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Evolution, Vol. 40, No. 5. (Sep., 1986), pp. 893-913.

Stable URL:
http://links.jstor.org/sici?sici=0014-3820%28198609%2940%3A5%3C893%3ATEOPPI%3E2.0.CO%3B2-F

Evolution is currently published by Society for the Study of Evolution.

EVOLUTION

INTERNATIONAL JOURNAL OF ORGANIC EVOLUTION

PUBLISHED BY

THE SOCIETY FOR THE STUDY OF EVOLUTION

Vol. 40 **September, 1986** **No. 5**

Evolution, 40(5), 1986, pp. 893–913

THE EVOLUTION OF PHENOTYPIC PLASTICITY IN LIFE-HISTORY TRAITS: PREDICTIONS OF REACTION NORMS FOR AGE AND SIZE AT MATURITY

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Abstract.—We used life-history theory to predict reaction norms for age and size at maturation. We assumed that fecundity increases with size and that juvenile mortality rates of offspring decrease as ages-at-maturity of parents increase, then calculated the reaction norm by varying growth rate and calculating an optimal age at maturity for each growth rate. The reaction norm for maturation should take one of at least four shapes that depend on specific relations between changes in growth rates and changes in adult mortality rates, juvenile mortality rates, or both. Most organisms should mature neither at a fixed size nor at a fixed age, but along an age-size trajectory. The model makes possible a clear distinction between the genetic and phenotypic components of variation. The evolved response to selection is reflected in the shape and position of the reaction norm. The phenotypic response of a single organism to rapid or slow growth is defined by the location of its maturation event as a point on the reaction norm.

A quantitative test with data from 19 populations and species of fish showed that predictions were in good agreement with observations ($r = 0.93$, $P < 0.0001$). The predictions of the model also agreed qualitatively with observed phenotypic variation in age and size at maturity in humans, platyfish, fruit flies, and red deer. This preliminary success suggests that experiments designed to test the predictions directly will be worthwhile.

Received August 19, 1985. Accepted May 7, 1986

This paper develops predictions for norms of reaction of the maturation event, then makes preliminary tests of the predictions. A norm of reaction represents the range of potential phenotypes that a single genotype could develop if exposed to a specified range of environmental conditions (Woltereck, 1909). Life-history traits are directly involved in survival and reproduction: size at birth, age and size at maturity, age- and size-specific birth and death rates, and so forth (Cole, 1954).

There are at least two reasons to introduce predictions about norms of reaction to work on life-history evolution. Although life-history theory is well-developed (Cole, 1954; Lewontin, 1965; Gadgil and Bossert, 1970; Schaffer 1974*a*, 1974*b*, 1979; Charlesworth and Leon, 1976; Leon, 1976; Michod, 1979; Law, 1979; Charlesworth, 1980; reviews in Stearns, 1976, 1977, 1980, 1982*a*, 1982*b*) and has had some success (e.g., Wilbur and Collins, 1973; Stearns and Crandall, 1981; papers in Dingle and Hegmann, 1982; reviews in Stearns, 1982*a*, 1982*b*; Stearns and Crandall, 1984; Roff, 1984), strong tests of the theory are not easy to carry out because tests of many predictions require that a response to selection be observed. Thus one reason to develop predictions for norms of reaction of life-history traits is the hope that some predictions will be testable within a single generation, or on existing data gathered for other purposes, and that the num-

ber of points at which the theory can be brought into risky contact with experiment and observation will be increased.

Second, although we have known since the work of Johannsen (1909) and Woltereck (1909) that the evolution of norms of reaction has important implications for ecology and genetics (Schmalhausen, 1949; Simpson, 1953; Waddington, 1957; Levins, 1968; Dobzhansky, 1970), and although population geneticists have long recognized that norms of reaction play an important role in their theory (Wright, 1931; Lewontin, 1974; Gupta and Lewontin, 1982; Via and Lande, 1985), we know of no predictions about the shape and position that norms of reaction should take. Thus a second reason for this work is to develop explicit predictions of norms of reaction in the hope that they will prove useful to evolutionary theory in general.

We set out to answer a specific question. How should an organism encountering an unavoidable stress that results in slower growth alter its age at maturity to keep fitness as high as possible despite the constraints imposed by slower growth? Much evidence suggests that such adjustments are possible and are made by many types of organisms. The question is whether the observed changes in age and size at maturity can be interpreted as life-history adaptations that maximize fitness under given constraints.

Background

The evidence comes from diverse sources. Among fish, faster growing plaice (Pitt, 1975) and Arctic char (Grainger, 1953) mature earlier and at the same size as slower growing ones. In rainbow trout, brown trout, and sticklebacks (Scott, 1962; Bagenal, 1969; Wootton, 1973), the fastest growing fish mature earliest. Some populations of Atlantic cod mature earlier and at smaller sizes than others (Pinhorn, 1969). Alm (1959) reviewed many cases in which more slowly growing fish matured later and at a smaller size, and a few cases in which they matured earlier and at a smaller size. Male platyfish (McKenzie et al., 1983) delay maturity and mature at smaller sizes when forced by food or temperature stress to grow more slowly, as do laboratory rats (Kennedy and Mitra, 1963) and wild house mice (Barnett, 1965, 1973). More slowly growing male slider turtles delay maturity and mature at smaller sizes, while more slowly growing females mature at the same age as rapidly growing females and at considerably smaller sizes (Gibbons et al., 1981).

Well-fed human females attain menarche at 12–13 years of age and are fit to conceive at 18, whereas food-stressed 19th century women attained menarche at 15–17 and were fit to conceive at 22 (Frisch, 1978). Bongaarts (1980) presents substantial evidence that malnutrition delays menarche in humans (but see Bullough, 1981). Well-fed women reach menarche 2–5 years earlier than malnourished women, and they appear to do it by growing faster and achieving a larger body size at maturity.

Thus five patterns are reported in the literature. When organisms are forced to grow more slowly, they 1) mature later at a smaller size, 2) mature later at the same size, 3) mature later at a larger size, 4) mature earlier at a smaller size, or 5) mature at the same age at a smaller size. As Gibbons et al. (1981) show, the two sexes can have different responses, e.g., (1) for males and (5) for females.

Part of this diversity of responses probably represents genetically based local adaptation rather than variation along a norm of reaction. Such cases include the Atlantic cod and slider turtles mentioned above. Part of this diversity also results from the diversity of factors influencing growth rates. Food stress produces different responses than temperature stress, and low temperatures during embryonic development produce different responses than do the low winter temperatures encountered by juvenile fish (Brett, 1979). The social mechanisms involved in intraspecific competition can interact with density and food ration to produce complex effects on growth rates (Magnuson, 1962), and can alter predation risk through differential exposure in natural situations (Hamilton, 1971; Werner et al., 1983).

Many other complications are possible and likely. It would be self-defeating to represent all ecological and behavioral complexities in these models. We have assumed that generalized "stress" affects growth and

that no matter what environmental factors cause them, changes in growth rates have similar impact on life-history evolution. We note, however, that if different factors produce different norms of reaction despite their identical effects on growth rate, the different responses have probably evolved because the differences among the factors are cues to differences in the association of changes in growth rates with changes in adult and juvenile mortality rates. These things we have modelled. In any particular case, close empirical analysis of the impact of different factors may be necessary to apply the models correctly.

The consequences of growth for the evolution of age at maturity have been explored by Gadgil and Bossert (1970), Wilbur and Collins (1973), Wilbur (1975), Schaffer and Elson (1975), Schaffer (1979), Stearns and Crandall (1981), and Roff (1984). Roff's paper summarizes a large body of fisheries data and should be compared with this one. The idea that age at maturity is determined by a balance between the advantages of short generation time and the advantages of large size is widespread in the life-history literature. The use of life-history theory to predict reaction norms was suggested by Stearns (1983) and developed by Stearns and Crandall (1984). This paper differs from Stearns and Crandall (1984) in that the theory has been simplified and more thoroughly explored, the number of case studies has been increased, and the predictions have been tested on additional data sets (with essentially the same results).

The Model

General Model: Assumptions and Definitions

This model assumes that a) fitness is defined by the Malthusian parameter r in the Euler-Lotka equation, and therefore that b) populations are in stable age distribution, c) no constraints inhibit evolution near local optima, and d) delaying maturity reduces juvenile mortality rate. It accounts for growth by assuming that fecundity is directly related to size. Thus a late-maturing organism has a higher initial fecundity rate than an earlier-maturing organism with the same growth rate.

Assumptions (a–c) are practical, but assumption (d) requires comment. We assume that parents that are older and larger when they first reproduce make offspring of higher quality with better chances of surviving to maturity. By considering the limiting case in which a newly-born female attempts to give birth, one can see that for most organisms mortality rates of offspring must rise to infinity as age at maturity of mothers decreases to 0. From this it follows that mortality of offspring must decrease in some manner as age at maturity of the mother increases.

There are few data available to justify this assumption, which seems plausible a priori. Stafford (unpubl.) analyzed factors influencing mortality in 107,038 infant deaths (National Center for Health Statistics, 1972) representing a sample for the entire U.S. in 1960–1961. He found that the infant mortality rate per 1,000 live births was high for mothers less than 15 years old (58.7), dropped for teenagers 15–19 years old (32.8), remained low (22.3–25.6) for women between 20 and 39 years old, then rose to 30.6 in women aged 40–44, and to 41.1 in women over 45. The left-hand portion of this relationship, the part covering actual and potential ages at maturity, can be well-fitted by describing juvenile mortality as an inverse function of age at maturity. Thus in at least one organism our assumption is justified. Whether such effects are widespread taxonomically is not yet known.

In some submodels, we assumed that changes in growth rate were associated with changes in juvenile or adult mortality rates. By this we did not mean that changes in growth cause changes in fitness. Instead, we meant to account for cases in which a change in growth rate acts as a reliable signal to the organism that well-defined changes in the demographic environment can be expected. Growth rate thus acts as a cue for upcoming environmental events that cause changes in adult or juvenile mortality and have done so dependably through evolutionary time. The problem is essentially one of life-history evolution in variable environments, and we assume that the optimal life history is one in which r is maximized in each environment encountered. Charlesworth (1980) has shown that this should hold for spatial

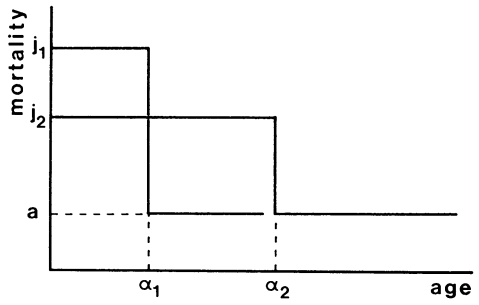

FIG. 1. Examples of mortality for two ages at maturity, α_1 and α_2. Juvenile mortality decreases as maturity is delayed, whereas adult mortality is not affected by age at maturity.

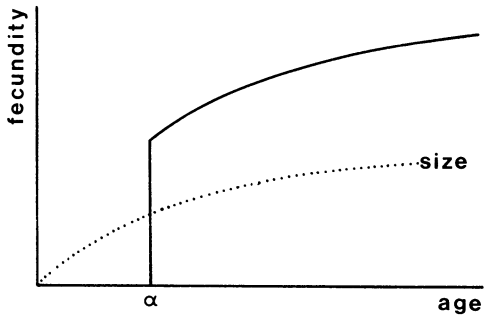

FIG. 2. An example of the relationship between fecundity and size. A linear function (fecundity = 2 × size) is shown.

variation affecting growth rate but that for temporal variation it will hold only if the environmental period is long in relation to generation time.

Calculating Optimal Age at Maturity

Definitions of symbols are given in Table 1. Writing x for age, k for growth rate, α for age at maturity, d for instantaneous mortality rate, a for adult mortality rate, and j for juvenile mortality rate, we defined functions for mortality (Fig. 1) as

$$d(x, \alpha, k) = \begin{cases} a(k) & \text{for } x \geq \alpha \\ j(\alpha, k) & \text{for } x < \alpha \end{cases}$$

and for fecundity (Fig. 2) as

$$m(x, k) = \begin{cases} b(s(x, k)) & \text{for } x \geq \alpha \\ 0 & \text{for } x < \alpha \end{cases}$$

where b represents birth rate and $s(x, k)$ determines size of organism.

According to our assumptions, fitness (r) is defined by the Euler-Lotka equation

$$\int_{\alpha}^{\infty} b(x, .)l(x, .)e^{-rx}\, dx = 1 \qquad (1)$$

where

$$l(x, .) = \exp\left[-\int_0^x d(x, \alpha, k)\, dx\right]$$
$$= \text{survivorship.}$$

Any value of age at maturity, α, at which fitness is locally maximal must satisfy

$$\frac{\partial r}{\partial \alpha} = 0 \qquad (2)$$

and

$$\frac{\partial^2 r}{\partial \alpha^2} < 0. \qquad (3)$$

As is shown in the Appendix, Equation (2) results in a curve, r_c, defining locally stationary values of r in terms of α,

$$r_c = \frac{1}{\alpha} \ln\left\{\frac{b(\alpha)l(\alpha)}{\dfrac{\partial[\alpha(a - j)]}{\partial \alpha}}\right\}, \qquad (4)$$

while Equation (3) limits α to those values satisfying

$$\frac{\partial r_c}{\partial \alpha} > 0. \qquad (5)$$

If α is restricted to those values that satisfy (5), optimal age at maturity can be found by inserting Equation (4) into the Euler-Lotka relation (1) and solving the resulting equation for α, which is equivalent to finding the intersection of curves (1) and (4) (Fig. 3). In examining this figure, one should recall that r_c is determined by taking the derivative of the Euler-Lotka equation with the integral equal to *some* constant c, and the intersection of the two curves in Figure 3 locates the point where this constant—the integral in the Euler-Lotka equation—equals 1. The value of α for which r_c is maximal, α_{max}, may be attained for a value of α at which the Euler-Lotka equation is not 1. If Eq. (5) holds, then α_{max} must come to the right, at a later age, than the optimal r satisfying the Euler-Lotka equation.

To summarize the argument, we can say

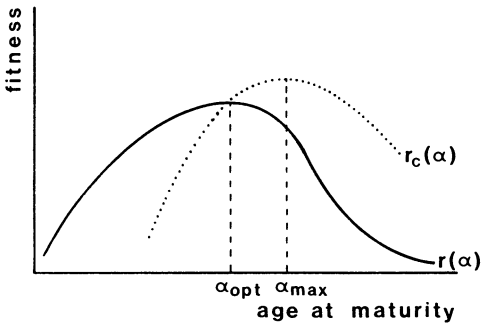

FIG. 3. For any age at maturity (α), there will be a value of r at which the derivative of r with respect to $\alpha = 0$ (ignoring discontinuities). These values of r define a curve, r_c, that represents one of the three conditions necessary for a local optimum in r with respect to α: a) first partial derivative = 0, b) second partial derivative negative, and c) integral in Euler-Lotka equation = 1. The second condition (b) implies that the partial derivative of r_c with respect to α must be positive, and this in turn implies that the maximum of r_c must be to the right of α_{opt}, as indicated in the figure. In brief, to find optimal age at maturity, one looks for the intersection of $r(\alpha)$, the curve defined by the Euler-Lotka equation, with $r_c(\alpha)$, the curve defined by $\partial r/\partial\alpha = 0$, provided that the intersection occurs where $\partial r_c/\partial\alpha > 0$.

TABLE 1. List of symbols.

Symbol	Meaning
x	age
α	age at maturity
r	Malthusian parameter, defined by Euler-Lotka relation
r_c	function defining critical r, defined by $\partial r/\partial\alpha = 0$
s	size (defined by Von Bertalanffy equation)
W	weight
l	length
k	growth rate
A	limiting size
B	difference between limiting size and size at birth, expressed as a proportion of limiting size
b	fecundity of mature organism
F	rate at which fecundity increases with size
H	intercept of line relating fecundity to size
a	adult mortality
a_0	adult mortality for growth rate equals 1
τ	power to which inverse of growth rate is raised
j	juvenile mortality
λ	constant shifting juvenile mortality up or down
σ	power to which inverse of growth rate is raised
γ	power to which inverse of age at maturity is raised

that finding optimal age at maturity comes down to finding values of α that simultaneously satisfy

$$\int_{\alpha}^{\infty} b(x)l(x)e^{-r_c x}\,dx = 1$$

and

$$\frac{\partial r_c}{\partial \alpha} > 0$$

where

$$r_c = \frac{1}{\alpha}\ln\left\{\frac{b(\alpha)l(\alpha)}{\frac{\partial[\alpha(a - j)]}{\partial\alpha}}\right\}.$$

Implementation

To define precise functions for mortality, fecundity, and growth, we assumed that adult and juvenile mortality rates are inversely related to growth rate and that delaying maturity decreases juvenile mortality rate. We assumed that fecundity grows linearly with size and that size at a given age follows the Von Bertalanffy equation. Specifically,

$$a(k) = a_0/k^{\tau}$$

$$j(\alpha, k) = \frac{\lambda}{k^{\sigma}\alpha^{\gamma}} + a_0$$

$$b(s(x, k)) = Fs(x, k) + H$$

$$s(x, k) = A(1 - Be^{-kx})$$

where $a(k)$ is adult mortality, $j(\alpha, k)$ is juvenile mortality, $b(s(x, k))$ is fecundity, and $s(x, k)$ is size. Note that fecundity rate increases steadily with age, whereas juvenile and adult mortality rates are independent of age, but dependent on age at maturity. A list of symbols is given in Table 1, and the relations among the variables and submodels are depicted in Figure 4. Note that as λ increases, juvenile mortality rates increase, and as γ increases, the curve relating juvenile mortality rates of offspring to age-at-maturity of parents bends towards the axes. The strength of association between growth rates and juvenile mortality rates is determined by σ; high values indicate a strong association between slow growth and high juvenile mortality. Similarly, τ determines

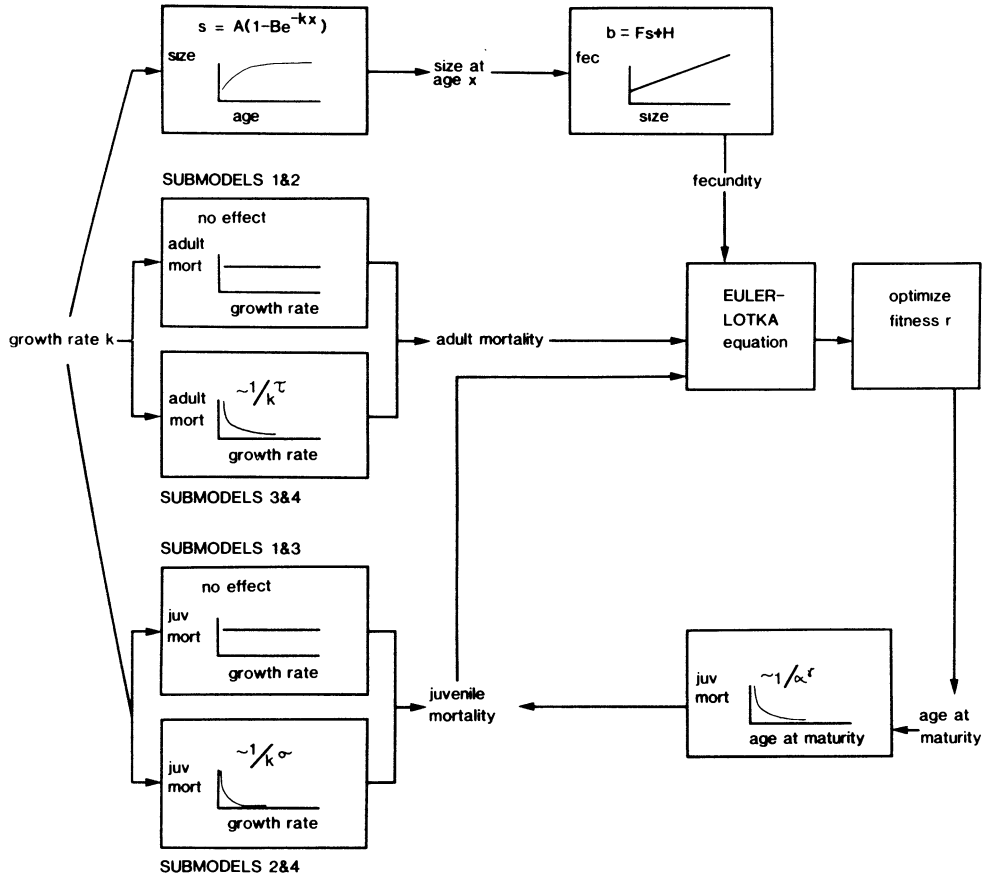

FIG. 4. The flow of the logic and the relationships among the submodels. Growth rates naturally affect size at a given age, and through size they affect fecundity. They can also be associated, not as causes but as correlates, with changes in adult and juvenile mortality rates. Given a certain relationship between fecundity and mortality on the one hand, and age on the other, one can solve the Euler-Lotka equation for the age at maturity that produces the optimal fitness, in this context assumed to be r. This optimal age at maturity in turn influences the instantaneous juvenile mortality rate, and this relationship is taken into account in calculating the optimal r.

the strength of association between growth rates and adult mortality rates; when $\tau = 0$, there is no association, and as τ increases, the association becomes stronger.

Inserting these functions into the Euler-Lotka relation (1) and letting $r = r_c$ leads to

$$\frac{(a + r_c)b(\alpha_0, k) + kb(\infty, k)}{(a + r_c)(a + r_c + k)} e^{-\alpha_0(J + r_c)} = 1. \quad (6)$$

Equation (6) can be solved numerically for α_0, which will be the optimal age-at-maturity if, evaluated at α_0,

$$\frac{\partial r_c}{\partial \alpha} > 0.$$

This model, which we now examine in detail, serves only as a base of reference. It is possible to model any trade-offs that can be represented in equations for $a(.)$, $j(.)$, $b(.)$, and $s(.)$, and then solve Eq. (6). We do not think it is more important, more general, or more realistic than the other models that we analyzed and that are mentioned below. What is general is the modeling framework, which provides a list of assumptions that have to be made and parameters that have to be estimated. In any particular case, these assumptions and parameter estimates must be checked and a specific model built for the organism in question. This we did for the case studies reported below.

Intuitive Interpretation

In this framework (Fig. 4), fitness depends on age at maturity both through generation time and through juvenile mortality. Fitness depends on size through the size-fecundity relationship, and size depends on growth rate and age. Delaying maturity decreases juvenile mortality rates and increases size, thereby endowing organisms with a higher lifetime expectation of offspring. In addition, if age at maturity affects juvenile mortality rates strongly enough, then delaying maturity increases the probability that offspring survive to maturity themselves. Delaying maturity can thus increase the probability that an organism will have a greater number of more successful offspring than earlier-maturing organisms.

However, fitness also increases when generation time decreases, thereby increasing the rate of increase in a clone by an effect analogous to compound interest. To reduce generation time, maturity must come earlier.

Thus there is a continual conflict as to whether an organism should mature later (to decrease juvenile mortality rates of offspring and to increase fecundity) or mature earlier (to reduce generation time). Its fitness is optimal when the increase in fitness resulting from the combined effects of reducing juvenile mortality rates and increasing fecundity by growing longer is exactly counteracted by the decrease in fitness resulting from the corresponding increase in generation time.

The interpretation of several crucial parameters—λ, σ, and γ—is more complex. The portion of juvenile mortality rate that is primarily imposed by the external environment is represented by λ. One can think of an increase in λ, for example, as a change in mortality rate caused by the immigration of a size-selective predator that can feed most efficiently on smaller prey. The prey organism might not be able to respond by developing a defense mechanism, but it might be able to alter age at maturity, which would result in changes in juvenile mortality rates caused by *internal* adjustments. Therefore, we think of mortality rates as having an external, inescapable component (λ) and an internal, adjustable and compensatory component linked through trade-offs to other life-history traits.

The *shape* of the curve relating juvenile mortality rates of offspring to age-at-maturity of parent is determined by γ, which is therefore primarily intrinsic, adjustable, and thus subject to selection. The strength of association between changes in growth rates and changes in juvenile mortality rates, is determined by σ, which contains both an intrinsic and an extrinsic component and is thus partially subject to selection.

Together with the parameters determining the growth curve and the fecundity-size relationship, these are the mathematical boundary conditions within which the local optimization of fitness is carried out. They are related to lineage-dependent biological constraints, but only loosely. The parameters simplify the constraints on the organism, and, because they do so for purposes of mathematical convenience, they mix together aspects of biology that would remain separate in other contexts.

With this caveat, one can say that the model parameters express differences among lineages that are related to the constraints implicit in different morphological plans and developmental systems and that the optimization procedure represents fine-tuning carried out by microevolutionary adjustment to local conditions. Because the modeling procedure provides for lineage-dependence, it can be applied to a wide variety of cases with some success (see below). However, one should remember that whatever success is achieved in prediction has nothing to do with comparative biology, because the comparative, lineage-dependent element enters the model only through curve-fitting (e.g., involving growth curves and size-fecundity relationships), not through prediction from deeper assumptions. Success in prediction means only that the phenotype is locally adjusted so that fitness is optimized within a rather narrow framework of constraints.

Behavior of the Model

Shape of the Norm of Reaction for Age and Size at Maturity

To test the behavior of the model, we calculated age and size at maturity over a

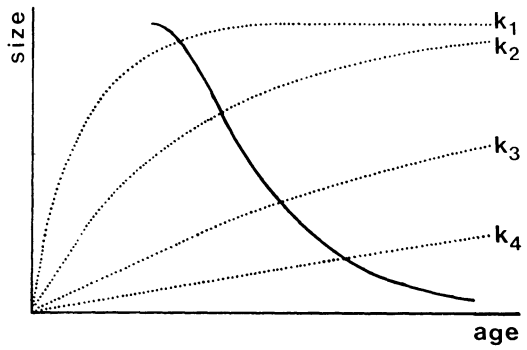

FIG. 5. A reaction norm for age and size at maturity. For each growth rate, k_i, specified, an optimal age at maturity is calculated by solving the Euler-Lotka relation for the α that results in optimal r. By connecting all the points defining optimal age at maturity and the size of the organism at that age, a norm of reaction for age and size at maturity can be defined in age-size space. In the figure, one such reaction norm (heavy solid line) and four growth curves (where $k_1 > k_2 > k_3 > k_4$) are shown.

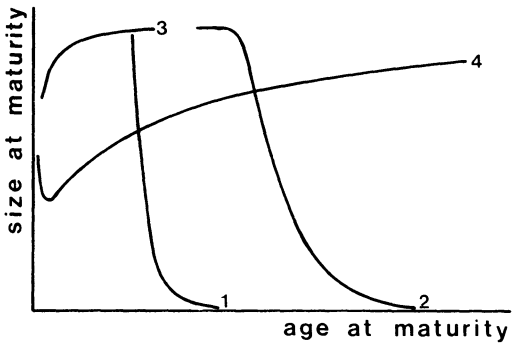

FIG. 6. Four shapes of reaction norms, depending on the way that growth rates are correlated with mortality rates, are predicted. The differences among the submodels that lead to the different predictions are summarized in Table 2: 1) L-shaped, 2) sigmoid, 3) paraboloid, and 4) keel-shaped.

range of growth rates, with all other parameters defining growth, fecundity, and mortality fixed, and plotted the resulting trajectories in age-size space (Fig. 5). The shape of the trajectory connecting optimal ages and sizes at maturity at different growth rates—the norm of reaction—turned out to be very sensitive to the parameters defining mortality rate (Fig. 6). We repeated the numerical analysis of the basic model on four submodels that differ in the way mortality rate depends on growth rate. These submodels represent different versions of the equations given above for $a(.)$, $j(.)$, $b(.)$, and $s(.)$. It is possible to follow the precise changes made in each model by referring to Figure 4 and Table 2. The reaction norms in Figure 6 form two series, one showing the effects of increasing the association between growth rates and juvenile mortality rates (from L-shaped [1] through sigmoid [2] to parabolic [3]) and the other showing the combined effects of associations between growth rates and both juvenile and adult mortality rates (the combination of the conditions that produce [1] and [2] will produce [4]). For all the sets of parameters tested, as growth rate decreased, age at maturity increased. Whether size at maturity also decreased depended on the submodel (in 2 and 4 it could increase) and on the parameters chosen. We did not find a combination of parameters or a submodel in which earlier maturity accompanied slower growth.

Submodel 1: No Dependence of Mortality Rates on Growth Rates.—When neither juvenile nor adult mortality rate changes when growth rate changes, the norm of reaction of the maturation event (the maturation trajectory) is L-shaped. Independence of mortality rates from growth rates is achieved by setting τ and $\sigma = 0$, so that adult mortality rate $a = a_0$, and juvenile mortality rate $j(\alpha) = (\lambda/\alpha^\gamma) + a_0$.

An L-shaped trajectory (trajectory 1 in Fig. 6) implies that over a range of fairly high growth rates, the organisms will appear to mature at fixed age, changing only their size at maturity, whereas over a range of fairly low growth rates the organisms will appear to mature at fixed size, changing only their age at maturity. This behavior is due to the different impact that a given change in growth rate has on fecundity at high and at low growth rates. For rapid growth, although a small decrease in growth rate has a large effect on fecundity at a given age (Fig. 7), a small increase in age will increase fecundity back to the former level. An organism must therefore delay maturity only slightly to maintain its fecundity rate. In contrast, when growth rates are small, age at maturity must be greatly increased to maintain a given fecundity rate.

Thus with slower growth, surviving to a

TABLE 2. List of submodels used to analyze model. Four submodels differing in the way that mortality depends on growth rate were analyzed. Each submodel revealed one or two distinct shapes for the trajectory connecting optimal age and size at maturity. The shapes of the trajectories are defined in Figure 6.

Submodel	Trajectory
1) No dependence of mortality on growth rate	L-shaped
2) Juvenile mortality increases as growth rate decreases	sigmoid-shaped for low σ* paraboloid for high σ
3) Adult mortality increases as growth rate decreases	L-shaped
4) Juvenile and adult mortality increase as growth rate decreases	keel-shaped

* σ is a measure of the effect of growth rate on juvenile mortality, i.e., low σ means little effect of growth rate, and high σ means large effect of growth rate.

size at which fecundity is high enough to produce a positive r becomes more important than reducing generation time. If the organism matures early enough, it will be so small and have such low fecundity that r will be negative. The optimal r must be positive to be biologically meaningful, and if slow growth and early maturation imply negative r values, then the optimal r must be associated with later maturity as growth slows. This conclusion is implicit in the relations of fecundity to size and of size to age.

Submodel 2: Juvenile Mortality Changes with Growth Rate.—This variation was implemented by holding $\tau = 0$ and letting σ take on different values, so that adult mortality rate $a = a_0$ and juvenile mortality rate $j(\alpha, k) = (\lambda/k^{\sigma}\alpha^{\gamma}) + a_0$. When σ is small, the association of growth rates and juvenile mortality rates is weak, but as σ increases, decreases in growth rates imply large increases in juvenile mortality rates. Figure 8 illustrates how trajectories of optimal age at maturity change shape as the growth-juvenile mortality relation strengthens.

Now consider the changes in juvenile mortality rate that strongly influence this change in the norm of reaction. For an age at maturity of 50 days, for $\lambda = 100$, for adult mortality $a_0 = 0.01$, and for rapid growth ($k = 0.33$), a change in σ from 0.0 to 0.3 only changes the juvenile mortality rate, j, from 0.060 to 0.076. However, for similar parameter values and slow growth ($k = 0.01$), a similar change in σ changes the juvenile mortality rate from 0.060 to 0.179. In all versions of these models, increases in juvenile mortality rates lead to delays in maturity. Therefore, since juvenile mortality rates increase more rapidly at low than at high growth rates, the delays in maturity are more pronounced at low than at high growth rates (Fig. 8), and the slopes of the maturation trajectories, which are negative, increase.

When the association of growth and juvenile mortality rates is moderate (e.g., $\sigma = 0.2$), the maturation trajectories become sigmoidal. With a sigmoidal maturation trajectory, size at maturity will appear to be fixed over ranges of high or low growth rates, while over a range of intermediate growth rates, size at maturity changes rapidly. If the association of growth rate and juvenile mortality rate becomes strong enough, the slope of the maturation trajectory will actually become positive: as growth rate decreases, both age and size at maturity increase, and the reaction norm looks like a paraboloid.

Submodel 3: Adult Mortality Changes with Growth Rate.—When adult mortality rate increases as growth rate decreases, and juvenile mortality rate is independent of growth rate, the trajectories are again L-shaped. They differ from the maturation trajectories of submodel 1, where growth rates and mortality rates are not associated, mainly in that their slope is greater. For growth rates less than one, age at maturity is greater in this model than in submodel 1.

Increasing adult mortality results in a shorter reproductive period, and therefore in lower lifetime fecundity. By delaying maturity, an organism can increase its fecundity at the onset of maturity without changing its reproductive period, thereby compensating for the loss in lifetime fecundity.

Submodel 4: Both Adult and Juvenile

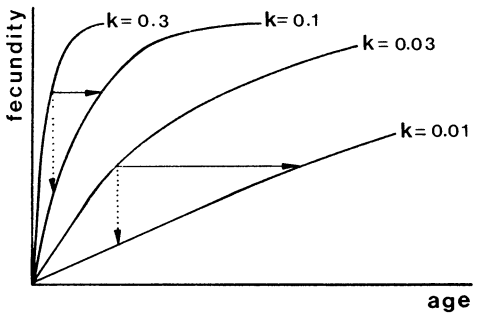

FIG. 7. The effects on fecundity of a given change in growth rate are different for rapid and for slow growth. For rapid growth, decreasing growth rate by a factor of three decreases fecundity markedly at a given age (dotted arrow). However, by increasing age only slightly, fecundity is raised back to its former level (solid arrow). For slow growth, a much greater increase in age is needed to compensate a similar change in growth rate. This explains the L-shape of some reaction norms.

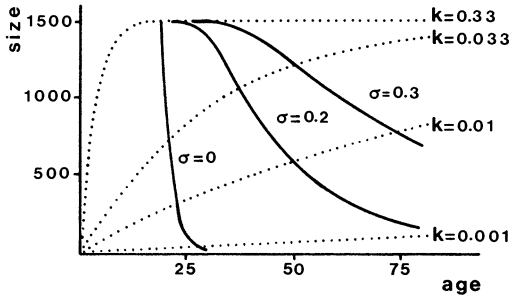

FIG. 8. Increasing the correlation between growth rates and juvenile mortality rates (i.e., increasing σ) increases the slope of the reaction norm.

Mortality Rates Change with Growth Rate.—When both adult and juvenile mortality rates increase as growth rates decrease, the maturation trajectories become keel-shaped. Along the portions of the trajectories to the left, where growth is rapid, organisms decrease their size at maturity and increase their age at maturity as growth rates decrease (Fig. 6). In contrast, along the portions of the trajectories to the right, where growth is slower, the model implies that it is advantageous to delay maturity so much that size at maturity is increased. This increase in size, however, occurs only for such slow growth, and for such a strong relationship between growth rates and adult mortality rates, that adult mortality exceeds juvenile mortality by a factor of 100 or more—a condition that is not likely to be encountered in nature.

Position of the Maturation Trajectory

The position of the maturation trajectory in age-size space depends on the fecundity rates and mortality rates specified. Age at maturity is most sensitive to juvenile mortality rate. Increases in λ and decreases in γ both delay maturity greatly (Fig. 9). Increases in adult mortality rates, although they also move the maturation trajectory to the right, have much less impact on age at maturity than similar changes in juvenile mortality rates. Changes in fecundity rates, however, have a strong influence on age at maturity. When the rate at which fecundity increases with size itself increases, the optimal maturation age shifts to the left: the organisms mature earlier at all growth rates.

These effects can again be explained intuitively. When fecundity increases rapidly with size, an organism can produce a given number of offspring when it is young and small. The fitness advantage comes through decreased generation time. When juvenile mortality rates are increased, the age at maturity up to which r is negative comes later in life because the organisms must grow longer to attain a high enough fecundity to have a positive r. In order to have an optimal r, which must be associated with later maturation than a merely positive r, the organism will mature later.

This explanation depends strongly on our assumption that juvenile mortality rate increases as age at maturity decreases. We readily admit that over a certain range of ages at maturity, juvenile mortality rates may not change much as age at maturity decreases. Over this range, decreases in age at maturity can actually lower the overall level of juvenile mortality by decreasing the time during which the juvenile stage is at risk. This influence combines with that of generation time to produce powerful selection for earlier maturity.

However, when maturation occurs much earlier than normal, in any organism, then it interferes with juvenile growth and development, and the resulting offspring, whether eggs or embryos, cannot be properly developed and provisioned. Thus at some point early in the life of all organisms,

the juvenile mortality rate of offspring must start to increase as maturation comes earlier, as we know it does in humans. Whether the balance of advantages and disadvantages is determined at a point where increases in juvenile mortality rates are balancing decreases in generation time, or at a point where generation time is balancing increases in fecundity, depends on the parameters used.

Summary of Predictions

Shape of Trajectory.—These models predict that norms of reaction for age and size at maturity should be described by one of four well-defined maturation trajectories (Table 2 and Fig. 6). In all cases, maturity is delayed as growth rates decrease, but the particular shape of the trajectory depends on whether or not changes in growth rates are associated with changes in adult mortality rates, juvenile mortality rates, or both, and if so, then how strongly associated.

Position of Trajectory (ceteris paribus).—We see the position of the average maturation trajectory of a population as the genetically determined prerequisite for individual development. It summarizes the evolutionary response of a particular organismic design to the demographic conditions encountered in the past, given the internal constraints of its lineage. For evolutionary equilibrium, then a) increasing the extrinsic juvenile mortality rate shifts the trajectory to the right—the organisms mature later; b) increasing the intrinsic rate of gain of fecundity with size shifts the trajectory to the left—the organisms mature earlier; and c) increasing the extrinsic adult mortality rate shifts the curve to the right—the organisms mature later, but not as late as they would for an equivalent change in juvenile mortality rate.

Position of the Maturation Event for a Particular Organism along the Maturation Trajectory.—A maturation trajectory describes the potential sizes and ages at maturity of a given genotype. In these models, organisms that are forced by external conditions to grow more slowly always delay maturity, but the precise manner in which they do so depends on conditions summarized in Table 2 and Figure 6. In practice, to predict a particular age and size at maturity, or to predict the shape of the maturation trajectory, one must define all the relevant parameters on which the predictions depend: adult and juvenile mortality rates, their association with growth rates, rate of gain of fecundity with size, and growth rates. This is done by building a submodel in each case.

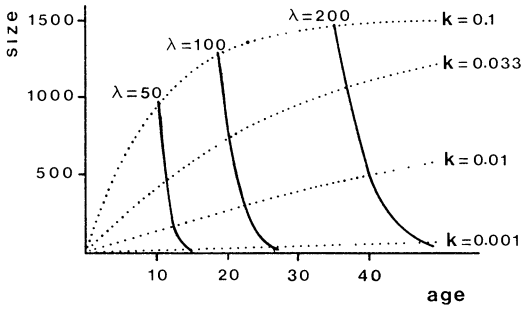

FIG. 9. Reaction norms showing optimal age and size at maturity for three levels of extrinsic juvenile mortality (determined by λ). Increasing λ shifts the reaction norms rapidly to higher ages at maturity, but does not change their shape.

None of the models developed here is appropriate for any particular organism unless a submodel has been worked out, as in the case studies below. This approach is flexible enough to deal with many cases, but the assumptions and parameters appropriate to any given case can only be determined after a careful consideration of the biology of that particular species. The models are not general, but the framework is.

ALTERNATIVE MODELS

Any life-history model that is even moderately realistic contains so many different relationships and so many parameters that one always wonders whether some important result might depend on a particular and sensitive, rather than a general and robust, feature of the model. To test the robustness of the results, four alternative models were analyzed. In each, we changed one of the basic assumptions.

Determinate Growth.—In this model, we assumed that the organism stops growing when it matures. This was the model used below for human females, and it would hold in general for organisms with determinate growth. Fecundity remains constant throughout the reproductive period, de-

pending only on juvenile growth rate and age at maturity:

$$b(x, k) = FA(1 - Be^{-k\alpha}) + H = b(\alpha, k).$$

All of the trajectories resulting from this model can be described by the shapes given above. They are L-, sigmoid-, paraboloid-, or keel-shaped. Under determinate growth, the model reacts to changes in parameters in the same way as the basic model. For example, increases in juvenile mortality rates shift the maturation trajectory to the right, and increasing the association between growth rates and mortality rates increases the slope of the trajectory. The only qualitative difference between this and the basic model is that the slope of the trajectories is slightly increased.

Fecundity Increases with the Cube of Length.—In this model, we assumed that fecundity increases with the cube of length, or approximately linearly with weight:

$$b(x, k) = FA(1 - Be^{-kx})^3 + H.$$

This model did not differ qualitatively from the basic model.

Juvenile Mortality Rate Decreases Exponentially with Age.—In the third model, we assumed that juvenile mortality, rather than staying constant throughout the juvenile period, decreases exponentially with age:

$$j(x, k) = \left(\frac{\lambda}{k^{\sigma}\alpha^{\gamma}}\right)e^{-kx} + a.$$

This represents a situation in which juveniles are growing out of risk. The principal qualitative difference between this model and the basic model is that the trajectories can become sigmoid-shaped even when juvenile mortality is independent of growth rate ($\sigma = 0$). As in the basic model, delaying maturity increases the probability that offspring will survive to maturity. In contrast to the basic model, the increase in survival is greatest for high growth rates, so that the tendency to delay maturity is greater for rapid than for slow growth. This leads to sigmoidal norms of reaction.

Juvenile Mortality Decreases with Size at Maturity.—In the fourth model, we made juvenile mortality rates size-dependent rather than age-dependent:

$$j(s(\alpha)) = \lambda[(A - s(\alpha))/A]^{\gamma} + a_0.$$

As in the other models, juvenile mortality rates increase as age at maturity decreases (and thus as the difference between limiting size, A, and size at maturity increases). In this model, however, juvenile mortality will never exceed $\lambda + a_0$. In all the maturation trajectories we have found for this case, both age and size at maturity increase as growth rate decreases (trajectory 3 in Fig. 6, a paraboloid).

Quantitative Test of the Model

These models are capable of predicting norms of reaction (see the case studies below), but they are also capable, given a growth rate, of predicting the location of the maturation event along the optimal trajectory. One encounters data on population means of age and size at maturity in combination with population means of growth rates much more frequently in the literature than one encounters data on the shape of the reaction norm. Therefore we used this feature to test quantitative predictions of age and size at maturation. We compared age at maturity observed in 19 populations of fish with age at maturity predicted by our model (Table 3). We only used data sets in which growth could be well represented by the von Bertalanffy equation. If the parameters of the equation were not given, we calculated them with the method of Fabens (1965).

We assumed either that fecundity was linearly related to length or that fecundity increased with the cube of length (approximately linearly with weight). The functions were thus either $b = FL + H$ or $b = FL^3 + H$. However, most papers gave fecundity as a function of the form $b = mL^n$. Whenever this function could be well approximated by a straight line, we chose the linear function because it had been used in the sensitivity analysis. If the cubic function fitted the data better, we used it. We limited ourselves to these functions because they can be readily integrated. In no case did we use a data set that these functions would have misrepresented. For example, Mann (1974) reports that the fecundity of dace in the River Stour can be described by

$$\lg(\text{fecundity}) = 4.038[\lg(\text{length})] - 5.474.$$

For lengths between 150 mm and 250 mm

TABLE 3. Predicted and observed age at maturity for 19 populations of fish. For each population, the predicted age at maturity was calculated for two values of γ, a measure of the effect of age at maturity on juvenile mortality. One of two different models was used. The length-model assumes that fecundity is linearly related to length, whereas the weight-model assumes that fecundity is linearly related to the cube of length.

Species	Location	Reference	Model	Observed (years)	Predicted ($\gamma = 2$) (years)	Predicted ($\gamma = 1.5$) (years)
Roach	R. Stour, England	Mann (1973)	weight	4.5	6.5	5.4
Dace	R. Stour, England	Mann (1974)	weight	4.3	4.8	4.3
Dace	R. Frome, England	Mann (1974)	weight	3.9	5.0	4.2
Turbot	North Sea	Jones (1974)	weight	4.0	7.0	6.0
Upland bully	New Zealand	Staples (1975)	length	1.0	1.1	0.9
Pike	R. Stour, England	Mann (1976)	weight	2.2	3.5	3.1
Haplochromis mloto	Lake Malawi, Malawi	Tweddle and Turner (1977)	length	1.4	1.4	1.0
Haplochromis intermedius	Lake Malawi, Malawi	Tweddle and Turner (1977)	length	1.9	1.7	1.2
Lethrinops parvidens	Lake Malawi, Malawi	Tweddle and Turner (1977)	length	3.1	1.6	1.5
Roach	L. Volvi, Greece	Papageorgiou (1979)	weight	3.8	4.3	4.0
Gudgeon	R. Frome, England	Mann (1980)	weight	2.1	3.0	2.4
Painted greenling	Monterey, U.S.A.	DeMartini and Anderson (1980)	weight	3.0	4.0	3.0
Painted greenling	Seattle, U.S.A.	DeMartini and Anderson (1980)	weight	3.2	4.7	3.7
Mosquito-fish	Garden Island, Australia	Trendall (1981)	length	0.11	0.09	0.09
Mosquito-fish	Oakley Dam, Australia	Trendall (1981)	length	0.21	0.15	0.10
Mosquito-fish	Lake Leschenaultia, Australia	Trendall (1981)	length	0.17	0.13	0.12
Mosquito-fish	Mill Point Road, Australia	Trendall (1981)	length	0.65	0.52	0.49
Haddock	North Sea	Hislop (1984)	weight	3.4	4.8	3.9
Cod	North Sea	Hislop (1984)	weight	4.5	6.7	5.6

(the range given by Mann), this curve can be very closely approximated by

$$\text{fecundity} = 0.00115(\text{length})^3 - 2{,}302.$$

When juvenile mortality was not given in the paper, we estimated it by assuming that on average two offspring of the first clutch survive to maturity. The available data did not allow us to calculate γ. However, it can be shown that if this model holds, γ must be greater than 1. We analyzed the data with the next larger integer, $\gamma = 2$, and, to test for sensitivity of the result to changes in γ, with $\gamma = 1.5$ as well. In addition, we assumed that adult and juvenile mortality rates were independent of growth rate, i.e., $\sigma = \tau = 0$.

The results are given in Table 3. For both values of γ tested, the correlation of predicted with observed ages at maturity was very strong. For $\gamma = 2$, $r = 0.93$ ($P < 0.0001$), and for $\gamma = 1.5$, $r = 0.94$ ($P < 0.0001$). In the first case, the standard error of the residuals in the regression of observations on predictions is 0.20, while in the second case, it is 0.16. Under the assumption that $\gamma = 2$, the predicted age at maturity explains 81.7% of the variation in the observed ages at maturity in this data set, while under the assumption that $\gamma = 1.5$, the prediction explains 93.3% of the variation. Similar results have been presented by Stearns and Crandall (1981, 1984) and Roff (1984).

FOUR CASE STUDIES

In order to develop a case study, one must estimate several parameters. The estimates we used for these case studies, and the sources from which we took the data, are given in Table 4.

Human Females.—As noted above, poorly nourished women mature three to four years later and at smaller sizes than do well-fed women. In Figure 10, we show how this difference can be interpreted as a single genotype sliding along a maturation trajec-

TABLE 4. Models and parameters used for case studies.

Case study	Function	Model	Parameters	Reference
Humans	growth	$W = W_\infty(1 - Be^{-kx})$ [no growth after maturation]	$W_\infty = 60$ kg $B = 0.94$ $k = 0.07 \ldots 0.2$	assumption
	fecundity	$b = FW + H$	$F = 0.0375$ $H = -1.25$	assumption
	juvenile mortality	$j = \frac{\lambda}{(k^\sigma \alpha^\gamma)}$	$\lambda = 21$ $\sigma = 1.4$ $\gamma = 3.5$	Bouvier and van der Tak (1976)
Drosophila melanogaster	growth	$l = l_\infty(1 - Be^{-kx})$	$l_\infty = 3$ mm $B = 0.9$ $k = 0.09 \ldots 0.32$	Shorrocks (1972)
	fecundity	$b = Fl + H$	$F = 212$ $H = -358$	Robertson (1957)
	juvenile mortality	$j = \frac{\lambda}{(k^\sigma \alpha^\gamma)}$	$\lambda = 30$ $\sigma = 0.25$ $\gamma = 2$	Sang (1950)
Red Deer				
females	growth	$W = W_\infty(1 - Be^{-kx})$	$W_\infty = 58.7$ kg $B = 0.89$ $k = 0.78$	Clutton-Brock et al. (1982)
	fecundity	$b = FW + H$	$F = 0.015$ $H = -0.054$	Clutton-Brock et al. (1982)
	juvenile mortality	$j = \frac{\lambda}{\alpha^2} + a_0$	$\lambda = 2.6$ $a_0 = 0.08$	Clutton-Brock et al. (1982)
males	growth	$W = W_\infty(1 - Be^{-kx})$	$W_\infty = 87.6$ kg $B = 0.93$ $k = 0.43$	Clutton-Brock et al. (1982)
	fecundity	$b = F(x - x_0)^2 + H$	$F = -0.54$ $H = 13$ $x_0 = 9\ (1.43 - k)^2$	Clutton-Brock et al. (1982)
	juvenile mortality	$j = \frac{\lambda}{\alpha^2} + a_0$	$\lambda = 5.5$ $a_0 = 0.17$	Clutton-Brock et al. (1982)

tory towards earlier maturity at larger sizes achieved through faster growth. This suggests that the ideas presented here are readily applied to events familiar to humans. It also suggests an important point concerning the demographic transition that is well known to human demographers. They may not, however, have realized that the maturation trajectory is predictable under adaptationist assumptions.

When a human population moves through the transition from underdeveloped to developed nation, the first demographic effect is a rapid drop in juvenile mortality achieved through improvements in medical care and nutrition. This effect has been widely recognized as responsible for the high population growth rates causing serious problems in the Third World. There is a simultaneous decrease in age at maturity and generation time that powerfully amplifies the effects of reduced juvenile mortality. Better nourished humans survive better, grow more rapidly, and mature earlier.

Since rate of population increase is most sensitive to changes in age at maturity (under such conditions), populations grow very rapidly in consequence (Cole, 1954; Lewontin, 1965; Caswell, 1978). Thus policies that encourage people in developing countries to delay reproduction will be just as effective, or even more effective, than pol-

icies that encourage people to limit family size. While this remark is not new, we make it here to connect it to the evolutionary explanation of the maturation trajectory that causes the difficulty. That connection is new.

If the reduction in juvenile mortality persists, then we expect an evolutionary response causing a shift of the whole trajectory to the left, further reducing average age at maturity, but only by about another 6 months (Fig. 10). This probably happened once during the shift from scavenging to hunting and gathering, then again during the shift to farming. This model suggests a further, postindustrial shift towards earlier maturity, presupposing no other adaptation affecting life histories.

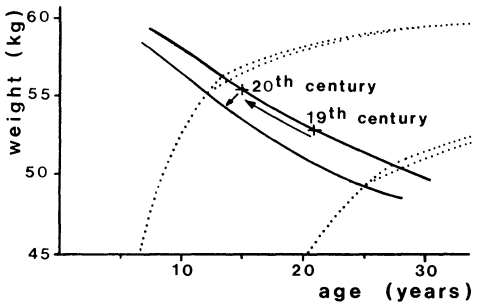

FIG. 10. The predicted reaction norms for human females. The upper reaction norm shows the presumed response to better nutrition that differentiates 19th from 20th century European females (long arrow). The lower reaction norm shows the predicted evolutionary response to lower juvenile mortality rates. The phenotypic response to nutrition is thought to account for most of the observed shift, about 3 years. The eventual evolutionary response to such a change in juvenile mortality could shift maturation another year earlier (short arrow).

Male Platyfish.—McKenzie et al. (1983) have described how age and size at maturity of male platyfish are effected by genes and environment. Male platyfish (Kallman and Schreibman, 1973) are polymorphic for size. Some of the size differences are caused by variation at a single locus. Male poeciliids virtually stop growing when they mature, and the alleles that Kallman found code for distinctly different maturation rules. McKenzie and his colleagues grew male platyfish of two different genotypes over a broad range of environmental conditions, varying temperature, food, water quality, and crowding. Their results (Fig. 11) show clearly distinct sets of maturation events. These sets contain about a hundred points for each genotype and consist of envelopes drawn around the maturation events, each of which is a point in age-size space. The envelopes suggest underlying L-shaped trajectories that would be consistent with the simplest assumption of no association between growth rates and mortality rates.

The variation that generates two envelopes rather than two lines in Figure 11 has two sources: variation at other loci, since genetic background is not controlled, and variation in genotype × environment interactions under different environmental conditions. The fact that the envelopes are clearly separated shows conclusively that the locus tested is a gene with major effects that remain detectable despite a strong attempt to exaggerate phenotypic plasticity. This case suggests that in nature we should expect to encounter families of trajectories generated by differences among genotypes. Only in clones raised in the laboratory will the trajectories themselves be directly measurable.

Drosophila.—Well-fed, uncrowded flies growing at 27°C start to reproduce when they are 11 days old and weigh about 1.0 mg. Poorly nourished or crowded flies start reproducing at 15 or 16 days or later, and weigh about 0.5 mg. This typical difference can be explained as a life-history adaptation to minimize the loss of fitness imposed by slower growth (Fig. 12).

The suggestion that organisms change age and size at maturity according to predictable rules as environmental conditions change has important consequences for population dynamics, which are often modeled in *Drosophila* (Prout, 1984). Their phenotypic plasticity is a form of density compensation, in which uncrowded organisms mature earlier, at larger sizes, and have higher lifetime fecundities than do crowded organisms. This effect is predictable, and we hope it will be included in more realistic models of population dynamics.

Red Deer.—Clutton-Brock et al. (1982) have presented beautifully detailed data on variation in reproductive success of male and female red deer on Rhum. We used their data to build a model that contrasts the maturation trajectories for male and fe-

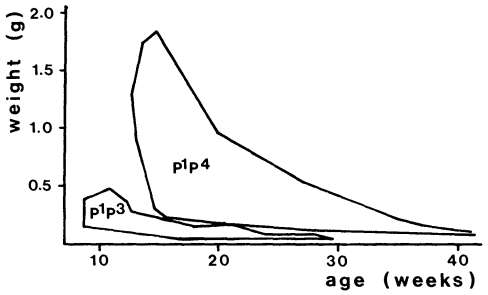

FIG. 11. Male platyfish have L-shaped norms of reaction that resemble those predicted under the simplest assumption of no relation between a change in growth rate and a change in age-specific mortality rates. Genotypic variation and genotype × environment interactions explain why one sees envelopes around the maturation events, each of which is located as a point within the envelope. p^1p^4 and p^1p^3 represent two different genotypes controlling a polymorphism in size at maturity probably maintained as an evolutionarily stable strategy (ESS). The larger morph has more elaborate courtship behavior and a shorter intromittent organ, while the smaller morph has simpler courtship behavior and a longer intromittent organ.

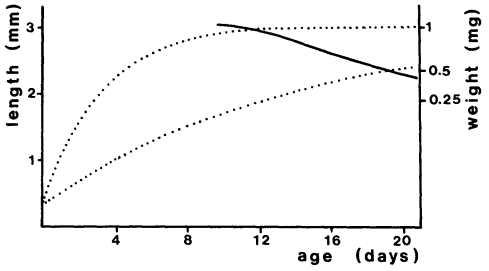

FIG. 12. It is well known that fruit flies delay maturity and mature at smaller sizes when stressed by poor food or high larval densities. The reaction norm shown is the optimal one predicted by our model given boundary conditions determined by experimental data.

male red deer. We assumed that the major consequence of small size in males is reduced access to mates and that variation in growth within a year-class would have to be assessed by comparison with the largest males in the vicinity, who may well have originated in other year-classes. Thus, there will usually be an old, large male in the vicinity with whom maturing males will have to fight in order to get access to mates. Smaller females, on the other hand, reproduce less frequently and produce offspring that have higher mortality rates, but no matter how small they are, they always have access to mates.

The model is consistent with observed differences in age at maturity of male and female red deer on Rhum, and it makes the prediction that variation in female maturation should be primarily size variation in the 3rd to 5th years of life, whereas variation in male maturation should be primarily age variation from the 4th to the 8th years of life (Fig. 13). The team on Rhum continues to gather the data that will test this prediction (Clutton-Brock, pers. comm.).

DISCUSSION

Although the evolution of norms of reaction and the impact of norms of reaction on evolution have long been recognized as important elements of evolutionary theory (Woltereck, 1909; Schmalhausen, 1949; Simpson, 1953; Dobzhansky, 1970), we know of no other predictions of the shape and position of reaction norms. The predictions about the positions of the reaction norms, while consistent with published statements (e.g., Gadgil and Bossert, 1970; Schaffer and Elson, 1975; Bell, 1977; Stearns and Crandall, 1981; Crandall and Stearns, 1982; Roff, 1984), provide new details. They appear to be successful in both qualitative and quantitative tests. We regard this success with some suspicion because the data were gathered for other purposes, and therefore plausible but indirect estimates had to be used to make any test at all. Moreover, these models do not explain why Alm (1959) and Pinhorn (1969) observed fish that mature earlier at smaller sizes when forced to grow more slowly.

Nevertheless the predictions were successful enough to suggest that experiments designed to test them directly would be worth doing, and we are doing them. It also suggests that similar work on other life-history traits, such as number and size of young, will prove rewarding.

Advantages of this Approach.—This framework of models unifies the explanation of phenomena that were not previously seen to be related. First, it resolves the discussion over whether organisms mature at a fixed size or at a fixed age. They do neither. Instead, they have evolved a norm of reaction that can be predicted as a well-defined curve of maturation events through

age-size space. Over part of the reaction norm, usually associated with slow growth rates, they will appear to mature at a fixed size. Over other parts, they may appear to mature at a nearly constant age in spite of changes in growth rates. However, there will almost always be portions of the reaction norm, usually at intermediate growth rates, where both age and size at maturity change rapidly as growth rates change.

Second, whereas Alm (1959) suggested that fish share a single, U-shaped reaction norm, we have found reaction norms with at least four shapes (none of them is U-shaped), and further exploration may reveal more. There appears to be a small number of possible reaction norms for maturation. How many is not yet known. The important point is that each one can be associated with specific relationships between growth rates and age-specific mortality rates.

Third, this model clearly distinguishes the genetic and environmental components of variation in age and size at maturity, and it shows how the two interact to determine an observation. Environmental variation produces variation in growth rates and age- and size-specific mortality rates. These changes operate over evolutionary time to mold the genetically determined shape and position of the maturation trajectory. They also work within a single generation to produce a purely phenotypic response that determines the point on the trajectory where a single organism matures. This point was made for morphological traits in *Daphnia* by Woltereck (1909), but without the connections to fitness provided by life-history theory.

Fourth, this model makes numerous predictions about both the shape and the position of reaction norms for maturation, some of which can be measured in a single generation. If one knows in advance, for example, that two populations have different demographic histories, then some of these predictions are testable simply by raising both populations in the laboratory under a range of environmental conditions. These models increase the number of places at which life-history theory comes into risky contact with experiments and observations.

One Prediction that Differs from Published Statements.—Our predictions about the influence of changes in juvenile mortality rates on the position of maturation reaction norms are in apparent conflict with published statements. We predict that increases in juvenile mortality rates result in delayed maturation. Cole (1954) and Williams (1966) made the opposite prediction. For example, Williams stated, "That organisms with high juvenile mortality rates have correspondingly rapid development is a significant generalization that I assume would be universally conceded" (p. 89) and "A given amount of acceleration of development in a high-mortality stage will increase fitness more that it would in a low-mortality stage. We should expect development to be most rapid in stages of high mortality" (p. 90), as is the case in amphibian metamorphosis, for example.

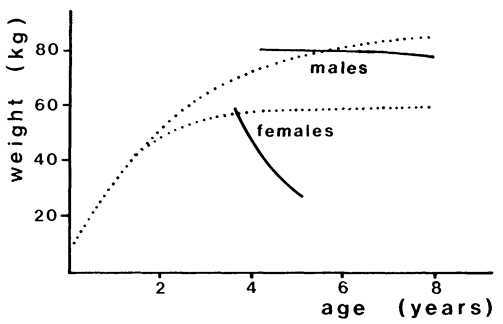

FIG. 13. The reaction norms of male and female Red Deer should differ both in their shape and in their location in age-size space. Female deer should mature at about 4 years under a wide range of growth rates, whereas male deer should delay their age at maturity considerably when forced to grow slowly. The growth curves represent average animals described by Clutton-Brock et al. (1982). This case makes the point that sexual selection operates on the evolution of reaction norms of life-history traits.

If the only impact of a change in developmental rate is on the risk that the developing individual will not survive to maturity, then Williams's statement is correct. Our prediction differs from his because we have introduced two additional effects: an effect of age and size of parent on juvenile mortality rates of offspring, and an effect of size on fecundity of parent. When these effects are strong, they can more than balance the fitness advantage of rapid development and lead to delayed maturity when juvenile mortality is increased.

First consider the effect of the fecundity-

size relationship. We can assume as an initial condition in the population before the increase in juvenile mortality rates occurs that at some smaller-than-normal size at maturity r would be negative because fecundity would be too low. Therefore maturity occurs at larger sizes than this, just how much larger being determined by other factors as discussed above. Somewhere between the small size where r is negative, and the actual size-at-maturity, there is a size where r is 0. Now imagine that juvenile mortality rates are increased, but that nothing else changes. The size at which r is 0 also increases, because only organisms with higher fecundities can maintain that r, and only larger organisms have higher fecundities. This means that the lower bound on sizes at maturity moves up, and if no other factors change, the actual sizes at maturity will also be larger.

Second, consider the effect that change in age and size at maturity is postulated to have on juvenile mortality rates of offspring. As extrinsic juvenile mortality rates are increased, the developing organism suffers higher risk if it remains juvenile longer while delaying maturity, as Williams pointed out. However, if by delaying maturity it can decrease the juvenile mortality rates of its offspring, then it will have balanced the extrinsic increase in mortality rates with an intrinsic decrease, and if the intrinsic effect is strong enough, it will delay maturity.

Interface with Ecological Factors Generating Differences in Growth Rates.—This approach could be applied to two interesting studies in which subtle interactions within and between species generate differences in growth rate: Wilbur and Collins's (1973) work on amphibian metamorphosis and Werner et al.'s (1983) work on predation risk to foraging sunfish.

Wilbur and Collins suggest that small random differences in growth rate within a cohort of amphibian larvae of similar initial size will be amplified by competition and by size-specific reactions to inhibitory waste products. The result is that the size distribution of larvae that at first had a normal distribution with low variance will be changed into a log-normal distribution with higher variance. A few individuals will rapidly grow to large size and metamorphose early, and many individuals will grow slowly and metamorphose late at small sizes. These organisms are displaying either L-shaped or sigmoid reaction norms.

Werner and his colleagues point out that when a cohort of bluegills is subjected to size-specific predation by bass, the smaller bluegills retreat to sheltered habitats with less food where they grow more slowly. The larger members of the cohort, which run less risk of being eaten, continue to forage as before in food-rich, risky habitat and grow more rapidly as a result. This suggests another condition under which Williams' prediction (see above) does not hold: when faster growth is directly associated with higher mortality, then organisms may choose slower growth and lower risk, rather than accelerating development.

In both cases, our approach can be applied if data can be gathered on how changes in growth rates are connected to changes in fitness through the fecundity-size relationship and through changes in juvenile and adult mortality rates. The critical step is achieved when the particular ecological factors affecting growth are translated into generally applicable relationships between growth and life-history traits. Once that step is made, the tools of life-history theory can be used to weigh different options in the currency of fitness.

Acknowledgments

This work began at Reed College while the senior author was supported by NSF DEB 78-22812, by an NIH Biomedical Research Support Grant, and by a Vollum Junior Faculty Sabbatical Award. Richard Crandall contributed significantly to the general approach. Joe Travis, Brian Charlesworth, Jim Cheverud, Derek Roff, and anonymous reviewers helped us to clarify the presentation. We thank the Zoology Institute and Computer Center of the University of Basel and the Swiss Nationalfond for support.

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Corresponding Editor: J. M. Cheverud

APPENDIX

Calculation of Locally Maximal Fitness

By differentiating the Euler-Lotka relation

$$\int_\alpha^\infty b(x)l(x)e^{-rx}\,dx = 1$$

with respect to α, we find

$$-b(\alpha)l(\alpha)e^{-r\alpha} + \int_\alpha^\infty \frac{\partial[b(x)l(x)]}{\partial\alpha}e^{-rx}\,dx - \frac{\partial r}{\partial\alpha}\int_\alpha^\infty xb(x)l(x)e^{-rx}\,dx = 0. \quad (A.1)$$

For our model

$$\frac{\partial[b(x)l(x)]}{\partial\alpha} = \frac{\partial[\alpha(a-j)]}{\partial\alpha}\, b(x)l(x)$$

so that (A.1) reduces to

$$\frac{\partial r}{\partial\alpha}\int_{\alpha}^{\infty} xb(x)l(x)e^{-rx}\,dx = \frac{\partial[\alpha(a-j)]}{\partial\alpha} - b(\alpha)l(\alpha)e^{-r\alpha}. \quad (A.2)$$

Setting $\frac{\partial r}{\partial\alpha} = 0$ results in

$$r_c = \frac{1}{\alpha}\ln\frac{b(\alpha)l(\alpha)}{\frac{\partial[\alpha(a-j)]}{\partial\alpha}}.$$

Evaluated at $r = r_c$, the derivative of (A.2) with respect to α is

$$\begin{aligned} K\frac{\partial^2 r}{\partial\alpha^2} &= \frac{\partial^2[\alpha(a-j)]}{\partial\alpha^2} - \frac{\partial[b(\alpha)l(\alpha)]}{\partial\alpha}e^{-r_c\alpha} \\ &\quad + r_c b(\alpha)l(\alpha)e^{-r_c\alpha} \\ &= \frac{\partial^2[\alpha(a-j)]}{\partial\alpha^2} - \frac{\partial[b(\alpha)l(\alpha)]}{\partial\alpha} \\ &\quad \cdot \frac{\frac{\partial[\alpha(a-j)]}{\partial\alpha}}{b(\alpha)l(\alpha)} \\ &\quad + \frac{\partial[\alpha(a-j)]}{\partial\alpha}\frac{1}{\alpha}\ln\frac{b(\alpha)l(\alpha)}{\frac{\partial[\alpha(a-j)]}{\partial\alpha}} \quad (A.3) \end{aligned}$$

where

$$K = \int_{\alpha}^{\infty} xb(x)l(x)e^{r_c x}\,dx > 0.$$

Note that

$$\begin{aligned} &\frac{\partial^2[\alpha(a-j)]}{\partial\alpha^2} - \frac{1}{b(\alpha)l(\alpha)}\cdot\frac{\partial[b(\alpha)l(\alpha)]}{\partial\alpha}\cdot\frac{\partial[\alpha(a-j)]}{\partial\alpha} \\ &\quad = -\frac{\partial[\alpha(a-j)]}{\partial\alpha}\cdot\frac{\partial}{\partial\alpha}\left\{\ln\frac{b(\alpha)l(\alpha)}{\frac{\partial[\alpha(a-j)]}{\partial\alpha}}\right\} \end{aligned}$$

so that (A.3) reduces to

$$\begin{aligned} K\frac{\partial^2 r}{\partial\alpha^2} &= \frac{\partial[\alpha(a-j)]}{\partial\alpha}\left[\frac{1}{\alpha}\ln\frac{b(\alpha)l(\alpha)}{\frac{\partial[\alpha(a-j)]}{\partial\alpha}}\right. \\ &\qquad \left. - \frac{\partial}{\partial\alpha}\left\{\ln\frac{b(\alpha)l(\alpha)}{\frac{\partial[\alpha(a-j)]}{\partial\alpha}}\right\}\right] \\ &= \frac{\partial[\alpha(a-j)]}{\partial\alpha}\left[r_c - \frac{\partial}{\partial\alpha}\{\alpha r_c\}\right] \\ &= \frac{\partial[\alpha(a-j)]}{\partial\alpha}\left[-\alpha\frac{\partial r_c}{\partial\alpha}\right]. \end{aligned}$$

Since $\frac{\partial[\alpha(a-j)]}{\partial\alpha} > 0$ for r_c to be defined, $\frac{\partial^2 r}{\partial\alpha^2} < 0$ implies that

$$\frac{\partial r_c}{\partial\alpha} > 0.$$