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ORIGINAL ARTICLE

Diseased Social Predators

Frank M. Hilker[1] · **Marta Paliga**[2] · **Ezio Venturino**[3]

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Abstract Social predators benefit from cooperation in the form of increased hunting success, but may be at higher risk of disease infection due to living in groups. Here, we use mathematical modeling to investigate the impact of disease transmission on the population dynamics benefits provided by group hunting. We consider a predator–prey model with foraging facilitation that can induce strong Allee effects in the predators. We extend this model by an infectious disease spreading horizontally and vertically in the predator population. The model is a system of three nonlinear differential equations. We analyze the equilibrium points and their stability as well as one- and two-parameter bifurcations. Our results show that weakly cooperating predators go unconditionally extinct for highly transmissible diseases. By contrast, if cooperation is strong enough, the social behavior mediates conditional predator persistence. The system is bistable, such that small predator populations are driven extinct by the disease or a lack of prey, and large predator populations survive because of their cooperation even though they would be doomed to extinction in the absence of group hunting. We identify a critical cooperation level that is needed to avoid the possibility of unconditional predator extinction. We also investigate how transmissibility and cooperation affect the stability of predator–prey dynamics. The introduction of parasites may be fatal for small populations of social predators that decline for other reasons. For invasive predators that cooperate strongly, biocontrol by releasing parasites alone may not be sufficient.

✉ Frank M. Hilker
frank.hilker@uni-osnabrueck.de

1 Institute of Environmental Systems Research, School of Mathematics/Computer Science, Osnabrück University, Osnabrück, Germany

2 Scuola Politecnica, DIID, University of Palermo, Palermo, Italy

3 Department of Mathematics, “Giuseppe Peano”, University of Torino, Turin, Italy

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1 Introduction

Group living is a widespread phenomenon in the animal kingdom. Carnivores form hunting groups, birds breed in colonies, fish school together, and herbivores form herds. Living in groups provides on the one hand many benefits due to cooperation (e.g., increased foraging efficiency, group defence, increased access to mates, and help from kin), but there are also numerous costs on the other hand (e.g., increased risk of disease or parasites, increased competition for food, and attraction of predators). While there has been considerable work on the evolution, function, and optimal group size of animal aggregations (Rubenstein 1978; Krause and Ruxton 2002; Beauchamp 2014), the impact of group living seems underexplored in dynamic population and community models.

In order to improve their skills in defence or hunting, many animals exhibit social behavior and cooperate with other members of their species. Cooperation is common in carnivores (e.g., African wild dogs (Courchamp and Macdonald 2001), spiders (Ward and Enders 1985), and chimpanzees (Watts and Mitani 2002)). Often, this behavior is a reflection of the environment in which the animals live; for example, there are many pack hunters in the Afrotropical region, where vast areas do not guarantee a sufficient food supply individually (Packer and Ruttan 1988; Rubenstein and Lovette 2007). Furthermore, harsh climatic conditions due to the Indian Ocean Dipole or the El Niño Southern Oscillation can enforce populations to cooperate for survival (McMahon et al. 1992).

At the same time, aggregation in groups comes with the risk of disease transmission. There is empirical evidence for a positive correlation between group size and both the prevalence and intensity of contagious parasites (Côté and Poulin 1995). For instance, the king penguin (*Aptenodytes patagonicus*) breeds in colonies of up to 500,000 individuals. Adults and chicks in large colonies are increasingly infested with ticks (*Ixodes uriae*) (Mangin et al. 2003), which reduce the incubation success of adults. Moreover, living in dense populations can promote transmission of some infectious diseases because of increased aggressive interactions, large social groups, or promiscuous mating systems (Loehle 1995; Altizer et al. 2003).

The interaction between disease transmission and cooperative behavior has been recently investigated in prey populations (Bate and Hilker 2014). As infection by a disease reduces prey density, this tends to weaken group defense against predators. Mathematical models show that prey infection allows predators to survive if they would go conditionally or unconditionally extinct in the absence of prey infection due to effective group defense. As for the stability of predator–prey dynamics, prey infection can stabilize oscillations to fixed points on the one hand, but also induce chaotic attractors and attractor crises on the other hand. Yet another effect of prey infection is the facilitation of stable (as opposed to oscillatory) coexistence of two consumers on one resource.

The objective of this paper is to investigate the impact of disease transmission in social predators. We shall be interested in the interplay of cooperative host behavior and infection dynamics, and how this interplay affects the persistence and stability of the predator–prey system. To this end, we extend a model of group-hunting predators (Teixeira Alves and Hilker 2017) by considering an infectious disease spreading in the predator population. We thus obtain an eco-epidemiological model that combines the effects of disease transmission and ecological interactions (see Venturino 2016, for a review of this type of models).

Group hunting is a form of foraging facilitation and can induce strong Allee effects (Berec 2010; Teixeira Alves and Hilker 2017), i.e., a positive relationship between population density and per capita growth rate (Allee 1931; Courchamp et al. 2008). Group hunting can have different consequences. On the one hand, it can mediate predator survival in ecological conditions where predators would go extinct without cooperation (Teixeira Alves and Hilker 2017). On the other hand, if pack hunting is too strong, the predation pressure may reduce prey population size to such levels that the predator population size decreases because of overexploitation (Teixeira Alves and Hilker 2017). Regarding the stability of predator–prey systems, group hunting tends to be destabilizing as it extends the parameter range of limit cycle oscillations (Berec 2010) and induces limit cycles that are not possible in the absence of cooperation (Teixeira Alves and Hilker 2017).

For the infectious disease in the predator population, we assume frequency-dependent disease transmission because this can drive the host population extinct for large enough transmissibilities. Moreover, we focus exclusively on density-mediated effects of the disease. That is, we assume the only impact of the disease is to reduce the host population density, but we do not consider trait-mediated effects, e.g., that the disease influences the group hunting behavior of infected predators. Nevertheless, the impact of disease mortality is hard to predict as there are different positive and negative feedback loops in the system. Increased disease-related mortality could be amplified by less effective group hunting or even lead to predator extinction by itself. By contrast, reduced predator densities could release prey from overexploitation by large predator populations and thus have a positive effect also on predators.

2 Model Derivation

2.1 Group Hunting and Predator Infection

In the absence of disease, the predator–prey model is of the form

$$\begin{aligned} \frac{\mathrm{d}N}{\mathrm{d}T} &= r\left(1-\frac{N}{K}\right)N - a(P)NP, \\ \frac{\mathrm{d}P}{\mathrm{d}T} &= -mP + \epsilon a(P)NP, \end{aligned} \tag{1}$$

where N and P represent the densities of the prey and predator populations, respectively. The prey population grows logistically with per capita growth rate r and carrying

capacity K. Predators have a natural per capita death rate m. Parameter ϵ is the efficiency with which predators convert consumed prey into their own growth. In order to incorporate cooperative hunting among predators, we assume that the attack (or search) rate per predator and prey $a(P)$ is a function of predator density. More specifically, we assume a linear relationship

$$a(P) = a_0 + a_1 P,$$

where $a_0 \geq 0$ is the density-independent attack rate in the absence of cooperative behavior and $a_1 \geq 0$ represents the strength of predator cooperation. If $a_1 = 0$, the attack rate is constant and model (1) corresponds to the Lotka–Volterra model with a linear functional response and with prey self-regulation. If $a_1 > 0$, model (1) corresponds to the foraging facilitation model by Teixeira Alves and Hilker (2017).

Next, we account for disease infection of predators. We consider a simple SI system and split the predator population $P = S + I$ into subpopulations of susceptibles (S) and infecteds (I). Consequently, we obtain two different attack rates

$$a_S(S, I) = a_{S0} + a_{SS} S + a_{SI} I,$$
$$a_I(S, I) = a_{I0} + a_{IS} S + a_{II} I,$$

of susceptible and infected predators, respectively. a_{S0} is the density-independent attack rate of a susceptible predator, and a_{SS} and a_{SI} represent how the attack rate of susceptible predators is increased by the presence of susceptible and infected predators, respectively. Parameters a_{I0}, a_{IS}, and a_{II} have analogous meanings for the attack rate of infected predators. In general, these parameters can be different from each other. For example, if infected predators cannot hunt, $a_{I0} = 0, a_{SI} = a_{II} = 0$, so that all hunting activity is done by susceptibles, $a_{S0}, a_{SS} > 0$. If $a_{IS} > 0$, infected predators benefit from group hunting with susceptibles who might tolerate infecteds at a kill or feed them by regurgitation. As another example, if infected predators participate in group hunting but are incapacitated due to disease, they are likely to not cooperate with their conspecifics in the same way as susceptibles do. In this case, we have:

$$a_{SI} < a_{SS}, \quad a_{II} < a_{SI}.$$

As a final example, predators could be more aggressive due to infection by a particular disease such as rabies. They can thus provide more resources to the population than before. In this case, we have:

$$a_{SI} > a_{SS}, \quad a_{II} > a_{SI}.$$

These extensions lead to a model with three populations (N, S, and I), which reads

$$\frac{\mathrm{d}N}{\mathrm{d}T} = r\left(1 - \frac{N}{K}\right) N - a_S(S, I) NS - a_I(S, I) NI, \tag{2}$$
$$\frac{\mathrm{d}S}{\mathrm{d}T} = -\beta \frac{SI}{P} - mS + \epsilon a_S(S, I) NS + (1 - \theta)\, \epsilon a_I(S, I) NI, \tag{3}$$

$$\frac{\mathrm{d}I}{\mathrm{d}T} = \beta\frac{SI}{P} - mI - \mu I + \theta\epsilon a_I(S, I)NI. \tag{4}$$

This model assumes frequency-dependent disease transmission, with transmission coefficient β. The potential impact of density-dependent transmission is considered in Discussion. Moreover, we assume that the disease can be transmitted vertically, i.e., from parent to offspring, where $\theta \in [0, 1]$ represents the fraction of newborns acquiring the infection from the parent. Furthermore, infected predators are subject to an additional disease-related per capita mortality μ. There is no recovery from the disease.

2.2 Simplifying Assumptions

Model (2)-(4) has thirteen parameters. In order to reduce the number of parameters and thus simplify the model, we assume that

$$a_0 = a_{S0} = a_{I0}, \quad a_1 = a_{SS} = a_{SI} = a_{IS} = a_{II}.$$

Biologically, this means that the disease does not influence the foraging behavior of predators. Hence, the attack rate is the same for susceptible and infected predators:

$$a_S(S, I) = a_I(S, I) = a_0 + a_1(S + I) = a_0 + a_1P.$$

Then, the model reads

$$\begin{aligned}
\frac{\mathrm{d}N}{\mathrm{d}T} &= r\left(1 - \frac{N}{K}\right)N - (a_0 + a_1P)\,NP,\\
\frac{\mathrm{d}S}{\mathrm{d}T} &= -\beta\frac{SI}{P} - mS + \epsilon\,(a_0 + a_1P)\,NS + (1 - \theta)\,\epsilon\,(a_0 + a_1P)\,NI,\\
\frac{\mathrm{d}I}{\mathrm{d}T} &= \beta\frac{SI}{P} - mI - \mu I + \theta\epsilon\,(a_0 + a_1P)\,NI.
\end{aligned}$$

We now perform a change of state variables, which allows us not only to deal with the singularity in the frequency-dependent transmission term, but also to distinguish disease-induced extinction cases from trivial extinction equilibria. To this end, we replace the state variables S and I by the new state variables P and i, which are the total predator population and its prevalence $i = I/P$, respectively. This yields

$$\begin{aligned}
\frac{\mathrm{d}N}{\mathrm{d}T} &= r\left(1 - \frac{N}{K}\right)N - (a_0 + a_1P)\,NP,\\
\frac{\mathrm{d}P}{\mathrm{d}T} &= -(m + \mu i)\,P + \epsilon\,(a_0 + a_1P)\,NP,\\
\frac{\mathrm{d}i}{\mathrm{d}T} &= i\,(1 - i)\,(\beta - \mu) - (1 - \theta)\,\epsilon\,(a_0 + a_1P)\,Ni.
\end{aligned}$$

We now nondimensionalize this model to ease its analysis. Introducing the dimensionless variables

$$n = \frac{\epsilon a_0}{m} N, \quad p = \frac{a_0}{m} P, \quad t = mT,$$

and dimensionless parameters

$$r' = \frac{r}{m}, \quad k = \frac{\epsilon a_0 K}{m}, \quad \alpha = \frac{m a_1}{a_0^2}, \quad \mu' = \frac{\mu}{m}, \quad \beta' = \frac{\beta}{m},$$

we obtain

$$\frac{\mathrm{d}n}{\mathrm{d}t} = r\left(1 - \frac{n}{k}\right) n - (1 + \alpha p)\, np, \tag{5}$$

$$\frac{\mathrm{d}p}{\mathrm{d}t} = -(1 + \mu i)\, p + (1 + \alpha p)\, np, \tag{6}$$

$$\frac{\mathrm{d}i}{\mathrm{d}t} = i\,(1 - i)\,(\beta - \mu) - (1 + \alpha p)\,(1 - \theta)\, ni, \tag{7}$$

where we have dropped the dashes of dimensionless parameters for notational convenience. This is the model we will analyze in the following. Our attention will be mainly focused on the impact of pack hunting, α, and disease transmissibility, β. Before proceeding we note that the infection will not persist in the predator population ($i \to 0$ as $t \to \infty$) if the transmission coefficient is smaller than the virulence ($\beta < \mu$) because $\mathrm{d}i/\mathrm{d}t < 0$ in that case.

2.3 Special Cases

Model (5)-(7) extends the Lotka–Volterra model with logistic prey growth by (i) disease spread in the predators and (ii) predator cooperation. We will briefly consider special cases before analyzing the full model.

2.3.1 Eco-Epidemiological Model Without Hunting Cooperation

In the absence of predator cooperation ($\alpha = 0$), model (5)-(7) reduces to a predator–prey model with infected predators. It corresponds to one of the models studied by Oliveira and Hilker (2010), who also considered density-dependent transmission but assumed in each case no vertical transmission, i.e., $\theta = 0$. In the case of frequency-dependent transmission, four different types of dynamical behavior are possible:

(a) Disease-free case: The disease cannot establish itself in the predator population. The system behaves like the classical Lotka–Volterra model with either (i) predators and prey coexisting at stable equilibrium or (ii) predators going extinct and prey reaching carrying capacity.
(b) Stable endemic coexistence: The disease establishes itself in the predator population, and all species (prey, predators, disease) coexist at a stable equilibrium.

(c) Oscillatory endemic coexistence: All species coexist in sustained oscillations, which are due to limit cycles.
(d) Disease-induced predator extinction: The disease drives the predator population to extinction, with the prey reaching carrying capacity.

In case (c), the disease induces limit cycle oscillations that do not exist in the predator–prey model without infection.

2.3.2 Hunting Cooperation Without Disease

If the disease does not establish itself in the predator population, i.e., $i = 0$, model (5)-(7) reduces to the predator–prey model with foraging facilitation considered by Teixeira Alves and Hilker (2017). We now briefly recap the main results because they are helpful to understand the dynamics of the full model.

If predators cannot survive in the absence of cooperative behavior, strong enough hunting cooperation translates into a demographic Allee effect and can mediate the survival of the predator population. In that case, there is bistability between predator extinction and one of the following dynamical outcomes

(a) Stable coexistence of predators and prey
(b) Oscillatory coexistence of predators and prey (limit cycle)

However, if hunting cooperation is too strong and the coexistence is oscillatory, the predator population collapses as it overexploits the prey and dips below the Allee threshold, i.e., the minimum viable population density due to the Allee effect. Mathematically, this is related to a homoclinic bifurcation. As a consequence, the dynamics is monostable and there is

(c) Predator extinction with prey at carrying capacity.

If predators and prey coexist in the absence of cooperative behavior, they continue to do so in the presence of hunting cooperation. In this case, there is no bistability, but rather

(d) Monostable coexistence of predators and prey.

Foraging facilitation tends to have a negative indirect effect on prey density. The direct effect on predator density is positive if hunting cooperation is not too strong, but negative if predators have grown to such a large population density that cannot be sustained anymore by the prey.

3 Equilibria and Stability Analysis

We now consider model (5)-(7) that combines hunting cooperation and predator infection. We will first present the basic reproduction numbers of the disease and of the predators. We will then explore the impact of hunting cooperation in an isocline analysis, where we restrict the three-dimensional dynamics onto a phase plane, assuming that the disease prevalence reaches its asymptotic value. Finally, we will summarize the results of a linear stability analysis.

3.1 Basic Reproduction Numbers

We begin with the basic reproduction number of the disease in the predator population, which can be defined as

$$\mathcal{R}_0(p^\circ, n^\circ) = \frac{\beta + \theta(1 + \alpha p^\circ)n^\circ}{1 + \mu}. \tag{8}$$

For this, we assume that predators and prey are at disease-free equilibrium densities p° and n°, respectively. The basic reproduction number gives the number of secondary infections caused by a single infected predator during its infectious period when introduced into a completely susceptible population of density p° and available prey n°. The mean lifetime of an infected predator is the reciprocal of $1 + \mu$, which is the sum of the dimensionless natural and disease-related per capita death rates. The secondary infections are due to horizontal transmission, β and vertical transmission, $\theta(1 + \alpha p^\circ)n^\circ$.

Hunting cooperation increases the basic reproduction number of the disease, provided that there is vertical transmission, i.e., $\theta > 0$. As horizontal disease transmission is frequency dependent, it is independent of predator density. Therefore, hunting cooperation affects disease spread only via vertical transmission.

If $\mathcal{R}_0(p^\circ, n^\circ) < 1$, the disease cannot spread in the predator population and disappears. The system will settle into the disease-free subsystem with hunting cooperation only (summarized in the previous section). If $\mathcal{R}_0(p^\circ, n^\circ) > 1$, the disease can spread and becomes endemic in the predator population.

Now, we consider the basic reproduction number of the predator population. It gives the average number of offspring produced by a single predator during its lifetime when introduced into a prey population in the absence of any other predators. It can be defined as

$$\mathcal{R}_p(i^*) = \frac{k}{1 + \mu i^*},$$

where the numerator is the dimensionless numerical response of a single predator in a prey population at carrying capacity. The denominator gives the death rate of a predator population infected at prevalence level i^*. Note that the prevalence level can be defined for frequency-dependent transmission because the contact rate is independent of predator density. The prevalence considered here corresponds to the proportion of infected predators when the total predator population tends to zero.

The predator basic reproduction number does not take into account the effect of hunting cooperation, because it is defined on the assumption that there are no other predators. However, the predator basic reproduction number involves the other ecological (predation), demographic (mortality), and epidemiological (virulence) processes. The condition $\mathcal{R}_p(i^*) > 1$ therefore expresses whether the predators, in the absence of hunting cooperation, can sustain themselves on the prey population available and in the presence of infection. If this is the case, we can say that the predators are *eco-epidemiologically sustainable*. If $\mathcal{R}_p(i^*) < 1$, the predators are *eco-epidemiologically unsustainable* and go extinct.

If the predator population is disease-free, $\mathcal{R}_p(0) = k$ as in Teixeira Alves and Hilker (2017). In the presence of infection, disease-related mortality reduces the predator basic reproduction number. This is plausible because the predators need to compensate disease-related deaths.

3.2 Phase Plane Approximation

At any nontrivial equilibrium with $p^* > 0$ and $n^* > 0$, the prevalence of infected predators equals

$$i^* = \frac{\beta - \mu - (1-\theta)}{\beta - \mu\theta}. \tag{9}$$

This follows from an isocline analysis (see "Appendix A"). The prevalence depends only on disease-related parameters (horizontal and vertical transmission as well as virulence) and is not affected by any ecological parameter.

Fixing the prevalence at its equilibrium level i^* defines a section of the three-dimensional state space of model (5)-(7). This allows us to approximate the dynamics in the predator–prey phase plane on the assumption that predators are infected with prevalence i^* and persist on prey. Figure 1 shows several phase planes for different values of critical parameter combinations.

- If $\mathcal{R}_p(i^*) > 1$, there is a unique nontrivial equilibrium E_{npi} of infected predators and prey, see Fig. 1b, d and "Appendix A" for details. The existence of this nontrivial equilibrium is independent of the strength of hunting cooperation.
- If the predators are eco-epidemiologically unsustainable, i.e., $\mathcal{R}_p(i^*) < 1$, the situation is more complicated. Hunting cooperation can induce the existence of multiple equilibria E_{npi}. The nontrivial nullclines of prey and predators can intersect twice, once, or not at all. Numerical simulations suggest that these different outcomes depend on the value of the cooperation parameter α (see also Sect. 4.2):
  - If α is smaller than a critical value, $\alpha < \alpha_c$, there is no intersection. The only stable equilibrium is the one where predators are extinct and prey at carrying capacity (Fig. 1a). Hence, predator cooperation is not strong enough to facilitate predator survival.
  - If $\alpha > \alpha_c$, there are two different coexistence equilibria E_{npi}, one stable and one unstable (Fig. 1c). In this case, we observe a strong demographic Allee effect: The predators either go extinct or coexist with the prey. The system is bistable, and the outcome depends on the initial condition. If the predator density is large enough, the hunting cooperation mediates predator survival. By contrast, if the predator density is too small, the predator population goes extinct (which could be caused by a lack of prey consumption or by the disease).
  - For the particular parameter value $\alpha = \alpha_c$, there is a unique intersection between the two nullclines, which suggests this is a saddle–node bifurcation point separating the two previous cases.

That is, hunting cooperation can mediate the survival of predators if they go extinct in the absence of foraging facilitation. For this to occur, the hunting cooperation needs to be strong enough. Numerical simulations suggest that the critical value $\alpha_c(i^*)$

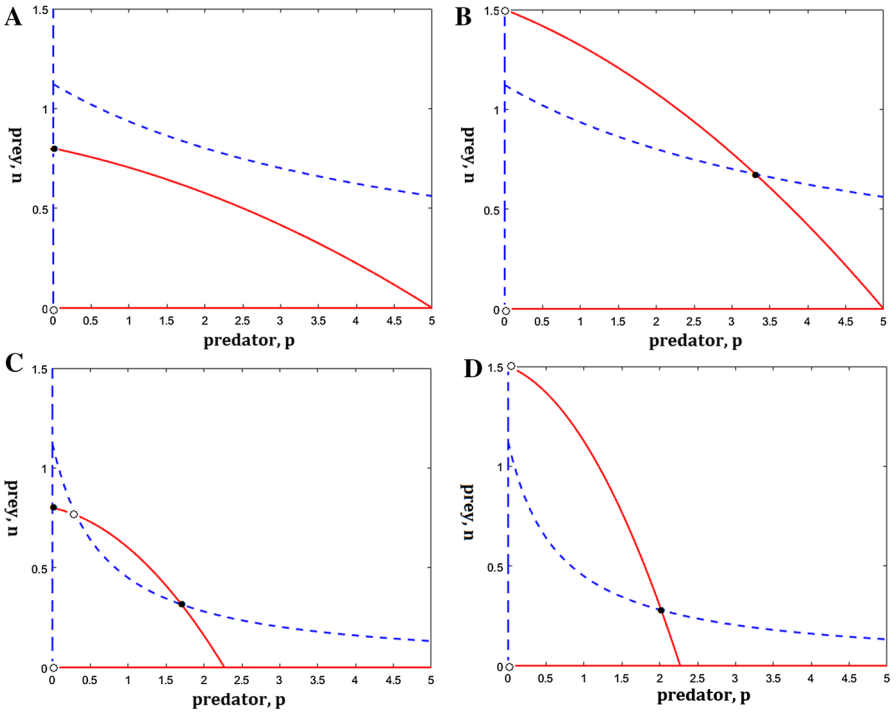

Fig. 1 Phase planes (p, n) with the prevalence fixed at nontrivial equilibrium level $i = i^*$. *Top panels* weak hunting cooperation ($\alpha = 0.2 < \alpha_c$), *bottom panels* strong hunting cooperation ($\alpha = 1.5 > \alpha_c$). *Left panels* eco-epidemiologically unsustainable predators ($k = 0.8 < k_c(i^*)$), *right panels* eco-epidemiologically sustainable predators ($k = 1.5 > k_c(i^*)$). The *dashed blue* lines indicate the predator nullclines and the *solid red lines* the prey nullclines. They are based on the assumptions to intersect the prevalence nullplane $i = i^*$ (for the nontrivial nullclines). The boundary equilibria and nullclines have been added from the three-dimensional model. The *filled circles* represent stable steady states, the *empty circles* the unstable steady states. The other parameter values are $r = 10$, $\mu = 0.3$, $\theta = 0.1$, $\beta = 2$ (Color figure online)

depends on the disease prevalence. In particular, $\alpha_c(i^*)$ increases with disease prevalence, which seems plausible because predators have to compensate higher infection levels by more intensive hunting cooperation. We will refer to the case $\alpha < \alpha_c(i^*)$ as *weak hunting cooperation* and the case $\alpha > \alpha_c(i^*)$ as *strong hunting cooperation*. The latter allows predators to survive in an eco-epidemiologically unsustainable environment.

3.3 Linear Stability Analysis

Table 1 summarizes the results from the equilibrium and stability analysis, using the threshold quantities identified in this Section. Details of the linear stability analysis are given in "Appendix B".

In the disease-free case, $\mathcal{R}_0(p^\circ, n^\circ) < 1$, the system settles either on the prey-only equilibrium or on a coexistence state between prey and susceptible predators. Due to the strong Allee effect mediated by hunting cooperation, there can be multiple

Table 1 Equilibria of models (5)–(7) and their existence and stability conditions

Equilibrium	Meaning	Existence	Stability
$E_0 = (0, 0, 0)$	Extinction of all species	Always	Unstable
$E_i = (0, 0, 1)$	Extinction of all species	Always	Unstable
$E_{\mathrm{n}} = (k, 0, 0)$	Prey only	Always	$\mathcal{R}_p(0) < 1$ and $\mathcal{R}_i < 1$
$E_{\mathrm{ni}} = (k, 0, i^\dagger)$	Disease-induced predator extinction	$\mathcal{R}_i > 1$	$\mathcal{R}_p(i^*) < 1$
$E_{\mathrm{np}} = (n^\circ, p^\circ, 0)$	Disease-free predator–prey coexistence	n/a	n/a
$E_{\mathrm{npi}} = (n^*, p^*, i^*)$	(Endemic) coexistence	$\mathcal{R}_0(0, 1) > 1$ necessary	n/a

See "Appendix B" for more details
The existence condition for E_{npi} is only necessary. n/a: not available

disease-free predator–prey equilibria E_{np}. In the presence of disease, predators survive if $\mathcal{R}_p(i^*) > 1$ or if strong hunting cooperation ($\alpha > \alpha_c(i^*)$) mediates their survival if they are eco-epidemiologically unsustainable.

Finally, we note that there can be two different prey-only equilibria. At the first one (E_{ni}), disease infection is strong enough to drive predators extinct. Consequently, there is a strictly positive prevalence in the limit process as the predator density approaches zero. If the predators would go extinct due to purely ecological reasons, the prevalence would be zero in the limit process (E_{n}). The disease-induced extinction equilibrium exists if the total predator population decays faster (namely at per capita rate $(1 + \alpha p)n - 1$) than the infected predators (at rate $\beta + \theta(1 + \alpha p)n - 1 - \mu$). As $p \to 0$ and $n \to k$, this condition can be expressed in terms of the prevalence reproduction number (cf. Oliveira and Hilker 2010),

$$\mathcal{R}_i = \frac{\beta + \theta k}{\mu + k} > 1.$$

4 Bifurcation Analysis

We now focus on the case that hunting cooperation can mediate predator survival in the disease-free system. That is, predators would go extinct in the absence of pack hunting because the prey supply does not sustain the predator population ($\mathcal{R}_p(0) < 1$), but cooperation allows predators to persist if their density is large enough ($\alpha > \alpha_c(0)$). In this case, the dynamics is bistable, see Fig. 1c. We are interested in the impact of predator infection, in particular whether the disease could potentially undermine the cooperation-mediated survival and drive the predators extinct, or whether predators are able to withstand and persist.

4.1 One-Parameter Bifurcations

Figure 2 shows bifurcation diagrams with varying disease transmissibility. For small values of disease transmissibility, the disease cannot establish itself in the predator

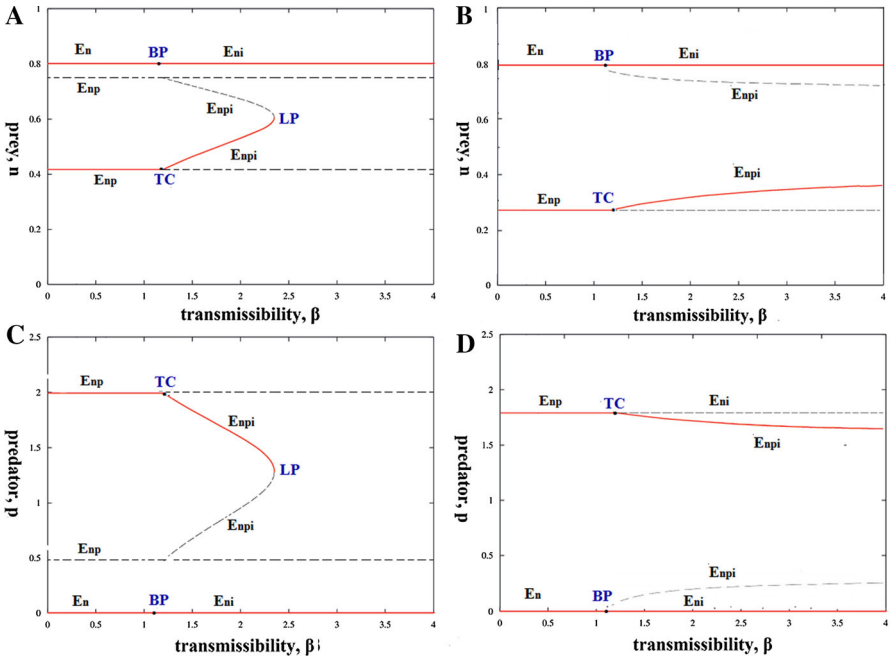

Fig. 2 Bifurcation diagrams with a bistable *baseline* scenario, in which cooperation can mediate predator survival in the disease-free system. *Left panels* ($\alpha = 0.7$) Sufficiently strong disease transmissibility undermines cooperation-mediated survival and always leads to predator extinction. *Right panels* ($\alpha = 1.2$) Hunting cooperation is so strong that predator survival remains possible for all values of β. *Top panels* show prey and *bottom panels* predator population densities at equilibrium, when varying β. The disease prevalence in the predator population is not shown here. It is given explicitly in Eq. (9) for the coexistence equilibrium and increases monotonically with β. *Solid red lines* represent stable equilibria, *dashed black lines* represent unstable equilibria. Equilibria are labeled according to Table 1. Bifurcation points are labeled as LP (limit point), TC (transcritical), and BP (branching point). Models (5)–(7), remaining parameter values: $\theta = 0.1$, $\mu = 0.3$, $r = 10$, and $k = 0.8 < k_c(0)$ (Color figure online)

population. The system remains disease-free and approaches either the coexistence state E_{np} or the prey-only state E_n. The disease-free equilibria are unaffected by transmissibility and therefore constant in Fig. 2. For larger transmissibilities, the disease invades the predator population. Two endemic coexistence equilibria E_{npi} emerge from the disease-free coexistence states E_{np}, one of which is stable and the other one unstable. The predator density at the stable branch of E_{npi} is depressed with increasing transmissibility, whereas prey density increases. All this holds true for both values of hunting cooperation considered in Fig. 2.

If hunting cooperation is strong but relatively small (left-hand side panels of Fig. 2), there is a limit point bifurcation when further increasing transmissibility. At that point, the two nontrivial equilibria coalesce and disappear. For larger values of β, predators go extinct due to the disease, and prey reach carrying capacity. The system has lost its bistability as the disease has become too strong.

By contrast, if hunting cooperation is strong and also sufficiently large (right-hand side panels of Fig. 2), there is no limit point bifurcation. Cooperation-mediated

predator survival remains possible for all values of the transmissibility considered. Nevertheless, the disease has a detrimental effect on predators. Not only does it reduce the predator density at the stable coexistence equilibrium E_{npi}, but numerical simulations (not shown here) suggest that it also tends to increase the Allee threshold, i.e., the minimum predator density required for persistence. Note that the Allee threshold depends on the prey and prevalence levels because we consider a three-dimensional system. The Allee threshold is therefore related to the boundary between the predator survival and extinction region; in particular, the Allee threshold is not identical with the unstable saddle point (Berec et al. 2001; Teixeira Alves and Hilker 2017, Francomano et al, in press). However, hunting cooperation is sufficiently strong that the system remains bistable and predator survival is always possible, for all values of the disease transmissibility.

4.2 Two-Parameter Bifurcation Diagram

To have a clearer picture of the effects of both disease and pack hunting, Fig. 3 shows a two-parameter bifurcation in the (α, β) parameter plane. If β is sufficiently small, the system remains disease-free. In this case, predators cannot survive if hunting cooperation is weak (E_n). However, if hunting cooperation is strong, there is bistability between E_n and predator–prey coexistence, which can be stable (E_{np}) or cyclic (cf. Teixeira Alves and Hilker 2017).

Let us now consider the case where β is large enough so that the disease becomes endemic. On the left-hand side of the limit point bifurcation curve, α is so small that hunting cooperation is weak and predators go extinct (E_{ni}). Crossing the limit point bifurcation curve, a strong Allee effect renders the system bistable: The predators either go extinct or coexist with prey. For larger values of α, there is a Hopf bifurcation, where E_{npi} becomes unstable and the populations oscillate. There is still bistability, namely between E_{ni} and the limit cycle oscillations.

We gather two important results from Fig. 3. First, the limit point bifurcation curve has an asymptote for increasing disease transmissibility, i.e., $\alpha_c(i^*) \to \alpha_c \approx 1.01$ as β becomes large. That is, for sufficiently large hunting cooperation ($\alpha > \alpha_c$), there is always the possibility of coexistence for at least some initial conditions even if disease transmissibility becomes extremely high. Second, the minimum value of α for Hopf bifurcations to occur is α_h. When fixing hunting cooperation at a value $\alpha > \alpha_h$, there can be two Hopf bifurcations when varying β, giving rise to a bubbling effect, see Fig. 4.

4.3 Impact of Vertical Transmission

Finally, we investigate the impact of vertical transmission. Figure 5 shows two-parameter bifurcation diagrams for increasing values of θ. We observe the following effects.

First, the higher the proportion of vertical transmission, the smaller the disease-free parameter region (orange color in Fig. 5). This makes sense as vertical transmission increases the basic reproduction number, see Eq. (8).

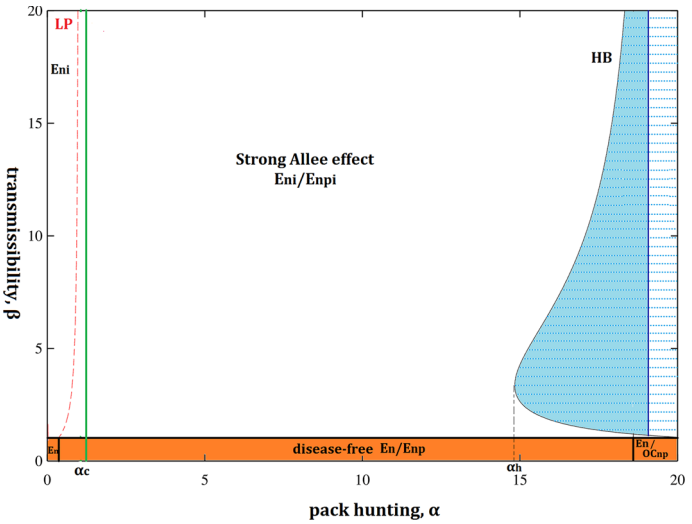

Fig. 3 Two-parameter bifurcation diagram in the parameter plane (α, β) for the case of an ecologically unsustainable predator population ($k < k_c(0)$). *Left* of the limit point curve (LP, *dashed red line*) the disease always drives the predators extinct. To the *right*, a strong Allee effect due to hunting cooperation facilitates predator survival. The *bold green line* is the asymptote α_c of the limit point curve for large transmissibilities; it indicates the value of α, above which cooperation is so strong that no infection could annihilate the strong Allee effect. The *thin black line* represents the Hopf bifurcation (HB) curve. α_h is the smallest value of α to induce a limit cycle. To its right, predators and prey oscillate if they coexist (*horizontal blue dots*). The bubbling effect is possible in the area *shaded* in *light blue*. The *bold blue line* is the asymptote of the Hopf bifurcation curve as $\beta \to \infty$, with $\alpha \to 19.2$. *Orange color* indicates the parameter region where the disease remains absent: In region En, the prey-only state is the only attractor. In region En/Enp, there is bistability due to cooperation-mediated predator survival for certain initial conditions. In region $En/OCnp$, coexistence is oscillatory due to a limit cycle. Equilibria are labeled according to Table 1. Model (5)-(7), remaining parameter values as in Fig. 2 (Color figure online)

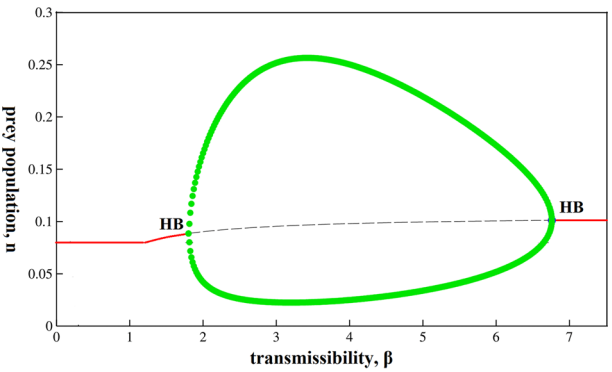

Fig. 4 Bubbling effect: Increasing horizontal disease transmission first destabilizes and then stabilizes the population dynamics of infected predators and prey. Shown is the bifurcation diagram for prey when varying disease transmissibility. Hunting cooperation is fixed at a value that allows predator survival due to a strong Allee effect ($\alpha = 17 > \alpha_c$). The *red line* indicates stable equilibria, and the *dashed black line* indicates unstable equilibria. The *bold green line* indicates the amplitudes of the limit cycles. HB marks Hopf bifurcations. The prey-only equilibrium and other unstable equilibria are not shown. Other parameter values are as in Fig. 3 (Color figure online)

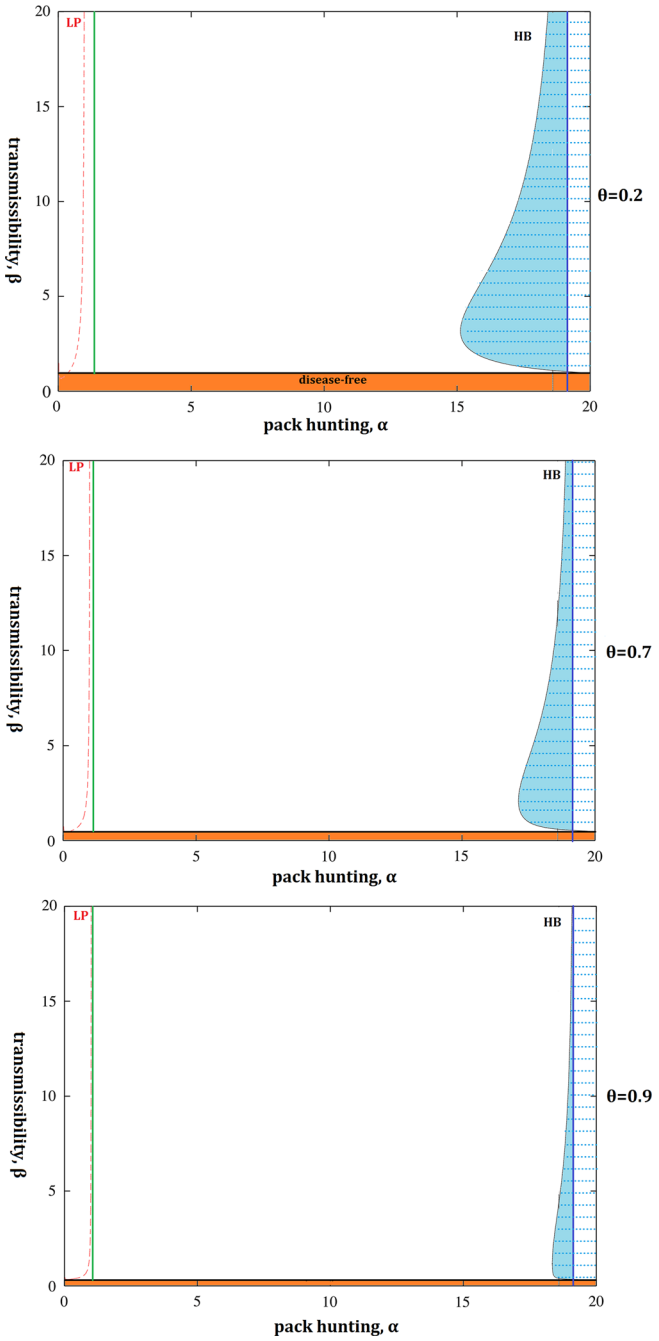

Fig. 5 Effect of vertical transmission: Two-parameter bifurcation diagrams with increasing values of vertical transmission θ. The area with vertical *blue dots* indicates the parameter region with the bubbling effect. Other curves, shadings, and parameter values are as in Fig. 3. *Top panel* $\theta = 0.2$, *middle panel* $\theta = 0.7$, *bottom panel* $\theta = 0.9$ (Color figure online)

Second, with increasing values of θ the limit point bifurcation curve moves closer to the asymptote α_c (green vertical line), and the asymptote itself increases slightly. That is, vertical transmission reduces the parameter region where hunting cooperation induces a strong Allee effect. This makes sense as a higher disease burden due to increased vertical transmission requires stronger pack hunting by the predators to survive.

Third, vertical transmission has a stabilizing effect as the parameter region with limit cycle oscillations shrinks when increasing θ. Moreover, fourth, the parameter region leading to the bubbling effect shrinks as well with increasing vertical transmission.

In the limiting case of complete vertical transmission, $\theta = 1$, the bifurcation diagram shows only the two asymptotes, namely α_c for the limit point bifurcation and the bold blue line for the Hopf bifurcation. In this case, the bubbling effect disappears completely.

5 Discussion and Conclusions

Living in groups can confer benefits to a population, but also promote adverse effects such as increased disease risk. Our model combines both pack hunting and disease transmission in the predator population. If disease transmissibility is high and hunting cooperation low, the impact of the disease prevails in the sense of driving the predator population extinct. However, if hunting cooperation is sufficiently large ($\alpha > \alpha_c$), we observe a different model behavior. In this case, the system is always bistable. That is, independently of the strength of the disease, the group behavior guarantees predator survival for large enough predator populations. Here, it is the pack hunting that prevails as it prevents unconditional predator extinction due to ecological or epidemiological reasons. In some intermediate region, the cooperation level needed to facilitate survival increases with disease transmissibility, as pack hunting needs to compensate disease virulence.

Cooperation-mediated survival is something that our model 'inherits' from the pack hunting model (Teixeira Alves and Hilker 2017). We show that it also occurs in the presence of disease. Note that multiple endemic equilibria, which are characteristic of the strong Allee effect, are not possible in the eco-epidemiological model without predator cooperation (Oliveira and Hilker 2010). Disease-induced host extinction is something that our model inherits from the eco-epidemiological model (Oliveira and Hilker 2010). Disease-induced extinction is typical for frequency-dependent transmission (e.g., Busenberg and van den Driessche 1990; Mena-Lorca and Hethcote 1992; Hilker 2010; Bate and Hilker 2013), which we assume here.

An interesting modification of our model would be to consider density-dependent transmission. We would expect that disease-induced predator extinction will not be possible anymore as disease transmission ceases when the predator population becomes small (cf. Oliveira and Hilker 2010). Furthermore, density-dependent transmission assumes that the larger the host population, the higher the disease transmission. This is another fundamental difference to frequency-dependent transmission and could be especially important in the presence of group behavior. On the one hand, one might therefore expect that increasing levels of hunting cooperation promote disease trans-

mission. On the other hand, the predator population density does not always increase with the level of hunting cooperation. This holds for the system without (Teixeira Alves and Hilker 2017) and with the disease. If the predation pressure due to pack hunting becomes too large, the prey density drops rapidly due to overexploitation and the predator density consequently decreases as well. We can see this also in Fig. 1b, d, where an increase in hunting cooperation leads to a smaller value of the predator density at the stable coexistence equilibrium. In summary, the effect of density-dependent transmission on the system could be mixed. This could also be affected by considering a limit to hunting cooperation as the attack rate cannot increase indefinitely with predator density (cf. Berec 2010).

The analysis in this paper reveals four threshold quantities that help us understand the system dynamics. First, the basic reproduction number of the disease expresses whether or not disease invades. Second, the predator basic reproduction number determines whether or not predators can be sustained in a given ecological and epidemiological setting without hunting cooperation. Third, $\alpha_c(i^*)$ marks the critical value of the hunting cooperation above which there is a strong Allee effect. Fourth, the prevalence reproduction number quantifies the existence of the disease-induced predator extinction state.

Our model considers disease spread in a host population that can be subject to a strong Allee effect. There are related models in the literature, where the strong Allee effect is not induced by pack hunting, but included in the demographics (Hilker et al. 2009; Thieme et al. 2009; Hilker 2009, 2010) or brought about by the coupling of mate finding and sterilizing pathogens (Berec and Maxin 2013). The dynamic behavior found in the current paper (e.g., bistability, disease-induced extinction) matches that found for frequency-dependent transmission in Hilker (2010). In particular, the bifurcation diagram of the infected predator density has a special form, see Fig. 6. The remarkable feature is that the density of infected predators abruptly collapses from

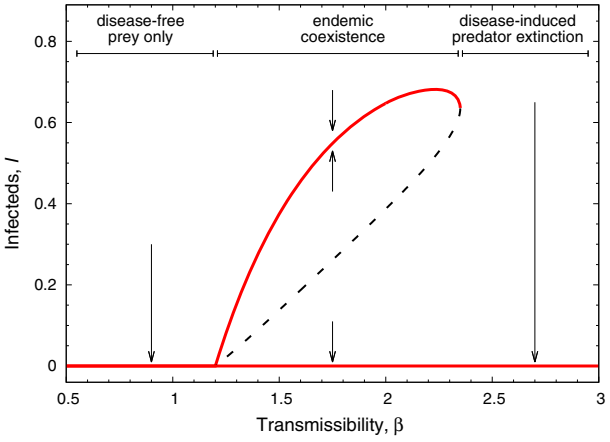

Fig. 6 Bifurcation diagram for the infected predators, $I = pi$ (not to be confused with the disease prevalence in the predator population). Parameter values: $\alpha = 0.7, k = 0.8$. Other parameter values are as in Fig. 1

a rather high value to zero. This happens at the limit point bifurcation and is related to disease-induced extinction, as the entire predator host population disappears. This is fundamentally different from a backward bifurcation, which also involves bistability. However, in the backward bifurcation scenario bistability occurs in the parameter range before disease invasion and there is no disease-induced extinction (cf.Hilker 2010).

Both pack hunting and disease transmission can each induce limit cycle oscillations (Oliveira and Hilker 2010; Teixeira Alves and Hilker 2017). It is therefore not surprising that we find limit cycles also in the combined model considered here. We also observe that disease can be destabilizing (Oliveira and Hilker 2010) but ultimately has a stabilizing effect when transmissibility becomes large (bubbling effect, Hilker and Schmitz 2008; Oliveira and Hilker 2010; Bate and Hilker 2013). Increased pack hunting induces limit cycles, just as in the model without disease (Teixeira Alves and Hilker 2017). In the presence of predator infection, however, the parameter domain of sustained oscillations becomes larger than in the absence of disease. This holds especially for intermediate transmissibilities.

In our simulations, we did not observe homoclinic bifurcations in which sustained oscillations suddenly disappear; however, homoclinic bifurcations could exist for other parameter values that were not explored in our simulations. Homoclinic bifurcations have been observed in the model without disease (Teixeira Alves and Hilker 2017).

Our model includes not only horizontal transmission, but also vertical transmission. We find that vertical transmission promotes disease persistence, requires more hunting cooperation for a strong Allee effect, strengthens the stabilizing effect of disease, and reduces the parameter regions with the bubbling effect.

What we have not considered in this paper is the Allee threshold, i.e., the critical predator density above which predators survive due to the strong Allee effect and below which they go extinct. This critical density is related to the separatrix between different basins of attraction, namely the one of the prey-only state on the one hand and the one of the coexistence state on the other hand. Francomano et al (2016, 2017, in press) have developed an algorithm to numerically approximate the Allee threshold and applied it to the model presented in this paper. Their simulations show that, for large hunting cooperation, the Allee threshold takes rather small densities so that the strong Allee effect is effectively more comparable to a weak Allee effect. This means that the cooperation-mediated survival is even more likely as it is less conditional on a large initial population density.

Our results suggest that strongly cooperating predators are ‘immune’ to disease-induced host extinction. From a wildlife management perspective, maintaining a large population size can be important to promote robustness against epidemics and invading parasites. Endangered populations, however, may be especially at risk when their declining population size cannot provide anymore the cooperation-mediated survival in conditions of disease-induced extinction. In the case of pest control, the introduction of parasites is a form of biological control, e.g., when predators represent an invasive species threatening biodiversity, and the aim is to protect the indigenous prey (Anderson 1982; Courchamp and Sugihara 1999; Bester et al. 2002; Oliveira and Hilker 2010). In this context, two conclusions seem important. First, biological control may not be successful in eradicating a predator pest that is very cooperative. Second, for

this reason it appears even more imperative to detect and control invasive species as early as possible (Vander Zanden et al. 2010) and to complement biological control by other measure to reduce pest population size (Numfor et al. 2017).

Appendix A: Isocline Analysis in the Endemic Predator–Prey Phase Plane

We perform an isocline analysis of the three-dimensional model (5)–(7), which will lead us to a reduced model in the two-dimensional phase plane. Any nontrivial equilibrium satisfies the following zero-growth conditions

$$\text{prey nullcline: } n = k\left[1 - \frac{1+\alpha p}{r}p\right], \tag{10}$$

$$\text{predator nullcline: } n = \frac{1+\mu i}{1+\alpha p}, \tag{11}$$

$$\text{prevalence nullcline: } i = 1 - \frac{(1+\alpha p)(1-\theta)}{\beta - \mu}n. \tag{12}$$

Substituting the value of n from equation (11) into equation (12), we find the nontrivial prevalence value at any equilibrium with $p^* > 0$

$$i^* = \frac{\beta - \mu - (1-\theta)}{\beta - \mu\theta},$$

which is exactly expression (9) shown in the main text.

We can now intersect the three-dimensional state space with the prevalence nullplane $i = i^*$ and work on the (p, n) plane, where $p > 0$ and $n > 0$. This simplifies the analysis because it allows us to find the intersection of curves (10) and (11) with $i = i^*$ constant (see Fig. 1).

The parabola (10) intersects the n–axis at the point $(0, k)$, while the hyperbola (11) intersects the n–axis at the point $(0, 1 + \mu i^*)$. That is, if the vertical intercept of the parabola (solid red dashed curve in Fig. 1) is higher than the vertical intercept of the hyperbola (dashed blue curve in Fig. 1), then there is a unique intersection of the parabola and hyperbola (Fig. 1b, d). This condition can be expressed as $k > 1 + \mu i^*$ or equivalently as $\mathcal{R}_p(i^*) > 1$. If $\mathcal{R}_p(i^*) < 1$, there can be 0, 1, or 2 intersections (Fig. 1a, c).

Appendix B: Existence and Stability of the Stationary States

To analyze the equilibrium point, we consider the Jacobian of system (5)–(7):

$$J = \begin{pmatrix} r\left(1 - \frac{2n}{k}\right) - p\alpha' & -p'n & 0 \\ \alpha' p & -(1+\mu i) + p'n & -\mu p \\ -\alpha'\theta' i & -\alpha\theta' n i & b'(1-2i) - \alpha'\theta' n \end{pmatrix},$$

where $b' = \beta - \mu$, $\theta' = 1 - \theta$, $\alpha' = 1 + \alpha p$, and $p' = 1 + 2\alpha p$. The system has the following possible equilibria:

1. $E_0 = (0, 0, 0)$. The trivial extinction state always exists and is always unstable, since the eigenvalues of the Jacobian evaluated at this equilibrium are

$$\lambda_1 = r > 0, \quad \lambda_2 = -1 + k, \quad \lambda_3 = \beta - \mu - (1 - \theta)k.$$

2. $E_{\mathrm{n}} = (k, 0, 0)$. This represents the disease- and predator-free equilibrium with the prey being at carrying capacity. It always exists and its eigenvalues are

$$\lambda_1 = -r < 0, \quad \lambda_2 = -1 + k, \quad \lambda_3 = \beta - \mu - (1 - \theta)k.$$

   Hence, E_{n} is stable if

$$k < 1, \quad \mathcal{R}_i = \frac{\beta + \theta k}{\mu + k} < 1.$$

3. $E_i = (0, 0, 1)$. This is the disease-induced extinction state with both predators and prey being absent. It always exists, but it is always unstable because its eigenvalues are:

$$\lambda_1 = r > 0, \quad \lambda_2 = -1 + \mu, \quad \lambda_3 = \beta - \mu - (1 - \theta)k.$$

4. $E_{\mathrm{ni}} = (k, 0, i^\dagger)$, where $i^\dagger = 1 - \frac{(1-\theta)k}{\beta - \mu}$. This is the state corresponding to disease-induced predator extinction, with the prey reaching carrying capacity. It exists if

$$\mathcal{R}_i = \frac{\beta + \theta k}{\mu + k} > 1.$$

   The eigenvalues are

$$\lambda_1 = -r, \quad \lambda_2 = k - (1 + \mu i^\dagger), \quad \lambda_3 = (\beta - \mu)(1 - 2i^\dagger) - (1 - \theta)k.$$

   That is, E_{ni} is stable if $k < 1 + \mu i^\dagger$, which translates into $\mathcal{R}_p(i^*) < 1$.
5. $E_{\mathrm{np}} = (n^\circ, p^\circ, 0)$ is the disease-free coexistence state of predators and prey. The values of n° and p° are cumbersome to obtain. If $k > 1$, E_{np} is unique. If $k < 1$, there can be up to two equilibria E_{np} which (dis-)appear in a saddle–node bifurcation in the disease-free plane (see Teixeira Alves and Hilker 2017). The stability of E_{np} is investigated numerically and discussed in the main text.
6. $E_{\mathrm{npi}} = (n^*, p^*, i^*)$, with $i^* = [\beta - \mu - (1 - \theta)](\beta - \mu\theta)^{-1}$ as shown in Appendix A. This equilibrium is the endemic coexistence state where all three species (prey, predators and disease) coexist. As for the disease-free coexistence equilibrium, we cannot find the explicit values of the prey and predator values. From $i^* > 0$, we obtain that a necessary existence condition for E_{npi} is

$$\mathcal{R}_0(0, 1) = \frac{\beta + \theta}{1 + \mu} > 1.$$

In Sect. 3, we find that if $\mathcal{R}_p(i^*) > 1$ the equilibrium exists and is unique and stable. If $\mathcal{R}_p(i^*) < 1$, there can be two, one or no equilibrium point E_{npi}. We study the existence and stability of E_{npi} numerically in the main text and show that there is a saddle–node bifurcation when varying α.

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