Ecological Complexity 18 (2014) 74–82

Contents lists available at SciVerse ScienceDirect

Ecological Complexity

journal homepage: www.elsevier.com/locate/ecocom

Original research article

Disease-induced modification of prey competition in eco-epidemiological models

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ARTICLE INFO

Article history:
Received 11 December 2012
Received in revised form 27 May 2013
Accepted 13 June 2013
Available online 22 July 2013

Keywords:
Differential host competition
Emergent carrying capacities
Bistability
Disease-induced extinction

ABSTRACT

Pathogens can change the strength of intraspecific competition experienced as well as exerted by their hosts. These modifications represent indirect effects of pathogens on host life-history traits and they have been largely overlooked—in both the theoretical and experimental literature. Here we consider an eco-epidemiological model that allows for differential competition amongst and between infected and uninfected prey individuals. We find that disease-induced modifications of competition can tremendously alter the stability and persistence of predator–prey systems. Specifically, differential prey competition can facilitate the coexistence of infected prey and predators, which is impossible if competitive abilities of healthy and diseased prey are equal. We also show that this scenario can be associated with bistability, in which case the populations coexist on the brink of disease-induced extinction. These results suggest that considering parasite-modified competition can be crucial in understanding the impact infectious diseases have on their host as well as on other species their host interacts with.

1. Introduction

Parasites can affect their host in many ways. The concept of virulence tries to encapsulate this in the damage a parasite inflicts upon its host, typically expressed as a reduction in host fitness due to infection (Read, 1994). Most mathematical models quantify virulence as an additional disease-related death rate or disease-induced reduction in fecundity (Day, 2002). However, there are many other direct and indirect effects of infection, which are also likely to affect parasite evolution (Alizon et al., 2009). For example, it is well-known that parasites alter their hosts with respect to resource requirements (Thompson et al., 2001), feeding rates (Rivero and Ferguson, 2003), behaviour (Poulin, 1995; Thomas et al., 1998; Lefèvre et al., 2009) including activity levels (Moore, 2002) and the response to various forms of stress (Brown and Pascoe, 1989; Renshaw et al., 1993; Bedhomme et al., 2005a).

There is some recent experimental evidence suggesting that parasitism also changes competitive abilities of hosts (Bedhomme et al., 2005). In general, however, little is known yet about these indirect effects of infection on host life-history traits. The literature is scarce and mostly focuses on parasitoids and macroparasites, both experimentally (Yan and Stevens, 1995; Bernstein et al., 2002; Lane and Mills, 2003; Sisterson and Averill, 2003; Koprivnikar et al., 2008) and theoretically (Bernstein, 1986; White et al., 2007), and also involves some plant hosts (Friess and Maillet, 1996; Damgaard and Jensen, 2002; Pagán et al., 2009).

Here, we investigate the consequences of differential (i.e. parasite-modified) competitiveness in a model that takes into account eco-epidemiological interactions. For this, we assume the host is subject not only to parasitism, but also to predation. This illustrates that the consequences of differential competitive abilities go beyond the host population itself, i.e. they profoundly impact other trophic levels as well. We find that it is crucial to identify how exactly infectious diseases alter the competitive strength of their host. In particular, we show that disease-modified intraspecific competition makes possible the coexistence of all species, which is impossible otherwise. However, we also show that the coexistence can be fragile (due to bistability) and dynamically complex (due to oscillations). These findings highlight the importance of extending the study of parasite-mediated modifications of host life-history traits beyond direct effects (e.g. on reproduction) to indirect effects (e.g. on competitive abilities).

2. The epidemiological skeleton

The basis of any eco-epidemiological model is the epidemiological skeleton, i.e. the submodel describing the infectious disease

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http://dx.doi.org/10.1016/j.ecocom.2013.06.002

dynamics. In this section, after briefly reviewing an epidemiological model with an explicit carrying capacity, we introduce a model that accounts for disease-modified intraspecific competition. This leads to emergent carrying capacities, and we will show that these are markedly different to explicit carrying capacities that have been employed in many eco-epidemiological models.

2.1. Explicit carrying capacity

A simple approach to modelling the spread of a disease in a host population and the interaction of susceptible (S) and infected (I) individuals is the following:

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S+I}{K}\right) - V(S,I), \tag{1}$$

$$\frac{dI}{dt} = r_I I\left(1 - \frac{S+I}{K}\right) + V(S,I) - \mu I, \tag{2}$$

Here, both subpopulations grow according to a logistic term with a common and *explicit carrying capacity* K. The underlying idea is that both susceptibles and infecteds contribute equally to the density-dependence resulting from resource competition. This model assumes that the disease is transmitted both vertically and horizontally—the latter with rate $V(S, I)$, which may be density-dependent or frequency-dependent or something inbetween (Begon et al., 2002). Parameters r_S and r_I are the intrinsic per-capita growth rates, i.e. when the total host population is rare. Disease-reduced fecundity is reflected by the constraint $r_I \leq r_S$. The parameter μ describes additional disease-induced mortality. This model has been used in several eco-epidemiological studies, but as we will see in the following, this formulation is limited in its applicability by the assumption of an explicit carrying capacity.

2.2. Emergent carrying capacity

A more general epidemiological skeleton is given by the equations

$$\frac{dS}{dt} = r_S S - (c_S S + c_{SI} I)S - V(S,I), \tag{3}$$

$$\frac{dI}{dt} = r_I I - (c_I I + c_{IS} S)I + V(S,I) - \mu I. \tag{4}$$

Here, the intrinsic per-capita growth rates of uninfected and infected hosts are given by r_S and r_I, respectively. They represent the net growth through density-independent reproduction and mortality. Intraspecific competition results in reduced population growth (due to increased mortality or decreased reproduction). Specifically, the parameters c_S and c_I characterise intra-class competition between susceptibles and infecteds, respectively, whereas the parameters c_{SI} and c_{IS} describe the inter-class impact of infecteds on susceptibles and the impact of susceptibles on infecteds, respectively.

Note that this model does not explicitly state a carrying capacity for the host population, as in the logistic growth formulation (1) and (2). Instead, the model is formulated in terms of intraspecific competition coefficients and we refer to this concept as *emergent carrying capacity*, following the notation of previous work on different variants of logistic growth (Bowers et al., 2003; Hoyle and Bowers, 2007). The emergent carrying capacity can be seen as an upper limit of population growth that arises from relevant processes like reproduction and competition. Rather than being a pre-determined number K, it is an emergent property based on actual life-history traits.

2.3. Explicit carrying capacity as a special case of emergent carrying capacity

Comparing the two formulations (1)–(2) and (3)–(4) it is clear that the explicit carrying capacity model can be obtained from the emergent carrying capacity model as the special case

$$c_S = c_{SI} = \frac{r_S}{K} \quad \text{and} \quad c_I = c_{IS} = \frac{r_I}{K}. \tag{5}$$

Here, two remarks are in order. Firstly, the competition coefficients are directly proportional to the intrinsic growth rates $r_{S,I}$. Hence, the intrinsic growth rates not only represent density-independent growth (as in the emergent carrying capacity model) but also determine the strength of density-dependent effects resulting from competition. This mingle–mangle is a popular fallacy of the logistic growth equation (Fulda, 1981; Kuno, 1991), which in particular may lead to confusion when it is applied in evolutionary models (Bowers et al., 2003; Hoyle and Bowers, 2007; Mallet, 2012).

Secondly and more importantly, there is a not so obvious assumption in the explicit carrying capacity model. The competitive pressure experienced by a susceptible individual is the same independently of whether it interacts with another susceptible or an infected individual ($c_S = c_{SI}$). The same holds true for the competitive pressure experienced by infected individuals ($c_I = c_{IS}$). That is, the coefficients describing the competitive pressure *received* by an individual are pairwise equal.

Moreover, the fact that the competition coefficients of the susceptible and infected equations differ is a result of the constraint $r_I < r_S$. If $r_I = r_S$, all four competition coefficients are equal. This may be a reasonable assumption if infection does not change competitive interactions at all, but it seems odd that the intrinsic growth rates are (the hidden) key in determining this.

The implicit assumption of pairwise equal competition coefficients is not only built in models using the explicit carrying capacity, but also shows up in other circumstances. Whenever models assume density-dependent birth or mortality rates, and this density-dependence is described by the total population, the competition coefficients within one equation are effectively equal. Such assumptions arise in many epidemiological models (e.g. Anderson et al., 1981; Gao and Hethcote, 1992; Zhou and Hethcote, 1994; Courchamp et al., 1995; Greenhalgh and Das, 1995; Barlow, 1996; Roberts, 1996; Lively, 2006; Hilker, 2009). The more general approach (3) and (4) takes into account that the infection status may change the competitive pressure exerted by susceptible and infected individuals, respectively.

3. The full eco-epidemiological model

3.1. Model description

We now proceed by putting a specialist predator P on top of the epidemiological skeleton (3) and (4) with emergent carrying capacities. The host population is thus not only a resource for the infection but also prey for the predator. We consider the following model of a general predator–prey community with an infection of the prey:

$$\frac{dS}{dt} = r_S S - (c_S S + c_{SI} I)S - \frac{\lambda SI}{S+I} - \frac{aSP}{h+(S+I)}, \tag{6}$$

$$\frac{dI}{dt} = r_I I - (c_I I + c_{IS} S)I + \frac{\lambda SI}{S+I} - \frac{aIP}{h+(S+I)} - \mu I, \tag{7}$$

$$\frac{dP}{dt} = \frac{\epsilon a(S+I)}{h+(S+I)}P - mP. \tag{8}$$

Here we assume that disease transmission is frequency-dependent with transmission coefficient λ, an assumption that is typically made for sexually transmitted diseases (Thrall et al., 1993) or diseases spreading in populations with social behaviour (Altizer et al., 2003). Consumption by predators is described by a saturating Holling type II functional response with maximal growth rate a, half-saturation constant h and resource conversion efficiency ϵ. Note that the predators are assumed not to distinguish between healthy and infected prey individuals. The parameter m describes the natural predator mortality rate. The S–P and I–P predator–prey subsystems, which are obtained for $I = 0$ or $S = 0$, respectively, are variants of the classical Rosenzweig–MacArthur model (1963).

Instead of using the healthy and infected subpopulations directly as dynamic variables, it is often convenient to work with the prevalence of the disease in the total population $N = S + I$. The prevalence is defined as the ratio $i = I/N$ of infecteds in the total population. Note that $S = (1 - i)N$ and $I = iN$. Rewriting the system in terms of the new variables removes the singularity at $S = I = 0$ and helps us to identify important parameter combinations:

$$\frac{dN}{dt} = \left[G(N,i) - \frac{aP}{h+N}\right]N, \tag{9}$$

$$\frac{di}{dt} = [r_i - (c_I - c_{SI})iN - (c_{IS} - c_S)(1-i)N](1-i)i, \tag{10}$$

$$\frac{dP}{dt} = \left[\frac{\epsilon aN}{h+N} - m\right]P. \tag{11}$$

We have defined two new expressions. Firstly,

$$G(N,i) = \underbrace{r_S(1-i) + (r_I - \mu)i}_{\text{density-independent growth of } S \text{ and } I} - [\underbrace{c_S(1-i)^2 + c_I i^2}_{\text{intra-class competition of } S \text{ and } I} + \underbrace{(c_{SI} + c_{IS})i(1-i)}_{\text{inter-class competition of } S \text{ and } I}]N$$

is the per-capita growth rate of the total population in the presence of disease but in the absence of predators. Secondly,

$$r_i = \lambda - \mu + r_I - r_S$$

is the net intrinsic spread rate of the disease prevalence in the population. This last quantity is not to be confused with the growth rate of infected individuals. It will appear several times during the following analysis and therefore deserves a closer look. One mechanism by which the disease can spread in the population is via horizontal transmission represented by the transmission coefficient λ, which thus appears as a positive contribution to r_i. On the other hand, the spread of the disease is adversely affected by an increased mortality of infected individuals as reflected by μ, because this decreases the time an infected individual can transmit the disease to susceptible hosts. The combined effect of vertical transmission and the growth of the susceptible portion as represented by the difference between the intrinsic growth rates r_I and r_S is a bit more subtle. Clearly, vertical transmission of the disease is beneficial for the disease and can increase prevalence, as reflected by the positive contribution of r_I. However, this effect is diminished (or reversed) by increased growth of the susceptible subpopulation, as this adds healthy individuals to the total population and thus leads to a decrease in disease prevalence.

It becomes apparent in formulation (9)–(11) that the spread of the disease in the prey population as described by the prevalence Eq. (10) is independent of the predator P. Conversely, the dynamics of the predators as given by Eq. (11) does not depend on the prevalence i. This is a direct consequence of the assumption that the predators do not or cannot discriminate between healthy and infected prey individuals—a scenario that has been investigated in a more general setting by Sieber and Hilker (2011).

A special case of this model with an explicit carrying capacity K has already been considered by Malchow et al. (2004) and Hilker and Malchow (2006). A peculiar feature of this special case is that stable stationary coexistence solutions do not exist. In the following, we will show that the more general formulation (6)–(8) makes coexistence equilibria possible and allows for complex bifurcation scenarios. Therefore, in the next section we will first investigate the existence and, where possible, the stability of the equilibria. Thereafter, we will take a closer look at some biologically plausible scenarios of intraspecific competitiveness.

3.2. Stationary solutions

To investigate the equilibria of Eqs. (9)–(11) we define a series of sets, on each one of which one of the three dynamical equations vanishes. These sets form surfaces in the three-dimensional state space and can be viewed as the analogous of the zero-isoclines (or *nullclines*) in two-dimensional systems. Consequently, we will refer to them as *nullsurfaces* from now on.

The rate of change dN/dt of the total population vanishes on the two nullsurfaces

$$\Gamma_N^0 = \{(0,i,P) \in \mathbf{R}^3_{\geq 0}\},$$
$$\Gamma_N = \left\{(N,i,P) \in \mathbf{R}^3_{\geq 0} | P = P^*(N,i) = G(N,i)\frac{h+N}{a}\right\},$$

where $\mathbf{R}_{\geq 0}$ denotes the non-negative real numbers. Similarly, from $di/dt = 0$ one obtains the surfaces

$$\Gamma_i^0 = \{(N,0,P) \in \mathbf{R}^3_{\geq 0}\},$$
$$\Gamma_i^1 = \{(N,1,P) \in \mathbf{R}^3_{\geq 0}\},$$
$$\Gamma_i = \left\{(N,i,P) \in \mathbf{R}^3_{\geq 0} | N = N^*(i) = \frac{r_i}{c_{IS} - c_S + i(c_S + c_I - c_{SI} - c_{IS})}\right\}, \tag{12}$$

and from $dP/dt = 0$

$$\Gamma_P^0 = \{(N,i,0) \in \mathbf{R}^3_{\geq 0}\},$$
$$\Gamma_P = \left\{(N,i,P) \in \mathbf{R}^3_{\geq 0} | N = N^* = \frac{mh}{\epsilon a - m}\right\}. \tag{13}$$

Nullsurfaces with a "0" superscript are referred to as trivial nullsurfaces and they correspond to the boundaries of the biologically relevant positive octant where one of the three species is absent. The functions $P^*(N,i)$, $N^*(i)$ and N^* will play a crucial role in the following, since they determine the shape of the non-trivial nullsurfaces. In particular, since N^* is constant, the surface Γ_P is always parallel to the Γ_N^0 surface.

Stationary solutions are obtained as the intersections of the nullsurfaces, and the shape and configuration of the nullsurfaces determine which equilibria actually exist. The trivial and semi-trivial equilibria and their biological meaning are summarised in Table 1. More details on existence and stability results can be found in the Supporting Information S1.

3.2.1. Non-trivial equilibria

The geometric intuition provided by the nullsurfaces is particulary useful to assess the existence of non-trivial coexistence equilibria. Non-trivial stationary solutions are given as the intersection $E_* = \Gamma_N \cap \Gamma_i \cap \Gamma_P$. The prey nullsurface Γ_N always intersects with both Γ_i and Γ_P. The existence of stationary solutions therefore depends on the relative positions of Γ_i and Γ_P. Since the predator nullsurface Γ_P is constant, it is mainly the

Table 1
Trivial and semi-trivial equilibria of system (9)–(11), see the Supporting Information S1 for more details.

Equilibrium	Biological meaning
E_0	Extinction
E_N	Prey only, disease-free
$E_\dagger$	Disease-induced extinction
E_{NP}	Predator–prey, disease-free
E_{IP}	Predator–prey, all prey infected
E_I	Prey only, all prey infected
E_{Nik}, $k=1, 2$	Prey only, disease endemic

prevalence nullsurface Γ_i that determines the existence of stationary solutions.

Here, we can distinguish three different cases, which are determined by the competition coefficients as follows:

$$c_{SI}+c_{IS}=c_S+c_I \quad \text{(equal inter- and intra-class competition)},$$
$$c_{SI}+c_{IS}>c_S+c_I \quad \text{(strong inter-class competition)},$$
$$c_{SI}+c_{IS}<c_S+c_I \quad \text{(weak inter-class competition)}.$$

If inter- and intra-class competition are equal, the existence of a unique coexistence equilibrium is impossible. This is because in this case the prevalence nullsurface Γ_i does not depend on i and is parallel to the predator nullsurface Γ_P (Fig. 1a). At this point we want to emphasize that, in the context of model (6)–(8), this outcome is typical for many common forms of transmission rates, including mass action (cf. Supporting Information S2).

The effect of disease-modified competition coefficients is to bend the nullsurface Γ_i of the disease prevalence and the direction of bending depends on the particular case of competition, since this determines whether $N_*(i)$ in the definition of Γ_i is increasing or decreasing with prevalence i. If inter-class competition is strong, the nullsurface bends towards the right-hand side (when looking from the disease-free N–P surface into the interior of the positive octant); cf. Fig. 1b. However, if inter-class competition is weak, the nullsurface bends towards the left-hand side; cf. Fig. 1c. In either case, this bending makes possible the existence of a unique coexistence steady state. Note that the prey density $N^*(i)$ at equilibrium increases (decreases) with increasing prevalence in the case of strong (weak) inter-class competition.

Let us consider the different cases in more detail.

1. The first case arises when the sum of the intra-class competition coefficients is equal to the sum of the inter-class competition coefficients, i.e. $c_S + c_I - c_{SI} - c_{IS} = 0$. Then the surface Γ_i does not depend on i and is parallel to the surface Γ_P. Note that this situation is the only possible scenario in the previously studied special case (5) of an explicit carrying capacity. The intersection of Γ_i and Γ_P is non-empty if and only if the surfaces coincide, which requires
$$\frac{mh}{\epsilon a-m}=\frac{r_i}{c_{IS}-c_S}.$$
In this case, E_* is a line of degenerate equilibria which can be described by the curve
$$\ell(i)=(N^*,i,P(N^*,i))$$
parametrised by the prevalence i. This case is structurally unstable and any slight perturbation of the system parameters will destroy this line of non-trivial equilibria (Hilker and Malchow, 2006).
2. On the other hand, the two cases satisfying $c_S + c_I - c_{SI} - c_{IS} \neq 0$ can give rise to the existence of a unique non-trivial stationary solution, which is given by
$$N^*=\frac{mh}{\epsilon a-m},$$
$$i^*=\frac{r_i-(c_{IS}-c_S)N^*}{(c_S+c_I-c_{SI}-c_{IS})N^*},$$
$$P^*=G(N^*,i^*)\frac{h+N^*}{a}.$$

Assessing the conditions that lead to either of these two equilibrium scenarios, it becomes clear that only the second scenario is biologically relevant. This is because the first case is non-generic in the context of the general model (9)–(11). For it to occur, two strict conditions have to be met by the system parameters, which is highly improbable in a real biological system. Stationary coexistence thus appears biologically unreasonable in this case and we will not consider it further. However, it should be noted that non-stationary coexistence is facilitated in the form of periodic (Hilker and Malchow, 2006) and chaotic attractors (Sieber and Hilker, 2011) over a considerable parameter range.

3.3. Bistable scenarios

In this section, we investigate the stability of the coexistence equilibrium. We will see that the emergence of this equilibrium is associated with the phenomenon of bistability. Two different forms of bistability are possible: between a diseased-induced extinction state and either (i) a disease-free state or (ii) a coexistence state. We will look at the stability conditions for each of these steady states individually.

Firstly, the most severe outcome of an epidemic is certainly the disease-induced extinction (D.I.E.) of the entire population. Such an extinction of the prey population leads inevitably to the subsequent extinction of the specialist predator P, thereby eradicating the entire predator–prey community. In the context of model (9)–(11), this state corresponds to the boundary equilibrium $E_\dagger$. Its local stability therefore determines whether disease-induced extinction poses a threat to the predator–prey community.

Linear stability analysis shows that $E_\dagger$ is locally stable whenever
$$r_I-\mu<0<r_i \tag{14}$$
is fulfilled. Recalling that r_i denotes the spread rate of the disease in the prey population while $r_I-\mu$ is the intrinsic growth rate of the infected subpopulation, the biological interpretation of this condition becomes clear. The positive spread rate means that, without any effects counteracting the advancing disease, a growing fraction of the total population becomes infected over time. The negative intrinsic growth rate means that the infected subpopulation is a sink for the population dynamics, e.g. more infected individuals are removed from the infected class by disease-induced mortality than are born into it. In combination, this implies that if the prevalence of the disease reaches some critical threshold, D.I.E. is the inevitable outcome. Note that this condition has also been derived by Hilker and Malchow (2006).

Secondly, the disease-free (i.e. $i=0$) predator–prey equilibrium E_{NP} is locally stable when it is stable in the well-known predator–prey subsystem and the additional condition
$$r_i<(c_{IS}-c_S)N^* \tag{15}$$
is fulfilled.

Hence, bistability between D.I.E. and the disease-free predator–prey equilibrium occurs when both stability conditions (14) and (15) hold simultaneously. This happens only if
$$c_S<c_{IS}. \tag{16}$$

Consequently, this rules out the possibility of bistability between a disease-free system and D.I.E. in the explicity carrying capacity scenario (5). With emergent carrying capacity, however, both the boundary equilibria $E_\dagger$ and E_{NP} can be stable at the same time.

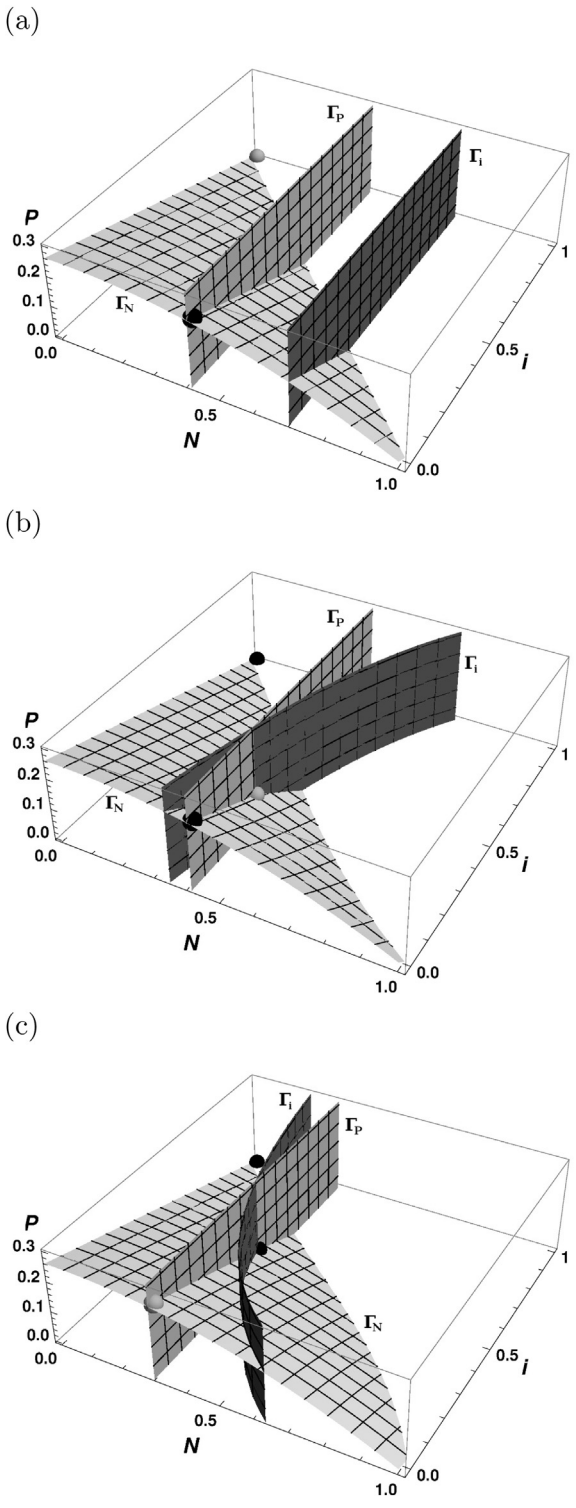

Fig. 1. Coexistence equilibria occur when all non-trivial nullsurfaces intersect. (a) In the case of equal inter- and intra-class competition, stationary coexistence is impossible because two of the nullsurfaces are parallel. (b) and (c) Differential competition coefficients bend the prevalence nullsurface (shown in dark) to the right (strong inter-class competition) or to the left (weak inter-class competition), thus facilitating unique coexistence equilibria.

The particular bifurcation scenario in the regime of bistability will also depend on the stability of the coexistence equilibrium E_* which in turn depends on the sign of the sum $c_S + c_I - c_{SI} - c_{IS}$. The two possible cases are discussed below and we assume that from now on the D.I.E. stability condition (14) is always fulfilled. For the time being, we also assume that the disease-free equilibrium E_{NP} is locally stable in the invariant predator–prey subsystem, ruling out oscillatory behaviour.

3.3.1. Strong inter-class competition

Firstly, we consider the case of strong inter-class competition, that is $c_S + c_I < c_{SI} + c_{IS}$. Assuming $c_S < c_{IS}$, cf. Eq. (16), we will use the predator mortality m as a bifurcation parameter. The corresponding bifurcation diagram is shown in Fig. 2a. There is a one-to-one correspondence between m and the prey density N^* prior to disease introduction, cf. Eq. (13). So increasing m corresponds to increasing N^* as well and thus shifting the planar predator nullsurface to the right-hand side.

If the prey density is too low, i.e.

$$N^* < \frac{r_i}{c_{IS} - c_S} = N^*(0),$$

the spreading disease will always lead to the extinction of the prey and subsequently of the predators, since according to the stability criteria (14) and (15) only the D.I.E. state $E_\dagger$ is stable.

At $N^* = N^*(0)$, the coexistence equilibrium E_* bifurcates from the disease-free equilibrium E_{NP} into the positive octant via a backward transcritical bifurcation. In this process, E_* becomes

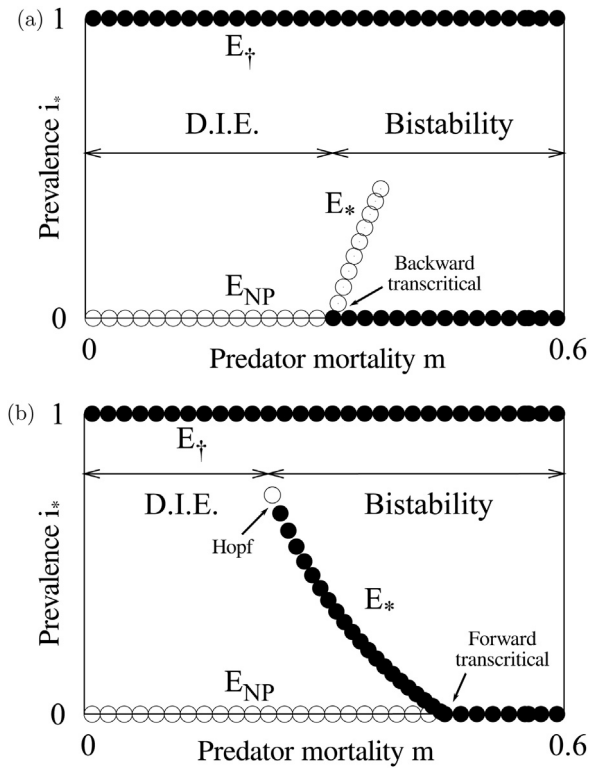

Fig. 2. Sketch of the bifurcation scenarios of the boundary equilibria $E_\dagger$, E_{NP} and the coexistence equilibrium E_* showing the regimes of disease-induced extinction (D.I.E.) and bistability. The predator mortality m as bifurcation parameter is a proxy for prey density at the disease-free equilibrium. (a) Backward transcritical bifurcation in the case of strong inter-class competition; (b) forward transcritical bifurcation in the case of weak intra-class competition. Empty (filled) circles denote unstable (stable) equilibria. In either case, the coexistence equilibrium disappears in transcritical bifurcations with the predator-free equilibrium E_{Ni1}. Parameter values: (a) $r_S = 1$, $r_I = 0$, $\epsilon = 0.3$, $a = 4$, $h = 1$, $\mu = 0.05$, $\lambda = 1.4$, $c_S = 1$, $c_I = 1$, $c_{SI} = 0.5$, $c_{IS} = 2$, (b) $c_{SI} = 0.25$, $c_{IS} = 1.25$, $\lambda = 1.2$.

unstable and E_{NP} stable, cf. Fig. 2a. This is where the regime of bistability begins. The outcome of an epidemic now crucially depends on the number of initially infected hosts: If too many hosts are infected in the early stages of an epidemic, disease-induced extinction is the ultimate outcome (Fig. 3a). By contrast, if only a few hosts are infected, the epidemic will abate after only a minor outbreak (Fig. 3b).

The bistability scenario has some interesting implications. All other things being equal, the parameter range of bistability increases with the size of the equilibrium prey population N^* prior to infection, since the right-hand side of inequality (15) increases with N^*. This means that highly effective predators which depress their prey to low densities put the whole community at a greater risk of being eradicated during an epidemic. For the predator this suggests a trade-off between effectiveness of predation and the very concrete risk of extinction by an infectious disease of the prey. This also implies that the risk of a collapsing predator–prey community is increased when the prey population is diminished by culling or removal of prey individuals as part of a containment strategy to control the epidemic.

It is also interesting to note that the scenario of strong inter-class competition implies that the parasite or pathogen responsible for the epidemic will always go extinct in the long-run, since the coexistence equilibrium is always unstable when it exists. Either it gets deprived of its host by disease-induced extinction, or the epidemic abates after a small outbreak, in which case the disease-free predator–prey community persists. As we will investigate now, this situation changes in the case of weak inter-class competition.

3.3.2. Weak inter-class competition

Now we assume $c_S + c_I > c_{SI} + c_{IS}$, i.e. weak inter-class competition. The bifurcation diagram is shown in Fig. 2b. Numerical simulations indicate that the coexistence equilibrium E_* is now typically stable, except for a very small parameter range right before the equilibrium leaves the positive octant. As a consequence, bistability is now possible between the coexistence equilibrium and the disease-induced extinction state. That is, the disease may be able to persist in the long-run (in contrast to the case of strong inter-class competition). The infection becomes endemic when the number of initially infected hosts is not too large, otherwise disease-induced extinction occurs (Fig. 3c and d). Our model thus predicts that a prerequisite for stable stationary coexistence of host, parasite and predators in a natural eco-epidemiological system is weak inter-class competition. And indeed the experimental system of Bedhomme et al. (2005) appears to be more of the weak inter-class competition type, although not far away from equal inter- and intra-class competition.

The coexistence equilibrium leaves the positive octant at $N^* = N^*(0)$ via a transcritical bifurcation with the prey only, disease endemic equilibrium E_{Ni1}. This is where the disease-free equilibrium E_{NP} becomes stable (Fig. 2b). In the region beyond this bifurcation point, the disease again has no chance of establishing itself in the long-run, the only possible outcomes being disease-induced extinction or the disease-free predator–prey state. Numerical bifurcation analysis also suggests that the coexistence equilibrium undergoes a Hopf bifurcation shortly before it leaves the positive octant (Fig. 2b). The associated population cycles lead to the non-stationary coexistence of all three species, but the parameter range of this case is extremely narrow (data not shown). However, the possibility of more robust non-stationary coexistence is explored in the next section.

It is also interesting to note that the bifurcation scenario shown in Fig. 2b implies that when the coexistence equilibrium exists, an increase in predator mortality will paradoxically always lead to increased predator densities. This is because the equilibrium E_* is stable for a large parameter range, and its

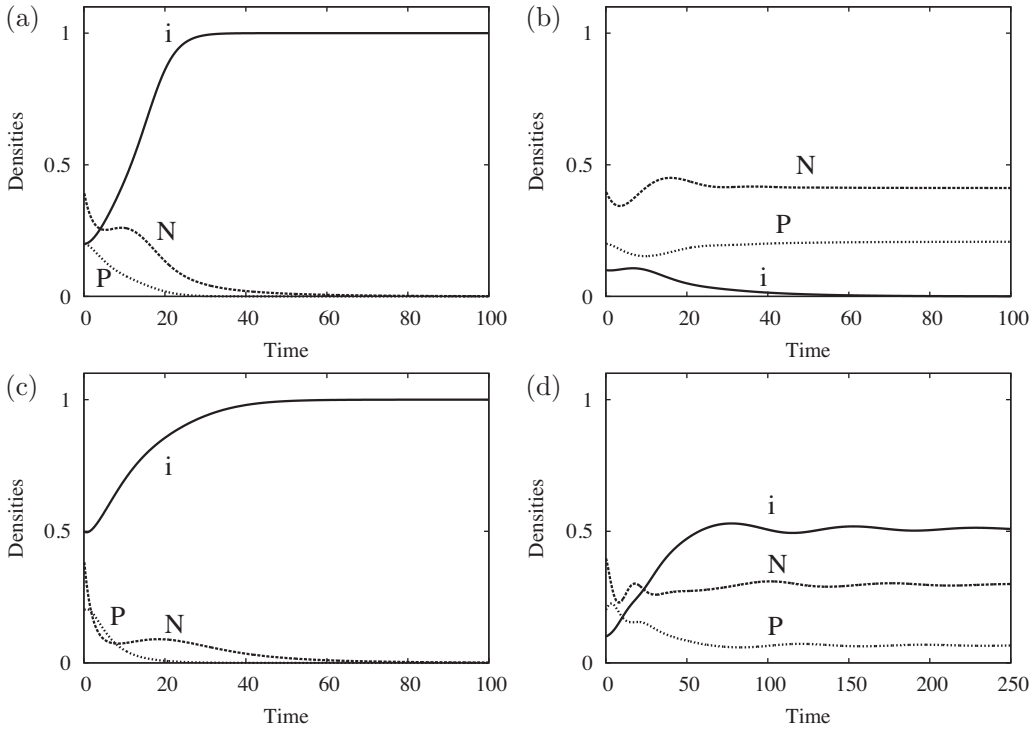

Fig. 3. Top row: Bistability between the disease-free predator–prey system and disease-induced extinction in the eco-epidemiological model with strong inter-class competition. (a) The initial condition with high prevalence ($i = 0.2$) drives the predator–prey community to extinction. (b) For a lower initial prevalence ($i = 0.1$) the disease is not able to spread, and prey and predators persist. Bottom row: Bistability between the coexistence equilibrium and disease-induced extinction in the eco-epidemiological model with weak inter-class competition. (c) A high initial prevalence ($i = 0.5$) drives the predator–prey community to extinction. (d) For a lower initial prevalence ($i = 0.1$), the coexistence equilibrium is approached. Parameters for (a,b) as in Fig. 2a with $m = 0.35$ and for (c,d) as in Fig. 2b with $m = 0.275$. Other initial conditions: $N = N^*$, $P = P_*$.

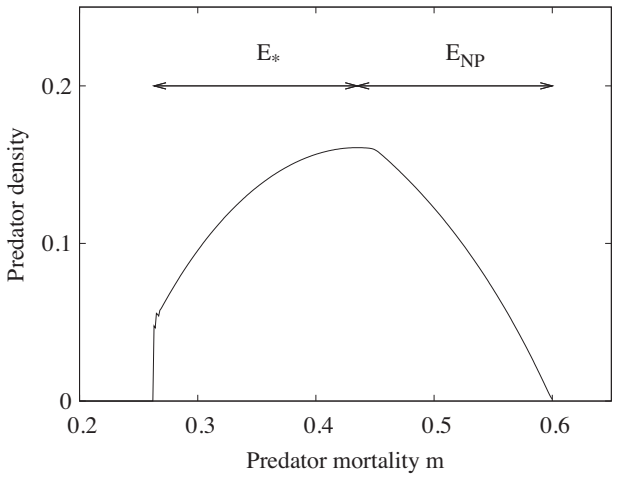

Fig. 4. Hydra effect: increasing predator mean density at the stable endemic equilibrium E_* with increasing predator mortality. After the transcritical bifurcation with the disease-free equilibrium E_{NP}, the predator mean density decreases again. Other parameters as in Fig. 3c and d.

predator component $P^*(N, i)$ is necessarily an increasing function of the predator mortality m—at least for intermediate mortality rates, since it enters the positive octant via a bifurcation from the equilibrium E_{Nik} with $P^*(N, i) = 0$ and it leaves the positive octant via the mentioned forward transcritical bifurcation with the disease free-equilibrium E_{NP} with $P^*(N, i) = P^*(N, 0) > 0$. The increase in predator density with increasing predator mortality is shown in Fig. 4. This phenomenon of a species mean density increasing with its own mortality has been termed hydra effect (Abrams, 2009). It has been shown for a wide range of classical predator–prey models that the condition for the hydra effect to occur is instability of the predator–prey equilibrium (Sieber and Hilker, 2012). Interestingly, the current results indicate that the presence of disease facilitates a hydra effect even when the predator–prey equilibrium is stable.

3.3.3. Bistability and non-stationary dynamics

Relaxing the assumption that the equilibrium E_{NP} is stable in the disease-free predator–prey subsystem tends to facilitate more complex bifurcation scenarios. In particular, the coexistence equilibrium can undergo a Hopf bifurcation leading to bistability between non-stationary coexistence of all three species and disease-induced extinction. This is illustrated in Fig. 5. Our numerical simulations show that the coexistence limit cycle may also undergo bifurcations to more complex behaviour such as chaos (not shown here).

4. Discussion and conclusions

We have argued that if competitive abilities differ due to infection, the explicit carrying capacity modelling approach does not seem appropriate. Instead, this calls for an emergent carrying capacity formulation, i.e. using differential competition coefficients for each of the four possible interactions amongst infected and susceptible individuals.

The explicit carrying capacity approach, however, has been used not only in epidemiological models (e.g. Gao and Hethcote, 1992; Courchamp et al., 1995; Barlow, 1996; Hilker, 2009) but also as the epidemiological skeleton in several eco-epidemiological models (Beltrami and Carroll, 1994; Chattopadhyay and Arino, 1999; Xiao and Chen, 2001; Hethcote et al., 2004; Singh et al., 2004; Malchow et al., 2004; Hilker and Malchow, 2006). We have therefore proposed and investigated a more general eco-epidemiological model that may be applied to biological systems with differential competitiveness in the infected prey population.

Emergent carrying capacities release the epidemiological skeleton from the rather restrictive constraints of the explicit carrying capacity model. In particular, the emergent carrying capacity model allows for the existence of a unique coexistence equilibrium, which is not possible in the explicit carrying capacity model with indiscriminate predation. The observation that the model with emergent carrying capacity is able to show more biologically relevant dynamics than the explicit carrying capacity model is in line with theoretical studies of evolutionary branching (speciation) in predator–prey models (Bowers et al., 2003; Hoyle and Bowers, 2007).

The possible occurrence of a unique coexistence equilibrium and the bistability scenario are of considerable theoretical and practical interest. While it is known from previous eco-epidemiological models with explicit carrying capacities that coexistence is possible when predators discriminate between prey types, and for example only feed on infected individuals (Chattopadhyay and Bairagi, 2001), models with indiscriminate predators have so far led to the conclusion that the dynamics are of an "all or nothing" type. Either the disease succesfully spreads

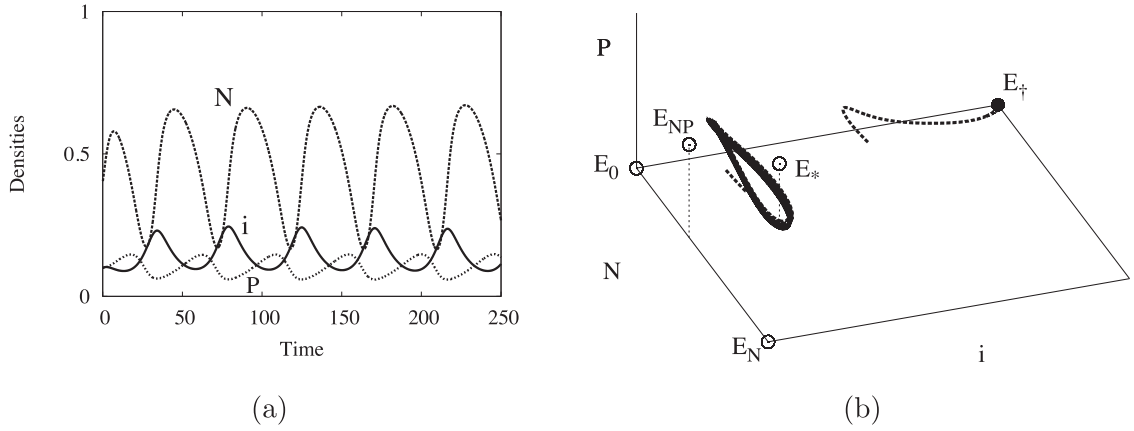

Fig. 5. (a) Coexistence cycle approached for the initial condition $N = N^*$, $i = 0.1$, $P = P_*$. For initial conditions with higher initial prevalence disease-induced extinction occurs. (b) Phase space with equilibria marked by circles and the orbits of two solutions approaching the disease-induced extinction state and the coexistence limit cycle. Filled circles denote a stable equilibrium and empty circles denote an unstable equilibrium. Parameters: $r_S = 1$, $r_I = 0$, $c_S = 1$, $c_I = 1$, $c_{SI} = 0.25$, $c_{IS} = 1.25$, $\epsilon = 0.3$, $a = 2$, $h = 0.1$, $m = 0.48$, $\lambda = 1.2$, $\mu = 0.05$.

displacing the predators and possibly leading to the collapse of the whole predator–prey community, which is a clearly Pyrrhic victory for the disease, or the epidemic abates with a subsequent return to the disease-free state. Only under certain conditions, namely when the predators are not too dominant and in addition the predator–prey system is cyclic, *non-stationary* coexistence is possible in the explicit carrying capcity model (Hilker and Malchow, 2006; Sieber and Hilker, 2011). Otherwise there is nothing between a disease-dominated or predator-dominated state, i.e. a competitive exclusion principle holds and "the winner takes it all" (Siekmann et al., 2010).

The emergent carrying capacity model, however, paints a more differentiated picture, with intermediate outcomes becoming possible, namely in the form of *stationary* (as well as non-stationary) coexistence of all three populations. This can be associated with bistability, i.e. a critical dependence on initial conditions and/or external perturbations. This latter case has profound implications for the control and management of infectious diseases, since any overcritical inflow of additional infected individuals may push the system from the healthy or endemic state on a trajectory to disease-induced extinction.

Even more dramatically, well-meant control measures such as host culling or other forms of removal of prey individuals may lead to this catastrophic crash of the system. This has been pointed out in the section on strong inter-class competition, but it holds equally for weak inter-class competition. Hence, the proposed general eco-epidemiological model (6)–(8) suggests that healthy predator–prey ecosystems or those in which a non-critical endemic disease is present may actually exist on the brink of disease-induced extinction, even though the dynamics are robust with respect to small perturbations.

There is yet another implication for the design of wildlife disease control programs. It has been hypothesised that predator removal results in more infections of the prey ('keeping the herds healthy' Packer et al., 2003). In contrast to this, our results in the case of weak inter-class competition suggest that predator removal (i.e. increasing their mortality m) can simultaneously increase predator density and decrease infection levels at the coexistence equilibrium (depending on initial conditions, cf. Figs. 2 and 4). Hence, predator removal can be doubly beneficial. Our results thus contradict the 'keeping the herds healthy' hypothesis and are in line with recent similar conclusions (e.g. Morozov and Adamson, 2011; Bate and Hilker, 2013).

In summary, the results presented here appear to challenge previous studies based on more restrictive assumptions that may hold only for particular biological systems. This paper highlights the need of an adequate description of the competition between healthy and infected subpopulations. As such basic interactions form part of any epidemiological skeleton that is embedded in a wider ecosystem context, modelling intraspecific competition may be crucial for understanding the dynamics of spreading diseases in food webs.

Acknowledgements

M.S. acknowledges support by funding from the BBSRC. H.M. acknowledges the funding of a visiting professorship by CAPES and DAAD. F.M.H. acknowledges partial support from the EU-ICT project Epiwork.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ecocom.2013.06.002.

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