



Eco-epidemiological interactions with predator interference and infection

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ABSTRACT

Predator interference is a form of competition between predator individuals over access to their prey. There is broad empirical evidence for interference to exist in different strengths in various types of ecological communities. At the same time, parasites are increasingly recognized to alter food web structure and dynamics. In order to investigate the eco-epidemiological interplay between interference and infection, we develop and analyze mathematical models of a predator–prey system, where the predators are subject to both interference and infectious disease. In the absence of infection, equilibrium predator density is known to show a non-monotonic response to interference by first increasing and then decreasing with increasing interference levels. We show that predator infection can change this pattern into a monotonically decreasing predator response to interference, provided the transmissibility is large enough and the pathogenicity is moderate such that the impact of disease on host population density prevails over interference effects. This holds for both types of disease transmission studied here, density-dependent and frequency-dependent. For density-dependent transmission, we find that intermediate values of interference can facilitate disease persistence, whereas the disease would disappear for small or large interference levels. By contrast, for frequency-dependent transmission, disease emergence is independent of interference levels. These dynamic interactions may be important for the understanding of potential biocontrol measures and of spread patterns of zoonotic diseases.

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1. Introduction

Parasites affect not only their host population but also species with which their hosts interact directly or indirectly (Anderson and May, 1986; Hatcher and Dunn, 2011; Sieber and Hilker, 2011). For example, in biological control parasites are introduced into populations of invasive predators with the aim of protecting endangered prey (e.g. controlling cats on Marion Island to rescue sea birds; Courchamp et al., 1999; Nogales et al., 2004). Similarly, predators can be used in population management to control the emergence of infectious diseases in prey hosts (Packer et al., 2003). As an example, predators can reduce rodent populations below critical levels for zoonotic diseases such as Lyme disease and hantavirus (Ostfeld and Holt, 2004).

Much of ecological food web theory rests on predator–prey interactions with so called prey-dependent functional responses

(Arditi and Ginzburg, 1989), i.e. the consumption rate per individual predator is assumed to depend only on prey density. Hassell and Varley (1969) showcased that a functional response decreases with the density of predators, and by now there is compelling empirical evidence for predator-dependent functional responses (e.g. Skalski and Gilliam, 2001; Kratina et al., 2009; DeLong and Vasseur, 2011; Zimmermann et al., 2015; Novak et al., 2017), the degree of which may depend on species and ecosystems. Many mechanisms have been proposed that can lead to a predator-dependent functional response, e.g. social interactions amongst predators (Abrams and Ginzburg, 2000), aggressive behavior resulting from population structure and cannibalism (Crowley and Martin, 1989; Rudolf, 2007), spatial aggregation of predators (Cosner et al., 1999), hunting cooperation (Teixeira Alves and Hilker, 2017), and intensified anti-predator behavior of the prey (Crowley and Martin, 1989; Lima, 2009).

If the functional response decreases (respectively increases) with predator density, there is predator interference (respectively facilitation). The dynamic interplay of predator facilitation and transmission of a pathogenic predator disease has recently been

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modeled mathematically by Hilker et al. (2017). They considered that facilitation was brought about by hunting cooperation and found the following distinction in outcomes. If predator cooperation is low and disease transmissibility is high, then the impact of infection in the sense of driving the host population extinct prevails over cooperation. However, if cooperation is strong enough, predator facilitation can mediate predator survival independently of the strength of the disease. That is, for invasive predators that cooperate strongly, biocontrol by releasing parasites alone may not be sufficient.

In this paper, we investigate the impact of predator interference on predator–prey systems with infectious diseases circulating in the predator population. Interference is a form of competition between individuals that occurs when the access to resources (e.g., prey) is reduced by aggressive or passive interactions with other individuals. This is sometimes called foraging interference or, if interference occurs between conspecifics, mutual interference. Here we will focus on the dynamic feedbacks caused by predator mutual interference and infection. One might expect that interference reduces predator population density—and thus also impedes density-dependent disease transmission. However, the impact of interference is not as simple as that. In basic predator–prey models, the predator population density at equilibrium increases with interference, provided that interference is small. This has been shown numerically (for the Beddington–DeAngelis functional response and logistic prey growth; DeAngelis et al., 1975) and analytically (for the Hassell–Varley and Beddington–DeAngelis functional responses and exponential prey growth; Arditi et al., 2004). By contrast, if interference is large, predator population density at equilibrium decreases with interference. The reason is that prey population density at equilibrium increases with interference (DeAngelis et al., 1975; Arditi et al., 2004); more food resources may thus supercompensate direct predator losses due to interference.

The interaction between interference and infection is therefore far from trivial—especially because disease itself can also regulate predator density and cascade to the prey level (Oliveira and Hilker, 2010; Hatcher and Dunn, 2011). We will consider two types of disease transmission, namely frequency-dependent transmission (FDT) and density-dependent transmission (DDT). Contact rates are independent of host population density in the former and proportional to host population density in the latter. These are two extremes in a continuum of possibilities, and the truth for many species may be somewhere in the middle.

The paper is organized as follows. In Section 2, we introduce our eco-epidemiological models. The functional responses are derived using the approach of “wasting times”, similarly to Beddington (1975). We will assume that there is no difference between susceptible and infected predators and that there is no prey handling time, so that we can focus on the impact of only predator interference.

Section 3 presents results from equilibrium and linear stability analysis, which we will complement by numerical simulations. In Section 4 we discuss our results and draw our conclusions.

2. Model description

2.1. Eco-epidemiological model

Consider a predator–prey system with an infectious disease in the predator population. Let X , S , and I be the population densities of prey, susceptible predators, and infected predators,

respectively, and τ be time. The mathematical model reads

$$\begin{aligned} \frac{dX}{d\tau} &= r \left(1 - \frac{X}{K} \right) X - f_S(S, I, X)S - f_I(S, I, X)I, \\ \frac{dS}{d\tau} &= -mS - \beta(S, I) + e_S f_S(S, I, X)S + (1 - \nu) e_I f_I(S, I, X)I, \\ \frac{dI}{d\tau} &= -mI - \mu I + \beta(S, I) + \nu e_I f_I(S, I, X)I, \end{aligned} \quad (1)$$

where $f_S(S, I, X)$ and $f_I(S, I, X)$ are generalizations of the Beddington–DeAngelis functional response extended to multiple predators types. They are derived in Appendix A and given by

$$\begin{aligned} f_S(S, I, X) &= \frac{a_S X}{1 + a_S h_S X + b_{SS} w_{SS} S + b_{IS} w_{IS} I}, \\ f_I(S, I, X) &= \frac{a_I X}{1 + a_I h_I X + b_{SI} w_{SI} S + b_{II} w_{II} I}, \end{aligned} \quad (2)$$

where a_S and a_I are the search rates and h_S and h_I the prey handling times of susceptible and infected predators, respectively. b_{SS} is the encounter rate between susceptible predators, b_{II} between infected predators, b_{SI} between susceptible and infected predators, and b_{IS} between infected and susceptible predators. w_{SS} , w_{II} , w_{SI} , and w_{IS} are the respective wasting times during these encounters.

The prey population grows logistically with intrinsic per-capita growth rate r and carrying capacity K . The prey is consumed by both susceptible and infected predators. The conversion efficiencies of susceptible and infected predators are e_S and e_I , respectively. The model accounts for vertical transmission of the predator disease: $\nu \in [0, 1]$ is the fraction of offspring that get infected from the parent. For $\nu = 0$, there is no vertical transmission while for $\nu = 1$ there is complete vertical transmission.

The predators are specialists and they have a natural per-capita death rate m . Infected predators have an additional disease-related per-capita mortality μ , henceforth called pathogenicity. There is no recovery from the disease. Function $\beta(S, I)$ describes horizontal disease transmission. In the case of density-dependent transmission (DDT),

$$\beta(S, I) = \beta SI,$$

and in the case of frequency-dependent transmission (FDT),

$$\beta(S, I) = \frac{\beta SI}{S + I}.$$

Note that the transmission parameter β has different dimensions for DDT and FDT.

2.2. Simplifying assumptions and non-dimensionalization

Due to the large number of parameters, we will make some simplifying assumptions. We start by assuming that susceptible and infected predators do not differ in their wasting times, handling times, encounter rates, and conversion efficiencies. This assumption may be well justified if the infection does not change predator behavior. We also assume that the predator encounter rates equal the rates with which predators find prey; this is likely to be the case unless predators detect each other at different distances than they detect prey (Turchin, 2003). More specifically:

$$\begin{aligned} w &:= w_{SS} = w_{IS} = w_{SI} = w_{II}, \\ a &:= b_{SS} = b_{IS} = b_{SI} = b_{II}, \\ h &:= h_S = h_I, a := a_S = a_I, e := e_S = e_I. \end{aligned}$$

In this case, susceptible and infected predators have the same functional response, and both $f_S(S, I, X)$ and $f_I(S, I, X)$ simplify to

the Beddington–DeAngelis functional response

$$f(X, Y) = \frac{aX}{1 + ahX + awY}. \tag{3}$$

If we additionally assume that the handling times are negligible ($h = 0$), then the functional response further simplifies to

$$f(X, Y) = \frac{aX}{1 + awY}. \tag{4}$$

This functional response has also been derived by [Beddington \(1975\)](#); see also [Turchin \(2003, pp. 84\)](#). It has been used before to focus on the effect of predator interference rather than on the effect of prey handling times ([Bate and Hilker, 2012](#)). We shall also consider it in the remainder of this paper. We have done some analysis and simulations also for the full Beddington–DeAngelis functional response (3), but as we did not find significant differences in our results, we chose to focus on the simpler version.

Before analyzing model (1) with functional response (4), we apply a coordinate transformation and nondimensionalize the equations. We substitute S and I by the total predator population density $Y = S + I$ and the prevalence of predator infection, $i = I/Y$. Then we can write the system (1), (4) as

$$\begin{aligned} \frac{dX}{d\tau} &= r \left(1 - \frac{X}{K} \right) X - \frac{aXY}{1 + awY}, \\ \frac{dY}{d\tau} &= -mY - \mu Yi + \frac{eaXY}{1 + awY}, \\ \frac{di}{d\tau} &= i \left(-\mu(i - 1) + \beta(i, Y) - \frac{eaX(1 - \nu)}{1 + awY} \right), \end{aligned} \tag{5}$$

where

$$\begin{aligned} \beta(i, Y) &= \beta i(1 - i)Y \quad \text{for FDT and} \\ \beta(i, Y) &= \beta i(1 - i)Y^2 \quad \text{for DDT.} \end{aligned}$$

We now nondimensionalize system (5) by applying the substitutions

$$N(t) = \frac{1}{K}X(\tau), \quad P(t) = \frac{a}{r}Y(\tau) \quad \text{and} \quad t = r\tau.$$

Defining

$$\begin{aligned} \frac{Kea}{r} &:= \tilde{a}, \quad \frac{m}{Kea} := \tilde{m}, \quad \frac{\mu}{Kea} := \tilde{\mu}, \quad \frac{\beta}{Kea} := \tilde{\beta}, \\ \text{and } rw &:= \tilde{w}, \end{aligned}$$

we get (for simplicity of notation, we will drop the tildes)

$$\begin{aligned} \frac{dN}{dt} &= (1 - N)N - \frac{NP}{1 + wP}, \\ \frac{dP}{dt} &= a \left(-mP - \mu Pi + \frac{NP}{1 + wP} \right), \\ \frac{di}{dt} &= ia \left((\beta - \mu)(1 - i) - \frac{N(1 - \nu)}{1 + wP} \right), \end{aligned} \tag{6}$$

for FDT, while for DDT we get

$$\begin{aligned} \frac{dN}{dt} &= (1 - N)N - \frac{NP}{1 + wP}, \\ \frac{dP}{dt} &= a \left(-mP - \mu Pi + \frac{NP}{1 + wP} \right), \\ \frac{di}{dt} &= ia \left((\beta P - \mu)(1 - i) - \frac{N(1 - \nu)}{1 + wP} \right). \end{aligned} \tag{7}$$

Table 1

Summary of equilibria and stability of the FDT model (6) with $h = 0$. * indicates when equilibrium values of P , N , or i are different from zero. See [Appendix B](#) for details and the definitions of A , B , and C . The reproduction numbers are given in the main text.

| $E = (N, P, i)$ | Feasibility conditions | Stability conditions |
|-------------------|--|---------------------------------|
| $E_0 = (0, 0, 0)$ | Always feasible | Unstable |
| $E_1 = (*, 0, 0)$ | Always feasible | $R_{0,0}^P < 1$ and $R_0^i < 1$ |
| $E_2 = (*, *, *)$ | $\beta - \mu > 0$ | $R_{0,i}^P < 1$ and $R_0^i > 1$ |
| $E_3 = (*, *, 0)$ | $R_{0,0}^P > 1$ | $R_{0,FDT} < 1$ |
| $E_4 = (0, 0, *)$ | Always feasible | Unstable |
| $E_* = (*, *, *)$ | $N_* > 0, i_* > 0,$ $A < 0$ and B or $C < 0$ or $A > 0$ and B or $C > 0$ | Routh–Hurwitz conditions |

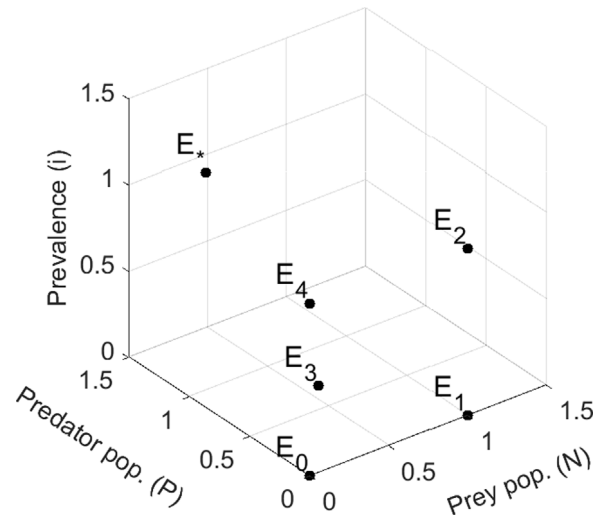


Fig. 1. Summary of the feasible and stable equilibria of the FDT model (6).

3. Results

3.1. Equilibria and stability analysis for FDT

In this section, we summarize the main results of the steady state and stability analysis of the FDT model (6). See [Table 1](#) and [Fig. 1](#) for an overview and [Appendix B](#) for details. We distinguish the following nontrivial cases.

(i) Prey-only case The equilibrium where the disease cannot establish and the predators go extinct while the prey reach their carrying capacity (in this case equal to 1 since the model was scaled) is $E_1 = (N_1 = 1, P_1 = 0, i_1 = 0)$. It is always feasible. It is stable when the following two conditions are satisfied. The first condition describes that the predators cannot survive on the prey population and is given by

$$R_{0,0}^P = \frac{1}{m} < 1. \tag{8}$$

$R_{0,0}^P$ is the ecological basic (indicated by the first 0 in the subscript) reproduction number of the predator population (indicated by the superscript) in the absence of infection (indicated by the second 0 in the subscript). The numerator is the (scaled) average offspring produced by a single predator during its lifetime when introduced into a prey population at carrying capacity (in dimensional terms this is eaK). The denominator is the scaled per-capita death rate and is the reciprocal of the average predator lifetime.

If $R_{0,0}^P < 1$, the ecological conditions are such that the predators cannot sustain themselves on the prey and go extinct.

The second stability condition describes that predator extinction is not due to infection. It is given by

$$R_0^i = \frac{\beta - \mu}{1 - \nu} < 1.$$

This can be interpreted as the prevalence reproduction number (Hilker and Schmitz, 2008; Oliveira and Hilker, 2010), as indicated by the i in the superscript. If $R_0^i < 1$, the infection dies out in the predator population before the predators go extinct. If $R_0^i > 1$, the infection remains prevalent in the limit process as the predators approach extinction. This leads to the next case.

(ii) Disease-induced predator extinction The equilibrium where predators go extinct due to the disease and the prey reach their carrying capacity is

$$E_2 = (1, 0, i_2), \quad i_2 = \frac{\beta - \mu - (1 - \nu)}{\beta - \mu}.$$

The strictly positive prevalence means that, as the predator density approaches zero in the extinction process, the prevalence approaches the value i_2 . That is, the infection sticks around in the extinction process and drives it forward. In particular, the infection does not go extinct before the predators do. E_2 is feasible if $\beta - \mu > 1 - \nu$. There are two stability conditions. The first one is

$$R_{0,i}^P = \frac{1}{m + \mu i_2} < 1.$$

$R_{0,i}^P$ is the ecological basic reproduction number of the predators in the presence of disease (indicated by the i in the second subscript position). In comparison with $R_{0,0}^P$, the difference is the shortened average predator lifetime due to pathogenicity, which affects a proportion i_2 of the predator population. If $R_{0,i}^P < 1$, the predator population goes extinct in the presence of disease. The second condition is $R_0^i > 1$, which guarantees that the infection prevalence is positive as the predator population vanishes. Note that disease-induced predator extinction is independent of the interference strength w .

In the case of complete vertical transmission, $\nu = 1$, we have $E_2 = (1, 0, 1)$ and the feasibility condition is not needed anymore.

(iii) Disease-free predator–prey case In the absence of disease, the equilibrium where both prey and predators coexist is

$$E_3 = \left(m(1 + wP_3), \frac{-(m-1)w - mw - 1 + \sqrt{4mw + (w-1)^2}}{2mw^2}, 0 \right). \tag{9}$$

It is feasible if $R_0^i > 1$. As E_1 is unstable if $R_0^i > 1$, this suggests the possibility of a transcritical bifurcation between these two equilibria. E_3 is stable if

$$R_{0,FDT} = \frac{\beta + \nu m}{m + \mu} < 1.$$

$R_{0,FDT}$ is the basic reproduction number of the disease in the predator population. It 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_3 and available prey N_3 ,

Table 2

Summary of equilibria and stability of the DDT model (7) with $h = 0$. * indicates when equilibrium values of P , N , or i are different from zero. See Appendix C for details. The reproduction numbers are given in the main text.

| $E = (N, P, i)$ | Feasibility conditions | Stability conditions |
|-------------------|------------------------|--------------------------|
| $E_0 = (0, 0, 0)$ | Always feasible | Unstable |
| $E_1 = (*, 0, 0)$ | Always feasible | $R_{0,0}^P < 1$ |
| $E_2 = (*, 0, *)$ | $\nu = 1$ | Unstable |
| $E_3 = (*, *, 0)$ | $R_{0,0}^P > 1$ | $R_{0,DDT} < 1$ |
| $E_4 = (0, 0, *)$ | Always feasible | Unstable |
| $E_* = (*, *, *)$ | $N_* > 0, i_* > 0$ | Routh–Hurwitz conditions |

assuming that predators and prey are at these equilibrium densities. The mean infectious period of an infected predator is the reciprocal of $m + \mu$, 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, νm .

(iv) Endemic coexistence case The equilibrium where predators and prey coexist and the disease is endemic in the predator population is given by

$$E_* = \left(\frac{1 + (w-1)P_*}{1 + wP_*}, P_*, \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu} \right),$$

where P_* is the positive root of a second-degree polynomial. See Appendix B for details, including feasibility and stability conditions.

It is noteworthy that the prevalence at E_* does not depend on the interference parameter w , i.e., $\partial i_*/\partial w = 0$. By contrast, the equilibrium prevalence decreases with increasing pathogenicity μ , $\partial i_*/\partial \mu < 0$. Finally,

$$\begin{aligned} \frac{\partial i_*}{\partial \beta} &> 0 \quad \text{for } \mu > m, \\ \frac{\partial i_*}{\partial \beta} &< 0 \quad \text{for } \mu < m. \end{aligned}$$

That is, with increasing disease transmissibility β the prevalence can increase or decrease, depending on the strength of the pathogenicity relative to the natural mortality.

A similar analysis of the impact of the infection and interference parameters on N_* and P_* is algebraically too involved, which is why we will investigate this numerically in Section 3.4.

3.2. Equilibria and stability analysis for DDT

Now we summarize the main results for the DDT model (7). See Table 2 for an overview and Appendix C for details. We distinguish the following four cases.

(i) Prey-only case The equilibrium E_1 where the predators go extinct while the prey reach their carrying capacity is $E_1 = (1, 0, 0)$. It is always feasible. It is stable if $R_{0,0}^P < 1$, where $R_{0,0}^P$ is the predator basic reproduction number in the absence of infection and is the same as in (8). In contrast to FDT, there is only one stability criterion. This is because disease-induced predator extinction is impossible for DDT (density-dependent transmission vanishes when the host population density approaches zero)—unless there is complete vertical transmission; see the next case.

(ii) Disease-induced predator extinction The disease-induced extinction state E_2 is only feasible for complete vertical

transmission, i.e., $\nu = 1$. In this case, $E_2 = (1, 0, 1)$, and it is always unstable.

- (iii) **Disease-free predator–prey case** The disease-free equilibrium E_3 where both prey and predators coexist is the same as in the FDT case, see (9). While the feasibility condition is also the same as for FDT, the stability condition is different and given by

$$R_{0,DDT} = \frac{\beta P_3 + \nu m}{m + \mu} < 1.$$

$R_{0,DDT}$ is the basic reproduction number of the disease for DDT. In comparison to $R_{0,FDT}$, the difference is that the secondary infections due to horizontal transmission depend on the predator equilibrium density P_3 , given in (9). Note that $R_{0,DDT}$ thus depends on the interference parameter w , whereas $R_{0,FDT}$ is not affected by interference.

- (iv) **Endemic coexistence case** The equilibrium E_* where prey, predators, and disease coexist is given by

$$E_* = \left(\frac{1 + (w - 1)P_*}{1 + wP_*}, P_*, \frac{\beta P_* - \mu - m(1 - \nu)}{\beta P_* - \mu \nu} \right),$$

where P_* is the positive root of a third-degree polynomial. See Appendix C for details, including feasibility and stability conditions. Note that the equilibrium prevalence depends on P_* . This means that for DDT the equilibrium prevalence does depend on the interference parameter w . This is different from FDT, for which the equilibrium prevalence is independent of predator interference.

3.3. Comparison between the models with FDT and DDT

In the absence of disease, the difference between FDT and DDT obviously does not matter. This implies that the ecological basic reproduction number of the predators (in the absence of disease) is the same for FDT and DDT. We note the following differences between the models with FDT and DDT.

- (a) The basic reproduction number of the disease differs between FDT and DDT, because the number of secondary infections depends on the predator population density for DDT. Consequently, the spread of predator diseases can be affected by predator interference due to its effect on population density. This is clearly reflected by the analytical equilibrium infection prevalence, which depends on predator density and thus on interference for DDT, but is independent of either for FDT.
- (b) The disease-induced host extinction state E_2 exists for DDT only for the particular case of complete vertical transmission and is always unstable. This is because disease transmission vanishes when the host population density approaches small enough values. For FDT, by contrast, disease transmission goes on even at small host population densities, which is why the disease-induced extinction state E_2 is feasible and stable in a certain parameter range. This is a well-known difference between FDT and DDT (e.g. Getz and Pickering, 1983; Zhou and Hethcote, 1994; Hilker et al., 2009; Oliveira and Hilker, 2010).
- (c) Another difference concerns the analytical tractability of the endemic coexistence equilibria. For FDT, we could find analytical expressions for N_* , P_* , and i_* . For DDT, by contrast, P_* is the positive root of a third-degree polynomial, and the expressions of N_* and i_* depend on it.

3.4. Numerical simulations

We now investigate the combined interplay of predator interference w and transmissibility β by varying their parameter values simultaneously. We will also analyze how the population density levels at equilibrium behave when the two varying parameters are predator interference w and pathogenicity μ . We will first focus on population density levels at equilibrium and then on (de-)stabilization brought about by infection and interference.

3.4.1. Population densities at equilibrium

Fig. 2 shows the asymptotic population densities and infection prevalence when the system has reached equilibrium. In the absence of disease (left of dashed curves; $R_{0,FDT} < 1$ and $R_{0,DDT} < 1$), predators and prey coexist at stable equilibria. For a fixed value of w in this parameter region, the asymptotic population densities of predators and prey naturally do not change when varying β . For a fixed value of β in the disease-free region, the asymptotic predator population density increases (respectively decreases) with increasing w when interference is low (respectively high). The asymptotic prey population density always increases with w . These asymptotic behaviors are the same for FDT and DDT.

Disease invasion into the predator–prey state is independent of predator interference for FDT and occurs for a fixed value of β ($R_{0,FDT} = 1$; vertical dashed line). For DDT, the disease invasion threshold depends on both β and w ($R_{0,DDT} = 1$; dashed curve). In particular, for a fixed value of β around 1.5, the disease dies out for small w , establishes for intermediate w , and dies out again for large w .

When the disease is endemic and predators and prey coexist (right-hand side of the dashed curves in Fig. 2), the impact of w on the asymptotic predator density translates from the disease-free into the endemic case, provided the transmissibility is not too large. That is, for intermediate ranges of β , the asymptotic predator densities initially increase and then decrease with w . See Fig. 3 for a zoomed-in diagram. For large values of β , by contrast, the asymptotic predator densities always decrease with w . The asymptotic prey densities also always increase with w , just as in the disease-free case. This holds for both FDT and DDT.

When increasing β in the endemic parameter region, asymptotic prey densities monotonically increase and asymptotic predator densities monotonically decrease. This holds again for both FDT and DDT. The asymptotic infection prevalence increases monotonically with β . For FDT, the prevalence is independent of w . For DDT, the infection prevalence can initially increase but then decreases with w , provided that transmissibility is low. For larger transmissibilities, the prevalence always decreases with w .

For FDT disease-induced extinction occurs for large values of β , independently of w (solid red line in Fig. 2). This is not possible for DDT.

In Fig. 4, we vary the pathogenicity μ rather than the disease transmissibility. The maximum depression of equilibrium predator host population size occurs for moderate values of μ . This holds for FDT and DDT (Figs. 4c and d, respectively). Small values of μ have little effect on the host population size, whereas large values of μ cause the eradication of the infection (Figs. 4e, f). This happens because infected individuals die more quickly than they can spread the disease. As a consequence, the equilibrium predator population density increases with large values of μ . Hence, low to moderate levels of μ are most effective in depressing host population size. In parameter regions where the predator hosts are less abundant, the prey population increases in size and vice versa (Figs. 4a, b).

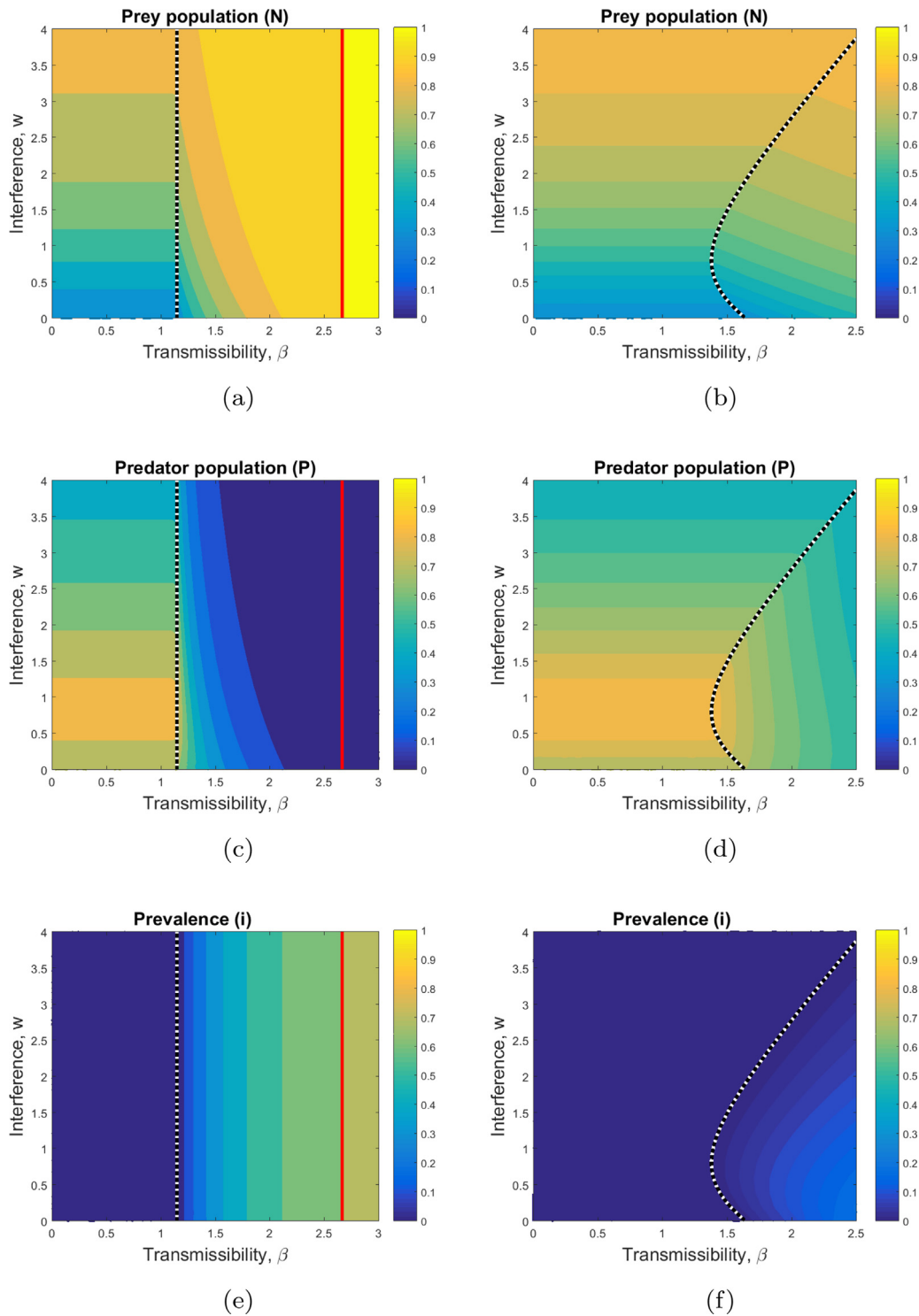


Fig. 2. Long-term values of the prey (top row, panels (a) and (b)), predators (middle row, panels (c) and (d)), and predator infection prevalence (bottom row, panels (e) and (f)) in the FDT (left column) and DDT (right column) model, obtained from numerical solutions of (6) and (7), respectively. On the left-hand side of the dashed black–white line ($R_{0,FDT} = 1$ and $R_{0,DDT} = 1$) the system is at the disease-free predator–prey equilibrium. Disease-induced predator extinction is possible only in the FDT model and occurs on the right-hand side of the solid red line ($R_{0,i}^p = 1$). Parameter values $a = 5.0$, $m = 0.3$, $\mu = 1$, and $\nu = 0.5$. Initial conditions: $N(0) = 1$, $P(0) = 1$, $i(0) = 0.6$.

When the predator infection is endemic, increasing interference can have two different qualitative effects on the equilibrium predator host density (Fig. 4c, d). For moderate values of μ , increasing interference always decreases the equilibrium predator density. For very small and large values of μ (approximately $0 < \mu \lesssim 0.1$ and shortly before the disease persistence threshold

shown in dashed line, respectively), the equilibrium predator density exhibits a non-monotonic response to interference by first increasing and then decreasing with w . In comparison to Fig. 2, where we varied transmissibility β , there are now two parameter regions with a non-monotonic host density response to interference; one of these regions is again close to the disease

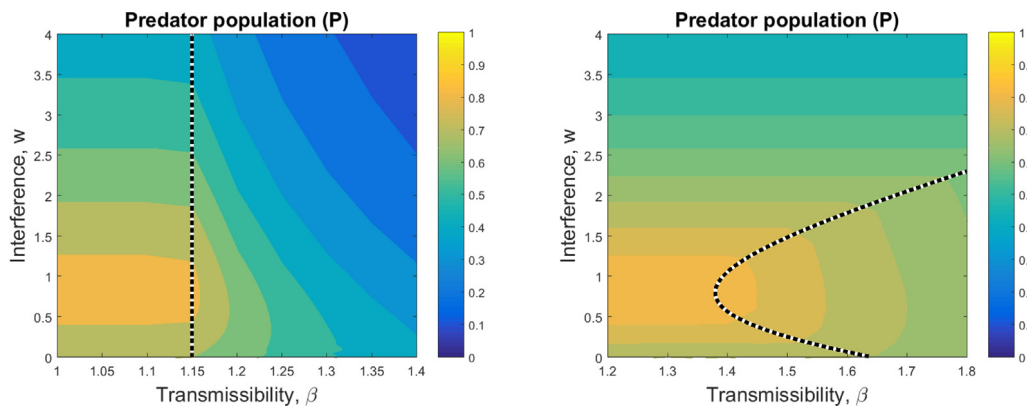


Fig. 3. A zoomed-in version of the asymptotic predator densities from Fig. 2, on the left-hand side of the frequency-dependent model as in Fig. 2 panel (c) and on the right-hand side of the density-dependent model as in Fig. 2 panel (d).

persistence threshold, and it is separated from the other one at very small values of μ by a wide parameter region with a monotonically decreasing predator response to interference.

The response of equilibrium prey density and predator infection prevalence to increasing interference is the same as in Fig. 2. That is, the equilibrium prey density monotonically decreases with w (Fig. 4a, b). In the case of FDT, prevalence is independent of w (Fig. 4e). In the case of DDT, there can be a transition of the disease being absent–present–absent with increasing w (Fig. 4f).

3.4.2. (De-)stabilizing effects

The disease-free predator–prey system corresponds to the Lotka–Volterra model with prey self-regulation. In the absence of disease ($R_{0,FDT} < 1$ or $R_{0,DDT} < 1$), the predator–prey coexistence equilibrium is therefore always globally asymptotically stable, if it exists. Upon disease invasion, the predator infection can first destabilize and then stabilize the system with increasing disease transmissibility. This is illustrated in Fig. 5 for both the FDT and DDT model. Increasing disease transmissibility induces a Hopf bifurcation if transmissibility is intermediate. As a consequence, the nontrivial equilibrium of prey and susceptible as well as infected predators gets destabilized, and limit cycles emerge. For a larger transmissibility value, however, there is another Hopf bifurcation, in which the limit cycles oscillations disappear and the nontrivial equilibrium gets stabilized. Hence, there is parameter region with intermediate transmissibility values leading to dynamic instability (hatched areas in Fig. 5).

With increasing interference strength, the parameter region of instability becomes smaller. If the system exhibits limit cycles, they disappear in a Hopf bifurcation for increasing interference values. If predator interference is sufficiently strong, no limit cycles occur for all transmissibility values investigated (Fig. 5).

4. Discussion and conclusions

In this paper, we have developed and analyzed dynamic eco-epidemiological models to investigate the mutual feedbacks between predator interference and disease transmission in the predator population. Let us first discuss the impact of interference on the spread of predator diseases. Whether or not there is an effect, depends on the type of disease transmission. For FDT, we have shown analytically that interference influences neither disease emergence, nor equilibrium prevalence levels, nor disease-induced host extinction. This is plausible because contact rates in FDT are assumed to be independent of host population density and thus do not depend on interference.

For DDT, by contrast, interference affects the basic reproduction number and thus disease invasion. One major finding of our

numerical experiments is that the transmissibility required for disease emergence is lowest for intermediate interference values. The highest pathogenicity level that can sustain an endemic infection occurs also for intermediate interference values. For smaller and larger interference values, disease emergence requires larger transmissibilities or lower pathogenicities. Similarly, asymptotic predator infection prevalences in our simulations are highest for intermediate interference values.

In other words, our results suggest that disease invasion can be facilitated by intermediate interference levels in the case of DDT. That is, ecological systems that favor low or high levels of interference are more likely to remain disease-free. This may be important for the understanding and prevention of epizootics and spillovers from zoonotic diseases. For example, if the aim is to avoid disease emergence, management actions could take this into account, e.g. by distributing (alternative) resources or designing dispersal corridors in ways that tend to enhance or alleviate interference interactions. DDT is typical for directly or environmentally transmitted diseases, but we would expect these results to hold also for other transmission functions that are a mixture between DDT and FDT (density-dependent at low host densities, frequency-dependent at high host densities; McCallum et al., 2001; Berec et al., 2017).

The underlying reason for these results is the non-monotonic response of equilibrium predator density to interference, with a maximum at intermediate interference values (DeAngelis et al., 1975; Arditi et al., 2004). This may seem counterintuitive as one might expect that predator density decreases with increasing interference. However, the negative direct effect of interference on predators can be supercompensated by a positive indirect effect on the prey, as equilibrium prey density increases with interference. The impact of interference on population densities is not that often considered though; most papers about predator interference focus on stability and limit cycles (DeAngelis et al., 1975; Alonso et al., 2002; Arditi et al., 2004; Rall et al., 2008; Berec, 2010; Li and Takeuchi, 2011), the derivation of the functional response (Beddington, 1975; Ruxton et al., 1992; Huisman and de Boer, 1997), or empirical evidence of predator-dependent functional responses (Hassell and Varley, 1969; Skalski and Gilliam, 2001; Kratina et al., 2009; DeLong and Vasseur, 2011; Zimmermann et al., 2015).

Let us now discuss the impact of disease on population densities and persistence. Another major finding in our numerical simulations is that the non-monotonic response of predators to interference also occurs in the presence of disease, provided the disease is not too ‘strong’, i.e., transmissibility is low and pathogenicity is large or very low. In these cases, predators can still benefit from increased interference even though they additionally suffer from increased pathogenicity. However, if the

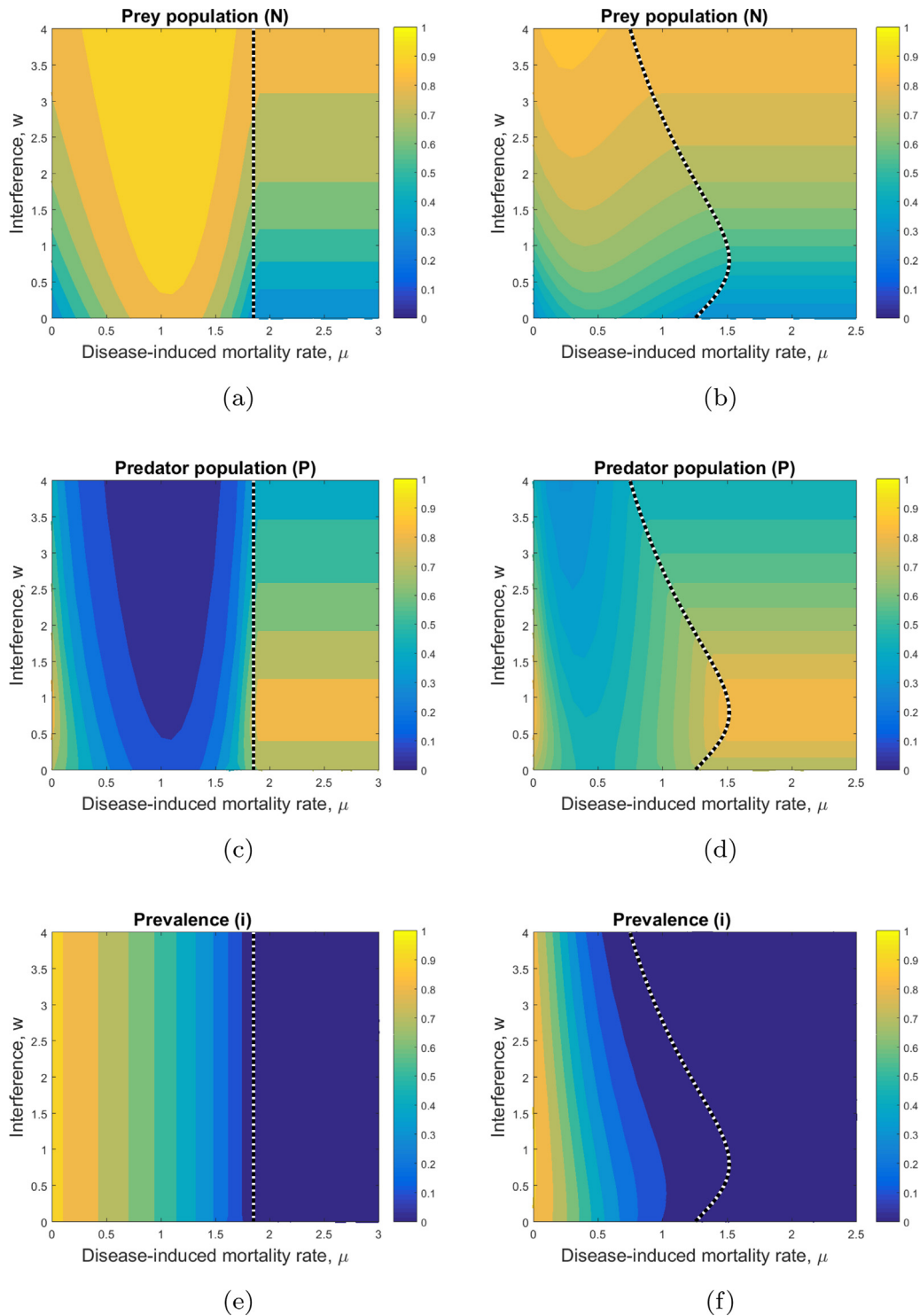


Fig. 4. Long-term values of the prey (top row, panels (a) and (b)), predators (middle row, panels (c) and (d)), and predator infection prevalence (bottom row, panels (e) and (f)) in the FDT (left column) and DDT (right column) model, when varying pathogenicity and predator interference strength. Results obtained from numerical solutions of (6) and (7), respectively. On the left-hand side of the dashed black–white line ($R_{0,FDT} = 1$ and $R_{0,DDT} = 1$) the system is at the disease-free predator–prey equilibrium. Parameters values $a = 5.0$, $m = 0.3$, $\beta = 2$, and $\nu = 0.5$. Initial conditions: $N(0) = 1$, $P(0) = 1$, $i(0) = 0.6$.

disease markedly depresses host population size due to high transmissibility or moderate pathogenicity, we do not observe the non-monotonic response of predators to interference; instead, equilibrium predator densities always decrease with interference. That is, large enough disease transmission or extreme pathogenicity levels at the low or high end drive the system into a state where predators cannot benefit anymore from interference

but are always impeded by the interplay of interference and infection. In some sense, the extra mortality due to disease always outweighs the potential benefit of interference in the form of increased prey population size and decreases competition for it. These observations hold for both FDT and DDT.

These results are consistent with well-known theory on biological control (Anderson and May, 1979; Anderson, 1979), when

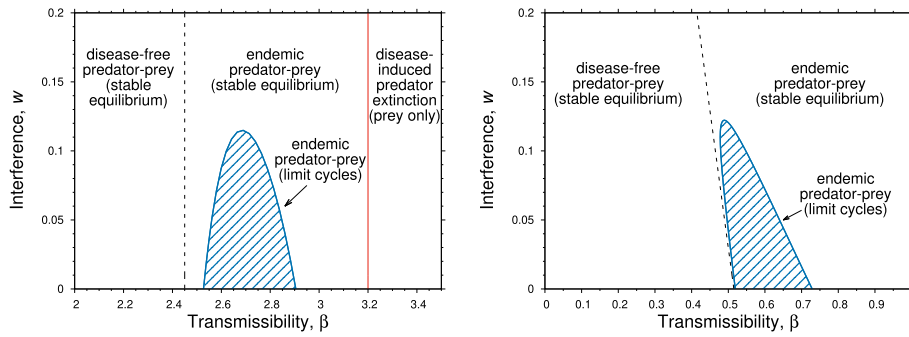


Fig. 5. Two-parameter bifurcation diagrams of (left) the FDT model (6) and (right) the DDT model (7), obtained by numerical continuation. In the hatched parameter region, there are nontrivial limit cycles. The blue bold curves mark Hopf bifurcations, the black dashed curves mark transcritical bifurcations indicating disease emergence ($R_0 = 1$), and the red solid curve marks a transcritical bifurcation indicating disease-induced predator extinction in the FDT model. Parameter values: (left) $a = 10.0$, $m = 0.5$, $\mu = 2$, $\nu = 0.1$. (right) $a = 5.0$, $m = 0.01$, $\mu = 0.5$, $\nu = 0$.

the aim is to regulate predators that are pests by introducing parasites. The parasites should not be too strong to maintain transmission over a long period of time, but they should not be too mild in order to avoid positive effects of interference on the host population.

Predator extinction due to infection is never possible for DDT, even with predator interference. For FDT, we have shown analytically that predator interference does not alter the condition for disease-induced predator extinction. For FDT, equilibrium prey densities always decrease with interference also in the presence of disease. For DDT, this is also the case in our numerical simulations.

We have also investigated how infection and interference affect system stability. In our simulations, interference had a stabilizing effect. That is, if there were limit cycle oscillations in the eco-epidemiological system, they were stabilized to asymptotically stable equilibrium points when increasing interference strength. This is consistent with the generally stabilizing effect attributed to predator interference in the literature (DeAngelis et al., 1975; Ruxton et al., 1992; Huisman and de Boer, 1997; Arditi et al., 2004; Berec, 2010). The (limited) simulations in this paper suggest that sufficiently strong interference overrides instabilities that may arise from combined infection and predation dynamics, for all values of the transmissibility.

In our simulations predator infection has been found capable of both destabilizing and re-stabilizing predator-prey systems. This is also consistent with the literature. On the one hand, predator infections have been reported to destabilize locally stable predator-prey equilibria for transmissibilities of intermediate level (Stiefs et al., 2009; Oliveira and Hilker, 2010; Bate and Hilker, 2013). On the other hand, infectious diseases in predators have been reported to stabilize unstable predator-prey systems for sufficiently large transmissibilities (Hilker and Schmitz, 2008; Oliveira and Hilker, 2010).

Predator-prey models like the one considered here are the building blocks for more complex food webs, and the meta-analysis of empirical data by DeLong and Vasseur (2011) suggests that predator interference is common. The importance of parasites in modifying entire community structures is increasingly recognized (Wood et al., 2007; Hatcher and Dunn, 2011). The results from this paper indicate that infection may alter population responses to interference and that interference in turn can influence infection levels and even alter disease invasion. Our models predict these effects for intermediate interference levels. There is little consensus in the literature about the strength of predator interference, except that it varies with prey and predator species, ecosystem context, and even predator density itself (Holdridge et al., 2016). DeLong and Vasseur (2011, p. 1) concluded that “interference is mostly intermediate in magnitude”, but they

measured interference magnitude in terms of a Hassell–Varley exponent; this stipulates the question whether their empirical findings can be translated into intermediate interference values in terms of our model.

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Appendix A. Functional response derivation

In this appendix, we will formulate the functional responses of predators. Similarly to Beddington (1975), we will take into account prey handling times and time wasted due to predator interference, but we will extend this derivation to multiple types of predators according to their infection status.

We assume that there are two types of predators, susceptible and infected ones with densities S and I , respectively. Let $Y = S + I$ be the total predator population density and X the prey population density. Further assume that susceptible predators spend their total time budget T^S either searching for prey (T_s^S), handling prey (T_h^S), or waste their time interfering each other (T_w^S). We assume the same for infected predators (for which the superscripts are replaced by I). Then

$$T^S = T_s^S + T_h^S + T_w^S, \\ T^I = T_s^I + T_h^I + T_w^I.$$

The overall time spent by susceptible and infected predators to handle prey is, respectively,

$$T_h^S = a_S X T_s^S h_S, \\ T_h^I = a_I X T_s^I h_I. \tag{10}$$

It is proportional to the density of prey caught during the searching time, where a_S and a_I are the rates at which susceptible and infected predators, respectively, find prey. h_S and h_I are the prey handling times of susceptible and infected predators, respectively.

The overall time wasted by susceptible and infected predators when encountering other predators is, respectively,

$$T_w^S = (b_{SS} S w_{SS} + b_{IS} I w_{IS}) T_s^S, \\ T_w^I = (b_{SI} S w_{SI} + b_{II} I w_{II}) T_s^I. \tag{11}$$

It is proportional to the density of prey caught during the searching time, where b_{SS} and b_{IS} are the rates at which susceptible predators encounter susceptible and infected ones, respectively. Similarly, b_{SI} and b_{II} are

the rates at which infected predators encounter susceptible and infected ones, respectively. w_{SS} and w_{IS} are the times wasted by susceptible predators when encountering susceptible and infected predators, respectively. Similarly, w_{SI} and w_{II} are the times wasted by infected predators when encountering susceptible and infected predators, respectively.

The time spent searching for prey is $T_s^j = T^j - T_h^j - T_w^j$, where $j = S, I$. Substituting T_h^j and T_w^j by the expressions in (10) and (11), respectively, gives

$$T_s^S = \frac{T^S}{1 + a_S X h_S + b_{SS} S w_{SS} + b_{IS} I w_{IS}}, \tag{12}$$

$$T_s^I = \frac{T^I}{1 + a_I X h_I + b_{SI} S w_{SI} + b_{II} I w_{II}}.$$

The number of prey caught by susceptible and infected predators is, respectively,

$$V^S = a_S X T_s^S, \tag{13}$$

$$V^I = a_I X T_s^I,$$

i.e., proportional to the product of prey and predator densities, to the search rate, and to the time spent searching. The functional response is the number of prey caught per unit time per predator. We therefore substitute the expressions (12) into (13) and then divide (13) by T^j , $j = S, I$, and by S or I . This gives the following functional responses of susceptible and infected predators, respectively,

$$f_S(S, I, X) = \frac{a_S X}{1 + a_S h_S X + b_{SS} w_{SS} S + b_{IS} w_{IS} I}, \tag{14}$$

$$f_I(S, I, X) = \frac{a_I X}{1 + a_I h_I X + b_{SI} w_{SI} S + b_{II} w_{II} I}.$$

Appendix B. Analysis of the FDT model

In this appendix, we determine the equilibria of the model with frequency-dependent transmission and analyze their local stability. Consider system (6), i.e.,

$$\frac{dN}{dt} = (1 - N)N - \frac{NP}{1 + wP},$$

$$\frac{dP}{dt} = a \left(-mP - \mu Pi + \frac{NP}{1 + wP} \right), \tag{15}$$

$$\frac{di}{dt} = ia \left((\beta - \mu)(1 - i) - \frac{N(1 - \nu)}{1 + wP} \right).$$

Proposition 1.

- (i) The trivial equilibrium $E_0 = (0, 0, 0)$ and the extinction equilibrium with positive prevalence $E_4 = (0, 0, 1)$ are always feasible and they are always unstable.
- (ii) The prey-only equilibrium $E_1 = (1, 0, 0)$ is always feasible, and it is stable if $m - 1 > 0$ and $\beta - \mu < 1 - \nu$ hold.
- (iii) The disease-induced predator extinction equilibrium

$$E_2 = \left(N_2 = 1, P_2 = 0, i_2 = \frac{\beta - \mu - (1 - \nu)}{\beta - \mu} \right)$$

is feasible if $0 < i_2 < 1$, and it is stable if $m - 1 + \mu i_2 > 0$ and $\beta - \mu > 1 - \nu$ hold.

- (iv) The disease-free predator-prey equilibrium $E_3 = (N_3, P_3, 0)$ with

$$N_3 = m(1 + wP_3),$$

$$P_3 = \frac{-(m - 1)w - mw - 1 + \sqrt{4mw + (w - 1)^2}}{2mw^2} \tag{16}$$

is feasible if $m - 1 < 0$. It is stable if $\beta - \mu < (1 - \nu)m$ holds.

- (v) The endemic coexistence equilibrium is

$$E_* = \left(\frac{1 + (w - 1)P_*}{1 + wP_*}, P_*, \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu} \right),$$

where P_* is the root of the second-degree polynomial $AP^2 + BP + C = 0$ with

$$A = (\mu + m)(\beta - \mu)w^2,$$

$$B = [2wC + (\beta - \mu\nu)(1 - w)], \tag{17}$$

$$C = (\mu + m)(\beta - \mu) - (\beta - \mu\nu).$$

For the feasibility of the endemic coexistence equilibrium we need to have

$$1 + (w - 1)P_* > 0, \quad \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu} > 0,$$

and one of the conditions

- (a) $A < 0$ and $B < 0$ or $C < 0$
- (b) $A > 0$ and $B > 0$ or $C > 0$

must hold, with $A, B,$ and C as defined in (17). For the stability of the endemic coexistence equilibrium the Routh–Hurwitz conditions for a third-degree polynomial must hold.

Proof. To get the equilibrium points we solve the system obtained from equating the RHS of (15) to zero. To study their stability we compute the Jacobian matrix of (15),

$$J = \begin{bmatrix} J_{11} & J_{12} & 0 \\ \frac{P}{1 + wP} & -m - \mu i - J_{12} & -\mu P \\ -\frac{(1 - \nu)i}{1 + wP} & \frac{(1 - \nu)Ni w}{(1 + wP)^2} & J_{33} \end{bmatrix}$$

with

$$J_{11} = 1 - 2N - \frac{P}{1 + wP},$$

$$J_{12} = \frac{-N}{(1 + wP)^2},$$

$$J_{33} = (2i - 1)(\mu - \beta) - \frac{(1 - \nu)N}{1 + wP},$$

and evaluate it at each equilibrium point. Once we have the characteristic polynomial we compute the eigenvalues and analyze the signs of their real parts.

- (i) This can be shown with a few calculations.
- (ii) The eigenvalues of $J|_{E_1}$ are $\lambda_1 = -1 < 0$, $\lambda_2 = -(m - 1)$, and $\lambda_3 = (\beta - \mu) - (1 - \nu)$. For the stability of E_1 , $m - 1 > 0$ and $\beta - \mu < 1 - \nu$ must hold.
- (iii) For $P = 0$ and $N = 1$, we obtain from the third equation of (15)

$$i_2 = \frac{\beta - \mu - (1 - \nu)}{\beta - \mu}.$$

For the feasibility of E_2 , $0 < i_2 < 1$ must hold. The eigenvalues of $J|_{E_2}$ are $\lambda_1 = -1 < 0$, $\lambda_2 = 1 - m - \mu i_2$, and $\lambda_3 = -[\beta - \mu - (1 - \nu)]$. For the stability of E_2 , $1 - m - \mu i_2 < 0$ and $\beta - \mu > 1 - \nu$ must hold.

- (iv) For $i = 0$, we get $N = m(1 + wP)$ from the second equation of (15) and substitute N in the first equation. Then we get a second-degree polynomial in P :

$$mw^2P^2 + ((m - 1)w + mw + 1)P + m - 1 = 0. \tag{18}$$

Notice that, according to Descartes’s rule, (18) has at least one positive root if $m - 1 < 0$, while the case of two positive roots is impossible.

The eigenvalues of $J|_{E_3}$ are $\lambda_1 = -(1 - \nu)m + \beta - \mu$, while λ_2 and λ_3 are the roots of

$$\lambda^2 + \left[m(1 + wP) + \frac{w m P}{1 + w P} \right] \lambda + \frac{m P}{(1 + w P)^2} + w m^2 P = 0, \tag{19}$$

where P is the positive root of (18). Assuming that E_3 is feasible, it is stable if $\beta - \mu < (1 - \nu)m$ holds. Notice that λ_2 and λ_3 are negative roots of (19), in fact all the coefficients of (19) are positive.

(v) We get

$$N = \frac{1 + (w - 1)P}{1 + wP} \tag{20}$$

from the first equation of (15). Summing up the second and the third equation of (15) we get

$$i = \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu}. \tag{21}$$

Substituting (20) and (21) into the third equation of (15), we get a second-degree polynomial in P :

$$AP^2 + BP + C = 0 \tag{22}$$

with

$$\begin{aligned} A &= (\mu + m)(\beta - \mu)w^2, \\ B &= [2wC + (\beta - \mu\nu)(1 - w)], \\ C &= (\mu + m)(\beta - \mu) - (\beta - \mu\nu). \end{aligned} \tag{23}$$

For the feasibility of the coexistence equilibrium we need to have the positivity of the prey population and of the prevalence, respectively,

$$1 + (w - 1)P_* > 0, \quad \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu} > 0.$$

For the positivity of the predator population, one of the conditions

- (a) $A < 0$ and $B < 0$ or $C < 0$ or
- (b) $A > 0$ and $B > 0$ or $C > 0$

must hold, with $A, B,$ and C defined in (23). Notice that it is impossible for (22) to have two positive roots, in fact we cannot have two alterations in the signs of the coefficients of the polynomial (22).

The eigenvalues of $J|_{E_*}$ are the roots of the characteristic polynomial $\lambda^3 + a_1\lambda + a_2\lambda + a_3 = 0$. a_1, a_2 and a_3 are too involved to be reported here. For the stability of E_* the Routh–Hurwitz conditions for a third-degree polynomial must hold, i.e., $a_1 > 0, a_3 > 0,$ and $a_1a_2 > a_3$. \square

Appendix C. Analysis of the DDT model

In this appendix, we determine the equilibria of the model with density-dependent transmission and analyze their local stability. Consider system (7), i.e.,

$$\begin{aligned} \frac{dN}{dt} &= (1 - N)N - \frac{NP}{1 + wP}, \\ \frac{dP}{dt} &= a \left(-mP - \mu Pi + \frac{NP}{1 + wP} \right), \\ \frac{di}{dt} &= ia \left((\beta P - \mu)(1 - i) - \frac{N(1 - \nu)}{1 + wP} \right). \end{aligned} \tag{24}$$

Proposition 2.

- (i) The trivial equilibrium $E_0 = (0, 0, 0)$ and the extinction equilibrium with positive prevalence $E_4 = (0, 0, 1)$ are always feasible. The disease-induced predator extinction equilibrium $E_2 = (1, 0, (\mu + 1 - \nu)\mu^{-1})$ is feasible if $\nu = 1$, i.e. only for complete vertical transmission. Furthermore all of them are unstable.
- (ii) The prey-only equilibrium $E_1 = (1, 0, 0)$ is always feasible, and it is stable if $m - 1 > 0$ holds.
- (iii) The disease-induced predator extinction equilibrium $E_2 = (1, 0, 1)$ is feasible if and only if $\nu = 1$, and it is always unstable.
- (iv) The disease-free predator–prey equilibrium $E_3 = (N_3, P_3, 0)$ with N_3 and P_3 as in (16) is feasible if the predator population is positive, i.e., $m - 1 < 0$. Furthermore, it is stable if $\beta P_2 - \mu < (1 - \nu)m$ holds.

Remark: The equilibrium point E_3 is the same as the one in Proposition 1, but the stability conditions are different.

(v) The endemic coexistence equilibrium is

$$E_* = \left(\frac{1 + (w - 1)P_*}{1 + wP_*}, P_*, \frac{\beta P_* - \mu - m(1 - \nu)}{\beta P_* - \mu \nu} \right),$$

where P_* is the root of a third-degree polynomial, for which no analytical expression was found.

For the non-negativity of the prey population and of the prevalence, we need to have

$$1 + (w - 1)P_* > 0 \quad \text{and} \quad \frac{\beta P_* - \mu - m(1 - \nu)}{\beta P_* - \mu \nu} > 0,$$

respectively.

For the stability of the endemic coexistence equilibrium the Routh–Hurwitz conditions for a third-degree polynomial must hold.

Proof. We proceed as for the FDT model in Appendix B. The Jacobian matrix of (24) is

$$J = \begin{bmatrix} J_{11} & J_{12} & 0 \\ \frac{P}{1 + wP} & -m - \mu i - J_{12} & -\mu P \\ -\frac{(1 - \nu)i}{1 + wP} & J_{32} & J_{33} \end{bmatrix},$$

where

$$\begin{aligned} J_{11} &= 1 - 2N - \frac{P}{1 + wP}, \\ J_{12} &= \frac{-N}{(1 + wP)^2}, \\ J_{32} &= \left[\beta(1 - i) + \frac{(1 - \nu)Nw}{(1 + wP)^2} \right] i, \\ J_{33} &= (2i - 1)(\mu - \beta P) - \frac{(1 - \nu)N}{1 + wP}. \end{aligned}$$

- (i) With a few calculations this is easy to find.
- (ii) The eigenvalues of $J|_{E_1}$ are $\lambda_1 = -1 < 0, \lambda_2 = -(m - 1),$ and $\lambda_3 = -(\mu + 1 - \nu)$. For the stability of E_1 $m - 1 > 0$ must hold.
- (iii) For $P = 0, N = 1,$ we obtain from the third equation of (24)

$$i = \frac{\mu + (1 - \nu)}{\mu}.$$

E_2 is feasible if $\nu = 1$, thus only for complete vertical transmission model. The eigenvalues of $J|_{E_2}$ are $\lambda_1 = -1 < 0, \lambda_2 = -(m + \mu - \nu),$ and $\lambda_3 = 1 - \nu + \mu > 0$. Hence, E_2 is unstable.

(iv) The equilibrium E_3 is the same as for FDT. The eigenvalues of $J|_{E_3}$ are $\lambda_1 = -(1 - \nu)m + \beta P - \mu$, while λ_2 and λ_3 are the roots of (19). Assuming that E_3 is feasible, it is stable if $\beta P - \mu < (1 - \nu)m$ holds. Notice that λ_2 and λ_3 are negative roots of (19), in fact all coefficients of (19) are positive.

(v) We get

$$N = \frac{1 + (w - 1)P}{1 + wP} \quad (25)$$

from the first equation of (24). Summing up the second and the third equation of (24) we get

$$i = \frac{\beta P - \mu - m(1 - \nu)}{\beta P - \mu \nu}. \quad (26)$$

Substituting (25) and (26) into the third equation of (24), we get a third-degree polynomial in P

$$A_1 P^3 + B_1 P^2 + C_1 P + D_1 = 0,$$

where

$$A_1 = (\mu + m)\beta w^2,$$

$$B_1 = (\mu + m)(2\beta w - \mu w^2),$$

$$C_1 = (\mu + m)(\beta - 2\mu w) + \mu \nu(w - 1),$$

$$D_1 = -(\mu + m - \nu)\mu.$$

For the feasibility of the coexistence equilibrium we need to have the positivity of the prey population and of the prevalence, respectively,

$$1 + (w - 1)P_* > 0, \quad \frac{\beta P_* - \mu - m(1 - \nu)}{\beta P_* - \mu \nu} > 0.$$

P_* can be found numerically.

The eigenvalues of $J|_{E_*}$ are the roots of the characteristic polynomial $\lambda^3 + b_1\lambda + b_2\lambda + b_3 = 0$. b_1 , b_2 , and b_3 are too involved to be reported here. For the stability of E_* the Routh–Hurwitz conditions for a third-degree polynomial must hold, i.e., $b_1 > 0$, $b_3 > 0$, and $b_1 b_2 > b_3$. \square

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