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ENVIRONMENTAL TOLERANCE

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The success of a species, its numbers, sometimes its size, etc., are determined largely by the degree of deviation of a single factor (or factors) from the range of optimum of the species. Victor E. Shelford (1913, p. 303)

These words, written by one of the founders of ecology at an early stage in his career, are so deeply embedded in ecological thought that they sound quite trite. Although Shelford's "law of tolerance" is still given significant coverage in some ecology texts (Odum 1971), it has largely been supplanted by the concept of the niche (Whittaker and Levin 1975), which underlies many ecological and evolutionary problems of current interest. Among investigators in evolutionary ecology and ecological genetics, there is much interest in the way in which natural selection interacts with the genome to determine a population's fitness response to different gradients of density-independent and density-dependent factors. In many areas of applied ecology, such as toxicity testing and the development of new crop varieties, a substantial proportion of research focuses on the sensitivity of different genotypes, populations, and/or species to environmental extremes.

In the following, we refer to the response of a genotype's total fitness over an environmental gradient as a tolerance curve. Our definition is a special case of the norm of reaction of Woltereck (1909) and Schmalhausen (1949), which relates the phenotypic expression of a genotype to its environment. Although a genotype-focused definition necessarily introduces some analytic and empirical difficulties, it is an essential starting point in any effort to understand the mechanisms underlying a population-level response to an environmental gradient. The significance of this point was considered first by Van Valen (1965) and later by Roughgarden (1972), who drew a distinction between the within- and between-phenotype components of niche width. The sensitivity of a population to environmental extremes is a function of both the between-individual variance in environmental optima and the within-individual breadth of adaptation.

The focus of this paper is threefold. First, we wish to point out some of the

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proximate causes that interact to shape a genotype's tolerance curve and some of the difficulties in identifying them. Second, we consider how temporal and spatial variation in the environment may influence the evolution of specific properties of the tolerance curve. Finally, we discuss a statistical protocol for the estimation of tolerance-curve parameters. We emphasize at the outset that in order to present some of the fundamental concepts of this paper without being overly technical, we have relied on a number of mathematical assumptions, particularly with respect to the form of certain distribution functions. Even with the simplifications, our results indicate that, from both an ecological and an evolutionary perspective, the complex issue of environmental tolerance is unlikely to be resolved with models that ignore explicit details about the genotype-phenotype-environment interface. Thus, although the concepts that we present are intended to be general, the extent to which our exact mathematical expressions can be carried over to natural populations will be resolved only after considerable empirical work.

In its simplest form, the tolerance curve of a genotype is defined by an environmental optimum and a measure of equitability of fitness over the environmental gradient (hereafter, the breadth of adaptation). Thus, we consider a form of bivariate evolution. At the least, a theory for tolerance-curve evolution requires the explicit incorporation of temporal and/or spatial variation in environmental parameters. Environmental heterogeneity, resulting from external circumstances or from modifying forces generated by the population itself, is ubiquitous and must play a leading role in shaping the breadth of adaptation of a population. In the absence of such variation, genetic variance at the population level and ecological generalism at the individual level is difficult to explain without invoking a balance between selection and mutation (or between selection and migration) (Lande 1976), frequency- or density-dependent selection (Slatkin 1979), or overdominance (Gillespie 1984).

As with all characters, the evolution of environmental tolerance may also depend greatly on certain aspects of population structure such as the mating system and dispersal strategy. Because of the considerable complexities of these issues, however, we have chosen to restrict our initial attention to the issues mentioned in the preceding paragraph. The focus of this study is an asexual population exposed to a single environmental gradient that is independent of population density and phenotypic composition. The confinement of our analysis to asexual populations does not seem overly restrictive when one considers the vast number of unicellular and multicellular organisms that periodically or entirely rely on asexual replication (Bell 1982).

There are additional reasons for choosing an asexual model. First, we wish initially to construct an evolutionary model that has a realistic genetic basis without being overly cumbersome. Environmental optima and breadths of adaptation are based on dozens, perhaps hundreds, of gene loci, and the complexities of linkage and gene interactions that might be involved could make a multiple-locus model extremely tedious. Since the genome of an asexual organism is functionally a single "supergene," such complexities do not enter an asexual model. Second, we demonstrate that a complete analysis of the parameters of the tolerance curve (i.e., the practical application of the theory) is possible only with organisms that can be propagated asexually.

At least three types of existing studies are related to the issue of tolerance-curve evolution. The most notable investigations have focused explicitly on the development of an evolutionary theory for niche width (Levins 1968; Roughgarden 1972; Bulmer 1974; Slatkin and Lande 1976; Felsenstein 1979; Christiansen and Loeschcke 1980; Slatkin 1980; Taper and Case 1985). In addition, one might consider the large number of theoretical studies on the maintenance of polymorphism at single loci in spatially and temporally variable environments (Levene 1953; Haldane and Jayakar 1963; Gillespie and Langley 1974; Karlin and Lieberman 1974; Gillespie 1976, 1978; Templeton and Rothman 1978, 1981; Matsuda and Gojobori 1979; Maynard Smith and Hoekstra 1980; Takahata 1981; and references therein). Finally, the literature on plant and animal breeding is replete with empirical and methodological studies on genotype-environment covariance (Bulmer 1980; Falconer 1981; Via and Lande 1985).

The niche-width models are most closely related to a theory for the evolution of environmental tolerance. Although theoretical studies on the evolution of niche width have probably provided a great deal more insight into this complex problem than would ever have emerged with purely verbal arguments, their primary emphasis has been on the distribution of specified phenotypes under different selective regimens. None of them has explicitly considered the proximate or ultimate causes of the tolerance curve itself, although most could be extended to such an analysis. Many of these studies (most notably Roughgarden 1972; Slatkin 1980; Taper and Case 1985) have focused on the extremely complicated case of density-dependent gradients and, of necessity, rely on numerous assumptions about rates of increase, carrying capacities, and competition coefficients for resources and consumers.

Two additional limitations of current niche theory may be pointed out. First, with the exception of Slatkin and Lande (1976), the theory of the evolution of niche width has considered only deterministic environments. Although spatial variation can legitimately be ignored under some population structures (Slatkin and Lande 1976; Felsenstein 1979), in general, this is not true for temporal heterogeneity (Slatkin and Lande 1976). Second, except in the study of Taper and Case (1985), the within-individual component of niche width has been assumed completely invariant. Under this restriction, variation in breadth of adaptation is purely a population phenomenon resulting from individual variation in environmental optima. Even though such an assumption is often required for mathematical tractability, and although it may be true that the intensity of selection on the environmental optimum may be greater than that on the breadth of adaptation, there seems to be little biological justification for assuming that a character as complex as breadth of adaptation will be immune to environmental and genetic effects.

The theory outlined below eliminates some of these problems. It also indicates, however, that Shelford may not have greatly overstated the case when he wrote, "The results of these five years of labor will not be pleasing to many zoologists because the principles of evolution, heredity, etc., have not been correlated. Their omission, however, has not been due to any prejudice against their introduction, but rather to the fact that they can only occasionally be related to this line of organization." (1913, p. vi.)

DETERMINANTS OF A GENOTYPE'S TOLERANCE CURVE

We represent the tolerance curve for individuals of phenotype \mathbf{z} over a single environmental gradient as $w(\mathbf{z}|\phi)$, where w represents fitness and ϕ is a continuously distributed environmental variable. Since we treat ϕ as independent of the density and frequency of phenotypes, the following theory is most relevant for physical gradients and some chemical ones (e.g., temperature, light intensity, moisture content, pH). It may also apply to essential nutrients that become toxic at high concentrations. Specifically, we rely on the bivariate phenotypic function

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

For this model, $w(z_1, z_2|\phi)$ is a Gaussian function with environmental optimum z_1 and "variance" z_2 . We adopt $(z_2)^{1/2}$ as a measure of a phenotype's breadth of adaptation, that is, of the sensitivity of a phenotype's fitness to environmental change. Note that as $(z_2)^{1/2}$ increases, the function $w(z_1, z_2|\phi)$ becomes flatter; that is, fitness is less influenced by a change in the environmental state, ϕ .

Our reliance on the Gaussian function derives from the common observation for density-independent gradients (usually at the population level) that extreme conditions are lethal or nearly so and that fitness is maximized at a single intermediate point on the gradient (Odum 1971; Ricklefs 1973). If these conditions are met, an individual tolerance curve that is not normal on the direct scale of measurement can almost always be rendered approximately normal by an appropriate scale transformation (Wright 1968). However, radical differences in the mathematical form of the tolerance curve between members of the same population would greatly compromise the generality of the following theory. In the case of radical differences among population members, a transformation to normality that was successful for one individual would be counterproductive for others. Note also that the Gaussian function has the property

$$\int_{-\infty}^{+\infty} w(z_1, z_2|\phi) d\phi = 1; \quad (2)$$

thus, any increase in the breadth of adaptation will be accompanied by a decline in fitness in the optimal environment. We therefore assume a "jack-of-all-trades is a master of none" conflict.

Before proceeding, let us compare equation (1) with the Gaussian fitness function that is frequently used in models of stabilizing selection of polygenic traits (Robertson 1956; Latter 1970; O'Donald 1970; Slatkin 1970, 1979; Lande 1976; Feldman and Cavalli-Sforza 1979; Kirkpatrick 1982; Lynch and Gabriel 1983; and references above). In these models ϕ , z_1 , and $(z_2)^{1/2}$ are measured on the phenotypic rather than the environmental scale. The optimal phenotype is then ϕ , and $(z_2)^{1/2}$ is inversely related to the intensity of stabilizing selection. Both ϕ and $(z_2)^{1/2}$ are assumed to be fixed by the environmental setting and equal for all individuals (for one exception to this treatment of $(z_2)^{1/2}$, see Taper and Case 1985). Under these circumstances, only the distribution of z_1 evolves.

Thus, our use of equation (1) to define the tolerance curve reflects a fundamental departure from the traditional interpretation of Gaussian fitness functions in

two ways: first, all parameters of our model are on environmental rather than phenotypic scales; and second, individuals vary in both z_1 and z_2 . The usual treatment of z_2 as a constant appears to be so well accepted that it is almost never explicitly stated as an assumption in stabilizing-selection models. In our model, however, absolute constancy of z_2 would imply that genetic variance for tolerance does not exist. This is certainly not true (Parsons 1983), and such an interpretation would obviously impose serious constraints on the analysis of problems related to the evolution of the breadth of adaptation. The phenotypes z_1 and z_2 are generally functions of many biochemical, physiological, and morphological attributes. Unless all the genes underlying these attributes are fixed, which seems unlikely, z_1 and z_2 are heritable traits subject to natural selection.

For the simple case in which the optimal environmental state of an individual is perfectly correlated with the phenotypic value of a single character, our use of an environmental instead of a phenotypic measure would be equivalent to a scale transformation. In most cases, however, the optimal environmental state depends on many characters, and any attempt to relate total fitness to a single trait might result in considerable inaccuracy (Lande and Arnold 1983). Direct use of the environmental gradient avoids this problem, since $w(z_1, z_2 | \phi)$ is a composite measure of fitness that integrates all properties of the phenotype. The cost of this approach is that it obviates the possibility of predicting the evolutionary dynamics of the individual characters defining fitness. This is a general problem of all models of phenotypic evolution. For example, a selection theory for height is not necessarily a theory for the length of individual parts.

Although we consider only a gradient of a single, independent environmental variable, we are not denying the importance of covariance between environmental variables. Just as the response of single characters to selection may be severely constrained by correlations between other selected characters resulting from pleiotropy, the evolution of a tolerance curve along one environmental gradient depends on the joint distribution of different environmental properties (such as temperature and humidity) to which individuals are exposed. For now, however, many of our general points can be made without recourse to multivariate modeling and matrix notation. In order to account for multiple environmental factors in future work, a multivariate form of equation (1) would need to be adopted, in which case z_1 and ϕ would become vectors and z_2 would become a "covariance" matrix.

Developmental Noise

For a single environmental gradient, clonal fitness is a function of two genotypic properties: g_1 , the optimal environmental state, and g_2 , the genetic contribution to the variance of the tolerance curve. However, through random noise in developmental pathways, maternal effects, and/or physiological acclimation, an individual's optimal environmental state and/or its breadth of adaptation may differ from the expectations g_1 and g_2 . Thus, the phenotype (z_1, z_2) of individual x of the clone may be written as $z_1(x) = g_1 + e_1(x)$ and $z_2(x) = g_2 + e_2(x)$, where the $e(x)$ represent deviations. By definition, the expected values of $e_1(x)$ and $e_2(x)$ are equal to zero. Quantitative geneticists generally refer to e_1 and e_2 as environmental

deviations. In order to avoid semantic difficulties, however, we refer to them as developmental noise. We rely on the usual assumption that the distribution of e_1 is independent of g_1 . Since $z_2 > 0$ by definition, however, the lower limit of e_2 is $-g_2$, and e_2 and g_2 cannot be strictly independent.

The tolerance curve for genotype (g_1, g_2) is

$$w(g_1, g_2 | \phi) = \int_0^{+\infty} \int_{-\infty}^{+\infty} w(z_1, z_2 | \phi) p(z_1, z_2 | g_1, g_2) dz_1 dz_2, \quad (3)$$

where the double integration is over the domain of possible (z_1, z_2) , and p denotes frequency. We assume that the conditional probability distributions for z_1 and z_2 are independent, such that

$$p(z_1, z_2 | g_1, g_2) = p(z_1 | g_1) p(z_2 | g_2). \quad (4)$$

The developmental variances for the two conditional distributions are V_{E1} and V_{E2} , respectively. We further assume that the joint action of many environmental effects on the environmental optimum results in a normal conditional distribution:

$$p(z_1 | g_1) = (2\pi V_{E1})^{-1/2} \exp[-(z_1 - g_1)^2 / 2V_{E1}]. \quad (5)$$

Although $p(z_2 | g_2)$ is also likely to be influenced by a number of different environmental factors, it cannot be strictly normal because a variance cannot be less than zero. We assume that no individuals are so narrowly adapted that $z_2 = 0$, which implies $p(0 | g_2) = 0$, and that $p(z_2 | g_2)$ has a single peak. A number of distribution functions satisfy these conditions (Lynch and Gabriel 1986). Those that we have focused upon share several qualitative properties: the mode is less than the mean of z_2 ; as V_{E2} approaches zero, $p(z_2 | g_2)$ approaches a normal distribution with mean of approximately g_2 and variance of approximately V_{E2} ; and as V_{E2} approaches infinity, $p(z_2 | g_2)$ becomes increasingly L-shaped. We rely on a function that most easily yields an analytic solution, a beta distribution of the second kind (Kendall and Stuart 1977, p. 163). In the Appendix we derive a special form of this function (eqs. A2, A3) that satisfies the conditions of expectation $E(z_2) = g_2$ and variance $\text{var}(z_2) = V_{E2}$.

The genotypic tolerance curve is obtained by substituting equations (1), (4), (5), (A2), and (A3) into equation (3) and integrating. The complete solution is given in the Appendix, where we also show that the genotypic tolerance curve is closely approximated by

$$w(g_1, g_2 | \phi) = (2\pi V)^{-1/2} \exp[-(g_1 - \phi)^2 / 2V] \quad (6)$$

with $V = V_{E1}(\alpha + \beta) / \beta$, $\alpha = g_2[g_2(g_2 + V_{E1}) + V_{E2}] / V_{E1}V_{E2}$, and $\beta = [g_2(g_2 + V_{E1}) / V_{E2}] + 2$, provided that α is greater than 5. Thus, for these conditions, $w(g_1, g_2 | \phi)$ is approximately normal with optimal environment g_1 and breadth of adaptation $V^{1/2}$. The necessary conditions for this normal approximation may not be very restrictive. If we adopt a scale such that $V_{E1} = 1$, as we do in the following analyses, then the criterion that α is greater than 5 will be met if g_2 is greater than approximately 1.0 and the coefficient of variation, $V^{1/2}_{E2} / g_2$, is less than approximately 1.0. Coefficients of variation in excess of 1.0 are extremely unusual for polygenic traits (Falconer 1981), and our following results suggest that a g_2 less than 1.0 is unlikely to persist in any environment exhibiting variability.

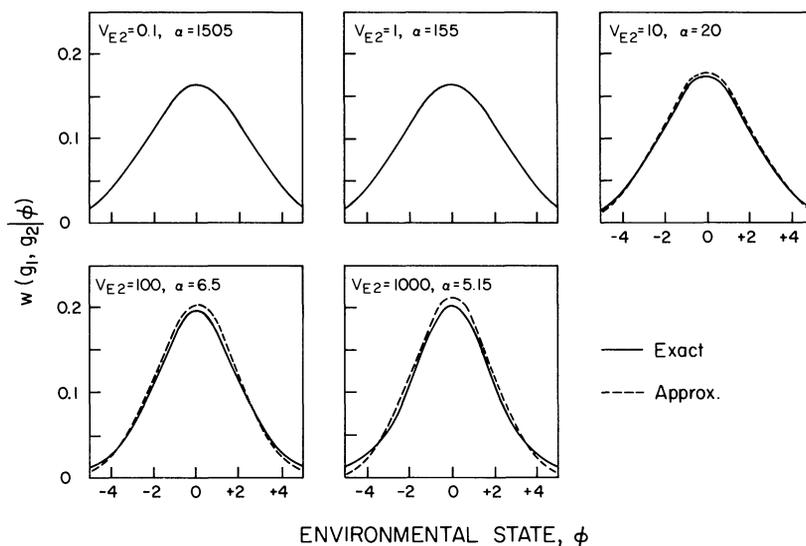


FIG. 1.—The influence of V_{E2} on the genotypic tolerance curve and the degree of correspondence between the “exact” and approximate solutions for $w(g_1, g_2|\phi)$, equations (A4) and (A7). The “exact” solutions were obtained by using a five-term Sterling’s approximation (Abramowitz and Stegun 1972, eq. 6.1.37) for the gamma functions and expanding the confluent hypergeometric function to 40 terms. The two solutions are indistinguishable on a graph for the cases of $V_{E2} = 0.1$ and 1.0 . In all cases, $g_1 = 0$, $g_2 = 5$, and $V_{E1} = 1$.

Equation 6 shows that the two types of developmental noise have opposite effects on $w(g_1, g_2|\phi)$. Although variance in the environmental optimum (V_{E1}) results in a broadening of the genotypic tolerance curve, V_{E2} causes it to narrow. The latter effect is not a peculiar property of the function for $p(z_2|g_2)$ that we have adopted in this paper (Lynch and Gabriel 1986). In the limiting case in which $V_{E2} = 0$, equation (6) applies exactly and the realized breadth of adaptation is $V^{1/2} = (g_2 + V_{E1})^{1/2}$. At the limit of applicability of equation (6) ($g_2 \geq 1$ and $V^{1/2}_{E2}/g_2 = 1.0$), $V^{1/2} \approx [(2g_2/3) + V_{E1}]^{1/2}$. For still larger V_{E2} , the exact solution of equation (3) shows that the genotypic tolerance curve becomes significantly leptokurtic, that is, narrower and more peaked near $\phi = g_1$ with elevated fitness in environments with extreme ϕ (fig. 1). Thus, depending on the relative levels of V_{E1} and V_{E2} , the equitability of a clone’s fitness over environmental states, that is, its realized breadth of adaptation, may be magnified or reduced relative to expectations based on its actual genetic attribute g_2 .

Spatial and Temporal Heterogeneity within a Generation

We next consider the role that some general selective forces may play in the evolution of tolerance curves. We will not consider selection-mutation balance or other equilibrium conditions for the maintenance of genetic variance for g_1 and g_2 . Our concern will simply be to identify the tolerance curve that maximizes the mean fitness of a clone exposed to spatial and temporal environmental variation.

In a spatially and temporally uniform setting with environmental state ϕ , equa-

tion (6) would define the fitness of genotype (g_1, g_2) . The optimal genotype under such circumstances would clearly be the extreme specialist with optimal environmental state $g_1 = \phi$ and with the minimum possible g_2 . Since such environments are biologically unrealistic, however, it is essential to determine how spatial and temporal variation in ϕ may influence the outcome of clonal selection operating on the tolerance curve. Let us first evaluate the consequences of within-generation spatial and temporal variance.

For a population growing in discrete generations, a mean environmental state, ϕ_t , may be identified for each generation t . If the environment is spatially complex, then it is likely that the mean environmental state experienced by each individual (ϕ_s) will deviate somewhat from ϕ_t . We assume that ϕ_s is normally distributed about ϕ_t with variance V_{ϕ_s} so that

$$p(\phi_s|\phi_t) = (2\pi V_{\phi_s})^{-1/2} \exp[-(\phi_s - \phi_t)^2/2V_{\phi_s}]. \quad (7)$$

We emphasize that V_{ϕ_s} is a measure of spatial heterogeneity perceived by a population, not an intrinsic property of the environment. It is likely that V_{ϕ_s} will be higher for a sedentary species than for a mobile species and that habitat selection will result in a further reduction in V_{ϕ_s} .

We incorporate within-generation temporal variance by allowing the environmental state experienced by each individual to be temporally distributed about ϕ_s with variance $V_{\phi_{tw}}$. An implicit assumption in such a treatment is that all microhabitats experience the same level of temporal heterogeneity. The expected fitness of genotype (g_1, g_2) in generation t is then the arithmetic mean of the ϕ_s -dependent fitnesses weighted by the probability of the occurrence of ϕ_s . We assume that temporal variation in ϕ influences the fitness of a clone geometrically (as when daily probabilities of survival interact multiplicatively to give annual survival),

$$w(g_1, g_2|\phi_t) = \int_{-\infty}^{+\infty} p(\phi_s|\phi_t) \left\{ \left[\prod_{i=1}^{\tau} w(g_1, g_2|\phi_{si}) \right]^{1/\tau} \right\} d\phi_s, \quad (8)$$

where τ is the number of discrete time steps of equal length per generation. The $w(g_1, g_2|\phi_{si})$ may be thought of as the multiplicative components of fitness in the life cycle. The item in braces, which is the lifetime fitness of genotype (g_1, g_2) in a habitat of mean state ϕ_s in generation t , may be abbreviated as $w(g_1, g_2|\phi_s)_t$. From equation (6),

$$w(g_1, g_2|\phi_s)_t = (2\pi V)^{-1/2} \exp\left[-\sum_{i=1}^{\tau} (g_1^2 - 2g_1\phi_{si} + \phi_{si}^2)/2\tau V\right],$$

where ϕ_{si} represents the environmental state experienced at time i . Assuming large τ and noting that $E(\phi_{si}^2) = \phi_s^2 + V_{\phi_{tw}}$, this further reduces to

$$w(g_1, g_2|\phi_s)_t = (2\pi V)^{-1/2} \exp\{-[(g_1 - \phi_s)^2 + V_{\phi_{tw}}]/2V\},$$

which upon substitution in equation (8) yields

$$w(g_1, g_2|\phi_t) = [2\pi(V + V_{\phi_s})]^{-1/2} \exp\left\{-\frac{1}{2} [(V_{\phi_{tw}}/V) + (g_1 - \phi_t)^2/(V + V_{\phi_s})]\right\}. \quad (9)$$

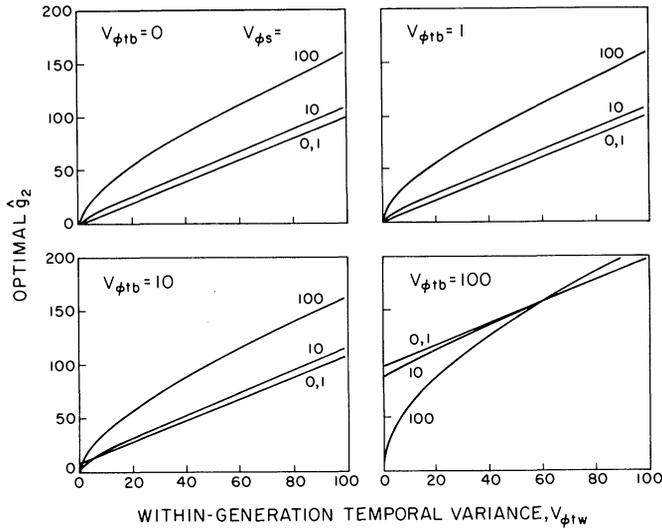


FIG. 2.—The optimal genetic parameter \hat{g}_2 for the case in which $V_{E2} = 0$, $g_1 = \phi_t$, and $V_{E1} = 1$. Since $\hat{g}_2 = \hat{V} - 1$ in this case, the optimal value of \hat{V} for any combination of $V_{\phi s}$, $V_{\phi tw}$, and $V_{\phi tb}$ can be taken from these graphs as $\hat{g}_2 + 1$. The influence of V_{E2} on \hat{g}_2 is obtainable from the definition of V in the text by back substitution.

This is the expected fitness of a clone with properties (g_1, g_2) in generation t . Note that in the derivation of equation (9) no assumptions were made about the temporal autocorrelation of fitness variation.

Equation (9) indicates that spatial heterogeneity alone is insufficient to discourage the evolution of specialization in an asexual population. In the absence of temporal variance in ϕ , the most-fit clones in each generation will be those with the minimum values for $|g_1 - \phi_t|$ and g_2 . Nevertheless, since $V_{\phi s}$ contributes to the breadth of $w(g_1, g_2|\phi_t)$, spatial heterogeneity can impede the rate at which the most specialized clones come to dominate by reducing the fitness differential between clones. However, within-generation temporal variation in ϕ encourages the evolution of generalism. The optimal realized breadth of adaptation is obtained by taking the derivative $dw(g_1, g_2|\phi_t)/dV$, and setting it and $(g_1 - \phi_t)$ equal to zero,

$$\hat{V}^{1/2} = \{1/2 V_{\phi tw} + 1/2 [V_{\phi tw} (V_{\phi tw} + 4V_{\phi s})]^{1/2}\}^{1/2}. \tag{10}$$

Some degree of generalization at the genetic level ($\hat{g}_2 > 0$) will be favored provided that $V_{\phi tw} > V_{E1}^2 / (V_{\phi s} + V_{E1})$. This follows from setting $\hat{V} = \hat{g}_2 + V_{E1}$ or $= 2\hat{g}_2/3 + V_{E1}$, the limits to the applicability of the approximation (9), and solving for the conditions for $\hat{g}_2 > 0$. Since V_{E1} contributes positively to \hat{V} , developmental noise for a clone's optimal environment causes a decrease in \hat{g}_2 . This result arises because a large V_{E1} allows the members of a clone to exploit a diversity of environments without the cost of evolving generalism.

Figure 2 (upper left panel) illustrates the dependence of \hat{g}_2 on $V_{\phi s}$ and $V_{\phi tw}$ for the special case in which $V_{E2} = 0$, with the scale set so that $V_{E1} = 1$. Since V declines from $g_2 + V_{E1}$ to approximately $2g_2/3 + V_{E1}$ as $V^{1/2}_{E2}/g_2$ increases from 0

to 1, the effect of V_{E2} within this range will be to increase \hat{g}_2 by a factor of 50% at the most. Thus, provided that $V^{1/2}_{E2}/g_2$ is within the range commonly observed for metric characters (0.1–0.5; Falconer 1981) and that V_{ϕ_s} is less than approximately $10V_{E1}$, then \hat{g}_2 is approximately equal to $V_{\phi_{tw}}$; that is, the optimal breadth of adaptation is expected to increase linearly with the square root of the temporal variance of ϕ . Only when V_{ϕ_s} is greater than $10V_{E1}$ does spatial heterogeneity promote a more discernible increase in \hat{g}_2 . The interactive effect of spatial and temporal heterogeneity on \hat{g}_2 arises because of the nonlinear nature of the Gaussian tolerance curve. Whereas $V_{\phi_{tw}}$ results in a net reduction in fitness for individuals in environments with ϕ_s near g_1 , in more extreme environments $w(g_1, g_2|\phi_t)$ is concave upward and temporal fluctuations in ϕ can actually enhance an individual's fitness.

LONG-TERM GEOMETRIC-MEAN FITNESS

Finally, we incorporate the between-generation component of temporal variance in the environmental state by letting ϕ_t be distributed with variance $V_{\phi_{tb}}$ and scaled such that the long-term mean environmental state (ϕ_t) is zero. Dempster (1955) showed that in asexual populations, in the absence of the opposing forces of mutation, migration, and drift, the clone with the maximum geometric-mean fitness always approaches fixation asymptotically. Thus, the appropriate fitness measure is now the expected geometric-mean fitness of genotype (g_1, g_2),

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

Substituting equation (9) and taking expectations, we find

$$w(g_1, g_2) = [2\pi(V + V_{\phi_s})]^{-1/2} \exp\left\{ -\frac{1}{2} [V_{\phi_{tw}}/V + (g_1^2 + V_{\phi_{tb}})/(V + V_{\phi_s})] \right\}. \quad (11)$$

In the derivation of equation (11) we have assumed that V_{ϕ_s} and $V_{\phi_{tw}}$ are constant between generations, but again we have made no assumptions about the temporal pattern of ϕ_t . A simple analytical solution of equation (11) for \hat{g}_2 cannot be obtained. It can be shown by differentiation, however, that between-generation temporal variance in ϕ always elevates \hat{g}_2 above that expected for environments with constant ϕ_t . Moreover, it can be seen from equation (11) that $V_{\phi_{tb}}$ and $V_{\phi_{tw}}$ have identical influences on genotypic fitness, and hence on \hat{g}_2 , in spatially homogeneous environments ($V_{\phi_s} = 0$). The significance of $V_{\phi_{tb}}$ is diminished in spatially heterogeneous environments because a relatively uniform distribution of possible environmental states is already present and a change in the mean environmental state between generations does little to change the distribution.

Figure 2 shows that because increasing $V_{\phi_{tb}}$ causes \hat{g}_2 to increase and because the effect is magnified in spatially homogeneous environments, an inverse relationship can sometimes arise between V_{ϕ_s} and \hat{g}_2 if $V_{\phi_{tw}}$ is less than approximately $V_{\phi_{tb}}/3$. This result arises in environments that are spatially and temporally uniform

within generations because most members of a clone suffer equally in extreme generations and persistence can only be accomplished by a generalist strategy. In a more spatially complex setting, even for generations in which $(g_1 - \phi_t)$ is extreme, there are always some spatial refugia and the cost of evolving generalism can be partially avoided.

METHODOLOGICAL CONSIDERATIONS

We now turn our attention from theoretical matters to some of the practical problems that must be considered when applying tolerance-curve theory to natural populations. Parameters of the tolerance curve are qualitatively different from most quantitative traits studied by evolutionary biologists in that the former can never be estimated with single individuals. Since the fitness of an organism can only be measured once, determining the width and location of the peak of the genotypic tolerance curve requires genotypes that can be replicated exactly and grown in different environments. Thus, the implementation of our theory in a genetic analysis is possible only with organisms that can be propagated by asexual means. This does not rule out the analysis of sexual populations, however, since many sexual organisms can also reproduce vegetatively.

In principle, the distribution of a genotype's fitness over a continuous environmental gradient can be determined in the field by monitoring individuals and their respective environmental states. This, of course, requires that members of individual genotypes can be identified with certainty. Alternatively, members of a known genotype can be grown in a number of discrete environmental states in the laboratory, glasshouse, or experimental plots. Special precautions are always necessary in a field setting, where the states of different environmental gradients may not be independent of each other, in which case recourse to a multivariate analysis might be necessary.

Given that the appropriate data are obtainable, the problem is to translate the individual estimates of fitness and environmental state into the parameters g_1 , g_2 , V_{E1} , and V_{E2} . Provided that the developmental deviations, e_1 , are symmetrically distributed around g_1 , then g_1 simply equals the location of the peak of the fitness function. As noted above, however, the "variance" of the distribution of fitness on ϕ is not an estimator of g_2 , but a complicated function of g_2 , V_{E1} , and V_{E2} . Nevertheless, when certain assumptions are met, statistical models can be derived that allow the resolution of the moments of the genotypic tolerance curve into estimates of g_2 , V_{E1} , and V_{E2} .

We offer the following analytical techniques for use when the assumptions of the models presented in the preceding section are met. That is, we assume that ϕ is measured on a scale such that $w(z_1, z_2 | \phi)$ is normal and that the developmental deviations e_1 and e_2 are approximately normally and independently distributed. These conditions will not have been met if the observed tolerance curve is asymmetrical, that is, if any of the odd moments of $w(g_1, g_2 | \phi)$ are significant, or if $w(g_1, g_2 | \phi)$ is significantly leptokurtic. Our assumptions of normality can be relaxed, and alternative models derived, but there is little point in pursuing this matter until the relevant data have been obtained. Our primary point is to demon-

strate that since the estimates of g_1 , g_2 , V_{E1} , and V_{E2} are obtainable in principle, the theoretical expectations generated above are testable.

The estimation procedure is quite routine for determining a genotype's environmental optimum (g_1) and realized breadth of adaptation ($V^{1/2}$) without regard to the underlying mechanisms. We have noted above that when the conditional phenotype distributions, $p(z_1|g_1)$ and $p(z_2|g_2)$, are approximately normal, the expected genotypic tolerance curve will also be normal. The first two moments (m_1 and m_2) of this fitness distribution over ϕ provide the desired information:

$$g_1 \approx m_1 = \frac{\sum_{i=1}^n w_i \phi_i}{\sum_{i=1}^n w_i}, \quad (12a)$$

$$V \approx m_2 = \frac{\sum_{i=1}^n w_i (\phi_i - m_1)^2}{\sum_{i=1}^n w_i}, \quad (12b)$$

where w_i and ϕ_i are the fitness and environmental-state measures for the i th individual, and a total of n individuals are evaluated. These equations apply when individuals are evenly sampled from all environments over the gradient. When such a protocol cannot be followed, ϕ_i should be the i th environmental state and w_i the mean fitness of individuals in that environment. Following Kendall and Stuart (1977, p. 245), the sampling variances for g_1 and V are approximately $\text{var}(g_1) = m_2/n$ and $\text{var}(V) = (m_4 - m_2^2)/n$, where

$$m_4 = \frac{\sum_{i=1}^n w_i (\phi_i - m_1)^4}{\sum_{i=1}^n w_i}.$$

In many situations, such as toxicity testing, $V^{1/2}$ provides adequate information on the breadth of adaptation. When gathered in conjunction with estimates of V_{ϕ_s} , $V_{\phi_{tw}}$, and $V_{\phi_{tb}}$, estimates of $V^{1/2}$ from different genotypes are also adequate to test much of the theory that we have developed above, because the genotypic property g_2 is expected to evolve subject to the constraint that V is optimized.

However, in order to test our idea that V_{E1} and V_{E2} influence the optimal g_2 , a more discriminating procedure is required to separate the effects of g_2 , V_{E1} , and V_{E2} on the breadth of adaptation. The basis for such an analysis is the influence that V_{E1} and V_{E2} have on the shape of the sampling distribution around the expected genotypic tolerance curve (fig. 3). In some cases, the patterns may be striking enough that a prognosis of the relative importance of V_{E1} and V_{E2} can be reached by examination of the distribution of (w_i, ϕ_i) in a scatter plot. When the variance in z_1 predominates (fig. 3c), a flat and narrow distribution of points appears around the environmental optimum. When the variance in z_2 predominates (fig. 3b), a narrow band of points appears near $g_1 \pm (g_2)^{1/2}$.

We have attempted to exploit this information in a number of ways to generate estimates of the four parameters (g_1 , g_2 , V_{E1} , V_{E2}) and have settled on the maximum-likelihood method as the most expedient approach. Suppose measures of fitness (w_i) and environmental state (ϕ_i) have been obtained for n individuals. The strategy of the maximum-likelihood procedure is to estimate the values of g_1 , g_2 , V_{E1} , and V_{E2} that maximize the joint probability of all of the observations of

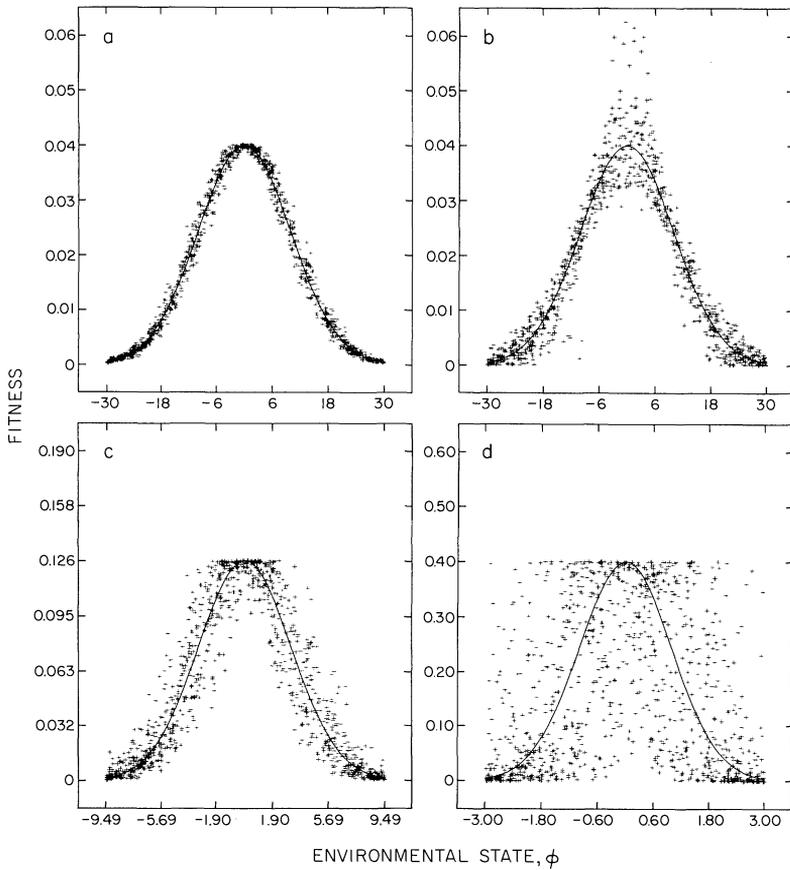


FIG. 3.—Examples of the expected sampling distribution of fitness over an environmental gradient. In each case, 1000 phenotypes were randomly drawn according to equations (5) and (A2) and randomly assigned an environmental state ϕ , yielding a fitness estimate defined by equation (1). In all cases, $g_1 = 0$ and $V_{E1} = 1$. *a*, $g_2 = 100$, $V_{E2} = 1$; *b*, $g_2 = 1000$, $V_{E2} = 1000$; *c*, $g_2 = 10$, $V_{E2} = 0$; *d*, $g_2 = 1$, $V_{E2} = 0$. *Solid line*, The expected fitness function for the deterministic situation in which $V_{E1} = V_{E2} = 0$.

(w_i, ϕ_i) . Letting p_i be the a posteriori probability of the observation (w_i, ϕ_i) conditional on estimates of g_1 , g_2 , V_{E1} , and V_{E2} , methods of numerical mathematics can be used to obtain the solution that maximizes $\prod_{i=1}^n p_i$ provided that a function for p_i is available.

There are several advantages to such an estimation procedure. It not only generates a joint solution for the four unknown parameters, but also yields the sampling variances. Measures of fitness over the entire range of ϕ are not essential for the procurement of accurate parameter estimations. The model can be used to analyze data sets in which multiple estimates of w_i are available in a few environments with fixed ϕ or data sets in which ϕ_i is essentially different for all individuals. Although maximum-likelihood estimation does require considerable compu-

TABLE 1

A COMPARISON OF MAXIMUM-LIKELIHOOD ESTIMATES OF THE TOLERANCE-CURVE PARAMETERS g_2 , V_{E1} , AND V_{E2} WITH KNOWN VALUES

	g_2	V_{E1}	V_{E2}	g_2	V_{E1}	V_{E2}
True value	4.000	1.000	1.600	1.000	0.250	0.100
Maximum-likelihood estimates	{ 3.815 4.505 4.700	{ 1.214 0.836 0.921	{ 0.808 1.515 1.568	{ 0.954 1.115 1.169	{ 0.304 0.277 0.226	{ 0.050 0.107 0.094
True value	10.000	1.000	1.000	5.000	1.000	3.000
Maximum-likelihood estimates	{ 9.964 10.592 10.306	{ 1.224 0.784 1.052	{ 0.822 1.202 1.088	{ 5.744 5.502 5.198	{ 1.099 1.087 0.652	{ 3.217 3.298 0.853
True value	1.000	0.250	0.010			
Maximum-likelihood estimates	{ 0.984 0.985 0.926	{ 0.186 0.264 0.228	{ 0.014 0.020 0.005			

NOTE.—It is assumed in these examples that accurate estimates of g_1 , which equals zero, are available before the analysis. In each example, random values of w_i and ϕ_i were obtained for 40 individuals in each of five environmental states, subject to the constraint that z_1 and z_2 are normally distributed (a close approximation in the case of z_2). For each set of known parameter values, three separate analyses of 200 different individuals were performed.

tational time, this is no longer a serious problem for laboratories possessing a microcomputer.

A derivation of the function p_i for the special case in which an individual experiences a constant level of ϕ throughout its life and α is less than 5 is presented by Gabriel (1986). Such a function is most relevant for situations in which individuals are grown in a controlled environment at fixed ϕ , as is often done in laboratory toxicity tests or in glasshouse gradient experiments. (In principle, using the equations we presented in the preceding section, p_i can also be derived for situations in which $V_{\phi tw} > 0$.) We tested the maximum-likelihood model by randomly drawing individuals of phenotype (z_1, z_2) according to the distributions $p(z_1|g_1)$ and $p(z_2|g_2)$ and assigning them an environmental state ϕ_i and fitness w_i defined by equation (1). The maximum-likelihood estimates of g_1 , g_2 , V_{E1} , and V_{E2} were then compared with the known parameters of $p(z_1|g_1)$ and $p(z_2|g_2)$. Table 1 illustrates the accuracy of this estimation procedure. With a total sample size of 200 individuals assorted among 5 environmental states, the maximum-likelihood estimates are generally quite close to the known values. (The algorithm for this maximum-likelihood procedure may be obtained from W. Gabriel.)

Ultimately, for a complete analysis of the tolerance curve from the standpoint of a population, it is useful to know not only the levels of V_{E1} and V_{E2} , but also the amount of genetic variance for the two tolerance-curve parameters, V_{G1} and V_{G2} . This is clearly necessary in order to evaluate the sensitivity of a population to environmental extremes. Such an analysis is also required for a determination of the potential evolutionary response of a population's mean optimal environmental state and the breadth of adaptation caused, for example, by selective challenges resulting from changes in $\bar{\phi}$, $V_{\phi s}$, $V_{\phi tw}$, and/or $V_{\phi tb}$.

In principle, the population parameters \bar{g}_1 , \bar{g}_2 , $V_{T1} = V_{G1} + V_{E1}$, and $V_{T2} = V_{G2} + V_{E2}$ can be derived by the same procedures outlined above. It is not a small task, however. Once mean estimates have been obtained for V_{E1} and V_{E2} by analyzing several unique genotypes, a population of mixed genotypes may be evaluated over the environmental gradient. The environmental optimum for such a mixed population is \bar{g}_1 . Provided that genotype-environment covariance is negligible and that the joint influence of genetic and environmental effects on z_1 and z_2 results in distributions of the form of equations (5) and (6), the maximum-likelihood method can be used, the estimated parameters now being \bar{g}_2 , V_{T1} , and V_{T2} instead of g_2 , V_{E1} , and V_{E2} . By subtraction of the independently derived estimates of V_{E1} and V_{E2} from V_{T1} and V_{T2} , the genetic variances and hence the broad-sense heritabilities (total genetic variance divided by phenotypic variance) of the tolerance-curve parameters may be obtained. Such an analysis requires that the genotypes be randomly distributed among environments, but again it is not necessary to investigate the entire range of ϕ . Of course, if one is simply interested in the population properties \bar{g}_1 , \bar{g}_2 , V_{T1} , and V_{T2} , then it is unnecessary to perform a complete analysis on any individual genotypes (since estimates of V_{E1} and V_{E2} are not required).

DISCUSSION

We have shown that in the context of an evolutionary analysis of tolerance curves at least five kinds of environmental variance (V_{E1} , V_{E2} , V_{ϕ_s} , $V_{\phi_{tw}}$, and $V_{\phi_{tb}}$) must be considered. Whereas any kind of temporal variance in the environment selects for more-broadly-adapted genotypes, temporal variance within generations ($V_{\phi_{tw}}$) plays a more important role than that between generations ($V_{\phi_{tb}}$), which becomes of negligible importance when the spatial component of variance is high. Spatial heterogeneity (V_{ϕ_s}) caused by structural complexity or immobility of individuals also often selects for more-broadly-adapted genotypes, but only when it operates in conjunction with temporal variance. Moreover, when $V_{\phi_{tw}}$ is much less than $V_{\phi_{tb}}$, spatial heterogeneity can actually select for a higher degree of specialization.

In a more general way, V_{ϕ_s} and $V_{\phi_{tw}}$ can be considered the total variance in additive and multiplicative effects on fitness resulting from environmental heterogeneity. (This is how they were treated in the preceding derivations.) The use of V_{ϕ_s} and $V_{\phi_{tw}}$ also formalizes Levins' (1968) concept of environmental grain. In a fine-grained environment, an individual passes through many "patches" in its lifetime. This implies relatively low V_{ϕ_s} , since most individuals will experience most patch types during their lives, and relatively high $V_{\phi_{tw}}$ resulting from the movement between different patches. In the most coarse-grained of environments, individuals spend their entire lives in single patches, thereby maximizing V_{ϕ_s} but reducing $V_{\phi_{tw}}$ to the temporal variance ambient in individual patches.

Elsewhere, from the standpoint of genic selection (Lynch and Gabriel 1986), we have pointed out that our results are not consistent with the verbal hypothesis of Ayala and Valentine (Ayala et al. 1975; Valentine 1976; Ayala and Valentine 1979), which implies a diminishing role for spatial heterogeneity in the selection

process in temporally variable environments. Nevertheless, despite the complex ways in which different forms of environmental variability can interact, figure 1 illustrates the dominant role played by $V_{\phi rw}$ and suggests that, for clones taken from different environments, the relationship between V and $V_{\phi rw}$ should have a slope between 1 and 2 if optimal V is in fact strongly selected. As indicated in the figure, such a relationship would hold approximately even if the other variance parameters varied by one to two orders of magnitude.

If developmental influences (V_{E1} and V_{E2}) play a major role in determining an individual's optimal environmental state (z_1) and breadth of adaptation ($z_2^{1/2}$), then the realized breadth of adaptation ($V^{1/2}$) for a genotype will often differ from that expected on the basis of genetic properties alone (i.e., $g_2^{1/2}$). This need not always be the case, however. Because V_{E1} causes an increase in V and V_{E2} causes a decrease, the two factors may sometimes counterbalance each other.

From the standpoint of the genotype, natural selection will tend to favor a g_2 subject to the constraint that V is optimized. Thus, species for which $V_{\phi s}$, $V_{\phi rw}$, and $V_{\phi tb}$ are identical may be expected to evolve very different g_2 's (while maintaining the same V) if interspecific variation in V_{E1} and V_{E2} is pronounced. Since V_{E1} and V_{E2} are functions of such physiological processes as acclimation ability and maternal effects and of developmental homeostasis, interspecific variation would not be surprising. It is also worth noting that such developmental-variance components as V_{E1} and V_{E2} are not fixed properties of a genotype, but may be substantially modified depending on the environmental background of factors other than the one of interest (Falconer 1981). Thus, the realized breadth of adaptation of a genotype might be radically altered when it is transplanted to a new setting (Via and Lande 1985). Obviously, any attempt to interpret empirical estimates of V_{E1} and V_{E2} should consider this.

It is important to recognize that in analyzing for the optimal parameters (\hat{g}_1 , \hat{g}_2) of the tolerance curve, we have assumed a fixed trade-off between the maximum fitness of an individual and its breadth of adaptation (eqs. 1 and 2). This is not totally realistic since there must occasionally arise mutants, for which the integral in equation (2) (call it k) does not equal 1. In that case, a clone with nonoptimal (g_1, g_2) and k greater than 1 could sometimes displace a clone with optimal (g_1, g_2) and k equal to 1, since the former's fitness in all environments would be elevated by a factor of k relative to the expressions given above. The outcome of such events can be assessed by comparing the solutions to equations such as (1), (6), (9), and (11): substitute the appropriate values of g_1 and g_2 for the two clones and multiply the mutant's fitness by k . If adaptive mutations for k arise much more frequently than those for g_1 and g_2 , natural populations may often appear to be "maladapted" with respect to the optimal environmental state and breadth of adaptation.

We have attempted to provide statistical procedures for parameter estimation such that the hypotheses generated by our model can be tested. The methodology that we recommend may also be of practical value in evaluating the sensitivity of genotypes and/or populations to environmental extremes, as is routinely done in assessments of environmental impacts and in the development of resistant varieties of economically important crops. A large number of reports in the literature already contain data on the relationship of partial or total fitness to physical and/or

chemical parameters. Unfortunately, they do not state whether the types of patterns generated in figure 3 are often observed, since the mean estimates of fitness rather than the individual data points are always reported.

The theory we have developed demonstrates that a substantial amount of valuable information on the mechanisms underlying the breadth of adaptation is concealed when data are simply averaged. When sufficient data are obtained, the maximum-likelihood method has the power to identify the extent to which the breadth of adaptation of a population is caused by g_2 , V_{E1} , V_{G1} , V_{E2} , and V_{G2} . Such information is essential if one wants to estimate the long-term flexibility of a population in the face of new selective challenges. Thus, the large pools of data generated by those in areas of applied ecology (and often filed away in laboratory notebooks or agency reports and ignored by those of us in less-applied research areas) may be of great value in gaining a deeper understanding of the adaptational aspects of environmental tolerance.

SUMMARY

A theory for the expression of a population's response to density-independent gradients of environmental factors is derived for the case of asexuality. It is shown that the environmental tolerance of a genotype is a function of at least four parameters: g_1 and V_{E1} , the environmental optimum and its developmental variance between individuals, and g_2 and V_{E2} , the expected genetic contribution to the breadth of adaptation and its developmental variance. The realized breadth of adaptation of a genotype ($V^{1/2}$) is a complex function of g_2 , V_{E1} , and V_{E2} , but we argue that, with an appropriate scale transformation, the tolerance curve of a genotype is approximately normal, with mean g_1 and standard deviation $V^{1/2}$. It is shown that temporal heterogeneity in the environment selects for more-broadly-adapted genotypes but that the within-generation component ($V_{\phi tw}$) plays a more prominent role than the between-generation component ($V_{\phi tb}$). Spatial heterogeneity selects for higher $V^{1/2}$ only when it occurs in conjunction with temporal variance within generations and only if $V_{\phi tb}$ is small relative to $V_{\phi tw}$. We argue that since g_2 is expected to evolve subject to the constraint that $V^{1/2}$ is optimized, species exposed to conditions favoring identical $V^{1/2}$ may evolve different g_2 if pronounced interspecific differences exist for V_{E1} and V_{E2} .

A maximum-likelihood method is shown to be capable of generating accurate estimates of the genotypic parameters g_1 , g_2 , V_{E1} , and V_{E2} with moderately large samples. We suggest how this procedure may be used to estimate analogous parameters for a population of mixed genotypes and to obtain estimates of the genetic variance for the environmental optimum and breadth of adaptation. The potential utility of this methodology for the analysis of data routinely generated in programs for environmental assessment and plant breeding is pointed out.

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APPENDIX

DERIVATION OF THE GENOTYPIC TOLERANCE CURVE

Because of the influence of environmental effects on phenotype, the many individuals of a given genotype generally form a continuum of phenotypes. If we define the fitness of a phenotype as a Gaussian function of two characters, z_1 (the optimal environmental state) and z_2 (where $z_2^{1/2}$ is the breadth of adaptation), such that

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

where ϕ is a continuously distributed environmental state, then the genotypic tolerance curve is defined by

$$w(g_1, g_2 | \phi) = \int_0^{+\infty} \int_{-\infty}^{+\infty} w(z_1, z_2 | \phi) p(z_1 | g_1) p(z_2 | g_2) dz_1 dz_2. \quad (A1)$$

We take the conditional phenotype distribution for the environmental optimum to be normal, with mean g_1 and variance V_{E1} ,

$$p(z_1 | g_1) = (2\pi V_{E1})^{-1/2} \exp[-(z_1 - g_1)^2 / 2V_{E1}].$$

For the conditional distribution of z_2 , we seek a function of the form $p(x) = x^{\alpha-1} (1+x)^{-(\alpha+\beta)} / B(\alpha, \beta)$, where $B(\alpha, \beta) = \Gamma(\alpha)\Gamma(\beta) / \Gamma(\alpha + \beta)$ is the beta function with Γ representing the gamma function. This is a beta distribution of the second kind, and its moments about the origin are known to be

$$E(x^n) = B(\alpha + n, \beta - n) / B(\alpha, \beta) = \Gamma(\alpha + n)\Gamma(\beta - n) / [\Gamma(\alpha)\Gamma(\beta)]$$

(Kendall and Stuart 1977, p. 163). The mean is therefore $E(x) = \alpha / (\beta - 1)$ and the variance $\text{var}(x) = \alpha(\alpha + \beta - 1) / [(\beta - 1)^2(\beta - 2)]$. In order to make x dimensionless, we let it equal z_2 / V_{E1} . Then, by change of variable, we obtain

$$p(z_2 | g_2) = (z_2 / V_{E1})^{\alpha-1} [1 + (z_2 / V_{E1})]^{-(\alpha+\beta)} / [V_{E1} B(\alpha, \beta)]. \quad (A2)$$

The required conditions that the mean and variance of $p(z_2 | g_2)$ be independent (i.e., that $E(z_2) = g_2$ and $\text{var}(z_2) = V_{E2}$) are satisfied when

$$\alpha = g_2 [g_2 (g_2 + V_{E1}) + V_{E2}] / V_{E1} V_{E2},$$

$$\beta = g_2 (g_2 + V_{E1}) / V_{E2} + 2.$$

V_{E1} serves only as a scaling factor in equation (A2) and does not influence the shape of the distribution; that is, if $V_{E1} = kV_{E1}$, then $z_2' = kz_2$, $g_2' = kg_2$, $V_{E2}' = k^2 V_{E2}$, and $p(z_2' | g_2') = p(z_2 | g_2)$.

Equation (A1) is solved by first integrating over z_1 to obtain

$$w(g_1, g_2 | \phi) = [V_{E1} (2\pi)^{-1/2} / B(\alpha, \beta)] \cdot \int_0^{+\infty} z_2^{\alpha-1} (z_2 + V_{E1})^{-(\alpha+\beta+1/2)} \exp\{- (g_1 - \phi)^2 / [2(z_2 + V_{E1})]\} dz_2,$$

which, by change of variable to $t = V_{E1} / (z_2 + V_{E1})$, becomes

$$w(g_1, g_2 | \phi) = [(2\pi V_{E1})^{1/2} B(\alpha, \beta)]^{-1} \int_0^1 (1-t)^{\alpha-1} t^{\beta-1/2} \exp[- (g_1 - \phi)^2 t / (2V_{E1})] dt. \quad (A3)$$

The integral may be expressed in terms of the confluent hypergeometric function (Abramowitz and Stegun 1972, eq. 13.2.1),

$$\int_0^1 (1-t)^{b-a-1} t^{\alpha-1} \exp(ct) dt = B(b-a, a) M(a, b, c),$$

where $a = \beta + 1/2$, $b = \alpha + \beta + 1/2$, $c = -(g_1 - \phi)^2/2V_{E1}$, and

$$M(a, b, c) = 1 + \frac{ac}{b} + \frac{(a)_2 c^2}{(b)_2 2!} + \dots + \frac{(a)_n c^n}{(b)_n n!} + \dots$$

$$(a)_n = \Gamma(a+n)/\Gamma(a)$$

(Abramowitz and Stegun 1972, eq. 13.1.2). Utilizing the transformation $M(a, b, c) = \exp(c)M(b-a, b, -c)$ (Abramowitz and Stegun 1972, eq. 13.1.27), we obtain

$$w(g_1, g_2 | \phi) = (2\pi V_{E1})^{-1/2} [B(\alpha, \beta + 1/2)/B(\alpha, \beta)] M(b-a, b, -c) \exp[-(g_1 - \phi)^2/2V_{E1}]. \quad (A4)$$

Provided that α is large ($\gg 1$), equation (A4) can be greatly simplified by using two approximations. First, since for these conditions $(b-a)_n \approx \alpha^n$ and $(b)_n \approx (\alpha + \beta)^n$ for the early terms in the convergent expansion $M(b-a, b, -c)$, the series closely approximates that of the exponential function, and therefore

$$M(b-a, b, -c) \approx \exp\{\alpha(g_1 - \phi)^2/[V_{E1}(\alpha + \beta)]\}. \quad (A5)$$

Second, the term containing the beta functions can be reduced by making use of Sterling's approximation for gamma of large x , which yields

$$\Gamma(x)/\Gamma(x + 1/2) \approx [x/(x + 1/2)]^x x^{-1/2} \exp 1/2.$$

From the definition of the base of natural logarithms, $\lim_{x \rightarrow \infty} [x/(x + 1/2)]^x = \exp -1/2$, which yields the further simplification

$$\Gamma(x)/\Gamma(x + 1/2) \approx x^{-1/2}. \quad (A6)$$

Noting that

$$B(\alpha, \beta + 1/2)/B(\alpha, \beta) = \Gamma(\alpha + \beta)\Gamma(\beta + 1/2)/[\Gamma(\beta)\Gamma(\alpha + \beta + 1/2)]$$

and substituting (A5) and (A6) into (A4), we obtain the approximation

$$w(g_1, g_2 | \phi) \approx (2\pi V)^{-1/2} \exp[-(g_1 - \phi)^2/2V], \quad (A7)$$

where $V = V_{E1}(\alpha + \beta)/\beta$.

Thus, provided that α is large, the genotypic tolerance curve is expected to be approximately normal, with optimal environment g_1 and breadth of adaptation $V^{1/2}$. In actuality, the exact solution (A3) demonstrates that although $w(g_1, g_2 | \phi)$ is always symmetrical, it is also leptokurtic. However, the difference between approximations (A4) and (A7) is essentially undetectable for α greater than approximately 25, and the normal approximation provides a good approximation for α even as small as 5 (fig. 1).

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