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Evolution of genetic variability in a spatially heterogeneous environment: effects of genotype–environment interaction

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Summary

Classical population genetic models show that disruptive selection in a spatially variable environment can maintain genetic variation. We present quantitative genetic models for the effects of disruptive selection between environments on the genetic covariance structure of a polygenic trait. Our models suggest that disruptive selection usually does not alter the equilibrium genetic variance, although transient changes are predicted. We view a quantitative character as a set of *character states*, each expressed in one environment. The genetic correlation between character states expressed in different environments strongly affects the evolution of the genetic variability. (1) If the genetic correlation between character states is not ± 1, then the mean phenotype expressed in each environment will eventually attain the optimum value for that environment; this is the evolution of phenotypic plasticity (Via & Lande, 1985). At the joint phenotypic optimum, there is no disruptive selection between environments and thus no increase in the equilibrium genetic variability over that maintained by a balance between mutation and stabilizing selection within each environment. (2) If, however, the genetic correlation between character states is ± 1, the mean phenotype will not evolve to the joint phenotypic optimum and a persistent force of disruptive selection between environments will increase the equilibrium genetic variance. (3) Numerical analyses of the dynamic equations indicate that the mean phenotype can usually be perturbed several phenotypic standard deviations from the optimum without producing transient changes of more than a few per cent in the genetic variances or correlations. It may thus be reasonable to assume a roughly constant covariance structure during phenotypic evolution unless genetic correlations among character states are extremely high or populations are frequently perturbed. (4) Transient changes in the genetic correlations between character states resulting from disruptive selection act to constrain the evolution of the mean phenotype rather than to facilitate it.

1. Introduction

Evolution in a spatially variable environment can produce changes in both the mean phenotypes of quantitative (polygenic) traits and their additive genetic variances and covariances. Thus a theory of the process of adaptation of quantitative traits to a heterogeneous environment requires dynamical equations for both the average phenotype (Via & Lande, 1985) and the genetic variability. In this paper we present a model describing the evolution of the genetic covariance structure of a quantitative trait that is under selection in a spatially variable environment.

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Genotype–environment interaction (g–e) clearly affects the evolution of the mean phenotype in a patchy environment (Via & Lande, 1985); the extent of g–e also strongly influences both the equilibrium genetic variability and the evolutionary dynamics of the additive genetic variances and covariances.

Our models use Falconer's (1952) idea that a metrical character expressed in two environments can be considered to be two genetically correlated *character states* each of which is expressed in only one of the environments. For example, the body size expressed by an insect population in which some individuals develop on each of two host plant species can be considered to be two character states, for example 'body size on plant species 1' and 'body size on plant species 2' (Via, 1984*b*). The additive genetic variance of each character state can be estimated

within the environment in which it is expressed; the additive genetic covariance between the character states expressed in different environments measures the extent to which they have the same genetic basis (Falconer, 1981). For example, an additive genetic correlation between character states of +1 means that the same alleles or sets of alleles make proportional contributions to the phenotypic value of the character state expressed in each environment. When the genetic correlation between character states is not perfect, changes in gene frequencies that affect a character state expressed in one environment may not affect other character states proportionally.

Genotype–environment interaction is equivalent to genetic variation in phenotypic plasticity (Via & Lande, 1985) and its magnitude is a reflection of the genetic correlations between character states expressed in different environments. Significant g–e generally corresponds to a genetic correlation between character states that is less than +1 (Robertson, 1959; Yamada, 1962; Fernando, Knights & Gianola, 1984; Via, 1984*b*). Because previous work shows that additive genetic correlations of ±1 are evolutionary special cases (Via & Lande, 1985), it is important to be able to detect them empirically. However, estimates of genotype–environment interaction *per se* do not directly measure the magnitude of the genetic correlations between character states expressed in different environments, and so estimates of g–e are less useful than genetic correlations in modelling phenotypic evolution in a heterogeneous environment (Robertson, 1959; Via, 1984*b*; Via & Lande, 1985). Estimates of the additive genetic correlation between character states can be obtained by performing a standard half-sib breeding design (Falconer, 1981) and allowing some members of every family to develop in each of the environments (e.g. Via, 1984*a*).

Phenotypic evolution in a heterogeneous environment can thus be considered as a problem in the evolution of a correlated suite of character states. Although each character state is only expressed in a particular environment, it is subject to correlated responses to selection acting on the character states expressed in all the other environments experienced by the population (Via & Lande, 1985). If character states expressed in different environments have a less than perfect genetic correlation, they have the potential for somewhat independent evolution. Classical genetic models for the maintenance of genetic variation that concern one locus with two alleles (reviewed in Hedrick, Ginevan & Ewing, 1976; Felsenstein, 1976) have not incorporated this possibility. Furthermore, previous treatments of the effects of spatial variation on quantitative genetic variation in a single character (Bulmer, 1971, 1980, ch. 10; Slatkin, 1978; Felsenstein, 1977, 1979) have considered only the special case in which there is no g–e, that is, in which the genetic correlation of character states in different environments was assumed to be +1. Our models allow the genetic correlation between character states expressed in different environments to assume any value from −1 to +1.

We first describe the evolutionary dynamics of the genetic covariance structure for a population under simultaneous selection in two environments. Four basic forces are involved: stabilizing selection within environments, disruptive selection between environments, pleiotropic mutation and recombination. We then present the equilibrium genetic covariance matrix of the character states. When the genetic correlation between character states in different environments is ±1, the equilibrium genetic variance in each environment is increased by permanent disruptive selection.

For the situation in which the genetic correlation between character states is not ±1, disruptive selection between environments has only a transient effect on quantitative genetic variances and covariances; temporary increases in genetic variance may result from periods of disruptive selection during the evolution of the mean phenotype toward a new optimum. Disruptive selection can thus inflate the observed genetic variation for populations in a heterogenous environment that are frequently subjected to temporal fluctuations in selection. The magnitude of this transient effect is estimated analytically and illustrated with numerical examples.

If genetic variances and covariances change during the course of evolution in a spatially variable environment, the evolutionary trajectory of the mean phenotype could be altered relative to that expected for a constant covariance structure (Via & Lande, 1985). Numerical analyses of the models presented here suggest, however, that the mean phenotype can generally be perturbed several phenotypic standard deviations from the joint optimum phenotype before the genetic covariance structure is changed by more than a few per cent during re-adaptation of the mean phenotype. Therefore, if the joint optimum phenotype does not change very often or by very much, evolutionary changes that occur in the genetic covariance structure may not produce substantial alterations in the trajectory of the mean phenotype that is predicted using a constant covariance matrix.

2. The model

(i) *Assumptions*

The model assumes a large panmictic population with discrete non-overlapping generations. Zygotes are dispersed randomly into different habitat types or environmental patches where they develop and undergo natural selection. We assume that the environment is 'coarse-grained' such that a given offspring only experiences selection in one habitat type in its lifetime. The order of events in the life cycle thus consists of individual development and selection within environments, followed by random mating and dispersal of zygotes. Under 'soft' selection (cf.

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Christiansen, 1974) the contribution of each patch to the mating pool is constant each generation. A fraction, q_i, of the population experiences the ith environment; this is the same as the c_i in Levene's (1953) and Christiansen's (1974) models. The characters of interest here are quantitative traits that play ecological roles in the adaptation of populations to different environmental circumstances (e.g. morphology, physiological pathways, behaviour and life history traits). For each character considered, a different phenotypic value may be expressed in each environment, corresponding to a condition of 'phenotypic plasticity' for that character (Bradshaw, 1965; Via & Lande, 1985). Within environments, each character state is assumed to be under stabilizing selection; these models are therefore not applicable to major components of fitness that are subject to continual directional selection.

The pleiotropic effects of alleles at the ith locus on two character states (one character with a possibly different expression in two environments) may be written as a vector $\mathbf{x}_i = [x_{i1}, x_{i2}]^T$, where T indicates transposition. Similarly, the vector of phenotypic values for the two character states can be written as $\mathbf{z} = [z_1, z_2]^T$. We assume that all of the genetic variation is additive. The phenotypic value for a particular character state can then be written as the sum of genetic and environmental effects as: $z = x + e = \sum_i (x_i + x_i') + e$, where x_i and x_i' are the allelic contributions from the two parents. Before selection in any environment, x and e are assumed to be independent with $\bar{e} = 0$ so that $\bar{z} = \bar{x}$. Although we consider a single character expressed in two environments for notational simplicity, the equations that follow can be generalized to apply to more characters or additional environments by increasing the dimensions of the vectors and matrices.

The 2×2 covariance matrix of allelic effects at the ith and jth loci on the character states expressed in each environment can be written as $\mathbf{C}_{ij} = \mathrm{E}[(\mathbf{x}_i - \bar{\mathbf{x}}_i)(\mathbf{x}_j - \bar{\mathbf{x}}_j)^T]$. For $i = j$, $\mathbf{C}_{ii}$ is the covariance matrix of allelic effects of the ith locus on the character states when genetic covariances among the states are attributable to pleiotropy. For $i \neq j$, $\mathbf{C}_{ij}$ is the covariance matrix of allelic effects due to linkage disequilibrium. The total effect of the alleles at the ith locus on the phenotype is defined as

$$\mathbf{C}_{iz} = \sum_j \mathbf{C}_{ij}. \tag{1}$$

With random mating there is no covariance between uniting gametes (Lande, 1977, 1980, 1984), and so the additive genetic variance–covariance matrix is

$$\mathbf{G} = 2\sum_i \sum_j \mathbf{C}_{ij} = 2\sum_i \mathbf{C}_{iz}. \tag{2}$$

Although $\mathbf{C}_{ij}$ need not generally be symmetric, $\mathbf{G}$ is symmetric. In this way, each character state can be described by a phenotypic mean ($\bar{z}_k$, $k = 1, 2$), a genetic variance (G_{kk}), and a genetic covariance with the character states expressed in the other environments (G_{kj}, $k \neq j$). In the 2×2 case examined here, the genetic variances of the character states expressed in different environments are on the diagonal of the matrix $\mathbf{G}$, and the genetic covariance between the character states is off the diagonal. If more than one character were studied in each environment, $\mathbf{G}$ would have the variances and covariances of characters within environments in blocks on the diagonal, and the covariances between character states expressed in different environments in the off-diagonal blocks.

Selection is assumed to be weak so that the composite distribution in the mating pool of allelic effects carried by individuals that were selected in different environments will be approximately Gaussian. In a coarse-grained environment, each individual experiences only one environment and thus expresses only one of the possible character states. The phenotypic covariance among character states in the two environments is therefore undefined (Falconer, 1981, p. 284). However, for the purpose of dynamical analysis, we can take $P_{12} = 0$ because selection operates independently in each environment. The phenotypic covariance matrix, $\mathbf{P}$, can thus be written as

$$\mathbf{P} = \begin{pmatrix} P_{11} & 0 \\ 0 & P_{22} \end{pmatrix}.$$

(ii) *Dynamics of the genetic covariance structure*

The matrices of allelic effects on the character states can be altered by four forces: stabilizing selection within each environment, disruptive selection between environments, mutation and recombination. Each of these will be considered in turn in order to formulate an expression for the evolutionary change in $\mathbf{C}_{ij}$, and thus in $\mathbf{G}$, that is expected each generation.

(1) *Stabilizing selection within each environment.* To approximate the action of weak stabilizing selection, a Gaussian form is assumed for the fitness function in the kth environment: $w_k(z_k) = \exp\{-(z_k - \theta_k)^2/2\omega_k^2\}$, where θ_k is the phenotypic optimum in the kth environment and ω_k is the width of the stabilizing selection function for the character state expressed in environment k.

Using the partial regressions of allelic effects on the phenotype expressed in each environment (Lande, 1977, 1980), the net effects of stabilizing selection over all environments on the covariance matrix of allelic effects at loci i and j can be written as

$$\sum_{k=1}^{2} -q_k \mathbf{C}_{iz}(\mathbf{W}_k + \mathbf{P})^{-1}\mathbf{C}_{jz}^T \tag{3}$$

where we define

$$\mathbf{W}_1 = \begin{pmatrix} \omega_1^2 & 0 \\ 0 & \infty \end{pmatrix} \quad \text{and} \quad \mathbf{W}_2 = \begin{pmatrix} \infty & 0 \\ 0 & \omega_2^2 \end{pmatrix}. \tag{4}$$

The off-diagonal elements of $\mathbf{W}_k$ are zero because

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selection acts independently in each environment and the width of the fitness function for the unexpressed character state in each environment is ∞ because that character state is not exposed to selection.

(2) *Disruptive selection between environments.* For the panmictic population considered here, the distribution of genotypic values before selection is identical in both environments because individuals are assumed to enter the environments at random. Even though each individual expresses only one of the two possible character states, the genotypic values of the unexpressed character state in each environment can change by correlated response to selection on the expressed character state (Via & Lande, 1985). Differences in selection between environments will cause the vectors of mean genotypic values to differ in each environment after selection. Then, when individuals from all environments mix in the random mating pool, any differences in the vectors of genotypic values among groups selected in different environments will increase the genetic variances in the mating pool. For the two-environment case, the effect of disruptive selection on the matrix of allelic effects is written as

$$\mathbf{D}_{ij} = q_1 q_2(\bar{\mathbf{x}}_i^{*(1)} - \bar{\mathbf{x}}_i^{*(2)})(\bar{\mathbf{x}}_j^{*(1)} - \bar{\mathbf{x}}_j^{*(2)})^T \quad (5)$$

with superscripts indicating the environment in which selection is occurring and asterisks denoting a value after stabilizing selection. Thus, $\bar{\mathbf{x}}_i^{*(1)} = (\bar{x}_{i1}^{*(1)}, \bar{x}_{i2}^{*(1)})^T$ is the vector of mean allelic effects at the ith locus in the first environment after stabilizing selection.

Using the fact that selection acts independently in each environment, the difference in mean allelic effects between portions of the population that are selected in different environments can be calculated by subtracting the regressions of allelic effects at the ith locus on the total genotypic value ($\bar{\mathbf{x}} = 2\sum_i \bar{x}_i$) in each of the two environments (Lande, 1977, 1980):

$$\bar{\mathbf{x}}_i^{*(1)} - \bar{\mathbf{x}}_i^{*(2)} = \bar{\mathbf{x}}_i^{(1)} + \mathbf{C}_{iz}\mathbf{G}^{-1}[\bar{\mathbf{x}}^{*(1)} - \bar{\mathbf{x}}^{(1)}] - \bar{\mathbf{x}}_i^{(2)} - \mathbf{C}_{iz}\mathbf{G}^{-1}[\bar{\mathbf{x}}^{*(2)} - \bar{\mathbf{x}}^{(2)}]. \quad (6)$$

In a panmictic population with random dispersal, the mean allelic effects in each environment before selection are identical, $\bar{\mathbf{x}}_i^{(1)} = \bar{\mathbf{x}}_i^{(2)}$. For soft selection, the overall mean fitness of the population can be defined as $\overline{W} = \overline{W}_1^{q_1}\overline{W}_2^{q_2}$, where $\overline{W}_k$ is the mean fitness in the kth environment (Via & Lande, 1985). Defining the gradient operator $\nabla = (\nabla_1, \nabla_2)^T = (\partial/\partial\bar{x}_1, \partial/\partial\bar{x}_2)^T$, we have $\nabla \ln \overline{W}_k = \mathbf{G}^{-1}(\bar{\mathbf{x}}^{*(k)} - \bar{\mathbf{x}}^{(k)})$ (Lande, 1979). Because fitness in each environment depends only on the character state that is expressed there, $\nabla \ln \overline{W}_1 = (\nabla_1 \ln \overline{W}_1, 0)^T$ and $\nabla \ln \overline{W}_2 = (0, \nabla_2 \ln \overline{W}_2)^T$, and (6) can be re-written as

$$\bar{\mathbf{x}}_i^{*(1)} - \bar{\mathbf{x}}_i^{*(2)} = \mathbf{C}_{iz}[\nabla \ln \overline{W}_1 - \nabla \ln \overline{W}_2] = \mathbf{C}_{iz}\left[\begin{pmatrix}\nabla_1 \ln \overline{W}_1 \\ 0\end{pmatrix} - \begin{pmatrix}0 \\ \nabla_2 \ln \overline{W}_2\end{pmatrix}\right]. \quad (7)$$

Multiplying this result as in equation 5 yields

$$\mathbf{D}_{ij} = q_1 q_2 \mathbf{C}_{iz} \cdot \begin{bmatrix}(\nabla_1 \ln \overline{W}_1)^2 & -\nabla_1 \ln \overline{W}_1 \nabla_2 \ln \overline{W}_2 \\ -\nabla_1 \ln \overline{W}_1 \nabla_2 \ln \overline{W}_2 & (\nabla_2 \ln \overline{W}_2)^2\end{bmatrix} \mathbf{C}_{jz}^T \quad (8)$$

where $\nabla_k \ln \overline{W}_k = (\theta_k - \bar{z}_k)/(\omega_k^2 + P_{kk})$ for the Gaussian fitness function.

The effect of disruptive selection between environments on the dynamics of the genetic covariance structure is completely accounted for by $\mathbf{D}_{ij}$. Equation 8 illustrates that when the mean phenotype vector of the population is at the joint optimum, the effect of disruptive selection vanishes because $\nabla_k \ln \overline{W}_k = 0$ for all k. Although the action of disruptive selection always increases the genetic variances, (8) also shows that the direction of change in the covariance depends on the relative signs of the forces of selection in the two environments: selection of the two character states in the same direction in both environments decreases the genetic covariance between the states, while the genetic covariance increases when the character states are selected in opposite directions in the two environments (see example in Fig. 1). Thus the changes in the genetic covariance between the two character states that are produced by disruptive selection are opposite to those that would facilitate evolution of the mean phenotype.

(3) *Mutation.* Mutations of small effect are assumed to produce an approximately multivariate normal distribution of allelic effects that is centred on the current mean allelic effect. Because a symmetric distribution of allelic effects is postulated, pleiotropic mutations influence the genetic covariance among character states but leave the covariance due to linkage disequilibrium unaffected. Mutation rates are assumed to be constant and equal for all alleles at a given locus although the rates may vary among loci. The 2×2 covariance matrix of mutational effects for alleles at loci i and j in gametes can therefore be written as

$$\delta_{ij}\mathbf{U}_i \quad (9)$$

where $\delta_{ij} = 1$ for $i = j$, and $\delta_{ij} = 0$ for $i \neq j$ (Lande, 1980). Models for estimation of the equilibrium genetic variance under mutation–selection balance that do not assume a Gaussian distribution of allelic effects have been formulated (Turelli, 1984), but dynamical equations based on such models are not currently available. To justify an approximately Gaussian distribution of allelic effects at each locus, we assume that stabilizing selection is weak and per-locus mutation rates are high (Turelli, 1984).

(4) *Recombination.* The recombination fraction between loci i and j is defined as $r_{ij} > 0$ for $i \neq j$, and $r_{ii} = 0$. As described in Lande (1980), recombination reduces the magnitude of covariance of allelic effects at loci i and j such that the contribution of recombination to $\Delta\mathbf{C}_{ij}$ is

$$-r_{ij}\mathbf{C}_{ij} \quad (10)$$

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From these four classes of terms (equations 3, 8, 9, 10) the change per generation in the covariance matrices of allelic effects for the two-environment case is

$$\Delta \mathbf{C}_{ij} = -q_1 \mathbf{C}_{iz}(\mathbf{W}_1+\mathbf{P})^{-1}\mathbf{C}_{jz}{}^T - q_2 \mathbf{C}_{iz}(\mathbf{W}_2+\mathbf{P})^{-1}\mathbf{C}_{jz}{}^T + \mathbf{D}_{ij} + \delta_{ij}\mathbf{U}_i - r_{ij}\mathbf{C}_{ij}. \quad (11)$$

The first two terms correspond to the weighted effects of stabilizing selection in the two environments, the third term is the effect of disruptive selection on the mean phenotype expressed in different environments, the fourth is the effect of mutation, and the last is the effect of recombination.

From equation (11), the dynamics of the genetic covariance matrix for an arbitrary number of loci that contribute equally to the phenotypic variance, have equal mutational parameters ($\mathbf{U}_i = \mathbf{U}$), and between which there is free recombination ($r_{ij} = \frac{1}{2}$, for $i \neq j$) is written as

$$\Delta \mathbf{G} = 2n[\Delta \mathbf{C}_{ii} + (n-1)\Delta \mathbf{C}_{ij}]. \quad (12)$$

In this symmetrical case, $\mathbf{C}_{iz} = \mathbf{C}_{jz}$ and $\mathbf{D}_{ij} = \mathbf{D}_{ii} = \mathbf{D}$, so substituting (11) into (12),

$$\Delta \mathbf{G} = 2n[-nq_1\mathbf{C}_{iz}(\mathbf{W}_1+\mathbf{P})^{-1}\mathbf{C}_{iz}{}^T - nq_2\mathbf{C}_{iz}(\mathbf{W}_2+\mathbf{P})^{-1} \cdot \mathbf{C}_{iz}{}^T + \mathbf{U} - [(n-1)/2]\mathbf{C}_{ij} + n\mathbf{D}]. \quad (13)$$

(iii) *Equilibrium genetic variance*

Genetic correlation between character states not ± 1. When $|\mathbf{G}| \neq 0$, that is, when the genetic correlation between the character states is not ± 1 and both states have non-zero genetic variance, the optimum mean phenotype in each environment will eventually be attained by the population. When the mean phenotype vector is at the joint optimum, there is no force of directional selection on the population, that is, $\nabla_k \ln \overline{W}_k = 0$ for $k = 1, 2$ and $\mathbf{D}_{ij}$ vanishes from (11). Thus disruptive selection between environments is not expected to contribute to the equilibrium genetic variance maintained in a population that has attained the joint optimum phenotype; in such a situation, the equilibrium solution for (11) is determined by a balance between pleiotropic mutation, recombination and the total force of stabilizing selection over all environments. Under weak selection ($\omega_k{}^2 \gg P_{kk}$) and loose linkage, $\mathbf{C}_{ii} \simeq \mathbf{C}_{iz}$ and (11) has an approximate solution of the form derived by Lande (1980),

$$\mathbf{C}_{ii} \simeq \mathbf{W}^{\frac{1}{2}}[\mathbf{W}^{-\frac{1}{2}}\mathbf{U}\mathbf{W}^{-\frac{1}{2}}]^{\frac{1}{2}}\mathbf{W}^{\frac{1}{2}}, \quad (14)$$

where $\mathbf{W}$ is constructed as a weighted sum of the matrices for the individual fitness functions for each environment such that $\mathbf{W}^{-1} = \sum_k q_k \overline{\mathbf{W}}_k^{-1}$. $\mathbf{W}$ is thus a diagonal matrix with elements $\omega_k{}^2/q_k$. When the mutational variances of the character states expressed in each environment are the same, we can write

$$\mathbf{U} = u\begin{pmatrix} 1 & \beta \\ \beta & 1 \end{pmatrix} \quad (15)$$

where β is the correlation of pleiotropic mutational effects on the character states. In this section, $|\beta|$ is assumed to have a magnitude less than unity. For simplicity, we also assume that $\omega_1 = \omega_2 = \omega$, $P_{11} = P_{22} = P$, and $q_1 = q_2 = \frac{1}{2}$. Then, substituting (15) into (14), the equilibrium matrix of allelic effects for the ith locus is

$$\mathbf{C}_{ii} \simeq \frac{\sqrt{2u\omega^2}}{2} \cdot \begin{bmatrix} \sqrt{(1+\beta)}+\sqrt{(1-\beta)} & \sqrt{(1+\beta)}-\sqrt{(1-\beta)} \\ \sqrt{(1+\beta)}-\sqrt{(1-\beta)} & \sqrt{(1+\beta)}+\sqrt{(1-\beta)} \end{bmatrix}. \quad (16a)$$

From (16*a*), the observed genetic correlation, γ, can be written as

$$\gamma \simeq [\sqrt{(1+\beta)}-\sqrt{(1-\beta)}]/[\sqrt{(1+\beta)}+\sqrt{(1-\beta)}] = \beta/[1+\sqrt{(1-\beta^2)}]. \quad (16b)$$

For reasons explained in the Appendix, the equilibrium genetic correlation in (16*b*) will always be closer to zero than is the correlation of mutational effects, $|\gamma| < |\beta| < 1$.

In the model discussed here, there is no selection for the different character states to be correlated ($\mathbf{W}$ is diagonal), but there can be correlation in the matrix of mutational effects ($\beta \neq 0$). It is of interest that equation 16*a* is of the same form as the equilibrium genetic structure derived by Lande (1984, equations 13*a*, *b*) when there is correlational selection ($\mathbf{W}$ not diagonal) but the characters are genetically independent. Note, however, that the equilibrium variance here is larger by a factor of $\sqrt{2}$ over that derived by Lande (1984) for two characters expressed in a single environment; in the present model, selection is effectively weakened because only part (in this case, one-half) of the population is selected in each environment each generation.

Genetic correlation between character states is ± 1. When the genetic correlation between the character states expressed in two environments is ± 1, the joint optimum phenotype may never be attained if the population is introduced into a new environment or the current environmental circumstances change (see Via & Lande, 1985 for examples). When the population mean deviates from the joint optimum phenotype, $\nabla_k \ln \overline{W}_k \neq 0$, disruptive selection between environments contributes to the equilibrium genetic variance (equations 8, 11). In the derivation of the equilibrium genetic variance for this situation, the same assumptions of symmetry made in the formulation of (16) are employed. At the point of equilibrium, the joint mean phenotype is equally far from the optimum in each environment, $\nabla_1 \ln \overline{W}_1 = -\varepsilon \nabla_2 \ln \overline{W}_2$, where $\varepsilon = \pm 1$

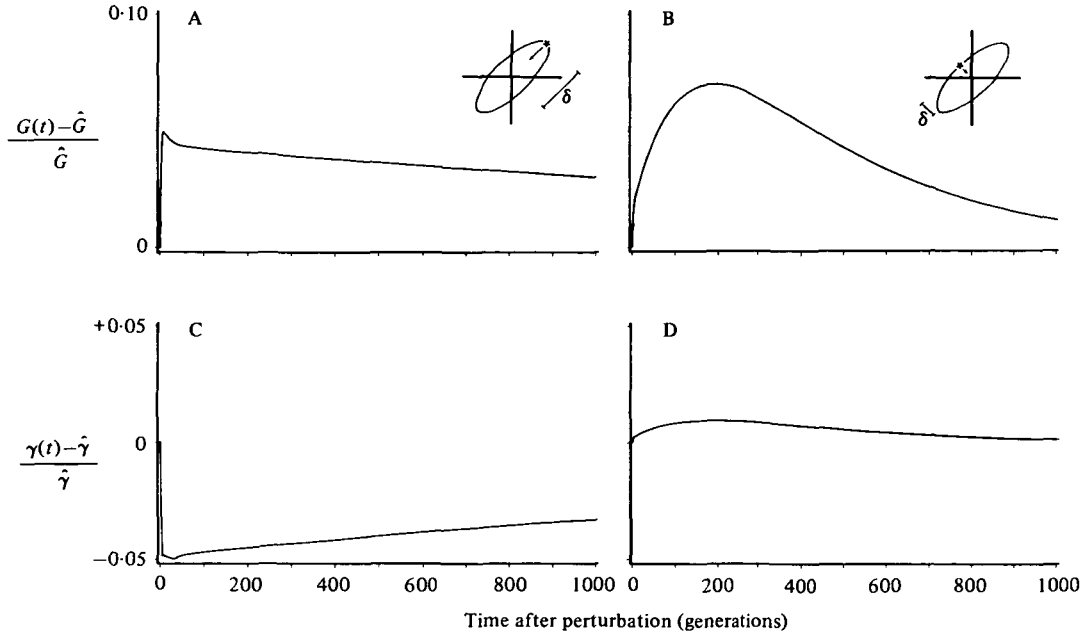

Fig. 1. Time course of change in the additive genetic variances of character states expressed in different environments or the additive genetic correlation between the character states following perturbations of the mean phenotype vector from the joint optimum. The changes in the genetic variance and genetic correlation over time are shown as a proportion of the equilibrium values $[(G(t)-\hat{G})/\hat{G}, (\gamma(t)-\hat{\gamma})/\hat{\gamma}]$. Insets indicate the direction of evolution of the mean phenotype vector after a perturbation along either the major axis (A, C) or the minor axis (B, D) of the genetic variation. Location of the mean phenotype immediately following the perturbation is denoted by the asterisk. In all cases the equilibrium genetic correlation is $\hat{\gamma} = 0{\cdot}78$, and because the examples are highly symmetrical (see text) the equilibrium heritability is the same for the character states expressed in each of the two environments, $\hat{h}^2 = 0{\cdot}42$. The environments have equal frequency, and the characters are equally variable, influenced by 10 equally mutable pleiotropic loci with free recombination. The magnitude of the perturbations (noted on the insets as δ) are in units of phenotypic standard deviations (σ_p): in A and C, $\delta = 3{\cdot}3\sigma_p$, while in B and D the perturbation that produced roughly the same increase in the genetic variance was only $\delta = 4{\cdot}3\sigma_p$. Although a perturbation in any direction temporarily increases the genetic variance (A, B), the sign of the change of the genetic correlation depends on the sign of the equilibrium genetic correlation, $\hat{\gamma}$, and the direction of the perturbation (C, D).

has the same sign as the genetic correlation between character states; that is, the forces of selection in the two environments are of equal magnitude but are directed perpendicular to the axis of the genetic correlation. When selection is weak, $\mathbf{W} \simeq \mathbf{W}+\mathbf{P}$, and when the genetic correlation between the character states is ± 1,

$$\mathbf{C}_{iz} \simeq \mathbf{C}_{ii} = c\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix} \quad \text{and for } i \neq j, \quad \mathbf{C}_{ij} = b\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix} \tag{17}$$

where b is a constant reflecting the magnitude of variance due to linkage disequilibrium. At equilibrium, when $\Delta\mathbf{C}_{ii} = \Delta\mathbf{C}_{ij} = 0$, (11) becomes

$$c^2\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix}\left[\frac{1}{2}\begin{pmatrix}\omega^{-2} & 0\\ 0 & \omega^{-2}\end{pmatrix} - \frac{(\nabla_1 \ln \overline{W}_1)^2}{4}\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix}\right] \cdot \begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix} = u\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix} \quad \text{for } i = j \text{ and} \tag{18a}$$

$$c^2\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix}\left[\frac{1}{2}\begin{pmatrix}\omega^{-2} & 0\\ 0 & \omega^{-2}\end{pmatrix} - \frac{(\nabla_1 \ln \overline{W}_1)^2}{4}\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix}\right] \cdot \begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix} = -b\begin{pmatrix}1 & \varepsilon\\ \varepsilon & 1\end{pmatrix} \quad \text{for } i \neq j. \tag{18b}$$

The two terms inside the square brackets correspond to the forces of stabilizing and disruptive selection, respectively. Equating (18*a*) and (18*b*) allows us to conclude that at equilibrium $b = -2u$, which is consistent with the magnitude of linkage disequilibrium predicted for the one character case (Bulmer, 1980, p. 178).

Assuming that $u \neq 0$, the equilibrium variance can be approximated by multiplying (18*a*) to yield four identical equations with the solution

$$c \simeq \sqrt{\frac{u\omega^2}{1-\omega^2(\nabla_1 \ln \overline{W}_1)^2}}. \tag{19a}$$

With Gaussian fitness functions, $\nabla_1 \ln \overline{W}_1 \simeq -(\bar{z}_1 - \theta_1)/\omega^2$. The equilibrium mean phenotype is a function of the initial conditions: $\hat{\bar{z}}_1 = \frac{1}{2}[\bar{z}_1(0) - \varepsilon\bar{z}_2(0) + (\theta_1 + \varepsilon\theta_2)]$. Using this expression for $\hat{\bar{z}}$, the solution in (19) can be written in terms of initial conditions as

$$c \simeq \omega^2\sqrt{\frac{u}{\omega^2 - (1/4)[\bar{z}_1(0) - \varepsilon\bar{z}_2(0) - (\theta_1 - \varepsilon\theta_2)]^2}}. \tag{19b}$$

If the width of the fitness function (ω) is not very small or the equilibrium mean phenotype is not very far from the optimum, $(\bar{z}_1 - \theta_1)^2 < \omega^2$, the denominator in (19) will be positive and less than unity. Then c in (19)

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will be larger than the diagonal elements of $\mathbf{C}_{ii}$ in (16) for genetic correlations close to ± 1, illustrating the increase in genetic variance expected from disruptive selection between environments that persists at equilibrium in this special case.

It should be noted that our formulae (18, 19) assume weak selection and break down in the case of no mutation, $u = 0$, because of the approximation $\mathbf{W} \simeq \mathbf{W} + \mathbf{P}$. Bulmer (1980, ch. 10) analysed more precisely the maintenance of genetic variability by disruptive selection on a single character in the two-niche model in the absence of mutation. In agreement with Felsenstein (1977), Bulmer (1980) found that in the absence of mutation, variance due to linkage disequilibrium was zero, that is, when $u = 0$ $b = 0$.

(iv) *Transient effects of disruptive selection on the genetic covariance*

When a population is perturbed away from the joint optimum, the genetic variance will increase and the genetic covariance between the character states will be changed by the force of disruptive selection between environments as described in (8). In order to estimate the maximum genetic variance attained by populations that are perturbed from the phenotypic optimum and to track the effect of the decreasing $\mathbf{D}_{ii}$ term on the genetic covariance matrix, the dynamics of $\mathbf{G}$ were studied numerically. For the numerical examples, (11) was coupled to equations describing the evolution of the mean phenotype (Via & Lande, 1985, Equation 5). The numerical examples were started with the mean phenotype at various distances from the optimum on one of the eigenvectors of the dynamical system for the mean phenotype, $\mathbf{y}_1 = (1/\sqrt{2})(1, 1)^T$ or $\mathbf{y}_2 = (1\sqrt{2})(1, -1)^T$ (Via and Lande, 1985). Initially, $\mathbf{G}$ was at equilibrium values that were estimated by iterating (11) with the mean phenotype at the optimum until the variance in successive generations was stable to 5 decimal places; in these symmetrical examples, $G_{11} = G_{22} = G$, $q_1 = q_2 = \frac{1}{2}$, and 10 equally mutable loci with free recombination contributed to the variability.

Results of the numerical studies indicate that when populations at the equilibrium genetic covariance structure are perturbed from the phenotypic optimum, the genetic variance increases to a maximum and then declines gradually back to the equilibrium value (Fig. 1 A, C). The genetic correlation between the character states also changes temporarily (Fig. 1 B, D). The sign of this change in the genetic correlation depends both on the sign of the equilibrium genetic correlation, $\hat{\gamma}$, and the direction of the perturbation of the mean phenotype. Often, the equilibrium genetic covariance matrix will not be re-attained until many generations after the mean phenotype has already reached its optimum.

In most cases the mean phenotype can be perturbed a considerable distance from the phenotypic optimum before disruptive selection causes an increase in genetic variation or a change in the genetic correlation of more than a few per cent (Fig. 2). Fig. 2 also shows that for Gaussian fitness functions, the maximum increase in genetic variance due to disruptive selection expressed as a proportion of the equilibrium variance

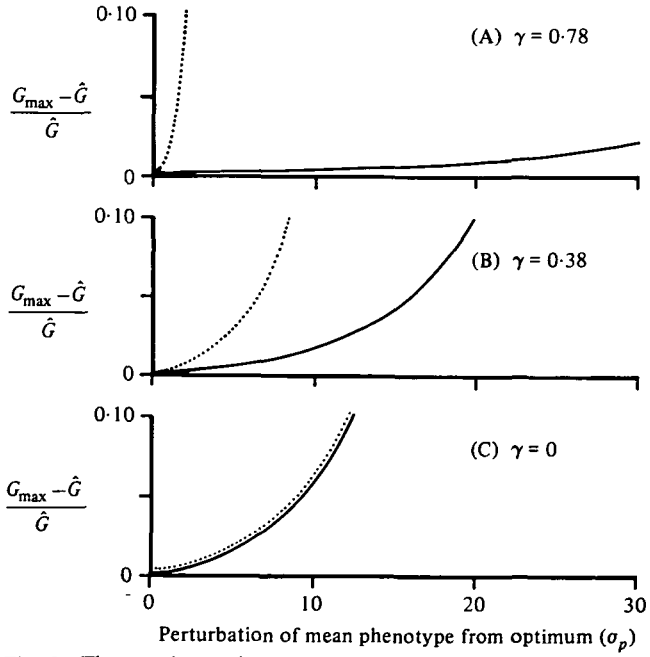

Fig. 2. The maximum increase expected in the genetic variance following perturbations of the mean phenotype of different magnitudes and directions, expressed as a proportion of the equilibrium genetic variance $[(G_{max} - \hat{G})/\hat{G}]$. For each of three values of the genetic correlation ($\hat{\gamma} = 0{\cdot}78$, $0{\cdot}38$, 0), the proportional increase in the genetic variance for perturbations in the directions of the major and minor axes of the genetic variation (see insets for Fig. 1 A and B) are shown as solid and dotted curves, respectively. When $\hat{\gamma} = 0$, these curves are coincident.

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$[(G_{max} - \hat{G})/\hat{G}]$ increases rapidly as the deviation from the optimum becomes greater; this is expected from (8). Finally, Fig. 2 illustrates that for $\hat{\gamma} \neq 0$, the maximum amount of change in the genetic variance during re-adaptation of the mean phenotype is much greater for perturbations of the mean in the direction of the minor axis of the genetic variation (against the genetic correlation) than it is for perturbations of the same magnitude in the direction of the major axis of the genetic variation.

Unequal mutational parameters for different loci. To determine whether asymmetry in mutational parameters across loci could produce a qualitative change in the results of the numerical examples discussed above, equation (11) was iterated for an additive genetic correlation of zero ($\hat{\gamma} = 0$), produced by summing mutational effects over two classes of loci: five loci had $\beta = +0{\cdot}5$, and five loci had $\beta = -0{\cdot}5$. If selection in a given direction were to produce more rapid change in the covariance structure for one class of loci than for the other, it would be noted as a difference between the dynamics of **G** for the symmetrical and asymmetrical cases (both with $\hat{\gamma} = 0$). No such difference was found in numerical examples in which two character states were selected to change either in the same direction or in different directions. Thus the results of the symmetrical cases illustrated in Figs. 1 and 2 are robust with respect to differences among loci in mutational parameters.

3. Discussion

The effect of a heterogeneous environment on genetic variability is a classical problem in population genetics. Single-locus genetic models suggest that polymorphism may often be maintained by spatial variation in selection (reviewed in Hedrick *et al.* 1976; Felsenstein, 1976). The models presented here extend the classical 'multiple-niche' models of evolution in variable environments in two ways: (1) we consider selection on quantitative (polygenic) traits instead of only the fitnesses of single-locus genotypes, and (2) we describe the dynamics of genetic variability in addition to the evolutionary equilibria.

We consider a simple 2-environment situation and regard a character expressed in these two environments as a pair of genetically correlated *character states* (Falconer, 1952; Via, 1984*b*; Via & Lande, 1985). In the coarse-grained model discussed here, any individual experiences only one environmental type and expresses only one of the possible character states. Each character state is independently selected toward an intermediate optimum phenotype in the environment in which it is expressed.

Under certain special conditions, the magnitude of the genetic correlation between character states expressed in different environments can affect the equilibrium genetic variability that is maintained in a polygenic trait under selection in a spatially variable environment. If the genetic correlation between the character states expressed in different environments is ± 1, the joint optimum phenotype will not be attained and there will be a persistent force of disruptive selection to move populations in each environment closer to the optimum phenotype for that habitat type. In such a circumstance, the genetic variance that would be maintained by balance of mutation and stabilizing selection within each environment is augmented by the effect of disruptive selection between environments (equations 18, 19). This result was also obtained by Felsenstein (1977), Slatkin (1978) and Bulmer (1980, ch. 10). The amount by which the equilibrium genetic variance is augmented by such a force of disruptive selection increases non-linearly (equation 19) with the distance of the equilibrium mean phenotype vector from the joint optimum.

In contrast, if the genetic variances of both the character states are greater than zero and the genetic correlation between the states is not ± 1, the average phenotype expressed in each environment will eventually reach the optimum value (Via & Lande, 1985). The equilibrium genetic variances of the two character states are then determined by a balance between mutation and the forces of stabilizing selection acting within environments (equation 16). For a pair of character states under independent forces of selection that are less than perfectly genetically correlated, there is thus no unique effect of disruptive selection in a heterogeneous environment on the genetic covariance structure of the character states; in such a case, the equilibrium in (16) is of a form identical to that for genetically distinct (not mutually pleiotropic) traits that experience selection to become correlated (Lande, 1984, equation 13*a*, *b*).

The occurrence of perfect genetic correlations among character states is an empirical problem. At present, few studies of natural populations exist that have estimated the genetic correlations between character states expressed in different environments (Via, 1984*b*; Weber, 1985; Shaw, 1986; Futuyma, personal communication). Unfortunately, experimental studies of this issue will require very large sample sizes to statistically distinguish high from perfect genetic correlations among character states.

If changes in the environment occur that alter the phenotypic optimum in any environment, the genetic covariance structure can be expected to change temporarily as a result of the action of disruptive selection between the environments (equations 8, 11). After such a perturbation, genetic variances will increase and, for a positive (negative) genetic correlation, the genetic covariance between character states will decrease (increase) if the character states in the two environments are selected in the same direction or increase (decrease) if they are selected in different directions. As shown in Fig. 1, the change in the genetic correlation between character states that is

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caused by disruptive selection between environments is opposite to that which would facilitate the evolution of the joint mean phenotype. Thus disruptive selection does not break unfavourable genetic correlations among character states expressed in different environments; rather, disruptive selection between environments acts to reduce favourable genetic correlations among character states and thereby to increase constraints on the evolution of the average phenotype.

Unlike single-locus models with only two alleles, the mean and the variance in quantitative genetic models are not completely determined by a single allele frequency, and can be assumed to evolve nearly independently. Thus one assumption of our models is that a perturbation of the mean phenotype vector from its joint optimum does not alter the effect of stabilizing selection within environments on the dynamics of the genetic variance (equations 3, 13). After a perturbation of the mean phenotype vector, the dynamics of **G** are determined solely by the approximately quadratic decay of the force of disruptive selection between environments as the mean phenotype vector moves towards the joint optimum (equation 8).

Numerical investigations of the dynamics of the genetic covariance matrix (equation 13) suggest that a population at the equilibrium genetic variance and covariance can experience a single perturbation of the mean phenotype vector by several phenotypic standard deviations from the joint optimum in most directions without an increase in the genetic variance or a change in the genetic correlation between the character states of more than a few per cent (Figs. 1, 2). The magnitude of the change in **G** for a given perturbation depends critically on the value of the genetic correlation between the character states; when the genetic correlation is not zero, evolution of the joint mean phenotype 'against' the genetic correlation (along the minor axis of genetic variation) will proceed more slowly than will evolution in the other direction.

The numerical examples in Fig. 2 suggest that the genetic covariance structure can be expected to remain nearly constant through time if the phenotypic optimum and the width of the fitness function (ω) are relatively stable and populations are perturbed only rarely and by moderate amounts. Under such circumstances, the dynamics of evolution of the joint mean phenotype in a large population inhabiting a heterogeneous environment may be reasonably described by models that assume a constant genetic covariance matrix (e.g. Via & Lande, 1985). If, however, the phenotypic optimum in any of the environments experienced by a population frequently shifts, or if the population is often perturbed more than a few standard deviations in a direction against the genetic correlation (see Fig. 1), the transient increases in the genetic variances caused by disruptive selection could accumulate. We analysed the transient effects of only a single episode of disruptive selection. The extent to which the cumulative effects of repeated episodes of disruptive selection between environments may affect the genetic variability in natural populations is an empirical problem which will require long-term studies of natural selection and population structure in heterogeneous environments.

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Appendix

In this model there is no selection for the character states expressed in different environments to be correlated because selection is assumed to operate independently in each environment. Nonetheless, stabilizing selection within environments does act to change the genetic covariance between the character states. Following the derivation in Lande (1977, p. 494), with equal strengths of stabilizing selection in two environments ($\omega_1 = \omega_2 = \omega$), $q_1 = q_2 = \frac{1}{2}$, γ as the additive genetic correlation between the character states, and v as the proportional decrease in the genetic variance in each environment after selection, the genetic variances after selection (denoted by asterisks) are

$$G_{11}^* = G_{22}^* = \tfrac{1}{2}[(1-v)+(1-v\gamma^2)]G_{11} = [1-(v/2)(1+\gamma^2)]G_{11} \quad \text{(A 1)}$$

and

$$G_{12}^* = \tfrac{1}{2}[(1-v)+(1-v)]G_{12} = (1-v)G_{12} \quad \text{(A 2)}$$

Thus, the genetic covariance between the character states expressed in different environments decreases as a result of forces of stabilizing selection acting independently on each character state within environments. Equations A 1 and A 2 illustrate that for $|\gamma| < 1$, the covariance between the character states actually decreases faster than the variance, because the covariance is affected equally by stabilizing selection in both environments, while the variance of each character state decreases less in the environment where it is not expressed.