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Adaptation, density dependence and the responses of trophic level abundances to mortality

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ABSTRACT

We use simple models to examine how the abundances of three trophic levels change in response to mortality imposed on each one of the levels. The models contain two factors whose joint effects have not been incorporated into previous analyses: direct density dependence ('self-damping') at trophic levels above the bottom level, and adaptive change on the middle trophic level. The adaptive change involves balancing foraging gains and risks of predation. The combination of this type of adaptation and self-damping leads to a wide variety of potential responses of trophic level abundances to increased per capita mortality at any one level. However, the signs of the responses at each level can often be predicted from a knowledge of the strength of direct density dependence together with three additional quantities: the shape of the relationship between resource intake and per capita growth rate for the middle level; the curvature of the function relating the fitness of the middle level species to its foraging effort; and the change in the ratio of predation vulnerability to foraging effort as effort changes. Some possible responses include a decrease in all three levels with increased mortality of the top level, and an increased density of either the middle or top level, following increased mortality imposed on it or its prey. We show that the responses of trophic levels to mortalities are similar for several different mechanisms of adaptive change on the middle level – micro-evolution or behaviour, species replacement and induced defence. Possible evidence for some of the novel predictions is discussed, as is the need for experimental studies of the consequences of mortality rates at all trophic levels, quantification of direct density dependence, and studies of the shapes of functional and numerical responses.

Keywords: adaptive foraging, bottom-up effect, density dependence, food web, inducible defences, top-down effect, tritrophic system.

INTRODUCTION

The abundances of the trophic levels of an ecosystem have a major effect on its functioning. Much food web research has therefore been concerned with how environmental factors

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affecting one trophic level influence the abundances of all levels in the system (Hairston *et al.*, 1960; Oksanen *et al.*, 1981; Abrams, 1993; Wootton and Power, 1993; Persson *et al.*, 1996; Polis and Winemiller, 1996; Brett and Goldman, 1997; Persson, 1999; Oksanen and Oksanen, 2000). However, many of the responses that have been observed are inconsistent with simple models (Leibold *et al.*, 1997; Chase *et al.*, 2000), and the current theoretical basis for understanding mechanisms that could lead to different responses is limited. The analysis presented here extends existing theory on three-level ecosystems in two ways: (1) it examines the responses of all three trophic levels to a standard perturbation at one level – that is, increased per capita mortality; and (2) it examines the combined effects of density dependence and adaptive balancing of food intake and predation risk on the responses to mortality. In analysing these issues, we look at several different mechanisms for adaptation of foraging effort and predation risk within a trophic level. The need for additional theory is reflected in the abundance of examples where the responses of trophic level abundances to factors affecting one level do not match the predictions of simple theory (Leibold *et al.*, 1997; see discussion below).

Much previous work on trophic level responses to environmental perturbations has been discussed under the rubric of 'top-down' effects of predator removal and 'bottom-up' effects of plant fertilization (e.g. Power, 1992; Persson *et al.*, 1996; Persson, 1999; Menge, 2000). This has led to a rather curious neglect of the consequences of environmental effects on the middle trophic level(s). While some studies have manipulated herbivore behaviour using predators (e.g. Schmitz *et al.*, 1997; Kotler *et al.*, 2001), environmental factors directly affecting middle trophic levels are often ignored in the top-down/bottom-up debate. By developing theory for the effects of per capita mortality on all levels of a system, we hope to encourage the use of more standardized perturbations when examining the control of different trophic levels on ecosystem properties. Past conclusions on the relative importances of bottom- and top-level control may have differed in part because of the different methodologies (fertilization *vs* species removal) employed to measure these effects (Chase *et al.*, 2000). Increases in per capita mortality of the basal species reduce the maximum per capita growth rate and equilibrium density of that species, so a decreased death rate should have effects similar to those of at least some types and levels of fertilization. Increases in mortality of the predator, if large enough, will eventually lead to predator disappearance. Thus, changes in per capita mortality of top and bottom trophic levels should often have effects that are qualitatively similar to those produced by the traditional approaches of fertilization and removal.

A second limitation of previous work is that it has not considered sufficiently many alternatives to simple food chain models in which the per capita growth rate of each consumer species depends on food and predator densities but does not depend directly on its own density (Oksanen *et al.*, 1981). Several features have been added as single-factor modifications to the simplest models, including direct density dependence of consumer growth (Wollkind, 1976; Gatto, 1991), heterogeneity in the trophic characteristics of species on one or more trophic levels (Leibold, 1989, 1996; Abrams, 1993) and adaptive behaviour (Abrams, 1995). However, there is a lack of models that ask whether two or more of these factors can interact to produce responses that differ from those predicted by models that only include a single additional factor. The two factors we consider here – direct density dependence and adaptive balancing of foraging costs and predation risk – are both likely to be present in many, if not all, food webs. Direct density dependence is almost inevitable in any population occupying a spatially heterogeneous environment where some locations are

better (for fitness) than others; in such cases, low-density populations have higher per capita growth rates, everything else being equal, because the individuals are able to occupy the best sites. A number of other factors, including intraspecific aggression, species-specific diseases and the presence of limiting non-food resources, can give rise to direct density dependence, and all of these are common occurrences (Abrams, 1986; Abrams and Ginzburg, 2000). Trade-offs between food intake and predation risk are also likely to arise in any spatially heterogeneous environment where individuals are not completely well-mixed and the foragers can behave adaptively; avoidance by foragers of areas with high risk allows food populations to build up in those areas, leading to a trade-off. Other reasons why such trade-offs should be extremely common are provided in Werner and Anholt (1993), Abrams (1995), Peacor and Werner (2000) and Bolker *et al.* (2003). Lima (1998) reviews the many empirical examples. A food intake–predation risk trade-off is also likely to characterize the traits of a pair of species on the middle trophic level because the trade-off greatly increases the ability of those species to co-exist (Armstrong, 1979; Leibold, 1989, 1996). Given that both direct density dependence and adaptive balancing of predation risk and food reward are very common, they are likely to be present together in many systems, and their joint consequences need to be understood.

Adaptive balancing of risks from higher trophic levels and food intake from lower trophic levels has been the subject of much recent work (Bolker *et al.*, 2003). However, different mechanisms that lead to such adaptive balancing have often been treated as having unique effects. Adaptive change within a trophic level may occur due to a variety of mechanisms, including shifts in individual behaviour (reviewed in Lima, 1998), evolutionary change (theory in Abrams and Matsuda, 1997; recent example in Yoshida *et al.*, 2003), shifts in the relative abundances or replacement of species on a particular level (Armstrong, 1979; Leibold, 1989, 1996; Bohannan and Lenski, 1999; Chase, 1999; Steiner, 2001) and changes in the characteristics of individuals due to a variety of non-genetic mechanisms, such as shifts in size- or age-structure, induced defences or maternal effects (e.g. Chase, 1999; Vos *et al.*, 2002). At the end of our analysis, we show that these various mechanisms are often expected to have similar effects on the responses of trophic level abundances to mortalities at particular levels.

The models analysed here include three trophic levels; this is both a common number of levels in natural systems (Cohen *et al.*, 1990) and is the smallest number of levels that allows indirect effects between non-adjacent trophic levels. Trophic levels are modelled as homogeneous populations; this simplification has led to accurate predictions in some cases (Wootton and Power, 1993; Oksanen and Oksanen, 2000). We examine the consequences of altered mortality at any one of the trophic levels, using extensions of Oksanen and co-workers' (1981) model. These extensions represent a full factorial set of combinations of direct density dependence at higher trophic levels and adaptive behaviour at the second trophic level. Given the restriction to three-level systems, the middle level is the only one that can show adaptive balancing of food intake and predation risk. We end the analysis by looking at models with alternative mechanisms for adaptive change.

THE MODELS

To examine the interaction of density dependence and adaptation, we begin with a model that lacks adaptation, but which has direct density dependence at all trophic levels. We adopt a simple form for the model; it is characterized by linear functional responses, and

the top two levels have per capita death rates that increase as a function of their density. The resource (R), consumer (N) and predator (P) populations change according to:

$$\frac{dR}{dt} = R[f(R) - CN - d_1] \tag{1a}$$

$$\frac{dN}{dt} = N[b(CR) - \delta_2 - d_2 - sP - \gamma(N)] \tag{1b}$$

$$\frac{dP}{dt} = P[EsN - d_3 - \alpha(P)] \tag{1c}$$

where f is a decreasing function that describes the density dependence of per capita population growth of the resource, and C and s are the slopes of the consumer and predator functional responses, respectively, and are referred to below as 'attack rates'. The per capita birth rate of the consumer is b, an increasing function of its resource intake rate, CR. The predator's per capita birth rate is directly proportional to its per capita intake rate of consumers, sN, with E denoting the conversion efficiency of consumers to predators. Direct density dependence in the non-basal species' per capita death rates is described by the increasing functions, γ for the consumer and α for the predator. This leads to models that are simpler than those in which density affects other components of population growth (C, s or b), but preliminary work on these alternatives (P. Abrams, unpublished results) suggests that they have similar effects. Density- and trait-independent per capita mortality rates are given by d_1, d_2 and d_3 for resource, consumer and predator, respectively. We will refer to these as extrinsic mortality rates. The trophic characteristics of the middle level that may be capable of coupled adaptive change in subsequent models are C and s. A trait that affects one or both of these consumption rate constants is also likely to affect the consumer's death rate, and the trait-dependent component of the consumer's per capita death rate is described by δ_2.

Our primary model of adaptive change assumes that a continuous trait affects foraging rate, predator vulnerability and non-predatory mortality. We use foraging rate, C, as a measure of the trait, and express vulnerability to predation, s, and non-predatory mortality, δ_2, as increasing functions of C. As noted above, trade-offs that couple mortality and C have been documented frequently (Lima and Dill, 1990; Werner and Anholt, 1993; Lima, 1998), and there are many theoretical reasons why they should be common (e.g. Abrams, 1995). The mean value of C changes adaptively over time at a rate that is proportional to the rate of increase of individual fitness with the trait. Such 'fitness gradient' models have been discussed in many previous works (Abrams *et al.*, 1993, Geritz *et al.*, 1998; Abrams, 2001). The proportionality constant may change as a function of the current mean trait value; this dependence is described by the function $v(C)$. A common feature of evolutionary change is that genetic variance becomes depleted as a trait approaches a limiting value, reducing the rate of change of the trait (Abrams *et al.*, 1993). If the trait is under purely behavioural control, the maximum rate of change towards a limiting value must also decrease as that limiting value is approached (Abrams, 2001). This again suggests that $v(C)$ decreases in this circumstance. Thus, the dynamics of the foraging trait are described by

$$\frac{dC}{dt} = v(C)[Rb' - \delta_2' - s'P] \tag{1d}$$

where the quantity in square brackets is the derivative of individual fitness with respect to the trait, C. Primes denote derivatives with respect to the arguments of the functions: $b' = \mathrm{d}b/\mathrm{d}(CR)$, $\delta_2' = \mathrm{d}\delta_2/\mathrm{d}C$, $s' = \mathrm{d}s/\mathrm{d}C$. Provided that a relatively narrow distribution of trait values is present in the population at a given time, equation (1d) can serve as a good approximation to models for change in the average value of either a behavioural or a quantitative genetic trait (Abrams *et al.*, 1993; Abrams, 2001; Abrams and Chen, 2002).

RESULTS

The responses of abundances to the extrinsic per capita mortality rates are obtained by differentiating the equilibrium conditions for equations (1a–d) with respect to each of the d_i. The resulting formulae can be simplified somewhat by assuming that the equilibrium of the system is locally stable. The expressions determining the signs of responses of abundances to mortalities are presented in Table 1, which also provides some additional details on their derivation. Table 2 evaluates the possible signs under a variety of assumptions. The remainder of this section provides more details about these general results under different assumptions about the components of equations (1).

Trophic level responses in the absence of adaptation: the implications of density dependence

In this case, the system lacks equation (1d). The dynamics of the three-species food chain in the absence of adaptation has been analysed many times previously (for a review, see Persson *et al.*, 1996). If there is an equilibrium with positive densities of all three species, it must be locally stable, given the linear functional responses assumed here (e.g. Jeffries, 1974; Pimm, 1982). The well-known model of Oksanen *et al.* (1981) assumes that there is no direct density dependence in equations (1b) and (1c), leading to the pattern of responses of abundances to mortalities summarized in matrix 1 of Table 2. Allowing direct density dependence in equations (1b) and (1c) leads to the slightly different responses given by matrix 2 in Table 2. This removes the three 'zeroes' present in matrix 1, which all stem from the assumption that predator population growth is independent of its own density. Given predator density dependence, an increase in the per capita death rate of a given species decreases its own equilibrium density, decreases the density of all species above it in the chain, and increases the density of the species immediately below it in the chain. An increase in the predator's extrinsic death rate, d_3, decreases the resource density because it increases consumer density. More generally, increasing the per capita death rate of a given species (trophic level) decreases the densities of all species above it in the chain, increases species that are an odd number of levels below it, and decreases the densities of the species that are an even number of levels below it. These results will be considered the standard against which our models with adaptation on the middle level are compared. The predictions of this model with density dependence of predators and consumers agree with the intuitive notions that: (1) the density of a species decreases when mortality is applied to it and (2) mortality imposed on a predator increases its prey population, while mortality imposed on a prey decreases its predator. They also yield what we will term the 'standard' trophic cascade responses – that is, mortality applied to the resource decreases all of the levels, while mortality applied to the predator increases the consumer level, but decreases predators and resources.

Table 1. Responses of equilibrium variables to altered death rates at one trophic level*

1. Increased mortality of the bottom level

$\frac{\partial \hat{P}}{\partial d_1}$ has sign of $-Es'\gamma' N(b' + CRb'') + EsC[R^2 b'' - \delta_2'' - s''P]$

$\frac{\partial \hat{N}}{\partial d_1}$ has sign of $C\alpha' b'(R^2 b'' - \delta_2'' - s''P) + ENss'(b' + CRb'') - b'ENCs'^2$

$\frac{\partial \hat{R}}{\partial d_1}$ has sign of $(\alpha'\gamma' + Es^2)(R^2 b'' - \delta_2'' - s''P) - \gamma' ENs'^2$

$\frac{\partial \hat{C}}{\partial d_1}$ has sign of $CEsb's' - (\alpha'\gamma' + Es^2)(b' + CRb'')$

2. Increased mortality of the middle level

$\frac{\partial \hat{P}}{\partial d_2}$ has sign of $EN[Cs' - s][b' + CRb''] - Esf'(R^2 b'' - \delta_2'' - s''P)$

$\frac{\partial \hat{N}}{\partial d_2}$ has sign of $-N\alpha'(b' + CRb'') + ENf's'^2 - f'\alpha'(R^2 b'' - \delta_2'' - s''P)$

$\frac{\partial \hat{R}}{\partial d_2}$ has sign of $ENs'[Cs' - s] - C\alpha'(R^2 b'' - \delta_2'' - s''P)$

$\frac{\partial \hat{C}}{\partial d_2}$ has sign of $-f's'sE + C\alpha'(b' + CRb'')$

3. Increased mortality of the top level

$\frac{\partial \hat{P}}{\partial d_3}$ has sign of $-\gamma' N(b' + CRb'') + (b'C^2 - f'\gamma')(R^2 b'' - \delta_2'' - s''P)$

$\frac{\partial \hat{N}}{\partial d_3}$ has sign of $Ns(b' + CRb'') - CNb's' + f's(R^2 b'' - \delta_2'' - s''P)$

$\frac{\partial \hat{R}}{\partial d_3}$ has sign of $-N\gamma' s' + sC(R^2 b'' - \delta_2'' - s''P)$

$\frac{\partial \hat{C}}{\partial d_3}$ has sign of $-b'C(s - Cs') - s'\gamma' f' - sC^2 Rb''$

* The equilibrium conditions for equations (1a–d) were differentiated implicitly with respect to each one of the three per capita mortality parameters, d_i, and the resulting equations were solved for the partial derivatives of the equilibrium values of each variable. The formulae for the partial derivatives of population densities can be factored into an expression multiplied by the determinant of the matrix of equilibrium conditions. The latter is proportional to the determinant of the Jacobian matrix and, therefore, is positive if the equilibrium is stable. The expressions for the partial derivatives were multiplied by the determinant, which does not alter their signs, to yield the preceding expressions.

Table 2. Potential biomass responses to increased extrinsic per capita mortality in food chain models

1. No adaptation or density dependence

	d_1	d_2	d_3
R	−	0	−
N	0	0	+
P	−	−	−

2. Density dependence but no adaptation

	d_1	d_2	d_3
R	−	+	−
N	−	−	+
P	−	−	−

3. Adaptation but no density dependence

	d_1	d_2	d_3
R	−	+ or −	−
N	+ or −	−	+ or −
P	−	+ or −	−

4. Adaptation and density dependence

	d_1	d_2	d_3
R	−	+ or −	−
N	+ or −	+ or −	+ or −
P	+ or −	+ or −	+ or −

5. Adaptation and density dependence: linear consumer fitness

	d_1	d_2	d_3
R	−	+ or −	−
N	+ or −	−	+ or −
P	−	+ or −	−

Trophic level responses in the presence of density dependence and adaptive behaviour or evolution within a homogeneous consumer population

Our analysis assumes that the system consisting of equations (1a–d) has an equilibrium with positive finite values of all four variables, and that that point is locally stable. Cycles may occur under equations (1) (Abrams, 1990) if the adaptive change in C is sufficiently rapid, and if the birth rate function, b, is strongly saturating (i.e. b has a large negative second derivative). The following results may not apply under these conditions. The existence of an equilibrium often requires that there is a minimum capture rate of resource, C, even when vulnerability to the predator, s, is as low as possible (Abrams and Chen, 2002). Table 1 provides expressions that determine the signs of the changes in densities and trait value with extrinsic mortality rates in this general case, and matrix 4 of Table 2 gives the potential signs of each response. Although these expressions are rather complicated, their signs depend on three key quantities, discussed below.

The curvature of consumer fitness with respect to the trait

The first of these three quantities is the curvature (second derivative) of the consumer's fitness as a function of the trait, C (i.e. $R^2b'' - \delta_2'' - s''P$) evaluated at the equilibrium point. This expression is a component of all nine formulae in Table 1 describing changes in populations with extrinsic mortalities. Because we have assumed that the trait maximizes individual fitness, $R^2b'' - \delta_2'' - s''P$ must be non-positive. A strongly curved fitness function means that this second derivative has a large negative value, and that there is strong stabilizing selection on the trait. Under these circumstances, C does not change greatly, and trait-mediated effects are small.

Relative sensitivity of predation risk and foraging gain to the trait

The second quantity that appears in many of the expressions for responses to extrinsic mortality is $Cs' - s$. This expression measures the relative sensitivity of predator vulnerability and foraging ability to the trait; it has the same sign as the derivative of the ratio (s/C) with respect to C. If this derivative, given by $(Cs' - s)/C^2$, is positive, an increase in C will imply a greater than proportional increase in s; thus a change in the trait will have a relatively greater impact on the consumer's interaction with its predator than its interaction with its resource. The term $Cs' - s$ only affects the response of a species to mortality at an adjacent level, and its sign reflects the relative sizes of the indirect effects due to changes in the coupled consumption rates, C and s. A large minimum vulnerability s generally makes s/C decline with C.

The non-linearity of the relationship between resource intake and fitness

The third quantity that is important in determining the sign of the responses of densities to extrinsic mortality is the expression $b' + CRb''$. This is equivalent to the derivative of CRb' with respect to CR, and is indicative of the non-linearity of the relationship between resource intake (CR) and consumer fitness. The sign of the expression $b' + CRb''$ determines whether an increase in resource density increases the optimal C (expression is positive) or decreases the optimal C (expression is negative) (Abrams, 1991a,b). Thus, the sign of this quantity can be determined by studying the short-term responses of foraging effort to food availability. A survey of responses of foraging traits to greater food density by Werner and Anholt (1993) suggests that many organisms decrease foraging effort when food becomes more abundant, although there are exceptions to this pattern (noted in Abrams, 1991b), and evolutionary trade-offs are largely unknown.

The three quantities discussed above, together with the strengths of direct density dependence (the slopes of the relationships between density and per capita mortality of the top two levels, α' and γ'), are sufficient to determine which of the potential signs given in matrix 4 of Table 2 actually describes the interaction. All three quantities can be estimated by measurements of functional and numerical responses; they do not require any fundamentally new type of empirical work. Only two of the entries in matrix 4 have a sign that is independent of the magnitudes of the three key quantities; R must decrease with increases in either d_1 or d_3. If there is adaptation, but no density dependence, responses of abundances to mortalities are described by matrix 3. A model with both processes, when all component functions are linear, is described by matrix 5. The general results of matrix 4 show that the combination of adaptation, direct density dependence in species above the basal level, and non-linearity implies a large number of potential responses that do not occur in models with none, one or two of these factors. The somewhat abstract results of Tables 1 and 2 are interpreted below.

Does middle-level adaptation alone change the nature of trophic cascades?

It is of interest to know how adaptation alone would alter trophic cascades (operationally defined here as the responses to mortalities on top or bottom levels). The responses with adaptive change alone can be derived from Table 1 by setting all terms involving the density dependent functions, γ and α, to zero. The results are summarized in matrix 3 of Table 2. Mortality applied to either predator or resource has the same qualitative effects on R and P predicted by models with density dependence and no adaptation – that is, the standard trophic cascade results for those species. However, unlike standard models, the consumer

here may either increase or decrease in response to mortality on top or bottom levels. The 'non-standard' decrease in N following increased mortality of the predator is most likely if the vulnerability to foraging effort ratio, s/C, increases with C, if birth rate saturates with increasing food intake, $b'' < 0$, and if resource density dependence is weak (f' close to zero). The 'non-standard' increase in N following increased mortality of the resource is likely if $b'' \geq 0$ and s/C decreases with C. Because opposite conditions promote non-standard top-down and bottom-up effects on N, it is very unlikely that both top-down and bottom-up effects on N would be non-standard.

The effects of consumer mortality on both resource and predator densities can be positive or negative when there is adaptation but no density dependence. It is possible that both are positive, both are negative, or that they have opposite signs. In the absence of adaptation, higher d_2 can only increase R and decrease P.

Counterintuitive responses

Mid-level adaptation alone, or the combination of such adaptation with density dependence, can lead to a number of counterintuitive responses: levels can increase in abundance in response to an increase in their own mortality; predators can increase in response to mortality of their prey; prey can decrease in response to mortality on their predators. This section outlines when such responses occur under equations (1).

The predator, P, may increase with increases in its own death rate, d_3, when $b' + CRb'' < 0$ (i.e. the optimum C decreases with R), consumer interference is relatively strong and resource density dependence is relatively weak. This increase in P is a consequence of an increase in C and hence s, following increased predator mortality. The predator's increased food intake more than compensates for its greater mortality. An increase in P with greater consumer mortality d_2 may occur if $b' + CRb''$ and $Cs' - s$ have the same signs and resource density dependence is weak (f' small in magnitude). If both $b' + CRb''$ and $Cs' - s$ are positive – for example, the increased consumer foraging (C) that results from an increase in R (following increased d_2) – combined with a larger than proportional increase in s with C leads to a greater availability of the consumer to the predator.

The consumer, N, may increase with increases in its mortality, d_2, when predator density dependence is strong, resource density dependence is weak and $b' + CRb'' < 0$. Increased mortality then decreases over-exploitation of the resource, over-compensating for the increase in mortality. The predator's numerical response is limited because of its own direct density dependence. These circumstances are also likely to produce a decrease in the consumer population with increased predator mortality, for similar reasons.

The resource, R, may decrease with an increase in the mortality of its own predator, the consumer species. This outcome requires that the ratio s/C decline with increasing C and that the predator's density dependence not be too strong. These conditions lead to an increase in C with higher d_2, which causes the decrease in R.

Consequences of increased mortalities for the equilibrium foraging effort, C

The short-term responses of the optimal C to changes in R or P are well known for the class of models studied here (Abrams, 1991a); C should decrease in response to an increase in P, and either increase or decrease in response to increased R, with the sign of the response given by $b' + CRb''$. In a food chain experiencing altered mortality, these short-term predictions may be changed by the longer-term changes in resource and predator densities. The key quantity, $b' + CRb''$, still influences the response of the equilibrium C to each

mortality, but indirectly caused changes in density also have an influence. Thus, for example, increasing resource death rate, d_1, may decrease foraging when $b' + CRb'' > 0$, but the resulting decrease in predator density may reverse that response.

Responses in linear systems

A model with density dependence and adaptation in which all components of the consumer fitness function are linear is of special interest. This is because it is equivalent to some analogous models with two consumer species, as is shown in the analysis of equations (2) below. In addition, all of the expressions in Table 1 simplify considerably in the linear case; these responses are summarized in matrix 5 of Table 2. Five of the nine responses to mortality applied to a single level have a definite sign in the linear case, while the remaining four have a sign that depends on how the ratio of vulnerability to foraging effort, s/C, changes with C (i.e. the sign of $Cs' - s$).

Interaction of density dependence and adaptive change

Density dependence and adaptive change interact in subtle and rather complicated ways to determine the responses of this tritrophic system to changes in mortality rates. In most cases where density dependence affects the direction of the response to mortality in Table 1, it can either increase or decrease the magnitude of the response, often depending on the shape of the relationship between fitness and resource intake. When $b' + CRb'' < 0$, the behavioural response of the middle species to increased resource density reduces its availability to the predator, which has an effect opposite to its own increase in population size: direct density dependence modulates the population response and, therefore, can change the sign of the overall response.

It is simple to construct models in which some of the responses discussed above are realized. Figure 1 is an example of responses to consumer mortality in a linear model

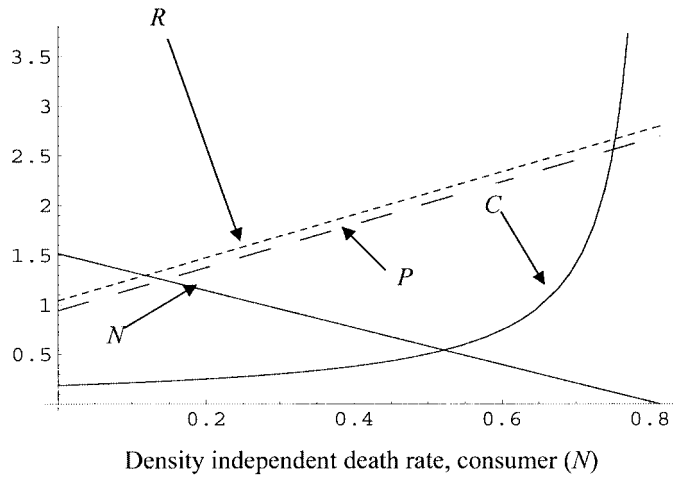

Fig. 1. The responses of resource (R), consumer (N), predator (P) and the consumer's trait (C) to an increasing mortality rate of the consumer in a linear system in which vulnerability increases rapidly with foraging effort (i.e. $Cs' > s$). The parameter values are: $r = 1$; $K = 4$; $C_0 = 0.3$; $\gamma = 0.2N$; $S_0 = 0.01$; $S_1 = 1$; $d_1 = 0$; $d_3 = 0.3$; $\delta_2 = 0.1$; $B_1 = B_2 = E = 1$. The predator increases as its prey's mortality is raised because of the prey's increased foraging, which implies greater predator vulnerability.

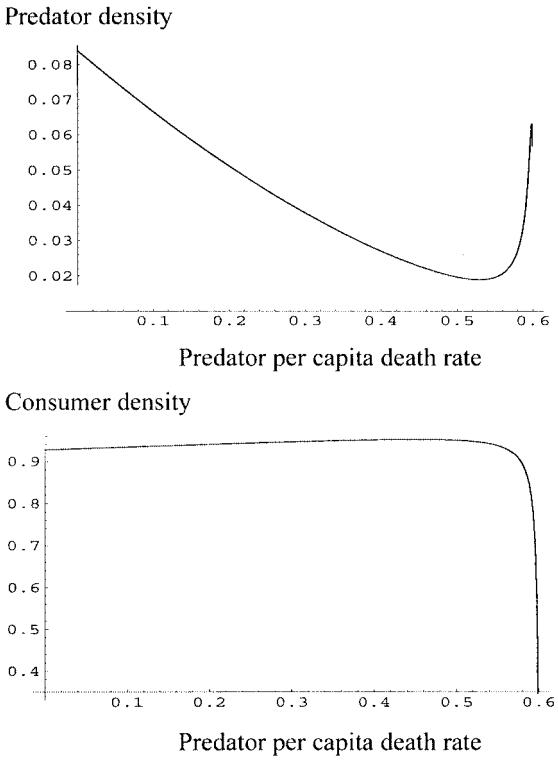

Fig. 2. The responses of consumer and predator abundances to increased per capita mortality of the predator population in the model described by equations (9) in Abrams (1995). The parameter values used in the calculations were: $B_3 = 0.75$; $B_2 = 2$; $B_1 = 1$; $\alpha_1 = 0.25$; $\alpha_2 = 1$; $D_2 = 0.1$; $D_3 = 0.3$; $k = 1$. Per capita density-independent mortality of the predator reduces the parameter B_3 in that model.

in which vulnerability increases rapidly with resource consumption, such that $Cs' > s$. As predicted for this case, increased mortality of the middle-level species increases the predator and resource populations. The equilibrium predator population is able to increase until mortality completely eliminates its prey because the prey's vulnerability to predation, s, increases at an accelerating rate with increased mortality. Figure 2 illustrates changes in predator and consumer abundances in a behavioural model first presented in Abrams (1995: equations 9 and 10). This model is characterized by a per capita growth rate function of the consumer that meets the criterion $b' + CRb'' < 0$; that is, consumer foraging decreases in response to greater resource densities. Here, when mortality is high, further increases in mortality of the predator can increase its own density and decrease that of the consumer species.

Trophic level responses when there are two consumer species

This section examines the first of two alternative models of middle-level adaptation – that is, shifting species composition. Several special cases of the two-consumer system have been analysed previously (Abrams, 1993; Leibold, 1996), but these have not considered the effect

of mortality at all three levels of the system, and most models have failed to include higher-level density dependence. Here we modify equations (1a–c) by adding a second consumer, and simplify the system by assuming that consumer birth rate increases linearly with food intake. This yields:

$$\frac{1}{R}\frac{dR}{dt} = f(R) - C_1N_1 - C_2N_2 - d_1$$

$$\frac{1}{N_i}\frac{dN_i}{dt} = BC_iR - d_{02} - \delta_{i2} - s_iP - G(N_i + N_j) \qquad i = 1, 2 \tag{2}$$

$$\frac{1}{P}\frac{dP}{dt} = E(s_1N_1 + s_2N_2) - d_3$$

We have assumed that the birth rate functions for the consumers have a common constant of proportionality, B, and that the energetic values of both prey to the predator are identical (and given by E). These assumptions reflect the lack of effect of C on either b or E in the corresponding behavioural model. Similarly, the consumer interference function, γ, is now simply a proportionality constant, G, multiplied by total consumer density. We have omitted the predator density dependence function, α, because it does not qualitatively alter any of the results. The two consumer per capita death rates have been expressed as the sum of a common component, d_{02}, and a component that is unique to the consumer species, δ_{i2}. The latter can be thought of as reflecting the trait-dependent effects. This decomposition of the mortality rates allows us to examine the system responses to identical increases in the extrinsic per capita mortality rates of both consumers by increasing d_{02}. The analysis of this system is simple because it can be transformed into the linear trade-off system considered in the previous section (see Appendix). As a consequence, all of the responses of trophic levels to mortality rates are identical to the linear continuous trait model (matrix 5 in Table 2). This equivalence of the models should not be surprising, because the two-species model can be considered as an evolutionary model in which an asexual population has two clones that differ in their values of the resource capture rate, C, their per capita death rate, d, and their vulnerabilities to a common predator, s. The condition that $Cs' > s$ in the behavioural model is equivalent to the condition in the two-species model that the species with the greater resource capture rate and vulnerability (say species 1) is also characterized by having a greater ratio s/C (i.e. $s_1/C_1 > s_2/C_2$ when $s_1 > s_2$ and $C_1 > C_2$). The other possibility is that $s_1/C_1 < s_2/C_2$ when $s_1 > s_2$ and $C_1 > C_2$, which is equivalent to $Cs' < s$ in the behavioural model.

In general, the component fitness functions in two-consumer models need not be linear. In the non-linear case, the equilibrium of the behavioural model is equivalent to that in a multi-prey model in which there is a continuous range of prey species whose values of C, s and δ_2 all lie on the trade-off curve assumed in the behavioural model. The assumption that there is an intermediate behaviour that maximizes fitness is equivalent to the assumption that the trade-off curve relating these parameters in different species is also concave downward.

Trophic level responses when consumers have inducible defences

This second alternative model of adaptation reflects an induced defence that is expressed in an all-or-none manner. Because of the presence of two phenotypes, the mean values of C, δ and s must all lie on a line connecting the two extreme values. If we assume, as in the two-

species model, that the two consumer types have the same efficiency of converting resources to offspring and have the same energetic value, E, to the predator, then the model becomes similar to the two-species model. The only difference is that one type can replace the other by phenotypic change rather than by demographic replacement. The dependence of replacement on fitness is similar in both cases. The equilibrium state of the induced defence model should be identical to that in the two-species model (and the linear behavioural/evolutionary model) provided that the two phenotypes have equal fitness at the equilibrium. If the mechanisms of induction and decay of the better-defended phenotype do not permit exact equalization of fitness, then one would expect slight deviations from the predictions of the two-species model. In addition, if the delay inherent in induction and decay led to population cycles, these cycles might result in an outcome that differed qualitatively from the behavioural model. By assuming that predator self-limitation is absent ($\alpha = 0$), that parents give birth to identical phenotypes, and that the two consumer phenotypes have identical values of B, E and γ, we can construct a model of induced defences that embodies the same trade-offs and structure as the two-consumer model given by equations (2). We require functions, m_{ij}, that describe the rate at which phenotype i transforms into phenotype j. If the transformation rate is an exponentially increasing function of the difference in fitness between the two consumer types, the following model is produced:

$$\frac{\mathrm{d}P}{\mathrm{d}t} = P(E(s_1N_1 + s_2N_2) - d_3) \tag{3a}$$

$$\begin{aligned}\frac{\mathrm{d}N_1}{\mathrm{d}t} = {} & N_1(BC_1R - d_{02} - \delta_{12} - s_1P) + MN_2(\exp[V(B(C_1 - C_2)R - (\delta_{12} - \delta_{22}) - (s_1 - s_2)P)]) \\ & - MN_1(\exp[V(B(C_2 - C_1)R - (\delta_{22} - \delta_{12}) - (s_2 - s_1)P)])\end{aligned} \tag{3b}$$

$$\begin{aligned}\frac{\mathrm{d}N_2}{\mathrm{d}t} = {} & N_2(BC_2R - d_{02} - \delta_{22} - s_2P) - MN_2(\exp[V(B(C_1 - C_2)R - (\delta_{12} - \delta_{22}) - (s_1 - s_2)P)]) \\ & + MN_1(\exp[V(B(C_2 - C_1)R - (\delta_{22} - \delta_{12}) - (s_2 - s_1)P)])\end{aligned} \tag{3c}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = Rf(R) - C_1RN_1 - C_2RN_2 \tag{3d}$$

The parameters that are common to this model and the two-consumer model (equations 2) have the same meaning. The rates at which the two consumer types transform into each other are described by the exponential terms. The constant M is the rate of transformation of one type into the other when the two types have equal fitness, and the constant V scales the exponential rate at which transformation increases with a difference in fitness. If the two types of consumers are not equally abundant when their fitnesses are equal, then the two transformation terms will be unequal, and the less abundant type will also have a slightly lower fitness at the equilibrium. Otherwise, the equilibrium should be close to that in the two-species model, and should respond to changes in per capita mortality in the same way. If M is small and V is large, the difference between the equilibrium point of this and the preceding two-consumer model should be minimal because the transformation rate is quite small when fitnesses are equal. Figure 3 compares the response of total consumer density to an increase in predator mortality rate in analogous models based on two species

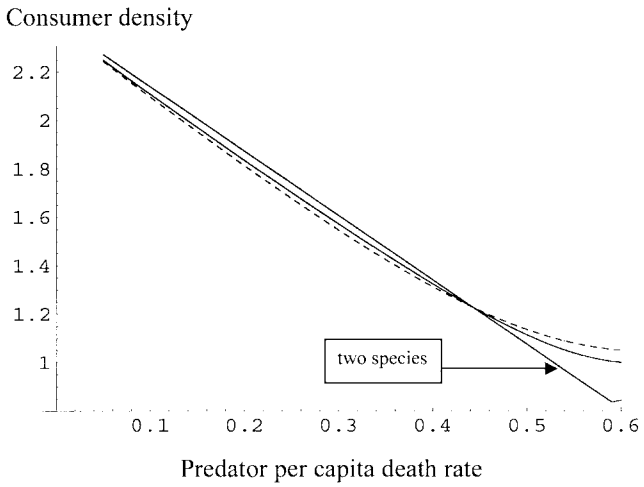

Fig. 3. The response of total consumer abundance to increased per capita mortality of the predator population in the induced defence model given by equations (5), with logistic growth of the resource: $f(R) = rR(1 - (R/K))$. The two curved lines give results for two different values of the constant, M, which gives the transformation rate per individual when the fitnesses of the two types are equal; the dashed line is for $M = 0.5$, while the solid line assumes $M = 0.05$. The straight line (labelled) gives the response of a comparable model in which there are two species with different vulnerabilities and resource capture rates; this line begins to increase at the far right-hand side because the better defended species goes extinct at that point. The other parameter values are: $E = 1$; $B = 1$; $r = 1$; $K = 4$; $V = 5$; $G = 0.1$; $C_1 = 1$; $C_2 = 0.3$; $s_1 = 0.71$; $s_2 = 0.01$; $d_{02} = 0.01$; $\delta_{12} = \delta_{22} = 0.1$.

(equations 2) and induced defences within a species (equations 3); two values of M are illustrated for the latter model. The densities are quite similar for all three cases, even when the transformation rate constant, M, is relatively large.

There is no simple equivalence between the dimorphic model considered here and a continuous trait (i.e. behavioural or evolutionary) model when the component fitness functions are non-linear. However, if it were possible to have a continuous range of phenotypes, rather than an all-or-none induction, the resulting polymorphic induced defence model should approximate a model with behavioural variation.

DISCUSSION

Predicting and explaining the responses of biomass to altered mortality in tritrophic systems

One of the main roles of simple models, such as those considered here, is to suggest potential mechanisms for observed changes in ecosystems. If, for example, all trophic levels decrease in abundance, previous theory (matrix 2 of Table 2) suggests that a decline in resource growth conditions is the most likely cause. However, increased mortality of the predator can have the same effect when there is adaptive change at the middle level and direct density dependence at all levels. We see the primary role of the simple theory developed here as a tool for hypothesis generation when trying to determine the causes of observed changes in the abundances of different trophic levels.

If there is both adaptation and density dependence, seven of the nine possible responses of the three trophic level abundances to the three level-specific mortality rates have an indeterminate sign (see matrix 4 in Table 2). However, the indeterminacy does not imply unpredictability. Knowing the strength of intraspecific density dependence and three quantities related to the consumer's adaptation to other levels often permits all of these signs to be determined. The three aspects of consumer level adaptation are: the curvature of the relationship between fitness and foraging effort (or the sensitivity of the optimal foraging effort to food and predator densities), the change in the ratio of predator vulnerability to foraging effort with an increase in effort, and the curvature of the function relating resource intake to consumer per capita population growth. The third quantity also determines the direction of change in optimal foraging effort with food abundance. Real ecosystems are vastly more complicated than these models. However, these three quantities are expected to have a major influence on biomass responses to mortality in systems that are more complicated than our simple models.

All three of the sign-determining quantities, and the strength of direct density dependence, are measurable in many systems, either directly or indirectly. The parameters C and s represent slopes of functional responses, and the change in their ratio with changes in C should be measurable with enough functional response measurements. The shape of b, the relationship between per capita growth rate and resource intake, should be measurable by experimentally varying resource availability. The sign, and a rough estimate of the magnitude of $b' + CRb''$, can be obtained from the sign and magnitude of the short-term response of consumer foraging effort to increased food density. Measurements of immediate responses of foraging effort to food density have been carried out in many previous studies (for reviews, see Werner and Anholt, 1993; Lima, 1998). Measuring the shape of the relationship between the fitness function and foraging effort requires that the latter be manipulated. However, information about the magnitude of the curvature of the function is also reflected in the degree of response of C to changes in P or R. Either a lack of capacity to adapt or a strongly curved fitness function is implied if the foraging trait shows little change in response to manipulation of either food or predator density. Either case leads to the simple determinate responses given by matrix 2 in Table 2. The direct effects of density on population growth can be measured using experiments that keep food and predator density constant while varying consumer density (e.g. Ganter, 1984). The population dynamic consequences of either adaptation or species replacement can be studied in a variety of organisms with short generation times (Bohannan and Lenski, 1999; Persson *et al.*, 2001; Matz *et al.*, 2002; Shertzer *et al.*, 2002; Vos *et al.*, 2002).

We have argued that the response structures of tritrophic systems in which similar types of consumer foraging adaptation are produced by different mechanisms should be similar. The dimorphic models are equivalent, in their long-term response of abundances to mortalities, to analogous continuous trait models in which the foraging–predation trade-off is linear. Non-linear models of behavioural adjustment are equivalent in the same way to models in which there is either evolution or species replacement, given that the underlying trade-off is identical.

Does increased mortality have the same effect as species addition/removal?

Because there are limits to the magnitude of adaptive change (set by limits to behavioural flexibility or to the range of characteristics of different potential consumer species), if

mortality rates are changed sufficiently, it is likely that adaptive change will cease because one form or phenotype is favoured to the exclusion of the other(s). Further changes in mortality will then have the effects expected in the absence of adaptation. This means that the direction of population responses to mortality will often shift when death rates become large enough. Moderate and extreme changes in mortality at one level may have opposite effects on the abundances of other levels. Another general lesson from the models presented here is that imposing mortality on a given trophic level may have qualitatively different effects than directly decreasing the abundance of that level. This is clear, because increased mortality may increase the abundance of the manipulated species. Even when that is not the case, there are many cases where press perturbations of the density of a species (as performed in Kneitel and Miller, 2002, for example) cause density changes opposite to those caused by the corresponding (increase or decrease) in mortality rates of that species. This is important, since alterations in mortality are generally much easier to carry out than are true press perturbations, where densities are changed and then maintained at constant levels.

Potential examples of different biomass responses and predictions

If the models of adaptive change analysed here are reasonable descriptions of tritrophic systems, then there should be some examples of trophic level abundances responding to mortality rates in directions other than those given in matrices 1 and 2 of Table 2, which lack adaptive change. In fact, examples of alternative responses are available for both aquatic and terrestrial systems (e.g. Lynch and Shapiro, 1981; Vanni, 1987; Brett and Goldman, 1996; Leibold *et al.*, 1997; Halaj and Wise, 2001; Shurin *et al.*, 2002). Predator removal may decrease herbivory (see Leibold *et al.*, 1997, and references therein; Halaj and Wise, 2001), which should not occur according to matrices 1 and 2 in Table 2, but is consistent with matrices 3–5 that include the possibility of adaptive change. The models with adaptation that we have analysed predict that the bottom two trophic levels (plants and herbivores) can respond in the same direction in response to altered mortality at the top level. Equivalent models without adaptation do not allow this (matrix 2). Leibold *et al.* (1997, fig. 5, p. 480) show a significant number of cases where the abundances of these two levels changed in the same direction in response to a fish manipulation (i.e. a top-down effect). We do not know of any studies that have demonstrated that the types of responses we predict were due to adaptive change in trophic characters within species. However, heterogeneity in edibility within tropic levels is often invoked to explain such non-standard trophic responses to enrichment and predator treatments (e.g. Balčiūnas and Lawler, 1995; Bohannan and Lenski, 1999; Steiner, 2001). Compositional changes within trophic levels are a likely explanation for alternative biomass responses in other cases as well (Leibold *et al.*, 1997; Chase *et al.*, 2000).

Many aquatic systems consist of nutrients, several types of algae differing in their vulnerability to consumers, and herbivores (Porter, 1977; Carpenter *et al.*, 1987; Leibold, 1996). The variation in algal edibility permits adaptation at that level. These systems should be candidates for observing increased herbivore abundance with increased mortality of all algae. Dreissenid mussels have greatly increased the mortality of most phytoplankton, and there is evidence to suggest that they may have increased or not significantly changed zooplankton density in many lakes, contrary to the expectations of previous theory (Noonburg *et al.*, in press).

We predict that many of the anomalous responses observed in previous studies will prove to be based on the interaction of changes in consumption-related traits of mid-level species with density dependence in mid- and/or top-level species. The review of experimental studies of 'top-down' and 'bottom-up' effects by Leibold *et al.* (1997) contains many examples where observed responses do not accord with theory that ignores adaptive change. These examples represent promising candidates for determining whether the changes in density can be explained using the theory presented here. We also predict that species that decrease foraging effort in response to increases in food density will often be involved in counterintuitive responses of abundance to mortality (including responses in their own abundance or their prey's abundance). We hope that these predictions will help spur research that measures both adaptive foraging responses and the responses of trophic level abundances to mortality rates.

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APPENDIX: EQUIVALENCE OF A LINEAR BEHAVIOURAL MODEL AND THE MODEL WITH TWO CONSUMER SPECIES

If there is a mixture of the two consumer species in equations (2), where we assume $C_1 > C_2$, the average consumption rate in the combined consumer population is

$$\overline{C} = \frac{C_1 N_1}{N_1 + N_2} + \frac{C_2 N_2}{N_1 + N_2} = C_1 q + C_2(1 - q) \tag{A1}$$

where q is the proportion of type-1 individuals in the population ($q = N_1/(N_1 + N_2)$). Similarly, the average trait-dependent per capita death rate and vulnerability are:

$$\bar{\delta}_2 = \delta_{12} q + \delta_{22}(1 - q) \qquad \bar{s} = s_1 q + s_2(1 - q) \tag{A2}$$

These relationships allow the average values of δ_2 and s to be expressed as a function of the average ($\bar{C}$), minimum (C_2) and maximum (C_1) values of C:

$$\bar{s} = \frac{s_2 C_1 - s_1 C_2}{C_1 - C_2} + \frac{(s_1 - s_2)\bar{C}}{C_1 - C_2} \tag{A3a}$$

$$\bar{D}_2 = \frac{\delta_{22} C_1 - \delta_{12} C_2}{C_1 - C_2} + \frac{(\delta_{12} - \delta_{22})\bar{C}}{C_1 - C_2} \tag{A3b}$$

Then, equations (2) in the text may be rewritten as

$$\frac{1}{R}\frac{\mathrm{d}R}{\mathrm{d}t} = f(R) - \bar{C}N_{\mathrm{tot}} - d_1 \tag{A4a}$$

$$\frac{1}{N_{\mathrm{tot}}}\frac{\mathrm{d}N_{\mathrm{tot}}}{\mathrm{d}t} = B\bar{C}R - d_{02} - \bar{\delta}_2 - \bar{s}P - GN_{\mathrm{tot}} \tag{A4b}$$

$$\frac{1}{P}\frac{\mathrm{d}P}{\mathrm{d}t} = E\bar{s}N_{\mathrm{tot}} - d_3 \tag{A4c}$$

with an additional equation for the rate of change of the mean value of C (which determines the mean δ_2 and s):

$$\frac{\mathrm{d}\bar{C}}{\mathrm{d}t} = (\bar{C} - C_2)(C_1 - \bar{C})\left(BR - \frac{\delta_{12} - \delta_{22}}{C_1 - C_2} - P\frac{s_1 - s_2}{C_1 - C_2}\right) \tag{A5}$$

where the ratio of the difference in δ_2's or s's to the difference in C's is equivalent to the derivative of the mean δ_2 (or s) with respect to the mean C (see equations A3). Thus, for the two-species model, equations (2) are equivalent to a version of equations (1) in which the functions and trade-offs are linear.