to a point just off the curve, it will return to the curve. In general, however, the population will not return to the same point. This suggests that genetic drift along the equilibria curve might be quite fast, as displacements from equilibrium can result in movement to a new equilibrium not very close to the starting point. Lande (1981), using a very different model of sexual selection, was able to solve for the rate of genetic drift and found that indeed it can be rapid. Lastly, it should be noted that experience iterating equations 1 and 4 indicates that the equilibria curve is also globally stable. Limit cycles have never been observed. Regardless of the initial conditions, if there is variation at the trait and preference loci, the population ultimately ends up somewhere on the curve.

DISCUSSION

The model explored here shows that the advantage which female mating preferences confer on certain male phenotypes can be a powerful agent in evolution. A chance mutant exhibiting sufficiently strong mating preference (satisfying the

appropriate inequality given following equation 5) can, if it reaches high frequency, always take its preferred male phenotype to fixation in the population. This is true even if the male trait is nearly lethal. When the slope of the equilibria curve is steep, small changes in the frequency of the preference will be visible as very abrupt changes in the composition of males in the population.

Because of the neutral stability of the preference allele frequency, forces not specified in the model will control evolution of the system near equilibrium. If the preference allele itself is associated with any direct fitness effects, positive or negative, a relatively rapid change in its frequency and consequently the frequency of the male trait would be expected. For example if males provide parental care or if certain preferences decrease the ability of females to obtain a mate, selection against the preference could result in relatively rapid loss of the preference and its associated male phenotype. Conversely, a force favoring a preference might result from selection to avoid matings with males of the inappropriate species or population, as suggested by Fisher (1958, p. 144–145).

But while these or other considerations may be important in particular cases, the minimal assumptions about the operation of sexual selection, as embodied in the present model, are sufficient to maintain the preference and the trait. In the absence of direct selection pressures on the preference alleles, weaker forces such as genetic drift can determine the evolutionary outcome. One interesting implication of this finding is that there is no stabilizing force intrinsic to sexual selection, so that it may be very easy for allopatric populations to diverge in their secondary sexual characters and hence in their isolating mechanisms, leading to accelerated speciation. This possibility, first suggested by Fisher (1958), has been demonstrated by Lande (1981).

Lande (1980, 1981) has developed models of the evolution of sexual selection based on diploid polygenic inheritance.

He supposed the males to be under normalizing natural selection, and analyzed evolution of the trait and preference under three different assumptions about how females choose mates in sexual selection. Lande found that in all cases there is a neutrally stable line of equilibria relating the male trait mean to the mating preference mean. In light of the disparate genetic assumptions used in his models and the one presented here, the analogy between the conclusions is striking. This strongly implies the major results do not depend critically on the genetical assumptions.

The polygenic and haploid models diverge with respect to the stability of the equilibria. Lande found that if the ratio of the covariance between the two characters and the additive genetic variance of the male trait is sufficiently large, the line of equilibria could itself become unstable, regardless of the linkage pattern. Then any perturbation moving the population off the line would be increasingly magnified through time. This would cause extremely rapid evolution of the trait in an unpredictable direction. In the model presented here, the equilibria can only become unstable in the biologically implausible case where there is no recombination between the loci. The root of this discrepancy lies in the levels of covariance between the trait and the preference that can accumulate in the two types of models. In the present model the upper limit of the covariance D is constrained because only two alleles code for each character. In Lande's models with continuously distributed characters, the covariance arising from the nonrandom mating can become large enough to cause the equilibria to be unstable. It can be shown that in a model hybrid between Lande's and mine based on two haploid loci with continuous, normally distributed allelic effects, the equilibria can become unstable, independent of the recombination fraction r . Thus while the present model leads to an indeterminate outcome because of the neutrally stable equilibria, constraints due to the very simple genetic assumptions add

some degree of stability. Lande's results show that the outcome of sexual selection may be even less predictable than the model presented in this paper suggests.

Although the model studied here assumes a haploid genetic system, it seems that the qualitative conclusions that have been reached also hold for diploid inheritance. It is apparently impossible to treat comparable two-locus, two-allele diploid models analytically because they require following nine (rather than three) simultaneous nonlinear equations. Other analytic models (e.g., O'Donald, 1977; Karlin and Raper, 1979) have therefore assumed static mating preferences and looked in detail at the evolution of the male trait. Diploid models of the simultaneous evolution of the trait and preference can however be iterated by computer to reveal some aspects of their behavior. This approach was used by O'Donald (1967, 1980) to study several models based on autosomal or sex linked loci and various assumptions about dominance. His results are consistent with the model developed here: using different initial conditions but the same parameter values, different equilibria are reached. This implies the existence of a neutrally stable curve of equilibria. I have duplicated some of his simulations and run an additional model based on additive gene effects using rules for mating analogous to those used in the analytic haploid model. The results confirm that the points of equilibria do fall on curves of a form similar to those shown in Figures 1 and 2. O'Donald did not recognize the significance of this pattern and instead concluded, as did Fisher, that "preferences start to evolve only if females prefer those male phenotypes that are advantageous in natural selection" (O'Donald, 1980, p. 197).

It has often been assumed that evolution somehow rescues populations from sexual selection. Mayr (1972, p. 101) wrote that "natural selection will surely come into play as soon as this sexual selection leads to the production of excesses that significantly lower the fitness of the species in interspecific encounters." Genetic models

of the evolution of sexual selection do not confirm this belief. The notion that evolution will necessarily extricate a species from the maladaptive tendencies of sexual selection is unfounded.

SUMMARY

The evolution of sexual selection is studied using a two-locus model of a polygynous population that follows both a male trait causing loss of viability and a female mating preference for that trait. The major conclusion is that such a mating preference is selected neither for nor against, but the mating advantage it confers on its preferred male type can maintain the less viable trait in the population. The equilibrium frequency of the preference is not uniquely determined by the forces of sexual selection, but this frequency determines the prevalence of the male trait in the population. If a mutant showing a mating preference for a male trait with reduced viability reaches high frequency, the trait can be taken to high frequency and consequently cause average male survivorship to deteriorate severely. Because the frequencies of the preference and hence the trait are indeterminate, it is possible that weak forces such as pleiotropy or genetic drift may control the direction and rate of their evolution. These conclusions still hold if a second, competing female mating preference that favors a more viable male type is introduced: neither that preference nor the more viable male trait will necessarily spread through the population.

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APPENDIX

Local stability of a point of equilibrium is shown by examining the eigenvalues of the linearized version of equations 4. Because the system is in three dimensions, the characteristic equation is a cubic. The problem is simplified by considering the least stable set of cases. Since the preference allele changes frequency only by its association with a trait allele that is changing frequency, tightening the linkage between the loci has the effect of destabilizing the model. However because the position of the equilibria curve is independent of r , finding that the curve is stable for the extreme of $r = 0$ implies that it is also stable for the same parameter values but with $r > 0$. When $r = 0$ we find from equations 4 that

$$\left. \frac{\partial \Delta t_2}{\partial D} \right|_{\text{equil.}} = \left. \frac{\partial \Delta p_2}{\partial D} \right|_{\text{equil.}} = \left. \frac{\partial \Delta D}{\partial D} \right|_{\text{equil.}} = 0 \quad (\text{A1})$$

$$\left\{ \begin{array}{l} \frac{1}{2} t_2 \frac{\partial H}{\partial t_2} \\ \frac{1}{2} \frac{\partial H}{\partial t_2} \left[(t_2 p_2 + D) \left(\frac{1}{1 - t_2} \right) - p_2 \left(\frac{t_2}{1 - t_2} \right) \right] \end{array} \right\} \quad \left\{ \begin{array}{l} \frac{1}{2} t_2 \frac{\partial H}{\partial p_2} \\ \frac{1}{2} \frac{\partial H}{\partial p_2} \left[(t_2 p_2 + D) \left(\frac{1}{1 - t_2} \right) - p_2 \left(\frac{t_2}{1 - t_2} \right) \right] \end{array} \right\}. \quad (\text{A2})$$

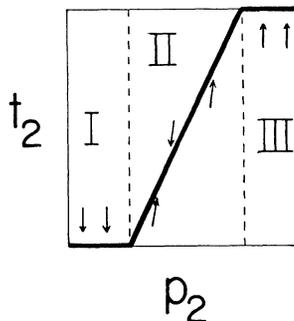


FIG. A1. Schematic of the stability analysis. The heavy curve represents the equilibria. See text.

and the model is neutrally stable in the D dimension as well as along the curve of equilibria. The stability matrix is shown below (expression A2).

One of the remaining roots of the resulting quadratic equation is zero, corresponding to the neutral stability of the equilibria curve. The remaining eigenvalue is

$$\lambda = \frac{1}{2} \left\{ t_2 \frac{\partial H}{\partial t_2} + \frac{\partial H}{\partial p_2} \left[(t_2 p_2 + D) \left(\frac{1}{1 - t_2} \right) - p_2 \left(\frac{t_2}{1 - t_2} \right) \right] \right\}. \quad (\text{A3})$$

A point of equilibrium is locally stable if this eigenvalue lies between -2 and 0 when evaluated at that point.

This eigenvalue was studied numerically along the part of the equilibria curve where $0 < t_2 < 1$, shown as Region II in Figure A1. Combinations of parameters were run over the ranges $1.001 < a_i < 4,096$ and $1 - a_1 < s < 1 - 1/a_2$ (the latter inequalities being required for internal equilibria to exist, as discussed in the text).

On the parts of the equilibria curve where the T locus is fixed, shown as Regions I and III in Figure A1, the eigenvalue is zero. Stability in these Regions can be inferred analytically. In Region I, it can be shown that $\Delta t_2 < 0$ for all values of t_2 , p_2 , and D . Likewise, in Region III $\Delta t_2 > 0$.

We therefore conclude that in all cases where the recombination rate between the two loci is greater than zero, the curve of equilibria is locally stable with respect to perturbations moving the population off the curve. The equilibria are neutrally stable with respect to disturbances that move the population along the curve.

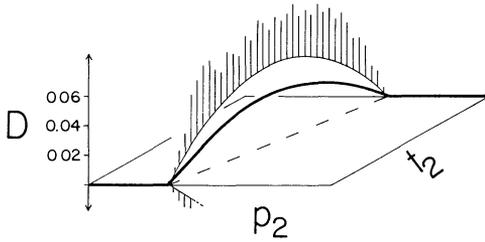


FIG. A2. Equilibria (heavy curve) shown in the three dimensions p_2 , t_2 , and D . When $r = 0$, the model becomes neutrally stable in the D (vertical) dimension as well as along the curve. Shaded regions of the equilibria plane are where numerical evaluation of the eigenvalue (equation A3) indicates the equilibria are unstable. Parameters for this example are as in Figure 1: $a_2 = 3$, $s = 0.4$.

In the biologically unlikely case that there is no recombination, $r = 0$ and the two loci become effectively a single locus with four alleles. Only in this special case is it possible to get unstable behavior of the equilibria. The model now becomes neutrally stable in the D dimension also. This forms a planar surface of equilibria (Fig. A2). There are regions on this surface where the linkage disequilibrium is sufficiently large that the equilibria are locally unstable. If a population starts at such a point, a small perturbation will cause it to move rapidly away from that region to a locally stable point on the equilibria plane.

ANNOUNCEMENT

BELTSVILLE SYMPOSIUM VII

Beltsville Symposium VII, Genetic Engineering: Applications to Agriculture, will be held May 16–19, 1982, at the Beltsville Agricultural Research Center. For further information, contact Dr. Lowell D. Owens, USDA-ARS, Room 116, Building 011A, BARC-West, Beltsville, MD 20705. Telephone (301) 344-4072.