QUANTITATIVE PREDICTIONS OF DELAYED MATURITY

STEPHEN C. STEARNS AND RICHARD E. CRANDALL
Reed College, Portland, Oregon 97202

What are the selective pressures that have driven the evolution of delayed maturity? K-selection (selection operating at high population densities) has been suggested as an explanation, but it does not fit all the data (Wilbur et al., 1974; Stearns, 1977) and it oversimplifies (Whittaker and Goodman, 1979). In a declining population, the rate of decrease is minimized when age at maturity is maximized (Hamilton, 1966; Mertz, 1971), but that explanation is probably not general, for such populations have an increased probability of extinction. Four other hypotheses, which we regard as more plausible, have been suggested: a delay in maturity results (a) in a gain in fecundity (Tinkle, 1969; Gadgil and Bossert, 1970; Tinkle et al., 1970; Wiley, 1974; Schaffer and Elson, 1975; Bell, 1977), (b) in an improvement in the juvenile survival of the offspring produced (Wiley, 1974; Hirshfield and Tinkle, 1977), (c) in a reduction in the cost of reproduction and an increase in adult survival rates (Schaffer, 1972; Wittenberger, 1979), or (d) in increased fitness in the face of unpredictable, catastrophic larval mortality, where the delay comes in the egg stage (Livdahl, 1979). These hypotheses are not mutually exclusive; all four could shape the life-history of some organism.

We have built optimality models in which reproduction and survival depend on the age at maturity of the organism. To keep the models tractable, we assumed that we could deal with a population as though it were a set of asexually reproducing, haploid clones, with each clone endowed with a different life-history. The optimization procedure tells us which clone will win the intraspecific competition for numerical dominance in future generations. To do this, we have assumed that the population is in stable age distribution, and that the definition of fitness is the rate at which a new allele grows in the population, where that allele affects age at maturity, survival, and fecundity. Thus the unit of selection in these models is the gene, not the individual or the population, but because we conceive the population as a set of haploid clones, the gene is effectively equivalent to the individual. For those who are uncomfortable with this simplification, we note that this analysis holds for the marginal effect of a gene substitution on phenotypes in a haploid, asexual population (cf. Charlesworth and Williamson, 1975). We also note that a clone can be growing exponentially in a stationary population. In these models, clones grow exponentially at a rate implied by the particular age at maturity and survivorship and fecundity schedules with which they are endowed. Thus the technical definition of fitness is the Malthusian parameter (Fisher, 1930), with neither frequency-dependence nor density-dependence, but we denote it as r—the intrinsic rate of increase of a clone.

Our models assume either the fecundity-gain hypothesis (a), the quality-of-young hypothesis (b), or both. We have found that either fecundity-gain or quality-of-young assumptions can yield predictions of a considerable delay in maturity when r is maximized. Moreover, when we used published data to predict optimal ages at maturity on the assumption that one, the other, or both effects were at work, the predictions were surprisingly good.

DEVELOPMENT OF THE MODELS

The models begin with the assumption of a stable age distribution (Lotka, 1913;

TABLE 1. *List of symbols.*

Symbol	Definition
α	Age at maturity
$\beta(\alpha,x)$	Fecundity, a function of both age at maturity and age
γ	H/F: the ratio of the intercept and slope of the function relating fecundity and age at maturity
ρ	An exponent defining the curvature of the hyperbola relating juvenile mortality to age
κ	A constant defining the height of the hyperbola relating juvenile mortality to age
$d(\alpha,x)$	Instantaneous mortality rate
d	Constant instantaneous adult mortality rate
$h(\alpha)$	Instantaneous juvenile mortality rate, a function of age at maturity of the parent
$l(\alpha,x)$	Probability of survival from birth to age x, a function of both age and age at maturity
$M(\alpha)$	The component of juvenile mortality that is affected by a delay in maturity
r	Two meanings: the instantaneous rate of population growth (in all equations), and the product-moment correlation coefficient (in statistical tests). See context.
x	Age
F	The rate of increase of fecundity with age at maturity
G	The rate of increase of fecundity with age following maturity
H	The intercept of the line relating fecundity to age at maturity
H^*	The constant value of fecundity for all reproductively active age classes in the Quality of Young Model

Fisher, 1930: see Table 1 for a list of symbols):

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

where

$$l(\alpha,x) = \exp[-\int_0^x d(\alpha,s)\,ds]. \qquad (2)$$

We then require that age at maturity must satisfy (1) and that population growth rate must satisfy

$$\partial r/\partial\alpha = 0. \qquad (3)$$

We shall say that r is *stationary* with respect to variation in α when (3) holds. Now (3) and (1) imply:

$$\beta(\alpha,\alpha)l(\alpha,\alpha)e^{-r\alpha}$$
$$= \int_\alpha^\infty \left[\frac{\partial\beta(\alpha,x)}{\partial\alpha}l(\alpha,x) \right.$$
$$\left. + \beta(\alpha,x)\,\frac{\partial l(\alpha,x)}{\partial\alpha} \right]e^{-rx}\,dx. \qquad (4)$$

Geometrically, we are noting that a given stable age distribution implies a certain relationship between population growth rate (r) and age at maturity (α) upon which we locate the maximum (Fig. 1). To obtain a prediction, one must specify the functions relating fecundity and survival to age at maturity. We do this three different ways, specified in the models that follow.

The Salamander Model (SAM).—This model was inspired by life history of the Appalachian Dusky Salamander, *Desmognathus ochrophaeus* (Tilley, unpubl.), which suggested the following assumptions. If we assume that the instantaneous death rate (d) takes one value (h_α) prior to maturity (α), that it may vary for different ages at maturity, and that it becomes a constant for all adult ages no matter what the adults' ages at maturity, then

$$d(\alpha,x) = \begin{cases} h(\alpha) & (x \le \alpha) \\ d & (x > \alpha). \end{cases} \qquad (5)$$

More specifically, we assumed that juvenile mortality is greater than adult mortality by an amount, $M(\alpha)$, that varies with age at maturity:

$$h(\alpha) = d + M(\alpha). \qquad (6)$$

Geometrically, this resembles a step of variable height that relates $h(\alpha)$, the in-
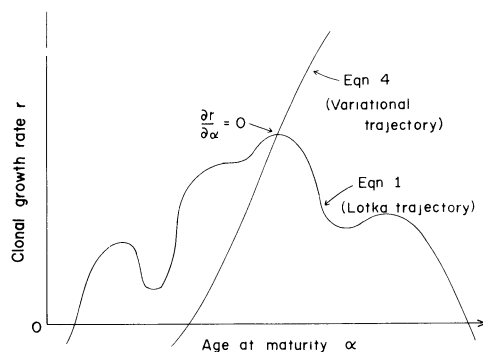
FIG. 1. A graphical interpretation of the general method for finding the age at maturity (α, on the abscissa) that maximizes clonal growth rate (r, on the ordinate). The Lotka-Euler Equation (1) implies some relationship between r and α: the Lotka trajectory. The requirement that $\partial r/\partial \alpha = 0$ implies some other relationship between r and α: the variational trajectory. Where the two lines cross, α maximizes r.
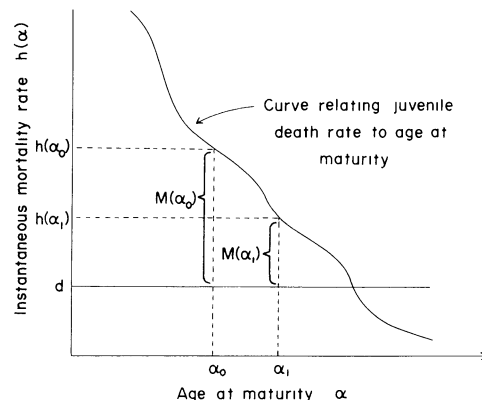

FIG. 2. A graphical interpretation of the general assumptions relating juvenile mortality, $h(\alpha)$, to age at maturity, α, and to constant adult mortality, d. A function of age at maturity, $M(\alpha)$, is added to the adult mortality rate to produce the juvenile mortality rate. We envision M as generally positive and declining as age at maturity increases.

stantaneous juvenile mortality rate, to α (Fig. 2).

Further, if we assume that fecundity increases linearly as maturity is delayed, but is constant thereafter, then

$$\beta(\alpha,x) = F\alpha + H. \tag{7}$$

This can be depicted as a series of steps taking their origin from a straight line with positive slope (Fig. 3). Finally, for convenience we defined a parameter, γ, as the ratio of the slope and the intercept of the line defining the growth in fecundity with delay in maturity:

$$\gamma = H/F. \tag{8}$$

From (5) we can relate survival to age:

$$l(\alpha,x) = \begin{cases} e^{-h(\alpha)x} & (x \leq \alpha) \\ e^{-h(\alpha)x}e^{-d(x-\alpha)} & (x > \alpha). \end{cases} \tag{9}$$

Substituting in (1), we get

$$1 = \int_{\alpha}^{\infty} (F\alpha + H)e^{-h(\alpha)\alpha}e^{-d(x-\alpha)}e^{-rx} \, dx \tag{10}$$

or

$$1 = \frac{(F\alpha + H)e^{-r\alpha}e^{-\alpha h(\alpha)}}{r + d}. \tag{11}$$

This defines the Lotka trajectory, the relationship of r and α for a clone in stable

age distribution that obeys the assumptions made.

$$r = -M(\alpha) - \alpha\frac{\partial M(\alpha)}{\partial \alpha} + \frac{1}{\alpha + \gamma} - d \tag{12}$$

which defines the variational trajectory, the solution to $\partial r/\partial \alpha = 0$. Where the lines defined by (11) and (12) cross, we have the solution to the problem: for what α is r stationary (Fig. 1)? Combining (11) and (12), and recalling (6), we get

$$1 = \frac{F(\alpha + \gamma)^2 e^{[-\alpha/(\alpha+\gamma)]}e^{\alpha^2 \partial M/\partial \alpha}}{1 + (\alpha + \gamma)[-M(\alpha) - \alpha\partial M/\partial \alpha]}. \tag{13}$$

The rate at which an allele (clone) multiplies is stationary with respect to variation in α if and only if α solves (13).

In a particular way, the Salamander Model has built into it both the fecundity-gain and the quality-of-young hypotheses. We therefore built models that made one or the other assumption, but not both, to generate predictions that would allow tests of both simple hypotheses, as well as the compound hypothesis embodied in (13).

The Linear Fecundity Model (LFM).— If we assume that mortality rates are in-
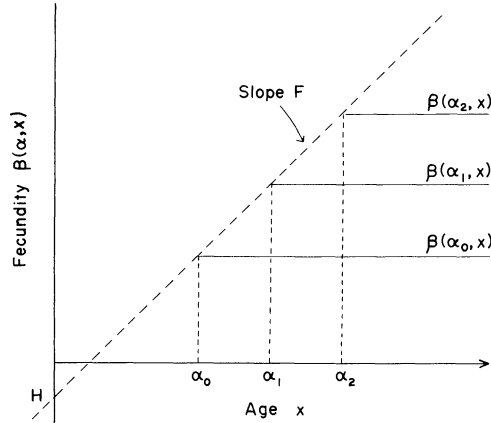
FIG. 3. A graphical interpretation of the assumptions relating fecundity to age at maturity and to age in the Salamander Model. We envision fecundity increasing linearly with age at maturity, then remaining constant at the initial level after the organism matures. H is the intercept and F the slope: $\beta(\alpha, x) F\alpha + H$.

FIG. 4. In the Linear Fecundity Model, a complication is introduced: we allow fecundity to vary linearly after first reproduction, with slope G, while preserving the other linear relation of fecundity to age at maturity. Symbols as in Fig. 3.

sensitive to change in age at maturity, then

$$d(\alpha, x) = d, \text{ a constant.} \quad (14)$$

Now we take advantage of the slight simplification of mortality assumptions to make the fecundity assumptions less restrictive. If we assume that fecundity grows with delayed maturity, and may then either grow or decline with age (x), then

$$\beta(\alpha, x) = F\alpha + Gx + H. \quad (15)$$

This assumption can be depicted as a fan of lines (the Gx element) whose origin can slide up and down a straight line with positive slope (the $F\alpha + H$ element: Fig. 4). Following a procedure similar to the one outlined for the Salamander Model, we find that age at maturity maximizes r when

$$1 = \frac{(F\alpha + G\alpha + H)^2}{F}$$
$$\left(1 + \frac{G}{F}\right)e^{-[\alpha F/(F\alpha + G\alpha + H)]}. \quad (16)$$

At the age at maturity which solves (16),
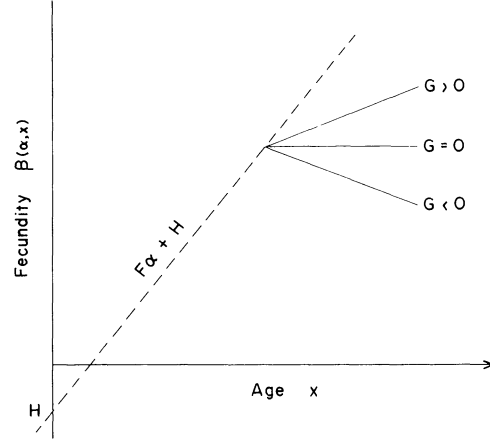
$$r = \frac{F}{F\alpha + G\alpha + H} - d. \quad (17)$$

Note that if juvenile mortality does not vary with age at maturity and equals the constant adult survival rate ($M(\alpha) = 0$, $h(\alpha) = d$), and if fecundity is constant following maturity ($G = 0$), then (13) and (16) reduce to the same relationship. This defines the relationship of the Salamander Model and the Linear Fecundity Model.

One additional feature of the Linear Fecundity Model is of interest: when $H = 0$, maturity will be delayed whenever $F > 0$ and $F + G > 0$. Thus so long as the fecundity gained by delaying maturity exceeds the rate at which fecundity may subsequently decline with age, delayed maturity will evolve. This implies that one could observe individuals whose fecundity declined with age, yet whose age at maturity had been delayed because their fecundity had increased as they put off maturation. While not paradoxical, this prediction is difficult to test. For example, we could not have used the estimation outlined below on a population of such individuals.

Now we consider the consequences of juvenile mortality that varies with age at maturity, but fecundity that does not.

The Quality of Young Model (QYM).— We make the same assumptions that we used to build the Salamander Model, but
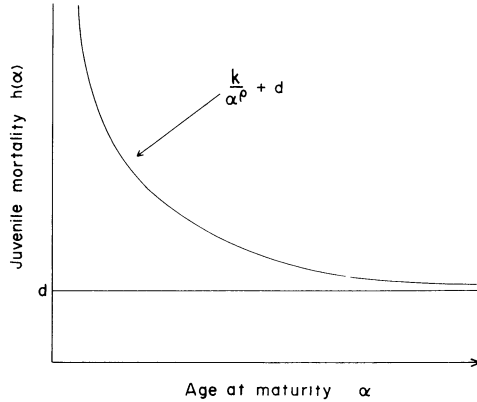
FIG. 5. A graphical interpretation of the relationship between juvenile mortality and age at maturity used in the Quality of Young Model. We envision juvenile mortality declining hyperbolically with age at maturity. κ is a constant which can be determined empirically: see text. ρ, the exponent of α, determines the rapidity of the hyperbolic decline in juvenile mortality. In the Quality of Young Model, the equations converged only if $\rho > 1$. Therefore we set $\rho = 2$, the next convenient value, for all the estimates reported in Table 1.

we also specify that fecundity is a constant for all ages at maturity,

$$\beta(\alpha, x) = H^*. \tag{18}$$

Moreover, if the juvenile mortality declines as some negative power of age at maturity, then

$$h(\alpha) = \kappa/\alpha^\rho + d. \tag{19}$$

This specifies that the curve relating juvenile mortality to age at maturity is a descending hyperbola that is asymptotic to the constant adult mortality rate, d (Fig. 5, cf. (6) and (2)). With these assumptions, age at maturity is optimal when

$$1 = \frac{H^* e^{-\rho\kappa\alpha^{-\rho+1}}}{\lambda(\rho - 1)\alpha^{-\rho}} \tag{20}$$

at which point the population growth rate is

$$r = \frac{\kappa(\rho - 1)}{\alpha^\kappa} - d. \tag{21}$$

Note that if fecundity is constant for all ages at maturity ($F = 0$) and if $H = H^*$, and if $M = \kappa/\alpha^\rho$, then the Salamander

Model (13) reduces to the Quality of Young Model (21). Thus both the Quality of Young Model and the Linear Fecundity Model (with $G = 0$) are included as special cases in the Salamander Model.

One feature of the Quality of Young Model is of special interest: there will only be a solution for finite positive α and r if $\rho > 1$. This means that juvenile mortality must drop off faster than $1/\alpha$ as maturity is delayed for the Quality of Young Model to predict any delay in maturity. For this reason we used the next convenient power, $\rho = 2$, for the calculations involved in testing these predictions. This is an additional a priori assumption about the relationship of juvenile mortality to age at maturity that we could not check with the available data.

METHODS

To test these ideas, we first constructed a general model as follows. We estimated the constants F, H, κ, d, and H^* empirically; we set $M(\alpha) = \kappa/\rho^2$ and $\gamma = H/F$; and we used (13) as the master equation for optimal α. The three particular models occupy certain parametric regions:

Linear Fecundity Model: $\kappa = 0$, H^* not used, $G = 0$;

Quality of Young Model: $F = 0$, $H = H^*$;

Salamander Model: G not used.

We used published data on salamanders (Tilley, 1979) and lizards (Blair, 1960; Tinkle, 1967; Tinkle and Ballinger, 1972; Vinegar, 1976) for the estimates of the constants. There are serious problems with inferring these parameters from the available data, for they require that one know what fecundity or mortality would have been if the organism had matured at a different age. We therefore took the following course: to infer fecundity (F,H), we worked with species where the relationships between size and fecundity and size and age are known. By estimating fecundity as a function of size, either length or weight, over the size range of naturally mature organisms, then extrapolating that

TABLE 2. *Predicted ages at maturity.*

Popu-lation	Empirically estimated constants					Observed α in yr	Predicted optimal age at maturity						Reference
							LFM		QYM		SAM		
	F	H	λ	d	H*		α	r	α	r	α	r	
a	1.32	10.6	0.54	0.08	11.7	0.83	#	#	0.56	1.63	0.59	1.61	1
b	29.1	21.0	0.13	0.80	45.2	0.83	#	#	0.14	6.05	0.18	4.30	2
c	28.8	−13.8	0.28	0.50	5.0	0.83	0.98	1.51	0.44	0.92	1.10	1.34	3
d	54.0	−15.0	0.07	0.83	14.3	0.75	0.62	2.13	0.12	3.77	0.65	2.01	3
e	12.0	−3.0	0.86	0.67	11.8	1.75	0.84	1.03	0.80	0.69	1.43	0.60	3
f	6.5	3.6	0.26	0.73	7.9	1.80	#	#	0.37	1.19	0.56	1.00	3
g	3.75	−2.9	0.28	0.60	4.6	1.61†	1.95	0.25	0.46	0.75	2.12	0.20	4
h	1.42	−1.46	4.25	0.26	5.6	5.0	2.86	0.29	3.23	0.15	5.40	0.11	5
i	1.78	−2.49	1.44	0.37	4.6	4.0	3.22	0.18	1.48	0.29	3.98	0.11	5

† Author reported a range of 1.0–2.0; we used the weighted arithmetic mean # Indicates that equation (3) has no solution with α, r > 0 a Side-blotched lizard, *Uta stansburiana*, Texas, b· Rusty lizard, *Sceloporus olivaceus*, Texas, c, d, e, f Eastern Fence lizard, *S. undulatus*, South Carolina, Texas, Ohio, Colorado; g· Striped Plateau lizard, *S virgatus*, Arizona, h, i Appalachian Dusky salamander, *Desmognathus ochrophaeus*, North Carolina, Whiteside and Dry Falls, respectively. (1) Tinkle (1967), (2) Blair (1960); (3) Tinkle and Ballinger (1972); (4) Vinegar (1975); (5) Tilley (unpubl)

relationship into the immature size classes, we obtained estimates of F and H.

Even though fecundity is fairly constant in all these populations after maturity, there is either a gain in size and fecundity from α to $\alpha + 1$, or sufficient variation in size and fecundity at age at maturity upon which to base a size-fecundity relationship. We always estimated F and H from the age at which the organisms naturally matured and from the next previous age class (the oldest juveniles). This gives the estimation procedure the flavor of a local sensitivity analysis with an a priori assumption that fecundity is a linear function of size near age at maturity.

Thus

$$F = [\beta(\alpha - x)]/[\alpha - (\alpha - x)], \quad (22)$$

where $\alpha - x$ represents the closest age class prior to maturity for which estimates could be made, and

$$H = \beta(\alpha) - F\alpha. \quad (23)$$

To estimate $M(\alpha)$ and $\partial M(\alpha)/\partial \alpha$ we assumed that

$$h(\alpha) = d + M(\alpha), \quad (24)$$

and that

$$M(\alpha) = \kappa/\alpha^2. \quad (25)$$

The instantaneous juvenile mortality rate was estimated as

$$h(\alpha) = 1 - [l(\alpha)]^{1/\alpha}. \quad (26)$$

The adult mortality rate, d, was estimated as the average of the instantaneous death rates over all adult age classes. Both can be calculated directly from a standard life table.

κ was estimated as the solution to the equation

$$\kappa = \alpha^2[h(\alpha) - d], \quad (27)$$

from which

$$\partial M/\partial \alpha = -2\kappa/\alpha^3. \quad (28)$$

As we noted above, we used the second power of α in the denominator of $M(\alpha)$ because we had found that ρ must be greater than 1 in the Quality of Young Model. The implication of this assumption is that juvenile mortality declines as the inverse square of age at maturity.

For example, we predicted that *Uta stansburiana* should mature at 0.59 yr from the Salamander Model, assuming $\beta(0.83 \text{ yr}) = 3.9/\text{clutch}$, $\beta(0.15 \text{ yr}) = 3.6/\text{clutch}$, giving, with 3 clutches/yr, $F = (11.7 - 10.8)/(0.83 - 0.15) = 1.32$, and $H = \beta(0.83 \text{ yr}) - 0.83F = 11.7 - 1.1 = 10.6$. For $h(\alpha)$ and d, $h(0.83) = 1 - [l(0.83)]^{1/0.83} = 0.87$; $d = (1/2)(0.04 + 0.12) = 0.08$; and $\kappa = (0.83)^2(0.87 - 0.08) = 0.54$. This example is represented by the first line in Table 2 (case a).

Once the empirical constants are specified, one can either calculate optimal age at maturity directly by numerically solving (13), (16), or (20), or one can plot r as
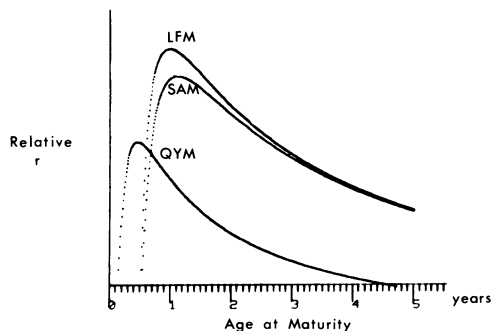
FIG. 6. The graphical method for solving (13), (16), and (20): for each set of assumptions (LFM, QYM, SAM) we solved the Lotka-Euler equation for a series of ages at maturity and plotted r as a function of α. The case shown is the Eastern Fence lizard, *Sceloporus undulatus*, South Carolina population, corresponding to Population c, Table 2.

a function of α, solving the Lotka-Euler equation (1) for each of a series of α's, and select as optimal the α for which the curve peaks (cf. Fig. 6). The two methods give the same answer, as we ascertained by using both.

RESULTS

We made nine predictions for each of the three models (Table 2). In the Linear Fecundity Model (16), only for six of nine estimates did the equation converge to an optimal age at maturity. For those six, the product-moment correlation of predicted with observed ages at maturity was $r = 0.896$ ($r^2 = 0.803$, $P < .05$). In the Quality of Young Model (20), for all nine estimates the equation converged to a solution, and the correlation of predicted with observed ages at maturity was $r = 0.929$ ($r^2 = 0.864$, $P < .01$). In the Salamander Model (13) for all estimates the equation converged, and $r = 0.956$ ($r^2 = 0.914$, $P < .01$).

DISCUSSION

Our predictions were fairly good despite the unrealistic assumptions of asexual haploid genetics with no frequency or density dependence and no seasonality. This may indicate that some conclusions of life-history theory are insensitive to the relaxation of those assumptions. If that is in
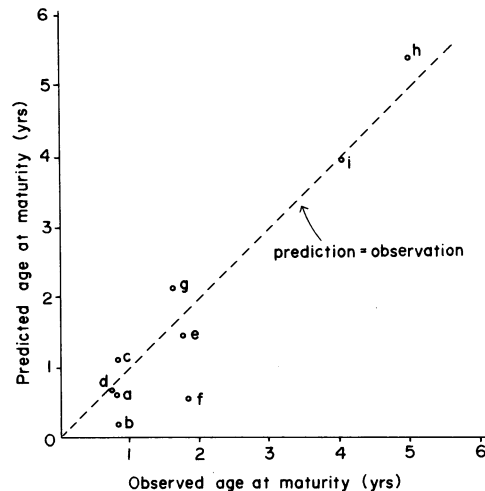

FIG. 7. Relation between observed and predicted ages at maturity for nine populations of lizards and salamanders. Note the poor fit for observed ages at maturity less than 2.0. a, b, c, d, e, f, g, h, i indicate the populations similarly labeled in Table 2. Predictions made with (13): the Salamander Model.

fact the case—and we do not yet know if it is—then future theoretical work could proceed with much simpler assumptions. We need more models which explore how much we can simplify while retaining the power of precise prediction.

One interpretation of the success of these predictions is that the fixation or disappearance of a new mutant depends critically on the rate at which it multiplies when it is at low frequency. If it survives to attain intermediate frequencies, then its eventual fixation is much more probable, for it is past the low-frequency range where drift is powerful. We think that the rate of multiplication of a new mutant at low frequencies is realistically modelled by asexual haploids with no frequency dependence.

Both the Linear Fecundity and the Quality of Young models can account for much of the variation among these populations and species in age at maturity, but the Salamander Model does so remarkably well (Fig. 7). One might suspect some hidden circularity in our reasoning, but when we attempted predictions for species in which fecundity increases dra-

matically after maturity (e.g., lake trout, smooth newts), the equations would not converge, indicating that we do not automatically get a prediction close to observation. Note also that the predictions are less accurate for observed ages at maturity of about 1 yr. This also argues against a hidden circularity. Seasonality constrains reproduction, and we have not built seasonality into these models. For example, at the predicted age at maturity of 0.59 yr, or 215 days, *Uta* hatched on June 17th, the earliest date recorded in Texas (Tinkle, 1967), would lay eggs on January 18th. Any eggs laid in January would probably be killed by cold. Seasonality affects a greater proportion of age at maturity for animals maturing near 1 yr than it does for those maturing later.

We expect (13) to work only for those organisms whose populations show significant size-fecundity relationships at maturity and shortly thereafter, followed by constant fecundity later in life: some lizards and salamanders, possibly turtles, but not most fish, birds, or mammals, for which other models must be built.

The shape of the r vs. α curves implied by the Lotka-Euler relationship deserves comment. In most cases, the Quality of Young Model implied a trajectory with a sharper peak than either the Linear Fecundity Model or the Salamander Model. We have not included in these models all factors that might mold age at maturity, just the two we expect to dominate. When other factors modify age at maturity, we expect the predictions from models that imply sharp peaks to be more robust. Thus we find the precision of the Salamander Model surprising and to a certain extent coincidental, because for that model the r vs. α curves are broad and flat for the two salamander predictions (cases h and i, Table 2): r_3 and r_5 are 73% and 91% of the population growth rate at the optimal age at maturity, $r_{3.98}$, for case i. In other words, the two cases (h and i) that contribute the most to the precision of our predictions are precisely the cases in which we expect our predictions to be least robust, most easily modified by

forces not accounted for in the models. Either there are no forces unaccounted for in those two cases, or if they exist they select for the same ages at maturity predicted here. Seasonality is a factor which should act to increase the apparent precision of predictions that are near an integral number of years, for it will constrain organisms to reproduce near age x which for other reasons might have matured anywhere between $x - 0.5$ and $x + 0.5$.

Given all that has been left out of the models, the predictions are remarkably accurate. However, this test does not demonstrate that there are only two basic reasons for delaying maturity, or that $M(\alpha) = \kappa/\alpha^2$ and that $\beta(\alpha) = F\alpha + H$ for lizards and salamanders. It does indicate that we understand two forces which can delay maturity, and that this line of thought looks promising enough to deserve more formal testing in a selection experiment. We also suggest that these models, or appropriate modifications of them be used as starting points in attempts to explain delayed maturity. If the fecundity-gain and the quality-of-young hypotheses can delay maturity, then one should not invoke more complex explanations unless one can show that those we have put forth here will not suffice.

SUMMARY

We predicted age at maturity in nine populations of lizards and salamanders with three models. The correlation of predicted with observed ages at maturity ranged from $r = 0.896$ to $r = 0.956$. We assumed that gene substitutions which change age at maturity also change fecundity or juvenile survival, or both; that populations can be modelled as sets of haploid, asexual clones that grow exponentially in stable age distribution; that clonal growth rate, r, defines fitness; and that optimization is an appropriate procedure.

LITERATURE CITED

BELL, G. 1977. The life of the smooth newt (*Triturus vulgaris*) after metamorphosis. Ecol. Monogr. 47:279–299.

BLAIR, W. F. 1960. The Rusty Lizard. Texas Univ. Press, Austin. 185 p.

CHARLESWORTH, B., AND J. A. WILLIAMSON. 1975. The probability of survival of a mutant gene in an age-structured population and implications for the evolution of life-histories. Genet. Res. 26:1–10.

FISHER, R. A. 1930. The Genetical Theory of Natural Selection. Clarendon Press, Oxford. 272 p.

GADGIL, M., AND W. BOSSERT. 1970. Life history consequences of natural selection. Amer. Natur. 104:1–24.

HAMILTON, W. D. 1966. The moulding of senescence by natural selection. J. Theoret. Biol. 12:12–45.

HIRSHFIELD, M. F., AND D. W. TINKLE. 1975. Natural selection and the evolution of reproductive effort. Proc. Nat. Acad. Sci. USA 72:2227–2231.

LIVDAHL, T. P. 1979. Environmental uncertainty and selection for life-cycle delays in opportunistic species. Amer. Natur. 113:835–842.

LOTKA, A. J. 1913. A natural population norm. II. J. Wash. Acad. Sci. 3:289–293.

MERTZ, D. B. 1971. Life history phenomena in increasing and decreasing populations, p. 361–399.

In G. P. Patil, E. C. Pielou, and W. E. Waters (eds.), Statistical Ecology, vol. 2. Pennsylvania State Univ. Press, University Park.

SCHAFFER, W. M. 1972. Evolution of optimal reproductive strategies. PhD Thesis. Princeton University, Princeton.

SCHAFFER, W. M., AND P. F. ELSON. 1975. The adaptive significance of variation in life history among local populations of Atlantic Salmon in North America. Ecology 56:577–590.

STEARNS, S. C. 1977. The evolution of life-history traits. Ann. Rev. Ecol. Syst. 8:145–171.

TINKLE, D. W. 1967. The life and demography of the side-blotched lizard, *Uta stansburiana*. Misc. Publ. Mus. Zool. Univ. Mich. 132:1–182.

TINKLE, D. W., AND R. E. BALLINGER. 1972. *Sceloporus undulatus:* a study of the intraspecific comparative demography of a lizard. Ecology 53:570–584.

TINKLE, D. W., H. M. WILBUR, AND S. G. TILLEY. 1970. Evolutionary strategies in lizard reproduction. Evolution 24:55–74.

VINEGAR, M. B. 1975. Demography of the striped plateau lizard, *Sceloporus virgatus*. Ecology 56:172–182.

WHITTAKER, R. H., AND D. GOODMAN. 1979. Classifying species according to their demographic strategy. I. Population fluctuations and environmental heterogeneity. Amer. Natur. 113:185–200.

WILBUR, H. M., D. W. TINKLE, AND J. P. COLLINS. 1974. Environmental certainty, trophic level, and resource availability in life history evolution. Amer. Natur. 108:805–817.

WILEY, R. H. 1974. Evolution of social organization and life-history patterns among grouse. Q. Rev. Biol. 49:201–227.

Corresponding Editor: H. M. Wilbur