

# The foraging–predation risk tradeoff and the evolution of multiple-defense plasticity

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## 1. Introduction

Tradeoffs have played a key role in our understanding of ecology and evolutionary biology. For example, interspecific performance tradeoffs have long been thought to govern the coexistence of species at the local scale, while tradeoffs between competitive and colonization abilities may help govern coexistence at larger spatial scales. Tradeoffs are also fundamental to understanding individual traits as well. Two such tradeoffs are the tradeoff between growth and reproduction and the tradeoff between foraging and predation risk. Both are highly general and highly influential in understanding individual ecology and evolution. The growth–reproduction tradeoff has been used by life history theory to explain the evolution of different growth and reproduction strategies. The foraging–predation risk tradeoff has been invoked by ecological theory as fundamental to understanding community dynamics.

Despite the recognition that these are fundamental tradeoffs faced by organisms in nature, relatively little theory in ecology or evolutionary biology has explicitly considered how different tradeoffs might interact with one another. One area where we can explore the interaction of multiple tradeoffs is in the evolution and expression of inducible defenses.

Inducible defenses encompass a broad range of phenotypically plastic traits, traits whose expression is determined by the environment, as well as genetics. These defenses including modifications in behavior, morphology, and life history that act to reduce predation risk (Tollrian and Dodson 1999). However, qualitatively different defense strategies (e.g., behavioral versus morphological defenses) have different costs - for example, behavioral defenses nearly always involve reduced foraging gain

(either through reduction in activity level or movement to a suboptimal foraging habitat (Lima and Dill 1990)), whereas life history defenses often modify the allocation of energy between growth and reproduction to optimize size. Essentially, for a given strategy, defense expression is determined by the relevant tradeoff.

However, given that each of these different defense strategies is a potential solution to the problem of predation risk, explaining why some species employ one defense instead of another involves exploring how different tradeoffs interact with one another to determine the evolution of different defense strategies. Furthermore, many species are capable of expressing multiple defenses, either simultaneously or across their lifetime (Hoverman et al. 2005; Boeing et al. 2006; Steiner and Pfeiffer 2007). In these cases, how does the interaction between the tradeoffs play out to generate integrated defense responses?

Here we focus on the interaction between the growth–reproduction tradeoff and the foraging–predation risk tradeoff to determine how these tradeoffs affect the expression of behavioral and life history defenses. We develop a general model for individual physiology and show that by varying the shape of the tradeoff between foraging gain and predation risk, we can generate different defense strategies, ranging from pure behavioral or life history defenses to integrated defense strategies. When multiple defenses are expressed simultaneously, the individual responses to the tradeoffs show a characteristic positive correlation. This implies that the tradeoffs can interact in a non-additive way. This allows one response to compensate for the cost of the other. This work demonstrates that a full understanding of individual life history (that is, the pattern of growth, reproduction, and mortality) requires an understanding of how different fundamental tradeoffs interact with one another at the level of the individual.

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## 2. Methods

### 2.1. Model description

Tradeoffs between different defenses arise at the level of individual physiology. Therefore the model must account for basic physiological processes (growth, maturation, reproduction) explicitly, focusing on how inducible defenses affect these processes. Optimal single or multiple defense investment can then be determined by explicit consideration of individual fitness.

We employ an individual-based physiological model where growth, reproduction, and death depend on the current state of the individual. The model we have developed is a general model for the physiology of an animal, based on dynamic energy budget models (Kooijman 2000). It is a variation on the physiologically-structured model developed by de Roos and colleagues (de Roos et al. 1990, 1992; de Roos 1997). Parameter values have been taken from de Roos et al. (1990), and the model and parameter values are well-verified from experimental data on the zooplankter *Daphnia magna* (de Roos 1997). Table 1 presents the variables and parameters used in the model, and gives default parameter values.

### 2.2. Characterizing investment in defense

Behavioral defenses are often characterized by changes in activity level or habitat to reduce the encounter rate with predators (Lima and Dill 1990; Tollrian and Dodson 1999). In our model, behavioral defense investment will be determined by  $\alpha(t)$ , the fraction of total foraging time that is spent active versus engaging in defensive behaviors such as hiding or remaining inactive. Thus,  $\alpha(t)$  will affect both foraging gain and predation risk; decreasing  $\alpha$  will reduce predation risk, but at the cost of reduced foraging gain.

Life history defenses are typically thought to result from changes in energy investment towards growth versus reproduction (Riessen 1999). Basic theory suggests that predator preference for large prey (positive size selection) favors increased allocation towards reproduction, leading to earlier and smaller maturation, whereas negative size selection favors increased allocation towards growth, leading to later and larger maturation (Taylor and Gabriel 1992; Ernande et al. 2004; Gardmark and

Dieckmann 2006).  $\kappa(t)$  is the fraction of ingested resources allocated towards growth versus reproduction; decreases in  $\kappa$  will lead to earlier maturation at a smaller size, whereas increases will lead to later maturation at a larger size.

The time dependence of both activity level  $\alpha(t)$  and growth allocation  $\kappa(t)$  reflects the fact that investment may change as a function of an individual's age, size, and physiological state.

We also want to make clear a distinction between the physiological responses to predation and the defenses against predation. In our case, changes in the shapes of  $\alpha(t)$  and  $\kappa(t)$  are responses to predation. These responses are responsible for producing the observed defenses (in this case, activity level and size). However, the important distinction is that the responses are independent of one another, but the defenses are not. In particular, size is affected both by  $\alpha(t)$  and  $\kappa(t)$ . This distinction is important for understanding the results presented in the rest of this paper.

### 2.3. 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:

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

where  $v_e$  is the maximum rate of energy consumption and  $\frac{\xi R}{1 + \xi R}$  is a Type II functional response. For this paper, resources will be held constant, so the type II functional response could be replaced with a dimensionless measure of resource abundance; we include it for full generality.

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 upon the surface area of the individual, which is proportional to  $\ell^2$ . 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.

Table 1: Model variables and parameters with default parameter values.

Symbol	Description	Units	Default value
$t$	Age	d	
$\ell$	Length	mm	
$\alpha$	Fraction of time spent foraging		
$\kappa$	Fraction of energy allocated to growth		
$b$	Number of eggs laid by an individual	eggs	
$p$	Survivorship of an individual		
$R$	Resource density	cells ml <sup>-1</sup>	500000
$v_e$	Maximum resource intake rate per unit surface area	cells mm <sup>-2</sup> d <sup>-1</sup>	1.8 x 10 <sup>6</sup>
$\xi$	Functional response shape parameter	ml cell <sup>-1</sup>	7.0 x 10 <sup>-6</sup>
$\ell_{\max}$	Maximum attainable length under unlimited resources	mm	6.0
$\gamma$	Rate constant of growth	d <sup>-1</sup>	0.15
$r_{\max}$	Maximum reproductive rate per unit surface area	eggs mm <sup>-2</sup> d <sup>-1</sup>	0.1
$\mu$	Background mortality rate	d <sup>-1</sup>	0.01
$\omega$	Slope of mortality-size curve	mm <sup>-1</sup>	0.5
$\ell_{\text{mid}}$	Median size preference of predator	mm	3
$P_{\max}$	Maximum predation rate	d <sup>-1</sup>	Varied between 0.1 and 1
$s$	Shape parameter for activity–mortality relation		Varied between 1 and 10

Some energy must then be utilized for maintenance. Maintenance costs are assumed to scale with body volume (proportional to the cube of length). This scaling is also general (see Kooijman (2000), pp. 89-94). Maintenance costs, therefore, create a tradeoff involving length: increased length increases both energy ingestion and maintenance, with maintenance costs increasing more rapidly as individuals get larger. This tradeoff leads to the optimal size at maturity being smaller than the maximum possible size, a point that will become important when considering life history defenses against predation.

#### 2.4. Growth and reproduction

The fraction of resources 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 tradeoff between current growth and future reproduction. When resources are constant, growth follows a modified von Bertalanffy growth equation (de Roos 1997; Kooijman 2000), which predicts growth in size to approach  $\ell_{\max}$  at a rate determined by the parameter  $\gamma$ , assuming that growth allocation  $\kappa(t)$  and activity level  $\alpha(t)$  are both con-

stant.

$$\frac{d\ell}{dt} = \gamma\kappa(t)(\ell_{\max}f(R, \alpha) - \ell(t)), \quad (2)$$

where

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

The remaining energy is allocated towards maturation or reproduction. Prior to reaching sexual maturity, individuals are assumed to allocate energy towards the development of non-somatic tissue (such as gonads or ovaries). Sexual maturity is reached upon investment of a fixed amount of energy into maturation. After reaching maturity, the rate of offspring production is determined by energy allotment towards reproduction  $1 - \kappa(t)$  and the maximum potential reproductive rate  $r_{\max}$ .

$$\frac{db}{dt} = r_{\max}(1 - \kappa(t)) \left( f(R, \alpha)\ell(t)^2 - \frac{\ell(t)^3}{\ell_{\max}} \right). \quad (4)$$

#### 2.5. Mortality

Finally, individuals experience mortality, with mortality due to predation dependent on both behavior and size. A large body of literature has

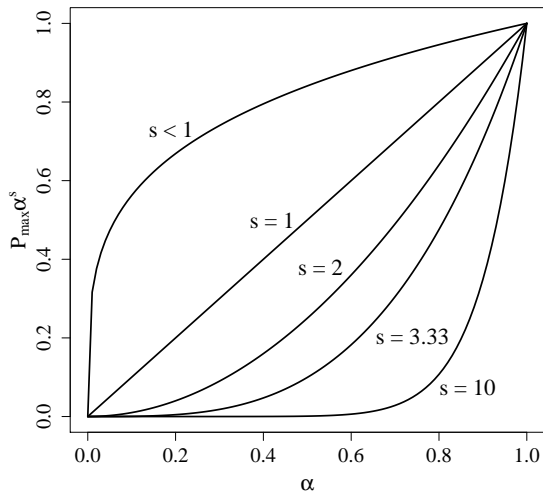


Fig. 1.— The relationship between activity level  $\alpha$  and predation rate for different values of  $s$ . See the text for the interpretation of  $s$ .

demonstrated increases in predation risk with increases in activity (Lima and Dill 1990; Werner and Anholt 1993; Werner and Peacor 2003); in our formulation, predation therefore depends upon the fraction of time spent foraging,  $\alpha(t)$ . Whereas it is clear that predation risk is an increasing function of activity level, the exact shape of the function relating activity level to predation rate is unknown. Previous theory has indicated that this shape is critical to determining the ecological consequences of behavioral defense (Abrams 1984, 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, but that introduces only one additional parameter.

What is an appropriate biological interpretation of the shape parameter  $s$ ? Often, specific shapes for the relationship between behavioral defense and predation rate are unspecified, focusing only on the curvatures (i.e., the value of the second derivative) (Abrams 1992). However, 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.

We assume that predation rate is zero when  $\alpha$  equals zero, and increases to a maximum  $P_{\max}$  when  $\alpha$  equals one, which yields the function  $P_{\max}\alpha^s$ . There are three basic shapes that can be produced, depending upon the value of  $s$  (Figure 1). If  $s = 1$ , the relationship between activity and predation risk is linear. This assumption is the default expectation (Gerritsen and Strickler 1977; Werner and Anholt 1993), and will hold whenever predators move at constant speed and demonstrate no preference. For this study, preference implies a behavioral response on the part of the predator to prey movement. If  $s > 1$ , the curve is convex, suggesting that predation rate increases at an increasing rate with activity level, i.e., the per capita predation rate increases with increases in activity level. This can be interpreted as indicating a behavioral preference on the part of predators for more active prey. Such preferential foraging has been demonstrated in a number of studies across a broad range of taxa (Snyder 1975; Furnass 1979; Wright and O' Brien 1982; Peterson and Ausubel 1984; Buskey et al. 1993; Sarno and Gubanich 1995; O'Keefe et al. 1998; Utne-Palm 1999, 2000). The biological mechanism behind this preference is likely due to predators cuing on prey movement. Utne-Palm (2000) showed that prey activity increased the reactive distance of foraging gobies (*Gobiusculus flavescens*). Furthermore, this study demonstrated that proportion of time swimming was a more important determinant of predation risk than either prey size or catchability. Figure 1 shows the different values of  $s$  that were investigated for this study. If  $s < 1$ , the curve is concave, and predation rate asymptotes at high activity levels. The affect of a concave relationship between activity level and mortality on the evolution of defenses was investigated, but the results were identical with the  $s = 1$  case, and so further discussion of the  $s < 1$  case will be omitted.

Based only on this relationship, we can infer cases where behavioral defenses and life history defenses are expected. As  $s \rightarrow \infty$ , predation risk drops so quickly with decreases in activity level that even a slight decrease will cause predation rate to drop to nearly 0. On the other hand, as  $s \rightarrow 0$ , predation rate is insensitive to changes in activity level. Thus, *a priori* we can predict that life history defenses will be observed for low

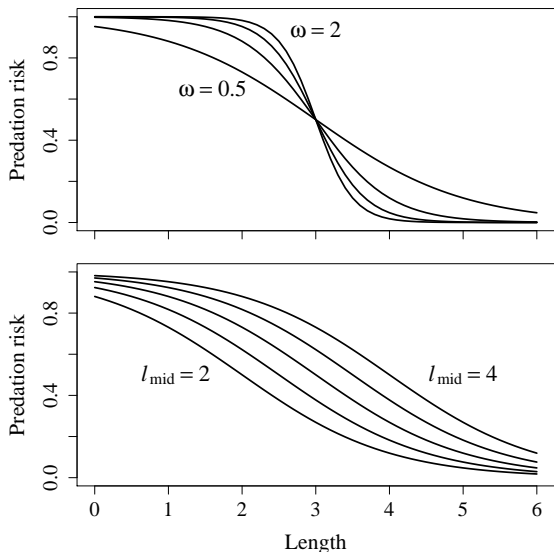


Fig. 2.— These graphs show how mortality changes with size, assuming different values for  $\omega$  and  $\ell_{\text{mid}}$ . For the simulations in the paper,  $\omega = 0.5$  and  $\ell_{\text{mid}} = 3$ .

$s$  values and behavioral defenses will be observed for high  $s$  values.

Predation rate is also dependent on size. In this study, predation risk is assumed to decrease with length, as happens when predator and prey are similar in size or predators are gape-constrained. This can create an effective size refuge against predation, whereby individuals can reduce predation risk by increasing size. This size refuge can cause individuals exposed to predators to grow to larger size than would be expected under predator-free conditions, but at the cost of increased maintenance demands.

A reasonable functional form for the relationship between size and predation rate is

$$P_{\text{max}}(1 - \tanh(\omega(\ell - \ell_{\text{mid}}))), \quad (5)$$

where  $P_{\text{max}}$  is the maximum predation rate (as above). The parameters  $\omega$  and  $\ell_{\text{mid}}$  characterize the size selectivity of the predator (Figure 2):  $\ell_{\text{mid}}$  gives the inflection point where predation rate is half its maximum and  $\omega$  is the slope of the predation rate-size curve.  $\ell_{\text{mid}}$  essentially characterizes the size preference of the predator, while  $\omega$  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. 2007).

Considering both the size- and behavioral-dependence of predation rate, along with a non-predation mortality term  $\mu$ , overall mortality rate is modeled as:

$$\frac{dp}{dt} = -(\mu + \alpha(t)^s (1 - \tanh(\omega(\ell - \ell_{\text{mid}}))) P_{\text{max}}) p. \quad (6)$$

## 2.6. Calculating the fitness of an individual

These three equations (eqs. 2, 3, and 5) can be used to determine the fitness of an individual. We used the fitness metric  $R_0$ , the net reproductive rate. It is calculated as

$$R_0 = \int_0^\infty \frac{db}{dt} p(t) dt. \quad (7)$$

which weights birth rate  $\frac{db}{dt}$  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).

## 2.7. Selection experiments

The responses to predation, activity level  $\alpha(t)$  and growth allocation  $\kappa(t)$ , determine the defense investment of an individual. Furthermore, the shapes of these functions will completely determine the fitness of an individual. Therefore, to optimize defense investment, one needs to determine the shapes of the functions that maximize individual fitness,  $R_0$ . However, the shapes of these functions are unknown, and could be quite complex. For example, growth allocation  $\kappa(t)$  could be equal to one up to some age, and then drop to zero. This would lead to determinate growth, where growth ceases as reproduction begins (Vincent and Pulliam 1980). Alternatively,  $\kappa(t)$  could be constant across an individual's lifetime, leading to indeterminate growth. Intermediate possibilities exist between these strategies. We used genetic algorithms to determine the optimal shapes for  $\alpha(t)$  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 the Appendix.

For any given environment, the output of the genetic algorithm was the activity level  $\alpha(t)$  and growth allocation  $\kappa(t)$  functions that maximized individual fitness. The shapes of the functions also determined defense investment, characterized by the average activity level ( $\hat{\alpha}$ ) across an individual’s lifespan (behavioral defense) and the size at maturity (life history defense). In this study, the maximum predation rate  $P_{\max}$  was varied between 0 and 1; an individual exhibits inducible defenses by a reduction in average activity level or an increase in size at maturity with changes in  $P_{\max}$ . Additionally, the value of  $s$  was varied between 1 and 10 to determine the effect of the shape of the foraging–predation risk tradeoff on the evolution of behavior and life history.

To determine how behavioral and life history defenses interact, three different selection scenarios were performed. In the first, both life history ( $\kappa(t)$ ) and behavior ( $\alpha(t)$ ) were under selection. In the second, only life history was under selection; behavior was assumed constant at the optimal behavior in the absence of predators,  $\alpha(t) = 1$ . In the third, only behavior was under selection; life history was fixed at the optimal life history in the absence of predators. This allows us to compare the optimal multi-defense strategy to the optimal single defense strategies to determine when multiple-defense strategies are favored, as well as to investigate the interaction between defenses in these cases. These experiments will be abbreviated as the LB selection experiment, the L selection experiment, and the B selection experiment, respectively.

### 3. Results

#### 3.1. Extreme tradeoffs favor single-defense strategies

As predicted based on the relationship between activity level and predation risk, at high values of  $s$ , only behavioral defenses are employed (Figure 3). This can be seen most clearly by comparing the average activity level  $\bar{\alpha}$  of the LB (black-and-white line) and B (gray line) strategies, seen in Figure 3C. The LB and B lines lay on top of one

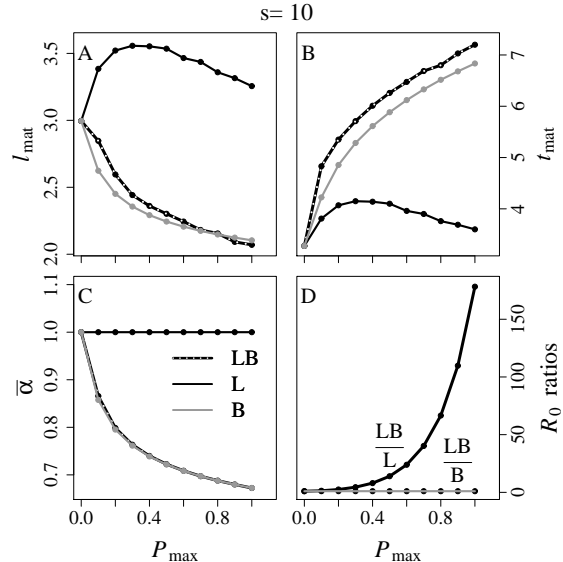


Fig. 3.— The optimal defense investments for  $s = 10$ . Panels A, B, and C plot the metrics for life history and behavioral defenses under each of the three selection scenarios. Defenses are indicated by an increase in size at maturity or by a decrease in average activity level  $\hat{\alpha}$  as  $P_{\max}$  increases. Panel D plots the relative fitness advantage of the multi-defense strategy over either pure defense strategy as the ratio of LB fitness to both the L and B fitness.

another, indicating that the multi-defense strategy closely follows the behavior-only strategy. However, the LB strategy tends to mature at a slightly larger size and slightly delayed from the B strategy (Figure 3A, B). This occurs because the optimal response  $\kappa(t)$  for the LB strategy prolongs allocation to growth. This partially compensates for the reduction in maturation size that results from decreased foraging gain.

At the low values of  $s = 1$ , only life history defenses are observed (Figure 4). Here the size at maturity of the LB strategy exactly matches that of the L strategy. Despite the fact that the relationship between activity and predation risk is linear (and not concave down), the optimal strategy is to forego the behavioral defense and “sprint” for the size refuge from predation.

The more interesting cases occur for intermediate values of  $s$ .

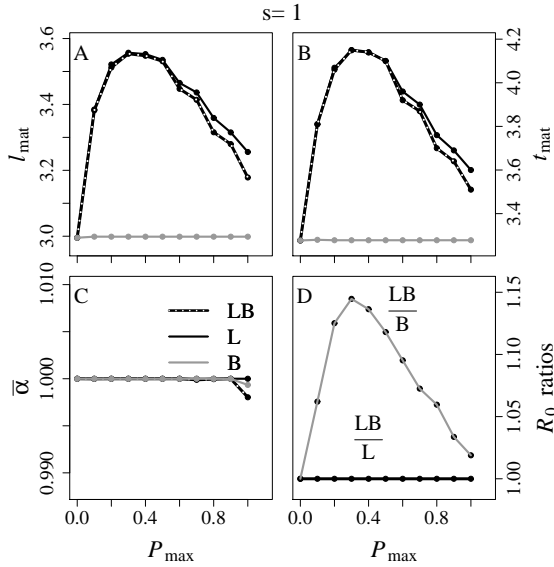


Fig. 4.— Optimal defense investments for  $s = 1$ . See the Figure 3 caption for more details.

### 3.2. Moderately nonlinear tradeoffs favor integrated multi-defense strategies

For  $s = 3.33$ , there is investment in both defenses at low  $P_{\max}$  values, as can be seen in Figure 5. For value of  $P_{\max} < 0.6$ , size at maturity has increased and average activity level  $\bar{\alpha}$  is reduced from the predator-free levels, indicating investment in both life history and behavioral defenses. However, as  $P_{\max}$  increases, the optimal strategy becomes investment only in behavioral defenses. This is due to the constraint on optimal life history defenses noted above - as predation rate increases, the optimal L strategy invests less and less in life history defense, whereas the optimal B strategy invests more and more in behavioral defense.

Furthermore, an important interaction between the different responses at low to intermediate  $P_{\max}$  values can be seen. At these values, the LB activity level  $\alpha$  is less than the B  $\alpha$  early in life, indicating that the LB strategy has increased expression of behavioral defenses early in ontogeny. Later in life, however, the LB curve is greater than the B curve, suggesting that behavioral defenses are relatively underexpressed. This suggests a pattern of compensation between the responses through ontogeny. By investing heavily

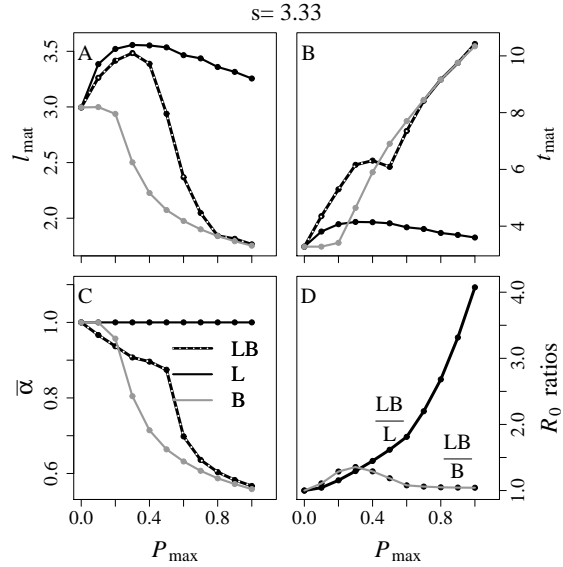


Fig. 5.— Optimal defense investments for  $s = 3.33$ . See the Figure 3 caption for more details.

in behavioral defenses early in life, survivorship is high. Once sexual maturity is reached, because the individual is large and can take advantage of the size refuge, it is able to stop investing in the behavioral defense without adversely affecting fitness.

A similar pattern of multi-defense expression is observed for  $s = 2$ , though it is even more exaggerated. When  $s = 3.33$ , the life history defense was not used at predator densities above 0.6, whereas now both life history and behavioral defenses are used at all but the highest predator density (see Figure 6A,C). Furthermore, these results reveal another important interaction between life history and behavior: the optimal B strategy does not reduce activity level, especially compared to the LB strategy. That the optimal B strategy would not invest in behavioral defense is counterintuitive, but can be explained by a careful examination of the patterns of growth, reproduction, and survivorship for the LB and B strategies. An evident cost of the behavioral defense is maturation at a reduced size. In order for the B strategy to employ behavioral defenses, the benefit in reduced mortality must compensate for this cost. However, the multi-defense strategy is able to compensate for this cost by modifying the life history response of

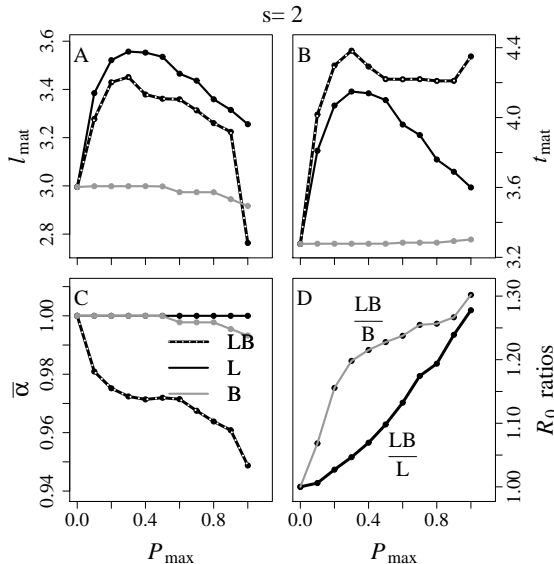


Fig. 6.— Optimal defense investments for  $s = 2$ . See the Figure 3 caption for more details.

allocation to growth.  $\kappa(t)$  to compensate for the decrease in size at maturity caused by reducing activity level.

#### 4. Discussion

Our results illustrate a number of important points regarding the optimal expression of inducible defenses. In particular, we predict that the shape of the foraging–predation risk tradeoff determines the optimal defense strategy. The fact that defense expression depends on the shape of this tradeoff has important implications for ecological research and interpretation of current theory on trait-mediated indirect interactions. Additionally, we predict that integrated multi-defense strategies will show a characteristic interaction between the responses to predation, (i.e., growth allocation,  $\kappa(t)$  and activity level,  $\alpha(t)$ ), revealing the importance of the interaction between the foraging–predation risk and growth–reproduction tradeoffs.

##### 4.1. The role of the foraging–predation risk tradeoff in determining optimal investment in the different defenses

The above results show that the shape of the foraging–predation risk tradeoff mediated by ac-

tivity level determines whether single or multiple defense investment by prey is optimal. At very high values of  $s$ , the relationship between activity level and predation rate is highly non-linear, so that predation rate decreases rapidly with small decreases in activity level, whereas foraging gain decreases linearly. This non-linearity in the foraging–predation risk tradeoff (predation risk is more sensitive to changes in  $\alpha$  than foraging gain) favors the evolution of behavioral defenses. Given our interpretation of  $s$  as the preference of the predator for more active prey, this model predicts that behavioral defenses would be more common in response to actively foraging predators.

Many species are known to exhibit behavioral defenses against negative size specific predation risk from active predators, including amphibians (Anholt et al. 2000; Urban 2007a), *Daphnia* (Pangle and Peacor 2006), fish (Abrahams and Healey 1993), and snails (Turner et al. 1999; Hoverman et al. 2005). Given the prevalence of behavioral defenses in nature (Lima and Dill 1990; Werner and Peacor 2003), our results suggest that non-linearity in the relationship between predation risk and activity may be the rule, rather than the exception. The results further highlight the importance of non-linearity in ecological systems and the need for direct empirical studies investigating the shapes of the relationships between dynamically variable traits and their ecological catalysts (Abrams 1995, 2001).

Looking across all environments ( $s$ - $P_{\max}$  combinations) there is a strong negative correlation between defenses, as investment in behavioral defenses tends to strongly reduce size at maturity. However, for intermediate  $s$  values, the defense expression pattern that emerges employs the behavioral defenses early in life when predation risk is highest and then reduces investment in behavioral defense as size increases and the size refuge from predation is approached. These compensatory dynamics between the responses to predation produce an integrated defense strategy and demonstrate the importance of the different timescales over which different defenses operate.

Behavioral defenses are effective immediately, and can be modified quickly and reversibly. Life history defenses, on the other hand, are only effective once a threshold of energy investment has been made. Additionally, they are not reversible -

individuals are not capable of shrinking. This difference in timescale affects the interaction between the two responses, and highlights the importance of considering defense investment across an individual’s lifetime, rather than performing a point optimization. Nearly all previous theoretical work on defense investments, especially work addressing the interaction between defenses (e.g., Steiner and Pfeiffer (2007)) has focused on the single optimal values of activity level  $\alpha$  and growth allocation  $\kappa$  and studied the interaction between these two points. However, this focus on point values will overlook compensatory dynamics that depend upon development. 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). Indeed, recent empirical work has shown a similar pattern of behavioral defense expression early in ontogeny, with expression of alternative defenses (either morphological or life history) late in ontogeny (Pettersson et al. 2000; Relyea 2003; Brodin et al. 2006).

Additionally, the selective advantage of multiple defense strategies over either single defense strategy is highest for these values of  $s$ . This suggests that trait integration should be more common in intermediate environments. This result corroborates the finding of Steiner and Pfeiffer (2007), who also showed that the selective advantage of a multiple defense strategy was highest in intermediate environments. If it is a general result that selection for multiple defense strategies is highest in intermediate environments, these results suggest that care must be taken in interpreting the results of trait expression experiments that use only two environments (a predator-free control and one level of predation).

At low values of  $s$ , only the life history defense is selected for because the foraging–predation risk tradeoff mediated by activity level is linear, or nearly so; i.e., changes in activity level have the same proportional affect on foraging gain and mortality risk. The optimal strategy is to forgo the behavioral defense in order to reach the size refuge as quickly as possible. Thus, size-selective predation combined with a predator with little to no preference for more active prey, e.g., a sit-and-wait predator, should select for life history defenses.

This pattern holds in nature, as the response of prey with flexible life histories to passive predators with negative size-dependent predation is often later maturation at larger size (Tollrian and Dodson 1999; Chase 1999b). For example, *Daphnia* are commonly preyed upon by the aquatic larval stage of the midge *Chaoborus*, a gape-limited sit-and-wait predator that detects prey from the wake generated by movement through the water column. Studies have shown that predation risk declines with increasing size (Pastorok 1981), and a common phenotypic response to *Chaoborus* is increased size at maturity (Riessen 1999).

The prediction that negative size-dependent predation should lead to life history defenses matches earlier models (Taylor and Gabriel 1992; Day et al. 2002). However, these models did not consider behavioral defenses; our results demonstrate the important interaction between size-dependent and activity-dependent predation risk. A model developed by Urban (2007a) also considers the interaction between behavior and life history and similarly showed that optimal prey defense could be to forgo a behavioral defense in favoring 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, analogous to our results. One caveat is that the model of Urban (2007a) was developed for organisms with complex life cycles and modeled only growth to a metamorphic event, with fitness assumed proportional to size at metamorphosis. This is a common assumption in models of complex life cycles that has empirical support (Benard 2004). However, this assumption creates an added benefit of increased size, which may increase the likelihood that life history defenses are optimal. Nevertheless, this result, and the empirical data motivating it (Urban 2007b) are additional evidence for the importance of the foraging–predation risk tradeoff in determining defense expression in nature.

#### 4.2. Implications for ecological dynamics

An extensive body of literature has investigated the importance of the foraging–predation risk tradeoff for ecological interactions (e.g., Abrams, 1992). This work has largely examined at how the shape of the tradeoff affects optimal behavior of prey, and how prey behavior then feeds

back to influence community structure and dynamics, through both direct and indirect effects (Werner and Peacor 2003; Bolker et al. 2003). A largely independent body of literature has looked at the consequences of life history plasticity for ecological dynamics (Chase 1999a,b). Our results illustrate how the foraging–predation risk tradeoff links these two bodies of literature. In particular, investigations of the consequences of the foraging–predation risk tradeoff that do not take defense expression into consideration may generate incorrect predictions. For example, investigating how a linear tradeoff affects behavior and how this behavioral modification affects ecological dynamics is misleading. Our results show that a linear tradeoff will not affect behavior at all, but rather will modify life history. This modification of life history may have very different ecological consequences. Chase (1999a) has shown that life history modification can alter a system from consumer-dominated to resource-dominated. Additionally, preliminary investigation of physiologically-structured population models (de Roos 1997) developed from the preceding physiological equations, allowing resources to be dynamic, predict that highly nonlinear tradeoffs (and corresponding optimal behavioral defense strategy) lead to cyclic dynamics, whereas linear tradeoffs lead to equilibrium dynamics (C. E. Cressler, *unpublished data*). Future research in this field needs to carefully establish how different tradeoffs affect both defense expression and ecological dynamics simultaneously. This interaction will be especially important for systems with predators having different  $s$  values, or systems in which the  $s$  values change through time, either stochastically or dynamically.

## 5. Conclusions

Trait expression is an area of research that has received renewed interest recently (Abrams 2001). Because of the important effects of dynamic traits on ecological interactions (Abrams 1995; Chase 1999a; Werner and Peacor 2003; Peacor and Werner 2004), understanding how fundamental physiological and ecological tradeoffs interact to influence trait expression becomes crucially important for understanding ecological communities. However, this understanding requires that empiricists and theoreticians move beyond think-

ing 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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## 6. Appendix: Genetic algorithm details

Genetic algorithms are a computational tool useful for performing optimization (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 for selection to act upon. 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, once 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 modelling 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*:

$$\mathbf{T} = t_0, t_1, \dots, t_m$$

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,  $\mathbf{P}_0, \mathbf{P}_1, \dots, \mathbf{P}_p$ , 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 only take values between 0 and 1. The number of control points specified depends upon the length of the knot vector and the degree of the B-spline, according to the relation  $p = m - n - 1$ . The "wiggleness" 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:

$$\begin{aligned} B_{i,0}(t) &= \text{1 if } t_i \leq t < t_{i+1} \text{ and } t_i < t_{i+1} \\ &= 0 \text{ otherwise} \end{aligned} \quad (8)$$

$$B_{i,n}(t) = \frac{t - t_i}{t_{i+n} - t_i} N_{i,p-1}(t) + \frac{t_{i+n+1} - t}{t_{i+p+1} - t_{i+1}} N_{i+1,p-1}(t) \quad (9)$$

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

$$\mathbf{B}(t) = \sum_{i=0}^p \mathbf{P}_i B_{i,p}(t) \quad (10)$$

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:

1. Upon 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."
2. The energetics equations are solved for each individual and a fitness is assigned to each individual.
3. Select parents to produce the next generation of solutions (described below)
4. Crossover occurs between the chromosomes of the two parents.
5. Mutation occurs with a fixed probability for each gene in the chromosome.
6. Return to step two for the next generation. This process is repeated for 1000 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 less than 0.005 for all runs, and was typically less than 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 weak 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

$$\begin{aligned} \mathbf{E}(R_0(i)) &= 1 + \frac{R_0(i) - \bar{R}_0}{2 * \sigma} \text{ for } \sigma \neq 0 \\ &= 1 \text{ for } \sigma = 0 \end{aligned} \quad (11)$$

where  $\bar{R}_0$  is the average fitness of all individuals in the population, and  $\sigma$  is the standard deviation of fitness. 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  $\sigma$  is large, the most fit individuals are not allocated the majority of the reproductive events. When  $\sigma$  is small late in the run, individuals with higher than average fitness stand out more, allowing evolution to continue.

Once 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  $\mathbf{E}(R_0(i)) + 1$  times, and no fewer than  $\mathbf{E}(R_0(i))$  times. Once 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.

This entire process is repeated for 1000 generations to determine the optimal solution. For each parameter combination, the algorithm was run 10 times. The results presented in the text show the  $\kappa(t)$  and  $\alpha(t)$  functions generated by av-

eraging across all individuals and across the replicate runs (800 individuals x 10 runs).

## REFERENCES

- Abrahams, M. and M. Healey, 1993. A comparison of the willingness of four species of pacific salmon to risk exposure to a predator. *Oikos* **66**:439–446.
- Abrams, P. A., 1984. Foraging time optimization and interactions in food webs. *American Naturalist* **124**:80–96.
- Abrams, P. A., 1992. Predators that benefit prey and prey that harm predators - unusual effects of interacting foraging adaptations. *American Naturalist* **140**:573–600.
- Abrams, P. A., 1995. Implications of dynamically variable traits for identifying, classifying, and measuring direct and indirect effects in ecological communities. *American Naturalist* **146**:112–134.
- Abrams, P. A., 2001. Describing and quantifying interspecific interactions: a commentary on recent approaches. *Oikos* **94**:209–218.
- Anholt, B. R., E. E. Werner, and D. K. Skelly, 2000. Effect of food and predators on the activity of four larval ranid frogs. *Ecology* **81**:3509–3521.
- Benard, M. F., 2004. Predator-induced phenotypic plasticity in organisms with complex life histories. *Annual Review of Ecology Evolution and Systematics* **35**:651–673.
- Benton, T. G. and A. Grant, 2000. Evolutionary fitness in ecology: Comparing measures of fitness in stochastic, density-dependent environments. *Evolutionary Ecology Research* **2**:769–789.
- Boeing, W. J., C. W. Ramcharan, and H. P. Riessen, 2006. Multiple predator defence strategies in daphnia pulex and their relation to native habitat. *Journal of Plankton Research* **28**:571–584.
- Bolker, B., M. Holyoak, V. Krivan, L. Rowe, and O. Schmitz, 2003. Connecting theoretical and empirical studies of trait-mediated interactions. *Ecology* **84**:1101–1114.

- Brodin, T., D. J. Mikolajewski, and F. Johansson, 2006. Behavioural and life history effects of predator diet cues during ontogeny in damselfly larvae. *Oecologia* **148**:162–169.
- Buskey, E. J., C. Coulter, and S. Strom, 1993. Locomotory patterns of microzooplankton - potential effects on food selectivity of larval fish. *Bulletin Of Marine Science* **53**:29–43.
- Chase, J. M., 1999a. Food web effects of prey size refugia: Variable interactions and alternative stable equilibria. *American Naturalist* **154**:559–570.
- Chase, J. M., 1999b. To grow or to reproduce? the role of life-history plasticity in food web dynamics. *American Naturalist* **154**:571–586.
- Day, T., P. A. Abrams, and J. M. Chase, 2002. The role of size-specific predation in the evolution and diversification of prey life histories. *Evolution* **56**:877–887.
- De Boor, C., 1978. A Practical Guide to Splines. Springer-Verlag.
- de Roos, A. M., 1997. A gentle introduction to physiologically structured population models. In S. Tuljapurkar and H. Caswell, editors, *Structured Population Models in Marine, Terrestrial and Freshwater Systems*, pages 119–204. Chapman and Hall, New York.
- de Roos, A. M., O. Dieckmann, and J. A. J. Metz, 1992. Studying the dynamics of structured population-models a versatile technique and its application to daphnia. *American Naturalist* **139**:123–147.
- de Roos, A. M., J. A. J. Metz, E. Evers, and A. Leipoldt, 1990. A size dependent predator-prey interaction - who pursues whom? *Journal Of Mathematical Biology* **28**:609–643.
- Ernande, B., U. Dieckmann, and M. Heino, 2004. Adaptive changes in harvested populations: plasticity and evolution of age and size at maturation. *Proceedings of the Royal Society of London Series B-Biological Sciences* **271**:415–423.
- Furnass, T. I., 1979. Laboratory experiments on prey selection by perch fry (*perca fluviatilis*). *Freshwater Biology* **9**:33–43.
- Gardmark, A. and U. Dieckmann, 2006. Disparate maturation adaptations to size-dependent mortality. *Proceedings of the Royal Society of London Series B-Biological Sciences* **273**:2185–2192.
- Gerritsen, J. and J. R. Strickler, 1977. Encounter probabilities and community structure in zooplankton: A mathematical model. *Journal of the Fisheries Research Board of Canada* **34**:73–82.
- Holland, J. H., 1975. Adaptation in Natural and Artificial Systems. University of Michigan Press.
- Hoverman, J. T., J. R. Auld, and R. A. Relyea, 2005. Putting prey back together again: integrating predator-induced behavior, morphology, and life history. *Oecologia* **144**:481–491.
- Kooijman, S. A. L. M., 2000. Dynamic Energy and Mass Budgets in Biological Systems. Cambridge University Press.
- Lima, S. L. and L. M. Dill, 1990. Behavioral decisions made under the risk of predation - a review and prospectus. *Canadian Journal Of Zoology-Revue Canadienne De Zoologie* **68**:619–640.
- Mitchell, M., 1998. An Introduction to Genetic Algorithms. MIT Press.
- Mitchell, M. and C. E. Taylor, 1999. Evolutionary computation: An overview. *Annual Review of Ecology and Systematics* **30**:593–616.
- Noonburg, E. G. and R. M. Nisbet, 2005. Behavioural and physiological responses to food availability and predation risk. *Evolutionary Ecology Research* **7**:89–104.
- O’Keefe, T. C., M. C. Brewer, and S. I. Dodson, 1998. Swimming behavior of daphnia: its role in determining predation risk. *Journal of Plankton Research* **20**:973–984.
- Pangle, K. L. and S. D. Peacor, 2006. Non-lethal effect of the invasive predator bythotrephes longimanus on daphnia mendotae. *Freshwater Biology* **51**:1070–1078.

- Pastorok, R. A., 1981. Prey vulnerability and size selection by chaoborus larvae. *Ecology* **62**:1311–1324.
- Peacor, S. D. and E. E. Werner, 2004. Context dependence of nonlethal effects of a predator on prey growth. *Israel Journal of Zoology* **50**:139–167.
- Peterson, W. T. and S. J. Ausubel, 1984. Diets and selective feeding by larvae of atlantic mackerel scomber-scombrus on zooplankton. *Marine Ecology-Progress Series* **17**:65–75.
- Pettersson, L. B., P. A. Nilsson, and C. Bronmark, 2000. Predator recognition and defence strategies in crucian carp, carassius carassius. *Oikos* **88**:200–212.
- Relyea, R. A., 2003. Predators come and predators go: the reversibility of predator-induced traits. *Ecology* **84**:1840–1848.
- Relyea, R. A., 2004. Integrating phenotypic plasticity when death is on the line. In M. Pigliucci and K. Preston, editors, *Phenotypic Integration: Studying the Ecology and Evolution of Complex Phenotypes*. Oxford University Press.
- Riessen, H. P., 1999. Predator-induced life history shifts in daphnia: A synthesis of studies using meta-analysis. *Canadian Journal of Fisheries and Aquatic Sciences* **56**:2487–2494.
- Rinke, K., S. Hulsmann, and W. M. Mooij, 2007. Energetic costs, underlying resource allocation patterns, and adaptive value of predator-induced life history shifts. *Oikos* **117**:273–285.
- Sarno, R. J. and A. A. Gubanich, 1995. Prey selection by wild american kestrels - the influence of prey size and activity. *Journal Of Raptor Research* **29**:123–126.
- Shertzer, K. W. and S. P. Ellner, 2002. State-dependent energy allocation in variable environments: Life history evolution of a rotifer. *Ecology* **83**:2181–2193.
- Snyder, R. L., 1975. Some prey preference factors for a red-tailed hawk. *Auk* **92**:547–552.
- Steiner, U. K. and T. Pfeiffer, 2007. Optimizing time and resource allocation trade-offs for investment into morphological and behavioral defense. *American Naturalist* **169**:118–129.
- Strand, E., G. Huse, and J. Giske, 2002. Artificial evolution of life history and behavior. *American Naturalist* **159**:624–644.
- Taylor, B. E. and W. Gabriel, 1992. To grow or not to grow: Optimal resource-allocation for daphnia. *American Naturalist* **139**:248–266.
- Tollrian, R. and S. I. Dodson, 1999. Inducible defenses in cladocera: Constraint, costs, and multipredator environments. In R. Tollrian and C. Harvell, editors, *The Ecology and Evolution of Inducible Defenses*, pages 177–202. Princeton University Press.
- Turner, A., S. Fetterolf, and R. Bernot, 1999. Predator identity and consumer behavior: differential effects of fish and crayfish on the habitat use of a freshwater snail. *Oecologia* **118**:242–247.
- Urban, M., 2007a. The growth-predation risk trade-off under a growing gape-limited predation threat. *Ecology* **88**:2587–2597.
- Urban, M. C., 2007b. Risky prey behavior evolves in risky habitats. *Proceedings Of The National Academy Of Sciences Of The United States Of America* **104**:14377–14382.
- Utne-Palm, A. C., 1999. The effect of prey mobility, prey contrast, turbidity and spectral composition on the reaction distance of gobiusculus flavescens to its planktonic prey. *Journal Of Fish Biology* **54**:1244–1258.
- Utne-Palm, A. C., 2000. Prey visibility, activity, size and catchability's (evasiveness) influence on gobiusculus flavescens prey choice. *Sarsia* **85**:157–165.
- Vincent, T. L. and H. R. Pulliam, 1980. Evolution of life-history strategies for an asexual annual plant model. *Theoretical Population Biology* **17**:215–231.
- Werner, E. E. and B. R. Anholt, 1993. Ecological consequences of the trade-off between growth and mortality-rates mediated by foraging activity. *American Naturalist* **142**:242–272.
- Werner, E. E. and S. D. Peacor, 2003. A review of trait-mediated indirect interactions in ecological communities. *Ecology* **84**:1083–1100.

Wright, D. I. and W. J. O' Brien, 1982. Differential location of chaoborus larvae and daphnia by fish - the importance of motion and visible size. *American Midland Naturalist* **108**:68-73.