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ABSTRACT

Parasites are important components of food webs. Although their direct effects on hosts are well-studied, indirect impacts on trophic networks, thus on non-host species, remain unclear. In this study, we investigate the consequences of parasitism on coexistence and stability within a simple trophic module: one predator consuming two prey species in competition. We test how such effects depend on the infected species (prey or predator). We account for two effects of parasitism: the virulence effect (parasites affect the infected species intrinsic growth rate through direct effects on fecundity or mortality) and the interaction effect (increased vulnerability of infected prey or increased food intake of infected predators). Results show that coexistence is favored when effects have intermediate intensity. We link this result to modifications of direct and apparent competitions among prey species. Given a prey infection, accounting for susceptible-infected population structure highlights that coexistence may also be reduced due to predator-parasite competition. Parasites affect stability by modulating energy transfer from prey to predator. Predator infection therefore has a stabilizing effect due to increased energy fluxes and/or predator mortality. Our results suggest that parasites potentially increase species coexistence. Precise predictions however require an assessment of various parasite effects. We discuss the implications of our results for the functioning of trophic networks.

Keyword: competition, predator-parasite relationship, interaction effect, paradox of enrichment, virulence.

Introduction

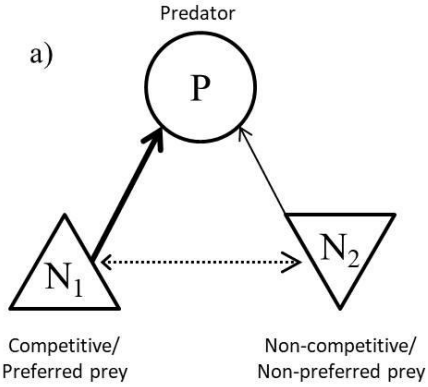
Many studies on food webs show that parasites are omnipresent, with a high biomass (Kuris et al., 2008) and making a large proportion of antagonistic interactions (Hudson et al., 2006; Amundsen et al., 2009). Although parasites are expected to have large impacts on diversity and stability (Poulin, 2010; Wood and Johnson, 2015), exact consequences appear difficult to estimate due to complexity of ecological networks and the diversity of parasite effects (Hatcher et al., 2006, 2014; Welch and Harwood, 2011). We therefore need an integrative perspective on the effects of parasitism in multi-species systems (ecosystem parasitology, Hatcher and Dunn, 2011; Tompkins et al., 2011). In the present work, we analyze the consequences of parasitism on coexistence and stability, using a trophic module approach.

We investigate two effects of parasites, called hereafter "virulence effects" and "interaction effects". Virulence effects embody the direct consequences of infection parasites typically reduce the fecundity and/or survival of their hosts (Coors and De Meester, 2011), thereby impacting their intrinsic growth rates. Such virulence effects are well-documented: Decaestecker et al. (2003) for instance showed reduced fecundity and increased mortality of *Daphnia magna* when infected by bacteria (*Pasteuria*) or fungi (Microsporidia). Similar effects of trematodes on *Daphnia obtusa* have been observed by Schwartz and Cameron (1993). Such virulence effects may propagate at the population level, decreasing host biomass and affecting competitive hierarchies among species (Decaestecker et al., 2015).

By affecting the phenotype of their hosts, parasites may also change the trophic interactions involving these hosts in the network (hereafter, "interaction effects"). This is well established for trophically-transmitted parasites as natural selection on the parasite may affect its host appearance or behavior (such modifications can then be seen as an extended phenotype of the parasite) in a way that increases its vulnerability to predation (trophic manipulation), thereby facilitating transmission (Lefèvre et al., 2009; Cézilly et al., 2010; Thomas et al., 2010; Jacquin et al., 2013). However, modifications of predator-prey interactions may also be simply an indirect effect of parasitism happening in the absence of trophic transmission (Peterson and Page, 1988; Hudson et al., 1992; Duffy et al., 2005). For instance, *Daphnia* infected by *Pasteuria ramosa* have a red coloration and are more catchable by *Anisop* (Goren and Ben-Ami, 2017). An infected predator may also increase its food intake (predation rate) to compensate the energetic costs incurred by the infection (Bernot and Lamberti, 2008; Dick et al., 2010; Lettini and Sukhdeo, 2010).

We here assess such consequences, defining coexistence as the possibility of maintaining all species (predators and prey species) and stability based on the occurrence of oscillating population dynamics. In our system, coexistence among the two prey species depends on the balance between direct (i.e. resource based) and apparent competition (i.e. competition mediated by the predator presence, Holt [1977]). As illustrated by classical experiments (Gause, 1934), direct competition is an important constraint for coexistence. The inclusion of a predator in a competitive system would affect coexistence through apparent competition (Holt, 1977). In such situations, one prey negatively affects the other prey by increasing the predator population. By combining the two competitions (direct and apparent competitions), coexistence is allowed when the most competitive species is also the most vulnerable prey (Holt et al., 1994). Consequently, coexistence requires a balance between direct and apparent competitions that may be affected by parasites. Virulence effects may for instance reduce competitive ability of the host species. Competition between *Daphnia magna* and *D. pulex*, usually favoring *D. magna*, may be reversed when *D. magna* are infected by microsporidian and bacteria (Decaestecker et al., 2015). Now consider parasitism on the predator. By decreasing predator density, a parasite with virulent effects may decrease apparent competition and thereby favor the preferred prey species, which is also the best direct competitor. Therefore, in prey infection as in predator infection, parasites with virulent effects likely affect coexistence by changing the relative intensity of the two types of competition (Fig. 1b). Interaction effects may be equally important. When they modify predation, parasites directly alter apparent competition. An increased

a)



b) **Predictions on coexistence**

Hosts \ Effects	Virulence	Interaction
Competitive/Preferred prey (N_1)	Maximal coexistence at intermediate intensity (balance between direct and apparent competition)	
Predator (P)		

c) **Predictions on stability**

Hosts \ Effects	Virulence		Interaction	
	Interaction heterogeneity[1]	Energy flow / Predator loss[2]	Interaction heterogeneity[1]	Energy flow / Predator loss[2]
Competitive/Preferred prey (N_1)	No effect	No effect	Stabilize (heterogenization)	Destabilize (increase energy flow)
Predator (P)	No effect	Stabilize (increase predator loss)	No effect or Destabilize (homogenization)	Destabilize (increase energy flow)

1 Based on McCann et al. (1998)
2 Based on Rip and McCann (2011)

Figure 1. Presentation of the trophic module and predictions. a) The module before infection consists of *P*, the predator, N_1, the competitive/preferred prey, and N_2, the non-competitive/non-preferred prey; solid arrow, the predation, dashed arrow, the direct competition. Predictions on how coexistence (Table b) and stability (Table c) depend on the infection scenario (identity of species infected, virulence or interaction effects).

predation on the best direct competitor, should favor the other prey species. Such parasite-induced modification of predation by snails (*Littorina littorea)* on ephemeral macroalgae, for instance, modify biomass and composition of intertidal communities (Wood et al., 2007). Under such scenarios, parasites favor coexistence at intermediate effects, while extremely high or low effects decrease coexistence by altering the balance between the two types of competition (Fig. 1b).

We also analyze the effects of parasites on the stability of the system (Fig. 1c). Considering the paradox of enrichment, a classical result in ecological theory is that stability decreases when larger energy flows (predation rate) occur for a given predator loss rate (predator mortality rate) (Rosenzweig, 1971; Rip and McCann, 2011). Consider virulence effects. For an infected prey, we do not expect effects on stability as neither predation rate nor predator mortality rate is modified. For an infected predator, however, infection increases the predator mortality rate and thus stabilizes the system. Now consider the interaction effect. As it directly increases the predation rate, it is expected to destabilize the system.

Next to such “energy flow” aspects, classical ecological theory also suggests that stability is enhanced when weak and strong interactions coexist within a trophic module (McCann et al., 1998). Heterogeneous systems made of few strong links and many weak links are more stable than homogeneous systems. Such stability constraints are not affected by virulence effects as they do not change the balance of interactions. On the contrary, interaction effects directly modify the distribution of interaction strengths and increasing predation on the most consumed prey should increase heterogeneity, and thus stability. When the predator is infected, the changes in the distribution of interaction strengths depend on how the parasite affects interaction rates. If all interaction rates increase in the same proportion stability should not be modified.

When all rates increase by a given, fixed change, interaction strengths should be homogenized and the system destabilized.

Many studies of parasite effects focus on one species (for instance describing virulence effects) or on a few species in interactions (for instance describing trophic interaction modifications by parasites). Nevertheless, the review by Hatcher et al. (2006) shows how the consequences of parasitism may extend to more complex systems. Here, we tackle the effects of parasitism on coexistence and stability, explicitly considering a predation-competition context (Fig. 1a). Using such a system allows us to consider "parasite-modified competition [and interactions] with apparent competition" as suggested by Hatcher et al. (2006).

We consider the infection of the prey, then of the predator species. In each case, we first tackle virulence effects (acting on reproduction when the infected species is a prey, and on mortality rates, when the infected species is the predator), then interaction effects (changes in trophic interactions). To allow a more tractable analysis, we first simplify the system, by considering that parasite effects are simple modifications of the host parameters. Such an approach is however limited, as it neglects important ecological feedbacks (e.g. parasite-predator competition when prey species are infected). Therefore, as a second step, we consider a system in which the host population is structured in susceptible and infected individuals. Our aims are to understand how the consequences of parasitism depend on the host trophic level or on parasite effect (virulence or interaction). We expect that prey parasitism increases coexistence (i.e. presence of the three species) when the best competitor is infected, as the parasite then decreases direct competition while increasing apparent competition. When the predator is infected, an intermediate level of parasitism is expected to favor coexistence (Fig. 1b). Concerning stability, we predict that virulence effects will not change stability, except when infected predators undergo large mortality rates (the system should then be stabilized) (Fig. 1c). Predicting how interaction effects alter stability is more difficult as they may modify both the energy transfer (destabilizing the system), and the distribution of interaction strengths within the module (Fig. 1c).

Model & Methods

General approach

To study the effects of parasites on a predation-competition system we proceed in two steps. First, we use an unstructured model in which the parasite dynamics are not explicitly included. We instead assume that parasite effects can be modeled by simple variations in the parameters of the host population dynamics.

We then model explicit parasite dynamics by structuring the host population in susceptible and infected individuals, as in Anderson and May (1986). Under such scenarios, an explicit competition between the parasite and the predator takes place under prey infection scenarios. It therefore gives a more complete account of the feedbacks that occur between the parasite and the trophic module.

Presentation of the unstructured system

We rely on the two prey-one predator model analyzed by Hutson and Vickers (1983), so that local and global stability conditions are already known. The model considers both intra and interspecific competition for the prey species and a linear functional response for the trophic interaction:

$$\begin{cases} \frac{dN_1}{dt} = N_1(r_1 - c_{11}N_1 - c_{12}N_2 - (a_1 + l)P) \\ \frac{dN_2}{dt} = N_2(r_2 - c_{21}N_1 - c_{22}N_2 - (a_2 + l)P) \\ \frac{dP}{dt} = P(e(a_1 + l)N_1 + e(a_2 + l)N_2 - m) \end{cases} \quad (1)$$

Table 1 Model parameters (as well as their default values) and variables (default values are based on values proposed in Hutson and Vickers [1983]).

Parameters	Descriptions	Default values	Dimensions
All models			
N_1, N_2, P	Species density	-	ind.m^{-2}
r_2	Intrinsic growth rate of prey species 2	18	d^{-1}
c_{11}	*Per capita* intraspecific competition rate of prey species 1	1	ind^{-1}.m^2.d^{-1}
c_{22}	*Per capita* intraspecific competition rate of prey species 2	8	ind^{-1}.m^2.d^{-1}
c_{12}	*Per capita* interspecific competition rate of prey species 2 on prey species 1	5	ind^{-1}.m^2.d^{-1}
c_{21}	*Per capita* interspecific competition rate of prey species 1 on prey species 2	4	ind^{-1}.m^2.d^{-1}
a_1	*Per capita* attack rate on prey species 1	1.2	ind^{-1}.m^2.d^{-1}
a_2	*Per capita* attack rate on prey species 2	0.5	ind^{-1}.m^2.d^{-1}
e	Conversion efficiency	1	dimensionless
m	Predator mortality rate	2.5	ind.m^{-2}.d^{-1}
Specific to unstructured model (Eq. (1))			
r_1	Intrinsic growth rate of prey species 1	10	d^{-1}
l	Increased food requirement of infected predators	1	ind^{-1}.m^2.d^{-1}
Specific to the structured infected prey model (Eq. (2))			
S_1, I_1	Density of the susceptible and infected individuals of prey species 1	-	ind.m^{-2}
f_1	Intrinsic fecundity rate of prey species 1	35	d^{-1}
m_1	Intrinsic mortality rate of prey species 1	0	d^{-1}
i	*Per capita* parasite transmission rate	20	ind^{-1}.m^2.d^{-1}
n	Virulence effect (decrease in infected prey fecundity rate)	-	d^{-1}
j	Interaction effect (increase in infected prey vulnerability)	-	ind^{-1}.m^2.d^{-1}
Specific to the structured infected predator model (Eq. (3))			
S_P, I_P	Density of the susceptible and infected predator individuals	-	ind.m^{-2}
m_i	Virulence effect (additional mortality of infected predator individuals)	-	d^{-1}
l_i	Interaction effect (additional energy requirement for infected predator individuals)	-	ind^{-1}.m^2.d^{-1}

with N_i the density of the prey species i, P the predator density, r_i the intrinsic growth rate of prey species i, c_{ii} its *per capita* intraspecific competition rate, c_{ij} the *per capita* effect of interspecific competition of species j on species i, a_i the attack rate on species i, e the conversion efficiency, m the predator intrinsic mortality rate and l the increased food requirement of infected predators. Parameter biological interpretation, dimensions and default values are given in Table 1.

Using system (1), we mimic the two effects (virulence effect and interaction effect) of the parasite. In case of prey infection (on N_1), the virulence effect is modeled through a decreased growth rate (r_1) and the interaction effect through an increased predation on infected hosts (a_1). In case of predator infection, the virulence effect is modeled through an increase of mortality rate (m) and the interaction effect by a simultaneous increase of the two attack rates (a_1 and a_2).

Presentation of the Susceptible-Infected structured systems

Structured model of prey infection

We now include infected prey population structure in the initial model (Eq. (1)) through a SI-structured model (Kermack and McKendrick, 1927; Anderson and May, 1986):

$$\begin{cases} \frac{dS_1}{dt} = S_1(f_1 - m_1 - c_{11}N_1 - c_{12}N_2 - a_1P) + I_1\big((f_1 - n) - iS_1\big) \\ \frac{dI_1}{dt} = I_1(iS_1 - c_{11}N_1 - c_{12}N_2 - (a_1 + j)P - m_1) \\ \frac{dN_2}{dt} = N_2(r_2 - c_{21}N_1 - c_{22}N_2 - a_2P) \\ \frac{dP}{dt} = P(ea_1N_1 + ejI_1 + ea_2N_2 - m) \end{cases} \quad (2)$$

with S_1 and I_1 the susceptible and infected prey densities ($N_1 = S_1 + I_1$), f_1 its intrinsic fecundity rate, m_1 its intrinsic mortality rate and i the *per capita* parasite transmission rate.

In this model, virulence effects are modeled through a reduction of fecundity (parameter n) while interaction effects are modeled through changes in prey vulnerability (parameter j).

Structured model of predator infection

We similarly consider a structured model in which predators are infected. The initial model (Eq. (1)) can then be rewritten:

$$\begin{cases} \frac{dN_1}{dt} = N_1(r_1 - c_{11}N_1 - c_{12}N_2 - a_1(S_P + I_P) - l_iI_P) \\ \frac{dN_2}{dt} = N_2(r_2 - c_{21}N_1 - c_{22}N_2 - a_2(S_P + I_P) - l_iI_P) \\ \frac{dS_P}{dt} = S_P(ea_1N_1 + ea_2N_2 - m) + I_P(e(a_1 + l_i)N_1 + e(a_2 + l_i)N_2 - iS_P) \\ \frac{dI_P}{dt} = I_P\big(iS_P - (m + m_i)\big) \end{cases} \quad (3)$$

with S_P and I_P the densities of susceptible and infected predators ($P = S_P + I_P$).

Virulence effects are considered through an increase in mortality rate (parameter m_i) while interaction effects modify the predation rate (parameter l_i), assuming that infected predators have larger energetic requirements.

Method of analysis of the different models

We systematically explored the consequences of the two effects of the parasite. We analyzed how they alter the coexistence of the three species (in the unstructured model) and of the four species (including the parasite) in the structured models. We then analyzed their consequences for stability by investigating the type of dynamics (stable point, cycles) occurring under different parasitism scenarios.

Our analysis relies on a number of assumptions. First, we assume that prey species 1 is the most competitive, thus excluding prey species 2 when predators and parasites are absent. This occurs when the intrinsic growth rate of species 1 (r_1) and its competition effect on species 2 (c_{21}) are high, given its intraspecific competition (c_{11}). Thus $r_1c_{22} > r_2c_{12}$ and $r_1c_{21} > r_2c_{11}$ allows this competition hierarchy (Hutson and Vickers, 1983; Case, 2000). Second, we assume that, before infection, the competitive prey (prey species 1) is also the most vulnerable to predation (thus $a_1 > a_2$). Thereby, the presence of the predator facilitates coexistence among prey species through apparent competition (Holt et al., 1994). Third, to emphasize the role of predation for coexistence, we focus on the cases where interspecific competition ($c_{12}c_{21}$) dominate intraspecific competition ($c_{11}c_{22}$), thus $c_{11}c_{22} < c_{12}c_{21}$, so that the two competitors cannot coexist in the absence of predators (Hutson and Vickers, 1983). Note that such constraints only apply before infection. The different scenarios of parasitism and the intensity of parasite effects indeed affect prey competitive abilities (when prey species are infected) and trophic interaction rates (through interaction

effects). In scenarios of prey infection, we consider that the host species is the most competitive species (i.e. species 1). Because parasites affect growth rate (r_1), the competitivity relationship between the two prey may be affected. Eventually, this may reverse the competitive hierarchy, or even allow different equilibria to be stable for a given set of parameters. As shown below, bistability between equilibria having either prey species 1 or prey species 2 is possible given parasite infection.

In principle, in the case of the unstructured model, it is possible to directly differentiate the stability theorems from Hutson and Vickers (1983), to understand how parameters affected by the parasite modify the stability conditions. Such analytical solutions are however quite complex and not biologically tractable. Thus for all scenarios, we preferred to perform numerical analyses using Mathematica® 11.1.1 (Wolfram research). First, using the structured models, we simulate the effect of a parasite addition in a non-infected system. Such simulations illustrate how the impacts vary depending on the parasitism effect (virulence or interaction) and on the infected species (predator or prey). For both the unstructured and the structured models, we then analyze the effects of parasitism more globally, through 2D-bifurcation diagrams (one dimension showing variations in virulence effects, the other variations in interaction effects). We analyze the local stability of each equilibrium and detect local bifurcations by studying the eigenvalues of the jacobian matrix for this equilibrium.

Results

Effects of parasite addition in the three-species structured models

Considering the high variety of expected effects of parasitism (Figs 1b-c), we start by presenting some examples of possible effects of adding a parasite on the coexistence and the stability of the three-species system. First note that virulence effects may increase coexistence because they reduce the competitive ability of the most competitive species. For instance, in Fig. 2a, parasitism on the most competitive species (species 1) eventually allows the invasion of the predator (due to interaction effects), then of the inferior competitor (prey species 2) However, virulence effects decrease coexistence when the predator is infected, as they then decrease apparent competition thereby favoring the most competitive species (Fig. 2e). Virulence effects also modulate top-down and bottom-up effects in the system. For instance, when the prey is infected, parasitism incurs a reduction of available energy for higher trophic levels, eventually leading to the loss of the predator (Fig. 2b).

Interaction effects also act on coexistence. First, they affect the degree of apparent competition among prey species as well as energy availability for higher trophic levels (bottom up effects). For instance, a comparison of Figs 2a and 2b show that for similar virulence effects, predator are not maintained if interaction effects are too weak (Fig. 2b) while larger interaction effects allow such a coexistence (Fig. 2a) by allowing a better energy transfer. Coexistence between the two prey species relies on the balance of direct and apparent competitions (Fig. 2a). Too low or too strong interaction effects however lead to the loss of one species, as it changes this balance between the two types of competition (Fig. 2e). These results are coherent with our predictions (Fig. 1b).

Concerning stability, consistent with our predictions (Fig. 1c), we observe that virulence effects do not change stability when the prey is infected (Figs 2a,b), as such effects neither affect the efficiency of energy transfers (interaction rates), nor the distribution of trophic interaction strengths. As expected, virulence effects stabilize the system when predators are infected (Figs 2d,e), as they increase predator mortality. Interaction effects change stability in more complex ways. While in case of prey infection they may stabilize the system by increasing the heterogeneity of interaction strengths (Fig. 2c), in case of predator infection, they may destabilize it by increasing interaction homogeneity or by increasing energy fluxes (Fig. 2f). On Figs 2d,e, we however note that stabilization by virulence effects dominates the complex consequences of interaction effects.

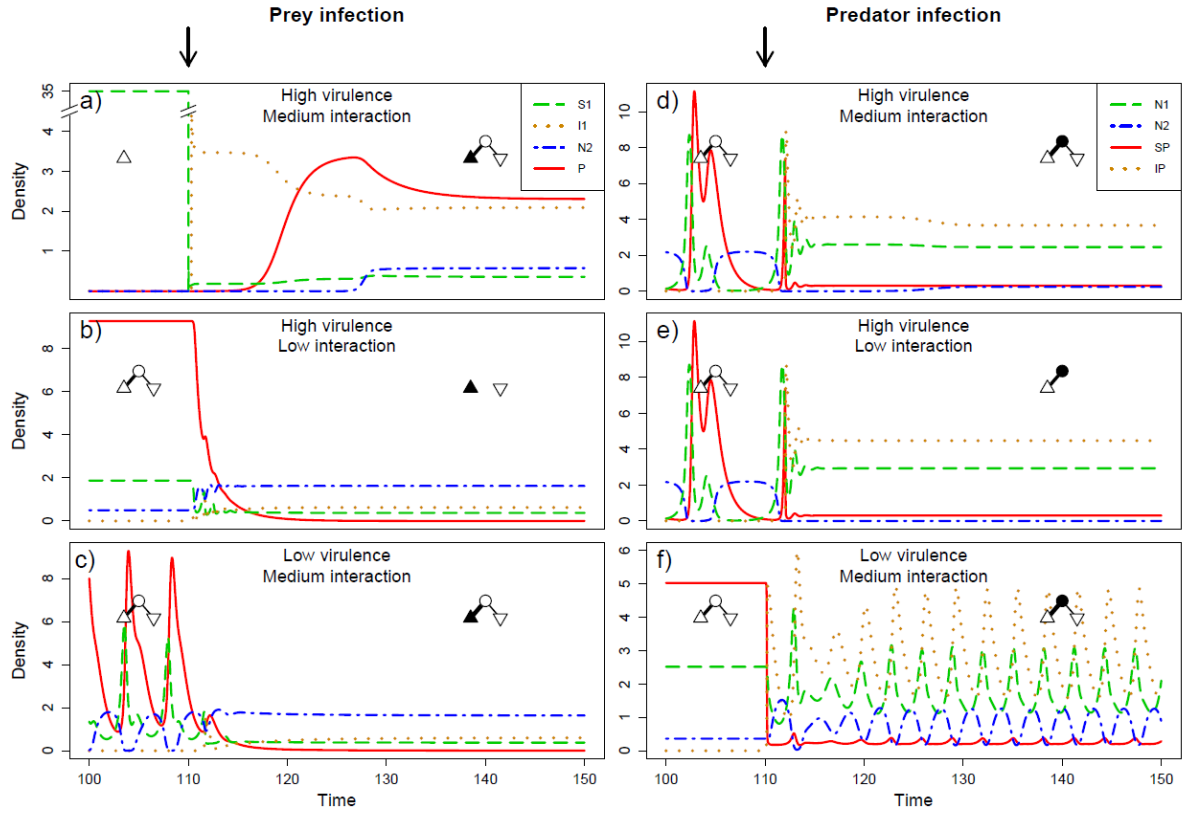


Figure 2. Effects of parasitism on coexistence and stability in a simple module. Note that we consider here a dynamic parasite (i.e. we use the structured model (Eq2) for prey infection (a-c) and (Eq3) for predator infection (d-f). The six panels represent various qualitative outcomes of parasitism: parasites increase coexistence (panels a), decrease coexistence (b,e), stabilize the system (c,e) or destabilize it (d,f). Arrows indicate the time of parasite introduction. Symbols show the composition of the system before and after adding the parasite: preferred prey 1 (triangle), non-preferred prey 2 (inverted triangle), predator (circle). Infected species are represented in black while non-infected are in white. Prey N_1 is shown in green dashed line, prey N_2 in blue dashed-dotted line, predator P in red solid line, infected prey (a-c) or predator (d-f) individual are shown in orange dotted line. Parameter values: as in Table 1, except a) $f_1 = 35$, $a_1 = 0.05$, $n = 33$, $j = 1$; b) $f_1 = 15$, $a_1 = 1.2$, $n = 12$, $j = 0.2$; c) $f_1 = 10$, $a_1 = 1.2$, $n = 3.5$, $j = 0.8$; d) $a_1 = 1.2$, $m = 2$, $m_i = 2.5$, $l_i = 0.5$; e) $a_1 = 1.2$, $m = 2$, $m_i = 2.5$, $l_i = 0.3$; f) $a_1 = 1.2$, $m = 3.2$, $m_i = 0.5$, $l_i = 0.5$. Note that y-axis of a) is broken.

Effects of parasitism in the unstructured model

Now that we have illustrated the possible qualitative effects of parasitism through simulation examples, we vary parasitism continuously in 2D-bifurcation diagrams. We first do so in the unstructured model (Eq. (1); Figs 3a,b and Fig. A1a,b). X-axis of the bifurcation diagram corresponds to the intensity of virulence effects (modeled as a reduction of growth rate for prey infection, and as an increase of mortality for predator infection), while interaction effects (modeled as an increase in vulnerability for prey infection and as an increase in food requirement for predator infection) are shown on the y-axis. We show variations in composition (Fold and Transcritical bifurcations) and stability (Hopf bifurcations) of the system, depending on the intensity of the effects.

We first analyze how parasitism constrains coexistence. In case of prey infection (Fig. 3a), intermediate virulence effects allow coexistence (regions 6/6u) provided the infected prey undergoes strong predation (arrow II). Such variations are consistent with our prediction that coexistence requires balance of direct and apparent competitions (Fig. 1b). Virulence effects impact coexistence as predicted: at medium intensity, we observe a shift in the dominant competitor (from region 1 to region 2). Note that the system exhibits bistability (arrow I, region 1-2, see also region 4-2 and bifurcation in Fig A1 a-I), as expected when interspecific competition dominates intraspecific competition (Case, 2000). In the area of bistability, two equilibria may be achieved, either competitor species 1 or competitor species 2 is present. Around regions

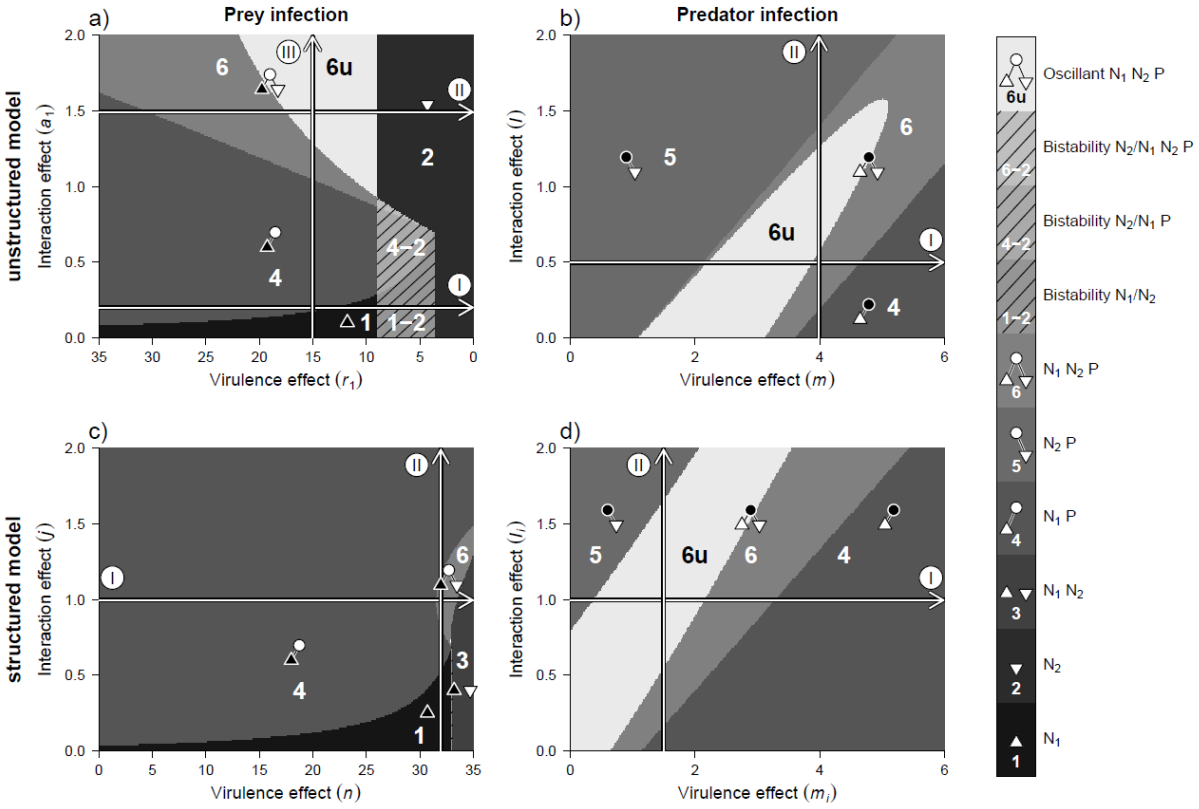


Figure 3. Composition and stability of the system depending on the intensity of virulence effects (x-axis), i.e. fecundity reduction in the case of prey infection or mortality increase in the case of predator infection, and interaction effects (y-axis), i.e. vulnerability increase in the case of prey infection or increased food requirement in case of predator infection. a,c) the host is the preferred prey; b,d) the host is the predator. a-b) show results of the unstructured model, c-d) the results of the structured model. Symbols indicate the composition of the system: preferred prey (triangle), non-preferred prey (inverted triangle), predator (circle). Infected species are represented in black. Arrows show the direction of increasing parasite effects: horizontal arrows for the virulence effect and vertical arrows for the interaction effects. Bifurcations along each arrow are represented in Fig. A1.

1-2 and 4-2 we have Fold bifurcations happening, a first (between regions 1 and 1-2) when species 1 loses its competitive dominance, a second (between regions 1-2 and 2) when the species 2 becomes the competitively dominant species. Regarding interaction effects, we observe they favor coexistence for the parameter range we consider (arrow III, regions 1 then 4 then 6). Nevertheless, our numerical analyses show that further increases in interaction effects would ultimately lead to a loss of the infected species (result not shown). Globally, all these results agree with our predictions (Fig. 1b), as parasitism affects coexistence by changing the balance between direct and apparent competitions.

Concerning predator infection, our numerical analysis show that prey coexistence is facilitated by intermediate intensities of virulence and interaction effects (regions 6/6u). This is illustrated on our bifurcation diagram (Fig. 3b, arrow I and II, Fig. A1b) and in coherence with our predictions (Fig. 1b). A high interaction effect or a small virulence effect (high apparent competition) induces the disappearance of the most consumed and most competitive prey (region 5). Contrarily, a small interaction effect or a high virulence (high direct competition) effect leads to loss of the least consumed and least competitive prey (region 4).

The intensity of parasitism also affects the dynamical stability of our system (Hopf bifurcations, i.e. presence/absence of oscillation). For prey infection (Fig. 3a and Fig A1a), virulence effects destabilize the system (arrow II, region 6u). This contradicts our prediction that stability should not be affected, as virulence effects do not modify interaction strengths. A possible hypothesis is that the increase in total prey density (not show) leads to a reduction in competition intensity. Such a decrease in population regulation could be

the cause of destabilization. Interaction effects destabilize the system (arrow III, region 6u). Such a destabilization may be due to increased energy fluxes from prey to predators (Fig. 1c). In case of predator infection (Fig. 3b and Fig A1b), virulence effects and interaction effects destabilize system when they have intermediate intensity (two Hopf bifurcations, around region 6u). We note that, when increasing simultaneously the two effects, higher stability is eventually achieved (no Hopf bifurcation).

Coexistence and stability in structured models of infection

We similarly analyzed the structured models of prey and predator infection (Eq. (2) and (3); Figs 3c,d and Fig. A1c,d). Coexistence (regions 6/6u) is favored at intermediate intensity of virulence and interaction effects regardless of the infected species. Thus, results on coexistence remain consistent with our predictions (Fig. 1b) and with the results observed for the unstructured model. Some finer differences however exist between the structured and unstructured models of prey infection (Fig. 3a vs Fig. 3c). Virulence effects still lead to a reduced competitive ability of the infected species, which favors the co-occurrence of the two competitors (arrow I, region 3). However, in the structured model, the explicit dynamics of parasite (susceptible-infected) provides an additional feedback loop between predator and parasite effects acting on prey species 1. This feedback reduces the parasite prevalence so that the different negative pressures acting on species 1 are more balanced. Consequently, its competitive ability is less reduced compared to the unstructured scenario. This species remains dominant (region 4) for a larger set of parameters, which reduced the possibility of coexistence (region 6). Hence, the coexistence area appears highly reduced compared to the unstructured model (regions 6/6u in Fig. 3 a vs c). The explicit dynamics of the parasite in the structured model also lead to a competition between predator and parasite populations (Fig. 4). Virulence effects favor the parasite in this competition. They lead to an increase in density of infected prey and to a decrease in predator density (Fig. 4, arrow I). Ultimately, by reducing the predator resources below a threshold density N^* (see the grey and white transcritical bifurcation), virulence effects ultimately lead to the extinction of the predator (Figs 3a-b, 4, S2). Contrarily, at large interaction effects, we observe a decrease in parasite prevalence while predator density increases (Fig. 4, arrows II and III). Interaction effects therefore reduce parasite resources below a threshold density S^* (see the black and white transcritical bifurcation), thereby favoring predators over parasites in their competition for prey. Consequently, while intermediate effects of parasitism are still required to maintain coexistence, the mechanism now relies not only on the balance between direct vs apparent competition among prey species, but also on a balanced competition between predators and parasites.

In scenarios of predator infection, structured and unstructured models give qualitatively similar results for coexistence (regions 6/6u in Fig. 3b vs Fig. 3d).

Effects of parasitism on stability are more idiosyncratic. For infected prey (Fig. 3c), the area of oscillations is greatly reduced in the structured system (region 6u too smaller to be indicate). This increased stability of structured prey infection system (compared to the non-structured case) is commonly observed for various sets of parameters (Fig. B1). When the parasite is maintained in the system, virulence and interaction effects seldom lead to an oscillating system (regions 6 and 6u in Fig. B1a,b) or stabilize an unstable one (regions 6 and 6u in Fig. B1c). Such stabilizing effects may be explained by the fact that the structured model explicitly accounts for an additional negative feedback between the predator and parasite populations. Regarding predator infection (Fig. 3d), virulence effects stabilize the coexistent system (arrow I, from regions 6u to 6), as predicted (Fig. 1c). Interaction effects (arrow II) first destabilize the system (from regions 6 to 6u), in coherence with our predictions (Fig. 1c). Further increases in interaction effects eventually lead to the destabilization of the module and to the loss of the prey species 1 (preferred by the predator) (from regions 6u to 5).

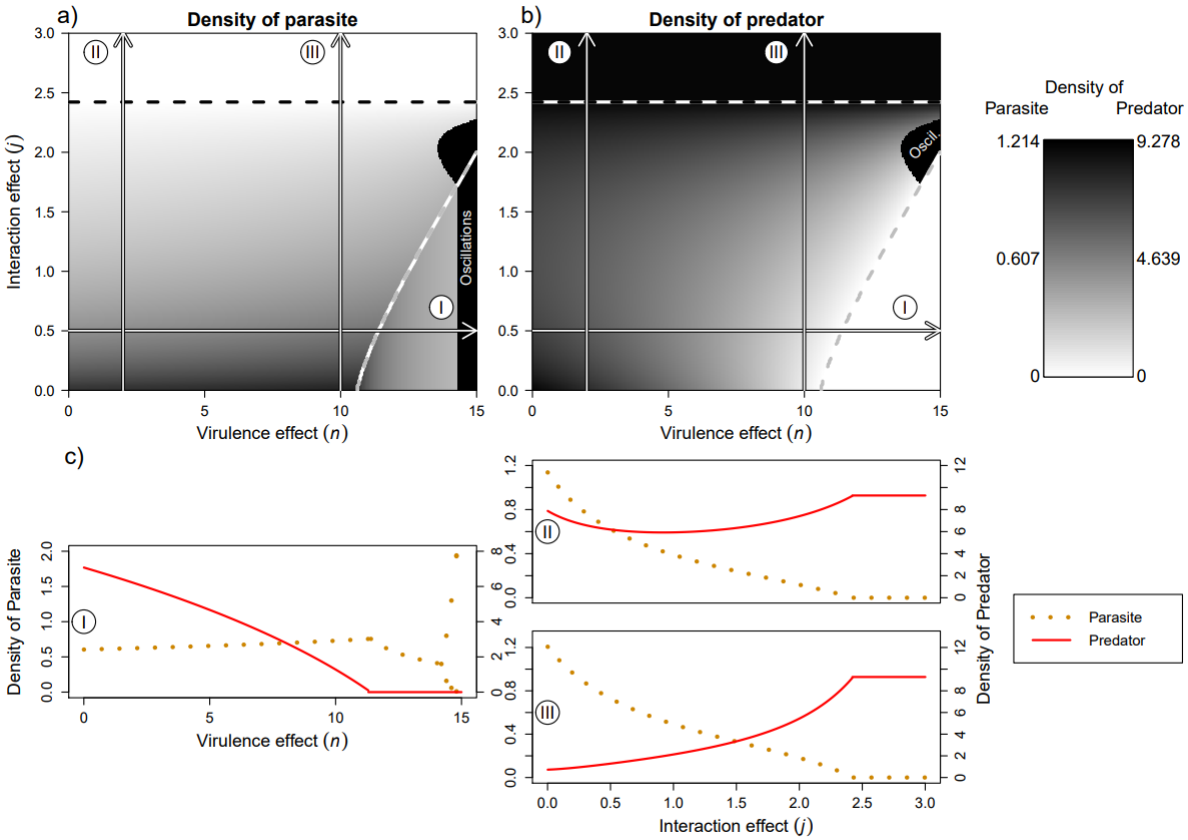


Figure 4. Analysis of predator-parasite competition. Parasite density (a) and of predator density (b) depending on the intensity of virulence (x-axis) and interaction effects (y-axis). Arrows show the direction of increasing parasite effects (arrow I shows increased virulence effects – reduction of host fecundity –, arrows II & III, increased interaction effects – increase of attack rate on infected). The dashed lines are transcritical bifurcations that show the loss of the parasite (above black/white) and the loss of the predator (right of grey/white). The right black area corresponds to oscillating systems. c) Three bifurcation diagrams, corresponding of the three arrows, showing the contrasting effects of virulence effects (that favor the parasite population) and of interaction effects (that favor the predator population). (1) $j = 0.5$, (2) $n = 5$ and (3) $n = 10$. The lines show predator density (red solid lines) and infection levels (orange dotted line).

Discussion

The present work uses simple models to highlight and understand mechanistically possible consequences of parasitism for coexistence and stability in predator-prey systems. We show that such consequences depend on the type of parasitism (virulence vs interaction effects), on the species that is infected (predator or prey), but that they can be understood to some extent based on classical ecological theories based on apparent competition (Holt, 1977) and interaction strength (McCann et al., 1998; Rip and McCann, 2011). More precisely, parasites affect coexistence within trophic level by changing the balance between direct and apparent competitions. In the case of infected prey, parasites also modify coexistence among trophic levels by altering bottom-up effects and through competition with predators. Assessing the latter effect however requires the construction of structured model that allow explicit variations in the parasite populations. While parasitism can affect stability positively or negatively depending on the scenario, the ratio between energy fluxes and predator mortality rates largely explain the effects of predator infection on system stability, as proposed in previous works (Rosenzweig, 1971; Rip and McCann, 2011).

Within a given trophic level (among prey species), we find that parasites may alter coexistence by affecting the relative intensity of direct and apparent competitions. First, parasites may reduce its host competitive ability (for instance through virulence effects), allowing coexistence with an inferior competitor

("parasite-mediated competition" *sensu* Hatcher and Dunn [2011]). Such a mechanism of coexistence is coherent with previous theoretical works (e.g. Anderson and May [1986]) and has also been observed in experiments and field investigations (Park, 1948; Price et al., 1988; Schall, 1992; Callaway and Pennings, 1998; Kiesecker and Blaustein, 1999; Decaestecker et al., 2015). For instance, Schall (1992) studied the coexistence of lizard species in Caribbean islands and observed that *Anolis wattsi* was present only when *A. gingivinus* was infected by the malarial parasite *Plasmodium azurophylum,* a parasite that has clear virulence effects.

Parasites infecting predators also affect coexistence and biomass distribution among trophic levels as they reduce top-down effects and alter apparent competition between prey species (Fig. A1b,d). By increasing predator mortality (e.g. through virulence effects), such parasites act as a top-predator (Wilmers et al., 2006) and may induce trophic cascades that increase prey density (Fig. A1b-I,d-I). Such parasite-mediated trophic cascades have been observed in nature (Buck and Ripple, 2017). For instance, Lindström et al. (1994) show that the infection of red foxes by *Sarcoptes scabiel* can lead to increased hare and grouse densities. However, such positive consequences of predator infection on prey abundances can be redistributed asymmetrically among prey species, as such parasites also alter apparent competition (Fig. A1b-I,d-I). Given virulence effects, the parasites of predators would lead to a release of apparent competition as they decrease predator populations. Contrarily, parasites incurring interaction effects may reinforce apparent competition. Such modifications of prey composition have been observed for both effects. Empirical studies showed that virulence effects reduce top-down control and affect prey composition (Dobson and Crawley, 1994; Hartley et al., 2009). Other experiments also showed that increased predation rate due to parasitism of the predator (i.e., interaction effects) can lead to a shift in the dominant prey species. Bernot and Lamberti (2008) for instance found that snails (*Physa acuta*) infected by a trematode (*Posthodiplostomum minimum*) have a greater grazing rate leading to a periphyton community dominated by *Cladophora* whereas, without parasite, periphyton is dominated by diatom and blue-green algae. Furthermore, system where infection reduce grazing rate presents similar results (Wood et al., 2007).

Parasites of prey species also affect higher trophic levels through bottom-up effects. They have a particular interaction with the predators of their host for which they become both a prey and a competitor (Sieber and Hilker, 2011), which is close to intraguild predation. Most previous studies focused on the effects of predators on infection levels. With this point of view (i.e. focusing on the effects of predators on parasites), Packer et al. (2003) developed the "healthy herd hypothesis" observed in theoretical and empirical works (Anderson and May, 1986; Lafferty, 2004; Duffy et al., 2005). By reducing host/prey density below a threshold, predators lead to parasite extinction. This effect increases when predator consumes preferentially infected prey (Packer et al., 2003; Hethcote et al., 2004), for instance due to interaction effects. Such observations are consistent with our results. In our model, interaction effects systematically favor predators in their competition with parasites. Such outcomes have also been observed experimentally. As shown by Duffy et al. (2005), the prevalence of parasite *Spirobacillus cienkowskii* in *Daphnia dentifera* population decreases when the abundance of bluegills (a predator of *Daphnia*) increases. Our work also clarifies conditions under which parasites have competitive or facilitative effects on predators, through modifications of bottom-up effects. When parasites have interaction effects, they allow the persistence of predators by making prey more available (facilitative effect) (Fig. A1c-II). Such effects are consistent with previous theoretical works (Hethcote et al., 2004). However, when parasites have mostly virulence effects, their negative impact on prey density may lead to the disappearance of the predator (competitive effect) (Fig. A1c-I). Such competitive effects are consistent with earlier theoretical works (e.g. Anderson and May [1986]). Parasites and predators then interact as competitors, sharing a common resource: the prey species. Such a competition ultimately reduces coexistence. Empirical examples of such dynamics exist. Banerji et al. (2015) showed experimentally that, by reducing prey density (*Paramecium caudatum*) parasites may lead to a reduction of predator density (*Didinium nasutum*).

Effects of parasites on stability seem to be highly context-dependent (Lafferty et al., 2008; Wood and Johnson, 2015). Previous theoretical and empirical studies report stabilizing effects through regulation of host populations (Anderson and May, 1978; Hilker and Schmitz, 2008; Cáceres et al., 2014) or through parasite-mediated coexistence (Dobson, 2004). Ong and Vandermeer (2015) for instance showed that adding not only predators, but also parasites allows for a more stable biological control. Other studies report destabilizing effects (Anderson and May, 1978, 1986; May and Anderson, 1978; Grenfell, 1992; Hudson et al., 1998), for instance due to increased vulnerability to predation (Ives and Murray, 1997), or when parasites create time lags in dynamics (May and Anderson, 1978; Hudson et al., 1998). In our model, stability outcomes are equally variable, as parasites may stabilize an unstable system or destabilize a stable one, even within a given parasitism scenario, depending on the considered set of parameters.

Nevertheless, our model highlights how some of these results on stabilization/destabilization can be related to general theories of stability in consumer-resource interactions. We systematically assessed two basic hypotheses: that heterogeneity in interaction strengths increases stability (McCann et al., 1998) and that stability depends on relative energy fluxes (ratio between attack rates and predator mortality rates [Rip and McCann, 2011; Rosenzweig, 1971]). Our model shows that the second hypothesis largely explains the patterns we observe in case of predator infection. We indeed observe that parasites of predators have a stabilizing effect in case of virulence effects (that increase in predator mortality), but a destabilizing effect when they induce interaction effects (that increase of attack rate). The stabilizing effects of virulent parasites infecting predators are consistent with previous theoretical results (Hilker and Schmitz, 2008) while the destabilization due to interaction effects had also been observed in model by Bairagi and Adak (2015).

While we categorized the effects of parasites in two types – virulence and interaction effects –, most parasites likely alter simultaneously life-history traits (with consequences for mortality and/or reproduction) and species behavior or physiology (with consequences for interaction strength). Interestingly, in some of our scenarios, coexistence can only be reached when combining the two effects. While early studies of parasitism focused on virulence effects (Park, 1948; Holt and Pickering, 1985), modifications of trophic interactions (i.e. interaction effects) have most often investigated in trophically-transmitted parasites within the framework of the manipulation hypothesis (Bethel and Holmes, 1977; Poulin and Maure, 2015). However, interaction effects may also be a by-product of virulent parasites, that lead by definition to modifications of host energy requirement or allocation (Hall et al., 2007). Hosts may then reduce or increase their activity with likely modifications of their vulnerability to predation or their consumption rate (Peterson and Page, 1988; Khokhlova et al., 2002; Wright et al., 2006; Wood et al., 2007; Gehman and Byers, 2017). The consequences of virulence and interaction effects at the community level remain understudied. Banerji et al. (2015) have however analyzed a tri-trophic food chain with a resource, a consumer, a predator and a parasite of the consumer. They showed that infection leads to variations in growth rate (implying virulence effects), changes in consumption rate (thus interaction effects), with implications for the dynamics of each species. In their case, infection of a *Paramecium* decreased its growth rate and cell size, increased its velocity and grazing rate, but did not modify its vulnerability.

We here combine in one model, predation, parasitism and competition to better understand their complex relationships and their consequences in food web. We showed that such effects depend on the type of parasitism (virulent vs interaction) as well as of the host trophic level. Parasites may act as competitors or facilitators for predators, and may help coexistence of the prey, particularly at intermediate intensity of effects, or when virulence and interaction effects occur simultaneously. By using successively unstructured and structured models, we showed that the effects of parasitism on stability act not simply as density dependent mortality, but in a more complex way, as pointed out in other theoretical works (Gerber et al., 2005). Our model is simplified in various ways. First, it relies on a module approach and how the impacts we document extend to more complex networks requires more complex simulations. Also, because we tried to focus on the effects of parasitism, we simplified the other interactions. For instance, we consider

that predators have a linear functional response. We may expect that the effect on stability will be similar with a type-II functional response (as in Hilker and Schmitz, 2008; Bairagi and Adak, 2015). Indeed, we rely on the theory of energy fluxes that was shown to apply to both type-I and type-II functional responses by Rip and McCann (2011). Also, the use of type-II functional response would imply to choose whether interaction effects alter maximal consumption rates and/or handling times. Currently, we could not find available data that would allow a guided choice on this issue. Such investigations would therefore be important. Our work however clearly stresses how existing feedbacks between predators and parasites can alter coexistence and stability. As current global changes simultaneously reshuffle local networks and the spatial propagation of various diseases, we hope it will help to understand some of the complex dynamics that will likely happen.

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Authors' contributions

LP, VM and NL designed the model and the computational framework and analyzed the data. LP performed analytic calculations and performed the numerical simulations. LP wrote the first draft, NL edited the first draft and all authors participated to further rewriting of the manuscript.

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Supplementary information

Supplementary information is available after the references:

- Appendix A: Bifurcations along virulence and vulnerability effect
- Appendix B: Composition and stability of prey-infected system for various set of parameters

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Appendix A: Bifurcations along virulence and vulnerability effect

Fig A1 shows bifurcation diagrams (local minima and maxima) of population densities when virulence effects or interaction effects are varied within the module. We observe that coexistence is most often observed for intermediate intensities of parasitism. Note the bistability area, delimited by two Fold bifurcations Fig. A1a-I. Regarding stability, we observe that the effects of parasitism can either stabilize (Fig. A1 d-I), destabilize (Fig. A1 a-II,a-III,d-II) or both (Fig. b) the dynamics of the system (Hopf bifurcation points at the transition of cyclic and stable node outcomes).

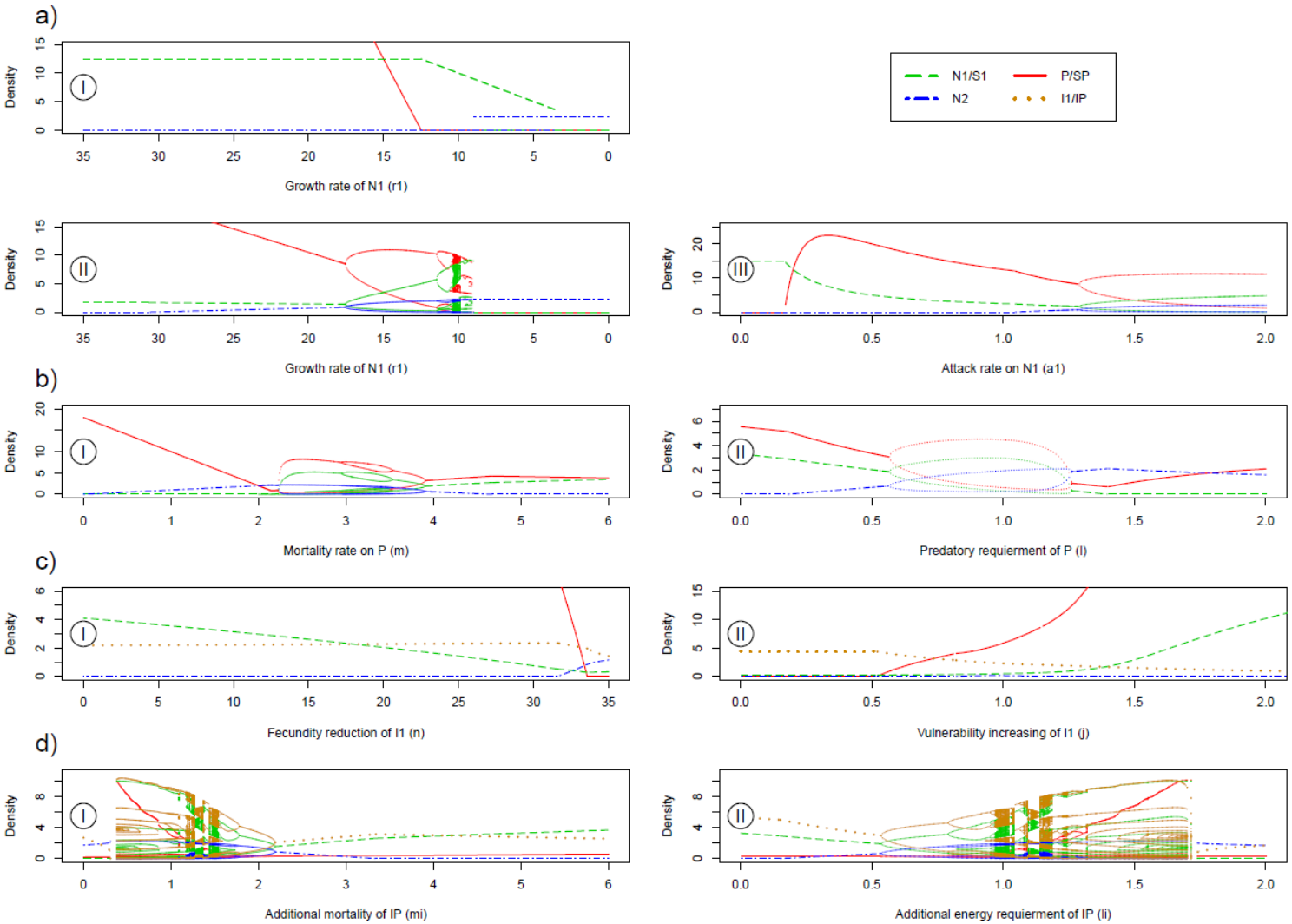

Figure A1. Bifurcations along arrows of Fig. 3. Left: bifurcation of virulence effect. Right: Bifurcation of interaction effect. a-d correspond to a-d of Fig. 3: a) prey infection with unstructured system, b) predator infection with unstructured system, c) prey infection with structured system, d) predator infection with unstructured system. Prey N_1 or S_1 is shown in green dashed line, prey N_2 in blue dashed-dotted line, predator P or S_P in red solid line, infected prey (c) or predator (d) individual are shown in orange dotted line. Parameter values: as in Fig. 3 except a) I: $a_1 = 0.2$; II: $a_1 = 1.5$; III: $r_1 = 15$; b) I: $l = 0.5$; II: $m = 4$; c) I: $j = 1$; II: $n = 32$; d I: $l_i = 1$; II: $m_i = 1.5$.

Appendix B: Composition and stability of prey-infected system for various set of parameters

Effects of prey parasitism on coexistence are investigated by studying how parasite effects lead to different states of the system. Parasites increase coexistence (if not maximal, Fig. B1a) at intermediate effect before they decrease it (Fig. B1). In particular, we observe the loss of the predator at high virulence effect and the loss of the parasite at high interaction effect (as shown in Fig. 4). Regarding stability, results are more idiosyncratic: parasites (virulent or interaction effect) destabilize (in a limited range) a stable system (Fig. B1 a-b) but stabilize an unstable one (Fig. B1 c-d).

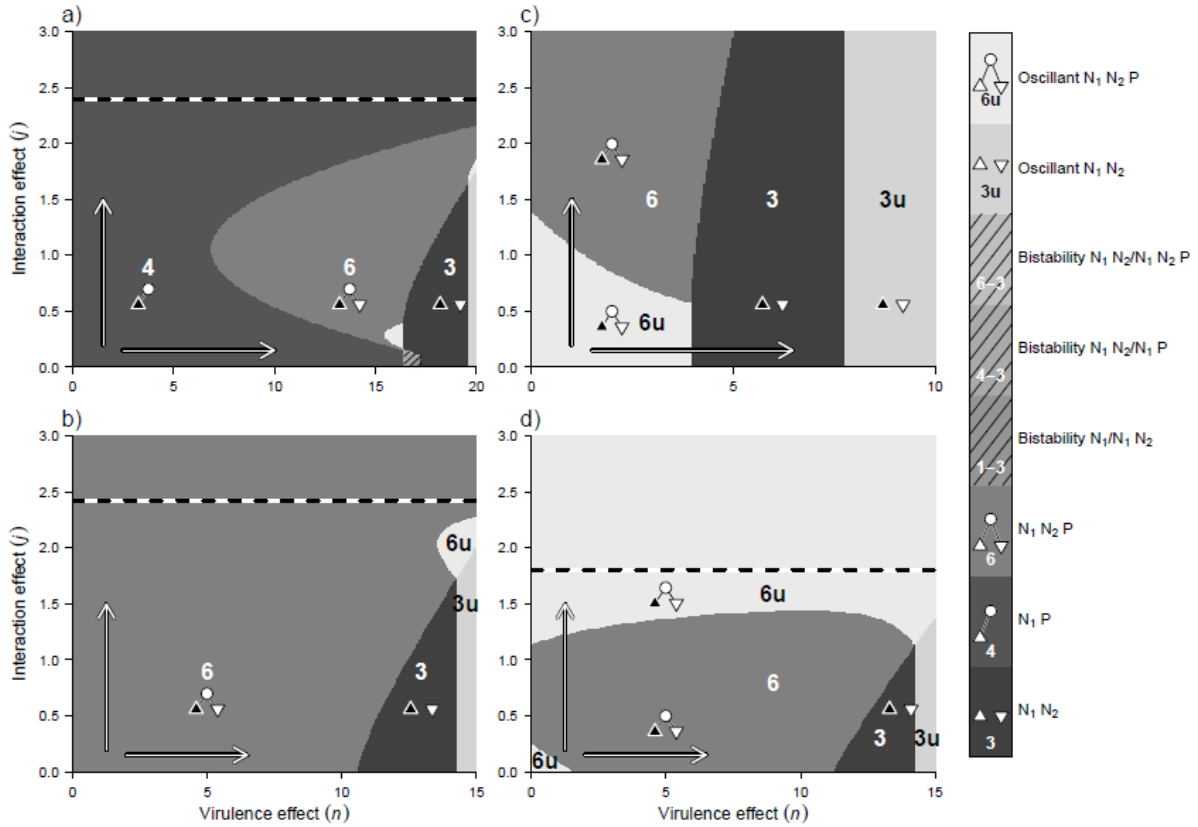

Figure B1. Variations in the composition and stability of the system given prey infection, for various initial compositions of the uninfected system. Symbols indicate the composition of the system: preferred prey (triangle), non-preferred prey (inverted triangle), predator (circle). Host species are represented in black. Arrows show the direction of increasing parasite effects. Horizontal dashed lines correspond to limits above which the parasite cannot persist. Parameter values: as in Table 1 except a) $f_1 = 20$, $a_1 = 0.6$; b) $f_1 = 15$, $a_1 = 1.2$; c) $f_1 = 10$, $a_1 = 1.2$; d) $f_1 = 15$, $a_1 = 1.5$.