The impact of mortality on predator population size and stability in systems with stage-structured prey

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Abstract

The relationships between a predator population’s mortality rate and its population size and stability are investigated for several simple predator–prey models with stage-structured prey populations. Several alternative models are considered; these differ in their assumptions about the nature of density dependence in the prey’s population growth; the nature of stage-transitions; and the stage-selectivity of the predator. Instability occurs at high, rather than low predator mortality rates in most models with highly stage-selective predation; this is the opposite of the effect of mortality on stability in models with homogeneous prey populations. Stage-selective predation also increases the range of parameters that lead to a stable equilibrium. The results suggest that it may be common for a stable predator population to increase in abundance as its own mortality rate increases in stable systems, provided that the predator has a saturating functional response. Sufficiently strong density dependence in the prey generally reverses this outcome, and results in a decrease in predator population size with increasing predator mortality rate. Stability is decreased when the juvenile stage has a fixed duration, but population increases with increasing mortality are still observed in large areas of stable parameter space. This raises two coupled questions which are as yet unanswered; (1) do such increases in population size with higher mortality actually occur in nature; and (2) if not, what prevents them from occurring? Stage-structured prey and stage-related predation can also reverse the ‘paradox of enrichment’, leading to stability rather than instability when prey growth is increased.

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1. Introduction

A large body of ecological theory investigates interactions between homogeneous populations. However, no natural population is truly homogeneous, and developmental changes in size usually entail changes in the foods and predators of the individuals in question (Metz and Diekmann, 1986; Ebenman and Persson, 1988; Werner, 1988). A large fraction of organisms undergo radical changes in physiology or morphology as they proceed through their life history (Ebenman and Persson, 1988). Thus, there is a legitimate worry regarding the applicability of simple models of homogeneous populations to natural systems. Size is one of the traits most frequently used to classify individuals in heterogeneous populations, because size is a major determinant of ecological interactions (Cohen et al., 1990). There are a variety of special cases where it has been shown that age- or size-structured models of interactions between species can make predictions that differ from those of models of homogeneous populations (Oster, 1976; Hastings, 1983; McNair, 1995; McCann and Yodzis, 1998; DeRoos et al., 2003). However, many aspects of the interactions between structured populations are poorly understood. Only a small fraction of the ecological questions addressed by models of homogeneous interacting species have been examined using models of structured populations. An
important relationship that has yet to be examined using models with stage structure is that between the mortality rate of a predator and its population size and dynamics.

It has long been known that increasing a predator’s mortality rate can increase its population size (Rosenzweig and MacArthur, 1963). This occurs in unstructured models when a vertical predator isocline intersects the prey isocline to the left of the maximum of the prey isocline (Rosenzweig and MacArthur, 1963). However, unstructured models in which the predator’s per capita growth rate is purely a function of prey density predict that the condition for a stable equilibrium is identical to the condition that the predator’s equilibrium density declines as its per capita mortality rate increases (Abrams, 2002). Conversely, if the model predicts an unstable equilibrium, the predator’s equilibrium density will increase with sufficiently small magnitude increases in its mortality rate. More recent work on similar predator–prey models has suggested that the predator population size may frequently increase as its own mortality rate increases in stable, as well as unstable systems. This outcome occurs when the standard predator–prey model is modified to allow adaptive change in a trait that predict that the condition for a stable equilibrium is identical to the condition that the predator’s equilibrium density declines as its per capita mortality rate increases (Abrams and Walters, 1996). The results described in the preceding paragraph leave open the question whether counterintuitive increases in predator population size with predator mortality can occur in stable systems in the absence of adaptive change, but in the presence of population structure that affects the interaction. Population structure with stage-related predation can result in effective refuges, which can often stabilize interactions (Abrams and Walters, 1996). The importance of adaptive change in characteristics on interactions within ecological communities is still somewhat controversial (Bolker et al., 2003). In contrast, there is little argument about the importance of size–structure in determining interactions in natural food webs (Cohen et al., 1990), and particularly in predator–prey systems involving fish (McCann and Yodzis, 1998; DeRoos and Persson, 2002; DeRoos et al., 2003; Walters and Martell, 2004). The present article explores the potential for increasing population size as the result of increased mortality in predator–prey systems in which the prey has a simple form of size or stage structure (size–structure in the predator is also discussed briefly). Models differ in assumptions regarding density dependence in the prey and the nature of the predator’s selectivity and its functional response.

2. Models

The analysis of stage structure presented here centers on predator–prey systems, in which one species (generally the prey) has 2 stages, with the population dynamics of each stage represented by an ordinary differential equation. Such models are not as flexible, and often not as realistic as delay-differential equation models having distributed delays (Nisbet, 1997). However, the ordinary differential equation models are accurate representations of distributed-delay models with exponentially distributed residence times within stages. Furthermore, stability analysis is relatively easy to carry out, unlike the case with most systems with distributed delays. Alternative stage structures are considered more briefly, including the cases of a fixed interval and gamma distribution of the juvenile stage lengths.

The analysis begins with a general framework in which the functional forms of the density-dependent fitness components in the prey and the predator’s functional response are unspecified. The analysis shows that the conditions for local stability of an equilibrium and for the predator to decrease in numbers when mortality increases, are no longer identical. In particular, predator numbers often increase with greater mortality in stable systems. This section is followed by analysis of several models with specific functions for predator functional response and prey density-dependence. These specific models begin with density-independent prey growth, and illustrate that the mortality-induced increase in predator population occurs frequently, given some simple and widely used population dynamical assumptions.

2.1. A general density-dependent model

The model considered here is described by the following equations:

\[
\frac{dN_1}{dt} = b_2(N_2)N_2 - d_1 N_1 - g(N_1)
\]

\[
- s_1 N_1 PH(e_1 s_1 N_1 + e_2 s_2 N_2),
\]
\[
\frac{dN_2}{dt} = gN_1 - d_2N_2 - s_2N_2PH(e_1s_1N_1 + e_2s_2N_2),
\]
\[
dP = P((e_1s_1N_1 + e_2s_2N_2)
\times H(e_1s_1N_1 + e_2s_2N_2) - D),
\]
where the variables, functions, and parameters are defined as follows: \(N_1\) and \(N_2\) are the population densities of juvenile and adult prey, \(P\) is the population density of the predator, \(b_2\) is the per capita birth rate of adult prey, which is a function of the density of adult prey, \(d_1\) and \(d_2\) are parameters giving the per capita death rates of juvenile and adult prey, \(D\) is the per capita death rate of the predator, \(g\) is the per capita transition (growth) rate of juveniles to adults in the prey population, \(s_i\) is the per capita capture rate of prey in class \(i\) by a searching predator and \(H\) is a function describing predator satiation (the proportional decrease in capture rate relative to predator that encounters vanishingly few prey) as a function of a weighted sum of its encounter rates of the two prey-stage classes. \(H\) is a decreasing function with a maximum value of 1 when its argument is 0. \(e_i\) is the conversion efficiency of ingested prey of class \(i\) into new predator individuals; \(e_i\) also represents the relative nutritional value of an individual of prey type \(i\) to the predator.

Density dependence in the birth rate reflects the assumption, common in fisheries, of a density-dependent stock-recruitment relationship. However, most of the qualitative results based on Eq. (1) do not depend on the locus of density dependence. The equilibria of Eq. (1) differ in their stability properties from the equilibria of an analogous model with homogeneous prey. These differences are particularly pronounced when the predator interacts differently with the two size classes. Because analysis of the general model becomes algebraically complicated, and because the goal is to compare stage-specific to indiscriminate predation, this section will treat the case of predation that is restricted to the juvenile prey class. The case with adult-only predation also produces differences between stability conditions and conditions for a hydra effect; see the section on alternative models below.

The question addressed here is whether the conditions for local instability of an equilibrium of Eq. (1) correspond to the conditions that make predator abundance increase with increasing mortality \(D\). To simplify the results, we assume that \(e_1 = e_2 = 1\). Proportional increases in the \(e_i\) and \(D\) do not change the equilibrium densities in this model. A larger \(e_i\) increases the range of values of \(D/e_i\) that yield a stable equilibrium. However, this effect is small in all numerical work we have done, and for simplicity, \(e_i = 1\) is assumed below. The condition for an equilibrium with positive densities of both predator and prey to be locally stable is then
\[
s_1b_2gH'(s_2 - d_2)\left((b_2 - d_2)g - d_1d_2\right)
+ H's_1(b_2 - d_2)g - d_1d_2\right)
\times [d_2^2 + b_2d_2g + 2b_2Dg - d_2DH(d_1 + g)]
+ H^2(s_1/H)D(d_2 + D)((b_2 - d_2)g - d_1d_2)^2
- b'g^2[d_2^2H^2 - d_2D(d_1 + g)H']
+ b_2g(H^2 + DH')] > 0.
\]

The analysis of this condition in the appendix establishes that: (1) a system with a linear functional response and no density dependence \((H' = 0\) and \(b'_2 = 0\)) is always stable. A system with no density dependence is stable if the satiation function has a sufficiently shallow slope \(H'\) small in magnitude. If \(H = 1\) (no satiation), but there is an Allee Effect in the prey \((b'_2 > 0)\) then the system can be stable provided that the Allee Effect is not too strong (i.e. if \(b'_2\) is relatively small in magnitude).

The condition for a hydra effect, i.e. for the equilibrium predator population, \(P\), to increase with its own mortality rate, \(D\), is shown in the appendix to be
\[
\frac{dP}{dD} = \frac{g^2b_2}{d_2s_1e_1(h^2 + DH^2) - d_2s_1e_1(h^2 + DH^2)}
\times [g(b_2 - d_2 - d_1d_2)].
\]

This expression is the difference between two rather complicated terms, both of which are usually negative (see appendix). If there is no density dependence, the first term is zero, and the entire expression is therefore positive for all levels of predator mortality, \(D\). Expression (3) is also positive if there is an Allee Effect. In such a case, there would be a hydra effect at low \(D\), when prey densities are low, but this effect might be reversed at higher values of \(D\), when normal density dependence prevails in the prey population. The equilibrium predator density will increase with a small increase in its own mortality when there is weak density dependence in prey natality, provided the functional response exhibits some saturation \((H < 0)\). If density dependence is sufficiently strong \((b'_2\) is sufficiently large in magnitude) then expression (3) will be negative, and predator numbers decrease as their mortality increases. In addition, the stability condition, Eq. (2), will always be satisfied when \(b'_2\) is large and negative. However, the conditions for stability and predator numbers that decline with mortality are clearly not equivalent. In particular, if there is no density dependence, the equilibrium will often be stable, but predator numbers will increase with increases in their own mortality (again assuming some saturation of the functional response).

Similar expressions have been derived for stability and for the effect of predator mortality on predator population size when one of the other prey demographic
parameters \((g, d_1, d_2)\) is density-dependent, and for models in which the predator specializes on adult prey. Because these analyses are quite similar to the preceding one they are not presented here. They share the characteristics of the above model that stability conditions do not coincide with the conditions for the predator to increase in numbers with an increase in its own mortality. They all show that such a counter-intuitive increase in numbers can occur at a locally stable equilibrium when the predator has a saturating functional response \((H\) is negative) and when the prey has sufficiently weak density dependence. Such an increase can also occur with a non-saturating response provided the prey’s per capita growth rate increases with its own population over some range of population sizes.

The general analysis presented above cannot easily reveal whether the predicted ‘hydra effect’ is large in its maximum magnitude, or whether it occurs over a wide or narrow range of parameter values. The following analysis pursues several models that make specific assumptions about the form of density dependence and the shape of the predator’s satiation function, so that these questions may be addressed.

2.2. Density-independent prey growth: Juvenile prey are vulnerable

Assuming density-independent prey growth allows a number of additional analytical results, and makes it easy to investigate the impacts of changing from stage restricted to generalist predation. In a model with homogeneous populations, the combination of density-independent prey growth and a type-2 predator functional response implies that the equilibrium predator density always increases with increased predator mortality; there is a hydra effect for all parameters. However, it could never be observed because the dynamics of the system are non-persistent; one or both species go extinct, or both populations grow indefinitely. The question addressed here is whether the same conclusion applies to a model with a stage-structured prey population and stage-limited predation. Hastings’ (1983) analysis showed that stability and persistence were possible in structured models. Here, that work is extended to consider the consequences of different predator mortalities. We initially assume that only the first prey stage (juvenile) is vulnerable to the predator, as in Eq. (1). Given a fixed probability per unit time for the transition from juvenile to adult prey, the dynamics of such a system can be described by

\[
\begin{align*}
\frac{d N_1}{dt} &= b_2 N_2 - d_1 N_1 - g N_1 - \frac{s N_1 P}{1 + sh N_1}, \\
\frac{d N_2}{dt} &= g N_1 - d_2 N_2, \\
\frac{d P}{dt} &= P \left( \frac{es N_1}{1 + sh N_1} - D \right).
\end{align*}
\]

The variables and parameters in this model have the same meanings introduced under Eq. (1), except that here, \(h\) is the predator’s handling time per prey item. This corresponds to a satiation function \(H = 1/(1 + s/\lambda N_1)\) in Eq. (1). Because only juveniles are consumed, the subscript 1 has been dropped from the attack rate, \(s_1\) and conversion efficiency, \(e_1\). As in the previous section, it is assumed that \(e = 1\).

The equilibrium densities of Eq. (4) under the above assumptions are:

\[
\begin{align*}
P &= \frac{g(b_2 - d_2) - d_1 d_2}{d_2 s(1 - Dh)}, \\
N_2 &= \frac{D g}{d_2 s(1 - Dh)}, \\
N_1 &= \frac{D}{s(1 - Dh)}.
\end{align*}
\]

The changes in predator and total prey populations with predator mortality are illustrated in Fig. 1. It is clear that the predator’s equilibrium population density increases with its own death rate, \(D\). All densities approach infinity as \(D\) approaches 1/\(h\). In order to have all species present at positive densities it is necessary that \(Dh < 1\) and that \(g > d_1 d_2/(b_2 - d_2)\). Because of the lack of density dependence in the prey, a larger prey population size always implies greater prey productivity. A greater predator death rate increases the prey population size at equilibrium, and hence increases prey production. This can be translated into increased predator numbers because of the predator’s saturating functional response, which reduces the prey’s risk from each predator individual as prey numbers increase.

The necessary and sufficient condition for local stability of this equilibrium of Eq. (4) can be worked out using the Routh–Hurwitz criteria. The general
stability condition can be derived from Eq. (2)

\[ 1 - Dh + \left( \frac{(Dh + d_1 h - 1)\left[d_1^2 + d_2 Dh(g + d_1) + b_2 g(1 - Dh)\right]}{d_2^2} \right) < 0. \]  

(6)

Several properties can be deduced from this inequality. It is necessary that \( 1 > h(D + d_2) \) for the equilibrium to be stable for any values of the other parameters. Because it is necessary that \( 1 > Dh \) for a positive equilibrium point, inequality (6) is usually satisfied unless the predator’s mortality or the mortality of the adult prey is high. The left-hand side of inequality (6) must become positive as \( D \) approaches its maximum value of \( 1/h \), so the equilibrium is unstable when \( D \) is large enough. If the juvenile stage is very short in duration (\( g \) is very large), condition (6) approaches the following: \( D < (1/h) - d_2 \). In this case, the equilibrium given by Eq. (5) is stable for most predator death rates if \( d_2 \) is small relative to \( 1/h \). Condition (6) is always satisfied when \( h = 0 \), although in this case, the (positive) equilibrium predator density is also independent of its mortality.

The left-hand side of the stability condition, Eq. (6), always increases as the predator’s death rate, \( D \), increases. This means that increasing the death rate can only destabilize the system. If the stability criterion is not satisfied for predator death rates close to zero, it will never be satisfied for higher death rates. If \( D \) approaches 0, inequality (6) simplifies to

\[ g > d_1^2 h / (b_2 (1 - d_2 h)). \]  

(7)

Expression (7) implies that the system is unstable for all predator death rates when \( g \), the transition rate to the adult stage, is sufficiently low. If condition (7) is not satisfied, then there will be stable equilibria when the predator death rate \( D \) is low, but unstable equilibria when \( D \) is high. The system described by Eq. (4) is often stable for a very large fraction of the permissible predator death rates. For example, if we set \( d_1 = d_2 = 0.1, \ h = 1, \ g = 0.1, \) and \( b_2 = 1 \), the range of predator death rates that produce a stable equilibrium always spans the majority of the range (>77%) of potential predator death rates (which lie between 0 and 1). Given these birth and death rates and handling time, the maximum predator death rate for stability lies between 0.7702 and 0.9 for all possible growth rates, \( g \). The appendix shows that the maximum \( D \) consistent with a stable equilibrium increases with \( g \) and approaches an asymptotic value at large \( g \). In the special case where the handling time is equal to 1, the maximum \( D \) for a stable equilibrium is \( 1 - d_2 \).

The derivative of the left-hand side of expression (6) with respect to \( g \) is negative provided that \( 1 > h(D + d_2) \); i.e., a larger growth rate makes stability more likely, and a smaller growth rate favors instability. However, \( g \) can become small enough to produce instability only if the death rate of juvenile prey is sufficiently low; if \( d_1 < d_2 h (b_2 - d_2) / (b_2 (1 - d_2 h - Dh)) \). A high enough juvenile death rate implies stability for all values of the growth rate, provided that \( D < (1 - d_2 h) / h \).

Enrichment of the prey population is expected to increase either \( b_2 \), or \( g \), or both of these parameters. It is clear that such enrichment decreases the left-hand side of the stability condition (expression (6)), given the necessary condition that \( 1 > h(D + d_2) \). This stabilizing effect contrasts with the outcome predicted for models with homogeneous predator and prey populations; i.e., Rosenzweig’s (1971) ‘paradox’, according to which enrichment can only destabilize the equilibrium.

The above analysis raises the question whether it is the prey’s class structure per se or the restriction of the predation to one of the classes that results in the stability of this model. One can examine how stability and dynamics change as the relative magnitudes of the two capture constants, \( s_1 \) and \( s_2 \), are made more similar, given that \( s_1 + s_2 \) is constant. As the two capture constants become more similar, instability becomes more likely. Equal predation on both prey results in non-persistent dynamics for most values of the other parameters in this density-independent model. However, stability does not require completely stage-restricted predation. Numerical analysis of local stability in models with moderately high selectivity (ratios of \( s_1/s_2 \) of approximately 3 or greater) showed that they are often stable for approximately the same range of predator death rates as models with complete specialization. In any case, it is clear that some degree of size-selective predation is essential for the stability of the systems described in this section.

The preceding analysis concentrated on the local stability of the internal equilibrium (where all populations are non-zero), and the dynamics for a limited range of initial densities. The domain of attraction of stable equilibria was quite large for parameters that were significantly different from their stability threshold, but the exact domains were not determined. Similarly, the dynamics around unstable equilibrium points were not examined systematically. For the parameters examined, when \( D \) was only slightly larger than the stability threshold, there was a locally attracting limit cycle surrounding the unstable point. Fig. 2A provides an example of the dynamics in such a case. Still larger values of \( D \) produce an outcome in which both prey and predator populations grow arbitrarily large from some or all initial conditions. In this case, the predator is unable to keep up with prey growth; the predator’s saturating functional response means that its maximum per capita growth rate is \((1/h) - D\), while the prey’s per capita growth rate approaches its natural rate of
2.3. Density-dependent models

The analysis in this section is motivated by the fact that strong density dependence in unstructured predator–prey models has the dual effects of stabilizing equilibria and eliminating the hydra effect. We would like to know if the same is true of the simple structured models outlined above. However, there are four parameters in the prey equations that may be density-dependent, and density-dependence may be the result of competition that is general or class-specific. Predation may be limited to juveniles, limited to adults, or some mixture. To limit the range of possibilities, we concentrate on models with class-restricted density effects and predation only on juveniles. Both of these assumptions characterize many aquatic predator–prey systems. Other cases are treated more briefly. We first consider models that reflect the form of the general model, where density-dependence operates via adult birth rate. This is followed by an analysis that adds density dependence in juvenile growth, because this produces quite different prey dynamics. Finally, we consider a range of alternative models more briefly to test the sensitivity of our conclusions to features such as the constant probability of growth into the adult stage.

2.3.1. Adult birth has linear density dependence

This is the simplest realization of the general model given by Eq. (1). In this case, \( b_2 = B_2(1 - zN_2) \), where \( B_2 \) represents the maximum per capita birth rate, and \( z \) is the proportional rate of decrease in per capita births with increased density. The satiation function \( H \) is based on Holling’s disk equation; for predation on juveniles only, \( H = 1/(1 + shN_1) \). Given this simple type of density dependence, it is possible to obtain analytical solutions for the equilibrium predator density, and for the stability criterion. The derivative of equilibrium predator density with respect to death rate is positive for a range of death rates from \( D = 0 \) to

\[
D = \frac{[hsd_2(g(B_2 - d_2) - d_1d_2) - zB_2g^2]}{[h(ssd/g)(B_2 - d_2) - d_1d_2 + zB_2g^2]}.
\]

If the density-dependence parameter, \( z \), is above the threshold value of \([hsd_2(g(B_2 - d_2) - d_1d_2)]/[B_2g^2]\), predator density always decreases with \( D \); there is no hydra effect. The stability condition for this model is quite complicated, but the effect of greater density dependence is to expand the range of \( D \) that results in a stable equilibrium. Locally unstable equilibrium points lead to a limit cycle for most parameters, although chaotic dynamics are still possible at parameter values for which similar dynamics occur in the density-independent model, provided density dependence is sufficiently weak. Fig. 3 plots both the stability boundary and the hydra effect boundary in the parameter space defined by

\[
(1/2)
\]

as its population becomes large relative to the predator’s. Chaotic dynamics occurred for some initial conditions for some parameter values with relatively low prey growth rates and relatively high predator death rates; an example is shown in Fig. 2B. The possibility of unlimited growth in this model makes it biologically inappropriate for analyzing global dynamics. The next several models therefore extend Eq. (4) by considering density dependence in the prey.

Fig. 2. (A) is an example of limit cycle dynamics in a system with density-independent growth of the prey, and the following parameters: \( [h = 1, s_1 = 1, d_1 = 0.1, d_2 = 0.05, b_2 = 1.0, g = 0.05, D = 0.95] \). In this case, the equilibrium becomes unstable when \( D = 0.93768 \). The dashed line is the juvenile prey population size, and the solid line is the predator population size. Fig. 1B illustrates chaotic dynamics in the model with density-independent prey growth. The parameter values are: \( s = 1, h = 1, d_1 = 0.005, d_2 = 0.1, g = 0.003, b_2 = 1, \) and \( D = 0.9 \). The dashed line is juvenile prey and the solid line is the predator population. Extinction of the predator can also occur for initial conditions farther from the equilibrium.
rates yield stable equilibria when range contracts rapidly as shown. Unstable equilibria occur for 0 for a very small area on the upper left-hand corner of the parameters exist, while the dashed line is the maximum death rate for which there solid line denotes the maximum death rate that permits the predator to exist, while the dashed line is the maximum death rate for which there is a hydra effect. All parameters produce a stable equilibrium except parameters other than a are identical to the density-independent model in Figs. 1 and 2: \( h = 1, s_1 = 1, d_1 = 0.1, d_2 = 0.05, B_2 = 1.0 \) and \( g = 0.05 \). The solid line denotes the maximum death rate that permits the predator to exist, while the dashed line is the maximum death rate for which there is a hydra effect. All parameters produce a stable equilibrium except for a very small area on the upper left-hand corner of the parameters shown. Unstable equilibria occur for 0.93768 < \( D \) < 1 for \( x = 0 \), but this range contracts rapidly as \( x \) increases; all permissible predator death rates yield stable equilibria when \( x > 0.009508 \).

predator death rate and strength of density dependence. The hydra effect occurs over a significant range of strengths of density dependence. In this figure the parameters other than \( x \) are identical to those used in the density-independent model. However, instability only occurs for a minute fraction of parameter space at high predator mortalities (see Fig. 3 legend).

As in the density-independent prey growth model, the degree of generalization of the predator has a great influence on stability. Identical predation rates on, and identical nutritional values of the two prey stages result in the pattern of instability for a wide range of low-to-moderately high predator death rates, with stability at the highest mortality rates. When predation is concentrated on one stage of the prey population, a density-dependent system is often stable for all predator death rates, and, if instability occurs, it is only for the highest death rates. If the two attack rates, \( s_1 \) and \( s_2 \) are varied while their sum is constant, there is generally an abrupt switch between a pattern of stability for all predator death rates (when the predator is relatively specialized on one size class) to a pattern of instability for a relatively wide range of low-to-moderately high predator death rates, with stability at the highest rates. For values of \( s_1 \) and \( s_2 \) close to the transition, there are often alternative attractors; a stable point and a limit cycle.

For example, given parameter values in Fig. 3 (\( B_2 = 1, g = 0.05, d_1 = 0.1, d_2 = 0.05 \), with \( h = 1 \) for both size classes, and \( e = 1 \) for both size classes) this transition occurs for attack rates close to \( s_1 = 0.7 \) and \( s_2 = 0.3 \). Both a stable point and a limit cycle exist for this particular set of parameters. In general, as the ratio of the two \( s \) values increases there is an abrupt transition from stability at low \( D \) (and possible instability at high \( D \)) when \( s_1 \) is close to 1, to instability at low \( D \) and stability at high \( D \) when \( s_1 \) approaches 0.5. This appears to be a relatively general feature of models with relatively weak density dependence. Fig. 4 examines a model with a different \( b_2 \) function than assumed above; it shows that the \( P \) vs. \( D \) relationship for three pairs of relative capture rates of juvenile and adult prey. The shape of this relationship changes relatively little, although the same type of switch in \( D \) values that are stable and unstable occurs at approximately the same level of predator specialization as in the model with linear density dependence.

### 2.3.2. Density-dependent juvenile growth and adult birth rates

The reason for considering this combination of density-dependent functions is its ability to produce alternative equilibria for the prey in the absence of the predator, which complicates the interaction. This model assumes that competitive interactions occur within each class of prey (adults only compete with adults, and juveniles with juveniles). This set of two density-dependent functions is plausible for many fish populations, where adult density dependence is often expressed...
in a stock–recruitment relationship, and where juvenile competition is likely to reduce the growth rate to the adult stage. Linear declines in both demographic parameters were chosen for simplicity. These assumptions yield the following model:

\[
\begin{align*}
\frac{dN_1}{dt} &= B_3(1 - x_2 N_2)N_2 - d_1 N_1 - G(1 - x_1 N_1)N_1 - \frac{sn_1 P}{1 + sh N_1}, \\
\frac{dN_2}{dt} &= G(1 - x_1 N_1)N_1 - d_2 N_2, \\
\frac{dP}{dt} &= P\left(\frac{esN_1}{1 + sh N_1} - D\right).
\end{align*}
\]

To understand the consequences of changing the mortality rate of predators in this system, it is also important to understand the dynamics of the prey population in the absence of predators. In this case, the model is given by

\[
\begin{align*}
\frac{dN_1}{dt} &= B_3(1 - x_2 N_2)N_2 - d_1 N_1 - G(1 - x_1 N_1)N_1, \\
\frac{dN_2}{dt} &= G(1 - x_1 N_1)N_1 - d_2 N_2.
\end{align*}
\]

For a range of parameter values, the prey-only model possesses two locally stable equilibria. The first is characterized by a large juvenile population; the resulting competition among juveniles makes growth into the adult class slow, producing a small adult population. The alternative stable equilibrium has a higher adult population; within-adult competition keeps the birth rate low, which produces a low-to-moderate juvenile size class. Fig. 5 shows the isocline diagram for Eq. (11) for such a case. The zero-growth isocline for the juvenile class consists of two branches, corresponding to the two solutions for \(N_2\) that produce zero growth in the first age class. The presence of two potentially stable prey equilibria means that there may be some predator parameter values for which the predator is able to invade one of the equilibria, but not the other (Fig. 6).

The full predator–prey model (Eq. (10)) is still simple enough that it is possible to get an explicit expression for the equilibrium predator density

\[
-\frac{d_1 (1 - Dh)^3 s^3 - G(-s + Dhs + Dz_1)[-s^2 d_2 (1 - Dh)^3 + B_2 (d_2 s^2 (1 - Dh)^2 + DGz_2 (Dz_1 - s(1 - Dh)))]}{d_2 s^2 (1 - Dh)^4}
\]

This expression is a rather complicated function of the predator’s death rate, but it is possible to verify that the predator often increases as its own mortality rate increases. For example, the following parameter set—
\(d_1 = 0.1;\quad d_2 = 0.05;\quad G = 0.1;\quad B_2 = 1;\quad h = 1;\quad s = 1;\quad x_1 = 0.1;\quad\text{and}\quad x_2 = 0.05—\) yields the relationship between predator mortality rate and predator density shown in Fig. 6. For this example, predator numbers increase with the equilibrium is unstable. There are alternative equilibria in the prey-only model, and only one of these two equilibria (the one with a larger number of juveniles) allows increase of the predator from low densities. However, in these cases, the initial increase in predator numbers shifts the prey densities to the low-juvenile-abundance equilibrium where a predator cannot maintain a viable population. Thus, the narrow
spike of positive predator equilibria on the right-hand side of Fig. 6 does not represent persistent systems.

Given expression (12) for the equilibrium predator density, a relatively simple condition reveals whether there exists a range of predator mortality rates, \( D \), close to \( D = 0 \), characterized by a hydra effect. This condition is

\[
shd_2(B_2 - d_1d_2 - d_2) - Gd_2z_{11}(B_2 - d_2) > 0.
\]

The first term in Eq. (13), \( shd_2(B_2 - d_1d_2 - d_2) \), must be positive at a positive equilibrium point, and the remaining two terms are negative and increase in magnitude with the growth rate, \( G \). It is therefore necessary that \( G \) be below some threshold if there is to be a range of low mortality rates over which increasing mortality increases the predator’s population.

### 2.3.3. Alternative models

We have analyzed cases with multiple density-dependent parameters in the case of predation on juveniles only. These do not alter the qualitative conclusions reached above, that the hydra effect and a locally stable equilibrium will both occur in systems with a saturating functional response and density dependence that is below a threshold in strength. The assumption of stage-specific density dependence was also relaxed by using the sum of the abundances of the two prey stages as the variable in the density-dependent components of the prey per capita growth rate. This again did not change the qualitative pattern of stability with a hydra effect when density dependence was relatively weak and the predator’s functional response was saturating. We also examined some more significant changes to the framework established by Eq. (1), and these are summarized below.

#### 2.3.3.1. Predation restricted to adults

If predators are considerably larger than their prey, it is possible that they will be restricted to consuming adults. The density-independent equations (4) can be modified to reflect this type of selectivity by changing the target of predation from prey classes 1 to 2. The simplest case is again when the prey have density-independent population growth. This yields the following equilibrium population sizes:

\[
N_1 = \frac{b_2D}{s(d_1 + g)(1 - Dh)},
\]

\[
N_2 = \frac{D}{s(1 - Dh)},
\]

\[
P = \frac{(b_2 - d_2)g - d_1d_2}{s(d_1 + g)(1 - Dh)}. \tag{14}
\]

These equilibrium population sizes display the same dependency on predator death rate, \( D \), as in the juvenile-vulnerable model, Eq. (4); all population sizes increase at an accelerating rate with \( D \). The local stability of the equilibrium point can be determined. In many respects, the results are similar to those for Eq. (4). Unlike the unstructured model, it is possible for the equilibrium point to be locally stable, or to be locally unstable and surrounded by a locally stable limit cycle. The stability condition is

\[
b_2g(1 - Dh)[h(d_1 + g) - (1 - Dh)] + h(d_1 + g) \\
\times[(d_1 + g)^2 + Dd_2(h(d_1 + g + D) - 1)] < 0. \tag{15}
\]

There is a range of predator death rates, starting at \( D = 0 \), that yields a stable equilibrium point provided the following condition is satisfied:

\[
(d_1 + g)^3h - gb_2(1 - h(d_1 + g)) < 0. \tag{16}
\]

As \( D \) approaches its maximum value of \( 1/h \), the stability condition (15) is always violated. Thus, given that condition (16) is satisfied for small \( D \), there will be a transition from stability to instability as the predator’s death rate is increased, as in the case of predation on juveniles. There are ranges of the growth rate, \( g \), for which condition (16) is not satisfied (i.e., the system is unstable even if the predator mortality rate is very low). If there is density dependence, this system can be stabilized over a wider range of parameter values, and it is again possible for the predator’s population to increase as its mortality rate increases in stable systems when density dependence is not too strong. Models with density dependence again allow outcomes in which there is a stable equilibrium at low predator death rates but an unstable one at higher death rates.

#### 2.3.3.2. Other types of stage structure

Many types of stage structure occur in natural populations, and it is impossible to determine the complete range of consequences of such structure. However, three extensions of the above model will be mentioned briefly here: (1) models in which the prey has a homogeneous population while the predator has a 2-class population structure; (2) models in which only the prey has a structured population, but it has more than two classes; (3) models with non-exponentially distributed residence times in the prey’s juvenile stage.

Simulations of a range of models have shown that size–structure in the predator has relatively minor effects on stability when prey consumption determines the predator’s per capita demographic rates. These models have stability properties similar to unstructured models. The prey’s dynamics are the key to stability in a homogeneous population model; the positive feedback produced by increased predator satiation with increased prey density generates this feedback, which leads to instability when density dependence in prey growth is weak enough. Stage structure in the prey provides a class of individuals that is free of (or has a reduced rate of) predation, which is usually stabilizing (Abrams and
Stage structure in the predator population does not have a comparable influence on stability. Two-class stage structure in both species produces dynamics that are generally similar to those observed when only the prey has size–structure, provided that both predator classes consume the same prey class.

When Eq. (4) are augmented so that the prey population has two juvenile stages (three total stages), the distribution of residence times in the entire juvenile phase is no longer exponentially distributed (McNair, 1995). This formulation also makes it possible to describe a graded transition, from very high vulnerability to the predator in the first stage, to lowered vulnerability in the second, to still lower (or no) vulnerability in the adult stage. A number of three- or more-stage analogues of the numerical examples for Eq. (4) were studied, and were generally found to have similar stability properties and similar relationships between predator population size and predator mortality.

Models with a fixed duration of the juvenile stage and a gamma distribution of juvenile stage lengths were also investigated. The model with linear density dependence in the adult birth rate (Model A of the previous section) was extended to a generic distribution of juvenile stage lengths as follows:

\[
\frac{dN_1}{dt} = B_2(1 - z_2N_2)N_2 - d_1N_1 - \frac{sN_1P}{1 + shN_1} - M(t),
\]

\[
\frac{dN_2}{dt} = -d_2N_2 + M(t),
\]

\[
\frac{dP}{dt} = P\left(\frac{\alpha}{1 + shN_1} - D\right),
\]

where \(M(t)\) is the rate at which juveniles mature. This will be an integral over all past times, of the birth rate at that time weighted by the distribution of stage lengths and the probability of survival to maturity (Gurney et al., 1986). The latter is obtained in terms of the per capita juvenile death rate at past times

\[
\Phi(t) = d_1 + \frac{sP(t)}{1 + shN_1(t)},
\]

by integrating the total juvenile death rate from birth to the present. This gives for the maturation rate

\[
M(t) = \int_0^\infty \beta(t')B_2(1 - z_2N_2(t - t'))N_2(t - t') \times \exp\left(-\int_{t-t'}^{t} \Phi(t') dt'\right) dt',
\]

where \(\beta(t')\) is the distribution of juvenile stage lengths.

The maturation rate is a complicated expression and it is hard to analyze this model for general distributions \(\beta(t')\). Progress can be made in two special cases; that of a fixed stage length and a gamma distribution of stage lengths. For a fixed stage length of duration \(\tau\) so that \(\beta(t') = \delta(t' - \tau)\) the maturation rate becomes

\[
M(t) = B_2(1 - z_2N_2(t - \tau))N_2(t - \tau) \times \exp\left(-\int_{t-\tau}^{t} \Phi(t') dt'\right),
\]

Eq. (17) then reduce to delay differential equations (Nisbet, 1997). For these equations the location of all non-trivial equilibria can be numerically calculated. This reveals that only one potentially stable equilibrium with a non-zero predator population can exist. In Fig. 7 the existence and hydra effect boundaries are shown as solid and dashed lines, respectively, in the \(D-x\) plane for a fixed value of \(\tau = 10\). It is also possible to linearize Eq. (17) and determine the boundary where this equilibrium becomes locally unstable (Murdoch et al., 2003). This is the dotted line of Fig. 7. Of course this tells us nothing about the global stability of the system, but numerical integration revealed that when stable, the equilibrium was reached from a wide range of initial conditions, and when unstable, a limit cycle was approached instead (Neves, 1975). No doubt more complex dynamics may exist in other regions of parameter space.

From Fig. 7, we see that the hydra effect applies for all \(D\) values except for a small range just below the point where predators can no longer exist. At larger \(D\) values the system may become unstable but increasing \(x\) mitigates this effect. The latter also decreases the range of the hydra effect. For these parameters the system is both stable and the hydra effect applies over a large proportion of predator death rates and competition

![Fig. 7. Boundaries for stability and the hydra effect in the model with a fixed length for the juvenile prey stage (\(\tau = 10\)). The strength of density dependence, measured by \(x\), is shown on the x-axis, while the predator’s per capita death rate, \(D\), is on the y-axis. The remaining parameters are \(h = 1\), \(s_1 = 1\), \(d_1 = 0.1\), \(d_2 = 0.05\) and \(B_2 = 1.0\). The solid line denotes the maximum death rate that permits the predator to exist, the dashed line is the maximum death rate for which there is a hydra effect and the dotted line indicates the boundary separating stable and unstable equilibria. The inset graph shows the stability boundaries for varying juvenile stage lengths.](image-url)
constants (63% of the area shown in the graph). The unstable region is larger with a fixed duration juvenile period than in the comparable model with an exponentially distributed juvenile phase. Direct comparison of the main graph in Fig. 7 to Fig. 3 is not possible because Fig. 3’s growth rate $G = 0.05$ yields a longer mean juvenile period (equivalent to $\tau = 20$). However, the inset in Fig. 7 shows the size of the unstable region for the $\tau = 20$ case, which is much larger than the corresponding region of Fig. 3, which was too narrow to illustrate effectively. The inset also reveals that increasing $\tau$ decreases stability. This results in a decrease in the area of stable hydra effect although this is somewhat offset by an increased range of the hydra effect itself. In the $\tau = 20$ case a stable hydra effect operates over 21% of the parameter range in Fig. 7.

The highly variable exponential stage length and the fixed stage length represent opposite extremes. We can explore intermediate cases using a gamma distribution

$$\beta(t) = \frac{\theta^t e^{-\theta t}}{\Gamma(t)}, \quad (21)$$

where $\Gamma(t)$ is the gamma function. This function gives a stage length with a mean value of $\tau = \frac{1}{\theta}$, a variance $\sigma^2 = \frac{1}{\theta^2}$ and coefficient of variation $V = 1/\sqrt{\tau}$. In the case of integer $\tau$, this distribution allows the system to be converted to $2\tau + 3$ ODEs (McNair, 1995). This conversion was used to generate Fig. 8, giving the positive predator population, hydra effect, and stability boundaries for a stage length $\tau = 20$ with $V = 0.22$ ($\tau = 20$, $\theta = 1$). Comparison with Figs. 3 and 7 ($\tau = 20$ inset) shows the same general properties as the other residence time distributions, but with an unstable region intermediate in size. The area of stable hydra effect is significant (61%). The inset of Fig. 8 shows that as the variability in the stage duration decreases, the system becomes less stable.

3. Discussion

The simplest predator–prey models are characterized by homogeneous populations and predator per capita growth rates that are solely a function of prey population size. These models predict that an increase in the population density of the predator in response to an increase in its own mortality only occurs in systems that exhibit cycles. This may be why the possibility of a predator increasing with increases in its own mortality (the ‘hydra effect’) has been ignored in applied disciplines within ecology, where cycles are often assumed to be uncommon. In the models investigated here, the presence of size-structure implies that stability and increasing population–mortality relationships are no longer coupled. It is common for the equilibrium population density of a predator to increase with increases in its own mortality in stable predator–prey systems. The predation-free class of prey provides a refuge, from which vulnerable prey are replenished. This phenomenon has been shown to be capable of producing stability in a variety of other models in which the predation-free state was not related to developmental stage (Abrams and Walters, 1996). The decoupling of stability and the hydra effect can also occur as the result of adaptive behavioral adjustment of traits related to the interaction in either or both of the species (Matsuda and Abrams, 2004; Abrams and Matsuda, 2005). This raises two questions; (1) do such increases in population size with higher mortality actually occur in nature; and (2) if not, what prevents them from occurring? As noted in previous articles based on homogeneous populations (Matsuda and Abrams, 2004, Abrams and Matsuda, 2005), there are no well-documented examples of the hydra effect, although several cases are consistent with such an effect. This is in part due to the absence of field or laboratory studies designed specifically to look for the effect, and because of the poor quality, variability, or short time spans of field observations of harvested systems (generally from fisheries). On the other hand, there are some suggestive examples (Abrams, 2002, Abrams and Matsuda, 2005). There are also other potential mechanisms for a hydra effect (Kuris and Lafferty, 1992).

In spite of the theory presented here, it is also possible that such increases in predator density with increased mortality are rare or non-existent. Because the effects can be eliminated by sufficiently strong density dependence in the prey population in all of the cases considered thus far, density dependence is a potential

![Fig. 8. Boundaries for stability and the hydra effect in the model with a gamma distribution of lengths for the juvenile prey stage. The stage had mean length $\tau = 20$ with a coefficient of variation $V = 0.22$. The solid line denotes the maximum death rate that permits the predator to exist, the dashed line is the maximum death rate for which there is a hydra effect and the dotted line indicates the boundary separating stable and unstable equilibria. The inset graph shows the stability boundaries for different $V$.](image-url)
explanation for the lack of hydra effects in past ecological studies. It is also possible that aspects of the predator–prey interaction that are not included in the simple models predicting a hydra effect could eliminate it. One candidate for such an explanation is the large number of interspecific interactions experienced by most harvested species in food webs. It is not yet clear what types of additional interactions would eliminate the hydra effect, although it is clear that competing predators that do not experience increased mortality could eliminate the effect under some circumstances (Abrams, 2003). Another possibility is the presence of predator-dependent functional responses, something Abrams and Ginzburg (2000) argued was the rule rather than the exception. Such responses reduce the occurrence of hydra effects in simple unstructured models (Abrams, 2002), and the same may be true of the models considered here. However, it is clear that a relatively strong hydra effect will not be completely eliminated by the presence of weak predator interference. Moreover, interference by itself can produce a hydra effect in a stable predator–prey system with no population structure or adaptive change in traits (Abrams and Matsuda, 2005).

There are at least two important implications of the results presented here for resource management. Firstly, it shows that the lack of population structure in previous models predicting hydra effects is not a necessary factor for the occurrence of such phenomena. Secondly, it suggests that prey size–structure combined with size-specific predation increases the likelihood of observing a hydra effect in a stable system. These conclusions are important for fisheries managers because the possibility of populations increasing as a consequence of harvest is contrary to assumptions that underlie most schemes of ‘adaptive management’. Adaptive management adjusts harvest rates or quotas based on responses of the population to past changes in harvest rates. However, the traditional assumption is that increased population size in spite of increased harvest reflects a situation where harvesting may safely be increased. Relationships such as those shown in Figs. 4 and 6 demonstrate that such adjustments will eventually result in a catastrophic collapse, when the population traverses the right-most section of the curves shown in the figures.

The models considered here also show that conditions for stability and instability in predator–prey systems differ dramatically between structured and unstructured populations, at least when predation is concentrated on a subset of the classes within the prey population. Not only are the structured models more stable under stage-related predation, but the effect on stability of increased predator mortality is opposite in the two cases; higher mortality is stabilizing in the traditional homogeneous models (Rosenzweig and MacArthur, 1963), while it is destabilizing in our size-specific predation models when stage length is sufficiently short or variable. Most of the models/parameters considered here become more stable rather than less stable when prey growth rate, \( g \), is increased. This was also true of the fixed stage-length model where stability decreased with increased juvenile stage lengths. Different parameters affecting prey growth have different effects on stability, and Rosenzweig’s (1971) ‘paradox of enrichment’ is certainly not an inevitable outcome of fertilization. In addition, unlike comparable unstructured models, the equilibrium abundances of both predator and prey generally change in response to changes in the prey’s demographic parameters.

The destabilizing effect of an increase in juvenile stage length for fixed delay models has been observed before. Particularly pertinent is a closely related host–parasitoid model (Murdoch et al., 1987). The latter is similar to the model considered in Section C except we have density-dependent host fecundity and a satiating parasitoid functional response. Murdoch et al. (1987) also use a stage-structured parasitoid population as well as host population. In fact, given a saturating parasitoid functional response and overlapping generations, the hydra effect is likely to apply quite generally to stage-structured host–parasitoid models.

A longstanding problem in ecology has been to reconcile the fact that the vast majority of measured functional responses exhibit satiation (Hassell, 1978; Jeschke et al., 2002) with the seeming stability (or at least lack of large amplitude cycles) in most systems for which we have long-term population records. Saturating functional responses in the simplest predator–prey models produce cycles whose amplitude is larger than that of most observed cycles over most of the range of potential parameters that yield cycles. One possible solution is direct density dependence in the predator (e.g., Abrams and Ginzburg, 2000). This is plausible in many cases, but measurements of such direct density dependence are almost non-existent (Ganter, 1984 is one of a handful of exceptions to this generalization). Adaptive avoidance behavior by prey (or adaptive foraging behavior in both species) can often produce effective predator density dependence (Abrams, 1993), but it can also produce instability in the case of a saturating predator response (Matsuda and Abrams, 1994; Abrams and Matsuda, 1997). There are some circumstances when adaptive predator avoidance can produce both stability and increasing population–mortality relationships. Other investigators have suggested that the observed stability stems from spatially localized interactions being more stable than interactions in well-mixed environments (McCauley et al., 1993; Jansen and DeRoos, 2000). The results presented here suggest that stage- or size-restricted predation may also provide an explanation for the stability of predator–prey systems. If this is true, then it should also be possible for predator
population size to increase with predator mortality in many predator–prey systems. The above explanation for the stability of predator–prey systems is given added plausibility by our observation that stability increased with increasing variation in the stage length. This has also been recently described for predator–prey models with linear functional responses (Eurich et al., 2005). Given that some variability might be expected in real systems then the stabilizing influence of stage-structure cannot be discounted.

Even if size- or stage-specific predation is a major cause of the observed stability of systems, this fact alone does not imply that increasing population-mortality relationships need be common. In the models investigated here, such relationships do not occur if the initial mortality of the predator is high enough or if competition within the prey is strong enough. The distributions of these two parameters in natural predator–prey systems are seldom known with any accuracy. It is also important to investigate the stability properties and hydra effect in models having continuous stage structure and in models having adaptive variation in growth rates. Nevertheless, models studied thus far suggest that it should not be a rare occurrence for a predator population to increase following an increase in its own mortality rate.

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Appendix

Stability and response of predator population to mortality in the model with a general functional response and general density dependence.

This appendix begins by analyzing the stability of an interior equilibrium of Eq. (1), given the assumptions that only one prey class is consumed (s1 = 0 or s2 = 0, γ = 1, εs = 1). The Jacobian matrix when s2 = 0 has the following form:

\[
\begin{bmatrix}
-d_1 - g - s_1 \rho (H + s_1 N_1 H') & b_2' N_2 + b_2 & -D \\
-b_1 & -d_2 & 0 \\
s_1 (H + s_1 N_1 H') & 0 & 0.
\end{bmatrix}
\]

Application of the Routh Hurwitz criteria to an equilibrium with both species present leads to the stability condition, Eq. (2) in the text. Both the above matrix and the stability condition use the equilibrium conditions for Eq. (1): (i) s1N1H = D; (ii) gbd2 - (d1 + g) = s1HP; and (iii) N2 = gN1/d2. The signs of the various terms in inequality (2) can be determined using the assumption that the functional response is increasing (so that H + s1N1'H > 0) and the fact that density dependence implies that \( b_2' < 0 \). It is important to note that, for a prey population to grow from low densities, it is necessary that \( g(b_2 - d_1) - d_2 d_2 > 0 \) (this is the criterion for \( N_1 = N_2 = 0 \) to be locally unstable in the system lacking the predator).

Expression (3) is derived by setting the right-hand sides of Eq. (1) to zero, and implicitly differentiating both sides with respect to \( D \). This yields a set of three equations which can be solved for the partial derivatives of \( N_1, N_2, \) and \( P \) with respect to \( D \). The value of \( \partial N_1/\partial D \) is set by the condition for equilibrium of the predator dynamics; \( sN_1H = D \) implies that \( \partial N_1/\partial D = 1/ [s(H + sN_1 H')] \). Similarly, \( \partial N_2/\partial D = (g/d_2) (\partial N_1/\partial D) \).

The sign of expression Eq. (3) for \( P \partial P/\partial D \) depends on the fact that the predator and prey densities are positive, which implies that \( g(b_2 - d_1) - d_2 d_2 > 0 \) and that \( D \) is less than the limit of \( sN_1H \) as \( N_1 \) approaches infinity. The sign of Eq. (3) also depends on the fact that the functional response is increasing, which implies that \( H + sN_1H' > 0 \). When evaluated at equilibrium, the increasing functional response condition implies that \( H^2 + DH' > 0 \).

There is a maximum \( D \) for stability in the density-independent model, which can be determined by solving inequality (6) after setting it to an equality. The result is

\[
D_{\text{max}} = \frac{Q + \sqrt{Q^2 - 4(d_1 d_2 - B_2 g + d_2 g)(B_2 d_2 gh - B_2 g + d_2^2 gh)}}{2(d_1 d_2 h - B_2 gh + d_2 gh)},
\]

where \( Q = d_1 d_2 h - 2B_2 gh + d_2 gh - d_1 d_2 h^2 + B_2 d_2 h^2 - d_2^2 gh \).

It can be shown that this expression increases with \( g \), and that the asymptotic value for large values of \( g \) is

\[
-d_2 h + d_2^2 h^2 + B_2 h^2 (2 - d_2 h) - \sqrt{d_2 h^2 (1 - d_2 h)^2 + b_2 Z_1 - 2B_2 d_2 Z_2}}{2b_2 (b_2 - d_2)}
\]

where \( Z_1 = -4 + 4d_2 h + 4h^2 - 4d_2 h^2 + d_2^2 h^4 \) and \( Z_2 = -2 + 2d_2 h + 2h^2 - 3d_2 h^2 + d_2 h^4 \).

Although the above expression is complicated, it does simplify to \( 1 - d_2 \) in the special case when \( h = 1 \).

References


