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Delayed reproduction and fitness in variable environments

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ABSTRACT **Many organisms delay the initiation of reproduction even though such delay is not adaptive in a constant environment. Theoretical arguments in this paper show that delaying reproduction can increase fitness in a sufficiently variable environment. This paper uses stochastic demography to analyze the fluctuating population structure produced by environmental uncertainty. The results explain previously puzzling features of life cycle delays observed in nature, predicting that populations of the same species living in environments of differing harshness can display different life history phenotypes, a number of distinct life history phenotypes can coexist neutrally within a single population, and genetic polymorphisms are easily maintained if heterozygotes have intermediate life history phenotypes.**

Many organisms delay the initiation of reproduction even though such delay is not adaptive in a constant environment. Biologists studying life cycle delays such as insect diapause (1–3), seed dormancy (4, 5), and cohort splitting (6–8) have suggested that delay in a life cycle is advantageous in a varying environment. This paper presents theoretical arguments to show that life cycle delays can increase fitness when environments are sufficiently variable. A significant and essential improvement is made over existing theory (9–11) by analyzing explicitly the changing age structure produced by environmental uncertainty. My results identify the stochastic evolutionarily stable state (ESS; ref. 12) for the extent of life cycle delay appropriate to any level of environmental variability. They also provide explanations for the geographic, phenotypic, and genetic variation in delays observed in many natural populations.

Possibly the most influential early theoretical work on the evolution of life cycle delays in a fluctuating environment is Cohen's 1966 paper (9), which does not analyze age structure. A considerable body of work has attempted refinements to this paper, but all of it ignores age structure; a recent review can be found in ref. 10. Livdahl (11) attempted a theoretical treatment including age structure, which as he pointed out is central to determining the consequences of a varying environment, but his demographic and genetic analyses fail to correctly describe stochastic variation. In recent years, a theory of stochastic demography has been developed (13, 14) that provides the essential theoretical basis for the present paper. I show that the stochastic dynamics of age structure may be important in understanding the evolution of many aspects of life histories.

Delay, Fitness, and Stochastic Demography

I begin by defining a relationship between delay and fitness and then consider its empirical applications. The discussion initially focuses on a simple model for which I obtain an exact analytical expression for fitness and discuss its empirical applications. The key biological conclusions from this simple model will be shown to hold for more complex life cycles.

Consider a population composed of immature and adult individuals, in which some immatures delay passage to adulthood. Suppose that a fraction p of immatures delays the transition to adulthood in each breeding season and that s is the survival rate of immatures. To describe environmental variability, let adult fertility be a time-dependent random variable (say M_t). The dynamics of the numbers I_t of immatures and A_t of adults are described in discrete time by the equation

$$\begin{bmatrix} I_{t+1} \\ A_{t+1} \end{bmatrix} = \begin{bmatrix} p & M_{t+1} \\ s(1-p) & 0 \end{bmatrix} \begin{bmatrix} I_t \\ A_t \end{bmatrix}. \quad [1]$$

Changes in this population depend on the sequence of distinct random fertilities M_1, M_2, etc., so the population composition does not reach a fixed value but changes over time. Stochastic demography (13) shows that for reasonable random sequences of fertility the long-run growth rate of such a population converges to a nonrandom number a, the stochastic analog of the Malthusian parameter. This a serves as a fitness determining the invasion of genes that alter the life cycle in the following sense (14). Suppose the population consists of diploid randomly mating individuals in which alleles 1 and 2 at a locus influence the life history in a random environment. Then allele 2 can initially increase in frequency in a population that is initially nearly homozygous for allele 1 if $a_{12} > a_{11}$; here a_{ij} is the stochastic growth rate of a population of individuals with the same life cycle as genotype ij. Thus, we may describe the evolutionary consequences of delay by determining its effect on the (stochastic) fitness a. By analogy with the deterministic situation, a stochastic ESS occurs at the extent of delay for which a is at a maximum.

Delay Is Not Adaptive in a Constant Environment

In a constant environment (with M_t fixed), a reduces to the Malthusian parameter r (15). I will show that r is decreased by an increase in the fraction p of individuals, which delays development, if the population is not declining. I will outline the calculation for a general life cycle described by a standard Leslie matrix (15), in which the first row contains fertilities and subdiagonal elements contain survival rates. Delay in any prereproductive age class i is described by modifying the matrix as follows: change the i,i element from 0 to p and multiply the $(i+1),i$ element by $(1-p)$. An example is the matrix on the right side of Eq. 1. Application of Caswell's (16) sensitivity formulae for r to such a modified matrix shows that

$$\frac{\partial r}{\partial p} \propto (V_{i+1} - V_i),$$

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Abbreviation: ESS, evolutionarily stable state.

where V_i is the reproductive value of age class i individuals. Noting that age class i does not reproduce (so the 1,i element of the matrix is zero), the definition of reproductive value as the left eigenvector of the modified Leslie matrix shows that $(V_{i+1} - V_i) < 0$ as long as $r > 0$. Thus, in a constant environment and an increasing population one expects no delay in any prereproductive age class. Under these conditions, any argument for the adaptive value of delay would have to invoke selection on some correlated trait (e.g., if delay were to result in enhanced survival or fertility, which more than offset the decrease in r due to delay alone).

Delay and Fitness: An Exact Relationship

Shifting to a variable environment in the model of Eq. **1**, suppose that M_t is a sequence of independent identically distributed random variables. In addition, assume that each M_t follows a γ distribution with coefficient of variation (ratio of standard deviation to mean) c. I now show how the stochastic dynamics of the population are analyzed to obtain an exact analytical formula for the long-run growth rate a. Readers uninterested in the technical details can go straight to Eq. **8** below.

In analyzing Eq. **1**, it is convenient to absorb s into M_t, so that in the equations that follow we can set $s = 1$. Defining $n = c^{-1/2}$, write $M_t = mG_t$, where G_t has γ density

$$g(w) = \frac{n^n}{\Gamma(n)} w^{n-1} e^{-wn}. \qquad [2]$$

The average of M_t is m and its variance is $m^2c^2 = (m^2/n)$. To calculate the long-run growth rate of the population in Eq. **1**, it is first necessary to obtain the stationary probability distribution of the population's age structure (13). It turns out to be easier to work instead with a transposed (dual) version of Eq. **1**. Let $\mathbf{X}_t$ be the transpose of the random matrix of Eq. **1** and consider the recursion

$$\mathbf{V}_t = \mathbf{X}_{t+1}\mathbf{V}_{t+1}. \qquad [3]$$

The vector $\mathbf{V}_t$ has a "backwards-in-time" evolution, but it has the same long-run growth rate a as the population vector in Eq. **1**—i.e.,

$$a = E \log\{[V_t(1) + V_t(2)]/[V_{t+1}(1) + V_{t+1}(2)]\}. \qquad [4]$$

Here, E indicates an average over both the random environmental distribution (Eq. **2**) and the stationary distribution of the (normalized version of) vector $\mathbf{V}_t$.

I will work with $\mathbf{V}_t$ because its stochastic properties can be obtained analytically. Define

$$R_t = \frac{(1-p)V_t(2)}{pV_t(1)}, \qquad [5]$$

where $0 < p < 1$ and the $V_t(i)$ are components of $\mathbf{V}_t$. Then the recursion (Eq. **3**) can be rewritten as

$$R_t = \frac{G_{t+1}}{z(1 + R_{t+1})}, \qquad [6]$$

where $z = p^2/m(1-p)$. The stationary probability density C of R_t obeys the following integral equation (which is derived and solved as in ref. 17),

$$C(x) = \int_0^\infty dy\, C(y)z(1+y)g[z(1+y)x],$$

with solution

$$C(x) = \kappa^{-1}x^{n-1}(1+x)^{-n}e^{-nzx}, \qquad [7]$$

where κ is fixed by normalization. Letting E_C indicate an average over the density C, and using Eqs. **4–6**, I find the exact analytical expression

$$a = \log p + E_C \log(1 + R). \qquad [8]$$

One can now compute a to any desired accuracy by quadratures (for the analytically minded, the expectation in Eq. **8** can be recast in terms of hypergeometric functions).

Life Cycle Delay Can Increase Fitness in a Variable Environment

I now explore the effects of delay on fitness a for the model (Eq. **1**) using the result (Eq. **8**). The calculation of a requires values of the extent of delay p and the product ms of the average fertility m and the survival rate s of young. Fig. 1 shows the relationship between a and the extent of life cycle delay p for different levels of environmental variability, computed with $m = 1.05$ and $s = 1$. For very small environmental variability (small c), a declines with p as would the deterministic Malthusian parameter r. When the environment becomes harsher and more variable (larger c), there is a dramatic shift in the curve and a increases with p to a maximum at an intermediate fraction of delay. Clearly, a fitness advantage can result from a nonzero probability of delaying the passage to adulthood.

Two other features of Fig. 1 have considerable biological significance. First, the delay fraction at which a is a maximum (the stochastic ESS of p) increases as the harshness of the environment (c) increases. Second, the maximum in a becomes very broad as c increases, so that in a highly variable environment there is very little difference in fitness between the ESS and a wide range of other delay phenotypes. I return to the biological consequences of Fig. 1 after showing that its features are robust to my choice of model and assumptions.

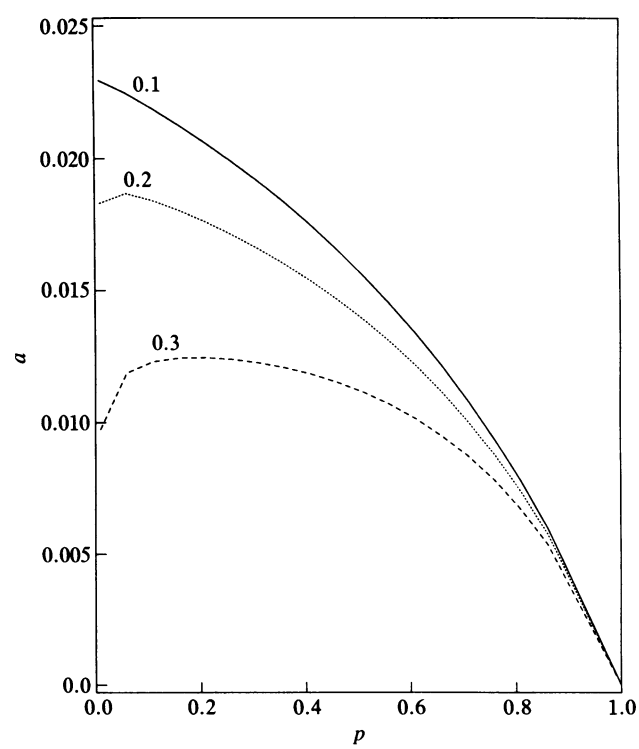

FIG. 1. Effect on fitness a of probability p of delayed passage from immature to adult. Curves are labeled with the coefficient of variation of fertility and are drawn through points computed from an exact analytical formula. See text for parameter values.

The properties of fitness a in Fig. 1 do not depend on the assumptions made about the random environment. Stochastic simulations of model **1** using fertilities distributed according to a lognormal distribution (instead of γ) yield results entirely similar to those in Fig. 1. The parameters used for the simulation are the same as in Fig. 1. This shows that the result is robust, although a stronger test should use a distribution of qualitatively different shape. I have also examined the effect of serial autocorrelation over time in the sequence of random fertilities and find the qualitative results unchanged. I will discuss autocorrelation further in the context of complex life cycles.

The Results Apply to Complex Life Cycles

I now show that the fitness–delay relationship for the two-age-class model carries over to more complex life cycles. I do so by studying another example, but one that makes clear how general cases can be dealt with. Consider a life cycle with two prereproductive stages, eggs and larvae, and two reproductive adult stages. Suppose that there is delay at the larval stage, and let p be the proportion of larvae delaying development to adulthood. As in the earlier example, I take the fertilities of adults to be variable over time; denote them by M_{3t}, M_{4t}. This population's dynamics are described by the modified Leslie matrix

$$\begin{bmatrix} 0 & 0 & M_{3t} & M_{4t} \\ s_1 & p & 0 & 0 \\ 0 & s_2(1-p) & 0 & 0 \\ 0 & 0 & s_3 & 0 \end{bmatrix} \qquad [9]$$

The survival rates are nonzero, $0 < s_i \leq 1$ for all i. As shown in general earlier, in a constant environment and an increasing population, increases in larval delay p will decrease the Malthusian parameter.

What happens when the four-age-class life cycle experiences environmental variation? I will determine the effects of delay by using a general analytical approximation to a and then present supporting numerical simulations. The analytical approximation (14) is accurate when the environmental variability is small and is obtained as follows. Let σ_i be the variance and $m_i > 0$ the average of M_{it}, and l_i be the survival rate to age class i with no delay. Let λ be the dominant eigenvalue of the population projection matrix with fertilities fixed at their averages; this λ solves a characteristic equation of the usual sort. Write $\lambda_1 = \lambda - p$ and define

$$u_i = l_i/\lambda^{i-1} \qquad i = 1, 2, \qquad [10]$$

and

$$u_i = l_i(1-p)/(\lambda^{i-2}\lambda_1) \qquad i = 3, 4. \qquad [11]$$

Here $u_1 = 1$ and the u_i, $i > 1$, are computed as the components of the right eigenvector of the average of the four-age-class random matrix. When $0 < p < 1$, this average matrix is nonnegative and irreducible, so it must have a positive eigenvector whose components are proportional to these u_i, $i \geq 1$. Therefore, it must be true that $\lambda > p$ when $0 < p < 1$. It is obvious that $\lambda > p$ when $p = 0$. And when $p = 1$, the average matrix is reducible and its largest eigenvalue is $\lambda = 1$. Therefore, we have $\lambda_1 > 0$ for $0 \leq p < 1$ and $\lambda_1 = 0$ at $p = 1$. I allow for the possibility that the adult fertilities may change in a correlated way, and write ρ for the correlation coefficient between M_{3t} and M_{4t} in each time interval. I assume no serial autocorrelation. Finally, setting $r = \log_e \lambda$ and applying directly the formulae of ref. 14, I find that

$$a \approx r - [\sigma_3^2 u_3^2 + \sigma_4^2 u_4^2 + 2\rho\sigma_3 u_3 \sigma_4 u_4]/(2\lambda^2\tau^2), \qquad [12]$$

where

$$\tau = 2 + m_4 u_4 + (\lambda/\lambda_1).$$

The quantities in Eq. **12** have the following biological meanings: if the environment is constant so that the fertilities are fixed at their average values, $r = \log_e \lambda$ would be the Malthusian parameter, u_i would be the stable proportion of individuals in age class i, and τ would be the mean generation length.

To see what Eq. **12** implies about delay vis-a-vis fitness, note the following: (*i*) as shown earlier for a constant environment, r decreases to 0 as delay p increases toward 1; (*ii*) for a fixed level of delay p, fitness a decreases as the variances σ_i increase; (*iii*) as the delay p increases toward 1, the quantities u_3 and u_4 decrease to zero, whereas the generation length τ increases toward infinity. This last point follows from the formula for τ plus the earlier observation that $\lambda_1 = 0$ at $p = 1$. For a given amount of environmental variance, this increase in τ with large p shows that the second term in Eq. **12** is much smaller for large p than for small p. Therefore, increasing environmental variance has much less effect on a when delay p is large than when it is small. Thus, in sufficiently harsh environments we can expect higher fitness with some delay than with no delay. This argument is illustrated in Fig. 2, in which analytical values of fitness computed using Eq. **12** are plotted versus delay, showing the emergence of a stochastic ESS in a sufficiently harsh environment. The parameters used in the calculation were $s_i = 1$ for all i, $m_3 = m_4 = 0.525$, and the coefficient of variation shown along with each plotted curve.

The above analysis is illustrated and confirmed by simulations. Fig. 3 is an example in which M_{3t}, M_{4t} are taken to be perfectly correlated and lognormally distributed with no serial autocorrelation. The parameters used for the simula-

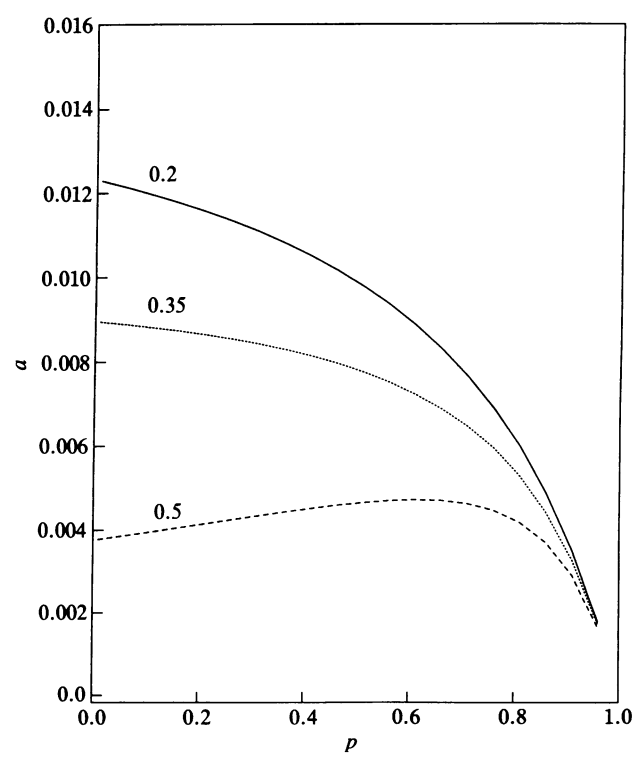

FIG. 2. Analytically computed fitness a vs. probability p of delayed larval development. Curves are labeled with a coefficient of variation that measures environmental variability and are drawn through points computed from Eq. **12**. Immature stages: eggs, larvae. Adults reproduce twice, their fertilities are perfectly correlated, and there is no serial autocorrelation. See text for parameter values.

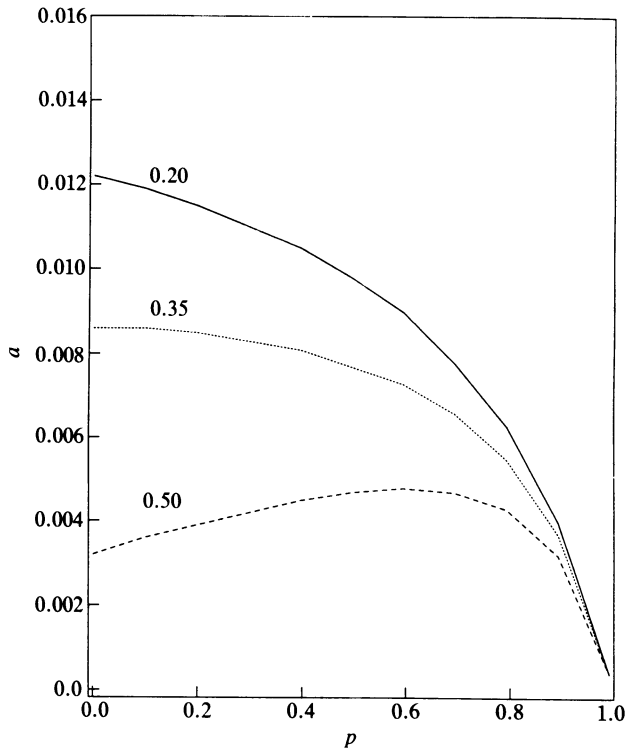

FIG. 3. Effect on fitness a of probability p of delayed larval development. Curves are labeled with a coefficient of variation, which measures environmental variability, and are drawn through points computed by numerical simulation. Immature stages: eggs, larvae. Adults reproduce twice, their fertilities are lognormally distributed and perfectly correlated, and there is no serial autocorrelation. See text for parameter values.

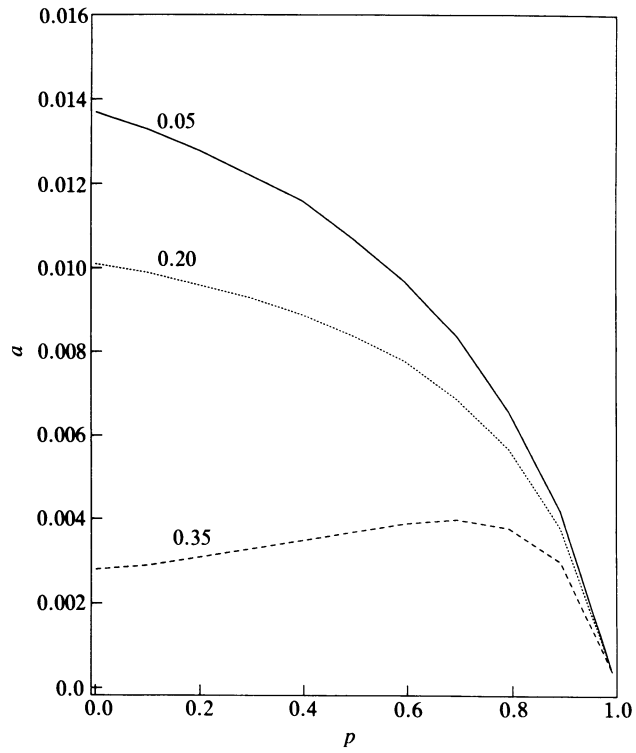

FIG. 4. Effect on fitness–delay relationship of serial autocorrelation in vital rates. Curves are drawn for the same model as in Fig. 3. Here the fertilities are perfectly correlated within each time interval and have a positive serial autocorrelation. The curves are labeled with the coefficient of variation of fertility in a single time interval and are computed from a series of stochastic simulations. See text for parameter values.

tion were $s_i = 1$ for all i, $m_3 = m_4 = 0.525$, and the coefficient of variation shown along with each plotted curve. Note first the appearance of an ESS at intermediate delay when the environment becomes sufficiently harsh, as predicted by the theory. As in Fig. 1, the maximum in fitness becomes significantly broader as the harshness of the environment increases. There is one striking difference between Figs. 1 and 3: the peak fitness in Fig. 1 occurs at a much lower value of delay p than in Fig. 3. The reason for the difference is found in the structure of the two life histories. The complex life history represented by Eq. **9** is iteroparous, and it is known (18, 19) that iteroparity in itself can result in a fitness advantage in variable environments. Thus, the extent of delay needed to produce a further fitness gain is expected to be larger for the iteroparous life cycle.

Simulations also reveal an interesting aspect of serial autocorrelation. For the four-age-class life cycle, Fig. 4 shows the effect of adding positive serial autocorrelation to the random sequence of fertilities. At each time, the fertilities are perfectly correlated and lognormally distributed. In the simulation, the fertilities have the form $\exp(\alpha_i + \beta_i X_t)$ where X_t is a sequence generated as a zero-mean, one-lag, autoregressive time series with standard normal shocks and a serial autocorrelation coefficient of +0.5. The other parameter values used were the same as for Fig. 3. Comparison of Figs. 3 and 4 shows that fitness behaves in essentially the same way in a serially autocorrelated environment as in a purely random one. However, positive serial autocorrelation clearly acts as a variance magnifier, so that a population exposed to a positively correlated environment of a given harshness behaves in the same way as a population exposed to a much harsher but serially uncorrelated environment.

Confronting Theory with Observations

The theory in this paper yields predictions concerning intrapopulation and interpopulation variability in life cycle delays; these predictions are supported by a wide range of evidence. The predictions are as follows:

(*i*) Populations of the same species living in different environments can be expected to display different amounts of prereproductive delay. Notice in the theory that the delay fraction at which a is maximal changes with c. Since geographically distinct populations (of one species) can often be expected to experience different levels of environmental harshness c, the present theory predicts that such populations would have different stochastic ESSs. Assuming selection is sufficient for the achievement of an ESS, such populations would display different amounts of prereproductive delay. Interpopulation variability within species correlated with environmental differences in habitat is well documented in many life history characteristics. Examples include cohort splitting in the rock slater *Ligia oceanica* (7) and the timing and distribution of reproduction in the freshwater leech *Erpobdella octoculata* (20).

(*ii*) Two or more distinct life history phenotypes can coexist neutrally in a single population. Note that the theoretically derived maximum in a is very broad for high c, showing that many different life cycles are nearly equally fit in harsh environments. Some of these life cycles have delays very different from the stochastic ESS. In consequence, two or more different but relatively neutral life history phenotypes can coexist in a single population. Examples of intrapopulation variability in life history abound. In cases in which phenotypic variability is documented but the possible genetic basis underlying it has not been characterized, it is possible that variation is being neutrally maintained. Examples in-

clude cohort splitting in the woodlice *Philoscia muscorum* (21), the timing of reproduction in *E. octoculata* (6, 22) and in the sea squirt *Botryllus schlosseri* (23), and development time in copepods (24). Other examples are given in ref. 23.

(*iii*) Genetic polymorphism can easily maintain distinct life histories in a single population. Note that the theoretically computed fitness a as a function of p is a concave curve with one peak. Therefore genetic polymorphisms for the extent of delay can be maintained as follows: if two homozygotes have values of p on different sides of the maximum, a polymorphism is possible whenever the heterozygote has an intermediate phenotype (because the heterozygote is fitter than its constituent homozygotes; ref. 14). Intermediate phenotypes for heterozygotes are commonly observed. Two studies illustrate very clearly genetic variation for life cycle delays: Istock (25) has shown that persistent heritable genetic variation underlies the variability in diapause fraction observed within populations of the pitcher-plant mosquito *Wyeomia smithii*; and Gilbert (26) has shown that genetic variation underlies the phenotypic variability observed in developmental time in the white butterfly *Pieris rapae*. Both cases lend support to the theory presented here. Other relevant examples are discussed in refs. 1, 3, and 27.

In theoretical work closely related to the present paper, Orzack and Tuljapurkar (18) and Roerdink (19) have analyzed the relationship between fitness and the extent of iteroparity in a variable environment. Their qualitative conclusions are very similar to those in *i–iii* above, suggesting that these may be general features of life history evolution in uncertain environments. As noted earlier, their work also bears on the different peak locations in Fig. 1 for a population in which adults reproduce once, and in Fig. 3 for a population in which adults reproduce twice. Delay has less marginal value for the population in Fig. 3 because repeated reproduction (iteroparity) in itself can increase fitness in a variable environment. I conclude that prereproductive delay and iteroparity are unlikely to evolve together. For example, dormant seed banks should be more common in annual plants than in perennials, and diapause should be relatively uncommon in insects that survive over more than one breeding season.

Life history theory based on fixed vital rates, or on simplistic nondemographic approximations for varying environments, is not capable of explaining the kinds of patterns described in *i–iii* above. Other critical assumptions of the optimality theory of life histories are not met in some of the examples cited here (20, 26). In my view, the selective forces due to uncertain environments provide an evolutionary explanation for many of the observations that disagree with classical optimality theory.

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