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Disease-induced stabilization of predator–prey oscillations

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ABSTRACT

Parasites are an integral part of virtually all food webs and species communities. Here we consider the invasion of a resident predator–prey system by an infectious disease with frequency-dependent transmission spreading within the predator population. We derive biologically plausible and insightful quantities (demographic and epizootiological reproduction numbers) that allow us to completely determine community composition. Successful disease invasion can have two contrary effects in driving its host population to extinction or in stabilizing predator–prey cycles. Our findings contradict predictions from previous models suggesting a destabilizing effect of parasites. We show that predator infection counteracts the paradox of enrichment. In turn, parasite removal from food webs can have catastrophic effects. We discuss the implications for biological control and resource management on more than one trophic level.

1. Introduction

Infectious diseases can have regulating effects not only on their host population, but also on other species their host interacts with (Anderson and May, 1986; Grenfell and Dobson, 1995; Hudson et al., 2001). Ecologists and epizootiologists alike become increasingly interested in the structuring effects of parasites and pathogens within food webs and multiple-species communities (Dobson and Hudson, 1986; Sait et al., 2000; Holt et al., 2003; Hudson et al., 2006; Lafferty et al., 2006, 2008). Here we consider a predator–prey model with an infectious disease circulating in the predator population. While the effect of parasites and pathogens in prey populations received a lot of attention, relatively little is known yet about the consequences of predator infection—both empirically and theoretically, see the review by Hatcher et al. (2006).

Parasites and pathogens can be broadly divided into microparasites (viruses, bacteria and protozoa) and macroparasites (helminths and other metazoan parasites). While microparasites effectively divide the host into distinct epidemiological classes (like susceptible and infective), mathematical models of macroparasitic infection need to keep track of the frequency-distribution of individual macroparasites within the host population. Transmission of microparasites can occur directly between definitive hosts (referred to as direct life cycle) or involve one or more intermediate host species (referred to as indirect cycle), cf. Dobson (1988).

Existing mathematical models suggest that disease introduction into the predator population tends to destabilize established predator–prey communities. This has been observed for microparasites with both direct (Anderson and May, 1986; Xiao and Van Den Bosch, 2003; Haque and Venturino, 2007) and indirect life cycles (Dobson, 1988; Fenton and Rands, 2006). Macroparasitic models generally have a tendency to unstable dynamics, because they consider the parasite burden in the host in an additional equation (Anderson and May, 1980; Dobson and Keymer, 1985; Dobson, 1988).

Here we show that the scenario of destabilization does not always hold true. The effect of disease introduction can be quite the opposite, namely to stabilize oscillatory predator–prey dynamics. We demonstrate this in one of the simplest models possible, for which we couple the classical Rosenzweig–MacArthur (1963) predator–prey model with a general *SI* (susceptible → infective) model for microparasites with direct life cycles. Our model thus brings together the two fields of ecology and epidemiology, as it extends classical epidemiological approaches (usually assuming constant host population sizes) by demography (varying population sizes) and ecological interactions (predation).

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Previous papers have predominantly focused on models with (i) a linear functional response of predators and (ii) density-dependent disease transmission (Dobson and Keymer, 1985; Anderson and May, 1986; Dobson, 1988; Lafferty, 1992; Han et al., 2001; Venturino, 1994, 2002; Xiao and Van Den Bosch, 2003). These are strongly simplifying assumptions. First, the feeding rate of predators usually saturates (e.g. Turchin, 2003). The only studies considering more realistic saturating functional responses so far are by Hadeler and Freedman (1989), Freedman (1990), Fenton and Rands (2006) (using Holling type II) and Haque and Venturino (2007) (using ratio-dependence). The paper by Hadeler and Freedman (1989) is particularly interesting as it explicitly studies the case of an oscillating resident predator–prey community. Upon disease invasion, the amplitude of the oscillation increases, indicating further destabilization. The other models do not consider the case of an oscillating resident predator–prey system or the disease-free model does not oscillate due to other modeling assumptions (Kribs-Zaleta, 2006).

Oscillating resident communities are particularly worthwhile to study, because prey–predator (or host–parasitoid, plant–herbivore, consumer–resource) systems are well-known examples of inherently fluctuating populations (e.g. Turchin, 2003). They represent interesting and relevant scenarios in biological control and resource management, because recurrent outbreaks in both terrestrial and aquatic ecosystems pose central problems in ecology (e.g. insect outbreaks and algal blooms, see Anderson and May, 1980; Hallegraeff, 1993; Dwyer et al., 2004; Hilker and Malchow, 2006). Oscillations are also a concern of biological conservation, because populations could reach such small abundances that they are likely to go extinct (e.g. Rosenzweig, 1971).

Second, density-dependent transmission assumes that the number of contacts between individuals increases linearly with host abundance, i.e. the population is well-mixed and each individual has the same probability of contact with any other individual. While this is the classical 'mass-action' assumption (Keeling, 2005) and has been demonstrated to hold for a number of diseases (e.g. Caley and Ramsey, 2001; Ramsey et al., 2002; Brown and Brown, 2004), sexually transmitted diseases (e.g. Thrall et al., 1993) or infections in populations with territorial or social behavior (e.g. Altizer et al., 2003) are clear examples suggesting frequency-dependent transmission, where the number of contacts between predators is independent of population size and remains constant. For instance, frequency-dependent transmission has been suggested for models of feline retroviruses in domestic cats (Courchamp et al., 1995; Fromont et al., 1998), phocine distemper virus in seals (Diekmann et al., 1995), cowpox virus in mixed populations of free-living rodents (Begon et al., 1999) and brucellosis in bisons (Dobson and Meagher, 1996). McCallum et al. (2001) and Begon et al. (2002) discuss the differences between frequency- and density-dependent transmission as well as other infection rates in more detail.

A key quantity in our analysis will be the demographic and epizootiological reproduction number. It can be defined as the expected number of offspring a typical individual produces in its life or, in epizootiology, as the expected number of secondary infections produced by a single infective individual in a completely susceptible population during its entire infectious period. While the concept of reproduction numbers was initially developed in demography already in the early 20th century (e.g. Lotka, 1925), they became a standard tool in epidemiology since the work of Anderson and May (1991) and Diekmann et al. (1990), cf. the review by Heesterbeek (2002). We will use reproduction numbers as helpful tools in determining the persistence (if they are larger than one) or extinction (if they are smaller than one) of a species. This will allow us to categorize the community composition of prey, predators and disease. The threshold concept inherent in reproduction numbers has been used in previous studies of eco-epidemiological models (e.g. Hadeler and Freedman, 1989; Han et al., 2001; Xiao and Chen, 2001; Hethcote et al., 2004). However, to our knowledge, this study is one of the first that bases the entire community classification completely on reproduction numbers and that also attributes biological meaning to all of these quantities.

This paper is organized as follows. The next section introduces the model with a saturating functional response and frequency-dependent disease transmission. In Section 3, we define the reproduction numbers that can completely explain the resulting community structure. Section 4 investigates parasite establishment in a resident predator–prey community in more detail numerically. This is where we observe disease-induced stabilization. Finally, we discuss our model and results, in particular the mechanisms favoring stabilization, and draw conclusions relevant for disease and community ecology.

2. Model description and assumptions

The model structure is shown in the transfer diagram of Fig. 1. The underlying predator–prey model is the classical Rosenzweig–MacArthur (1963) model:

$$\underbrace{\frac{dX}{dT}}_{\text{prey rate of change}} = \underbrace{bX\left(1-\frac{X}{K}\right)}_{\text{growth}} \underbrace{-\frac{aX}{H+X}Y}_{\text{predation}}, \tag{1}$$

$$\underbrace{\frac{dY}{dT}}_{\text{predators rate of change}} = \underbrace{e\frac{aX}{H+X}Y}_{\text{conversion of prey consumed}} \underbrace{-dY}_{\text{natural mortality}}. \tag{2}$$

The logistically growing prey population X has an intrinsic per-capita growth rate b and a carrying capacity K. The functional response of predators is of Holling type II with a maximum consumption rate a, the half-saturation constant is H and the conversion efficiency is e. The predators Y have a linear death rate with per-capita mortality d. Considering the spread of an infectious disease within the predator population, we assume that the total predator population $Y = S + I$ can be split into a susceptible and an infective part. That is, there is no recovery from the disease. Then we obtain two differential equations for the

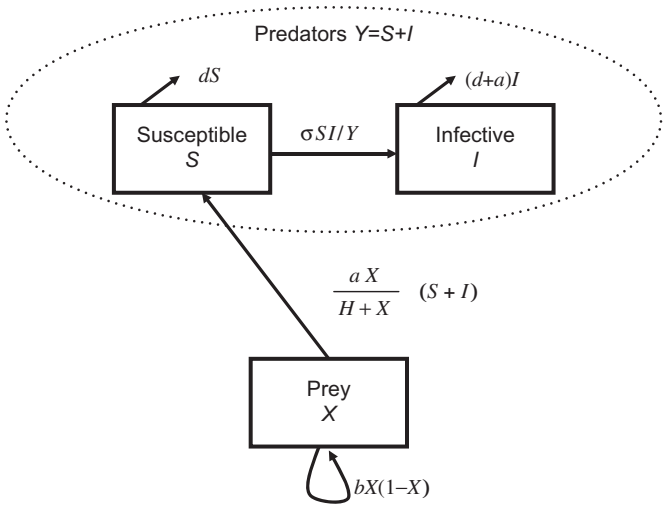

Fig. 1. Transfer diagram of model (5)–(7) in terms of susceptible and infective predators and the prey.

single equation (2):

$$\underbrace{\frac{dS}{dT}}_{\substack{\text{susceptibles}\\ \text{rate of change}}} = \underbrace{e\frac{aX}{H+X}(S+I)}_{\substack{\text{growth by conversion of}\\ \text{prey consumed}}} \underbrace{-dS}_{\substack{\text{natural}\\ \text{mortality}}} \underbrace{-\sigma\frac{SI}{S+I}}_{\substack{\text{disease}\\ \text{transmission}}}, \tag{3}$$

$$\underbrace{\frac{dI}{dT}}_{\substack{\text{infectives}\\ \text{rate of change}}} = \underbrace{\sigma\frac{SI}{S+I}}_{\substack{\text{disease}\\ \text{transmission}}} \underbrace{-(d+\alpha)I}_{\substack{\text{natural and disease-}\\ \text{related mortality}}}. \tag{4}$$

Infective predators suffer an additional disease-related mortality (virulence) α. Transmission takes place directly between infective and susceptible predators, i.e. we consider a microparasite with direct life cycle. The incidence (new cases per unit time) is assumed to be frequency-dependent with transmission coefficient σ.

Our model is now described by the three equations (1), (3) and (4). Let us rewrite the equations for S and I in Y and i variables with i being the prevalence $i = I/(S+I)$, i.e. the fraction of the host population being infective. This has two advantages. First, it removes the singularity in the disease transmission terms for $S+I=0$. Second, in the case of host population extinction ($Y=0$), it allows to distinguish between ecological (e.g. too high predator mortality) and epidemiological factors (i.e. disease-induced extinction) as underlying mechanisms. In the process of vanishing host population size, the prevalence becomes zero in the former case, while the prevalence remains strictly positive in the latter case. This reflects that disease transmission is ongoing even in small populations (cf. Hethcote, 2000; de Castro and Bolker, 2005a).

The model equations can be simplified by introducing dimensionless variables. Choosing $N = X/K$ for the prey population, $P = Y/(eK)$ for the predator population and $t = eaT$ for time, we reduce the number of parameters from seven to five, namely

$$r = \frac{b}{ea}, \quad h = \frac{H}{K}, \quad m = \frac{d}{ea},$$
$$\beta = \frac{\sigma}{ea}, \quad \mu = \frac{\alpha}{ea},$$

which are all positive. We thus arrive at the dimensionless model

$$\frac{dN}{dt} = rN(1-N) - \frac{NP}{h+N}, \tag{5}$$

$$\frac{dP}{dt} = \frac{NP}{h+N} - mP - \mu Pi, \tag{6}$$

$$\frac{di}{dt} = \left[(\beta-\mu)(1-i) - \frac{N}{h+N}\right]i, \tag{7}$$

which is formulated in terms of the prey population, total predator population and disease prevalence within the predators. This is the model we will analyze in the following sections.

3. Community composition: disease and predator reproduction numbers

This section deals with the community composition of model system (5)–(7). It can be completely explained by four threshold quantities that are demographic and epizootiological reproduction numbers. Note that it can also be obtained independently by a mathematical stability analysis (see Appendix A). We will show that the asymptotic long-term behavior, which is determined by stable equilibria $\boldsymbol{E} = (N^*, P^*, i^*)$, can be retrieved by biologically plausible reproduction numbers.

We start by considering a resident predator–prey community without disease. A necessary condition for these two species to coexist is that the predators have a positive net growth rate. We can formulate this in terms of the *disease-free demographic reproduction number*

$$R_0^P = \frac{1}{(h+1)m} \tag{8}$$

that gives the expected number of offspring ($1/(h+1)$) of an average predator individual in its lifetime ($1/m$). It is based upon the assumptions that the prey are at carrying capacity ($N=1$) and the disease is absent ($i=0$). Clearly, if $R_0^P > 1$, the predators can sustain itself on the prey (second column in Table 1). As in the Rosenzweig–MacArthur model, the disease-free coexistence state $\boldsymbol{E}_4 = (N_4^*, P_4^*, 0)$ can be either stable or unstable and surrounded by a limit cycle.

Regarding disease dynamics, we consider the case $\beta > \mu$ throughout this paper. Otherwise the prevalence declines to zero monotonically, cf. Eq. (7), and we are left with the disease-free predator–prey subsystem. The condition for successful disease invasion of the resident predator–prey community can be expressed by the *epizootiological reproduction number*

$$\mathscr{R}_0 = \frac{\beta}{m+\mu}. \tag{9}$$

$\mathscr{R}_0$ can be defined as the average number of secondary cases (β) due to a single infective individual introduced into a completely susceptible host population during its infectious period ($1/(m+\mu)$). If $\mathscr{R}_0 > 1$, all three species (prey–predator–disease) can coexist (third column in Table 1). Numerical simulations shown in the following section indicate that the enzootic coexistence state $\boldsymbol{E}_5 = (N_5^*, P_5^*, i_5^*)$ is either stable or unstable and surrounded by sustained limit cycle oscillations.

If the disease causes too many deaths in its host population, however, the predators will go extinct. The corresponding quantity

Table 1
Community composition and the stability of equilibria (N^*, P^*, i^*)

	$R_0^P > 1$			$R_0^P < 1$	
	$\mathscr{R}_0 < 1$	$\mathscr{R}_0 > 1, R_i^P > 1$	$\mathscr{R}_0 > 1, R_i^P < 1$	$\mathscr{R}_i < 1$	$\mathscr{R}_i > 1$
$\boldsymbol{E}_1 = (1,0,0)$	Unstable	Unstable	Unstable	Stable	Unstable
$\boldsymbol{E}_3 = (1,0,i_3^*)$	–	–/Unstable	Stable	–	Stable
$\boldsymbol{E}_4 = (N_4^*, P_4^*, 0)$	Stable/cyclic	Unstable	Unstable	–	–
$\boldsymbol{E}_5 = (N_5^*, P_5^*, i_5^*)$	–	Stable/cyclic	–	–	–
	Prey–predator	*Prey–predator–disease*		*Prey only*	

R_0^P and R_i^P are the demographic reproduction numbers of the disease-free and infective predator population, respectively. $\mathscr{R}_0$ is the epizootiological reproduction number of the disease. $\mathscr{R}_i$ is the reproduction number of the disease prevalence within the predator population and can be substituted by $\mathscr{R}_0/R_i^P$. Details of the stationary states are in Appendix A. '–' indicates that an equilibrium does not exist or is unfeasible. 'Cyclic' means that an equilibrium is unstable and surrounded by a limit cycle. The equilibria $\boldsymbol{E}_0 = (0,0,0)$ and $\boldsymbol{E}_2 = (0,0,1)$ are always unstable and therefore omitted.

is the predators' *enzootic demographic reproduction number*

$$R_i^P = \frac{1}{(h+1)(m+\mu i_5^*)} = \frac{\beta}{(\beta-\mu)(\mu+m)(h+1)} \tag{10}$$

that takes into account the additional mortality rate μ for the infective part of the population (i_5^*). The subscript of R_i^P now labels the assumption of the predators being subject to enzootic infection. If $R_i^P<1$, the predators go extinct with only the prey population remaining and reaching carrying capacity, see the stationary state $\boldsymbol{E}_3=(1,0,i_3)$ and the fourth column in Table 1. This disease-induced extinction is one of the two mechanisms leading to a vanishing predator population. The other one is realized if the predators are not able to establish themselves ($R_0^P<1$, two right columns in Table 1). In this situation we can distinguish between two cases. In the first one ($\mathscr{R}_i<1$), the disease goes extinct before the predators do. In the second one ($\mathscr{R}_i>1$), the disease remains prevalent in the vanishing predator population. The former and the latter correspond to the stationary states $\boldsymbol{E}_1=(1,0,0)$ and $\boldsymbol{E}_3=(1,0,i_3^*)$, respectively. Which one of these will be attained depends on the threshold quantity

$$\mathscr{R}_i = (\beta-\mu)(h+1) = \frac{\mathscr{R}_0}{R_i^P}. \tag{11}$$

This gives the number of secondary infections discounted by the fact that the host population size is changing (cf. Thieme, 1992). Consider the following: if the prevalence is small compared to the predator population size, the infective part of the population decays at an approximate exponential rate $\beta-m-\mu$ (see Eq. (4)), whereas the total population size decays approximately at rate $1/(h+1)-m$ (see Eq. (6)). The difference between these two exponential rates defines the value of $\mathscr{R}_i$ and basically determines if disease prevalence will grow in small predator populations ($\mathscr{R}_i>1$) or decay ($\mathscr{R}_i<1$). As $\mathscr{R}_i$ can also be obtained directly from Eq. (7) for the prevalence, we refer to it as the *prevalence reproduction number*.

Note that $\mathscr{R}_i$ can also be written as the ratio of the reproduction numbers of infectives ($\mathscr{R}_0$) and the total host population in the presence of disease (R_i^P), cf. Eq. (11). We can therefore base our classification on three rather than four pertinent thresholds.

The entire community composition, i.e. persistence of (i) prey alone, (ii) prey and predator or (iii) prey, predator and disease, can be predicted by biologically meaningful reproduction numbers. The prey can always grow and survive. Hence, the stationary states $\boldsymbol{E}_0=(0,0,0)$ and $\boldsymbol{E}_2=(0,0,1)$ are always unstable. The classification by the reproduction numbers thus coincides with the results from linear stability analysis, as shown in Table 1.

4. Disease-induced stabilization of predator–prey oscillations: numerical simulations

The subject of this section is the nonlinear aspect involved in the invasion of a resident predator–prey community by a pathogen. First, we will use numerical bifurcation analysis and simulations to show how disease stabilizes predator–prey oscillations. Next, we will give an intuitive explanation of this stabilizing effect. Finally, we will summarize the dynamical behavior to be expected for different disease characteristics.

Fig. 2 illustrates how community dynamics changes with varying transmissibility. We assume that predators are strong enough to exist on the prey in the disease-free system, i.e. $R_0^P>1$. For $\beta<0.9$, equivalently $\mathscr{R}_0<1$, the disease cannot establish within the resident predator–prey system. The resulting dynamics are large-amplitude oscillations of predators and prey, i.e. $\boldsymbol{E}_4=(N_4^*,P_4^*,0)$ is unstable and surrounded by a limit cycle.

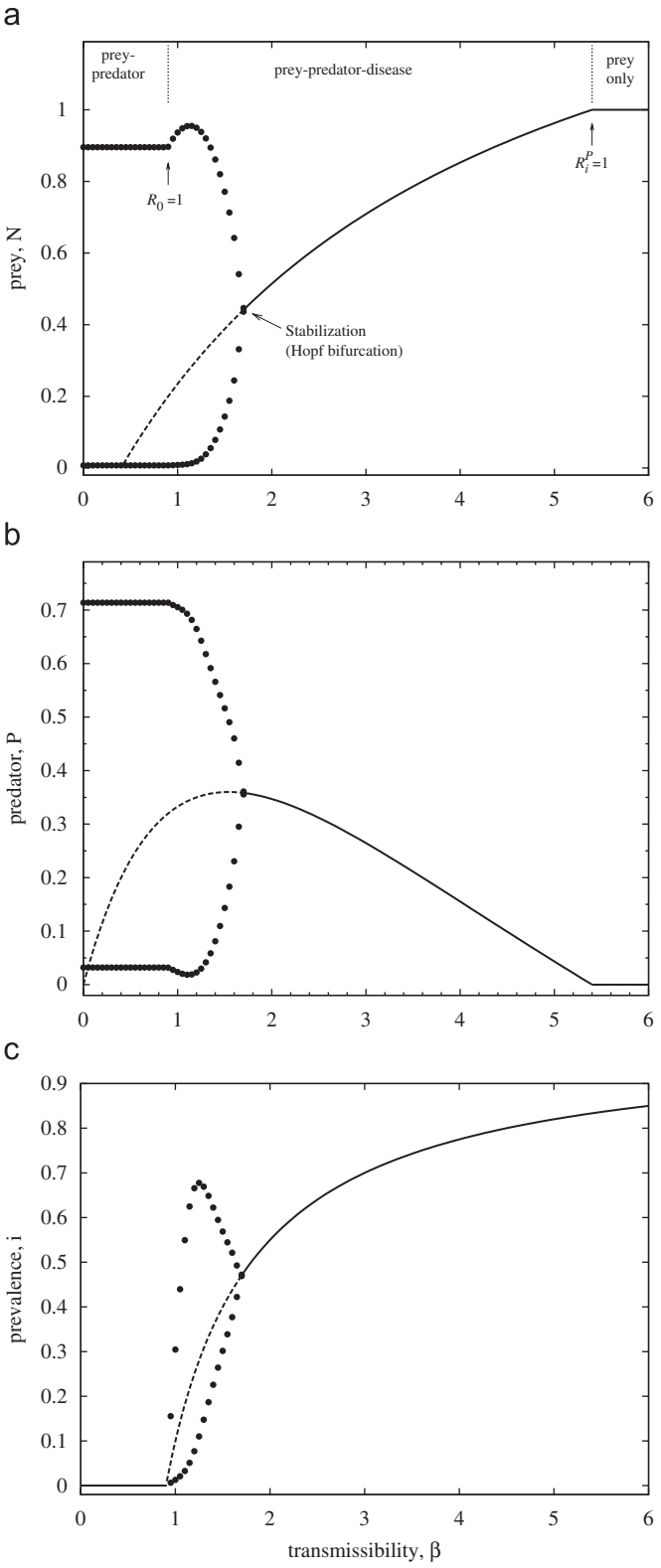

Fig. 2. Disease invasion and stabilization of a cyclic predator–prey community. Solid (dashed) lines represent stable (unstable) equilibria. Circles indicate the maximum and minimum amplitudes of limit cycle oscillations. Unstable (semi-) trivial equilibria are not shown for the sake of clarity. Parameter values: $r=1$, $h=0.2$, $m=0.5$, $\mu=0.4$.

As the predator–prey subsystem does not depend on disease parameters, the cycle as well as the corresponding stationary state remain constant for all transmissibility values in this range.

Upon disease establishment ($\mathscr{R}_0 > 1$), the two-species cycle in the predator–prey subsystem becomes a three-species cycle of enzootic coexistence. The amplitudes of the cycles shrink with increasing transmissibility, and the oscillations eventually stabilize if $\beta_H \approx 1.669$ is passed. Mathematically, this corresponds to a backward Hopf bifurcation: the enzootic stationary state $\boldsymbol{E}_5 = (N_5^*, P_5^*, i_5^*)$ that is unstable for $\beta < \beta_H$ gains local stability and the surrounding limit cycle attractor disappears. Hence, all three species are now in stable equilibrium. Increasing prevalence (larger β) continues to depress the host population size and ultimately drives it to extinction from $\beta = 5.4$ on (equivalently, $R_i^P < 1$). Without predators, the prey grow to carrying capacity.

To understand why the predator–prey oscillations get stabilized by the disease, it is instructive to consider the nullplanes of system (5)–(7) in three-dimensional state space (Fig. 3). Fixing the prevalence at $i = 0$, we obtain a projection in the disease-free predator–prey system with a single-humped prey nullcline and a vertical predator nullcline. It is well-known that the predator nullcline being on the right-hand side from the hump results in a stable equilibrium (Rosenzweig, 1971). In the top of the hump a Hopf bifurcation occurs. Enriching the prey population by increasing the carrying capacity changes the shape and height of the prey nullcline and moves its peak to the right, i.e. the predator nullcline may be left from the peak. This destabilizes the equilibrium and generates limit cycles with increasing amplitude (paradox of enrichment, Rosenzweig, 1971). Fig. 3 illustrates a large-amplitude cycle in the predator–prey subplane.

Increasing the predator mortality shifts the vertical predator nullcline towards the right-hand side (e.g. Case, 2000). Its impact is therefore stabilizing. Disease invasion in the predator population has a similar effect, as infection induces additional mortality in the predators and thus effectively increases their mortality. The sum of natural and disease-induced per-capita mortality could be replaced by a total mortality

$$\widehat{m}(i) = m + \mu i. \tag{12}$$

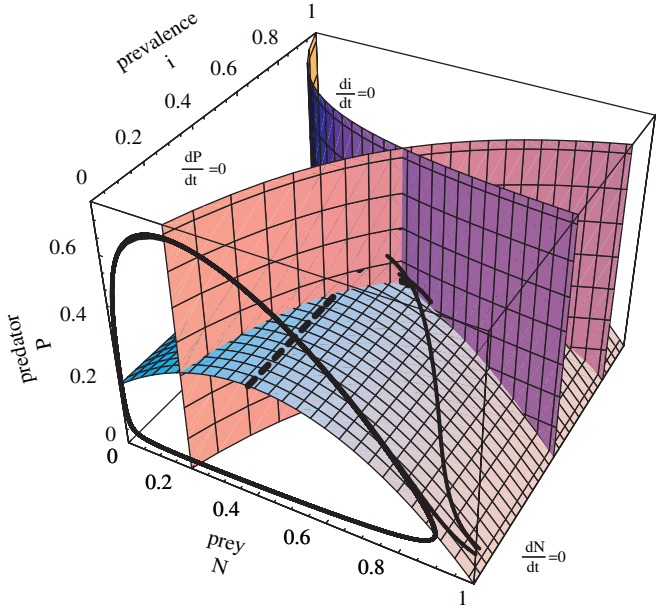

Fig. 3. Disease prevalence bends the predator nullplane towards the right-hand side and back over the hump of the prey nullplane (dashed line), thus counteracting the paradox of enrichment. Solid lines are two example trajectories of the disease-free subsystem showing limit cycle oscillations and the enzootic three-species system showing damped oscillations. Parameter values as in Fig. 2 with $\beta = 2$.

Solving for the predator nullplane (by setting the right-hand side of Eq. (6) equal to zero),

$$N = \frac{h(m + \mu i)}{1 - (m + \mu i)} = \frac{h\widehat{m}(i)}{1 - \widehat{m}(i)}, \tag{13}$$

one can clearly see the impact of invading disease. The prey population increases with disease prevalence in the predators. This eventually pushes the equilibrium point back over the hump (shown by the dashed line) into the region where predator–prey dynamics are stable. An example is given in Fig. 3, where the disease moves the intersection of all three nullplanes right from the hump. The trajectory spirals towards a stable equilibrium, i.e. the disease damps the oscillations inherent in the predator–prey dynamics. Note that the expression in (13) is always positive (i.e. $\widehat{m}(i) < 1$, which is guaranteed by $R_i^P > 1 \Leftrightarrow \widehat{m}(i) < 1/(h+1)$).

The disease-induced mortality depends on the prevalence and is therefore a dynamic variable, cf. Eq. (12). In fact, the disease-related mortality does not completely bear analogy with the natural mortality. Numerical simulations indicate that the predator nullcline projection actually needs to be shifted a bit further than just right from the hump. This is because the system with disease is three-dimensional.

Finally, let us consider how the composition and stability of the prey–predator–disease community change when varying not only transmissibility β, but also virulence μ. This is illustrated in the two-parameter bifurcation diagram in Fig. 4. Note that β and μ are the only disease-related parameters. That is, Fig. 4 could help answering important questions in biological control: Which kind of virus should be introduced, in order to yield a certain behavior?

The dashed line in Fig. 4 corresponds to $\mathscr{R}_0 = 1$. It delineates the parameter region of disease-free dynamics and indicates the establishment of the disease. In our case, the two-species limit cycle in the predator–prey subsystem bifurcates into the interior of the three-species state space and becomes an enzootic cycle. The bold line is the Hopf bifurcation line, corresponding to the limit cycle disappearance and gain of stability for the enzootic coexistence state (disease-induced stabilization). The dotted/solid line corresponds to $R_i^P = 1$. That is, disease-induced extinction takes place when the enzootic equilibrium disappears in a transcritical bifurcation and exchanges stability with the disease-induced extinction state. Note that the one-parameter

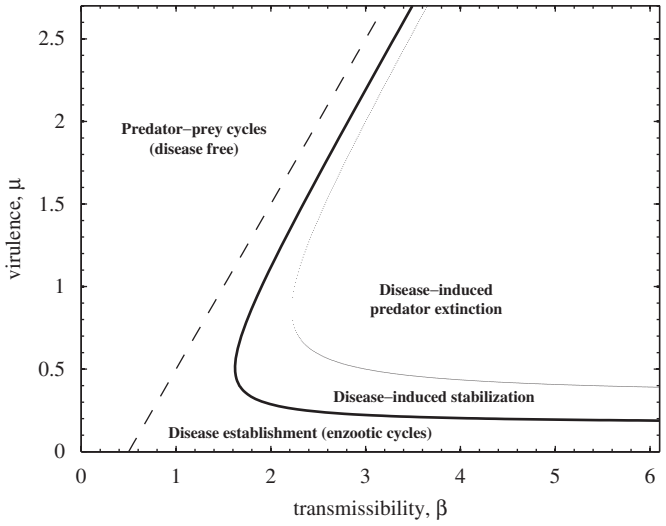

Fig. 4. Community composition and stability in the pathogen-related parameter plane (β, μ). The bold line indicates the stabilization of limit cycle oscillations. It is obtained by a two-parameter continuation of the Hopf bifurcation. The dashed and dotted/solid line correspond to $\mathscr{R}_0 = 1$ and R_i^P, respectively. Other parameter values as in Fig. 2.

bifurcation diagram in Fig. 2 can be retrieved from the two-parameter bifurcation diagram when fixing $\mu = 0.4$.

Fig. 4 shows that disease-induced stabilization as well as disease-induced extinction are nested within the parameter region where the disease can establish ($\mathscr{R}_0 > 1$). They can therefore be seen as more 'extreme' events in the case of successful parasite invasion. Both scenarios require certain minimum values for the transmissibility as well as for the virulence. In the case of disease-induced stabilization, in which we are interested here, this can be easily understood. First of all, the transmissibility needs to be high enough to ensure a large enough prevalence that shifts the predator nullcline across the hump (cf. Fig. 3). Similarly, the virulence is required to be high enough, in order to sufficiently increase host mortality. In particular, there is a minimum virulence level $\mu_{min} \approx 0.19$, below which disease-induced stabilization cannot take place—even for high transmissibilities. Stabilization is most likely to occur in parameter regions with low/intermediate virulences $\mu > \mu_{min}$ and intermediate or high transmissibilities (e.g. parameter region around the label 'disease-induced stabilization' in Fig. 4).

5. Discussion and conclusions

Nonlinear interactions between predators and prey are well-known to generate endogenous oscillations. We have shown, to our knowledge for the first time, that these fluctuations can be stabilized by an infectious disease spreading within the predator population. This challenges the current view of destabilizing disease impacts (Dobson and Keymer, 1985; Anderson and May, 1986; Dobson, 1988; Hadeler and Freedman, 1989; Xiao and Van Den Bosch, 2003; Fenton and Rands, 2006; Haque and Venturino, 2007), which also similarly exists for disease infecting prey populations (e.g. Anderson and May, 1986; Beltrami and Carroll, 1994; Hall et al., 2005; Hilker and Malchow, 2006; Roy and Holt, 2008). Moreover, our results appear to contradict the observation of de Castro and Bolker (2005b) that parasite-induced cycles are more likely to occur in larger communities.

Our findings are also of relevance for biological control, as infectious diseases can be used as control agents of undesirable species such as biological invaders. This study interestingly suggests that parasites can have regulating effects on more than one trophic level and be utilized for management purposes in multi-species systems. The introduction of disease cannot only control or eradicate the predator, but also allow the prey species to recover. For example, pathogens could potentially be used to control mammal pest species such as feral domestic cats (predators) on oceanic islands that have devastating impacts on native prey species (e.g. sea birds), cf. Courchamp and Sugihara (1999), Courchamp et al. (2003) and Nogales et al. (2004).

What is the stabilizing mechanism in our model? The effect of the disease is solely to increase predator mortality, which decreases predator population size and the predation pressure on the prey. This, in turn, increases prey population size and the density dependence felt by the prey population, which is a stabilizing factor. Infection thus indirectly couples predator mortality with prey population size. A similar inhibition of the predator population by high densities of the prey occurs in the presence of toxic prey species (Roy and Chattopadhyay, 2007) or can be achieved by vertical migration of zooplankton (Morozov et al., 2007). A qualitatively very similar shift of the predator nullplane as in Fig. 3 emerges if the predators have their own (top-)predators, cf. figure 1 in Rosenzweig and MacArthur (1963) and figure 2 in Oksanen et al. (1981). (In other models, top-predators can also further destabilize the dynamics, e.g. Hastings and Powell, 1991; Rinaldi and De Feo, 1999.) These forms of indirect predator density dependence have in fact been discussed as a stabilizing mechanism (Abrams and Walters, 1996), in particular when the functional response of the top-predator is linear (Abrams and Roth, 1994). Note that the impact of disease prevalence on the predator mortality is linear, too, although the situation in our model is more complex as the prevalence is also coupled with the prey population.

Disease transmission in our model is frequency-dependent. This is known to be stabilizing in single-species models with exponential (Busenberg and van den Driessche, 1990) and logistic growth (Zhou and Hethcote, 1994) as well as an Allee effect (Hilker et al., 2007). Since the stabilization takes place for a large enough prevalence (Fig. 3) or transmissibility (Fig. 2), this provides another intuitive explanation of the stabilization observed, namely a prevailing impact of the (stabilizing) disease over the (oscillatory) predator–prey dynamics.

Density-dependent disease transmission, in contrast, appears to have a more destabilizing influence (e.g. Hilker et al., accepted for publication). The difference between these two transmission modes is that the per-capita contact rate of individuals increases linearly with population size (density-dependent) or remains constant (frequency-dependent). As a consequence, the number of secondary infections in our model is constant and $\mathscr{R}_0$ does not depend on predator population size. Hence, there is no critical community size (e.g. Anderson et al., 1981) that is necessary for disease establishment. If there were such a condition, disease invasion in a fluctuating population would vary periodically, cf. Anderson et al. (1981, section 13) and Hadeler and Freedman (1989).

The stabilizing impact of disease invasion does not seem to be restricted to frequency-dependent transmission. Numerical simulations indicate that density-dependent infection can stabilize predator–prey oscillations as well. However, the dynamics appears to be more complicated (involving bistability and unstable limit cycles due to a subcritical Hopf bifurcation) and will be described in more detail elsewhere. Considering that virulent infectious diseases increase predator mortality, we expect stabilization by disease invasion to be a rather common phenomenon in the Rosenzweig–MacArthur model independent of the transmission mode (e.g. frequency- or density-dependent or something else). The prevalence of disease simply needs to be large enough to sufficiently increase the total predator mortality, cf. Eq. (12).

Fig. 2 shows a moderate increase in the amplitudes of predator–prey oscillations just after disease invasion ($\mathscr{R}_0$ slightly larger than 1). This effect also occurs for different parameter sets. Adding parasites as a third species to an established community could be expected to result in more complex rather than stabilized dynamics. For instance, infective predators can be regarded as another trophic level depressing susceptible predators and feeding upon the prey, which is similar to an intraguild predation food web. Theory predicts contrasting effects of the omnivory link established by the top-predator/disease. On the one hand, it can destabilize positive equilibria in communities with linear functional responses (Holt and Polis, 1997) and even lead to chaos (Tanabe and Namba, 2005). On the other hand, omnivory tends to have a stabilizing effect on communities with saturating functional responses (McCann and Hastings, 1997). We have not observed any cases in which infection might possibly induce more complex dynamics like chaos, but we have not performed systematic simulations nor considered the case in which disease might possibly destabilize stable predator–prey dynamics.

The paradox of enrichment continues to puzzle both theoretical and experimental ecologists (e.g. Luckinbill, 1974; McCauley and Murdoch, 1990; Tilman and Wedin, 1991; Myerscough et al., 1996; Kirk, 1998; McCauley et al., 1999). Our findings suggest that

the additional predator mortality induced by infectious diseases has a stabilizing impact and counteracts the paradox of enrichment. This can be nicely explained in the phase plane projections, because the predator nullcline is shifted towards the stable region. However, it retains its constant vertical shape. Hence, enrichment will still destabilize the system.

Although disease introduction in our model does not 'reverse' the paradox of enrichment, it offers another potential explanation for why natural populations tend to be stable. Many species have a plethora of parasites and pathogens, making it possible that inherently cyclic behavior can be stabilized. In practice, however, it will be difficult to distinguish whether a particular system is stabilized due to disease or any other factor. Therefore, any metrics that allow to identify disease as the causing mechanism would be helpful.

Emerging and re-emerging infectious diseases and their invasion of resident species communities are pervasive. This study provides insightful ecological and epidemiological reproduction numbers for understanding how parasites structure community composition. All quantities are biologically plausible and could, in principle, also be measured from the field. Moreover, this study indicates that two very different outcomes are possible upon disease introduction: (1) the host population can either be driven to extinction or (2) an otherwise unstable resident community can be stabilized. Adding or removing parasites from food webs might therefore have unexpected and dramatic consequences, possibly leading to extinctions or outbreaks on more than one trophic level. This highlights the importance of including infectious disease agents in food webs, which has begun to be recognized only recently (Lafferty et al., 2008).

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Appendix A. Existence and stability of stationary states: linear stability analysis

System (5)–(7) has the following six equilibria $\boldsymbol{E}_j = (N_j^*, P_j^*, i_j^*)$, $j = 0, \ldots, 5$.

0. $\boldsymbol{E}_0 = (0,0,0)$. The trivial extinction state is always a saddle point. The eigenvalues are

$$\lambda_1 = r > 0,$$
$$\lambda_2 = -m < 0,$$
$$\lambda_3 = \beta - \mu.$$

1. $\boldsymbol{E}_1 = (1,0,0)$ with eigenvalues

$$\lambda_1 = \frac{1}{h+1} - m \lesseqgtr 0 \quad \text{if } R_0^P \lesseqgtr 1,$$
$$\lambda_2 = -r < 0,$$
$$\lambda_3 = \beta - \mu - \frac{1}{h+1} \lesseqgtr 0 \quad \text{if } \mathscr{R}_i \lesseqgtr 1.$$

The disease-free prey-only state is stable if the predators are too weak to establish ($R_0^P < 1$) and the disease does not have the potential to drive the predators to extinction ($\mathscr{R}_i < 1$). Otherwise $\boldsymbol{E}_1$ is a saddle point.

2. $\boldsymbol{E}_2 = (0,0,1)$. The trivial extinction state due to disease is always a saddle point with eigenvalues

$$\lambda_1 = r > 0,$$
$$\lambda_2 = -\mu - m < 0,$$
$$\lambda_3 = -(\beta - \mu).$$

3. $\boldsymbol{E}_3 = (1,0,i_3^*)$ with $i_3^* = 1 - 1/(\beta-\mu)(h+1) = (\mathscr{R}_i - 1)/\mathscr{R}_i$. The disease-induced extinction state exists only for $\mathscr{R}_i > 1$, in which case the disease-free prey-only state is unstable. The eigenvalues are

$$\lambda_1 = -r < 0,$$
$$\lambda_2 = \frac{1}{h+1} - m - \mu\left(\frac{\mathscr{R}_i - 1}{\mathscr{R}_i}\right) \lesseqgtr 0 \quad \text{if } R_i^P \lesseqgtr 1,$$
$$\lambda_3 = \frac{1}{h+1} - (\beta - \mu) \lesseqgtr 0 \quad \text{if } \mathscr{R}_i \gtreqless 1.$$

Hence, $\boldsymbol{E}_3$ is stable if $R_i^P < 1$ and a saddle point otherwise.

4. $\boldsymbol{E}_4 = (N_4^*, P_4^*, 0)$ with $N_4^* = mh/(1-m)$ and $P_4^* = rh(1 - m(h+1))/(1-m)^2 = rh/(1-m)^2\,(R_0^P - 1)/R_0^P$. The predator–prey coexistence state in the disease-free subsystem exists if $R_0^P > 1$. The eigenvalues are

$$\lambda_{1,2} = c_1 \pm \sqrt{c_2(-1)},$$
$$\lambda_3 = \beta - \mu - m \lesseqgtr 0 \quad \text{if } \mathscr{R}_0 \lesseqgtr 1.$$

$\mathscr{R}_0 < 1$ is a necessary condition for stability of $\boldsymbol{E}_4$. If $\mathscr{R}_0 < 1$ holds true, the disease vanishes and we have the same situation as in the predator–prey system: a pair of complex conjugate eigenvalues determined by parameter combinations c_1 and c_2 that depend solely on the ecological parameters r, h and m.

5. $\boldsymbol{E}_5 = (N_5^*, P_5^*, i_5^*)$ with

$$N_5^* = \frac{(\beta-\mu)(m+\mu)h}{\beta - (\beta-\mu)(m+\mu)},$$
$$P_5^* = rh\beta\frac{\beta - (\beta-\mu)(m+\mu)(h+1)}{[\beta-(\beta-\mu)(m+\mu)]^2} = \frac{rh\beta^2}{[\beta-(\beta-\mu)(m+\mu)]^2}\,\frac{R_i^P - 1}{R_i^P}$$

and

$$i_5^* = \frac{\beta - m - \mu}{\beta} = \frac{\mathscr{R}_0 - 1}{\mathscr{R}_0}.$$

The enzootic coexistence state exists if both $\mathscr{R}_0 > 1$ and $R_i^P > 1$. Its stability is investigated by means of continuation analysis and numerical simulations in Section 4.

Table 1 summarizes the existence and linear stability conditions of the stationary states. There is one special case that occurs only for the particular parameter combination $\beta = \mu$. In this case $\boldsymbol{E}_6 = (0,0,i_6^*)$ with $i_6^* \in (0,1)$ is a continuum of stationary states. These equilibria are unstable saddle points, i.e. their eigenvalues are $\lambda_1 = r > 0$, $\lambda_2 = -m - \mu i_6^* < 0$, $\lambda_3 = (\beta-\mu)(1 - 2i_6^*)$. As the parameter condition is unlikely to be met exactly in nature, this special case is not considered in the main text.

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