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Phenotypic Plasticity in Life-History Traits: Demographic Effects and Evolutionary Consequences[1]

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SYNOPSIS. Although much life-history theory assumes otherwise, most life-history traits exhibit phenotypic plasticity in response to environmental factors during development. Plasticity has long been recognized as a potentially important factor in evolution, is known to be under genetic control, and may or may not be adaptive. The notion of adaptive plasticity contrasts with the idea that developmental homeostasis is a major evolutionary goal. The conflict was resolved in principle by Ashby's cybernetic analysis of homeostasis, which showed how plasticity in "response variables" might act to screen "essential variables" from the impact of environmental disturbance. To apply this analysis to life-history plasticity, it must be incorporated into a demographic model. An approach is presented here using life cycle graphs and matrix projection models. Plasticity in response to temporal variation leads to time-varying matrix models: plasticity in response to spatial variation leads to models structured by criteria other than age. The adaptive value of such plasticity can be assessed by calculating its effects on a suitable measure of fitness: long-term growth rate for time-invariant models, expected growth rate discounted by variance for time-varying models. Three examples are analyzed here: plasticity in the rate of development from one instar to the next in a stage-classified model, plasticity in multiplicative yield components, and plasticity in dormancy as a response to environmental cues. Development rate plasticity is adaptive if reproductive value increases from the instar in question to the next, maladaptive otherwise. Plasticity in yield components reduces fitness variance, and hence is adaptive, if the responses of successive developmental steps (*e.g.*, flowers/stem, seeds/flower) are negatively correlated. Plasticity in dormancy is adaptive if it responds to the same factor(s) influencing mortality, but with opposite sign. A number of important problems, including trade-offs between genetic and phenotypic adaptation and the distinction between continuous and discontinous plasticity remain to be solved.

INTRODUCTION

Phenotypic plasticity in life-history traits is a pervasive phenomenon, but one which has yet to be satisfactorily incorporated into life-history theory. This paper is an attempt in that direction. I hope to show some possible approaches, to outline the kind of results these approaches may yield, and to reveal some of the difficulties still to be overcome in any truly successful solution of the problem.

An organism's genes and its environment together determine its phenotype, not as a structure frozen in time, but as a life cycle which unfolds dynamically over the whole lifespan of the individual (Bonner, 1965, 1974). The incorporation of this epigenetic process into evolutionary theory has long been recognized as a major problem (*e.g.*, Waddington, 1957, 1968; Lewontin, 1974; Bonner, 1982). One aspect of development is the *plasticity* of the phenotype, *i.e.*, the sensitivity of the phenotype to the environment (Bradshaw, 1965). My focus here is on life-history traits, as distinct from the morphological and physiological traits on which attention has traditionally focused. Indeed, to the extent that fitness has a demographic basis, morphological or physiological plasticity is evolutionarily irrelevant unless it affects life-history traits.

Life-history plasticity is interesting for several reasons. It is conspicuous by its absence in genetically naive life-history theory, which predicts optimal strategies in different environments, and makes the unspoken assumption that selection will fix those genotypes yielding the appropriate strategy in the appropriate circumstances. Tests of such theories are usually conducted by comparing observed and predicted phenotypes in different populations

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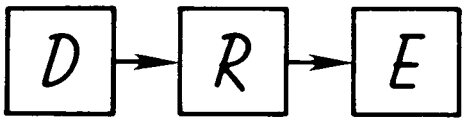

FIG. 1. Ashby's (1956) schematic representation of homeostasis. D represents a disturbance process, originating in the external environment. E is a set of "essential variables"; the fitness of the organism depends on these variables remaining within certain bounds. R is a response system. It is homeostatic to the extent that its actions screen E from the behavior of D.

or species. Stearns (1977) has reviewed much of this data and assessed its quality. One of his criteria was whether the genetic basis of the phenotypic differences had been demonstrated. But given that most life-history theory is purely strategic, and specifies no genetic mechanisms, one might argue that the source of the phenotypic differences is irrelevant. Even if the theory is regarded as making genetic predictions, it must be remembered that the plasticity of a given character is itself a trait and may be under genetic control. Dobzhansky (1951) pointed out that, "Genes act through the developmental patterns which the organism shows in each environment. What changes in evolution is the norm of reaction of the organism to the environment." Bradshaw (1965) summarizes abundant evidence that plasticity is under genetic control on a trait-by-trait basis. If the amount and nature of plasticity may themselves be adaptive, how can they be incorporated into life-history theory and how can one test their adaptiveness?

The last question, it seems to me, is crucial. It is clear that glib acceptance of the "adaptationist programme" (Gould and Lewontin, 1979) can lead to confusion and misinterpretation of evolutionary patterns. This is especially true when dealing with plasticity as a trait in itself, since the physicochemical basis of life automatically makes epigenesis susceptible to outside influences. Schmalhausen (1949), for example, distinguished adaptive "modifications," elicited by evolutionarily familiar stimuli, from maladaptive "morphoses" resulting from unfamiliar factors. Distinguishing adaptive variation from the unavoidable influence of the environment will not always be easy.

The possibility of adaptive explanations for plasticity raises two important issues. One is the conflict between plasticity and homeostasis. On the one hand, the possible advantages of plasticity are obvious. Darwin (1881) conjectured that plasticity would be favored in a species subject to "repeated and great changes of conditions." Wright (1931) called it "perhaps the chief object of selection." Baldwin (1902) proposed that plasticity had tended to increase over evolutionary time, while "congenital influence" (*i.e.*, direct genetic control) had decreased; Lewontin (1957) proposed essentially the same thing. On the other hand, homeostasis, the maintenance of a constant *milieu interieur* in the face of a changing external environment, has long been viewed as one of the most important forms of adaptation (Bernard, 1878; Cannon, 1932; Lerner, 1954; Waddington, 1957).

The second issue is the choice between phenotypic plasticity and genotypic change as means of adaptation. Are there situations which consistently favor one over the other? And are the two independent, or do they interact with each other?

These are some of the problems raised by recognizing the possibility that plasticity may itself be an adaptive trait. The next section reviews some theoretical approaches to these problems.

BACKGROUND

This is not the place for a review of patterns of life-history plasticity, valuable though such a review would be (see Bradshaw, 1965; Jain, 1979; Stearns, 1982). Suffice it to say that plasticity has been demonstrated in most life-history traits: survival (*e.g.*, White and Harper, 1970), fecundity (*e.g.*, Kahn and Bradshaw, 1976; Matsuo, 1975), offspring size in humans (*e.g.*, Weinstein and Haas, 1977) but not seed size in plants (*e.g.*, Harper *et al.*, 1970), the sensitivity of fecundity to density (Lavie *et al.*, 1978), energy allocation (*e.g.*, Hickman, 1975), development rate (*e.g.*, Birley, 1979; Smith-Gill and Berven, 1979), the dormancy of offspring (*e.g.*, Marcus, 1982), etc.

The interpretation of this diversity of patterns requires a conceptual framework. Let us consider some possibilities.

The cybernetic approach. The most complete analysis of homeostasis and plasticity is due to Ashby (1952, 1956). His theory describes the state of the organism in terms of a set E of "essential variables" (Fig. 1), which must be kept within a certain range for continued survival. The essential variables are subject to disturbances, D, which originate in the external environment. The goal of the homeostatic or regulating system, R, is to protect the essential variables from disturbance, thus preserving the integrity of the organism.

The uncertainty in behavior of each of the components in Figure 1 can be measured by its entropy, $H = -\Sigma p_i \ln(p_i)$, where p_i is the probability of occurrence of the i^{th} state. Ashby (1956) pointed out that

$$H(E) \geq H(D) + H(R \mid D) - H(R),$$

where H(E), H(D), and H(R) are the uncertainties in the essential variables, the environmental disturbance process, and the regulator, and $H(R \mid D)$ is the conditional uncertainty in the regulator, given the state of the environment. The goal of homeostasis is to reduce H(E). Clearly, there are two possible tactics: increasing the plasticity of the regulator (H(R)), or linking the behavior of the regulator more tightly to the disturbance, thus reducing $H(R \mid D)$.

Here is a conceptual solution to the contrast between plasticity and homeostasis. Plasticity is adaptive to the extent that it contributes to homeostasis, and the goal of reducing variability in the essential variables requires sufficient plasticity in the regulating system. Ashby called this the Law of Requisite Variety, and spoke of homeostasis as "variety destroying variety."

In an important but little known paper Bateson (1963, see also 1979) applied Ashby's ideas to evolution. He pointed out that an immediate consequence of the Law of Requisite Variety is the existence of an economics of plasticity. If flexibility in the regulating system protects the essential variables, multiple, conflicting demands may so reduce the available plasticity that homeostasis breaks down. Thus organisms may face the problem of allocating a limited supply of plasticity.

Within an evolving population, the plasticity required for regulation may arise at any of a number of levels: shallow, easily reversible behavioral and physiological processes, deeper developmental changes, which are irreversible within the lifetime of the individual, or genetic adaptation reversible only over generations (*e.g.*, Gause, 1942). If plasticity at any level must be conserved, Bateson argues, the system should match the level at which adaptation takes place to the frequency with which the environment changes. Plasticity which is reversible on a short notice is wasted if it is used to adapt to conditions which change only rarely; plasticity which operates on a long time scale is ineffective in dealing with short-term disturbances. The situation is even more complicated by the existence of negative interactions between genetic adaptation and phenotypic plasticity. Gause (1942) suggested such interactions, based on experiments with salt tolerance in *Paramecium.* Much the same phenomenon seems to lie behind the negative genetic correlations in crop plants between average yield and the sensitivity of yield to stress (Rosielle and Hamblin, 1981).

The formal approach of Ashby and Bateson clarifies the interaction of plasticity and homeostasis, and highlights the difficulty of assessing the adaptiveness of phenotypic differences merely by seeking their genetic basis. However, it leaves several important questions unanswered. It is not clear what the essential variables are, nor how they are to be distinguished from the regulatory variables. Only the most rudimentary description of the individual life cycle is included. Finally, this approach focuses on the allocation of regulatory variability, but assumes that such variability is correctly organized to protect the essential variables. It ignores the problem of what that organization entails. This last problem may be approached by fitness sets.

A fitness set approach. Levins (1968*a*) introduced fitness sets to deduce adaptive strategies in variable environments. As a basically strategic approach, it has little to say about the choice between adaptation

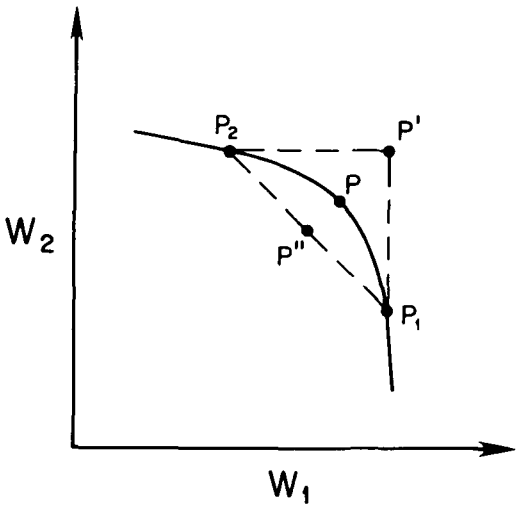

FIG. 2. Phenotypic plasticity in a fitness set analysis (after Levins, 1968*a*). The axes on the graph represent fitness in environments 1 and 2, respectively. If a phenotype originally fixed at P becomes flexible, and is able to correlate its development with the upcoming environment, developing into P_1 in environment 1 and P_2 in environment 2, its effective fitness is increased to that of P'. In the absence of a correlation with the upcoming environment, its effective fitness is reduced to a point on the line connecting P_1 and P_2 (*e.g.*, P").

at somatic or genetic levels, but assuming at the outset that plasticity is involved, it can be used to assess its adaptiveness. Levins (1963, 1968*b*) presented the following analysis. Consider the fitness set shown in Figure 2. Suppose that a genotype canalized to produce phenotype P becomes plastic, and responds to some cue correlated with upcoming environments, so as to develop to phenotype P_1 in environment 1 and phenotype P_2 in environment 2. Its effective fitness is changed to P'; such a shift is clearly adaptive. This result depends crucially on a sufficiently positive correlation of the developmental plasticity with the upcoming environment. With a convex fitness set, even a zero correlation, leading to phenotype P", reduces the effective fitness.

Levins (1968*a*, 1969*a*) also introduced the important distinction between cogradient and contragradient variation (see Berven *et al.*, 1979 for a detailed discussion). In cogradient variation, the phenotypic response (the norm of reaction) and the genetic response to a given factor are parallel; in contragradient variation they oppose each other. It is tempting to use the existence of contragradient variation as evidence for the maladaptiveness of the norm of reaction. This is probably often true, but not always. When an adaptive response switches from one level to another, it need not affect the same traits in the same way. For example, panting with a sudden increase in altitude is almost certainly an adaptive physiological response. Yet, the essential variable here has to do with oxygen uptake, and that can be protected in many ways. Populations living permanently at different altitudes might respond to selection by increasing lung capacity, hemoglobin levels, etc. If they did, high altitude populations might well have lower ventilation rates than low altitude populations at any given oxygen level (Frisancho, 1975). The physiological and genetic responses would exhibit contragradient variation in rate of breathing, yet both would be adaptive.

A DEMOGRAPHIC FRAMEWORK

The analysis of plasticity in life-history traits must be able to incorporate environmental variability, plasticity in response variables, homeostasis of essential variables, and the correlation of responses with environmental cues into a demographic framework flexible enough to accommodate a diversity of life cycles. I will use here a generalization of the discrete-state, projection matrix approach of Leslie (1945), Lefkovitch (1965), and others; for details, see Caswell (1978, 1982*a*, *b*). The analysis begins with a life cycle graph (Fig. 3), in which the numbered nodes represent life cycle stages and the arcs indicate the transitions possible between t and t + 1. The dynamics resulting from this life cycle are described by

$$\mathbf{n}(t+1) = \mathbf{A}\mathbf{n}(t) \qquad (1)$$

where $\mathbf{n}(t)$ is a vector whose entries give the abundance of the different stages at time t, and $\mathbf{A}$ is a projection matrix in which a_{ij} is the coefficient on the arc from n_j to n_i.

The demographic analysis of (1) is straightforward. The asymptotic growth rate is given by λ, the largest of the eigenvalues of **A**. The stable stage distribution is given by the corresponding right eigenvector, **w**, defined by $\mathbf{Aw} = \lambda\mathbf{w}$. The reproductive value vector is given by the corresponding left eigenvector, **v**, where $\mathbf{v}'\mathbf{A} = \lambda\mathbf{v}'$. These quantities can be obtained directly from the life cycle graph (Hubbell and Werner, 1979; Caswell, 1982*a*).

The growth rate λ is the customary measure of fitness for life-history studies (see, *e.g.*, Emlen, 1970; Charlesworth, 1980 for justifications and limitations). To evaluate the selective pressure on any trait, it is necessary to evaluate the sensitivity of λ to changes in that trait. This sensitivity is given by Caswell (1978)

$$\frac{\partial\lambda}{\partial a_{ij}} = \frac{v_i w_j}{\langle \mathbf{v},\mathbf{w}\rangle} \qquad (2)$$

for any element in the matrix **A**, where v_i is the i^{th} element of **v**, and w_i the j^{th} element of **w**. The net selective pressure on a trait x which affects many entries in **A** simultaneously is

$$\frac{d\lambda}{dx} = \sum_{i,j} \frac{\partial\lambda}{\partial a_{ij}} \frac{\partial a_{ij}}{\partial x}. \qquad (3)$$

With this framework, it is possible to examine the demographic and evolutionary consequences of plasticity, considered as a trait in itself. The approach is to insert plasticity into some portion of the life cycle, evaluate its effects on dynamics, and then try to infer its consequences for fitness. The first step is to see how plasticity manifests in demographic models.

Demographic Effects of Plasticity

To evaluate the demographic effects of plasticity, I begin by eliminating genetic variation (thereby, taking one step back from the purposes of this symposium), and letting the life cycle graph represent the growth of a clone. In the absence of plasticity, every individual of a given age in such a population would be identical, regardless of the environment it had experienced. The life cycle graph of such a population looks like Figure 3a; the corresponding matrix is a time-invariant Leslie matrix (*i.e.*, a matrix with survival probabilities on the subdiagonal, fecundities in the first row, and zeros elsewhere).

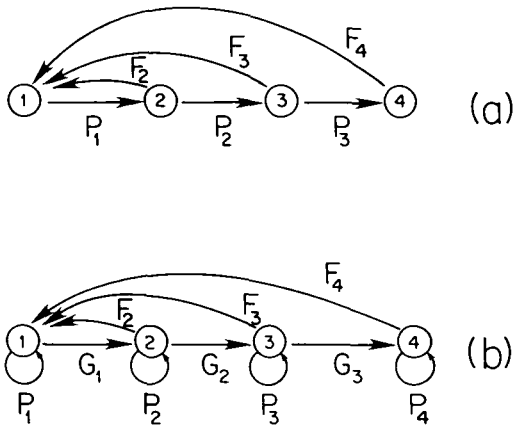

FIG. 3. Life cycle graphs for (a) age-classified populations and (b) size- or stage-classified populations.

If plasticity is present, individual phenotypes will be modified by the environment, with results depending on the pattern of environmental variability. In a uniform environment, the plasticity is irrelevant, all individuals are identical, and **A** is still a constant Leslie matrix. If the environment varies, however, the nature of the projection matrix will change in a manner which depends on the grain of the variation, and on whether the variability is spatial or temporal.

Temporal variation. The effect of temporal variation depends on its scale relative to the time scale of the population projection (*i.e.*, the difference between t and t + 1). If the variation is fine-grained relative to this scale, the demography still appears time invariant, and individuals of the same age are identical. The transition probabilities are averages (the appropriate average may depend on the details of the situation, *e.g.*, Hastings and Caswell, 1979) over the within-time-step variation. For example, if a population is projected on an annual time scale, survival probabilities from year to year are averages (in some sense) of seasonal values, but if the only variation is within years, they will remain constant from year to year. Coarse-grained variation, on

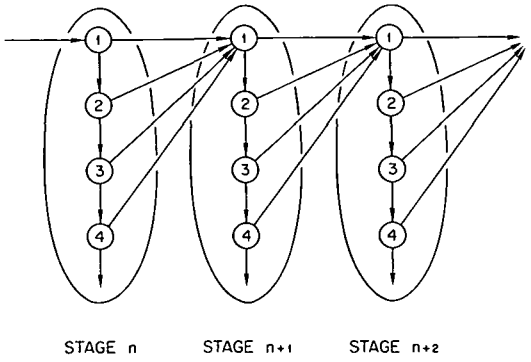

FIG. 4. A life cycle graph for a population with discrete developmental stages ("instars") and variability in development rate. Individuals may remain in an instar for varying lengths of time (indicated by the small numbered circles within each in star) before developing into the next.

the other hand, leads to a time-varying demography:

$$\mathbf{n}(t+1) = \mathbf{A}_t\mathbf{A}_{t-1}\mathbf{A}_{t-2} \ldots \mathbf{A}_2\mathbf{A}_1\mathbf{n}(0). \quad (4)$$

The variability may be cyclic (*e.g.*, Skellam, 1966; MacArthur, 1968) or random (*e.g.*, Cohen, 1979*a*, *b*; Tuljapurkar and Orzack, 1980; Tuljapurkar, 1983). Another possible description is

$$\mathbf{n}(t+1) = \mathbf{A}(t)\mathbf{n}(t) \quad (5)$$

where $\mathbf{A}(t)$ is a matrix of random variables with specified means, variances, and covariances.

Spatial variation. In the presence of plasticity, pure spatial variation generates differences between individuals, based on their location during ontogeny. If the variability is fine-grained relative to the scale on which the population is defined, individuals within the population must be classified according to something other than (or in addition to) age (Wilbur, 1980). Growth rates may be plastic, so that individuals of the same age may be of different sizes (*e.g.*, trees: Daniel *et al.*, 1979; herbaceous plants: Werner and Caswell, 1977; fish: Elson, 1957; Policansky, 1983), or plasticity in development rate may lead to individuals of the same age being in different instars (*e.g.*, cladocerans: Brown, 1929) or developmental stages (*e.g.*, amphibians: Smith-Gill and Berven, 1979; insects: Birley, 1979). A portion of the life cycle graph for such a population is shown in Figure 4. If age is completely irrelevant, or if the population is near a stable age distribution, this graph can be collapsed to the size-classified life cycle of Figure 3b. The resulting projection matrix (see Hartshorn, 1975; Enright and Ogden, 1979 for applications to tree populations) is a time-invariant stage-classified matrix.

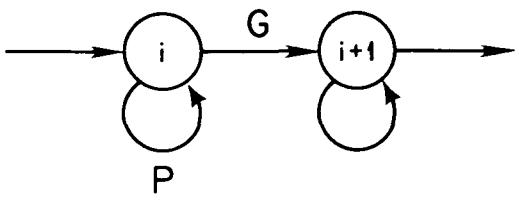

FIG. 5. Two adjacent stages obtained by collapsing the graph of Figure 4.

Coarse-grained spatial variation leads to different subpopulations in different environments, and can be described by multipatch models (*e.g.*, Rogers, 1975), in which $\mathbf{A}$ is a partitioned matrix describing both population growth within and migration between patches.

In summary, then, temporal environmental variability changes the constancy of $\mathbf{A}$, while spatial variability changes the structure of $\mathbf{A}$. In the next section, I will use this framework to incorporate plasticity into several important traits, and go on to examine its evolutionary consequences.

EVOLUTIONARY CONSEQUENCES: THREE EXAMPLES

Plasticity in development rate

Let us consider first a case where fine-grained spatial variability leads to a life cycle graph like that shown in Figure 4. We collapse this graph to the stage-classified description of Figure 3b, and consider the two adjacent stages shown in Figure 5. Development rate determines instar duration, d; because of the environmental variation, d will have a probability distribution f(d) (Sharpe *et al.*, 1977), characterized by a mean $\bar{d}$ and a variance σ_d^2. Since we are

considering a competing-clones model of life history evolution, σ_d^2 is completely environmental in origin, resulting from the developmental plasticity of the organism. What selective pressures act on σ_d^2? Are there situations where increased plasticity, leading to a greater variance in development time, will be favored?

To answer these questions, we need to express the parameters G (the probability of growth from i to i + 1) and P (the probability of remaining in instar i) as functions of $\bar{d}$ and σ_d^2. Suppose that the population has a stable age distribution within each instar, with c_x the proportion of the population which has been in the instar for x time units. Then the proportion of the instar which molts in (t, t + 1) is

$$G = \int_0^\infty c_x f(x)\, dx \Big/ \int_0^\infty c_x\, dx. \qquad (6)$$

Let η_x denote the probability than an individual has not molted by age x (if molting were mortality, η_x would be a survivorship function). Then $c_x = \eta_x \exp(-rx)$ and $f(x) = (-1/l_x)\, d\eta_x/dx$. Thus G simplifies to

$$G = \int_0^\infty e^{-rx} f(x)\, dx \Big/ \int_0^\infty e^{-rx}\eta_x\, dx \qquad (7)$$

where r is the growth rate of the whole population.

Using cumulant-generating functions (see Keyfitz, 1977, pp. 129–131 for an essentially similar calculation in a different context) it can be shown that

$$\ln(G) \simeq r\left[\frac{\sigma_d^2}{\bar{d}}\right] - \ln(\bar{d}) \qquad (8)$$

(assuming that r is small and f(d) approximately normal). Note that if there is no variance, $G = 1/\bar{d}$, which is a frequently used approximation. If the mortality M is independent of age, the probability of remaining in the instar from t to t + 1 is just

$$P = 1 - G - M. \qquad (9)$$

Equations (8) and (9) define P and G, and thus the transition between instars i and i + 1. When will selection favor increases in σ_d^2? We need to calculate the effect of σ_d^2 on fitness:

$$\frac{d\lambda}{d\sigma_d^2} = \frac{\partial\lambda}{\partial G}\frac{\partial G}{\partial\sigma_d^2} + \frac{\partial\lambda}{\partial P}\frac{\partial P}{\partial\sigma_d^2} \qquad (10)$$

$$= \frac{v_{i+1}w_i}{\langle \mathbf{v},\mathbf{w}\rangle}\frac{\partial G}{\partial\sigma_d^2} + \frac{v_i w_i}{\langle \mathbf{v},\mathbf{w}\rangle}\frac{\partial P}{\partial\sigma_d^2} \qquad (11)$$

where the selective pressures on G and P are obtained from Eq. (2). From (8), $\partial G/\partial\sigma_d^2 = Gr/\bar{d}$, and from (9) $\partial P/\partial\sigma_d^2 = -Gr/\bar{d}$. Thus

$$\frac{d\lambda}{d\sigma_d^2} = \frac{Grw_i}{\bar{d}\langle \mathbf{v},\mathbf{w}\rangle}(v_{i+1} - v_i). \qquad (12)$$

Assuming $r > 0$, the direction of selection on σ_d^2 depends on the increment in reproductive value from instar i to i + 1. If this increment is positive, increases in development rate plasticity in instar i will be favored, and vice-versa. Applying the same analysis to $\bar{d}$, it can be shown that $d\lambda/d\bar{d}$ and $d\lambda/d\sigma_d^2$ are of opposite sign. Thus, if $v_{i+1} > v_i$, it pays to decrease $\bar{d}$, minimizing the average time required to get to the stage with the higher reproductive value. The increase of σ_d^2 favored under the same conditions leads to some individuals developing sooner, others later. The conclusion from (12) is that, as long as $r > 0$, the benefit from the former individuals outweighs the cost of the latter.

Since stage-specific reproductive value curves increase over early stages (at least prior to the stage of first reproduction: Caswell, unpublished), and may later decrease, plasticity may be subjected to different selective pressures at different stages in the life cycle. These results predict a positive correlation of σ_d^2 and Δv, and a negative correlation between the mean and variance of instar duration. Since variances in developmental stage duration are themselves variable (Birley, 1979), with the appropriate data, these predictions could be tested.

Temporal variation and fitness

The analysis of coarse-grained temporal variation requires a measure of fitness appropriate to a time-varying projection matrix. Different choices of fitness measures lead to markedly different predictions (Hastings and Caswell, 1979). Here, I will adopt the theory of risk-aversion (borrowed from micro-economics; see Hey

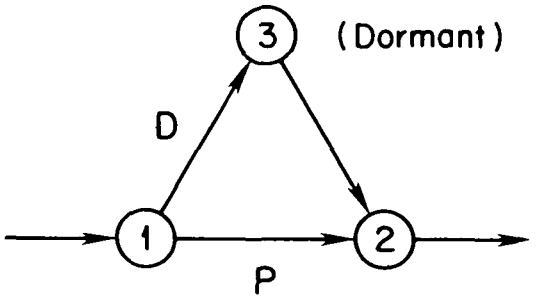

FIG. 6. A portion of a life cycle graph containing a dormant stage (n_3).

[1979] and Keeney and Raiffa [1976], and introduced to population biology by Real [1980*a*, *b*]). This approach is both tractable and flexible; it can be easily related to other measures of fitness in variable environments (*e.g.*, Levins, 1968*a*; Schaffer, 1974; Cohen, 1968; Gillespie, 1977; Eshel, 1981). What follows is a brief summary; for more details, see Real's papers cited above.

Let W denote a measure of fitness (what economists would call a utility function). In a constant environment, we have been assuming $W = \lambda$. In a variable environment, λ will vary, and fitness will be some function, $W = f(\lambda)$. If the utility function follows the so-called von Neumann-Morganstern axioms (von Neumann and Morganstern, 1947), then the optimal strategy under uncertainty is to maximize the expectation of W.

What is the expectation of W? The simplest approximation is

$$E(W) = E(f(\lambda)) \simeq E(\lambda), \qquad (13)$$

but this leads to such difficulties as the expected population size diverging to infinity while the probability of extinction approaches one (Lewontin and Cohen, 1969). A second, more interesting, approximation is

$$E(W) \simeq E(\lambda) - k\sigma_\lambda^2, \qquad (14)$$

where k is known as a coefficient of risk-aversion. If k is positive, selection between strategies with equal mean fitness favors that with the smaller variance in fitness.*

There may also exist risk-prone strategies, in which $k < 0$.

Now suppose that the matrix A is subject to random variation, so that the entries are random variables with expected values $\overline{a_{ij}}$, variances $V(a_{ij})$, and covariances $C(a_{ij},a_{kl})$. For small fluctuations, $E(\lambda)$ and σ_λ^2 can be approximated by

$$E(\lambda) \simeq \lambda(\overline{a_{ij}}) \qquad (15)$$

$$\sigma_\lambda^2 \simeq \sum_{i,j}\left(\frac{\partial\lambda}{\partial a_{ij}}\right)^2 V(a_{ij}) + 2\sum_{i,j}\sum_{k,l}\left(\frac{\partial\lambda}{\partial a_{ij}}\right)\left(\frac{\partial\lambda}{\partial a_{kl}}\right)C(a_{ij},a_{kl}). \qquad (16)$$

Since the partial derivative terms in (16) are given by (2), it is possible to evaluate the contribution of variances and covariances to σ_λ^2.

Here we find a connection with the ideas of Ashby and Bateson on homeostasis: λ itself is playing the role of an essential variable. It is possible to reduce σ_λ^2, and thus increase W, by decreasing $V(a_{ij})$ or producing negative covariance $C(a_{ij},a_{kl})$ between traits. Both of these represent strategies for a regulator to decrease the transmission of variety from the environment to the essential variables. The former could be characterized as a protective strategy, the latter as a reactive strategy.

Plasticity and variance: Multiplicative traits. It is clear that plasticity in morphological, physiological, or behavioral traits may reduce $V(a_{ij})$, but what about plasticity in life-history traits themselves. *A priori*, such plasticity should lead to increased, not decreased variance, but one important exception is the case of traits whose value is determined by the product of a sequence of subsidiary traits. Reproductive output, for example, appears in the life cycle graph as a single parameter, say F_i, where $n_1(t+1) = \Sigma F_i n_i(t)$. But, in fact, reproductive output is the product of a series of allocation parameters, expressed sequentially through the development of the individuals, *e.g.*,

F_i = (pods/plant)(seeds/pod) · (initial seedling survival).

Plasticity in any one of these terms will increase $V(F_i)$, but this variance can be re-

* The geometric mean of λ is one commonly used fitness measure; it is risk-averse with $k = 1/2\lambda$.

duced by negative covariance between components. Such negative correlations among yield components are well-known in crop plants. Adams (1967), Grafius (1961), and Grafius and Thomas (1971) have documented a sequence of developmental determination in such plants, with negative correlations between successive stages. Perturbations of early stages are damped out by plastic responses at later stages; the result is a marked reduction in the variance in final yield.

Plasticity and covariance: Dormancy. Theoretical approaches to adaptive plasticity emphasize the importance of the correlation between the environment and the response (Ashby, 1956; Bateson, 1963; Levins, 1968*b*). Such correlations can produce negative covariance between life-history traits, thereby reducing σ_λ^2 and increasing W. Consider, for example, the problem of dormancy. Many organisms have the possibility of entering a dormant state at some stage in their life cycle, often in response to specific environmental cues (*e.g.*, Villiers, 1975; Dingle, 1978; Tauber and Tauber, 1981). Several theoretical analyses of the phenomenon exist (Cohen, 1970; Levins, 1969*b*; Taylor, 1980*a*, *b*); the following sketch places demographic plasticity explicitly in the foreground.

Figure 6 shows a segment of a life cycle in which an individual may survive directly from n_1 to n_2 with probability P(t), or may become dormant for one time unit with probability D(t), returning to the mainstream of the life cycle one time unit later. Consider first the effect of dormancy on fitness in a constant environment, where $W = \lambda$.

Dormancy affects fitness in two ways: directly through its effect on D, and indirectly through its effect on P (that is, if more individuals remain dormant, fewer proceed directly from n_1 to n_2). The net effect is

$$\frac{d\lambda}{dD} = \frac{\partial\lambda}{\partial D} + \frac{\partial\lambda}{\partial P}\frac{\partial P}{\partial D}.$$

If the dormant individuals are randomly selected from those who would live and those who would die without dormancy,

$$\frac{\partial P}{\partial D} = \frac{-P}{(1-D)}.$$

Thus $$\frac{d\lambda}{dD} = \frac{\partial\lambda}{\partial D} - \frac{P}{(1-D)}\frac{\partial\lambda}{\partial P}.$$

The partial derivatives with respect to P and D can be evaluated using Eq. (2); the result is

$$\frac{d\lambda}{dD} = \frac{w_1}{\langle \mathbf{v},\mathbf{w}\rangle}\left[v_3 - \frac{P}{(1-D)}v_2\right]. \quad (17)$$

Since $v_3 = G\lambda^{-1}v_2$ (Caswell, 1982*a*), an increase in D will be favored only if

$$G\lambda^{-1} > \frac{P}{(1-D)},$$

that is, if survival from n_1 to n_2 is very poor, but "germination" of dormant individuals is very good.

Now suppose that survival varies in response to some environmental factor e, so that $P(t) = \bar{P} + \phi_P e(t)$, and consider the possible evolution of plasticity in dormancy. Plasticity in D would result in $D(t) = \bar{D} + \phi_D e(t)$. (The expressions for P(t) and D(t) assume that the mean value is independent of the sensitivity to e(t); this is probably not true in general [Rosielle and Hamblin, 1981].) How does the extent of plasticity, measured by $|\phi_D|$, affect W?

If e(t) is scaled so that its mean is zero, plasticity will have no effect on E(λ), and will affect W only through σ_λ^2:

$$\frac{dW}{d\phi_D} = -k\frac{d\sigma_\lambda^2}{d\phi_D}. \quad (18)$$

The variance and covariance terms are given by $V(D) = \phi_D^2\sigma_e^2$ and $C(P,D) = \phi_P\phi_D\sigma_e^2$, and the partial derivative terms by $\partial\lambda/\partial D = v_3w_1/\langle \mathbf{v},\mathbf{w}\rangle$ and $\partial\lambda/\partial P = v_2w_1/\langle \mathbf{v},\mathbf{w}\rangle$. Thus,

$$\frac{dW}{d\phi_D} = \frac{-2k\sigma_e^2v_3w_1^2}{\langle \mathbf{v},\mathbf{w}\rangle}(\phi_Dv_3 + \phi_Pv_2). \quad (19)$$

If the population is risk-averse, so that $k > 0$, then increases in plasticity are favored only if ϕ_D and ϕ_P are of opposite sign, that is, only if there is negative covariance between P and D. Such a population would be expected to evolve a dormancy response to the same environmental factor which reduces the survival probability (or to some cue correlated with this factor). The intensity of this selection is greater, the more risk-averse the population and the greater the environmental variance. An optimum

level of plasticity in dormancy is reached when

$$D = -\frac{v_2}{v_3}\phi_P = -\phi_P\frac{\lambda}{G}. \qquad (20)$$

CONCLUSIONS AND PROSPECTS

These three examples have shown how plasticity in life-history traits may have beneficial effects, increasing expected fitness, reducing variance, or generating negative covariance between traits. It is clear, however, that much remains to be done to incorporate phenotypic plasticity into life history theory. Some conspicuous unsolved problems include:

(1) The incorporation of genetics, purposely excluded from the analysis here. Without this inclusion, it is impossible to examine trade-offs between genetic and purely phenotypic adaptation (Bateson, 1963), or the distinction between cogradient and contragradient selection (Levins, 1968*a*).

(2) The incorporation of different modes of plasticity. There is an important distinction between continuously variable plasticity ("dependent morphogenesis" in the sense of Schmalhausen [1949] or "modulation" in the sense of Smith-Gill [1983]) and discontinuous developmental switches (the "auto-regulative development" of Schmalhausen [1949], or the "conversion" of Smith-Gill [1983]). The approach here has focused on modulation, although conversion is *a priori* more likely to be adaptive.

(3) Approximations. All of the analyses presented here rely on a number of approximations, assuming that environmental change and phenotypic response are both small enough they can be approximated by continuous, differentiable, and even linear functions. Many conspicuous types of phenotypic plasticity, however, involve major changes, confronting the organism with life or death decisions which may render such approximations invalid. Like most life-history theory, I have also assumed that the population is near its stable age distribution; severe environmental fluctuations make this assumption less likely.

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