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Adaptive and demographic responses of plankton populations to environmental change

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Abstract

Because of their large population sizes, short generation times, and clonal mode of propagation, microorganisms should often be the first members of a community to respond evolutionarily to temporal changes in the environment. Because the planktonic microbial community directly or indirectly influences all other members of aquatic ecosystems, it is useful to have a general theory for the magnitude and limits of such response. Models are presented for the expected dynamics of evolutionary change for the mean and variance of a quantitative character under natural selection toward a fixed or a moving optimum. It is also shown how the rate of population growth is related to the phenotypic composition of the population and the selective aspects of the environment. These models, which lead to the identification of extinction thresholds for the rate of environmental change beyond which a population cannot maintain itself, provide a heuristic basis for understanding the response of ecosystems to environmental perturbations. The analyses also indicate that clones of microorganisms isolated into novel laboratory environments are likely to undergo substantial evolutionary change over periods of a few hundred days, which raises questions about the utility of such cultures for inferring ecological properties of natural populations.

Models for the growth of natural microbial populations typically assume that the species involved are static evolutionary entities that do not adapt genetically to biotic or abiotic sources of environmental change. However, it is well known that relatively small populations confined to laboratory chemostats and initiated as clones can often evolve new variants in response to selective challenges. In nature, it is common for local population sizes of planktonic organisms to be in the neighborhood of 10^{12} or many orders of magnitude larger. The mutation rate per locus is on the order of 10^{-6} per generation and the characters upon which natural selection operates are often encoded by tens to hundreds of loci (Falconer 1981; Lande 1981), so the opportunities for fa-

vorable mutations are enormous in such populations. When the generation time is on the order of hours (bacteria) to days (algae and most zooplankton), such mutations can spread rapidly throughout a population by natural selection. Thus, it would be surprising if species inhabiting microbial communities such as the plankton did not exhibit evolutionary responses to annual cycles of the environment or to more long-term environmental change.

Our purpose here is to examine the extent to which genetic variation can influence the seasonal dynamics of large populations of microorganisms. We are also interested in the conditions required for population persistence in the face of a long-term environmental trend such as pollution or global warming. Because microorganisms play central roles in biogeochemical cycles that influence all higher level organisms either directly or indirectly, the questions we are asking also have relevance for issues concerning the stability and resilience of ecosystems. For reasons outlined in the preceding paragraph, it seems likely that the

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Notation

z	Phenotypic value; observed value of any character (e.g. cell size, thermal optimum, lipid content)
g	Genotypic value; expected phenotypic value of a given clone
e	Environmental deviation of z from g caused by nongenetic factors
$p(g)$	Probability distribution for genotypic values
$p(z g)$	Probability distribution for phenotypic values conditional on g
μ_g	Mean genotypic (and phenotypic) value in the population†
σ_g^2	Genotypic variance in the population
σ_e^2	Variance of environmental deviations
σ_z^2	Phenotypic variance in the population, equal to $\sigma_g^2 + \sigma_e^2$
σ_m^2	Mutational rate of input of new genetic variance
$\hat{\sigma}_g^2$	Genotypic variance in a population at selection-mutation equilibrium
r_z	Instantaneous rate of increase for individuals with phenotypic value z
r_g	Instantaneous rate of increase for clone with genotypic value g
r_m	Instantaneous rate of increase for individuals with the optimum phenotypic value θ
\bar{r}_m	Instantaneous rate of increase for a population with the optimal mean phenotypic value θ
r^*	Instantaneous rate of increase for individuals with the optimal genotypic value
\bar{r}	Instantaneous rate of increase for the population
θ	Optimum phenotypic value
σ_w	Width of the fitness function; inversely proportional to the strength of selection
α	Equal to σ_m/σ_w
P_t	Fractional deviation of the genetic variance at time t from the equilibrium expectation
Q	Fractional distance from the optimum phenotype traversed during time t
k	Rate of change of phenotypic optimum in a linearly varying environment; amplitude of the oscillation of the optimum in a periodic environment
$ k_c $	Critical rate of environmental change beyond which a population cannot evolve rapidly enough to avoid extinction
ω	Periodicity of a cycle in the phenotypic optimum
ζ	Equal to $\arctan(\omega/\alpha)$; phase displacement of the cycle in the mean phenotype from the optimum
t_{\min}, t_{\max}	Time in a cycle, starting at $t = 0$, when the population mean phenotype reaches a minimum or maximum

† Mean phenotypic values and mean genotypic values are equivalent because mean environmental deviations in the population are zero by definition.

microbial community will often be the first segment of an ecosystem to exhibit an evolutionary or demographic response to an environmental perturbation. These changes can be propagated as ecological effects to larger organisms which have not had sufficient time for genetic change.

The models that we introduce below are similar in spirit to those presented earlier for the evolutionary dynamics of a quantitative character in a parthenogenetic species with discrete generations (Lynch and Gabriel 1983). Here, however, the focus is on continuous-growth models without age structure. Such an approach provides reasonable approximations for short-lived organisms with overlapping generations. We will assume that the population size is sufficiently large that random genetic drift can be ignored and that the distribution of phenotypes is approximated closely by a continuous function. We also assume an asexual mode of reproduction—the most frequent situation in most planktonic organisms (bacteria, algae, fungi, protozoa, rotifers, and cladocera). This greatly simplifies genetic models by eliminating the need to explicitly incorporate complexities such as linkage and nonadditive gene action.

For purposes of exposition, we assume that individual fitness (and the population rate of increase) is determined by a single quantitative character (or a series of characters that can be combined in a simple linear fashion), which may be approximately true for a zooplankton population under intense size-selective predation, a phytoplankton population limited by light or a specific nutrient, or a bacterial population experiencing a substantial shift in a density-independent aspect of the environment. However, selection often operates simultaneously on a system of independent or partially correlated characters (Lande and Arnold 1983). Although the types of models that we introduce are readily generalized to incorporate multivariate selection (Lande 1979), it is not easy to implement such changes without a loss of clarity. We wish to avoid that, and such modifications would not alter our conclusions qualitatively. The symbols used here are summarized in the list of notation.

Gaussian selection

A relatively common form of natural selection is the case in which there is an intermediate optimum phenotype with fitness declining monotonically with the absolute deviation from the optimum (Johnson 1976; Endler 1986). Here we let z be a continuously distributed character, such as size, shape, thermal or pH optimum, upon which natural selection operates. The rate of increase takes on its maximum value when $z = \theta$, and it declines in a symmetrical manner with deviations to either side of the optimum. Because our focus is on continuously growing populations with overlapping generations, the appropriate measure of fitness is the instantaneous rate of increase. For an individual with phenotype z , it can be represented by

$$r_z = r_m - \frac{(z - \theta)^2}{2\sigma_w^2} \quad (1)$$

where θ is the optimum phenotype, σ_w the width of the fitness function, and r_m the rate of increase for the optimum phenotype. A negative value of r_z implies that individuals of phenotype z do not produce enough offspring to replace themselves. Note that as σ_w increases, the curvature of the fitness function, and hence the intensity of selection, declines. The parameters r_m , θ , and σ_w are all estimable properties of natural populations (Lande and Arnold 1983; Mitchell-Olds and Shaw 1987; Schluter 1988).

Quantitative characters are usually the products of multiple genetic loci and environmental effects; as such, they generally exhibit a continuous range of variation. The phenotypic value (z) of an individual is a directly observable property that can be broken into the sum of two components: a genotypic value (g), which is the expected phenotype of members of the clone, and an environmental deviation (e), which represents the deviation of an individual's phenotype from g caused by environmental effects, developmental noise, etc. In any given environment, environmental deviations are equally likely to increase or decrease the phenotypic value relative to its expectation. So for the population as a whole, the mean environmental effect is zero, and the mean

genotypic value is equal to the mean phenotypic value.

If the conditional phenotype distribution for the clone $p(z|g)$ is assumed to be measured on a scale that yields normality with mean g and environmental variance σ_e^2 , then the genotypic rate of increase is

$$r_g = \int p(z|g)r_z dz = r^* - \frac{(g - \theta)^2}{2\sigma_w^2} \quad (2)$$

Here $r^* = r_m - (\sigma_e^2/2\sigma_w^2)$ is the rate of increase of the optimum genotype. By producing nonoptimal phenotypes, environmental variance causes $r^* < r_m$.

If, in addition, the genotype distribution $p(g)$ is assumed to be normal with mean μ_g and variance σ_g^2 , then the population rate of increase is

$$\bar{r} = \int p(g)r_g dg = \bar{r}_m - \frac{(\mu_g - \theta)^2}{2\sigma_w^2} \quad (3)$$

Here $\bar{r}_m = r_m - (\sigma_z^2/2\sigma_w^2)$ is the population rate of increase when the mean phenotype is at the optimum, and $\sigma_z^2 = \sigma_g^2 + \sigma_e^2$ is the phenotypic variance. A positive rate of population growth requires that

$$\frac{r_m - \bar{r}}{r_m} = \frac{(\mu_g - \theta)^2 + \sigma_z^2}{2\sigma_w^2 r_m} < 1. \quad (4)$$

Thus, even if the mean phenotype is at the optimum, the population growth rate will be negative if the phenotypic variance is large relative to σ_w^2 . The quantity on the left of Eq. 4 can be thought of as a measure of the selective load on a population; it is a measure of the fractional reduction of the population rate of growth below the maximum possible r_m .

For an asexually reproducing population, the rate of change in clone frequency is a linear function of the deviation between the genotypic and population rate of growth,

$$\frac{dp(g)}{dt} = p(g)(r_g - \bar{r}) \quad (5)$$

(Crow and Kimura 1970). Noting that the

mean genotypic value in the population is $\mu_g = \int p(g)g \, dg$ and assuming no transmission of environmental effects across generations, we find that the rate of evolution of the mean phenotype resulting from natural selection is

$$\frac{d\mu_g}{dt} = \int g \frac{dp(g)}{dt} \, dg. \quad (6)$$

It can be shown that the fitness function given by Eq. 2 preserves a normal distribution of genotypic values, which reduces Eq. 6 to

$$\frac{d\mu_g}{dt} = \frac{\sigma_g^2(t)[\theta - \mu_g(t)]}{\sigma_w^2}. \quad (7)$$

Thus, the mean phenotype of the population always evolves toward the optimum, and it does so at a rate that is directly proportional to the genetic variance for the trait, the distance from the optimum, and the intensity of selection around the optimum.

Selection also influences the genetic variance. It can be shown that, provided the distribution of genotypic values remains normal, the rate of loss of genetic variance via selection is simply $-\sigma_g^4(t)/\sigma_w^2$ per unit time. Thus, in this model natural selection causes an erosion of genetic variance at a rate that depends only on σ_w^2 —not on the distance of the mean phenotype from the optimum (cf. Lande 1976; Lynch and Gabriel 1983).

Polygenic mutation (Lynch 1988) will result in the continuous introduction of new genetic variation at the rate σ_m^2 . We assume that the conditional distribution of mutational effects is normal with mean zero. The dynamics of genetic variance are then

$$\frac{d\sigma_g^2}{dt} = \sigma_m^2 - \frac{\sigma_g^4(t)}{\sigma_w^2}. \quad (8)$$

This equation shows that, provided σ_w^2 remains constant, the opposing forces of selection and mutation drive the genetic variance to an equilibrium level equal to

$$\hat{\sigma}_g^2 = \sigma_m \sigma_w. \quad (9)$$

Kimura (1965) obtained a parallel result, by

a different route, for single loci in sexual populations.

The solution to Eq. 8 is

$$\sigma_g^2(t) = \hat{\sigma}_g^2 + \left\{ \left[\frac{1}{\sigma_g^2(0) - \hat{\sigma}_g^2} + \frac{1}{2\hat{\sigma}_g^2} \right] \exp(2\alpha t) - \frac{1}{2\hat{\sigma}_g^2} \right\}^{-1} \quad (10)$$

where $\alpha = \sigma_m/\sigma_w$. If we let

$$P_t = \frac{\hat{\sigma}_g^2 - \sigma_g^2(t)}{\hat{\sigma}_g^2}$$

be the fractional deviation of the genetic variance at time t from the expected equilibrium, Eq. 10 implies that the time to pass from a starting condition P_0 to P_t is

$$t = \frac{1}{2\alpha} \ln \left[\frac{P_0(2 - P_t)}{P_t(2 - P_0)} \right], \quad (11a)$$

which reduces to

$$t = \frac{1}{2\alpha} \ln \left(\frac{2 - P_t}{P_t} \right) \quad (11b)$$

for the special case in which the initial genetic variance is zero (i.e. starting from a single clone so that $P_0 = 1$). For this extreme situation, it takes $0.55/\alpha$ and $1.83/\alpha$ time units to build to 50 and 95% of the equilibrium level of variance.

It is common laboratory procedure to establish pure clones of microorganisms from a single wild-caught individual and to utilize the derivative cultures in laboratory experiments for many years, often to make inferences about the ecological properties of the field population. An implicit assumption of this procedure is that the culture does not undergo significant evolutionary change subsequent to its establishment. However, since laboratory culture conditions often bear little resemblance to the natural environment (Roszak and Colwell 1987), it seems very likely that newly established cultures will be under strong selection for a change in the mean of one or more characters. Initially, the response to such selection will be negligible because the genetic

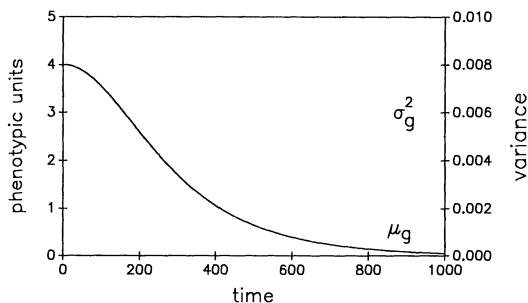


Fig. 1. Expected evolutionary dynamics for the mean and genetic variance of a quantitative character in a clonal population under selection toward a new optimum phenotype, starting with zero genetic variance and assuming that physiological acclimation is not occurring. The optimal phenotype is scaled to be $\theta = 0$, the initial mean is $\mu_g(0) = 4$, the mutational variance $\sigma_m^2 = 2.5 \times 10^{-5}$, and the width of the fitness function $\sigma_w = 1$.

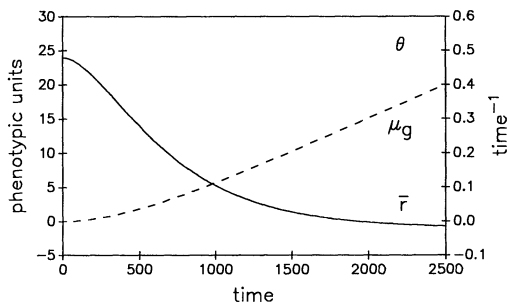


Fig. 2. The response of the mean phenotype (μ_g) and the population rate of increase (\bar{r}) to a long-term change in the optimum phenotype ($\theta = 0.01t$). μ_g and θ are given on the left scale in arbitrary phenotypic units; \bar{r} is given on the right in arbitrary time units. $\sigma_e = 1$, $\sigma_w = 5$, and $\sigma_m = 0.01$. The population starts out at the initial optimum with $\bar{r}_m = 0.5$, gradually lags behind the optimum until a steady state \bar{r} slightly less than zero is attained, and then declines to extinction.

variance will be near zero, but the introduction of adaptive variants via polygenic mutation will gradually allow evolutionary progress. The preceding equations can be used to estimate the magnitude of evolutionary change that can be expected to occur.

Setting $\sigma_g^2(0) = 0$ and assuming that the optimum phenotype remains stable at θ , we obtain the expected evolutionary dynamics by substituting Eq. 10 into 7 and solving:

$$\mu_g(t) = \theta + \frac{2[\mu_g(0) - \theta]\exp(\alpha t)}{1 + \exp(2\alpha t)}. \quad (12)$$

If we let

$$Q = \frac{\mu_g(0) - \mu_g(t)}{\mu_g(0) - \theta}$$

be the fraction of the initial distance from the optimum phenotype traversed, the expected time to reach Q is

$$t = \frac{1}{\alpha} \ln \left\{ \frac{1 + [Q(2 - Q)]^{1/2}}{1 - Q} \right\}, \quad (13)$$

which for $Q = 0.5$ and 0.95 reduces to $t = 1.32/\alpha$ and $3.69/\alpha$.

The preceding results show that the temporal dynamics of evolutionary change depend critically on α , the ratio of the square root of the mutational variance and the cur-

vature of the fitness function. If α is on the order of 10^{-2} – 10^{-3} d^{-1} , as suggested below, substantial evolutionary change can be expected in a few hundred days if the genotypic value of the colonizing individual is a good distance from the optimum (Fig. 1). Clear evidence of phenotypic evolution has been obtained in laboratory isolates of *Daphnia* maintained for such periods (Banta 1939), and suggestive evidence exists for a number of marine phytoplankters (Wood 1988). The literature also contains numerous anecdotal reports regarding the need for prolonged periods to stabilize the properties of laboratory cultures of microorganisms. Quantitative documentation of such phenomena is extremely rare, but Bomber et al. (1989) provided a detailed report on a gradual ~ 200 -d phase of doubling of the rate of cell division in laboratory isolates of the toxic dinoflagellate *Gambierdiscus toxicus*. They favor physiological acclimation as an explanation for their results, but the necessity of 30 cell divisions for such change seems remarkable, and the possibility that the change was genetic cannot be ruled out.

Response to a temporal change in the environment

Suppose now that the optimum phenotype is changing linearly in time at rate k so that

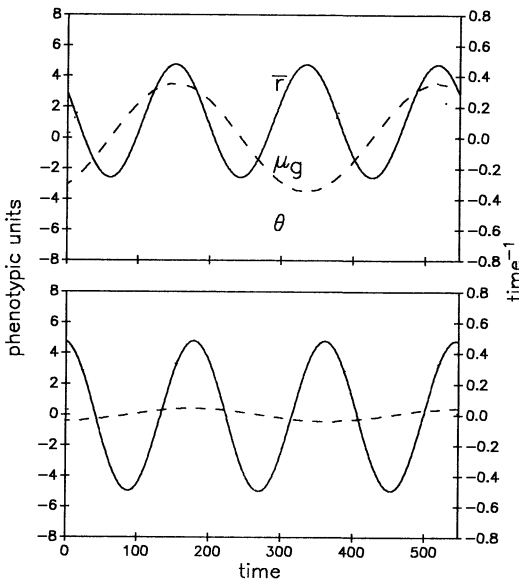


Fig. 3. Equilibrium cycles of evolutionary change in response to a cycle in the optimum phenotype. The mean and optimum phenotypes, μ_g and θ , are given in arbitrary units on the left, and the population rate of growth \bar{r} as d^{-1} on the right. In both examples, $k = 7$, $\omega = 2\pi/365$, $\bar{r}_m = 0.5$, $\sigma_w = 5$, and $\sigma_e = 1$. In the lower panel, the rate of polygenic mutation is low ($\sigma_m = 0.005$); consequently, the annual evolutionary change is very slight, and a large fraction of the year is spent in a decline phase. In the upper panel, $\sigma_m = 0.05$, the mean phenotype tracks the optimum more closely, and a smaller portion of the year is spent in a decline phase. Note the different mean phenotypes during the two annual phases of maximum growth.

$$\theta(t) = \theta(0) + kt, \quad (14)$$

but that the intensity of selection around the optimum remains constant. If we assume that the genetic variance has reached its equilibrium value, substituting Eq. 9 and 14 into Eq. 7 leads to the solution

$$\begin{aligned} \mu_g(t) = & [\mu_g(0) - \theta(0) + (k/\alpha)]\exp(-\alpha t) \\ & + [\theta(t) - (k/\alpha)]. \end{aligned} \quad (15)$$

Thus, as $t \rightarrow \infty$, historical effects due to starting conditions (given by the first term) die out, and the population mean phenotype settles into a steady state lag, $k\sigma_w/\sigma_m$, behind the moving optimum (Fig. 2).

Substituting the steady state lag for the deviation of the mean from the optimum ($\mu_g - \theta$) in Eq. 4, we obtain the extinction threshold

$$|k_c| = (2\bar{r}_m\sigma_m^2)^{1/2}. \quad (16)$$

Populations that are exposed to rates of environmental change in excess of this value for prolonged periods must decline toward zero. The extinction threshold increases with \bar{r}_m , the population growth rate when the mean phenotype is at the optimum, and with the rate of polygenic mutation σ_m^2 , which provides the genetic variance required for adaptive change.

Periodic environments

Most microbial populations experience regular temporal periodicity of important aspects of the environment (e.g. annual cycles of temperature, pH, selective predation, etc.). The selection resulting from such change can be represented by an optimum phenotype that is a sine function of time:

$$\theta(t) = k \sin(\omega t). \quad (17)$$

Here $k = (\theta_{\max} - \theta_{\min})/2$ is a measure of the amplitude of the oscillation, and ω is a measure of the periodicity of the cycle (equal to $2\pi/365$ for an annual cycle if t is measured in days). With this model, the average annual optimum is scaled to be equal to zero.

If we assume again that σ_g^2 remains stable in time at its expected equilibrium, the solution to Eq. 7 now becomes

$$\begin{aligned} \mu_g(t) = & \left[\mu_g(0) + \frac{\alpha\omega k}{\alpha^2 + \omega^2} \right] \exp(-\alpha t) \\ & + \frac{k \sin(\omega t - \zeta)}{[1 + (\omega/\alpha)^2]^{1/2}} \end{aligned} \quad (18)$$

with $\zeta = \arctan(\omega/\alpha)$. As in the linear model (Eq. 15), the first term in this equation, which depends on the starting conditions and decays at a rate of α per unit time, describes a transient contribution to the evolutionary dynamics of the mean phenotype. The second term describes the equilibrium cycle for the change in mean phenotype into which the population eventually settles.

When the equilibrium evolutionary dynamics have been attained, the mean phenotype will cycle with the same periodicity as the environment but out of phase with it by the amount ζ (Fig. 3). The time of appearance of the maximum mean phenotype

always lags behind the time at which θ_{\max} occurs. This lag increases with decreasing α , i.e. with a decreasing rate of polygenic mutation or a decrease in the intensity of selection. Thus, in an annual cycle, the mean of a character is expected to exhibit a phase of evolutionary increase during the initial phase of the decline in the optimum (and vice versa for the initial phase of increase in the optimum). This phase shift occurs because the extreme values of μ_g are always less than the extreme values of θ .

The implication of these observations is that the mean phenotypes for a population in a cyclical environment should be less strongly correlated with immediate environmental conditions than with those at some time in the past. Within a cycle (starting at time zero), the population mean phenotype reaches a minimum at time

$$t_{\min} = \frac{2\pi + \arctan(-\alpha/\omega)}{\omega} \quad (19a)$$

and a maximum at

$$t_{\max} = t_{\min} - \frac{\pi}{\omega}. \quad (19b)$$

These points represent the two times per cycle in which the population mean phenotype coincides with the optimum phenotype. The population attains its maximum rate of increase at each of these times, but the optimal (and observed) phenotypic means during the two periods are expected to differ to a degree that increases with increasing α (Fig. 3).

The second term in Eq. 18 shows that the amplitude of the annual evolutionary cycle of the mean phenotype is a fraction $[1 + (\omega/\alpha)^2]^{-1/2}$ of the amplitude of the environment. Thus, the amplitude of evolutionary change increases from zero when α is small to the amplitude of the cycle of optimum phenotypes as α becomes very large. In the latter case, the population mean tracks the optimum because of the frequent production of adaptive variants (high σ_m), or because of strong selection (low σ_w), or both.

Equations 17 and 18 can be used in conjunction with Eq. 3 to evaluate the periods during a cycle for which population growth rate is positive. The degree to which \bar{r} varies

throughout a cycle depends on the extent to which the population is capable of tracking the shifting optimum. Pronounced pulses of productivity are expected in populations that are incapable of adapting to the seasonal change.

However, even if the population does periodically experience positive growth, long-term persistence requires that

$$\int_0^{2\pi/\omega} \bar{r}(t) dt \geq 0.$$

For a population in selection-mutation balance, this is equivalent to

$$|k_c| < 2\sigma_w \{\bar{r}_m [1 + (\alpha/\omega)^2]\}^{1/2} \quad (20)$$

where k_c again represents an extinction threshold. If the amplitude of environmental change exceeds this value, persistence will not be possible because the short periods of positive growth will be offset by long periods of population decline.

Discussion

For both linear and periodic trends in the environment, our results suggest that the ratio $\alpha = \sigma_m/\sigma_w$ is of fundamental importance for describing the dynamics of phenotypic evolution in a large population of asexual organisms. A doubling of σ_m has the same effect on the equilibrium evolutionary trajectory as a halving of σ_w . In principle, σ_w can be measured for a character under stabilizing selection by monitoring the fitness of different phenotypes and obtaining maximum-likelihood (or similar) estimates of the parameters of Eq. 1. This type of analysis has been applied to many terrestrial organisms, but we are unaware of any attempts to measure the width of the fitness function directly for any quantitative trait in a natural microbial population. Moreover, members of the plankton cannot be easily marked in the field, so it is unlikely that direct estimates of the intensity of selection (σ_w) will be forthcoming.

Thus, it is worth noting that α is equivalent to $\sigma_m^2/\hat{\sigma}_g^2$. Both components of this expression can be estimated in the laboratory by ANOVA (Lynch 1984a; Wood et al. 1987)—the numerator from a collection of asexual sublines that have been allowed

to diverge by spontaneous mutation, the denominator from a collection of clones extracted from the field. If the experimental design controls for maternal effects (Lynch 1984a), the between-line component of variance provides a clean estimate of genetic variance. In the case of a mutation-accumulation experiment, this must be divided by time to provide an estimate of σ_m^2 . On the basis of this and similar approaches, estimates of the rate of polygenic mutation have become available for numerous characters in a variety of species of higher plants and animals (Lynch 1988). On a per generation basis, these estimates range from 10^{-4} to $10^{-2} \sigma_e^2$. Such values need to be scaled appropriately to be compatible with our continuous time model.

To provide a rough idea of the order of magnitude that α is likely to be, we consider some data from the only planktonic organism that has been analyzed extensively from a quantitative genetic perspective—the freshwater cladoceran *Daphnia pulex*. Estimates of the rate of polygenic mutation are available for several life-history characters (Lynch 1984a). If we assume a generation time of ~ 10 d,

$$\sigma_m^2/\sigma_e^2 \approx 2 \times 10^{-4} \text{ d}^{-1}.$$

For the same characters, Lynch (1984b) used a selection analysis to infer indirectly from a field study that the equilibrium level of heritability ($\hat{\sigma}_g^2/\hat{\sigma}_z^2$) under asexual reproduction is ~ 0.07 . Direct heritability estimates from another natural population reproducing by obligate parthenogenesis average ~ 0.03 (Lynch et al. 1989). For such small values, the expected heritability is essentially $\sigma_m\sigma_w/\sigma_e^2$. Taking 0.05 to be an estimate of this quantity and dividing into 2×10^{-4} , we obtain $\alpha = \sigma_m/\sigma_w \approx 4 \times 10^{-3} \text{ d}^{-1}$, which is intermediate to the values utilized in Fig. 3.

Thus, the *Daphnia* data are consistent with the hypothesis that clonally reproducing microorganisms are capable of significant phenotypic evolution in the face of a temporally changing environment. It is clear, however, that data on α for other characters and other species are required before any general conclusions can be drawn. It must also be emphasized that laboratory esti-

mates of variance components may provide a biased picture of the field situation if there is significant genotype \times environment interaction (Riska et al. 1989).

Other indirect methods for estimating α can be imagined. Consider the case, for example, when a population is seen to be evolving toward an identifiable optimum phenotype and accurate estimates of the population mean phenotype are available t time units apart. If it can be reasonably assumed that the optimum has been stable throughout the period of observation and that the genetic variance has remained at its equilibrium value, then rearrangement of Eq. 15 with $k = 0$ leads to

$$\alpha = -\frac{1}{t} \ln(1 - Q) \quad (21)$$

where Q has been defined above. Following the same logic, it should be possible to estimate α for newly established laboratory cultures with

$$\alpha = \frac{1}{t} \ln \left\{ \frac{1 + [Q(2 - Q)]^{1/2}}{1 - Q} \right\}. \quad (22)$$

As an example of the application of the latter formula, we provide a rough calculation from the data of Bomber et al. (1989). They observed that the average toxicity of cell cultures increased from ~ 2.5 units in new isolates toward a steady state level of ~ 20.0 units. About half the distance between these two points had been traversed by 150 d. For $Q = 0.5$, Eq. 22 simplifies to $\alpha = 1.32/t$. Thus, if we assume that the cultures were really evolving and not changing their mean phenotype by physiological adjustment,

$$\alpha \approx 9 \times 10^{-3} \text{ d}^{-1}.$$

As noted above, difficulties arise in interpreting experiments such as that of Bomber et al. (1989). Are long-term trends in the phenotypes of laboratory cultures a result of physiological acclimation or of genetic change? There is a simple way to evaluate this problem empirically. If the change is due to acclimation to the laboratory environment, then the same response should be seen in very large cultures as in very small ones (e.g. those maintained by single-cell

descent), provided they are raised under the same conditions. If the change is genetic, it should not be observed in very tiny populations, which would not provide sufficient opportunities for beneficial mutations.

The models presented above assume that at any point in time there is a single intermediate optimum phenotype toward which the population is evolving. Such stabilizing selection appears to be the norm for many characters in natural populations (Endler 1986), but there are many alternatives to the quadratic fitness function that yield an intermediate optimum. Consider, for example, the situation in which the predominant selective pressure is on the uptake ability for a critical nutrient, which would lead to a hyperbolic relationship between rate of increase and nutrient concentration. Such a function can often be characterized by two parameters: r_{\max} , the maximum rate of growth which occurs at high nutrient concentration, and K_s , the half-saturation concentration (i.e. the nutrient concentration at which the growth rate is equal to $r_{\max}/2$). Suppose now that z is an underlying property of the phenotype that jointly determines the maximum growth rate and the half-saturation concentration. Specifically, let there be a tradeoff such that phenotypes that grow relatively rapidly in enriched environments grow relatively slowly at low nutrient levels. Such a functional relationship will lead to an optimal value of z which depends on the nutrient concentration. Thus, unless the genotypic values for the maximum growth rate and the half-saturation constant are independent, selection for nutrient uptake kinetics can also be viewed as a form of stabilizing selection.

We have assumed that the conditional genotype distribution of the progeny of an individual is Gaussian with a mean equal to the genotypic value of the parent and with a small variance $T\sigma_m^2$, where T is the generation time, which is equivalent to assuming that a vanishingly small fraction of progeny are nonmutants for the character under consideration but that most mutants are also essentially indistinguishable from their parent. The potential for evolutionary change in microbial populations has been documented repeatedly in laboratory experi-

ments, but almost all existing work focuses on mutations with major phenotypic effects, as in studies of the evolution of new biochemical functions (Hall 1983). The evidence is strong, however, that the vast majority of viable mutations in higher organisms have very small effects that are indiscernible on an individual basis (Lynch 1988). There is no compelling reason to think that the situation is different in microorganisms. Even the bacteria (with their constituent plasmids and mobile genetic elements) have a large array of mutational mechanisms with structural effects ranging from single nucleotide replacement to insertion, deletion, or rearrangement of entire blocks of DNA (Terzaghi and O'Hara 1990). Probably no procaryotic or eucaryotic genome is ever replicated without some error. Thus, for quantitative characters such as size, shape, development time, physiological rates, etc., which may be influenced by a large portion of the genome, our assumption of a continuum of mutational effects seems reasonable.

Only a few attempts have been made to document the existence of quantitative genetic variation in natural populations of microorganisms, but nearly all such attempts seem to have been successful. The existing studies on *Daphnia* have been mentioned above. King (1972, 1977) has documented interclonal variation of fitness parameters in planktonic populations of rotifers. The evidence for population genetic variation in marine phytoplankton has been reviewed by Wood (1988). Brand et al. (1981) summarized evidence for interclonal variation in the marine diatom *Thalassiosira pseudonana* for nutrient uptake abilities, sensitivity to exotic chemicals, and cell division rates. Wood et al. (1987) demonstrated that ~20% of the morphological variation in a collection of *Thalassiosira tumida* from the Weddell Sea was due to genetic causes. Significant differences in cell potencies and growth rates exist among clones of *G. toxicus* (Bomber et al. 1989). Natural populations of *Escherichia coli* and *Salmonella typhimurium* are known to contain variants for numerous biochemical-physiological properties (Neidhardt et al. 1987). In all of these cases, the phenotypic variants appear

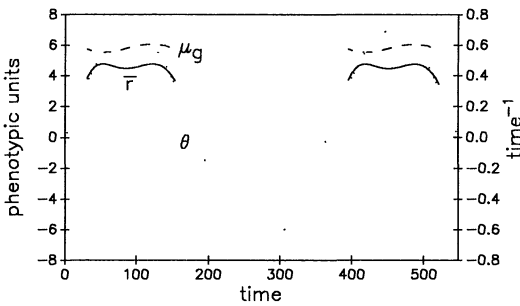


Fig. 4. Equilibrium cycles of evolutionary change for a regularly diapausing species exposed to a periodic environment. The parameters are the same as in the upper panel of Fig. 3.

to be distributed along a continuum rather than in discrete classes, which is consistent with the assumptions we have made above.

We have not explicitly incorporated density dependence into our formulations. Such effects are likely to be important in most populations but, assuming that they modify r_g of all genotypes in the same fashion, they will not influence the evolutionary dynamics of the mean phenotype; only the population dynamics would be affected. The same is true of density-independent factors that influence the population in a genotype-independent manner.

Many species may avoid times of anticipated population decline through the production of diapausing resting stages. Such behavior is counterproductive when there is a long-term trend in the environment because the absence of evolution during the resting stage can only cause the mean phenotype to lag further behind the moving optimum. But in a predictably periodic environment, a contracted phase of active population growth can greatly enhance the range of environments within which a species can survive. The consequences of this type of life-history strategy can be studied by numerical methods with the formulae given above. As in the case of a continuously growing population, a regularly diapausing population eventually settles into an equilibrium evolutionary trajectory determined by the rate of polygenic mutation, the width of the fitness function, the amplitude of the environmental fluctuations, and the portion of the environmental cycle

within which the population is active. In Fig. 4, for example, the population appears for a brief period of time straddling the peak in the optimum phenotype dynamics, which results in a relatively weak annual cycle of evolutionary change. In principle, populations can also avoid the selective load resulting from a periodic environment, without going through a diapause phase, by the use of phenotypic plasticity for the character under selection. Incorporation of a phenotypic response function into the preceding models should also be relatively straightforward.

Many microbial species have a capacity for periodic sexual reproduction. Elsewhere (Lynch and Gabriel 1983) we have shown that such a life cycle enables a population to respond very rapidly to a selective challenge immediately following the sexual generation. At that time, recombination is expected to release substantial amounts of genetic variation for quantitative characters that have been under selection previously. Clonal selection then results in the efficient erosion of expressed genetic variance down toward the expectation given in Eq. 9, while hidden genetic variance accumulates via the buildup of linkage disequilibrium. The cycle is then repeated following the next phase of sexual reproduction. The general dynamic models that we have presented are still valid for such populations, provided the flush of genetic variance at the onset of each period of clonal reproduction is properly accounted for.

Our approach to estimating thresholds for environmental conditions beyond which a population cannot be maintained is similar in spirit to models that have been developed in other contexts. For example, Skellam (1951) and Kierstead and Slobodkin (1953) considered the critical patch size below which the productivity of a plankton population is overwhelmed by dispersal across the perimeter of the patch. Lande (1987) developed a model for the degree of habitat fragmentation above which a mobile territorial species would be unable to sustain itself. Pease et al. (1989) considered the critical rate at which the movement of an environmental cline across a landscape can overwhelm the ability of a population to

disperse and adapt genetically. These types of models are of use in identifying general properties of the environment which if exceeded lead to a discontinuity in the structure of a community.

Our results provide some rough guidance as to the rates of environmental change that an asexual population can sustain for an extended period of time. For simplicity, consider a population with a maximum rate of increase for the optimal genotype of 0.5 d^{-1} . Then, from Eq. 16, a daily rate of environmental change that exceeds the square root of the daily rate of input of mutational variance for the selected character will cause the population to steadily decline to extinction. Accepting the above observation that $\alpha \ll \omega$, Eq. 20 implies that the critical amplitude of change in the optimum phenotype in a periodic environment is essentially independent of mutational variance. The extinction threshold is $\sim 2\sigma_w(\bar{r}_m)^{1/2}$. Thus, for our example, an amplitude of annual fluctuation for the optimum of a selected character that exceeded 1.4 times the width of the fitness function would be sufficient to exclude a population.

These types of results may be of some use in assessing the fragility of microbial communities to prolonged periods of environmental change. They may also provide some insight as to how seasonality can result in the permanent exclusion of species from environments that appear to periodically provide ideal growth conditions.

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