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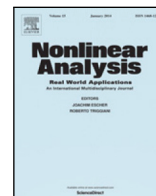
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The role of manipulative trophically transmitted parasites in the stability of a predator–prey community

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ABSTRACT

In this paper, we are interested in the effect of a trophically transmitted parasite on the structure and dynamics of a resident predator–prey community. The parasite, apart from increasing the mortality rates of its hosts, can also change their physiology or their behaviour, which is known as trait-mediated indirect interaction. We assume that parasite transmission, which entails rapid physiological or behavioural change, is a fast process with respect to the community dynamics, including prey and predator growths and predation. This existence of different time scales allows us to provide analytical results to understand some conditions under which the parasite change the dynamics of the predator–prey community. Thus, we are able to find conditions under which indirect trait-mediated interactions induced by the parasite lead to a coexistence between predators and prey that would not occur in its absence. We also provide conditions, evolutionary deleterious, that ensure the extinction of a predator–prey community that would be viable without parasite intervention. Finally, we show situations in which the action of the parasite destabilizes the predator–prey system without eliminating it, producing oscillations, the mechanism of which is analysed.

1. Introduction

There is an increasing evidence that parasites play a crucial role in structuring biological communities [1] and ecosystems [2]. They can sometimes account for a significant portion of total biomass in natural ecosystems [3].

The general purpose of community studies is to analyse the effects of species interactions on populations densities. The effects that individuals of two species can have on each other can be direct, as it is the case of predation or interference competition. At the same level of importance are the indirect interactions where the effect of one species on another is achieved through a third one. Parasites are a source of indirect interactions in a community. By killing their hosts, they cause so-called density-mediated indirect interactions, while by changing the physiology or behaviour of their hosts, they cause what are known as trait-mediated interactions [4,5].

Obviously, the life span of parasites must be long enough to complete their life cycle. This means that the life strategy of parasites often involves moving from a host to another. The movement between hosts is often achieved by trophic transmission [6], leading to the class of *trophically transmitted parasites*. The needs of parasites go sometimes against host interests and might even be fatal

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for the host. The response of parasites to solve this conflict of interests is host manipulation, i.e. the ability to alter the adequate feature (behaviour, appearance) of their host so as to increase their own fitness [7]. Host manipulation occurs in a large variety of hosts and parasites, and it can take many different forms [8].

Host manipulation by parasites has important consequences in community and ecosystem dynamics [9]. Manipulative parasites can strengthen the trophic links involved in their transmission. Numerous trophically transmitted parasites induce phenotypic changes in their hosts to produce an increase of the predation vulnerability of intermediate hosts to definitive hosts. This phenomenon is called enhancement and *enhancer-parasites* represent a large proportion of the parasites in food webs [2,3]. In contrast to empirical evidence for the abundance of enhancer-parasites in natural systems, some modelling works [10] foresee that extensive enhancement would destabilize predator–prey dynamics and, therefore, lead to parasite extinction. A proposed framework to save this apparent contradiction is the switcher-paradigm [11]. The host-manipulating parasite successively induces reduced predation, decrease of prey vulnerability, followed by predation enhancement with parasite maturation, what can lead to higher parasite persistence [12].

In this work we want to participate in the preceding discussion by introducing in a simple model of a trophically transmitted parasite a new feature not considered previously. The phenotypic changes induced by parasites in their hosts occur, as any trait-mediated effect, on a shorter timescale than population-density mediated effects [5]. Thus, we represent our model by a fast-slow system of ordinary differential equations. We identify the phenotypic change with the fact of being infected by the parasite. Then, we distinguish two time scales in our model and consider parasite transmissions acting at the fast one. The direct predator–prey interactions and the density-mediated indirect interactions between parasites and their hosts are assumed to occur at the slow time scale.

We consider a predator–prey community, with the prey being the intermediate host of the parasite and the predator the definitive host. Assuming the parasite is a microparasite, we can include it in the model in the simplest possible way, which consists of distinguishing between individual infected or uninfected in each species. A model with a similar structure is treated in Haderler and Freedman [13], that is considered one of the seminal works, together with Anderson and May [14], of the field called mathematical eco-epidemiology. An abundant literature on it can be found in Venturino [15]. The model proposed in Haderler and Freedman [13] is studied analytically, obtaining conditions on the viability of the parasite–predator–prey system. It is shown, for instance, that, in the case where the predator–prey community is not viable in the absence of the parasite, parasitism could lead to predator persistence whenever a certain transmission threshold is exceeded. A more recent and influential reference on the subject of manipulative trophic transmitted parasite is Fenton and Rands [10]. It includes a suite of models with a similar structure to that of Haderler and Freedman [13]. An equilibria stability analysis leads the authors to conclude that host manipulation keeps predator–prey community persistence though can greatly alter its quantitative dynamics, being its impact largely dependent on the predator functional response. With the same structure in Rogawa et al. [16] it is presented a model justifying that host manipulation can stabilize community dynamics, particularly when the manipulation is more intense. It treats separately the manipulation of the intermediate and the definitive hosts. In Iritani and Sato [11], de Vries and van Langevelde [12] they proposed models with two infected prey stages, in the first one parasites inducing predation suppression and in the second one enhancement. In both of them the switching strategy is presented as stabilizing and favouring community persistence. A general conclusion of all these references is that host manipulation is a consequence of self-organized behaviour of the parasite populations leading to a permanent coexistence with its hosts, what plays a major role in community stability.

This paper aims to present an original eco-epidemiological model that is complex enough to produce different types of manipulative effects but still simple enough to keep tractable. This allows us to understand the relative role of each parameter in different situations. Namely, the model is able to reproduce the positive impact of a parasite on a non viable community. It also contains the negative effect of a parasite killing a predator that would persist in the absence of the parasite. Finally the model can produce fluctuations induced by the presence of parasite, which are based on a complex mechanism involving all the protagonists in the community sequentially. The relative simplicity of our model allowed us to get analytical results that could show this mechanism.

In the next section, we describe the model and the assumptions on which it is based. The Section 3 introduces the reduction method and the reduced model with the assumptions required for the reduction method. This section also provides the dynamics of the reduced model. When the reduction is valid, these results also provide the dynamics of the complete model. But we will show that depending on the parameter values, there are situations where the reduction method is not always valid. The Section 4 explains and illustrates the effect of the parasite on the predator–prey community and provides the explanations for each effect. The last section discusses the results and concludes the paper.

2. The model

We consider a simple trophically transmitted parasite model. Fig. 1 is a scheme describing the model structure, the parameters and their description are provided in Tables 1 and 2 provides the processes and flows. The parasite uses the prey species as an intermediate host to transmit to the definitive host that is the predator species. We assume that the parasite is a microparasite and so we introduce it in the model by distinguishing between uninfected and infected individuals in both host species. We thus have four state variables: N_U and N_I are the densities of uninfected and infected prey and P_U and P_I the densities of uninfected and infected predators.

In the absence of the parasite the predator–prey interaction is described by a classical Lotka–Volterra model with logistic growth of the prey and linear functional response. The prey intrinsic growth rate is $r > 0$ and its density-dependent regulation is reflected in the carrying capacity $K > 0$. Parameter $a > 0$ in the predation term is a rate per predator, and $e > 0$ is the predator conversion

Table 1
Description of the model parameters.

Parameter	Description
Ecological parameters	
r	Intrinsic growth rate of the prey population
K	Carrying capacity of the prey population
a	Attack rate
e	Efficiency conversion
m	Predator <i>per capita</i> mortality rate
Epidemiological parameters	
β	Contact rate for prey infection
λ	Contact rate for predator infection
ϕ	Infected prey fertility reduction rate
φ	Infected predator fertility reduction rate
α	Prey vulnerability enhancement rate
μ	Infected prey mortality induced by the parasite
ν	Infected predator mortality induced by the parasite
General	
ϵ	Time scale parameter

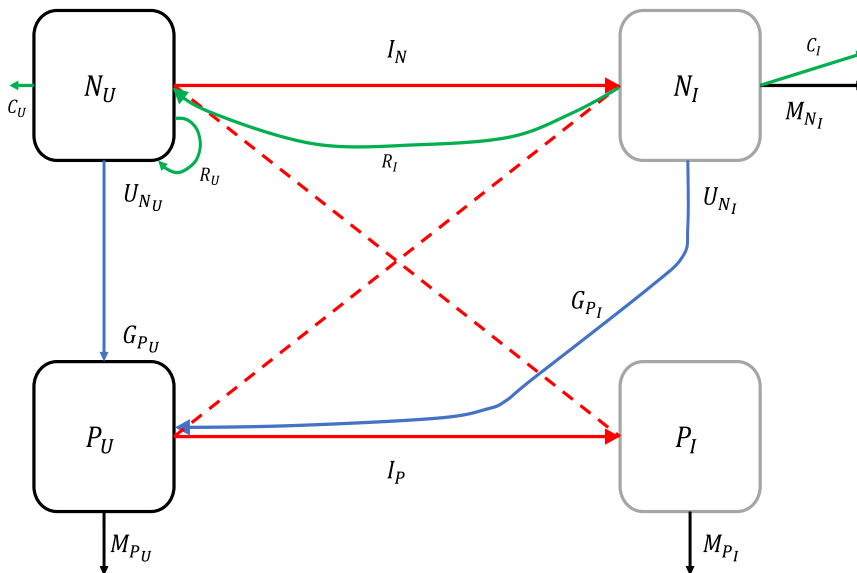


Fig. 1. Scheme of the model (2). Blue arrows represent trophic flows, red arrows represent epidemiological interactions (dashed lines) and flow (solid lines) and black arrows illustrate the death flow. Green lines correspond to reproduction and intraspecific competition death, the processes included in the logistic growth model. The blue flow G_P , leaving the N_I compartment and reaching the P_U compartment means that energy got from infected prey is converted in predator reproduction and only uninfected predators are produced. G_{P_U} measures reproduction of predators that only met uninfected prey. The detailed description of flows is provided in Table 2. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

efficiency of consumed prey. The per-capita predator mortality rate is $m > 0$. If we call N and P the densities of prey and predators, respectively, the model without parasite becomes

$$\begin{aligned}
 N' &= rN \left(1 - \frac{N}{K}\right) - aNP \\
 P' &= eaNP - mP
 \end{aligned}
 \tag{1}$$

It entails a stabilization towards the coexistence equilibrium

$$N^* = \frac{m}{ea}, \quad P^* = \frac{r}{a} \left(1 - \frac{m}{eaK}\right),$$

provided that $N^* < K$. In the opposite case, $N^* \geq K$, predators get extinct and prey tend to their carrying capacity K [17].

The manipulation of the parasite can act both on the prey, the intermediate host species, and on the predator, the definitive host species [16]. We consider three possible effects on the infected prey: reduction of fertility that can reach castration, increase of vulnerability to predation, and increase of mortality. On the other hand, the parasite effect on the infected predators are decrease of fertility, and increase of mortality.

We assume that there is no vertical transmission of the parasites, i.e., all prey and predator newborns are uninfected. The parasite passes into the environment from infected predators. It is then picked up by the prey that so become infected. In turn,

Table 2
Description of flows represented in Fig. 1.

Flow	Description	Value
R_U	Reproduction of uninfected prey individuals	$\epsilon r N_U$
R_I	Reproduction of infected prey individuals	$\epsilon r \phi N_I$
C_U	Competition induced death of uninfected prey	$\epsilon r N_U \frac{N_U + N_I}{K}$
C_I	Competition induced death of infected prey	$\epsilon r N_I \frac{N_U + N_I}{K}$
U_{N_U}	Uptake of uninfected prey by predators	$\epsilon a N_U (P_U + P_I)$
G_{P_U}	Predator reproduction associated to predation of uninfected prey	$\epsilon e a N_U (P_U + \phi P_I)$
U_{N_I}	Uptake of infected prey by predators	$\epsilon (a + \alpha) N_I (P_U + P_I)$
G_{P_I}	Predator reproduction associated to predation of infected prey	$\epsilon e (a + \alpha) N_I (P_U + \phi P_I)$
I_N	Prey infection	$\beta N_U P_I$
I_P	Predator infection	$\lambda (a + \alpha) P_U N_I$
M_{P_U}	Uninfected predator mortality	$\epsilon m P_U$
M_{N_I}	Infected prey mortality due to parasite	$\epsilon \mu N_I$
M_{P_I}	Infected predator mortality	$\epsilon (m + \nu) P_I$

predators become infected by feeding on infected prey. We consider that all predators participate equally in supplying parasites to the environment and that all prey have access to it. This leads us to propose a transmission rate from the parasite to the prey proportional, with transmission coefficient $\beta > 0$, to the product $N_U P_I$. It is contacts between infected prey and uninfected predators that lead to trophic transmission of the parasite. We then assume that the transmission rate of the parasite from the prey to the predator is proportional, with a coefficient $\lambda > 0$, to the predation term corresponding to these two sub-populations. It is clear that greater consumption of prey entails an increased risk of transmission.

The fact that the demographic and ecological process is supposed to be slow compared to the transmission of the parasite is described by means of parameter $\epsilon \ll 1$. The full model in terms of the time scale associated to the transmission process reads as follows:

$$\begin{aligned}
 N'_U &= -\beta N_U P_I + \epsilon \left(r (N_U + \phi N_I) - r N_U \frac{N_U + N_I}{K} - a N_U (P_U + P_I) \right) \\
 N'_I &= \beta N_U P_I + \epsilon \left(-r N_I \frac{N_U + N_I}{K} - (a + \alpha) N_I (P_U + P_I) - \mu N_I \right) \\
 P'_U &= -\lambda (a + \alpha) N_I P_U + \epsilon (e (a N_U + (a + \alpha) N_I) (P_U + \phi P_I) - m P_U) \\
 P'_I &= \lambda (a + \alpha) N_I P_U + \epsilon (-(m + \nu) P_I)
 \end{aligned} \tag{2}$$

In the fast part of the system, the parasite transmission, we assume that recovery does not exist. The model with recovery will be treated elsewhere.

Concerning the slow part of the system, the trophic relationship, we use the parameters above described (r , K , a , e , and m), and the parameters associated to parasite influence.

All prey participate in reproduction, although infected ones may see their fertility rate decreased by a factor $\phi \in [0, 1]$ due to the effects of the parasite. The fact that parasite leads to infected prey castration can be assumed by setting $\phi = 0$. Both infected and uninfected prey participate at the same level in competition for resources and, therefore, have the same density-dependent regulation.

All predators, infected and uninfected, participate equally in the predation process. The increase of vulnerability to predation due to parasite is defined by parameter $\alpha > -a$. When $\alpha = 0$, infected prey are as likely to be preyed upon as uninfected prey; when $\alpha > 0$, predators are more likely to capture infected prey; and, when $\alpha \in (-a, 0)$ the predator preference is for uninfected prey. Parameter $\mu \geq 0$ represents the additional parasite-induced host mortality of infected prey. Similarly, for infected predators, $\phi \in [0, 1]$ is the factor describing the decrease of fertility, $\phi = 0$ stands for their castration, and $\nu \geq 0$ is the added parasite-induced mortality.

3. Model reduction

The reduction method is based on the Geometrical Singular Perturbation Theory (GSPT) initiated by Fenichel [18,19]. Appendix A.1 provides the mathematical details. Roughly, the method consists in transforming the differential system (2) into a slow-fast form (3) (see below for details). Then we study the fast part of the slow-fast system by setting $\epsilon = 0$. If the resulting subsystem has a hyperbolic stable equilibrium, we then replace the fast variables (here N_U and P_U) by their fast equilibrium value, which usually depends on the slow variables (here N and P) in the slow part. These two steps are detailed below. Let us note here that the requirement for applying the method is that the fast equilibrium is hyperbolic stable. This can be analysed through the Jacobian matrix of the fast subsystem (see below). The eigenvalues of this matrix usually depend on the slow variables. When the values of the slow variables move, the previous requirement can be true at the beginning and become false after some time. In this case, the reduction method is only valid during a fixed period and other arguments are needed to complete the study when

the required conditions are lost. The mathematical method can be extended to such situation [20–22] and it has been applied in ecological models [23].

In this section, we build a reduced model with two state variables, the total prey density $N(t) = N_U(t) + N_I(t)$ and the total predator density $P(t) = P_U(t) + P_I(t)$, such that the solutions of the reduced model are ε -approximations of the N and P solutions of the initial model (2). In order to proceed with the reduction, we first write model (2) into the so-called slow–fast form, by making the following change of coordinates $(N_U, N_I, P_U, P_I) \mapsto (N_U, P_U, N, P)$, obtaining:

$$\begin{aligned} N'_U &= -\beta N_U(P - P_U) + \varepsilon \left(r(\phi N + (1 - \phi)N_U) - rN_U \frac{N}{K} - aN_U P \right) \\ P'_U &= -\lambda(a + \alpha)(N - N_U)P_U + \varepsilon \left(e((a + \alpha)N - \alpha N_U)(\varphi P + (1 - \varphi)P_U) - mP_U \right) \\ N' &= \varepsilon \left(r(\phi N + (1 - \phi)N_U) - rN \frac{N}{K} - ((a + \alpha)N - \alpha N_U)P - \mu(N - N_U) \right) \\ P' &= \varepsilon \left(e((a + \alpha)N - \alpha N_U)(\varphi P + (1 - \varphi)P_U) - (m + \nu)P + \nu P_U \right) \end{aligned} \tag{3}$$

3.1. Fast system

The fast system associated to system (3) is obtained by setting $\varepsilon = 0$. Considering N and P as nonnegative parameters, we analyse the next two-dimensional system on the domain of interest $\mathcal{H} := [0, N] \times [0, P]$

$$\begin{aligned} N'_U &= -\beta N_U(P - P_U) \\ P'_U &= -\lambda(a + \alpha)(N - N_U)P_U \end{aligned} \tag{4}$$

It possesses the parasite-free fast equilibrium

$$E^* = (N, P),$$

and the all infected fast equilibrium

$$E_0^* = (0, 0). \tag{5}$$

\mathcal{H} is positively invariant for system (4).

The Jacobian matrix associated to system (4) is

$$J(N_U, P_U) = \begin{pmatrix} -\beta(P - P_U) & \beta N_U \\ \lambda(a + \alpha)P_U & -\lambda(a + \alpha)(N - N_U) \end{pmatrix}$$

The trace is negative on $\mathcal{H} - \{E^*\}$ so the Bendixon criterium implies that there is no closed orbit inside.

E^* is a saddle and its stable manifold has an empty intersection with $\mathcal{H} - \{E_0^*\}$. On the other hand, we have

$$J(0, 0) = \begin{pmatrix} -\beta P & 0 \\ 0 & -\lambda(a + \alpha)N \end{pmatrix} \tag{6}$$

so that E_0^* is locally asymptotically stable provided $N \neq 0$ and $P \neq 0$. The Poincaré-Bendixon theorem yields that all solutions in $\mathcal{H} - \{E^*\}$ tend to E_0^* . Hence global asymptotic stability follows as well.

3.2. Reduced system

If we substitute the fast equilibrium E_0^* (5) into the equations for the slow variables N and P in system (3) and change to the slow time scale, keeping the same notation for the derivative, we obtain the reduced system

$$\begin{aligned} N' &= r\phi N - \frac{r}{K}N^2 - (a + \alpha)NP - \mu N \\ P' &= e\varphi(a + \alpha)NP - (m + \nu)P \end{aligned} \tag{7}$$

To analyse model (7) we distinguish two cases: $r\phi - \mu \leq 0$ and $r\phi - \mu > 0$.

1. In the absence of predators, parasites prevent prey from growing, $r\phi - \mu \leq 0$:

In this case we have for any initial condition, $N(0) = N_0 > 0$ and $P(0) = P_0 > 0$, that

$$N'(t) \leq -\frac{r}{K}N^2(t),$$

which straightforwardly yields

$$N(t) \leq \frac{N_0 K}{K + rN_0 t} \xrightarrow{t \rightarrow \infty} 0.$$

Now, for any positive value $d < m + \nu$ we can find $t_d > 0$ such that for $t \geq t_d$

$$N(t) \leq \frac{m + \nu - d}{e\varphi(a + \alpha)},$$

what implies that

$$P'(t) \leq -dP(t)$$

and, therefore, that also $P(t)$ tends to 0. So the trivial equilibrium is globally asymptotically stable.

2. In the absence of predators, parasites do let prey grow, $r\phi - \mu > 0$:

In this case model (7) can be written in the form of model (1):

$$\begin{aligned} N' &= r_1 N \left(1 - \frac{N}{K_1} \right) - a_1 N P \\ P' &= e_1 a_1 N P - m_1 P \end{aligned} \tag{8}$$

where $r_1 = r\phi - \mu$, $K_1 = \frac{r\phi - \mu}{r} K$, $a_1 = a + \alpha$, $e_1 = e\varphi$ and $m_1 = m + \nu$.

Calling $N_1^* = m_1 / (e_1 a_1)$, that stands for the prey density balancing predator growth, we can distinguish the two different asymptotic behaviours that can exhibit model (8):

- (a) If $N_1^* \geq K_1$ there is no positive equilibrium and solutions tend to the free predator equilibrium $\mathcal{E}_U^* = (K_1, 0)$.
- (b) If $N_1^* < K_1$, the positive solutions of model (8) tend to the coexistence equilibrium

$$\mathcal{E}^* = (N_1^*, P_1^*) = \left(\frac{m_1}{e_1 a_1}, \frac{r_1}{a_1} \left(1 - \frac{N_1^*}{K_1} \right) \right). \tag{9}$$

3.3. Link between the reduced model (7) and the complete model (2)

In case 2(b) Fenichel's theorem does apply and, therefore, the asymptotic behaviour of the solutions of the initial model (2) can be obtained with the help of the fast equilibria (5) and the asymptotic behaviour of the reduced model (8). The approximate behaviour consists in individuals in both hosts populations becoming rapidly infected and their total densities tending to \mathcal{E}^* . Fig. 2 illustrates that both models exhibit similar densities of the total populations in this case, and the reduced model is obtained with only infected individuals in both populations.

On the other hand, in cases 1 and 2(a) we note that both, N and P , or just one, P , slow variables tend to 0 so that at least one of the eigenvalues of the Jacobian matrix (6) tends to vanish and thus Fenichel's theorem does not apply for an infinite time. As mentioned at the beginning of this section, the analysis of model (2) cannot be done with the help of the reduced model (7) after a certain time.

In case 2(a), two sub-cases can be considered: either $K < N_1^*$ or $K > N_1^*$. In the former subcase, the predator goes to extinction and consequently the parasite as well, which allows the prey to reach the carrying capacity with only uninfected individuals. The next section provides the proof. In the latter subcase, fluctuations may take place. This is also discussed in Section 4.

In case 1, we can show that fluctuations can take place according to the parameter values. This is discussed in Section 4 and the mathematical proof is provided in Appendix A.1.

We saw previously that several cases occur according to the parameter values. Since we are interested in the impact of the parasite on the predator-prey system, we now translate the above conditions on parameters that distinguish the different cases in conditions on parameters describing parasite effects. For instance, case 1 corresponds to the condition $r\phi - \mu < 0$. This condition explicitly shows that it is satisfied either if the extra mortality induced by the parasite on the prey is large enough ($\mu > r\phi$) or if the reduction of the prey fertility is strong enough ($\phi < \frac{\mu}{r}$). In cases 2 (a and b), the reversed inequality holds. Furthermore, case 2 (b) corresponds to the condition $N_1^* < K_1$ that can be rewritten as $\alpha > -a + \frac{(m + \nu)r}{e\varphi K(r\phi - \mu)}$ for instance. This last condition can be interpreted as a large impact of the parasite on the prey catchability, it corresponds to strong enhancement. The case 2(a) is subdivided in two subcases. Either $K_1 < N_1^* < K$, which is equivalent to $-a + \frac{m + \nu}{e\varphi K} < \alpha < -a + \frac{(m + \nu)r}{e\varphi K(r\phi - \mu)}$ or $K_1 < K < N_1^*$ which is equivalent to $\alpha < -a + \frac{m + \nu}{e\varphi K}$. The former subcase corresponds to an intermediate value of α (intermediate enhancement) and the latter subcase corresponds to a small value of α , either small enhancement if $\alpha > 0$ or even preference for uninfected prey if $\alpha < 0$.

4. Effects of the parasite on the predator-prey community

In this section we present results on the asymptotic behaviour of the solutions of the model (2) that cover all the previous cases and subcases. They correspond to four exclusive conditions on the parameters. For each of them, we interpret the results under the epidemiological point of view, that is, we expose the effect of parasite on the system. We show that the presence of the parasite may change the fate of the predator-prey community, which could be different if the parasite was absent. Let us just remind that in absence of parasite, our model (2) is equivalent to model (1). Consequently, in absence of parasite, either the model reaches a positive equilibrium if $K > \frac{m}{ea}$ or it reaches an equilibrium without predator if $K < \frac{m}{ea}$.

Several models have been described in the previous section. We have two equivalent versions of the complete model, the initial model (2) and its mathematically equivalent slow-fast form (3), needed to apply Fenichel's theorem. We shall refer in the paper to model (2) as the complete model. Furthermore, Fenichel's theory allowed us to get a reduced approximation of the complete model with model (7). When $r_1 = r\phi - \mu > 0$, this model has a mathematically equivalent form with model (8). We introduced this new

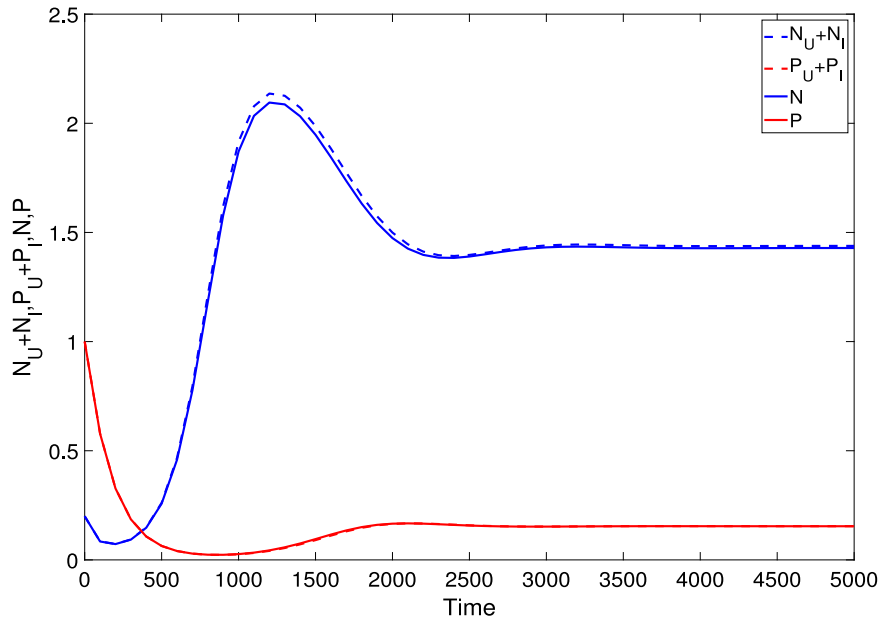


Fig. 2. This figure illustrates the approximation of the abundances of the total populations $N_U + N_I$ and $P_U + P_I$ with the complete model (2) by the abundances of the total populations N and P simulated with the reduced model (8). Here, the parameter values are $\epsilon = 0.01$, $\beta = 8$, $a = 0.1$, $e = 0.2$, $K = 3$, $m = 0.3$, $r = 1$, $\alpha = 2$, $\lambda = 5$, $\mu = 0.1$, $\nu = 0.3$, $\varphi = 1$, and $\phi = 0.9$. The initial conditions are $N_U(0) = 0.1$, $N_I(0) = 0.1$, $P_U(0) = 0.5$ and $P_I(0) = 0.5$ for the complete model and $N(0) = 0.2$ and $P(0) = 1$ for the reduced model.

version because it has a very common form and it is well-known. Thus each time it is meaningful, that is when $r_1 > 0$, we refer to model (8) as the reduced model.

The section is organized as follows. In the first subsection, we deal with the case 1 described in the previous section, that is $r_1 = r\phi - \mu < 0$, the parasite prevents the prey from growing and we show that it can lead to fluctuations. The other subsections assume that $r_1 > 0$ and a decreasing impact of the parasite on the prey catchability (α) from Section 4.2 where $\alpha > -a + \frac{(m+\nu)r}{e\varphi K(r\phi-\mu)}$ (case 2.b of the previous section) to Section 4.4 where $-a < \alpha < -a + \frac{m+\nu}{e\varphi K}$. Section 4.3 corresponds to the intermediate situation $-a + \frac{m+\nu}{e\varphi K} < \alpha < -a + \frac{(m+\nu)r}{e\varphi K(r\phi-\mu)}$. Thus Sections 4.3 and 4.4 deal with the two subcases 2.a of the previous section.

4.1. Parasite prevents prey from growing ($r\phi - \mu < 0$)

We consider here the situation where $r_1 = r\phi - \mu < 0$, or in other words in which the prey intrinsic growth rate r reduced by the fertility factor ϕ becomes less than the extra mortality rate μ induced by the parasite. At first glance, we may conclude that the prey, having a negative growth rate, would go to extinction, leading then the specialist predator to extinction as well. However, when the predator density is low, the parasite cannot spread in the prey population, thus the effect of parasites decreases. What would thus be the dynamics in this case? Actually, as it is illustrated on Fig. 3, the model can exhibit either a stable equilibrium or fluctuations. In both cases, the reduced model presented in the previous section cannot be used.

We prove in Appendix A.1 (Theorem 2) that fluctuations can take place, explain why and provide some sufficient conditions for their existence. Note that the fluctuations are induced by the presence of the parasite because if all individuals were initially uninfected, the model would reach an equilibrium. Roughly speaking, the mechanism of the fluctuations can be described as follows: when the populations are infected, the prey collapses because its fertility is reduced and its mortality increased, which leads to the reduction of predators and hinders the spread of the parasite; once the predator and infected prey densities are low, prey can grow, causing predators to grow again and facilitating the spread of the parasite, starting a new cycle. From a mathematical point of view, the proof of the existence of fluctuations is done by showing that there exists a sequence of times T_n tending to infinity such that if n is even, the number of prey $N(T_n)$ is very low (of order of ϵ) and that when n is odd, $N(T_n)$ is ϵ -close to $\frac{m+\nu}{e\varphi(a+\alpha)}$. The existence of this sequence is obtained by showing that the (N, P) components of solutions of the complete model (2) can be sequentially approximated by solutions of the reduced model (7) and by solutions of the reduced model (22) alternatively. Model (22) is an approximation of the complete model (2), also obtained from Fenichel’s theorem, but valid in the vicinity of $\{P = 0\}$, that is when the predator density is rather low.

Furthermore, if the attack rate a is small enough, it may imply that the reduced predator growth prevents fluctuations. Indeed, Fig. 3 shows that the parameter a has an impact on the dynamics: when a is small, the model reaches an equilibrium while when a is large enough, oscillations appear and their amplitude is larger for larger a . Moreover, for a given a , an increase of the parameter

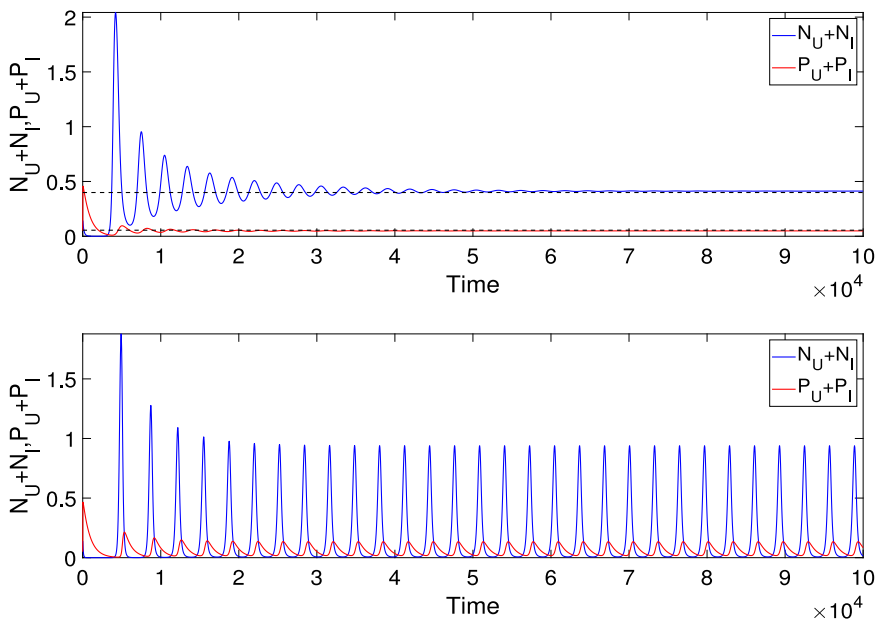


Fig. 3. This figure illustrates the dynamics of total prey and predator populations for the complete model (2) in the case where $r\phi - \mu < 0$. Two examples are provided, an equilibrium is reached in the first one (top panel) while fluctuations take place in the second one (bottom panel). Here, the parameter values are $\epsilon = 0.01, \beta = 0.2, e = 0.2, K = 3, m = 0.01, r = 1, \alpha = 0.7, \lambda = 1, \mu = 0.1, \nu = 0.1, \phi = 1$, and $\phi = 0.05$. The top panel is obtained with $a = 0.8$ while the bottom panel is obtained with $a = 2.5$. The initial conditions for both simulations are $N_U(0) = 0.1, N_I(0) = 0.04, P_U(0) = 0.42$ and $P_I(0) = 0.05$. Note that we added the equilibrium value (black dashed lines on the top panel) of model (22) to illustrate that it provides a good approximation of the complete model (2) when it has a stable equilibrium.

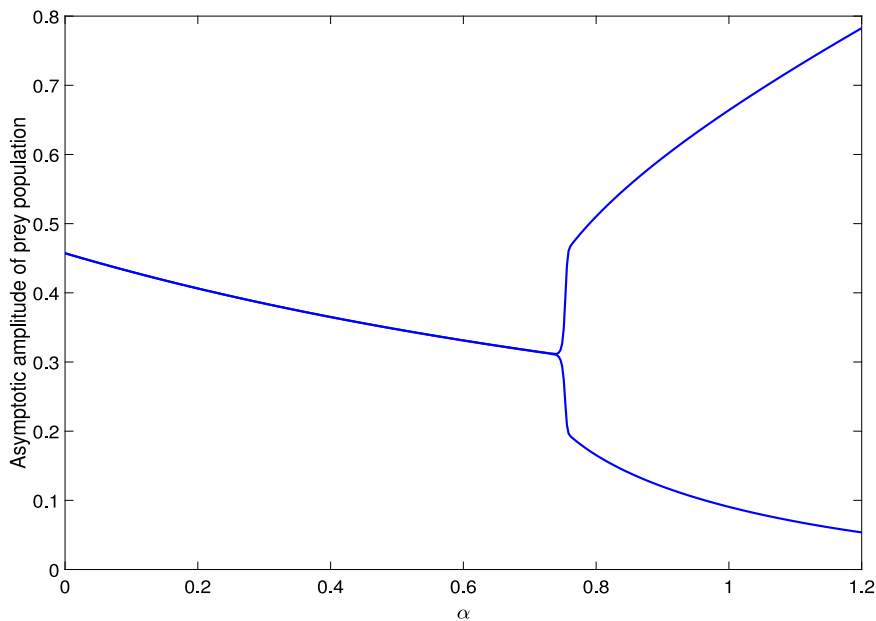


Fig. 4. This figure illustrates the effect of increasing the vulnerability to predation α on the dynamics of model (2) in the case where $r\phi - \mu < 0$. When α is small (no or low manipulation), the population reaches an equilibrium, the asymptotic total prey population density, which is shown in the figure. Increasing α leads to a destabilization of the equilibrium and the population tends to fluctuate between the two values represented in the figure. The parameter values are $\epsilon = 0.01, \beta = 0.2, a = 1.2, e = 0.2, K = 2.8, m = 0.01, r = 1, \phi = 0.05, \lambda = 1, \mu = 0.1, \nu = 0.1, \phi = 1$, and α is varied in the range $[0, a]$. The initial conditions are $N_U(0) = 0.5, P_U(0) = 1, N_I(0) = 0.5$ and $P_I(0) = 1$.

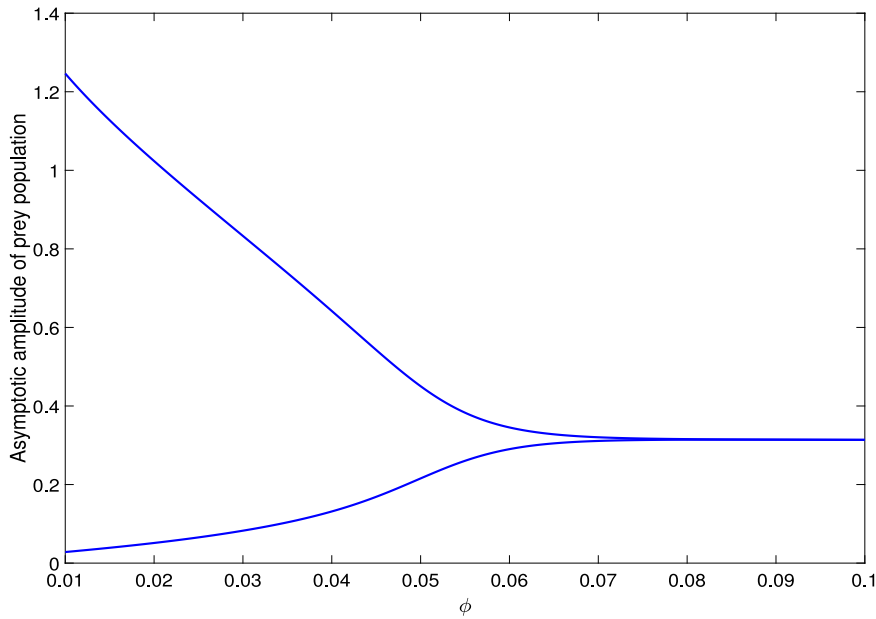


Fig. 5. This figure illustrates the effect of the infected prey fertility reduction parameter ϕ on the dynamics of the model (2) in the case where $r\phi - \mu < 0$. When ϕ is very small (strong fertility reduction), the prey population density tends to oscillate between the two values represented in the figure. Increasing ϕ leads to stabilization on the equilibrium shown in the figure. The parameter values are $\epsilon = 0.01$, $\beta = 0.2$, $a = 1.2$, $e = 0.2$, $K = 2.8$, $m = 0.01$, $r = 1$, $\alpha = 0.7$, $\lambda = 1$, $\mu = 0.1$, $\nu = 0.1$, $\varphi = 1$, and ϕ is varied in the range $[0.01, 0.1]$. The initial conditions are $N_U(0) = 0.5$, $P_U(0) = 1$, $N_I(0) = 0.5$ and $P_I(0) = 1$.

α leads to an increase of the fluctuation amplitude. More precisely, increasing the value of parameter α can either destabilize an equilibrium for small a (see Fig. 4) or just increase the amplitude of oscillations for large a .

In this subsection, the presence of the parasite leads to a negative prey growth rate $r\phi - \mu$ but if the prey density becomes low enough, the predator decreases and this reduces the impact of the parasite. Thus the dynamics of the full model is clearly a balance between positive interactions (impact of prey on predators) and negative indirect feedback of the parasite which needs predators to survive. If one of the processes become too intense, fluctuations take place: it can be the manipulation of the prey by the parasite (parameter α), the reduction of the prey fertility (parameter ϕ), see Fig. 5, just to give two examples.

4.2. Parasite has a strong impact on the prey catchability ($r\phi - \mu > 0$, large α)

We assume here that $r_1 = r\phi - \mu > 0$ and that $\alpha > -a + \frac{(m+\nu)r}{e\varphi K(r\phi-\mu)}$, which is equivalent to $K_1 > N_1^*$, the case 2.b of the previous section. Let us remind that under these conditions, the reduction method assumptions are satisfied, as soon as ϵ is small enough, which is assumed here. If we also assume that $eaK - m < 0$ (which is compatible with the conditions on the parameters assumed previously in this subsection), the complete model (2) exhibits a bistability. Depending on the initial conditions, either the trajectory goes to the equilibrium $(K, 0, 0, 0)$ or the trajectory reaches a stable equilibrium where both N and P are strictly positive and provided in formula (9). Let us remind that the equilibrium $(K, 0, 0, 0)$ corresponds to a situation where the predator is absent and all the prey are uninfected. However, the other equilibrium corresponds to a situation with prey and predator populations coexisting. Furthermore, the set $\{(N_U, N_I, P_U, P_I), N_U = N > 0, N_I = 0, P_U = P, P_I = 0\}$ is included in the stable manifold of the equilibrium $(K, 0, 0, 0)$. Accordingly, if there is no parasite in the system at $t = 0$, then the predator population will go to extinction and the prey population reaches its carrying capacity. However, if the numbers of infected prey and infected predators are not too small initially, then a positive equilibrium with prey and predators is reached. The notable issue is that, under the previous conditions, we could have extinction of the predator without parasite while the predator reaches a positive equilibrium when the parasite is present. Fig. 6 illustrates this phenomenon. This means that the persistence of the trophic community is the consequence of the pathogen interaction.

The results explained just above are rather straightforward now if we use model (3), the slow-fast version of the complete model (2). The equilibrium with no predator and no parasite, with these coordinates, is $(K, 0, K, 0)$. We first show that this equilibrium is locally hyperbolic stable with a small basin of attraction, that is the size of the basin of attraction is of order of ϵ . model (3) admits $(K, 0, K, 0)$ as a locally asymptotically stable equilibrium because, the Jacobian matrix of system (3) at this equilibrium has the following eigenvalues:

$$\gamma_1 = -\epsilon(m - eaK), \gamma_2 = -\epsilon r, \gamma_3 = -\epsilon(r + \mu), \gamma_4 = -\epsilon(m + \nu),$$

which are all strictly negative since we assumed that $eaK - m < 0$. However, as it can be seen on the previous expressions, the eigenvalues are of order of ϵ and this means that the basin of attraction of this equilibrium can be very small.

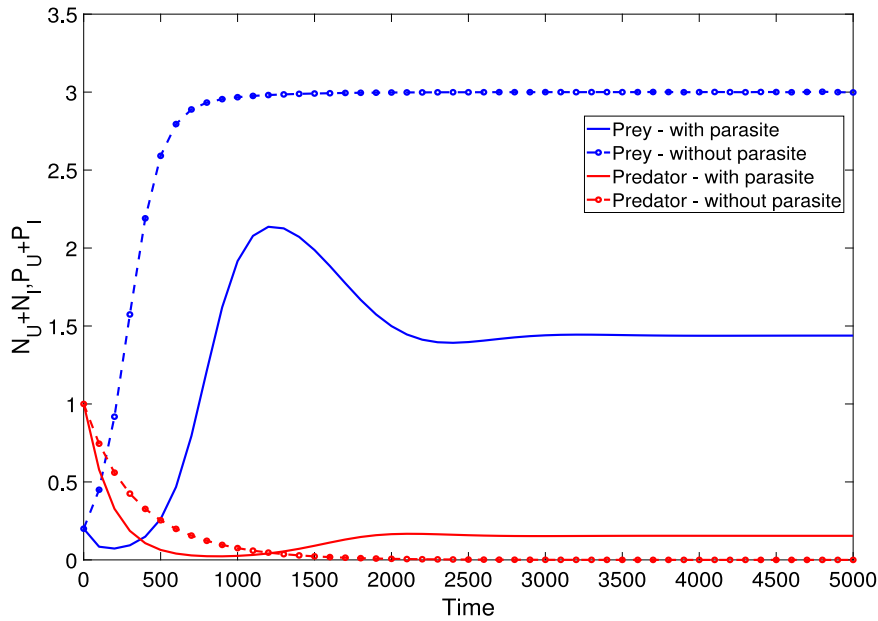


Fig. 6. This figure illustrates that in model (2) the parasite can favour predator–prey coexistence in the case where $r\phi - \mu > 0$ and large α . If parasites are initially present (continuous curves), predators can invade, whereas the opposite happens in the absence of parasites (dashed curves). Here, the parameter values are $\epsilon = 0.01$, $\beta = 8$, $a = 0.1$, $e = 0.2$, $K = 3$, $m = 0.3$, $r = 1$, $\alpha = 2$, $\lambda = 5$, $\mu = 0.1$, $\nu = 0.3$, $\phi = 1$, and $\phi = 0.9$. The initial conditions are $N_U(0) = 0.2$, $N_I(0) = 0$, $P_U(0) = 1$ and $P_I(0) = 0$ for the simulation without parasite and $N_U(0) = 0.1$, $N_I(0) = 0.1$, $P_U(0) = 0.5$ and $P_I(0) = 0.5$ for the simulation with parasite.

Moreover, let us consider an initial condition with no parasite. Since $N_I = 0$ and $P_I = 0$ are invariant conditions under the flow of (2), it follows that this model can be reduced to model (1). And as we reminded in the first section, if $eaK - m < 0$, the predator gets extinct. Thus each trajectory with an initial condition of the form $(N, 0, N, P)$ in model (3) is in the basin of attraction of $(K, 0, K, 0)$.

Let us now consider an initial condition with $0 < N < K$ and $P > 0$. As explained in Section 3, the complete model can thus be reduced and the reduced model governing the total population densities N and P has a globally asymptotically stable positive equilibrium (9). Accordingly, the complete model has a stable equilibrium that can be reached by certain initial conditions and where the prey and predator populations coexist.

This illustrates the fact that a large enough enhancement (parameter α) transforms a non viable predator–prey community into a permanent coexisting one. Parasite may play a key role in community stability [16].

4.3. Parasite has an intermediate impact on prey catchability (intermediate values of α)

Here we focus on the case where $r\phi - \mu > 0$ and α has an intermediate value:

$$-a + \frac{m + \nu}{e\phi K} < \alpha < -a + \frac{(m + \nu)r}{e\phi K(r\phi - \mu)} \tag{10}$$

The second inequality in (10) is equivalent to $K_1 < N_1^*$, which means that with the reduced model (8), the predator population would go to extinction. However, as we discussed in Section 2, in this case the reduced model is not a good approximation of the full model (2) because, since the predator density goes to zero, it leaves the domain where the Fenichel’s theorem applies. We provide the mathematical treatment of this case in Appendix A.2. Roughly, the dynamics can be understood as follows. First, because the infection is a fast process, the number of infected prey and predator will increase and then the reduced model (8) provides a good approximation of the full model during a given time length. This corresponds to a decrease of the predator population (both infected and uninfected subpopulations). After some time, the approximation fails and since the predator is low, we can reduce the full model to a 3-dimensional system (see Appendix A.2 for more details). This reduced model can have several types of equilibrium according to the value of the carrying capacity K : one with predator extinction and another one with coexistence of prey and predators, the latter at a low density. More precisely, if the carrying capacity is lower than $K_c = \frac{m + \nu}{e\phi a}$, the predator goes to extinction, the parasite disappears and the uninfected prey population reaches the carrying capacity K . If the carrying capacity is larger than K_c , the predators can be maintained, but at a very low density, which leads the prey almost uninfected and close to the carrying capacity. We show in Appendix A.2 that the threshold for the carrying capacity that distinguished the two previous dynamics is $K_c = \frac{m + \nu}{e\phi a}$ and this threshold belongs to the range of values of K compatible with the parameter conditions of this subsection. The appendix also contains simulations that illustrate the two configurations highlighted in this subsection.

4.4. The parasite has a low impact on prey catchability (low value of α)

We now assume that $r\phi - \mu > 0$ and

$$-a < \alpha < -a + \frac{m + \nu}{e\phi K} \tag{11}$$

Condition (11) is equivalent to $K < N_1^*$, which also involves that $K_1 < N_1^*$ since $K_1 < K$ always holds. We also assume here that $eaK - m > 0$ thus if there is no parasite initially, as we already said and shown in Section 2, the prey and predator populations coexist at equilibrium (like in model (1) which is equivalent to our complete model when the parasite is absent). We now see in this subsection that in presence of the parasite, the predator is excluded.

The proof of the predator exclusion in model (2) is still based on the Fenichel’s theorem, but it requires something more than the mere information obtained from the reduced model (7). We work on model (3), equivalent to model (2). For a set of initial conditions $(N_U(0), P_U(0), N(0), P(0))$ of model (3), one can apply Fenichel theory for ϵ small enough (Appendix A.1.1 provides a statement of the theorem) and, then, the solutions of model (8) are used to approximate $N(t)$ and $P(t)$ in model (3), as long as $P(t)$ keeps large enough. Under the conditions (11), P decreases to 0 in model (8) and, thus, after some time, the approximation fails. When it fails, P (and so also P_U) is of order of ϵ and we can, therefore, normalize the variables P and P_U in model (3) as follows:

$$P = \epsilon \bar{P} \text{ and } P_U = \epsilon \bar{P}_U \tag{12}$$

and so model (3) takes the following form:

$$\begin{aligned} N'_U &= \epsilon \left(-\beta N_U (\bar{P} - P_U) + r(\phi N + (1 - \phi)N_U) - \frac{r}{K} N_U N \right) + O(\epsilon^2) \\ \bar{P}'_U &= -\lambda(a + \alpha)(N - N_U)\bar{P}_U + \epsilon \left(e((a + \alpha)N - \alpha N_U)(\phi \bar{P} - (1 - \phi)\bar{P}_U) - m\bar{P}_U \right) \\ N' &= \epsilon \left(r(\phi N + (1 - \phi)N_U) - \frac{r}{K} N^2 - \mu(N - N_U) \right) + O(\epsilon^2) \\ \bar{P}' &= \epsilon \left(e((a + \alpha)N - \alpha N_U)(\phi \bar{P} - (1 - \phi)\bar{P}_U) - (m + \nu)\bar{P} + \nu\bar{P}_U \right) \end{aligned} \tag{13}$$

This is again a slow-fast system, which is equivalent to model (3) (and (2)) as long as the trajectory remains in the region of the phase space where condition (12) is valid. One then applies the Fenichel’s theory as done in Section 3. First, we set $\epsilon = 0$, which tells us that \bar{P}_U rapidly reaches a value close to 0, and then we replace \bar{P}_U by $O(\epsilon)$ in the slow equations. We make a change of time for using a slow time scale, and keeping the same notations for the time derivative, the model (13) reduces to:

$$\begin{aligned} N'_U &= -\beta N_U \bar{P} + r(\phi N + (1 - \phi)N_U) - \frac{r}{K} N_U N + O(\epsilon) \\ N' &= r(\phi N + (1 - \phi)N_U) - \frac{r}{K} N^2 - \mu(N - N_U) + O(\epsilon) \\ \bar{P}' &= e((a + \alpha)N - \alpha N_U)\phi \bar{P} - (m + \nu)\bar{P} \end{aligned} \tag{14}$$

Note that $N \leq K$ is a positively invariant region, thus one can assume without loss of generality that $N_U(t) \leq N(t) \leq K$, which allows us to conclude that:

$$\bar{P}' \leq (e\phi(a + \alpha)K - (m + \nu))\bar{P} = e\phi(a + \alpha)(K - N_1^*)\bar{P}$$

This inequality, together with (11), allows us to prove that \bar{P} tends to 0 at least exponentially fast. Thus, after some time, \bar{P} is ϵ close to 0. Taking this into account and changing the variable N_U by $N_I = N - N_U$, the other two equations of (14) can be written as follows

$$\begin{aligned} N'_I &= -(r\frac{N}{K} + \mu)N_I + O(\epsilon) \\ N' &= r(N - (1 - \phi)N_I) - \frac{r}{K} N^2 - \mu N_I + O(\epsilon) \end{aligned} \tag{15}$$

The first equation in (15) shows that $N_I(t)$ also tends at least exponentially fast to 0. Note that in this case, we cannot use directly the model (15) as an approximation of the complete model (3) but a new renormalization adding $N_I = \epsilon \bar{N}_I$ would lead to a similar dynamics. Thus, we can show that P and N_I tends to 0 and this leads the variable N to follow the equation:

$$N' = rN(1 - \frac{N}{K})$$

and we can conclude that $N(t)$ and $N_U(t)$ tend to K .

To summarize, if the parasite is initially absent then the populations of prey and predators coexist at equilibrium, whereas, if the parasite is initially introduced into the system, the predators become extinct and, therefore, the parasites too. This is illustrated in Fig. 7.

5. Conclusions

In this paper, we present an original model to study the effects of a manipulative trophically transmitted parasite on a predator-prey community. The model has been built trying, on the one hand, to include enough complexity and, on the other hand, to preserve to a large extent mathematical tractability. The complexity allows us to represent in some detail the impact of the parasite on the predator-prey community. Mathematical tractability entails understanding the processes involved and their results. We show

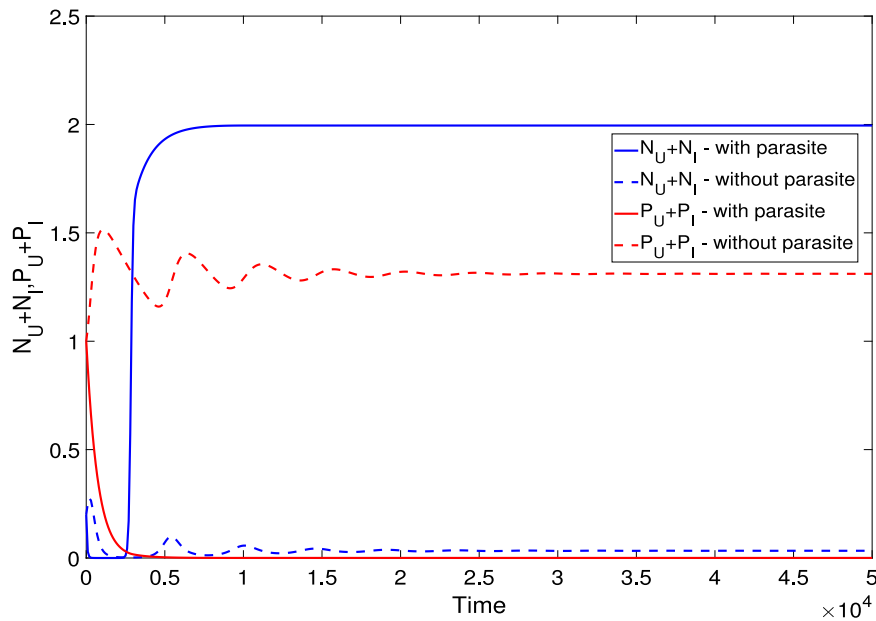


Fig. 7. This figure illustrates the extinction of the predator population induced by the parasite in model (2) in the case where $r\phi - \mu > 0$ and low α . Indeed, when there is no parasite in the initial condition, the predator and the prey densities reach a positive equilibrium value (dashed curves). However, in presence of parasite, the predator goes extinct (continuous curves). The parameter values are $\epsilon = 0.01$, $\beta = 0.2$, $a = 1.5$, $e = 0.2$, $K = 2$, $m = 0.01$, $r = 2$, $\phi = 0.1$, $\lambda = 1$, $\mu = 0.1$, $\nu = 0.1$, $\varphi = 0.1$, and $\alpha = 0.7$. The initial conditions are $N_U(0) = 0.5$, $N_I(0) = 0$, $P_U(0) = 0.5$ and $P_I(0) = 0$ for the simulation without parasite and $N_U(0) = 0.4$, $N_I(0) = 0.1$, $P_U(0) = 0.4$ and $P_I(0) = 0.1$ for the simulation with parasite.

that the model can reproduce patterns observed in real cases, see references in Section 9.4 of Hughes et al. [8]. One of them is the permanence of the predator–prey community induced by the parasite, that is, the coexistence of the community in the presence of the parasite for values of the parameters that would imply the extinction of the predator in its absence. We also show that the parasite can generate population densities fluctuations in circumstances that without the parasite would lead to a stable equilibrium situation.

We provide analytical conditions under which we can conclude the fate of the population densities. Our analytical results are illustrated and completed by numerical simulations. The mathematical analysis of the model requires methods of reduction to simplify the study. These methods are based on the fact that changes between the infection states (uninfected versus infected) are much faster than the ecological processes (growth, predation, and mortality). Roughly speaking, a first step consists in using a quasi-steady state assumption and the Fenichel’s theorem to reduce the dimension of the model. In case where the assumptions of the theorem are only valid during a transient time, we complete the study by considering different possible reduction models whenever possible.

Five parameters of our model allow us to represent different aspects of the manipulation of prey or predators by the parasite (see Table 1). We obtain results that help us to better understanding the effect of these parameters on the dynamics of the predator–prey system.

An important result is obtained when $r\phi - \mu > 0$ (the parasite allows the prey to grow in the absence of the predator). In this case, increasing the parameter α has a stabilizing effect, which can even lead to predator–prey coexistence in the form of stable equilibrium in circumstances that, without the parasite, would drive the predator to extinction, as it is explained in Section 4.2. Parasites that follow this strategy are called enhancer-parasites [11]. The results of the presented model (2) suggest, according to empirical evidence [24], that enhanced vulnerability leads to a stable coexistence equilibrium. This contrasts with the results obtained from other models [10] which, instead, predict a destabilization of the system.

On the other hand, a different result, when $r\phi - \mu < 0$ (the parasite prevents the prey from growing in the absence of the predator), shows that the parasite can lead to populations fluctuations, which is obtained when the prey vulnerability α is increased, as it is illustrated in Fig. 4.

In the absence of the parasite, the asymptotic behaviour of model (2) consists only of stable equilibria. Depending on the ecological parameters values, the system would tend to maintain only the prey population (poor environment) or to the prey–predator coexistence (rich environment). We show that increasing the prey mortality induced by the parasite μ or decreasing the infected prey fertility ϕ may lead the predator–prey community to fluctuate, see Fig. 5 for an illustration of the effect of ϕ reduction. These results are detailed in Section 4.1.

We provide a mechanism based on the mathematical arguments of the appendix, that is summarized as follows. Suppose the parasite has a strong impact on both fertility and mortality of infected prey, to the point that infected prey have a low or even negative growth rate. As parasite transmission is rapid compared to population dynamics, this reduction in growth rate can be

considered to affect all prey. Also assume that when the prey growth rate is positive, the carrying capacity of the prey in the presence of the parasite is so small that the predator cannot survive in a constant environment. In this case, the predator will start to collapse, and then the parasite is no longer able to infect the prey. Prey then slowly will increase with mostly uninfected individuals. Since there is still a positive, albeit small, number of predators in the system, and the number of prey has increased sufficiently, the predators begin to grow again. This increases the transmission of the parasite, which again leads to the spread of the infection among the prey. This is the point at which the rapid development of the parasite is repeated, which is followed by the infection of almost all the prey, its collapse, then the near extinction of the predators and the parasite, and so on. This is a heuristic explanation of the proof given in the appendix, which allows us to understand under which conditions this will occur.

In the same way, we can conclude with our study that decreasing the infected predators fertility φ leads to an increase of N_1^* , which can result in predator extinction, as detailed in Section 4.4. Increasing the infected predator extra-mortality induced by the parasite ν has the same effect.

Results of this paper have been obtained from the model (2) which is obviously based on specific assumptions. Our results are not sensitive on some of them, for instance we simulated the model with a Holling Type II functional response instead of the linear one and could see that, for the range of parameters we used, the qualitative results were unchanged. It is therefore a future work to extend them on a structurally different model based on Holling-Tanner formulation. This would allow to consider predator reproduction in a very different way to validate the present results in a more general context. Another extension that we aim to deal with concerns the existence of a recovery state.

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Appendix

A.1. Appendix 1: Case where $r\phi - \mu < 0$

This appendix is dedicated to the mathematical analysis of the complete model (2) when $r\phi - \mu < 0$, and we mainly show the existence of a periodic solution and we provide conditions under which the complete model (2) exhibits fluctuations. We remind first that this model is equivalent to model (3), which reads:

$$\left\{ \begin{aligned} \frac{dN_U}{d\tau} &= -\beta N_U(P - P_U) + \varepsilon \left(r(1 - \phi)N_U + r\phi N - \frac{r}{K} N_U N - aN_U P \right) \\ \frac{dP_U}{d\tau} &= -\lambda(a + \alpha)(N - N_U)P_U + \varepsilon \left(e((a + \alpha)N - \alpha N_U) \left((1 - \phi)P_U + \varphi P \right) - mP_U \right) \\ \frac{dN}{d\tau} &= \varepsilon \left(r(1 - \phi)N_U + r\phi N - r\frac{N^2}{K} - aNP - \alpha(N - N_U)P - \mu(N - N_U) \right) \\ \frac{dP}{d\tau} &= \varepsilon \left(e((a + \alpha)N - \alpha N_U) \left((1 - \phi)P_U + \varphi P \right) - (m + \nu)P + \nu P_U \right) \end{aligned} \right. \tag{16}$$

where $(N_U, P_U, N, P) \in \mathcal{E}$ with \mathcal{E} the phase space defined as:

$$\mathcal{E} = \{(N_U, P_U, N, P) / N \geq 0, P \geq 0, 0 \leq N_U \leq N, 0 \leq P_U \leq P\}$$

This part is devoted to the study of the dynamics of the trophically transmitted parasite model when the model reduced to the slow manifold leads to $(N, P) = (0, 0)$ as a stable equilibrium. Since, in this case, the real parts of the Jacobian matrix of the fast system tend to 0, the trajectory enters a region of the phase space where the Fenichel’s theory does not apply. We then provide elements to understand the dynamical behaviour of the system and prove that fluctuations occur in the complete system. But in order to clarify our approach, we first remind Fenichel’s theorem and this gives us the arguments and notations to explain what is going on when the assumptions are not satisfied.

A.1.1. Statement of Fenichel’s theorem

We assume that in the previous model, ε is a small positive parameter. We add the equation $\frac{d\varepsilon}{d\tau} = 0$. For all $N \geq 0$ and all $P \geq 0$, $(0, 0, N, P, 0)$ is an equilibrium. The jacobian matrix of the system at this equilibrium is:

$$J(N, P) = \begin{pmatrix} -\beta P & 0 & 0 & 0 & * \\ 0 & -\lambda(a + \alpha)N & 0 & 0 & * \\ 0 & 0 & 0 & 0 & * \\ 0 & 0 & 0 & 0 & * \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{17}$$

This matrix admits three vanishing eigenvalues and 2 negative eigenvalues $-\beta P$ and $-\lambda(a + \alpha)N$, which are strictly negative as long as $N > 0$ and $P > 0$. We can apply the following theorem due to Fenichel [18,19]:

Theorem 1 (Fenichel, 1979). *With the above notation, for every compact set $\mathcal{K} \subset (\mathbb{R}^{++})^2$, and for every positive integer $k \in \mathbb{N}$, there exists a strictly positive real number ε_0 and a C^k map denoted by Ψ , defined from $\mathcal{K} \times [0, \varepsilon_0]$ to \mathbb{R}^2 such that:*

- $(\Psi(N, P, 0), N, P, 0)$ is an equilibrium for all $(N, P) \in \mathcal{K}$;
- the graph of Ψ is an invariant manifold \mathcal{M} under the flow of the differential system;
- this graph is tangent to the centre space $E^C(N, P)$ of $J(N, P)$ at each equilibrium point $(\Psi(N, P, 0), N, P, 0)$.

Let $\varepsilon \in [0, \varepsilon_0]$ as in the previous theorem and let denote by \mathcal{M}_ε the graph of the map Ψ restricted to $\mathcal{K} \times \{\varepsilon\}$. Since the non vanishing eigenvalues have strictly negative real parts for all $(N, P) \in \mathcal{K}$, the manifold \mathcal{M}_ε is attracting for model (16), that is all trajectories starting from an initial condition $(N_U(0), P_U(0), N(0), P(0))$ such that $(N(0), P(0)) \in \mathcal{K}$ jumps to \mathcal{M}_ε and remains close to this manifold as long as the required conditions of Theorem 1 are satisfied.

Remark. note that if N or P is close to 0, one or both negative eigenvalues is also close to 0.

In order to apply this theorem, we first consider the case $\varepsilon = 0$, which gives the following system:

$$\begin{cases} \frac{dN_U}{d\tau} = -\beta N_U(P - P_U) \\ \frac{dP_U}{d\tau} = -\lambda(a + \alpha)(N - N_U)P_U \\ \frac{dN}{d\tau} = 0 \\ \frac{dP}{d\tau} = 0 \\ \frac{d\varepsilon}{d\tau} = 0 \end{cases} \tag{18}$$

called the fast system. Clearly, (N_U, P_U) tends to $(0, 0)$, or in other words, the function Ψ of the previous theorem satisfies $\Psi(N, P, 0) = (0, 0)$. Let us consider a compact set \mathcal{K} in the positive domain of the (N, P) variables. There is a positive number ε_0 such that for all $\varepsilon < \varepsilon_0$, there exists an invariant manifold \mathcal{M}_ε on which the model (16) can be written as the following slow system:

$$\begin{cases} \frac{dN}{dt} = r_1 N \left(1 - \frac{N}{K_1}\right) - (a + \alpha)NP + O(\varepsilon) \\ \frac{dP}{dt} = e(a + \alpha)\varphi NP - (m + \nu)P + O(\varepsilon) \end{cases} \tag{19}$$

where $t = \varepsilon\tau$, $r_1 = r\phi - \mu$ and $K_1 = K \frac{r_1}{r}$.

Assumption (A₁). we assume here that $r_1 < 0$.

It follows from assumption (A₁) that $\frac{dN}{dt} < 0$ and thus $N(t)$ tends to $O(\varepsilon)$ as t goes to infinity. Consequently, $P(t)$ shall also go to $O(\varepsilon)$. These results follow from the reduced slow system. But as we said above, when N or P become too small, the reduction Fenichel’s theorem does not apply anymore.

A.1.2. Fluctuations: result and proof

We show now that fluctuations take place under some conditions that are provided in the proof. We first introduce a notation that allows to give a clear statement of the result. We denote by ρ , the following combination of parameters:

$$\rho = \frac{r\alpha^2(m + \nu)\left(r(m + \nu) + (\mu - r\phi)e\varphi K(a + \alpha)\right)}{\left((r(1 - \phi) + \mu)Ke\varphi(a + \alpha)^2 + (r\phi - \mu)\alpha Ke\varphi(a + \alpha) - 2r\alpha(m + \nu)\right)^2} \tag{20}$$

Assumption (A₂). We assume that the combination of parameters in formula (20) is very small: $\rho = O(\varepsilon)$, while $\alpha = O(1)$. Note that we provide in the remark at the end of the proof several ways to satisfy this assumption.

Theorem 2. *We assume (A₁) and (A₂) for model (16). For all $\xi_N > 0$ arbitrarily small, there exists $\varepsilon > 0$ such that for all ε in $[0; \varepsilon_0]$, there exists a compact set in the phase space such that for all solutions with an initial conditions in this compact set, there exists a sequence*

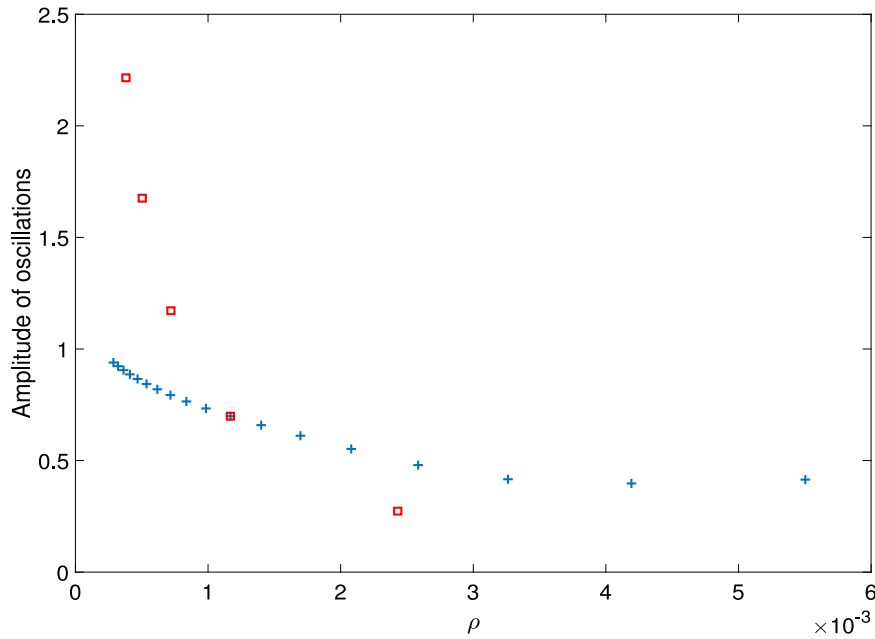


Fig. 8. This figure illustrates the relation between the amplitude of the fluctuations of the prey abundance and the value of the parameter ρ . The simulations are done with model (2) and the parameter values are $\epsilon = 0.01, \beta = 0.2, e = 0.2, m = 0.01, r = 1, \alpha = 0.7, \lambda = 1, \mu = 0.1, \nu = 0.1, \varphi = 1, \phi = 0.05$, with initial conditions $(N_U(0), N_I(0), P_U(0), P_I(0)) = (0.1, 0.04, 0.42, 0.05)$. Two sets of simulations are done. The first one (blue crosses) is obtained by varying a from 0.8 to 2.5 (note that increasing a leads to decrease ρ here). The second one (red symbols) is obtained by varying K from 2 to 6 (note again that an increase of K corresponds here to a decrease of ρ). It turns out as explained in the text that the lower the parameter ρ , the larger the fluctuation amplitude. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

$\{T_n\}_{n \in \mathbb{N}}$ with $T_n \rightarrow +\infty$ when n goes to infinity, $N(T_n) = O(\xi_N)$ if n is even and $N(T_n) = N_1^* + O(\epsilon)$ where $N_1^* = \frac{m + \nu}{e\varphi(a + \alpha)}$ for all odd n . This means that the prey population fluctuates with an amplitude of order N_1^* (which does not decrease to 0 when ϵ goes to 0).

The technical condition $\rho = O(\epsilon)$ is a sufficient condition in our theorem. We illustrate this on Fig. 8, where several combinations of parameters giving different values of ρ are used to simulate the model (2). For low values of ρ , the model exhibit fluctuations and the amplitude of these fluctuations decreases when ρ is increased.

To prove the theorem, we first need the two following lemmas.

Lemma 3. Let $\xi > 0$ a real number. There exists a compact set $\mathcal{K} \subset \{(N, P)/N \geq 0, P \geq 0\}$ such that all trajectories of model (19) starting in \mathcal{K} leave \mathcal{K} by the set $\{N = \xi\}$ if ϵ is small enough.

Proof. Let P_M be a large real number. We define M_1 (see Fig. 9) as the intersection between the trajectory of model (19) starting from $M_0 = (K, P_M)$ and the line $\{N = \xi\}$, and let Γ_{01} the trajectory between M_0 and M_1 : this trajectory exists since $N(t)$ tends to 0. Let us now remind that $N_1^* = \frac{m + \nu}{e\varphi(a + \alpha)}$ and that the straight line $N = N_1^*$ divides the half plane in two region, P is increasing when $N > N_1^*$ and decreasing when $N < N_1^*$. We then consider the point M_2 at the intersection between the trajectory of model (19) starting from $M_3 = (N_1^*, \xi)$ and the line $\{N = \xi\}$. We denote by Γ_{32} the trajectory between M_2 and M_3 (note that we can chose P_M large enough such that the trajectory Γ_{01} is above the trajectory Γ_{32} in the 2D phase space $\{(N, P)/N \geq 0, P \geq 0\}$). We finally define the point $M_4 = (K, \xi)$. The set bounded by Γ_{01} , the line $[M_1, M_2]$, the trajectory Γ_{32} , the line $[M_3, M_4]$ and the line $[M_4, M_0]$ is a compact set denoted \mathcal{K} . It is straightforward to check that all trajectories starting in \mathcal{K} leave this set by the line $[M_1, M_2] \subset \{N = \xi\}$. \square

Lemma 4. Let ξ be a positive real such that $\xi < N_1^*$ and ϵ small enough. All trajectories of model (16) starting outside regions $\mathcal{R}_N = \{(N_U, P_U, N, P); N \leq \xi\}$ and $\mathcal{R}_P = \{(N_U, P_U, N, P); P \leq \xi\}$ but in the vicinity of an invariant manifold of Fenichel's theorem will enter \mathcal{R}_N and then leave it through \mathcal{R}_P .

Proof. According to Lemma 3, the trajectory enters the region \mathcal{R}_N . Once in this region, from the last equation of model (16), one gets:

$$\begin{aligned} \frac{dP}{dt} &\leq e(a + \alpha)N\varphi P - (m + \nu)P \\ &\leq e((a + \alpha)\xi\varphi - (m + \nu))P \end{aligned}$$

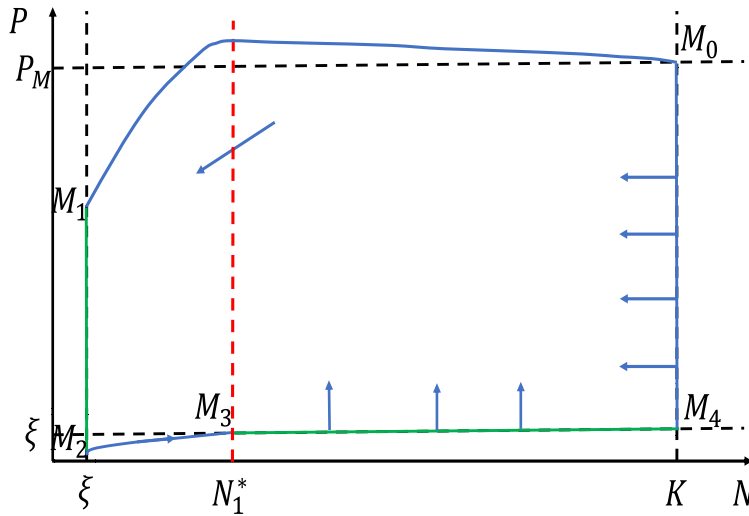


Fig. 9. Scheme illustrating the set in the (N, P) plane from which all trajectories of model (19) leave at the boundary $\{N = \xi_N\}$, as set in the proof of Lemma 3.

and it follows that $P(t) \leq P_0 \exp((e(a + \alpha)\xi\varphi - (m + \nu))t)$ which tends to 0, thus the trajectory will enter the region \mathcal{R}_p . \square

We can now prove the theorem.

Proof. Let us consider a trajectory of model (16) which is initially neither in region \mathcal{R}_N nor in region \mathcal{R}_p . For ϵ small enough and according to Fenichel’s theorem, after a short transient time, this trajectory is close to an invariant 2-dimensional manifold on which the dynamics is described by model (19). Lemmas 3 and 4 allow us to conclude that there is a time $T_0 > 0$ such that the trajectory enters \mathcal{R}_N at time T_0 , thus $N(T_0) = \xi$. Moreover, the trajectory will later enter region \mathcal{R}_p .

In order to follow the trajectory after it enters region \mathcal{R}_p with ξ a small number, we make the following rescaling. We set $P_U = \epsilon^2 \bar{P}_U$ and $P_I = \epsilon \bar{P}_I$ in model (2), which leads to the following model:

$$\begin{cases} \frac{dN_U}{d\tau} = -\epsilon\beta N_U \bar{P}_I + \epsilon \left(r(N_U + \phi N_I) - rN_U \frac{N_U + N_I}{K} \right) + O(\epsilon^2) \\ \frac{dN_I}{d\tau} = \epsilon\beta N_U \bar{P}_I - \epsilon \left(rN_I \frac{N_U + N_I}{K} + \mu N_I \right) + O(\epsilon^2) \\ \frac{d\bar{P}_U}{d\tau} = -\lambda(a + \alpha)N_I \bar{P}_U + e \left(aN_U + (a + \alpha)N_I \right) \varphi \bar{P}_I + O(\epsilon) \\ \frac{d\bar{P}_I}{d\tau} = \epsilon\lambda(a + \alpha)N_I \bar{P}_U - \epsilon(m + \nu)\bar{P}_I \end{cases} \quad (21)$$

model (21) is a slow-fast system to which we can apply Theorem 1. From this, we can show that \bar{P}_U shall reach a so-called "fast" equilibrium which depends on N_U , N_I and \bar{P}_I . Moreover, as long as N_I is not too small (of order of ϵ), the long-term dynamics (or so called slow dynamics) is governed by:

$$\begin{cases} \frac{dN_U}{dt} = -\beta N_U \bar{P}_I + r(N_U + \phi N_I) - \frac{r}{K} N_U (N_U + N_I) + O(\epsilon) \\ \frac{dN_I}{dt} = \beta N_U \bar{P}_I - r \frac{N_I}{K} (N_U + N_I) - \mu N_I + O(\epsilon) \\ \frac{d\bar{P}_I}{dt} = e(aN_U + (a + \alpha)N_I) \varphi \bar{P}_I - (m + \nu)\bar{P}_I + O(\epsilon) \end{cases} \quad (22)$$

We now study this model by neglecting the terms of order of ϵ . There are 2 equilibria with $\bar{P}_I \neq 0$. Indeed, vanishing the \bar{P}_I equation and assuming that $\bar{P}_I \neq 0$, we obtain:

$$N_I = \frac{m + \nu}{e\varphi(a + \alpha)} - \frac{a}{a + \alpha} N_U = N_1^* - \frac{a}{a + \alpha} N_U \quad (23)$$

In order to find the number of uninfected prey, we add the two first equations in model (22) and replace N_I by the previous expression, we thus obtain a second degree equation:

$$c_2 N_U^2 + c_1 N_U + c_0 = 0$$

where

$$\begin{aligned} c_2 &= \frac{r}{K} \left(\frac{\alpha}{a + \alpha} \right)^2 \\ c_1 &= \frac{2r}{K} \frac{\alpha}{a + \alpha} N_1^* - r - (\mu - r\phi) \frac{a}{a + \alpha} \\ c_0 &= (\mu - r\phi) N_1^* + \frac{r}{K} (N_1^*)^2 \end{aligned}$$

Note that we are assuming that the parameters satisfy that

- $c_1 < 0$;
- $c_2 > 0$;
- under assumption (A_1) , $c_0 > 0$.

Let us define Δ as:

$$\Delta = c_1^2 - 4c_0c_2 = c_1^2 \left(1 - 4 \frac{c_0c_2}{c_1^2} \right) \tag{24}$$

It is a straightforward calculation to show that:

$$\Delta = c_1^2 (1 - 4\rho)$$

where ρ is defined as:

$$\rho = \frac{r\alpha^2(m + \nu) \left(r(m + \nu) + (\mu - r\phi)e\phi K(a + \alpha) \right)}{\left((r(1 - \phi) + \mu)Ke\phi(a + \alpha)^2 + (r\phi - \mu)\alpha Ke\phi(a + \alpha) - 2r\alpha(m + \nu) \right)^2}$$

We consider a parameter combination such that $\rho = O(\epsilon)$ (assumption (A_2)), thus we can write ρ as $\rho = \epsilon\bar{\rho}$. According to these assumption and notation, $\Delta = c_1^2(1 - 4\epsilon\bar{\rho})$, thus $\Delta > 0$ and the solutions of Eq. (24) are real numbers defined as:

$$\begin{aligned} N_U^+ &= \frac{-c_1 + \sqrt{\Delta}}{2c_2} > 0 \\ N_U^- &= \frac{-c_1 - \sqrt{\Delta}}{2c_2} > 0 \end{aligned}$$

Vanishing the equation of N_U allows us to calculate the equilibria values for \bar{P} and this shows that two positive equilibria exists. Since $c_1 < 0$, one can write $\sqrt{\Delta}$ as:

$$\sqrt{\Delta} = -c_1 + O(\epsilon) \tag{25}$$

which implies that we can write $N_U^- = \epsilon\bar{N}_U^-$ and $N_U^+ = -\frac{c_1}{c_2}$. For each of these values of N_U , we can associate an equilibrium of model (22). We show in Lemma 5 that the equilibrium associated to N_U^+ is not possible in the positive domain. Thus there exists only one positive equilibrium for model (22). The density of infected predators associated to this equilibrium is given by:

$$\bar{P}_I^- = \frac{r}{\beta} \left(1 + \phi \frac{N_1^*}{N_U^-} + \phi \frac{a}{a + \alpha} - \frac{1}{K} \left(N_1^* + \frac{\alpha}{a + \alpha} N_U^- \right) \right) \tag{26}$$

With the assumptions of the theorem, it follows that $\bar{P}_I^- = O(1/\epsilon)$ and thus $P_I^- = O(1)$. More precisely, P_I^- can be as large as we want provided ρ is small enough. And since $P^- \geq P_I^-$, P can be larger than the number ξ chosen in Lemma 4 for instance. Thus there exists a time $T > T'_0$ such that $P(T) = \xi$. Since for $t \in]T_0; T'_0[$ the trajectory is in \mathcal{R}_P , P is thus increasing at time T'_0 while it was decreasing at time T_0 . There is thus a time $T_1 \in]T_0; T'_0[$ such that $\frac{dP}{dt} = 0$ with $P \neq 0$. This occurs in \mathcal{R}_P for $N(T_1) = N_1^* + O(\epsilon)$. Thus we have $N(T_0) = O(\xi_P)$ and $N(T_1) = N_1^* + O(\epsilon)$. When P_I is large enough, the approximation of model (16) by (22) is no longer valid. It means that the trajectory of model (16) is entering I at time T'_0 , thus the same reasoning can be applied recursively to show Theorem 2.

It remains to prove that the equilibrium (N_U^-, N_I^-, P_I^-) is stable when $N - U^- = O(\epsilon)$ for model (22). In order to do this, we consider the Jacobian matrix of the corresponding differential system at the equilibrium:

$$J_{E_q} = \epsilon \begin{pmatrix} -\beta\bar{P}_I^- + r - \frac{r}{K}(2N_U^- + N_I^-) & r\phi - r\frac{N_U^-}{K} & -\beta N_U^- \\ \beta\bar{P}_I^- - r\frac{N_I^-}{K} & -r\frac{N_U^- + 2N_I^-}{K} - \mu & \beta N_U^- \\ e\alpha\phi\bar{P}_I^- & e(a + \alpha)\phi\bar{P}_I^- & 0 \end{pmatrix}$$

The eigenvalues of this matrix are the solutions of the following characteristic equation:

$$\gamma^3 + a_1\gamma^2 + a_2\gamma + a_3 = 0$$

with

$$a_1 = r\phi\frac{N_I^-}{N_U^-} + 3r\frac{N_I^-}{N_U^-} - r\frac{N_U^- + N_I^-}{K} + O(\epsilon) > 0$$

$$\begin{aligned}
 a_2 &= (\beta P_I^- + r - r \frac{N_I^-}{K}) 2r \frac{N_I^-}{K} - r\phi(\beta P_I^- - r \frac{N_I^-}{K}) + O(\epsilon) \\
 a_3 &= \left(\beta e(a + \alpha)\phi r(1 + \frac{N_I^-}{K}) \bar{P}_I^- + (\mu - r\phi)\beta e a \phi \bar{P}_I^- + 2\beta e a \phi \frac{r}{K} N_I^- \bar{P}_I^- \right) N_U^- + (\epsilon^2)
 \end{aligned}$$

The equilibrium is locally asymptotically stable if (Routh - Hurwitz criterion) one has:

$$\begin{aligned}
 a_1 &> 0 \\
 a_3 &> 0 \\
 a_1 a_2 &> a_3
 \end{aligned}$$

Since $N_U^- = O(\epsilon)$, it is straightforward to see that a_1, a_2 and a_3 are all strictly positive and since $a_3 = O(\epsilon)$, one gets that $a_1 a_2 > a_3$. Thus the equilibrium is stable. \square

Remark. actually, we built an continuous map from \mathcal{K} to \mathcal{K} which thus has a fixed point from Brouwer's theorem, and this fixed point corresponds to a periodic solution of model (16). Thus the model admits a periodic solution.

Remark. the relation $\rho = O(\epsilon)$ can be reached by many ways. Among them, we mention $K = O(1/\epsilon)$ or $a = O(1/\epsilon^{1/3})$ for instance.

Lemma 5. The equilibrium (N_U^+, N_I^+, P_I^+) when $\rho = 0(\epsilon)$ in the proof of Theorem 2 is not in the positive domain.

Proof. In order to prove this result, we first show that $N_U^+ + N_I^+ > K$. Indeed, we have $N_U^+ = -\frac{c_1}{c_2} + O(\epsilon)$ and a straightforward computation leads to:

$$N_U^+ + N_I^+ = \frac{a + \alpha}{a} (1 + (\frac{\mu}{r} - \phi) \frac{a}{a + \alpha}) K > K \tag{27}$$

The region of the phase space $\{(N_U, N_I, P_I), 0 \leq N_U, 0 \leq N_I, 0 \leq P_I, K < N_U + N_I\}$ cannot contain any equilibrium because the derivative of $N_U + N_I$ is strictly negative in this region for model (22). Thus if the derivative of this expression is vanishing, it needs that $P_I < 0$. This completes the proof. \square

A.2. Appendix 2: Case where $r\phi - \mu > 0, K_1 < N_1^* < K$

In this case, for adequate initial conditions, the complete model (3) can be approximated by the reduced model (8) for a finite length of time. Indeed, as said in the main text, since we assume that $K_1 < N_1^*$, the predator population goes to extinction in the reduced model, and since the state variable $P(t)$ is decreasing to 0, the assumptions required for the Fenichel's theorem are not satisfied after a given time T_0 . More precisely, let us consider a compact set $\mathcal{K} \subset \{(N, P)/N > 0, P > 0\}$, there exists $\epsilon_0 > 0$ such that for all $\epsilon < \epsilon_0$, the theorem applies as long as the slow state vector $(N(t), P(t))$ remains in \mathcal{K} . Since $P(t)$ goes to 0 with the slow system, there is a time T_0 at which the slow trajectory leaves the compact set \mathcal{K} . After this time, it is required to consider the full system but since we know that $P(t)$ is close to 0 (and then that P_u is small as well), we have to consider the full system in a region of the phase space where the following change of variables is meaningful:

$$P = \epsilon \bar{P} \quad P_U = \epsilon \bar{P}_U \tag{28}$$

With this change of variables, the full model reads:

$$\begin{aligned}
 N'_U &= -\beta N_U (\bar{P} - \bar{P}_U) + \epsilon r(\phi N + (1 - \phi)N_U) - \epsilon r N_U \frac{N}{K} + O(\epsilon^2) \\
 \bar{P}'_U &= -\lambda(a + \alpha)(N - N_U)\bar{P}_U + \epsilon (e((a + \alpha)N - \alpha N_U)(\phi \bar{P} + (1 - \phi)\bar{P}_U) - m\bar{P}_U) \\
 N' &= \epsilon \left(r(\phi N + (1 - \phi)N_U) - rN \frac{N}{K} - \mu(N - N_U) \right) + O(\epsilon^2) \\
 \bar{P}' &= \epsilon (e((a + \alpha)N - \alpha N_U)(\phi \bar{P} + (1 - \phi)\bar{P}_U) - (m + \nu)\bar{P} + \nu\bar{P}_U)
 \end{aligned} \tag{29}$$

where the time derivative is done with respect to the fast time τ .

Again, under conditions required by the Fenichel's theorem, this model can be reduced to a 3-dimensional system where \bar{P}_U can be replaced by 0 because in the previous model, the fast part contains only the \bar{P}_U equation and this equation has only $\bar{P}_U = 0$ as a globally hyperbolic stable equilibrium. This equilibrium is hyperbolic stable as long as N and N_U are not too close.

The reduced model reads:

$$\begin{aligned}
 N'_U &= -\beta N_U \bar{P} + r(\phi N + (1 - \phi)N_U) - rN_U \frac{N}{K} + O(\epsilon) \\
 N' &= r(\phi N + (1 - \phi)N_U) - rN \frac{N}{K} - \mu(N - N_U) + O(\epsilon) \\
 \bar{P}' &= e\phi((a + \alpha)N - \alpha N_U)\bar{P} - (m + \nu)\bar{P} + O(\epsilon)
 \end{aligned} \tag{30}$$

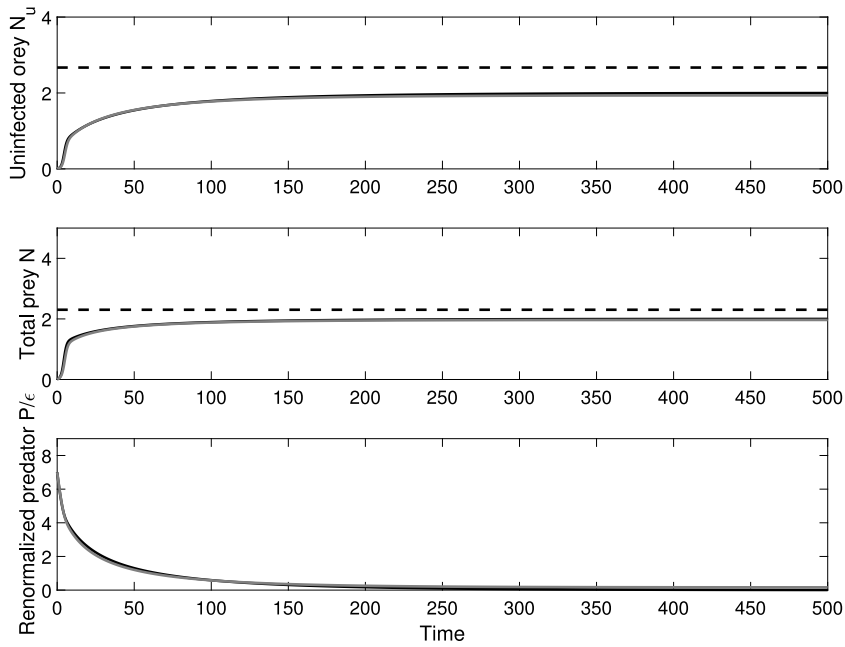


Fig. 10. This figure compares the complete and the reduced models when the equilibrium E_1 of model (30) is stable. The grey curve corresponds to the complete model (2) while the black one is got with the reduced model (30). As it can be seen, the curves of the complete and reduced models are very close. Parameter values are $\beta = 0.2, a = 2.5, e = 0.2, K = 2, m = 0.01, r = 2, \phi = 0.1, \lambda = 1, \mu = 0.1, \nu = 0.1, \varphi = 0.1, \alpha = 0.7$ and $\epsilon = 0.01$.

where the time derivative is now with respect to the slow time t . We study this model neglecting the terms $O(\epsilon)$ in each equation. In order to describe the equilibria in a simple way, let us introduce some notations. We set:

$$\delta = \frac{\alpha}{a + \alpha} \quad B = r_1 + \frac{r - r_1}{\delta} \quad \text{and} \quad \Delta = B^2 - 4 \frac{r}{K} \frac{r - r_1}{\delta} N_1^* \tag{31}$$

model (30) may admit 3 equilibria:

$$E_1 = \begin{pmatrix} K \\ K \\ 0 \end{pmatrix} \quad E_2 = \begin{pmatrix} N_U^+ \\ N^+ \\ P^+ \end{pmatrix} \quad E_3 = \begin{pmatrix} N_U^- \\ N^- \\ P^- \end{pmatrix} \tag{32}$$

where

$$N^+ = K \frac{B + \sqrt{\Delta}}{2r}, \quad N_U^+ = \frac{N^+ - N_1^*}{\delta} \quad \bar{P}^+ = \frac{r}{\beta} \left(\phi \frac{N^+}{N_U^+} + (1 - \phi) - \frac{N^+}{K} \right) \tag{33}$$

and similarly

$$N^- = K \frac{B - \sqrt{\Delta}}{2r}, \quad N_U^- = \frac{N^- - N_1^*}{\delta} \quad \bar{P}^- = \frac{r}{\beta} \left(\phi \frac{N^-}{N_U^-} + (1 - \phi) - \frac{N^-}{K} \right) \tag{34}$$

Lemma 6. *The equilibrium E_1 is locally stable for model (30) if and only if $K < \frac{m + \nu}{\epsilon \varphi a}$. Moreover, under the same conditions, $(K, 0, K, 0)$ is a stable equilibrium for model (3).*

Proof (Sketch of Proof). The local stability of E_1 easily follows from the Jacobian matrix study. However, in this case, since N_U and N are getting closer, the trajectory will leave the domain of application of the reduction theorem (30). Since this occurs when N and N_U are close, we consider the full model still setting $P = \epsilon \bar{P}$ and $P_U = \epsilon \bar{P}_U$, but also adding $N_u = N - \epsilon \bar{N}_1$. Doing this change of variables leads again to conclude that $\bar{P}(t)$ and $\bar{P}_U(t)$ tend to 0 and then $N(t)$ and $N_U(t)$ tend to K . Thus the equilibrium E_1 remains a local attractor for the state (N_U, N, \bar{P}) for the complete model and \bar{P}_U also goes to zero. \square

In this case, the epidemic is excluded from the system and it is illustrated on Fig. 10.

We now state the lemma for the existence of positive equilibria.

Lemma 7. *Let us assume that r, ϕ and μ are such that $r_1 = r\phi - \mu$ be strictly positive. There exists a range of values of m, ν, e and φ such that the two following inequalities are simultaneously satisfied:*

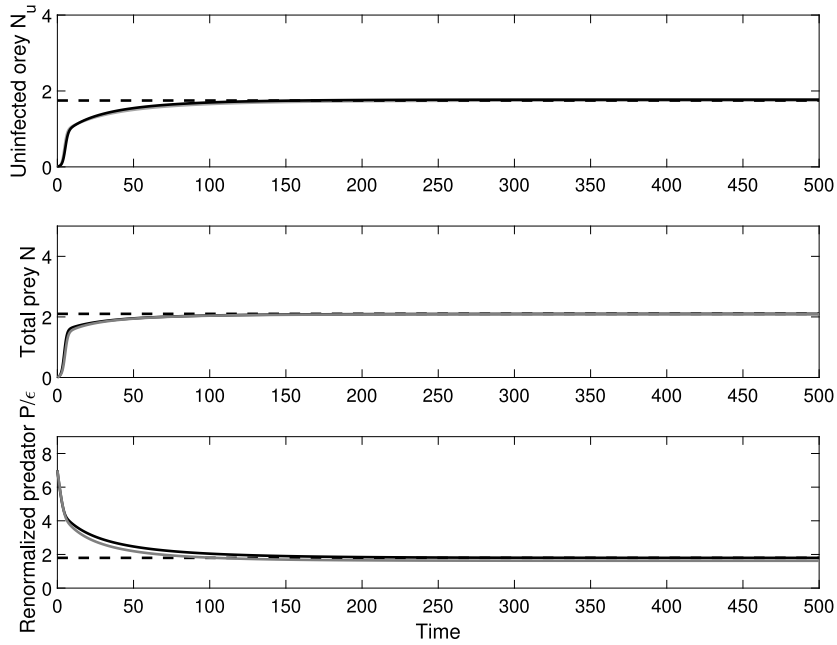


Fig. 11. This figure compares the complete and the reduced models when the equilibrium E_3 of model (30) is stable. The grey curve corresponds to the complete model (2) while the black one is got with the reduced model (30). As it can be seen, the curves of the complete and reduced models are very close. Moreover, the dashed lines represent the equilibrium E_2 coordinates. Parameter values are $\beta = 0.2$, $a = 2.5$, $e = 0.2$, $K = 2.5$, $m = 0.01$, $r = 2$, $\phi = 0.1$, $\lambda = 1$, $\mu = 0.1$, $\nu = 0.1$, $\varphi = 0.1$, $\alpha = 0.7$ and $\epsilon = 0.01$. In this case, we have $K > \frac{m + \nu}{e\varphi a}$.

- $K_1 < N_1^*$
- $\Delta > 0$

Furthermore, under these conditions, the model (30) admits 2 positive equilibria E_2 and E_3 provided previously.

Proof. The first item can be written as follows:

$$\frac{m + \nu}{e\varphi} > \frac{r_1}{r} K(a + \alpha) \tag{35}$$

Moreover, the second item is equivalent to $B^2 > 4 \frac{r}{K} \frac{r - r_1}{\delta} N_1^*$. This inequality can be written as follows:

$$\frac{m + \nu}{e\varphi} < \frac{\alpha K}{4r(r - r_1)} \left(r_1 + \frac{r - r_1}{\delta} \right)^2 \tag{36}$$

Thus in order to get both items together, the next inequality is necessary and sufficient:

$$\frac{r_1}{r} K(a + \alpha) < \frac{\alpha K}{4r(r - r_1)} \left(r_1 + \frac{r - r_1}{\delta} \right)^2 \tag{37}$$

Some simple calculus allows to show that this last inequality is equivalent to:

$$((\delta + 1)r_1 + r)^2 > 0 \tag{38}$$

which is obviously true. Consequently, all combinations of parameters m , ν , e and φ satisfying the following constraint fulfil the items of the lemma.

$$\frac{r_1}{r} K(a + \alpha) < \frac{m + \nu}{e\varphi} < \frac{\alpha K}{4r(r - r_1)} \left(r_1 + \frac{r - r_1}{\delta} \right)^2 \tag{39}$$

Under the conditions provided by these items, N^- and N^+ are well defined and are both positive, because from the definition of Δ it is clear that $B > \sqrt{\Delta} > 0$ and thus $0 < N^- < N^+$. The inequality $N_1^* > K_1$ is equivalent to $N^- > N_1^*$ and thus in this case we also have $N^+ > N_1^*$ which ensures that N_U^+ and N_U^- are positive. Finally, regarding \bar{P}^- , we have:

$$\bar{P}^- = \frac{r}{\beta} \left(\phi \left(\frac{N^-}{N_U^-} - 1 \right) + 1 - \frac{N^-}{K} \right) \tag{40}$$

Since $K > N^- > N_U^-$, the right hand side of the previous equality is obviously positive. We also show that $\bar{P}^+ > 0$ in a very similar way.

As a consequence, model (30) admits E_2 and E_3 as positive equilibria. \square

In the latter case, we illustrate on Fig. 11 that the reduced model (30) and the full model (3) are very close and that the system reaches the equilibrium E_2 (dashed line).

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