

Predation shifts coevolution toward higher host contact rate and parasite virulence

Authors: Jason C. Walsman^{*a} and Clayton E. Cressler^b.

^{*}Corresponding author. Email: walsmanjason@gmail.com

^aDepartment of Biological Sciences, University of Pittsburgh; Pittsburgh, PA USA.

^bSchool of Biological Sciences, University of Nebraska; Lincoln, NE USA.

Abstract:

Hosts can avoid parasites (and pathogens) by reducing social contact, but such isolation may carry costs, e.g., increased vulnerability to predators. Thus, many predator-host-parasite systems confront hosts with a trade-off between predation and parasitism. Parasites, meanwhile, evolve higher virulence in response to increased host sociality and consequently, increased multiple infections. How does predation shift coevolution of host behaviour and parasite virulence? What if predators are selective, i.e., predators disproportionately capture the sickest hosts? We answer these questions with an eco-coevolutionary model parametrized for a Trinidadian guppy-*Gyrodactylus spp.* system. Here, increased predation drives host coevolution of higher grouping, which selects for higher virulence. Additionally, higher predator selectivity drives contact rate higher and virulence lower. Finally, we show how predation and selectivity can have very different impacts on host density and prevalence depending on whether hosts or parasites evolve, or both. For example, higher predator selectivity led to lower prevalence with no evolution or only parasite evolution but higher prevalence with host evolution or coevolution. These findings inform our understanding of diverse systems in which host behavioural responses to predation may lead to increased prevalence and virulence of parasites.

Keywords: host-parasite coevolution, predator-prey, social behaviour, parasite avoidance, eco-evolutionary modelling, group living

1. Background

Predation is often expected to reduce the incidence, prevalence, or transmission of infectious diseases of prey, “keeping the herds healthy and alert” [1, 2] by reducing host density and thus the spread of density-dependent diseases. However, there are certain conditions under which predation can increase, not decrease, disease in its prey [3-5]; for example if predators remove enough recovered individuals, increasing the supply of susceptible individuals in a

density-limited host population, then predators can increase infection prevalence [4]. The effect of predators on disease, however, is expected to be much more negative if predators disproportionately capture infected hosts more than uninfected hosts (“selective predation”) [4, 6-11]. Selective predation occurs across predator-prey/host-parasite systems including host species such as red grouse [9], mud crabs [12], snowshoe hares [13], salmon [14], and zooplankton [15].

But while predation has strong consumptive effects on prey populations, the non-consumptive effects of predation may be just as strong [16]. Non-consumptive effects occur when prey traits change due to the threat of predation, through evolution or plasticity, thus altering how prey interact with their biotic and abiotic environments. Predation drives many non-consumptive effects relevant to parasites [17, 18], e.g., when growing larger to defend against predation makes hosts more vulnerable to infection [5]; recent theory has begun describing how the consumptive and non-consumptive effects of predation, acting simultaneously, will affect parasite ecology [19, 20] and, more recently, evolution [21]. One notable, non-consumptive effect of predation, prey grouping, should influence diverse infectious diseases across animal taxa. Many animal taxa, invertebrates [22] and vertebrates [23] alike, group for defence against predators. For prey infected with parasites that rely on contact or proximity for transmission, grouping may strongly influence parasite transmission and, ultimately, evolution [24, 25]. In such cases, increased predation effectively increases the cost of parasite avoidance. Here we use a mathematical model to show that this ubiquitous, non-consumptive effect can have large effects on disease outcomes which shift with selective predation.

Further, parasitic organisms are renowned for adaptation in response to such selective pressures [26]. For example, host mortality unrelated to infection can lead to the evolution of

higher virulence [26, 27]. However, theory shows that this result depends strongly on whether mortality interacts with virulence; e.g., if selective predation is especially effective against more virulent parasites, predation is more likely to select for lower virulence [28]. Even nonselective predation can drive the evolution of lower virulence if it reduces the likelihood of multiple infections, as multiple infections often favour higher virulence [27]. However, defensive grouping in response to predation could increase multiple infections and virulence [21, 29]. Thus, predation and predator selectivity may strongly shift host grouping and parasite virulence, with implications for prevalence and host density.

We tease apart these interactions with biologically-relevant parameter values from the well-characterized predator-prey/host-parasite system of generalist piscivores, Trinidadian guppy prey/hosts (*Poecilia reticulata*) and guppies' helminth parasites (*Gyrodactylus* sp.). The focal system, and many other host-parasite systems across taxa, meet multiple model assumptions: hosts trade-off predator defence against parasite transmission, parasites have a trade-off that selects for intermediate virulence [21, 30], parasites compete in multiple infections which lead to selection for increased virulence [21, 31], and selective predation likely raises the effective cost of virulence [15]. In our focal system, infection intensity (likely correlated with the rate of parasite reproduction on the host), is linked with transmission rate and virulence [21] and possibly also with selective predation [32]. With these assumptions, the model helps explain how strong predation can lead to high parasite prevalence and virulence, as observed in high predation guppy populations [21], even if predation is very selective. However, these assumptions are likely to be met in many predator-prey/host-parasite systems, suggesting that our model insights may apply very broadly, contributing to a richer understanding of how

coevolution in even modestly ecologically complex communities can generate novel evolutionary outcomes.

2. Methods

(a) Model ecology

We model the densities of susceptible (S), infected (I) and recovered (R) hosts in the presence of predation. Hosts grow logistically with maximum reproductive rate (a) and a sensitivity to crowding (q); hosts suffer mortality from background sources (d) and from predation proportional to the predation level (P) and inversely proportional to their contact rate with other hosts, a measure of grouping (c). We chose P/c because it is the simplest function that captures our intuition that per-capita predation rates should decrease nonlinearly with c ; note that this function does not consider an interaction between host density and the efficacy of host grouping for defence. Infection transmits from infected hosts to susceptible hosts dependent on c and parasite transmissibility (T), which captures all aspects of transmission rate other than c . Infected hosts suffer additional death from parasite virulence (v) and may also experience a higher predation rate depending on the harmfulness of the infection (ϕv); ϕv quantifies the degree of extra preference of the predator for infected hosts ($\phi = 0$ for no preference). Infected hosts can recover (at rate y ; $y = 0$ for the case of no recovery). Recovered hosts suffer mortality and waning immunity (at rate z ; $z = 0$ for the case of permanent immunity):

$$\frac{dS}{dt} = (a - qH)H - dS - \frac{P}{c}S - cTIS + zR \quad (1a)$$

$$\frac{dI}{dt} = cTIS - dI - \frac{P}{c}(1 + \phi v)I - vI - yI \quad (1b)$$

$$\frac{dR}{dt} = yI - dR - \frac{P}{c}R - zR \quad (1c)$$

Here, predation is constant (see Fig. S1 for analysis with a dynamic predator, which gives many similar outcomes but see Fig. S1n for a difference); this assumption is reasonable in many systems where predators are generalists whose dynamics are not tied to those of any particular prey [33] or if predator generation times are much longer than those of prey. We focus on outcomes at the model's single endemic equilibrium, which is stable if the parasite $R_0 > 1$ (for our parameters). We use a biologically relevant parameter range for the guppy-*Gyrodactylus* system [21] and focus on model behaviour in this range. The uncharacterized parameter in the guppy-*Gyrodactylus* system is the selectivity of predation (value of ϕ); thus, we explore a broad range of ϕ from 0-100 so that, at the highest values of ν in our focal parameter range, infected hosts are predated upon ~ 2.7 times more than susceptible hosts. This range is likely relevant to many systems where similarly large or even larger factors have been found [9, 12, 13, 15].

(b) Host evolution of contact rate

We model evolution and coevolution with Adaptive Dynamics [34], which models evolution as a sequence of invasion events by “mutant” genotypes invading a monomorphic population of “residents.” For hosts, we model evolution of contact rate, assuming that each host genotype has a genetically fixed contact rate. An invading host genotype with contact rate c_m gains protection from predation but risks transmission of infection that depends on the geometric mean of its contact rate and that of the resident [c_r , so $\sqrt{c_m c_r}$ in equation (2)]; the choice of geometric mean allows the contact rate traits of both the individual invader and the resident population to influence the invader's overall contact rate. Either can avoid sociality by having

zero contact rate [similar to Bonds et al. 2005; 35]. Equations (2a-c) give the dynamics of a mutant host (S_m, I_m, R_m) invading a resident population at equilibrium (H_r^*, I_r^*):

$$\frac{dS_m}{dt} = [a - qH_r^*]H_m - dS_m - \frac{P}{\sqrt{c_m c_r}} S_m - \sqrt{c_m c_r} T I_r^* S_m + zR_m \quad (2a)$$

$$\frac{dI_m}{dt} = \sqrt{c_m c_r} T I_r^* S_m - dI_m - \frac{P}{\sqrt{c_m c_r}} (1 + \phi v) I_m - vI_m - \gamma I_m \quad (2b)$$

$$\frac{dR_m}{dt} = \gamma I_m - dR_m - \frac{P}{\sqrt{c_m c_r}} R_m - zR_m \quad (2c)$$

From equations (2a-c), we can derive an expression for the invasion fitness of a mutant host [using $dH_m/dt = dS_m/dt + dI_m/dt + dR_m/dt$; equation (3) derived from equation (2)]:

$$\frac{1}{H_m} \frac{dH_m}{dt} = a - qH_r^* - d - \frac{P}{\sqrt{c_r c_m}} - \frac{P}{\sqrt{c_r c_m}} \phi v p_m - v p_m \quad (3)$$

Host evolution depends on balancing mortality from predators against mortality from parasites as host genotypes do not differ in fecundity. A mutant host (H_m) has fecundity and suffers background mortality (d), mortality from non-selective predation ($P/\sqrt{c_r c_m}$), additional mortality from selective predation ($P/\sqrt{c_r c_m} \phi v p_m$) that depends on the infection prevalence suffered by the mutant (p_m), and mortality from parasitism ($v p_m$). In practice, it is difficult to calculate prevalence for the mutant (p_m) but the proportion of time spent infected serves as a simpler proxy [as done by 36]; this calculation of fitness finds the same invasion success/failure as a Next Generation Matrix approach [as used by 37; see supplementary code for proof]. From equation (3), we can derive an expression for the selection gradient of contact rate, G_c , showing how this depends on mortality from the three sources identified above:

$$G_c = \frac{\partial}{\partial c_m} \frac{1}{H_m} \frac{dH_m}{dt} \Big|_{c_m = c_r} \quad (4a)$$

$$G_c = \underbrace{\frac{P}{2c_r^2}}_{1. \text{ Mortality from predation}} \underbrace{-\frac{P}{c_r} \phi v \frac{\partial p_m}{\partial c_m} + \frac{P}{2c_r^2} \phi v p_m}_{2. \text{ Mortality from selective predation}} \underbrace{-v \frac{\partial p_m}{\partial c_m}}_{3. \text{ Mortality from parasitism}} \quad (4b)$$

Term 1 represents how predation increases the selection gradient, driving higher contact rate.

Term 2 is more complex as it contains a negative term (driving contact rate down) that represents additional mortality from selective predation if hosts become infected; this negative is

counteracted by a positive term (driving contact rate up), representing the benefit of contact rate for protecting infected hosts from selective predation. Term 3 represents how mortality from

parasitism decreases the selection gradient, driving lower contact rate. We find the value of

contact rate that is a Continuously Stable Strategy (c_{css}), namely, a singular point (a value of c_m where $G_c = 0$) which is convergence stable and evolutionarily stable; these criteria mean that, in

the neighbourhood of the singular point, strategies closer to it can invade those further from it

while no strategy in that neighbourhood can invade the singular point.

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151 (c) Parasite evolution of virulence

We model parasite fitness dependent on a link between the virulence of each parasite

genotype (v_i) and its transmissibility (T_i) with a concave relationship between virulence and

transmissibility that is known to promote evolutionary stability [$k_2 < 1$, see equation (5a); 38].

Parasite genotypes with higher v_i suffer from a shorter infectious period but benefit from higher

T_i . In the Adaptive Dynamics analysis, we calculate the invasion fitness of a parasite genotype

invading a resident population at equilibrium (S_r^*, I_r^*) [equation (5b), simplified by cancellation of

I_m from all terms]. The invader can infect both susceptible hosts and hosts infected by the

resident genotype i.e., we assume superinfection at a rate that depends on a superinfection

parameter [σ , equation (3b); following the form used by 27]; at the same time, the resident

parasite can infect hosts infected by the mutant parasite. We assume the superinfection parameter does not depend on parasite virulence because this dependence may be negative or positive in various systems [31] and has not been characterized in our focal system; note that such a dependence could alter some results, such as leading to evolutionary branching in parasite virulence [27]. We find the parasite's CSS (v_{CSS}) from the selection gradient of virulence (equation 5c), separated into terms quantifying 1. the benefit of transmitting to susceptible hosts; 2. the benefit of transmitting to infected hosts (possible because of superinfection); 3. the cost of virulence in terms of additional host death from selective predation and virulence.

$$T_i = k_1 v_i^{k_2} \quad (5a)$$

$$\frac{1}{I_m} \frac{dI_m}{dt} = cT_m S_r^* - d - \frac{P}{c} (1 + \phi v_m) - v_m - \gamma + \sigma c T_m I_r^* - \sigma c T_r I_r^* \quad (5b)$$

$$G_v = \frac{\partial}{\partial v_m} \frac{1}{I_m} \frac{dI_m}{dt} \Big|_{v_m = v_r}$$

$$= \underbrace{c S_r^* \frac{dT_m}{dv_m}}_{1. \text{ Transmission to } S} + \underbrace{\sigma c I_r^* \frac{dT_m}{dv_m}}_{2. \text{ Transmission to } I} + \underbrace{\left(-\phi \frac{P}{c} - 1 \right)}_{3. \text{ Cost of virulence}} \quad (5c)$$

(d) Coevolutionary outcomes

Host contact rate evolution depends on parasite virulence and predation [$c_{\text{CSS}}(v, P)$] and vice versa [$v_{\text{CSS}}(c, P)$]. These feedbacks can lead to a point that is a coevolutionary, stable attractor [$c_{\text{coCSS}}(P), v_{\text{coCSS}}(P)$]. For a pair of trait values (c, v) to be a coCSS, c must be an uninvadable singular point for the case of host-only evolution (likewise v for the parasite) and the joint dynamics must be stable (the coevolutionary version of convergence stability; see electronic supplementary material for details). Coevolutionary convergence stability may depend on assumptions regarding the speed of host and parasite evolution [39]; we make no such

assumptions but in our focal parameter range, singular point intersections are coCSSes for any positive rates of evolution (see supplementary material for cases where outcomes will depend on the speed of evolution). In addition to model outcomes for traits (c and v), we also focus on two key population outcomes: host density (H^*) and prevalence ($p^* = I^*/H^*$); these population outcomes depend on predation, contact rate, and virulence.

Table 1. Values and units of state variables and parameters. As discussed in Methods, all parameter values are sourced from Walsman et. al 2022 [21] except for ϕ and we use a lower range of predation (but $\phi > 0$ compensates somewhat by making overall predation higher and see the supplementary material for results with higher predation). State variables then parameters listed, each in order of appearance in equation (1).

Symbol	Meaning	Value and/or units
S	Susceptible host density	hosts·m ⁻²
I	Infected host density	hosts·m ⁻²
R	Recovered host density	hosts·m ⁻²
a	Maximum host birth rate	0.106 day ⁻¹
q	Host sensitivity to crowding	0.017 m ² ·host ⁻¹ ·day ⁻¹
d	Background host death rate	1.30 x 10 ⁻³ day ⁻¹
P	Predation	0-0.005 day ⁻¹
c	Host-host contact rate	m ² ·host ⁻¹ day ⁻¹
T	Transmissibility of infection	Unitless
z	Rate of immunity loss	0.033 day ⁻¹

ϕ	Selectivity of predation	0-100 day
ν	Virulence of infection	day ⁻¹
γ	Recovery rate	0.020 day ⁻¹
k_1		0.0199
k_2		0.277
σ	Superinfection parameter	1.21

3. Results

(a) Host evolution of contact rate

Our method allows clear interpretation of host evolution. From equation (4b), predation on all hosts drives higher contact rate (pathway 1 can only be positive). Selective predation is more complex (pathway 2 can be positive or negative). Parasitism, meanwhile, can only select for decreased contact rate (pathway 3 can only be negative).

Importantly, selection favours host genotypes that minimize mortality at the individual level (evolutionary stability condition for a CSS) not the average mortality in the population. As a result, hosts evolve higher contact rate than necessary for parasites to spread (c_{CSS} higher than $c_{boundary}$ in Fig. 1a) and the parasites become endemic (Fig. 1b). As contact rate and prevalence increase with predation, host density decreases (Fig. 1c; here, $c_{boundary}$ is the contact rate that maximizes H^* at a given predation level). When predation is selective, results are qualitatively similar in that hosts evolve higher contact rate that leads to higher prevalence and lower host density but with some different evolutionary dynamics (see Fig. S2).

Link text here for Figure 1.

(b) Coevolutionary outcomes

(i) Summary of coevolutionary outcomes

First, (i) we summarize coevolutionary outcomes then later we explain (ii) how those outcomes arise for traits and (iii) host density and prevalence. We contextualize coevolutionary outcomes in contrast to outcomes from no evolution, only host evolution, or only parasite evolution. Any non-evolving trait is held fixed at its average value from the coevolutionary case (averaged across predation and selectivity ranges). Without evolution (Fig. 2a-d), increasing predation depresses host density and prevalence, especially depressing prevalence when predation is more selective (blue curve lower than orange in Fig. 2d). With host evolution (Fig. 2e-h), hosts evolve increasing contact rate in response to increasing predation. Increasing contact rate increases prevalence and can eventually lead to ‘resistance is futile’ [40] (i.e., behavioural resistance through decreased contact rate) and hosts evolve maximum contact rate to minimize predation risk (blue curve in Fig. 2e; see Fig. S2 for more detail). In the main text, we limit this runaway evolution to some maximum contact rate to maintain biological realism (we chose $c_{\max} = 5$). With only parasite evolution (Fig. 2i-l), parasites evolve mostly flat virulence with predation or strongly decreasing virulence if predation is selective; virulence is mostly flat with non-selective predation because predation increases mortality but also decreases infected host density and the force of superinfection (see Table S2). Host density declines somewhat with predation but less so when it is more selective. Prevalence decreases with predation, but parasite evolution makes prevalence less responsive to selectivity.

With coevolution, predation selects for increasing contact rate, especially with higher selectivity. Meanwhile, parasites evolve increasing virulence but less so with increasing selectivity. Host density declines, somewhat more with more selective predation. Prevalence

increases with predation and more so for higher selectivity. Because coevolutionary prevalence is higher at higher selectivity, death from parasitism may actually be slightly higher with higher selectivity (unlike the no evolution case, see Fig. S3). At high selectivity and predation (higher P than shown here), there is no stable coCSS (see Fig. S4).

Link text here for Figure 2.

(ii) Explanation of coevolutionary trait outcomes

To clarify how host contact rate coevolves along a predation gradient, we must disentangle how predation directly shifts contact rate and how it indirectly shifts contact rate by shifting virulence. We find how predation directly and indirectly shifts host evolution through the three pathways mentioned above. As predation and virulence shift contact rate, all three impact the host selection gradient (G_c) but the selection gradient must remain zero at any evolutionary singular point (of which CSSes and coCSSes are a subset):

$$G_c(c_{\text{coCSS}}, v_{\text{coCSS}}, P) = 0 \quad (6)$$

Equation (6) allows us to implicitly differentiate with respect to P [equation (7a)] and rearrange [equation (7b)] to disentangle how c_{coCSS} changes into direct and indirect effects of predation:

$$0 = \frac{dG_c}{dP} = \frac{\partial G_c}{\partial c_{\text{coCSS}}} \frac{dc_{\text{coCSS}}}{dP} + \frac{\partial G_c}{\partial v_{\text{coCSS}}} \frac{dv_{\text{coCSS}}}{dP} + \frac{\partial G_c}{\partial P} \quad (7a)$$

$$\frac{dc_{\text{coCSS}}}{dP} = \frac{1}{-\frac{\partial G_c}{\partial c_{\text{coCSS}}}} \left(\underbrace{\frac{\partial G_c}{\partial P}}_{\text{Direct}} + \underbrace{\frac{\partial G_c}{\partial v_{\text{coCSS}}} \frac{dv_{\text{coCSS}}}{dP}}_{\text{Indirect}} \right) \quad (7b)$$

Note that $\partial G_c / \partial c_{\text{coCSS}}$ is negative at any CSS (convergence stability). Equation (7b) indicates that the direct and indirect effects of predation on contact rate evolution will be proportional to the effects on the selection gradient ($\partial G_c / \partial P$ and $\partial G_c / \partial v_{\text{coCSS}}$). These effects can be disentangled, in

turn, into how predation or virulence shift predation on all hosts (pathway 1 in 4b), selective predation (pathway 2), or mortality from parasitism (pathway 3).

This analysis shows that predation directly selects for contact rate while evolution of higher virulence somewhat resists this pattern, with our parameter values (middle of P range, coevolutionary trait values at each ϕ). At all selectivity levels, the overwhelming effect of predation on contact rate evolution is a positive, direct one through pathway 1 (see Table S1). The second-most important effect of predation is a negative, indirect one through coevolution of higher virulence, which leads to a benefit of lower contact rate for avoiding mortality from parasitism (pathway 3). This approach can also be useful to disentangle how selectivity affects contact rate evolution (taking derivatives of G_c with respect to ϕ instead of P). In short, increased ϕ increases the importance of high c to protect infected hosts compared to the importance of low c to avoid infection (for our parameter values).

This same approach can be applied to virulence evolution and its relevant three pathways, for which more pathways have an appreciable effect. For our parameters, the direct effects of predation through these three pathways yields a net negative effect on virulence evolution if predation is selective (see Table S2). Increased predation directly increases the death rate of infected hosts and thus the equilibrium density of susceptible hosts, $S_r^* = [d+P/c (1+\phi v_r)+v_r+\gamma]/(cT_r)$. Increased S_r^* in turn increases the benefit of transmission to susceptible hosts ($cS_r^* dT_m/dv_m$; P directly increases pathway 1). Increased predation decreases the density of infected hosts I_r^* , thus decreasing the benefit of transmission to infected hosts (pathway 2). Further, predation always increases the cost of virulence when it is selective ($\phi > 0$; pathway 3). Pathway 3 gets much stronger as selectivity ϕ increases so that the total, direct effect of predation changes with increasing ϕ from weak selection for higher virulence to strong selection for lower

virulence. We label these direct effects as consumptive as they come from consuming hosts while the non-consumptive effects arise by increasing coevolutionary host contact rate.

Increased contact rate decreases the death rate of infected hosts and thus has a small, negative effect on virulence through pathway 1 (note that c coefficient cancels c in denominator of S^*_I). Increased contact rate strongly increases the benefit of transmission to infected hosts (pathway 2) while decreasing the cost of virulence (pathway 3), selecting for virulence. As selectivity ϕ increases, the magnitudes of all the effects of contact rate increase. Across the selectivity range, the overall non-consumptive effect of predation is mostly to select for higher virulence through pathway 2 (transmission to I because of multiple infections).

So far, we have explained how contact rate and virulence feedback on each other but not how the mutual feedbacks lead to a coCSS. We do so by plotting c_{CSS} across a range of fixed v values, and v_{CSS} across a range of fixed c values (Fig. 3). This shows how predation moves both host and parasite CSS curves, thereby altering the intersection of the curves (the coCSS). Non-selective predation leads to the coevolution of higher contact rate and virulence mostly through the effect of predation on contact rate and the effect of contact rate on virulence (Fig. 3a; see Table S2). When predation is very selective, predation directly increases contact rate even more (see Figs. 2e, 3b). Further, increased predation has a large, negative, direct, consumptive effect on virulence evolution (pathway 3 in Table S2; Fig. 3b), pushing virulence lower. Lower virulence then leads to an even further increased contact rate (indirect effect of predation), which in turn partially ameliorates the decrease in virulence (indirect effect of predation). The sum of these direct and indirect effects make v_{coCSS} slightly lower and c_{coCSS} higher when ϕ is larger.

Link text here for Figure 3.

(iii) Explanation of coevolutionary population outcomes

We develop more mathematical insight into the effect of predation on population outcomes by partitioning its effect into three pathways: the ecological effects, the effect through contact rate evolution, and the effect through virulence evolution. For example, the net effect of predation on host density (dH^*/dP) is:

$$\underbrace{\frac{dc}{dP} \frac{\partial H^*}{\partial c}}_{\text{Effect through } c} + \underbrace{\frac{dv}{dP} \frac{\partial H^*}{\partial v}}_{\text{Effect through } v} + \underbrace{\frac{\partial H^*}{\partial P}}_{\text{Ecological effect}} = \frac{dH^*}{dP} \quad (8)$$

Conceptually, the first two terms in equation (8) are readily characterized as the eco-coevolutionary, indirect effects of predation through contact rate (c) or virulence (v) evolution (trait responses in Fig. 4a and trait impacts in Fig. 4b). The third term is the direct, ecological effect of predation on host density. Together, these give the net effect of predation on eco-coevolutionary host density. Some of these results are more sensitive to parameter values than others, e.g., increasing selectivity can make the ecological effect of predation on host density more or less negative; we report values at the middle of our predation range ($P = 0.0025$) and the corresponding coevolutionary traits for each selectivity level (see Fig. 2m, n).

Predation increases coevolutionary contact rate and virulence. At higher selectivity, predation has a larger positive effect on contact rate and smaller positive effect on virulence (Fig. 4a; see also Fig. 2m, n, Fig. 3). Increasing contact rate decreases host density (see Fig. 1) and slightly more so when predators are more selective. Increasing virulence decreases host density, especially when virulence leads to harsher predation on infected hosts (higher selectivity). The impact of each trait on host density (Fig. 4b) multiplied by the response of that trait to predation (Fig. 4a) gives the effect of predation on host density through that trait [equation (8)]. Ecologically, increasing predation decreases host density (Fig. 2c) but selective predation can somewhat ameliorate this decrease. The effects of predation via contact rate and virulence

evolution exacerbate this trend and reverse the impact of selectivity; increasing predation strongly decreases host density and most when predation is more selective due to the larger increase in contact rate. Therefore, coevolution exacerbates how much selective predation reduces host density.

The effects of predation and selectivity on prevalence (p^*) are more mixed and differ strongly with coevolution (see Figs. 4d, e). Without evolution, predation decreases prevalence and most strongly when predation is most selective (Fig. 4e). With coevolution, predation and selectivity increase contact rate, leading to the biggest net increase in prevalence for the case of strong, selective predation (Fig. 4e). These effects on prevalence and host density arise because selection on individual hosts drives high contact rate to avoid predation (Fig. 1), spreading parasites and selecting for increased lethality in those parasites.

Link text here for Figure 4.

The effects of predation differ from the effects of simple increases in host background death rate, d , because d does not have a strong interaction with contact rate. Host mortality only indirectly affects selection on contact rate by ecologically decreasing the prevalence of infection. Thus, contact rate only increases weakly with increasing d , only weakening the ecological trend of higher d leading to lower prevalence (for host evolution or coevolution; see Fig. S5). This contrasts with predation which strongly increases contact rate, driving a net increase in prevalence (for host evolution or coevolution; see Fig. 2).

4. Discussion

The effects of selective predation on parasite prevalence and virulence follow standard expectations for microparasite models without predator defence/contact rate evolution; with such

evolution, newer results emerge. First, consider predation as a source of non-parasite induced mortality and how it impacts prevalence and virulence. Increased host mortality generally leads to declining prevalence and thus decreasing investment in the resistance that prevents infection [41]; we get equivalent results when increasing d (Fig. S5), which does not interact directly with contact rate. Predictably [21], we get very different results when the mortality source (predation) selects for higher contact rate, leading to increasing contact rate and increasing prevalence with predation (Figs. 1, 2). Increased host mortality is also expected to lead to increased virulence if multiple infections are unimportant or decreased virulence if they are important [27]; we find this result without contact rate evolution (see orange curve in Fig. 2j) but with contact rate evolution, increasing contact rate drives much stronger selection for virulence (compare Fig. 2j, n; see also Table S2). Second, consider how selectivity affects prevalence and virulence. More selective predation is expected to decrease prevalence more [4, 6-11], which we find without contact rate evolution (Fig. 2d). With contact rate evolution, higher selectivity can actually lead to higher prevalence (Fig. 2h, p). For virulence, more selective predation (dependent on virulence) is expected to select for decreased virulence [28] but we find that contact rate evolution greatly weakens this effect (compare Fig. 2j to n). In all of these cases, prevalence and virulence are greatly amplified by the fact that selection on individual host contact rate leads to “selfish” evolution of high contact rate that spreads disease.

Our coevolutionary model outcomes hinge strongly on such host evolution. Selection on individual contact rate unsurprisingly leads to “selfish” evolution of high contact rate that is harmful to the host population, with high prevalence and lowered host density. This arises because selection on individual fitness (evolutionary stability condition of a CSS) depends on the direct infection costs and predation benefits of higher contact rate for the individual; this

individual selection does not account for how increasing contact rate allows parasites to become more abundant, harming the population as a whole. Selection for high contact rate that harms the host population in terms of prevalence and total density may be a common feature when the benefits of sociality (here avoiding predation from a static predator) are more static than the cost in terms of a dynamic parasite population (see Fig. S6 for the case of a dynamic predator, in which hosts evolve a *lower* contact rate than would maximize host density). However, our analysis does not consider host relatedness, and we might expect that inclusion of relatedness will lead to kin selection on contact rate that could drive the evolution of lower contact rate. Such group selection likely helps explain the existence of strong “social distancing” in populations of ants [42] or honeybees [43], where kin selection is extremely strong.

Our modelling results highlight other, key assumptions and predictions for empirical testing. One key assumption to test would be the interaction of virulence and predator selectivity. Predator selectivity seems likely to interact with virulence as hosts with more virulent infections will be less able to avoid predators. Predator selectivity has often been found to increase with the number of parasites per host [9, 13-15] with suggestive evidence in our focal system [32]; further, infection intensity is linked to the virulence of parasites that reproduce within hosts such as *Gyrodactylus* spp. worm [21] and others [44, 45]. These data point to a potentially critical interaction of virulence and selective predation worth empirical testing.

In terms of predictions, one key test would be the pattern of infection prevalence along a gradient of selective predation. If predation strongly selects for lower, infection-preventing resistance (here higher contact rate), we predict that even selective predation can increase prevalence (and virulence for systems with multiple infections). But if predation does not interact with anti-infection defence, increased selective predation should decrease prevalence and

virulence as previously expected. Higher predation guppy populations suffer higher prevalence of worm ectoparasite infection and have more virulent parasites [21], as our model predicts, but the selectivity of predation in this focal system has been indirectly supported by one study [32] and challenged by another [46].

It will also be interesting to study how these results might interact with other forms of host defence against parasitism. For example, guppy [47] and mandrill hosts [48] exhibit inducible, avoidance behaviour when conspecifics are obviously infected. Inducible avoidance behaviours like this may follow different evolutionary patterns than constitutively lower contact rate, as we model, as immunological constitutive and inducible defences sometimes follow similar and sometimes different patterns [49]. Moreover, if susceptible hosts can effectively shun infected hosts, this exclusion may be a mechanism for producing predator selectivity (e.g., infected hosts end up with a lower effective c value due to behavioural avoidance from uninfected hosts). If avoidance increases with virulence, this phenomenon may break the trade-off that drives virulence evolution in this system by reducing the fitness benefits of virulence; this behaviour may also alter the trade-offs driving the evolution of shoaling behaviour itself. Further, behavioural defences likely coevolve simultaneously with physiological defences; indeed, more physiologically susceptible guppy [50] or finch hosts [51] seem to rely on stronger avoidance behaviour. While inducible avoidance of infected individuals may provide different results, this phenomenon may be adequately captured by constitutively lower contact rates and selective predation for certain model outputs; thus, future exploration could determine when inducible avoidance yields different results than our constitutive trait approach. Both considerations of constitutive/inducible defence and physiological/behavioural defence will likely prove fruitful areas for further exploration of behavioural defences against infection.

Overall, these results demonstrate the importance of non-consumptive effects and parasite adaptation for understanding the effects of predators, especially selective ones, on parasite dynamics. Previous intuition regarding “healthy herds” holds well for the ecological effects of predation and predator selectivity but poorly when host contact rate and parasite virulence coevolve. Managers relying on selective predators to keep the herds healthy could be unpleasantly surprised if selective predation bunches the herd together, accelerating the spread of increasingly lethal parasites. Here, we provide intuition for how selective predation may lead to surprising results through host-parasite coevolution: predation drives increased contact rate as hosts group for defence; increased contact rate increases multiple infections, selecting for higher virulence. The resulting increased prevalence and virulence of parasites drives host density down. More selective predation directly decreases virulence but leads to a further increase in contact rate, partially restoring virulence and increasing the prevalence of infection. These core mechanisms in our model may prove relevant for eco-evolutionary host density and prevalence outcomes in diverse animal hosts.

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Data accessibility

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