Interactions between Behavioral and Life-History Trade-Offs in the Evolution of Integrated Predator-Defense Plasticity

Clayton E. Cressler,[1,*] Aaron A. King,[1,2] and Earl E. Werner[1]

1. Department of Ecology and Evolutionary Biology, University of Michigan, Ann Arbor, Michigan 48109; 2. Department of Applied Mathematics, University of Michigan, Ann Arbor, Michigan 48109

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abstract: Inducible defense, which is phenotypic plasticity in traits that affect predation risk, is taxonomically widespread and has been shown to have important ecological consequences. However, it remains unclear what factors promote the evolution of qualitatively different defense strategies and when evolution should favor strategies that involve modification of multiple traits. Previous theory suggests that individual-level trade-offs play a key role in defense evolution, but most of this work has assumed that trade-offs are independent. Here we show that the shape of the behavioral trade-off between foraging gain and predation risk determines the interaction between this trade-off and the life-history trade-off between growth and reproduction. The interaction between these fundamental trade-offs determines the optimal investment into behavioral and life-history defenses. Highly nonlinear foraging–predation risk trade-offs favor the evolution of behavioral defenses, while linear trade-offs favor life-history defenses. Between these extremes, integrated defense responses are optimal, with defense expression strongly depending on ontogeny. We suggest that these predictions may be general across qualitatively different defenses. Our results have important implications for theory on the ecological effects of inducible defense, which has not considered how qualitatively different defenses might alter ecological interactions.

Keywords: phenotypic plasticity, inducible defense, foraging–predation risk trade-off, predator-prey, evolutionary computation.

Introduction

The risk of predation is a powerful force in the evolution of species traits, and many behavioral, morphological, and life-historical traits confer defense against predators. However, many of these traits are not fixed but are phenotypically plastic, varying on the basis of environmental context (Tollrian and Harvell 1999). A large body of empirical work documents both patterns of defense expression and their efficacy in reducing the risk of predation. Recent research has also explored the consequences of these defenses to interactions with other species in the food web (Werner and Peacor 2003; Relyea 2004*a*). Moreover, empirical studies have documented that closely related species often employ qualitatively different defense strategies (De Meester et al. 1995; Rundle and Brönmark 2001; Mikolajewski and Johansson 2004) or that species are capable of expressing multiple defenses, either simultaneously or across ontogeny (Relyea 2004*a*; Hoverman et al. 2005; Boeing et al. 2006; Steiner and Pfeiffer 2007). Different clones of *Daphnia*, for example, are capable of expressing up to eight different inducible defenses simultaneously, including modifications in life history, behavior, and morphology (Boeing et al. 2006).

Given that different defenses employed alone, simultaneously, or serially during ontogeny are evolutionary solutions to the risk of predation, the question arises as to what factors favor the evolution of one defensive strategy over another or how they are jointly employed. Understanding how these traits are integrated is central to understanding the evolution of the phenotype, as well as how these traits influence population dynamics, interactions with other species, and patterns of species coexistence (Miner et al. 2005).

Theory elucidating the ecology and evolution of inducible defenses has largely focused on single traits (reviewed in Bolker et al. 2003). Comparatively little theory has been developed to explain the evolution of qualitatively different defense strategies (e.g., behavioral vs. morphological defenses) or the manner in which multiple defense strategies are integrated in the phenotype. At the core of this problem is the issue of how trade-offs associated with expression of defenses interact and thereby influence the evolution and expression of these defenses.

For example, modification of behavior or life history involves fundamental trade-offs for the organism. For behavioral defense, the trade-off is often between foraging gain and predation risk, as a ubiquitous behavioral response to predation is a reduction of foraging activity or

* Corresponding author; e-mail: cressler@umich.edu.

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the movement to a suboptimal foraging habitat (Lima and Dill 1990). For life-history defenses, there is often a trade-off between the allocation of energy to growth or reproduction that leads to a trade-off between age versus size at maturity (Roff 2001). Moreover, these trade-offs are not independent of one another: any modification of behavior may affect the pattern of energy allocation; for example, if an individual reduces its activity level in response to predation risk, growing to the same size at maturity will require either a prolonged growth phase or an increase in the amount of energy allocated per unit time. There has been some empirical work that has recognized that trade-offs may be interacting, although the end goal of these projects was to attribute patterns of behavior and life history to only one trade-off rather than any interaction between trade-offs (Ball and Baker 1996; Beckerman et al. 2007). Recent theory has also begun to explore the implications of interacting trade-offs (Steiner and Pfeiffer 2007). This work is central to developing a more complete understanding of life-history evolution, since trade-offs form the foundation of life-history theory. However, most models treat defense investment as a constant parameter (Abrams and Rowe 1996; Day et al. 2002; Steiner and Pfeiffer 2007) rather than a function of individual physiology. This omission effectively ignores the reality that defense investment incurs trade-offs arising at the level of individual physiology and that the relative costs and benefits of defense investment will change as individuals age and grow (Clark and Harvell 1992).

Here we explore how variation in the shape of the ecological trade-off between foraging gain and predation risk affects the interaction between this trade-off and the physiological trade-off between growth and reproduction, which in turn determines the optimal expression of behavioral and life-history (body size) defenses under negative size-dependent predation. We do this both to elucidate the nature of multiple defense expression and to make explicit predictions for this common scenario. Our choice of these defensive traits is motivated by the generality of their effects across organisms. First, body size and behavior are traits that are critical to species fitness; many ecological interactions are size dependent, and body size influences nearly all physiological processes, including resource ingestion, growth, reproduction, and mortality (Werner and Gilliam 1984; Kooijman 2000; de Roos et al. 2003). Thus, any modification of body size in response to predation risk will have important ecological consequences. Behavior is similarly fundamental; because of its role in resource acquisition, behavior affects many of the same physiological processes as body size and has been shown to strongly impact ecological processes (Bolker et al. 2003; Werner and Peacor 2003). Furthermore, the relationship between body size and behavior is complicated by the joint dependence of these traits on ontogeny. Changes in body size through ontogeny can affect predation risk and, therefore, the efficacy of different behaviors, and behavior can influence body size through its effect on foraging gain and growth rates. Finally, the shape of this trade-off is predicted to have strong impacts on the ecological consequences of behavior (Abrams 1992; Bolker et al. 2003), but there has been no systematic exploration of its consequences for defense expression.

Using an individual-based physiological model, we investigate how the optimal investment into life-history and behavioral defenses varies under an activity-mediated trade-off between foraging gain and predation risk. This extends previous theory (Abrams and Rowe 1996; Day et al. 2002; Steiner and Pfeiffer 2007; Urban 2007*a*) in a number of ways. Most importantly, by treating behavior and energy allocation separately and allowing both to vary with age, we are able to achieve a more complete understanding of how organisms balance competing trade-offs and how this interaction can give rise to complex, multivariate responses. We discuss the implications of this result for the evolution of trait integration and for the study of the ecological consequences of inducible defense.

Methods

Model Description

To investigate the interaction between trade-offs, we employ an individual-based physiological model where growth, reproduction, and death depend on the current state of the individual. The model is based on the physiologically structured model developed by Kooijman and Metz (1984). The structure and parameterization of the original model were modified to allow for flexibility in life history and behavior. These changes are discussed in more detail in appendix A in the online edition of the *American Naturalist*, which also contains the derivation of the growth equation. A basic description of the equations and key parameters is presented below. Parameter values have been taken from de Roos et al. (1990), except as noted in appendix A. The energetic assumptions underlying these equations are very general (Kooijman 2000), so this model represents a general conceptual model for investigating how organisms balance competing life-history trade-offs. Table 1 presents the variables and parameters used in the model and provides default parameter values.

Characterizing Investment in Defense

Behavioral defenses are often characterized by changes in activity level or habitat that reduce the encounter rate with predators (Lima and Dill 1990; Tollrian and Harvell 1999).

Table 1: Model variables and parameters with default parameter values

Parameter	Description	Units	Default value
t	Age	days	
$\ell(t)$	Length	mm	
$\alpha(t)$	Fraction of time spent foraging		
$\kappa(t)$	Fraction of energy allocated to growth		
$b(t)$	Birthrate	eggs day^{-1}	
$p(t)$	Survivorship of an individual		
R	Resource density	cells mL^{-1}	10^6
v_x	Maximum resource intake rate per unit surface area	cells mm^{-2} day^{-1}	1.8×10^6
ξ	Functional response shape parameter	mL cell^{-1}	7.0×10^{-6}
$\hat{\ell}$	Maximum attainable length under unlimited resources	mm	6.0
$\hat{g}$	Rate constant of growth	day^{-1}	.5
$\hat{r}$	Rate of offspring production per unit surface area	mm^{-2} day^{-1}	.14
μ	Background mortality rate	day^{-1}	.01
ω	Slope of mortality-size curve	mm^{-1}	.5
ℓ_{mid}	Median size preference of predator	mm	3.5
P_{h}	Half of maximum predation rate	day^{-1}	0–.2
s	Shape parameter for activity-mortality relation		1–10

In our model, behavioral defense investment will be determined by $\alpha(t)$, the fraction of total available foraging time that is spent in active foraging as opposed to engaging in defensive behaviors. Life-history defenses are typically thought to result from changes in energy investment between growth and reproduction, often in response to size-dependent predation (Taylor and Gabriel 1992; Ernande et al. 2004; Gårdmark and Dieckmann 2006). We define $\kappa(t)$ as the fraction of net production allocated to growth versus reproduction.

The trade-offs involved in defense expression are mechanistically built into the model by considering how $\alpha(t)$ and $\kappa(t)$ affect physiological processes. Increasing activity level increases foraging gain but also predation risk, so $\alpha(t)$ will directly affect both energy acquisition and mortality. Prolonged allocation to growth will reduce predation risk from negative size-selective predators by increasing size, but it will also delay reproductive maturity, so $\kappa(t)$ will directly affect both growth and reproduction and indirectly affect mortality. The time dependence of $\alpha(t)$ and $\kappa(t)$ reflects the fact that investment may change as a function of an individual's age, size, and physiological state (Clark and Harvell 1992).

Foraging Gain and Maintenance Costs

Acquisition of energy from the environment is the key process underlying growth and reproduction. The rate of energy ingestion is modeled as

$$I(R, \ell, \alpha, t) = v_x \alpha(t) \frac{\xi R}{1 + \xi R} \ell^2, \tag{1}$$

where v_x is the maximum rate of resource consumption, R is the constant resource abundance, and $\xi R/(1 + \xi R)$ is a Type II functional response. Two features of this formulation are noteworthy. First, ingestion rate depends linearly on activity level, $\alpha(t)$, so total resource ingestion is proportional to the fraction of time actively foraging. Second, energy ingestion depends on the surface area of the individual, which is proportional to ℓ^2. The dependence of feeding rate on surface area is quite general, applying to many different feeding modes (see the discussion in Kooijman 2000, pp. 66–71). This dependence implies that energy ingestion will increase as individuals increase in size; this is the mechanism by which increased size increases reproductive potential. Some energy must be used for maintenance, with maintenance costs scaling with body volume (see Kooijman 2000, pp. 89–94).

Growth and Reproduction

We assume that maintenance costs are taken directly from ingested resources. Individuals then allocate surplus energy between growth and reproduction. This assumption makes our model a net production model (sensu Noonburg et al. 1998) rather than a net assimilation model, in which energy is allocated to cover both growth and maintenance (de Roos 1997). Both net assimilation and net production models can be justified on biological grounds; our choice of model results from our desire that $\kappa(t)$ be free from any constraints. By contrast, net assimilation models require a rule that specifies how energy is to be reallocated when maintenance costs are high.

The fraction of net production allocated to growth is controlled by the time-varying function $\kappa(t)$, with $1 -$

$\kappa(t)$ being allocated to maturation and reproduction. This reflects the inherent trade-off between current growth and future reproduction. When resources are constant, growth follows a modified von Bertalanffy growth equation (Kooijman and Metz 1984; de Roos 1997; Kooijman 2000), which predicts growth in size to approach $\hat{\ell}$ at a rate determined by the parameter $\hat{g}$, assuming that growth allocation $\kappa(t)$ and activity level $\alpha(t)$ are both constant:

$$\frac{d\ell}{dt} = \hat{g}\kappa(t)(\hat{\ell}f(R, \alpha) - \ell(t)), \tag{2}$$

where

$$f(R, \alpha) = \alpha(t)\frac{\xi R}{1 + \xi R}. \tag{3}$$

The remaining energy is allocated to maturation or reproduction. Before reaching sexual maturity, individuals are assumed to allocate energy to the development of reproductive tissue: sexual maturity is reached upon investment of a fixed amount of energy into maturation (see app. B in the online edition of the *American Naturalist*). The birthrate $b(t)$ is then determined by energy allotment to reproduction $1 - \kappa(t)$ and the rate of offspring production per unit surface area $\hat{r}$:

$$b(t) = \hat{r}(1 - \kappa(t))\left(f(R, \alpha)\ell(t)^2 - \frac{\ell(t)^3}{\hat{\ell}}\right). \tag{4}$$

Mortality

We consider mortality risk that depends on both behavior and size. A large body of literature has demonstrated increases in predation risk with increases in activity (Lima and Dill 1990; Werner and Anholt 1993; Werner and Peacor 2003). While this pattern is clear, we are unaware of any studies that have directly measured the relationship between foraging activity and predation risk, despite theory indicating that this shape is critical in determining the ecological consequences of behavioral defense (Abrams 1992). We follow Noonburg and Nisbet (2005) and assume that predation risk scales with $\alpha(t)^s$, a simple function that is flexible enough to take a variety of shapes from concave to convex. Furthermore, an examination of the relationship between activity level and predation rate for different values of s suggests a possible biological interpretation of s as an indicator of the foraging behavior of the predator.

Depending on the value of s, $\alpha(t)^s$ can take three basic shapes (fig. 1). If $s = 1$, then the relationship between activity and predation risk is linear. This assumption is the default expectation (Gerritsen and Strickler 1977; Werner and Anholt 1993). It will hold, for example, when predators move at constant speed and demonstrate no behavioral response to prey movement. If $s > 1$, then the curve is convex, suggesting that the per capita predation rate increases with increases in activity level. This can be interpreted as indicating a preference among predators for more active prey. Such preferential foraging has been demonstrated in a number of cases (Furnass 1979; Wright and O'Brien 1982; Peterson and Ausubel 1984; O'Keefe et al. 1998; Utne-Palm 2000). The biological mechanism behind this preference is likely predators cuing on prey movement. If $s < 1$, then the curve is concave and predation rate asymptotes at high activity levels. The effect of a concave relationship between activity level and mortality on the evolution of defenses was investigated, but the results were identical to those in the case where $s = 1$; further discussion of the $s < 1$ case is omitted.

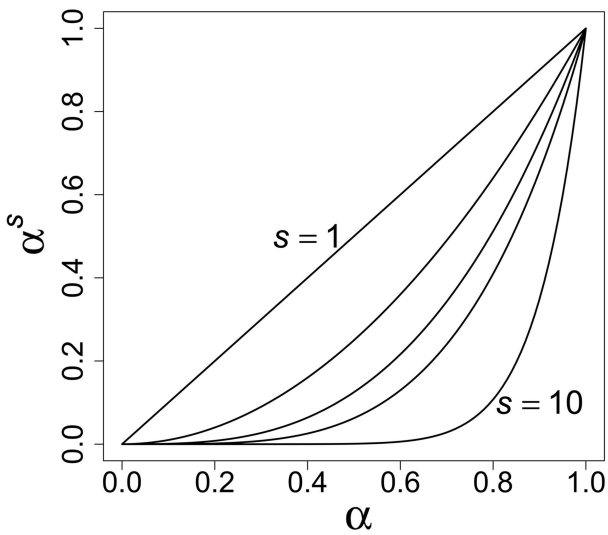

Figure 1: Relationship between activity level α and predation rate for different values of s. See the text for the interpretation of s.

This formulation makes possible two a priori predictions about the effect of s on optimal defense expression. As s approaches infinity, even a slight reduction in activity level will reduce predation rate to almost 0, whereas as s approaches 0, reduction in activity level will have no effect on predation risk. From this observation, we predict that high values of s will favor behavioral defenses, whereas low values of s will favor either life-history defenses or a strategy that forgoes any investment in defense.

Predation rate is also dependent on size. Here we assume that predation risk decreases with length, as occurs when predator and prey are similar in size or predators are gape

constrained. This creates a size refuge against predation, so that individuals exposed to predators may increase growth at the cost of delayed maturation. A reasonable functional form for the relationship between size and predation rate is

$$P_h\{1 - \tan h[\omega(\ell - \ell_{mid})]\}, \tag{5}$$

where P_h is half the maximum predation rate. The parameters ω and ℓ_{mid} characterize the size selectivity of the predator (fig. 2): ℓ_{mid} gives the inflection point where predation rate is equal to P_h and ω is the slope of the predation rate-size curve. The parameter ℓ_{mid} characterizes the size preference of the predator, while ω characterizes how size limited the predator is: larger values suggest that the predator is very limited by prey size; smaller values indicate a predator that is less size limited (Rinke et al. 2008).

Considering both the size and behavior dependence of predation rate and a nonpredation mortality term μ, the dynamics of survivorship $p(t)$ is described by

$$\frac{dp}{dt} = -(\mu + \alpha(t)^s\{1 - \tan h[\omega(\ell - \ell_{mid})]\}P_h)p. \tag{6}$$

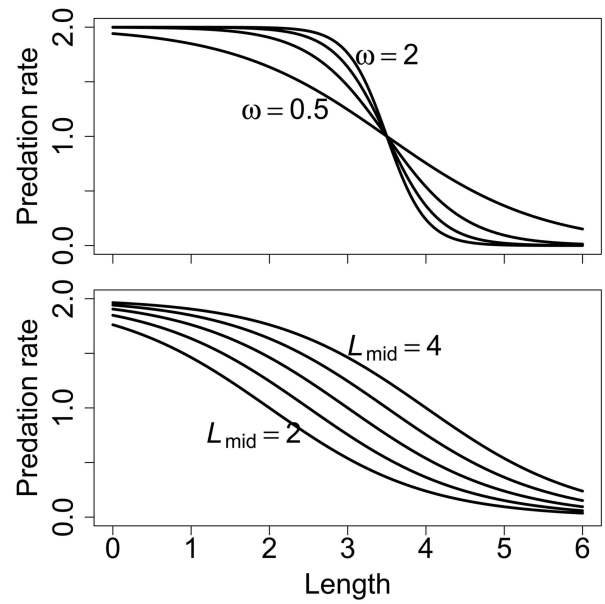

Figure 2: Illustration of how mortality changes with size, assuming different values for ω and ℓ_{mid}. For the simulations in the article, $\omega = 0.5$ and $\ell_{mid} = 3.5$.

Calculating the Fitness of an Individual

Equations (2)–(4) and (6) can be used to determine the fitness of an individual. Fitness was measured by the net reproductive rate R_0:

$$R_0 = \int_0^\infty b(t)p(t)dt. \tag{7}$$

Net reproductive rate weights birthrate $b(t)$ by survivorship $p(t)$ to determine the expected number of offspring produced by an individual over the course of its life. This fitness metric is appropriate whenever population size and the environment are constant between generations, as we assume (Benton and Grant 2000).

Selection Experiments

To find the optimal defense investment, one must determine the shapes of activity-level and growth-allocation functions that maximize individual fitness. However, the shapes of these functions are unknown and might be quite complex. We used genetic algorithms to determine the optimal shapes $\alpha(t)$ and $\kappa(t)$ (Holland 1975), an approach that has been successfully applied to other questions in evolutionary ecology (Shertzer and Ellner 2002; Strand et al. 2002). Details regarding function specification and genetic algorithm implementation can be found in appendix C in the online edition of the *American Naturalist*.

For any given environment, we characterize behavioral defense investment by the average activity level across an individual's life span, while life-history defenses are characterized by size at maturity. We report the age at maturity to complete the depiction of the life-history strategy. Size and age at maturity are determined by the interaction between $\alpha(t)$ and $\kappa(t)$. In this study, the predation rate P_h was varied between 0 and 0.2; over this range, individual fitness is always greater than the replacement level ($R_0 > 1$). Plasticity in defenses is seen by a reduction in average activity level or an increase in size at maturity with changes in P_h. The value of s was varied between 1 and 10 to determine the effect of the shape of the foraging–predation risk trade-off on the evolution of behavior and life history.

Three different selection scenarios were performed. In the first, only growth allocation was under selection; behavior was assumed to be constant at the optimal value in the absence of predators, $\alpha(t) = 1$. In the second, only behavior was under selection; growth allocation was fixed at the optimal allocation pattern in the absence of predators. In the third, both growth allocation $\kappa(t)$ and activity level $\alpha(t)$ were under selection. These experiments will be abbreviated as the L (life history only), B (behavior only), and LB (life history and behavior) selection experiments, respectively. By fixing either growth allocation or activity

level, we fix the growth-reproduction and foraging–predation risk trade-offs, respectively. Comparing the results when both growth allocation and activity level are flexible to these cases allows us to investigate how the trade-offs interact to determine optimal behavior and life history.

Results

A Short Guide to Interpreting the Results

To facilitate the presentation of the results, figure 3 shows how the shapes of the growth allocation and behavior functions independently affect maturation size and age. Figure 3*B* and 3*C* shows the maturation size and age for different $\kappa(t)$ functions (fig. 3*A*), assuming behavior is constant at $\alpha = 1$. In all of the results that follow, the optimal growth allocation function has this same basic shape. Note that age at maturity does not correspond to the age at which energy allocation switches to reproduction. This is because energy is allocated to maturation first; only after the fixed maturation requirement is met does reproduction begin. The duration of the delay between switching allocation to maturation and the onset of reproduction is determined by the size at maturity (see app. B for more details). The black line in figure 3*A* is the optimal predator-free growth allocation. Figure 3*E* and 3*F* shows the maturation size and age for different $\alpha(t)$ functions (fig. 3*D*);

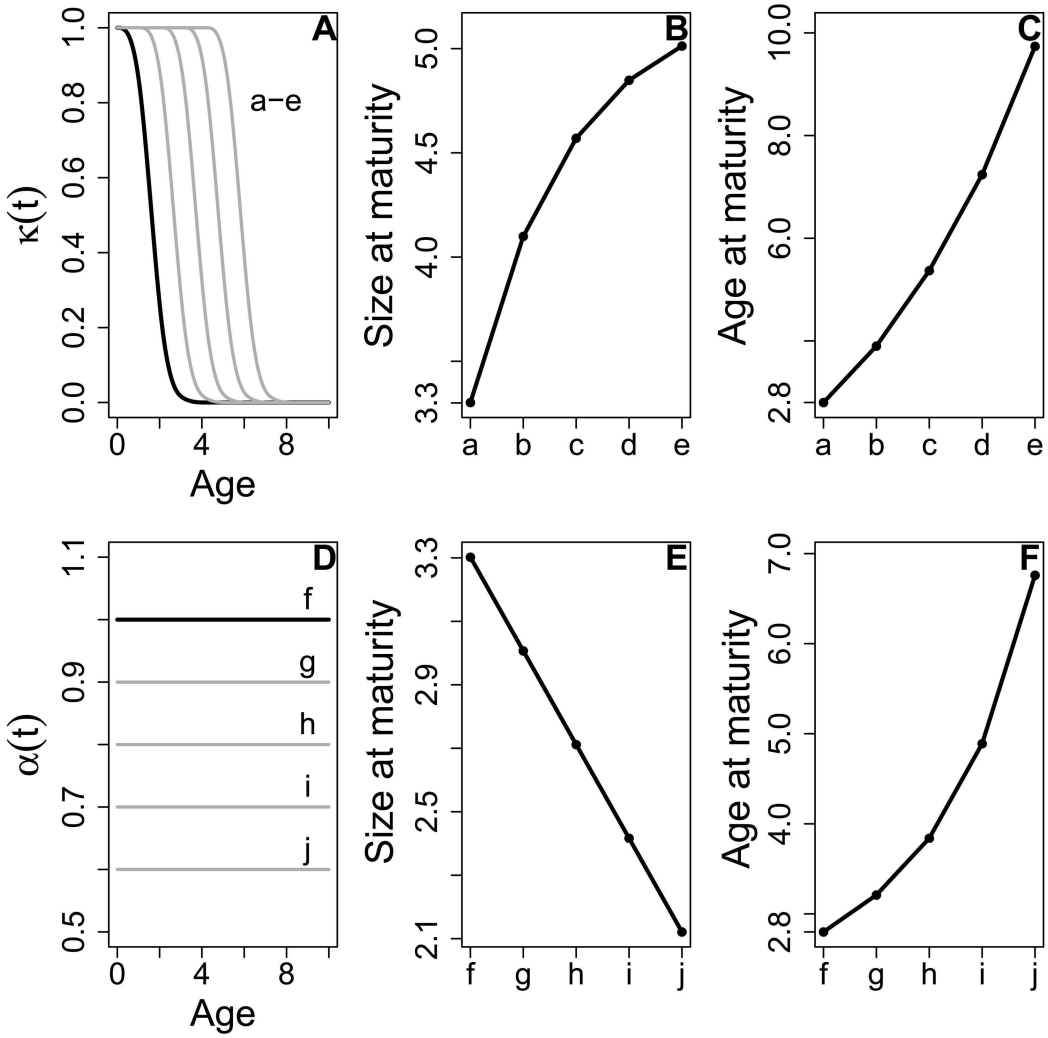

Figure 3: *A–C*, Effects of different allocation functions on size and age at maturity. *D–F*, Effects of different behavior functions. Here we assume constant behavior; observed behavior $\alpha(t)$ was more complex (see fig. 5 for an example). The black lines (*a* in *A* and *f* in *D*) show the optimal growth allocation pattern and activity level in the absence of predation. For *A–C*, activity level is held at the optimal predator-free $\alpha(t)$ value, while for *D–F*, growth allocation is determined by the optimal predator-free $\kappa(t)$ value.

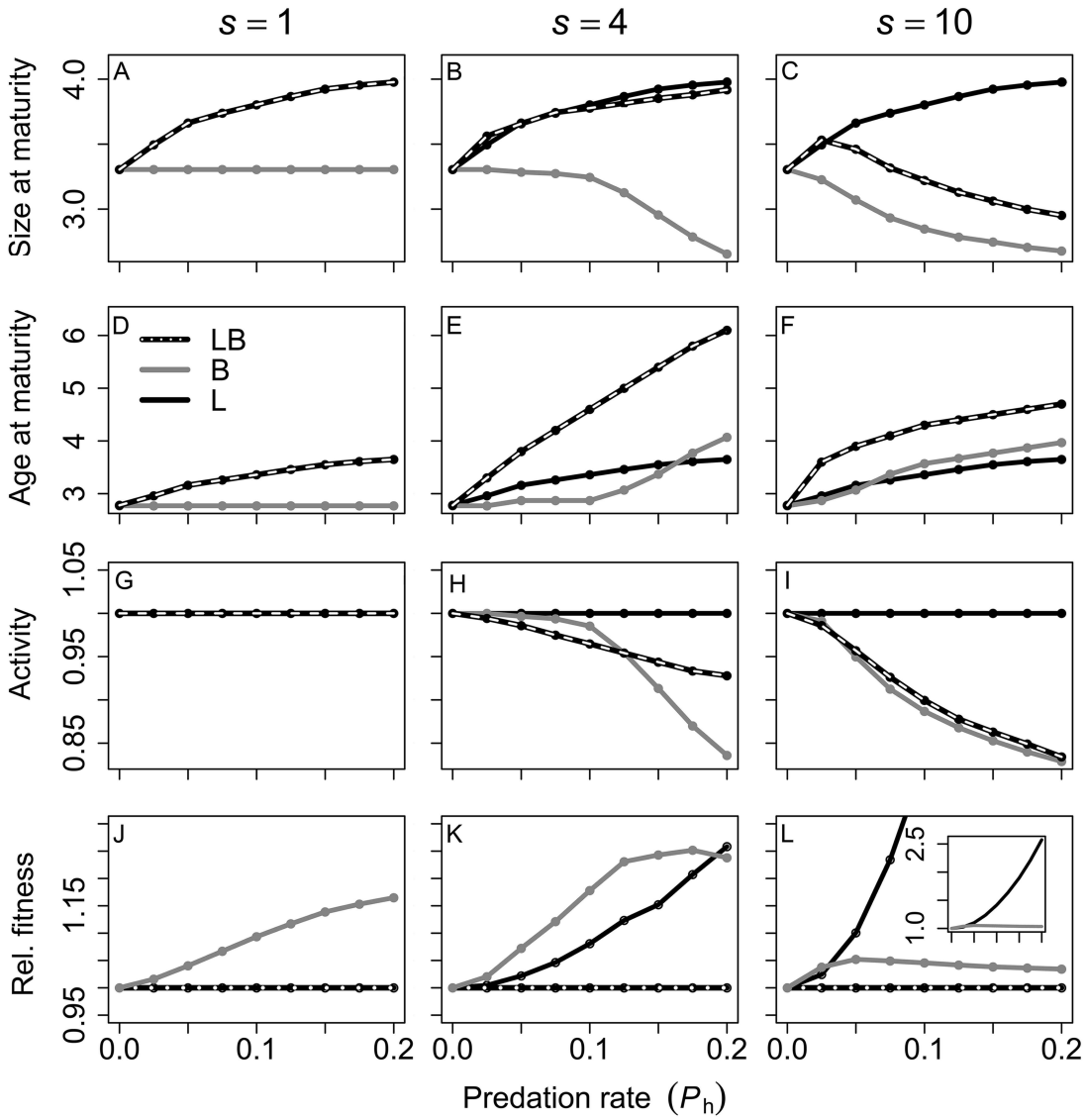

Figure 4: Optimal defense investments for $s = 1$, 4, and 10. For each s value, the metrics for life-history (L) and behavioral (B) defenses are plotted for each of the three selection scenarios. The LB combined strategy is shown by the black-and-white dashed line, the B strategy is shown by the gray line, and the L strategy is shown by the black line. Note that for $s = 1$, the LB and L strategies are identical; only the LB strategy can be seen in these plots. The final row shows the relative fitness advantage of the LB strategy. The black-and-white dashed line is LB relative to itself (fixed at 1), the gray line is LB relative to B, and the black line is LB relative to L.

the black line in figure 3*D* is the optimal predator-free behavior.

As the growth phase is prolonged, size at maturity increases, as does age at maturity. This is entirely straightforward, since more energy is allocated to growth and the switch to allocating energy to maturation and reproduction occurs at later ages. Decreasing activity level causes size at maturity to decrease, because less energy has been ingested by the age when allocation switches to maturation and reproduction. Age at maturity increases because it takes longer to meet the maturation requirement as size at maturation decreases, because reduced size reduces energy intake (app. B).

Linear Trade-Offs Favor Life-History Defenses

Above we predicted that low s values would lead to life-history defenses. It turns out that even a linear trade-off,

which is the expected trade-off shape for a predator with no foraging preference, is enough to produce this result (see fig. 4 for the results when $s = 1$). In figure 4*A*, 4*D*, 4*G*, and 4*J*, the LB and L strategies are identical. No behavioral defense is being used (fig. 4*G*). The optimal strategy is to suffer the high mortality risk resulting from high activity levels and "sprint" for the life-history refuge by not investing in behavioral defense. The main cost of behavioral defense is delayed maturation. This delay has a direct negative impact on fitness; this cost must be offset by benefit of increased survivorship for investment in behavioral defense to be worthwhile. In such an environment, behavioral defense is not effective enough to offset the delay.

Highly Nonlinear Trade-Offs Favor Behavioral Defenses

For high values of *s*, defense expression is dominated by behavioral defenses, as we predicted. The mean activity levels for the LB and B strategies are nearly identical across P_h values (fig. 4*I*). However, the LB strategy is able to compensate for one of the major costs of behavioral defenses (reduced size) by prolonging allocation to growth. This can be seen in the increased size at maturity for the LB compared with the B strategy (fig. 4*C*). In fact, for $P_h < 0.075$, the LB strategy actually invests in both defenses simultaneously, as is seen in the increase in size from $P_h = 0$. The cost of prolonged allocation to growth is delayed maturation, but the increase in birthrate for maturing at a larger size compensates for this delay, giving the LB strategy a significant fitness advantage over both the L and the B strategies. We note that for higher values of P_h (not shown), the LB and B strategies converge, though R_0 drops below replacement.

Moderately Nonlinear Trade-Offs Favor Multidefense Strategies

With moderate values of *s*, there is investment in both defenses simultaneously (fig. 4*B*, 4*H*). This is achieved through a specific interaction between the different defense investment functions. Figure 5 shows a characteristic case. The LB activity level α is less than the B α early in life, indicating that the LB strategy increases expression of behavioral defenses early in ontogeny (fig. 5*B*). Later in life, however, the LB curve lies above the B curve, suggesting that behavioral defenses are relatively underexpressed. Simultaneously, the LB strategy prolongs the allocation to growth $\kappa(t)$ (fig. 5*A*). This allows it to compensate for the decrease in size at maturity caused by reducing activity level. The early reduction in activity level, coupled with prolonged allocation to growth, allows the individual to invest in both defenses. This suggests that the optimal strategy uses $\kappa(t)$ and $\alpha(t)$ to compensate for the costs of defenses through ontogeny.

Discussion

The Shape of the Foraging–Predation Risk Trade-Off Determines the Optimal Defense Strategy

Physiological and ecological trade-offs provide a framework for understanding life-history evolution. However, most life-history theory has assumed that these trade-offs operate independently of one another (Steiner and Pfeiffer 2007). This is unrealistic, as multiple trade-offs can play a role in determining a single trait; optimal trait expression will then be determined by balancing the costs of interacting trade-offs. In this study we consider two fundamental trade-offs: the physiological trade-off between growth and reproduction and the ecological trade-off between foraging gain and predation risk. The first trade-off is mediated by the pattern of energy allocation. Increased allocation to growth reduces size-dependent predation risk, but it carries the cost of delayed reproductive maturity. The second trade-off is mediated by activity level. Reducing activity level reduces behavior-dependent predation risk, but at the cost of reduced growth and delayed maturity. That both trade-offs share a benefit but differ in cost suggests that the optimal pattern of behavior and energy allocation will depend on the shapes of these underlying trade-offs.

We show that by varying the shape of the foraging–predation risk trade-off, we can arrive at very different patterns of covariation between life history and behavior. When the foraging–predation risk trade-off is highly nonlinear and accelerating ($s \gg 1$), behavioral defenses are highly effective and we predict that behavior and life history will be dominated by this trade-off. On the other hand, when the trade-off is linear or decelerating ($s \leq 1$), we predict that behavior and life history are determined by the trade-off between growth and reproduction. However, for the moderately accelerating trade-off shape, behavior and life history are determined by both trade-offs. The interaction between the two trade-offs leads to a multiple-defense strategy, with behavior and life history integrated across ontogeny (Pigliucci 2003).

A number of models have considered how predation jointly modifies behavior and life history (Abrams 1991; Abrams and Rowe 1996; Steiner and Pfeiffer 2007; Urban 2007*a*). Abrams and Rowe (1996) considered how size-independent predation, assuming a concave-up relationship between behavior and predation risk (analogous to $s > 1$), affected optimal age and size at maturity and behavior. In the case most similar to our model, where both traits are flexible and nonpredation mortality does not

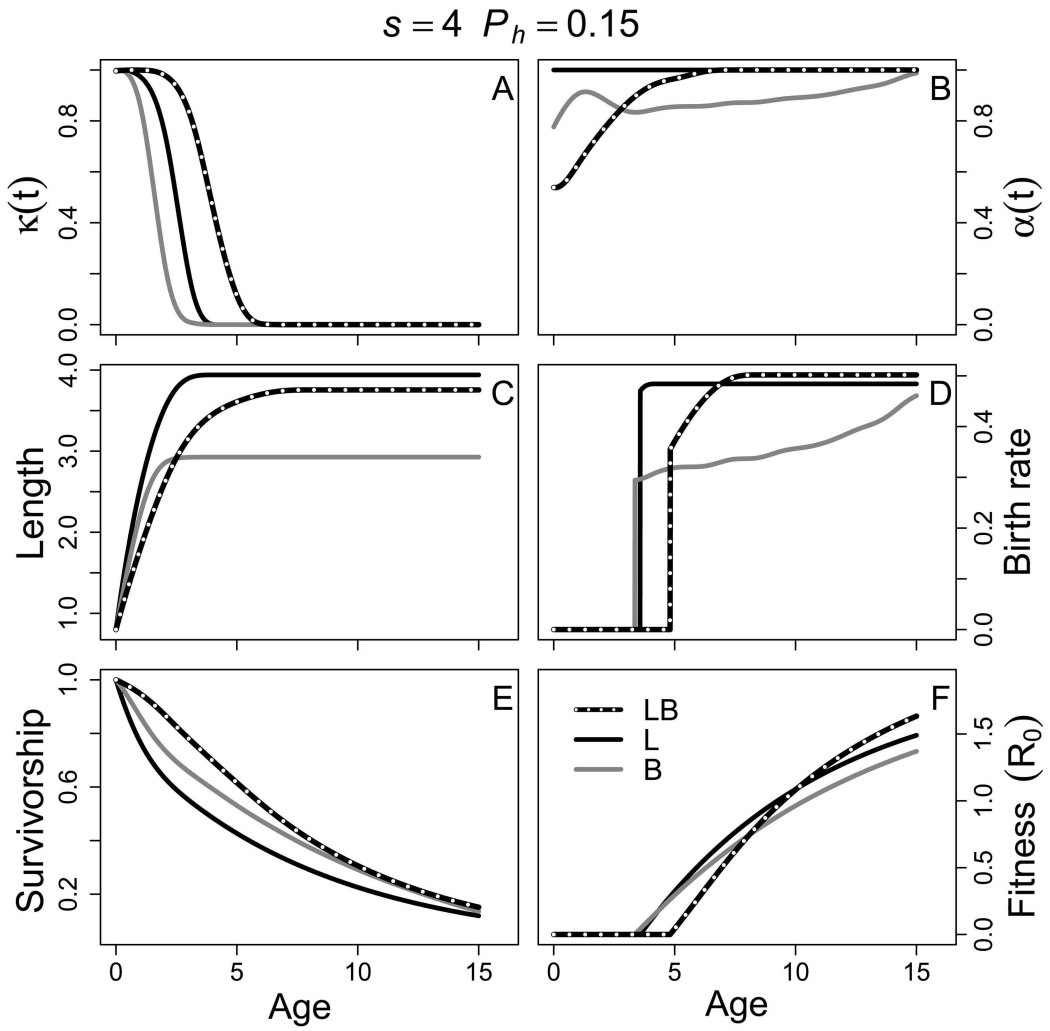

Figure 5: Behavioral (B) and life-history (L) responses and pattern of growth, reproduction, and mortality that result from each strategy for $s = 4$ and $P_h = 0.15$. The multiple-response experiment invests in both defenses across ontogeny by using each response to compensate for the cost of the other defense. This produces the integrated multiple-defense strategy shown in figure 4.

depend on behavior (table 1 of Abrams and Rowe 1996), the direct response to predator density was to reduce size at maturity, decrease growth effort, and decrease growth rate. Age at maturity could increase or decrease, depending on predator density. The model assumptions and predictions agree with the predictions from the $s = 10$ case for our model.

Our predictions for the linear ($s = 1$) case match those of Urban (2007*a*). This model showed that the optimal prey defense strategy could be to forgo a behavioral defense in favor of reaching a size refuge as quickly as possible. This result was much more likely when the relationship between growth and predation risk was linear or decelerating, which is analogous to our results. This result was motivated by empirical data (Urban 2007*b*) and provides additional evidence for the importance of the foraging–predation risk trade-off in determining defense expression in nature.

However, neither the two models described above nor any of the other models that have considered the effect of predation on both life history and behavior have allowed responses to vary with age. As such, they miss the interactions between the responses that allow for multiple-defense strategies to evolve. Intuitively, we expect that investments in behavioral and life-history defense will be negatively correlated. Increasing investment in behavioral defense by reducing activity will reduce size at maturity and, hence, investment in life-history defense. However,

this intuition ignores how the functions underlying defense investment (i.e., growth allocation $\kappa(t)$ and activity level $\alpha(t)$) interact with one another through ontogeny. For moderate s values, the pattern that emerges in these underlying traits is to reduce activity level early in life, when predation risk is highest, while simultaneously keeping growth allocation high. As size increases, activity level is increased until the individual reaches the size refuge. It is the compensatory dynamics between these responses that gives rise to the integrated multiple-defense strategy.

This compensation suggests the importance of timescale in defense expression. Behavioral defenses are effective immediately and can be modified quickly and reversibly. Life-history defenses, on the other hand, are effective only at reducing predation risk after a threshold of energy investment has been made. They are also not reversible: individuals of most species are typically not capable of shrinking. This difference in timescale affects the interaction between the two defenses and highlights the importance of considering defense investment across an individual's lifetime (Clark and Harvell 1992; Relyea 2004*b*; Hammill et al. 2010).

Empirical Evidence for the Theoretical Predictions

There are a number of different mechanisms that could lead to a concave-up nonlinear relationship between activity and predation risk. This nonlinearity is generated whenever predator-encounter rates increase at an increasing rate as prey individuals become more active. Simple mechanisms that could produce such a result are active prey selection, where predators choose more active over less active prey (Snyder 1975; Sarno and Gubanich 1995), or increased perception of more active prey (O'Keefe et al. 1998; Utne-Palm 2000). Additionally, predators could increase their own activity levels or foraging speeds in response to more active prey. The prey-detection method of the predator (e.g., visual hunting vs. mechanoreception) is probably less important than the hunting mode (e.g., active vs. passive predators). Predators that feed via sit-and-wait or filter feeding are less likely to demonstrate preferences and are likely to impose a linear foraging–predation risk trade-off.

Empirical work provides evidence for many of our predictions. Many species are known to exhibit behavioral defenses against negative size-specific predation risk from active predators, including amphibians (Anholt et al. 2000; Urban 2007*b*), *Daphnia* (Pangle and Peacor 2006), fish (Abrahams and Healey 1993), and snails (Hoverman et al. 2005). Studies also observe that predators that prefer small prey and forage passively select for life-history defenses (Crowl and Covich 1990; Chase 1999; Tollrian and Harvell 1999).

However, most of the foregoing studies measured only one phenotypic trait. When multiple defensive traits are measured within the same study, it is almost always found that individuals express multiple defenses, either simultaneously or across ontogeny (Relyea 2001; Hoverman et al. 2005; Boeing et al. 2006). Existing theory, which has focused largely on single traits, does not predict how complex multivariate defense strategies may evolve or how defensive traits may covary with one another. Our model shows that, for behavior and life history, the pattern of covariation depends on the interaction between physiological and behavioral trade-offs underlying these defenses. This suggests a general framework for understanding patterns of covariation between other defensive traits. Furthermore, recent empirical work suggests that integration of multiple defenses may be the rule, rather than the exception. In particular, a number of studies have shown exactly the pattern observed here: high investment in behavioral defense early in life but reduced investment through ontogeny (Pettersson et al. 2000; Relyea 2003; Brodin et al. 2006; Hammill et al. 2010). Additionally, work in positive size-dependent predation systems has shown a similar effect, albeit operating in the opposite direction. For example, *Daphnia* often do not engage in behavioral defense (diel vertical migration) until they reach large sizes, because at small sizes they are protected against predation (Leibold et al. 1994). A recent review of integrated defense responses to predation highlighted the importance of studying trait expression through ontogeny to uncover the important interaction between development and defense expression (Relyea 2004*b*).

Applicability of These Results to Other Systems

The assumptions of our model were chosen to maximize the potential for interaction between the ecological and life-history trade-offs. That is, we focused on defenses that, all else being equal, are negatively related to one another: reduced activity reduces growth rate and, thus, size at maturity. However, it is worth asking how the insights gained from these results could be used to predict defense expression in situations where the size dependence of predation took a different form and defensive traits other than behavior and growth allocation were modified. We focus here on the cases of positive size-dependent predation and morphological defenses.

Theoretical and empirical research have shown that a common response to positive size-dependent predation is to reduce the allocation to growth and increase the allocation to reproduction, leading to early maturation at reduced size (Taylor and Gabriel 1992; Tollrian and Harvell 1999; Ernande et al. 2004). Behavioral defenses, on the other hand, tend to lead to late maturation at reduced size

(Beckerman et al. 2007). Since both behavioral and life-history defenses against positive size-dependent predation would lead to reduced size at maturity, investment in both defenses simultaneously would seem to be easier. We have investigated how variation in the shape of the foraging–predation risk trade-off affects behavior and life history under positive size-dependent predation (app. D in the online edition of the *American Naturalist*). Our results show that multiple-defense strategies are common (present even for $s = 1$), but a switch to behavior-only defenses occurs for lower values of s. This provides an alternative explanation for life-history patterns attributed to either altered energy allocation or behavior in previous studies (Ball and Baker 1996; Beckerman et al. 2007).

Morphological defenses are ubiquitous in natural systems (Tollrian and Dodson 1999). Development of morphological defenses is typically thought to require reallocation of energy from growth and reproduction (Steiner and Pfeiffer 2007). Additionally, both life-history and morphological defenses have indirect positive effects on fitness through reduced predation rate. Clark and Harvell (1992) predicted that investment in such traits is best done early in life, with allocation to reproduction done only later in life. These similarities suggest that morphological and life-historical defenses may show similar patterns of covariance with behavioral defenses. Empirical work supports this supposition. For example, Hammill et al. (2010) has shown that the ciliate *Euplotes* expresses behavioral defenses initially in response to predators, with morphological defenses being expressed later. This temporal separation of defense expression is exactly analogous to the predictions made by our model. Theoretical work by Steiner and Pfeiffer (2007), however, predicts that integration may be common between behavior and morphology. They found that increasing predator density increased investment in both morphological and behavioral defenses. Investment in single defense was found only when morphological defense effectiveness was increased until the behavioral defense was no longer necessary. This would be similar to decreasing the value of ℓ_{mid} here.

Implications for Ecological Theory

Understanding how trade-offs interact with one another is essential to understanding phenotypic evolution. Two of the best-studied trade-offs involve the behaviorally mediated trade-off between foraging and predation mortality and the physiologically mediated trade-off between growth and reproduction. Both of these trade-offs involve fundamental traits that have wide-ranging impacts on individual-, population-, and community-level processes (Werner and Gilliam 1984; de Roos et al. 2003). Here we show that varying the shape of the foraging–predation risk trade-off modifies the interaction between the trade-offs, leading to the evolution of qualitatively different defense strategies.

The foraging–predation risk trade-off has been widely cited as important in understanding ecological dynamics (Abrams 1992; Werner and Peacor 2003). Our results suggest that this trade-off has important implications for optimal investment in different defenses as well, and we predict that the differences in defense strategy between closely related species or clones of single species may be explainable in terms of differences in the shapes of the foraging–predation risk trade-off. This has important implications for ecological theory, as it suggests that previous work that has considered how only behavior modifies ecological interactions is limited. Integrated multiple-defense strategies are common in nature, and our model predicts that they should arise under common conditions. However, no theory has been developed yet that explores the ecological consequences of integrated defense strategies. Furthermore, consideration of the ecological dynamics that results from different defense strategies opens up the question of the role of feedback between ecological and evolutionary dynamics in driving selection on behavior and life history. Such feedback can generate diversifying selection, permitting the coexistence of multiple defense strategies (Abrams et al. 1993; Day et al. 2002).

Trait expression is an area of research that has recently received renewed interest (Abrams 2001). Because of the important effects of dynamic traits on ecological interactions (Abrams 1995; Werner and Peacor 2003), understanding how ecological factors promote the expression of different characters becomes crucially important for understanding ecological communities. However, this understanding requires that empiricists and theoreticians move beyond thinking about single traits to a fuller consideration of how the expression of multiple traits varies across ontogeny and across different environments.

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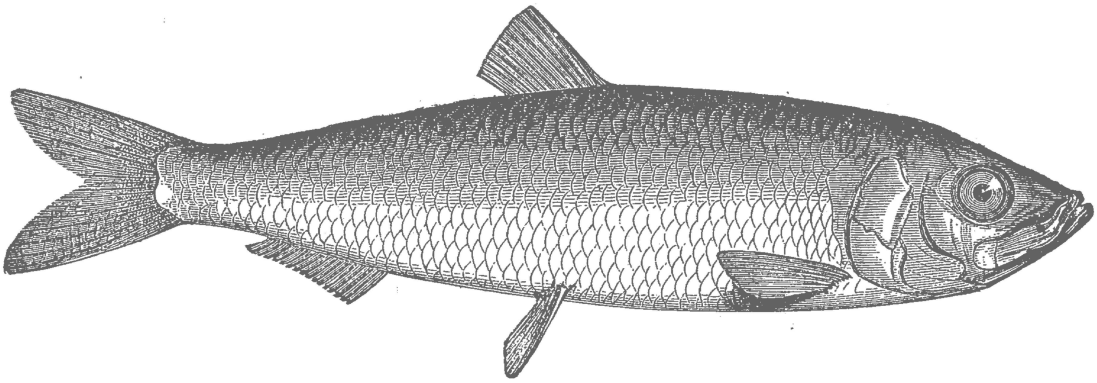

Herring *Clupea elongata* have "almost deserted the waters about Provincetown, where I have formerly seen them in immense schools very near the shore." From "The Habits and Migrations of Some of the Marine Fishes of Massachusetts" by James H. Blake (*American Naturalist*, 1870, 4:513–521).

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Appendix A from C. E. Cressler et al., "Interactions between Behavioral and Life-History Trade-Offs in the Evolution of Integrated Predator-Defense Plasticity"

(Am. Nat., vol. 176, no. 3, p. 276)

Derivation of the Growth Equation: The Behavior-Dependent Functional Response

Energy ingestion is assumed to be proportional to the surface area (which is itself proportional to the square of length, ℓ^2) of an individual. This assumption is well-justified for many species (Kooijman 2000, pp. 66–71). For an individual of a given size, energy ingestion is further assumed to depend on resource density, as well as on the behavior of the individual according to a modified version of a Type II functional response. Letting $f(R, \alpha)$ be the functional response, the rate of energy ingestion is

$$I(R, \ell, \alpha) = f(R, \alpha)\ell^2. \tag{A1}$$

The functional response $f(R, \alpha)$ can be derived following the logic developed by Holling (1959). The function $\alpha(t)$ represents the fraction of total possible foraging time that is actually spent foraging. Note that α is not the fraction of total time; the underlying assumption is that a fixed amount of an individual's time budget is spent on other processes (mating, brooding, territory defense, etc.) and that this amount does not change. The dependence of α on t reflects the fact that the amount of time spent foraging may change with the age (and physiological state) of an individual.

Let T be the total time possible for foraging; αT is the actual amount of time spent foraging. The remainder of the total possible time $(1 - \alpha)T$ is assumed to be spent engaging in defensive behaviors. Dividing foraging time into its component processes, $\alpha T = T_h + T_s$, where T_h is total time spent handling food items and T_s is the total time spent searching for food. Total time handling food should be $r \times h$, where r is the total number of calories ingested and h is the handling time for each calorie. Following Holling, $r = k \times R \times T_s$, where k is the search efficiency (volume searched per unit time) and R is the resource density (calories per unit area or volume). The parameters h and k are also dependent on the surface area of an individual, as larger individuals handle more food and search a greater area per unit time. Thus, the number of food items taken per unit of time spent foraging is then

$$\frac{r}{\alpha T} = \frac{kRT_s}{T_h + T_s} = \frac{kRT_s}{kRT_s h + T_s} = \frac{kR}{1 + khR}, \tag{A2}$$

and the functional response (items taken per unit of time allocated to foraging) is

$$f(R, \alpha) = \frac{r}{T} = \frac{\alpha kR}{1 + khR}. \tag{A3}$$

We then make the following substitutions: $k = v_x\xi$ and $h = 1/v_x$. Parameters v_x and ξ are defined in table 1 in the text. By simplifying, the rate of energy ingestion is

$$I(R, \ell, \alpha, t) = v_x\alpha(t)\frac{\xi R}{1 + \xi R}\ell^2. \tag{A4}$$

Growth and Reproduction

The article by de Roos et al. (1990) can be referenced for a full derivation of the growth equation. Here we highlight the differences between their model and ours. The key difference is in the placement and interpretation of the parameter κ. Kooijman and Metz (1984) and de Roos et al. (1990) interpreted κ as representing the fraction of energy allocated to both growth and maintenance, making their models net assimilation models (because assimilated energy is partitioned between growth, maintenance, and reproduction; Noonburg et al. 1998). Because of this assumption, if the energy allocated to growth is not enough to meet basic maintenance requirements, then energy must be reallocated to growth from reproduction, necessitating a more complicated formulation. With our formulation, maintenance costs are "taken off the top" and net production is partitioned between growth and reproduction (Noonburg et al. 1998). This allows $\kappa(t)$ to be free from an a priori constraint. In the event that total energy ingestion is not enough to meet basic maintenance requirements, an individual is assumed to die from starvation. Biologically, this is not completely realistic, as most organisms maintain an energy reserve that can sustain them during periods of low resources; however, since resources are constant in these experiments, this simplification does not affect our results.

This change in assumption does, however, modify the physiological bases for the growth parameters in our model. In the Kooijman and Metz (1984) model, energy allocation determines the maximum size an individual can reach, with a fixed growth rate per unit. We view maximum length as fixed and κ as modifying the growth rate per unit production. To be consistent with empirical work, we preserve the maximum length at the value reported by de Roos et al. (1990). We then set maximum growth rate to be equal to 0.5 so that if κ were held constant at the de Roos et al. (1990) value (0.3), our growth rate $\hat{g}\kappa$ would equal their γ.

Our approach to reproduction agrees with that of Kooijman and Metz (1984) in that we view κ as modifying the rate of reproduction. Therefore, $\hat{r}(1 - \kappa)$ is equivalent to the parameter $r_{\max}$ in Kooijman and Metz (1984), and if we hold κ fixed at 0.3, then $\hat{r}(1 - \kappa) = r_{\max}$.

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Appendix B from C. E. Cressler et al., "Interactions between Behavioral and Life-History Trade-Offs in the Evolution of Integrated Predator-Defense Plasticity"

(Am. Nat., vol. 176, no. 3, p. 276)

Modeling Sexual Maturation

The original model of de Roos et al. (1990) assumed that maturation occurred at a fixed size (2.5 mm). This was reasonable because the authors also assumed that κ was fixed at 0.3. Since we are allowing $\kappa(t)$ to vary in time, it is inappropriate to think of maturation as occurring at a fixed size. Instead, it is more reasonable to think of maturation as occurring once some investment into germ tissue was met. In a study by de Roos et al. (1990), the authors noted that there must be energy allocated to maturation; before reaching size at maturity, the 0.7 fraction of ingested energy was implicitly assumed to be allocated to maturation. Thus, using the original de Roos et al. (1990) model, with its constant $\kappa = 0.3$, it is possible to analytically determine the amount of energy allocated to sexual maturity when maturation occurs at a fixed size. This amount was then set as a threshold in our model; maturation occurred when enough energy had been allocated to reproduction to meet this threshold. Thereby, the timing and size at maturity were free to change with the allocation strategy employed by an individual.

Figure 3 in the text depicts the size at maturity that results from different allocation functions, including the optimal predator-free allocation function. To give a sense of how size at maturity influences the amount of time required to reach sexual maturity, we take advantage of the fact that all of the κ splines closely approximate a "bang-bang" allocation strategy (sensu Vincent and Pulliam 1980), where energy is allocated only to growth and then only to reproduction. Figure B1 plots time required to reach sexual maturity for different maturation sizes. In creating this plot, we have assumed that, before reaching this size, all energy was allocated to growth, and after maturation size was reached, all energy was allocated to maturation. The time delay decreases over smaller sizes but then increases rapidly due to the nonlinearity in net production rate (ingestion rate scales with surface area but maintenance costs scale with body volume).

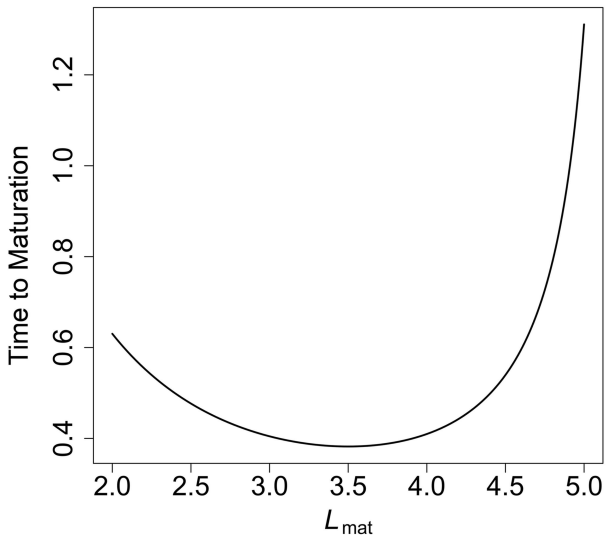

Figure B1: Time required to reach sexual maturity as a function of the size at maturity. This assumes that before reaching size at maturity, all energy was allocated to growth, with allocation to maturation occurring only thereafter.

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Appendix C from C. E. Cressler et al., "Interactions between Behavioral and Life-History Trade-Offs in the Evolution of Integrated Predator-Defense Plasticity"

(Am. Nat., vol. 176, no. 3, p. 276)

Genetic Algorithm Details

Genetic algorithms are a useful computational tool for performing optimizations (Holland 1975). Conceptually, genetic algorithms are an approximation of natural selection; natural selection operates via the biased reproduction of individuals with higher fitness, with random variation acting to maintain diversity on which selection can act. In order for the analogy to hold up and genetic algorithms to work properly, the following components are needed (Mitchell and Taylor 1999): (1) A population of candidate solutions for the problem to be solved. The solutions are encoded according to a representation scheme. Extending the analogy of natural selection, these solutions represent chromosomes, and the units of encoding are genes. (2) A fitness criterion that assigns a numerical value to each chromosome, determining its quality as a solution to the problem at hand. (3) A way of "mating" different candidate solutions to create a new population of solutions when the current population has been assigned fitnesses. This reproduction scheme typically incorporates elements of selection, mutation, and crossover.

For the given problem of finding the optimal defense strategy, the analogy with natural selection is very natural; the candidate solutions are the $\alpha(t)$ and $\kappa(t)$ functions, and the fitness function is R_0, defined in the text and determined by solving the energetics equations. Because of this, it becomes tempting to view the genetic algorithm as modeling evolution. However, these algorithms are designed only as optimization tools and do not attempt to model the actual process of evolution via natural selection.

Since we are attempting to determine the optimal shapes of the $\alpha(t)$ and $\kappa(t)$ functions, we need a way of encoding these functions. B-splines are particularly useful, as they are flexible enough to take any shape along a given interval of interest and can be encoded as a sequence of points. To define a B-spline, we must first specify a vector known as a knot vector:

$$\mathbf{t} = t_0, t_1, \ldots, t_m.$$

These knots span the interval of interest; in this case, the interval is the life span of an individual. A B-spline is a linear combination of basis functions, polynomials of degree n that are defined between each knot. The B-spline also has degree n; here, $n = 3$.

A B-spline is also characterized by a sequence of points, $p_0, p_1, \ldots, p_p$, known as control points. The control points are the encoding scheme; an individual's $\alpha(t)$ and $\kappa(t)$ functions are specified by the particular sequence of control points. Thus, each individual has a unique set of control points. These control points determine the value of the function at any point in the interval specified by the knot vector. Since $\alpha(t)$ and $\kappa(t)$ are proportions between 0 and 1, as long as each control point is between 0 and 1, the B-spline will also take only values between 0 and 1. The number of control points specified depends on the length of the knot vector and the degree of the B-spline, according to the relation $p = m - n - 1$. The "wiggliness" of the B-spline is controlled by the number of knots and control points. For these experiments, the number of knots was specified to be 28, so each individual was characterized by two vectors of 24 control points determining the $\alpha(t)$ and $\kappa(t)$ functions.

To calculate the value of the B-spline for any point in the interval $[t_0, t_m]$, we must first calculate the value of the basis functions:

$$b_{i,0}(t) = \begin{cases} 1 & \text{if } t_i \le t < t_{i+1}, \\ 0 & \text{otherwise,} \end{cases} \tag{C1}$$

$$b_{i,n}(t) = \frac{t - t_i}{t_{i+n} - t_i} N_{i,p-1}(t) + \frac{t_{i+n+1} - t}{t_{i+p+1} - t_{i+1}} N_{i+1,p-1}(t). \tag{C2}$$

Then the value of the spline at the point t within the interval $[t_0, t_m]$ is (De Boor 1978):

$$B(t) = \sum_{i=0}^{p} p_i b_{i,p}(t). \tag{C3}$$

In this way, the value of $\alpha(t)$ and $\kappa(t)$ can be determined for any t.

Using this encoding scheme and the fitness function defined by R_0, the genetic algorithm operates in the following way. On initialization, the algorithm randomly generates 800 candidate solutions (individuals) who are defined by two vectors of 24 control points specifying the $\alpha(t)$ and $\kappa(t)$ functions, with each control point generated as a random draw from the distribution $U[0, 1]$. The vectors are the "chromosomes" and the control points are the "genes." The energetics equations are solved for each individual and a fitness is assigned to each individual. Parents are then selected to produce the next generation of potential solutions (described below). The next generation will be of the same size as the current generation. Crossover occurs between the chromosomes of the two parents. Mutation occurs with a fixed probability for each gene in the chromosome. This completes one generation of the genetic algorithm. This process is repeated for 1,000 generations, which was long enough for convergence to occur in all cases. Convergence was determined by calculating the standard deviation of fitness across all individuals in the population; the standard deviation was <0.005 for all runs, and it was typically <0.0005.

There are many different approaches to selection (outlined in Mitchell 1998). A good selection method should have a strength of selection that is relatively constant throughout the run of the genetic algorithm. Some methods have the problem that selection is very strong early in the run and weaker late in the run as each individual is more similar. This can lead to premature convergence of the algorithm. To circumvent this problem, we used sigma scaling of raw R_0 values (Mitchell 1998). The expected number of reproductive events for an individual, i, was calculated as

$$E(R_0(i)) = \begin{cases} 1 + \dfrac{R_0(i) - \overline{R}_0}{2 \times \sigma} & \text{for } \sigma \neq 0, \\ 1 & \text{for } \sigma = 0, \end{cases} \tag{C4}$$

where $\overline{R}_0$ is the average fitness of all individuals in the population and σ is the standard deviation of fitness. If this value is negative, we set it equal to 0. The scaling works by smoothing out fitness differences when there is a lot of variation but emphasizing fitness differences when the population is homogeneous. Early in the algorithm, when σ is large, the most fit individuals are not allocated the majority of the reproductive events. When σ is small late in the run, individuals with higher than average fitness stand out more, allowing evolution to continue.

When individual fitnesses have been scaled to an expected value, individuals are paired as parents according to stochastic universal sampling, which has zero bias and minimum spread (Mitchell 1998). This sampling algorithm ensures that every individual will be chosen as a parent no more than $E(R_0(i)) + 1$ times and no fewer than $E(R_0(i))$ times. This also ensures that the size of the population remains constant at 800 for each generation. When individuals are paired, each reproductive event creates two new offspring whose chromosomes are generated by crossover between the two parents and mutation of the parental genes. For each offspring, a crossover point is chosen along the length of the chromosome; the offspring inherits the control points of opposite parents on opposite sides of the crossover point. Crossover acts to create variation while preserving combinations of control points that yield high fitness. Mutation occurs with fixed probability (0.01); mutations alter the value of the gene by drawing a new gene value from a normal distribution with mean equal to the current gene value and a standard deviation of 0.1. We constrain genes to take values between 0 and 1; if mutation alters the value outside of this interval, it is placed on the appropriate boundary.

This entire process is repeated for 1,000 generations to determine the optimal solution. For each parameter

combination, the algorithm was run 10 times. We then use a mutation-only search to determine the optimal strategy. This was done by first finding the strategy with the highest fitness in each of the 10 replicates. These strategies were then subjected to mutation, where a single gene was chosen at random and mutated to a random value drawn from a normal distribution with a mean at the current gene value and a standard deviation of 0.01. If the resulting strategy had a higher fitness, this mutation was accepted; if not, the strategy remained unchanged. This process was repeated for 10,000 mutation events. In all cases, the 10 replicate data sets had converged to strategies whose fitnesses were within 0.01% of one another. The results presented in the text show the strategy with the highest fitness.

Literature Cited Only in Appendix C

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 DOI: 10.1086/655425

Appendix D from C. E. Cressler et al., "Interactions between Behavioral and Life-History Trade-Offs in the Evolution of Integrated Predator-Defense Plasticity"

(Am. Nat., vol. 176, no. 3, p. 276)

Results for Positive Size-Dependent Predation

We also investigated the consequences of positive size-dependent predation on the interaction between trade-offs. Positive size dependence in predation risk (that is, predators that prefer large prey items) is common in many of the systems referenced in the main text (e.g., *Daphnia* [De Meester et al. 1995], damselflies [McPeek and Peckarsky 1998], dragonflies [Mikolajewski and Johansson 2004], and snails [Rundle and Brönmark 2001]). Previous theory suggests that the life-history response to positive size-dependent predation is to reduce allocation to growth versus reproduction, leading to the defense of early maturation at reduced size (Taylor and Gabriel 1992; Ernande et al. 2004). However, as we show in the main text, a consequence of behavioral defense investment is reduced energy gain, which can lead to reduced size at maturity. Some experimental work has also revealed that predator-induced life history could be a result of changes in physiology or behavior (Ball and Baker 1996; Beckerman et al. 2007). Thus, understanding how life history and behavior might covary under positive size-dependent predation is also a compelling question.

To modify the main text equations to consider positive size dependence, only one change was necessary: instead of size-dependent mortality taking the form

$$1 - \tan h[\omega(\ell - \ell_{\mathrm{mid}})], \tag{D1}$$

it takes the form

$$1 + \tan h[\omega(\ell - \ell_{\mathrm{mid}})]. \tag{D2}$$

The effect of this change can be seen in figure D1.

To determine the optimal behavior and life history, we followed the same protocol as in the main text. We found optimal behavior and life history using genetic algorithms for three experiments: when both activity level and growth allocation were flexible (LB experiment), when activity level was flexible but growth allocation was fixed at the optimal predator-free level (B experiment), and when growth allocation was flexible but activity level was fixed at the optimal predator-free level (L experiment). Again, s was varied between 1 and 10 and P_{h} was varied between 0 and 0.2. Life-history defenses were employed whenever size at maturity is smaller than the predator-free value and age at maturity is earlier. Behavioral defenses were employed whenever average activity level was <1.

Results for $s = 1$ and $s = 2$

For both $s = 1$ and $s = 2$, both defenses were employed (fig. D2), although the investment in behavioral defense is relatively weak. That both defenses are employed is demonstrated by the fact that maturation occurs at small sizes (fig. D2*A*, D2*B*) and early (fig. D2*E*, D2*F*), while activity level is slightly reduced (fig. D2*I*, D2*J*). This shows that multiple defense strategies are more common for negative size-dependent predation, since linear foraging–predation trade-offs led to life-history-only strategies for positive size-dependent predation. Life-history-only strategies may be present for values of s that are <1, that is, where predation rate saturates with increases in activity level.

Results for $s = 4$

For $s = 4$ (a moderately nonlinear relationship between behavior and predation rate), we see investment in both defenses at low predation rate (as evidenced by the decrease in size at maturity [fig. D2*C*] and the reduced activity level [fig. D2*K*]). The strategy does not reduce age at maturity due to the cost of behavioral defense (fig. D2*G*), but neither does it mature late. However, for higher levels of predation rate, we see investment only in behavioral defense as age at maturity increases above the predator-free level.

Results for $s = 10$

For $s = 10$, only the behavioral defense is used (fig. D2*D*, D2*H*, D2*L*). Predator-induced changes in size at maturity and age at maturity are caused by the reduced activity level and energy intake of the organism, rather than by an altered growth allocation.

Conclusions

Under positive size-dependent predation risk, we still observe both pure behavioral and integrated multidefense strategies. Pure life-history strategies presumably are found as s is reduced further. The switch to pure behavioral strategies is predicted to happen at lower values of s. The reason for the earlier switch is fairly intuitive. Reduced activity level reduces energy intake; this tends to caused delayed maturation at smaller size. Under negative size-dependent predation, this has multiple costs to the organism: (1) reproduction starts later in life, (2) birthrate is reduced because of reduced size, and (3) mortality risk is higher because of reduced size. The benefit is, of course, reduced mortality. In order for behavioral defenses to be optimal, this benefit must outweigh all of these costs. However, under positive size-dependent predation, the third cost is not present and the benefits of activity level reduction need only outweigh the first two costs. The result that integrated multiple-defense strategies are possible can provide alternative explanations for previous experimental work, which has attempted to attribute predator-induced life-history patterns to either behavioral or life-history defenses, but not both. In particular, Beckerman et al. (2007) found evidence for both defenses contributing to observed life history, but they interpreted the results as more strongly supporting the hypothesis that life history was the result of changes in energy allocation.

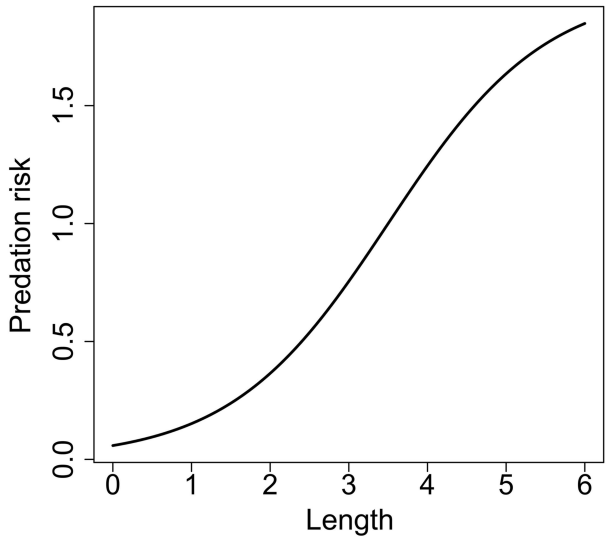

Figure D1: Scaling of predation rate by length under positive size-dependent mortality risk; $\omega = 0.5$ and $\ell_{\text{mid}} = 3.5$, as in the main text.

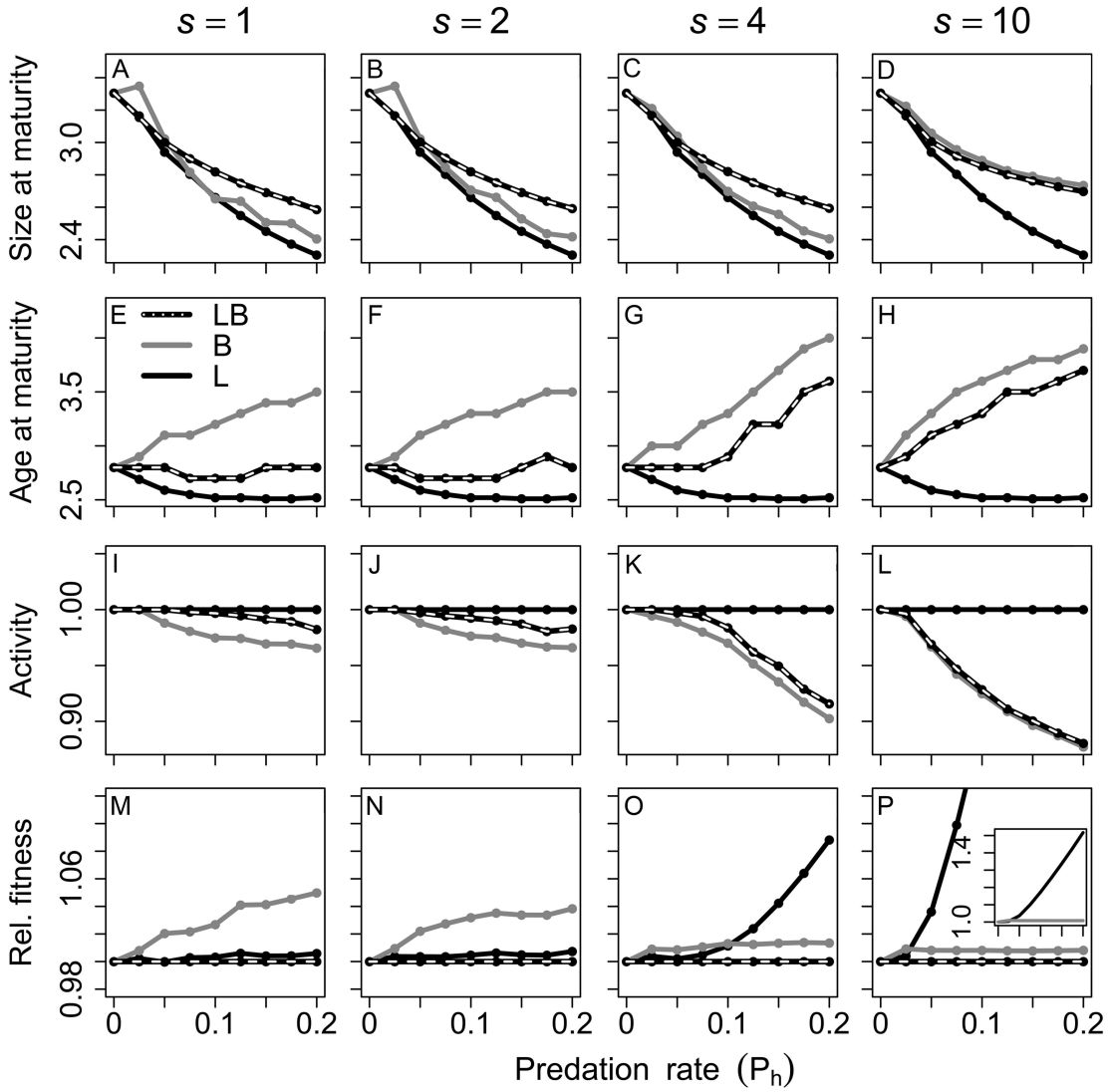

Figure D2: Optimal behavior and life history for different shapes of the foraging–predation risk trade-off (higher *s* values lead to increasingly nonlinear trade-offs). The optimal multidefense strategy is shown by the black-and-white dashed line, the optimal behavior-only strategy is shown by the gray line, and the optimal life-history-only strategy is shown by the black line.

Literature Cited Only in Appendix D

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