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Ecological Modelling 179 (2004) 29–38

ECOLOGICAL MODELLING

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Reactivity and transient dynamics of predator–prey and food web models

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Received 9 September 2003; received in revised form 14 April 2004; accepted 3 May 2004

Abstract

Stability and resilience characterize the asymptotic responses of perturbations to the equilibria of ecological models. Short-term responses, however, can differ markedly from asymptotic responses. Perturbations to a stable equilibrium may, for example, produce trajectories that initially move away from, rather than towards, the equilibrium. The maximum short-term rate of departure from the equilibrium is called the "reactivity", and stable equilibria with positive reactivity are called "reactive". These transient responses can be large and long-lasting, and have been reported in a variety of ecological models. In this paper we explore the reactivity of predator–prey and food web models. We show that coexistence equilibria are reactive in all predator–prey and food web models in which at least one species has a per capita growth rate that is independent of its own density. These constitute the vast majority of published models. When density-dependent mortality of the top predator is included in the form of a non-linear "closure term", reactivity always decreases, and may be eliminated altogether. Our results imply that short-term amplification of perturbations is a real possibility for predator–prey interactions and food webs.

Keywords: Perturbations; Transients; Food chains; Food webs; Stability

1. Introduction

The equilibria of ecological models are often characterized by their stability and resilience, i.e. by whether small perturbations to the equilibrium eventually decay and, if so, how fast. Thus, stability and resilience characterize asymptotic dynamics. An equilibrium is locally asymptotically stable if a small perturbation eventually decays, regardless of what happens to the perturbation in the short term.

In some stable systems, however, the immediate response to some perturbations is a trajectory that moves away from, rather than towards, the equilibrium. This transient growth can be significant and long-lasting. Equilibria that exhibit this property are said to be "reactive" (Neubert and Caswell, 1997). Reactivity has now been documented in a number of ecological models (Neubert and Caswell, 1997; Chen and Cohen, 2001; Neubert et al., 2002; Ives et al., 2003; Marvier et al., 2003), and Ives et al. (2003)have developed methods for estimating reactivity from time series.

The short-term growth of a perturbation is often as important as its long-term decay. Ecosystem managers and conservation biologists must deal with short-term responses for economic and political reasons. Also, real ecosystems typically do not complete their response to a perturbation before the next one occurs. Instead, they are buffeted by a more or less continual

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doi:10.1016/j.ecolmodel.2004.05.001

series of perturbations, and transient behavior may be the norm rather than the exception in nature.

In this paper, we investigate the transient dynamics of predator–prey and, more generally, food web models. In Section 2, we briefly review the indices used to characterize responses to perturbations. In Section 3, we show that perturbations to coexistence equilibria of food web models are always reactive if the per-capita mortality rates of top predators are density-independent. The influence of density-dependent predator mortality is discussed in Section 4. In Section 5 we give an example using the MacArthur–Rosenzweig predator–prey model. Results are discussed in Section 6.

2. Responses to perturbation

Consider a system of non-linear differential equations modelling the interaction between m species whose population densities are collected in the vector $\boldsymbol{n}(t)$:

$$\frac{\mathrm{d}\boldsymbol{n}}{\mathrm{d}t} = \boldsymbol{f}(\boldsymbol{n}). \tag{1}$$

The equilibria of system (1) are those points $\hat{\boldsymbol{n}}$ that satisfy

$$\boldsymbol{f}(\hat{\boldsymbol{n}}) = 0. \tag{2}$$

To first approximation, the dynamics of a small perturbation ($\boldsymbol{x}(t)$) of the equilibrium are governed by the linear system

$$\frac{\mathrm{d}\boldsymbol{x}}{\mathrm{d}t} = \boldsymbol{A}\boldsymbol{x}, \tag{3}$$

where the $m \times m$ matrix $\boldsymbol{A}$ is the Jacobian matrix with elements

$$a_{ij} = \left.\frac{\partial f_i}{\partial n_j}\right|_{\hat{\boldsymbol{n}}}. \tag{4}$$

2.1. Asymptotic responses: $t \to \infty$

The asymptotic behavior of the solutions to system (3) determine the stability of the equilibrium point. If $\boldsymbol{x}(t) \to 0$ as $t \to \infty$ for all initial conditions $\boldsymbol{x}(0)$, the equilibrium point is said to be *asymptotically stable*. A sufficient condition for stability is that all of the eigenvalues of $\boldsymbol{A}$, call them $\lambda_i(\boldsymbol{A})$, have negative real parts. From here on, we consider only stable equilibria.

To quantify the stability of an equilibrium point, Pimm and Lawton (1977), introduced a measure they called *resilience*. Resilience, for which we here use the symbol ν_∞, is the asymptotic rate of decay of a typical perturbation and is calculated as

$$\nu_\infty(\boldsymbol{A}) = -\mathrm{Re}(\lambda_1(\boldsymbol{A})), \tag{5}$$

where $\lambda_1(\boldsymbol{A})$ is the eigenvalue with largest real part. We will assume, from here on, that this eigenvalue is unique. The more resilient the equilibrium, the more rapidly perturbations decay in the long run.

2.2. Instantaneous responses: $t \to 0$

Stability and resilience determine the long-run response to perturbations. They say nothing, however, about the short-term response. In fact, a small perturbation to a stable equilibrium point can exhibit significant growth in the short term.

Neubert and Caswell (1997) introduced a measure of the short-term transient growth of perturbations called *reactivity*, that we here denote as ν_0. Reactivity is defined as the maximum, taken over all initial conditions, of the instantaneous rate of amplification of a small perturbation,

$$\nu_0 = \max_{||\boldsymbol{x}(0)||} \left(\frac{1}{||\boldsymbol{x}||} \frac{\mathrm{d}||\boldsymbol{x}||}{\mathrm{d}t} \right)\Bigg|_{t=0}. \tag{6}$$

The reactivity ν_0 can be calculated from the Jacobian matrix as

$$\nu_0(\boldsymbol{A}) = \lambda_1(H(\boldsymbol{A})). \tag{7}$$

The matrix $H(\boldsymbol{A})$,

$$H(\boldsymbol{A}) = \frac{1}{2}(\boldsymbol{A} + \boldsymbol{A}^{\mathrm{T}}), \tag{8}$$

is called the Hermitian part of the matrix $\boldsymbol{A}$. As the Hermitian part of any matrix is symmetric, its eigenvalues are real. Equilibria with positive reactivity ($\nu_0 > 0$) are called *reactive*. The larger the reactivity, the faster a perturbation can possibly grow. In contrast, the size of any perturbation to a non-reactive equilibrium decays monotonically.

2.3. Transient responses: $0 < t < \infty$

Resilience describes the response to perturbation in the limit as $t \to \infty$. Reactivity characterizes responses in the limit $t \to 0$. There is often interesting transient behavior between these two limits. A bound on the amplification of a perturbation that holds for all time is given by the *amplification envelope*:

$$\rho(t) \equiv \max_{\boldsymbol{x}(0)\neq 0} \frac{||\boldsymbol{x}(t)||}{||\boldsymbol{x}(0)||} = |||e^{\boldsymbol{A}t}|||, \tag{9}$$

where $|||\cdot|||$ is the matrix norm induced by the Euclidean vector norm (Neubert and Caswell, 1997). This matrix norm is easily calculated in many mathematical software packages. In Matlab, the matrix norm is given by the command `norm()`.

Reactivity and resilience are related to the slope of the natural logarithm of the amplification envelope:

$$\nu_0 = \lim_{t\to 0^+} \frac{\mathrm{d}}{\mathrm{d}t} \ln \rho(t), \tag{10a}$$

$$\nu_\infty = \lim_{t\to \infty} \frac{\mathrm{d}}{\mathrm{d}t} \ln \rho(t). \tag{10b}$$

Other properties of the amplification envelope, like its maximum, $\rho_{\max}$, the time after the perturbation when the maximum occurs, $t_{\max}$, and the return time, $T_R = \int_0^\infty \rho(t)\,\mathrm{d}t$, are useful for describing the shape of the amplification envelope and for characterizing the transient response to perturbations (Neubert and Caswell, 1997). In this article, we will limit our attention to resilience and reactivity.

2.4. Examples

Fig. 1a shows the amplification envelopes for two matrices,

$$\boldsymbol{A_1} = \begin{pmatrix} -1 & 0 \\ 0 & -10 \end{pmatrix} \quad \text{and} \quad \boldsymbol{A_2} = \begin{pmatrix} -1 & 15 \\ 0 & -10 \end{pmatrix}. \tag{11}$$

$\boldsymbol{A_1}$ and $\boldsymbol{A_2}$ have the same dominant eigenvalue ($\lambda_1 = -1$), and hence the same resilience ($\nu_\infty(\boldsymbol{A_1}) = \nu_\infty(\boldsymbol{A_2}) = 1$). As both eigenvalues are real, the matrices $\boldsymbol{A_1}$ and $\boldsymbol{A_2}$ represent linearizations around a stable node. Asymptotically, perturbations governed by $\boldsymbol{A_1}$ and $\boldsymbol{A_2}$ decay at the same rate, but the transient dynamics are very different (Fig. 1a). The system described by $\boldsymbol{A_2}$ exhibits transient growth of perturbations, as indicated by the hump in the amplification envelope; its reactivity is $\nu_0(\boldsymbol{A_2}) \approx 3.25 > 0$. The system described by $\boldsymbol{A_1}$ cannot exhibit transient growth because $\nu_0(\boldsymbol{A_1}) = -1 < 0$.

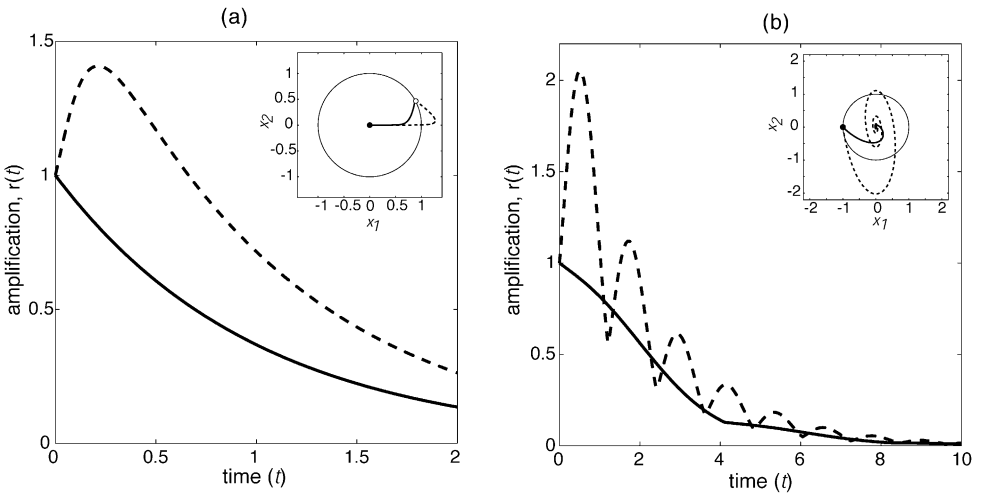

Fig. 1. Amplification envelopes ($\rho(t)$) for the matrices $\boldsymbol{A_1}$ (a, solid line), $\boldsymbol{A_2}$ (a, dashed line), $\boldsymbol{A_3}$ (b, solid line), and $\boldsymbol{A_4}$ (b, dashed line), as given by equations (11). Insets show the responses to typical perturbations in the phase plane. Equilibria whose linearizations give matrices $\boldsymbol{A_2}$ and $\boldsymbol{A_4}$ are reactive.

Fig. 1b shows amplification envelopes for the two matrices

$$\boldsymbol{A_3} = \frac{5}{6}\begin{pmatrix} -1 & -1 \\ 1 & -0.2 \end{pmatrix} \quad \text{and} \quad \boldsymbol{A_4} = \begin{pmatrix} -1 & -1 \\ 7 & 0 \end{pmatrix}, \tag{12}$$

which represent linearizations around stable foci. $\boldsymbol{A_3}$ and $\boldsymbol{A_4}$ have the same resilience ($\nu_\infty(\boldsymbol{A_3}) = \nu_\infty(\boldsymbol{A_4}) = 0.5$), but their reactivities are different ($\nu_\infty(\boldsymbol{A_3}) \approx -0.17$, $\nu_\infty(\boldsymbol{A_4}) \approx 2.54$). As a result, the system governed by $\boldsymbol{A_4}$ exhibits transient growth of perturbations, but that governed by $\boldsymbol{A_3}$ does not.

3. Food web models

We now turn to food web models. We will show that in a wide class of such models any equilibrium point at which all species coexist must be reactive. We start with the simplest consumer–resource model (a two-species predator–prey model).

3.1. Predator–prey models

Predator–prey models often take the form

$$\frac{\mathrm{d}n_1}{\mathrm{d}t} = n_1 f_1(n_2), \tag{13a}$$

$$\frac{\mathrm{d}n_2}{\mathrm{d}t} = f_2(n_1, n_2), \tag{13b}$$

where n_1 is the predator density and n_2 the prey density. As written, (13a) assumes that the per capita growth rate of the predator, $f_1(n_2)$, may depend on the prey density but is independent of the predator density. This density-independence of the predator growth rate is crucial to our results. Most published predator–prey models fall into this class.

If the model has an equilibrium point $\hat{\boldsymbol{n}} = [\hat{n}_1, \hat{n}_2]^{\mathrm{T}}$ such that $\hat{n}_1 > 0$, then $f_1(\hat{n}_2) = 0$. As a consequence, the Jacobian matrix has the form

$$\boldsymbol{A} = \begin{pmatrix} f_1(n_2) & n_1 \dfrac{\mathrm{d}f_1}{\mathrm{d}n_2} \\ \dfrac{\partial f_2}{\partial n_1} & \dfrac{\partial f_2}{\partial n_2} \end{pmatrix}\Bigg|_{\boldsymbol{n}=\hat{\boldsymbol{n}}} = \begin{pmatrix} 0 & a_{12} \\ a_{21} & a_{22} \end{pmatrix}. \tag{14}$$

As $a_{11} = 0$, the stable equilibrium point $\hat{\boldsymbol{n}}$ must be reactive. We can prove this by using the Routh–Hurwitz criteria to show that $\lambda_1(H(\boldsymbol{A}))$ is positive.

For *any* real 2×2 matrix $\boldsymbol{B}$, the Routh–Hurwitz criteria

$$\mathrm{tr}\,\boldsymbol{B} < 0, \quad \det \boldsymbol{B} > 0 \tag{15}$$

are necessary and sufficient conditions for the eigenvalues of $\boldsymbol{B}$ to have negative real parts (e.g. Murray (2002)). We assume that $\hat{\boldsymbol{n}}$ is stable, and that the Jacobian $\boldsymbol{A}$ has no eigenvalue equal to zero (a generic property of linear operators in $\mathbb{R}^n$ (Hirsch and Smale, 1974)). This implies that $\mathrm{tr}\,\boldsymbol{A} < 0$ and $\det \boldsymbol{A} > 0$.

If $H(\boldsymbol{A})$ violates either of the Routh–Hurwitz criteria, then $\lambda_1(H(\boldsymbol{A})) > 0$ and the equilibrium point is reactive. For $\boldsymbol{A}$ as given by (14), we have

$$\mathrm{tr}\,H(\boldsymbol{A}) = \mathrm{tr}\,\boldsymbol{A} < 0, \tag{16}$$

and

$$\det H(\boldsymbol{A}) = -\tfrac{1}{4}(a_{12} + a_{21})^2 < 0. \tag{17}$$

Since $\det H(\boldsymbol{A}) < 0$, the Routh–Hurwitz criteria (15) are violated, and any stable coexistence equilibrium of any predator–prey system of the form (13) is reactive.

3.2. Food chains and food webs

We now consider a linear food chain of m species, in which n_1 is the top predator and n_m is the basal species:

$$\frac{\mathrm{d}n_1}{\mathrm{d}t} = n_1 f_1(n_2), \tag{18a}$$

$$\frac{\mathrm{d}n_i}{\mathrm{d}t} = f_i(n_{i-1}, n_i, n_{i+1}), \quad \text{for } i = 2, \ldots, m-1, \tag{18b}$$

$$\frac{\mathrm{d}n_m}{\mathrm{d}t} = f_m(n_{m-1}, n_m). \tag{18c}$$

As written, (18a) implies that the per capita growth rate of the top predator depends on the density of its prey but is independent of its own density.

Linearizing (18) around the coexistence equilibrium gives the model (3) with

$$\boldsymbol{A} = \begin{pmatrix} 0 & a_{12} & & & & & \\ a_{21} & a_{22} & a_{23} & & & & \\ & a_{32} & a_{33} & a_{34} & & & \\ & & \ddots & \ddots & \ddots & & \\ & & & \ddots & \ddots & a_{m-1,m} & \\ & & & & a_{m,m-1} & a_{mm} \end{pmatrix}. \tag{19}$$

The only non-zero elements fall along the three central diagonals of $\boldsymbol{A}$. The first element on the main diagonal, a_{11}, is always zero, because $f_1(\hat{n}_2) = 0$ at the coexistence equilibrium. To show that the coexistence equilibrium is always reactive, we must show that $H(\boldsymbol{A})$ has at least one positive eigenvalue. To do that, we will make use of the following fact: any real, symmetric, matrix $\boldsymbol{B}$ with $b_{11} = 0$ and no zero eigenvalue has at least one positive eigenvalue (see Appendix A). Thus, if $H(\boldsymbol{A})$ has no zero eigenvalues, it must have at least one positive eigenvalue (since its first diagonal element is zero). As a result, any coexistence equilibrium of the food-chain model (18) is reactive, unless $\lambda_1(H(\boldsymbol{A})) = 0$. If $\lambda_1(H(\boldsymbol{A})) = 0$, then $H(\boldsymbol{A})$ is singular, but nonsingularity is a generic property of linear operators (Hirsch and Smale (1974), p. 157). Thus, any food chain model whose linearization is of the form (19) is either reactive or can be approximated arbitrarily closely by one that is reactive.

The same argument can be applied to *any* food web model in which one species, typically a top predator, has a per capita growth rate independent of its own density. Without loss of generality, one can label this species n_1. The Jacobian matrix will then necessarily have a zero as its first diagonal element, and as a result the equilibrium must be reactive. If, on the other hand, there is no species in the web whose per capita growth rate is independent of its own density, the above arguments do not apply. This situation often arises when the mortality rates of top predators—the so-called *closure terms*—are density dependent. We examine this case next.

4. Closure terms

Food web models are typically assembled from three types of components: (i) source terms that describe the dynamics of basal species that do not feed on other species in the web; (ii) consumption terms that link consumers and their resources in the web; and (iii) terms describing the mortality of top predators which are not fed upon by other species in the web. In plankton food webs, part of the mortality of top predators may result from higher-order predators (e.g. fish) that are not included in the model. Acknowledging their role in truncating the food web, the top-predator mortality terms have been dubbed *closure terms* (Steele and Henderson, 1992, 1995; Totterdell, 1993).

Most food web models include only density-independent mortality of the top predators, so the closure terms are proportional to the population densities of the species whose mortality they describe. If the closure term does reflect, at least in part, the action of higher-order predators, then the functional or numerical responses of those predators might produce apparent density-dependent mortality of the top predator, and thus a non-linear closure term.

Steele (1976) first recognized that "what appears at first as a minor technical problem, the closing of the upper end of the food chain, in fact has significant implications for the outputs ...." The effects of non-linear closure terms on the stability, asymptotic dynamics, and bifurcation structure of food chain models have been studied by Gilpin (1975), Steele (1976), Steele and Henderson (1981, 1992, 1995), Bazykin et al. (1981), Hainzl (1988, 1992), Gatto (1991), Edwards and Brindley (1996, 1999), Caswell and Neubert (1998), Edwards and Bees (2001) and Edwards and Yool (2000). Here, we examine the effects of non-linear closure terms on transient dynamics. As these non-linearities significantly increase the difficulty of mathematical analysis, we will limit our attention to the two-species predator–prey model:

$$\frac{\mathrm{d}n_1}{\mathrm{d}t} = [g_1(n_2) - \mu(n_1)]n_1, \tag{20a}$$

$$\frac{\mathrm{d}n_2}{\mathrm{d}t} = g_2(n_1, n_2), \tag{20b}$$

where n_1 and n_2 are the respective population densities of the predator and the prey. A particular example of this model is the MacArthur–Rosenzweig predator–prey model that we examine in more detail in the next section.

The closure term in model (20) is $\mu(n_1)n_1$. Caswell and Neubert (1998) suggested that if the hazards of density-independent and density-dependent mortality are independent one should write

$$\mu(n_1) = (m + \epsilon n_1^\alpha), \tag{21}$$

where m is the density-independent hazard and ϵn_1^α is the density-dependent hazard. By varying m and ϵ it is possible to change the per capita mortality from completely density-independent ($\epsilon = 0$) to completely density-dependent ($m = 0$). Varying α changes the dependence of mortality on density from decelerating ($0 < \alpha < 1$) to linear ($\alpha = 1$) to accelerating ($\alpha > 1$). The linear case corresponds to a Type I functional response in the higher-order predators, and the decelerating case crudely approximates a Type II functional response. The accelerating case seems biologically unrealistic, so we will limit our attention to $0 < \alpha \leq 1$.

If the Jacobian of (20) at a stable coexistence equilibrium $(\hat{n}_1, \hat{n}_2)$ is given by the matrix $\boldsymbol{A}$, the resilience and reactivity of the equilibrium are

$$\nu_\infty = -\frac{1}{2}\mathrm{Re}\left[a_{11}+a_{22}+\sqrt{(a_{11}-a_{22})^2+4a_{12}a_{21}}\right], \tag{22}$$

and

$$\nu_0 = \frac{1}{2}\left(a_{11}+a_{22}+\sqrt{(a_{11}-a_{22})^2+(a_{12}+a_{21})^2}\right). \tag{23}$$

In turn, $a_{11} = -\alpha\hat{n}_1^\alpha$; the other entries in $\boldsymbol{A}$ depend on α only implicitly through their dependence on $\hat{n}_1$ and $\hat{n}_2$.

The effects of non-linear closure on transient dynamics can be studied by varying α. Increasing α not only increases the non-linear response but also changes the total mortality rate μ. In order to isolate the effects of the nonlinearity, any time we change α we will simultaneously change ϵ so as to hold μ constant at $\hat{\mu}$. Given this restriction, equations (20) then imply that:

$$\frac{\mathrm{d}\hat{n}_1}{\mathrm{d}\alpha} = \frac{\mathrm{d}\hat{n}_2}{\mathrm{d}\alpha} = 0. \tag{24}$$

The relationship between resilience and the strength of density-dependent closure is complicated. Differentiating the resilience (22), and using (24), we obtain:

$$\frac{\mathrm{d}\nu_\infty}{\mathrm{d}\alpha} = \frac{\partial\nu_\infty}{\partial a_{11}}\frac{\partial a_{11}}{\partial\alpha}, \tag{25a}$$

$$= \frac{\alpha^2\hat{n}_1^{\alpha-1}}{2}\mathrm{Re}\left(1+\frac{a_{11}-a_{22}}{\sqrt{(a_{11}-a_{22})^2+4a_{12}a_{21}}}\right). \tag{25b}$$

Thus, $\mathrm{d}\nu_\infty/\mathrm{d}\alpha > 0$ unless $(a_{11}-a_{22})^2+4a_{12}a_{21} > 0$ and $\sqrt{(a_{11}-a_{22})^2+4a_{12}a_{21}} < -(a_{11}-a_{22})$. The relationship between reactivity and α, however, is more straight-forward. Differentiating the reactivity (23), and again using (24), we obtain:

$$\frac{\mathrm{d}\nu_0}{\mathrm{d}\alpha} = \sum_{i,j}\frac{\partial\nu_0}{\partial a_{ij}}\left(\frac{\partial a_{ij}}{\partial\alpha}+\sum_{k=1}^{2}\frac{\partial a_{ij}}{\partial\hat{n}_k}\frac{\mathrm{d}\hat{n}_k}{\mathrm{d}\alpha}\right) \tag{26a}$$

$$= \frac{\partial\nu_0}{\partial a_{11}}\frac{\partial a_{11}}{\partial\alpha} \tag{26b}$$

$$= -\frac{\alpha^2\hat{n}_1^{\alpha-1}}{2}\left(1+\frac{a_{11}-a_{22}}{\sqrt{(a_{11}-a_{22})^2+(a_{12}+a_{21})^2}}\right) \tag{26c}$$

$$< 0. \tag{26d}$$

Thus, increasing the strength of the density-dependence in the closure term, while holding the total per capita mortality rate constant, always decreases reactivity. But can density-dependent closure eliminate the possibility of transient growth? That is, by increasing α, can we make a reactive equilibrium nonreactive? To see if we can, we will investigate the transient dynamics of a particular predator–prey model next.

5. An example: the MacArthur–Rosenzweig predator–prey model

The MacArthur–Rosenzweig predator–prey model with a nonlinear closure term is:

$$\frac{\mathrm{d}N_1}{\mathrm{d}T} = \left[\frac{CAN_2}{N_2+B} - (M + EN_1^\alpha)\right]N_1, \tag{27a}$$

$$\frac{\mathrm{d}N_2}{\mathrm{d}T} = RN_2\left(1-\frac{N_2}{K}\right) - \frac{AN_2N_1}{N_2+B}. \tag{27b}$$

This model incorporates logistic prey growth with growth rate R and carrying capacity K, and a Holling

Type II functional response with saturation level A, and half-saturation constant B. The yield coefficient C transforms units of prey into predators. Setting

$$n_1 = \frac{AN_1}{RB}, \quad n_2 = \frac{N_2}{B}, \quad t = RT, \quad \gamma = \frac{CA}{R}, \quad \kappa = \frac{K}{B}, \quad m = \frac{M}{CA}, \tag{28a}$$

and

$$\varepsilon = \frac{EA^{\alpha-1}}{BCR^{\alpha}}, \tag{28b}$$

transforms the model into dimensionless form:

$$\frac{dn_1}{dt} = \gamma\left[\frac{n_2}{n_2+1} - (m + \varepsilon n_1^{\alpha})\right] n_1, \tag{29a}$$

$$\frac{dn_2}{dt} = n_2\left(1 - \frac{n_2}{\kappa}\right) - \frac{n_1 n_2}{n_2+1}. \tag{29b}$$

Neubert and Caswell (1997) studied the transient dynamics of system (29) in the case when the closure term is linear (i.e. when $\varepsilon = 0$). In this case, the unique positive equilibrium is given by

$$\hat{n}_1 = (1+\hat{n}_2)\left(1 - \frac{\hat{n}_2}{\kappa}\right), \tag{30a}$$

$$\hat{n}_2 = \frac{m}{1-m}, \tag{30b}$$

and the Jacobian matrix is

$$\boldsymbol{A} = \begin{pmatrix} 0 & \dfrac{\gamma\hat{n}_1}{(1+\hat{n}_2)^2} \\ -m & 1 - \dfrac{2\hat{n}_2}{\kappa} - \dfrac{\hat{n}_1}{(1+\hat{n}_2)^2} \end{pmatrix}. \tag{31}$$

The eigenvalues of $\boldsymbol{A}$ have negative real parts, and therefore the equilibrium is stable, when:

$$\frac{\kappa-1}{2} < \hat{n}_2 < \kappa. \tag{32}$$

(This corrects a typographical error in Neubert and Caswell (1997).)

In Fig. 2, we show the resilience and reactivity of this equilibrium as functions of the rescaled maximum predator growth rate γ. There is no simple relationship between these two quantities. For $\gamma < 1$, the equilibrium point is a stable node, and resilience grows monotonically with γ. For $\gamma > 1$, the equilibrium point is a stable focus, and resilience is independent of γ. Reactivity is lowest for $\gamma \approx 2.5$, and increases dramatically as γ exceeds this value.

When model (29) has nonlinear closure, the equilibrium cannot be written in closed form. The Jacobian,

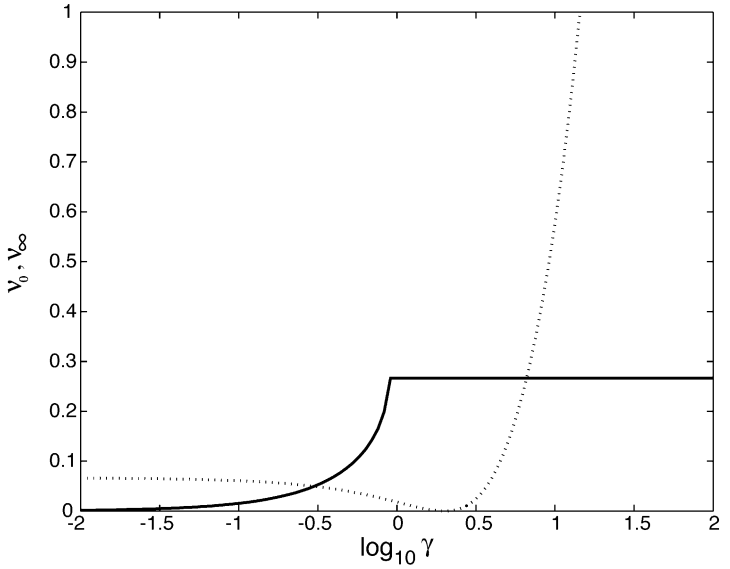

Fig. 2. The reactivity (v_0, dashed line) and resilience (v_∞, solid line) of the coexistence equilibrium of model (29) as functions of the rescaled maximum predator growth rate γ. The other parameters are set as $m = 0.4$, $\varepsilon = 0$, $\kappa = 1$.

written in terms of the equilibrium population sizes, is

$$\boldsymbol{A} = \begin{pmatrix} -\gamma\alpha\varepsilon\hat{n}_1^{\alpha} & \dfrac{\gamma\hat{n}_1}{(1+\hat{n}_2)^2} \\ -\dfrac{\hat{n}_2}{1+\hat{n}_2} & 1 - \dfrac{2\hat{n}_2}{\kappa} - \dfrac{\hat{n}_1}{(1+\hat{n}_2)^2} \end{pmatrix}. \quad (33)$$

We are interested in the reactivity and resilience calculated from $\boldsymbol{A}$ as α changes in a way that maintains the per capita mortality level; i.e. increasing α while holding $m + \varepsilon\hat{n}_1^{\alpha}$ constant.

To facilitate comparison with the results shown in Fig. 2, we fixed the equilibrium at $(\hat{n}_1, \hat{n}_2) = (\frac{5}{9}, \frac{2}{3})$ by setting $m + \varepsilon\hat{n}_1^{\alpha} = 0.4$. We set $m = 0.2$ so that $\varepsilon\hat{n}_1^{\alpha} = 0.2$. We then computed the resilience and reactivity of the equilibrium over a range of γ and α values.

Fig. 3a shows that nonlinear closure can qualitatively change the relationship between the predator's maximum per capita rate of increase (γ) and resilience (v_∞). For $\alpha = 0$, resilience is a nondecreasing function of γ (Fig. 2); for $\alpha > 0$, resilience has a peak and decreases for large γ. For γ fixed and small, resilience increases with α. For γ large, resilience is maximized at intermediate values of α. In Fig. 3b, we see that nonlinearity in the closure term can produce even more dramatic effects on reactivity. For $\alpha = 0$, the equilibrium point is always reactive (except for a single value of γ for which $v_0 = 0$), but for $\alpha > 0$ and intermediate values of γ it is not.

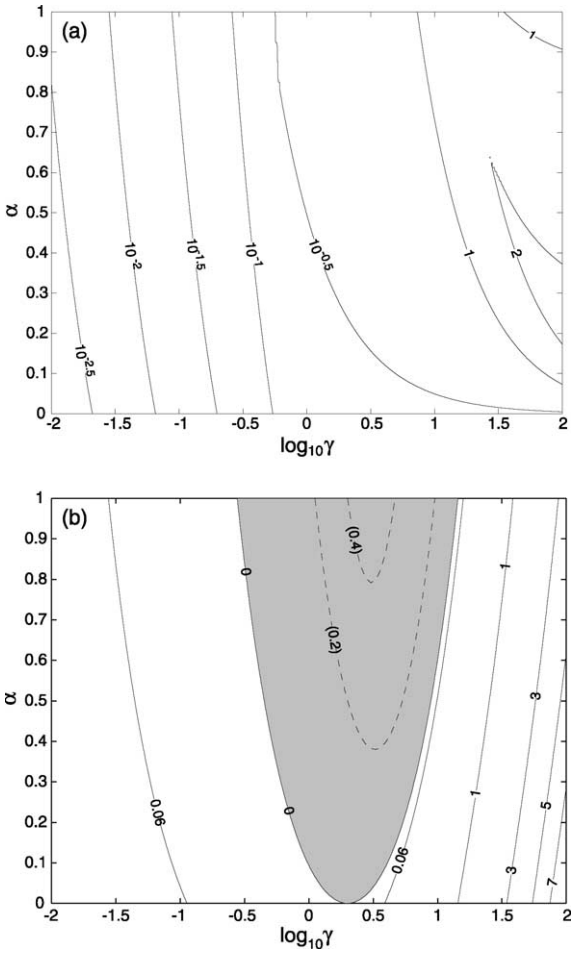

Fig. 3. Contours of (a) resilience and (b) reactivity for the coexistence equilibrium of model (29) as functions of $\log_{10}\gamma$ and the dimensionless parameter α. In this figure, $\kappa = 1$, $m = 0.2$, and the per capita predator mortality rate $m + \varepsilon\hat{n}_1^{\alpha}$ was held constant at 0.4. Numbers in parentheses are negative.

6. Discussion

We have shown that reactivity is a property of the equilibria of a class of predator–prey and food web systems described by ordinary differential equations. That class—those with at least one species with density-independent mortality—is large and important. An idea of just how large is given by the fact that it includes every predator–prey, chemostat and food web model in the recent texts by Gurney and Nisbet (1998) and Kot (2001), all but two of the predator–prey models in the monograph of Murdoch et al. (2003), and all but one of the many food web models in DeAngelis (1992). In addition to these theoretically-oriented models, it includes such empirical nutrient cycling models as those for fjord ecosystems (Ross et al., 1993) and the oceanic mixed layer (Fasham et al., 1990).[1]

The lack of density-dependent mortality in food web models does not, of course, guarantee that it is rare in nature. But to the extent that these models reflect

[1] We note that many empirical food web models contain parameters that are forced by periodically varying seasonal conditions. Although we have presented the reactivity of equilibria, periodic attractors, resulting from external forcing or from intrinsic limit cycles, may also be reactive. The proper definition of reactivity in such cases is still an open problem.

the mechanisms of actual food webs, reactivity is the rule, not the exception. Thus, we can expect that perturbations of stable food webs will, in the short term, be amplified. Even small perturbations may produce large excursions from equilibrium. The consequences of this can be profound. A short-term study may fail to detect the equilibrium, or any tendency to return to it, at all. If perturbations occur repeatedly (as is certainly true in nature), whether the system returns to equilibrium between perturbations will depend more on the reactivity than on resilience. We conjecture that reactive systems will have higher variance around their equilibria than non-reactive systems.

Acknowledgements

We thank the participants in the 2000 Nantucket Mathematical Ecology Retreat for helpful discussions. This research was supported in part by funding from the National Science Foundation (DEB-9973518, OCE-0083976). WHOI Contribution 11179.

Appendix A

Here, we prove that if $\boldsymbol{B}$ is a real, symmetric, $m \times m$ matrix with $b_{11} = 0$ and no zero eigenvalue then $\boldsymbol{B}$ has at least one positive eigenvalue. This fact is a corollary of the *inclusion principle* (Horn and Johnson, 1985):

Theorem 1. *Let $\boldsymbol{C}$ be an $m \times m$ Hermitian matrix, let r be an integer with $1 \leq r \leq m$, and let $\boldsymbol{C_r}$ denote any $r \times r$ principal submatrix of $\boldsymbol{C}$ (obtained by deleting any $m - r$ rows and the corresponding columns from $\boldsymbol{C}$). Let the eigenvalues of $\boldsymbol{C}$, $\eta_i(\boldsymbol{C})$, be arranged such that $\eta_1(\boldsymbol{C}) \leq \eta_2(\boldsymbol{C}) \leq \cdots \leq \eta_m(\boldsymbol{C})$. For each integer k, such that $1 \leq k \leq r$ we have:*

$$\eta_k(\boldsymbol{C}) \leq \eta_k(\boldsymbol{C_r}) \leq \eta_{k+m-r}(\boldsymbol{C}). \tag{A.1}$$

Applying this theorem to $-\boldsymbol{B}$, with $r = 1$, $k = 1$, and $-b_{11}$ as the corresponding principal submatrix, shows that $\eta_1(-\boldsymbol{B}) \leq \eta_1(-b_{11}) = 0$. Thus, the smallest eigenvalue of $-\boldsymbol{B}$ is not positive. The largest eigenvalue of $\boldsymbol{B}$ must therefore be nonnegative. If $\boldsymbol{B}$ has no zero eigenvalues, its largest eigenvalue must be positive.

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