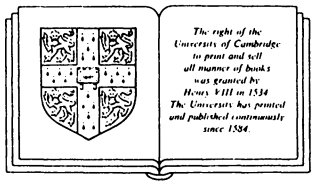
Evolutionary physiological ecology

Edited by
P. Calow
Department of Zoology
University of Sheffield



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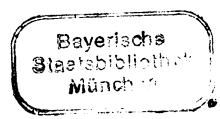

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CONTRIBUTORS

(First authors only)

B.L. BAYNE
Institute for Marine Environmental Research, Prospect Place, Plymouth, PL1 3DH, U.K.

P. CALOW
Department of Zoology, University of Sheffield, Western Bank, Sheffield S10 2TN, U.K.

E. GNAIGER
Institute Zoophysiologie, Universitat Innsbruck, Peter-Mayr-Strasse 1a, A-6020, Innsbruck, Austria.

J.P. GRIME
Unit of Comparative Ecology (NERC), University of Sheffield, Western Bank, Sheffield S10 2TN, U.K.

T.B.L. KIRKWOOD
National Institute for Medical Research, The Ridgeway, Mill Hill, London NW7 1AA, U.K.

A.L. KOCH
Department of Biology, Indiana University, Bloomington, Indiana 47405, U.S.A.

M. LYNCH
Department of Ecology, Ethology and Evolution, Shelford Vivarium, 606 E. Healey Street, University of Illinois, Champaign, Illinois 61820, U.S.A.

B.K. McNAB
Department of Zoology, University of Florida, Gainesville, Florida 32611, U.S.A.

R.M. SIBLY
Department of Zoology, University of Reading, Whiteknights, Reading, RG6 2AJ, U.K.

L.B. SLOBODKIN
Department of Ecology and Evolution, Division of Biological Sciences, State University of New York at Stony Brook, Stony Brook, New York 11794, U.S.A.

O.T. SOLBRIG
Gray Herbarium, Harvard University, 22 Divinity Avenue, Cambridge, Mass. 02138, U.S.A.

R.LeB. DUNBRACK
Department of Fisheries and Oceans, Pacific Biological Station, Nanaimo, B.C. V9R 5K6, Canada.

EVOLUTION OF THE BREADTH OF BIOCHEMICAL ADAPTATION

M. Lynch
W. Gabriel

INTRODUCTION

The existence of variation at the gene level is now accepted to be ubiquitous among species, although the extent of variation is known to differ among species (Nei, 1975; Ayala, 1976; Nei & Koehn, 1983; Nei & Graur, 1984). Much, but not all, of this variation can be explained by the neutral theory of molecular evolution (Kimura, 1983) which assumes that the dominant forces underlying the dynamics of gene frequency change are mutation and the random drift resulting from finite population size. While the neutral theory has been particularly successful in explaining the approximate constancy of the long-term rate of gene substitution, there are many short-term and localized properties of structural gene polymorphisms that can only be explained by selection, and a consensus is gradually emerging that the fit of the neutral theory to existing data is improved when the theory is modified to incorporate a second kind of drift due to random variation in selection intensity (Matsuda & Gojobori, 1979; Takahata & Kimura, 1979; Takahata, 1981; Nei & Graur, 1984).

These recent developments are of interest since a very substantial amount of mathematical theory on the maintenance of genetic polymorphisms via spatial and temporal heterogeneity in the environment has been formulated, largely as an alternative to the neutral theory (for comprehensive reviews and syntheses see Karlin & Lieberman, 1974; Christiansen & Feldman, 1975; Felsenstein, 1976; Hedrick et al., 1976; Gillespie, 1978). The object of most of this theory has been to demonstrate how environmental variation can serve as a form of balancing selection, thereby preserving genetic polymorphisms. With the exception of the studies of Takahata & Kimura (1979); Takahata (1981) and Tier (1981), most of this theory either does not incorporate mutation or does so only in a very restrictive manner (two alleles with reversible mutation), and therefore has

very little to contribute to our understanding of the evolution of biochemical adaptation. That is, while the theory may explain the degree of heterozygosity maintained when alleles are exposed to different schedules and spatial patterns of selection intensity, it does not consider the underlying determinants of an allele's sensitivity to environmental fluctuations.

In addition to its potential role in promoting polymorphisms, environmental heterogeneity may also select for genes whose biochemical products can maintain their functional integrity in the face of such variation. However, perhaps because environmental heterogeneity comes in many forms (spatial and temporal variance, both within and between generations, and in the genetic as well as the environmental background) and is generally subjectively defined, little mathematical theory or empirical work has been focused on the problem of breadth of biochemical adaptation. Only a verbal argument presented by Ayala and Valentine (Ayala et al., 1975; Valentine, 1976; Ayala & Valentine, 1979) bears directly on the issue.

Drawing from the fitness set theory of Levins (1968) as well as from considerable electrophoretic data that indicate a positive correlation between the stability of trophic resources and the level of genetic variation in pelagic and benthic marine invertebrates, the Ayala-Valentine hypothesis states that functionally-broad alleles that encode for a highly generalised phenotype are strongly favoured in temporally variable environments. Narrowly-adapted alleles are purported to be strongly selected against under these conditions, thereby resulting in a high degree of homozygosity. In more temporally stable environments, the spatial component of heterogeneity is thought to take precedence such that microhabitat specialization results in the maintenance of a variety of narrowly-adapted alleles through balancing selection. The Ayala-Valentine hypothesis is unique in its focus on both the properties of loci (polymorphism and heterozygosity) and genes (functional breadth), and it has subsequently been invoked in modified form to explain patterns of genetic variation in marine decapods (Nelson & Hedgecock, 1980) and marine fishes (Smith & Fujio, 1982).

The hypothesis can also be criticised on a number of grounds (Soule, 1976; Nei & Graur, 1984). First, there are a number of species, such as the cave fish Astyanax mexicanus (Avise & Selander, 1972) and fossorial mammals (Nevo et al., 1974), which are thought to live in highly stable environments but have exceptionally low levels of genetic variation. Any criticism on these grounds is weak, since it can always be argued that a small effective population size is responsible for the reduced amount of

heterozygosity. A second and more serious problem is the subjective nature of the hypothesis. Unless the level of environmental heterogeneity can be objectively defined from the standpoint of the organism, there is little hope for testing the hypothesis. Finally, it is not clear that the verbal reasoning of Ayala & Valentine has led to the correct prediction. For example, it is not clear why microhabitat specialization cannot be equally or more pronounced in temporally variable environments than in stable ones. The existing theoretical work on the relation of environmental heterogeneity to heterozygosity alone is exceedingly complex, and with slight changes in assumptions, different investigators have often reached radically different conclusions. Modification of the existing theory to allow for the evolution of environmental sensitivity at the gene level is likely to further complicate the theory for genetic variability to an even greater degree.

Despite the shortage of attention that the problem of breadth of biochemical adaptation has received from evolutionary biologists, there is no a priori reason to expect it to be any less important as a means of genetic adaptation to variable environments than the maintenance of heterozygosity. Indeed, several empirical studies suggest that something other than the promotion of polymorphisms must occur when populations are exposed to increasing levels of environmental heterogeneity. While Powell (1971), Powell & Wistrand (1978) and McDonald & Ayala (1974) all found strikingly higher levels of genetic variance in laboratory populations of Drosophila exposed to spatial and temporal heterogeneity compared to controls, more recent studies (Minawa & Birley (1978); Mackay (1980), (1981); Haley & Birley (1983); Zirkle & Riddle (1983) have obtained either mixed or contrary results. Moreover, Nei (1980) has pointed out that, even in the presence of environmental heterogeneity, heterozygosity in the populations of Powell (1971), Powell & Wistrand (1978), and McDonald & Ayala (1974) was eroded more rapidly than could be accounted by random sampling drift; i.e. even in the studies with results most concordant with theoretical predictions, environmental heterogeneity enhanced the rate of loss of genetic variance relative to expectations under selective neutrality. Clearly, the problem of what environmental heterogeneity selects for is far from resolved.

Although we recognize the importance of genetic variation in heterogeneous environments, we will not concern ourselves with this issue here, focusing instead on the development of a theory for the evolution of the breadth of adaptation at the gene level. We therefore address the Ayala-Valentine hypothesis only in part. Much of what follows will take on an

adaptational tone since we are primarily concerned with identifying the optimal level of functional flexibility of an allele in an effort to formalize the argument of Ayala and Valentine. Elsewhere, we will examine the extent to which the intensity of selection for alleles, and hence the likelihood of the evolution of the optimum and of the maintenance of heterozygosity, is modified by the level of environmental variation (Lynch & Gabriel, in prep.). We will also present our mathematical derivations in detail elsewhere, restricting our attention here only to the most fundamental definitions and formulae.

THE GENIC FITNESS FUNCTION

In order to explore the consequences of environmental heterogeneity for the evolution of gene properties, we require a theory that explicitly links the fitness of an allele to its environment. In the following, we define the genic fitness function, $w(g_1,g_2|\phi)$, as the expected fitness of an allele over a continuous environmental gradient, ϕ . It is easiest to think of ϕ as a density-independent parameter such as temperature, and the allele as a gene encoding for an enzyme. Associated with any allele will be a mean environmental optimum (g_1) and a measure of functional flexibility (g_2). A precise definition of g_2 will follow below, but in effect it is a measure of the equitability of the fitness of an allele over the environmental gradient. It is important to note that g_1 and g_2 are not <u>in vitro</u> properties of an allele. On the contrary, they are functions of the integrated phenotype, being defined as the average environmental optimum and breadth of all <u>individuals</u> containing the allele.

In the derivation of a fitness estimate for allele (g_1,g_2) two sources of variation must be taken into consideration. First, because of mutation, recombination, segregation, and gene flow, any gene is likely to be found in a multitude of genetic backgrounds. Moreover, variation in the environment is likely to further magnify the diversity of phenotypes within which a gene resides. The environment within which an individual develops may have long-lasting effects on the phenotype (Falconer, 1981), and short-term changes may be accomplished by physiological acclimation or behavioral modification (Hochachka & Somero, 1973; Ricklefs, 1979). Thus, while (g_1,g_2) is defined to be the expected phenotype of an individual with the allele, the actual genic fitness function is determined by the conditional phenotype distribution $p(z_1,z_2 \mid g_1,g_2)$, as well as by the phenotypic fitness function, $w(z_1,z_2 \mid \phi)$ (Fig. 1), such that:

$$w(g_1,g_2|\phi) = \int_0^{+\infty} \int_{-\infty}^{+\infty} w(z_1,z_2|\phi).p(z_1,z_2|g_1,g_2).dz_1 dz_2 \quad (1)$$

The second source of variation that influences the fitness of a gene is the variance in the environmental parameter ϕ due to spatial heterogeneity and temporal fluctuations. We will take this topic up in some detail in the following section. First, however, we consider the fundamental relationship of the fitness of a gene to the environment.

We assume that the environmental state, ϕ , is measured on a scale such that the phenotypic fitness function is normalised,

$$w(z_1,z_2|\phi) = (2\pi z_2)^{-1/2} \exp[-(z_1-\phi)^2/2z_2] \quad (2)$$

For an individual with phenotype (z_1,z_2), z_1 is the environmental state in which fitness is maximized, and z_2, the "variance" of the fitness function. Note that z_1 and z_2 are measured on different scales, a small inconvenience that can be eliminated by square root transformation of the latter. Throughout this paper will refer to $\sqrt{z_2}$ as the environmental breadth of a phenotype. Note also that a cost to being a generalist is implicit in equation (2), since any increase in $\sqrt{z_2}$ results in a reduction of fitness in the optimal environment while increasing fitness in more extreme environments. Such a cost is implicit in the Ayala-Valentine hypothesis and throughout the evolutionary ecological literature (MacArthur, 1972; Pianka, 1978), although as emphasised by Futuyma et al., (1984) and Huey & Hertz (1984) empirical tests of the idea are almost totally lacking.

We further assume that the genes that contribute to an individual's environmental optimum and breadth have additive effects both within and between loci (for supportive arguments for enzymatic loci, see Gillespie & Langley, 1974 and Kacser & Burns, 1981) and that the conditional phenotype distributions for z_1 and z_2 are independent so that

$$p(z_1,z_2|g_1,g_2) = p(z_1|g_1).p(z_2|g_2) \quad (3)$$

The conditional phenotype distribution for the environmental optimum, $p(z_1|g_1)$, is taken to be normal with mean g_1, and variance V'_{T1}. V'_{T1} includes the variance in environmental effects that contribute to the optimum, V_{E1}, and all of the genetic variance for the trait conditional on one copy of the gene being present at the locus. Thus,

Fig. 1. Mean fitness function for a gene (solid line) and for a few of the phenotypes within which it is found.

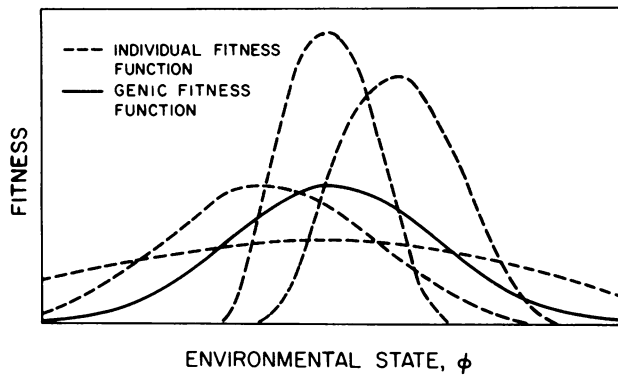
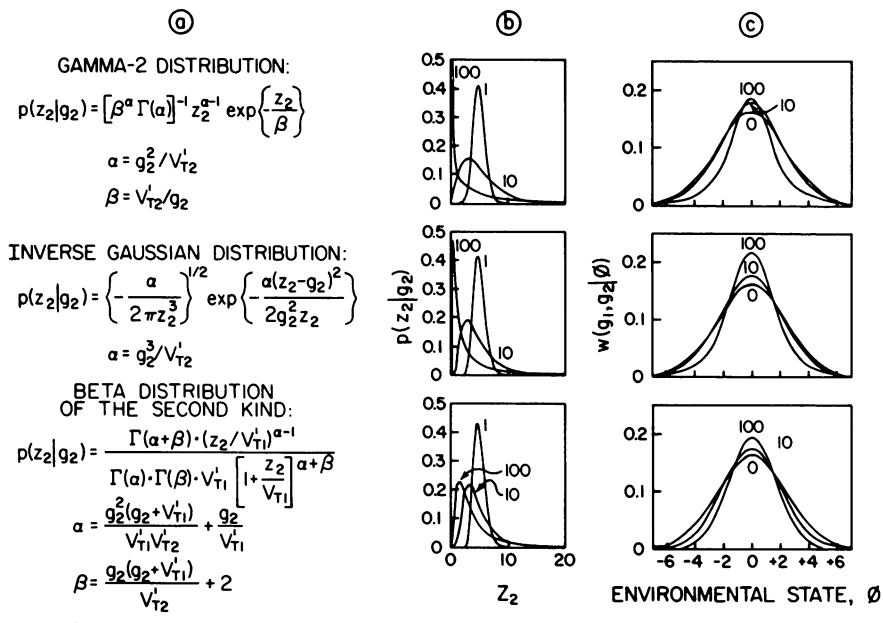


Fig. 2. Three conditional phenotype distributions for the square of environmental breadth (z_2), shapes of the distributions for V'_{T2} = 1, 10, 100, and shapes of the genic fitness function when applied to equation (1) with $g_1=0$, $g_2=5$, and $V'_{T1}=1$. For gamma-2 and inverse Gaussian distributed z_2, the genic fitness function was obtained by numerical integration (Gauss-Laguerre quadrature), whereas the approximate analytical solution (equation (5)) was used in the case of the beta distribution of the second kind.



$$V'_{T1} = \rho_1 V_{G1} + V_{E1} \qquad (4)$$

where V_{G1} is the total genetic variance for the optimum. Note that ρ_1 takes on a minimum value of 0.5 when no other loci encode for the environmental optimum, since only a single gene is free to vary. This low value may also be approached if the locus under consideration is in extreme linkage disequilibrium with other loci encoding for the environmental optimum. However, if linkage is not strong, $\rho_1 \to 1$ as the number of loci increases.

Since a variance cannot be <0, the conditional distribution for environmental breadth cannot be normal. Moreover, it does not seem biologicallly reasonable for z_2 to ever equal zero exactly. In order to separate genetic from environmental effects a distribution for which the variance is independent of the mean is desirable. Three distributions that meet these requirements are outlined in Fig. 2a. In each case the expectation of z_2 is g_2 and the variance of z_2 is V'_{T2} (defined in the same manner as V'_{T1}). All three distributions are very similar if the coefficient of variation $[(V'_{T2})^{\frac{1}{2}}/g_2] < 0.5$, and although they differ quantitatively for larger V'_{T2}, their qualitative behaviour is the same (Fig. 2b). When $(V'_{T2})^{\frac{1}{2}} \ll g_2$, $p(z_2 | g_2)$ is approximately normal; but as V'_{T2} increases, the conditional distribution becomes increasingly asymmetrical with the mode approaching the origin. In the following analyses we rely on the beta distribution of the second kind for $p(z_2 | g_2)$ as it is the only case for which we have been able to obtain an analytical solution for the genic fitness function.

The solution to equation (1) for normally distributed $p(z_1 | g_1)$ and beta distributed $p(z_2 | g_2)$ is somewhat involved and is presented elsewhere (Lynch & Gabriel, in press). It is sufficient to note here that if the scale is set such that $V'_{T1} = 1$, then provided $g_2 > 1$ (an assumption that is supported by analyses in the next section) and the coefficient of variation $[(V'_{T2})^{\frac{1}{2}}/g_2] < 1$ (a liberal upper limit for most quantitative traits), the genic fitness function is closely approximated by

$$w(g_1, g_2 | \phi) = (2\pi V')^{-1/2} \exp[-(g_1-\phi)^2/2V'] \qquad (5)$$

where

$$V' = V'_{T1} + \frac{g_2\{g_2(g_2+V'_{T1}) + V'_{T2}\}}{g_2(g_2+V'_{T1}) + 2V'_{T2}} \quad . \qquad (6)$$

Thus, the genic fitness function is approximately normal with maximum fitness in environment $\phi = g_1$, and variance V'. In the following we will refer to $\sqrt{V'}$ as the realised environmental breadth for a gene. Note that for the special case in which $V'_{T2} = 0$, equation (5) reduces to the exact solution of equation (1) under those circumstances:

$$w(g_1,g_2|\phi) = [2\pi(g_2+V'_{T1})]^{-1/2} \exp[-(g_1-\phi)^2/2(g_2+V'_{T1})] \quad (7)$$

and that at the upper limit of the applicability of the normal approximation, $[(V'_{T2})^{1/2}/g_2] \simeq 1$. the genic fitness function is approximately

$$w(g_1,g_2|\phi) = \{2\pi[(2g_2/3)+V'_{T1}]\}^{-1/2} \exp\{-(g_1-\phi)^2/2[(2g_2/3)+V'_{T1}]\} \quad (8)$$

Thus, depending on the genetic and environmental background, an allele's realised environmental breadth is expected to fall within the range of $[(2g_2/3)+V'_{T1}]$ and $[g_2+V'_{T1}]$.

In summary, V'_{T1} and V'_{T2} have conflicting effects on the genic fitness function. Variance for the environmental optimum always results in a flattening of the fitness function, thereby endowing an allele with functional flexibility, whereas variance for environmental breadth has the opposite effect. The latter effect is not unique to the beta distribution of the second kind (Fig. 2c).

SPATIAL AND TEMPORAL HETEROGENEITY

Having defined the fundamental relationship between genic fitness and ϕ, we now proceed to evaluate the realised fitness of an allele with properties (g_1,g_2) in environments characterised by different degrees of spatial and temporal variation. For a population growing in discrete generations, we identify the mean environmental state over all microhabitats in generation t as ϕ_t. If the environment is spatially heterogeneous relative to the mobility of individuals, then it is likely that the actual mean environmental state experienced by an individual will be somewhat different from ϕ_t. We will take the mean environmental state experienced by individuals, ϕ_s, to be normally distributed with expectation ϕ_t and variance $V_{\phi s}$. Note that $V_{\phi s}$ is a measure of spatial heterogeneity perceived by the population. It is likely that $V_{\phi s}$ will be higher for a sedentary species than for a mobile species in the same environment. However, this need not always be the case, since dominance hierarchies in behaviourally sophisticated

organisms might actually inflate the variance in mean environmental states experienced by different individuals.

The second source of environmental variation that we incorporate is the temporal variance in ϕ experienced by individuals within a generation, $V_{\phi tw}$. We will assume that the level of temporal heterogeneity experienced by individuals is independent of their mean environmental state, but make no assumptions regarding the temporal distribution of ϕ.

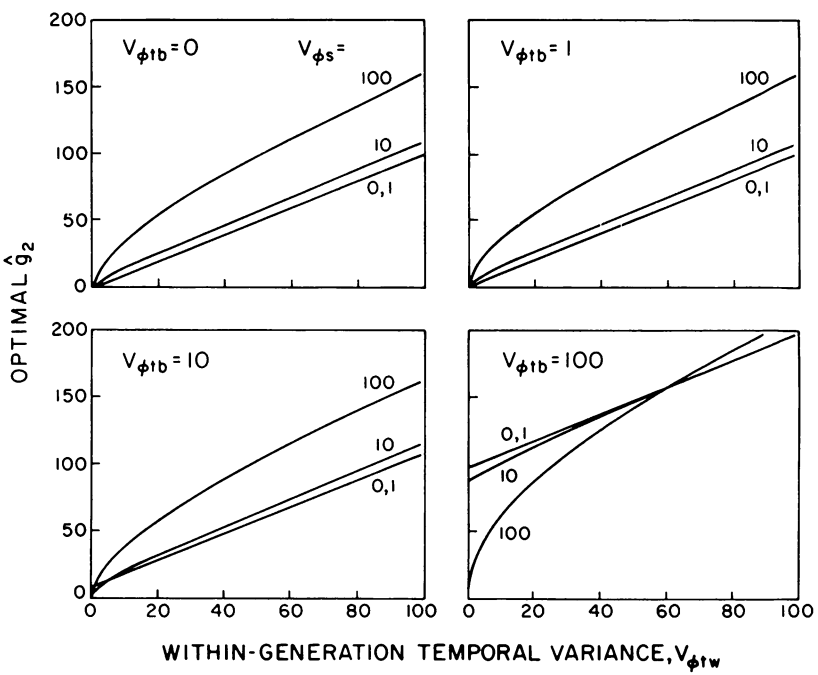
Before proceeding, it is worth considering the relationship of $V_{\phi s}$ and $V_{\phi tw}$ to the concept of environmental grain (Levins, 1968) which is frequently alluded to in the ecological genetic literature. An organism is said to perceive its environment as fine-grained if it passes through many patches in its life time. As individuals spend a greater proportion of their lives in a single microhabitat, the environment is said to be more coarse-grained. While the concept of grain is used in the context of spatial heterogeneity, it also has an element of temporal heterogeneity embedded in it. In our terminology, a fine-grained environment is one in which $V_{\phi s}$ is relatively low but $V_{\phi tw}$ is relatively high. That is, $\phi_s \simeq \phi_t$ for most individuals since they all pass through most patch types in their life times, but the movement between patches with different environmental states increases $V_{\phi tw}$. In the most fine-grained of environments, $V_{\phi s}$ will be essentially zero, but even in the most coarse-grained of environments, while $V_{\phi tw}$ will be reduced, it cannot be less than the temporal variance of ϕ which occurs within a microhabitat. Thus, the definition of environmental grain, which has heretofore been used primarily from a heuristic standpoint, is formalized by the use of $V_{\phi s}$ and $V_{\phi tw}$.

A more general interpretation of $V_{\phi s}$ and $V_{\phi tw}$ is to consider them to be the total variance in additive and multiplicative effects on fitness resulting from environmental heterogeneity. While we have defined the spatial component of environmental variation such that it has an additive influence on genic fitness, temporal variance in ϕ within generations influences fitness geometrically (as when daily probabilities of survival interact multiplicatively to determine annual survivorship). Using techniques that we present elsewhere (Lynch & Gabriel, in press), the fitness of allele (g_1,g_2) in generation t is found to be

$$w(g_1,g_2,t) = \{2\pi(V'+V_{\phi s})\}^{-1/2}.\exp\left\{-\frac{1}{2}\left[\frac{V_{\phi tw}}{V'}+\frac{(g_1-\phi_t)^2}{V'+V_{\phi s}}\right]\right\} \qquad (9)$$

Finally, in order to determine the relative long-term advantages of different alleles, we incorporate the variance in ϕ_t between generations. In extending our analysis across generations, we adopt the geometric mean fitness of a gene as a measure of its relative success (Dempster, 1955). In order for us to make any analytical progress, it is necessary to assume constancy of the genetic background (ρ_1, ρ_2 , V_{G1}, and V_{G2}) throughout the period of selection. Approximate constancy of these parameters can be expected to result from selection-mutation balance in an environment that

Fig. 3. The optimum g_2 of a gene product as a function of the spatial ($V_{\phi s}$), within-generation temporal ($V_{\phi tw}$), and between-generation temporal ($V_{\phi tb}$) variance. Solutions are for the case in which $g_1 = \bar{\phi}_t$ (i.e. the optimal environmental optimum has been attained), $V'_{T1} = 1$, and $V'_{T2} = 0$.



does not change between generations, but stochastic variation in ϕ_t must promote variance in the genetic background. Provided the distribution of ϕ_t is stochastically stable, we anticipate that stochastically stable distributions will also arise for ρ_1, ρ_2, V_{G1}, and V_{G2}, thereby preventing (or retarding) complete fixation or loss of alleles with different properties in effectively infinite populations (Gillespie, 1978). We hope to address this complicated issue in the future. For now we simply take the position that the expected frequency of an allele will be positively correlated with its geometric mean fitness as defined below.

Setting the environmental scale so that the long-term mean environmental state (average ϕ_t) is zero, and letting the variance in ϕ_t between generations be $V_{\phi tb}$, the geometric mean fitness of an allele with properties (g_1,g_2) is found using the techniques in Lynch & Gabriel (in press),

$$w(g_1,g_2) = \lim_{T\to\infty} \left[\prod_{t=1}^{T} w(g_1,g_2,t)^{1/T} \right]$$

$$= \{2\pi(V'+V_{\phi s})\}^{-1/2} \exp\left\{ -\frac{1}{2}\left[\frac{V_{\phi tw}}{V'} + \frac{g_1^2 + V_{\phi tb}}{V' + V_{\phi s}} \right] \right\} \qquad (10)$$

As in the case of within-generation temporal variance, this derivation makes no assumptions about the temporal distribution of ϕ_t between generations.

We are now in a position to evaluate the influence of the various forms of environmental variance on the optimal properties of an allele $(\hat{g}_1,\hat{g}_2)$. It is immediately clear that the environmental optimum that maximizes geometric mean fitness is $\hat{g}_1 = 0$, i.e., the long-term mean environmental state. Estimation of the optimal environmental breadth, $\hat{g}_2$, is less straight-forward because of the complexity of V'. However, the limits to $\hat{g}_2$, can be obtained quite readily by computer by noting from equations (7) and (8) that $[(2\hat{g}_2/3) + V'_{T1}] \leq V' \leq [\hat{g}_2 + V'_{T1}]$ and that from the standpoint of the individual, it is the realised environmental breadth (V') that must be optimized. The minimum value for $\hat{g}_2$, which arises when V'_{T2} is zero, is given in Fig. 3 as a function of $V_{\phi s}$, $V_{\phi tw}$, and $V_{\phi tb}$. The maximum $\hat{g}_2$, which we assume to be approached as $(V'_{T2})^{1/2}/g_2 \to 1$, is approximately 1.5 times the plotted values.

Several conclusions can be drawn from this analysis. First, in accordance with Ayala & Valentine's expectation, temporal variance in the environment always results in selection for more broadly adapted or "functionally flexible" alleles. However, the scale of temporal variance matters a great deal. In an extremely fine-grained environment ($V_{\phi s} = 0$),

$V_{\phi tw}$ and $V_{\phi tb}$ have identical influences on $\hat{g}_2$, but as $V_{\phi s}$ becomes large, the influence of the between-generation variance becomes negligible. The significance of temporal variation between generations is diminished in highly spatially heterogeneous environments because a relatively even distribution of environmental states is already present in different microhabitats, and a shift in $\bar{\phi}_t$ between generations does little to change it.

Second, and in partial agreement with Ayala & Valentine, spatial heterogeneity is not a sufficient condition for the evolution of broadly adapted alleles. In temporally invariant environments, functionally narrow alleles (those with the lowest possible g_2) will have the highest expected fitness. However, provided that the between-generation variance is not too large, spatial heterogeneity in temporally unstable environments accentuates selection for generalism in environments with higher $V_{\phi tw}$. This result is in conflict with the implicit assumption of the Ayala-Valentine hypothesis that spatial heterogeneity plays a diminishing role in molding adaptive genetic properties in temporally variable environments.

Finally, we note that if the between-generation component of temporal variance is much greater than the within-generation component, an inverse relationship may actually arise between $V_{\phi s}$ and $\hat{g}_2$. Such an effect is not predicted by the verbal hypothesis of Ayala & Valentine. It appears to result because a narrowly adapted allele in a spatially homogeneous environment is highly sensitive to environmental changes between generations, whereas the mean fitness of a more broadly adapted allele is relatively constant from generation to generation. In a highly spatially heterogeneous environment a specialist allele would nearly always be located in some favorable microhabitats even in generations with extreme ϕ_t. For example, if ϕ were temperature, then in a year with an extremely high temperature, a microhabitat that is on average excessively cool would have a temperature close the long-term average.

DISCUSSION

The sensitivity of a biochemical pathway to environmental fluctuations most likely evolves in response to two conflicting forces : the necessity of a generalised strategy in a heterogeneous environment, and the cost of evolving generalism (the "jack-of-all-trades is a master-of-none" argument). At the level of biochemical adaptation, environmental heterogeneity includes not only spatial and temporal variance external to the individual (our $V_{\phi s}$, $V_{\phi tw}$, and $V_{\phi tb}$), but also variation in the genetic

environment resulting from a gene's residence in a number of genetic backgrounds (our $\rho_1 V_{G1}$ and $\rho_2 V_{G2}$) and variation in the developmental background induced by the environment (our V_{E1} and V_{E2}). We may, therefore, expect the evolution of biochemical/physiological strategies of organisms to be as much constrained by population genetic phenomena (degree of inbreeding, migration, linkage and chromosomal structure) as by extrinsic environmental factors.

The study of the breadth of biochemical/physiological adaptation from an evolutionary perspective is clearly in an early embryonic state. Indeed, we know of no explicit attempts to test the Ayala-Valentine hypothesis nor of any data sets to which our own theory may be applied. A substantial amount of comparative work has been done on the response of isozymic reactions to changes in the physical/chemical environment, but virtually all of this work has either been done in vitro or in fixed genetic backgrounds (Hochachka & Somero, 1973; Koehn et al., 1983; Watt, 1985). Any attempt to measure $\hat{g}_1$ and reconcile it with an adaptationist argument must realise that the relevant measures of biochemical properties are those obtained in vivo rather than in vitro, a point recently emphasised by several biochemical geneticists (Middleton & Kacser, 1983; Powell & Amato, 1984; Watt, 1985), and that meaningful measures can only be obtained by examining an allele's properties in a full complement of genetic backgrounds rather than in a single artificially constructed background.

An important reason why an in vivo measure may provide an inaccurate description of the in vitro properties of an allele underlying a polygenic trait was pointed out by Lande (1976). The only constraint on a polygenic trait under stabilizing selection is that the aggregate effect of all constituent loci results in a phenotype near the optimum. Subject to this single constraint, the mean effects of constituent loci are free to change in an infinite number of ways via the interaction of drift, mutation, and selection.

In this first attempt at a formal theory for the breadth of biochemical adaptation we have made a number of assumptions regarding shapes of distributions, additivity of allelic effects, an effectively infinite number of possible allelic types, a cost to specialization, and zero covariance between the mean and variance of environmental states within microhabitats. Such assumptions, some of which have been rationalised above, have been necessary in order for us to make progress in the development of a theory that is mechanistic and couched in terms that are measurable in natural

populations. Regardless of these assumptions, however, it is clear that at least seven types of variance in the genetic and environmental background have an influence on the optimal environmental breadth of an allele. These are summarised with their implications for the evolution of the breadth of biochemical adaptation in Table 1.

Perhaps the most striking result of our analysis is the conclusion that not all types of variation in the background of an allele encourage the evolution of broad adaptation ("functional flexibility" or "generalism") for the gene product. If the variance for the environmental optimum (V_{T1}) is high, either because of high V_{E1} or $\rho_1 V_{G1}$, then some individuals containing the allele are likely to be at their optimal environmental state in most generations, and the cost of evolving generalism can be avoided. Moreover, as discussed above, depending on the temporal stability of the environment, spatial variance, caused by structural complexity of the environment and/or

Table 1. Components of variance in the background of an allele and the influence that they have on the evolution of breadth of biochemical adaptation.

Factor		Increased breadth of biochemical adaptation, $\sqrt{g_2}$, is favoured if the factor
$\rho_1 V_{G1}$,	conditional genetic variance for environmental optimum	decreases
V_{E1},	environmental variance for the environmental optimum	decreases
$\rho_2 V_{G2}$,	conditional genetic variance for environmental breadth	increases
V_{E2},	environmental variance for environmental breadth	increases
$V_{\phi s}$,	spatial component of the variance in environmental state as perceived by individuals	depends on $V_{\phi tw}$ and $V_{\phi tb}$
$V_{\phi tw}$,	within-generation component of temporal variance in environmental state as perceived by individuals	increases
$V_{\phi tb}$,	between-generation component of temporal variance in environmental state as perceived by individuals	increases

immobility of individuals, can sometimes select for reduced environmental breadth.

Although some of our conclusions may be quantitatively sensitive to changes in the assumptions underlying our model, our results clearly indicate that the complexity of the relationship between environmental heterogeneity and the evolution of genetic adaptations should not be taken lightly. Although Ayala and Valentine were not explicit in formulating their hypothesis that temporal instability of the environment results in selection for broadly adapted alleles, they appear to adopt $V_{\phi tw}$ as their measure of temporal heterogeneity. Our results indicate that the relation between $V_{\phi tw}$ and $\hat{g}_2$ in different species and/or populations will be highly dependent on the degree to which spatial heterogeneity and between-generation variation in ϕ_t are correlated with $V_{\phi tw}$.

The most appropriate test of our theory for a specific locus would involve the measurement of life-time fitness of many individuals known to contain the allele of interest, but otherwise randomly taken from a natural population. By performing such measures at various points along the environmental gradient and subsequently curve-fitting, estimates of g_1 and $\sqrt{V'}$ can be obtained for the allele. Thus, in the context of evolutionary theory, the measurement of the breadth of biochemical adaptation need not involve any biochemical analysis other than the electrophoresis needed to identify the genotypes of individuals. It does, however, require the analysis of many more individuals than an in vitro biochemical investigation.

Although the further partitioning of the realised environmental breadth, V', into its components ($g_2, \rho_1 V_{G1}, \rho_2 V_{G2}, V_{E1}$, and V_{E2}) would be of interest, it would also require extensive quantitative genetic analysis and for many purposes may be unnecessary. Simple estimates of $\sqrt{V'}$ are relevant to our theory. For while we have focused on the optimization of g_2 in this paper, the evolution of g_2 is actually determined by the more fundamental constraint that $\sqrt{V'}$ be optimized. Since we have scaled our parameters in this paper such that $V'_{T1} = 1$, the optimal values for V' are obtainable from Fig. 3 (for the case $V'_{T2} = 0$) by simply adding 1 to $\hat{g}_2$.

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