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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 profitabilities. In this work, we study the eco-evolutionary consequences of prey infection 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 (interaction 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 interaction 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 interaction 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.

Keyword: Adaptive foraging, virulence, vulnerability, predator diet, profitability, parasitism

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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.

We can distinguish direct and indirect effects of infection on host fitness. Direct effects on fitness (virulence effects hereafter) occur when parasites reduce host fecundity, increase host mortality or both. For instance, increased mortality is described in semi-captive populations of elephants (*Elephas maximus*), infected by various parasites (Lynsdale et al. 2017) or observed in laboratory for fishes (Brassard et al. 1982), guppies (*Lebistes reticulatis*) and brook trout (*Salvelinus fontinalis*), infected by a trematode (*Diplostomum spathaceum*). Infection-induced

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fecundity reduction is documented for various animals like water fleas (*Daphnia sp.*) hosting bacterial, viruses or fungi (Decaestecker et al. 2003). Such virulence effects 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 (interaction 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). 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. Virulence and interaction effects can therefore act in opposite ways on profitability and infection by a single parasite species can induce both effects, which make the consequences on predator foraging strategies 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

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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 interaction 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 interaction 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 interaction 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 to decompose 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

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(concomitant predation). Second, predators can limit parasite transmission by reducing host density (e.g. Dallas et al. 2018). Such negative effects should be amplified when parasites increase host vulnerability through interaction effects (Packer et al. 2003; 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 interaction effects, as they increase prey vulnerability (Hethcote et al. 2004; 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, interaction 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 interaction 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 interaction effects have the opposite effect. Our results also show that evolution favors niche separation among parasites and predators, thereby promoting coexistence.

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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_i the total prey species i population, S_i and I_i 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, N_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_{ii} the *per capita* competition rate of prey i, c_{ij} the *per capita* competition effect of prey j on prey i, a_i the *per capita* attack rate of the predator on prey i, e the conversion efficiency. Virulence effects are implemented through a decreased fecundity in the infected prey (parameter n) and interaction 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.

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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	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	-	ind^{-1}.m^2.d^{-1}
a_2	*Per capita* attack rate on prey species 2	-	ind^{-1}.m^2.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	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}
	Trade-off (Eq. (2))		
s	Shape of the trade-off	-	Dimensionless
k_0	Total Allocation in foraging effort	1.5	ind^{-1}.m^2.d^{-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.

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: $s > 1$), andk_0 corresponds to the total allocation of energy or time in predation.

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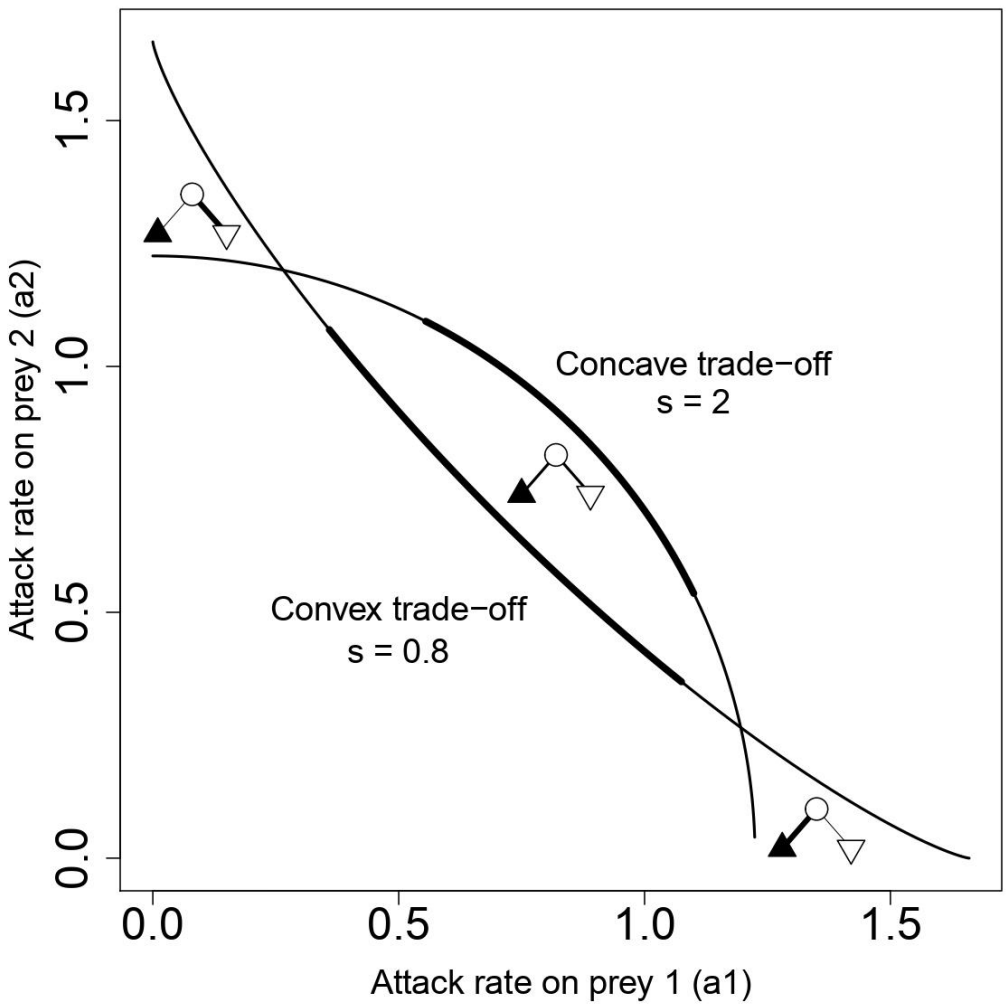

Figure 1. Concave (s>1) and convex (s<1) trade-off of predation investment in each prey. Modules schematize intensity of each trophic link. Symbols represent modules composition: predator (circle), competitive prey (triangle), alternative prey (inversed triangle), infected species are represented in 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 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):

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

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

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 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

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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:

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

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

$$c_{22} = -{\bar{a}_1}^{s-2} k_0 (k_0 - {\bar{a}_1}^s)^{\frac{1}{s}-2} N_2^*(s-1) < 0 \quad (6)$$

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 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. Figures 3 and A1 are Pairwise Invasibility Plots (PIP, Diekmann 2004), 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

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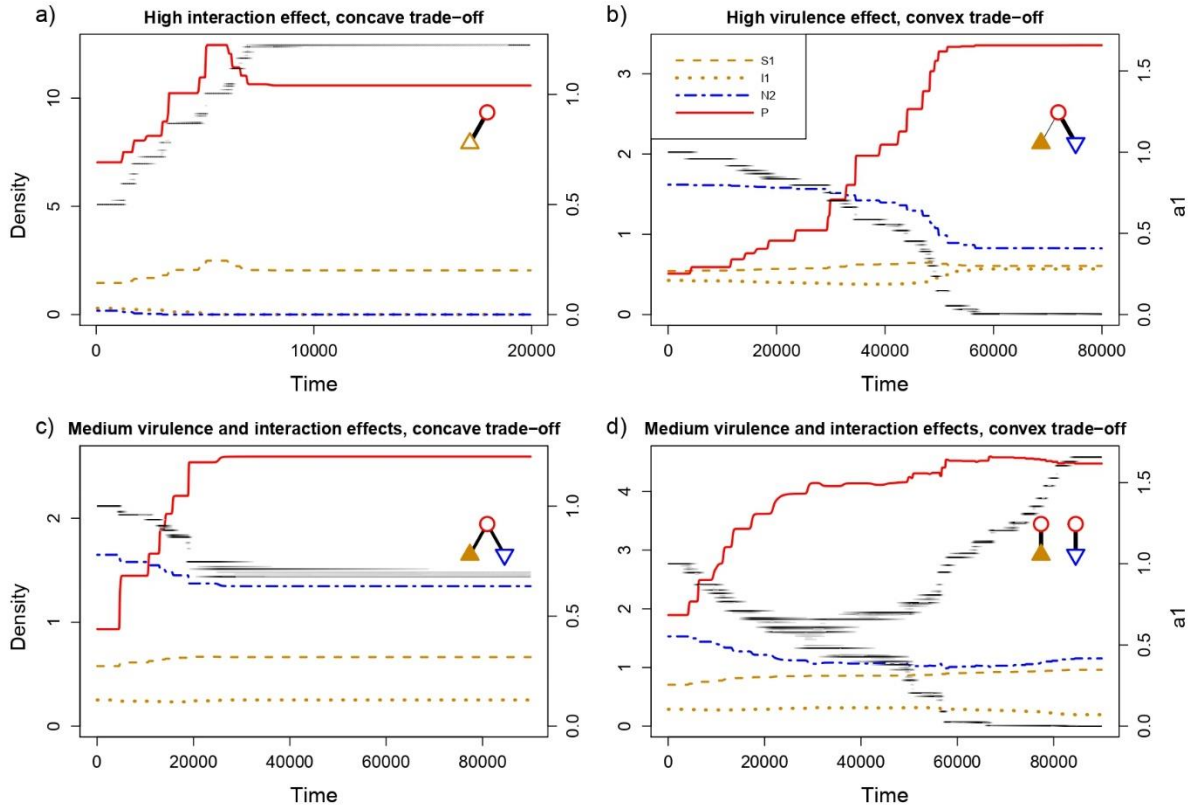

Figure 2. Various eco-evolutionary dynamics (species densities and trait value of the predator) of the module depending of trade-off shape and parasitism effects. a,c) concave trade-off, b,d) convex trade-off. a) high interaction effect, b) high virulence effect, c,d) medium virulence and interaction 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) $n = 15$, $j = 3$, $s = 0.8$.

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. 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. 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

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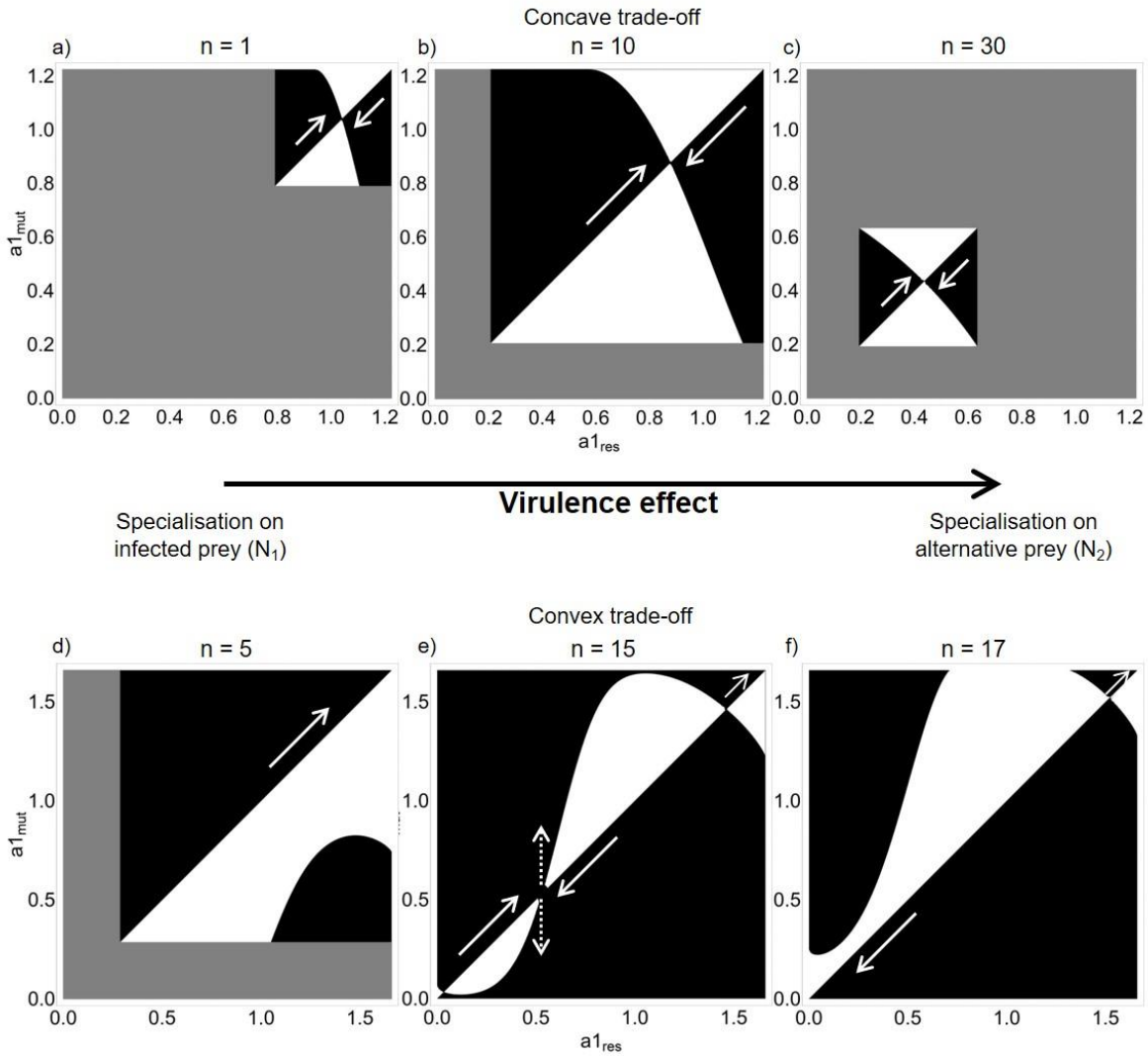

Figure 3. Pairwise Invasibility Plots of evolution of predator diet (i.e. evolution of a_1) for a concave (a-c) and a convex (d-f) trade-offs, when increasing intensity of virulence effect. On PIP, black area corresponds to a positive relative fitness of mutants, white area corresponds to a negative 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 interaction effect.

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

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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 interaction effects constrains their occurrence. Virulence effects are depicted on Fig. 3, interaction effects on Fig. A1 and both effects investigated concurrently on Fig. 4.

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 interaction 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 interaction effects move the

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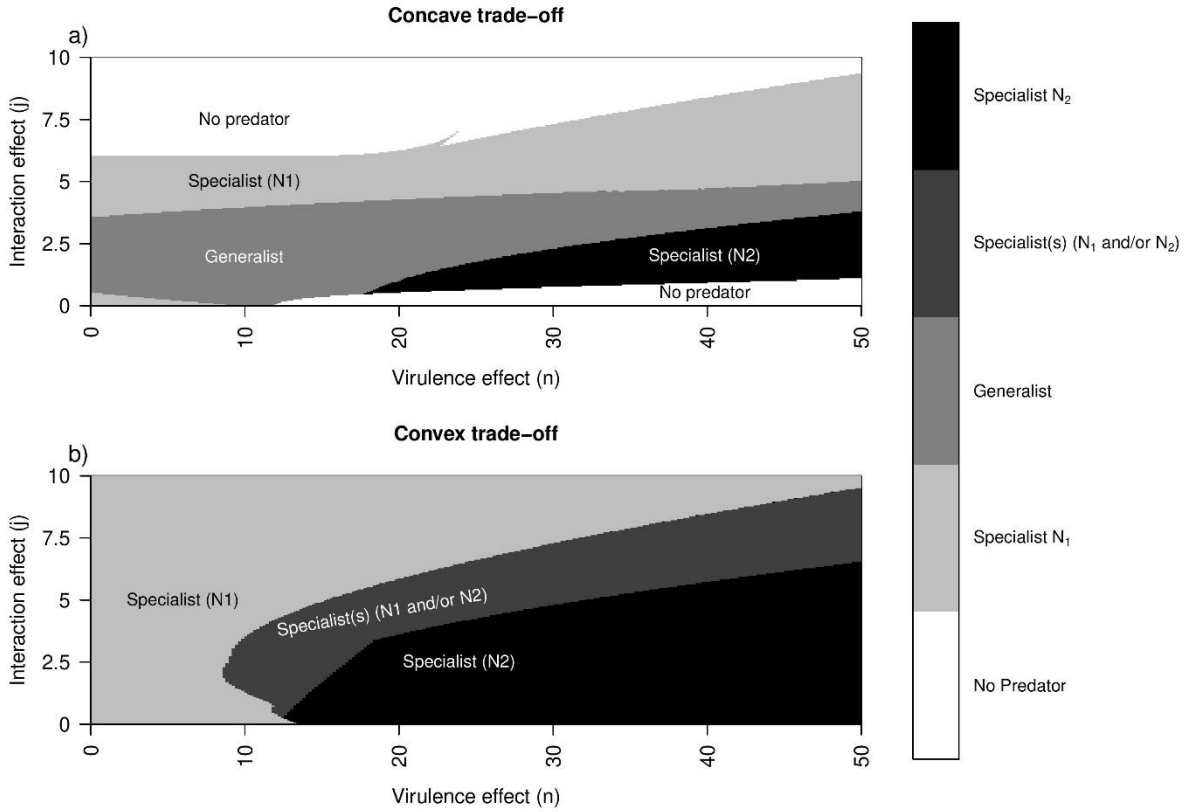

Figure 4. Predator diet after evolution, function of virulence effect (x-axis) and interaction effect (y-axis) of the parasite, with concave (a) and convex (b) trade-offs. Parameter values: see Table 1, except a) s = 2, b) s = 0.8.

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 interaction effects (fig. A1d-f).

Figure 4 summarizes these antagonistic consequences of virulence and interaction effects, and how they depend on the shape of the trade-off function. As previously described, specialist strategies are selected when either virulence or interaction 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 interaction 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

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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 by changing the relative weight of direct and apparent competition (Prosnier et al. 2018), we here highlight that evolutionary dynamics in response to parasitism may influence coexistence in trophic modules.

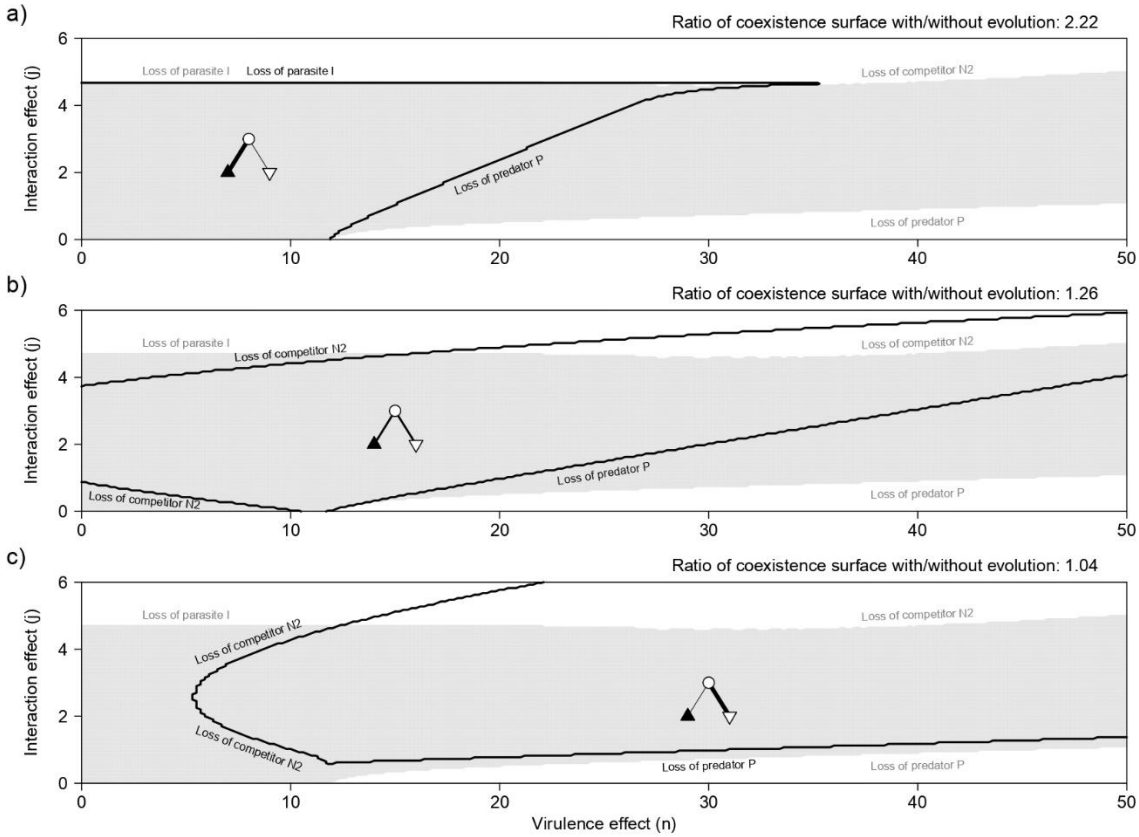

Figure 5. Coexistence of the three species, with convex trade-off, with evolution (grey area) and without evolution (inside the black delimitation). When the predator is a) specialist on the infected prey N_1, b) generalist, c) specialist on the alternative prey N_2. The species loss at the limits are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

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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 enhance coexistence. 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 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 interaction 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

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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 interaction) 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 interaction effects favor predation on the host species. Such results are consistent with our predictions based on host profitability: virulence effects reduce profitability whereas interaction effects increase it. Evolution also favors coexistence among prey and between predators and parasites.

As expected, virulence and interactions effects have antagonistic consequences on the evolution of predator diet. Virulence effects (i.e. decreased host fecundity) lead to specialisation 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, interaction effects (i.e. increase in host vulnerability) lead to specialisation on the infected prey. Again, turning to the definition of fitness (Eq. 2), the first term is increased when interaction 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 interactions 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 interaction effects and concave trade-offs. It is replaced by a coalition of two specialists given convex trade-offs. Such a link between trade-

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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). These evolutionary results may be linked to few empirical observations. For instance, a change in the diet due to prey infection has been observed by Sato et al. (2012). In this case, a nematomorph parasite infecting a cricket species causes the suicide of its host in nearby rivers. This change in behavior makes crickets a highly profitable resource for trouts (i.e. example of a high interaction effect). In qualitative agreement with the results of our model, Sato et al. (2012) observe a reduction in the consumption of other benthic resources.

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 interaction 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

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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 key avenue to pursue. 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 diversity and could explain why "a healthy system to be one that is rich in parasite species" (Hudson et al. 2006). Also, predator diet evolution is known to create negative frequency dependence among prey species likely

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extends to more diverse systems (Kondoh 2003; Loeuille 2010; Valdovinos et al. 2010), and we show that parasitism could increase its phenomenon. Finally, we stress that the consequences of evolution vary between virulence and interaction effects. This may have important consequences for the management of ecological networks. For instance, it suggests that depending on the type of parasite occurring within an ecosystem, the effects in terms of extinction may largely differ. While precise predictions are out of the scope of the current article, we hope that our model may help to understand part of these complex dynamics.

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References

Banerji, A., A. B. Duncan, J. S. Griffin, S. Humphries, O. L. Petchey, and O. Kaltz. 2015. Density- and trait-mediated effects of a parasite and a predator in a tri-trophic food web. Journal of Animal Ecology 84:723–733.

Brassard, P., M. E. Rau, and M. A. Curtis. 1982. Infection dynamics of Diplostomum spathaceum cercariae and parasite-induced mortality of fish hosts. Parasitology 85:489–493.

Buck, J. C. 2019. Indirect Effects Explain the Role of Parasites in Ecosystems. Trends in Parasitology 35:835–847.

Buck, J. C., and W. J. Ripple. 2017. Infectious Agents Trigger Trophic Cascades. Trends in Ecology & Evolution 32:681–694.

Cézilly, F., F. Thomas, V. Médoc, and M.-J. Perrot-Minnot. 2010. Host-manipulation by parasites with complex life cycles: adaptive or not? Trends in Parasitology 26:311–317.

Charnov, E. L. 1976*a*. Optimal Foraging: Attack Strategy of a Mantid. The American Naturalist 110:141–151.

———. 1976*b*. Optimal foraging, the marginal value theorem. Theoretical population biology 9:129–136.

Cressler, C. E., D. V McLeod, C. Rozins, J. Van Den Hoogen, and T. Day. 2016. The adaptive evolution of virulence: a review of theoretical predictions and empirical tests. Parasitology 143:915–930.

Dallas, T. A., M. Krkošek, and J. M. Drake. 2018. Experimental evidence of a pathogen invasion threshold. Royal Society Open Science 5:171975.

Decaestecker, E., A. Vergote, D. Ebert, and L. De Meester. 2003. Evidence for strong host clone-

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parasite species interactions in the daphnia microparasite system. Evolution 57:784–792.

Dieckmann, U., and R. Law. 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. Journal of mathematical biology 34:579–612.

Diekmann, O. 2004. Beginner's Guide to Adaptive Dynamics. Banach Center Publications 63:47–86.

Duffy, M. A., S. R. Hall, A. J. Tessier, and M. Huebner. 2005. Selective predators and their parasitized prey: Are epidemics in zooplankton under top-down control? Limnology and Oceanography 50:412–420.

Egas, M., U. Dieckmann, and M. W. Sabelis. 2004. Evolution restricts the coexistence of specialists and generalists: the role of trade-off structure. The American naturalist 163:518–531.

Emlen, J. M. 1966. The Role of Time and Energy in Food Preference. The American Naturalist 100:611–617.

Eshel, I. 1983. Evolutionary and Continnoas Stability. Journal of Theoretical Biology 103:99–111.

Gause, G. F. 1934. The struggle for existence. The Williams & Wilkins Company.

Geritz, S. A. H., É. Kisdi, G. Meszéna, and J. A. J. Metz. 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. Evolutionary Ecology 12:35–57.

Geritz, S. A. H., J. A. J. Metz, É. Kisdi, and G. Meszéna. 1997. Dynamics of Adaptation and Evolutionary Branching. Physical Review Letters 78:2024–2027.

Goren, L., and F. Ben-Ami. 2017. To eat or not to eat infected food: a bug's dilemma. Hydrobiologia 798:25–32.

Hethcote, H. W., W. Wang, L. Han, and Z. Ma. 2004. A predator - Prey model with infected prey. Theoretical Population Biology 66:259–268.

Hilker, F. M., and K. Schmitz. 2008. Disease-induced stabilization of predator–prey oscillations. Journal of Theoretical Biology 255:299–306.

Holt, R. D. 1977. Predation, apparent competition, and the structure of prey communities. Theoretical population biology 12:197–229.

Holt, R. D., J. Grover, and D. Tilman. 1994. Simple Rules for Interspecific Dominance in Systems with Exploitative and Apparent Competition. The American Naturalist 144:741–771.

Hudson, P. J., A. P. Dobson, and K. D. Lafferty. 2006. Is a healthy ecosystem one that is rich in parasites? Trends in Ecology & Evolution 21:381–385.

Hudson, P. J., A. P. Dobson, and D. Newborn. 1992. Do Parasites make Prey Vulnerable to Predation? Red Grouse and Parasites. The Journal of Animal Ecology 61:681.

Hudson, P. J., A. P. Dobson, and D. Newborn. 1998. Prevention of Population Cycles by Parasite Removal. Science 282:2256–2258.

Hutson, V., and G. T. Vickers. 1983. A criterion for permanent coexistence of species, with an application to a two-prey one-predator system. Mathematical Biosciences 63:253–269.

Ingram, T., R. Svanbäck, N. J. B. Kraft, P. Kratina, L. Southcott, and D. Schluter. 2012. Intraguild predation drives evolutionary niche shift in threespine stickleback. Evolution 66:1819–1832.

Kondoh, M. 2003. Foraging Adaptation and the Relationship Between Food-Web Complexity and Stability. Science 299:1388–1391.

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Křivan, V., and J. Eisner. 2003. Optimal foraging and predator-prey dynamics III. Theoretical Population Biology 63:269–279.

Lafferty, K. D. 2004. Fishing for lobsters indirectly increases epidemics in sea urchins. Ecological Applications 14:1566–1573.

Lafferty, K. D., A. P. Dobson, and A. M. Kuris. 2006. Parasites dominate food web links. Proceedings of the National Academy of Sciences 103:11211–11216.

Loeuille, N. 2010. Consequences of adaptive foraging in diverse communities. Functional Ecology 24:18–27.

Lynsdale, C. L., H. S. Mumby, A. D. Hayward, K. U. Mar, and V. Lummaa. 2017. Parasite-associated mortality in a long-lived mammal: Variation with host age, sex, and reproduction. Ecology and Evolution 7:10904–10915.

MacArthur, R. H., and E. R. Pianka. 1966. On Optimal Use of a Patchy Environment. The American Naturalist 100:603–609.

McNaughton, S. J. 1992. The propagation of disturbance in savannas through food webs. Journal of Vegetation Science 3:301–314.

Metz, J. A. J., R. M. Nisbet, and S. A. H. Geritz. 1992. How should we define "fitness" for general ecological scenarios? Trends in ecology & evolution 7:198–202.

Packer, C., R. D. Holt, P. J. Hudson, K. D. Lafferty, and A. P. Dobson. 2003. Keeping the herds healthy and alert: implications of predator control for infectious disease. Ecology Letters 6:797–802.

Prosnier, L., V. Médoc, and N. Loeuille. 2018. Parasitism effects on coexistence and stability within simple trophic modules. Journal of Theoretical Biology 458:68–77.

Pyke, G. H., H. R. Pulliam, and E. L. Charnov. 1977. Optimal Foraging: A Selective Review of Theory and Tests. The Quarterly Review of Biology.

Raffel, T. R., L. B. Martin, and J. R. Rohr. 2008. Parasites as predators: unifying natural enemy ecology. Trends in Ecology & Evolution 23:610–618.

Rueffler, C., T. J. M. Van Dooren, and J. A. J. Metz. 2006. The Evolution of Resource Specialization through Frequency-Dependent and Frequency-Independent Mechanisms. The American Naturalist 167:81–93.

Sato, T., T. Egusa, K. Fukushima, T. Oda, N. Ohte, N. Tokuchi, K. Watanabe, et al. 2012. Nematomorph parasites indirectly alter the food web and ecosystem function of streams through behavioural manipulation of their cricket hosts. (D. Post, ed.)Ecology Letters 15:786–793.

Sieber, M., and F. M. Hilker. 2011. Prey, predators, parasites: intraguild predation or simpler community modules in disguise? Journal of Animal Ecology 80:414–421.

Stewart, T. E., M. E. Torchin, and C. E. Cáceres. 2018. Invisible Parasites and Their Implications for Coexisting Water Fleas. Journal of Parasitology 104:101–105.

Valdovinos, F. S., R. Ramos-Jiliberto, L. Garay-Narváez, P. Urbani, and J. A. Dunne. 2010. Consequences of adaptive behaviour for the structure and dynamics of food webs. Ecology Letters 13:1546–1559.

van Velzen, E., and R. S. Etienne. 2013. The evolution and coexistence of generalist and specialist herbivores under between-plant competition. Theoretical Ecology 6:87–98.

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Appendix

A) Diet evolution due to parasitism: interaction effect

When only interaction effects occur (Fig. A1), a higher interaction 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

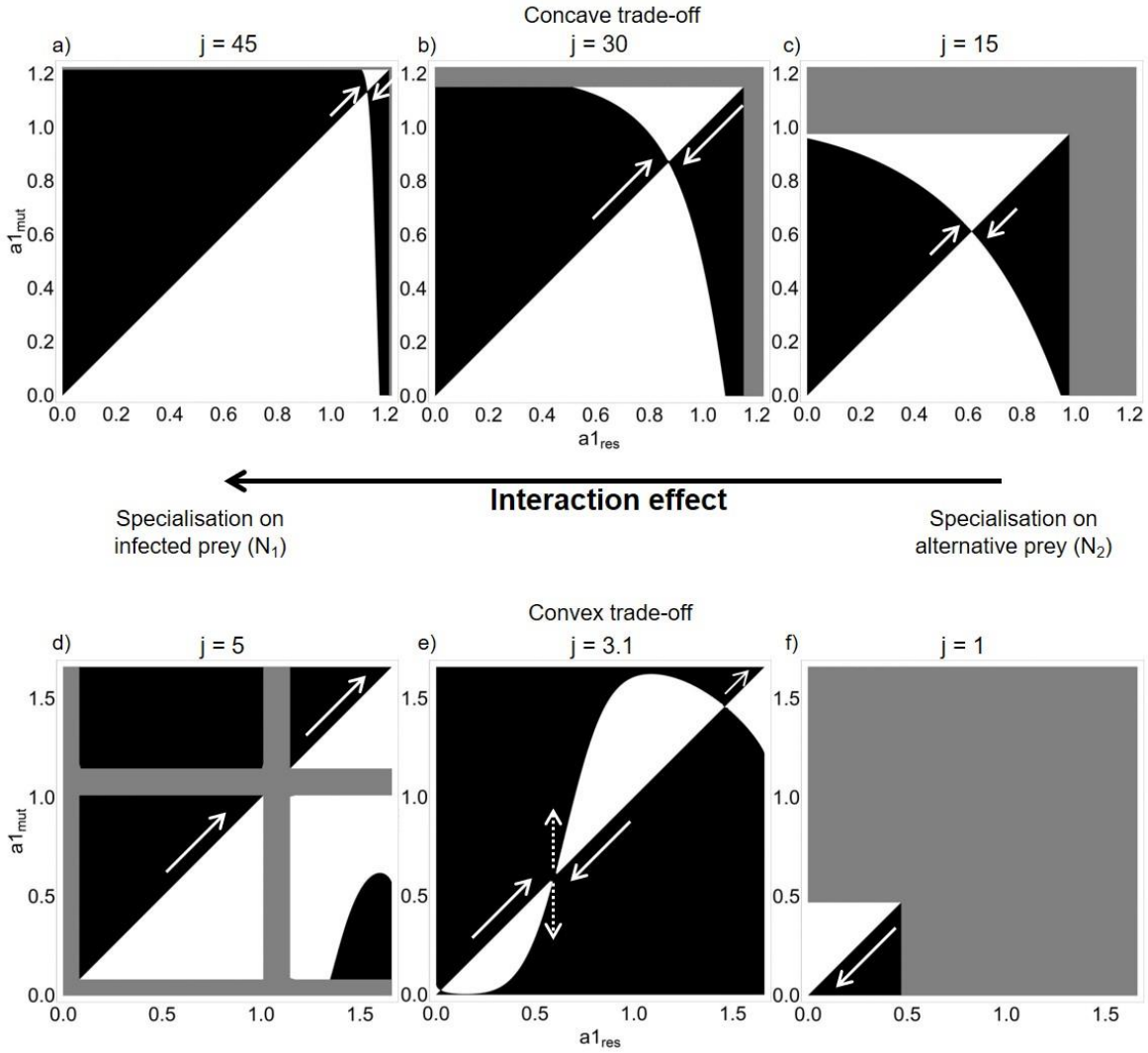

Figure A1. Pairwise Invasibility Plots of evolution of predator diet (i.e. evolution of a_1) for a concave (a-c) and a convex (d-f) trade-offs, when increasing intensity of interaction effect. On PIP, black area corresponds to a positive relative fitness of mutants, white area corresponds to a negative 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.

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(concave trade-offs, A1b), or to diversification (convex trade-offs, A1e). To go further and assuming concave trade-offs, increasing the interaction 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.

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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),

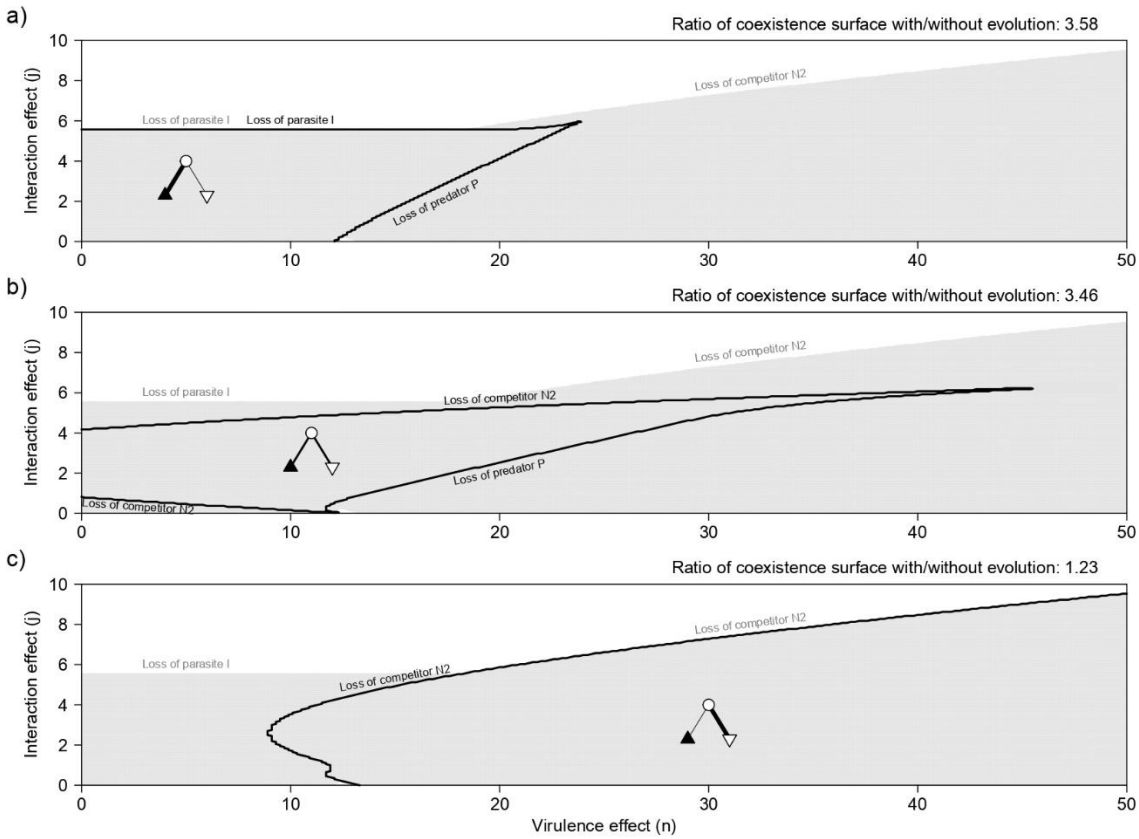

Figure B1. Coexistence of the three species, with concave trade-off, with evolution (grey area) and without evolution (inside the black delimitation). When the predator is a) specialist on the infected prey N_1, b) generalist, c) specialist on the alternative prey N_2. The species loss at the limits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

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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.