ABSTRACT

The equilibrium distribution of a quantitative character subject to frequency- and density-dependent selection is found under different assumptions about the genetical basis of the character that lead to a normal distribution in a population. Three types of models are considered: (1) one-locus models, in which a single locus has an additive effect on the character, (2) continuous genotype models, in which one locus or several loci contribute additively to a character, and there is an effectively infinite range of values of the genotypic contributions from each locus, and (3) correlation models, in which the mean and variance of the character can change only through selection at modifier loci. It is shown that the second and third models lead to the same equilibrium values of the total population size and the mean and variance of the character. One-locus models lead to different equilibrium values because of constraints on the relationship between the mean and variance imposed by the assumptions of those models. The main conclusion is that, at the equilibrium reached under frequency- and density-dependent selection, the distribution of a normally distributed quantitative character does not depend on the underlying genetic model as long as the model imposes no constraints on the mean and variance.

FREQUENCY- and density-dependent selection on a quantitative character can result from interactions between individuals in a population in which the extent of the interaction depends on the measure of the character in each individual. The interaction could be either direct or indirect and may depend on other species in the community. There would be direct interactions in a species if, for example, the outcome of a dispute over a food item depended on relative size. An individual's access to food would depend on the size distribution of the individuals it encountered. There would be indirect interactions if the ability to utilize food items of different size depended on body size. The extent of interaction between different individuals in such a species would depend on the distribution of food sizes for which they compete and on the distribution of body sizes.

In this paper, I shall consider the evolutionary equilibrium distribution of a quantitative character subject to frequency- and density-dependent selection. The motivation for this paper is provided by the fact that there are a number of biological conditions under which there would be expected to be frequency-
and density-dependent selection acting on a normally distributed character. The goal is to find the equilibrium distribution of such a character and determine how sensitive the equilibrium distribution is to the assumptions of the underlying genetical models.

I have discussed in another paper (Slatkin 1979a) the more general problem of the effect of frequency- and density-dependent selection on a polymorphic species. The relevant result from that paper is that natural selection will tend to equilibrate the fitnesses of the different phenotypes in a population to the extent possible, given the genetic flexibility in the frequencies of the different phenotypes. Thus, we could expect that, if there are relatively few distinct phenotypes, there would be sufficient genetic flexibility through the frequencies of the various gametes and, possibly, genetically modifiable parameters, that the fitnesses of the different phenotypes would be equal at any equilibrium.

For a quantitative character, the implications of the general analysis are different. There is an effectively infinite number of phenotypic classes distinguished by different values of the character and relatively little genetic flexibility in the distribution of the character. Most quantitative characters are normally distributed, at least on a transformed scale. While it is possible that natural or artificial selection can alter the mean and variance of the distribution, it is likely to be difficult to change its functional form. Therefore, there are effectively only two parameters that can be changed by selection, the mean and variance, and the general results cannot be used. It is not correct to assume that, at equilibrium, all individuals have the same fitness. However, we shall see in the present paper that, given the assumptions that a character be normally distributed and there be no constraints on the mean and variance, it is possible to characterize the evolutionary equilibrium distribution of a quantitative character in a way that does not depend on the details of the genetic basis of the character. Different predictions are obtained when there are constraints on the mean and variance.

There are currently three different approaches to modelling the evolution of quantitative characters. The first is through a one-locus model in which the different genotypes have fixed effect. Included in this category are models in which there is more than one locus, but with the same allele frequency at each (e.g., Bulmer 1971, 1974). The second is through one-locus or multiple-locus models in which there is an effectively infinite number of alleles at each locus, which can have a continuous range of effects (e.g., Kimura 1965; Lande 1976a, 1977; Cavalli-Sforza and Feldman 1976; Felsenstein 1977). The third is through a correlation or phenotypic model of the inheritance of the character in which the parameters of the model are subject to genetic modification (Slatkin and Lande 1976). Each of these approaches differs in the assumptions that are made about the genetical basis of the character. We shall see here that, for understanding the equilibrium distribution, the important difference between these approaches is in the genetic constraints placed on the changes in the mean and variance of a normally distributed quantitative character. When there are no constraints, these models lead to the same predictions about the equilibrium...
mean and variance of the character, but the predictions differ if there is some implicit or explicit constraint.

THE SELECTION MODEL

We shall consider models of a single population and a quantitative character of measure \( z \) that is normally or approximately normally distributed in the population. Let \( N(t) \) be the number of newborn individuals and \( p(z,t) \) be the distribution of \( z \) among newborns before selection has acted in generation \( t \). In all cases that we shall consider, \( p(z,t) \) will be normal, either by assumption or as a consequence of the genetical model, and we shall denote the mean and variance of \( p(z,t) \) by \( \bar{z}(t) \) and \( \sigma_z^2(t) \).

Let \( W(z) \) be the absolute fitness of individuals of type \( z \). When there is assumed to be frequency- and density-dependent selection, \( W(z) \) depends on both \( N(t) \) and \( p(z,t) \). Throughout, we shall assume that the only time dependence in \( W \) is through \( N(t) \) and \( p(z,t) \). The function, \( W \), is assumed to take into account the various ecological interactions among individuals that lead to frequency- and density-dependent selection. A convenience form of \( W \) and one that has some empirical basis is

\[
W(z) = 1 + r - \frac{rN}{k(z)} \int \alpha(z - z') p(z') \, dz'
\]

where \( k(z) \) represents the resources that can be utilized by an individual of type \( z \), \( \alpha(z - z') \) represents the competition between individuals of types \( z \) and \( z' \) for the limiting resource, and \( 1 + r \) is the maximum fitness in the absence of competition. Throughout, the integrals will be assumed to be over all values of \( z \). This functional form of \( W \), which is analogous to the form of the Lotka-Volterra competition equations, was introduced by Roughgarden (1972) in the context of competition between lizards of different size for insects of different size. It is a convenient form to use because many of the results can be obtained analytically, but we shall see that the conclusions will not depend on the form of \( W \), only on its general properties.

In some of the examples, we shall use particular forms for \( k(z) \) and \( \alpha(z - z') \). Let

\[
k(z) = \frac{k_o}{\sqrt{2\pi}\sigma_z} \, e^{-(z-z_o)^2/2\sigma_z^2} ,
\]

where \( k_o \) represents the total resource availability, \( z_o \) represents the value of the character for which the maximum resources are available and \( \sigma_z^2 \) is a measure of the range of available resources. Let

\[
\alpha(z - z') = e^{-(z-z')^2/2\sigma_z^2} ,
\]

where \( \sigma_z^2 \) is a measure of the extent of competition between individuals of types \( z \) and \( z' \) and can be interpreted as an indication of the resources that are shared by the two types (Roughgarden 1972).
Given a particular functional form for $W(z)$, a model of a population must lead to recursion formulae for $N(t)$, $\bar{z}(t)$ and $\sigma_z^2(t)$. The recursion formulae for the mean and variance depend on the assumptions about the genetical basis of the character, and these formulae will be obtained for each of the three models presented in the next sections. However, for any reasonable genetical model, changes in population size depend on the mean absolute fitness in any generation—assuming discrete nonoverlapping generations. Thus, the recursion formula for $N(t)$ is

$$N(t+1) = N(t) \int W(z) p(z,t) \, dz.$$  \hspace{1cm} (4)

**ONE-LOCUS MODELS**

We begin with a brief review of a one-locus model of a quantitative character. Consider a monoecious, randomly mating species with discrete nonoverlapping generations and assume that there is a single locus with two possible alleles, $A_1$ and $A_2$. Assume that there is a quantitative character with measure $z$, and assume that the mean value of the character in the three genotypes $A_1A_1$, $A_1A_2$, and $A_2A_2$ is $-a$, $0$, and $a$. That is, each allele at the locus has an additive effect $a/2$ and there is no dominance. Assume, also, that the distribution of $z$ among the individuals with the same genotype is normal with the mean equal to the genotypic mean and the variance equal to $\sigma_e^2$, the environmental component of the variance.

If we let $x$ be the frequency of $A_1$, then

$$\bar{z} = (1 - 2x)$$ \hspace{1cm} (5)

and

$$\sigma_z^2 = 2a^2x(1 - x) + \sigma_e^2,$$ \hspace{1cm} (6)

where $\bar{z}$ and $\sigma_z^2$ are the mean and variance of $z$ in the population. Equations (5) and (6) follow from the assumption of random mating, and it is clear that the values of $\bar{z}$ and $\sigma_z^2$ are completely determined by the value of $x$, the only independent variable. In fact, can eliminate $x$ from (5) and (6) to obtain the relationship

$$\sigma_z^2 = \frac{a^2}{2} \left(1 - \frac{\bar{z}^2}{a^2}\right) + \sigma_e^2$$ \hspace{1cm} (7)

that is imposed by the assumptions of the one-locus model. There is a similar relationship between $\bar{z}$ and $\sigma_z^2$ if there is more than one locus, at which the additive effects are independent and of the same magnitude and at which the allele frequencies are the same. (cf., Bulmer 1971, 1974.) Since we are concerned with a normally distributed, quantitative character, we must assume that $a$ is small enough that the character is approximately normally distributed with a mean and variance given by (5) and (6), even though it cannot be exactly so because the distribution of $z$ is the sum of three normal distributions with different mean values. That is, we must assume $a << \sigma_e$.

Let the fitnesses of the three genotypes $A_1A_1$, $A_1A_2$, and $A_2A_2$ be $W_1$, $W_2$ and
Those fitnesses are computed by averaging $W(z)$ over the distribution of $z$ for each genotype. That is,

$$W_i = \int W(z) \, g(z + a_i) \, dz,$$

where $g(z)$ is normal with mean 0 and variance $\sigma_z^2$ and $a_i = -a, 0, a$ for $i = 1, 2, 3$. There are two conditions for the equilibrium values of $x$ and $N$. The first is the condition for genetic equilibrium, which can be written as

$$\hat{x}(W_1 - W_2) = (1 - \hat{x})(W_3 - W_2),$$

where the carat indicates the equilibrium value. The second is the condition for the demographic equilibrium,

$$\bar{W} = \hat{x}^2 W_1 + 2\hat{x}(1 - \hat{x}) W_2 + (1 - \hat{x})^2 W_3 = 1,$$

which follows (4).

Equation (10) implicitly defines $\hat{N}$ as a function of $x$ for any fixed value of $x$, including $\hat{x}$. Substituting this function in (9), we can obtain a single equation of the unknown equilibrium allele frequency, $\hat{x}$. There is no reason to assume that there must be a solution for $\hat{x}$ in $(0,1)$ or that there must be only one such solution. However, at any polymorphic equilibrium, (9) implies that either

$$W_1 = W_2 = W_3$$

or

$$W_1 \neq W_2 \text{ and } W_3 \neq W_3$$

must be satisfied. However, (11a) requires that a single independent variable, $\hat{x}$, simultaneously solve two independent algebraic equations. That is unlikely to be possible unless there are special symmetries in the fitness function that make such a solution possible. This result is a special case of the general case in which there is an insufficient number of independent allele frequencies (one) to permit the equilibration of the three independent genotypic fitnesses (SLATKIN 1979a).

We can illustrate this result and make it more concrete by using the particular form for $W(z)$ given in (1)–(3), for which analytic results can be obtained. We evaluate the $W_i$ using (8) and the assumption in (1) that $p(z')$ is normal with mean and variance given by (5) and (6). The algebra is straightforward and the results are

$$W_i = 1 + r - A \exp\left[B(a_i)/2D - C(a_i)\right],$$

where $A$ is a constant independent of $a_i (=-a, 0, +a)$ and

$$B(a_i) = \frac{a_i}{\sigma_e^2} + \frac{\bar{z}}{\sigma_z^2 + \sigma_a^2} - \frac{z_0}{\sigma_h^2}$$

$$C(a_i) = \frac{a_i^2}{\sigma_e^2} + \frac{\bar{z}^2}{\sigma_z^2 + \sigma_a^2} - \frac{z_0^2}{\sigma_h^2}$$

$$D = \frac{1}{\sigma_e^2} + \frac{1}{\sigma_z^2 + \sigma_a^2} - \frac{1}{\sigma_h^2}.$$
When (11a) is satisfied, then the condition that \( W_2 = W_3 \) implies
\[
\sigma_z^2 = \sigma_a^2 + \sigma_e^2.
\] (15)
Together, (14) and (15) imply that (11a) can be satisfied only when both
\[
\bar{z} = z_0
\] (16a)
and
\[
\sigma_z^2 = \sigma_a^2 - \sigma_e^2
\] (16b)
are satisfied. If \( \sigma_z^2 - \sigma_a^2 < \sigma_e^2 \), then there is no equilibrium solution such that (11a) is satisfied because \( \sigma_z^2 \geq \sigma_e^2 \). Otherwise, there can be such an equilibrium solution only if both parts of (16) are satisfied. However, \( \bar{z} \) and \( \sigma_z^2 \) are related by (7), which is imposed by the assumptions of the one-locus model. Therefore, (11a) cannot be satisfied at the equilibrium except for particular and special choices of the parameter values.

CONTINUOUS GENOTYPE MODELS

Kimura (1965), Lande (1976a) and others analyzed another type of model of a quantitative character that has an additive genetic basis. The essential difference between this type and the one-locus model discussed above is that the constraint on the functional relationship between the mean and variance imposed by the one-locus model (i.e., equation 7) is removed by making the assumption that, at each locus, there is an effectively infinite range of genotypic values. Kimura (1965) and Lande (1976a, 1977) analyzed a model of a character subject to density- and frequency-independent optimizing selection and show that a significant phenotypic variance can be maintained by independent mutations of small effect. Here, we will consider the effect of frequency- and density-dependent selection in the absence of mutations. We will discuss later the potential effects of mutations in this model.

The features of the multilocus, continuous genotype model introduced by Lande (1976a, 1977) that are relevant here are:

1. A quantitative character with measure \( z \) is determined additively by \( n \) loci. The value of the character in an individual is
\[
z = g + e = \sum_{i=1}^{n} (x_i + x_i') + e,
\] (17)
where \( x_i \) and \( x_i' \) are the additive effects at the paternal and maternal gametes at the \( i \)th locus and \( e \) is the environmental component, which is independent of the genotypic values and has zero mean and variance \( \sigma_e^2 \).

2. A complete description of the population in any generation is in terms of the total numbers \( N \) and the joint distribution of allelic effects \( F(x_1, \ldots, x_n; x_1', \ldots, x_n'; t) \). Assuming, as we are here, random mating, then after mating and before selection, the maternal and paternal gametes are independently distributed. We will also assume no sex-specific differences so that, before selection, \( F(x_1, \ldots, x_n; x_1', \ldots, x_n'; t) \) can be written as the product of the two sets of gametic effects
$F(x_1, \ldots, x_n; x_1', \ldots, x_n'; t) = f(x_1, \ldots, x_n; t) f(x_1', \ldots, x_n'; t)$. \hfill (18)

As shown by \text{Lande} (1976a), selection can induce correlations between the paternal and maternal gametes so such a separation is not possible after selection. We will denote $F$ after selection by $F_w(\ldots)$.

(3) In many cases, the effect of selection can be described in terms of the means, variances and covariances of the allelic contributions from each locus. Following the notation of \text{Lande} (1977), let $\bar{x}_i$ be the mean allelic effect at locus $i$, $C_{ij}$ be the covariance between either the maternal or paternal allelic effects at locus $i$ and locus $j$, and $C'_{ij}$ be the covariance between maternal and paternal allelic effects at locus $i$ and locus $j$ before selection. The quantities $\bar{x}_{iw}$, $C_{ijw}$ and $C'_{ijw}$ are similarly defined after selection. Both of these sets of quantities can be obtained by direct integration of $F$ or $F_w$. It follows from (18) that $C'_{ij} = 0$.

(4) Recursion formulae for $\bar{x}_i(t)$ and $C_{ij}(t)$ in terms of the selection acting each generation can be obtained if we assume that the regression of the $x_i$ on $z$ is linear. This would be the case if $f(x_1, \ldots, x_n; t)$ were multivariate normal, but it can be true for more general functional forms as well. Following \text{Lande} (1977), we write the regression before selection as

$$x_i - \bar{x}_i(t) = \frac{C_{iz}}{\sigma_z^2} (z - \bar{z}(t)) + e_{i,z}, \hfill (19)$$

where $e_{i,z}$ is the independent residual element of the variance, which we will assume to be homoscedastic (i.e., $e_{i,z}$ has the same variance for all $z$). It follows from the definition of the $C_{ij}$ that in (19)

$$C_{iz} = \sum_{j=1}^n C_{ij}(t). \hfill (20)$$

As discussed by \text{Lande} (1977), if selection acts only on $z$, then the regression of $x_{iw}$ on $z_w$ is still linear with the same regression coefficient and the same residual element of variance. From this fact, \text{Lande} (1977, p. 188) derived formulae for $C_{ijw}(t)$ in terms of the $C_{ij}(t)$ and the quantity

$$k(t) = 1 - \frac{\sigma_{z,w}^2(t)}{\sigma_z^2(t)}, \hfill (21)$$

where $\sigma_{z,w}^2$ is the variance of $z$ after selection:

$$C_{ijw}(t) = C_{ij}(t) - k(t)C_{iz}C_{jz} \frac{C_{iz}}{\sigma_z^2(t)} \hfill (22a)$$

and

$$C'_{ijw}(t) = - k(t)C_{iz}C_{jz} \frac{C_{iz}}{\sigma_z^2(t)} \hfill (22b)$$

(cf., \text{Lande} 1977, equations 6a and 6b). From (22), the recurrence formulae for the $C_{ij}(t)$ in the absence of mutations is

$$\Delta C_{ij} = - r_{ij} C_{ij}(t) - k(t)C_{iz}C_{jz} \frac{C_{iz}}{\sigma_z^2(t)}, \hfill (23)$$
where $r_{ij}$ is the recombination fraction between locus $i$ and $j$, which is 0 if $i = j$. Equation (23) is one of the two basic equations of the continuous genotype model. The other, for the change in the $\tilde{x}_i(t)$ is

$$
\Delta \tilde{x}_i = \frac{C_{ij}}{\sigma^2} [\tilde{z}_w(t) - \tilde{z}(t)]
$$

(\text{LANDE 1976a}).

Summing (24) over $i$ and multiplying by two, we obtain the usual formula for quantitative genetics

$$
\Delta \tilde{z}(t) = h^2 [\tilde{z}_w(t) - \tilde{z}(t)]
$$

(25)

where $h^2$, the heritability is $\sigma_g^2/\sigma^2$, the ratio of the additive genetic variance to the total variance, and

$$
\sigma_g^2 = 2 \sum_{i,j=1}^n C_{ij}.
$$

Before we can discuss the effect of frequency- and density-dependent selection of a character governed by the continuous genotype model, we must make an additional assumption. \text{KIMURA} (1965) showed that at a single locus subject to weak stabilizing selection, mutations that increase the variance would tend to make the distribution of $z$ normal. \text{LANDE} (1976a) used \text{KIMURA'S} result and assumed that $f(x_1, \ldots, x_n; t)$ is multivariate normal. He then showed that the multivariate normal form is approximately preserved under recombination, "nor-optimal" selection and mutation.

In these studies of density- and frequency-independent selection, the normal distribution of $z$ results from the balance between selection and mutation. In considering density and frequency-dependent selection in the absence of mutations, the assumptions of the continuous genotype model do not require that $z$ be normally or even approximately normally distributed. The function $f(x_1, \ldots, x_n)$ could, in principle, take any functional form and lead to a distribution of $z$ that is far from normal. Here, we will assume that $f$ has a form such that $z$ is approximately normally distributed. That would be the case if $f$ were multivariate normal, but, if the environmental component of the variance were sufficiently large, $z$ could be approximately normal for a larger variety of forms of $f$. The justification for this assumption is biological, not mathematical. The purpose of this paper is to investigate the effect of frequency- and density-dependent interactions that depend on the value of a normally distributed phenotypic character. The analysis in that problem is required by the large number of characters that are approximately normally distributed, at least on a transformed scale. In each of the three models that we consider for the genetic basis of such a character, we must impose an assumption to ensure normality. While much more complex models of quantitative characters that are not normally distributed can be constructed and analyzed, it is not yet clear what use they would have.

With the assumption that $z$ be normally distributed, we can compute $W(z)$ from the assumptions about the density- and frequency-dependent interactions.
From \( W(z) \), we can then compute \( \tilde{z}_w(t) \) and \( k(t) \). We will be concerned only with the equilibrium values of \( \tilde{z} \) and \( \sigma_z^2 \). From (25) we must have

\[
\tilde{z}_w = \tilde{z}
\]  

(26)

at equilibrium. Equation (23) with \( i=j \) implies that \( k=0 \) at equilibrium because \( r_{ii} = 0 \) by definition. Therefore

\[
\sigma_{z_0}^2 = \sigma_z^2
\]  

(27)

at any equilibrium for which the \( C_{ii} \) are not all zero. In addition, if \( k=0 \), then (23) with \( i\neq j \) imply that

\[
C_{ij} = 0
\]  

(28)

for all \( i\neq j \) because \( r_{ij} \neq 0 \) for \( i\neq j \), by assumption.

Therefore, we can characterize the equilibrium reached under frequency- and density-dependent selection by the conditions that the mean and variance of \( z \) are not changed by selection and by the condition that there be no linkage disequilibrium between any pair of the controlling loci. In fact, these are the conditions that must be satisfied at any equilibrium of the continuous genotype model when only selection, and not mutation and migration, is acting. However, nontrivial results are obtained only when there is frequency dependence. Whether the equilibrium described by these conditions is stable must be determined in each case. As is typical of models of involving frequency-dependent selection, the equilibrium conditions alone do not provide enough information to determine local stability.

The assumption that \( z \) be normally distributed in each generation does not affect the characterization of the equilibrium. However, without that assumption, the distribution \( p(z) \) would not be completely determined by the \( x_i \) and \( C_{ij} \). Therefore, without the assumption of normality, a complete solution for \( f \) would be required in order to compute \( p(z) \) and \( W(z) \).

We can illustrate these results by considering the model of selection given in equations (1) to (3). For this model, there is only one equilibrium possible at which there is a nonzero genetic variance. That equilibrium can exist only when

\[
\sigma_\eta^2 - \sigma_\alpha^2 > \sigma_e^2
\]  

(29)

and is given by

\[
\bar{z} = z_0, \quad \sigma_z^2 = \sigma_k^2 - \sigma_e^2, \quad N = \frac{k_0}{\sqrt{2\pi\sigma_z^2}}
\]  

(30)

The equilibrium values of \( \sigma_z^2 \) and \( \bar{z} \) are determined from (26) and (27). The value of \( N \) is then found from the condition that \( \bar{W} = 1 \), which follows from (4). There is a second equilibrium that is always present at which all \( C_{ij} = 0 \). At that equilibrium, any value of \( \bar{z} \) is possible and, for each \( \bar{z} \), there is an equilibrium value of \( N \).

In (30), only the values of \( \bar{z} \) and \( \sigma_z^2 \) are specified. The partitions of the allelic means and variances among the loci is arbitrary and the equilibrium is neutrally stable with respect to any perturbations of \( \bar{z}_i \) and \( C_{ij} \) that leave \( \bar{z} \) and \( \sigma_z^2 \)
unchanged. The same feature appeared in Lande's (1976a) analysis, but in that case, only the components of the mean are undetermined by the equilibrium condition. The components of the variance are fixed by the mutational pressure at each locus.

The particular choice of functions in (1) to (3) is such that, when (30) exists, $W(z)$ is exactly one and $f(x_1, \ldots, x_n)$ is multivariate normal. In fact, $f$ is the product of $n$ independent normal distributions because all the covariance terms are zero. In this case, the assumption about the normality of the distribution of $z$ is unnecessary, since that property follows from the structure of the model. By a straightforward analysis of the recursion equations for $N(t), \bar{x}_i(t)$ and $C_{ij}(t)$, it can be shown that, when (30) exists, it is stable for $r < 2$, which is the condition for the demographic stability of such a difference equation model of population growth. It also can be shown that the other equilibrium, with $\sigma_z^2 = \sigma_e^2$, is unstable when (29) is satisfied and stable when it is not.

Felsenstein (1977) obtained results similar to these for a slightly different model of frequency-dependent selection that also preserves multivariate normality. Bulmer (1974) used a selection model similar to the one used here, but he used a multiple-locus model with two alleles at each locus and required that the allele frequencies at each locus be the same at equilibrium. This assumption, which imposes a constraint on the equilibrium mean and variance similar to (7), leads to the prediction that $\sigma_{e^2} < \sigma_e^2$ at a stable equilibrium. Bulmer's (1974) result differs from equation (27) because of the constraint. Although the magnitude of the difference between the predictions of these two models is small, that difference has some important implications for Bulmer's analysis of ecological character displacement (Slatkin 1979b).

We can summarize the above results in a convenient form by rewriting (26) and (27) as

$$\bar{z} = \int z p(z) W(z) dz$$

and

$$\sigma_z^2 = \int (z - \bar{z})^2 p(z) W(z) dz,$$  \hspace{1cm} (32)

and then transforming them to the expressions

$$f \frac{\partial p(z)}{\partial \bar{z}} W(z) dz = 0$$

and

$$f \frac{\partial p(z)}{\partial \sigma_z^2} W(z) dz = 0.$$  \hspace{1cm} (34)

Equations (33) and (34) are obtained from (31) and (32) by using the assumption that $p(z)$ is normal with mean $\bar{z}$ and variance $\sigma_z^2$. Note that in (31) and (32), it is not necessary to renormalize the distribution after selection by dividing by $\bar{W}$ because $\bar{W} = 1$ at equilibrium. Lande (1976b) obtained a different expression of the equilibrium value of the mean reached under pure frequency dependence because, in that case, $\bar{W}$ is not necessarily one at equilibrium. His equation reduces to (33) when the additional condition required by the density dependence is imposed.
The equilibrium state reached under density- and frequency-dependent selection could be strongly affected by a steady flux of mutations at each locus. The mutations would, in general, act to increase $\sigma_z^2$ and the resulting equilibrium value would be larger than in the absence of mutations. The equilibrium in that case must have the property that selection each generation reduces the variance by the same amount as the mutations increase it. Consequently, the distribution of $z$ at equilibrium would have to be such that the frequency- and density-dependent interactions lead to stabilizing selection on $z$.

**PHENOTYPIC MODELS**

Both models considered so far are based on the assumption of a particular genetic basis for a phenotypic character. An alternative to this is a model based directly on assumptions about the relationship between the measures of a character in parents and offspring. We will consider only the simplest such model here to illustrate the equivalence of this approach to the genetical models when only equilibrium distributions are required. As before, consider a quantitative character, $z$, and assume that the distribution of $z$ in the offspring of any pair of parents is the same and is normal with mean $\bar{z}$ and variance $\sigma_z^2$. In genetical terms, we are assuming that the character has zero heritability, but that is an interpretation of this model in terms of a genetical model rather than a part of the phenotypic model itself.

Assume that there is frequency- and density-dependent selection described by a given function, $W(z)$. If $p(z)$ is normal with a fixed mean and variance, the only independent variable is $N$, which must be found from (4) with $N(t+1) = N(t)$, the condition that the mean absolute fitness be one. From (4), $N$ is implicitly a function of $\bar{z}$ and $\sigma_z^2$.

We next consider the effect of a modifier locus that affects $\bar{z}$ and $\sigma_z^2$. We then assume that there are two possible alleles, $M_1$ and $M_2$, at the modifier locus. This is a standard method for analyzing long-term evolutionary trends in genetically modifiable parameters and is more fully discussed in Slatkin (1978). Assume initially that the modifier locus is monomorphic for $M_1$ and that $N$ has reached the equilibrium value required by (4). Let that value be $N(\bar{z}, \sigma_z^2)$. Now assume that $M_2$ is introduced in a small frequency and that the offspring of the $M_1M_1 \times M_2M_2$ matings are distributed normally with mean $\bar{z} + \delta \bar{z}$ and variance $\sigma_z^2 + \delta \sigma_z^2$, where $\delta \bar{z}$ and $\delta \sigma_z^2$ are assumed to be small. Assume that $M_2$ can cause only one type of deviation in the offspring distribution. There must be some relationship between $\delta \bar{z}$ and $\delta \sigma_z^2$, unless it is also assumed that the mutant changes only the mean or the variance, in which case $\delta \bar{z}$ or $\delta \sigma_z^2$ is zero. We can express this relationship as

$$\delta \sigma_z^2 = \frac{d\sigma_z^2}{d\bar{z}} \delta \bar{z} = g(\bar{z}, \sigma_z^2) \delta \bar{z}$$

where the function $g$ characterizes the effect of the particular mutant.

In considering one mutant that is characterized by a particular function, $g$, we are imposing a constraint on the relationship between $\bar{z}$ and $\sigma_z^2$ of the same
type as in the one-locus model. In fact, (35) could be treated as a first-order
differential equation that could, in principle be solved to give \( \sigma_z^2 \) as a function
of \( \bar{z} \), as in (7). However, the use of modifier alleles to determine evolutionary
trends requires that different kinds of modifiers be considered. The basic idea,
which is discussed more fully in Slatkin (1978), is to determine the changes
in the population, given its current state and given the type of genetic variation
that is supposed to be present. Assumptions about the types of variation possible
must be an integral part of the model. That is, in using modifier alleles, the class
of possible modifiers must be clearly defined.

To illustrate the use of modifier alleles in the present context, consider
a modifier that changes only the mean, \( \bar{z} \), in the heterozygotes from \( \bar{z} \) to
\( \bar{z} + \delta \bar{z} \), but does not change \( \sigma_z^2 \). In the absence of more information about the
way in which the mean can evolve, we should consider modifiers with both
positive and negative values of \( \delta \bar{z} \). Different numerical values of \( \delta \bar{z} \) would lead
to different predictions, but we could analyze the whole class of modifiers at
once. If there were some biological reason that, at a given value of \( \bar{z} \), further
decreases were not possible, then we would require \( \delta \bar{z} > 0 \), thus restricting the
class of modifiers considered. If, in addition, we knew that increases in \( \bar{z} \) larger
than a certain magnitude were not possible, then we would have to impose an
upper bound on \( \delta \bar{z} \) as well. The key point is that the constraints, if any, on the
changes due to the modifiers must be imposed by conditions outside the model
itself.

In the present use of modifier alleles, a particular modifier is associated with
particular numerical values of \( \delta \bar{z} \) and \( \delta \sigma_z^2 \). All modifiers that preserve a certain
functional relationship between \( \bar{z} \) and \( \sigma_z^2 \) are characterized by a particular func-
tion, \( g \), in (35). Modifiers with both positive and negative effects would be in
that class. If there were some biological reason to constrain changes in \( \bar{z} \) and \( \sigma_z^2 \)
to (35), then that would be the only class of modifiers to consider. In the absence
of any such constraint, we must consider different types of modifiers that are
characterized by different functions. We will see that it is sufficient to consider
only one other such class, characterized by a different function, \( g'(\bar{z}, \sigma_z^2) \).

To determine the selection coefficient on \( M_z \) for the class of modifiers defined
by (35), we note first that the mean absolute fitness of the \( M,M \) individuals
is one, because, by assumption, the population was in equilibrium before \( M_z \) was
introduced, and second that \( M_z \) is sufficiently rare that the frequencies of the
other two genotypes do not change \( W \). The selection coefficient of \( M_z \) is then
found from the mean fitness of the heterozygotes alone because the \( M,M_z \) indi-
viduals are too infrequent to affect the change in the frequency of \( M_z \). If \( s \) is the
selection coefficient on \( M_z \), then, approximately

\[
s = \int p(z, \bar{z} + \delta \bar{z}, \sigma_z^2 + \delta \sigma_z^2) \ W(z) \, dz - 1 ,
\]

and by expanding \( p \) in a power series in \( \delta \bar{z} \)

\[
s = \delta \bar{z} \ s_1 + \delta \bar{z}^2 \ s_2 + o(\delta \bar{z}^3) ,
\]

where

\[
s_1 = \int \left( \frac{\partial p}{\partial \bar{z}} + g(\bar{z}, \sigma_z^2) \ \frac{\partial p}{\partial \sigma_z^2} \right) \ W(z) \, dz
\]

(38)
and $s_2$ is a similar term with second derivatives. Throughout, $W(z)$ is evaluated at $\bar{z}$, $\sigma_z^2$ and $\bar{N}(\bar{z}, \sigma_z^2)$.

From (37), we can determine the evolutionary trends in the parameters, $\bar{z}$ and $\sigma_z^2$ due to selection on any modifier that is characterized by the particular function, $g$. If we consider the class of such modifiers, including those with positive and negative values of $\delta \bar{z}$, then we can ensure that no such modifier could increase in frequency if

$$s_1 = 0$$

$$s_2 < 0 .$$

(39a)

(39b)

Equation (39a) is the condition for the equilibrium values of $\bar{z}$ and $\sigma_z^2$ and (39b) is the condition for their stability. Equations (38) and (39a) lead to the condition

$$\int \left( \frac{\partial p}{\partial \bar{z}} + g \frac{\partial p}{\partial \sigma_z^2} \right) W(z) dz = 0 ,$$

(40)

which must be satisfied if $\bar{z}$ and $\sigma_z^2$ are to be unchanged by the selection of modifiers with small effects of this type. As discussed in Slatkin (1978), modifiers with larger effects could move the population from the equilibrium values determined by (40), but modifiers with small effects would tend to return the population to those values.

We have obtained a condition for the evolutionary equilibrium reached by the mean and variance due to selection on one class of modifiers. However, unless there is some particular reason for restricting the possible evolutionary changes of modifiers of this type, we must consider other types of modifiers as well that are characterized by different functional relationships between $\bar{z}$ and $\sigma_z^2$. If we consider another class of modifiers for which the relationship between the mean and variance is determined by a different function, say $g'(\bar{z}, \sigma_z^2)$, then the above argument leads to the conclusion that, at the evolutionary equilibrium for modifiers of that type,

$$\int \left( \frac{\partial p}{\partial \bar{z}} + g' \frac{\partial p}{\partial \sigma_z^2} \right) W(z) dz = 0$$

(41)

must be satisfied.

In order to find the equilibrium conditions when there are assumed to be no constraints on $\bar{z}$ and $\sigma_z^2$, we must consider the implications of (40) and (41) for any pair of functions $g$ and $g'$. However, unless $g' = g$, (40) and (41) are two linearly independent equations that together imply

$$\int \frac{\partial p}{\partial \bar{z}} W(z) dz = 0$$

(42)

and

$$\int \frac{\partial p}{\partial \sigma_z^2} W(z) dz = 0$$

(43)

at the equilibrium values of $\bar{z}$ and $\sigma_z^2$. These are exactly the conditions obtained for the equilibrium reached under the assumptions of the continuous genotype model. [cf., equations (33) and (34).] The stability of the equilibrium has to
be checked separately. With this model, the conditions for stability are (42) and (43) with the first partial derivatives replaced by second derivatives. It is easy to show that those conditions guarantee the stability against selection of any type of modifier of the mean and variance. However, the conditions for stability obtained in this way are not necessarily the same as those obtained from the continuous-genotype model, although in many cases, the same conditions will hold.

DISCUSSION

My main conclusion is that, for a normally distributed quantitative character subject to frequency- and density-dependent selection, the equilibrium mean and variance of the character can be generally determined by considering the effect of selection only at the phenotypic level. This is the case in the above models when the mean and variance are not constrained. As discussed in Slatkin (1978), the genetic basis of the character can be important when fluctuating selection is considered, because the transient response to selection is determined by the relationship between the time scale of change in the selection function and the time scale of response of the phenotypic distribution. The latter time scale is determined by the details of the assumptions about the genetic basis of the character. In considering only the equilibrium conditions, however, the relative time scales do not enter, so that the equilibrium results do not depend on the genetic basis except through any constraints imposed.

In models involving frequency-dependent selection, there are many conditions under which the fitnesses of the different types in a population are equal at an equilibrium. The tendency for frequency-dependent selection to equilibrate fitnesses would be expected on intuitive grounds, and I have shown elsewhere (Slatkin 1979a) that if there is sufficient genetic flexibility in the population, then the fitnesses must be equal at any equilibrium. However, that result does not apply to the case of a normally distributed character. There are only two variables, the mean and variance of the character’s distribution, that can be changed and an effectively infinite number of types—individuals with different measures of the character—so that it is unlikely that all fitnesses can be equal at equilibrium. That is, only for special functional forms of $W(z)$ could $\bar{z}$ and $\sigma^2_z$ be chosen to make $W(z) = 1$ for all $z$. For the particular choice of $W(z)$ defined by equations (1) to (3), there are such values [equation (30)], and that fact leads to the determination of the stability criteria.

If $W(z)$ has even a slightly different form from that given in (1) to (3), $W(z)$ is no longer equal to one at the equilibrium. This can be illustrated with two simple examples, shown in Figures 1 and 2. In Figures 1 and 2, equations (1) and (3) were used, but in Figure 1, (2) was replaced by

\[
k(z) = k_o \quad |z| < 15
\]
\[
= 0 \quad |z| > 15
\]

and in Figure 2 by

\[
k(z) = k_d e^{-|z|/2\sigma_n^2} \quad z < 0
\]
\[
= k_d e^{-|z|/2\sigma_n^2} \quad z > 0 .
\]
2.0

\[ W(z) \]

\[ \tilde{W} \]

\[ z \]

\[ -24 \quad -12 \quad 0 \quad 12 \quad 24 \]

**Figure 1.** A graph \( W(z) \) vs. \( z \)—the solid line—at the equilibrium reached under the continuous genotype or phenotypic models, with \( W(z) \) defined by (1), (3) and (44). Also shown is the distribution of fitness as a function of genotypic values—the dashed line. The vertical marks indicate two standard deviations. \( (r = 0.9, z_0 = 0, \sigma_e^2 = 10, \sigma_2^2 = 50, \sigma_x^2 = 20.5) \). The results shown for these two cases are for both the continuous genotype model and for the phenotypic model with modifiers, as long as there are no constraints imposed in either model. The one-locus model would, in general, produce a different equilibrium, \( W(z) \).

When equations (1) to (3) are assumed, the results have some bearing on the “niche-variation” hypothesis of Van Valen (1965), which states that an increase in the diversity of food resources used by a species would lead to an increase in phenotypic variation in the species. Van Valen tested this hypothesis by comparing coefficients of variation in body size in bird species that were known to use a wide variety of food types with those that use a smaller variety. He found a greater coefficient of variation in the first group of species, but subsequent workers \( (e.g., \text{Soule and Stewart} 1970) \) have not found such a relationship in other species. The results here suggest that both the diversity of resources \( (\sigma_r^2) \) and the intensity of competition between individuals \( (\sigma_a^2) \) determine the equilibrium variance under frequency- and density-dependent selection. An increase in \( \sigma_r^2 \) would lead to an increase in \( \sigma_a^2 \) only when \( \sigma_a^2 \) did not increase by the same amount. In attempting to test the niche-variation hypothesis, both the diversity of resources and the intensity of competition within species would have to be taken into account.
These results also have some implications for the use of optimality criteria in predicting the outcome of natural selection. It is well known among population geneticists that the mean fitness or population size of a population subject to frequency-dependent selection is not maximized. But, there is still some tendency among ecologists to assume that the distribution of a character will adjust itself in some optimal manner. Such an assumption is usually made in the absence of other simple criteria for predicting the outcome of natural selection. For example, Roughgarden (1972) argues that, in a diploid species, the “niche width”, that is, the phenotypic variance, will evolve to maximize the total population size. Using the model of selection defined by equations (1) through (3), it can be shown that the population size is maximized as a function of the phenotypic variance at

$$\sigma^2_z = \sigma^2_w - \sigma^2_w/2,$$

which differs from the equilibrium variance reached in the absence of constraints by $\sigma^2_w/2$. In this model, the equilibrium population size is not strongly dependent on the phenotypic variance, but there can be a large difference between the equilibrium and “optimal” variance.

In considering a single phenotypic character, there is no a priori reason to impose any constraints on the way in which the mean and variance can change. However, when several characters are considered, there may be severe constraints on the changes in the mean and variance of any of them due to other characters.
For example, many characters are strongly correlated with body size, so that selection for increase in the mean or variance in some character would be constrained unless the mean and variance in body size changed correspondingly or unless the extent of correlation between body size and the character were changed. Insufficient attention has been given to the evolutionary importance of such constraints, although Lande (1979) has made significant progress on this problem.

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LITERATURE CITED


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