

EVOLUTION IN A VARIABLE ENVIRONMENT

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Over the past four decades, several authors have stressed that it is not just good performance, on the average, that matters in evolution but that variation in performance also plays an important role in determining long-term evolutionary trends. Several independent lines of research have arisen from considering different kinds of variation. Dempster (1955) introduced a model in which temporal fluctuations in reproductive success for competing genotypes favor the genotype with the highest geometric-mean reproductive success. Levene (1953) studied a case in which the relative success of alleles varies spatially within a generation. These two papers established the dichotomy between temporal and spatial variation that most authors continue to use. Gillespie (1974a) considered still another kind of variation, variation in the reproductive success of each individual, in which the amount of variation depends on factors such as developmental homeostasis. Gillespie's (1974a) model has led to extensive discussion of the idea of evolutionary "bet hedging" (Slatkin 1974; Seger and Brockmann 1987).

In still another lineage of models, several authors have considered the problem of how variability in resource acquisition by different individuals affects reproductive success (Caraco 1980; Real 1980; Rubenstein 1982). These models suggest that individuals tend to avoid behavioral strategies that lead to variation in resource acquisition and have led to discussions of "risk aversion" (e.g., Stephens and Krebs 1986).

In this paper we develop a framework for analyzing these different types of variation. The key to our approach is the partitioning of the variance of reproductive success of a genotype into parts attributable to the variance of individual reproductive success and parts attributable to correlations in reproductive success among individuals. We use our approach to show the simple relationships among the models mentioned above, which have previously been treated in different ways. We generalize these models to include any correlation structure in the reproductive success among individuals of the same and different genotypes. We also apply our method to two examples: a model of developmental homeostasis, and a model of competition between semelparity and iteroparity when there is variable resource acquisition. Finally, we discuss some generalizations that

emerge from considering these models together, in particular, the geometric-mean principle and ideas of bet hedging and risk aversion.

THE GENERAL MODEL

In this section we develop the main equations for describing gene-frequency change. We first consider haploid individuals carrying alleles A_1 and A_2 at the locus of interest, with initial frequencies q_1 and q_2 . We define the reproductive success of an individual as the total number of offspring produced by that individual. The average genotypic reproductive successes of A_1 and A_2 and the average population reproductive success are, respectively,

$$\begin{aligned} R_1 &= \frac{1}{Nq_1} \sum_{i=1}^{Nq_1} (\mu_1 + \alpha_{1i}) = \mu_1 + \bar{\alpha}_1, \\ R_2 &= \frac{1}{Nq_2} \sum_{j=1}^{Nq_2} (\mu_2 + \alpha_{2j}) = \mu_2 + \bar{\alpha}_2, \\ \bar{R} &= q_1 R_1 + q_2 R_2, \end{aligned} \quad (1)$$

where N is population size, μ_1 and μ_2 are the expected reproductive successes of A_1 and A_2 individuals, and the α_{1i} and α_{2j} are the random deviations from the expectations for particular individuals. The α_{1i} and α_{2j} have zero expectations and variances of σ_1^2 and σ_2^2 . We assume throughout that σ_1^2 and σ_2^2 are of the same order of magnitude as $\mu_1 - \mu_2$ and that both are small relative to $\bar{\mu} = q_1\mu_1 + q_2\mu_2$. Under these assumptions, we can set $\bar{\mu} = 1$ without loss of generality.

The frequency of A_1 after one time period is

$$q'_1 = q_1(R_1/\bar{R}) = q_1F_1, \quad (2)$$

where we define F_1 as the relative fitness of A_1 . Because reproductive success is a random variable, frequency changes are also random variables. The change in the frequency of the A_1 type over one time interval is $\Delta q_1 = q'_1 - q_1$. The expected change in gene frequency is

$$E(\Delta q_1) = E(q'_1) - q_1 = E(q_1 R_1 / \bar{R}) - q_1. \quad (3)$$

One feature of equation (3) is worth noting here. The right-hand side contains the expectation of the ratio of two random variables, and the denominator \bar{R} depends on q_1 , so that the dependence of $E(\Delta q_1)$ on q_1 may be much more complicated than is suggested by equation (2). In particular, even if R_1 and R_2 are independent of q_1 , $E(\Delta q_1)$ induces the action of frequency-dependent selection on A_1 when the R 's are subject to stochastic variation. We discuss the significance of this observation later.

We cannot analyze equation (3) under arbitrary assumptions about reproductive success because the right-hand side includes the expectation of a ratio of two random variables for which no general formula is available. We proceed as above by assuming that differences in reproductive success between individuals are small relative to average reproductive success in the population, expanding the

denominator of equation (3) in a Taylor series in $\bar{\alpha}_1$ and $\bar{\alpha}_2$, and keeping only the first two terms:

$$E(\Delta q_1) \approx q_1 q_2 \{(\mu_1 - \mu_2) + [\text{cov}(R_2, \bar{R}) - \text{cov}(R_1, \bar{R})]\}. \quad (4)$$

Equation (4) is equivalent to Gillespie's (1977) approximation.

To predict the long-term gene-frequency changes, we need the variance of Δq_1 , $\text{var}(\Delta q_1)$. If $E(\Delta q_1)$ and $\text{var}(\Delta q_1)$ are of the same order of magnitude, then by diffusion theory we can describe the directional tendency of gene-frequency change by the ratio $E(\Delta q_1)/\text{var}(\Delta q_1)$ (Ewens 1979, chap. 4; Karlin and Taylor 1981, chap. 15). From equation (2) we can obtain, using the same type of approximation as in equation (4),

$$\text{var}(\Delta q_1) \approx q_1^2 q_2^2 \text{var}(R_1 - R_2). \quad (5)$$

Equations (4) and (5) depend on variances and covariances in average genotypic reproductive successes R_1 and R_2 and can be used to translate measures of reproductive success into relative genotypic fitnesses. We can relate the variance of *individual* reproductive success to the variance and covariance of average *genotypic* reproductive success by noting, from equation (1), that

$$\begin{aligned} \text{var}(R_1) &= \rho_1 \sigma_1^2, \\ \text{var}(R_2) &= \rho_2 \sigma_2^2, \end{aligned} \quad (6)$$

$$\text{cov}(R_1, R_2) = \rho_{12} \sigma_1 \sigma_2,$$

where ρ_1 , ρ_2 , and ρ_{12} are the correlations in reproductive success between randomly chosen pairs of A_1 , A_2 , or A_1 and A_2 individuals, respectively.

By substituting equations (1) and (6) into equations (4) and (5), we obtain

$$E(\Delta q_1) \approx q_1 q_2 \{(\mu_1 - \mu_2) + [q_2 \rho_2 \sigma_2^2 - q_1 \rho_1 \sigma_1^2 + (q_1 - q_2) \rho_{12} \sigma_1 \sigma_2]\}, \quad (7)$$

$$\text{var}(\Delta q_1) \approx q_1^2 q_2^2 (\rho_1 \sigma_1^2 + \rho_2 \sigma_2^2 - 2 \rho_{12} \sigma_1 \sigma_2). \quad (8)$$

Gillespie (1974*b*) and Karlin and Taylor (1981, chap. 15) presented results that are of a form similar to equations (7) and (8), but they did not partition genotypic variance into individual-level variations, σ^2 , and the correlations among individuals, ρ , as described in equation (6). This partitioning is the key to our analysis because the interactions between each individual and the environment and the interactions among individuals are the biological determinants of the genotypic variances.

TYPES OF VARIATIONS

Types of biological variability experienced by populations have usually been divided into distinct categories, such as developmental or within-individual fluctuations, temporal environmental changes, spatial variability among distinct patches, and variable rates of resource acquisition for particular behavioral strategies or physiological pathways. Each of these categories has been modeled in a different way, and each has a separate literature. We show that all of these

categories can be cast in the framework presented in the preceding section and that our method of partitioning genotypic variance into individual variance and correlations among individuals provides a simple and natural way of generalizing previous models.

Gillespie's Model of Individual Variation

Gillespie (1974a) introduced a model in which the reproductive success of each of the N haploid individuals in the population depends on its interactions with the environment during development. The reproductive successes of different individuals are independent because, by Gillespie's assumptions, different individuals experience different conditions and develop in an uncorrelated way. Nevertheless, the finite population size ensures that an individual's reproductive success correlates with the average reproductive success of its genotype. The correlation of two randomly chosen A_1 's is $\rho_1 = 1/(Nq_1)$, because there are Nq_1 individuals of type A_1 , and hence a chance $1/(Nq_1)$ of choosing the same individual twice. By the same reasoning, $\rho_2 = 1/(Nq_2)$. Clearly, $\rho_{12} = 0$ because different individuals experience different conditions. Substituting these values into equation (7), we find that $E(\Delta q_1) > 0$ for any gene frequency when

$$\mu_1 - \sigma_1^2/N > \mu_2 - \sigma_2^2/N. \quad (9)$$

Since this condition no longer depends on q , it is sufficient to describe long-term evolutionary advantage without the need to consider polymorphism and frequency dependence. Here, an allele with a *long-term advantage* is more likely to become fixed than a neutral allele with the same initial gene frequency.

With our approach, we can easily generalize Gillespie's result. In equation (7), if $\rho_{12} = 0$, then $E(\Delta q_1) > 0$ when $\mu_1 - q_1\rho_1\sigma_1^2 > \mu_2 - q_2\rho_2\sigma_2^2$. Since this condition depends on q_1 and q_2 , the direction of expected gene-frequency change may switch according to current gene frequency. However, if $\rho_1 = k_1/q_1$ and $\rho_2 = k_2/q_2$, where the k 's are independent of the q 's, then the condition for an expected increase in A_1 is

$$\mu_1 - k_1\sigma_1^2 > \mu_2 - k_2\sigma_2^2. \quad (10)$$

Under Gillespie's assumptions, $k_1 = k_2 = 1/N$. Our generalization allows for correlations among individuals caused, for example, by developing under similar environmental conditions.

In the Appendix, we show that equation (10) describes a condition for long-term advantage and therefore includes Gillespie's result in equation (9) as a special case. Below, we apply equation (10) to show how this result can be used to make specific predictions.

Temporal and Spatial Variation under Global Population Regulation

Dempster (1955) studied a model in which the success of an allele varies from one generation to the next in response to the environment, but within each generation, all alleles of the same type have identical success. Variation occurs among generations (temporal) rather than within generations (spatial). For a

haploid model, Dempster concluded that the allele with the highest geometric-mean success would prevail. Several other workers have analyzed more-general models of temporal variation in selection intensities (e.g., Kimura 1954; Haldane and Jayakar 1962; Gillespie 1973, 1978; Hartl and Cooke 1973; Karlin and Liberman 1974).

In Dempster's model, all alleles of the same type have identical reproductive success within a generation, $\rho_1 = \rho_2 = 1$, and by assumption, $\rho_{12} = 0$. The expected change in allele frequency is therefore (see eq. [7])

$$E(\Delta q_1) \approx q_1 q_2 [(\mu_1 - q_1 \sigma_1^2) - (\mu_2 - q_2 \sigma_2^2)]. \quad (11)$$

This equation illustrates an important general feature of evolution in variable environments, namely, that rare types have an inherent advantage when there is global regulation of population density. This apparent frequency dependence is an example of the phenomenon that we mentioned just below equation (3): taking the expectation of the ratio of two random variables, both of which depend on q_1 , can induce frequency dependence even if the reproductive successes are not frequency-dependent in each generation. To understand the frequency dependence in this particular example, suppose q_1 is small. In equation (4), $\text{cov}(R_1, \bar{R})$ is relatively small compared with $\text{cov}(R_2, \bar{R})$; hence, A_1 has an advantage. The opposite is true when A_2 is rare. A graphic description of rare-type advantage is shown in figure 1.

In spite of the inherent rare-type advantage, polymorphism is not maintained in this model (Gillespie 1973; Hartl and Cook 1973; Karlin and Liberman 1974) because of the high variance of gene-frequency change (see eq. [8]). When one allele is very rare, reproductive success and relative fitness are linearly related (fig. 1*b*), and thus, geometric-mean reproductive success determines the course of gene-frequency evolution (Gillespie 1973; Hartl and Cook 1973; Karlin and Liberman 1974; diploidy and multiple alleles have been treated in several papers, including Gillespie 1974*b*, 1978, 1980; Levikson and Karlin 1975; Turelli and Gillespie 1980; Turelli 1981).

We can generalize the Dempster model by allowing variation in reproductive success among individuals of the same genotype, which in effect allows for some spatial variation. This type of spatial variation differs from models of spatial variation considered in the following section. Here, we are assuming global regulation of the population. In the following section, we consider the Levene model and generalizations of it, in which regulation of population size occurs in each patch. We represent the effects of spatial variation by allowing ρ_1 and ρ_2 to be less than one, which reduces the variance of average genotypic success. The general condition for the A_1 's to increase can be expressed by comparing geometric-mean reproductive successes:

$$\mu_1 - \rho_1 \sigma_1^2 / 2 > \mu_2 - \rho_2 \sigma_2^2 / 2. \quad (12)$$

This condition applies for any haploid model in which the average correlation in reproductive success among individuals does not depend on gene frequency, population regulation is global, and the correlation between types ρ_{12} is zero.

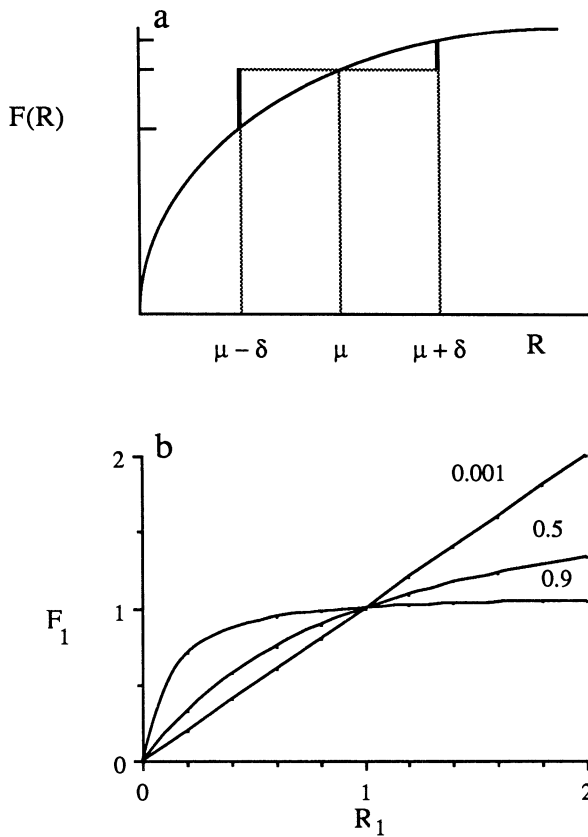


FIG. 1.—*a*, Increasing variation causes a decrease in expected value when there are diminishing returns. Here, average reproductive success, R , is μ , but deviations of $\pm \delta$ occur, with increases and decreases at equal frequencies. The gain in relative fitness, F , for an increase of δ units of reproductive success is less than the corresponding loss in fitness when reproductive success is $\mu - \delta$. Average fitness therefore declines as the frequency and magnitude of deviations increase. Note that the discount to fitness depends on the curvature of the relationship of fitness and reproductive success. *b*, The curvature of fitness versus reproductive success depends on the correlation between genotypic reproductive success and population reproductive success. The numbers above each curve represent different strengths of this correlation. If two haploid genotypes are uncorrelated, then, because $\bar{R} = q_1 R_1 + q_2 R_2$, the correlation between genotypic reproductive success and population success, ρ , is the frequency of the genotype. Note that there is little curvature when a genotype is rare, which explains the rare-type advantage discussed in the text.

Spatial Variation and Local Population Regulation

Levene (1953) assumed that the environment is divided into a large number of patches and that competition for limited reproductive opportunities occurs locally and independently within each patch. In a haploid model, one allele is favored in some patches, and the alternative allele is favored in others. Unlike models of global population regulation, the Levene model does maintain polymorphism.

Gillespie (1974*b*, 1978) has presented a diffusion analysis in which the number of locally regulated patches increases from one (Dempster) to infinity (Levene). The results in this section are not new, but our method for deriving them is. The advantage of our method is that it shows simply the relationship between this model of spatial variation and other models of spatial and temporal variation.

As in Gillespie's (1974*b*, 1978) models, we combine temporal and spatial variation with mode of population regulation. Let there be K independent patches. After each generation, each patch contributes the same fixed number of propagules (adults) to a global pool; then propagules recolonize the patches from this global pool. The number of successful colonists is large enough that the variance of gene frequency among patches is negligible. Within each patch, there is no variance of success among alleles of the same type; put another way, within patches, $\rho_1 = \rho_2 = 1$ and $\rho_{12} = 0$. Let σ_1^2 and σ_2^2 be the variances of the reproductive successes of A_1 's and A_2 's chosen randomly from different patches.

To carry out our analysis, we have to modify equation (3) to account for the regulation of population size in each patch. The local regulation of population size induces frequency-dependent interactions at the level of each patch for the same reasons discussed above. If local regulation occurs in each of K patches, and each patch contributes equally to the global population, then

$$E(\Delta q_1) = \left[\frac{1}{K} \sum_k E(q_1^{(k)} R_1^{(k)} / \bar{R}^{(k)}) \right] - q_1, \quad (13)$$

where $R_1^{(k)}$ and $\bar{R}^{(k)}$ are the average reproductive successes, respectively, of A_1 individuals and of all individuals in each patch.

The relationship between the Levene model of spatial variation and the Dempster model of temporal variation can be better appreciated by formulating the Levene model as a model of temporal variation. As noted by Gillespie (1974*a*, 1978), the Levene model of K patches, in which A_1 is favored in pK patches and A_2 is favored in $(1 - p)K$ patches, can be regarded as a model of K patches in which each patch has a probability p in each generation that A_1 will be favored and a probability of $1 - p$ that A_2 will be favored. With that view, the principal difference between the Levene model and the Dempster model is whether population size is regulated globally or locally.

If the deterministic component of success does not dominate (the μ 's), then rare types have an intrinsic advantage. Since, in the standard Dempster model, this rare-type advantage is not sufficient to overcome the large variance of gene-frequency change, no polymorphism is maintained. In the present case, because the process is averaged over K patches, the variance of gene-frequency change is the variance of the average change over all patches, or $1/K$ multiplied by the variance within each patch. The variance of gene-frequency change declines as K increases, which allows the rare-type advantage to come into play and maintain polymorphism. Because Levene's model assumes in effect that K is large, the rare-type advantage is deterministic. Intermediate values of K have been studied by Gillespie (1974*b*).

As K becomes large, the change in gene frequency given in equation (7) becomes essentially a deterministic process (a random Levene model in Gillespie

and Langley 1976). We can therefore solve for the equilibrium by setting $E(\Delta q_1) = 0$, which yields

$$q_1/q_2 = (\mu_1 - \mu_2 + \rho_2\sigma_2^2)/(\mu_2 - \mu_1 + \rho_1\sigma_1^2), \quad (14)$$

where the ρ 's, which are within-group correlations in this equation, describe the amount of spatial variation in reproductive success within a locally regulated patch. This equation can also be obtained as an asymptotic result from Gillespie's (1974*b*) formula for the stationary density of gene frequency by allowing his measure of patchiness to become large.

Diploidy

Thus far we have considered only haploid individuals. In this subsection, we consider diploid individuals and illustrate our method with a result for an N -patch model. We treat diploid individuals as patches that are cohabited by haploid genotypes, thus generating particular patterns of correlations among the haploid genotypes.

Equation (4) for $E(\Delta q_1)$ still applies, but we need new expressions for R_1 , R_2 , and \bar{R} . First, for the deterministic aspect of selection, dominance relationships can be treated in the usual way: replace the reproductive-success differential $\mu_1 - \mu_2$ with $(\mu_1 - \mu_2)[2q_2 + 2h(q_1 - q_2)]$, where h is a parameter for the degree of dominance. Second, the random components of reproductive success need to be recalculated in order to obtain the covariance terms. If we let the random effects of each allele be additive—for example, if α_1 and α_2 are the random effects of A_1 and A_2 alleles such that an A_1A_2 heterozygote has a random reproductive-success component $\alpha_1 + \alpha_2$ —then the random components $\delta(\)$ for reproductive-success averages for A_1 's, A_2 's, and the population as a whole are

$$\delta(R_1) \approx q_1\bar{\alpha}_1^{(\text{hom})} + q_2(\bar{\alpha}_1^{(\text{het})} + \bar{\alpha}_2^{(\text{het})})/2, \quad (15)$$

$$\delta(R_2) \approx q_2\bar{\alpha}_2^{(\text{hom})} + q_1(\bar{\alpha}_1^{(\text{het})} + \bar{\alpha}_2^{(\text{het})})/2, \quad (16)$$

$$\delta(\bar{R}) \approx q_1^2\bar{\alpha}_1^{(\text{hom})} + q_2^2\bar{\alpha}_2^{(\text{hom})} + q_1q_2(\bar{\alpha}_1^{(\text{het})} + \bar{\alpha}_2^{(\text{het})}), \quad (17)$$

where $\bar{\alpha}_1^{(\text{hom})}$ and $\bar{\alpha}_1^{(\text{het})}$ are the average random components per A_1 allele in homozygotes and heterozygotes, respectively, with similar definitions for $\bar{\alpha}_2^{(\text{hom})}$ and $\bar{\alpha}_2^{(\text{het})}$. These equations can be obtained by writing expressions similar to equation (1) for diploids and assuming approximate Hardy-Weinberg frequencies.

The simplest application, which is similar to Gillespie's (1974*a*) N -patch model, is the case in which the random effect of each allele is independent of the effect of its homologue and of all other alleles in the population. A straightforward calculation then shows that $\text{cov}(R_2, \bar{R}) - \text{cov}(R_1, \bar{R}) = (\sigma_2^2 - \sigma_1^2)/4N$, where σ_1^2 and σ_2^2 are the variances of the effects of randomly chosen A_1 and A_2 alleles. When the deterministic effects of the alleles are additive ($h = 1/2$), then the condition for the increase of the A_1 allele is $(\mu_1 - \sigma_1^2/4N) > (\mu_2 - \sigma_2^2/4N)$, which differs from the haploid case only by the factor of four in the denominator of the variance terms (see above). Since the correlations depend on the inverse of gene frequency, substitution into equation (7) yields a frequency-independent condition.

Variable Resource Acquisition and Nonlinearity

The previous models ignore the particular mechanisms that cause variation in reproductive success. For example, foraging success may vary among individuals and lead to variation in viability and fecundity. If foraging success and reproductive success are linearly related, then foraging success can be regarded as a scaled measure of reproductive success. However, because a nonlinear relationship between resource acquisition and reproductive success is probably common, variation in foraging success affects both the mean and the variance of reproductive success.

Several recent papers have assumed a nonlinear relationship between resource acquisition and reproductive success and then calculated the effect of variable resource acquisition on average reproductive success (Caraco 1980; Real 1980; Rubenstein 1982; further references in Stephens and Krebs 1986). These models have used average *individual* reproductive success to determine which strategies are likely to evolve. This method fails to translate individual reproduction into relative genotypic success and is therefore insufficient to predict evolutionary patterns. We extend these models by relating variable resource acquisition to the mean and variance of *genotypic* reproductive success, which we can then translate into relative genotypic success by the methods introduced above.

Begin by letting the reproductive success of the j th individual of the i th genotype be

$$X_{ij} = a_i + c_i[1 + g(\gamma_{ij})].$$

The term a_i is the component of reproductive success that does not depend on the trait of current interest, and c_i is the average reproductive success of this trait; thus, the relative magnitudes of a_i and c_i determine the fraction of average reproductive success explained by this particular trait. Fluctuations in resource acquisition are described by the random variable γ_{ij} , which has a mean of zero and a variance V_i . The function g translates fluctuations in resource acquisition into variation in reproductive success. By Taylor-series expansion, the expectation and variance of $g(\gamma_{ij})$ are approximately $(1/2)g''(0)V_i$ and $g'(0)V_i$, respectively, where primes denote first and second derivatives (for a discussion of the applicability of this approximation, see Stephens and Krebs 1986, pp. 145–146).

We can now write the expectation and variance of *individual* reproductive success for the i th genotype as

$$\mu_i \approx E(X_{ij}) = a_i + c_i[1 + (1/2)g''(0)V_i], \quad (18)$$

$$\sigma_i^2 \approx \text{var}(X_{ij}) = c_i^2 g'(0)V_i. \quad (19)$$

Using these equations, we can express the variance of *genotypic* reproductive success as $\rho_i \sigma_i^2$ and apply all aspects of the above theory. Clearly, if the correlations among individuals expressed by the ρ 's are not small, then one must take account of the variance of genotypic reproductive success when describing relative genotypic success. For example, if a significant component of variable foraging success by a particular strategy depends on environmental fluctuations, then the success of individuals pursuing the same strategy will be highly correlated. A

study of relative genotypic success is required in such a case, as illustrated in the second example below.

TWO EXAMPLES OF MODELS OF EVOLUTION IN A VARIABLE ENVIRONMENT

Developmental Homeostasis

Our formulation focuses on correlations in reproductive success among individuals. Such correlations arise when a group of individuals experience the same conditions. As a specific example in which groupings are fixed, suppose that there are K discrete flowering periods during a season and that each individual flowers during one period only. All A_2 types have strict canalization and flower during the same period, and each A_1 type has weak developmental homeostasis and flowers in any particular period with probability $1/K$. Conditions for pollination success or seed set vary among periods in an unpredictable and uncorrelated manner, because of variation in pollinator service, weather, etc. Furthermore, assume that the correlation in reproductive success between individuals flowering in a single period is λ . Under these assumptions, $\rho_2 = \lambda$, $\rho_1 = \lambda/K$, and $\rho_{12} = \lambda/K$, and for simplicity, we assume that the variance of reproductive success caused by vagaries of environment and pollination is the same for all individuals, $\sigma_1^2 = \sigma_2^2 = \sigma^2$.

Because the correlations have constant values in this model, the genotype with the larger geometric-mean reproductive success dominates (eq. [12]). If K is large, then the condition under which A_1 has an advantage is $\mu_1 > \mu_2 - \rho_2\sigma^2/2$.

Correlations and variations in reproductive success caused by patterns of developmental homeostasis can therefore be of sufficient magnitude to outweigh sizable differences in expected reproductive success. In this particular model, developmental homeostasis and strict canalization are disfavored by selection. Bull (1987) has examined models that reach a similar conclusion. Others (Slatkin and Lande 1976; Orzack 1985) have reached similar conclusions using models that allow for the possibility of temporal autocorrelations in environmental conditions. We have not analyzed the effects of temporal autocorrelations, although our general approach of considering the variances and covariances of individual reproductive success might be useful in considering autocorrelated environments.

Variable Resource Acquisition, Nonlinearity, and Life History

In this subsection, we show how variable resource acquisition fits within our framework by extending a model that compares semelparous with iteroparous reproduction (Charnov and Schaffer 1973; Hastings and Caswell 1979; Bulmer 1985). Here, variation in behavioral success affects both the expectation and the variance of genotypic reproductive success; and the variance of genotypic reproductive success in turn affects relative genotypic success. Previous studies of variable behavioral success have ignored the translation from reproductive success to relative genotypic success.

Let X_{ij} be the reproductive success of the j th individual of the i th genotype,

$$X_{ij} = p_i + [b_i - f(p_i)][1 + g(\gamma_{ij})], \quad (20)$$

where p_i is the probability that an adult survives to the next season after reproducing; b_i is the expected number of surviving offspring (birthrate) if $p_i = 0$; $f(p_i)$ is the reduction in the expected number of offspring as a function of parental survival; and γ_{ij} is the random component for the number of surviving offspring of the j th individual of the i th genotype.

Before analyzing this model, we make a few simplifying assumptions. First, let $p_1 = 0$; thus, the genotype $i = 1$ is semelparous. Next, assume that the expectation of γ_{ij} is zero and its variance is V_i for all i and j ; in words, the percentage of fluctuation in offspring production has the same long-term distribution for all individuals of a particular genotype, even though different individuals may experience different conditions in any particular season.

We can obtain the mean μ_i and the variance $\rho_i\sigma_i^2$ of genotypic reproductive success in the manner leading up to equations (18) and (19):

$$\begin{aligned}\mu_i &\approx p_i + [b_i - f(p_i)][1 + (1/2)g''(0)V_i]; \\ \rho_i\sigma_i^2 &\approx \rho_i[b_i - f(p_i)]^2g'(0)V_i.\end{aligned}$$

Solutions for two-allele haploid models with global regulation take one of two forms. If the correlations ρ are inversely proportional to gene frequency, $\rho_i = k_i/q_i$, then semelparity prevails when $\mu_1 - k_1\sigma_1^2 > \mu_2 - k_2\sigma_2^2$ (see eq. [10]). If, instead, the correlations are independent of gene frequency, then the genotypic geometric means determine long-term success, and the condition for semelparity is $\mu_1 - \rho_1\sigma_1^2/2 > \mu_2 - \rho_2\sigma_2^2/2$ (see eq. [12]).

Variable success in resource acquisition has two effects. First, when g is a diminishing-returns function, the expected reproductive success decreases with increasing variability because g'' is negative. This is the effect studied by previous models of variable resource acquisition (e.g., Caraco 1980; Real 1980; Rubenstein 1982). Second, the variability in individual resource acquisition affects the variance of average genotypic reproductive success, as in models discussed previously. The magnitude of the variance of average genotypic success depends, as always, on the correlation among individuals of that genotype. If environmental fluctuations affect resource acquisition by all members of the same genotype in the same way, then ρ_i is large, and this second effect is significant. However, if genotype affects the variance of an individual's success in a way that is not correlated with that of other members of the same genotype, then expected reproductive success μ is typically the dominant effect.

THE GEOMETRIC-MEAN PRINCIPLE

The geometric-mean principle states that, of two competing genotypes, the genotype with the higher geometric-mean fitness increases in frequency (see, e.g., Seger and Brockmann 1987). The difficulty in applying this principle is that it is not necessarily clear to which definition of fitness the geometric mean applies. In fact, several types of geometric-mean principles have been proposed. If the fitness is relative fitness, defined in equation (2) as the ratio of a genotype's reproductive success to the mean reproductive success, then the geometric-mean principle is

always true. For a haploid model, this can be proved simply by recursive application of equation (2). For a diploid model, equation (2) is still correct if F_1 is the marginal allelic fitness of A_1 . Consequently, the allele with the higher geometric-mean marginal fitness increases in frequency.

When stated in terms of relative or marginal fitness, the geometric-mean principle is always true but not very useful. The problem is that in a stochastic model there is usually no simple relationship between geometric-mean relative fitness and measurable quantities such as individual or genotypic reproductive success. In particular, relative fitness is unpredictable because it depends on gene frequencies that fluctuate stochastically (eq. [3]). Therefore, one cannot use the fact that the geometric-mean principle is true for relative fitness to conclude that the geometric-mean reproductive success or the geometric-mean success in foraging tends to increase under selection. We have shown that the actual course of gene-frequency changes depends on the detailed assumptions about how spatial, temporal, developmental, and behavioral variation affect the reproductive success of each individual and the correlations in reproductive success among individuals.

Gillespie's (1974a) model of individual variation is an example in which the geometric-mean reproductive success does not predict the outcome of selection. We showed that, in a generalization of Gillespie's two-allele haploid model, the sign of $(\mu_1 - k_1\sigma_1^2) - (\mu_2 - k_2\sigma_2^2)$ determines which allele tends to increase in frequency, where μ_i and σ_i^2 are the mean and variance of reproductive success of an individual of the i th genotype, and k_i is defined by $\rho_i = k_i/q_i$, where ρ_i is the correlation in reproductive success between two individuals of genotype i . As discussed above, Gillespie's (1974a) result follows for the special case in which different individuals of the same genotype have uncorrelated reproductive successes.

In our generalization of Gillespie's (1974a) model, the difference in geometric-mean reproductive successes of genotypes A_1 and A_2 is approximately $[\mu_1 - k_1\sigma_1^2/(2q_1)] - [\mu_2 - k_2\sigma_2^2/(2q_2)]$. This expression depends on gene frequencies, q , and may change sign as gene frequencies change. The correct condition to predict which allele will increase in frequency does not depend on q (see the preceding paragraph). Therefore, geometric means are not useful for this important class of models.

There are geometric-mean principles that can be applied. For example, geometric-mean reproductive success does predict the outcome of selection in Dempster's (1955) model and its generalizations. In a Levene (1953) model, Seger and Brockmann (1987) noted that a different geometric-mean principle applies; namely, selection favors the maximization of the geometric-mean reproductive success of different patch types (Li 1955; Cannings 1973). Although this result is true for the Levene model, it is a special case that cannot easily be generalized. In any model, it is possible to find some quantity whose geometric mean tends to increase; this quantity in a particular model does not provide a guide to determining the appropriate quantity in other models.

An idea similar to the geometric-mean principle is "variance discounting" (Caraco 1980; Real 1980; Stephens and Krebs 1986). Under variance discounting, the expected reproductive success of a behavior is the expected amount of the

resources acquired minus a function of the variance of the resources acquired (see eq. [18]). Variance discounting of this sort is a valid principle to the extent that average reproductive success is sufficient to predict evolutionary change in these models. This is true only when the behaviors on which the models focus do not cause a variance of reproductive success among individuals pursuing the same or different strategies. As we have shown in many cases, the dynamics of gene-frequency evolution also depend on the variance of reproductive success among individuals (e.g., eq. [19]).

To summarize, in some cases, a geometric-mean or related principle does predict the outcome of selection in a variable environment. Without a careful analysis, however, it is not clear how each of these principles applies to a variety of evolutionary problems. We see our approach as a preferable alternative to applying one of these principles in a general way. Environmental or developmental variation affects the means and variances of individual reproductive success and the correlations in reproductive success among individuals. These quantities determine which alleles, and hence which strategies, will be favored by natural selection.

BET HEDGING AND RISK AVERSION

The idea of bet hedging (Slatkin 1974; Seger and Brockmann 1987) can be understood in terms of our partitioning the genotypic variance of reproductive success into correlations among individuals and the variance of individuals. Bet hedging can be thought of as occurring at two levels, the level of the genotype and the level of the individual (Seger and Brockmann 1987). At the level of the genotype, bet hedging occurs if a trait or behavior reduces the correlations in reproductive success among individuals of a genotype, in effect increasing the number of independent samples, or bets, for each genotype. Our model of flowering periods and developmental homeostasis illustrates this genotypic-level bet hedging.

Bet hedging at the individual level can also be understood by correlations. Variance of individual reproductive success can be partitioned into variance of the success of particular offspring and the correlation in success among offspring (similar to eq. [6]). Putting one's eggs in different baskets reduces the correlations among offspring and thus reduces individual-level variance.

Bet hedging, the reduction in correlations either among individuals of the same genotype or among offspring of an individual, is always favored when there are no associated costs, since it reduces the variance of a genotype's average reproductive success. The interesting question is how this reduction in variance is balanced against particular costs. Our method provides a formal structure for comparing these costs and benefits.

An idea related to bet hedging is "risk aversion." Stephens and Krebs (1986) and Real and Caraco (1986) summarized recent work on this subject. Risk aversion can be defined within our approach as a trait or behavior that increases average reproductive success by lowering the variance of resource acquisition; this depends on reproductive success increasing at a diminishing rate with increasing resources (eq. [18]).

Bet hedging differs from risk aversion because, at the particular level being analyzed, bet hedging reduces the variance of success, whereas risk aversion increases average success. This difference can be illustrated by considering once again the case of variable resource acquisition at the individual level. In comparisons of two strategies in which each behavioral episode has a higher or lower variance of resource reward, choosing the lower-variance reward is a case of risk aversion. Risk aversion is favored because of the diminishing relation between resources and reproduction. By contrast, sampling from several independent patches in order to reduce the correlation among resource rewards for each episode, and thereby the overall variance of reproductive success, is a case of bet hedging. Bet hedging is favored because a lower variance of individual reproductive success is advantageous to genotypic success as, for example, in the case presented in the subsection "*Gillespie's Model of Individual Variation*," above.

Sometimes it is not so easy to decide whether a behavior should be considered an example of risk aversion or bet hedging. An analysis comparing these two categories can help to clarify the biological assumptions and conclusions of a model. Consider Real's (1980) paper, which is widely cited in the context of risk aversion and variance discounting. There are actually two parts to Real's model. First, he assumed that reproduction increases at a diminishing rate as resources increase; therefore, a relatively high variance of resource acquisition leads to a relatively low expected reproductive success as in equation (18). Second, Real partitioned the total variance of individual resource acquisition into the variance associated with each type of resource-acquiring behavior that an individual pursues and the correlations among the successes of each of these behaviors, a partitioning of variance similar to equation (6) but at a different level. Real (1980) stressed that, within an individual, diverse behaviors with low correlations among themselves reduce the variance of resource acquisition and therefore increase expected reproductive success. The first part of the model, that high variance of resources reduces expected reproductive success, is consistent with our description of risk aversion. Real's main point, however, is that diverse behaviors lead to a lower variance of resource acquisition, consistent with our definition of bet hedging as a reduction in variance by increased sampling. Which description one chooses depends on the level being analyzed. As a final note, Real's analysis did not address the effect of variable resource acquisition on the variance of reproductive success and is therefore an incomplete study of evolutionary consequences. A fuller analysis is contained in equations (18) and (19).

CONCLUSIONS

We have developed a theoretical framework with which to analyze the evolutionary consequences of variable environments. Our approach emphasizes that means and variances of individual reproductive success and correlations in the reproductive successes of different individuals are sufficient to predict the long-term course of evolution under a wide variety of conditions. Different assumptions about developmental, behavioral, spatial, and temporal variation in factors that affect reproductive success lead to different variances of and correlations in reproductive success. By focusing on processes at the individual level, we have

shown the relationship among several kinds of models of variation. We have also shown the relationship of such ideas as bet hedging, risk aversion, and variance discounting to the more conventional models of selection.

Our analysis is not completely general. We assume that environmental variation affects reproductive success in such a way that the mean and variance of the changes in allele frequency are sufficient to predict the course of evolution. That is the assumption necessary to use the mathematical tools of diffusion theory. In effect, this assumption restricts our method to cases in which the variance of reproductive success is small relative to the mean. We did not analyze cases in which there are correlations in environmental conditions between generations. Our approach does, however, allow the analysis of a large class of models that have been and will continue to be of interest to evolutionary biologists.

SUMMARY

We develop a general model for the effects of variation in reproductive success on gene-frequency change and phenotypic evolution. Our approach is based on distinguishing among individual, genotypic, and population-level reproductive success and on relating these three levels through correlations. For example, the variance of genotypic reproductive success can be expressed by individual-level variance and by the correlations among individuals. We use these correlations to show the simple relationship among earlier models of selection on the variance of reproductive success, of temporal variation in selection, of spatial variation in selection, and of variation in behavioral traits. Our approach also applies to diploid individuals by regarding diploidy as a way to induce correlations in reproductive success between pairs of alleles. We apply our method to patterns of developmental homeostasis, the evolution of iteroparity, and the effects of variability in resource acquisition under nonlinear gains. Finally, we discuss the uses and limitations of the geometric-mean principle, and we provide a precise description and formal methods of analysis for bet hedging and risk aversion.

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APPENDIX

We prove a general result about diffusion theory that we used to extend Gillespie's (1974a) model of individual variation in reproductive success. Our notation here is based on that of Ewens (1979, chap. 4). We assume finite population size, and therefore accessible boundaries, and a haploid model with global population regulation. We continue to make the particular assumptions about magnitudes of effects in equation (1) so that a diffusion analysis is valid.

We claimed in the text that if $E(\Delta q_1) > 0 \forall q_1$, then A_1 has a greater probability of fixation

than a neutral gene; that is, the probability of fixation is greater than q_1 . This claim justifies the use of the condition presented in equation (10). We now prove this claim.

Let the frequency of some allele be q and its probability of fixation be $P(q)$. If the allele is neutral, then $P(q) = q$, and we must show that $P(q) > q$ when $E(\Delta q) > 0 \forall q$. Let $E(\Delta q) = a(q)$ and $\text{var}(\Delta q) = b(q)$. Now, by definition,

$$\psi(y) = \exp \left\{ -2 \int^y [a(z)/b(z)] dz \right\}.$$

The probability of fixation at a frequency of one is

$$P(q) = \int_0^q \psi(y) dy / \int_0^1 \psi(y) dy$$

(Ewens 1979).

We need to show that $P(q) > q$ or, equivalently,

$$\frac{1}{q} \int_0^q \psi(y) dy > \int_0^1 \psi(y) dy.$$

Note that $\psi(y)$ is a monotonically decreasing function of y since, by assumption, $a(z)/b(z)$ is always positive. Thus, the left-hand side of this inequality, which is the average area under $\psi(y)$ over the interval $(0, q)$, must be greater than the right-hand side, which is the average area under $\psi(y)$ over the interval $(0, 1)$.

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