The Effects of Predation on the Age and Size of Maturity of Prey

Peter A. Abrams; Locke Rowe

Evolution, Vol. 50, No. 3 (Jun., 1996), 1052-1061.

Stable URL:
http://links.jstor.org/sici?sici=0014-3820%28199606%2950%3A3%3C1052%3ATEOPOT%3E2.0.CO%3B2-W

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

Evolution, 50(3), 1996, pp. 1052–1061

THE EFFECTS OF PREDATION ON THE AGE AND SIZE OF MATURITY OF PREY

PETER A. ABRAMS[1,2] AND LOCKE ROWE[3]
[1]*Department of Ecology, Evolution, and Behavior, University of Minnesota, 1987 Upper Buford Circle, Saint Paul, Minnesota 55108*
[3]*Department of Zoology, University of Toronto, Toronto, Ontario M5S 1A1, Canada*
E-mail: lrowe@zoo.toronto.edu

Abstract.—The effects of nonselective predation on the optimal age and size of maturity of their prey are investigated using mathematical models of a simple life history with juvenile and adult stages. Fitness is measured by the product of survival to the adult stage and expected adult reproduction, which is usually an increasing function of size at maturity. Size is determined by both age at maturity and the value of costly traits that increase mean growth rate (growth effort). The analysis includes cases with fixed size but flexible time to maturity, fixed time but flexible size, and adaptively flexible values of both variables. In these analyses, growth effort is flexible. For comparison with previous theory, models with a fixed growth effort are analyzed. In each case, there may be indirect effects of predation on the prey's food supply. The effect of increased predation depends on (1) which variables are flexible; (2) whether increased growth effort requires increased exposure to predators; and (3) how increased predator density affects the abundance of food for juvenile prey. If there is no indirect effect of predators on prey food supply, size at maturity will generally decrease in response to increased predation. However, the indirect effect from increased food has the opposite effect, and the net result of predation is often increased size. Age at maturity may either increase or decrease, depending on functional forms and parameter values; this is true regardless of the presence of indirect effects. The results are compared with those of previous theoretical analyses. Observed shifts in life history in response to predation are reviewed, and the role of size-selective predation is reassessed.

Key words.—Age at maturity, development time, food supply, foraging effort, growth rate, life history, optimization, predation, size at maturity.

Received March 24, 1994. Accepted May 4, 1995.

Because of their importance in determining fitness, age and size at maturity are two of the most frequently studied parameters of the life history (see reviews in Roff 1992; Kozłowski 1992; Stearns 1992). Predation typically exerts strong selection pressures on life-history traits (Endler 1986), and some studies of populations subjected to different forms or intensities of predation have responded with evolutionary shifts in either or both age and size of maturity. Phenotypic plasticity in these traits is also common. Both genetic and phenotypic shifts have usually been attributed to the size selectivity of the predator (see Discussion). However, in most cases, prey mortality rates have not been measured directly, so it is not clear whether the imposed mortality was acutally size selective. Our goal here is to show, using mathematical models, how nonselective predation affects age and size of maturity. Such theory is needed if the life-history effects of predation per se are to be separated from those of predator selectivity.

The need for such theory, and for specifying the form of predation, is illustrated by the well-documented evolutionary shifts in age and size at maturity of Trinidadian guppies, caused by shifts between two predator regimes. Explanations for these observed evolutionary shifts have been based primarily on differences in the size selectivity of the two primary predators (Reznick and Endler 1982; Reznick et al. 1990). Yet recent direct estimates of the size-mortality relationships under two predator regimes demonstrate differences in absolute mortality rates, but not in the relative mortalities of different size classes (Reznick et al. 1996). Thus, the size-selectivity hypothesis appears to be either incomplete or incorrect in this case (Reznick et al. 1996). This leads to the question, can nonselective predation account for the same observations?

In this paper, we analyze a series of simple optimization models to determine how nonselective predation should affect the age and/or size of maturity in organisms having a simple life history. The life history is characterized by juvenile and adult stages, and the predator can prey on either juveniles only, or both adults and juveniles. The models are designed to investigate three independent mechanisms by which predation may affect adaptive shifts in age and size at maturity. First, greater predation represents a higher mortality, and this, in itself, may select for changes in the life history. Second, because predation risk usually increases with increased growth rate, increased predator density is expected to increase the costs of rapid growth and thus the trade-off between growth and mortality in the juvenile stage. The increased cost of rapid growth is expected to affect both growth rate and perhaps the optimal age and size of maturity. Thirdly, predation changes prey population densities and may change prey foraging behavior. Both of these effects are likely to change density-dependent components of the life history, including the abundance of the prey's food. The altered food density (or other density-dependent factors) may then have an indirect effect on the optimal age and size of maturity.

We investigate several forms of the life history, ranging from those with constrained age or size at maturity to those where both traits are flexible. In contrast to much of the previous theory (Roff 1992; Stearns 1992), but in accord with the empirical literature (Sih 1987; Lima and Dill 1990; Nylin et al. 1993), we allow growth effort to vary in response to increased predation risk. Thus, our theory provides predictions for the direct effect of predation on optimal growth effort, and on both age and size at maturity. To compare our

[2] Present address: Department of Zoology, University of Maryland, College Park, Maryland 20742; E-mail: abrams@zool.umd.edu

results with previous theory, we also consider a case where growth effort is fixed. In each case, we allow for indirect effects on prey food supply and thereby provide predictions regarding combined direct and indirect effects. Finally, we review some relevant empirical work, suggest empirical issues that require more attention, and contrast our theory with previous theory.

Models and Analysis

All of the models discussed here focus on the juvenile stage of the life history. This is because, given the assumptions made below, traits expressed by the end of the juvenile stage determine the optimal age and size of maturity. We allow for flexibility in traits that increase growth rates, but also increase mortality (e.g., time spent foraging). We consider three formulations of the life history that may be arranged along a contiuum. At one end are life histories where development time is fixed. For example, strongly seasonal environments may constrain the transformation to maturity to a brief period of time (see Ludwig and Rowe 1990; Rowe and Ludwig 1991). At the other end are cases where size at maturation is constrained to a narrow range of values. Only size at maturity in the first case, and only age at maturity in the second case, are free to evolve or respond plastically to the environment. Although few animals may be under such strong constraints on age or size of maturation, considering the ends of this continuum is illustrative. Finally, both age and size at maturity may be plastic and/or free to evolve. We also compare all of these results with those that follow from assuming that growth rates are fixed, but that both age and size at maturity are plastic. This has been the most common assumption in previous life-history models (e.g., Roff 1992).

In all of the models fitness is measured by expected lifetime reproduction (R_o). This implies either a stable population or a population with discrete generations. All of the models considered here are variants of a model presented in Abrams et al. (1996). Age at maturity (development time) will be denoted by T. Size at maturity, denoted S, is determined by development time and mean proportional growth rate, denoted by g. The underlying model of individual growth is exponential. The rate of increase in size, s, during the juvenile period, ds/dt, is given by the product of growth rate and size. Thus, size at maturity, S, is exp(gT), assuming that size is measured in units equal to the initial size. Alford and Jackson (1993) have shown that this is often an accurate description of juvenile growth. We assume that initial size is not evolutionarily flexible. If it were, initial size would have to be considered as a variable in determining optimal life histories. Juvenile growth rate, g, is the result of the interaction of traits related to metabolism and foraging with the food supply. The value of a costly trait that promotes growth will be denoted C, and will be referred to as "effort." Effort could be any morphological or behavioral trait that increases food consumption or efficiency of food utilization, but decreases some other component of fitness. Potential examples are: (1) physiological traits that increase growth rate for a given food intake, but have a negative pleiotropic effect on ability to survive adverse physical conditions (e.g., decreased storage capacity); or (2) the proportion of time an organism spends searching for food, which determines exposure to predators. The mean availability of food over the course of the juvenile period will be denoted R. Thus, growth rate is a function of C and R. Growth rate increases with increases in either C or R (i.e., $\partial g/\partial C > 0$ and $\partial g/\partial R > 0$). It is important for the following analysis to know the signs of the second partial derivatives, $\partial^2 g/\partial C^2$ and $\partial^2 g/\partial C \partial R$. Each second derivative can potentially be positive or negative. However, it seems most likely that $\partial^2 g/\partial C^2$ will be negative, because of the likelihood of diminishing returns from increased development of a particular growth-promoting trait. It also seems likely that $\partial^2 g/\partial C \partial R$ will be positive. This mixed second derivative describes how an increase in food affects the slope of the relationship between g and C, and the growth increment from a unit increase in effort will usually be larger when more food is available. However, if the growth rate is close to some upper limit, then increased food may make growth less sensitive to effort, by bringing it closer to the asymptotic value.

The cost of increased growth effort is an increased mortality rate. Here, there are two possible mechanisms. Increased effort may increase exposure to a particular set of predators, as is likely when C represents a trait like time spent foraging. Alternatively, increased effort may increase other components of mortality. The mortality rate due to the focal predator is denoted m(C)P, where P is the density of the focal predator. Mortality rate due to other factors is u(C). For convenience, we will refer to these two types as predatory and nonpredatory mortality, even though u may include mortality from other predators. The probability of survival to maturity is therefore given by $\exp(-uT - mPT)$. Both m and u are increasing and are likely to have nonnegative second derivatives; increasing effort increases the costs at an accelerating rate (see Abrams 1991a,b; Abrams et al. 1996 for a discussion of the possibility of a negative second derivative). Effort and mortality may change systematically as an animal grows during the juvenile period, but even in such cases, it may be possible to represent mean mortality as a function of mean effort.

Reproductive rate is assumed to increase with size at maturity. For mathematical convenience, this relationship will be defined in terms of logarithms. The logarithm of expected reproduction is related to the logarithm of size at maturity by the function ϕ, where ϕ is increasing or unimodal. Thus, expected reproduction is $\exp(\phi(g(C, R)T))$. In general, there are some constraints on reproductive rate other than those related to size. As a result, the second derivative of the reproduction function, ϕ'', will often become negative at large values of gT. However, if there is size-dependent contest competition among adults, then ϕ'' may be positive, even at large sizes. If reproduction is a power function of size, ϕ is linear and $\phi'' = 0$.

Adult fitness may depend on the difference between the actual development time (age at maturity) and the time that would maximize the adult component of fitness (e.g., the development time that results in maturity when high quality oviposition sites are most abundant). In this case adult fitness is multiplied by the factor $\exp(-f(T))$, where f is either increasing (when $T = 0$ maximizes adult fitness) or has an intermediate minimum at the adult-optimal time to maturity. In either case, the second derivative of f (denoted f″) will be positive, because the cost of a given change in timing increases as it departs from the optimal value.

TABLE 1. Assumptions about fitness components.

	Derivatives with definite signs*	Derivatives with indeterminate signs; most likely** sign given
g (C, R)	$\partial g/\partial R > 0$; $\partial g/\partial C > 0$	$\partial^2 g/\partial C^2 \leq 0$; $\partial^2 g/\partial R^2 \leq 0$; $\partial^2 g/\partial C \partial R \geq 0$
ϕ (gT)	$\phi' > 0$	$\phi'' \leq 0$
f (T)	$f'' > 0$	$f' > 0$
u (C)	$u' > 0$; $(u'' \geq 0)$	
m (C)	$m' > 0$; $(m'' \geq 0)$	

* Parentheses denote relationships that are much more likely than the opposite, but whose sign is not completely determinate.
** These signs reflect the authors' biological intuitions.

The expected adult reproductive output depends on adult mortality. However, when mortality is independent of size, it reduces the lifetime reproductive outputs of all adults by the same proportion. This means that it simply rescales adult fitnesses, and can be ignored when determining the evolutionarily favored trait values. We also assume that, if there are effects of population density on adult survival or reproduction, these affect all sizes proportionately, and therefore do not affect the relative fitnesses of differently sized individuals. Growth during the adult stage is not modeled explicitly. However, the present model is consistent with adult growth, provided that expected adult fitness can still be measured as an increasing function of size at maturity. Semlitsch et al. (1988) found that this assumption was met for an amphibian that grew significantly after maturity.

In summary, the optimal life history is defined as those values of C and T that maximize fitness (denoted W), which is the probability of survival to maturity multiplied by the expected reproductive output of an adult of the resulting size (S); i.e.,

$$W = \exp\{-m(C)TP - u(C)T + \phi(g(C, R)T) - f(T)\} \tag{1a}$$

Maximizing fitness is equivalent to maximizing the logarithm of fitness, which is the reason for using logarithmic relationships in the above discussion. Thus, fitness may also be measured by:

$$\ln(W) = -m(C)TP - u(C)T + \phi(g(C, R)T) - f(T) \tag{1b}$$

We have discussed the shapes of the various functions above, and this discussion is summarized in Table 1. The table divides our assumptions about the signs of the derivatives of functions into those we consider to apply generally, and those where at least two alternatives are possible. In the latter case, we identify the alternative that we feel is most probable for most biological systems. The remainder of the analysis seeks to determine how the optimal values of C and T change as predator density changes. These two traits determine the optimal size at maturity, S. We consider the simpler cases of fixed age at maturity and fixed size at maturity before considering the more general case in which neither of these traits is fixed. When nonpredatory mortality is insignificant ($u \approx 0$), the results simplify considerably, so the life-history responses for this case are presented separately. The results are summarized in Tables 2 and 3, and the mathematical analysis is largely relegated to the appendices.

Fixed Age at Maturity

In this case, the age at maturity, T, may be scaled to unity, and does not enter into the calculations. Similarly, the time-dependent function, f, can be ignored, because its value is constant when time to maturity is constant. Expected survival is given by $\exp(-u(C) - m(C)P)$. Expected adult fitness is

TABLE 2. Responses of optimal time to maturity (T_o), growth effort (C_o), size (S_o), and growth rate ($g(C_oR)$) to increased predation, when nonpredatory mortality (u) is insignificant. Zero denotes no change, ↑ denotes an increase in the optimal value, and ↓ denotes a decrease. Parentheses denote comparatively unlikely responses.

Variable	T_o	C_o	S_o	$g(C_oR)$
A. Direct responses to predator density				
Model type				
Fixed age	0	0 or ↓	0 or ↓	0 or ↓
Fixed size	↑	↓	0	↓
Flexible	↑ or ↓	↓	↓	↓
Fixed effort	↓	0	↓	0
B. Indirect responses to predator density via food supply				
Model type				
Fixed age	0	↑ or ↓	↑	↑
Fixed size	↓ (↑)	↑ or ↓	0	↑ (↓)
Flexible	↑ or ↓	↑ or ↓	↑	↑ (↓)
Fixed effort	↑ or ↓	0	↑	↑

TABLE 3. Responses of optimal time to maturity (T_o), growth effort (C_0), size (S_o), and growth rate ($g(C_oR)$) to increased predation (general case). Zero denotes no changes, ↑ denotes an increase in the optimal value, and ↓ denotes a decrease. Parentheses denote comparatively unlikely responses.

Variable	T_o	C_o	S_o	$g(C_oR)$
A. Direct responses to predator density				
Model type				
Fixed age	0	0 or ↓	0 or ↓	0 or ↓
Fixed size	↓ or ↑	↑ or ↓	0	↑ or ↓
Flexible	↑ or ↓	↑ or ↓	↓	↑ or ↓
Fixed effort	↓	0	↓	0
B. Indirect responses to predator density via food supply				
Model type				
Fixed age	0	↑ or ↓	↑	↑
Fixed size	↓ (↑)	↑ or ↓	0	↑ (↓)
Flexible	↑ or ↓	↑ or ↓	↑	↑ (↓)
Fixed effort	↑ or ↓	0	↑	↑

an increasing function of the logarithm of size at maturity; because $T \equiv 1$, size at maturity is equal to the growth rate. This allows the two functions, ϕ and g, to be combined into a single function, denoted $g^*(C, R)$, that indicates the effects of effort and food density on both growth rate and the logarithm of reproductive output. Thus, expected reproductive output is $\exp(g^*(C,R))$, and fitness is maximized if $g^*(C,R) - u(C) - m(C)P$ is maximized. The growth-and-reproduction function, g^*, is increasing. Appendix 1 derives the effect of predation on the optimal effort, C_o, which is proportional to its effect on the optimal size.

Appendix 1 shows that, if increased growth requires increased exposure to predation, the direct effect of higher predator density is to decrease growth effort so prey individuals decrease risk. Given the fixed age at maturity, this implies a smaller size. If predation is simply an unavoidable mortality source ($m' = 0$), it has no direct effect on the optimal life history. However, the indirect effect of increased predator density, caused by increased food supply, is typically an increase in growth rate and hence size at maturity. The increased growth rate may be accompanied by increased or decreased growth effort, depending on the shape of the growth rate function. Because the direct and indirect effects of increased predator density on growth rate and size at maturity are of opposite sign, their combined effect cannot be generalized, although under many conditions, the indirect effect will be larger.

Fixed Size at Maturity

In this situation, time to maturity, T, must be inversely proportional to the growth rate, g, for size to remain constant. This means that expression (1b) for the logarithm of fitness may be written as:

$$\ln(W) = \Phi - [m(C)P + u(C)]/g(C, R) - f(1/g(C, R)), \quad (2)$$

where Φ is the constant value of the reproductive output function ϕ. Appendix 2 derives the effects of increased predation on optimal effort and time to maturity.

Appendix 2 shows that the direct effect of predation depends on the relative magnitudes of the effects of effort on nonpredatory and predatory mortality. The results may be summarized as follows. If risk of predation is unaffected by the prey's growth strategy ($m' = 0$), increased predator density has the direct effect of increasing growth effort, which decreases risk by decreasing the optimal age at maturity. If there is a trade-off between predation risk per unit time and growth effort, more rapid growth increases risk per unit time at the same time it reduces the period of exposure to predators. The direct effect of predators is then often to increase age at maturity and decrease growth rate. However, this is generally true only when the nonpredatory costs of effort are small relative to predatory costs (see Table 2). The indirect effects via increased food usually counteract the direct responses. When there are indirect effects, the net effect of predators cannot be predicted without a detailed knowledge of the system.

Flexible Age and Size at Maturity

In the most general case, age and size at maturity are both adaptively flexible, and fitness expression (1b) cannot be simplified. The analysis of this case is presented in the second section of Appendix 2. The direct response of size to predation is to decrease. However, the direct responses of optimal age at maturity and growth effort to predation are more difficult to generalize; there are biologically reasonable circumstances under which C_o or T_o (but not both) may increase in response to increased P. If the nonpredatory costs of effort are insignificant, growth rate and growth effort decline in response to predation, but time to maturity may increase (Table 2). When nonpredatory costs of growth effort are significant, effort may decrease (to reduce risk per unit time) or increase (to reduce total time of exposure to risk). Time to maturity can increase when effort exhibits a larger proportional decrease.

Indirect effects again complicate the situation. Although the responses of C_o and T_o to increased food may be positive or negative, the response of size to increased food is virtually always positive (and must be positive whenever $\partial^2 g/\partial C \partial R$ is positive). Thus, the indirect response of size to the increased food caused by predation counteracts the direct response. As noted in earlier cases, the net effect on size depends on the population dynamic details, but it is easy to find functions that make the indirect positive response larger than the direct negative response (i.e, optimal size at maturity increases with increased predation).

Fixed Growth Effort

In this last section we consider a life history where growth effort (C) is fixed. This is a common assumption of previous life-history models. It may actually occur when the theoretically optimal value is greater than the largest possible effort. Here we consider the effect of such a fixed growth effort within our modeling framework. In this case, the value of C can be scaled to $C = 1$, and the fitness expression becomes $\phi(g(R)T) - f(T) - m_1PT - u_1T$, where the subscript 1's denote that these functions now are constants. The resulting expression for the effect of predator density on T_o is:

$$\partial T_o/\partial P = [-m_1 + (\partial R/\partial P)(\partial g/\partial R)(\phi' + gT\phi'')]/[-g^2\phi'' + f''] \quad (3)$$

When there is no indirect effect of predators on food, this expression must be negative; age at maturity will decrease in response to predation. Because C is constant, reducing T is the only way to reduce predation risk, and a shorter development period also entails a smaller size.

If there is an indirect effect of predators on food, the effects on size and age at maturity again depend on population dynamic assumptions. However, when all density dependence occurs via effects on the juvenile food supply, the indirect effect of predators on food is given by $\partial R/\partial P = m_1/(\phi' \partial g/\partial R)$. This follows from results in Appendix 1; in conjunction with Eq (3), it implies that the summation of direct and indirect effects of predators on age at maturity is positive (negative) when ϕ'' is positive (negative). Furthermore, the net direct and indirect effects on the logarithm of size are given by

$(m_1 Tf'')/[\phi'(-g^2\phi'' + f'')]$; this must be positive, implying that predators increase the optimal size of their prey. (Note that the denominator of this fraction must be positive if there is an intermediate optimum value of T.)

Summary

All of the preceding results are summarized in Table 2 (for the special case of u = 0) and Table 3 (for the general case). Although there are many possible responses to juvenile-stage predation, the direct response of size at maturity is almost always a decrease when it is free to vary adaptively. If one of the two adaptive variables, growth effort (C) or age of maturity (T), is fixed, the direct response of the other to predation is a decrease (or no change). However, when both C and T can vary, they may either increase or decrease as a direct response to predation. The outcome depends largely on the sensitivity of predation risk to growth effort. A high sensitivity generally means that effort decreases as a direct response to predation, while a low sensitivity means that effort increases. The optimal age at maturity often changes in the opposite direction to growth effort. When reproductive output is proportional to size (or is any power function of size) both effort and age at maturity must decrease in response to increased predation, assuming no indirect effects. There are certainly many more studies demonstrating a predation-related cost to growth than nonpredation related costs (Lima and Dill 1990; Werner and Anholt 1993). Under these conditions, the only direct response to predation that is indeterminate in sign is that of age at maturity when age, effort, and size are all flexible (Table 2).

In most cases, the food-mediated indirect effect on prey size favors an increase in size, and the combined direct and indirect responses of optimal size to predation can be positive. The indirect responses of effort and age at maturity are more difficult to predict. It is usually necessary to know something about the two mortality rate functions, m and u, and the size-advantage function, ϕ, to make predictions. In many cases, however, the indirect effect will be opposite to and larger in magnitude than the direct effect.

Before concluding this analysis, something more should be said about the possibility that predators consume adults as well as juveniles. As noted before, there is no direct effect of predation on adults on the optimal age and size at maturity, provided the adult-stage predation affects the fitnesses of all adult sizes proportionally. However, predation on adults can affect the magnitude of the indirect effect of predators on the juvenile food supply. All else being equal, predation on adults (as well as juveniles) will result in smaller juvenile population size, and therefore an even larger increase in the juvenile food supply. By the same token, if predation only occurs at the adult stage, there will only be indirect effects on age and size at maturity, which are both likely to be positive.

Discussion

The theory we have presented here differs in three main ways from most previous theory on prey life-history responses to increased predation. First, we allow prey to adaptively vary their growth effort (e.g., risk taking) in a continuous fashion in response to increased predation. At least at the behavioral level, there is much evidence for such adaptive adjustment of growth effort to predation risk (reviews in Sih 1987; Lima and Dill 1990). Gotthard et al. (1994) present evidence for physiological costs of increased growth in a butterfly species with flexible growth rates, indicating the presence of nonpredatory costs of growth. We show that inclusion of flexible growth rates can have dramatic effects on the predictions. Second, we focus on nonselective rather than size-selective predation (see also Kozłowski and Wiegert 1987; Kozłowski and Uchmanski 1987; Kozłowski 1992; Kawecki 1993). This analysis demonstrates that evolution (or plastic adjustment) of age and size at maturity are expected from changes in the magnitude of predation alone. Finally, we allow for indirect effects (increased prey food supply) of increased predation. We show that indirect effects often work in opposition to, and may even exceed, the direct effects of predation on the components of the life history.

We begin this discussion by reviewing previous theory on the effect of predation on age and size of maturity. In the remaining discussion, we review some well known empirical examples of predator effects on prey life histories and suggest some alternative explanations based on the theory we have developed here. We then make some suggestions for further experimental work, which we think will help bring into focus the mechanisms underlying life-history evolution.

Previous Theory

Most previous models of optimal age and/or size of maturity have not included either explicit trade-offs between growth rate and instantaneous risk of mortality or indirect effects via prey resources (e.g., Gadgil and Bossert 1970; Taylor et al. 1974; Law 1979; Michod 1979; Charlesworth 1980; Roff 1981; Kusano 1982; Stearns and Koella 1986; Kozłowski and Uchmanski 1987). These studies are reviewed in Roff (1992). He points out that those models that have assumed age-dependent rather than stage-dependent mortality (e.g., Law 1979; Michod 1979) predict that higher mortality on young age classes will increase age at maturity. On the other hand, if the mortality is stage-dependent (as is true in our models), higher juvenile mortality favors earlier maturity, which, given a fixed growth rate, also means maturity at a smaller size. Kawecki (1993) investigated a model of life-history evolution in patchy environments, and noted that reduced competition within patches as the result of higher stage-specific mortality could favor a later age of maturity. This seems to have been the only previous consideration of indirect effects from predation. It should also be noted that the effects of predation differ in growing and stable prey populations. In the former case, higher predation reduces r, which generally favors later maturation (Roff 1992).

Several previous works have specifically focussed on the relationship between age of maturity and size at maturity in environments characterized by different, but fixed, growth rates (Stearns and Koella 1986; Gebhardt and Stearns 1988; Perrin and Rubin 1990; Houston and McNamara 1992; Kawecki and Stearns 1993). All of these studies call the relationship between age and size of maturity a "reaction norm." Because a reaction norm describes phenotype as a function

of the environment, this usage is only defensible if there is a simple mapping between one of the two traits and an environmental variable. If growth rates are fixed, there may be a simple functional relationship between size and environment. However, this is clearly not the case when growth rate is flexible in response to food or predator density, as is true in most of our models (see also Abrams et al. 1996). In this case, "reaction norm" can be applied to the separate relationships between growth rate or age of maturity and any of several environmental variables. Although it is not their main focus, some of the articles listed above do contain results regarding the response of age and size at maturity to mortality. Stearns and Koella (1986) and Stearns (1992) suggest that greater externally imposed juvenile mortality increases both age and size of maturity. This contradicts both the present results and those of Roff (1981) and Kusano (1982). It seems to be due to two key assumptions: (1) that earlier maturity implies a higher juvenile mortality rate, independent of size of maturity; and (2) greater externally-imposed mortality increases the mortality rate due to early maturation in a multiplicative way. There is very little evidence to support the first assumption in most organisms (Stearns and Koella [1986] cite the fact that offspring of very young human females have higher infant mortality rates.) There appears to be no evidence for the second assumption.

There have been several previous studies in which a trade-off between growth and mortality was incorporated into the model. The best known of these is Gilliam's (Gilliam 1982; Gilliam and Fraser 1987), "minimize μ/g" rule. This describes the optimal growth strategy of organisms under no time constraints in a stable population. This model is equivalent to our model for a fixed size at maturity, under the additional assumption that there is no seasonality ($f = 0$). Werner (1986) has applied the "minimize μ/g" rule to examine optimal age and size at metamorphosis in amphibians. Aquatic and terrestrial habitats were assumed to have different size specific growth and mortality curves. This is somewhat different from the question of the optimal age of maturity, because considerable growth can occur between metamorphosis and first reproduction. Werner's analysis predicts that higher larval mortality (due to predators or other factors) results in earlier metamorphosis (see also Rowe and Ludwig 1991). However, this does not consider the possibility that both growth and mortality curves in a given habitat can be affected by behavior. Houston et al. (1993) have recently reanalyzed the effects of predation under Gilliam's (1982) model, which applies to the case of a fixed size with no indirect effects. Their results are essentially identical to those reached here, although they did not consider the case when growth-effort affects both predatory and nonpredatory mortality.

Ludwig and Rowe (1990) and Rowe and Ludwig (1991) also analyzed models of age and size of maturity that included a growth/mortality trade-off mediated by predation risk, and (unlike Gilliam's models) included environmental seasonality. Their dynamic programming models assume that the trade-off is mediated by the choice between two potential habitats, one of which has more predators. Except for this discrete choice, and the potential dependence of foraging effort on condition and time, their models are similar to the models of flexible size and time considered here. However, they did not assess the direct or indirect effects of predation on age and size at maturity. Instead, they assessed the relationship between size at metamorphosis (and maturity) and time in the season for a group of individuals varying in condition. It is likely that dynamic programming models like these will be required to investigate optimal strategies under size-selective predation.

Evidence from Nature

There have been relatively few published studies of the response of size and age at maturity to predation. In none that we know of was there any attempt to measure the indirect response of prey food density. In some cases, the predators are known to be selective with respect to prey size. However, the role of this selectivity in producing the observed life-history responses in prey is unclear. In some cases it is possible that responses of nonfocal predators modified or reversed the pattern of size-selectivity the experimenter was attempting to achieve.

Perhaps the most thorough study of the effect of predation on prey life histories is that by Reznick and collaborators (Reznick and Endler 1982; Reznick 1989; Reznick et al. 1990) on the Trinidadian guppy (*Poecilia reticulata*) system. In general, guppies occuring naturally in stream reaches with the predator, *Crenicichla,* mature at a smaller size and a younger age. Moreover, experimental introductions of *Crenicichla* into non-*Crenicichla* reaches results in similar evolution of the life history. These evolutionary shifts were originally attributed to a preference of *Crenicichla* for large sized guppies. However, there is conflicting evidence on size selectivity (Seghers 1973; Mattingly and Butler 1994).

The guppy research is unique in that there has now been direct measurement of the selectivity of the actual mortality regime, which includes the indirect effects of the focal predator on other predators (Reznick et al. 1996). The ratio of mortality rates of differently sized guppies was similar in localities with and without *Crenicichla,* although *Crenicichla* increased total mortality. The source of these mortality differences may or may not result from nonselectivity of *Crenicichla.* Other components of the predator community may covary with *Crenicichla.* Moreover, if size-specific antipredator behaviors occur, then mortality patterns do not necessarily reflect predation risk (Abrams 1993). Nevertheless, there is now no clear evidence of size selective effects.

The observed evolutionary responses of guppies are consistent with the theory presented here for nonselective predation, in the absence of large indirect effects on prey food supply. When both age and size at maturity are flexible, we predict maturation of prey at a smaller size. We also predict that age at maturity will often decrease, and must decrease when adult fitness is approximately proportional to size. The presence of predation on adults and possible presence of size-selectivity do not change our predictions, unless, by growing large enough, individuals can significantly reduce predation. This is unlikely here, because *Crenicichla,* if selective at all, favors large sizes. Indirect effects of increased guppy food supply in *Crenicichla* localities do not appear to be strong. Reznick et al. (1990) found that male guppies on high food

ration matured at larger sizes (and earlier). Because guppies actually matured at smaller sizes in *Crenicichla* localities, we conclude that any positive effect on guppy food supply, resulting from decreased guppy density, was not large enough to outweigh the direct effect of increased predation.

A variety of laboratory studies have examined the effects of predation on age and size at maturity. Crowl (1990) and Crowl and Covich (1990) found that the freshwater snail, *Physella virgaga virgata,* increased both age and size of maturity in the presence of predation by crayfish, which were shown to feed selectively on the smallest snails. The prey's response was incompatible with our predictions for the direct effects of indiscriminate predation, and compatible with models of predator selectivity for small prey. However, the response is also compatible with predictions based on the indirect effect of increased food supply. Skelly and Werner (1990) showed that American toads (*Bufo americanus*) reduced growth rate and size at metamorphosis (maturity) in response to predation risk in the laboratory (see also Wilbur and Fauth, 1990). These results are in accord with our predictions for direct effects of nonselective predation. Skelly and Werner (1990) also showed that the magnitude of the change in size at maturity was much greater when food density was reduced than when a predator was introduced. This suggests that, under some circumstances, the indirect response via food densities could exceed the direct response, and size at metamorphosis could increase.

An indirect, resource-mediated response was found by Werner (1991) in a similar study of the juvenile growth rates of bullfrogs (*Rana catesbiana*) and green frogs (*Rana clamitans*) raised together, both in the presence and absence of a caged predator. Bullfrogs increased growth rate and green frogs decreased growth rate in response to the predator, although both species reduced activity rates by comparable proportions. The increased growth rate of bullfrogs indicates an indirect, resource-mediated response to predation in the opposite direction of that of green frogs (Werner 1991). In this case, the food density changed because of the change in foraging activity (growth effort) rather than a change in consumer population density.

Several studies have noted shifts in development time and size at maturity in populations of water fleas, *Daphnia,* exposed to predation. These include Dodson and Havel (1988), Leibold and Tessier (1991), Spitze (1991), and Black (1993). In most of these studies, there was some size-selectivity by the predator, but there is again no direct evidence that the observed responses were due to the size-selectivity.

Future Empirical Work

The preceding review reveals two main gaps in the empirical literature. The first is in our understanding of the true form of size-specific predation risk. Of the studies reviewed above, only Reznick et al. (1996) have actually measured mortality rates of size classes in the field regimes under which the life history has evolved. Several other studies have laboratory or field enclosure estimates of size selectivity. However, the problem with these single predator assays is that they do not take into account potential indirect effects of the focal predator on other predators. Most species live in environments with many predator species (Lima 1992), and removal (or addition) of a focal species may have indirect effects on other predator species, which may have very different selectivities from the focal predator. Such indirect effects are implicated in Reznick's study of guppy mortality, and in the community-level consequences of bluegill sunfish (*Lepomis macrochirus*) removal studied by Gilinsky (1980). Furthermore, as noted above, mortality is not neccessarily an accurate reflection of predation risk. We suggest that a more thorough accounting of the size-specificity of predation under the different predation regimes should be undertaken, before changes in the life history are attributed to size-selective predation.

Second, emprical studies in both the laboratory and field have not taken into account the potential for indirect effects of predators on their prey's food supply. Although community ecologists have begun to study such indirect effects (e.g. Kerfoot and Sih 1987; Carpenter and Kitchell 1993), evolutionary biologists interested in life histories apparently have not done so. We show that most of the predicted direct responses to predation considered here can be reversed by strong indirect effects on food supply. Responses of food densities can be measured in the field and comparisons of field results with laboratory studies having controlled food supplies provide a method to assess the magnitudes of food-mediated indirect effects.

The theory presented here also suggests that more needs to be known about the costs of growth and the fitness advantages of larger size. More rapid growth can be achieved by changes in behavior, physiology, or morphology. Only those costs related to foraging behavior seem to have received detailed study (e.g., Lima and Dill 1990; but see Gotthard et al. 1994). Knowing the linearity or nonlinearity of the size-advantage function will often allow qualitative predictions to be made in some cases where Table 3 indicates an uncertain response. In other cases, roughly determining the relative sensitivities of predatory and nonpredatory mortality to growth effort would allow a qualitative prediction. It is not necessary to determine the exact form of the functions to make some predictions about the effects of nonselective mortality.

Acknowledgments

PAA was supported by a Mid-Career Fellowship in Environmental Biology (National Science Foundation IBN 9202342) and by National Science Foundation grant DEB 9419716. LR was supported by an NSERC postdoctoral fellowship. Our collaboration was made possible by the hospitality of the Zoology Department at the University of British Columbia. We thank D. Reznick for sharing his thoughts and his unpublished work, and T. Kawecki and M. Mangel for commenting on earlier drafts.

Literature Cited

Abrams, P. A. 1991a. The relationship between food availability and foraging effort: Effects of life history and time-scale. Ecology 72:1242–1252.

———. 1991b. Strengths of indirect effects generated by optimal foraging. Oikos 62:167–176.

———. 1993. Why predation rates should not be proportional to predator density. Ecology 74:726–733.

ABRAMS, P. A., O. LEIMAR, S. NYLIN, AND C. WIKLUND. 1996. The effect of flexible growth rates on optimal sizes and development times. Am. Nat. 147:381–395.

ALFORD, R. A., AND G. D. JACKSON. 1993. Do cephalopods and larvae of other taxa grow asymptotically? Am. Nat. 141:717–728.

BLACK, A. R. 1993. Predator-induced phenotypic plasticity in *Daphnia pulex*: Life history and morphological response to *Notonecta* and *Chaoborus*. Limnol. Oceanogr. 38:986–996.

CARPENTER, S. R., AND J. F. KITCHELL. 1993. Trophic cascades in lakes. Cambridge Univ. Press, Cambridge.

CHARLESWORTH, B. 1980. Evolution in age-structured populations. Cambridge Univ. Press, Cambridge.

CROWL, T. A. 1990. Life history strategies of a freshwater snail in response to streampermanence and predation: Balancing conflicting demands. Oecologia 84:238–243

CROWL, T. A., AND A. P. COVICH. 1990. Predator-induced life-history shifts in a freshwater snail. Science (USA) 247:949–951.

DODSON, S. I., AND J. E. HAVEL. 1988. Indirect prey effects: Some morphological and life-history responses of *Daphnia pulex* exposed to *Notonecta undulata*. Limnol. Oceanogr. 33:1274–1285.

ENDLER, J. A. 1986. Natural selection in the wild. Princeton Univ. Press, Princeton, NJ.

GADGIL, M., AND W. H. BOSSERT. 1970. Life historical consequences of natural selection. Am. Nat. 104:1–24.

GEBHARDT, M. D., AND S. C. STEARNS. 1988. Reaction norms for developmental time and weight at eclosion in *Drosophila mercatorum*. J. Evol. Biol. 1:335–354.

GILINSKY, E. 1980. The role of fish predation and spatial heterogeneity in determining benthic community structure. Ecology 65:455–468.

GILLIAM, J. F. 1982. Habitat use and competitive bottlenecks in size-structured fish populations. Ph.D. diss. Michigan State Univ., East Lansing.

GILLIAM, J. F., AND D. F. FRASER. 1987. Habitat selection under predation hazard: Tests of a model with foraging minnows. Ecology 68:1856–1862.

GOTTHARD, K., S. NYLIN, AND C. WIKLUND. 1994. Adaptive variation in growth rate: Life hsitory costs and consequences in the speckled wood butterfly, *Pararge aegeria*. Oecologia (Berlin) 99:281–289.

HOUSTON, A. I., AND J. M. MCNAMARA. 1992. Phenotypic plasticity as a state-dependent life-history decision. Evol. Ecol. 6:243–253.

HOUSTON, A. I., J. M. MCNAMARA, AND J. M. C. HUTCHINSON. 1993. General results concerning the trade-off between gaining energy and avoiding predation. Phil. Trans. Roy. Soc. Lond. B. 341: 375–397.

KAWECKI, T. J. 1993. Age and size at maturity in a patchy environment: Fitness maximization vs. evolutionary stability. Oikos 66:309–317.

KAWECKI, T. J., AND S. C. STEARNS. 1993. The evolution of life histories in spatially heterogeneous environments: Optimal reaction norms revisited. Evol. Ecol. 7:155–174.

KERFOOT, W. C., AND A. SIH, EDS. 1987. Predation: Direct and indirect impacts on aquatic communities. Univ. Press of New England, Hanover, NH.

KOZŁOWSKI, J. 1992. Optimal allocation of resources to growth and reproduction: Implications for age and size at maturity. Trends Ecol. Evol. 7:15–19.

KOZŁOWSKI, J., AND J. UCHMANSKI. 1987. Optimal individual growth and reproduction in perennial species with indeterminate growth. Evol. Ecol. 1:214–230.

KOZŁOWSKI, J., AND R. G. WIEGERT. 1987. Optimal age and size at maturity in annuals and perennials with determinate growth. Evol. Ecol. 1:231–244.

KUSANO, T. 1982. Postmetamorphic growth, survival, and age at first reproduction of the salamander, *Hyobius nebulosus tokyoensis* Tago in relation to a consideration on the optimal timing of first reproduction. Res. Popul. Ecol. 24:329–344.

LAW, R. 1979. Optimal life histories under age-specific predation. Am. Nat. 114:399–417.

LEIBOLD, M., AND A. J. TESSIER. 1991. Contrasting patterns of body size for *Daphnia* species that segregate by habitat. Oecologia 86:342–348.

LIMA, S. L. 1992. Life in a multi-predator environment: Some considerations for anti-predator vigilance. Ann. Zool. Fennici 29:217–226.

LIMA, S. L., AND L. M. DILL. 1990. Behavioural decisions made under the risk of predation: A review and prospectus. Can. J. Zool. 68:619–640.

LUDWIG, D., AND L. ROWE. 1990. Life history strategies for energy gain and predator avoidance under time constraints. Am. Nat. 135:686–707.

MATTINGLY, H., AND M. V. BUTLER. 1994. Laboratory predation on the Trinidadian guppy: Implications for the size-selective predation hypothesis and guppy life history evolution. Oikos 69: 54–64.

MICHOD, R. E. 1979. Evolution of life histories in response to age-specific mortality factors. Am. Nat. 113:531–550.

NYLIN, S., C. WIKLUND, P-O. WICKMAN, AND E. GARCIA-BARROS. 1993. Absence of trade-offsbetween sexual size dimorphism and early male emergence in a butterfly. Ecology 74:1414–1427.

PERRIN, N., AND J. F. RUBIN. 1990. On dome shaped norms of reaction for size-to-age at maturity in fishes. Funct. Ecol. 4:53–57.

REZNICK, D. N. 1989. Life history evolution in guppies (*Poecilia reticulata*). 2. Repeatability of field observations and the effects of season on life histories. Evolution 43:1285–1297.

REZNICK, D. N., AND J. A. ENDLER. 1982. The impact of predation on life history evolution inTrinidadian guppies (*Poecilia reticulata*). Evolution 36:160–177.

REZNICK, D. N., H. BRYGA, AND J. A. ENDLER. 1990. Experimentally induced life-history evolution in a natural population. Nature 346:357–359.

REZNICK, D. N., M. J. BUTLER IV, AND H. F. RODD. 1996. Life-history evolution in guppies (*Poecilia reticulata*). VI. Differential mortality as a mechanism for natural selection. Evolution *In press.*

ROFF, D. A. 1981. On being the right size. Am. Nat. 118:405–422.

———. 1992. The evolution of life histories: Theories and analysis. Chapman and Hall, New York.

ROWE, L., AND D. LUDWIG. 1991. Size and timing of metamorphosis in complex life cycles: Time constraints and variation. Ecology 72:413–427.

SEGHERS, B. H. 1973. An analysis of geographic variation in the anti-predator adaptations of the guppy, *Poecilia reticulata*. Ph.D. diss., Univ. of British Columbia, Vancouver, BC, Canada.

SEMLITSCH, R. D., D. E. SCOTT, AND J. H. K. PECHMANN. 1988. Time and size at metamorphosis related to adult fitness in *Ambystoma talpoideum*. Ecology 69:184–192.

SIH, A. 1987. Predators and prey lifestyles: An evolutionary and ecological overview. Pp. 203–224 *in* W. C. Kerfoot and A. Sih, eds. Predation: Direct and indirect impacts on aquatic communities. Univ. Press of New England, Hanover, NH.

SKELLY, D. K., AND E. E. WERNER. 1990. Behavioral and life historical responses of larval American toads to an odonate predator. Ecology 71:2313–2322.

SPITZE, K. 1991. *Chaoborus* predation and life-history evolution in *Daphnia pulex*: Temporal pattern of population diversity, fitness, and mean life history. Evolution 45:82–92

STEARNS, S. C. 1992. The evolution of life histories. Oxford Univ. Press, New York.

STEARNS, S. C., AND J. C. KOELLA. 1986. The evolution of phenotypic plasticity in life-history traits: Predictions of reaction norms for age and size of maturity. Evolution 40:893–914.

TAYLOR, H. M., R. S. GOURLEY, C. E. LAWRENCE, AND R. S. KAPLAN. 1974. Natural selection of life history attributes: an Analytical approach. Theor. Popul. Biol. 5:104–122.

WERNER, E. E. 1986. Amphibian metamorphosis: Growth rates, predation risk, and the optimal size at transformation. Am. Nat. 128:319–341.

———. 1991. Nonlethal effects of a predator on competitive interactions between two anuran larvae. Ecology 72:1709–1720.
WERNER, E. E., AND B. R. ANHOLT. 1993. Ecological consequences of the trade-off between growth and mortality rates mediated by foraging activity. Am. Nat. 142:242–272.
WILBUR, H. M., AND J. E. FAUTH. 1990. Experimental aquatic food webs: Interactions between two predators and two prey. Am. Nat. 135:176–204.

Corresponding Editor: R. Holt

APPENDIX 1

Life History Responses to Predation with a Fixed Age at Maturity

When development time is fixed, sufficient conditions for C to maximize fitness are:

$$\partial(\ln(W))/\partial C = \partial g^*/\partial C - u' - m'P = 0 \qquad \text{(A1-1a)}$$

$$\partial^2(\ln(W))/\partial C^2 = \partial^2 g^*/\partial C^2 - u'' - m''P < 0 \qquad \text{(A1-1b)}$$

where primes denote derivatives. Implicit differentiation of the necessary condition (A1-1a) yields the expression for the effect of predator density P on the optimal growth effort, C_o:

$$\partial C_o/\partial P = \{-m' + (\partial R/\partial P)(\partial^2 g^*/\partial C \partial R)\} \div \{-\partial^2 g^*/\partial C^2 + u'' + m''P\}, \qquad \text{(A1-2)}$$

where all derivatives are evaluated at the optimal C specified by Eq. (A1-1a), and R is food density. The total effect of P on the optimal size is indicated by the sign of its effect on g* evaluated at the optimal C;

$$\partial g^*/\partial P = (\partial g^*/\partial C)(\partial C_o/\partial P) + (\partial g^*/\partial R)(\partial R/\partial P), \qquad \text{(A1-3)}$$

where all derivatives are evaluated at C_o and the population dynamical equilibrium.

If $\partial R/\partial P = 0$, Eq. (A1-3) shows that the effect of the predator on prey size is given by the sign of Eq. (A1-2). The numerator of Eq. (A1-2) reduces to $-m'$, which must be nonpositive; the denominator of Eq. (A1-2) is always positive because of condition (A1-1b). The only case when $m' = 0$ is when traits conferring more rapid growth do not increase exposure to predation. Thus, increased predator density should usually decrease (and never increase) growth rate and size.

If there is an indirect effect via food, the effects of food on effort and the total effect of predators on the growth rate of prey can both be derived from Eqs. (A1-2,3), and are given by:

$$\partial C_o/\partial R = \partial^2 g^*/\partial C \partial R/\{(-\partial^2 g^*/\partial C^2 + u'' + m''P\}, \qquad \text{(A1-4a)}$$

$$\partial g^*/\partial P = \{-m'(\partial g^*/\partial C)\}/\{-\partial^2 g^*/\partial C^2 + u'' + m''P)\} + (\partial R/\partial P)\{(\partial^2 g^*/\partial C \partial R)(\partial g^*/\partial C) + (\partial g^*/\partial R)(-\partial^2 g^*/\partial C^2 + u'' + m''P)\} \div \{(-\partial^2 g^*/\partial C^2 + u'' + m''P)\} \qquad \text{(A1-4b)}$$

C_o increases (decreases) with food density if $\partial^2 g^*/\partial C \partial R$ is positive (negative); an increase is therefore more likely for reasons given above. The total effect of predators on growth rate is the sum of two ratios, the second of which is the indirect effect. All of the quantities within parentheses in the indirect term of (A1-4b) must be positive, except for $\partial^2 g^*/\partial C \partial R$, which may be either positive or negative (based on the signs of the corresponding derivatives of g and ϕ in Table 1). Thus, the indirect effect of predation via food supplies will frequently be an increase in growth rate and size.

The assumption that C is the proportion of time spent foraging means that g*(C,R) in equation (2) can be represented as a function of a single variable, g*(CR), where CR is mean food intake rate over the juvenile period. This means that the various partial derivatives of g* appearing in Eq. (A1-4b) are:

$$\partial g^*/\partial C = Rg^{*\prime};\ \partial g^*/\partial R = Cg^{*\prime};\ \partial^2 g^*/\partial C \partial R = g^{*\prime} + CRg^{*\prime\prime};\ \partial^2 g^*/\partial C^2 = R^2 g^{*\prime\prime} \qquad \text{(A1-5)}$$

In this example, we assume that all density dependence in per capita growth rate operates through effects on the juvenile food supply. This means that increases in the food supply R must increase fitness enough to exactly offset the decreased fitness caused directly by predation:

$$(\partial/\partial P)[g^*(CR) - u(C) - m(C)P] = -m + (\partial R/\partial P)Cg^{*\prime} = 0, \qquad \text{(A1-6)}$$

This implies that $\partial R/\partial P = m/(Cg^{*\prime})$. When both this and the formulas in (A1-5) are substituted into Eq. (A1-3), the result is:

$$\partial g^*/\partial P = g^{*\prime}[R\partial C_o/\partial P + (\partial R/\partial P)C_o] = \{(m - C_o m')(u' + C_o m') + mC_o(u'' + m''P)\} \div \{C_o(-R^2 g^{*\prime\prime} + u'' + m''P)\} \qquad \text{(A1-7)}$$

The denominator of (A1-7) must be positive by Eq. (A1-1b). The numerator may be positive or negative. However, it must be positive (given the assumptions that $u'' > 0$ and $m'' > 0$) whenever $m > C_o m'$. There are many biologically reasonable relationships between foraging time and predation risk that will satisfy this inequality for some or all parameter values. In particular, if the exposure of nonforagers is sufficiently high, m must be greater than Cm'.

Equation (A1-6) assumes that the density dependence in the juvenile stage operates solely through effects on the food supply, R. The decreased juvenile density due to predation may affect other mortality factors. In this case, R would not increase as much as predicted by Eq. (A1-6), and the net effect on size would more likely be negative. The derivation leading to Eq. (A1-7) also assumed that there was no density dependence in the adult stage, and that the juvenile-stage predator did not consume adults. If there is density dependence in the adult stage, then increasing predator density, by reducing recruitment to the adult stage, increases the adult component of fitness. If the population is stable (as assumed) this means that the juvenile component of fitness must decrease. Thus, food (R) does not increase as much as predicted by Eq. (A1-6). Evolution of increased size in response to predation is still possible, but less likely than in a comparable system with no adult density dependence. If the juvenile-stage predators also consume adults (but there is no adult density dependence) then increased predation has the direct effect of lowering both the juvenile and the adult components of fitness. Thus, R must increase more than specified by Eq. (A1-6) if the population is to remain constant. This makes it more likely that the net effect on size is positive.

APPENDIX 2

Optimal Life Histories under All Condition Other than a Fixed Time to Maturity

Fixed Size at Maturity

The sufficient conditions for an optimal life history are:

$$-g(Pm' + u') + \partial g/\partial C\{mP + u + f'\} = 0 \qquad \text{(A2-1a)}$$

$$-g(Pm'' + u'') + \partial^2 g/\partial C^2(mP + u + f') - (f''/g^2)(\partial g/\partial C)^2 < 0 \qquad \text{(A2-1b)}$$

Implicit differentiation again yields an expression for the total effect of P on C_o:

$$dC_o/dP = \{(\partial g/\partial C)m - gm' + (\partial R/\partial P)[(u + mP + f')(\partial^2 g/\partial C \partial R) - (\partial g/\partial R)(Pm' + u' + (f''/g^2)(\partial g/\partial C))]\} \div \{g(Pm'' + u'') - \partial^2 g/\partial C^2(mP + u + f') + (f''/g^2)(\partial g/\partial C)^2\} \qquad \text{(A2-2)}$$

where again, the right hand side is evaluated at C_o.

When there are no indirect effects ($\partial R/\partial P = 0$), expression (A2-2) must have the same sign as $(\partial g/\partial C)m - gm'$. Equation (A2-1a) implies that this is identical to the quantity, $[gu' - u(\partial g/\partial C) - f'(\partial g/\partial C)]/P$. The derivative of the seasonality function, f', is likely to be positive, because fitness decreases with age at maturity in most seasonal environments (see Rowe and Ludwig 1991). Thus, when growth effort, C, only affects mortality due to the focal predator(s) (i.e., $u' = 0$), both growth effort and growth rate will usually decrease, and age at maturity will increase in response to greater predation. Predation in this case favors less risky and less rewarding growth strategies. If effort only affects nonpredatory components of mortality ($m' = 0$) then Eq. (A2-2) must be positive in the absence of indirect effects. In other words, growth rate and effort increase, while age at maturity decreases in response to greater predation. When effort affects both u and mP, the magnitudes and shapes of these functions determine the responses to increased P.

The response of optimal effort to food, and the summed direct and indirect responses of growth rate to predator density are:

$$\partial C_o/\partial R = \{(u + mP + f')(\partial^2 g/\partial C\partial R) - (\partial g/\partial R)(Pm' + u' + (f''/g^2)(\partial g/\partial C))\}/(-Z), \quad \text{(A2-3a)}$$

$$dg/dP = (\partial g/\partial C)(\partial C_o/\partial P) + (\partial g/\partial R)(\partial R/\partial P) = \{(\partial g/\partial C)^2 m - (\partial g/\partial C)gm'\}/(-Z) + (\partial R/\partial P)\{[(u + mP + f')((\partial g/\partial C)(\partial^2 g/\partial C\partial R) - (\partial^2 g/\partial C^2)(\partial g/\partial R) - (1/g)(\partial g/\partial R)(\partial g/\partial C)^2) + g(\partial g/\partial R)(Pm'' + u'')]\}/(-Z), \quad \text{(A2-3b)}$$

where Z denotes the left hand side of (A2-1b). Growth effort, C_o may either increase or decrease in response to food density (i.e., [A2-3a] may be either positive or negative). Thus, the indirect response of effort to predators may either reinforce or counteract the direct effect. The indirect component of the predator's total effect on growth is given by those terms in Eq. (A2-3b) that are multiplied by $\partial R/\partial P$; $-(1/g)(\partial g/\partial R)(\partial g/\partial C)^2$ must be negative, but the other terms must be or are likely to be positive. This suggests that the indirect effect from increased food usually increases growth rate even if it does not increase growth effort. This opposes the direct effect of predators whenever the direct effect is reduced effort (which we argued seems more likely than increased effort). The assumption of constant size means that the indirect effect on age at maturity opposes the effect on growth rate; i.e., age of maturity usually decreases in response to greater food supplies. Here again, it is possible to use specific models to show that the indirect effect may be larger than the direct effect. If, for example, growth effort, C, represents time spent foraging, the indirect effect of predators on prey size is often larger than the direct effect; the proof of this assertion is analogous to that presented in Appendix 1. In the less common case where the predatory costs of increasing effort are small relative to nonpredatory costs, then indirect and direct effects usually have the same sign, and growth rate will increase while age at maturity decreases with increased predation.

Flexible Age and Size

The conditions for the two variables, C and T, to maximize this fitness expression become:

$$\partial(\ln(W))/\partial C = (\phi'\partial g/\partial C - u' - m'P)T = 0 \quad \text{(A2-4a)}$$

$$\partial(\ln(W))/\partial T = g\phi' - f' - u - mP = 0 \quad \text{(A2-4b)}$$

$$\partial^2(\ln(W))/\partial C^2 = T[\phi'\partial^2 g/\partial C^2 + T\phi''(\partial g/\partial C)^2 - u'' - m''P] < 0 \quad \text{(A2-4c)}$$

$$\partial^2(\ln(W))/\partial T^2 = g^2\phi'' - f'' < 0 \quad \text{(A2-4d)}$$

$$(\partial^2(\ln(W))/\partial C\partial T)^2 - (\partial^2(\ln(W))/\partial C^2)(\partial^2(\ln(W))/\partial T^2) = (gT\phi''\partial g/\partial C)^2 - T(\phi'\partial^2 g/\partial C^2 + T\phi''(\partial g/\partial C)^2 - u'' - m''P)(g^2\phi'' - f'') < 0 \quad \text{(A2-4e)}$$

The effect of a parameter such as predator density on the optimal C and T may be found by implicit differentiation of eqs. (A2-4a,b). The resulting general expression for the effects of P on C_o, T_o, and $\ln(S_o)$ are quite lengthy, so the formulas presented below are for the case when there are no indirect effects on food ($\partial R/\partial P) = 0$):

$$\partial C_o/\partial P = (-T_o/Z)[g\phi''(m'g - m\partial g/\partial C) - m'f''] \quad \text{(A2-5a)}$$

$$\partial T_o/\partial P = (-T_o/Z)[T_o\phi''(\partial g/\partial C)(-gm' + m\partial g/\partial C) + m\phi'\partial^2 g/\partial C^2 - m(u'' + m''P)] \quad \text{(A2-5b)}$$

$$\partial\ln(S_o)\partial P = (-T_o/Z)[gm\phi'\partial^2 g/\partial C^2 - gm(u'' + m''P) - T_o m'f''\partial g/\partial C] \quad \text{(A2-5c)}$$

where Z is the left hand side of inequality (A2-4e), which is negative, and all expressions are evaluated at the optimal values of C and T.

None of the three derivatives above has a definite sign. However, it is very likely that, in the absence of indirect effects, size will decrease in response to greater predator densities; size must decrease unless $\partial^2 g/\partial C^2$ is positive and large in magnitude. This is unlikely, both because growth rate is likely to approach some maximum as effort becomes large, and because condition (A2-4c) sets un upper limit on the value of $\partial^2 g/\partial C^2$ that is consistent with intermediate optimal values of C and T. Thus, we predict that size will generally decrease in response to increased predation. However, either effort or time to maturity may increase; Eqs. (A2-5a,b) may be positive or negative. If nonpredatory costs (u') of growth effort are small, the direct response of C_o is to decrease, but T_o may increase or decrease. If predatory costs (m') are small, T_o must decrease, but C_o may increase or decrease. Some more definite conclusions can be drawn if assumptions are made about the shape of some of the fitness components. If, for example, reproduction is a power function of size ($\phi'' = 0$), which includes the case of reproduction directly proportional to size), both C_o and T_o always decrease in response to predation (see also Abrams et al. 1996).

The indirect effects of predators via increased food may be found by implicit differentiation of Eqs. (A2-4a,b) with respect to R. The resulting formulas are presented below:

$$\partial C_o/\partial R = (-T_o/Z)[(\partial g/\partial C)(\partial g/\partial R)(\phi'')(\phi' g + T_o f'') - \phi'(\partial^2 g/\partial C\partial R)(g^2\phi'' - f'')] \quad \text{(A2-6a)}$$

$$\partial T_o/\partial R = (-T_o/Z)[T_o g\phi'\phi''(\partial g/\partial C)(\partial^2 g/\partial C\partial R) - (\partial g/\partial R)(\phi'\phi'' T(\partial g/\partial C)^2 + (\phi' + gT\phi'')(\phi'\partial^2 g/\partial C^2 - u'' - m''P)] \quad \text{(A2-6b)}$$

$$\partial\ln(S_o)\partial R = (-T_o/Z)\{-g(\partial g/\partial R)(\phi')(\phi'\partial^2 g/\partial C^2 - u'' - m''P) + Tf''[\phi'(\partial g/\partial C)(\partial^2 g/\partial C\partial R) - (\partial g/\partial R)(\phi'\partial^2 g/C^2 - u'' - m''P)]\} \quad \text{(A2-6c)}$$

where Z is the left hand side of inequality (A2-4e). The responses of C_o and T_o can each be positive or negative depending on the forms of the functions. Both of these expressions simplify considerably in the case when reproduction is a power function of size (and $\phi'' = 0$). In this case, age at maturity always increases, and growth effort increases whenever $\partial^2 g/\partial C\partial R$ is positive, which is likely to be satisfied in most cases. The only term in expression (A2-6c) for $\partial\ln(S_o)/\partial R$ that can ever be negative is the term involving $\partial^2 g/\partial C\partial R$, which is more likely to be positive than negative. Thus, size almost always increases in response to greater food. The response of growth rate to food is given by $dg/dR = \partial g/\partial R + (\partial g/\partial C)(\partial C_o/\partial R)$. This formula may be expanded using (A2-6a). The resulting expression is rather long; it has more positive than negative terms, and is thus more likely to be positive than negative. Growth rate can only decrease in response to increased food if C_o decreases with R, and if the decrease in C_o is large enough to offset the increase in R.