LONG-TERM CORRELATED RESPONSE, INTERPOPULATION COVARIATION, AND INTERSPECIFIC ALLOMETRY

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Abstract.—A model of long-term correlated evolution of multiple quantitative characters is analyzed, which partitions selection into two components: one stabilizing and the other directional. The model assumes that the stabilizing component is less variable than the directional component among populations. The major result is that, within a population, the responses of characters to selection in the short term differ qualitatively from those in the long term. In the short term, the responses depend on genetic correlations between characters, but in the long term they are only determined by the fitness functions of stabilizing and directional selection, independent of genetic and phenotypic correlations. Treating the stabilizing component as a constant and assuming the directional component to vary among populations, I present formulas for the interpopulation covariation and interspecific allometry, which are functions of the intensity matrix of stabilizing selection. Particular attention is paid to the relationship between intra- and interpopulation correlations.

Received April 21, 1987. Accepted September 21, 1987

One of the basic principles in quantitative genetics is that the correlated response of a character Y to selection on a character X is proportional to the genetic correlation between the characters X and Y (Falconer, 1981). This is equivalent to saying that, if selection is directional on some characters, the responses on other characters will be determined by the genetic correlations between the selected and unselected characters. Apparently this principle depends on the assumption that there is no correlational selection acting on the characters.

Correlational selection, usually analyzed in terms of multivariate stabilizing selection, could influence the prediction of the correlated response based on the information of genetic covariation alone. It could even change the whole pattern of response predicted from genetic correlations. If stabilizing selection is regarded as one of the common selection forces acting on quantitative characters in natural and artificial populations, its likely effect on the long-term correlated response should be taken into account in modeling of evolution of quantitative characters.

In this paper, I examine the effects of multivariate stabilizing and directional selection on long-term correlated response, interpopulation covariation, and interspecific allometry. First, I present a multivariate analysis of the effects of stabilizing and truncation selection on long-term correlated response, with the assumption of multivariate normal distributions of phenotypes and breeding values. This part is intended to model selection in laboratory experiments, with natural (stabilizing) selection toward some optimum phenotypes and artificial (truncation) selection directed to extreme phenotypes (Zeng and Hill, 1986). The result of this analysis shows that the responses of characters to selection in the short term differ qualitatively from those in the long term. In the short term, the responses depend on genetic correlations between characters as expected, but in the long term they are only determined by the fitness functions of stabilizing and directional selection, independent of genetic and phenotypic correlations.

To model natural population situations, two models of the differentiation of populations are analyzed in the second section to predict the pattern of interpopulation covariation, with specific reference to the relationship between intra- and interpopulation correlations. In the first model, it is assumed that an original population was maintained by stabilizing selection at the initial optimum and that a group of de-
scendant populations diverged from their common ancestral population as a result of directional selection following environmental changes or niche shifts. Each local population would then tend to move to its new local optimum, which depends on the direction and intensity of directional selection and the shape of stabilizing selection. In the second model, only directional selection is analyzed. Although both models tend to give a similar pattern of relationship between intrapopulation and interpopulation correlations, the causes are different in the two models. In the first model, the relationship is due to correlational selection, whereas in the second it is due to genetic correlation. Some caution in the study of interspecific allometry is also advised.

**Long-term Correlated Response to Stabilizing and Truncation Selection**

A common assumption in multivariate analysis of inheritance is that both phenotypes and genotypes of quantitative characters follow multivariate normal distributions, an assumption made in this analysis as well. This is essentially based on the central limit theorem that as the number of factors influencing genotypes as well as phenotypes increases, the distributions approach multivariate normality. For quantitative characters controlled by multiple genes, the assumption of normality is usually satisfied, at least approximately, after some appropriate scale transformation (Wright, 1968).

Consider a population that has been maintained at the optimum for n quantitative characters by stabilizing selection. To this population we apply artificial (truncation) selection on m of these characters toward extreme phenotypic values. These and other characters of the population will then evolve in an artificial environment along the directions determined by truncation and stabilizing selection. Eventually, the responses to selection will cease when a balance is reached between stabilizing and truncation selection. In this section, I investigate long-term responses to selection on these n characters.

Let \( x \) be a column vector of phenotypic measurements of these n quantitative characters and suppose that the corresponding vectors of additive genetic effects, \( y \), and environmental deviations, \( e \), follow independent multivariate normal distributions with \( x = y + e \), so that the probability-density functions of \( y \) and \( e \) are

\[
g(y) = (2\pi)^{-\frac{n}{2}} |G^{-1}|^{\frac{1}{2}} \cdot \exp \left\{ -\frac{1}{2}(y - \mu_y)^T G^{-1} (y - \mu_y) \right\}
\]

\[
h(e) = (2\pi)^{-\frac{n}{2}} |E^{-1}|^{\frac{1}{2}} \cdot \exp \left\{ -\frac{1}{2}(e - \mu_e)^T E^{-1} (e - \mu_e) \right\}.
\]

(1)

where \( \mu_y \) and \( \mu_e \) are the mean vectors of \( y \) and \( e \), and \( G \) and \( E \) are the genetic and environmental covariance matrices. For convenience, it is assumed that \( G \) and \( E \) are nonsingular and that \( \mu_e = 0 \). The superscript T denotes transposition. The distribution of \( x \) is then also multivariate normal, with mean \( \mu_x = \mu_y \) and covariance matrix \( P = G + E \), and is given by

\[
f(x) = (2\pi)^{-\frac{n}{2}} |P^{-1}|^{\frac{1}{2}} \cdot \exp \left\{ -\frac{1}{2}(x - \mu_x)^T P^{-1} (x - \mu_x) \right\}.
\]

(2)

The assumption of independent multivariate normal distributions of \( y \) and \( e \) implies that the regression of genetic effects on phenotypes is linear and homoscedastic with

\[
E(y | x) = \mu_y + GP^{-1}(x - \mu_x) \quad (3a)
\]

and

\[
\text{Var}(y | x) = (I - GP^{-1})G \quad (3b)
\]

where \( I \) is the identity matrix. A well known result due to Pearson (1903) is that selection on \( x \) does not influence the regression of \( y \) on \( x \). Therefore, the change in \( y \) due to selection on \( x \) can be inferred from the regression coefficients.

Let us first consider stabilizing selection. Stabilizing selection is taken to be a Gaussian function
\[ w(x) = \exp \left\{ -\frac{1}{2} (x - \theta)^T W^{-1} (x - \theta) \right\}, \]

where \( W \) is a positive definite symmetric matrix and \( \theta \) is the optimum vector. The matrix \( W \) is a measure of the intensity of stabilizing selection. The diagonal elements, \( w_{ii} \), of \( W \) approximate the strength of stabilizing selection acting directly on each character; higher values of \( w_{ii} \) reflect weaker stabilizing selection. The off-diagonal elements, \( w_{ij} (i \neq j) \), approximate the strength of correlational selection acting on different characters jointly, which will be shown to determine the correlated response to selection in the long term and also to be, in part, responsible for the interpopulation covariation (cf. Lande and Arnold, 1983; Lande, 1984).

With (2) and (4) the distribution of \( x \) after stabilizing selection has the density function

\[ f'(x) = \frac{f(x) w(x)}{\int f(x) w(x) \, dx}, \]

which is still multivariate normal. The denominator of the right side of (5) is a constant. The mean vector \( \mu_*' \) and covariance matrix \( P' \) of \( f'(x) \) can be found from

\[ P'^{-1} = P^{-1} + W^{-1} \]

and

\[ P'^{-1} \mu_*' = P^{-1} \mu_x + W^{-1} \theta \]

by equating like terms in (5), which gives \( \mu_*' = W(W + P)^{-1} (\mu_x + PW^{-1} \theta) \) and \( P' = W(W + P)^{-1} P \).

Now we consider truncation selection, in which those individuals that satisfy a certain criterion will be saved for reproduction and others will be discarded. The criterion can be based on a linear combination of multiple characters (index selection). Let \( I = b^T x \) be the selection index where \( b^T = [b_1 \ldots b_m \ 0 \ldots 0] \), \( m < n \), and \( b_i \) is the index coefficient of the \( i \)th character, and suppose that a fixed proportion of individuals with highest value on the index are selected for reproduction in each generation. After truncation the means of the characters become

\[ \mu_i^* = \mu_i' + \sum_{r=1}^{m} b_i \sigma_{i'r} \frac{v}{\sigma_{rr}} \]

\[ (i = 1, \ldots, n), \]

where \( v \) is the intensity of truncation selection, \( \sigma_{i'r} \) is the phenotypic covariance between the characters \( i \) and \( r \) after stabilizing selection and \( \sigma_{rr} = [b^T W (W + P)^{-1} P b]^{-1} \) is the standard deviation of the index. Since the variance of the index after truncation is known to be \( \sigma_{ii}^* = \sigma_{ii} [1 - v(v - z)] \) (e.g., Bulmer, 1980 p. 153) where \( z \) is the standard deviate of the truncation point, the variances and covariances of the \( x_i \)'s after truncation are

\[ \sigma_{ij}^* = \sigma_{ij} - \sum_{r=1}^{m} b_i \sigma_{ir'} \sum_{r=1}^{m} b_r \sigma_{rr'} \frac{v(v - z)}{\sigma_{rr'}} \]

\[ (i, j = 1, \ldots, n) \]

by using the result of Pearson (1903) and Aitken (1934). Equations (6) and (7) can be expressed in matrix notation as

\[ \mu_y^* = W(W + P)^{-1} (\mu_x + PW^{-1} \theta) + W(W + P)^{-1} Pa \]

and

\[ P^* = W(W + P)^{-1} P - W(W + P)^{-1} \cdot Pbb^T P(W + P)^{-1} Wc \]

with \( a = b \nu / \sigma_{ii} \) and \( c = \nu(v - z) / \sigma_{ii} \).

It follows immediately that the means and covariances of \( y \) after selection are

\[ \mu_y^* = E[y | x] = \mu_y + \text{GP}^{-1} [W(W + P)^{-1} \cdot (\mu_x + PW^{-1} \theta + Pa) - \mu_x] \]

\[ = \mu_y + G(W + P)^{-1} \cdot (\theta + Wa - \mu_x) \]

\[ G^* = E[y^2 | x] - \mu_y^2 = \text{GP}^{-1} P^* \text{P}^{-1} G + (I - \text{GP}^{-1}) G \]

\[ = G - \text{GP}^{-1} (I - P^* P^{-1}) G \]

from (3), (8), and (9).

If mating is random and there is no sexual dimorphism, the mean genetic values of the offspring will equal those of their parents (Falconer, 1981). That is, if we let \( \mu_* (t) \) be the vector of means of \( y \) after selection in generation \( t \) and \( \mu_* (t + 1) \) be that before
evolution of the vector of means

\[ \mu_{t+1} = \mu_r + G_t(W + P_r)^{-1} \theta + Wa_r - \mu_r \] 

(13)

from (10) and (12), where \( G_t \) and \( P_r \) are the genetic and phenotypic covariance matrices in generation \( t \).

Changes in \( G_t \) are influenced not only by selection, but also by mutation, recombination, and the genetic structure of the population. If the genetic variances and covariances can be maintained in the population by a balance among selection, mutation, and recombination (e.g., Lande, 1980b; Turelli, 1985), \( G_t \) will converge to an equilibrium genetic covariance matrix \( G_{\infty} \) which is positive definite (Karlin, 1979).

In this case, a balance for the population mean vector will also be reached, which is given by

\[ \mu_{\infty} = \theta + Wa_{\infty} \] 

(14)

from (13) by letting \( \mu_{t+1} = \mu_r \).

It is of interest to compute the response in the first generation of selection when \( \mu_0 = \theta \). This is

\[ \mu_1 = \theta + G_t(W + P_r)^{-1}(\theta + Wa_r - \theta) \]

\[ \approx \theta + G_t(I + P_rW^{-1})^{-1}a_r \]

(15)

for weak stabilizing selection [compare with (14)]. Furthermore, if \( G_t \) and \( a_r \) are assumed to be constant across generations, we can find, from (13),

\[ \mu_1 = \theta + Wa_r - [I - G_t(W + P_r)^{-1}]' \cdot (\theta + Wa_r - \mu_0). \]

(16)

Equation (14) shows that the means of \( x \) and \( y \) at the selection limit are independent of both the genetic covariance matrix \( G \) and the phenotypic covariance matrix \( P \). They are determined only by the two kinds of

<table>
<thead>
<tr>
<th>Character</th>
<th>( a_r )</th>
<th>( h^2 )</th>
<th>( w_{hi} )</th>
<th>( x_1 )</th>
<th>( x_2 )</th>
<th>( x_3 )</th>
<th>( x_4 )</th>
<th>( x_5 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( x_1 )</td>
<td>1.0</td>
<td>0.2</td>
<td>12.0</td>
<td>-</td>
<td>0.15</td>
<td>-0.20</td>
<td>-0.50</td>
<td>0.45</td>
</tr>
<tr>
<td>( x_2 )</td>
<td>2.0</td>
<td>0.4</td>
<td>30.0</td>
<td>0.40</td>
<td>-</td>
<td>0.20</td>
<td>-0.40</td>
<td>-0.05</td>
</tr>
<tr>
<td>( x_3 )</td>
<td>0.8</td>
<td>0.6</td>
<td>14.4</td>
<td>0.20</td>
<td>0.25</td>
<td>-</td>
<td>-0.25</td>
<td>0.20</td>
</tr>
<tr>
<td>( x_4 )</td>
<td>0.5</td>
<td>0.5</td>
<td>5.0</td>
<td>-0.25</td>
<td>-0.35</td>
<td>0.05</td>
<td>-</td>
<td>-0.35</td>
</tr>
<tr>
<td>( x_5 )</td>
<td>1.5</td>
<td>0.8</td>
<td>15.0</td>
<td>0.60</td>
<td>0.35</td>
<td>0.20</td>
<td>-0.20</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 1. Hypothetical parameters of the population and the intensities of stabilizing selection. Genotypic correlations are above the diagonal and phenotypic correlations are below. Heritability \( (h^2) \) is the ratio of genetic variance to phenotypic variance.
Table 2. Correlations in $W$ matrix for the example of the effects of correlational selection depicted in Figure 2.

<table>
<thead>
<tr>
<th>Character</th>
<th>$x_2$</th>
<th>$x_3$</th>
<th>$x_4$</th>
<th>$x_5$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$x_1$</td>
<td>-0.05</td>
<td>0.35</td>
<td>0.10</td>
<td>-0.40</td>
</tr>
<tr>
<td>$x_2$</td>
<td></td>
<td>-0.25</td>
<td>0.20</td>
<td>-0.10</td>
</tr>
<tr>
<td>$x_3$</td>
<td></td>
<td></td>
<td>-0.15</td>
<td>-0.05</td>
</tr>
<tr>
<td>$x_4$</td>
<td></td>
<td></td>
<td></td>
<td>0.10</td>
</tr>
</tbody>
</table>

Selection intensities, one from stabilizing selection embodied in the matrix $W$ and the other from truncation selection embodied in the vector $\mathbf{a}$ (where $\mathbf{a} = \mathbf{b}/\sigma_i$). In other words, the whole complicated system of genetic and phenotypic correlations becomes irrelevant in determining the means at the limit to stabilizing and truncation selection, although the means do depend on $\mathbf{G}$ and $\mathbf{P}$ matrices before $\mu_\infty$ is reached. This indicates that the correlated responses of characters to selection depend primarily on the genetic correlations between characters in the short term (15), but in the long term these correlated responses depend more on the influence of correlated stabilizing selection (14). Indeed, if stabilizing selection acts independently on the characters, i.e., the off diagonals, $w_{ij}$ ($i \neq j$), of $W$ are zero, the characters will evolve independently in the long term, even though they are correlated genetically (except when the genetic correlations are perfect, i.e., $-1$ or $1$). The means of those characters that are not directly truncated will finally return to their original optimum values, no matter what kind of correlated responses from directional selection they have experienced previously, a situation illustrated in Figure 1 (for the hypothetical parameters given in Table 1). But if $w_{ij} \neq 0$ ($i \neq j$), as seems likely, the means of nontruncated characters will tend to respond to directional selection through the correlated influence of stabilizing selection (Fig. 2, Table 2). The degree of the correlated change between the two characters is determined by $\gamma_{w_{ij}} = w_{ij}/\sqrt{w_{ii}w_{jj}}$.

**Interpopulation Covariation**

Evolution of correlated characters in natural populations is sometimes considered as a process of optimum shift due to environmental change (e.g., Lande, 1980a). In this model, environmental change or niche shifts would shift the optimum phenotype and cause directional selection toward the new optimum phenotype. Letting $\Phi$ be the new optimum, the new fitness function would be changed from (4) to

$$w(x) = \exp \left\{ -\frac{1}{2} (x - \Phi)^T W^{-1} (x - \Phi) \right\},$$

which is equivalent to

$$w(x) = \exp \left\{ \mathbf{a}^T x - \frac{1}{2} (x - \theta)^T W^{-1} (x - \theta) \right\},$$

where $\mathbf{a} = W^{-1} (\Phi - \theta)$ is the intensity vector of directional selection. One of the assumptions implicit in this model is that all char-
acters considered are adaptively significant, so that there is at least one fixed optimum vector of the population phenotypes in an environment. For a population adapting to a new environment, however, it is possible that only a few characters are critical for adaptation and are the direct targets of selection. Other characters may evolve as a consequence of correlational selection and genetic correlations. Therefore, the final population means might depend on the balance of different forces of selection and the genetic structure of the population, if we ignore the effect of random genetic drift.

Moreover, the optimum-shift model predicts that, if different populations are assumed to be at different adaptive peaks, there is no necessary relationship between multivariate patterns of covariation within and between populations. Although there has been some debate about the existence of the positive relationship between multivariate patterns of variation within and between populations (Klug and Kerfoot, 1973; Sokal, 1976, 1978; Johnson and Mickevich, 1977; Pierce and Mitton, 1979; Baker, 1980; Atchley et al., 1982; Rohlf et al., 1983), the positive relationship between intra- and interpopulation correlations is consistent over published data for morphological characters of *Pemphigus* (Sokal, 1962; Sokal et al., 1980; Sokal and Riska, 1981) and the tick *Haemaphysalis leporispalustris* (Thomas, 1968). The data show that all high intrapopulation correlations are associated with high interpopulation correlations and that the magnitudes of interpopulation correlations are generally higher than those of corresponding intrapopulation correlations, whereas intermediate and low level intrapopulation correlations could be paired with a relatively wide range of interpopulation correlations. Since the measurement of correlation is unitless and free from the influence of sample mean, the relationship between the correlations must be more reliable than the relationship between the variances. Thorpe (1976) and Sokal (1978) have postulated that the development of interpopulation correlations is circumscribed by the nature of intrapopulation correlation affecting the characters concerned. To account for these relationships between intra- and interpopulation correlation, I consider two selection models that can explain the development of interpopulation correlation.

Model I: Stabilizing and Directional Selection. — In modelling the differentiation of quantitative characters among populations, I assume that all populations sampled for study were originally from one population which was maintained by stabilizing selection at the initial equilibrium \( \theta \). The populations diverged from their common ancestor, due to different directional selection in different local populations as a result of environmental changes or niche shifts. Let \( \mathbf{a}_k \) be the intensity vector of directional selection on the characters in the \( k \)th population and suppose that \( \mathbf{a}_k \) varies from population to population with mean vector \( \mathbf{a} \) and covariance matrix \( \mathbf{A} \) among the populations. Then, assume that for the \( k \)th population the fitness function is changed to

\[
  w_k(x_k) = \exp \left\{ \mathbf{a}_k^T x_k - \frac{1}{2} (x_k - \theta)^T \mathbf{W}^{-1} (x_k - \theta) \right\}.
\]  

(17)

We thus have also assumed in Equation (17) that the intensity matrix of stabilizing selection, \( \mathbf{W} \), is constant among populations. Biologically, this means that, for all the populations concerned, individuals are selected approximately in the same fashion, although the equilibrium combinations of characters may differ because of the variation in the vector \( \mathbf{a} \) among populations. This may not be true, of course. In reality, there must be some variation in the \( \mathbf{W} \) matrix among populations as well. That is, different environments may impose different intensities or even different patterns of stabilizing selection on characters. Such a situation is likely to happen for distantly related populations or species; however, for closely related populations or species, the variation of the \( \mathbf{W} \) matrix among populations might be small enough to be ignored without serious error.

The fitness function (17) is equivalent to

\[
  w_k(x_k) = \exp \left\{ -\frac{1}{2} (x_k - \theta - \mathbf{W} \mathbf{a}_k)^T \right\} \cdot \mathbf{W}^{-1} (x_k - \theta - \mathbf{W} \mathbf{a}_k) \right\}.
\]
That is, the population is selected toward a new equilibrium, \( \theta + W\mathbf{a}_k \), which will eventually be approached regardless of the pattern of genetic covariation of the population, unless genetic variation is absent in some selected characters (\(|G| = 0\)) (Lande, 1979; Via and Lande, 1985; present study [see above]). There is a difference between this model and the optimum-shift model (e.g., Lande, 1980a). In the present model, the "new optimum" resulting from the change of directional selection intensities on some critical characters is influenced by the shape of the fitness function. In the optimum-shift model, the new optimum is preassigned to that environment and has no necessary relationship with the shape of the fitness function. Thus, the interpopulation covariation under the optimum-shift model will depend on the geographical and chronological pattern of adaptive peaks, which may have no necessary relationship with the pattern of the fitness function and the pattern of genetic and phenotypic covariation of the population.

In the present model, the covariance matrix, \( \mathbf{K} \), among the population means at the new equilibria is expected to be

\[
\mathbf{K} = \mathbf{W}(\mathbf{E}(\mathbf{a}_k) - \alpha \mathbf{a}_k^T)^T \mathbf{W}
\]

since \( \mathbf{E}(\mathbf{a}_k) = \mathbf{W} \mathbf{E}(\mathbf{a}_k) = \mathbf{W} \alpha \) (after taking \( \theta = 0 \)), which is a product of three covariance or covariance-like matrices, two of them being the intensity matrix of stabilizing selection. In equation (18), \( \mathbf{A} \) is the covariation matrix of \( \mathbf{a}_k \) among the populations.

Now we turn to the problem of the relationship between intra- and interpopulation correlations. The intensity and pattern of stabilizing selection, \( \mathbf{W} \), can directly influence the maintenance of intrapopulation phenotypic and genetic variation and covariation (Lande, 1975, 1980b, 1984; Turrielli, 1985). It is generally expected that, in the face of strong stabilizing selection, less intrapopulation variation and covariation will be maintained and also less interpopulation variation and covariation will result [Equation (18)]. In this way, the intrapopulation variations and covariations are linked by stabilizing selection. A comparison between the correlation structures of \( \mathbf{W} \) and \( \mathbf{K} \) might give some indication about the relationships between intrapopulation correlations. Let us consider first a simple, two-character case in which

\[
\mathbf{K} = \begin{bmatrix}
  k_1^2 & \gamma_{k1}k_2 \\
  \gamma_{k1}k_2 & k_2^2
\end{bmatrix},
\]

\[
\mathbf{W} = \begin{bmatrix}
  w_1^2 & \gamma_{w1}w_2 \\
  \gamma_{w1}w_2 & w_2^2
\end{bmatrix},
\]

and

\[
\mathbf{A} = \begin{bmatrix}
  a_1^2 & \gamma_{a1}a_2 \\
  \gamma_{a1}a_2 & a_2^2
\end{bmatrix},
\]

where \( k_1^2 \) is the variance of character 1 among population means, \( w_1^2 \) is the intensity of stabilizing selection on character 1, \( a_1^2 \) is the variance of the directional-selection differential on character 1 among populations, \( \gamma_{k1} \) is the interpopulation correlation between characters 1 and 2, \( \gamma_{w1} \) is the selective correlation imposed by stabilizing selection within populations, and \( \gamma_{a1} \) is the selective correlation exerted by directional selection among populations. Then, from (18) we have

\[
k_1^2 = w_1^2[\gamma_{w1}w_1w_2a_1a_2 + \gamma_{w1}w_2w_2a_2^2],
\]

\[
k_2^2 = w_2^2[\gamma_{w1}w_1w_2a_1a_2 + \gamma_{w2}w_2w_2a_2^2],
\]

\[
\gamma_{k1}k_2 = \gamma_{w1}w_1w_2[w_1^2a_1^2 + \left( \frac{1}{\gamma_{w1}} + \gamma_{w2} \right) \cdot \gamma_{w1}w_1w_2a_1a_2 + w_2^2a_2^2],
\]

and

\[
|\gamma_k| \geq \left| \frac{2\gamma_{w1} + \gamma_{a1}(1 + \gamma_{w2})}{1 + \gamma_{w1}^2 + 2\gamma_{w1}\gamma_{a1}} \right|,
\]

which is equal only when \( w_1a_1 = w_2a_2 \). If \( \gamma_{w1} \) and \( \gamma_{a1} \) have the same sign, then

\[
|\gamma_k| \geq \left| \frac{2\gamma_{w1}}{1 + \gamma_{w1}^2} \right| \geq |\gamma_{w1}|. \quad (19)
\]

This inequality tells us that the value of \( \gamma_k \) is generally expected to be larger in magnitude than \( \gamma_{w1} \) unless \( \gamma_{a1} \) differs from \( \gamma_{w1} \) in
Table 3. Interpopulation correlation ($\gamma_k$) for different combinations of values of $\gamma_w$, $\gamma_a$, and $d_2$, with $a_1 = 1$ and $w_1^2 = w_2^2 = 20$.

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<th>$\gamma_w$</th>
<th>$\gamma_a = -0.3$</th>
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<th>0.01</th>
<th>0.1</th>
<th>0.01</th>
<th>0.1</th>
<th>0.01</th>
<th>0.1</th>
<th>0.01</th>
<th>0.1</th>
<th>0.01</th>
</tr>
</thead>
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<td>0.99</td>
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</tbody>
</table>

Note that if directional selection acts only on one character, i.e., either $a_1$ or $a_2 = 0$, then $\gamma_k = 1$, regardless of the values of $\gamma_w$, $\gamma_a$, and $w_2$. When $\gamma_w = 0$, then $\gamma_k = \gamma_a$, unless $a_1$ or $a_2 = 0$. Some examples of $\gamma_k$ values for different combinations of values of $\gamma_w$, $\gamma_a$, and $d_2$ are given in Table 3. If the intrapopulation correlation is generally not very different from $\gamma_w$ in value, Table 3 shows that a high intrapopulation correlation should always be associated with a high interpopulation correlation of the same sign, but a low intrapopulation correlation could be paired with a low or high, positive or negative, interpopulation correlation. This is the pattern of relationship postulated by Thorpe (1976) and Sokal (1978) and observed most clearly in the data set of Sokal and Riska (1981). The result of Smith (1981) also supports this relationship. The inequality (19) can be extended to a general $n$-character case in which

\[
|\gamma_{kj}| \geq \frac{\sum_{i=1}^{n} \gamma_{wij} \gamma_{wij}}{\left(\sum_{i=1}^{n} \gamma_{wij}^2 \sum_{i=1}^{n} \gamma_{wij}^2\right)^{1/2}} \geq |\gamma_{wij}|. \tag{20}
\]

Model II: Directional Selection. Alternatively, we could analyze the interpopulation variation without stabilizing selection, and consider directional selection only. Let us consider, for example, the fitness function (17) without the quadratic term and let $\mu_0 = 0$. Then we have

\[
\mu_k = \sum_{i=1}^{t} G_k/a_{ki}, \tag{21}
\]

which is analogous to equation (15), where $t$ is the number of generations since population $k$ diverged from the base population. Note that in this model there is no theoretical limit to selection as long as $|G| > 0$. If $G_k$ is assumed to be approximately constant across the generations, (21) can be written as

\[
\mu_k = G_k \sum_{j=1}^{t} a_{kj} = G_k c_k \tag{22}
\]

where $c_k$ is interpreted as the cumulative selection differential vector in the $k$th population. Let $c_k$ have mean vector $\beta$ and covariance matrix $C$ among populations, and further assume that $G$ is also approximately the same in different populations. The interpopulation covariance matrix then becomes

\[
K = GCC \tag{23}
\]

after applying an argument similar to that in model I. Similarly, let

\[
G = \begin{bmatrix} g_1^2 & \gamma_\beta g_1 g_2 \\ \gamma_\beta g_1 g_2 & g_2^2 \end{bmatrix}
\]

and

\[
C = \begin{bmatrix} c_1^2 & \gamma_c c_1 c_2 \\ \gamma_c c_1 c_2 & c_2^2 \end{bmatrix},
\]

in which $g_1^2$ and $g_2^2$ are the additive genetic variances of characters 1 and 2, $\gamma_\beta$ is the genetic correlation of characters 1 and 2, and $c_1^2$, $c_2^2$, and $\gamma_c$ are the variances and correlation of the cumulative selection differentials among populations. Then,

\[
|\gamma_k| \geq \frac{2\gamma_\beta + \gamma_c(1 + \gamma_\beta^2)}{1 + \gamma_\beta^2 + 2\gamma_\beta \gamma_c} \geq \frac{2\gamma_\beta}{1 + \gamma_\beta^2} \geq |\gamma_\beta|, \tag{24}
\]
LONG-TERM CORRELATED RESPONSE

if \( \gamma_8 \) and \( \gamma_c \) have the same sign. This is certainly an oversimplified calculation since, in reality, \( G \) must change from generation to generation and from population to population. The change of \( G \) over generations and populations does not, however, alter the qualitative prediction of the relationship between \( \gamma_8 \) and \( \gamma_c \) under this model.

We have analyzed two selection models: model I, with stabilizing and directional selection, predicts the relation \( \mu_k = W_a k + \theta \) at equilibrium and the interpopulation covariance matrix \( K = WAW \); and model II, with directional selection only, predicts \( \mu_k = Gc_k + \theta \) and, consequently, the interpopulation covariance matrix \( K = GCG \).

Clearly, in model II, the covariation of characters among populations is related to the covariation within populations through the genetic covariance matrix. This is a consequence of the so-called Secondary Theorem of Natural Selection (Robertson, 1968; Crow and Nagylaki, 1976) which states that the rate of change in a character is a function of its additive covariance with fitness. However, it should be pointed out that this theorem applies only when the character is linearly related to fitness. When the relationship is quadratic, such as the fitness function (17) in model I, the rate of change will also depend on the quadratic relationship of the characters with fitness. This is especially so in the long term [Equation (14)], and, consequently, the interpopulation covariation in model I is a function of this quadratic relationship [Equation (18)].

**Interspecific Allometry**

The term “allometry” generally refers to the relationships between body size and size-related characters. There are three types of allometry: i) ontogenetic or growth allometry (relationships between characters in a single growing individual); ii) intraspecific or static allometry (between different individuals all at the same stage of growth); and iii) interspecific or evolutionary allometry (between different species at the same stage of growth) (Huxley, 1932; Gould, 1966, 1977; Cheverud, 1982; Fleagle, 1985; Lande, 1985). The widely used allometric formula is the regression equation

\[
\log(Y) = \log(d) + \log(X),
\]

where \( X \) is body size, \( Y \) is another character, \( d \) is a constant, and \( b \) is the allometric coefficient. Denoting the logarithms of body size and the other character as \( x = \log(X) \) and \( y = \log(Y) \) with phenotypic standard deviations \( p_x \) and \( p_y \), and correlation \( \gamma_p \), the allometric coefficient for intraspecific allometry can be expressed as the phenotypic regression coefficient within population

\[
b = \frac{\gamma_p p_y}{p_x}.
\]

For interspecific allometry, Reeve (1950) and Lande (1979) noticed that, if species diverged morphologically because of natural selection acting on overall body size and change in other characters is merely a correlated response (a common null hypothesis in the comparison of closely related species), there would be a precise relationship between interspecific allometry and genetic allometry of characters within populations. This can be shown as follows (Lande, 1979). Let

\[
R_x = \frac{g^2}{2 p_x^2} S_x
\]

and

\[
R_y = \frac{\gamma_8 g_x g_y}{2 p_x^2} S_x
\]

be the direct response of \( x \) and the correlated response of \( y \) to selection for \( x \), respectively, where \( S_x \) is the selection differential for \( x \), \( \gamma_8 \) is the genetic correlation, and \( g_x \) and \( g_y \) are the genetic standard deviations. The ratio of the correlated response of \( y \) to the direct response of \( x \) will be

\[
\frac{R_y}{R_x} = \frac{\gamma_8 g_y}{g_x}.
\]

“If the relative values of the genetic covariances do not change much during evolution, the [evolutionary] graphs will tend to be straight lines on logarithmic coordinates, that is, ‘allometric’ in the sense of Huxley (1932)” (Lande, 1979 p. 404). This is a simple and elegant illustration of the relationship between the coefficients of interspecific allometry and genetic regression.
Lande (1979) has also analyzed the case of differentiation of populations by random genetic drift, which gives the same allometric slope as above.

This interpretation of interspecific allometry has become popular in the literature. As a result, genetic allometry within populations has frequently been used synonymously with interspecific allometry, and the relationship between phenotypic and genetic covariance patterns has been used to explain the similarity or difference between observed intraspecific and interspecific allometry (Atchley and Rutledge, 1980; Cheverud, 1982; Wolpoff, 1985). Since the issue is fundamental to the study of allometry, some underlying assumptions of Lande’s (1979) analysis must be noted. As emphasized by Lande (1979), the expected correspondence between interspecific allometry and genetic regression holds only if the character y is strictly neutral during the process of evolution. Any sort of selection on y may destroy the precise connection between the two coefficients. Equation (25) is only a predictor of the interspecific allometry under the specific model, assuming no selection on y. This can be seen clearly from the formulas of interpopulation covariation derived above. For example, the coefficient of interspecific allometry under model II, given in Equation (26) (below), reduces to (25) only when \( c_y = 0 \) (\( c_y^2 \) is the variance of the cumulative directional selection differentials on y among the populations compared). Thus, selection on y will bring about a higher interspecific slope than that predicted by (25) (if \( \gamma_y > 0 \)), as noted by Lande (1979) in the discussion of brain : body size allometry.

Moreover, if there is stabilizing selection on body size and size-related characters in addition, the expected interspecific allometry is given by Equation (27) (below) from model I. In this case, the seemingly obvious relationship of (25) does not exist in the long term. The interspecific allometry will depend on the shape as well as the intensities of the selection function.

**Discussion**

The long-term correlated evolution of quantitative characters is analyzed in this paper as a process of the joint influence of stabilizing and directional selection under the assumption that differentiation of populations is due to different intensities of directional selection on some adaptively significant characters. The process that is modelled is similar to that involved in animal and plant improvement, in which directional selection is applied primarily on some economically important characters. I analyze this model as a complement to the directional-selection-only model (e.g., Lande, 1979) and Lande’s (1980a) optimum-shift model. The major characteristic of this model is that long-term correlated responses to selection are determined by the shape of the fitness function rather than by genetic covariation. As a result, interpopulation covariation and interspecific allometry are shown to be functions of the intensity matrix of stabilizing selection.

The model described in this paper has some limitations. First, it relies on the assumption of approximately constant intensity matrix, W, among populations. Although it does not seem unreasonable to
assume that the W matrix is less variable among closely related populations compared with the intensity vector of directional selection, a, there is no empirical evidence to justify this. Variation of the W matrix among populations would not influence the prediction of long-term response within a population, however, since the response depends on only the intensities of selection acting on that population, but would complicate prediction of interpopulation covariation and cause departure from the simple theory. Second, the results depend on a multivariate normal relationship between phenotypes and breeding values. This means that, to satisfy the assumption, not only must the number of genes influencing the characters be large, but also the effects of genes probably need to be additive, and environmental deviations must be normally distributed. In spite of these limitations, the analysis still has some general implications. It reveals some qualitative features about correlated evolution of quantitative characters, which have not been described previously.

Finally, it should be pointed out that the predictions for long-term response and interpopulation covariation do not necessarily depend on the assumption of truncation as the type of directional selection. Although truncation selection was used in the first part of the analysis in order to model the type of selection in plant and animal breeding, other types of directional selection could give similar results.

ACKNOWLEDGMENTS

I thank C. C. Cockerham, W. G. Hill, R. Lande, T. F. C. Mackay, B. Riska, H. Tachida, and M. Turelli for helpful discussions of this topic and comments on earlier drafts of this paper. This is paper No. 11016 of the Journal Series of the North Carolina Agricultural Research Service, Raleigh, NC 27695-7601. This investigation was supported in part by National Institute of Health Research Grant GM 11546 from the National Institute of General Medical Science.


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Corresponding Editor: M. Slatkin