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CONTEXT DEPENDENCE OF NONLETHAL EFFECTS OF A PREDATOR ON PREY GROWTH

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ABSTRACT

Predators can have a large influence on their prey through induced changes in prey phenotype. Such "nonlethal" predator effects have been abundantly demonstrated empirically in both terrestrial and aquatic systems. But the extent to which changes in species traits alter short-term responses such as growth rate or probability of survival is not clear. Here we develop models to examine the nonlethal effects of predators on prey growth. Our analyses illustrate how the nonlethal effects of predators on individual prey growth depend on environmental context; e.g., factors such as focal species density, competitor density, resource dynamics, and the timescale over which the interactions occur. This context dependence arises because of complex interactions of three mechanisms; (1) the direct negative effect of induced reduction in foraging rates, which is opposed by (2) the potential positive effects of reductions in intra- and interspecific competition, and (3) resource responses to reduced foraging. We present new empirical work, and review previous work, on larval-anuran growth that is in general support of model predictions. The framework presented here can serve to facilitate the design and interpretation of experimental results and predict how the nonlethal predator effect on prey growth in natural systems will vary over time and space.

INTRODUCTION

There is a large empirical literature demonstrating the existence of nonlethal predator effects in a diversity of ecological systems. The presence of a predator can induce large phenotypic modifications in prey (reviewed in Lima and Dill, 1990; Kats and Dill, 1998; Lima, 1998; Tollrian and Harvell, 1999) that influence the growth and survival rate of the reacting prey (e.g., Werner et al., 1983; Huang and Sih, 1990; Relyea, 2000; McPeek et al., 2001; Peckarsky et al., 2001) and, indirectly, other species that interact with the prey (reviewed in Abrams et al., 1996; Werner and Peacor, 2003). These "nonlethal" or

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"trait-mediated" effects (Abrams et al., 1996) can dramatically affect species population dynamics and the emergent properties of ecological communities (reviewed in Bolker et al., 2003). The potential implications of such effects on population and community dynamics underscore the importance of performing empirical studies that accurately quantify their magnitude and influence.

Recent studies, however, have indicated mixed responses of prey to the nonlethal presence of predators. These responses can range from negative to positive effects of the predator on individual prey growth. These responses suggest that the environmental context in which the focal predator–prey system is embedded may influence the magnitude and direction of the response of prey to presence of predators. For example, the nonlethal effect of predators on the growth rate of anuran larvae has been shown to depend on densities of these anuran larvae and their competitors (Huang and Sih, 1990; Werner and Anholt, 1996; Van Buskirk and Yuriwitz, 1998; Peacor and Werner, 2000; Relyea, 2000). The mechanisms underlying this context dependence likely are related to the fact that predator-induced changes in prey traits are indirectly reflected back through other species, including resources, to affect the growth rate of the reacting prey, and the degree to which this occurs is dependent on the context of the predator–prey interaction examined.

Such context dependence makes it difficult to interpret empirical measurements of nonlethal predator effects on prey growth rate, or the resulting trait-mediated effects on other species. Experiments performed in the laboratory or in mesocosms generally exclude much of the natural context of predator–prey interactions, and thus can strongly underestimate or overestimate the importance of nonlethal predator effects in the field. In addition, the environmental context of predator–prey interactions likely varies temporally and spatially in the field. Therefore, even results of field experiments or comparisons of nonlethal effects across communities or gradients may be an inaccurate or misleading assessment of the magnitude of nonlethal predator effects unless combined with an assessment of environmental context.

In this paper, we examine how phenotypic responses of prey to predators affect individual prey growth rates. We examine the changes in prey growth due to both direct effects of trait-changes on prey attack rates, and the indirect effects through resources and interspecific competitors. We present a model that isolates these different factors and makes predictions concerning the effect of focal species and competitor densities, factors affecting resource growth rate, and experimental duration, on prey growth responses. We test these model predictions with new and published empirical studies of larval anuran–predator interactions.

A MODEL OF NONLETHAL PREDATOR EFFECTS ON CONSUMER GROWTH

In this section we extend a previously introduced model (Peacor and Werner, 2000; Peacor, 2002) to illustrate how predator-induced changes in the phenotype of a prey species affect its foraging and growth rate. The purpose of the model is to clarify the multiple mechanisms that combine to affect prey growth rate, and the manner in which

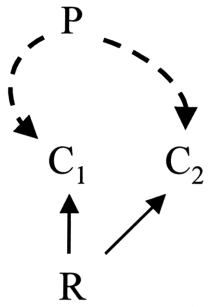

Fig. 1. The model food web consisting of a predator (P), a focal prey species (C_1), a competitor species (C_2), and resources (R). Dotted arrows represent predator-induced phenotypic responses that cause a reduction in foraging rates, while solid arrows represent consumption and point in the direction of energy flow. Nonlethal predator effects on C_1 are examined in the absence of predation on C_1 and C_2.

they interact. In order to isolate and to examine these mechanisms, we do not include effects of the predator on prey density in the model. Thus, the model characterizes the nonlethal (i.e., trait-mediated) effect of a predator in a simple food web (Fig. 1) as manipulated in many empirical investigations (i.e., where predators are present and induce changes in prey traits, but are manipulated in a way that blocks predation, see, e.g., Soluk and Collins, 1988; Werner, 1991; Wissinger and McGrady, 1993; McIntosh and Townsend, 1996; Beckerman et al., 1997; Abramsky et al., 1998). We discuss how these effects interact with the lethal effects of the predator in the Discussion.

NONLETHAL EFFECT OF A PREDATOR ON A SINGLE PREY SPECIES

First consider the effect of a predator on individual growth rates of a single prey species. The presence of a predator will *directly* affect prey growth rate through its effect on the latter's traits, many of which influence resource acquisition rates. For example, when an individual forager modifies traits such as activity level to decrease predation risk, this response will often decrease its ability to acquire resources (Werner and Anholt, 1993). We can represent this effect with the equation

$$a = a'(1 - \Delta) \tag{1}$$

where a and a' are the prey's attack rates on its resource in the presence and absence of the predator, respectively, and Δ is a dimensionless parameter representing the relative negative effect of predator presence on this attack rate. Phenotypic responses to predators potentially are a function of the predator and other species densities, but for purposes of this analysis we treat Δ as a constant. Most experimental assays of the nonlethal effects of predators on growth rates of prey tacitly assume this scenario. We assume that growth rate is proportional to attack rate and resource level. Growth rate in

predator presence is then

$$g = baR = ba'(1 - \Delta)R \tag{2a}$$

and in predator absence is

$$g' = b'a'R' \tag{2b}$$

The coefficient b is the conversion efficiency of acquired resources into prey mass, and R is resource level. It is possible that predator presence will affect b (Loose and Dawidowicz, 1994; McPeek et al., 2001), but here we assume effects on b are negligible ($b' = b$) in order to focus on the effects of trait changes on the prey–resource interaction (i.e., the attack rate). It is a simple matter, however, to modify the following analysis to include changes in b if they are important.

Constant resource level

Consider first the effect of a predator on prey growth rate in a system in which resource level is constant. The effect of this reduction on growth rate (resulting from eq 2) is illustrated in Fig. 2a, in which the intersection of the resource acquisition rate (i.e., the rate resources are removed from the system) and the resource renewal rate

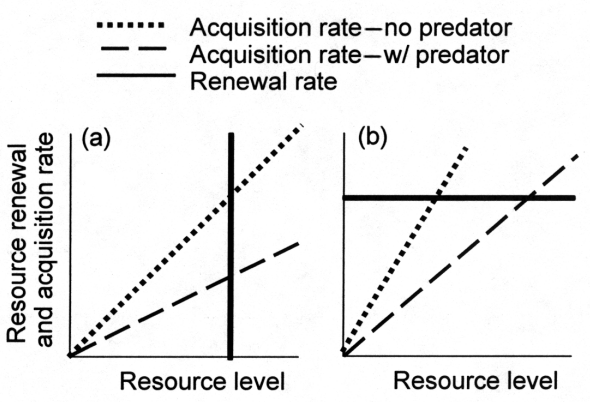

Fig. 2. Effect of a 50% predator-induced reduction in prey attack rate ($\Delta = 0.5$) on the resource acquisition rate of prey in a system with (a) constant resource level and (b) constant resource renewal. Dotted and dashed lines represent the resource acquisition rate (i.e., removal rate) as a function of resource level in the presence (dashed lines) and absence (dotted lines) of the nonlethal predator effect on attack rate. The solid line represents the resource renewal rate. The intersection of the resource acquisition rate and renewal rate determines the rate at which resources are being acquired by the prey at steady state, which is proportional to the prey growth rate. The predator causes a reduction in growth rate that is (a) proportional to the reduction in the attack rate if resources are constant, but (b) nonexistent if resources are renewed at a constant (resource-independent) rate.

determines the rate at which the consumers acquire the resources at steady state. In a system with a constant resource level, the reduction in this steady state level is proportional to the reduction in the attack rate (1-Δ), which is in turn proportional to the consumer growth rate. Therefore, predator presence will reduce growth rate in proportion to the reduction in attack rate,

$$g = g'(1 - \Delta) \tag{3}$$

Constant resource renewal

Next consider the effect of a predator on prey growth rate in a system in which resources are renewed at a constant rate, J, independent of resource density. Then, the resource growth rate is

$$\frac{dR}{dt} = J - aNR \tag{4}$$

where N is the density of the prey, and therefore aN is the net attack rate of the prey population. Hereafter we denote the positive term of resource growth rate the resource renewal rate (J in eq 4), and the negative term due to consumption by prey the resource acquisition rate (aNR in eq 4). We assume that resource renewal is fast relative to the resource acquisition rate and therefore resource density at steady state (i.e., $dR/dt = 0$) is

$$R = \frac{J}{aN} \tag{5}$$

Therefore the growth rate, g, of a prey individual is

$$g = \frac{Jba}{aN} = \frac{Jb}{N} \tag{6}$$

Equation 6 indicates that there is no net nonlethal effect of the predator on prey growth rate,

$$g = g' \tag{7}$$

because growth is independent of the attack rate.

Two opposing mechanisms underlie the prediction of zero net effect of the predator on prey growth; the negative direct effect of reduced attack rates is precisely counterbalanced by an indirect positive effect on resource level (due to the predator-induced reduction in intraspecific competition). This result is illustrated in Fig. 2b, in which a 50% reduction in attack rate is associated with a doubling of the resource level, leading to a zero net nonlethal effect of the predator on individual prey growth rate. In this example there is a reciprocal relationship between a decrease in the attack rate and an increase in the resource level that will hold if the functional response is expressed as a

product of the attack rate and resource level (i.e., f(aR) as in the Holling Type I [used here] and Holling Type II functional responses). Note, however, that the two opposing mechanisms are predicted to counterbalance each other even in the absence of this reciprocal relationship, as, for example, in the Type III functional response (Real, 1979) where the attack rate increases as a function of resource level.

A second way of describing this prediction is to consider the amount of resources passing through the system and how these resources are divided among individual prey. At steady state, by definition, a system with constant (density-independent) resource renewal rate will have the same input and output (through uptake by prey) of resources, i.e., the predator will not affect the amount of resources consumed by the prey population. Thus, if the predator does not affect the *relative* rates that individuals are foraging, and the net amount of resources acquired by all consumers is unaffected, then a focal individual captures the same amount of resource per unit time, and grows at the same rate. In this case, an individual will forage at a slower rate in the presence of the predator, but at the same time, the ability of intraspecific competitors to capture the resources also will be reduced by the same amount.

These two explanations for the constant growth rate in the presence and absence of the predator are equivalent, but use different variables to describe the opposing predator effects. In the first, an individual's ability to capture resources is counterbalanced by an increase in absolute resource level. In the second, the flow of resource through a system (i.e., the net amount of resources available for consumption) is unaffected by predator presence, and the predator-induced reduction in an individual's resource attack rate is counterbalanced by a reduction in attack rate of competitors. Below we examine more complex scenarios, and will use this latter framework to describe the results as we believe it more clearly elucidates the context dependence of the nonlethal effect of the predator.

Convex resource renewal

Next consider a more complex case in which resource renewal is not constant. Generally, resource renewal rate is related to resource standing crop, and is convex with a single maximum (Harper, 1977; Watkinson, 1980). This convex shape arises because an increase in R at low R leads to an increase in growth potential, while an increase in R at high R leads to increased self-limitation. The logistic equation is commonly used to describe this relationship, and thus the equation for resource growth rate becomes

$$\frac{dR}{dt} = rR\left(1 - \frac{R}{K}\right) - aNR \tag{8}$$

K is the resource carrying capacity, and r is the per capita intrinsic resource growth rate at low density in the absence of self-limitation. We again assume that a steady state is reached (i.e., $dR/dt = 0$). The growth rate of an individual prey when resources are at steady state is then

$$g = baK\left(1 - \frac{a}{r}N\right) \tag{9}$$

In this case, the nonlethal effect of the predator indirectly affects resource renewal rates, the magnitude of which strongly depends on the net attack rate of the prey population (i.e., on the product of density and individual attack rate). Therefore, the net nonlethal effect of the predator on prey growth rate can be very different at different prey densities or attack rates. As in the previous analysis, we assume individuals of the same species respond to the predator equivalently, and therefore the manner in which the resources are divided among prey is unaffected by predator presence. Consequently, the effect of the predator on individual prey growth rate will be proportional to its indirect effect on resource renewal rate. In Fig. 3 we illustrate the effect of a predator-induced reduction in net attack rates of the prey population on resources. When net attack rates are low (i.e., small aN), there is little impact on the resources, and therefore a reduction in attack rate has only small effects on resource level. However, small increases in resource level are associated with a reduction in the resource renewal rate nearly proportional to the reduction in prey attack rate (Fig. 3a). Thus, the predator has a negative effect on prey growth that is nearly proportional to the reduction in prey attack rate. At intermediate net attack rates, a reduction in attack rate will have little effect on resource renewal rates, and thus there is no effect on prey growth rate (Fig. 3b). This case then approximates the scenario presented above where resource renewal rates are constant, and the direct negative effect of a reduced attack rate is offset by the reduction in competition. At yet higher net attack rates a different scenario emerges (Fig. 3c); the decreased net attack rate causes an *increase* in the resource renewal rate. Thus, in addition to the resource level being higher, the net amount of resources that flow through the system is also higher. Individual prey therefore grow at an increased rate in the

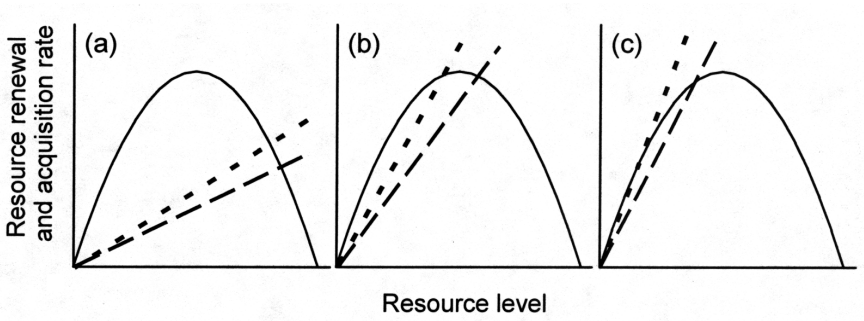

Fig. 3. Effect of a 25% predator-induced reduction in prey attack rate ($\Delta = 0.25$) on resource acquisition rate of prey in a system with logistic resource growth with (a) low, (b) intermediate, and (c) high net attack rate of the prey. Dotted, dashed, and solid lines as in Fig. 2. The effect of the predator-induced reduction on prey attack rate, and hence on resource acquisition rate and in turn prey growth rate, is negative and nearly proportional to the decrease in attack rate at low net attack rates, negligible at intermediate net attack rates, and positive at high net attack rates.

presence of the predator. In summary, as the magnitude of the net attack rate of the prey population increases from low to high levels, the net nonlethal effect of the predator on prey growth can proceed from negative, to negligible, to positive, because the indirect effect of the predator on resource renewal changes from negative, to negligible, to positive.

For clarity, we have examined a simple representation with logistic resource growth and a linear relationship between prey attack rates and resource density. However, the qualitative mechanisms and patterns of the predator effects presented here are robust to scenarios with more complex relationships. For example, a resource refuge from prey consumption can reduce the indirect positive effect of the predator on the resource renewal rate, but the model is not qualitatively changed, i.e., the mechanisms introduced above all must be considered to predict the net effect of the predator. It will be important, however, to accurately represent the functional relationship between resource acquisition rates and prey and resource density, and the relationship between resource renewal rate and resource level, to make quantitative predictions.

Finite resource without renewal

We next consider a system with a finite resource that is not renewed. This system would mimic, for example, natural systems existing on seasonal supplies of detritus, or experimental systems in which a finite nonreproducing resource is used. In this case, prey will reduce the level of resources over time, and the amount of resources available to an individual will be affected by predator presence as time progresses. The average

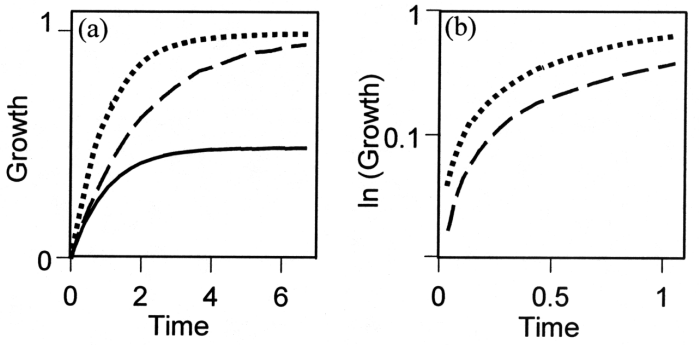

Fig. 4. Growth of individuals in a single prey species population with a finite resource and in the presence (dashed line) and absence (dotted line) of a 50% reduction in attack rate (i.e., $\Delta = 0.5$). Growth and time units are arbitrary. In the presence of the predator, prey initially grow 50% slower, but this negative effect disappears over time as a larger proportion of the resources are acquired. Figure 4b shows the natural logarithm of growth in the lower time region in order to illustrate the initial relative predator effects difficult to discern in Fig. 4a. If in predator presence only a lone individual from a large population responds to the predator, that individual will grow half as fast (Fig. 4a, solid line) over the entire time period.

growth for an individual prey in a system with a finite resource with and without a predator-induced reduction in attack rate is illustrated in Fig. 4. We assume that individuals acquire resources proportional to their attack rate and resource level. Therefore, after a finite time period, t, the total amount of resources acquired (and hence individual growth) is proportional to $1\text{-}e^{Na'(1-\Delta)t}$ (Royama, 1971). Initially, there is a strong negative effect of the predator on growth that is proportional to the induced reduction in attack rate. Over time, however, this negative effect gradually disappears, until the predator effect is nonexistent. It is, of course, a trivial result that the predator will have no effect on total biomass gain of the population over a large period of time if all food is eaten. However, it is important to keep in mind that from the perspective of an individual forager, two counteracting mechanisms are operating that lead to no net effect on growth. There is a negative direct effect on attack rate that is opposed by an indirect positive effect through the reduced intraspecific competition. Indeed, if only one individual responds to predator presence, while all of the other individuals do not, the initial large negative effect on the responding individual in predator presence would persist indefinitely for that individual (Fig. 4a), as opposed to disappearing as in the case where all individuals respond to the predator.

NONLETHAL EFFECT OF THE PREDATOR WITH COMPETITORS OF THE FOCAL PREY SPECIES

Thus far we have considered a homogeneous population of prey. Now consider the nonlethal effect of a predator on the growth rate of an individual focal prey species (C_1) in the presence of competitors (C_2) of that focal prey. If the phenotypic responses of the two prey species to predator presence differ in magnitude, the presence of the predator will affect their competitive relationship, which will contribute to the net effect of the predator on focal prey growth rate. To illustrate this process, first consider a constantly renewing resource. The resource growth rate is

$$\frac{dR}{dt} = J - a_1 R N_1 - a_2 R N_2 \tag{10}$$

where the subscripts 1 and 2 refer to the focal prey and competitor species, respectively. At steady state, resource density is

$$R = \frac{J}{a_1 N_1 + a_2 N_2} \tag{11}$$

Therefore the growth rate of the focal prey species in predator absence is

$$g_1' = b_1' J \frac{a_1'}{a_1' N_1 + a_2' N_2} \tag{12}$$

and in predator presence is

$$g_1 = b_1 J \frac{a_1'(1-\Delta_1)}{a_1'(1-\Delta_1) N_1 + a_2'(1-\Delta_2) N_2} \tag{13}$$

Δ_1 and Δ_2 are the predator-induced reduction in the attack rate of C_1 and C_2, respectively. As in the previous case with constant resource renewal, the predator presence does not affect the net amount of resources acquired by all consumers. However, in contrast to the case where competitors are absent, predator presence can have large negative or positive effects on prey growth rate depending on the relative effects on C_1 and C_2 attack rates. For example, if the nonlethal effect of the predator on the attack rate of C_1 is greater than that of C_2 (i.e., $\Delta_1 > \Delta_2$), then the predator will have a negative effect on C_1's growth rate. That is, the net amount of resources acquired by all individuals is the same, but predator presence reduces the competitive ability of C_1 relative to C_2, and hence has a negative effect on the amount of resource garnered by C_1. This effect is illustrated in Fig. 5, where the growth rates of C_1 in predator absence and presence are shown over a range of the relative net attack rates of C_1 and C_2 for the case where C_1 responds to the predator and C_2 does not. Recall that the net attack rate of each prey population defines the total impact of a species on the resource, and is the product of density and individual attack rates ($a_i N_i$). At very low net attack rates of C_1 relative to that of C_2, the reduction in the growth rate of C_1 is proportional to the attack rate reduction (Fig. 5). However, at increasingly larger C_1 net attack rates, the nonlethal effect of the predator on C_1's growth rate gradually disappears until it becomes negligible (as expected from the previous analysis at constant resource renewal without a competitor).

If the resource renewal is density dependent (e.g., logistic), then the competitor can influence the nonlethal effect of the predator on focal prey growth in two ways. First, the competitor response to predation will influence the indirect effect of the predator on

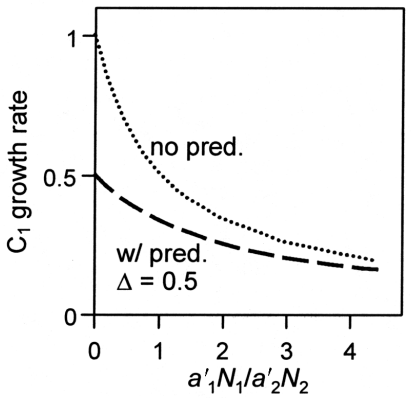

Fig. 5. Growth rate of a focal prey species, C_1, in the presence (dashed line) and absence (dotted line) of a 50% predator-induced reduction in attack rate (i.e., $\Delta_1 = 0.5$) and in the presence of a competitor, C_2, that does not respond to the predator (i.e., $\Delta_2 = 0$). Growth rate is shown as a function of the ratio of the net attack rate of the focal prey and competitor in predator absence. The predator has a strong negative effect at a relatively high net attack rate of the competitor, but very little effect at a low net attack rate of the competitor. (The parameters used were $a'_1 = a'_2$, $N_2 = 1$, and N_1 varied from near 0 to 4.4).

resource level and renewal rates. For example, if the net competitor attack rate is large compared to that of the focal prey species, then it will primarily be the competitor species response that dictates changes in the resource, not that of the focal consumer species. Second, the competitor response will influence the manner in which available resources are partitioned between the competitors. As an example of the interaction of these two factors, consider a system with high focal prey net attack rate. In the absence of a competitor, the predator-induced reduction in prey foraging is predicted to have a net positive effect on prey growth rate (Fig. 3c). If a competitor that does not respond to the predator is present, this will reduce the indirect positive effect on resource renewal, and reduce the fraction of resources acquired by the focal species, thus reducing the indirect positive effect on the focal species in two ways.

GENERAL EQUATION OF PREY GROWTH

The previous analyses suggest a general equation of the nonlethal predator effect on prey growth rate in a finite time period. From eq 2, the total growth of an individual is

$$G = bRa'(1 - \Delta)t \tag{14}$$

where b, R, and a' represent their average values in the finite time period t (integration over time would provide a more accurate representation). We can modify this equation algebraically to make more explicit the different mechanisms discussed above by which the predator affected prey growth,

$$G = bTp \tag{15a}$$

where

$$p = \frac{a_1'(1-\Delta_1)}{\sum_{i=1}^{n} a_i' N_i (1-\Delta_i)} \tag{15b}$$

and

$$T = Rt\sum_{i=1}^{n} a_i' N_i (1-\Delta_i) \tag{15c}$$

In the absence of the predator this equation applies with $\Delta_i = 0$. T is the total amount of resources acquired by all consumers (i.e., the focal prey species and n competitor species). p represents the fraction of T consumed by the focal individual. Equation 15 makes explicit four factors underlying the net nonlethal effect of a predator on prey growth. The predator may affect (1) the conversion efficiency of resources into growth (we have not included this factor in our analysis), (2) the amount of resources acquired by all foragers through reduced net attack rates and ensuing indirect effects on resource levels (eq 15c), (3) an individual's intrinsic ability to capture resources (the numerator of eq 15b), and (4) intra- and interspecific competition (the denominator of eq 15b).

When combined, the third and fourth mechanisms describe the predator's effect on the manner in which T is divided among individual prey.

All previous model results can be described within this framework (eq 15). In the case of constant resource renewal, the predator does not affect T, and therefore the effect on prey growth is proportional to the effect on p. There is therefore no predator effect on growth of the prey in the absence of interspecific competitors, but the predator affects prey growth by modifying the proportion of resources captured by the focal individual in the presence of interspecific competitors. In contrast, for density-dependent resource renewal, the predator indirectly affects T, and its effect on prey growth can be positive, negligible, or negative. In the absence of competitors, the predator effect on prey growth is proportional to this effect on T, because p is unaffected. With competitors, we must consider the nonlethal predator effect on T and p simultaneously. For a finite nonrenewing resource, T is equivalent to the amount of resources depleted, and therefore is negatively affected by predator presence initially, but as time progresses, the effect of the predator on T decreases until it is nonexistent. Therefore, the nonlethal predator effect on prey growth is initially negative and proportional to Δ, but as time progresses, asymptotes to 0. However, when competitors are present, we again must consider the nonlethal predator effect on p.

EMPIRICAL TESTS OF THE MECHANISMS OF NONLETHAL PREDATOR EFFECTS

In this section we present empirical studies that illustrate the operation of the mechanisms outlined in the model section. These empirical studies permit us to qualitatively test predictions formulated from the mechanisms examined above and demonstrate the range of possible responses to the presence of a predator. The studies we present were all conducted with anuran larvae and dragonfly naiads as predators. We formulated predictions based on model analyses taking into account important deviations of the experimental situation from model assumptions. For example, individual prey growth during experiments leads to changes in size and, therefore, attack rates. We validated the predictions using a simulation model that included such additional factors.

SINGLE PREY POPULATION WITH NONREPRODUCING RESOURCES

We show above that the nonlethal effect of the predator on prey growth will be influenced by the interaction between the net attack rate and resource levels. In experiments tadpoles grow over time and therefore tadpole attack rates increase, suggesting that the nonlethal predator effect on tadpole growth should change as they grow. In these experiments, artificial (nonreproducing) resources were supplied at a constant rate. Tadpoles were stocked at very small sizes, and therefore the net attack rate of the tadpole population initially was low. Hence, we predicted that the predator-induced reduction in the attack rate would have little indirect effect on resources and cause a reduction in growth rate proportional to the decrease in attack rates. As tadpoles grow, however, net attack rate will increase due to increased individual attack rates, and eventually tadpoles

will consume nearly all of the resources provided regardless of any predator-induced reduction in attack rate (Laurila et al., 1998; Relyea, 2001, 2002; Peacor, 2002). This latter situation therefore parallels the model scenario of a finite nonreproducing resource (Fig. 4), where a direct negative effect on an individual's attack rate is counterbalanced by a reduction in competition, and the average individual growth rate is unaffected by predator presence (eq 7). Unlike the finite resource case, however, resources are being continually added in the experiment, and therefore tadpole growth is predicted to converge in predator presence and absence to a value that increases over time, rather than to a finite value. Thus, in such experiments, conditions will change from essentially a constant nonlimiting resource where there is a strong negative predator effect on growth, to the case where resources are limiting, and there is no predator effect on growth. Disappearance of the negative effect on prey growth will occur when the net attack rate is large enough that tadpoles acquire nearly all resources in both the presence and absence of the predator. Therefore, the negative effect should disappear earlier at higher tadpole densities and at lower resource levels.

To test these predictions, we quantified the time course of nonlethal effects of caged larval dragonfly predators (*Anax junius*) on growth of larval wood frogs (*Rana sylvatica*) at different tadpole densities and resource levels. By caging *Anax*, nonlethal effects of the predator are isolated from any effects due to density reduction (Kats et al., 1988; Werner, 1991; McCollum and Van Buskirk, 1996). Wood frog tadpoles react behaviorally to chemical cues from larval dragonfly predators by reducing activity levels and spending a larger proportion of time on the substrate (Van Buskirk and Yuriwicz, 1998; Relyea, 2000; Peacor, unpublished data).

The experiment was performed in mesocosms filled with 300 L of well water containing 150 g of oak leaves added to provide structural complexity for tadpoles. Zooplankton, phytoplankton, and periphyton collected from a nearby pond were also added. Pools were covered with lids constructed of 60% woven shade cloth to deter colonization by insect competitors and predators. Treatments were instituted 9 days after mesocosms were constructed on June 7, 1999. Each pool received wood frog tadpoles obtained from nine egg masses (collected from one pond in Scio Township, MI), hatched in wading pools, and fed rabbit chow. Each pool received two small predator cages (constructed from slotted plastic drainpipe with ends enclosed by fiberglass window screening) and in half of the pools (i.e., predator treatments) *Anax* were added to the cages. These *Anax* were fed three–six wood frog tadpoles (total mass ~ 300 mg) every other day.

We employed a 4×2 factorial design, with four different resource level/tadpole density combinations crossed with caged *Anax* presence and absence. One resource level/tadpole density combination received 80 small wood frog tadpoles (22 ± 8 mg, mean $\pm$ SD) and ground rabbit chow distributed evenly throughout each pool every other day at a rate of approximately 1.5 g per day. To test the prediction that the nonlethal effect of the predator initially would be strongly negative and then disappear, after 5, 11, 19, and 26 days tadpoles were removed from the pools, counted, and weighed, and returned to the pools. To test the prediction that the negative effect on growth would

disappear more quickly at high prey density, a high density treatment was implemented with twice the initial tadpole density (160 tadpoles/pool) but with the same resource level as the previous treatment. To test the prediction that the negative effect on growth would disappear more quickly at low resource levels, two further treatments with 1/4 (intermediate resource level) and 1/8 (low resource level) the base resource level were implemented with 80 tadpoles. Thus, there was a base resource level/tadpole density treatment, and three manipulations that reduced the resource level per tadpole by either increasing tadpole density or reducing resource level. For the statistical analysis of average mass, we log transformed the average mass data in order to test for treatment effects on relative, rather than absolute, differences in mass (Wootton, 1994). Each of the eight treatment combinations was replicated four times.

The effects of caged *Anax* on wood frog growth were strongly dependent on time, density, and resource manipulation (significant 3-way interaction term using repeated measures, 2-way ANOVA, $p = 0.002$). The results were consistent with predictions (Fig. 6). Initially the predator had a strong negative effect on tadpole growth. However, as tadpoles grew and impact on resources increased (indicated by the increasing effect of resource level and tadpole density on average growth), this negative effect disappeared in all treatments (Fig. 6). Further supporting predictions, *Anax* effects on tadpole growth disappeared earlier at higher tadpole density and at lower resource levels. This effect can be seen by the third sampling date (significant interaction term between the resource level/tadpole density treatments and predator presence, 2-way ANOVA, $p = 0.015$), where there is a strong negative effect of the predator at the low density/high resource treatment (t-test, $p = 0.006$), but not in the other three treatments (t-tests, $p > 0.16$ for all three). Note that at the second sampling date the predator had an equivalent negative effect on mean growth in the three low density treatments; growth was 30.2 +/– 2.1% (mean +/– SE derived from error propagation), 27.0 +/– 1.0%, and 28.0 +/– 0.9% lower in predator presence in the high, intermediate, and low resource level treatments, respectively (Fig. 6). However, in the high density treatment, the predator effect was significantly lower, 11.2 +/– 0.6 % (2-way ANOVA, significant interaction between resource level/tadpole density treatments with predator treatments on average mass, $p = 0.009$, but nonsignificant interaction if the high density treatment was omitted from the analysis, $p = 0.7$). Average tadpole survivorship in the experiment was high, 92.5 +/– 1.0 and 89.0 +/– 1.0 % in the no predator and predator treatments, respectively. While significant (ANOVA, $p = 0.036$), the effect of the predator on survivorship was very small (4% reduction). There was no effect of resource level/tadpole density on survival (2-way ANOVA, $p = 0.8$). Therefore, effects on survivorship did not confound growth results.

An experiment by Van Buskirk and Yuriwitz (1998) also demonstrated the predicted effects of the predator on prey growth as a function of density of the consumer. These authors reported the effect of caged *Anax* on wood frog growth in 1300 l cattle tank mesocosms. All tanks were initiated at equivalent tadpole densities, but three removal rates (tadpoles were removed with nets at a rate of 0, 1.1, and 3.4% per day) were instituted to simulate predation. As predicted, at low final tadpole density (i.e., high removal rate), caged *Anax* had a large negative effect on tadpole growth rate, while at

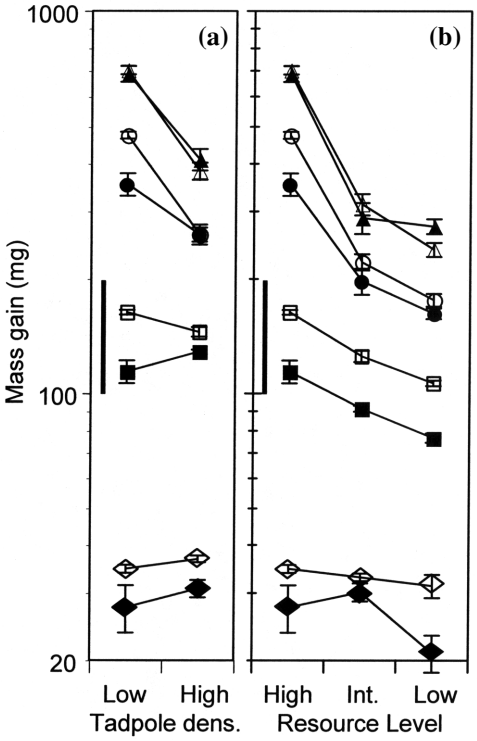

Fig. 6. Effect of caged *Anax* predators on wood frog tadpole mass gain (final minus initial mass) at four consecutive sampling dates at (a) different wood frog densities (low = 80 tadpoles, high = 160 tadoles) and (b) different resource addition levels (high, intermediate, and low correspond to a daily ration of X, X/4, and X/8, respectively). Diamonds, squares, circles, and triangles represent the first (day 5), second (day 11), third (day 19), and fourth (day 26) sampling dates, respectively. Comparing solid and empty symbols, which represent caged-*Anax* presence and absence, respectively, provides the effect of the caged *Anax* at a given date. Thus, the lowest paired lines (diamond symbols) show the effect of the predator on mass gain at the earliest measurement of mass gain, and moving up the graph shows the change in the predator effect as time progressed. Mass gain is presented on a log scale to clarify the relative nonlethal effect of the predator. Vertical bars are a guide to the eye that indicate a 50% difference in mass gain. Note that the same data are used for the low tadpole density in (a) and high resource level in (b).

high tadpole density, caged *Anax* had no effect on final size (Fig. 7). The final effect of the predator on tadpole growth was similar to what we observed in the previous experiment at the third sampling date (Fig. 6). Presumably, if the experiment of Van Buskirk and Yuriwitz was extended in time, the negative effect at the higher removal rate would also disappear when the tadpoles reached a large enough size to have a large impact on resources.

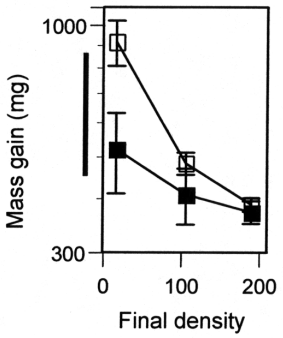

Fig. 7. Wood frog mass gain in the presence (filled squares) and absence (empty squares) of caged *Anax* at different simulated predator thinning rates. Mass gain is plotted as a function of the final density after thinning. Vertical bar as in Fig. 6. Data from Van Buskirk and Yuriwitz (1998).

DYNAMIC RESOURCE GROWTH RESPONSES

We have conducted several experiments to examine predator impacts through resource renewal rates. In the first of these experiments (Peacor, 2002), we examined the growth of small bullfrog tadpoles in the presence and absence of caged *Anax* in experimental cattle tank mesocosms. In order to create a situation where resource renewal rates could increase in predator presence, high net attack rates were instituted using a high (200) density of small bullfrog (*Rana catesbieana*) tadpoles in each mesocosm. In order to achieve varying levels of resource renewal rates, nutrients (nitrogen and phosphorous) were added daily at three different levels. Large bullfrog tadpoles were added at a low density to serve as a bioassay of resource levels since they responded weakly, if at all, to caged *Anax* presence (Werner and Anholt, 1996). We expected that the magnitude of the resource response to a predator-induced reduction in a small tadpole attack rate would vary as a function of the different nutrient addition levels, and large bullfrog tadpole growth served as a bioassay of this difference.

Results were consistent with the proposed mechanism (Fig. 8). Caged *Anax* had a strong positive effect on the net biomass gain of small and large tadpoles, which indicates that *Anax* had an indirect positive effect on resource renewal. Therefore, the direct negative effect of caged *Anax* on small bullfrog growth (foraging rate) was offset by the indirect positive effect on resource availability. The positive effect on small tadpole growth was only observed at the higher two nutrient treatments where the indirect positive effect on resource renewal was presumably the largest (as indicated by a larger indirect positive effect on large bullfrog growth). That is, at the low nutrient level where resource response was smallest, the negative and positive effects of the predator counterbalanced each other, resulting in no net effect on small tadpole growth. Different nutrient addition levels therefore led to qualitatively different effects of the predator on tadpole growth. In addition, these results differ from the previous experiments that used a finite nonreproducing resource.

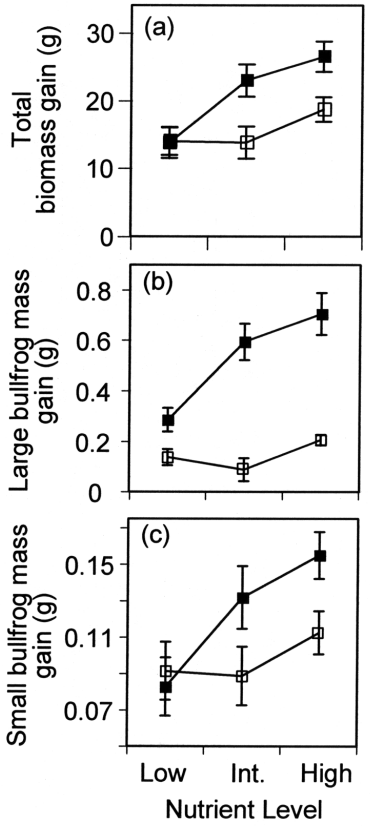

Fig. 8. The effect of a nonlethal predator on (a) total biomass gain of small and large bullfrog tadpoles, (b) average large bullfrog growth, and (c) average small bullfrog growth at three nutrient levels (low, intermediate, and high). Symbols as in Fig. 6. Data from Peacor (2002).

A second experiment also using bullfrog tadpoles illustrates the changing importance of the resource response mechanism over time. In this experiment (Werner and Peacor, in prep.), we manipulated nutrients, nonlethal presence and absence of *Anax*, and removal of tadpoles to simulated predation. Individual growth of the tadpoles (resulting in increasing population biomass) again increased pressures on the resources over time. We present one set of results from this experiment (high nutrient, removal, in the presence and absence of the *Anax*) where we can see the nonlethal effect of the predator on tadpole growth range from negative, to negligible, to positive over time as pressure on resources increased (Fig. 9). Early in the experiment when resources were abundant and individual growth fast, there was a significant negative effect of presence of the *Anax* on individual growth. As population biomass increased over time, individual growth diminished, resource limitation became important, and the two treatments con-

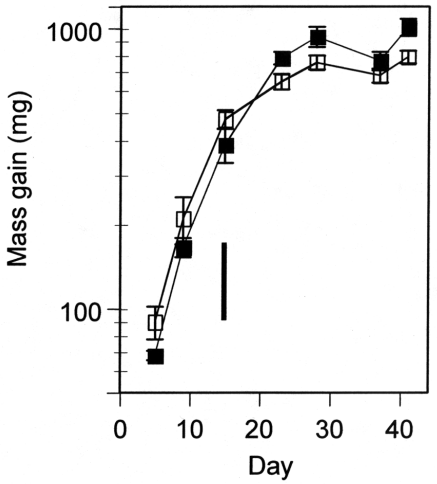

Fig. 9. Mass gain (mg) of small bullfrogs (initial mass = 15 mg) in the presence and absence of caged *Anax* over time. Symbols and vertical bar as in Fig. 7. Initially mass gain was approximately 25% lower in predator presence, but this negative effect disappeared, and at the end of the experiment, mass gain was approximately 28% greater in predator presence. From Werner and Peacor, in press.

verged. By the end of the experiment, however, individual growth was significantly greater in the presence of the predator due to the increasing relative importance of the resource response to reduced foraging. That is, reduced foraging in response to the presence of the predator enabled a positive response of the periphyton that overbalanced the direct negative effects of the predator presence.

COMPETITOR PRESENCE AND RESOURCE PARTITIONING

Thus far we have isolated and examined mechanisms that affect prey growth rate in the absence of competitors (or at low levels of interspecific competition). Presence of a predator can influence relative foraging rates of competitors and hence relative competitive abilities affecting how resources are divided among species (or size classes). We have performed a number of studies that examine this mechanism. In these experiments, we used levels of nonreproducing resources and consumer densities that insured virtually all resources were consumed in the presence and absence of the predator. This enabled us to isolate the effect of the predator on the division of the resources by the focal prey and competitor.

The model predicts that if the net amount of resources acquired by all consumers is nearly equivalent in the presence and absence of the predator, then the nonlethal effect of the predator will vary greatly as a function of the relative net attack rates of a focal prey species and competitor. For example, if the focal prey species responds to the predator, but the competitor does not, then the nonlethal presence of a predator will have a

negative effect at low, but little effect at high net attack rates of the focal prey species (Fig. 5).

Two published studies permit a test of this prediction as density of a focal prey species changes. In an experiment by Werner and Anholt (1996), small bullfrog larvae were raised at three densities (25, 50, and 100) in the presence and absence of caged *Anax* and eight large bullfrog competitors in 1300 L cattle tank mesocosms. In contrast to the small bullfrogs, large bullfrogs do not react to caged *Anax*, and therefore $\Delta_2 = 0$. (There also were 30 small green frog (*Rana clamitans*) tadpoles in the tanks whose reaction and growth response to caged *Anax* was similar to small bullfrogs (Werner, 1991; Peacor and Werner, 1997), and for the sake of this analysis were approximated as small bullfrog equivalents.) This experiment ran longer (56 days) than required to reduce resources to very low levels (Peacor, unpublished data). As predicted, caged *Anax* had a strong negative effect on small bullfrog growth at low small tadpole density, but had a negligible effect at high density (Fig. 10a).

A second experiment performed by Peacor and Werner (2000), using the same cattle tank mesocosm system, also corroborated the model predictions. In the presence of nonreacting large green frog competitors, caged *Anax* had a large negative effect on small green frog growth at low small green frog density, but no effect at high small green frog density (Fig. 10b). Note that if competitors were not present in these two experiments, the nonlethal effect of the predator on small tadpole growth is predicted to be negligible at all small tadpole densities (as observed in the last stage of the first wood frog experiment reported above). However, in the presence of large tadpoles, the

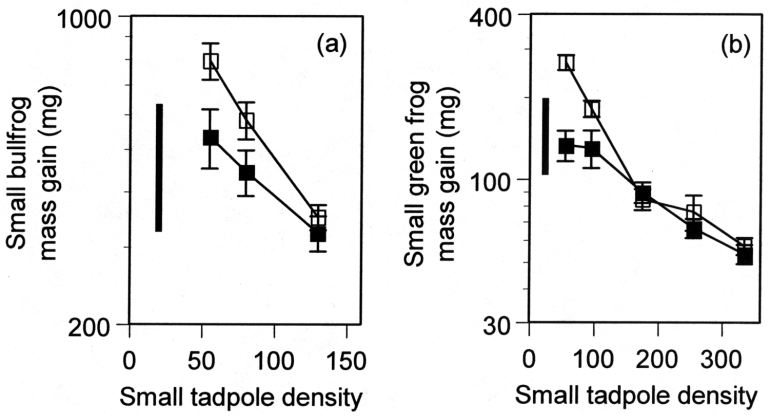

Fig. 10. Nonlethal predator (caged *Anax*) effect on prey mass gain as a function of prey density, in the presence of a nonresponding competitor. Symbols and vertical bar as in Fig. 7. (a) Small bullfrog growth as a function of small tadpole density (30 small green frogs + small bullfrogs) in the presence of large bullfrogs. Data from Werner and Anholt, 1996. (b) Small green frog tadpole growth as a function of small tadpole density (15 small bullfrogs + small green frogs) in the presence of large green frogs. Data from Peacor and Werner, 2000.

negative effect on the growth of small tadpoles occurred because the large tadpoles acquired a larger proportion of a finite resource in the presence of *Anax*.

Consider next the effect of the predator on a prey that reacts less strongly to the predator than to a competing species. We can use the previous two studies to also examine this situation by considering the larger non-reacting size class as the focal prey, and the responding small tadpoles as the competitor. Therefore, in contrast to the previous case, Δ_1 is 0, but Δ_2 is not. While the large tadpoles are not susceptible to *Anax* and do not modify their behavior, we can nevertheless consider them as idealized prey of *Anax* in order to clarify the nonlethal effect of a predator on the growth of a prey that responds more weakly than a competitor. At low competitor density, the nonlethal predator is predicted to have no effect on growth because the focal prey does not respond to the predator and there are no competitive effects. However, as competitor density increases, a predator-induced reduction in competitor attack rate will cause an increasingly larger proportion of resources to be made available to focal prey individuals. Using eqs 12 and 13, this positive effect on focal prey growth is predicted to increase rapidly as the net attack rate of the responding prey (i.e., density) increases (and is asymptote at $1/(1-\Delta_2)$).

In Peacor and Werner (2000), growth of nonreacting large green frogs was measured in the presence and absence of caged *Anax* over a large range of small tadpole density (as above, we group small bullfrogs and green frogs together in this discussion as their reaction to predator presence was similar). Small tadpole density ranged from moderately low (total net biomass accumulation of the population 0.7 times that of large green frog tadpoles) to very high (total net biomass accumulation 4 times that of large tadpoles). The relative net attack rates of the small tadpoles compared to those of the large tadpoles therefore spanned a large range. The model prediction was supported; at low density there was a small positive effect of predator presence on large tadpole growth that increased from approximately 10% to 60% as small tadpole density increased (Fig. 11a). Results from Werner and Anholt (1996) were also consistent with the model prediction. Small tadpole density did not extend to as low a level as in the previous example, so the positive effect was large over the entire range of small tadpole density, but relatively larger at high small tadpole density (Fig. 11b).

In the above experiments, we used a non-reacting consumer to clarify nonlethal predator effects on multiple prey populations. If both prey respond to the predator, and one responds more (i.e., $\Delta_1 > \Delta_2$), then the latter's competitive ability will decline relative to that of the other species. As a consequence, it will grow at a slower rate and the other species at a faster rate. Note that this hypothesis requires that nearly all resources are consumed in predator presence and absence, otherwise both species would simply grow more slowly. This prediction was examined in a laboratory experiment (Werner, 1991) in which small green frog and bullfrog tadpoles were raised together in the presence and absence of caged *Anax*, and fed at a low rate such that all resources were consumed under both treatments. In this study, the relative reduction in the activity of green frog tadpoles in response to caged *Anax* was greater than that of bullfrog tadpoles. Model predictions were supported as the presence of caged *Anax* increased the

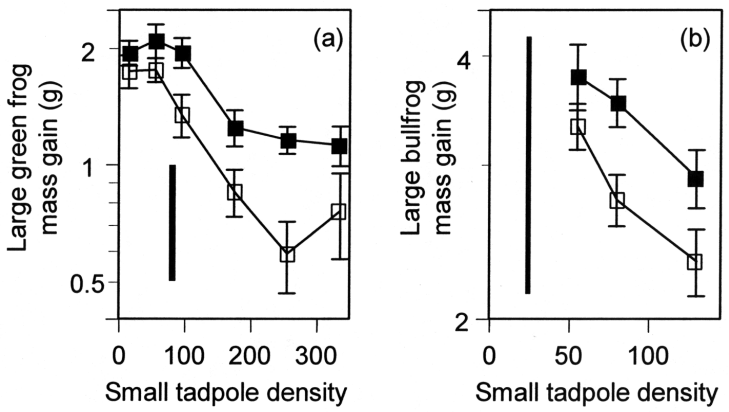

Fig. 11. Large tadpole mass gain as a function of small tadpole density in the presence and absence of caged *Anax* predators. (a) Large green frog growth over a range of small tadpole density (15 small bullfrogs + small green frogs). Data from Peacor and Werner, 2000. (b) Large bullfrog growth over a range of small tadpole density (30 small green frogs + small bullfrogs). Symbols and vertical bar as in Fig. 7. Data from Werner and Anholt, 1996.

competitive ability of small bullfrogs relative to that of green frogs, i.e., bullfrog growth increased in the presence of caged *Anax*, while that of green frogs decreased. Note that the presence of a competitor was required for these growth rate changes. In similar single species laboratory experiments with limited resources, typically the predator will have no effect on growth because the tadpoles acquire all resources in predator presence and absence (Laurila et al., 1998; Relyea, 2001, 2002; Peacor, 2002).

DISCUSSION

There has been a long and venerable history of theory and empirical work on predator–prey interactions. The vast majority of this work focuses exclusively on the density (or lethal) effects of the predator on the prey population. It is becoming increasingly clear that this body of work provides an incomplete picture of the effects of predators on prey populations, and that a more representative theory of predator–prey interactions must incorporate the nonlethal components of the predator's effects; i.e., the effects through induced changes in prey phenotype. There is now substantial empirical evidence that these nonlethal effects can contribute strongly to the net effects of predators in interactions with prey (Huang and Sih, 1991; Wissinger and McGrady, 1993; Peacor and Werner, 2001; reviewed in Werner and Peacor, 2003).

In order to explore some of the components of a more representative theory, we developed a framework to examine nonlethal effects of predators on individual prey growth rate. Nonlethal predator effects arise through several interacting mechanisms, including direct negative effects on resource acquisition rates, positive indirect effects

via reductions in intra- and interspecific competition, and positive indirect effects via resource renewal rates. We develop a heuristic model that clarifies how these mechanisms combine to influence individual prey growth, and how this influence depends on environmental factors such as focal species density, competitor density, resource dynamics, and the timescale over which the interactions occur.

Our empirical results illustrate the range of effects predicted by the model. The rich nature of the time dependence of the nonlethal predator effect on prey growth was illustrated in a system with a finite nonreproducing resource (Fig. 6). Initially, the mechanism dominating the growth effect results was the induced reduction in the amount of resources acquired. However, over time, as tadpoles grew and the resources were depleted at a greater rate, the negative effect of the predator on growth disappeared as reduction in intraspecific competition due to predator presence counteracted the direct negative effects. As predicted from the interaction of these two mechanisms, this disappearance progressed more quickly at high tadpole density, and low resource levels, where the depletion of resources occurred more rapidly.

Further, in experiments with a limiting artificial (nonreproducing) resource, we demonstrated how the nonlethal effects of a predator can affect the manner in which resources are divided among prey species that respond differentially to the predator (Figs. 10 and 11). We showed that if a (nonresponding) competitor was present, then the predator had a negative effect on prey growth because the focal prey species acquired a smaller proportion of the resource, but had a positive effect on the growth rate of a nonresponding consumer. When both prey responded to the predator, the predator had a positive effect on the growth of the prey that reduced its foraging effect the least, and a negative effect on the prey that reduced its foraging activity the most.

Finally, we were able to show the potential for a predator to indirectly affect the total amount of resources available to the prey population. Changes in prey attack rates on resources can affect the numerical response of a resource, and therefore a predator-induced reduction in prey attack rate can have a large effect on resource renewal rates and levels. This mechanism also counters the direct negative effect of the predator. At high net attack rates of the prey, a predator-induced reduction in foraging rate can lead to a large increase in resource renewal rates, which in turn can cause a net positive effect on prey growth rate. This prediction was observed in two experiments (Figs. 8 and 9). The operation of this mechanism is clear in these experiments because the net effects of predator presence are positive. It is important to note, however, that the underlying mechanism may be operating even when the net effect is negative or negligible (e.g., in the first experiment at the low nutrient level). In similar scenarios in natural systems, therefore, predator effects on prey could go unnoticed even if there are large negative and positive effects that counterbalance each other. It is important to quantify the two opposing effects in order to predict the effect of the predator as environmental conditions change over space or time. Clearly understanding the mechanistic basis of the nonlethal effects of predators on prey will be necessary to understand and predict predator effects on a food web.

In the experimental results reviewed here, deviations from model predictions were

observed that may yield insight into tadpole phenotypic response to predator presence. While the general trends in the two experiments examining the predator effect on prey growth in the presence of a nonreacting competitor (Fig. 9) were consistent with model predictions, there was a quantitative deviation from expectations. Small tadpole biomass gain was approximately equal to that of large tadpoles in the highest density treatment in the Werner and Anholt (1996) experiment, and at the intermediate density in the Peacor and Werner (2000) experiment. These results suggest that the net attack rates of the two competing size classes were similar at these densities. The model predicts that under this condition, the predator will have a negative effect on the growth of small tadpoles (Fig. 5). However, in both empirical studies, there was no effect on growth under these conditions; i.e., the negative effect of caged *Anax* on small tadpole growth disappeared more quickly than that predicted by the model as density (net attack rate) increased. One possible explanation for this discrepancy is that the phenotypic response to predator presence was smaller at higher small tadpole densities. Prey are predicted to react less strongly to predators at reduced resource levels that occur at higher prey densities (Gilliam, 1982; Abrams, 1990; Houston et al., 1993; Werner and Anholt, 1993; Grand and Dill, 1999). Alternatively, behavioral ecological theory predicts that prey should respond less to predators at high prey densities due to dilution effects of predation risk (Pulliam, 1973; Bertram, 1978; Dehn, 1990; McNamara and Houston, 1992; reviewed in Elgar, 1989; Lima, 1990) or due to density dependent differences in risk assessment of predator cues (Peacor, 2003). This latter mechanism may be responsible for the density dependent pattern in the wood frog experiment (Fig. 6). At the second sampling date in this experiment, the effect of predator presence on relative tadpole growth was equivalent across resource levels, but significantly lower in high-density treatments. Because the relative effect of the predator on tadpole growth was very similar across a wide range of resource levels, this density effect was not likely due to potential changes in resource level caused by increased tadpole density. This pattern suggests that wood frog tadpoles respond less strongly to predator presence at high conspecific density independent of their effect on resource levels. Thus, these additional relations may have to be included in the model to achieve quantitative fidelity to empirical results.

Our theoretical analyses examined idealized systems and were used to clarify three different potential mechanisms by which the nonlethal effect of a predator can affect prey growth. A number of potential factors were not included in the model analysis that may be important to include in particular systems. For example, we assumed that the effect of predator presence on attack rate (Δ) is constant in the derivation of model predictions. But the magnitude of phenotypic responses to predator presence is predicted to be a function of resource and conspecific densities as discussed above. Δ may also be a function of prey size, and therefore change with time as an individual grows. We also have not addressed potential effects of a predator on conversion efficiency of resources to growth. In the presence of the predator, consumers may adjust physiological traits or behavior (e.g., habitat shifts) that affect metabolic rates and assimilation efficiency (Loose and Dawidowicz, 1994; McPeek et al., 2001). Therefore, to attain more quantitative predictions, modifications of the model to more accurately depict such features

may be required. As illustrated in the previous paragraph, our model can serve as a "null model", deviations from which can be a signal of important biological processes occurring in a particular system.

Because large fluctuations in species' densities at local spatial scales are common, our analysis predicts that the nonlethal predator effect on growth rate of consumers will vary greatly over time and space as a function of fluctuations in the density of the focal prey species, competitors, and resources and/or predators (through combined lethal and nonlethal effects, see below). For example, consider a system with a focal consumer and competitor species that respond strongly and weakly to predator presence, respectively. If the competitor density is low, the nonlethal effect of the predator on focal prey growth is predicted to be negligible at very high focal prey densities and have a very large indirect positive effect on the competitor. This picture would be dramatically altered when the relative species' densities were reversed; the predator then is predicted to have a strong negative effect on focal prey growth and no effect on the competitor growth. This leads to the counterintuitive result that it is precisely where the direct effect on prey growth is negligible that the indirect effect on other species is large (Peacor and Werner, 2000), and illustrates the necessity of examining species behavior to interpret predator effects on prey growth.

The context dependence of the magnitude and sign of the nonlethal predator effect on prey growth indicates the potential difficulties of predicting the net effects of a predator. For example, the measurement of the nonlethal effect of a predator on prey growth rate in the laboratory or mesocosms may not reflect the magnitude in the field because of differences in interspecific competition levels or resource dynamics. Consider, for example, a natural system in which there is relatively little intraspecific competition for a relatively abundant resource, and therefore there are strong negative nonlethal predator effects on the focal species' growth rate. If, in a laboratory or mesocosm study, relatively high densities or low resource levels were employed, growth rate would be unaffected by the predator, and therefore greatly underestimate the effect in the natural setting. A similar problem would arise if resources were limited in the presence of interspecific competition from a competitor that reacts more weakly to the predator. In the natural system the nonlethal predator could have a large negative effect, but such an effect would not be detected in a mesocosm experiment conducted in the absence of the interspecific competitors. Further, the duration of an experiment is a critical factor and must be carefully considered. An empirical study performed over a short time period with abundant food could indicate large predator effects on growth, when in fact in natural settings the influence is small.

Our model analyses were directed at the nonlethal component of the predator's effect on prey growth in order to isolate and clarify the underlying mechanisms. Clearly, to understand the overall influence of nonlethal effects of predators it will be necessary to understand their influence in combination with lethal (i.e., density) effects. Since the model enables prediction of nonlethal predator effects on prey growth rate as a function of prey density, we can use it to examine the manner in which lethal and nonlethal predator effects combine to affect individual prey growth. If density effects are small,

then we can approximate the net effect of the predator on prey growth rate using the previous analysis of nonlethal effects, which may be negative, negligible, or positive. At increasingly higher predation rates, however, both lethal and nonlethal effects become important. Individual prey growth rate will be positively affected by reductions in prey density due to the freeing of resources. In addition, because the nonlethal effects of the predator on prey growth are dependent on prey density, a change in density due to lethal effects can change their expression. Consider, for example, a single prey population at a high enough density that resources (with logistic resource growth) are suppressed in a system. In the absence of high predation rates, the nonlethal effect of the predator on growth may be positive. But at high predation rates, the net attack rate of the prey population will be lowered due to reduced densities, and the nonlethal predator effect will be negative. The density effect therefore has effectively increased the relative importance of the direct negative effect on prey growth resulting from reduced attack rates. In this case, the nonlethal and lethal effects operate synergistically to negatively affect population growth rate through reductions in density and individual growth rate. However, at high net attack rates, both predation and a predator-induced reduction in foraging activity can act to release resources and cause large increases in resource levels. In this case, the lethal and nonlethal effect combine to reduce overexploitation of resources and hence to positively affect individual growth and potentially population growth rate and density.

Several empirical studies permit an analysis of the combined impact of lethal and nonlethal predator effects. In the experiment of Van Buskirk and Yuriwitz (1998) a lethal predator effect was simulated by manually removing tadpoles. In our experiment on wood frog growth the different wood frog density treatments can represent different levels of predation (i.e., lower densities correspond to higher predator rates). Similarly, higher resource levels in this experiment, and hence lower intraspecific competition, can be viewed as the consequence of higher lethal effects. In all cases, the density (lethal) effect on surviving tadpole growth rate was positive (Figs. 6 and 7) due to a reduction in intraspecific competition for a limited resource. However, this effect was greatly reduced when caged *Anax* were present (third sampling date in Figs. 6 and 7). That is, the negative nonlethal effect of the predator on growth rate greatly reduced the lethal positive effect due to released resources. We also can use the same data to examine how the lethal effect influenced the magnitude of the nonlethal effect of the predator on growth. The magnitude of the nonlethal effect was large and negative at high simulated predation rates, but negligible at low simulated predation rates. The mechanisms underlying this effect are those used to explain the difference in the nonlethal effect at different prey densities. This example illustrates the manner in which the lethal and nonlethal effects can combine to affect prey growth. The model derived here can be used as a guide to predict the net result, which will vary across systems as a function of prey density, resource dynamics, competitor presence, and the relative magnitude of lethal and nonlethal effects.

Do the short-term effects on individual prey growth influence long-term population abundance and dynamics, and emergent community properties such as diversity and

structure? To incorporate phenotypic responses into theory to address these questions, it is necessary to include parameters that describe how a phenotypic response will affect the interaction between species. These parameters could represent either particular traits, or phenomenological constants that encapsulate the combined effects of many trait responses. For example, a predator will affect prey foraging rate on a resource through changes in many different behavioral (including, e.g., time spent feeding, speed, time spent searching for high-resource patches, and habitat preference), morphological, and physiological traits. Such a phenomenological constant is therefore analogous to the parameter Δ that we use in the model to represent the nonlethal effect of a predator on prey attack rates . Given the complexity of measuring all the trait changes responsible for Δ, and the difficulty in quantifying their relationship to attack rates or interaction coefficients, Δ would be difficult to derive from trait changes directly. Further, we have shown in this paper that equivalent predator-induced changes in prey attack rates on resources can have very different effects on prey growth rate. Therefore, it is not straightforward to extract parameters needed in theory from measurement of fitness responses such as growth rate. However, we have argued elsewhere (Peacor and Werner, in press) that Δ can be estimated from the predator effect on prey growth rate in experiments after small time intervals (e.g., at the first and second sampling date in the wood frog experiment presented here, Fig. 6). A review of the literature using this method indicates that Δ lies in the range of 0.2–0.8 in many systems, and thus lies in a range that is most likely large enough to strongly influence model predictions of population and community dynamics.

The prediction, verified experimentally, that the magnitude of the nonlethal predator effect on growth is largest at high removal rates, suggests that nonlethal effects may contribute strongly to net predator effects even for predator–prey interactions with very high predation rates. One might assume that nonlethal effects are more likely to be unimportant when density effects are large. However, the model and empirical studies suggest that it will be at low densities (i.e., high predation rates) when the (negative) nonlethal effect on growth is most severe. A predator-induced phenotypic modification that causes a reduction in growth on the order of 50%, as seen in the experiments presented here, will most likely have a very large effect on prey fitness. Growth is an important component of fitness, and reductions in growth rate can have numerous negative effects on fitness, such as increasing the time in which an individual is vulnerable to predation, and reducing the probability of metamorphosis in ephemeral habitats. Further, the interactive effect of the nonlethal effect of the predator on prey growth as a function of the lethal effect suggests that nonlethal predator effects on prey growth could destabilize population dynamics by leading to larger amplitudes populations cycles. That is, when densities are low (associated with large lethal effects), the nonlethal effect on growth is most severe, and when densities are high, the nonlethal effect on growth is weakest.

In conclusion, the success of the framework developed here in predicting experimental results over a large range of conditions indicates that it can be used to assist in the design and interpretation of experiments, and help in the interpretation of observations

of nonlethal predator effects in natural systems. The context dependence of nonlethal predator effects illustrated in the model and empirical results should serve to alert researchers to the potential rich nature of this process and the potential difficulty of making general inferences from the results of laboratory and mesocosm studies, or from limited observations of natural systems. It is clear that it will be necessary to incorporate the nonlethal effects of predator into predator–prey theory to understand dynamics in many systems.

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