Evolution of predator foraging in response to prey infection favors species coexistence

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Abstract

As acknowledged by Optimal Foraging theories, predator diets depend on prey profitability. Parasites, ubiquitous in food webs, are known to affect simultaneously host vulnerability to predation and host energy contents, thereby affecting profitability. In this work, we study the eco-evolutionary consequences of prey infection by a non trophically-transmitted parasite, with a simple lifecycle, on predator diet. We also analyze the consequences for coexistence between prey, predators and parasites. We model a trophic module with one predator and two prey species, one of these prey being infected by a parasite, and distinguish between two effects of infection: a decrease in host fecundity (virulence effect) and an increase in vulnerability to predation (facilitation effect). Predator foraging may evolve toward specialist or generalist strategies, the latter being less efficient on a given resource. We show that the virulence effect leads to specialisation on the non-infected prey while the facilitation effect, by increasing prey profitability, favors specialisation on the infected prey. Combining the two effects at intermediate intensities promotes either generalist predators or the diversification of foraging strategies (coexistence of specialists), depending of trade-off shape. We then investigate how the evolution of predator diet affects the niche overlap between predator and parasite. We show that facilitation effects systematically lead to a high niche overlap, ultimately resulting in the loss of the parasite. Virulence effects conversely favor coexistence by allowing a separation of the predator and parasite niches.

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1. Introduction

The consequences of parasitism on food webs regarding trophic cascades (Buck and Ripple 2017) or in terms of stabilisation (Hilker and Schmitz 2008; Prosnier et al. 2018) or destabilisation (Hudson et al. 1998; Prosnier et al. 2018) are most often investigated through the lens of ecology. On the other hand, parasite evolution is frequently addressed through the evolution of virulence or transmission, sometimes in a context of trophic interactions (Cressler et al. 2016). However, modulating host phenotype and abundance may also have evolutionary consequences for the species interacting with the hosts, including predators. In this work, we study how infection of a prey species by a specialist parasite with a simple life cycle (i.e. with one host) affects the evolution of the predator foraging strategy.

A possible way to understand variations in predator diet is to consider Optimal Foraging Theory, under which predators interact only with the prey species that enhance their net energy intake rate (Pyke et al. 1977). The relative profitability of prey species, defined as the ratio between energetic gain (prey energetic value) and energetic cost (search and handling times), determines the occurrence of trophic links and therefore the degree of generalism of predators (Emlen 1966; MacArthur and Pianka 1966; Charnov 1976*a*, 1976*b*). Although parasite-induced alterations in host phenotype are likely to modify the two components of profitability, the consequences on the structure of natural communities remain poorly understood.

Infection induces modifications in host energy allocation, either because parasites use energy for their own development, or because hosts allocate energy to the immune response at the expense of other functions. This energy reallocation may (negatively) affect host fitness through direct and indirect effects. Direct effects on fitness (virulence effects hereafter) occur when parasites reduce host fecundity, increase host mortality or both. Increased mortality has been described very often (Brassard et al. 1982; Prins and Weyerhaeuser 1987; Lynsdale et al. 2017).

Infection-induced fecundity reduction is documented for various animals like water fleas (*Daphnia sp.*) hosting bacterial, viruses or fungi that reduce offspring production by 7-66% (Decaestecker et al. 2003). Such virulence effects should decrease the density of hosts (prey), which make them less profitable towards predators, search time being increased. Indirect effects on fitness through increased vulnerability to natural enemies and particularly predators (facilitation effects hereafter) results from changes in host phenotype including behavior, morphology or physiology. This can be part of an adaptive transmission strategy in trophically-transmitted parasites (Cézilly et al. 2010) but also occurs in non-manipulative parasites (Goren and Ben-Ami 2017), as the simple lifecycle parasite without trophic transmission that we consider in this article. For instance, red grouses infected by nematodes are more consumed by mammalian predators due to higher scent emissions, as parasites induce physiological modifications (Hudson et al. 1992). *Anisops* prefer *Daphnia* infected by *Pasteura ramosa* in dark condition but the non-infected *Daphnia* in light condition (Goren and Ben-Ami 2017), possibly due to modifications in host colouration and mobility. Making the host more attractive (e.g. increased conspicuousness) or more catchable (e.g. decreased mobility) may decrease the search and handling times of predators and thus increase profitability. Note that separating virulence and facilitation effects is a simplification we use to highlight important differences. In reality, both effects may be to some degree correlated: host resources diverted by the parasite for its own development affect simultaneously host fecundity and/or morality (i.e. virulence effect) and host behaviour (i.e. resulting in a facilitation effect). Because virulence and facilitation effects can therefore act in opposite ways on profitability and infection by a single parasite species that induces both effects, the consequences on predator foraging strategies could be unintuitive.

Previous theoretical investigations on the evolution of predator foraging in the absence of parasite suggest that the type of trade-off regarding the relative intensity of predation on each

prey included in the diet largely affects predator diet evolution (Egas et al. 2004; Rueffler et al. 2006). Concave trade-offs usually select generalist predators (i.e. that consume both prey) whereas convex trade-offs lead to specialist predators (i.e. that consume only one prey) or to diversification (i.e. coexistence of various predator strategies). These works however consider only symmetrical resources (i.e. all prey are identical), so that they cannot account for trait modifications due to parasitism. Infection dynamics can also interact with predation dynamics in complex ways. Here, based on Optimal Foraging, we expect that predators should specialise on the prey whose profitability has been increased by infection. It follows that virulence effects should lead to either generalist strategies or to specialisation on the alternative (i.e. non-infected) prey while specialisation on the infected prey should be observed with facilitation effects.

Beside evolutionary outcomes, we also investigate whether and how the evolution of predator diet affects species coexistence in the trophic module. Coexistence between two prey species depends on a balance between intrinsic competitivity (i.e., which prey species would dominate in the absence of predators and parasites (Gause 1934)) and the intensity of apparent competition (i.e., predator-mediated competitions (Holt 1977)). Prey coexistence is favored when the most competitive species is also the one preferred by the predator (Holt et al. 1994). Because virulence and facilitation effects constrain both direct and apparent competition, the ecological dynamics linked to infection have been shown to influence prey coexistence (Prosnier et al. 2018). Here, we go beyond and tackle the feedback of the evolution of predator diet on these ecological effects with the idea that it may alter apparent competition and thereby prey coexistence in predictable ways. An increase in coexistence is expected in case of virulence effects as evolution should favor the predators that prefer the alternative prey, the negative effects of parasitism being then compensated by a release from predation. Under

facilitation effects increasing profitability, parasitism should amplify specialisation on the infected species, which may lead to prey exclusion and impair coexistence.

Understanding the conditions of coexistence between parasites and predators requires decomposing the relative effects they have on each other. Regarding the effects of predators on parasites, a classical hypothesis in ecology is that predators may limit parasite prevalence within populations (Healthy Herd Hypothesis, Packer et al. 2003; Lafferty 2004; Duffy et al. 2005) in several ways. First, predators indirectly consume parasites when consuming infected prey (concomitant predation) (Johnson et al. 2010), for instance the signal crayfish (*Pacifastacus leniusculus*) reduces the abundance (but not the prevalence) of parasites of its isopod and snail prey (Pulkkinen et al. 2013). Second, predators can limit parasite transmission by reducing host density, for instance, Dallas et al. (2018) showed in the water flea *Daphnia dentifera* that under a density threshold of 80 ind.L-1, its fungal pathogen *Metschnikowia bicuspidata* cannot be maintained. Such negative effects should be amplified when parasites increase host vulnerability through facilitation effects, as suggested by the models developed by Packer et al. (2003) and Prosnier et al. (2018). Now consider the effects of parasites on the predators of their host. Virulence effects, by reducing host density, decrease predator density through resource limitation (bottom-up effects). Such bottom-up reduction in predator density has for instance been experimentally observed with paramecia infected by *Holospora undulata* and consumed by *Didinum nasutum* (Banerji et al. 2015). Conversely, parasites could enhance predator density when they induce facilitation effects, as they increase prey vulnerability (theoretical works of Hethcote et al. [2004] and Prosnier et al. [2018]).

Accounting for the evolution of predator diet should therefore largely influence the outcomes of these ecological effects. If virulence effects promote specialisation on the alternative prey, evolutionary dynamics should lead to niche partitioning between parasites and predators,

thereby increasing coexistence. On the other hand, facilitation effects should increase niche overlap and thus competition between predators and parasites.

To understand these eco-evolutionary dynamics, we model a trophic module with one predator and two prey in competition, the most competitive prey being infected and structured in susceptible and infected individuals. The parasite induces virulence and facilitation effects, modeled respectively by a reduction of host fecundity or an increase in host vulnerability to predation. In agreement with our predictions, we show that virulence effects favor specialization on the alternative prey while facilitation effects have the opposite effect. Our results also show that evolution favors niche separation among parasites and predators, thereby promoting coexistence.

2. Model presentation and analysis

2.1. Predation-infection model

The model considers two prey species sharing a predator. We assume both intraspecific and interspecific competition among prey and we model predation through linear functional responses. The infected prey (labeled 1) is also the most competitive and its population is structured in susceptible and infected individuals (S_1 and I_1 respectively). Parasite transmission is horizontal and we assume that infected individuals do not recover. Considering these hypotheses, ecological dynamics follow the set of equations:

$$\begin{cases} \frac{dS_1}{dt} = S_1(f_1 - m_1 - c_{11}N_1 - c_{12}N_2 - a_1P) + I_1\big((f_1 - n) - iS_1\big) \\ \frac{dI_1}{dt} = I_1(iS_1 - c_{11}N_1 - c_{12}N_2 - (a_1 + j)P - m_1) \\ \frac{dN_2}{dt} = N_2(r_2 - c_{21}N_1 - c_{22}N_2 - a_2P) \\ \frac{dP}{dt} = P(ea_1N_1 + ejI_1 + ea_2N_2 - m) \end{cases} \quad (1)$$

, with N_x the total prey species x population, S_x and I_x respectively the susceptible and infected individuals (thus $N_1 = S_1 + I_1$), f_1 the intrinsic fecundity rate of the infected prey, m_1 its intrinsic mortality rate, r_2 is the intrinsic growth rate of the alternative prey (with $r_2 = f_2 - m_2$), P the predator density, m its intrinsic mortality rate, i the *per capita* transmission rate of the parasite, c_{xx} the *per capita* competition rate of prey x, c_{xy} the *per capita* competition effect of prey y on prey x, a_x the *per capita* attack rate of the predator on prey x, e the conversion efficiency. Virulence effects are implemented through a decreased fecundity in the infected prey (parameter n) and facilitation effects through an increased attack rate on the infected prey (parameter l). The biological interpretation, dimensions and default values of parameters are given in Table 1.

Prey N_1 being the most competitive, we assume that $r_1c_{22} > r_2c_{12}$et $r_1c_{21} > r_2c_{11}$ (Hutson and Vickers 1983) so that it excludes the alternative prey in the absence of predators and parasites.

Table 1. Biological interpretation, dimensions and default values of model's parameters. Used values are those proposed in Prosnier et al. (2018) and based on Hutson and Vickers (1983).

Parameters	Descriptions	Defaults values	Dimensions
	Model (Eq. (1))		
N_1, N_2, P	Species densities	-	ind.m^{-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	25	d^{-1}
m_1	Intrinsic mortality rate of prey species 1	10	d^{-1}
r_2	Intrinsic growth rate of prey species 2	18	d^{-1}
c_{11}	*Per capita* intraspecific competition rate of prey species 1	1	m^2.ind^{-1}.d^{-1}
c_{22}	*Per capita* intraspecific competition rate of prey species 2	8	m^2.ind^{-1}.d^{-1}
c_{12}	*Per capita* interspecific competition rate of prey species 2 on prey species 1	5	m^2.ind^{-1}.d^{-1}
c_{21}	*Per capita* interspecific competition rate of prey species 1 on prey species 2	4	m^2.ind^{-1}.d^{-1}
a_1	*Per capita* attack rate on prey species 1	-	m^2.ind^{-1}.d^{-1}
a_2	*Per capita* attack rate on prey species 2	-	m^2.ind^{-1}.d^{-1}
e	Conversion efficiency	1	Dimensionless
m	Predator mortality rate	2.5	ind.m^{-2}.d^{-1}
i	*Per capita* parasite transmission rate	35	m^2.ind^{-1}.d^{-1}
n	Virulence effect (decrease in infected prey fecundity rate)	-	d^{-1}
j	Facilitation effect (increase in infected prey vulnerability)	-	m^2.ind^{-1}.d^{-1}
	Trade-off (Eq. (2))		
s	Shape of the trade-off	-	Dimensionless
k_0	Total Allocation in foraging effort	1.5	m^{2s}.ind^{-s}.d^{-s}

The ecological model was previously analyzed by Prosnier et al. (2018). They show that coexistence is possible only given restrictive conditions. Between-prey coexistence is possible if the competitive ability of the best competitor has to be partially reduced by virulence effect (decreased fecundity), and if the predator consumes preferentially the infected prey (facilitation effect). Too large virulence effects allow the parasite to competitively exclude the predator, while too large facilitation effects allow the predator to overconsume infected prey, thereby leading to the parasite extinction.

2.2. Trade-off constraints on adaptive foraging

We assume that the foraging strategy of the predator varies along a trade-off function that describes allocation (e.g. attack rate) between the two prey species. The time or energy devoted to the consumption of one prey reduces predation on the other. We also assume that generalist strategies have lower attack rates on a given prey compared to a specialist of the same prey. Such trade-offs (Fig. 1) can be modelled using the following function (Egas et al. 2004):

$$a_1{}^s + a_2{}^s = k_0 \leftrightarrow a_2 = (k_0 - a_1{}^s)^{1/s} \qquad (2)$$

, where s affects the trade-off curvature (convex: $s < 1$, linear: $s = 1$ or concave: $s > 1$), and k_0 corresponds to the total allocation of energy or time in predation.

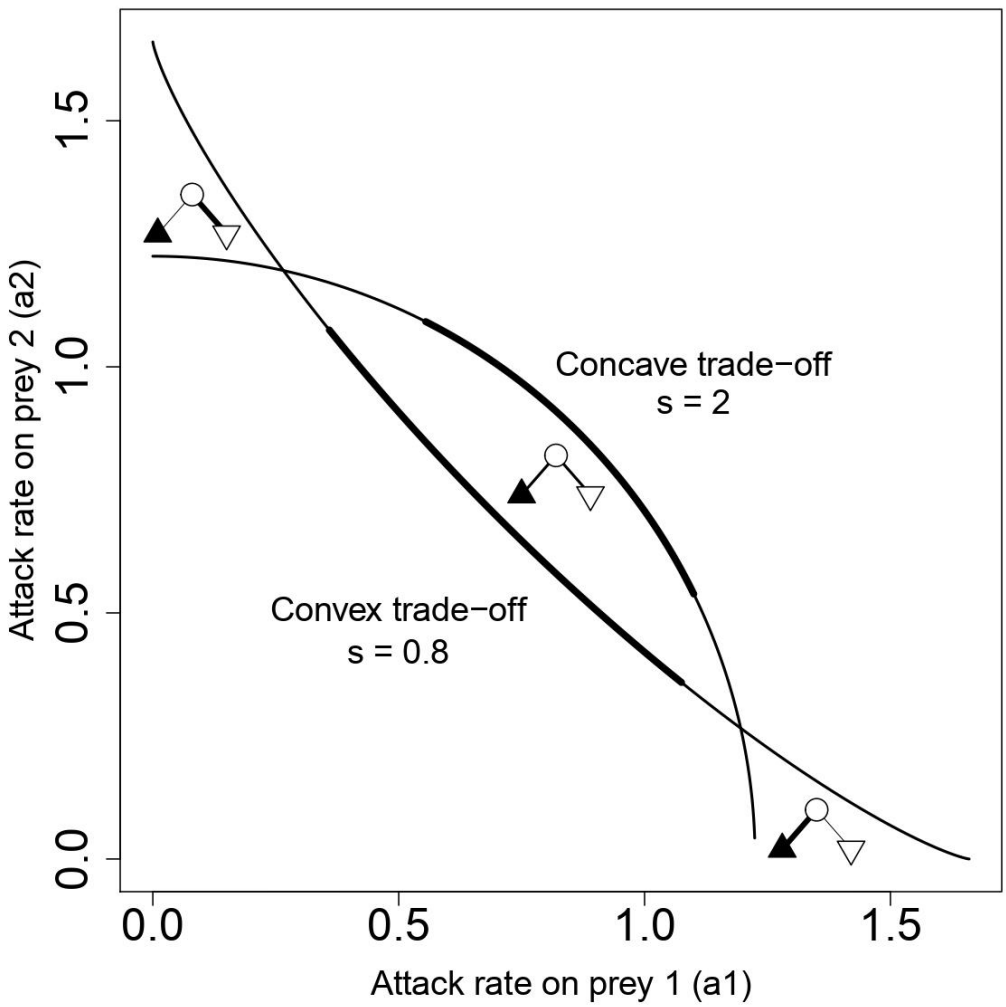

Figure 1. Concave (s>1) and convex (s<1) trade-off of predation investment in each prey. Modules schematize intensity of each trophic link. Symbols represent modules composition: predator (circle), competitive prey (triangle), alternative prey (inversed triangle), infected species are represented in black, healthy species in white. Bold lines show values for generalist predator and thin lines values for specialist predator. See Table 1 for parameter values.

2.3. Predator diet evolution

We use adaptive dynamics (Dieckmann and Law 1996; Geritz et al. 1998) to analyse diet evolution (i.e. variations in a_1). Adaptive dynamics assume that evolutionary and ecological dynamics occur on separate timescales, i.e. rare mutations, of small phenotypic effects. Under such conditions, resident phenotypes a_1 reach the ecological equilibrium before the next mutation occurs and evolution can be understood based on the invasibility of this equilibrium by nearby mutants (phenotype a_{1m}). The relative fitness of mutants can then be defined by their

intrinsic growth rate when rare in these equilibrium conditions (invasion fitness, Metz et al. 1992):

$$\omega(a_{1m}, a_1) = \frac{1}{P(a_{1m})} \frac{dP(a_{1m})}{dt}\bigg|_{\substack{P(a_{1m})\to 0 \\ P(a_1)\to P^*(a_1)}} \tag{3}$$

$$\omega(a_{1m}, a_1) = ea_{1m}({S_1}^* + {I_1}^*) + ej{I_1}^* + e(k_0 - {a_{1m}}^s)^{1/s}{N_2}^* - m$$

From equation (3), we see that the mutants having high attack rates on prey N_1 (a_{1m}) can invade (i.e. the fitness function is more likely positive) when N_1 population is large enough, or when the density of the alternative prey N_2 is small enough.

Variations of trait a_1 (with a_2 deduced using equ. (2)), can be described using the canonical equation of adaptive dynamics (Dieckmann and Law 1996):

$$\frac{da_1}{dt} = \frac{1}{2}\mu\,\sigma^2 P^*(a_1) \frac{\partial\omega(a_{1m}, a_1)}{\partial a_{1m}}\bigg|_{a_{1m}\to a_1} \tag{4}$$

Where μ is the *per capita* mutation rate, σ^2 the phenotypic variance linked to mutation process, and P^* the equilibrium density of residents (phenotype a_1). In equation (4), the selection process is described by the slope of the fitness landscape around the resident phenotype, $\frac{\partial\omega(a_{1m},a_1)}{\partial a_{1m}}\Big|_{a_{1m}\to a_1}$. The direction of evolution entirely depends on the sign of this slope, so that a_1 increases when the slope is positive and decreases when the slope is negative.

From equation (4), note that phenotypes no longer vary when the gradient is null, so that evolutionary singularities $\bar{a}_1$correspond to:

$$\frac{\partial\omega(a_{1m}, a_1)}{\partial a_{1m}}\bigg|_{a_{1m}\to a_1\to\overline{a_1}} = 0 \tag{5}$$

The dynamics around these singularities can be characterized using two criteria (Eshel 1983; Geritz et al. 1997; Diekmann 2004). Invasibility describes whether the singularity can be

invaded by nearby mutants (i.e. non-invasible strategies are ESS). Convergence describes if, starting close to the singularity, selected mutants are even closer to it, so that the strategy is eventually reached. Second derivatives of the fitness $\omega(a_{1m}, a_1)$ allow us to characterize invasibility and convergence (Diekmann 2004). We define c_{22} as:

$$c_{22} = \left.\frac{\partial^2 \omega}{\partial {a_{1m}}^2}\right|_{a_{1m} \to a_1 \to \bar{a}_1} \tag{6}$$

A singularity is non-invasible if $c_{22} < 0$, thus if

$$c_{22} = -{\bar{a}_1}^{s-2} k_0 (k_0 - {\bar{a}_1}^s)^{\frac{1}{s}-2} N_2^* (s-1) < 0 \tag{7}$$

From this equation we show that non-invasible singularities only occur with a concave trade-off (s>1).

Each singularity $\bar{a}_1$corresponds to a certain level of foraging generalism, as low values indicate that the predator mostly feeds on the alternative resource, while high values of $\bar{a}_1$indicate that the predator mainly feeds on the host. We consider a predator as a specialist if more than 75% of its diet is based on one prey (thin lines on Fig. 1).

2.4. Various outcomes of diet evolution

In our system, the predator specialises on the most competitive prey (N_1) in the absence of parasite (Not shown). In the presence of parasites, we can observe four outcomes regarding diet evolution, which are depicted on Figures 2, 3 and A1. Figure 2 represents the temporal dynamics of the four populations (uninfected prey, infected prey, non-host prey and predator) and the values of the attack rate on a_1 – simulations were performed with Scilab (version 5.5.2). Figures 3 and A1 are Pairwise Invasibility Plots (PIP, Diekmann 2004) – that were generated using Mathematica® 11.1.1 (Wolfram research), by plotting the sign of the relative fitness (Equ

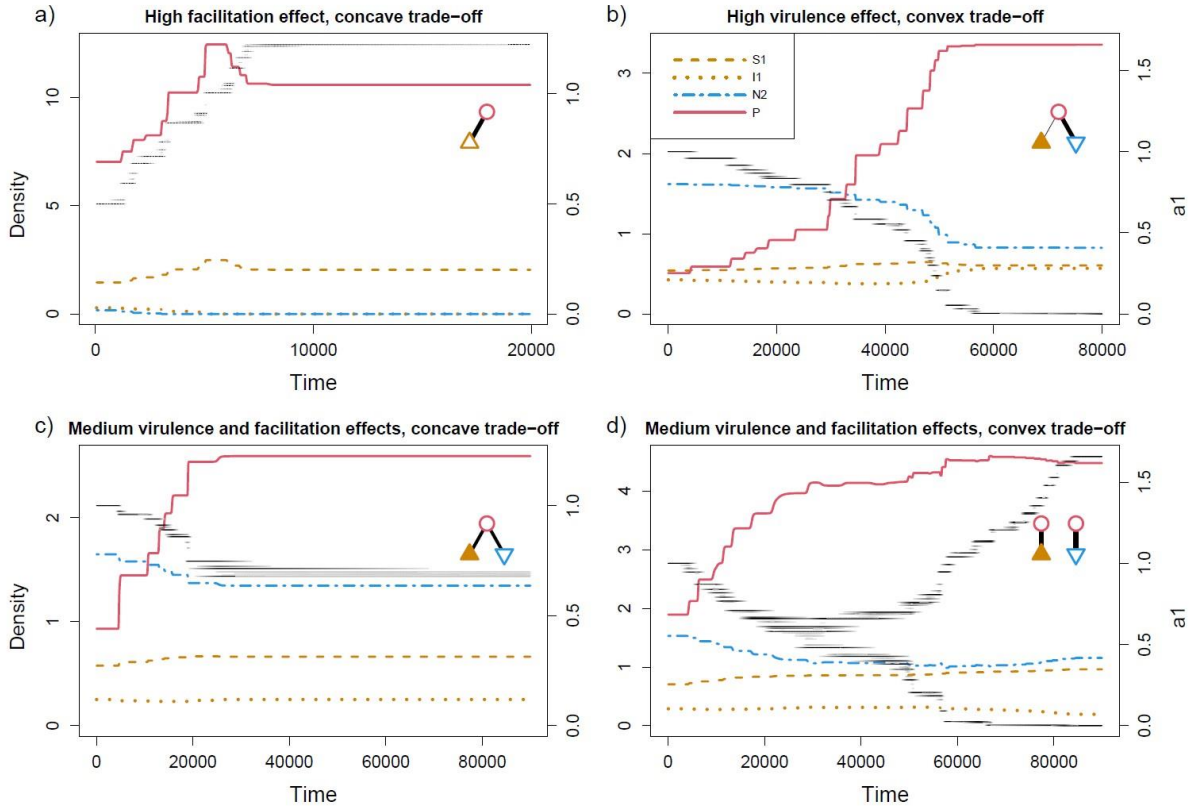

Figure 2. Various eco-evolutionary dynamics (species densities and trait value of the predator) of the module depending of trade-off shape and parasitism effects. a,c) concave trade-off, b,d) convex trade-off. a) high facilitation effect, b) high virulence effect, c,d) medium virulence and facilitation effect. Symbols show the module composition and the intensity of trophic link at the end of the simulation: predator (circle), competitive prey (triangle), alternative prey (inversed triangle). Infected species are show in fill, healthy species in empty. Healthy-host species S_1 is show in orange dashed line, infected-host species I_1 in orange dotted line, alternative prey N_2 in blue dashed-dotted line, predator P in red solid line. Values of a_1, the evolving trait, is shown in dark (grey scale indicates the density of the predator that have each trait value). Parameter values: see Table 1, except: a) n = 20, j = 5, s = 2; b) n = 15, j = 2, s = 0.8 ; c) n = 20, j = 2, s = 2 ; d) n = 15, j = 3, s = 0.8.

(3)) when all species equilibriums are positive –, which describe the relative fitness $\omega(a_{1m}, a_1)$: the value of the evolving trait a1 of the resident population (x-axis) relative to that of the mutant (y-axis). The mutant has a positive relative fitness and thus can invade the population in the dark area, and a negative relative fitness in the white area. Note that because mutations are small and rare, resident and mutant trait values are close to the diagonal $x = y$, and thus the evolution of the trait follows the diagonal, depending of the fitness value above and below the diagonal. Evolutionary directions are shown with white arrows.

Generally, we observe a specialization on the infected prey with high facilitation effects and a specialization on alternative prey with high virulence effects. At intermediate effects we

obtain generalist predators with concave trade-off, and diversification and evolutionary multistability with convex trade-off. In detail, we show that the predator can be generalist (Figs 2c, 3b, A1b,c), which requires that the singularity $\bar{a}_1$ is a Continuously Stable Strategy (CSS, i.e. convergent and non-invasible) with intermediate values (see figure 1). As previously showed (eq. 7), this only occurs for concave trade-offs. Predators can also be specialists, either on the host (Fig. 2a) or on the alternative prey (Fig. 2b), which can be achieved in four ways.

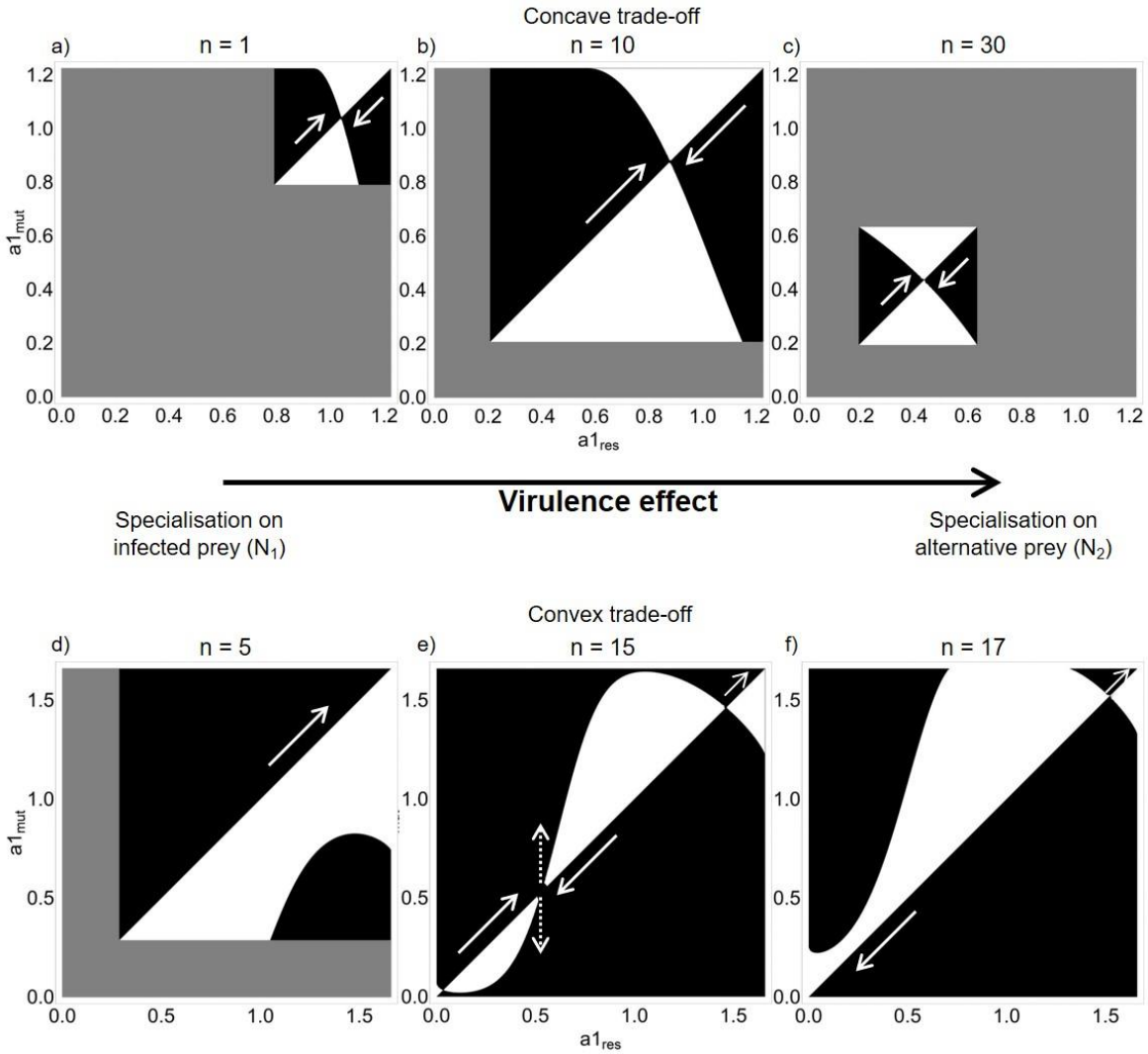

Figure 3. Pairwise Invasibility Plots of evolution of predator diet (i.e. evolution of a_1) for a concave (a-c) and a convex (d-f) trade-offs, when increasing intensity of virulence effect. On PIP, black area corresponds to a positive relative fitness of mutants, white area corresponds to a negative relative fitness of mutants, grey area shows no-coexistence of the system. The white solid arrows show the direction of evolution, the white dotted arrows show evolutionary branching. a-c) CSS, d) no singularity, e) one EBP and two repellors, f) one repellor. Parameter values: see Table 1, except a) $n = 1$, $j = 1$, $s = 2$; b) $n = 10$, $j = 1$, $s = 2$; c) $n = 30$, $j = 1$, $s = 2$; d) $n = 5$, $j = 3$, $s = 0.8$; e) $n = 15$, $j = 3$, $s = 0.8$; f) $n = 17$, $j = 3$, $s = 0.8$. Note that Fig A1 shows the results with the facilitation effect.

First, specialists could correspond to CSS of large or small values (Fig. 3a,c, A1a). Alternatively, the singularity may be a Repellor (i.e. non-convergent and invasible), with evolution leading to specialization on one species or another depending on the initial diet. In our model, repellors only occur with convex trade-offs as they are invasible (eq. 7) (Fig. 3f). A last possibility to obtain a specialist is that no singularity exists, when the fitness gradient (eq. 3) is always positive or negative. Evolution then leads to a continuous increase or decrease of a_1, so that complete specialization is selected (Fig. 3d, A1d,f). Differentiating the fitness function (3) to get the fitness gradient, it can easily be shown that the loss of one prey systematically selects specialization on the other prey (Fig. 2a). Finally, we can obtain a diversification of the predator strategies (polymorphism) with the coexistence of two specialists, one on each prey (Figs 2d, 3e, A1e). Such evolutionary dynamics correspond to Evolutionary Branching Points (EBP): a singularity that is convergent and invasible. Such branchings occur only for convex trade-offs where the singularity is invasible (eq. 7). Note that these outcomes are not mutually exclusive, as several singularities may coexist for a given set of parameters, yielding complex evolutionary dynamics. For instance, on Fig. 3e, two repellors coexist with a branching point, so that, depending of its initial diet, a predator can evolve to specialisation on the host (if the initial a_1 is high, i.e. higher than the value of the highest repellor), on the alternative prey (if the initial a_1 is low, i.e. lower than the value of the lowest repellor), or to diversification (i.e. a coalition of two specialists as illustrated on Fig. 2d) (Fig. 3e and A1e).

2.5. Parasites affect diet evolution

Now that we have described the possible outcomes, we investigate how the intensity of the parasite-induced virulence and facilitation effects constrains their occurrence. Virulence effects are depicted on Fig. 3, facilitation effects on Fig. A1 and both effects investigated concurrently on Fig. 4 – Data were generated with Mathematica®, using analytical form of the fitness

gradient and the equilibriums of the four species: for each parameters couple we obtain the numerical value of the fitness and then search the zero-crossing along an a_1 gradient; we reported either the corresponding value (for a null fitness) of a_1 for the concave trade-off, either the number of zero-crossing for the convex trade-off; for each zero-crossing we control for the existence of all species.

When only virulence effects occur (Fig. 3), as expected, a higher virulence changes the outcome of evolution from specialisation on the infected prey to specialisation on the alternative prey. Conversely, but also in agreement with our predictions, higher facilitation effects eventually lead to specialisation on the infected prey (Appendix A). In both cases, intermediate effects either lead to generalist strategies (concave trade-offs, Fig. 3b, A1b), or to diversification (convex trade-offs, Fig. 3e, A1e). To go further and assuming concave trade-offs, increasing the virulence effects move the CSS toward lower values of a_1, so that the predator increasingly specializes on the alternative prey (Fig. 3a-c), while increasing facilitation effects move the CSS toward higher values of a_1 so that the predator increasingly relies on the host species (Fig. A1a-c). Similarly, for convex trade-offs, higher virulence effects move the system from a complete specialization on the host (Fig. 3d) to a situation in which most dynamics would lead to specialization on the alternative prey (fig. 3f). Analogous variations can be described when decreasing the facilitation effects (fig. A1d-f).

Figure 4 summarizes these antagonistic consequences of virulence and facilitation effects, and how they depend on the shape of the trade-off function. As previously described, specialist strategies are selected when either virulence or facilitation effects are strong (Fig. 4, light grey and black areas), regardless of the shape of the trade-off. However, most parasites are likely to induce virulence and facilitation effects simultaneously, so that the evolutionary outcome may often be in between these extreme cases (i.e. in the middle of the panels of Fig. 4). Such evolutionary outcomes depend on the shape of the trade-off. Concave trade-offs allow

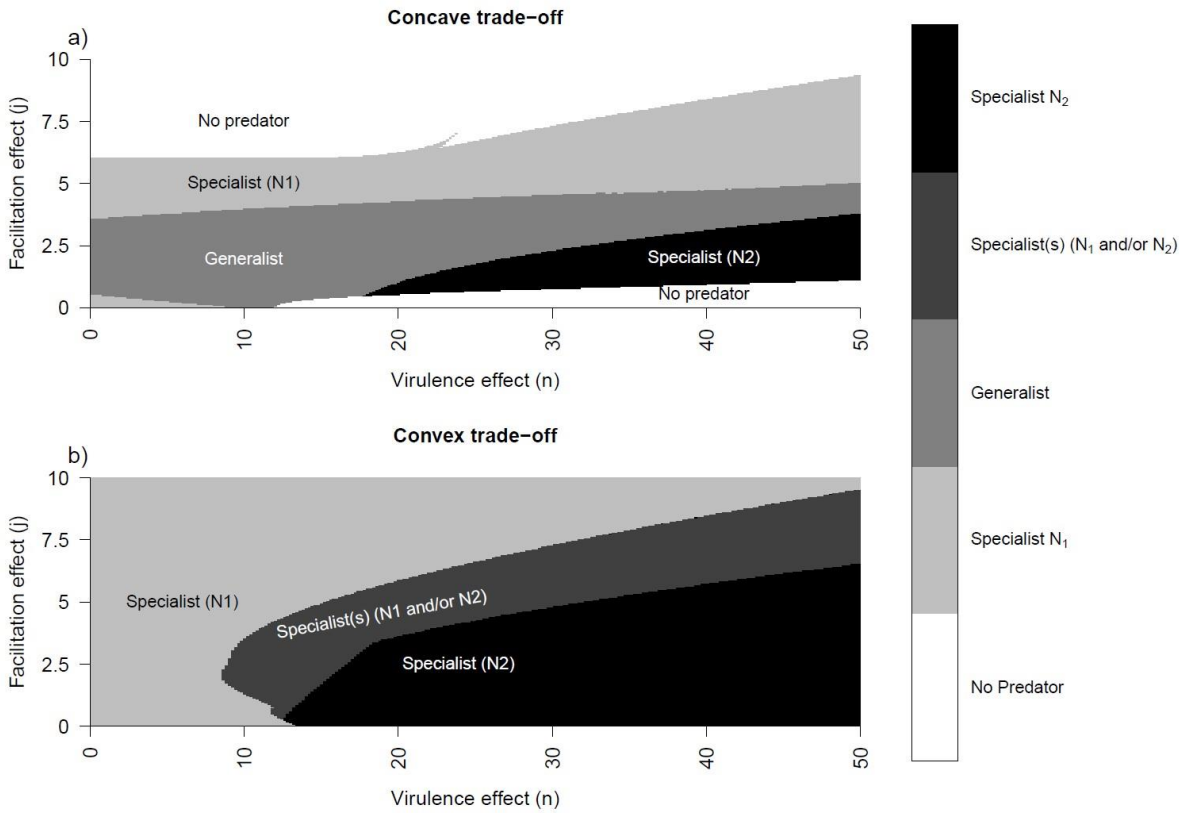

Figure 4. Predator diet after evolution, function of virulence effect (x-axis) and facilitation effect (y-axis) of the parasite, with concave (a) and convex (b) trade-offs. Parameter values: see Table 1, except a) s = 2, b) s = 0.8.

generalist strategies (medium grey, Fig. 4a) while convex trade-offs favor the diversification of predator diets, potentially leading to the coexistence of specialist strategies (dark grey, Fig. 4b). Finally, note on Figure 4 that extreme effects may lead to the loss of coexistence within the module (white areas), highlighting the fact that evolutionary responses of predators to prey infection may have far reaching consequences for community structure. We now analyze such consequences for species coexistence in further details.

2.6. Consequences on coexistence

While we know that parasitism may directly constrain coexistence of prey by changing the relative weight of direct and apparent competition (Prosnier et al. 2018), here we highlight that evolutionary dynamics in response to parasitism may favor coexistence in trophic modules. To clarify the role of evolution, on figure 5, we show the coexistence conditions with (grey area) and without (area delimited by the solid line) diet evolution. For the “without evolution”

scenarios, we consider as reference cases in which the predator is specialised on the infected prey (Fig. 5a), on the alternative prey (Fig. 5c), or is a generalist (Fig. 5b).

Our results suggest that evolution systematically enhances the size of coexistence area. The coexistence area is increased by a factor 2.22 compared to a predator specialist on the host prey (Fig. 5a), of 1.26 compared to a generalist predator (Fig. 5b), and of 1.04 compared to a predator specialist on the alternative prey (Fig. 5c). Evolution particularly acts at low and high intensities of virulence effects. At low intensities where the combination of predation and competition could result in the exclusion of the alternative prey (Fig. 5c), evolution allows coexistence because it selects predator diets that are more specialized on the host prey. Thereby, evolution increases apparent competition on the competitively dominant prey. At high intensities, the

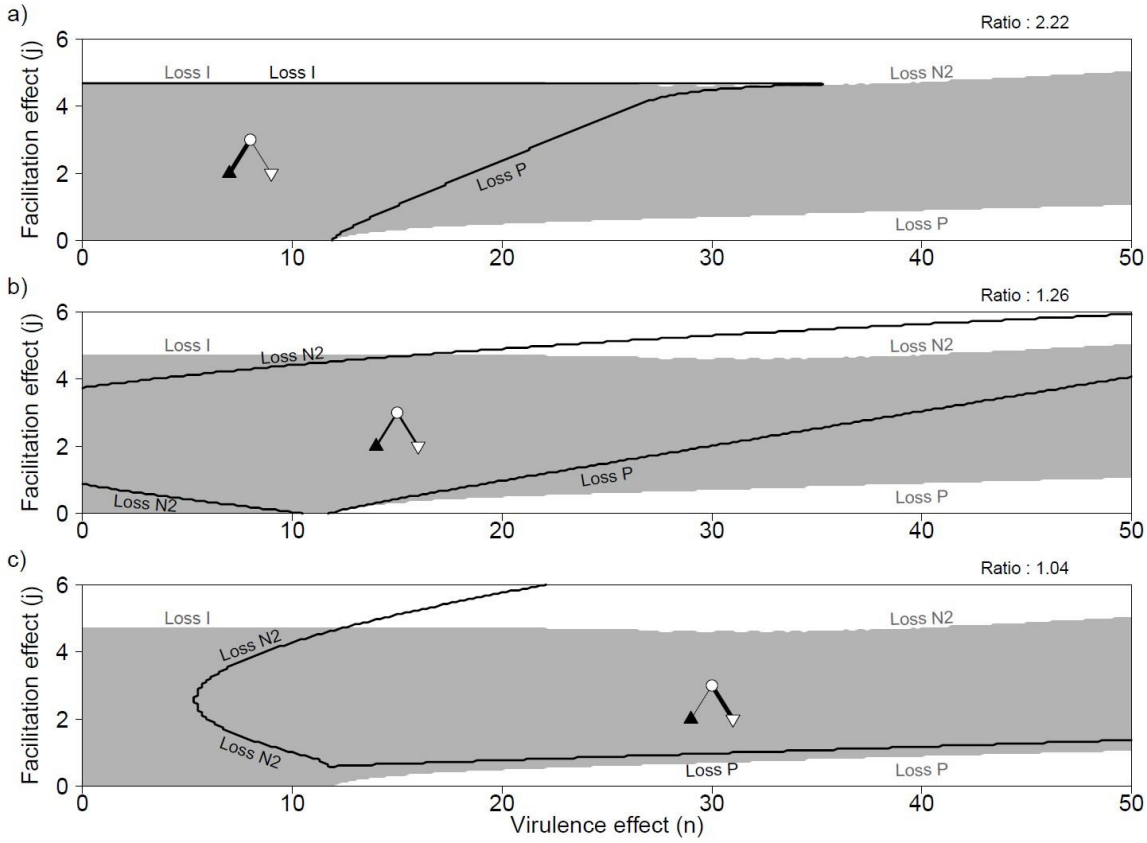

Figure 5. Coexistence of the three species, with convex trade-off, with evolution (grey area) and without evolution (inside the black delimitation). When the predator is a) specialist on the infected prey N_1, b) generalist, c) specialist on the alternative prey N_2. The species loss at the limits are written, in dark without evolution, in grey with evolution. Ratio is the ratio of coexistence surface with/without evolution. Parameters value: see Table 1 except s=2. Note that Fig B1 shows the results with a concave trade-off.

predator is excluded because the parasite decreases the amount of prey available, especially in case of specialisation on the host prey (Fig. 5a). Here evolution promotes coexistence in that it favors the diets more oriented toward the alternative prey. Note that while the global effect of evolution is to enhance coexistence, it may decrease it for particular effects. For instance, when both virulence and facilitation effects are high, from an ecological point of view a balance exists between apparent and direct competition among prey and coexistence is possible without evolution (e.g. Fig. 5b,c, see also Prosnier et al. 2018). Evolution however increases the weight of apparent competition and results in the exclusion of the alternative prey. While Fig. 5 assumes a concave trade-off, we note that the effects of evolution on coexistence are similar for convex trade-offs (see Appendix B).

3. Discussion

Parasites likely affect food webs in many ways. Because of their virulence and the resulting decrease in fecundity, they can constrain the availability of prey populations, thereby leading to resource limitation for predators. Alternatively, parasite-induced phenotypic alterations can strengthen trophic interactions, making infected prey more susceptible to predation. While these ecological consequences of parasitism for food webs have received some attention (McNaughton 1992; Banerji et al. 2015; Buck and Ripple 2017; Buck 2019), parasitism also affects coevolution between prey and predator species. Here, our model shows that the evolution of predator diet depends on the type of parasitism effect (virulence or facilitation) and its intensity. We show that virulence effects usually lead to the selection of increased predation on the alternative (i.e. non host) prey, while facilitation effects favor predation on the host species. Such results are consistent with our predictions based on host profitability: virulence effects reduce profitability whereas facilitation effects increase it. Evolution also favors coexistence among prey and between predators and parasites.

As expected, virulence and facilitation effects have antagonistic consequences on the evolution of predator diet. Virulence effects (i.e. decreased host fecundity) lead to specialization on the alternative prey. Because virulence decreases the host prey population, the first part of the fitness function (Eq. 2) is decreased and foraging strategies that focus more on the alternative prey are favored. Conversely, facilitation effects (i.e. increase in host vulnerability) lead to specialization on the infected prey. Again, turning to the definition of fitness (Eq. 2), the first term is increased when facilitation effects are present, so that strategies focusing more on the host species are selected. Consequently, the evolution of predator diet can here be understood in the context of optimal foraging (Emlen 1966; MacArthur and Pianka 1966; Charnov 1976*a*, 1976*b*). Predators can be generalists when virulence and facilitation effects have intermediate intensities and/or when they act simultaneously. Thus, we expect diet to shift progressively from specialism on one prey to the other prey when the two effects vary. Generalism requires balanced virulence and facilitation effects and concave trade-offs. It is replaced by a coalition of two specialists given convex trade-offs. Such a link between trade-off shape and diversification is consistent with previous theoretical works on the evolution of specialization (Egas et al. 2004; Rueffler et al. 2006; van Velzen and Etienne 2013).

The evolution of predator diet influences coexistence between the two prey species, because it depends on the balance between direct and apparent competition. It also influences the predator-parasite coexistence, by modulating the resource overlap between the two species. Concerning the coexistence of prey species, virulence effects reduce host competitivity and may ultimately lead to its exclusion (Prosnier et al. 2018). With evolution, however, a decrease in host density selects a lower consumption by the predator, which balances the decrease in host competitive ability thereby favoring its persistence. Similarly, if the alternative prey were to become rare, evolution would select diets that are more focused on the host prey, so that coexistence would again be favored. In a nutshell, adaptive foraging creates a negative

frequency-dependence that promotes the persistence of the rare prey, a mechanism already pointed out by several works (Kondoh 2003; Křivan and Eisner 2003; Loeuille 2010) but not to our knowledge in the context of parasitism. Conversely, given facilitation effects, evolution selects higher predation on the host, so that the negative effects of parasitism on prey coexistence are magnified (i.e. both parasitism and evolution increase predation on the host prey).

We observe an increase in predator-parasite coexistence due to niche partitioning between parasite and predator when parasites induce virulence effects. This is consistent with approaches that unify parasite and predator under the same natural enemy concept (Raffel et al. 2008). Here, the parasite and the predator can be viewed as intraguild predators since they compete for the same trophic resource (i.e. the host population) while the predator also kills the parasite through concomitant predation (i.e. when it consumes infected individuals) (Sieber and Hilker 2011). Niche partitioning between intraguild predators due to evolutionary dynamics has already been shown (Ingram et al. 2012). While the widespread co-occurrence of parasites and predators in food webs (Lafferty et al. 2006) can be puzzling given the competition that exists between the two groups, our results on the role of diet evolution may offer a possible explanation. Stewart et al. (2018) described what could be interpreted as niche partitioning between the parasites and predators of *Ceriodaphnia* in the lake Gatun (Panama). However, direct comparison with our theoretical work is difficult as the evolutionary processes remain unknown. An empirical validation of our results would involve experimental works based on short-lived organism to allow rapid evolution. For instance, that could be the system used by Banerji et al. (2015): *Paramecium caudatum* infected by *Holospora undulata* and consumed by a rotifer, *Didinium nasutum*, which allows ecological dynamics in less than two months. We could add a competitor to *P. caudatum* through another species of *Paramecium* or another ciliate like *Stylonychia pustulata* as done by Gause (1934).

In this work, we show how parasites may affect the evolution of predator diet and how in return adaptive foraging alters species coexistence. How such relationship between evolution of foraging and parasitism affects more complex food webs would be a natural extension of the present work. For instance, while coexistence of predators and parasites may not be easily explained from an ecological point of view (due to competition), our results suggest that evolution can provide the niche partitioning needed to allow the maintenance of their diversity and could explain why "a healthy system to be one that is rich in parasite species" (Hudson et al. 2006). However, at this point, it seems premature to extrapolate our results to complex food webs, as empirical information is lacking for several important aspects. We chose to discuss two points: the functional response and the predator infection.

Because we aimed at studying a variety of evolutionary scenarios (flexible trade-offs, different effects of parasitism), we voluntarily simplified the ecological dynamics. We for instance use a type-I functional response. While type-II functional responses are sometimes preferred, it is unclear, in the case of facilitation effects of the parasite, which parameters of the functional response would be modified (i.e. handling times or attack rates). Also, we expect that our main results concerning the effect of adaptive foraging on species coexistence and predator-parasite niche separation to be robust. For instance, given a type II function, it is easy to show that when the infected prey is low due to parasitism effects, evolution will still favor consuming the alternative prey, thereby promoting prey coexistence and niche separation, as in the type I case. These results are consistent with literature that suggest that adaptive foraging creates a frequency-dependent process that is utterly stabilizing, regardless of the functional response used (Loeuille 2010; Valdovinos et al. 2010).

Another interesting point, that it comes in mind when studying parasitism and predation concurrently, and in particular with facilitation effects, is the possibility for the predator to be infected by the parasite when it eats an infected prey – i.e. a trophic transmission (see for

instance the model of Fleischer et al. (2020), where predator evolve in response to trophically-transmitted parasites). Considering our results, if parasites have also a virulent effect on the predator, we should expect that it magnifies the niche separation, because the parasite should be both a competitor and a parasite for the predator. However, in some well-known cases niche separation is not possible: when the predator is the final host (i.e. parasites with complex cycle). Because parasites then need to infect the predator to complete their cycle, we deduce from our results some constraints on the virulence and the facilitation effects due to the evolution of predator diet. The virulence should be lower both for the prey and the predator (see the model of Kuris, 2003, for a low virulence for the predator, but not for the prey, and the review of Fayard et al. (2020) for a low virulence on the intermediate host). However, if avirulence is not possible (due to evolutionary or biological constraints), the parasite should maximize the facilitation effect (i.e. the intermediate host should be prey by the final host before its death). Because predation on host is necessary for the parasite, the niche overlap should maintain coexistence between the predator and the parasite, contrary to our system where such situations would lead to parasite extinction. Moreover, it should be beneficial to the predator (Øverli and Johansen 2019). Consequently, we show from our work and the resulting discussion, that studying facilitation effects is also interesting for non trophically-transmitted parasites, and virulence effects for trophically-transmitted parasites. However, and despite their non-independence, both lacking in past works.

Finally, despite some simplifications of our model, the possible generalization of the results and the various scenarios studied allow us to consider possible effects of parasitism in food webs that seem currently understudied. Our study should be a base both for empirical works, as detailed by Fleischer et al. (2020) on the stickleback model that could prey on benthic and/or pelagic prey variously infected, and for theoretical works on the complex implications of parasitism on food webs.

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Appendix

A) Diet evolution due to parasitism: facilitation effect

When only facilitation effects occur (Fig. A1), a higher facilitation effect always leads evolution from specialisation on the alternative prey to specialisation on the infected prey, thus antagonist to virulence effects. Intermediate intensity either lead to generalist strategies

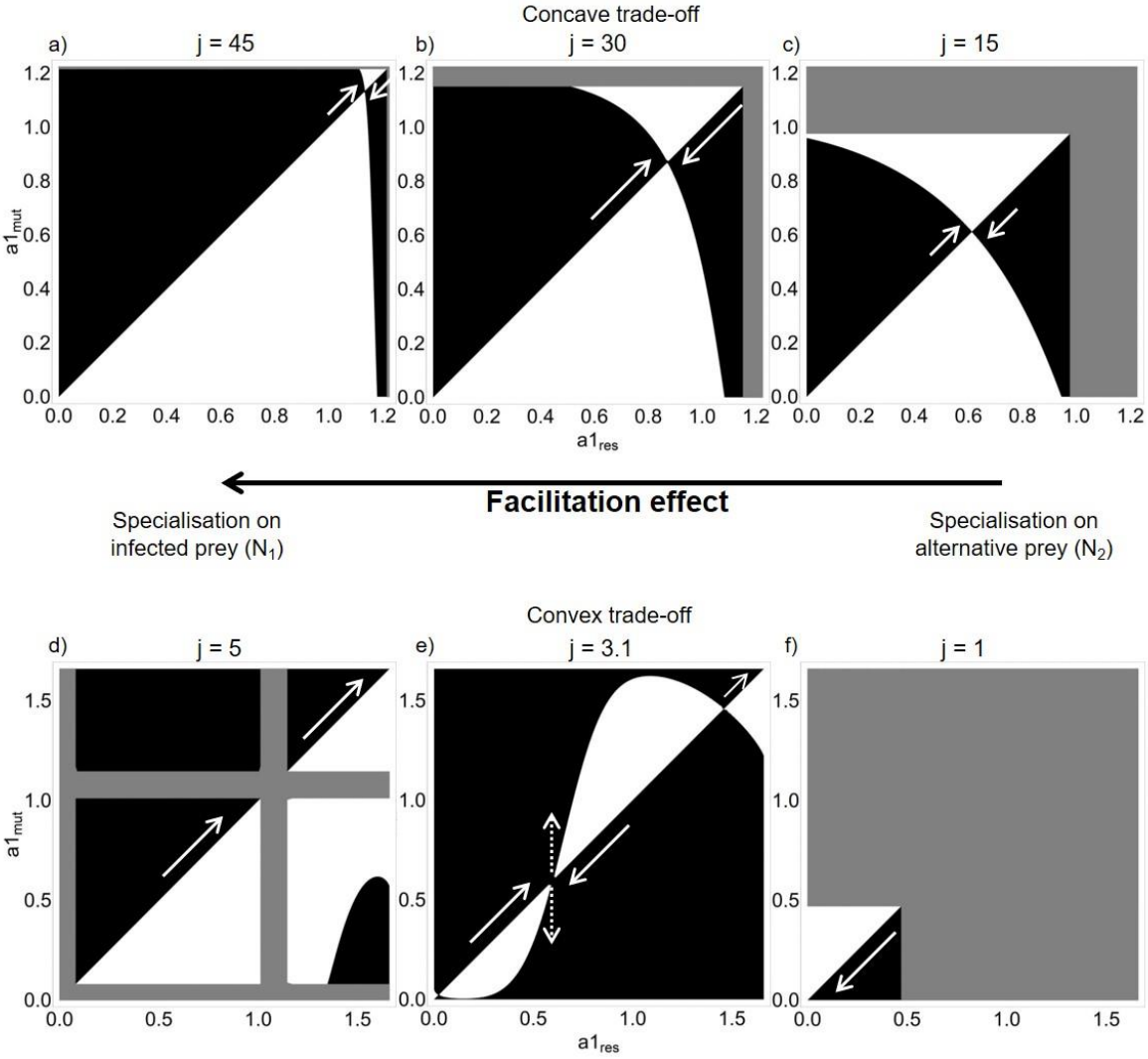

Figure A1. Pairwise Invasibility Plots of evolution of predator diet (i.e. evolution of a_1) for a concave (a-c) and a convex (d-f) trade-offs, when increasing intensity of facilitation effect. On PIP, black area corresponds to a positive relative fitness of mutants, white area corresponds to a negative relative fitness of mutants, grey area shows no-coexistence of the system. The white solid arrows show the direction of evolution, the white dotted arrows show evolutionary branching. a-c) CSS, d) no singularity, e) one EBP and two repellors, f) no singularity. Parameter values: see Table 1, except a) n = 20, j = 45, s = 2 ; b) n = 20, j = 30, s = 2 ; c) n = 20, j = 15, s = 2 ; d) n = 15, j = 5, s = 0.8 ; e) n = 15, j = 3.1, s = 0.8 ; f) n = 15, j = 1, s = 0.8. Note that Fig 3 shows the results with the virulence effect.

(concave trade-offs, A1b), or to diversification (convex trade-offs, A1e). To go further and assuming concave trade-offs, increasing the facilitation effects move the CSS toward higher values of a_1, so that the predator increasingly specializes on the infected prey (Fig. 1Aa-c). Similarly, for convex trade-offs, higher virulence effects move the system from a complete specialization on the alternative prey (Fig. A1f), to a situation in which most dynamics would converge to diversification (i.e. the coexistence of two specialists, Fig. A1e), eventually leading to situations in which almost all initial diets will lead to specialization on the host (fig. A1d). However, note that, in some case, as on Fig. A1d if you start with $0.1 < a_1 < 1$ you may loss coexistence of the system through the loss of the parasite.

B) Consequences on coexistence: concave trade-off

Similarly, as for convex trade-off, we compare the coexistence conditions with (grey area) and without (area delimited by the solid line) diet evolution. For the "without evolution" scenarios, we consider the cases where the predator is specialised on the infected prey (Fig. B1a), the alternative prey (Fig. B1c), or is a generalist (Fig. B1b).

Our results show that coexistence with evolution is higher than coexistence without evolution. The coexistence area is increased by a factor 3.58 compared to a predator specialist on the host prey (Fig. B1a), of 3.46 compared to a generalist predator (Fig. B1b), and of 1.23 compared to a predator specialist on the alternative prey (Fig. B1c). Evolution particularly acts at low and high intensities of virulence effects. At low intensities where the combination of predation and competition could result in the exclusion of the alternative prey (Fig. B1c),

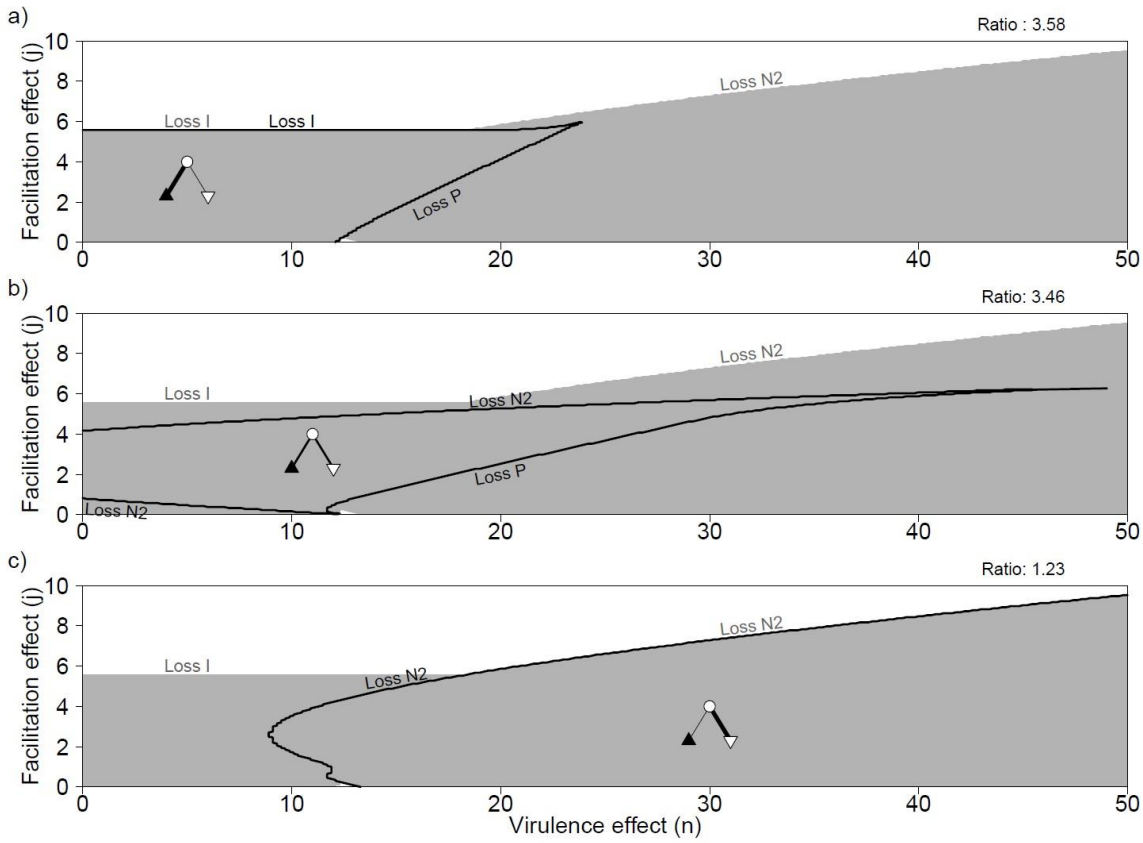

Figure B1. Coexistence of the three species, with concave trade-off, with evolution (grey area) and without evolution (inside the black delimitation). When the predator is a) specialist on the infected prey N_1, b) generalist, c) specialist on the alternative prey N_2. The species loss at the limits are written, in dark without evolution, in grey with evolution. Ratio is the ratio of coexistence surface with/without evolution. Parameters value: see Table 1 except s=0.8. Note that Fig. 5 shows the results with a convexe trade-off.

evolution allows coexistence because it selects predator diets that are more specialized on the host prey. Thereby, evolution increases apparent competition on the prey that is, without evolution, competitively dominant. At high intensities, the predator might be excluded because the parasite decreases the amount of prey available, especially in case of specialization on the host prey (Fig. B1a). Here evolution promotes coexistence in that it favors the diets more oriented toward the alternative prey.