

Optimal Size at Maturity in Size-Structured Populations

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Maturity is a critical point in the life cycle, and determining the optimal age or size at maturity is a classical problem in life history theory. Most organisms spend some time in non-reproductive immature stage. To explain the widespread occurrence of delayed maturity, a number of authors made mathematical models with the age-specific fecundity and survival rate, specified functional forms of fecundity and survival rate, and examined the possibility that two hypotheses hold. One hypothesis is that delayed maturation is advantageous when it leads to more fecundity of mature individuals. The other one is that the offspring produced by parents with delayed maturation may have higher survival in their juvenile period. In the present paper, we use the size-structured McKendrick-von Foerster equation as our basic model and obtain five conditions for delayed maturation. Two of these correspond to the above two hypotheses. The other three are new: we show that a discontinuous decrease at maturation in survival or growth rate can favor delayed maturation. We explore several numerical examples showing the operation of these mechanisms.

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1. Introduction

Maturity is a critical point in the life cycle, and determining the optimal age or size at maturity is a classical problem in life history theory (Cole, 1954; Gadgil & Bossert, 1970; Charnov, 1979). Most organisms spend some time in non-reproductive immature stage. However, it can be shown (e.g. Caswell, 1989, p. 154) that, in a deterministic environment, selection never favors delayed maturity, provided that the population is non-decreasing [the situation in stochastic environments is fundamentally different (Tuljapurkar, 1990), but we shall not consider the effects of stochasticity here].

The explanation for the selection against delayed maturity is simple: fitness depends on the rate at which an allele can propagate through the population and, all other things being equal, reproducing sooner increases that rate. To explain the widespread occurrence of delayed maturity in the face of this selection pressure, a number of authors have developed models in which the cost of delayed maturity is balanced by a benefit elsewhere in the life cycle (e.g., Bell, 1980; Roff, 1981, 1984; Kozlowski & Wiegart, 1987; Stearns & Crandall, 1981, 1984; Crandall & Stearns, 1982; Stearns & Koella, 1986; Stearns, 1992). In some of these models, delayed maturity leads to increased adult fecundity, because fecundity is correlated with size and growth slows at maturity. Delayed maturity produces larger adults with higher reproductive output, and this benefit may outweigh the cost of delayed reproduction. In other models, delayed maturity leads to increased juvenile survival of the offspring. Again, the benefit of increased juvenile survival may outweigh the cost of delayed reproduction.

Although most previous studies have used ageclassified demographic models to calculate the

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population growth rate (r) or lifetime reproductive output (R_0) as measures of fitness, the underlying ideas are based on size-related vital rates. Thus most of these models have written age-specific survival and/or fecundity as functions of size and used a growth function to relate size and age. They then search for an age at maturity that maximizes r or R_0 .

In this paper, we begin with a model formulated directly in terms of size rather than age. Our analysis is thus most appropriate for the many species, including plants, fish, and some invertebrates (e.g. Caswell, 1989), for which size is a better state variable than age. In many such species, there are distinct size thresholds for maturity, rather than the age thresholds addressed in previous models [e.g. crabs (Somerton & MacIntosh, 1983), trees (data in Caswell, 1986), herbaceous plants (Gross & Werner, 1983, Caswell, 1986), fishes (Alm, 1959)]; see Fig. 1. Somatic growth appears explicitly in our demographic model, and survival and reproduction are expressed directly as functions of size, rather than age. Whereas most previous studies have focused on specific functional forms for survival, reproduction, and growth, we will be able to outline conditions for the evolution of delayed maturity in general, and then explore their consequences with specific functional forms.

We find several factors that might favor delayed maturity that have not appeared in previous studies; we will argue that these are in fact the most biologically plausible and the most empirically accessible of such factors.

Age-classified population dynamics can be recovered as a special case of a size-classified model with a growth rate exactly equal to unity. Therefore, our methods and results can also be applied to the case of age-structured populations.

2. The Model and Optimal Size at Maturity

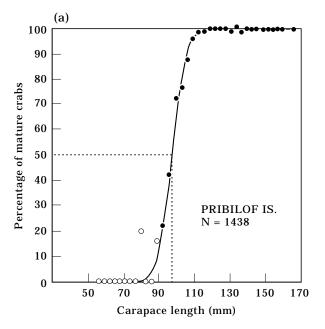
We use the size-structured McKendrick-von Foerster equation (Van Sickle, 1977; Metz & Diekman, 1986; DeRoos, 1996) as our basic model. It describes the state of the population by the size density function n(w, t), where w is size and t is time. Size may be measured by length, weight, or the size of a body part, depending on the application. The vital rates are given by the growth function g(w), the mortality rate $\mu(w)$ and the fecundity m(w), all of which are functions of size. The dynamics of a population with initial size w_0

and size at maturity M are given by

$$\begin{cases} \frac{\partial n(w,t)}{\partial t} + \frac{\partial [g(w;M)n(w,t)]}{\partial w} \\ = -\mu(w;M)n(w,t) \quad \text{(1a)} \end{cases}$$

$$\int_{M}^{\infty} m(w;M)n(w,t)dw = g(w_0;M)n(w_0,t), \quad \text{(1b)}$$

Equation (1b) is a boundary condition that sets the flow of individuals at the initial size $w = w_0$ equal to the integrated reproductive output of all mature individuals.



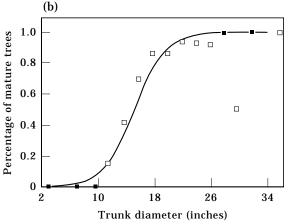


FIG. 1. Examples of size-dependent maturation in animals and plants. (a) Percentage of female blue king crabs mature, as a function of carapace length in mm [from Somerton & MacIntosh (1983)]. (b) Percentage of western pine trees bearing cones, as a function of trunk diameter in inches [from Caswell (1986), based on data of Zon (1915)].

In the above equations, we have written g(w; M), $\mu(w; M)$ and m(w; M) as functions of both size and size at maturity, because size at maturity may affect the vital rates.

In a constant environment, n(w, t) converges asymptotically to a stable size distribution, and the population increases exponentially at a rate, r given by the solution to the characteristic equation (Van Sickle, 1977):

$$\int_{M}^{\infty} \frac{m(w; M)}{g(w; M)} l(w, w_0; M) e^{-r\tau(w, w_0; M)} dw = 1.$$
 (2)

Equation (2) is the size-classified analog of the Euler-Lotka equation for age-structured populations. For any specific functional forms of the vital rates, the population growth rate (or Malthusian parameter),
$$r$$
 depends on the size at maturity M . The optimal maturation size is the value of M that maximizes r ; to find it, we differentiate both sides of eqn (2) with respect to M and set the derivative equal to zero (see Appendix A):

$$\frac{\partial r}{\partial M} = \frac{1}{\int_{M}^{\infty} \frac{m}{g} l\tau \ e^{-r\tau} dw} \times \left\{ -\frac{m(M; M)}{g(M; M)} l(M, w_0; M) e^{-r\tau(M, w_0; M)} + \int_{M}^{\infty} \frac{\partial}{\partial M} \left(\frac{m}{g}\right) l \ e^{-r\tau} dw - \int_{M}^{\infty} \frac{m}{g} l \ e^{-r\tau} \frac{\partial \tau}{\partial M} dw \right\}, \tag{5}$$

In this equation, $l(w, w_0; M)$ represents the probability of surviving from birth (at size w_0) to size w; it can be written in terms of the growth rate and mortality functions as

$$l(w, w_0; M) = \exp\left[-\int_{w_0}^{w} \frac{\mu(x; M)}{g(x; M)} dx\right]$$

and $l(w_0, w_0; M) = 1$. (3)

where

$$s(w, w_0; M) = \int_{w_0}^{w} \frac{\mu(x; M)}{g(x; M)} dx.$$
 (6)

Therefore, a necessary condition for the existence of an interior optimal maturation size (M^*) , is $\partial r/\partial M=0$ at $M=M^*$. The following two equations are thus necessary conditions for the optimal maturation size:

$$\begin{cases}
\int_{M^*}^{\infty} \frac{m(w; M^*)}{g(w; M^*)} l(w, w_0; M^*) e^{-r^*\tau(w, w_0; M^*)} dw = 1 \\
-\frac{m(M^*; M^*)}{g(M^*; M^*)} l(M^*, w_0; M^*) e^{-r^*\tau(M^*, w_0; M^*)} + \int_{M^*}^{\infty} \frac{\partial}{\partial M} \left(\frac{m}{g}\right) l e^{-r^*\tau} dw \\
-\int_{M^*}^{\infty} \frac{m}{g} l \frac{\partial s}{\partial M} e^{-r^*\tau} dw - r \int_{M^*}^{\infty} \frac{m}{g} l e^{-r^*\tau} \frac{\partial \tau}{\partial M} dw = 0.
\end{cases} \tag{8}$$

In eqn (2), $\tau(w, w_0; M)$ represents the time required to grow from the initial size w_0 to size w. It can be written as

$$\tau(w, w_0; M) = \int_{w}^{w} \frac{1}{g(x; M)} dx.$$
 (4)

Equation (7) is equivalent to the characteristic equation (2) when $M = M^*$ and $r = r^*$ and eqn (8) is equivalent to $\partial r/\partial M = 0$ at $M = M^*$. We can obtain M^* and r^* (r when $M = M^*$) by solving eqns (7) and (8) simultaneously.

3. Conditions for Delayed Maturation

There are four terms in the parenthesis of the right hand side of eqn (5). The second, third, and fourth of these involve the derivatives of the vital rates by maturation size M. The first term is always negative, which implies that if the vital rates are independent of M, r always decreases when maturity is delayed, and maturation at the initial size is optimal. The net selection pressure on M depends on the balance of this term and the possibly positive effects from the second, third, and fourth terms. At least one positive term among the latter three is necessary for delayed maturation to be favored.

In order to clarify the conditions under which selection may favor delayed maturity, we now consider the condition under which each of the latter three terms is equal to zero.

(A)
$$\frac{\partial (m/g)}{\partial M} = 0$$
 (from the second term).

The necessary and sufficient condition for $\partial(m/g)/\partial M=0$ is that

$$\frac{m(w; M)}{g(w; M)} \equiv f_1(w), \tag{9}$$

where $f_1(w)$ may depend on size, but is independent of M. The second term in eqn (5) is positive if

Condition 1 m/g depends positively on maturation size.

(B)
$$\frac{\partial s}{\partial M} = 0$$
 (from the third term).

 $\partial s/\partial M$ is equal to zero if and only if (see Appendix B)

$$\frac{\mu(w; M)}{g(w; M)} \equiv f_2(w)$$
 and $f_2(M-) = f_2(M+)$, (10)

where $f_2(M-)$ and $f_2(M+)$ represent the values of $f_2(w)$ when w approaches to M from the left side and from the right side, respectively. The second equation in eqn (10) requires that μ/g is a continuous function of w at maturation size. Therefore, the third term in eqn (5) can be positive (see Appendix B) when

Condition 2 μ/g depends negatively on maturation size, i.e.

$$\frac{\partial(\mu/g)}{\partial M} < 0,$$

or,

Condition 3

$$\frac{\mu(M-;M)}{g(M-;M)} < \frac{\mu(M+;M)}{g(M+;M)}.$$

Either the negative dependence of the mortality (normalized by growth) on size at maturity, or a discontinuity in normalized mortality at maturation can be a selective pressure for delayed maturation.

(C)
$$\frac{\partial \tau}{\partial M} = 0$$
 (from the fourth term).

Appendix c shows that $\partial \tau / \partial M = 0$ if and only if

$$g(w; M) \equiv f_3(w)$$
 and $f_3(M-) = f_3(M+)$. (11)

Therefore, the fourth term in eqn (5) can be positive if

Condition 4 g depends positively on maturation size, i.e.

$$\frac{\partial g}{\partial M} > 0,$$

or

Condition 5
$$g(M-;M) > g(M+;M)$$
.

That is, either the increase of the size-specific growth rate with delayed maturation, or a discontinuity in growth rate at maturity can be a selective pressure for delayed maturation.

4. Biological Interpretation

We have identified five Conditions, at least one of which is necessary for the optimal size at maturity to exceed w_0 .

- Normalized fecundity is positively dependent on M. or
- normalized mortality is negatively dependent on M, or
- (3) normalized mortality increases discontinuously at M, or
- (4) growth depends positively on M, or
- (5) growth decreases the discontinuously at M.

Conditions 1 and 2 correspond, respectively, to the fecundity advantage and the mortality advantage hypotheses (Bell, 1980; Roff, 1981, 1984; Kozlowski & Wiegart, 1987; Stearns & Crandall, 1981, 1984; Crandall & Stearns 1982; Stearns & Koella, 1986; Stearns 1992). Condition 4 is new. It involves the effect of size at maturity on growth rate, and has not

The condition for delayed maturation in age-structured population		
Condition	Mathematical expression	Biological meaning
1	$\frac{\partial m(w; M)}{\partial M} > 0$	Fecundity increases when maturation is delayed.
2	$\frac{\partial \mu(w; M)}{\partial M} < 0$	Mortality decreases when maturation is delayed.
3	$\mu(M-) < \mu(M+)$	Mortality increases abruptly after maturation.
4	$\frac{\partial g(w; M)}{\partial M} > 0$	_
5	g(M-) > g(M+)	_

Table 1

The condition for delayed maturation in age-structured population

been considered before because growth in size does not appear explicitly in age-classified models.

Conditions 3 and 5 are also new, and are perhaps the most important of our results. In conditions 1, 2 and 4 the vital rates are explicit functions of maturation size (M). This requires that two individuals of the same size should have different vital rates because they matured at different sizes. This appeared reasonable in earlier, age-classified models, because maturity affects growth, and thus size at a given age. Size, in turn, affects survival and/or reproduction. In our size-classified model, this somewhat tortured chain of causation is unnecessary because individuals are classified directly in terms of size. Thus, if maturity affects the vital rates at later stages in our model, individuals of the same size will have different survival or fertility, depending on when they matured. This is inconsistent with the notion that size is a valid individual state variable (Metz & Diekman, 1986). It certainly does not agree with the hypothesized causal chain linking maturation and the vital rates.

Conditions 3 and 5, by contrast, describe the immediate effect of maturation on the size-specific vital rates. Individuals just smaller than M are expected to have vital rates discontinuously different from individuals just larger than M. But, under these conditions, mature individuals of the same size have the same vital rates, no matter what their sizes at maturity. Thus conditions 3 and 5 are the most biologically reasonable of the various conditions yielding delayed maturity. The pattern of energy allocation changes at maturity. As more energy is allocated to reproduction, less is available for allocation to growth, maintenance, and predator avoidance. This means that there will be a difference in growth and/or mortality between mature and immature individuals of the same size.

AGE-STRUCTURED POPULATIONS AS A SPECIAL CASE

The classical McKendrick-von Foerster equation can be obtained as a special case of eqns (1a) and

(1b) by identifying w with age, in which case g(w; M) = 1. The characteristic eqn (2) is then the Euler–Lotka equation for an age-structured population. The possible conditions for delayed maturation are summarized in Table 1. Conditions 4 and 5 cannot hold because g(w; M) is constant. Delayed maturation can be favored if one of Conditions 1–3 holds. Conditions 1 and 2 correspond to the fecundity advantage and the survival advantage hypotheses, as noted above. Condition 3 is a new hypothesis in this context; it suggests that delayed maturation may be favored when mortality increases discontinuously at maturity.

6. Numerical Examples

In this section, we present three numerical examples for specific forms of the survival, growth and fecundity functions. The first is a size-structured model in which fecundity is proportional to body size, mortality is constant, and growth is described by the von Bertalanffy equation. We will show that a reduction in growth rate at maturity can select for delayed maturation. In the second example, we add to this model a size refuge from predation and show that under some circumstances the optimal size at maturity coincides with this size. Finally, we consider an age-structured example, and show how discontinuous mortality (Condition 3) may select for delayed maturity in the absence of the fecundity or survival advantage hypotheses.

A SIMPLE SIZE CLASSIFIED MODEL

The vital rates are given by

$$\begin{cases} \text{fecundity} & m(w) = mw \\ \text{mortality} & \mu(\text{constant}) \end{cases}$$

$$\begin{cases} \text{growth } rate & g(w) = \begin{cases} k_1(w_{\text{max}} - w) & w < M \\ k_2(w_{\text{max}} - w) & w > M \end{cases}$$

$$(12)$$

The growth rate is given by the von Bertalanffy equation, with a growth coefficient k that differs before and after maturity. This change could result from the shift of resources from growth into gametes or reproductive tissue. We assume that fecundity is proportional to body size, and mortality is constant, both of these rates being independent of M

Substituting eqn (12) into eqns (2) and (5), we obtain

$$m \exp[-A(r+\mu)] \left\{ \frac{w_{\text{max}}}{r+\mu} - \frac{w_{\text{max}} - M}{r+\mu + k_2} \right\} = 1$$

$$A = -\frac{1}{k_1} \ln \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_0} \right) \tag{13a}$$

and

$$\frac{dr}{dM} \left\{ A \exp[-A(r+\mu)] + \frac{mw_{\text{max}}}{(r+\mu)^2} - \frac{m(w_{\text{max}} - M)}{(r+\mu + k_2)^2} \right\}$$

$$=\frac{m}{k_1(r+\mu+k_2)(w_{\max}-M)}\times$$

$$\{w_{\text{max}}(k_1 - k_2) - M(r + \mu + k_1)\}.$$
 (13b)

When $k_1 \le k_2$, the r.h.s. of eqn (13b) is always negative. Therefore, the optimal maturation size is the minimum size (w_0) . When $k_1 > k_2$, the r.h.s. of eqn (13b) may be positive. Therefore, delayed maturation may be favored when the growth rate decreases abruptly at maturity. The optimal maturation size, M^* , is

$$M^* = \frac{w_{\text{max}}(k_1 - k_2)}{(r^* + \mu + k_1)},\tag{14}$$

where r^* is the Malthusian parameter at the optimal value of M, obtained by simultaneously solving eqn (13a) and eqn (13b) with the left hand side set equal to zero.

Figure 2 shows how r responds to the change in M as a function of k_1 and k_2 . As the growth rate after maturity, k_2 , declines, delayed maturation becomes optimal and the optimum size at maturity increases. Figure 3 explores the response of the optimal maturation size M^* to changes in the various parameters. Mortality has no effect on M^* in this example [Fig. 3(a)]. The mathematical reason is that the Malthusian parameter and mortality always appear together in eqns (13) and (14) as a sum (r + m). The biological reason is that the mortality is the same both at immature and mature stages. Figure 3b shows

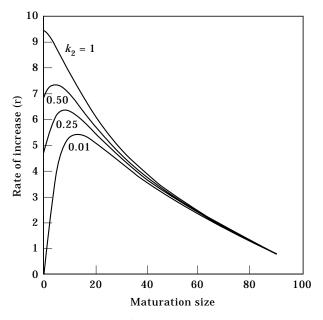


Fig. 2. The response of Malthusian parameter to the change in M as a function of k_2 for a given value of $k_1 = 1.0$, f = 1.2 and $\mu = 1$. As the growth rate after maturity, k_2 , declines, delayed maturation becomes optimal and the optimum size at maturity increases.

how M^* depends on the growth rate coefficients before and after maturation $(k_1 \text{ and } k_2)$. If $k_1 < k_2$, M^* is equal to zero and delayed maturation is not favored. If $k_1 > k_2$, M^* increases with the difference between k_1 and k_2 . The more abrupt the change of growth rate at maturity is, the larger the optimal maturation size is. M^* also depends on the fecundity [Fig. 3(c)]; the larger the fecundity coefficient, the smaller the optimal maturation size. There is an interaction between the growth rate and fecundity coefficients. At low immature growth rates, M^* is relatively insensitive to changes in fecundity. When immature growth is rapid, M^* is more sensitive to fecundity.

EFFECTS OF A SIZE REFUGE FROM PREDATION

Some species are subject to attack by a size-specific predator, which preys on them only up to some size w_p . Individuals beyond this size are invulnerable to this predator, so w_p delimits a 'size refuge' from predation. Paine (1976) drew attention to this phenomena and its potential importance in permitting coexistence of predators and prey. In some bivalves (B. Ripley, personal communication; see Hardwick-Witman, 1985) the size at maturity is close to the size refuge w_p . This has been explained by noting that there is an advantage to growing as fast as possible through the vulnerable size range; if reproduction slows down growth it should be postponed until after w_p .

Mortality changes from μ_1 to μ_2 at size w_p ; growth rate To analyze this case, we change the vital rate functions of the previous model to

fecundity
$$m(w) = mw$$

$$\begin{cases}
\mu_1 & w < w_p \\
\mu_2 & w > w_p
\end{cases}$$
growth rate $g(w) = \begin{cases} k_1(w_{\text{max}} - w) & w < M \\ k_2(w_{\text{max}} - w) & w > M \end{cases}$
(15)

changes from k_1 to k_2 at maturity; fecundity is proportional to body size.

Clearly, the presence of a size refuge alone (i.e. if $k_1 = k_2$) is not sufficient to select for delayed maturity, since none of Conditions 1–5 are then satisfied. But if $k_1 > k_2$ the size threshold may influence the location of the optimal size at maturity.

Two cases must be considered, depending on the relationship between w_p and M. If $M < w_p$, substituting eqn (15) into eqns (2) and (5), we obtain

$$m \exp[-A(r + \mu_1) + B(\mu_1 - \mu_2)]$$

$$\left\{ \frac{w_{\text{max}}}{r + \mu_2} - \frac{w_{\text{max}} - M}{r + \mu_2 + k_2} \right\} = 1$$

$$A = -\frac{1}{k_1} \ln \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_0} \right), \quad B = -\frac{1}{k_2} \ln \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_p} \right)$$
(16a)

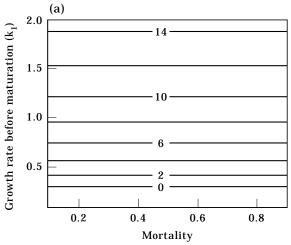
and

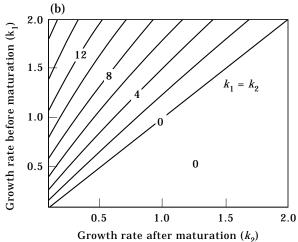
$$\frac{dr}{dM} \left\{ A \exp[A(r + \mu_1) - B(\mu_1 - \mu_2)] + \frac{mw_{\text{max}}}{(r + \mu_2)^2} - \frac{m(w_{\text{max}} - M)}{(r + \mu_2 + k_2)^2} \right\}$$

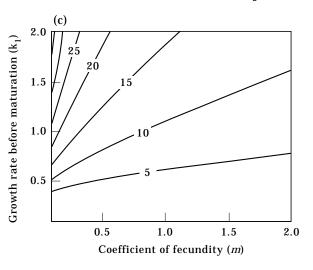
Fig. 3. The contour map of the optimal maturation size (M^*) for $W_{\max} = 100$, $w_0 = 0$. The attached numerics with contour lines represent the value of M^* . (a) $k_1 = 0.3$, f = 1.2: Mortality has no effect on the optimal maturation age (M^*) in this example. The optimal maturation size increases with the increase of growth rate before maturation. The optimal maturation size is equal to zero in the region under the contor line with 0. (b) $\mu = 0.3$, f = 1.2: The optimal maturation size depends on both the coefficients of growth rate before and after maturation $(k_1$ and $k_2)$. If $k_1 < k_2$, it is equal to zero and delayed maturation is not favored. If $k_1 > k_2$, it increases with the difference between k_1 and k_2 . (c) $\mu = 0.3$, $k_2 = 0.3$: It also depends on the coefficient of fecundity. The more the coefficient of fecundity is, the optimal maturation size decreases.

$$= \frac{m}{r + \mu_2 + k_2} + \left\{ \frac{m w_{\text{max}}}{(r + \mu_2)^2} - \frac{m (w_{\text{max}} - M)}{(r + \mu_2 + k_2)^2} \right\}$$

$$\left\{ -\frac{r + \mu_1}{k_1 (w_{\text{max}} - M)} + \frac{\mu_1 - \mu_2}{k_2 (w_{\text{max}} - M)} \right\}$$
 (16b)







(see Appendix D for details). In this case, the mathematical expression for the optimal maturation size is slightly complicated.

In the case of $M > w_p$, we also obtain the Euler–Lotka equation and a equation on the first derivative.

In this case, the optimal maturation size is

$$M^* = \begin{cases} \frac{w_{\text{max}}(k_1 - k_2)}{(r^* + \mu_2 + k_1)} & \text{when } k_1 > k_2 \\ 0 & \text{when } k_1 < k_2 \end{cases}$$
 (17)

Equation (17) is similar to eqn (14). Figure 4 shows the result of changing w_p when $k_1 = k_2$; as expected there is no internal optimum for maturation size, although as w_p increases, r declines because more of the life cycle is subject to mortality from predation.

Figure 5 shows the result of changing w_p in a situation $(k_1 > k_2)$ in which an optimal maturation size is possible. When the predation mortality is severe ($\mu_1 = 1$, $\mu_2 = 0.1$) and the effect of maturity on growth is large $(k_1 = 1, k_2 = 0.1)$, the optimal size at maturity M^* is close to w_p , once w_p exceeds a certain threshold [\approx 15 in Fig. 5(b)]. When the predation effect is weaker $[\mu_1 = 0.6, \mu_2 = 0.5 \text{ in Fig. 5(c, d)}],$ or the maturity effect is weaker $[k_1 = 1, k_2 = 0.6]$ in Fig. 5(e, f)] there is still an optimal size at maturity, but it no longer approximates w_p . Thus, on the basis of this model, the correlation between size at maturity and the size refuge from predation is to be expected in situations where both the cost of reproduction and the predator effect are strong, but not otherwise.

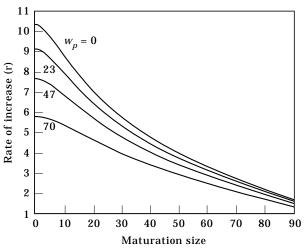


Fig. 4. The relation between Malthusian parameter and maturation size when $k_1 = k_2 = 1$. f = 1.2, $\mu_1 = 1$, $\mu_2 = 0.1$: There is no internal optimum for maturation size, although as w_p increases, r declines.

AN AGE-STRUCTURED MODEL: DISCONTINUOUS MORTALITY

Finally, we analyse an age-classified model in which a discontinuity in mortality at maturity can provide the selective force for delayed maturation, even when neither the fecundity advantage nor the juvenile survival advantage hypotheses apply. The vital rates are

$$\begin{cases}
\text{fecundity} & m(w) = mw^n \\
\text{mortality} & \mu(w) = \begin{cases} \mu_1 & w < M \\ \mu_2 & w > M \end{cases}
\end{cases}$$
(18)

Substituting eqn (18) into eqns (2) and (5), and setting $w_0 = 0$ and g(w; M) = 1 (because the model is age-structured) we obtain

$$\frac{m \exp\{-(r+\mu_1)M\}}{r+\mu_2} \sum_{k=0}^{n} \frac{n!}{(n-k)!} \frac{M^{n-k}}{(r+\mu_2)^k} = 1 \quad (19a)$$

and $\frac{dr}{dM} \left\{ \frac{1}{r+\mu_2} + M + \frac{\sum\limits_{k=0}^{n} \frac{n!}{(n-k)!} \frac{k(r+\mu_2)^{k-1} M^{n-k}}{(r+\mu_2)^{2k}}}{\sum\limits_{k=0}^{n} \frac{n!}{(n-k)!} \frac{M^{n-k}}{(r+\mu_2)^k}} \right\}$

$$= -(r + \mu_1) + \frac{\sum_{k=0}^{n} \frac{n!}{(n-k)!} \frac{(n-k)M^{n-k-1}}{(r+\mu_2)^k}}{\sum_{k=0}^{n} \frac{n!}{(n-k)!} \frac{M^{n-k}}{(r+\mu_2)^k}}.$$
 (19b)

When n = 0, the right-hand side of eqn (19b) becomes $-(r + \mu_1)$, which is always negative. Therefore, when fecundity does not increase with age, delayed maturity is not favored. When n = 1, the right-hand side of eqn (19b) becomes

$$\frac{-(r+\mu_1)(r+\mu_2)M+(\mu_2-\mu_1)}{(r+\mu_2)M+1},$$
 (20)

the sign of which depends on the difference in mortality before and after maturation $(\mu_2 - \mu_1)$. If $\mu_2 > \mu_1$, delayed maturation can be favored; the optimal maturation age, M^* , is

$$M^* = \frac{\mu_2 - \mu_1}{(r^* + \mu_1)(r^* + \mu_2)},\tag{21}$$

where r^* is the Malthusian parameter at the optimal maturation age, obtained by solving eqns (19a) and (19b) simultaneously, with the l.h.s. of eqn (19b) set equal to zero. Figure 6 shows a contour map of

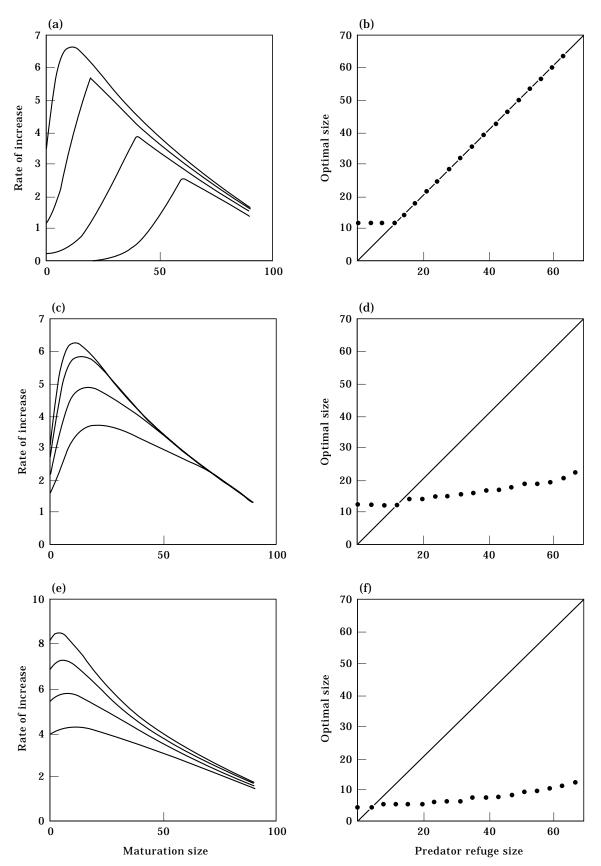
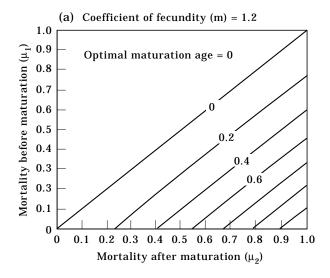


Fig. 5. The result of changing w_p in a situation that $k_1 > k_2$. (a and b) When the predation mortality is severe ($\mu_1 = 1$, $\mu_2 = 0.1$) and the effect of maturity on growth is large ($k_1 = 1$, $k_2 = 0.1$), the optimal size at maturity M^* is close to w_p , once w_p exceeds a certain threshold. (c and d) When the predation effect is weaker ($\mu_1 = 0.6$, $\mu_2 = 0.5$), or (e and f) the maturity effect is weaker ($k_1 = 1$, $k_2 = 0.6$) there is still an optimal size at maturity, but it no longer approximates w_p .



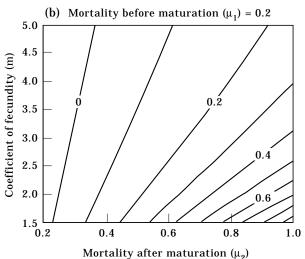


Fig. 6. The contour map of the optimal maturation age. (a) For a given coefficient of fecundity, the optimal maturation age increases with the difference between the mortalities after and before maturation ($\mu_2 - \mu_1$). If the difference is equal to or less than zero, the optimal age becomes zero. (b) For a given mortality before maturation, the optimal maturation age also depends on the proportional coefficient of fecundity (m). As m increases, it decreases. It implies that large fecundity causes that late maturation is unnecessary.

 M^* as a function of the parameters. M^* increases as the difference of mortalities between after and before maturation increases, becoming zero when the two mortalities are equal. The optimal maturation age also depends on the coefficient of fecundity (m). As m increases, M^* decreases. This implies that high fecundity may remove the selective pressure for later maturation.

When n > 1, the sign of the r.h.s. of eqn (19b) depends on the mortalities after and before maturation. Therefore, delayed maturation may be favored. M^* is expected to decrease as n increases

because the higher potential of fecundity leads to early maturation.

7. Discussion

As explained in the introduction, two hypotheses on delayed maturation has been already proposed. We propose here the third hypothesis, based on Conditions 3 and 5, that the abrupt change of mortality or growth rate after maturation can be a selective force for delayed maturation. While the first two hypotheses insisted that delayed maturation gives an advantage for the number of offspring or survival rate of offspring, our hypothesis insists that a relative disadvantage after maturation leads to delayed maturation. Although our hypothesis holds theoretically as presented in the present paper, a question remains unsolved. That is what kind of mechanism leads to the abrupt change of mortality or growth rate after maturation.

In general, maturation and reproduction after it are accompanied with some cost. For example, in perennial plants, formation of reproductive organs or investment to seeds consumes much assimilating products and it leads to the decreases of growth rate or of survival rate (Eis et al., 1965; Sohn & Policansky, 1977; Law, 1979). In fish, the development of spermary or ovary also consumes much energy and the similar decreases are observed. It is generally called 'cost of reproduction'. Therefore, the abrupt changes after maturation in mortality or growth rate are expected to occur with high probability due to cost of reproduction. The assumption of our hypothesis could be caused by cost of reproduction. Growth or mortality may change with maturity for other than cost of reproduction. Behavior involved with reproduction may make mature individuals more susceptible to predators, or reduce their opportunities for feeding, or involve them in potentially injurious territorial disputes. For some species, maturity involves a metamorphosis that completely changes the habitat of the mature individual (e.g. amphibians and insects with aquatic larvae).

Conditions 3 and 5 show that, whatever their source, such differences may be enough to select for delayed maturity. The empirical or observational, not theoretically, studies would be necessary in order to certify whether cost of reproduction or other reasons could be a selective force for delayed maturation.

We note that finding empirical evidence for these changes is more direct than finding dependence of the vital rates on the size at maturity, which is the assumption of the first and the second hypotheses. The third hypothesis requires only a comparison of the vital rates of mature and immature individuals of the same size, or the identification of a discontinuity in a size-specific growth or mortality function at the size at maturity.

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APPENDIX A

In eqn (2), r is implicitly a function of the maturation size (M) as:

$$\int_{M}^{\infty} \frac{m(w; M)}{g(w; M)} l(w, w_0; M) e^{-r(M)\tau(w, w_0; M)} dw = 1. \quad (A.1)$$

Therefore, differentiating both sides of eqn (A.1) with respect to M,

$$-\frac{m(M; M)}{g(M; M)} l(M, w_0; M) e^{-r\tau(M, w_0; M)}$$

$$+ \int_{M}^{\infty} \frac{\partial}{\partial M} \left(\frac{m}{g}\right) l e^{-r\tau} dw - \int_{M}^{\infty} \frac{m}{g} l \frac{\partial s}{\partial M} e^{-r\tau} dw$$

$$+ \int_{M}^{\infty} \frac{m}{g} l e^{-r\tau} \left\{ -\frac{\partial r}{\partial M} \tau - r \frac{\partial \tau}{\partial M} \right\} dw = 0,$$

where

$$s(w, w_0; M) = \int_{w_0}^{w} \frac{\mu(x; M)}{g(x; M)} dx$$
 [eqn (6)].

Since $\partial r/\partial M$ and r are not functions of the integral variable (w), we can obtain eqn (5).

APPENDIX B

We, here, obtain the condition such that $\partial s / \partial M = 0$. Since $M < w < \infty$ [from eqn (2)], s can be written as

$$s(w, w_0; M) = \int_{w_0}^{M} \frac{\mu(x; M)}{g(x; M)} dx + \int_{M}^{w} \frac{\mu(x; M)}{g(x; M)} dx.$$
(B.1)

Thus,

$$\frac{\partial s}{\partial M} = \frac{\mu(m-;M)}{g(M-;M)} - \frac{\mu(M+;M)}{g(M+;M)} + \int_{w_0}^{w} \frac{\partial \left(\frac{\mu}{g}\right)}{\partial M} dx.$$
(B.2)

From eqn (B.2), the condition such that $\partial s/\partial M = 0$ is that

$$\frac{\partial \left(\frac{\mu}{g}\right)}{\partial M} = 0 \quad \text{and} \quad \frac{\mu(M-;M)}{g(M-;M)} = \frac{\mu(M+;M)}{g(M+;M)}. \quad (B.3)$$

That is,

$$\frac{\mu(w; M)}{g(w; M)} \equiv f_2(w)$$
 and $f_2(M-) = f_2(M+)$. (10)

From eqn (B.2), the third term on the r.h.s. of eqn (5) can be positive if:

Condition 2

$$\frac{\partial(\mu/g)}{\partial M} < 0,$$

or

Condition 3

$$\frac{\mu(M-;M)}{g(M-;M)} < \frac{\mu(M+;M)}{g(M+;M)}$$

APPENDIX C

The derivation of the mathematical condition that $\partial \tau / \partial M = 0$ is similar to Appendix B.

$$\tau(w, w_0; M) = \int_{w_0}^{M} \frac{1}{g(x; M)} dx + \int_{M}^{w} \frac{1}{g(x; M)} dx.$$
(C.1)

Thus,

$$\frac{\partial \tau}{\partial M} = \frac{1}{g(M-;M)} - \frac{1}{g(M+;M)}$$
$$- \int_{w}^{w} \frac{1}{g^2} \frac{\partial g}{\partial M} dx. \quad (C.2)$$

Therefore, the condition such that $\partial \tau / \partial M = 0$ is

$$\frac{\partial g}{\partial M} = 0$$
 and $g(M - ; M) = g(M + ; M)$. (C.3)

That is,

$$g(w; M) \equiv f_3(w)$$
 and $f_3(M-) = f_3(M+)$. (11)

From eqn (C.2), the 4th term on the r.h.s. of eqn (5) can be positive if:

Condition 4

$$\frac{\partial g}{\partial M} > 0,$$

or

Condition 5

$$g(M - ; M) > g(M + ; M).$$

APPENDIX D

The survivorship, $l(w, w_0)$, depends on both the coefficients of growth rate and the mortalities. The values of these parameters depend on size as shown in Fig. D1. Therefore,

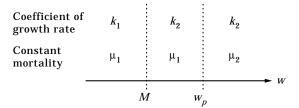
$$l(w, w_0) = \exp\left\{-\int_{w_0}^{M} \frac{\mu_1 dw}{k_1 (w_{\text{max}} - w)} - \int_{M}^{w_p} \frac{\mu_1 dw}{k_2 (w_{\text{max}} - w)} - \int_{w_p}^{w} \frac{\mu_2 dw}{k_2 (w_{\text{max}} - w)}\right\}$$

$$= G_1 (w_{\text{max}} - w)^{\mu_2/k_2}$$
 (D.1)

in Case 1, where

$$G_{1} = \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_{0}}\right)^{\mu_{1}/k_{1}} \left(\frac{w_{\text{max}} - w_{p}}{w_{\text{max}} - M}\right)^{\mu_{1}/k_{2}} (w_{\text{max}} - w_{p})^{-\mu_{2}/k_{2}}.$$
(D.2)

Case I



Case II

Coefficient of growth rate
$$k_1$$
 k_1 k_2

Constant μ_1 μ_2 μ_2 μ_2
 W_0 M

Fig. D1. The values of the coefficients of growth rate and the mortalities. The survivorship, $l(w, w_0)$, depends on both parameters in each size class.

Similarly,

$$l(w, w_0) = G_2(w_{\text{max}} - w)^{\mu_2/k_2},$$
 (D.3)

where

$$G_{2} = \left(\frac{w_{\text{max}} - w_{p}}{w_{\text{max}} - w_{0}}\right)^{\mu_{1}/k_{1}} \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_{p}}\right)^{\mu_{2}/k_{1}} (w_{\text{max}} - M)^{-\mu_{2}/k_{2}}$$
(D.4)

in Case 2. Therefore, substituting eqns (15), (D.1) and (D.2) into eqns (2) and (5), we obtain eqn (16) in Case 1 and

$$m \exp[-A(r + \mu_1) + B(\mu_1 - \mu_2)]$$

$$\left\{ \frac{w_{\text{max}}}{r + \mu_2} - \frac{w_{\text{max}} - M}{r + \mu_2 + k_2} \right\} = 1$$

$$A = -\frac{1}{k_1} \ln \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_0} \right), \quad B = -\frac{1}{k_2} \ln \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_p} \right)$$

$$G_{2} = \left(\frac{w_{\text{max}} - w_{p}}{w_{\text{max}} - w_{0}}\right)^{\mu_{1}/k_{1}} \left(\frac{w_{\text{max}} - M}{w_{\text{max}} - w_{p}}\right)^{\mu_{2}/k_{1}} (w_{\text{max}} - M)^{-\mu_{2}/k_{2}} \qquad \frac{dr}{dM} \left\{ A \exp[A(r + \mu_{1}) - B(\mu_{1} - \mu_{2})] + \frac{mw_{\text{max}}}{(r + \mu_{2})^{2}} - \frac{m(w_{\text{max}} - M)}{(r + \mu_{2} + k_{2})^{2}} \right\}$$
in Case 2. Therefore, substituting eachs (15), (D.1)

$$= \frac{m}{r + \mu_2 + k_2} - \left\{ \frac{m w_{\text{max}}}{(r + \mu_2)} - \frac{m (w_{\text{max}} - M)}{(r + \mu_2 + k_2)} \right\} \times \frac{r + \mu_2}{k_1 (w_{\text{max}} - M)} \quad \text{(AD.5b)}$$

in Case 2 eqn (17) can be obtained from the r.h.s. of eqn (D.5b) be equal to zero.