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# Revealing the role of predator-dependent disease transmission in the epidemiology of a wildlife infection: a model study

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**Abstract** It is well known that predation/harvesting on a species subjected to an infectious disease can affect both the infection prevalence and the population dynamics. In this paper, I model predator–prey–pathogen interactions in the case where the presence of a predator indirectly affects the transmission rate of the infection in its prey. I call this phenomenon the predator-dependent disease transmission. Such a scenario can arise, for example, as a consequence of anti-predator defence behaviour, debilitating the immune system of the prey. Although being well documented, the predator-dependent disease transmission has rarely been taken into account in ecoepidemiological models. Mathematically, I consider a classical S-I-P ecoepidemiological model in which the infected and/or the healthy host can be consumed by a predator where the coefficient in the mass action transmission term is predator-dependent. Investigation of the model shows that including such a predator-dependent disease transmission can have important consequences for shaping predator–prey–pathogen interactions. In particular, this can enhance the survival of the predator, restricted in a system with a predator-independent disease transmission. I demonstrate the emergence of a disease-mediated strong Allee effect for the predator population. I also show that in the system with predator-dependent disease transmission, the predator can indirectly promote epidemics of highly virulent infectious diseases, which would die out in a predator-free system. Finally, I argue that taking into account predator-dependent disease transmission can have a destabilizing effect in a eutrophic environment, which can potentially cause the extinction of both species. I also show that including the predator-dependent disease transmission may increase the infection prevalence, and this fact will question the 'keeping herds healthy' hypothesis concerning the management of wildlife infections by natural predators.

**Keywords** Ecoepidemiology · Transmission rate · Predator–prey model · Paradox of enrichment · Anti-predator defence behaviour

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## Introduction

Population dynamics of a species subjected to an infectious disease are often affected by trophic pressure exerted by the predators as well as by harvesting. Various programmes for the management of disease in natural populations take this fact into account by suggesting the control of the infection through predation (Hudson et al. 1998; Stiling and Moon 2005; Choisy and Rohani 2006; Hawlena et al. 2010; Greenman and Hoyle 2010). The use of natural predators for disease control can be tricky. Indeed, it has been theoretically shown that predation on infected populations can both increase and decrease the infection prevalence of the disease (Packer et al. 2003; Holt and Roy 2007; Roy and Holt 2008). On the other hand, it has been recently demonstrated that on larger timescales, predation can affect the evolutionary course of pathogen virulence of its prey and consequently result in a selection of highly viral strains (Choo et al. 2003; Morozov and Adamson 2011).

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Interestingly, even on a short timescale, the presence of predators can indirectly influence the transmission rate and the virulence of the disease, thus affecting management strategies. Indeed, the presence of predators often modifies the behaviour as well as the habitat use of prey individuals. As a result, the prey population can become more vulnerable to a disease/pathogen in the presence of predators, and so the transmission rate of disease becomes a function of the total amount of the predator. Let us assume, for the sake of simplicity, that the incidence rate (i.e. the number of infected individuals per unit of time) can be described via a classical mass action relation, $\lambda IS$, in an S-I model (Anderson and May 1979), where $S$ is the number of susceptible and $I$ is the number of infected individuals in the prey population. In this case, the transmission coefficient $\lambda$ can be a function of the amount of predator $P$ in the ecosystem, i.e. $\lambda(P)$. I call this phenomenon predator-dependent disease transmission, and, as is further shown, this can play a crucial role in shaping predator–prey–disease interactions.

Various mechanisms can allow for the predator-dependent disease transmission. For instance, this can be a consequence of an anti-predator behaviour (such as the inducible defence; Tierney et al. 1993; Rigby and Jokela 2000; Matz and Kjelleberg 2005; Kortet et al. 2007; Friman et al. 2009). For example, in the presence of predators, a freshwater snail spends a long time hiding inside its shell. This makes it more vulnerable to parasites since the organism cannot expel the blood which is necessary for proper immune system functioning (Rigby and Jokela 2000). Another example is the extra exposure to infection by cercaria of tadpoles in the presence of predators; an increase in the susceptibility of the tadpoles to a trematode infection becomes possible since tadpoles usually reduce their activity in the presence of predatory fish (Thiemann and Wassersug 2000). Predator-dependent disease transmission is also observed in a common zooplankton species *Daphnia* (Yin et al. 2011) in the presence of fish, but an understanding of the exact mechanisms of this phenomenon has not yet been reached. One possible explanation is that in the presence of predators, the contact rate of *Daphnia* larva with parasites increases (Baker and Smith 1997). Also, it has been reported that *Daphnia* usually descends in the water column to avoid predation by fish, and this may increase the chance of becoming infected by parasitic spores from the sediment (Decaestecker et al. 2002). More generally, some zooplankton species contain reserves of carotenoids which are necessary to build up their immune system, and it has been shown that in the presence of predators in the ecosystem, zooplankters reduce the amount of carotenoids in their body to be less visible for predation, making them more vulnerable to infection (van der Veen 2005). Another widespread mechanism causing an increase in the transmission rate is a result of the grouping of prey in the presence of predators through the formation of fish schools, avian flocks, herds of herbivores, etc. (e.g. Edgerly 1994). A dense 'packing' of individuals in groups would signify a larger transmission rate due to the increased number of contacts (Alexander 1974; Freeland 1976; Cote and Poulin 1995; Krasnov et al. 2002; Altizer et al. 2003).

Although being empirically well documented (a large number of other references can be easily provided), the effects of predator-dependent disease transmission on the dynamics of predator–prey–disease interactions have been largely overlooked in the modelling literature so far. This paper is aimed to bridge the existing gap. Mathematically, I consider a classical predator–prey model in which the prey population is subjected to an infectious disease. The predator can consume both infected and healthy prey (without becoming infected itself); however, the attack rates on infected and healthy prey are different. The disease transmission coefficient is considered to be a function of the total amount of predators/predator density. Both possible scenarios are investigated where the presence of predators enhances and impedes the transmission of the disease (some ecological scenarios including a reduction of disease transmission in the presence of a predator are discussed in "Discussion").

Modelling results show that taking into account predator dependence in the transmission rate can have important consequences for the dynamics of predator–prey–disease interactions. In particular, a predator-dependent disease transmission can allow the predator to establish in the system, which can be otherwise impossible. Furthermore, I show the emergence of a disease-mediated strong Allee effect where the survival of a supercritical population number of predators becomes possible by maintaining a high transmission rate of disease among its prey, whereas a small number of predators will always go extinct. This effect occurs in both cases of enhancing and reducing the transmission in the presence of the predator. I find that enhancing of disease transmission by predators can promote a highly virulent infectious disease, which would die out in the absence of predators. Taking into account predator-dependent disease transmission (in the case of enhancement of transmission) would have a destabilizing effect on predator–prey interactions in a eutrophic environment, which can potentially cause the extinction of both species. Finally, I shall discuss the potential effects of predator-dependent disease transmission on the infection prevalence. In particular, I show that the infection prevalence may increase in the presence of a predator, thus questioning the 'keeping herds healthy' hypothesis.

## Modelling framework

The general model consists of three differential equations for the density of the healthy prey $S$, the infected prey $I$ and

the predator $P$. I assume that the predator is able to consume both the healthy and the infected prey; however, the parameters describing the consumption of $S$ and $I$ (e.g. the attack rates) can be different. I also consider that the predator is a specialist, i.e. the prey population constitutes its only food source. Note that such a modelling approach is rather standard in the literature, combining a S-I model and a Rosenzweig–MacArthur predator–prey model (e.g. Chattopadhyay and Bairagi 2001; Greenman and Hoyle 2010; Rhodes and Martin 2010).

$$\frac{\mathrm{d}S}{\mathrm{d}t} = rS\left(1-\frac{S+I}{K}\right) - \lambda(P)SI - f_{\mathrm{s}}(S,I)P \tag{1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \lambda(P)SI - DI - f_{\mathrm{I}}(S,I)P \tag{2}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = (\omega_s f_{\mathrm{s}}(S,I) + \omega_{\mathrm{I}} f_{\mathrm{I}}(S,I) - \delta)P \tag{3}$$

where $K$ is the carrying capacity of the prey population. I assume that only healthy individuals can reproduce; however, the infected subpopulation still influences overall prey growth by competing for resources and thus contributing to the carrying capacity; $r$ is the maximum per capita growth rate of the healthy prey.

The consumption of healthy and infected prey by the predator is described by the terms $f_{\mathrm{S}}$ $(S, I)P$ and $f_{\mathrm{I}}$ $(S, I)P$, respectively, where $f_{\mathrm{S,I}}$ are the corresponding predator functional responses. When consuming infected prey, the predator is assumed not to succumb to the infection. In this paper, I shall investigate the case where the predator functional response is of Holling type I (linear response) and that the consumption of each subpopulation of prey (i.e. $S$ or $I$) occurs independently of the other subpopulation (no explicit prey selectivity). This can be described by

$$f_{\mathrm{S}}(I,S) = bS, \tag{4}$$

$$f_{\mathrm{I}}(I,S) = aI. \tag{5}$$

I shall briefly discuss in "Discussion" the influence of saturation in the predator functional response. The other parameters describing the predator dynamics are the trophic efficiency coefficients $\omega_{\mathrm{S}}$ and $\omega_{\mathrm{I}}$ and the natural mortality $\delta$. For the sake of simplicity, we assume that trophic efficiency coefficients are the same $\omega_S = \omega_I = \omega$.

The transmission of the disease is parameterized via the classical mass action term $\lambda SI$ (Anderson and May 1979; McCallum et al. 2001). The mass action scenario can be observed in the case where the number of contacts between individuals is not fixed but is proportional to the host density. Another possible mechanism arises when the infection is caused by parasites which have a free-living stage and are released by infected host individuals. In the case of a fast equilibrium for the density of the free-living stage, this density will be proportional to that of the infected host. This can be seen from a simple example given by the following differential equation for the density of the free-living stage $V$: $\mathrm{d}V/\mathrm{d}t = \beta I - \gamma V$, where the production of free-living parasites is proportional to the number of infected hosts $I$ and the mortality $\gamma$ of $V$ is suggested to be constant (e.g. Rhodes and Martin 2010). Suggesting a fast equilibrium for $V$, we have each time $V \approx \beta I/\gamma$. Since the incidence rate is proportional both to the density of the free-living parasites $V$ and to the number of healthy hosts $S$, the classical mass action arises.

The coefficient of proportionality in the mass action term $\lambda$ is called the transmission coefficient, and in this paper, I assume that it depends on the amount of predators, $P$, in the system. Various mechanisms can allow for the dependence $\lambda(P)$, and different parameterisations will result in different model outcomes. To proceed further, we need to explicitly specify the functional dependence $\lambda(P)$ since without doing so, even the question about the number of the system's equilibria cannot be fully addressed. In most of this paper, I shall consider the simplest relation between $\lambda$ and $P$, i.e. the linear dependence which can be considered as the first term in the Taylor expansion of $\lambda(P)$ about the predator-free state $P=0$:

$$\lambda = \lambda_0 + \alpha P \tag{6}$$

where $\lambda_0$ is the transmission rate in the absence of predators; $\alpha$ describes the effect of the presence of predator. I shall consider both scenarios where the presence of predator results in (1) an increase of transmission rate of the disease ($\alpha > 0$) and (2) a decrease of transmission rate ($\alpha < 0$). Note that in the case of $\alpha < 0$, it is necessary to require that the transmission rate (Eq. 6) always be nonnegative, i.e. $\lambda$ is given by 6 for $\lambda_0 + \alpha P > 0$; it is zero otherwise.

Based on standard approaches (e.g. Mukherjee 1998; Roy and Chattopadhyay 2005), one can easily prove the boundedness as well as the positive invariance of solutions of the general model (Eqs. 1–3). To better understand the model properties, we shall first separately investigate two limiting cases where the predator only consumes infected prey ("Predator only consumes infected prey") and where the predator only feeds on healthy prey ("Predator only consumes healthy prey"). Finally, in "Predator consumes both healthy and infected prey", the general case will be addressed where the predator can consume both the infected and the healthy prey.

## Modelling results

### Predator only consumes infected prey

In this subsection, we shall consider the particular case of Eqs. 1–3 where the predator consumes infected prey only. Note that this scenario is frequently observed in field and laboratory observations (Kabata 1985; Hudson et al. 1992, 1998; Friend 2002; Johnson et al. 2006; Duffy and Sivars-Becker 2007; Liao et al. 2008). The resultant equations are obtained from the general model by setting $f_S(I,S) \equiv 0$ or $b=0$ in Eq. 4. To better understand the effects of predator-dependent disease transmission on the system dynamics, we shall briefly discuss the case $\alpha=0$, i.e. the presence of the predator has no effect on $\lambda$. Investigation of the system with $\alpha=0$ shows that the unique interior equilibrium is always stable wherever it exists (see Appendix 1). The increase of $\lambda_0$ always results in a decrease in the stationary density $S^*$ of healthy prey; however, the dependence of $P^*$ on the transmission rate is non-monotonic, having a maximum for an intermediate $\lambda_0$. The stationary density of infected prey for $P^*>0$ is always constant: $I^*=\delta/(\omega a)$. As in the classical Rosenzweig–MacArthur predator–prey model with a linear functional response, an increase in the carrying capacity $K$ does not affect the system stability (see Appendix 1).

Now we can take into account the effects of predator dependence of the transmission rate $\lambda$. The system's stationary states for $\alpha\neq0$ are computed in Appendix 1. The system always has a disease-free and predator-free state ($K$,0,0). Establishment of infection in the system becomes possible for $D<K\lambda_0$. It can be easily shown that in this case, the basic reproduction ratio/number of the disease becomes larger than 1. Overall, this ratio is defined as the number of secondary cases caused by an infected individual just after disease introduction in the absence of immunity (Diekmann et al. 1990), which in the current model is given by $R_0=\lambda(P)S/(D+aP)$, with $P$ and $S$ being the stationary densities of species. For the disease-free and predator-free state, we have the basic reproduction ratio given by $R_0=\lambda_0 K/D$. One can see that the condition for establishing the infection in the system is the same as for $\alpha=0$, i.e. in the absence of a predator-dependent disease transmission. Depending on the model parameters, the system can have a predator-free stationary state ($S^1$,$I^1$,0) and there can be up to two interior states ($S^*$,$I^*$,$P^*$), with all species densities being positive. The coordinates of the stationary states as well as the local stability analysis can be found in Appendix 1. It is convenient to use $\alpha$ and $\lambda_0$ as bifurcation parameters and consider parametric portraits in the $\alpha$–$\lambda_0$ plane. The structure of these $\alpha$–$\lambda_0$ portraits largely depends on the value of the carrying capacity $K$.

Figure 1 shows the bifurcation portrait of the system for a small carrying capacity ($K=3$) as well as the equilibrium densities $S^*$ and $P^*$as functions of the parameter $\alpha_0$ for a fixed $\lambda_0$. The bifurcation diagram is constructed based on the analytical results in Appendix 1 as well as an extensive numerical simulation. There are five possible parametric domains corresponding to different dynamical regimes (see Fig. 1a). In domain 1, the unique interior equilibrium ($S^*$, $I^*$,$P^*$) is stable. Numerical simulation shows that all trajectories will be finally attracted by this state, i.e. it is globally stable. Domain 2 is characterized by the existence of two interior stationary states, one of which is a stable node/focus, the other one being a saddle point. The non-saddle state is stable; however, the system has another attractor—a predator-free equilibrium ($S^1$,$I^1$,0). Thus, depending on initial conditions, the system will evolve either to the interior equilibrium ($S^*$,$I^*$,$P^*$) or to the predator-free state. The above bistability remains in domain 3 (large $\alpha$ and small $\lambda_0$), where the two attractors are: the stationary state ($S^*$,$I^*$,$P^*$) and the disease-free and predator-free state ($K$,0,0). In this case, one can interpret the results in terms of the basic reproduction number. Indeed, the initial value of $R_0=\lambda_0 K/D<1$ in the predator-free system can become larger than 1 in the case of introduction of a sufficiently large (supercritical) amount of predator, resulting to a further epidemics. In the other domains, 5 and 4, the only possible attractors are the predator-free state ($S^1$,$I^1$,0) and the state ($K$,0,0), respectively. Those points are globally stable.

Figure 1b, c shows variation in the equilibrium densities $S^*$ and $P^*$ as functions of $\alpha$ for a fixed value of $\lambda_0$ (the value of $I^*$ is always constant, $I^*=\delta/(\omega a)$). Figure 1b shows variation in the species densities when $\lambda_0$ is small and the predator cannot establish in the system until its influence on the transmission rate becomes large enough (implying a supercritical $\alpha$). For the value of $\alpha$, corresponding to a limit point (LP), two stationary states emerge via a saddle–node bifurcation, one of them being a saddle point (not shown in the figure). Note that in this case, the system is bistable—the predator-free state ($S^1$,$I^1$,0) is always stable for the given $\lambda_0$. The stationary densities of this state ($S^1=D/\lambda$, $P^1=0$) are also indicated in Fig. 1b. Figure 1c represents the situation for a larger $\lambda_0$, where the predator is able to establish in the system without affecting the transmission rate (i.e. for $\alpha=0$). In this case, the dependence of $P^*$ on $\alpha$ is non-monotone, having a maximum for an intermediate $\alpha$; $S^*$ monotonically decreases with $\alpha$. Note that domain 1 is not bounded for $\alpha<0$, which always implies a positive $\lambda(\alpha,P^*)>0$. This follows from the fact that for $\lambda(\alpha,P^*)<0$, the derivative $dI/dt$ would be negative and no interior stationary state would exist in this case.

Interesting preliminary conclusions can be made based on Fig. 1. A positive predator dependence of the transmission rate (a supercritical $\alpha>0$) can guarantee the survival of the predator, which is otherwise impossible for small $\lambda_0$ and $\alpha=0$. Another important feature is the bistability in the

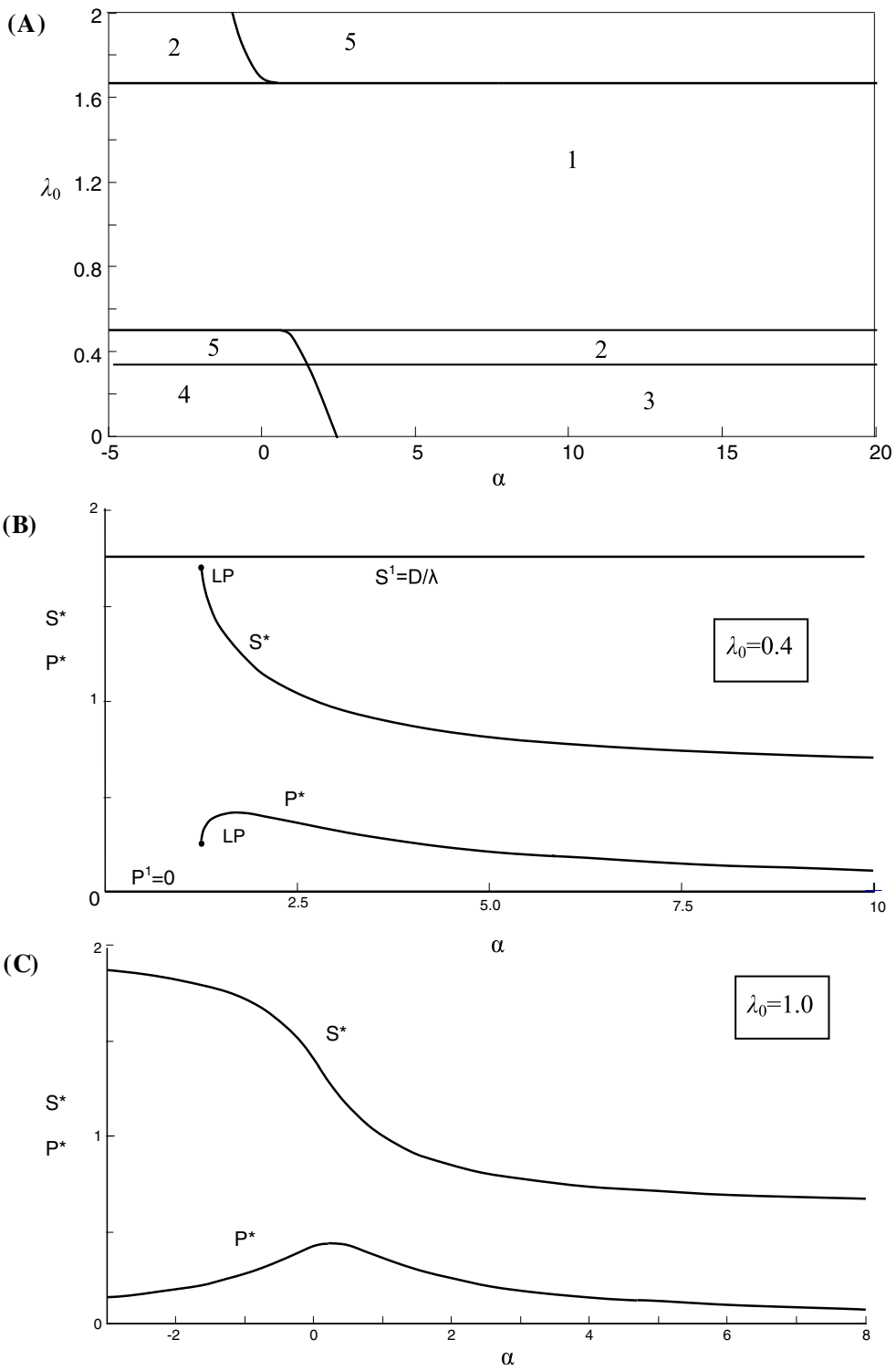


**Fig. 1** **a** Bifurcation diagram in the $\alpha$–$\lambda_0$ plane in the case where the predator only consumes infected prey. The meaning of the domains and the corresponding regimes is summarized in Table 1. The carrying capacity of the system is small ($K$=3). LP denotes the limit point (a saddle–node bifurcation). The other parameters are: $r$=1; $\delta$=0.1; $D$=1; $a$=1; $\omega$=0.25. **b**, **c** The stable stationary densities $S^*$, $P^*$ as functions of $\alpha$ plotted for $\lambda_0$=0.4 and $\lambda_0$=1.0, respectively. In the case of bistability (**b**), the other stable state is shown with densities $S^1$ and $P^1$

system with a predator-dependent transmission rate (domains 2 and 3). This signifies that a small initial predator population cannot establish in the system, whereas it becomes possible for a population with a large size. This phenomenon is analogous to the strong Allee effect in ecology, and I shall call it a disease-mediated strong Allee effect. Note that the bistability in the system can also be observed for $\alpha<0$ (domain 2). This occurs for large $\lambda_0$, for which the establishment of a predator is not possible with a predator-independent transmission rate ($\alpha$=0).

Figure 2 represents the bifurcation portrait for a large carrying capacity ($K$=20). Along with domains 1–5 (which

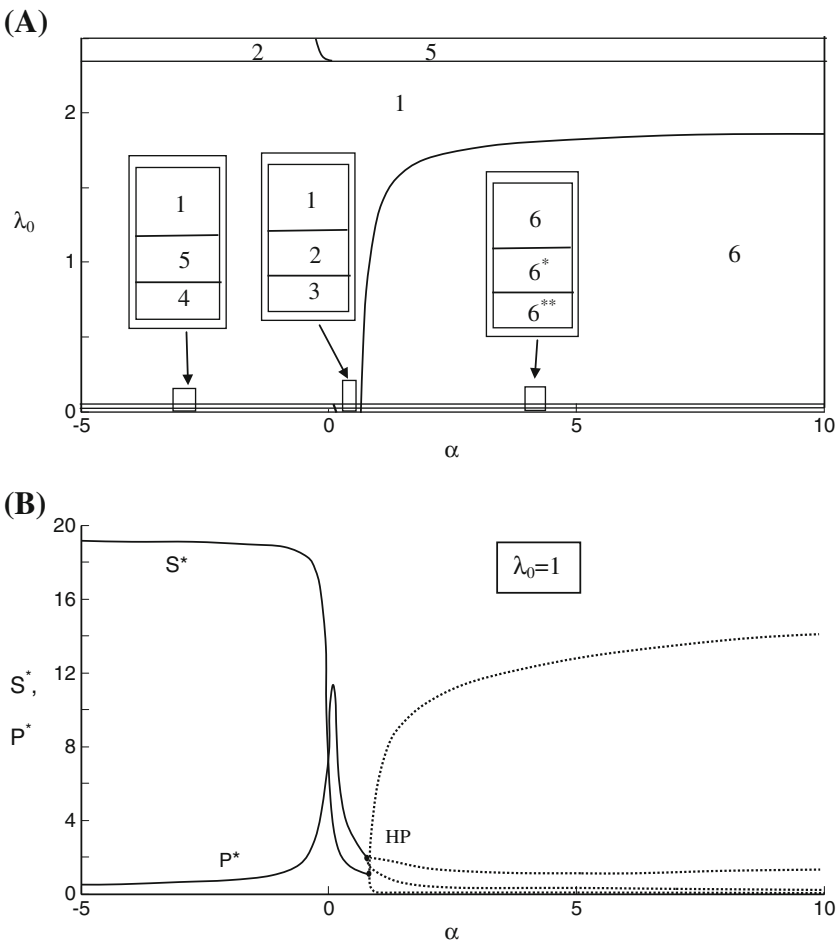


**Fig. 2** **a** Bifurcation diagram in the $\alpha$–$\lambda_0$ plane in the case where the predator only consumes the infected prey. The carrying capacity of the system is large ($K$=20). The meaning of the domains and the corresponding regimes is summarized in Table 1. **b** Stable stationary densities $S^*$ and $P^*$ (*solid lines*) and the amplitudes of oscillations (*dotted lines*) as functions of $\alpha$ ($\lambda_0$=1.0). *HP* Hopf bifurcation point. The other parameters are the same as in Fig. 1

have the same meaning as in Fig. 1), there appear domains 6, 6* and 6**. Those domains are characterized by oscillatory dynamics of species (the interior stationary state loses its stability via a Hopf bifurcation). In domain 6, the only system attractor is the limit cycle. Domains 6* and 6** are modifications of 6; they are characterized by the coexistence of two attractors: a stable limit cycle and the stable predator-free stationary state, respectively. An increase in $K$ results in the shrinking of all other domains, except the domain of oscillations 6. The most important observation is that the effects of predator dependence on the transmission rate would lead to a destabilization for large carrying capacities, which does not occur for $\alpha$=0. Figure 3b shows the species equilibrium densities (solid lines) as well as the maximal and minimal values along the limit cycle with an increase of $\alpha$ (dotted lines). One can see that the amplitude of oscillations of prey density increases with $\alpha$, with the minimal values approaching zero. This can potentially cause the extinction of species as in the classical paradox of enrichment in predator–prey models (cf. Rosenzweig 1971; McCauley et al. 1999).

For the sake of convenience, the above patterns of dynamics corresponding to different bifurcation domains in Figs. 1 and 2 are summarized in Table 1 (domains 1–6**). Table 1 holds both for the current section (i.e. for $b$=0) and for the other cases with $b$>0 (two next sections). Note also that along with the mentioned regimes, the model can show some other patterns of dynamics; in particular, there is a possibility to have chaotic dynamics for large $K$. The transition to chaos in the system was found to take place via the classical period-doubling scenario. For the sake of brevity, I do not show those results.

## Predator only consumes healthy prey

The model is obtained from Eqs. 1 to 3 by setting $f_S(I,S) \equiv 0$ or $a$=0 in Eq. 4. Note that such a scenario can be used to model the avoidance of infected prey individuals by a predator due to a risk of acquiring disease (Pfennig 2000; Packer et al. 2003; Al-Zyoud and Sengonca 2004; Jones et al. 2005; Roy and Chattopadhyay 2005; Venturino 2010). Note also that sometimes pathogens can induce behavioural responses that minimize the predation risk to their host in order to benefit their own transmission (Parris et al. 2006). As in the previous subsection, we shall briefly summarize the properties of the model without predator dependence of

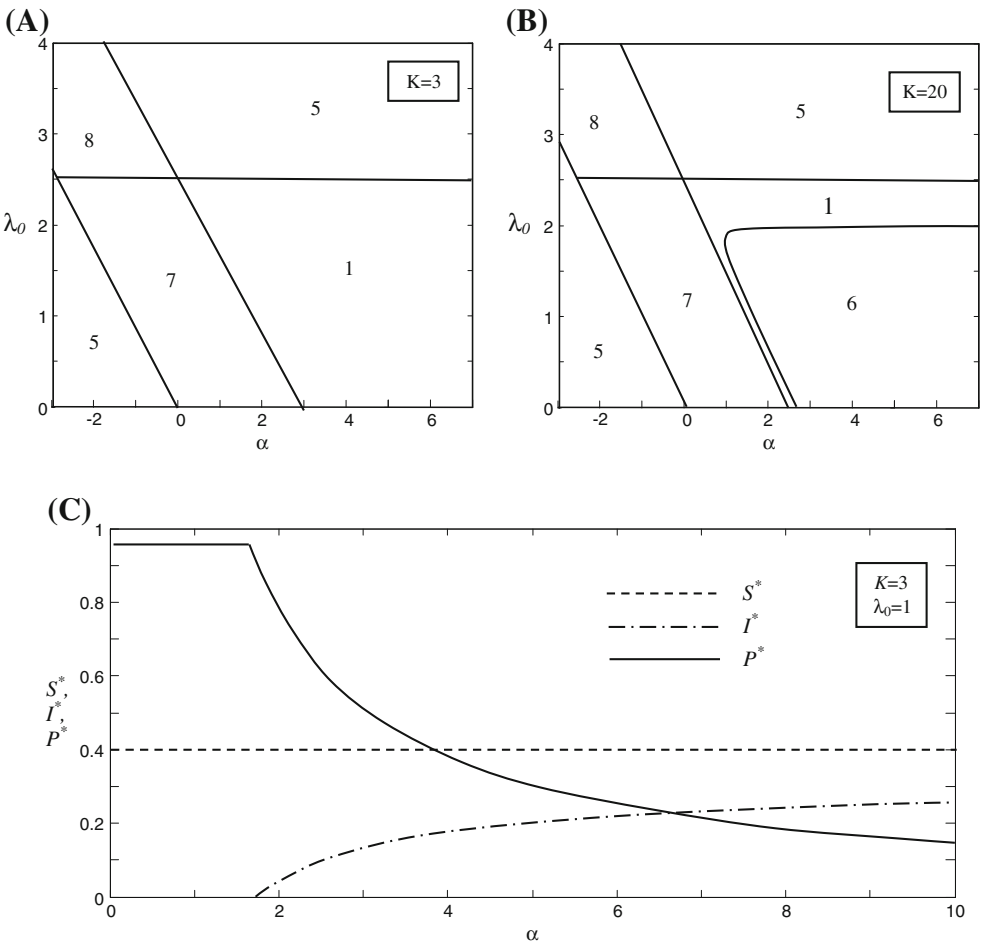


**Fig. 3** Bifurcation diagram in the $\alpha$–$\lambda_0$ plane in the case where the predator only consumes healthy prey, constructed for different degree of eutrophication. The meaning of the domains and the corresponding regimes is summarized in Table 1. **a** The carrying capacity is small ($K$=3). **b** The carrying capacity is large ($K$=20). The other model parameters are: $r$=1, $\delta$=0.1, $D$=1, $b$=1, $\omega$=0.25, $r$=1. **c** The stable stationary species densities as functions of $\alpha$ plotted for $\lambda_0$=1, $K$=3

the transmission coefficient ($\alpha$=0). The initial three-component model, S-I-P, will eventually evolve to a two-component model, i.e. either the predator or the disease will die out. In particular, the feasible stationary states are ($K$,0,0), ($S^1$*,0,$P^1$*) and ($S^1$,$I^1$,0), where ($S^1$*,0,$P^1$*) is the disease-free equilibrium and ($S^1$,$I^1$,0) the predator-free equilibrium. The predator and the infection compete with each other for the common resource, which is the healthy prey $S$. Depending on the system parameters, there can be two possible outcomes of such competition (see Appendix 2): (1) a disease-free predator–prey S-P system and (2) a predator-free S-I system. Similar outcomes have been found in other epidemiological models where infection and predation compete for a shared host/prey (Siekmann et al. 2010).

Typical parametric portraits for $\alpha \neq 0$ in the $\alpha$–$\lambda_0$ plane are shown in Fig. 3. Figure 3a gives a parametric portrait for a small carrying capacity ($K$=3) suggesting four possible domains. The meaning of the domains is summarized in Table 1. Domain 5 is a predator-free domain: the initial three-component model will eventually evolve to the stationary state ($S^1$,$I^1$,0). This is characterized by large $\lambda_0$ and positive $\alpha$. In domain 7, the infection will eventually vanish and the model will become a predator–prey model with the stable equilibrium ($S^1$*,0,$P^1$*). For domain 1 (small $\lambda_0$, large positive $\alpha$), all three species—$S$, $I$, $P$—will coexist in a stable mode. Domain 8 is characterized by a bistability, which, however, differs from that of domains 2 and 3. The trajectory goes either to ($S^1$,$I^1$,0) or to ($S^1$*,0,$P^1$*); the interior equilibrium also exists, but it is now a saddle point. The appearance of domain 1 of the coexistence of $S$, $I$, $P$, which is impossible for $\alpha$=0, can be interpreted as the indirect effects of predation on the disease transmission-promoting epidemics of severe diseases by increasing the overall $\lambda$ (i.e. increasing the epidemic threshold). The condition for establishing disease in the predator–prey system is given by (see Appendix 2)

$$\lambda_0 > D\omega b/\delta - \alpha P^{1*}. \tag{7}$$

Note that condition 7 can be obtained directly by considering the basic reproduction ratio of the disease, $R_0$. For the system, where the predator can only consume the healthy prey, $R_0$ is given by $R_0$=$\lambda(\alpha,P)S/D$, with $P$ and $S$ being stationary densities. In the absence of predators ($S$=

**Table 1** Description of the domains of the bifurcation diagrams of models 1–3 shown in Figs. 1, 2, 3 and 4

| Domain number | Occurrence in the limiting cases ($a$=0 or $b$=0) | Description of patterns of dynamics (phase space) |
|---|---|---|
| 1. | Yes | The interior equilibrium ($S^*$,$I^*$,$P^*$) is globally stable. All trajectories starting from positive initial conditions will eventually tend to this state. |
| 2. | Requires $a>0$ | The system attractors are the interior equilibrium ($S^*$,$I^*$,$P^*$) and the predator-free equilibrium ($S^1$,$I^1$,0). Depending on the initial conditions, the trajectories will tend to one of those equilibria. |
| 3. | Requires $a>0$ | The system attractors are the interior equilibrium ($S^*$,$I^*$,$P^*$) and the disease-free and predator-free equilibrium ($K$,0,0). Depending on the initial conditions, the trajectories will tend to one of those equilibria. |
| 4. | Yes | The disease-free and predator-free state ($K$,0,0) is globally stable. All trajectories starting from positive initial conditions will eventually tend to this state. |
| 5. | Yes | The predator-free equilibrium ($S^1$,$I^1$,0) is globally stable. All trajectories starting from positive initial conditions will eventually tend to this state. |
| 6. | Yes | The interior equilibrium ($S^*$,$I^*$,$P^*$) is unstable. All trajectories starting from positive initial conditions will eventually tend to a stable limit cycle, the only system attractor characterized by all $S$,$I$,$P>0$. |
| 6* | Requires $a>0$ | The interior equilibrium ($S^*$,$I^*$,$P^*$) is unstable. The system has two attractors: the predator-free equilibrium ($S^1$,$I^1$,0) and the stable limit cycle characterized by all $S$,$I$,$P>0$. All trajectories starting from positive initial conditions will eventually tend either to ($S^1$,$I^1$,0) or to the stable limit cycle. |
| 6** | Requires $a>0$ | The interior equilibrium ($S^*$,$I^*$,$P^*$) is unstable. The system has two attractors: the disease-free and predator-free equilibrium ($K$,0,0) and the stable limit cycle characterized by all $S$,$I$,$P>0$. All trajectories starting from positive initial conditions will eventually tend either to ($K$,0,0) or to the stable limit cycle. |
| 7 | Requires $b>0$ | The disease-free stationary state ($S^1{*}$,0,$P^1{*}$) is the global attractor. All trajectories starting from positive initial conditions will eventually tend to this equilibrium. |
| 8 | Requires $b>0$ | The system has two attractors: the predator-free equilibrium ($S^1$,$I^1$,0) and the disease-free equilibrium ($S^1{*}$,0,$P^1{*}$). All trajectories starting from positive initial conditions will eventually tend to one of those equilibria. |

$K$, $P$=0), the epidemic threshold is obtained from condition $K\lambda_0/D>1$, which is impossible for large virulence $D$. In the presence of predators ($S=S^1{*}$, $P=P^1{*}$), the basic reproduction number of the disease becomes $R_0=(\lambda_0+\alpha P^1{*})S^1{*}/D$; thus, for a large value of $\alpha$ (a high degree of predator dependence of the transmission rate), the value of $R_0$ can be larger than 1, thus resulting in epidemics of a disease having a large $D$.

Figure 3c shows the variation of the stationary species densities $S^*$, $I^*$, $P^*$ with increasing $\alpha$ ($K$= 3). One can see that the density of infected prey $I$ is increasing with $\alpha$, thus enhancing the infection prevalence; the predator density shows a monotone decrease.

In the case of a eutrophic environment (large $K$), domain 1 of the stable coexistence of $S$, $I$, $P$ shrinks in size (see Fig. 3b). Domain 6 appears in the diagram where the interior state ($S^*$,$I^*$,$P^*$) becomes unstable and species densities oscillate around this state. Thus, the predator-dependent transmission results in a stability loss of the system. Domain 6 grows in size with $K$. Note that the loss of stability for large $K$ and $\alpha>0$ can be proven analytically based on the stability conditions in Appendix 2. Increasing $\alpha>0$ for a fixed $\lambda_0$ and $K$ results in an increase of the amplitude of oscillations with the minimal densities through the cycle becoming very close to zero (this can be interpreted as an extinction of species). Thus, as in "Predator only consumes infected prey", predator-dependent disease transmission has a destabilizing effect, which is not possible in the system with $\alpha$=0.

## Predator consumes both healthy and infected prey

Based on the results of the previous sections, we shall briefly consider a more realistic situation where the predator consumes both healthy and infected prey, i.e. $a$, $b>0$ in Eqs. 4 and 5. For instance, grazing zooplankton do not discriminate between infected and non-infected phytoplankton (Malchow et al. 2004; Rhodes and Martin 2010), and this type of predation is also called the indiscriminating predation (see Sieber and Hilker 2011). The equilibrium points and their stability are addressed in Appendix 3. In particular, the system can have the semi-trivial stationary states ($K$,0,0), ($S^1{*}$,0,$P^1{*}$) and ($S^1$,$I^1$,0) with the coordinates given by the same expressions as for the system with $a$=0 (see Appendix 2). There can be at most two interior equilibrium points. The condition for the establishment of the disease (the epidemic thresholds) in the absence of a predator is the same as in the previous cases: $D<K\lambda_0$;

however, in the presence of a predator, it is given by (see Appendix 3)

$$\lambda_0 > D\omega b/\delta + P^{1^*}(a\omega b/\delta - \alpha). \quad (8)$$

The possibility of epidemics can also be predicted based on a computation of the basic reproduction number given by $R_0=\lambda(\alpha, P)S/(D+aP)$, with $S$ and $P$ being stationary densities. When the predator is present in the system ($S=S^1*$, $P=P^1*$), the basic reproduction number is defined by $R_0=(\lambda_0+\alpha P^1*)S^1*/(D+aP^1*)$. One can easily see that compared with the scenario where the predator consumes only healthy prey, partial consumption of infected prey ($a>0$) will increase the epidemic threshold, thus preventing the establishment of the disease. I should also emphasize that the predator-dependent disease transmission always decreases the epidemic threshold, making the value $R_0$ larger than for $\alpha=0$.

One can prove that for $\alpha=0$, the interior equilibrium points with $a$, $b>0$ (if they exist) are always stable, i.e. no sustainable oscillations (see Appendix 3). To have a good understanding of what will happen in the system for $\alpha\neq0$, we shall follow the qualitative changes in the previous bifurcation diagrams of models 1–3 obtained for the two limiting cases: $a=1$, $b=0$ and $a=0$, $b=1$ (Figs. 1, 2 and 3). The parameters $a$ and $b$ have the meaning of the attack rates on the infected and healthy prey population, respectively. First, we shall increase $b$ for a fixed $a=1$ from 0 to1. Alternatively, we shall increase $a$ for $b=1$ from 0 to1. Note that variation of the parameters $a$, $b$ can also be interpreted as changing the selectivity of predation.

The result of variation of the attack rates is shown in Fig. 4 constructed for a small carrying capacity ($K=3$; Fig. 4a, b) and for a pronounced degree of eutrophication ($K=20$; Fig. 4c). The other parameters are kept the same as in the previous figures. Due to the limited space of this paper, I do not show here the other bifurcation diagrams obtained for intermediate $a$ and $b$. The meaning of the numbers of domains in Fig. 4 is the same as in the previous figures, and it is summarized in Table 1. To follow the evolution of the parametric diagram for $K=3$ when the attack rate $b$ on the healthy predator increases, one can progressively compare the diagrams in Figs. 1a, 3a and 4a, b. After $b\approx0.3$ until $b=1$ ($a$ is fixed, $a=1$), the bifurcation diagram looks qualitatively similar to that of Fig. 3a. Note that an increase of $a$ from 0 to 1 for $b=1$ does not qualitatively affect the bifurcation portrait shown in Fig. 4a. When the carrying capacity of the system is large (Fig. 4c), the transition in the bifurcation diagrams can be followed by comparing Figs. 2a, 4c and 3b. I skip the intermediate diagram where domain 2 does not exist. After $b\approx0.1$ until $b=1$, the bifurcation diagrams look qualitatively similar to that of Fig. 3b, and reciprocally, an increase in $a$ from 0 to 1 for a fixed $b=1$ does not result in any qualitative changes of the bifurcation portrait in Fig. 3b.

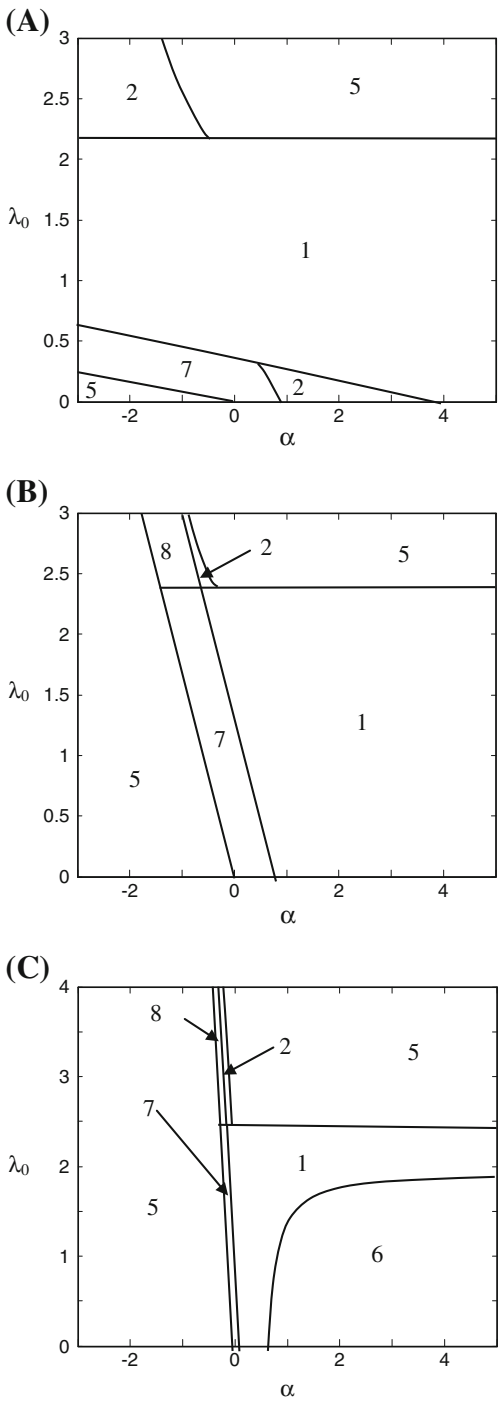


**Fig. 4** Bifurcation diagrams in the $\alpha$–$\lambda_0$ plane for the full model (Eqs. 1–3), i.e. the predator consumes both infected and healthy prey ($a$,$b>0$). **a**, **b** The carrying capacity of the system is small ($K=3$); the attack rates on the healthy prey for (**a**, **b**) are respectively $b=0.14$ and $b=0.2$. **c** The carrying capacity of the system is large ($K=20$); the attack rate on the healthy prey is $b=0.03$. The other parameters are the same as in Fig. 4 ($a=1$). The meaning of the domains and the corresponding regimes is summarized in Table 1

I should emphasize that the structure of parametric diagrams and the above-discussed transition between the diagrams shown in Figs. 1, 2, 3 and 4 is robust with respect to the variation of other model parameters.

## Discussion

Predators and pathogens often compete together for the same target species which becomes a prey and a host at the same time. This simultaneous pressure from both the predators and the pathogens on a given host/prey can be beneficial for both of these natural enemies. Indeed, it is well known that infected prey individuals become easy to catch by the predator, and this property has been taken into account in a large number of ecoepidemiological models (e.g. Chattopadhyay and Bairagi 2001; Packer et al. 2003; Venturino 2010). On the other hand, the presence of a predator can enhance pathogens' success in a prey population by debilitating its resistance to the infection. This fact has also been well recognized in the ecological literature (Baker and Smith 1997; Rigby and Jokela 2000; Thiemann and Wassersug 2000; Decaestecker et al. 2002; Navarro et al. 2003; Matz and Kjelleberg 2005; van der Veen 2005; Kortet et al. 2007; Caceres et al. 2009; Friman et al. 2009; Yin et al. 2011); however, so far, it has rarely been taken into account in theoretical models. As an exception, I can cite the work of Caceres et al. (2009) on a similar topic where the consumption of infected prey by a predator was considered to help the spore dispersal of a parasite (the predator-spreader hypothesis).

My theoretical investigation using a fairly simple ecoepidemiological model (Eqs. 1–3) demonstrates that a predator-dependent transmission rate may have an important role in shaping predator–prey–disease interactions. Firstly, a predator-dependent disease transmission rate can promote the survival of a (specialist) predator (e.g. Figs. 1 and 2), which is otherwise impossible. Indeed, the presence of predators increases the transmission rate, thus decreasing the epidemic threshold (see Eq. 7). As a result, the presence of predators creates a special environment with high contamination properties which can guarantee their own survival. Note that for a very large transmission rate $\lambda_0$, the mechanism of predator survival via their influence on the transmission rate is different and implies a decrease in the transmission rate ($\alpha<0$). Based on the model equations, one can show that for large $\lambda_0$ (and $\alpha=0$), the predator cannot establish in the system since the number of healthy and infected prey becomes small and cannot maintain the predator's growth. In such a case, survival of the predator can be made possible by a *decrease* of the total transmission rate $\lambda$ (implying $\alpha<0$), and thus increasing the equilibrium values of $S^*$ and $I^*$.

Second, the persistence of a predator in the ecosystem by influencing the disease transmission rate of its prey can be conditional (e.g. domains 2 and 3 in Fig. 1). In other words, for a successful establishment/invasion of the predator, its initial amount must be supercritical since the system shows bistability. In the model phase space, the two stable equilibrium points $(S^1,I^1,0)$ and $(S^*,I^*,P^*)$ are separated by a saddle point with $S,I,P>0$. The observed bistability is analogous to the strong Allee effect in ecology where the growth rate of a population growth becomes negative at small population densities (e.g. Allee 1938; Dennis 1989; Courchamp et al. 1999, 2008). In our case, a supercritical amount of predators creates a highly contaminating environment with a suitable food supply which cannot be maintained by smaller predator densities. Such a scenario of the emergence of a strong Allee effect (which can be referred to as a disease-mediated strong Allee effect) has not been reported in the literature yet.

Third, the predator-dependent disease transmission can promote epidemics of a highly virulent infectious disease. This conclusion can be made, for example, based on Figs. 3 and 4 as well as on conditions 7 and 8 and the computation of the basic reproduction numbers. Indeed, in the system with small $\alpha$, a disease with virulence $D$ being too large compared with its transmission rate $\lambda_0$ cannot establish in the population due to high prey mortality. The predator, by indirectly affecting the transmission rate, may increase the value of $\lambda$ and thus allows for the survival of highly viral disease strains. It is important to say that such a decrease of the epidemic threshold has another consequence: it both allows for the establishment of the infection and the survival of the predator—in the system with a high constant $\lambda$ ($\alpha=0$), the establishment of an infection will signify extinction of the predator (see Fig. 3). Note that this finding undermines the hypothesis 'keeping the herds healthy', which claims that the population size of prey ($S$) subjected to infection will rise in the presence of a predator, and thus a high level of predation ought to be beneficial for conservation purposes (Hudson et al. 1998; Packer et al. 2003; Stiling and Moon 2005).

Finally, the effects of predation on the transmission rate will be destabilizing in ecosystems with high levels of eutrophication (large $K$). This property of the model remains regardless of whether the predator consumes mostly infected or healthy prey (cf. Figs. 2, 3 and 4). Note that in the system with $\alpha=0$ and the linear functional response, the system dynamics is always stable (a globally stable interior equilibrium). Thus, taking into account the predator-dependent transmission rate makes the system vulnerable to eutrophication even for a linear predator functional response. This can be of importance since for large $\alpha$ and $K$ species, densities throughout the oscillation cycle may approach dangerously close to zero and result in

a stochastic extinction (cf. Rosenzweig 1971; McCauley et al. 1999).

An important issue that we have not addressed so far concerns the effects of the predator-dependent transmission rate on the disease prevalence, which is determined as the ratio between the infected individuals and the total number of individuals in the population: $I/(I+S)$. I have plotted the disease prevalence as a function of $\alpha$ in Fig. 5a in the case the predator only consumes infected prey. Different curves show the influence of the per capita growth rate of the prey $r$ on the disease prevalence. I only consider the model parameters for which the equilibrium species densities are stable, allowing the prevalence $I^*/(S^*+I^*)$ to be computed analytically. One can see from the graphs that an increase in $\alpha$ results in an increase in the disease prevalence.

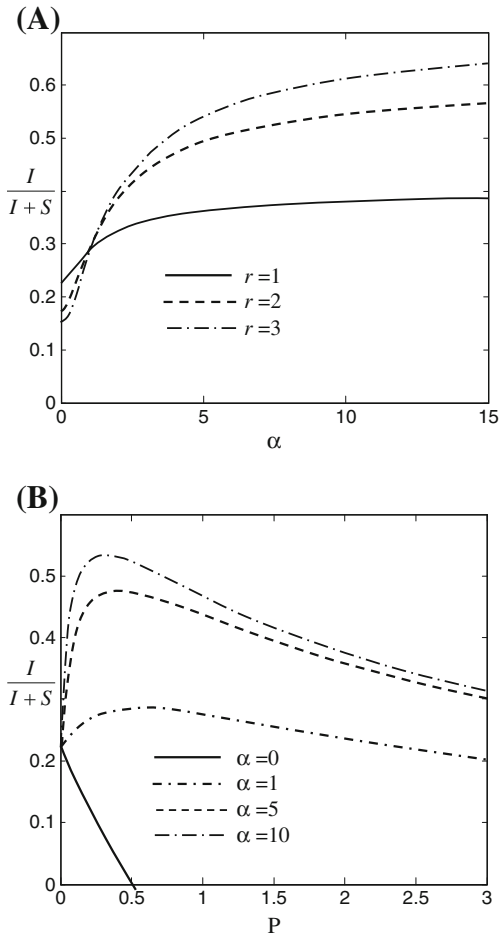


**Fig. 5** Influence of the predator-dependent disease transmission on the infection prevalence of prey, $I/(S+I)$. **a** The infection prevalence in the full system (Eqs. 1–3) with a specialist predator. Different curves are constructed for different per capita prey growth rates $r$. The model parameters are $\lambda=1$, $\delta=0.1$, $D=1$, $a=1$, $\omega=0.25$, $K=3$. **b** The infection prevalence in the system with a generalist predator ($P$ is constant). Different curves are constructed for different $\alpha$ measuring the effect of predators on the disease transmission in the prey. The model parameters are $\lambda=1$, $\delta=0.1$, $D=1$, $a=1$, $\omega=0.25$, $K=3$

Note that the graphs shown in Fig. 5a do not allow us to directly compare my results with those of Packer et al. (2003) regarding the applicability of the optimistic hypothesis on 'keeping the herds healthy' by predators (see also Ostfeld and Holt 2004). In the cited works, the predator was considered to be a generalist and not a specialist, as in our model (Eqs. 1–3). A comprehensive investigation of an S-I-P model, similar to Eqs. 1–3 with a *generalist* predator, should be done elsewhere. Here, I shall briefly address a particular scenario where a generalist predator shows a slow population growth and its biomass can be considered to be approximately constant. In this case, one can use only Eqs. 1 and 2 with the predator density $P$ being a control parameter. In Fig. 5b, I show the infection prevalence in such a system as a function of the predator density $P$. Different curves are traced for different $\alpha$. One can see that for $\alpha=0$, the predator always causes a monotonic decrease in the disease prevalence (this is the original 'keeping the herds healthy' hypothesis). However, taking into account the predator-dependent disease transmission (intermediate and large $\alpha$) can result in an increase of the disease prevalence for small and intermediate amounts of predator: only large $P$ will 'keep the herds healthy'. For small $P$, the effects of enhancing disease transmission in the presence of predators prevail over the 'beneficial' effects of the removal of infected prey. Interestingly, this scenario seems to be in good agreement with some laboratory experiments (Yin et al. 2011). Note that for the scenario where the predator only consumes the healthy prey, the results are qualitatively the same.

In the previous sections, I considered that the functional response of the predator was linear. A more realistic scenario would include saturation in the predation rate. I will briefly address a particular case where the saturation in the functional response is described by a Holling type II term with the following parameterization:

$$f_1(I,S) = a\frac{I}{I\beta+1} \tag{9}$$

where $1/\beta$ is the half-saturation density. For the sake of simplicity, I consider the scenario where the predator only consumes infected prey ($b=0$). It can be proven analytically that the system can have only one interior non-saddle equilibrium (result not shown). Interestingly, the bifurcation structure in the $\alpha$–$\lambda_0$ space remains the same as in Figs. 1 and 2. An increase in $\beta$ results in the shrinking of the domains of predator persistence. Another important property of the system, taking into account saturation in predation, is that destabilization occurs for smaller values of the carrying capacity $K$. Intuitively, such a behaviour is fairly unsurprising since destabilization becomes a joint result of both factors: predator-dependent disease transmission and the paradox of

enrichment—like destabilization which is frequently observed in predator–prey systems with Holling II functional response (cf. Rosenzweig 1971; McCauley et al. 1999).

In this paper, I considered that the predator can both enhance the transmission of infection ($\alpha>0$) and hinder it ($\alpha<0$). Examples of study cases resulting in $\alpha>0$ have been given in "Introduction". Scenarios involving a decrease of the transmission rate in the presence of predators ($\alpha<0$) are less common. However, a few such examples can be cited. Some algae can excrete substances (infochemicals) which attract their predators (zooplankton) because the predators also consume spores causing fungal infection of the algae (Kagami et al. 2004). Similar effects are reported when predators actively consume free-living stages of disease of their prey (e.g. Johnson et al. 2010). Another example includes the formation of animal groups in the presence of predators for defence purposes. Although inside each group the transmission of disease is usually enhanced (e.g. Cote and Poulin 1995), on a larger space scale, this can reduce the risk of infection of the whole metapopulation of prey since intergroup communication can be small (Watve and Jog 1997; Wilson et al. 2003). Note that in the presence of predators, some animals can hide in refuges/shelters and avoid frequent contacts with conspecies, thus reducing infection risk (Behringer and Butler 2010). Finally, in an infected population, predators may mainly focus their consumption on the most infected individuals, which are often also the most infectious in the population (high rate transmitters or the disease). Such selective grazing in the infected part of prey can potentially reduce the transmission rate of the disease (Johnson et al. 2006).

I would like to point out several future directions for related research. Firstly, it will be important to obtain more experimental and field data on the dependence of disease transmission rates on the amount of predators, i.e. the shape of $\lambda(P)$. The results have been obtained based on linear dependence; however, preliminary investigation for a quadratic dependence shows that new patterns of dynamics (e.g. multiple interior equilibrium points) can emerge. My opinion is that revealing the shape of $\lambda(P)$ experimentally is not necessarily a complicated task—a number of works have been published showing that a high density of predator kairomones results in a high infection rate of preys (e.g. Yin et al. 2011), and one simply needs to reproduce the same experiments for several other kairomones densities. Another important extension will be theoretically considering different parameterizations of $\lambda(P)$, for instance, taking into account possible saturation of transmission for large predator densities. In this paper, I have studied the scenario where the predator is a specialist, but in a number of real-world cases, it can be a generalist, thus having alternative food sources. Thus, for practical applications, it will be important to address the effects of predator-dependent disease transmission in predator–prey–disease interactions with a generalist predator. Also, an important extension will be to consider another parameterization of the transmission rate, in particular the frequency-dependent pathogen transmission which is widely used in the modelling literature (e.g. McCallum et al. 2001; Malchow et al. 2004; Hilker and Schmitz 2008). Interestingly, recent publications even argue that the incidence rate should be neither purely frequency- nor density-dependent but a mixture of both (e.g. Ryder et al. 2007); thus, it will be rather interesting to model the influence of a predator-dependent disease transmission in such situation. Finally, it will be interesting to consider the effect of evolution of virulence on the behaviour of the given system on larger (evolutionary) timescales.

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## Appendix 1

Here, I analytically investigate the local stability property of the model in the case where the predator consumes infected prey only. The model has the following stationary states:

1. The trivial stationary state (0,0,0). It is always unstable.
2. The disease-free and predator-free stationary state ($K$,0,0). It is stable for $D>K\lambda_0$.
3. The predator-free stationary state ($S^1$,$I^1$,0), with $S^1=D/\lambda_0$, $I^1=r(K\lambda_0-D)/[\lambda_0(K\lambda_0+r)]$. This state is stable if

$$\delta > \frac{\omega\, ar(K\lambda_0 - D)}{\lambda_0(K\lambda_0 + r)}, D < K\lambda_0 \tag{10}$$

4. The interior stationary state(s) ($S^*$,$I^*$,$P^*$) where the stationary values are determined by

$$S^* = \frac{rK\omega\, a - r\delta - \delta K(\lambda_0 + \alpha P^*)}{r\omega\, a}, \tag{11}$$

$$I^* = \frac{\delta}{\omega\, a}, \tag{12}$$

The value of $P^*$ is defined from the following quadratic equation:

$$Q_1 P^2 + Q_2 P + Q_3 = 0, \tag{13}$$

where the coefficients $Q_i$ are defined by $Q_1 = \alpha^2\delta K$, $Q_2 = \alpha\,\delta\, r + 2\lambda_0\delta\,\alpha\, K - \alpha\, a\, rK\omega + a^2 r\,\omega$, $Q_3 = r\, a\,\omega(D - \lambda_0 K) + \lambda_0 r\delta + \delta K\lambda_0^2$

Based on Eq. 13 and the expressions for $Q_i$, it is easy to conclude that the interior stationary state is unique (only

one positive solution of Eq. 13) in the case $Q_3<0$; $Q_2>0$. Note that the condition $Q_3<0$ is the opposite of the first inequality in Eq. 10. In the case where $Q_3>0$, there can be two positive solutions of Eq. 13, which obviously requires $Q_2^2 - 4\,Q_1 Q_3 > 0$. In the case that none of the above conditions is satisfied, the system does have interior stationary states.

The stability of the interior states is determined by the Jacobian $J$ at those states

$$J\left(S^*, I^*, P^*\right) = \begin{pmatrix} -\frac{rS^*}{K} & -S^*\left(\frac{r}{K} + \lambda_0 + \alpha P^*\right) & -\alpha S^* I^* \\ (\lambda_0 + \alpha P^*) I^* & 0 & \alpha S^* I^* - aI^* \\ 0 & \omega a P^* & 0 \end{pmatrix} \tag{14}$$

The eigenvalues of $J$ are given by the following characteristic equation:

$$\mu^3 + \Omega_1 \mu^2 + \Omega_2 \mu + \Omega_3 = 0, \tag{15}$$

where the coefficients $\Omega_i$ are given by

$$\Omega_1 = -\frac{rS^*}{K},$$

$$\Omega_2 = \frac{\omega a^2 K I^* P^* + \lambda_0 (r + K\lambda_0) S^* I^* + (2\lambda_0 K\alpha + \alpha r + \alpha^2 K P^* - \omega \alpha a K) S^* I^* P^*}{K},$$

$$\Omega_3 = -\frac{a\omega(\alpha K \lambda_0 I^* + \alpha^2 K I^* P^* + r\alpha S^* - ra) S^* I^* P^*}{K}.$$

According to the Routh–Hurwitz stability criterion, stability of ($S^*$,$I^*$,$P^*$) takes place if and only if the following conditions hold

$$\Omega_i > 0, \qquad \Omega_1 \Omega_2 > \Omega_3. \tag{16}$$

I used numerical simulations to evaluate the stability conditions (Eq. 16). Note that with the help of conditions 16 and the explicit expressions for the equilibrium densities, one can prove that for $K\to\infty$, we have $\Omega_3<0$, i.e. the interior equilibrium becomes unstable. Numerical computation shows that the loss of stability takes place via a supercritical Hopf bifurcation with a birth of stable limit cycle.

Note that in the absence of predator dependence of the transmission rate ($\alpha=0$), the system may only have one interior stationary state for which the values of $S^*$ and $P^*$ are given by

$$P^* = \frac{rKa\omega\lambda_0 - r\delta\lambda_0 - \delta\lambda_0^2 K - a\omega rD}{ra^2\omega}. \tag{17}$$

$$S^* = \frac{rK\omega a - r\delta - \delta K\lambda_0}{r\omega a}. \tag{18}$$

Application of the Routh–Hurwitz stability criterion shows that

$$\Omega_1\Omega_2 - \Omega_3 = r\,(S^*)^2 I^* \lambda_0 \frac{(r + \lambda_0 K)}{K^2} > 0.$$

In other words, the interior stationary state is always locally stable for $\alpha=0$.

## Appendix 2

Here, the local stability analysis of the model (Eqs. 1–3) is done in the case where the predator consumes healthy prey only. The model has the following stationary states.

1. The trivial stationary state (0,0,0). It is always unstable.
2. The disease-free and predator-free stationary state ($K$,0,0). This state is locally stable provided that $D> K\lambda_0$ and $\omega bK<\delta$.
3. The disease-free stationary state ($S^1$*,0,$P^1$*) with $S^1$*=$\delta/\omega b$ and $P^1$*=$r(1-S^I/K)/b$. This state is locally stable provided $\lambda_0<D\omega b/\delta-\alpha P^1$*.
4. The predator-free stationary state ($S^1$,$I^1$,0) with $S^1=D/\lambda_0$, $I^1=r(K\lambda_0-D)/[\lambda_0(K\lambda_0+r)]$. This state is locally stable for $D> K\lambda_0$ and $\lambda bS^1<\delta$.

5. The unique interior state ($S^*$,$I^*$,$P^*$), where the stationary values are determined by

$$S^* = \frac{\delta}{\omega b} \tag{19}$$

$$I^* = \frac{(Kb\omega - \delta)\alpha\delta r + (\lambda_0\delta - bD\omega)b^2K\omega}{\omega b\alpha(r\delta + Db\omega K)} \tag{20}$$

$$P^* = \frac{Db\omega - \delta\lambda_0}{\alpha\delta}. \tag{21}$$

Note that in the absence of any effects of predation on the transmission rate ($\alpha$=0), the interior stationary state does not exist. The stability of the interior state is determined by the Jacobian $J$

$$J(S^*, I^*, P^*) = \begin{pmatrix} -\frac{rS^*}{K} & -S^*\left(\frac{r}{K} + \lambda_0 + \alpha P^*\right) & -\alpha S^* I^* - bS^* \\ (\lambda_0 + \alpha P^*)I^* & 0 & \alpha S^* I^* \\ \omega b P^* & 0 & 0 \end{pmatrix} \tag{22}$$

The eigenvalues of $J$ are given by the following characteristic equation:

$$\mu^3 + \Xi_1\mu^2 + \Xi_2\mu + \Xi_3 = 0, \tag{23}$$

where the coefficients $\Xi_i$ are given by

$$\Xi_1 = -\frac{rS^*}{K},$$

$$\Xi_2 = \frac{I^*\left(-\lambda r - 2\lambda\alpha P^* K - \alpha r P^* - \alpha^2 P^{*2} K - \lambda^2 K - \omega b P^* K\alpha\right) - \omega b^2 P^* K}{K} S^*,$$

$$\Xi_3 = -\omega\frac{b\alpha\lambda K + bP^*\alpha^2 K + b\alpha r}{K} S^{*2} I^* P^*$$

Thus, the interior stationary state is stable in the case $\Xi_1\Xi_2 > \Xi_3$. In this paper, I used numerical simulations to evaluate the above stability condition.

## Appendix 3

Here, I investigate the equilibrium points of models 1–3 in the case where the predator consumes both the infected prey and the healthy prey. The local stability analysis is done based on considering the Jacobian matrix computed at the equilibrium points (for the sake of brevity, the corresponding Jacobian matrices are not shown here).

1. The trivial stationary state (0,0,0). It is always unstable.
2. The disease-free and predator-free state ($K$,0,0). The stability condition of the state is the same as in the case where the predator only consumes healthy prey.
3. The disease-free stationary state with the density of predator above zero ($S^1$*,0,$P^1$*), with $S^1{*}=\delta/\omega b$ and $P^1{*}=r(1-S^1{*}/K)/b$. This state is locally stable if $\lambda_0 < D\omega b/\delta + P^1(a\omega b/\delta - \alpha)$.
4. The predator-free stationary state ($S^1$,$I^1$,0), with $S^1$ and $I^1$ the same as in the system when prey consumes either only infected prey or healthy prey. This state is locally stable for $D < K\lambda_0$ and $\omega b S^1 + \omega a I^1 < \delta$.
5. The interior stationary state(s) ($S^*$,$I^*$,$P^*$) with all species densities being positive

$$S^* = \frac{D + aP^*}{\lambda_0 + \alpha P^*}, \tag{24}$$

$$I^* = \frac{\delta\alpha P^* + \delta\lambda_0 - b\omega(D + aP^*)}{\omega a(\lambda_0 + \alpha P^*)}, \tag{25}$$

where $P^*$ is given by the following quadratic equation:

$$R_1 P^{*2} + R_2 P^* + R_3 = 0, \tag{26}$$

with the coefficients $R_i$ defined by $R_1 = Q_1$, $R_2 = Q_2 - b\omega(ar + KD\alpha)$, $R_3 = Q_3 - (r + K\lambda_0)\omega bD$.

Here, the coefficients $Q_i$ were defined earlier in Appendix 1. From Eq. 25, one can conclude that the

number of the interior stationary states cannot be larger than 2.

The stability of the interior state is determined by the Jacobian $J$ at those states

$$J(S^*, I^*, P^*) = \begin{pmatrix} -\frac{rS^*}{K} & -S^*\left(\frac{r}{K} + \lambda_0 + \alpha P^*\right) & -\alpha S^* I^* - bS^* \\ (\lambda_0 + \alpha P^*)I^* & 0 & \alpha S^* I^* - aI^* \\ \omega bP^* & \omega aP^* & 0 \end{pmatrix}. \tag{27}$$

The corresponding expression for the characteristic equation is rather cumbersome and is not shown here. The stability analysis of the interior stationary states has been done via the Routh–Hurwitz criterion with the help of numerical simulation.

In the particular case where there is no predator dependence of the transmission rate ($\alpha$=0), the predator stationary species density becomes ($a \neq b$)

$$P^* = \frac{rb\omega D - r\delta\lambda_0 - ra\omega D + K\lambda_0(b\omega D + ra\omega - \delta\lambda_0)}{ra\omega(a-b)}. \tag{28}$$

The values of $I^*$ and $S^*$ can be found from Eqs. 24 and 25. Stability analysis based on Eq. 27 at $\alpha$=0 shows that the interior stationary state (Eq. 5) is always stable. Because of rather cumbersome expressions, I do not show the proof here.

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