Genet. Res., Camb. (1989), **53**, *pp.* 215–221 *Printed in Great Britain*

A genetic model of interpopulation variation and covariation of quantitative characters

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(Received 14 March 1988 and in revised form 27 October 1988)

Summary

Evolutionary consequences of natural selection, migration, genotype–environment interaction, and random genetic drift on interpopulation variation and covariation of quantitative characters are analysed in terms of a selection model that partitions natural selection into directional and stabilizing components. Without migration, interpopulation variation and covariation depend mainly on the pattern and intensities of selection among populations and the harmonic mean of effective population sizes. Both transient and equilibrium covariance structures are formulated with suitable approximations. Migration reduces the differentiation among populations, but its effect is less with genotype–environment interaction. In some special cases of genotype–environment interaction, the equilibrium interpopulation variation and covariation is independent of migration.

1. Introduction

Whether population differentiations is related to within-population variation is an interesting question in evolutionary biology. Kluge & Kerfoot (1973) observed some positive correlation between the amounts of interlocality differentiation and intra-population variance for several morphometric characters. Subsequently, the 'Kluge–Kerfoot phenomenon' was reported in several other studies (Sokal, 1976, 1978; Johnson & Mickevich, 1977; Pierce & Mitton, 1979; Baker, 1980; Atchley, Rutledge & Cowley, 1982). However, Rohlf, Gilmartin & Hart (1983) showed that these observations did not prove the existence of the correlation between interlocality differentiation and intralocality variation, and that the 'Kluge–Kerfoot phenomenon' could be simply a statistical artifact because '[t]he observed correlation is due to the measures of within- and among-population variability both being functions of a third variable – the sample mean.'

Theoretically, if a group of populations diverge for a short time, a positive relationship between within- and among-population variation is expected. Both neutral and selection models predict that the differentiation of populations is directly related to within-population genetic variation. In the long term, however, the relationship becomes less clear. Conventionally, it is regarded that in the long term populations could converge to their local optima (Bulmer, 1971*a*,*b*; Lande, 1980*a*). If these optima display a geographic cline or some ecological patterns (Felsenstein, 1977; Slatkin, 1978), among-population variation would not necessarily be related to within-population variation.

It is plausible that not all characters have local optimum values. Characters are likely to evolve together; while some shift to their new local optima, others change as a consequence of genetic correlation in the short term and selective correlation in the long term (Zeng, 1988). In a model incorporating constant stabilizing selection and spatially differential directional selection (Zeng, 1988), it is shown that at equilibrium among-population variation could still be related to within-population variation, but indirectly, through the joint influence of stabilizing selection on the maintenance of within-population variation and the development of among-population variation.

In this paper, the previous work is extended to include migration, genotype–environment interaction, and random genetic drift. I will begin with an introduction of the selection model and explore further some of its properties. Then I will treat migration, genotype–environment interaction, and random genetic drift individually in different sections.

2. The model

Consider a group of isolated populations which diverged from a common ancestral population due to disruptive selection caused by environmental changes or niche shifts. It is assumed initially that population

size is infinite in each population and there is no migration or genotype–environment interaction, but later these assumptions will be removed.

Let $\boldsymbol{x}$ be a column vector of phenotypic measurements of n quantitative characters and suppose that the corresponding vectors of additive genetic effects, $\boldsymbol{y}$, and environmental deviations, $\boldsymbol{e}$, follow independent multivariate normal distributions with $\boldsymbol{x} = \boldsymbol{y} + \boldsymbol{e}$. Then $\boldsymbol{x}$ is also multivariate normally distributed with mean vector $\boldsymbol{\mu}_x = \boldsymbol{\mu}_y + \boldsymbol{\mu}_e$ and covariance matrix $\boldsymbol{P} = \boldsymbol{G} + \boldsymbol{E}$, where $\boldsymbol{\mu}_y$, $\boldsymbol{\mu}_e$ and $\boldsymbol{G}$, $\boldsymbol{E}$ are the mean vectors and covariance matrices of $\boldsymbol{y}$ and $\boldsymbol{e}$, respectively. Both $\boldsymbol{G}$ and $\boldsymbol{P}$ are assumed to be positive definite. Assuming also that $\boldsymbol{\mu}_e = \boldsymbol{0}$, then $\boldsymbol{\mu}_x = \boldsymbol{\mu}_y$. These assumptions are conventional for multivariate analyses of quantitative inheritance.

Natural selection on phenotypes is partitioned into two components: directional and stabilizing, and assumed throughout to have a general form that for the ith population the fitness function is

$$w_i(\boldsymbol{x}) = \exp\{\boldsymbol{a}_i^T \boldsymbol{x} - \tfrac{1}{2}(\boldsymbol{x}-\boldsymbol{\theta})^T \boldsymbol{W}_i^{-1}(\boldsymbol{x}-\boldsymbol{\theta})\} \quad (i = 1, 2, \ldots, m), \quad (1)$$

where the superscript T denotes matrix transposition and m is the number of populations. $\boldsymbol{\theta} = [\theta_r]$ $(r = 1, 2, \ldots, n)$ is the column vector of original mean phenotypes of the population before differentiation of subpopulations (θ_r is the rth element of $\boldsymbol{\theta}$). $\boldsymbol{a}_i = [a_{i,r}]$ $(r = 1, 2, \ldots, n)$ is a column vector which approximates the intensities of directional selection in the ith population, and $\boldsymbol{W}_i = [W_{i,rs}]$ $(r, s = 1, 2, \ldots, n)$ is an $n \times n$ positive definite symmetrical matrix which approximates the strength of stabilizing selection in the ith population (Zeng, 1988). [The fitness function (1) has been used by Felsenstein (1977) as a general type of selection acting on n-linked loci.] In the following the subscripts h, i, j and k are used to index populations and p, q, r and s to index characters.

Selection is assumed to be weak so that genetic variation can be maintained by the balance between mutation and selection (Lande, 1980*b*; Via & Lande, 1987). The covariance matrix of genotypes can then be assumed to remain roughly constant as evolution of mean phenotypes proceeds.

Under these assumptions it has been shown (Lande, 1980*a*; Zeng, 1988) that the mean phenotypes of characters in the ith population in successive generations, $\boldsymbol{\mu}_i = [\mu_{i,r}]$ $(r = 1, 2, \ldots, n)$, can be written as

$$\boldsymbol{\mu}_i(t+1) = \boldsymbol{\mu}_i(t) + \boldsymbol{G}_i(\boldsymbol{W}_i + \boldsymbol{P}_i)^{-1}\{\boldsymbol{\theta} + \boldsymbol{W}_i\boldsymbol{a}_i - \boldsymbol{\mu}_i(t)\}, \quad (2)$$

where $\boldsymbol{\mu}_i(t)$ is the mean vector at generation t and $\boldsymbol{G}_i$ and $\boldsymbol{P}_i$ are the genetic and phenotypic covariance matrices in the ith population. At equilibrium $[\boldsymbol{\mu}_i(t) = \boldsymbol{\mu}_i(t+1)]$,

$$\boldsymbol{\mu}_i = \boldsymbol{\theta} + \boldsymbol{W}_i\boldsymbol{a}_i, \quad (3)$$

or in component $\mu_{i,r} = \theta_r + \sum_{s=1}^{n} W_{i,rs}a_{i,s}$ unless $\boldsymbol{G}_i$ is singular.

Since the ultimate responses are determined only by $\boldsymbol{W}_i$ and $\boldsymbol{a}_i$, the interpopulation variation at equilibrium depends only on the variation of $\boldsymbol{W}_i$ and $\boldsymbol{a}_i$ among populations. Let $\boldsymbol{a}_i$ be random variables among populations with mean vector $\boldsymbol{a}$ and covariance matrix $\boldsymbol{A}$. $\boldsymbol{W}_i$ could be regarded as random variables as well, but this would greatly complicate the expression of interpopulation covariances. Instead, for simplicity, we ignore the variation of $\boldsymbol{W}_i$ among populations and take $\boldsymbol{W}$ as constants. The interpopulation covariance matrix at equilibrium is then approximately

$$\boldsymbol{K} \simeq \boldsymbol{WAW}(m-1)/m \simeq \boldsymbol{WAW} \quad (4)$$

for m not very small, where m is the number of populations (Zeng, 1988).

If the original mean phenotypes, $\boldsymbol{\theta}_i$'s, are not the same among the populations and $\boldsymbol{\theta}_i$'s have a mean vector $\boldsymbol{\theta}$ and covariance $\boldsymbol{\Theta}$, equation (4) becomes

$$\boldsymbol{K} \simeq \boldsymbol{\Theta} + \boldsymbol{WAW}, \quad (5)$$

provided that $\boldsymbol{\theta}_i$ is independent of $\boldsymbol{a}_i$. If in addition the genetic and phenotypic covariance matrices in a population remain approximately constant during phenotypic evolution, the expected mean phenotypes at generation t in the ith population after the population occupied a new environment will be approximately $\boldsymbol{\mu}_i(t) \simeq \boldsymbol{\theta}_i + [\boldsymbol{I} - \{\boldsymbol{I} - \boldsymbol{G}_i(\boldsymbol{W}_i + \boldsymbol{P}_i)^{-1}\}^t]\boldsymbol{W}_i\boldsymbol{a}_i$ (Lande, 1980*a*; Zeng, 1988), where $\boldsymbol{I}$ is an $n \times n$ identity matrix. The interpopulation covariance matrix at generation t can then be approximated as

$$\boldsymbol{K}(t) \simeq \boldsymbol{\Theta} + [\boldsymbol{I} - \{\boldsymbol{I} - \boldsymbol{G}(\boldsymbol{W}+\boldsymbol{P})^{-1}\}^t] \times \boldsymbol{WAW}[\boldsymbol{I} - \{\boldsymbol{I} - \boldsymbol{G}(\boldsymbol{W}+\boldsymbol{P})^{-1}\}^t]^T \quad (6)$$

by neglecting the variations of $\boldsymbol{G}_i$, $\boldsymbol{P}_i$ and $\boldsymbol{W}_i$ among populations, where $\boldsymbol{G}$ and $\boldsymbol{P}$ are the mean covariance matrices of $\boldsymbol{G}_i$ and $\boldsymbol{P}_i$ among populations.

3. Migration

Migration of individuals from one population to another can reduce variation among populations. If populations are not totally isolated from each other, the effect of migration should be taken into account in analysing interpopulation variances and covariances. Suppose there are m large populations distributed in m heterogeneous environments. Selection occurs within each environment. After selection a proportion f_{ij} of individuals migrate from the ith to the jth population, with $\boldsymbol{F} = [f_{ij}]$ $(i, j = 1, 2, \ldots, m)$ as the forward migration matrix (Bodner & Cavalli-Sforza, 1968). It is clear that $f_{ij} \geqslant 0$ and $\sum_{j=1}^{m} f_{ij} = 1$. In order to write the approximate transformation of mean phenotypes of populations in successive generations, we need to define the backward migration matrix $\boldsymbol{M} = [m_{ij}](i, j = 1, 2, \ldots, m)$ where m_{ij} is the proportion of individuals in the ith population originating from the jth population after selection and migration.

https://doi.org/10.1017/S0016672300028196 Published online by Cambridge University Press

If c_i is the relative number of individuals in the ith population ($c_i > 0$ and $\sum_{i=1}^{m} c_i = 1$), then

$$m_{ij} = c_i f_{ij} \Big/ \sum_{k=1}^{m} c_k f_{kl} \quad \left(m_{ij} \geqslant 0 \quad \text{and} \quad \sum_{j=1}^{m} m_{ij} = 1 \right)$$

(Bodmer & Cavalli-Sforza, 1968). We assume that m_{ij} is constant across generations and random mating occurs after migration within each local population.

The mean vector of $\boldsymbol{\mu}_i(t)$ in the next generation depends on the migration matrix $\boldsymbol{M}$ and the mean vectors of other populations. Let

$$\tilde{\boldsymbol{\mu}}(t)^T = [\boldsymbol{\mu}_1(t)^T \dots \boldsymbol{\mu}_m(t)^T], \quad \tilde{\boldsymbol{\theta}}(t)^T = [\boldsymbol{\theta}_1(t)^T \dots \boldsymbol{\theta}_m(t)^T],$$

and $\tilde{\boldsymbol{a}}(t)^T = [\boldsymbol{a}_1(t)^T \dots \boldsymbol{a}_m(t)^T]$, which all have length nm. Let also

$$\tilde{\boldsymbol{G}} = [\boldsymbol{G}_{ij}], \quad \tilde{\boldsymbol{P}} = [\boldsymbol{P}_{ij}], \quad \text{and} \quad \tilde{\boldsymbol{W}} = [\boldsymbol{W}_{ij}] \quad (i,j = 1,2,\dots,m),$$

where $\boldsymbol{G}_{ii} = \boldsymbol{G}_i$, $\boldsymbol{P}_{ii} = \boldsymbol{P}_i$, $\boldsymbol{W}_{ii} = \boldsymbol{W}_i$ and $\boldsymbol{G}_{ij} = \boldsymbol{0}$, $\boldsymbol{P}_{ij} = \boldsymbol{0}$, $\boldsymbol{W}_{ij} = \boldsymbol{0}$, $i \neq j$. The equation (2) can be written in another way as

$$\tilde{\boldsymbol{\mu}}(t+1) = \tilde{\boldsymbol{\mu}}(t) + \tilde{\boldsymbol{G}}(\tilde{\boldsymbol{W}} + \tilde{\boldsymbol{P}})^{-1}\{\tilde{\boldsymbol{\theta}} + \tilde{\boldsymbol{W}}\tilde{\boldsymbol{a}} - \tilde{\boldsymbol{\mu}}(t)\}.$$

With migration the recurrent equation is changed to

$$\tilde{\boldsymbol{\mu}}(t+1) = \tilde{\boldsymbol{M}}[\tilde{\boldsymbol{\mu}}(t) + \tilde{\boldsymbol{G}}(\tilde{\boldsymbol{W}} + \tilde{\boldsymbol{P}})^{-1}\{\tilde{\boldsymbol{\theta}} + \tilde{\boldsymbol{W}}\tilde{\boldsymbol{a}} - \tilde{\boldsymbol{\mu}}(t)\}], \quad (7)$$

where $\tilde{\boldsymbol{M}} = \boldsymbol{I} \otimes \boldsymbol{M}$ is the direct product of $\boldsymbol{I}$ and $\boldsymbol{M}$. Here it is assumed that selection is weak and that the means of populations are not very different so that the approximation of normal genetic and phenotypic distributions remains valid with migration. If the means of populations do become appreciably different, this approximation is not very accurate. From (7) we can write

$$\tilde{\boldsymbol{\mu}}(t) = (\tilde{\boldsymbol{M}} - \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}})^t \tilde{\boldsymbol{\mu}}(0) + [\tilde{\boldsymbol{I}} - (\tilde{\boldsymbol{M}} - \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}})^t] \times (\tilde{\boldsymbol{I}} - \tilde{\boldsymbol{M}} + \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}})^{-1} \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}}(\tilde{\boldsymbol{\theta}} + \tilde{\boldsymbol{W}}\tilde{\boldsymbol{a}}) \quad (8)$$

and at equilibrium

$$\tilde{\boldsymbol{\mu}} = (\tilde{\boldsymbol{I}} - \tilde{\boldsymbol{M}} + \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}})^{-1} \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}}(\tilde{\boldsymbol{\theta}} + \tilde{\boldsymbol{W}}\tilde{\boldsymbol{a}}), \quad (9)$$

where $\tilde{\boldsymbol{H}} = \tilde{\boldsymbol{G}}(\tilde{\boldsymbol{W}} + \tilde{\boldsymbol{P}})^{-1}$ and $\tilde{\boldsymbol{I}}$ is an $nm \times nm$ identity matrix, unless $\tilde{\boldsymbol{I}} - \tilde{\boldsymbol{M}} + \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}}$ is singular. [Strictly speaking (8) and (9) hold only when $\boldsymbol{G}_{ii} = \boldsymbol{G}_{jj}$ for all i and j, see below.] Since $\tilde{\boldsymbol{H}}$ is a positive definite diagonal block matrix, $\tilde{\boldsymbol{I}} - \tilde{\boldsymbol{H}}$ is non-singular and $\boldsymbol{M}$ is a stochastic matrix, $\tilde{\boldsymbol{I}} - \tilde{\boldsymbol{M}} + \tilde{\boldsymbol{M}}\tilde{\boldsymbol{H}}$ is expected to be nonsingular and the equilibrium (9) exists.

With (8) and (9) we can theoretically calculate the interpopulation variance and covariance matrix $\boldsymbol{K}$ at equilibrium or at any generation. It is difficult, however, to give a formula for $\boldsymbol{K}$ similar to (6) in this situation. But we observe that, when $m_{ij} = 1/m$ for all i and j, there is no variation expected among population means and all populations form a single large panmictic population. At the other extreme, when $\tilde{\boldsymbol{M}} = \tilde{\boldsymbol{I}}$ (no migration or gene flow among local populations), the interpopulation variances and covariances are defined by (6). With migration the interpopulation variances and covariances are therefore smaller than or equal to those defined by (6).

4. Genotype–environment interaction

Environmental modification of phenotypes of individuals inhabiting heterogeneous environments can give rise to genotype–environment interaction. The phenotypes of a character observed in different environments may not be the same genetically, but can be viewed as being genetically correlated. Selection on a phenotype expressed in one environment may then cause correlated responses in other phenotypes which are expressed in other environments (Falconer, 1981). Via & Lande (1985) have used this approach to examine the effect of genotype–environment interaction on the evolution of population means of a quantitative character, which are under soft and hard selection, in two environments. In the following I will illustrate some effects of genotype–environment interaction on the modelling of interpopulation variances and covariances under the selection function of (1).

Following Via & Lande (1985), the expression of a character in a given environment is considered to be a *character state*. If there are m populations distributed in m different environments, there will be m character states for each of n quantitative characters, a total of nm expressed character states. Although each state is only expressed in one environment, we have to define nm, not n, character states in each population. The other states which are not expressed in one environment need to be defined because the genes which determine them will be carried by migrants to the alternate environments where they will be expressed. In general, for n characters and m environments there are nmm character states defined in the analysis.

Let $\hat{\boldsymbol{x}}$ be the array of these nmm character states which is defined as

$$\hat{\boldsymbol{x}} = \begin{bmatrix} \tilde{\boldsymbol{x}}_1 \\ \vdots \\ \tilde{\boldsymbol{x}}_m \end{bmatrix} \quad \tilde{\boldsymbol{x}}_i = \begin{bmatrix} \boldsymbol{x}_{i1} \\ \vdots \\ \boldsymbol{x}_{im} \end{bmatrix} \quad \boldsymbol{x}_{ij} = \begin{bmatrix} x_{ij1} \\ \vdots \\ x_{ijn} \end{bmatrix} \quad (i,j = 1,2,\dots,m)$$

were x_{ijr} is the state of character r which is expressed in the jth environment, but carried by individuals located in the ith environment. When $i \neq j$, the character state is not expressed and will evolve only by correlated responses to selection on the expressed states. Let $\hat{\boldsymbol{\mu}}$ be the population mean vector of $\hat{\boldsymbol{x}}$. Thus

$$\hat{\boldsymbol{\mu}} = [\tilde{\boldsymbol{\mu}}_i], \tilde{\boldsymbol{\mu}}_i = [\boldsymbol{\mu}_{ij}],$$

and

$$\boldsymbol{\mu}_{ij} = [\mu_{ijr}] \; (i,j = 1,2,\dots,m; r = 1,2,\dots,n).$$

Similarly the initial mean vector, $\hat{\boldsymbol{\theta}}$, is defined as

$$\hat{\boldsymbol{\theta}} = [\tilde{\boldsymbol{\theta}}_i], \tilde{\boldsymbol{\theta}}_i = [\boldsymbol{\theta}_{ij}], \quad \text{and} \quad \boldsymbol{\theta}_{ij} = [\theta_{ijr}];$$

and the vector of directional selection intensities

$$\hat{\boldsymbol{a}} = [\tilde{\boldsymbol{a}}_i], \boldsymbol{a}_i = [\boldsymbol{a}_{ij}], \quad \text{and} \quad \boldsymbol{a}_{ij} = [a_{ijr}].$$

https://doi.org/10.1017/S0016672300028196 Published online by Cambridge University Press

Since x_{ij}'s ($i \neq j$) are not expressed and selection does not act on them directly, $a_{ij} = 0$ ($i \neq j$) and $a_{ii} = a_i$.

The phenotypic variances and covariances of x_{ii} are defined by P_{ii} ($= P_i$), but the phenotypic covariances of x_{ki} and x_{kj} ($k \neq i$ or $k \neq j$) are undefined since x_{ki} is not expressed if $k \neq i$. Similarly the stabilizing selection intensities are also only defined on x_{ii} as W_{ii} ($= W_i$). The genetic covariances of x_{ki} and x_{kj} for all k are, however, defined by G_{ij} since the states are genetically correlated. When $i = j$, $G_{ii} = G_i$. Then the selection responses on $\tilde{\mu}_i$ at generation t before migration is

$$\Delta\tilde{\mu}_i(t) = \tilde{G}\tilde{V}_i(t) \quad (i = 1, 2, \ldots, m) \tag{10}$$

where $\tilde{G} = [G_{ij}]$ ($i, j = 1, 2, \ldots, m$) is the genetic covariance matrix, and $\tilde{V}_i(t) = [V_{ij}(t)]$ ($j = 1, 2, \ldots, m$) is the vector of selection gradients (Lande, 1979). In this vector

$$V_{ii}(t) = (W_{ii} + P_{ii})^{-1}\{\theta_{ii} + W_{ii}a_{ii} - \mu_{ii}(t)\}$$

from (2) and $V_{ij}(t) = 0$ when $i \neq j$ because selection does not act on x_{ij}. Although x_{ij} ($i \neq j$) is not exposed to selection, μ_{ij} has correlated responses through the genetic covariances. Equation (10) can be expressed in another way as

$$\Delta\tilde{\mu}_i(t) = \tilde{G}\tilde{R}_{ii}\{\tilde{\theta}_i + \tilde{s}_i - \tilde{\mu}_i(t)\}, \tag{11}$$

where

$$\tilde{R}_{ii} = [R_{ii,jk}] \ (j, k = 1, 2, \ldots, m), \ R_{ii,ii} = (W_{ii} + P_{ii})^{-1}, \quad R_{ii,jk} = 0 \ (i \neq j \text{ or } i \neq k);$$

and

$$\tilde{s}_i = [s_{ij}] \ (j = 1, 2, \ldots, m), \ s_{ii} = W_{ii}a_{ii}, \ s_{ij} = 0 \ (1 \neq j).$$

After migration and random mating, the mean vector of character states becomes

$$\hat{\mu}(t+1) = \hat{M}[\hat{\mu}(t) + \hat{G}\hat{R}(\hat{\theta} + \hat{s} - \hat{\mu}(t))], \tag{12}$$

where

$$\hat{M} = \tilde{I} \otimes M; \ \hat{G} = \tilde{G} \otimes I; \ \hat{R} = [\tilde{R}_{ij}] \ (i, j = 1, 2, \ldots, m), \quad \tilde{R}_{ij} = \tilde{0} (i \neq j); \quad \text{and} \quad \hat{s} = [\tilde{s}_i] \ (i = 1, 2, \ldots, m).$$

Again this is an approximation and subject to the assumption that the distribution of genotypes and phenotypes within populations remain approximately multivariate normal in each generation before selection.

If $\hat{I} - \hat{M} + \hat{M}\hat{G}\hat{R}$ is non-singular, we can write from (12)

$$\hat{\mu}(t) = (\hat{M} - \hat{M}\hat{G}\hat{R})^t\hat{\mu}(0) + [\hat{I} - (\hat{M} - \hat{M}\hat{G}\hat{R})^t] \times (\hat{I} - \hat{M} + \hat{M}\hat{G}\hat{R})^{-1}\hat{M}\hat{G}\hat{R}(\hat{\theta} + \hat{s}) \tag{13}$$

and at equilibrium

$$\mu = (\hat{I} - \hat{M} + \hat{M}\hat{G}\hat{R})^{-1}\hat{M}\hat{G}\hat{R}(\hat{\theta} + \hat{s}) = \hat{J}(\hat{\theta} + \hat{s}), \tag{14}$$

where $\tilde{I}$ is an $nmm \times nmm$ identity matrix,

$$\hat{J} = I \otimes \tilde{J} \quad \text{and} \quad \tilde{J} = [J_{ij}], \ J_{ij} = [J_{ij.kh}] (i, j, k, h = 1, 2, \ldots, m),$$

$J_{ij.jj} = 1$ for every i and j, and $J_{ij.kh} = 0$ when $j \neq k$ or $J \neq h$. For example, when $m = 2$,

$$\tilde{J} = \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 \\ 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix}.$$

(We observe that $\hat{M}\hat{J} = \hat{J}$ and $\hat{M}\hat{G}\hat{R}\hat{J} = \hat{M}\hat{G}\hat{R}$ from the definitions of $\hat{M}$, $\hat{G}$, $\hat{R}$ and $\hat{J}$, so $(\hat{I} - \hat{M} + \hat{M}\hat{G}\hat{R})^{-1}\hat{M}\hat{G}\hat{R} = \hat{J}$ when $\hat{I} - \hat{M} + \hat{M}\hat{G}\hat{R}$ is non-singular). It can be shown that the conditions for $\hat{I} - \hat{M} + \hat{M}\hat{G}\hat{R}$ to be non-singular are (i) $\hat{G}$ is non-singular; and (ii) the stochastic matrix M is irreducible. ($\hat{R}$ is singular and of rank nm.) Thus we have

$$\mu_{ij} = \theta_{jj} + W_{jj}a_{jj} \tag{15}$$

for every i and j. Equation (15) is equivalent to (3) when $i = j$. This shows that, if $\tilde{G}$ is non-singular and M is irreducible, the populations in different environments at equilibrium will converge to the same vector of mean breeding values, but not necessarily to the same vector for expressed character states, so that indirectly selected character states attain the same values as their directly selected counterparts (i.e. $\mu_{ij} = \mu_{jj}$ for every i and j) (Via & Lande, 1985); and the equilibrium variation of population means of expressed character states is determined by (5), independent of the influence of the migration structure. However, before reaching the equilibrium the variation will depend on the migration structure (M) and the degree of genotype–environment interaction, and is smaller than that defined by (6).

When the migration matrix M is reducible (i.e. individuals in some populations never migrate to other populations, directly or indirectly), (13) and (14) will not exist and (15) will not hold not for all i and j, but for those i and j which are equal (i.e. for expressed characters). For example, when $M = I$, at equilibrium

$$\mu_{jj} = \theta_{jj} + W_{jj}a_{jj}$$

and

$$\mu_{ij} = \theta_{ij} + G_{ij}G_{jj}^{-1}W_{jj}a_{jj} = \theta_{ij} + G_{ii}^{\frac{1}{2}}\Gamma_{ij}G_{jj}^{-\frac{1}{2}}W_{jj}a_{jj},$$

where Γ_{ij} is a diagonal matrix whose rth diagonal element ($r = 1, 2, \ldots, n$) is the genetic correlation between x_{kir} and x_{kjr}. It is this Γ_{ij} matrix that measures the degree of genotype–environment interaction between x_{ki} and x_{kj}. When there is no genotype–environment interaction (e.g. $\Gamma_{ij} = I$),

$$\mu_{ij} = \theta_{ij} + G_{ii}^{\frac{1}{2}}G_{jj}^{-\frac{1}{2}}W_{jj}a_{jj},$$

which reduces to $\mu_{ij} = \theta_{ij} + W_{jj}a_{jj}$ only when $G_{ii} = G_{jj}$.

$\tilde{G}$ can be singular. It happens if genetic correlations between some character states or partial correlations

https://doi.org/10.1017/S0016672300028196 Published online by Cambridge University Press

are perfect (1 or -1), or genetic variance of a character is exhausted. This perhaps happens quite often in reality when n and m are not small. When $\tilde{\boldsymbol{G}}$ is singular, as long as $\boldsymbol{G}_{ii}$ is non-singular for every i, (12) still converges to some equilibrium which is not defined by (15). The equilibrium will depend on $\boldsymbol{M}$ and $\tilde{\boldsymbol{G}}$. An extreme case has been given by (9) in which $\boldsymbol{\Gamma}_{ij} = \boldsymbol{I}$ for every i and j.

5. Drift under stabilizing selection

Random genetic drift due to finite population size is another source of interpopulation variation. Even without selection populations can diverge from their original means with the rate depending on the nature and amount of genetic variation within populations, mutation schemes, and population sizes (Wright, 1951; Robertson, 1952; Clayton & Robertson, 1955; Lande, 1976, 1979; Chakraborty & Nei, 1982; Lynch & Hill, 1986; Cockerham & Tachida, 1987). When populations are selected for different optimum phenotypes, the divergence due to drift is constrained by stabilizing selection (Lande, 1980*a*). Let $\bar{\boldsymbol{x}}_i(t)$ be the column vector of mean phenotypes of n quantitative characters in the ith population with effective size N_i at generation t and $\boldsymbol{D}_i(t)$ represent the dispersion matrix for the probability distribution of $\bar{\boldsymbol{x}}_i(t)$ among populations. The expectation of $\bar{\boldsymbol{x}}_i(t)$ is $\boldsymbol{\mu}_i(t)$. While equation (2) gives the dynamics of the expectation of $\bar{\boldsymbol{x}}_i(t)$ in successive generations without migration, the recursion for the dispersion matrix is given approximately by (Lande, 1980*a*)

$$\boldsymbol{D}_i(t+1) = \boldsymbol{D}_i(t) - \boldsymbol{G}_i(\boldsymbol{W}_i+\boldsymbol{P}_i)^{-1}\boldsymbol{D}_i(t) - \boldsymbol{D}_i(t)(\boldsymbol{W}_i+\boldsymbol{P}_i)^{-1}\boldsymbol{G}_i + \boldsymbol{G}_i + \boldsymbol{G}_i/N_i \quad (16)$$

to first order in $\boldsymbol{G}_i(\boldsymbol{W}_i+\boldsymbol{P}_i)^{-1}$. This was obtained by using the approximation of a normal probability distribution of $\bar{\boldsymbol{x}}_i(t)$ and neglecting variation in $\boldsymbol{G}_i$ across generations. Unless N_i is very small, the approximation is appropriate. $\boldsymbol{G}/_i N_i$ is the expected rate of dispersion per generation if there is no stabilizing selection. However, under selection the dispersion is constrained. So starting with $\boldsymbol{D}_i(0)$ zero, the dispersion matrix of $\bar{\boldsymbol{x}}_i(t)$ is

$$\boldsymbol{D}_i(t) = [\boldsymbol{I} - \{\boldsymbol{I} - 2\boldsymbol{G}_i(\boldsymbol{W}_i+\boldsymbol{P}_i)^{-1}\}^t](\boldsymbol{W}_i+\boldsymbol{P}_i)/(2N_i). \quad (17)$$

The expected interpopulation variation of characters due to drift, $\boldsymbol{K}_d(t)$, is the mean of $\boldsymbol{D}_i(t)$ among populations. Neglecting the variation of $\boldsymbol{G}_i$, $\boldsymbol{P}_i$, and $\boldsymbol{W}_i$ among populations, we have

$$\boldsymbol{K}_d(t) \simeq [\boldsymbol{I} - \{\boldsymbol{I} - 2\boldsymbol{G}(\boldsymbol{W}+\boldsymbol{P})^{-1}\}^t](\boldsymbol{W}+\boldsymbol{P})/(2N), \quad (18)$$

where N is the harmonic mean of N_i among the populations. Then the expected total interpopulation variation becomes

$$\boldsymbol{K}(t) \simeq \boldsymbol{\Theta} + [\boldsymbol{I} - \{\boldsymbol{I} - \boldsymbol{G}(\boldsymbol{W}+\boldsymbol{P})^{-1}\}^t] \times \boldsymbol{WAW}[\boldsymbol{I} - \{\boldsymbol{I} - \boldsymbol{G}(\boldsymbol{W}+\boldsymbol{P})^{-1}\}^t]^T + [\boldsymbol{I} - \{\boldsymbol{I} - 2\boldsymbol{G}(\boldsymbol{W}+\boldsymbol{P})^{-1}\}^t](\boldsymbol{W}+\boldsymbol{P})/(2N) \quad (19)$$

from (6) and (18), since the dispersion of the mean vectors from expectations is independent of evolution of the expected means of populations (Lande, 1980*a*). At equilibrium

$$\boldsymbol{K} \simeq \boldsymbol{\Theta} + \boldsymbol{WAW} + (\boldsymbol{W}+\boldsymbol{P})/(2N). \quad (20)$$

In this formula the second term on the right hand side is the contribution of differential selection to the interpopulation variation and the third is of drift which is constrained by stabilizing selection. (The first is the initial variation.) Thus if $\mathrm{tr}(\boldsymbol{WAW}) \gg \mathrm{tr}(\boldsymbol{W}+\boldsymbol{P})/2N$ [tr( ) is the trace of a matrix, i.e. the sum of the diagonal elements of a matrix], it will be expected that most of the variation among population means at equilibrium is due to differential selection among populations rather than random genetic drift for the characters concerned, and vice versa. For example, in the one dimension case, the interpopulation variation will be largely due to differential selection if $A_{11} \gg 2{\cdot}6 \times 10^{-4}$ for $N = 100$, $P_{11} = 1$, and $W_{11} = 20$.

With (19) we can also have the expected interpopulation variation after one generation selection and sampling which is approximately

$$\boldsymbol{K}(1) \simeq \boldsymbol{\Theta} + \boldsymbol{GAG} + \boldsymbol{G}/N \quad (21)$$

after making the approximation $\boldsymbol{W}+\boldsymbol{P} \simeq \boldsymbol{W}$ for weak stabilizing selection (i.e. magnitudes of eigenvalues of $\boldsymbol{W}$ much larger than those of $\boldsymbol{P}$). This shows that $\boldsymbol{K}$ is mainly a function of $\boldsymbol{G}$ in the short term, but of $\boldsymbol{W}$ in the long term.

The relative contribution of the differential selection and drift on interpopulation variation depends also on the number of generations since populations diverged. For instance, in the one dimension case if we take $W_{11} = 20$, $P_{11} = 1$, $G_{11} = 0{\cdot}5$, $N = 100$, and $A_{11} = 0{\cdot}001$, we can show from (19) that the short-term population variation is mostly due to drift (accounting for 82% when $t = 5$ and 69% when $t = 10$), although in the long term the differential selection accounts for most of the variation (78% at the equilibrium). Since $\boldsymbol{W}_{ii}$ is generally much larger than $\boldsymbol{G}_{ii}$ under weak stabilizing selection, it is expected that for given $\boldsymbol{A}_{ii}$ and N the relative contribution of the differential selection to interpopulation variation increases with time until reaching its maximum, unless $\boldsymbol{A}_i = \boldsymbol{0}$.

Thus it is possible that the pattern of interpopulation variation is different in different time scales. In the short term, especially immediately after separation of populations, the drift proportion on interpopulation variation attains the maximum, and $\boldsymbol{K}$ could be expected to be approximately proportional to $\boldsymbol{G}$. But since differential selection contribution to the interpopulation variation increases with time, $\boldsymbol{K}$ would be expected to be a quadratic function of $\boldsymbol{W}$ in the long term. In that case the overall level of correlation of $\boldsymbol{K}$ matrix is likely to be higher than that of $\boldsymbol{W}$ matrix (Zeng, 1988).

https://doi.org/10.1017/S0016672300028196 Published online by Cambridge University Press

6. Discussion

Interpopulation variation and covariation of quantitative characters can principally originate from four sources; differential selection among populations, random genetic drift, genotype–environment interaction, and mutation. Migration (or gene flow) increases homogeneity of populations. In this paper I used a specific selection model (1) to examine the effects of different forces (except mutation) on interpopulation variation.

Under selection and drift, the interpopulation covariance matrix $\boldsymbol{K}$ is likely to be approximately proportional to the genetic covariance matrix $\boldsymbol{G}$ in the short term. In the long term $\boldsymbol{K}$ is a quadratic function of the stabilizing selection matrix $\boldsymbol{W}$. This implies that the overall level of correlation of $\boldsymbol{K}$ (interpopulation correlations) is likely to be in the magnitude of that of $\boldsymbol{G}$ in the short term, and can increase with time, thus tending to be larger than that of the phenotypic covariance matrix $\boldsymbol{P}$ (intrapopulation correlations) if the overall level of correlation of $\boldsymbol{W}$ is not less than that of $\boldsymbol{G}$ and $\boldsymbol{P}$.

For quantitative characters under multivariate stabilizing selection, it is usually assumed that genetic and phenotypic variation and covariation of the characters are selected to conform to the shape of the fitness surface acting on them (Schmalhausen, 1949; Waddington, 1957; Olson & Miller, 1958). Studies on functionally related traits also show that these traits tend to be highly integrated morphologically, and their phenotypic correlation structure conforms to the functional relationship structure (Bader & Hall, 1960; Cheverud, 1982). Some studies (Cheverud, 1982; Cheverud, Rutledge & Atchley, 1983; Cheverud & Leamy, 1985; Kohn & Atchley, 1988) suggest that the genetic correlation structure among morphological traits is more highly integrated than the phenotypic correlation structure. However, these observations of high genetic correlations may partly be due to the bias toward high integration of the correlation matrix inherent in the method of estimating the genetic covariance matrix (Hill & Thompson, 1978; Cheverud, 1988). This is because the estimator of between-group covariance matrix tends to bias eigenvalues of the matrix toward extremes (Hill & Thompson, 1978) and a correlation matrix with higher dispersed eigenvalues is more tightly integrated. Observations on morphological characters of *Pemphigus* also suggest that the overall level of interpopulation correlations is higher than that of intrapopulation correlations (Thomas, 1968; Sokal, Bird & Riska, 1981). This is consistent with the result obtained in this paper. (For interpopulation correlations these studies computed both the product-moment correlation matrix based on locality means and the interlocality–component correlation matrix based on covariance component analysis. The overall level of interlocality–component correlations is slightly higher than that of product–moment correlations (Thomas, 1969).)

Genotype–environment interaction causes further divergence among populations, because different suites of genes or different gene effects may be expressed in different environments. The effect of migration on interpopulation variation is directly related to the extent of genotype–environment interaction. When there is no genotype–environment interaction, the interpopulation variation will be reduced by migration, and the structure of migration among populations will be critical in determining population differentiation. However, with genotype–environment interaction the effect of migration is reduced. This is because genotype–environment interaction permits somewhat independent evolution of characters in different environments. In the special case that the genetic covariance matrix $\tilde{\boldsymbol{G}}$ is non-singular, migration will not influence the equilibrium interpopulation variation and covariation of expressed character states, but will reduce the rate of convergence to the equilibrium variation. The within-population variation at the equilibrium will also be free from the effect of migration and disruptive selection (Via & Lande, 1987).

The model of population differentiation examined in this paper is restricted in many ways. In addition to the general assumptions usually made in multivariate quantitative genetic analyses (e.g. polygenic inheritance, multivariate normality of phenotypic and additive genetic distributions), other assumptions have been made, including (i) the fitness function (1); (ii) simultaneous divergence of progeny populations from the base population; and (iii) uniform $\boldsymbol{W}$ matrices among populations [(6), (18) and (19) also rely on the assumptions of constant $\boldsymbol{G}$ and $\boldsymbol{P}$ matrices over time and across populations]. The fitness function (1) appears to be quite general and can encompass different types of selection (Felsenstein, 1977). The model analysed here differs from that of Lande (1980*a*) in that $\boldsymbol{a}_i$ is assumed to be independent of $\boldsymbol{W}$. It is also different from the Brownian motion model of Felsenstein (1985). Uniform $\boldsymbol{W}$ matrices among populations are made for convenience of analysis, so are $\boldsymbol{G}$ and $\boldsymbol{P}$ matrices. Variation of these matrices among populations will greatly complicate the analysis. Although these matrices are likely to change as populations diverge, there is some evidence to indicate that both phenotypic and genetic covariance matrices (and particularly correlation matrices) are likely to be similar in closely related populations (Lofsvold, 1986; Kohn & Atchley, 1988). A branching phylogeny will affect transient pattern of covariation among lineages, but will not influence the equilibrium covariation.

This is paper No. 11828 of the Journal Services of the North Carolina Agricultural Research Service, Raleigh, NC 27695-7606. 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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