

ON THE EVOLUTION OF RELIABLE INDICATORS OF FITNESS

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Traits often have conflicting effects on different components of fitness. Life-history evolution is replete with examples of traits that increase survival and decrease fecundity, or vice versa, and this fact is recognized in life-history models based on reproductive effort (e.g., Gadgil and Bossert 1970; Schaffer 1974; Michod 1978). Missing from this theory is a consideration of how such traits evolve when there are prior viability differences present in the population that are heritable. This issue is of special relevance to the evolution of communication signals, because there are a variety of reasons why animals should be selected to assay the fitness of individuals they interact with, whether in mating or in combat (Maynard Smith 1982).

Recently, there has been considerable interest in traits that differ in expression depending on the fitness of the individual concerned, since such traits occupy a central position in current controversies concerning the evolution of male displays by sexual selection (Maynard Smith 1985; Kirkpatrick 1986*a,b*; Pomiankowski 1987*a*). Two major competing theories have been proposed to explain the evolution of conspicuous male traits, or displays, that increase a male's mating success but decrease his viability. The first is Fisher's (1958) runaway theory, and the second is the so-called good-genes theory, which originated in one form or another with Trivers (1972) and Zahavi (the handicap principle; Zahavi 1975, 1977). The good-genes view is that male displays provide information about male fitness that is used by females in choosing mates. The work presented below addresses logical and possible historical relations between Fisher's runaway process and the good-genes view.

The model presented below addresses the origin and maintenance of reliable indicators of prior viability differences present in the population. We use the term *viability class* to refer to these prior, or baseline, viability differences, which are assumed to be present in the population before selection acts on the trait of interest. Since the trait of interest in this study is one that has conflicting effects on two multiplicative components of fitness, overall fitness of an individual is a function of both its viability class and the effects on fitness, both positive and negative, of the trait of interest (see, e.g., table 1).

Our motivation in constructing the model came from our desire to understand whether male display could evolve as a reliable indicator of genetic fitness, as postulated by the good-genes view of sexual selection. Male display is one example of a trait that has conflicting effects on two components of fitness, in this case, viability and mating success. We view this problem of evolution of male display as a special case of the more general problem of the evolution of traits that have conflicting effects on fitness in a population in which there are prior heritable differences in viability. Thus, we develop the model and its analysis for the more general setting and apply our results to the evolution of male display as a specific case.

MODEL

GENETIC SYSTEM AND FITNESS

For reasons of mathematical simplicity, we consider a haploid, two-locus genetic model. Let the A locus affect a difference in viability, with the intrinsic viability of A and a individuals being assumed to be 1 and $1 - s$, respectively. Thus, there are two viability classes, A and a , in our model. However, the overall fitness of an individual is a function not only of these prior viability differences but also of the conflicting effects on fitness of the trait of interest. As discussed in more detail below, these effects may vary with viability class.

We assume that genetic variation for viability class is maintained at a single, haploid locus by a force, such as migration or mutation, which, each generation, converts μ A alleles to less fit a alleles, without affecting the statistical association (i.e., linkage disequilibrium) between the alleles at the viability-class locus and alleles at the second modifier locus. This is but one possible representation of the mechanism by which genetic variation in viability class could be maintained.

We assume that the low-viability and high-viability classes are initially alike in their expression of the trait. We then ask, under what conditions does a mutant allele at the modifier locus increase in frequency if it modifies the expression of the trait in one, or both, of the two viability classes?

Let the M locus be a modifier locus that affects a difference in the expression of the trait in one or both of the viability classes. The trait is assumed to have antagonistic effects on two components of fitness that act multiplicatively. For example, it might increase (or decrease) fertility or mating success, and decrease (or increase) viability. Explicit formulations of the effects of the trait are given below.

The two components of fitness, viability, v_i , and fertility, t_i , that are affected by the trait are assumed to interact multiplicatively so that the overall fitness is $v_i t_i$. A simple additive model is considered (table 1A) in which the trait, T , accrues a viability cost per unit of trait, c_A and c_a , in high- and low-viability-class individuals, respectively. The total decrease in viability accrued by the trait is assumed to equal the product of the value of the trait and the cost per unit of trait. The fertility of each genotype is assumed to be directly proportional to the value of its

TABLE 1
LIFE TABLE FOR A MODEL POPULATION

GENOTYPE			
<i>AM</i>	<i>Am</i>	<i>aM</i>	<i>am</i>
A. ADDITIVE MODEL			
$v_1 = 1 - Tc_A$	$v_2 = 1 - T(1 + \alpha)c_A$	$v_3 = 1 - Tc_a - s$	$v_4 = 1 - T(1 + \delta)c_a - s$
$t_1 = T$	$t_2 = T(1 + \alpha)$	$t_3 = T$	$t_4 = T(1 + \delta)$
v_1t_1	v_2t_2	v_3t_3	v_4t_4
B. ADDITIVE SEXUAL-SELECTION MODEL			
$v_1 = 1 - Tc_A$	$v_2 = 1 - T(1 + \alpha)c_A$	$v_3 = 1 - Tc_a - s$	$v_4 = 1 - T(1 + \delta)c_a - s$
$t_1 = T$	$t_2 = T(1 + \alpha)$	$t_3 = T$	$t_4 = T(1 + \delta)$
$w_1 = 1$	$w_2 = 1$	$w_3 = 1 - s$	$w_4 = 1 - s$
1	1	1	1

NOTE.— T is the value of the trait that accrues cost c_A or c_a in individuals with genotypes A and a , respectively, at the fitness locus. The parameters α and δ are the effects of the modifier allele; s is the selective coefficient against the a allele. In the additive model, v_i is viability; t_i , fertility; and v_it_i , fitness. In the additive sexual-selection model, v_i is male viability; t_i , male mating; and w_i , female viability. Female fertility is one for all genotypes.

trait. Genotypes with the new modifier allele, m , have new values for the trait, $T(1 + \alpha)$ and $T(1 + \delta)$, for individuals having high and low fitness, respectively, with concomitant changes in their viabilities and fertilities. Thus, the modifier allele interacts with both components of fitness, viability and fertility.

The values of the trait that maximize individual fitness can be obtained by differentiating fitness, v_it_i , with respect to T in the two unmodified viability classes, $i = 1, 3$. For the additive model given in table 1A, these values are

$$T_A = 1/2c_A \quad \text{and} \quad T_a = (1 - s)/2c_a, \quad (1)$$

for the high- and low-viability classes, respectively. Whether genetic modification allows the two classes to attain the different values of the trait that maximize their individual fitness is a central question in our analysis below.

In the specific case of sexual selection, the trait is male display and thus is only expressed in males. In this case, males are assumed to fit the simple additive model just discussed, whereas females lack the trait and thus experience only selection determined by their viability class (table 1B).

Three kinds of modifiers are distinguished in the following analysis according to the effect of the modifier in the two viability classes considered (table 2). The first kind of modifier has equal effect in both viability classes. In the case of sexual selection, modifiers of equal effect correspond to the "Fisher stage" of the process, in which the effects of prior viability differences are not considered in the evolution of the display. The second kind of modifier affects only one of the viability classes, and the modifier and the viability class are thus in one-to-one correspondence. The third kind of modifier affects both viability classes but in an antagonistic fashion, increasing the trait in one viability class and decreasing it in the other.

TABLE 2
EFFECT OF MODIFIERS

Modifier Type	α	δ
Equal	δ	
One-to-one	$\pm 0.1^*$	0.0
	0.0	± 0.1
Antagonistic	+0.1	-0.1
	-0.1	+0.1

NOTE.— α , Effect on high-fitness class; δ , effect on low-fitness class.

* The value of 0.1 is used as an example only. The value could be any small number measuring the effect of the modifier allele on the trait.

RECURRENCE EQUATIONS

Let $i = 1, 2, 3, 4$ index genotypes $AM, Am, aM,$ and am , respectively. Define $x_{i,t}$ as the frequency of genotype i among newborns in generation t , which is the same for males and females. Let \mathbf{x}_t be the vector of gamete frequencies before viability and fertility selection in generation t :

$$\mathbf{x}_t = [x_{1,t}, x_{2,t}, x_{3,t}, x_{4,t}]$$

For each gamete type i we define a recombination matrix \mathbf{R}_i :

$$\begin{aligned}
 \mathbf{R}_1 &= \begin{bmatrix} 1 & 1/2 & 1/2 & 1/2(1-r) \\ 1/2 & 0 & 1/2 r & 0 \\ 1/2 & 1/2 r & 0 & 0 \\ 1/2(1-r) & 0 & 0 & 0 \end{bmatrix}, \\
 \mathbf{R}_2 &= \begin{bmatrix} 0 & 1/2 & 0 & 1/2 r \\ 1/2 & 1 & 1/2(1-r) & 1/2 \\ 0 & 1/2(1-r) & 0 & 0 \\ 1/2 r & 1/2 & 0 & 0 \end{bmatrix}, \\
 \mathbf{R}_3 &= \begin{bmatrix} 0 & 0 & 1/2 & 1/2 r \\ 0 & 0 & 1/2(1-r) & 0 \\ 1/2 & 1/2(1-r) & 1 & 1/2 \\ 1/2 r & 0 & 1/2 & 0 \end{bmatrix}, \\
 \mathbf{R}_4 &= \begin{bmatrix} 0 & 0 & 0 & 1/2(1-r) \\ 0 & 0 & 1/2 r & 1/2 \\ 0 & 1/2 r & 0 & 1/2 \\ 1/2(1-r) & 1/2 & 1/2 & 1 \end{bmatrix}.
 \end{aligned} \tag{2}$$

The k, l element of the recombination matrix for gamete i is frequency of i gametes produced by a mating between genotypes k and l .

The following two matrices with the fitnesses of females and males (table 1A) on

the diagonal are helpful in calculations:

$$\mathbf{W} = \begin{bmatrix} w_1 & 0 & 0 & 0 \\ 0 & w_2 & 0 & 0 \\ 0 & 0 & w_3 & 0 \\ 0 & 0 & 0 & w_4 \end{bmatrix}, \quad (3)$$

$$\mathbf{Z} = \begin{bmatrix} z_1 & 0 & 0 & 0 \\ 0 & z_2 & 0 & 0 \\ 0 & 0 & z_3 & 0 \\ 0 & 0 & 0 & z_4 \end{bmatrix}.$$

After viability and fertility selection in generation t , but before the force that maintains variation operates, the frequency of genotype i is

$$y_i = \frac{(\mathbf{x}_t \mathbf{Z}) \mathbf{R}_i (\mathbf{x}_t \mathbf{W})^T}{\mathbf{x}_t (\mathbf{1W})^T \mathbf{x}_t (\mathbf{1Z})^T}, \quad (4)$$

with $\mathbf{1} = [1, 1, 1, 1]$. In equation (4) the superscript T indicates the matrix transpose.

At this point the force maintaining the less fit a allele in the population is assumed to operate. The full two-locus recurrence relations then become

$$\mathbf{x}_{t+1}^T = \begin{bmatrix} y_1(1 - \mu) \\ y_2(1 - \mu) \\ y_3 + \mu y_1 \\ y_4 + \mu y_2 \end{bmatrix}. \quad (5)$$

ANALYSIS

LOCAL STABILITY ANALYSIS, MALES = FEMALES

We first consider the case of no sex differences in expression of the trait or the modifier allele. In this case $\mathbf{W} = \mathbf{Z}$. We are interested in the conditions for increase for the m allele when it is rare. Recall that this allele modifies the expression of the trait in one or both of the two viability classes. Consider the mutation-selection equilibrium corresponding to fixation of the M allele. It can be shown that this equilibrium is given as

$$\hat{\mathbf{x}} = [\hat{x}_1, 0, \hat{x}_3, 0], \quad (6)$$

with

$$\hat{x}_1 = \frac{-z_3 + z_1(1 - \mu)}{z_1 - z_3} \quad \text{and} \quad \hat{x}_3 = 1 - \hat{x}_1 = \frac{z_1 \mu}{z_1 - z_3}. \quad (7)$$

The equilibrium given by equation (6) corresponds to a mutation-selection equilibrium at which Haldane's principle applies. Haldane's principle (1932) states that the effect of mutational variation on average fitness is equal to the rate at which variation is produced by mutation, μ , and is independent of the effect of mutation on individual fitness, s . At the equilibrium given by equation (6), it can be seen

that the average fitness of the population is given by

$$\bar{z} = z_1(1 - \mu), \quad (8)$$

which is independent of s .

After obtaining the Jacobian matrix

$$\mathbf{J} = \left[\frac{\partial x_{i,t+1}}{\partial x_{j,t}} \right]_{\hat{x}}, \quad i, j = 1, 3, 4, \quad (9)$$

with the constraint

$$x_2 = 1 - x_1 - x_3 - x_4,$$

the eigenvalues are obtained by solving for the roots of the characteristic equation,

$$\det(\mathbf{J} - \lambda \mathbf{I}) = 0. \quad (10)$$

If the absolute value of the dominant eigenvalue is greater than one, the mutation-selection equilibrium is unstable and the modifier allele, m , increases in frequency. In most cases considered, the eigenvalues are positive over the range of biological interest, and our primary interest, therefore, is in the conditions under which the dominant eigenvalue is greater than one.

$$\text{Males} = \text{Females}, \quad 'r' = 0$$

We consider separately the case of $r = 0$, since in this case the three roots of equation (10) can be obtained explicitly. Two of the roots can each be associated with the condition for increase for one of the two rare gametes Am and am , which are independent of one another. These two eigenvalues are always nonnegative and equal to

$$\begin{aligned} \lambda_{am} &= z_4/\bar{z}, \\ \lambda_{Am} &= z_2(1 - \mu)/\bar{z}. \end{aligned} \quad (11)$$

The lack of recombination prohibits the am gamete from increasing in frequency in the usual case of modifier mutations of small and continuous effect, since $\lambda_{am} < 1$, if δ is small. This can be seen intuitively from the observation that the am gamete contains the new modifier allele with the low-viability allele. By definition, the low-viability class begins with fitness less than the average fitness by some discrete amount. Consequently, a small change in T cannot make up for this intrinsic fitness difference. Although, for large δ , a narrow range of parameter conditions can be found in which $\lambda_{am} > 1$, these conditions are considered to be biologically unrealistic and are not considered here.

A graph of λ_{Am} as a function of T is given in figure 1 for a specific case of modifiers of equal effect that increase (fig. 1a) and decrease (fig. 1b). The curves in figure 1 strictly hold for the parameter values $r = 0$, $s = 0.1$, $\mu = 0.2$, $c_a = 0.2$, and $c_A = 0.1$. These parameter values are used as a basis for many of the figures that follow. However, the qualitative nature of the curves in these figures is indicative of a wide range of parameter values. The quantitative effects of the

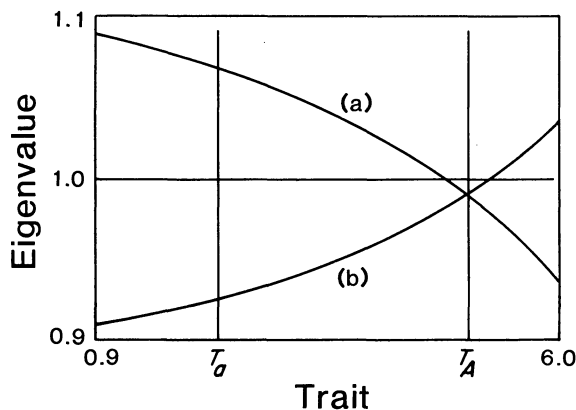


FIG. 1.—The dominant eigenvalue as a function of trait value for modifiers of “equal effect” that (a) increase or (b) decrease the trait by amount 0.1. No recombination. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = |\delta| = 0.1$, $s = 0.1$, $\mu = 0.2$, $c_a = 0.2$, $c_A = 0.1$, $r = 0$. See the text for further explanation.

basic parameters r , s , μ , c_A , and c_a are discussed when relevant. The biologically meaningful range of T for these parameter values is $T \in (0.83, 10.00)$. If T is outside this range, fitnesses and frequencies become negative. For the purposes of illustrating the most interesting range of eigenvalue curves, the range of (0.90, 6.00) was chosen for the figures. The eigenvalue curves do not cross the $\lambda_{\text{dom}} = 1.0$ line outside this range. The eigenvalue curves in figure 1 intersect at $T = 1/c_A(2 + \alpha)$, which approaches the optimum for the high-viability class, T_A , as α approaches zero. The finite magnitude of the effect of the modifier, α , creates a region of neutrality around T_A (fig. 1). As the effect of the modifier gets small ($\alpha \rightarrow 0$), this region vanishes, and the a and b curves in figure 1 intersect the $\lambda_{\text{dom}} = 1.0$ line at T_A exactly.

Consequently, at any point in the evolution of T , selection will be acting on modifiers that take T closer to the optimum for the high-viability class. These initial-increase conditions have been confirmed by computer simulation of recurrence equations (5). In all cases studied, if the modifier increased initially, it increased to fixation. The modification of T to T_A occurs whether the modifier affects only the high-viability class or both the high- and low-viability classes, since the effect of the modifier on the low-viability class is irrelevant to the dominant eigenvalue, λ_{Am} , and λ_{am} is always less than one. The steepness of the curves decreases as the magnitude of the effect, $|\alpha|$, becomes small.

In summary, with no recombination, evolutionary modification takes the value of the trait to the optimum value for the high-viability class. This value is then evolutionarily stable in the face of modifiers that change the value of the trait. Since there is no recombination and $\lambda_{am} < 1$, no modification occurs in the low-viability class unless it is a side effect of the effect of the modifier in the high-

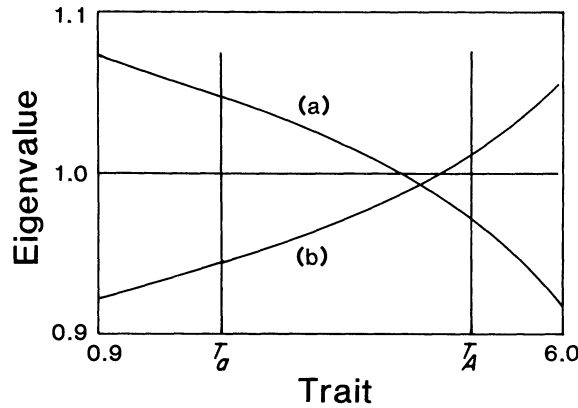


FIG. 2.—The dominant eigenvalue as a function of trait value for modifiers of equal effect that (a) increase or (b) decrease the trait by amount 0.1. Free recombination. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = |\delta| = 0.1, s = 0.1, \mu = 0.2, c_a = 0.2, c_A = 0.1, r = 0.5$. See the text for further explanation.

viability class.

$$\text{Males} = \text{Females}, 'r' \neq 0$$

When recombination is allowed, the situation changes and each viability class can optimize its value of the trait, so long as the one-to-one kind of modifiers occur (table 2). The roots of the characteristic equation (10) are given as $\lambda_1 = z_3/\bar{z} < 1$, always, and

$$\lambda_2, \lambda_3 = [-B \pm (B^2 - 4AC)^{1/2}]/2A, \tag{12}$$

where

$$\begin{aligned} A &= 1 - \mu, \\ B &= [r(z_4\hat{x}_1z_1 + z_0\hat{x}_3z_3) - z_1z_2(1 - \mu) - z_1z_4]/z_1^2, \\ C &= z_2z_3(1 - r)/z_1^2. \end{aligned}$$

We have solved equations (12) for various T values for the various kinds of modifiers given in table 2.

Modifiers of equal effect.—Consider first the case of modifiers that have the same effect in both high- and low-viability classes, $\alpha = \delta$ (table 2). In the case of sexual selection, we interpret this assumption as embodying the Fisher phase of the process of evolution of the male display, in which the issue of prior viability differences in males is not considered. An example of the eigenvalue functions for modifiers that increase (fig. 2a) and decrease (fig. 2b) expression of the trait is given in figure 2 for free recombination, $r = 0.5$. We see in figure 2 that the evolutionarily stable equilibrium for the trait is now intermediate between the optima for the two viability classes, being closer to T_A than to T_a . The corre-

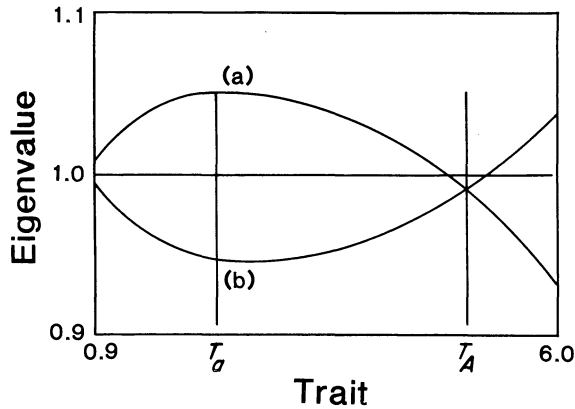


FIG. 3.—The dominant eigenvalue as a function of trait value for one-to-one modifiers that (a) increase or (b) decrease the trait by amount 0.1 in the high-fitness class. Free recombination. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = 0.1$, $|\delta| = 0$, $s = 0.1$, $\mu = 0.2$, $c_a = 0.2$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation.

sponding curves in the case of modifiers of a trait that is expressed only in males is given below in figure 10. As recombination decreases from $r = 0.5$ to $r = 0$, the intersection of the curves approaches T_A . The qualitative nature of these eigenvalue curves is independent of the parameter values, although the steepness of the curves and their exact point of intersection do depend on the parameter values chosen.

In summary, if the trait is constrained to be expressed equally in both viability classes, natural selection takes the trait to a value that is a compromise between the optima for the two viability classes.

One-to-one modifiers.—We now consider the cases of one-to-one modifiers that affect either the high-viability class or the low-viability class but not both (table 2). Figure 3 is the graph of an example of the case

$$|\alpha| = 0.1, \quad \delta = 0, \quad (13a)$$

and figure 4 is the corresponding graph for

$$\alpha = 0, \quad |\delta| = 0.1, \quad (13b)$$

both assuming free recombination. As can be seen in these figures, both viability classes can now optimize their trait values, so long as the appropriate modifiers exist. Although the qualitative nature of these eigenvalue curves are independent of the precise parameter values assumed, certain quantitative aspects do depend on these values. Consider, for example, the effect of the equilibrium frequency of the low-viability class on the evolution of modifiers of the less fit class (condition 13b). Figure 5 is the graph of the case of $\mu = 0.01$ to contrast with $\mu = 0.2$ in figures 1–4. The intensity with which selection acts on modifiers of the low-

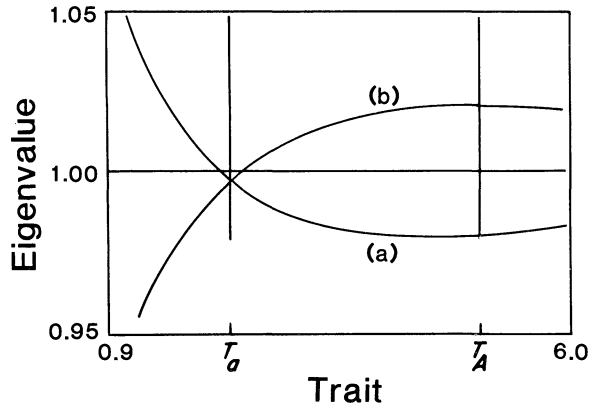


FIG. 4.—The dominant eigenvalue as a function of trait value for one-to-one modifiers that (a) increase or (b) decrease the trait by amount 0.1 in the low-fitness class. Free recombination. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = 0$, $|\delta| = 0.1$, $s = 0.1$, $\mu = 0.2$, $c_a = 0.2$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation.

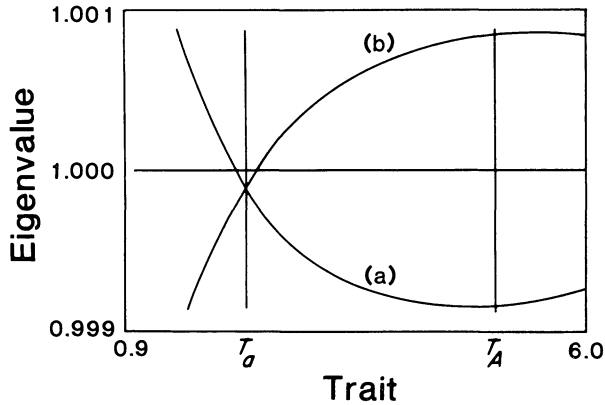


FIG. 5.—The dominant eigenvalue as a function of trait value for one-to-one modifiers that (a) increase or (b) decrease the trait by amount 0.1 in the low-fitness class. Free recombination. Same as figure 4 but with lower mutation rate. Notice different scale of y-axis. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = 0$, $|\delta| = 0.1$, $s = 0.1$, $\mu = 0.01$, $c_a = 0.2$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation.

viability class, as measured by the magnitude of the eigenvalues, is much reduced for the smaller mutation rate (note the different scale for the y-axis), although the qualitative nature of the curves is the same. Presumably, this occurs because the low-viability class on which selection operates is less abundant. Similar effects occur as a result of increases in s .

In figures 1–5, the eigenvalue curves are calculated assuming that each viability class has the same value of T before the modifier is introduced. In the case of modifiers of equal effect (figs. 1, 2), the curves depict continuous evolutionary trajectories driven by a cascade of modifiers each increasing (or decreasing) T , since as modifiers increase to fixation, the assumption of equal T in the two viability classes is maintained. For example, beginning at some value of T , say $T = 1$, if the eigenvalue curve is greater than one, the modifier, with $\alpha = \delta = 0.1$, will increase in frequency to fixation and take T for both viability classes to the next incremental value, $T = 1.1$. At this point what was the new modifier now becomes the old allele at the M locus, and the eigenvalue curve at $T = 1.1$ determines whether another modifier of similar effect will increase in frequency. Such modifiers will continue increasing until T reaches the evolutionarily stable region bracketed by the crossing of the eigenvalue curves with $\lambda = 1$ in figures 1 and 2. In the case of sexual selection, we interpret this evolutionarily stable state (ESS) as the equilibrated stage for the male trait described verbally by Fisher (1958) and mathematically by Lande (1981).

For modifiers of unequal effect, the eigenvalue curves in figures 3 and 4 no longer depict such a continuous evolutionary scenario, since the T values for the two viability classes begin to diverge as soon as the first modifier increases to fixation. In this case, to be considered next, the evolutionary scenario driven by a cascade of modifier mutations depends on the T value at which the modification process begins. Nevertheless, the curves in figures 3 and 4 show that, at any point in the evolution of T driven by modifiers of equal effect or by some other process maintaining the assumption of equal T in the two classes, there will be selection for one-to-one modifiers of the kind given.

We now assume that evolution has led to the evolutionarily stable value for modifiers of equal effect (fig. 2; for the Fisher equilibrium in the case of sexual selection, see fig. 10), and we ask when one-to-one modifiers will increase in frequency. An example is given in figure 6 for the case considered previously, in which the Fisher T value for modifiers of equal effect is $T = 4.43$ (fig. 2). For this situation, figures 6*a* and 6*b* consider the cases of one-to-one modifiers that affect only the high-viability class or the low-viability class, respectively. In figure 6*a* or 6*b*, the T value for the low-viability class or high-viability class, respectively, remains at its initial value of $T_i = 4.43$, whereas the T value for the other class changes because of evolutionary modification. The graphs in figure 6 represent continuous evolutionary trajectories of modifiers of the assumed kind beginning at $T = 4.43$. As can be seen from figure 6*a*, one-to-one modifiers of the high-viability class, which increase T from its initial value of $T_i = 4.43$, increase in frequency until T approaches T_A , the optimum for the high-viability class. Modifiers of the high-viability class that decrease T do not increase in frequency. The converse is the case for one-to-one modifiers of the low-viability class, and, in this case, the trait in the low-viability class approaches T_a , the optimum for that class (fig. 6*b*).

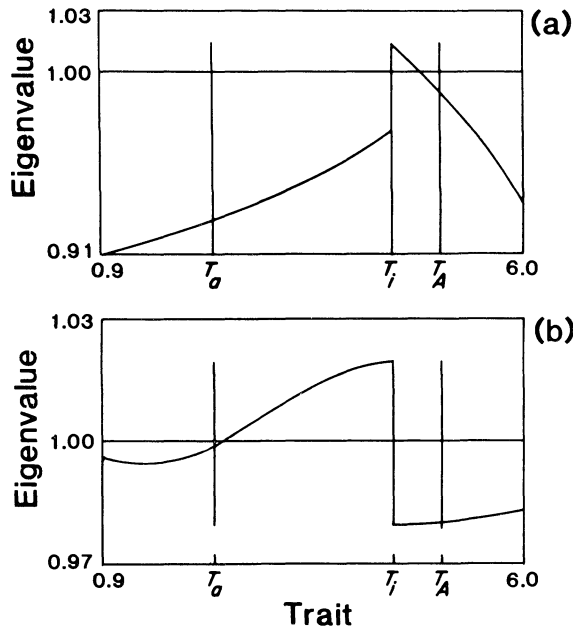


FIG. 6.—The dominant eigenvalue as a function of trait value for one-to-one modifiers of (a) the high-fitness class or (b) the low-fitness class, assuming an initial trait value of $T_i = 4.43$. Free recombination. Eigenvalue curves to the left (right) of T_i are for modifiers that decrease (increase) the trait. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: (a), $|\alpha| = 0.1$, $|\delta| = 0$; (b), $|\alpha| = 0$, $|\delta| = 0.1$; $s = 0.1$, $\mu = 0.2$, $c_a = 0.2$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation.

The optimization of T by evolutionary modification does not require differential costs of the trait in the two viability classes, although it is facilitated by such differences. That differential cost, in the sense of $c_A \neq c_a$, is not necessary, as can be seen in figure 7 for the case of $c_A = c_a = 0.1$, $\mu = 0.2$, $r = 0.5$, $s = 0.2$, and one-to-one modifiers with a magnitude of effect equal to 0.05. The strength of selection as determined by the magnitude of the eigenvalues is reduced by the assumption of equal costs, however. (Note that in fig. 7a, the eigenvalue curve appears to cross the $\lambda = 1.0$ line in the region between $T = 0.9$ and $T = T_a$. This evolutionary trajectory of decreasing T in the high-fitness class could not be taken, since the eigenvalue curve begins with a modulus less than one. Even if it were taken, however, as the curve approaches one, the equilibrium frequencies of one of the genotypes becomes negative. Consequently, the curve does not become greater than one in a region of biological relevance.)

In summary, evolutionary modification of the trait will take its value toward its optimum value in the two viability classes assuming that modifiers can act independently in the two viability classes.

Antagonistic modifiers.—We now consider modifiers with antagonistic effects in the two viability classes, that is, modifiers that increase the trait in one class and

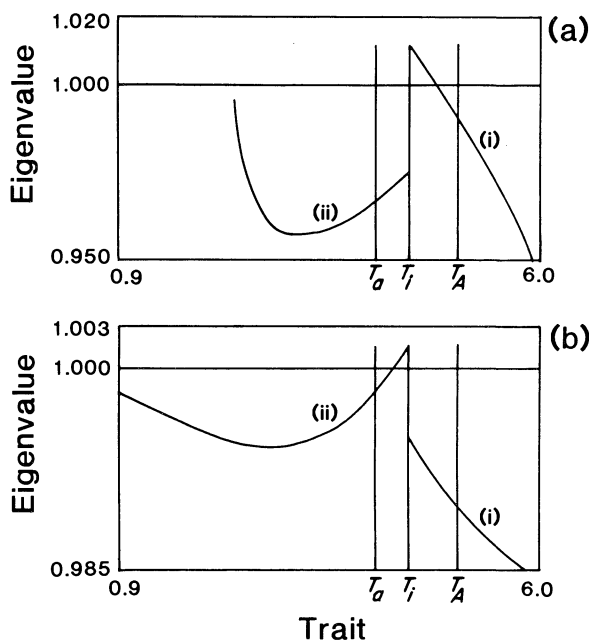


FIG. 7.—The dominant eigenvalue as a function of trait value for one-to-one modifiers of (a) the high-fitness class or (b) the low-fitness class, assuming an initial trait value of $T_i = 4.43$. Free recombination, equal cost. Eigenvalue curves to the left (ii) (right, i) of T_i are for modifiers that decrease (increase) the trait. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: (a), $|\alpha| = 0.1$, $|\delta| = 0$; (b), $|\alpha| = 0$, $|\delta| = 0.1$; $s = 0.1$, $\mu = 0.2$, $c_a = 0.1$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation.

decrease it in the other (table 2). Such antagonistic effects may result from modifiers that associate the trait with a physiological parameter that is itself correlated with viability class, such as resting metabolic rate, fat level, or protein level (Røskaft et al. 1986). Figure 8a is the graph of an example of the case

$$\alpha = -0.1, \quad \delta = +0.1, \quad (14a)$$

and figure 8b is the corresponding graph for

$$\alpha = +0.1, \quad \delta = -0.1, \quad (14b)$$

both assuming free recombination and T initially equal in the two classes. This is only one of many possible examples of such modifiers. The constraint of equal magnitude of effect in both viability classes serves to represent the issue of modifiers with antagonistic effects, but it may be an artificial assumption in some cases. If we were to assume that the magnitude of the effect of the modifier is proportional to the distance of the initial T from the optima of the viability classes, T_A and T_a , optimization should be able to occur. However, our interest here lies in illustrating possible constraints on the optimization process.

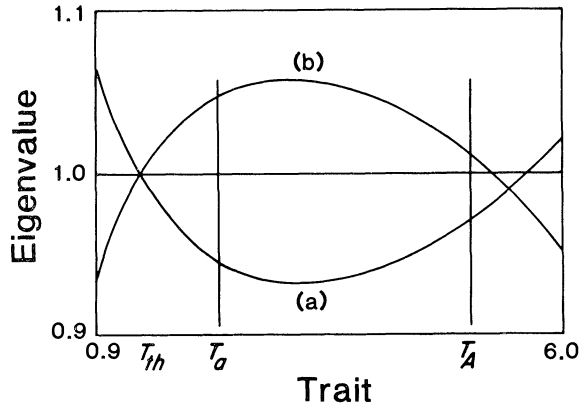


FIG. 8.—The dominant eigenvalue as a function of trait value for antagonistic modifiers that (a) decrease or (b) increase the trait by amount 0.1 in the high-fitness class. Free recombination. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: (a), $\alpha = -0.1, \delta = +0.1$; (b), $\alpha = +0.1, \delta = -0.1$; $s = 0.1, \mu = 0.01, c_a = 0.2, c_A = 0.1, r = 0.5$. See the text for further explanation of the threshold value, T_{th} .

As can be seen in figure 8 for the case of antagonistic modifiers with equal magnitude of effect, there is now a threshold value for $T = T_{th}$. On the one hand, if the initial $T = T_i$ is such that $T_i < T_{th}$, then modifiers that increase T in the low-viability class and decrease it in the high-viability class (eq. 14a) will increase in frequency. However, antagonistic modifiers of the kind given in equation (14b) cannot increase if $T_i < T_{th}$. On the other hand, if $T_i > T_{th}$, then the converse is the case, and modifiers that increase T in the high-viability class but decrease it in the low-viability class will themselves increase in frequency. In either case, the modification process does not lead to a cascade of modification, since the fitness of one or the other viability class will quickly decrease to zero. This occurs because, for small initial T values, there is little room for T to decrease further in either of the viability classes, since both classes are far from their optima, and the modifier is taking one of the viability classes even farther from its optimum. This occurs because antagonism in the effects of the modifiers is assumed. For this reason, we consider the case of evolutionary trajectories beginning at intermediate T values.

In figure 9 are graphed the evolutionary trajectories beginning at the evolutionarily stable values for modifiers of equal effect, $T_i = 4.43$ (fig. 2). Since the evolutionary trajectory of T in the two viability classes is different, the eigenvalue curve is first graphed as a function of T in the high-viability class (fig. 9a). Since T in the low-viability class is also changing, in figure 9b, changes in T in the low-viability class are graphed against changes in T in the high-viability class. As can be seen in figure 9, the high-viability class dominates the process. Modifiers that increase T in the high-viability class but decrease T in the low-viability class (eq. 14b) increase in frequency, taking T in the high-viability class to a value just

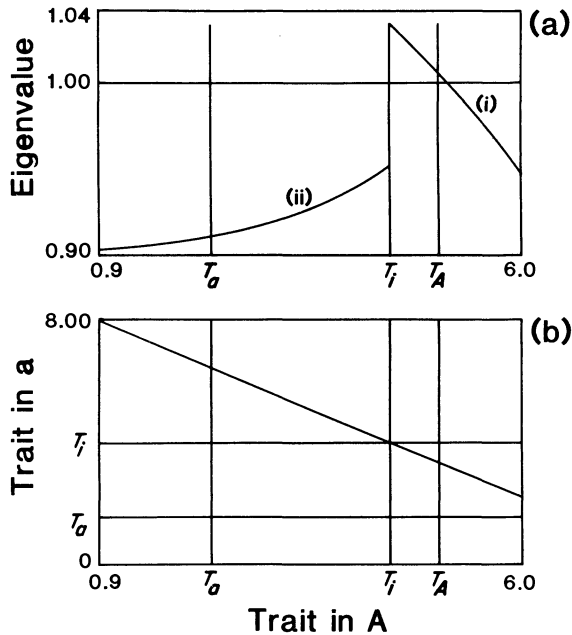


FIG. 9.—(a), The dominant eigenvalue as a function of trait value for antagonistic modifiers of high-fitness class beginning at $T_i = 4.43$. (b), T in low-fitness class graphed against T in high-fitness class. Free recombination. (a), Eigenvalue curves to the left (ii) (right, i) of T_i are for modifiers that decrease (increase) the trait in the high-viability class. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = 0.1$, $|\delta| = 0.1$, $s = 0.1$, $\mu = 0.01$, $c_a = 0.2$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation of T_{th} .

greater than T_A , the optimum for the high-viability class (fig. 9*a*(i)). Concomitantly, T in the low-viability class decreases but does not reach T_a (fig. 9*b*). Modifiers that increase T in the low-viability class but decrease T in the high-viability class do not increase in frequency (fig. 9*a*(ii)).

In summary, if the effects of the modifier on the two viability classes are interdependent, evolutionary modification of the trait may be prevented from taking the trait to its optimum value in each viability class. Nevertheless, the trait will usually come to have different values in the two viability classes.

LOCAL STABILITY ANALYSIS, MALES \neq FEMALES

We now consider the case of evolution of male display in which expression and modification of the trait are confined to males. The fitness of the four female gametes now depends only on their genotype at the fitness locus (table 1B), and the male population has the trait- and modifier-dependent fitnesses already considered. Using matrices (3) with the additive model given in table 1B, equations (5)

can be used to study this problem. Analytical analysis of the problem is complicated by the fact that the equilibrium frequency at the viability locus now corresponds to a mutation-selection balance involving different selection in the two sexes. The equilibrium frequency is now given as a root of the following quadratic equation, which cannot be factored:

$$\hat{x}_1 = [-B - (B^2 - 4AC)^{1/2}]/2A, \tag{15}$$

with

$$A = (z_3 - z_1)s,$$

$$B = -A - C - \mu z_1,$$

$$C = \frac{1}{2} z_1(1 - s) + \frac{1}{2} z_3 - z_3(1 - s) - \frac{1}{2} \mu [z_1(1 - s) + z_3].$$

To obtain the eigenvalues, the partial derivatives forming the Jacobian matrix (eq. 9 using table 1B) must now be evaluated at \hat{x}_1 given by equation (15). Doing so yields

$$\begin{aligned} \lambda_1 &= (y_{3,3} - y_{3,1})(1 - \mu), \\ \lambda_2, \lambda_3 &= [-b \pm (b^2 - 4c)^{1/2}]/2, \end{aligned} \tag{16}$$

where

$$b = -(1 - \mu)y_{4,4} - y_{1,1} - y_{3,1} + \mu(y_{1,4} + y_{3,4}),$$

$$c = (y_{1,1} + y_{3,1})(1 - \mu)y_{4,4} - (1 - \mu)y_{4,1}(y_{1,4} + y_{3,4}),$$

with

$$y_{i,j} = \left(\frac{\partial y_{i,t+1}}{\partial y_{j,t}} \right)_{\hat{x}},$$

with the understanding that $\hat{x} = (\hat{x}_1, 0, 1 - \hat{x}_1, 0)$ with \hat{x}_1 given by equation (15).

Figure 10 is the graph of the case corresponding to the previous figures for modifiers of equal effect but for the fitness specification given in table 1B. Limiting the trait to males has shifted the compromise Fisher T value for modifiers of equal effect more to the center between the two optima. However, nothing qualitatively new concerning the evolution of modifiers of the male trait has emerged in our analysis of the sexual-selection case that has not been discussed in the case of equal males and females, above. For this reason, we do not present here the complete set of eigenvalue curves for the sexual-selection case specified in table 1B.

DISCUSSION

Fisher (1958) argued that a trait in males that is preferred by females may become an extravagant display as a consequence of this preference during, what he called, a runaway process. During this process, the genes for female choice become associated with those for the male display, since if both female choice and male display are heritable, choosy females tend to have sons with a display and

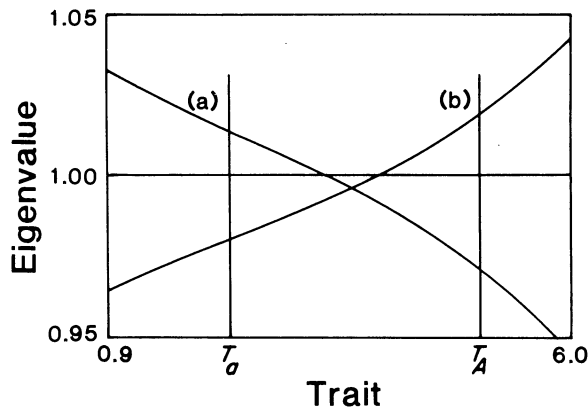


FIG. 10.—The dominant eigenvalue as a function of trait value for modifiers of equal effect that (a) increase or (b) decrease the trait by amount 0.1. Sexual-selection model with free recombination. $T_a = 2.25$ and $T_A = 5.00$ are the optimum trait values for low-fitness and high-fitness classes, respectively. A dominant eigenvalue greater than one (less than one) indicates that the modifier increases (decreases) in frequency from an arbitrarily small initial frequency. Parameter values: $|\alpha| = |\delta| = 0.1$, $s = 0.1$, $\mu = 0.2$, $c_a = 0.2$, $c_A = 0.1$, $r = 0.5$. See the text for further explanation.

the (unexpressed) genes for choosiness. As a result of female choice, more-extreme males gain more matings. Therefore, the two factors, female choice and male display, reinforce one another, leading to increasingly extreme males. This runaway process stops at the point at which the increasing mating benefits of the display no longer compensate for the decreasing survival. “[A]t this point both plumage elaboration [i.e., male display] and the increase in female preference will be brought to a standstill, and a condition of relative stability will be attained” (Fisher 1958, p. 152). Under some circumstances, there can be a line of equilibria at which each intensity of female choice accounts for a unique value of male display (frequency, if it is a single allele [Kirkpatrick 1982]; size or conspicuousness, if it is a continuous character [Lande 1981]). Fisher’s scenario and the more recent genetic models that demonstrated its validity have been interpreted by theorists as implying that male displays may be only aesthetic (attractive), with no information about other characters in males (see, e.g., Arnold 1985).

In contrast, Zahavi (1975, 1977) claimed that females prefer males with displays because displays are indicators of genetic fitness. Thus, genes for female choice become associated with those for both fitness and male display. Under Fisher’s view, choosy females pass on more genes to future generations because of the increased mating success of their sons, whereas, according to Zahavi’s view, choosy females pass on more genes because of the increased viability of both their sons and daughters. Female choice evolves as a consequence of its association with the genes determining viability (Pomiankowski 1987a).

There are three main issues in explicating Zahavi’s view: (1) the maintenance of heritable variation in fitness, (2) the evolution of female choice, and (3) the maintenance of the display as a faithful indicator of genetic fitness. The mainte-

nance of variation in fitness is a large problem in evolutionary theory, one that has been explicitly represented here but not satisfactorily addressed at a deep level. Concerning female choice, we have implicitly assumed that it exists, and, as a result, our model does not address the evolution of female choice. Our main purpose has been to understand a mechanism by which a display may originally become a faithful indicator of genetic fitness as well as a mechanism by which it can be maintained as such.

Heritable variation in viability is necessary for the process of evolutionary modification studied above. Indeed, the strength of the action of selection on modifiers has been shown to depend on the magnitude of this variation. As just mentioned, the maintenance of heritable variation in fitness is a fundamental problem in evolutionary theory, one that is surrounded by controversy and strong opinions. The emphasis in the present paper is more on the implications of this variation for the evolution of reliable indicators of fitness, rather than on the mechanism by which this variation is maintained. The model of single-locus mutation or migration studied above is not intended to represent realistically the *process(es)* by which genetic variation in viability is maintained. In real genomes, many loci contribute to viability differences between individuals. For reasons of mathematical tractability, we consider variation in viability at only a single locus, but we interpret this locus as representing the whole genome. We have assumed realistic mutation rates at the level of the genome, but when coupled with the assumption of a single locus, these same rates appear unrealistic. This is a limitation of our model. However, we do not believe it invalidates the conclusions drawn, so long as there is some mechanism that maintains significant levels of heritable variation in fitness.

In contrast with previous suggestions (Zahavi 1975; Andersson 1982; Kodric-Brown and Brown 1984; Nur and Hasson 1984), high cost is not required for the trait to become a reliable indicator of prior viability differences in the population. What seems to be required in our model is simply that the overall fitness of the two viability classes be maximized at different values of the trait (Nur and Hasson 1984). In most cases, greater absolute cost pushes the optimum T values farther apart, and, as a result, greater costs should facilitate the process of modification. In addition, different relative costs, with the low-viability class paying a greater cost than the high-viability class ($c_a > c_A$), enhance the prior fitness difference resulting from viability class and push the optimum T values farther apart. Consequently, both greater absolute cost and differences in relative cost can facilitate the process of modification of the trait in the different viability classes. However, neither of these factors is logically necessary. There may be different optima for the two viability classes, if the absolute magnitudes of the costs are small, if the costs per unit of trait are equal for the two viability classes, or even if the high-viability class pays a higher cost than the low-viability class.

In figure 7, we show that unequal costs are not necessary for the process of evolutionary modification to proceed in different directions in the two viability classes, at least for the additive model considered there. A multiplicative model of viability effects, in which the viability of the low-viability class was $v_3 = (1 - s) \cdot (1 - Tc_a)$ with $v_1 = 1 - Tc_A$, would not yield different optima for the two viability

classes unless the costs were different (L. Heisler, pers. comm.). In addition, when the cost is the same in the two viability classes ($c_a = c_A$), the relative effect on the low-viability class is still greater, since this class has less viability to begin with. Although this is true, it misses the point that the cost per se is not the basic issue. The basic issue is the maximization of individual fitness at different trait values for the different viability classes. Cost is one factor, perhaps an important one in practice, which can contribute to this.

A major problem with the good-genes view in general has been that the mechanism that maintains male display as a faithful indicator of fitness has not been satisfactorily addressed. Even when the male character initially functions as an indicator of fitness before it responds to female choice (see, e.g., Fisher 1958), it may lose this feature during the runaway process. Fisher (1915), O'Donald (1980), and Heisler (1984, 1985) suggested that the increasing values of the male character (now a display) should become dissociated from fitness. If this is correct, then when the evolutionary trajectory of the system lands at some point on the line of equilibria, male display would be aesthetic, not an indicator of fitness. If female choice is costly, both choice and display will be unstable (Pomiankowski 1987*b*).

This difficulty can be resolved in two ways that are supported by dynamic models. First, male display may evolve initially as an amplifier of preexisting detectable differences in viability class, on which female choice is already based. A display that functions as an amplifier benefits its high-fitness male carriers but decreases mating success of its low-fitness male carriers. Consequently, the amplifying effect of the display is likely to result in a correlation between viability class and the display's expression (Hasson 1989). This approach is based on the assumption that certain traits such as feather condition, by their nature, are faithful indicators of fitness.

Second, Dominey (1983) and Nur and Hasson (1984) suggested that male display may become an indicator of fitness after the runaway process has come to a halt, at the equilibrium between the viability cost and mating benefit conferred by the display. At this point, the display attains its Fisher T value (assuming the same initial T for all males). Optimization processes operating on the expression of the display in males were assumed to lead the display to be expressed differentially in males of different viability classes. In this paper, we have tested this assumption of differential expression of the trait in different viability classes by examining the evolution of genetic modifiers that change the value of the trait by an amount that depends on viability class. We have shown that, as a consequence of prior variation in viability class, such genetic modifiers will evolve and maintain different values of the trait in different viability classes. Consequently, evolutionary modification of the trait should allow it to be used as a reliable indicator of viability class.

In the model analyzed here, sexual recombination was necessary for traits to reach their optimum values. As a consequence of this optimization process, such traits could be used as reliable indicators of viability class. As shown above, this optimization process depends on the presence of sexual recombination as well as on the existence of modifiers that can act independently in each viability class. This role of sex as a facilitator of fitness optimization is in contrast to the many

situations in which sex can constrain adaptation and optimization (see, e.g., Michod 1984, 1986; Gayley and Michod 1990). It is yet another example of the often conflicting and model-dependent effects that sex has at the population level (Michod 1990).

SUMMARY

We study the evolution of traits that have conflicting effects on two components of fitness in a population in which there are prior heritable viability differences. Examples of such traits may include the conspicuous male traits, or displays, that increase a male's mating success but decrease his viability. We consider the simple case of two viability classes controlled by a single haploid locus and a second modifier locus, which modifies the expression of the trait in one or both of the viability classes. Variation at the viability-class locus is maintained by a balance between selection and a force, like mutation or migration, that restores the less fit class. As a result of the prior differences in viability and the effects of the trait, overall fitness in each viability class is maximized at different values (optima) of the trait. In the absence of modifiers that can differentially express the trait in the different viability classes, the evolutionarily stable value of the trait is a compromise between the optimum values for the two fitness classes. Modifiers that can act independently on the trait in the two viability classes allow the trait to take on its optimum values in the two viability classes. Since the optimum of the trait for the high-viability class is greater than that for the low-viability class, evolutionary modification should allow such traits to be used as reliable indicators of genetic fitness. Differential cost of the trait in the two viability classes is not required for the trait to be maintained as a reliable indicator of fitness in an additive model of viability effects considered here. However, in a multiplicative model of viability effects, differential cost would be necessary. What seems to be required in all cases is simply that the overall fitness of the two viability classes be maximized at different values of the trait. Constraints on the effects of the modifiers in the two viability classes can interfere with this optimization process. However, the trait still evolves to have different values in the two viability classes. We apply our results to the good-genes theory of sexual selection, which postulates that the male display is a reliable indicator of male fitness.

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