EVOLUTION AND EXTINCTION
IN RESPONSE TO
ENVIRONMENTAL CHANGE

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INTRODUCTION

Extensive geological and paleontological research indicates that life historically has experienced periods of major environmental change, resulting from astronomical and tectonic events, on time scales ranging up to hundreds of thousands of years (Hays et al., 1976; Ruddiman and McIntyre, 1981; Hide and Dickey, 1991). Species have often responded to these changes by migrating, adapting, or speciating (Bartlein and Prentice, 1989; Davis, 1981; Cronin and Schneider, 1990), but in many cases they have simply gone extinct (Nitecki, 1984; Elliot, 1986; Jablonski, 1991). Because the rates of environmental change now being induced by human activity are well beyond those normally experienced by most organisms (Malone and Roederer, 1985; Bolin et al., 1986; Abrahamson, 1989; Schneider, this volume; Groom and Schumaker, this volume), there is justifiable concern about the possibility of a global environmental catastrophe.

Due to the complex structure of ecological communities, it is conceivable that the inability of one or a few critical species to cope with long-term environmental change will have cascading effects on other interdependent species (Holt, 1990), possibly producing a major alteration of community structure. It is therefore of some importance that we begin to develop a general understanding of the constraints on the ability of populations to adapt to environmental change. Since almost all quantitative characters exhibit some genetic variation, prolonged directional change in the envi-
Evolution, Extinction, and Environmental Change

Environment generally will result in adaptive evolution. The critical question is whether, in the face of unfavorable environmental change, a population can evolve rapidly enough to avoid extinction.

The salient issues are summarized in Figure 1, in which it is assumed that the source of environmental change is external to the ecosystem and uninfluenced by changes in local community composition. Populations can profit from environmental change (e.g., populations at their polar limits may benefit from global warming), but our focus will be on situations where the environment is shifting toward conditions that are clearly unsuitable for a locally adapted population. The initial direct impact of such environmental change will be a reduction in reproductive capacity and/or viability resulting from maladaptation to the new conditions. Provided the necessary genetic variation exists, natural selection will result in the gradual evolution of more suitable phenotypes, but any substantial reduction in population size provoked by the environmental change will reduce the opportunities for such adaptation. The amount of genetic variation that can be maintained in a population decreases as population size decreases, and when populations are reduced to very small sizes, the chance fixation of unconditionally deleterious alleles may lead to a further decline in fitness.

These circumstances may be modified in mobile species if the change in environment induces individuals to migrate in search of more suitable habitat. Any further reduction in local population size by emigration will reduce the local evolutionary potential even more, especially if the popu-

![Diagram](image)

**Figure 1.** A simplified picture of the factors influencing the response of local population size to environmental change. Only the influences denoted by black lines are considered explicitly in this paper.
lation is geographically isolated (e.g., an island population or a preserve surrounded by unsuitable habitat). Provided the emigrants are successful, however, dispersal can replace evolution as a survival strategy, at least temporarily. But when environmental change operates over a broad enough geographic range or over a long enough time scale so that the current set of phenotypes is unsuitable in all habitats, evolution provides the only route to species survival.

In addition to the direct impact of an environmental change on a species' physiological, growth, or reproductive characters, it can exert substantial indirect influences through changes in the abundances of interacting species such as prey, predators, competitors, parasites, or hosts. In the long run, the net influence of such indirect effects can be detrimental or beneficial depending on the total change in community composition, and they may sometimes overwhelm the direct effects. Since the consequences of both dispersal and community structure change are likely to be highly species-specific, neither of those issues will be pursued any further here, although their potential significance should not be underestimated.

Ideally, an understanding of the evolutionary and demographic consequences of long-term environmental change ought to be provided by empirical research. But for many species this is impractical, if not impossible, due to long generation times, difficulties in rearing, ethical issues, etc. Because of the urgency of the problem, the development of mathematical models seems well warranted. Ultimately, such models will require the integration of population dynamics theory with evolutionary theory, an area that has received very little attention in the past. We present some initial results in this paper.

Consider a character under stabilizing selection in an environment changing such that the optimum phenotype increases (or decreases) with time. To a degree depending on the magnitude of the additive genetic variance for the trait, the mean phenotype in the population will lag behind the changing optimum. This lag in adaptation will cause a reduction in mean population fitness that, if substantial enough, will result in a decline in population size. If the rate of environmental change is sufficiently slow, the mean phenotype will remain close enough to the optimum that extinction can be avoided. However, there must be a critical rate of environmental change beyond which the selective load is so great that a positive population growth rate cannot be maintained.

The purpose of this paper is to evaluate how this critical rate of environmental change depends on certain aspects of the genetic system. For the sake of simplicity, we assume that individual fitness is determined predominantly by a single continuously distributed character subject to the standard laws of quantitative inheritance (Falconer, 1989). The character under consideration might be an attribute of size or shape, thermal or pH optimum, etc. We examine two extreme situations: sexual populations of arbitrary size, and asexual populations that are effectively infinite in size.
SELECTION IN A CHANGING ENVIRONMENT

Natural selection commonly favors an intermediate phenotype, with fitness declining approximately quadratically with the absolute deviation from the optimum (Johnson, 1976). The focus of this paper is on the situation in which the optimum phenotype θ exhibits temporal change. We will be considering continuously growing populations with overlapping generations, so the appropriate measure of fitness is the instantaneous rate of increase. Thus, at any point in time, the phenotypic fitness function (i.e., the expected fitness of an individual with phenotypic value z) can be represented by

\[ r_z = r_m - \frac{(z - \theta)^2}{2\sigma_w^2} \]  

(1)

where \( r_m \) is the rate of increase for the optimum phenotype, and \( \sigma_w \) is the width of the fitness function. Note that as \( \sigma_w \) increases, the curvature of the fitness function, and hence the intensity of selection, declines. A negative value of \( r_z \) implies that individuals of phenotype \( z \) do not produce enough offspring to replace themselves. From Equation (1), this is seen to occur whenever the absolute deviation of \( z \) from the optimum exceeds \((2r_m\sigma_w^2)^{1/2}\). The parameters \( r_m \), \( \theta \), and \( \sigma_w \), at least in principle, are all estimable properties of natural populations (Lande and Arnold, 1983; Mutchell-Olids and Shaw, 1987; Schluter, 1988; Lynch et al., 1991).

Quantitative characters are usually influenced by multiple genetic loci and environmental effects. Consequently, they generally exhibit a continuous distribution of variation that is approximately normal on some scale of measurement. The observed phenotypic value of an individual can be represented as the sum of two components: an additive genetic value (\( g \)), and a nonheritable residual deviation (\( e \)) from \( g \) caused by nonadditive gene action, environmental effects, and developmental noise. Assuming the distribution of additive genetic values has mean \( \bar{g} \) and variance \( \sigma_g^2 \), and the distribution of residual deviations has mean 0 and variance \( \sigma_e^2 \), then the rate of population growth is

\[ r = r_m - \frac{(\bar{g} - \theta)^2 + \sigma_e^2}{2\sigma_w^2} \]  

(Lynch et al., 1991), where \( \sigma_g^2 = \sigma_e^2 + \sigma_e^2 \) is the phenotypic variance. Note that because the mean residual deviation (\( \bar{e} \)) is assumed to be equal to zero (i.e., we are modeling only genetic change), \( \bar{g} \) is equivalent to the mean phenotype in the population.

We interpret \( r \) as the maximum rate of population growth that would be observed in the absence of density-dependent influences. Thus, if \( r < 0 \) for very long, the population is clearly doomed to extinction. Note that even if the mean phenotype is at the optimum (\( \bar{g} = \theta \)), the population rate of growth will generally be less than the maximum possible (\( r_m \)), since any
level of phenotypic variance will result in the production of nonoptimal phenotypes. Thus, although genetic variance provides the fuel for adaptive evolution, it also imposes a load on the population.

We will assume that the phenotype is measured on a scale such that the initial optimal phenotype is equal to zero. Due to the environmental change, the optimum increases at an expected rate $k$ per unit time. The actual optimum at time $t$ is then

$$\theta(t) = kt + \varepsilon_\theta$$  \hspace{1cm} (3)

where $\varepsilon_\theta$ represents stochastic temporal variation in the optimum around its expected trajectory. We assume that $\varepsilon_\theta$ is normally distributed with no correlation in time, mean equal to 0, and variance equal to $\sigma_\theta^2$. The width of the fitness function is assumed to be constant.

**FINITE SEXUAL POPULATIONS**

As a consequence of human disturbance, many species, especially vertebrates and vascular plants, have been reduced to isolated populations containing tens to hundreds of individuals. For purely demographic reasons, such species are highly vulnerable to extinction even in fairly constant environments (Lande, 1988), but when they are confronted with the additional problem of an environmental change, their vulnerability to extinction will be magnified even further.

The development of a theory for critical rates of environmental change for finite sexual populations introduces a number of technical difficulties. For infinite populations in a deterministic environment [i.e., $\theta(t) = kt$, $\sigma_\theta^2 = 0$], the dynamics of the mean phenotype can be evaluated explicitly (Lynch et al., 1991). But for the case under consideration, there is no single deterministic outcome. Random genetic drift of the mean phenotype, combined with stochasticity of the environment, results in a probability distribution of mean phenotypes realized among a hypothetical set of replicate populations. We have employed diffusion theory to evaluate this distribution.

We start with the stochastic differential equation

$$\frac{d\overline{\theta}}{dt} = \frac{\sigma_\theta^2(kt + \varepsilon_\theta - \overline{\theta})}{\sigma_\theta^2} + \varepsilon_\theta$$  \hspace{1cm} (4)

where $\varepsilon_\theta$ is the change in the mean phenotype caused by random genetic drift, and the nonstochastic portion of the equation follows from procedures for computing the change in the mean of a normally distributed character under quadratic selection (Lynch et al., 1991). The remaining results in this section (several steps to which we omit) are obtained by expressing this model in the form of a classical Ornstein-Uhlenbeck diffusion process (Karlin
and Taylor, 1981), under the assumption that the genetic variance, \( \sigma_g^2 \), is essentially constant in time. Justification for this assumption is given in the appendix, where it is also argued that provided \( N_e \) is less than \( 10^3 \) or so, \( \sigma_g^2 = 2N_e \sigma_m^2 \), with \( N_e \) being the effective population size and \( \sigma_m^2 \) the rate of input of genetic variance by mutation.

The expected dynamics of the mean phenotype conditional on a given value of \( \bar{g} \) are

\[
E\left( \frac{d\bar{g}}{dt} \mid \bar{g} \right) = \frac{\sigma_g^2 k t - \bar{g}}{\sigma_w^2} \tag{5}
\]

This shows that the expected mean phenotype always evolves towards the optimum, and it does so at a rate that is proportional to the additive genetic variance and to the inverse of the squared width of the fitness function.

Random genetic drift causes the variance of the distribution of mean phenotypes to increase by the amount \( \sigma_g^2/N_e \) per generation (Lande, 1976), and stochasticity in the environment further inflates the variance in the probability distribution of the mean phenotype by the amount \( \sigma_g^2 \sigma^2_w/\sigma_w^4 \). Thus, the variance in the rate of change of the mean phenotype conditional on a given value of \( \bar{g} \) is

\[
V\left( \frac{d\bar{g}}{dt} \mid \bar{g} \right) = \frac{\sigma_g^2}{N_e} + \frac{\sigma_g^2 \sigma^2_w}{\sigma_w^4} \tag{6}
\]

The asymptotic distribution of \( \bar{g} \) for large \( t \) can be viewed as that expected after sufficient time has passed for a stochastic balance to be reached between the forces of selection, drift, and mutation. This distribution can be shown to be normal with mean

\[
E(\bar{g}) = kt - k \frac{\sigma^2_g}{\sigma_w^2} \tag{7}
\]

and variance

\[
V(\bar{g}) = \frac{\sigma_w^2}{2N_e} + \frac{\sigma^2_g \sigma^2_w}{2\sigma^4_w} \tag{8}
\]

Equation (7) shows that the expected mean phenotype evolves asymptotically at exactly the same rate as the change in the optimum (\( k \)) but lags behind the optimum by an amount \( k \sigma^2_w/\sigma_g^2 \). The variance of \( \bar{g} \) consists of a random genetic drift term, \( \sigma^2_w/(2N_e) \), identical to that found by Lande (1976), and an additional term due to the environmental stochasticity.

Taking the expectations of Equation (2) over the distributions of \( \bar{g} \) and \( \theta \), the expected long-term fitness of a population in stochastic equilibrium with the changing environment is found to be

\[
E(r) = r_m - \frac{\sigma^2_g}{2\sigma^2_w} - \frac{k^2 \sigma^2_g}{2\sigma^4_w} - \frac{1}{4N_e} - \frac{\sigma^2_\theta}{2\sigma^2_w} \left( \frac{\sigma^2_g}{2\sigma^4_w} + 1 \right) \tag{9}
\]
Four types of loads contribute to the reduction in the expected population growth rate below its maximum possible value, \( r_m \). In order of their appearance in Equation (9), first, as noted above, phenotypic variance results in a net loss of fitness due to the production of nonoptimal phenotypes. Second, a lag load results from the deviation of the expected mean phenotype from the moving optimum. With increasing \( \sigma_e^2 / \sigma_w^2 \), the population mean tracks the moving optimum more closely, and the lag load is reduced. A third type of load results from the stochastic dispersion of the mean phenotype from its expectation due to random genetic drift. As shown by Lande (1976, 1980), the drift load has the interesting property of being independent of both the genetic variance and the width of the fitness function. While the amount of drift in the mean phenotype increases with the genetic variance, so does the response to selection, and the influences of those two factors on \( r \) cancel exactly. Similarly, although a flatter fitness function provides a greater opportunity for drift, the fitness consequences of a unit change in the mean phenotype are reduced, and again the two factors cancel. Fourth, environmental stochasticity causes a dispersion of the mean phenotype from the path of the expected optimum due to both the past selection response to the variation in \( \theta \) and to the current deviation of the realized \( \theta \) from its expectation. The first component of the environmental stochasticity load increases with the genetic variance, which causes the mean phenotype to carry a “memory” of past stochastic variation.

The critical rate of environmental change, beyond which the population crosses the extinction threshold, is the absolute value of \( k \) that satisfies \( E(r) = 0 \),

\[
k_c = \frac{\sigma_e^2}{\sigma_w^2} \left[ 2 \bar{r}_m - \frac{1}{2N_e} - \sigma_e^2 \left( \frac{\sigma_e^2}{2\sigma_w^2} + 1 \right) \right]^{1/2}
\]

(10)

where \( \bar{r}_m = r_m - (\sigma_e^2 / 2\sigma_w^2) \) is the maximum rate of population increase when the mean phenotype is at the optimum. For the special case of large population size \( (N_e \to \infty) \) and a deterministic environment \( (\sigma_e^2 = 0) \), Equation (10) simplifies to

\[
k_c = \sigma_e^2 \sqrt{2 \bar{r}_m / \sigma_w^2}
\]

(11)

Noting that the genetic variance is necessarily less than the phenotypic variance, an upper bound to the critical rate of environmental change can be obtained from this expression. In units of phenotypic standard deviations,

\[
k'_c = k_c / \sigma_e < \sigma_e \sqrt{2 \bar{r}_m / \sigma_w^2}
\]

(12)

Since it is much easier to estimate \( \sigma_e^2 \) than \( \sigma_g^2 \), and since \( \bar{r}_m \) is also estimable, this expression may have some practical utility for identifying situations in which an extinction threshold will definitely be exceeded. For example, if the maximum possible rate of population increase is 0.5 per generation,
and the width of the fitness function is equivalent to ten phenotypic standard deviations, then \( k'_c = 0.1 \), implying that the population could not possibly sustain a prolonged change in the optimum value of the selected trait in excess of 0.1 phenotypic standard deviations per generation.

Using a commonly observed value for mutational heritability, \( \sigma'^2_w/\sigma^2_w \), to compute the genetic variance, Figure 2 illustrates the relationship between \( k'_c \) (\( k'_c \) in units of phenotypic standard deviations) and the effective population size for three values of \( \sigma^2_w \) and \( \sigma^2_w \), for the case in which the potential rate of increase is \( r_m = 0.67 \) (equivalent to a potential population doubling per generation). For the parameter values used in this figure, there is a nearly log-linear increase of \( k'_c \) with population size, due largely to the dependence of \( \sigma^2_c \) on \( N_e \). Note also that, counterintuitively, the critical rate of environmental change actually declines with decreasing selection intensity (increasing \( \sigma^2_w \)). This is due to the increased lag load that develops when selection is weak.

LARGE ASEXUAL POPULATIONS

Since microbes such as bacteria, fungi, and algae play critical roles in all biogeochemical cycles and form the base of most food chains, their abundances influence most higher-level organisms either directly or indirectly. Therefore, it seems especially important to develop a general understanding of the constraints on the evolution of microorganisms. Random genetic drift is unlikely to play much of a role in the evolutionary dynamics of microbial populations, since their population sizes are usually effectively infinite, and the drift load can be assumed to be negligible. However, most microbial populations reproduce predominantly by asexual means, and the absence of segregation and recombination is expected to lead to reduced levels of genetic variation, and therefore to lower evolutionary potential relative to the expectation in otherwise similar sexual populations (Lynch and Gabriel, 1983).

As in the case of sexual populations, asexual populations asymptotically settle into an expected rate of evolution equal to the rate of change in the environment, lagging behind it by a constant amount (Lynch et al., 1991). The expected asymptotic dynamics of the mean phenotype are as described by Equation (7). Noting that \( \tilde{\sigma}_p^2 = \sigma_m \sigma_w \) (Lynch et al., 1991; Appendix), the asymptotic distribution of the mean phenotype is obtainable from Equations (7) and (8). The extinction threshold is then simply

\[
k_c = \sigma_m \left[ 2\tilde{\sigma}_m - \frac{\sigma_0^2}{\sigma_w} \left( \frac{\sigma_m}{2\sigma_w} + 1 \right) \right]^{1/2}
\]  

(13)

Thus, the critical rate of environmental change increases with the rate of polygenic mutation, \( \sigma^2_m \), which provides the genetic variance required for adaptive evolution. It also increases with the maximum population growth
FIGURE 2. Critical rate of environmental change $k'$ (in units of phenotypic standard deviations), beyond which the expected rate of population growth becomes negative, given as a function of the effective population size and the width of the fitness function. Results are given for $r_m = 0.67$ (equivalent to a potential population doubling per unit time), $\sigma_m^2 = 0.001$, $\sigma_e^2 = 1$ (a typical observed mutational heritability is $\sigma_m^2/\sigma_e^2 = 0.001$; Lynch, 1988), and letting $\sigma_e^2 = 2N_e\sigma_m^2$ (Appendix). In the upper panel, the population cannot maintain a positive growth rate at any $N$, when $\sigma_e^2 = 10$.  

242
rate, \( \bar{r}_m \), and it decreases with environmental stochasticity, \( \sigma^2_e \). Note that when the temporal change in \( \theta \) is deterministic (\( \sigma^2_\theta = 0 \)), \( k_e \) is independent of the width of the fitness function. Because the genetic variance is proportional to \( \sigma^2_w \), and the lag load is proportional to \( \sigma^2_e/\sigma^2_g \), the latter is independent of \( \sigma^2_w \).

Using the results in the Appendix, the critical rate of environmental change per generation for an asexual species is found to be only about \( (2n_e)^{-1/2} \), where \( n_e \) is the effective number of loci—as large as that for a sexual species. Thus, all other things being equal, recombination provides sexual species with an evolutionary advantage. However, with their shorter generation times, it is possible that asexual microorganisms can sustain larger values of \( k \) on an absolute time scale. For example, all other things being equal, on an absolute time scale, \( k_e \) for an asexual population would match that of a sexual population if the latter’s generation time were \( \sqrt{2n_e} \) longer.

**TRANSMISSION DYNAMICS**

Up to now, the results that we have described apply to the steady-state situation in which the expected lag in the population mean phenotype has been attained. The general relevance of the theory depends on the time it takes to reach this state. The transient dynamics for an effectively infinite asexual population in a deterministic environment are worked out in Lynch et al. (1991). Here we present the more general case.

If it is assumed that the genetic variance is initially at its equilibrium value, the dynamics of the expected mean phenotype can be obtained from the solution to Equation (4),

\[
E[\bar{g}(t)] = \left[ \bar{g}(0) + (k/\alpha) e^{-\alpha t} \right] + kt - (k/\alpha)
\]

where \( \alpha = \sigma^2_g/\sigma^2_w \). This expression converges to Equation (7) for large \( t \). The time scale for the approach to the equilibrium lag is proportional to \( 1/\alpha \). That is, a doubling of \( \alpha \) speeds up the dynamics by a factor of two. Moreover, a doubling of \( \sigma^2_g \) has the same influence on the expected evolutionary trajectory as a halving of \( \sigma^2_w \).

Provided the effective population size and the environmental stochasticity have been constant in the past, the initial realized mean \( \bar{g}(0) \) actually will be distributed about the expectation \( \theta = 0 \) with variance given by Equation (8). However, it will take some time for the evolutionary trajectory for any single population to develop the steady-state variance in time. Using diffusion theory, it is possible to solve for the transient dynamics in the variance of the population mean phenotype,

\[
V[\bar{g}(t)] = \left( \frac{\sigma^2_{2N_e}}{2N_e} + \frac{\sigma^2_{2N_e}}{2\sigma^2_w} \right) \left[ 1 - e^{-2\alpha t} \right]
\]

(15)
This converges to Equation (8) as \( t \to \infty \), the time scale being half that noted above for the dynamics of the mean.

An example of the dynamics of the distribution of \( \bar{g} \) is given in Figure 3. The expected lag of the mean phenotype behind the optimum, \( \bar{g} - kt \), attains 50 percent of the equilibrium lag by \( t = 0.7/\alpha \) generations, which for the example given (\( \sigma^2_g = 1.0 \) and \( \sigma^2_w = 25 \)) occurs in only 17 generations. The variance of the distribution of \( \bar{g} \) attains 50 percent of its equilibrium value in half that time. Note that due to the relatively high level of environmental stochasticity in this example, the expected equilibrium lag is approximately 1.8 phenotypic standard deviations, and there is always some possibility that the realized mean phenotype will equal or exceed the optimum.

**DISCUSSION**

Although the theory we have presented relies on a number of simplifying assumptions, it provides a useful starting point for investigating the potential for populations to adapt to long-term environmental change. Obviously, due to functional and developmental constraints, no character can be expected to evolve directionally for an indefinite period of time. Our results are

![Figure 3](image-url)

**FIGURE 3.** Probability distribution of the mean phenotype as a deviation from the expected optimum, \( \theta = kt \), as a function of \( t \) generations. The initial mean is assumed to be \( \bar{g} = 0 \), the genetic variance to be at its equilibrium expectation \( 2N_e \sigma^2_g \), \( \sigma^2_g = 0.001 \), \( k = 0.05 \), \( \sigma^2_w = 25 \), \( \sigma^2 = 10 \), and \( N_e = 500 \). The heaviest (leftmost) curve is the asymptotic distribution approached as \( t \to \infty \). If it is assumed that the environmental variance is \( \sigma^2_x = 1 \), which is reasonable for the value of \( \sigma^2_m \) used (Lynch, 1988), then the phenotypic variance is equal to 2, and each unit on the abscissa is equivalent to 0.7 phenotypic standard deviations.
meant to identify rates of environmental change that can be sustained when adaptive potential is present. For that reason and others discussed below, the critical rates that we have identified are likely to be upper bounds.

The models that we have employed are based on the premise, first suggested by Fisher (1918), that most complex traits are products of a large number of loci with individually small effects. This premise has stood up to a substantial body of empirical work (Falconer, 1989). Results from long-term selection experiments on numerous characters, in both plants and animals, show very clearly that directional changes on the order of 10 or more phenotypic standard deviations can often be accomplished in less than 100 generations (Jones et al., 1968; Kress, 1975; Dudley, 1977; Eisen, 1980; Weber and Diggins, 1990). Changes of this magnitude are well outside the range of variation seen in the base population at the start of an experiment, but they need not involve mutation. As selection jointly advances the frequencies of favorable alleles at multiple loci, very large changes in the mean phenotype can be accomplished with the genetic variance residing in the base population alone. But there is now compelling evidence that polygenic mutation makes significant additional contributions to long-term selection response (Lynch, 1988; Mackay, 1989; Keightley and Hill, 1990; Weber and Diggins, 1990; Caballero et al., 1991). Dykhuizen (1990) reviews these issues in microbial populations, which usually respond to selective challenges via multiple mutational steps.

These observations imply that the critical rates of environmental change that we have identified can be sustained evolutionarily for a large number of generations before functional or developmental constraints become important. For example, from Equation (13), \( k_c \) for an asexual population is necessarily less than \( \frac{2\mu_m}{\sigma_m^2} \). Assuming \( \mu_m \) is on the order of 0.5 or less, and noting that \( \sigma_m^2 \) is on the order of \( 10^{-3} \) (Lynch, 1989), then an upper limit to the rate of environmental change (and the steady-state rate of evolution associated with it) that can be sustained by an asexual population is roughly 0.05 phenotypic standard deviations per generation. For finite sexual populations, from Equation (11), \( k_c \) is necessarily less than \( \frac{2N_e\sigma_m^2}{[2\mu_m/(\mu_m^2+2\sigma_m^2)]^{1/2}} \), which is almost certainly less than \( (2N_e\sigma_m^2/(\mu_m^2+[2\mu_m])^{1/2} \) for a viable population. So for populations smaller than \( N_e = 500 \), the critical rate of environmental change will be less than a phenotypic standard deviation per generation, perhaps considerably so. Note that since the population mean phenotype is expected to evolve at exactly the rate \( k \) at equilibrium, these estimates of \( k_c \) can also be viewed as maximally sustainable rates of evolution.

A number of additional issues need to be addressed in future research. For example, our work only deals explicitly with a single panmictic population in a spatially homogeneous environment, a situation particularly relevant to species that are fragmented into isolated demes. Another common type of population structure, one being increasingly imposed on organisms
by human activity, involves semi-isolated demes interconnected by narrow corridors of suitable habitat. Little theoretical work has been done on stabilizing selection with a moving optimum in a subdivided population. It seems likely, though, that population subdivision would reduce the critical rate of environmental change due to an increase in the lag and drift loads in local populations. Fease et al. (1989) have examined the situation in which a spatial cline in the optimum moves across a continuous landscape inhabited by a population with restricted dispersal ability, pointing out that there is a critical rate of environmental movement beyond which the population's ability to migrate and/or locally adapt is overwhelmed by the selective load.

As noted in the Appendix, there is still considerable debate as to the appropriate way to model the genetic variance for characters under stabilizing selection. When there is a directional component to selection and population sizes are on the order of $10^3$ or less, there are reasons to expect the predictions of the neutral model to approximate the equilibrium genetic variance closely, an assumption that we relied on in the preceding examples for sexual populations. But to verify this, the theory for Gaussian selection with a moving optimum needs to be further developed mathematically. In our derivations, we also ignored fluctuations in the genetic variance that might exist between generations. This should be roughly valid for effectively infinite asexual populations, but less so for small sexual populations. Further work needs to be done to evaluate how violations of these assumptions influence the quantitative predictions of our theory. Since the critical rates of environmental change identified above are nonlinear functions of the genetic variance, a high degree of variation in the latter would affect the predictions.

Throughout this chapter, we have treated the effective population size as a constant, thereby assuming that the population maintains a roughly constant density until the extinction threshold has been crossed. In the future, it will be useful to model the process more explicitly by allowing the population density to respond directly to the environmental change. One way of approaching this problem is to couple the equations for the dynamics of the mean phenotype and the genetic variance with a population dynamics equation, such as the logistic model. As noted above, when the population size is finite and the environmental change has a stochastic element, the realized population rate of increase varies around its expectation, sometimes becoming negative. This will cause the population to decline below its carrying capacity occasionally, which will temporarily increase the drift load, and if prolonged, will cause a reduction in the genetic variance. Recovery from the loss of genetic variance may then require a long period of replenishment by polygenic mutation. By leading to an even greater lag load in a changing environment, these two genetic consequences of a population bottleneck will further reduce the population's
ability to maintain itself, and may cause it to become extinct. We anticipate that these kinds of synergistic effects will lead to critical rates of environmental change that are somewhat lower than those defined above.

These kinds of synergistic interactions between random genetic drift and the loss of population fitness have been explored recently in a somewhat different context—finite populations in a constant environment, with recurrent mutation to unconditionally deleterious alleles (Gabriel et al., 1991; Lynch and Gabriel, 1991). In small populations, there is always some possibility that a mildly deleterious mutation will increase in frequency, sometimes even going to fixation. This causes a further decline in population size, which makes it easier for the next round of deleterious mutations to drift to high frequency, which again reduces the population size, and so on, ultimately leading to population extinction. This process, referred to as a mutational meltdown, will obviously be exacerbated in a changing environment that precipitates a decline in population size, and should be explored in the future.

Because we have focused only on the long-term average maximum growth rate of a population under selection, rather than on the actual population dynamics, our results have an additional limitation. We have only identified the critical rates of prolonged environmental change beyond which a population is doomed to certain, rapid extinction. However, even if the long-term average growth rate is predicted to be positive, random genetic drift and/or environmental stochasticity can cause any population to temporarily deviate far enough from the optimum for a long enough time that extinction might occur. The likelihood of such an event will be magnified if the rate of environmental change is already close to the extinction threshold, but it will also depend on the effective population size, the rate of polygenic mutation, the intensity of selection, and the degree of environmental stochasticity. These issues can be addressed by extending future work to the expected distributions of extinction times.

Although our focus has been on Gaussian selection with a moving optimum, the approach can be extended readily to other forms of selection. For example, Hill (1982) showed that under long-term truncation selection (and under the assumptions of our genetic model), a population asymptotically settles into a steady-state evolutionary trajectory, with the mean phenotype lagging behind the advancing truncation point by a constant amount. The asymptotic rate of evolution is simply $\sigma^2_g / \sigma^2$, where $i$ is the standardized selection intensity, and as noted above, $\sigma^2 = 2N_e \sigma^2_m$. Provided the fraction of the population eliminated by selection per generation ($p$) is in the range of 0.25 to 0.995, then $i = 0.8 + 0.4 \ln[p/(1 - p)]$ (Smith, 1969).

In units of phenotypic standard deviations, the critical rate of increase (or decrease) of the truncation point is simply $k^* = h^2 i_c$, where $h^2$ is the equilibrium heritability of the trait $(2N_e \sigma^2_m / \sigma^2)$, and $i_c$ is the critical selection intensity beyond which the population cannot maintain itself. As a simple
example, consider a low-fecundity population that requires at least 50 percent survival (from selection) to maintain itself. Then, $p = 0.5$, $i_e = 0.8$, and $k_e' = 0.8k^2$—the population would be doomed to certain extinction if the rate of movement of the truncation point exceeded $0.8k^2$ phenotypic standard deviations per generation. Because $\sigma_m^2/\sigma_e^2$ is on the order of $10^{-3}$, when $N_e$ is small $k^2$ is on the order of $2N_e \times 10^{-3}$. For the above example, this implies a critical rate of environmental change of approximately $1.6N_e \times 10^{-3}$ phenotypic standard deviations per generation.

In closing, we note that our heuristic models suggest some simple experiments that might be done to further clarify our understanding of the consequences of environmental change for evolution and extinction. For practical reasons, such experiments will need to be performed with species that have short generation times and that are grown easily in controlled laboratory or field environments. By exposing a large set of replicate populations to a stepwise or continual change in an important environmental parameter, such as temperature, it should be possible to acquire some information on the mean and variance of evolutionary rates and of extinction times. It will be especially useful to perform such experiments at several different effective population sizes and at several levels of stochastic variation of the environmental parameter. Obviously, the results of such “natural selection” experiments are likely to be rather species-specific, but given the nearly complete absence of critical work on the problem (with the exception of that on bacteria) and the significant contribution that such studies would make to the further development of the theory, a well-designed series of empirical studies is essential to further progress in this area.

**SUMMARY**

Due to the ubiquity of genetic variation for quantitative characters, populations that are exposed to environmental change are expected to adapt gradually via evolutionary changes in one or more traits. However, natural selection will result in a reduction in average viability and/or fecundity. If the rate of environmental change is sufficiently fast, the reduction in fitness due to selection will overwhelm the population’s ability to maintain itself, even though adaptation may be occurring, and extinction will ensue. We present some simple models that lead to predictions for the critical rate of environmental change beyond which extinction is inevitable. Results are given that suggest the relationship of this rate to the intensity of selection, the magnitude of polygenic mutation, the effective population size, and the reproductive system.
APPENDIX:
ADDITIVE GENETIC VARIANCE UNDER
GAUSSIAN SELECTION

Following the procedures of Kimura (1965) and Latter (1970), an explicit expression can be obtained for the dynamics of the genetic variance expected under the joint action of random genetic drift, mutation, and Gaussian selection. Under the assumptions of normality, the dynamics of the expected genetic variance are given by

\[
\frac{d\sigma^2_g}{dt} = \sigma^2_m - \frac{\sigma^4_g}{2n_e\sigma^2_w} - \frac{\sigma^2_g}{2N_e}
\]

(A1)

where the three terms respectively describe the input of new variance via polygenic mutation, and the losses of variance by selection and by random genetic drift. \(\sigma^2_m = 2\mu E(a^2)\) is the rate of input of new genetic variance via mutation, and \(n_e\) is the effective number of segregating factors, which is roughly the chromosome number plus the mean number of crossovers per meiotic event. The above expression is a Riccati equation, the solution to which is

\[
\sigma^2_g(t) = \sigma^2_e + \left[\left(\frac{1}{\sigma^2_e(0) - \sigma^2_e} + \frac{1}{2\phi n_e\sigma^2_w}\right) e^{\phi t} - \frac{1}{2\phi n_e\sigma^2_w}\right]^{-1}
\]

(A2)

where \(\phi = 1/(2N_e) + \sigma^2_g/n_e\sigma^2_w\). The expected genetic variance for sexual populations under drift–mutation–selection equilibrium, \(\sigma^2_g\), is obtained by solving the preceding equation as \(t \to \infty\).

\[
\sigma^2_g = \sqrt{n_e\sigma^2_m/2N_e} + 2n_e\sigma^2_w\sigma^2_m - (n_e\sigma^2_w/2N_e)
\]

(A3)

When drift prevails over selection (\(N_e\) small, \(\sigma^2_w\) large), \(\sigma^2_g = 2N_e\sigma^2_m\), the neutral result (Lynch and Hill, 1986). The same result is approached asymptotically as \(n_e\) becomes very large, since the intensity of selection operating on individual loci becomes infinitesimally small.

There has been considerable debate as to the validity of Kimura's "infinite-allele" approach versus an alternative non-Gaussian "house-of-cards" approach to modelling the genetic variance of quantitative traits in sexual populations (Lande, 1975; Turelli, 1984; Barton and Turelli, 1987; Slatkin, 1987). However, most of the debate has concentrated on populations of effectively infinite size under stabilizing selection with the mean phenotype coinciding with the optimum. For finite populations under directional (truncation) selection, the equilibrium genetic variance is remarkably close to the neutral expectation, provided the distribution of mutational effects is symmetrical about zero (Hill, 1982; Keightley and Hill, 1987; Hill and Keightley, 1988). Favorable mutations make a higher average contribution to the heterozygosity while they are segregating than do neutral mutations,
but this is essentially balanced by the shorter time to fixation for the former. These results, combined with other recent observations that the "house-of-cards" model also yields solutions close to the neutral expectation for $N_e < 10^3$ or so (Keightley and Hill, 1988; Bürger et al., 1989; Houle, 1989), provide fairly strong justification for the use of $\sigma_g^2 = 2N_e \sigma_m^2$ for small sexual populations. This is a particularly useful result because both $N_e$ and $\sigma_m^2$ are estimable parameters (Lande and Barrowclough, 1987; Lynch, 1988).

More generally, for nonsymmetrically distributed mutational effects, $\sigma_g^2 = 4PN_e \sigma_m^2$, where $P$ is the fraction of mutations with effects in the direction of selection (Keightley and Hill, 1987).

The assumption that the genetic variance is constant in time cannot be strictly true for finite populations, but some justification for such an approximation can be given for sexual populations of at least moderate size. Imagine a set of replicate populations all exposed to the same stochastic processes, and assayed for their additive genetic variance at the same point in time. For neutral quantitative characters, the squared coefficient of variation of the additive genetic variance among replicates is approximately

$$\frac{8 + [E(a^4)/4\mu E^2(a^2)]/(3N_e)}{(3N_e)},$$

where $\mu$ is the total gametic mutation rate for the trait, and $E(a^2)$ and $E(a^4)$ are the second and fourth moments of mutational effects (Keightley and Hill, 1989; Zeng and Cockerham, 1991). If the distribution of mutational effects is highly leptokurtic [i.e., $E(a^4) >> E^2(a^2)$] and/or the gametic mutation rate is low (i.e., $\mu << 1$), the second term can dominate. When mutational effects are normally distributed with mean zero, i.e., $E(a^4) = 3E^2(a^2)$, the above expression reduces to

$$\frac{8 + (3/4\mu)\mu}{(3N_e)}.$$

Computer simulations of Keightley and Hill (1989) showed that these expressions work quite well for populations under stabilizing selection when the mean is at the optimum, but the model that we are considering (with a moving optimum) has not been investigated. The most we can say at this point is that the coefficient of variation of the additive genetic variance is unlikely to be less than $(3/N_e)^{1/2}$, and it may be several times higher.

For effectively infinite asexual populations, the explicit definition of the equilibrium genetic variance needs to be modified. Letting $N_e \to \infty$ in the previous equation, the equilibrium level of genetic variance for an effectively infinite sexual population is found to be $\sigma_g^2 = \sigma_m^2/(2n_e)^{1/2}$. In the absence of sex, the genome functions as a single nonsegregating unit, so $2n_e = 1$, and $\sigma_g^2 = \sigma_m^2$ (Lynch et al., 1991).

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