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RESEARCH ARTICLE

Parasite-driven cascades or hydra effects: Susceptibility and foraging depression shape parasite–host–resource interactions

Jason Cosens Walsman[1] | Alexander Thomas Strauss[2] | Spencer Ryan Hall[3]

[1]Indiana University and University of Pittsburgh, Pittsburgh, PA, USA

[2]Indiana University and University of Georgia, Athens, GA, USA

[3]Indiana University, Bloomington, IN, USA

Correspondence
Jason Cosens Walsman.
Email: jcw105@pitt.edu

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Abstract

1. Parasites kill hosts but also can indirectly increase the abundance of their resources. Given this resource feedback, how much will parasites decrease host density? Can they increase host density? Seeking answers, we integrate trait measurements, a resource–host–parasite model and experimental epidemics with plankton. This combination reveals how parasites may decrease or increase host density. This spectrum of outcomes reflects tension between parasite-driven mortality (a density-mediated effect) and foraging depression upon contact with parasite propagules (a trait-mediated one).
2. In the model, mortality rises when higher susceptibility to infection increases infection prevalence. These large epidemics release resources while suppressing hosts (creating a trophic cascade). In contrast, when hosts are less susceptible and parasites depress host foraging, a resource feedback can elevate host density during epidemics (creating a hydra effect), particularly at higher carrying capacity of resources. This combination creates the hydra effect because it elevates primary production relative to per-host consumption of resources (two key determinants of host density).
3. We test these predictions qualitatively with trait measurements and a mesocosm experiment. Clonal lines of zooplankton hosts differed in their foraging depression and susceptibility. Then, with these different host genotypes, we created epidemics in mesocosms supplied with either low or high nutrients (to manipulate carrying capacity). Hydra effects and trophic cascades both arose and in the trait–nutrient combinations predicted by the model.
4. Hence, we show how tension between trait- and density-mediated effects of parasites can govern the fate of host density during epidemics—from trophic cascades to hydra effects.

KEYWORDS
disease ecology, experimental epidemics, hydra effect, theoretical ecology, trait-mediated effects, trophic cascade

1 | INTRODUCTION

Virulent parasites threaten host populations across taxa (Dobson et al., 2008). Parasites impose fitness costs, increasing mortality and/or reducing fecundity of infected hosts. This harm to fitness can decrease host density (Daszak et al., 2000), potentially contributing to extinction of host populations (Ebert et al., 2000; Vredenburg et al., 2010). Additionally, harmful outbreaks of parasites can severely damage crops (Fry & Goodwin, 1997) and livestock (Cleaveland et al., 2001; Horan & Fenichel, 2007). Furthermore, epidemics that depress host density can trigger conservation crises, for example, in mammals (Roelke-Parker et al., 1996), birds (Cooper et al., 2009) and amphibians (Vredenburg et al., 2010). Declines of host populations during epidemics can also indirectly release resource species consumed by hosts; such parasite-driven trophic cascades have been observed in numerous parasite–host–resource systems (Buck & Ripple, 2017). These increased resources may then increase host fitness, complicating the net effect of parasites on host populations. But, these net effects matter for community composition (Wood et al., 2007) or biological control (Boivin et al., 2012). Hence, it becomes valuable to predict how strongly parasites will suppress host density and release their resources.

The strength of parasite-driven trophic cascades should depend on the traits that control interactions among parasites, hosts and their resources. One key trait is the susceptibility of hosts to infection. Since susceptibility promotes infection of new hosts, it typically increases the proportion of hosts infected (Dwyer & Elkinton, 1993; Strauss et al., 2015, 2018; Thrall & Burdon, 2000). Once infected, individual hosts can suffer reductions of fitness such as increased mortality (Ebert et al., 2000). Multiplied by higher prevalence, such virulence increases population-level mortality and depresses host density (Hall et al., 2011; Hochachka & Dhondt, 2000). Therefore, higher susceptibility should lead to stronger 'trophic cascades', with stronger host suppression and larger resource release. In this sense, susceptibility to parasites acts like attack rate of predators in predator–prey-resource systems: both modulate mortality of victims and increase cascade strength (see Appendix Section 1a or Shurin & Seabloom, 2005). Hence, mortality imposed by parasites (and predators) can strengthen trophic cascades.

However, natural enemies can also depress their victim's foraging rate. For example, cues from predators can reduce foraging rates of their prey, for example, if prey forage less to avoid predation (Laundré et al., 2010; Morgan, 1988). Similarly, the foraging rate of hosts can slow due to virulent effects of infection, behavioural response to infection or behavioural avoidance of propagules (Hite et al., 2017, 2020; Raveh et al., 2011; Strauss et al., 2019). Foraging reduction might penalize hosts with lower nutritional intake and fecundity (Buck et al., 2018). Then again, hosts which accidentally ingest parasites and slow foraging might benefit from reduced exposure (Hite et al., 2020). Additionally, such foraging depression should also indirectly release resources from consumption pressure (Philpott et al., 2004). For hosts that slow their foraging in response to parasites, higher resource density may compensate for slower feeding. Hence, depressed foraging could impose mixed consequences for both prey and hosts: Costly reduction of energy intake could be offset by released resources and lower mortality (Beckerman et al., 1997; Morgan, 1988). Given this mix, the net effect of foraging depression in these scenarios is unclear.

Here, we show how the interplay between mortality and foraging depression controls the strength of parasite-driven trophic cascades but can also produce hydra effects. In a hydra effect, a source of mortality (e.g. predator or parasite) leads to higher—not lower—density (e.g. of prey or hosts). One mechanism producing the hydra effect ('prudent resource exploitation') involves trait-mediated indirect effects on the victim's resources (Matsuda & Abrams, 2004). In this mechanism, higher resource density can support more victims because of how resource production is divided among victims (Schröder et al., 2014). More specifically, a hydra arises when foraging depression increases resource production more than per capita consumption of resources (Abrams, 2009). Alternatively, if higher resource consumption demands (due to accompanying more mortality) exceed the increase in resource production, then enemies drive a trophic cascade. We apply this theory to parasites that cause both mortality (a density-mediated effect) and foraging depression (a trait-mediated effect). Furthermore, we predict and empirically test how host and resource traits govern the range from hydra effects to strong trophic cascades during epidemics.

Hence, we illustrate how susceptibility, foraging depression and carrying capacity of the resource lead to both cascades and hydra effects in a single system by interweaving models and experiments. We measured susceptibility and foraging depression traits of several clonal genotypes of zooplankton hosts and used them to parameterize the model. The model predicts that the parasite drives stronger trophic cascades as susceptibility of hosts increases (with or without foraging depression). However, hosts with lower susceptibility but strong foraging depression can experience hydra effects, especially at high carrying capacity of the resource. We tested these predictions using those same host genotypes in a mesocosm experiment. As predicted, populations of more susceptible hosts suffered larger epidemics (higher prevalence) of a fungal parasite. These larger epidemics more strongly suppressed host density and released algal resources. Furthermore, hydra effects emerged for resistant (low susceptibility) genotypes with strong foraging depression in systems receiving high nutrients. Hence, parasites (like predators) can trigger hydra effects through the 'prudent resource exploitation' mechanism. Furthermore, tension between mortality, foraging depression and resource production predictably governs the range from strong cascades to hydra effects during epidemics.

2 | MATERIALS AND METHODS

2.1 | Experimental system

We parameterize and test our model in a planktonic system. We use isoclonal lines of the freshwater zooplankton host (*Daphnia dentifera*) to manipulate focal traits (Strauss et al., 2015). Hosts

Symbol	Meaning	Units	Value
t	Time	day	Varies
R	Density of resources	µg chl *a*/L	Varies
S	Density of susceptible hosts	hosts/L	Varies
I	Density of infected hosts	hosts/L	Varies
H	Total host density, $S + I$	hosts/L	Varies
Z	Density of parasite propagules	parasites/L	Varies
p	Prevalence of infection, $\frac{I}{S+I}$	unitless	Varies
c	Conversion efficiency, hosts	hosts/µg chl *a*	0.18[a]
d	Background mortality rate	day^{-1}	0.011[a]
f_0	Foraging rate, maximum	L $host^{-1}$ day^{-1}	0.0138[b]
$f(Z) = f_0 e^{-\alpha Z}$	Foraging rate, function of Z	L $host^{-1}$ day^{-1}	See Figure 1a
K	Carrying capacity, resources	µg chl *a*/L	94.3; 10–100[a]
m	Loss rate of parasites	day^{-1}	1.93[c]
r	Intrinsic rate of increase, R	day^{-1}	0.52[c]
u	Susceptibility to parasites	host/parasite	5.81×10^{-5}; 0.157–4.35×10^{-4}[a]
v	Pathogen-induced mortality	day^{-1}	0.045; 1.05×10^{-5}–0.06[a]
α	Foraging depression, hosts	L/parasite	3.45×10^{-6}; 0–2.27×10^{-5}[b]
σ	Parasite production per host	parasites/host	1.32×10^{5}[a]

[a]Biologically reasonable (fm. Strauss et al., 2015).
[b]Reasonable given data.
[c]A reasonable estimate.

TABLE 1 Symbols for state variables (top) and traits and other parameters (bottom) in the dynamical model (Equation 1). Default values are accompanied by ranges

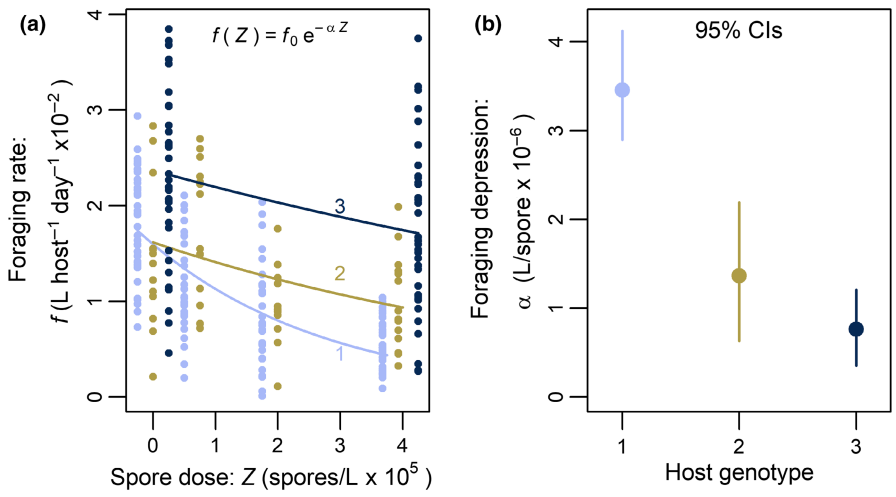

FIGURE 1 Differences among genotypes in the coefficient of foraging depression (α). (a) Fits of an exponential decay model for foraging rate with spores, $f(Z) = f_0 e^{-\alpha Z}$ to data from a foraging experiment. Larger values of α indicate stronger depression of foraging rate with spore dose, Z. Points are jittered horizontally for clarity. (b) The coefficient of foraging depression (α) for genotype 1 was stronger than for the others (2 and 3). Error bars are bootstrapped 95% CIs

incidentally ingest free-living propagules of a virulent fungal parasite (*Metschnikowia bicuspidata*; Ebert, 2005) while filter feeding on algal resources (*Ankistrodesmus falcatus*). Infected hosts have elevated death rate, and they release infectious propagules upon death. The short generation times of hosts, resources and parasites enable multigeneration feedbacks during experimental epidemics. Our work did not require ethical approval.

2.2 | Estimation of susceptibility and foraging traits

We define susceptibility as the probability a host becomes infected given a single exposure. For each of three clonal genotypes, we estimated susceptibility from the number of hosts becoming infected following controlled time and dose of propagules (previously reported in Strauss et al., 2015). For these same host genotypes,

we quantified the depression of foraging rate when hosts contact spore propagules. Foraging rate of individual hosts was measured for 8 hr at different spore concentrations (Strauss et al., 2019). Since terminal infection develops over a longer period (~9 days, Stewart Merrill et al., 2019), the measured depression of foraging rate reflects behaviour more than a symptom of infection. We fit foraging data for genotypes 1 and 2 (by Strauss et al., 2019, reanalysed here) and for genotype 3 (previously unpublished) as an exponential function of spores (more appropriate than linear according to Strauss et al., 2019): $f(Z) = f_0 e^{-\alpha Z}$ (where f_0 is the maximum foraging rate; see Appendix Section 2a). This exponential function prevents the unrealistic case of a negative feeding rate at high Z. It also fit better than a linear function (overall ΔAIC = 12.22). (A piecewise linear function could alleviate negative feeding. However, it created a differentiability issue for local stability analysis, so we avoided it.) We determined the significance of differences in these coefficients of foraging depression (α) by bootstrapping 95% CIs (Efron & Tibshirani, 1993 see Appendix Section 2a for details). All analyses were performed in Rstudio (R Core Team, 2019).

2.3 | Resource–host–parasite model

We analyse a minimal model of logistically growing resources (R), susceptible hosts (S), infected hosts (I) and free-living parasite propagules (spores, Z). This model captures key biology of focal planktonic system (see also Table 1):

$$\text{Resources: } \frac{dR}{dt} = rR\left(1 - \frac{R}{K}\right) - f(Z)(S+I)R, \tag{1a}$$

$$\text{Susceptible hosts: } \frac{dS}{dt} = cf(Z)(S+I)R - dS - uf(Z)SZ, \tag{1b}$$

$$\text{Infected hosts: } \frac{dI}{dt} = uf(Z)SZ - (d+v)I, \tag{1c}$$

$$\text{Spores: } \frac{dZ}{dt} = \sigma(d+v)I - mZ. \tag{1d}$$

Resources grow logistically, a reasonable assumption allowing primary productivity to (potentially) increase during epidemics. Resource productivity is population-level recruitment of new resources per unit time, $rR(1 - R/K)$, and depends on intrinsic rate of increase r and carrying capacity K (Equation 1a). Resources are consumed by susceptible (S) and infected (I) hosts foraging at rate $f(Z) = f_0 e^{-\alpha Z}$ (parameterized above) which declines exponentially with the density of spores (Z). This is a trait-mediated effect of parasites and ranges from strong (large α) to absent ($\alpha = 0$); because hosts reproduce clonally and host clones show consistent variation in α, model populations have a certain, fixed value of α. We assume equal foraging rate and foraging depression for susceptible and infected classes as a first, simplest approach (see Penczykowski et al., 2022 for a reduced foraging rate by infected hosts). Both host classes, S and I, convert resources into susceptible offspring with conversion efficiency c (Equation 1b). Hence, transmission is horizontal. For simplicity, infection does not lower fecundity here, but we consider it in the Appendix Section 1g. Total host density is $H = S + I$. Hosts experience background mortality at rate d (Equation 1b) due to predation, senescence and other factors. Susceptible hosts encounter parasites while foraging; their susceptibility, u, determines the proportion of hosts infected per encounter. Exposure and susceptibility jointly determine the transmission rate (often denoted β) from the environment to susceptible hosts, $uf(Z) = uf_0 e^{-\alpha Z}$ (Equation 1b). Infection converts susceptible hosts into infected hosts (Equation 1c). They suffer elevated death rate due to virulence of infection, $d + v$ (where v is pathogen-induced mortality; Equation 1c). Death of infected hosts is the density-mediated effect of parasites. When infected hosts die, they release σ parasite propagules (Z) back into the environment (Equation 1d). Losses of parasite propagules occur at background rate m (Equation 1d), for example, due to sinking, consumption (Strauss et al., 2015), solar radiation (Overholt et al., 2012) etc.

The model has a single, stable, endemic equilibrium for the range of parameter values considered. Since the exponential form of $f_0 e^{-\alpha Z}$ prevented general analytical solutions (though see Equation 2 for partial solutions), we found equilibrial densities at each parameter set with the rootSolve package (Soetaert, 2009) and evaluated the stability of each using local stability analysis with the Deriv package (Clausen & Sokol, 2020) in Rstudio version 3.6.0 (R Core Team, 2019). We then determined the effects of prevalence (p), susceptibility (u), foraging depression (α) and carrying capacity (K) on resource (R) and host (H) densities using a mixture of analytical and numerical techniques. We defined the strength of trophic cascades (resource release with host suppression) or hydra effects (host increase) as ratios of the stable endemic equilibrium to the disease-free boundary equilibrium. Since the mesocosm epidemics likely did not reach equilibria, we compared the closest analogue: We qualitatively matched them to transient dynamics of the model using the deSolve package (Soetaert et al., 2010) and used the equilibria to predict longer term outcomes (see Appendix Section 1c for parameter values in which oscillations or bistability [when $\alpha > 0$] arise).

2.4 | Mesocosm test of model predictions

2.4.1 | Experimental design

We test predictions of the dynamic model with populations of interacting algal resources, zooplankton hosts and fungal parasites. To manipulate host traits, we stocked 50 L mesocosms with hosts of either one clonal genotype differing in foraging depression (α) and susceptibility (u) traits or a 50:50 mixture of two genotypes. In these mixtures, traits of mixed populations were bounded by those of the two genotypes. Furthermore, despite the potential for change

in clonal frequency, initial trait values qualitatively predicted the patterns of density outcomes (see below). Using a weighted average of traits from observed genotype frequencies (not shown) did not improve model fit or qualitatively change any outcomes (using same methods as Walsman et al., 2021). Then, to manipulate carrying capacity of algal resources, we added either low or high supply of inorganic nutrients (see Appendix Section 2b for more experimental details). Finally, disease treatments were inoculated once with fungal spores (propagules). Altogether, the design crossed six susceptibility treatments (genotypes 1, 2, 3, 1&2, 1&3 or 2&3) × two nutrient supply treatments (low or high) × two parasite treatments (present or absent), all replicated three times for 72 total mesocosms. With twice-weekly sampling, we measured algal density, host density and infection prevalence over c. seven host generations (~1 generation every 10 days; see Appendix Sections 2b,c).

2.4.2 | Statistical analyses

We assume that temporal averages approximate equilibria from the model as supported by simulations. In that model, connections between susceptibility (u) and prevalence (p) prove important mathematically. Hence, we first tested for their relationship (and with nutrient supply) using beta regression with the BETAREG package (Cribari-Neto & Zeileis, 2010; see Appendix: Section 2d for more details). Next, we fit a linear model of $\log_{10}$-transformed algal or host density, respectively, predicted by disease, susceptibility and nutrients with interactions between disease and susceptibility and disease and nutrients (see Equation S7 for details). Fitting log densities reduces bias while having more normal error distributions (Hedges et al., 1999). If susceptibility increases the strength of trophic cascades, there should be a significant positive interaction between susceptibility and disease for $\log_{10}$ resource density (since resources increase) and negative interaction for $\log_{10}$ host density (since hosts decline; see Appendix Section 2e). Additionally, we tested for a hydra effect in two genotype treatments at high nutrient supply using a nested ANOVA (see Appendix Section 2f). For each genotype, $\log_{10}$ host density was the response variable with mesocosms nested within disease treatment and repeated measurements nested within mesocosms. This nesting incorporated the power while appropriately handling the autocorrelation of repeated measurements. Of the original 72 mesocosms, nine were removed as outliers (most due to population extinction; see Appendix Section 2c). All analyses were performed in Rstudio (R Core Team, 2019).

3 | RESULTS

3.1 | Estimation of susceptibility and foraging traits

The host genotypes cover a wide span of trait space, enabling significant manipulation of the traits of host populations by stocking different genotypes. The three genotypes span almost an order of magnitude of susceptibility (Bristol 10: $u_1 = 5.81 \times 10^{-5}$, A4-3: $u_2 = 1.09 \times 10^{-4}$, Standard: $u_3 = 3.93 \times 10^{-4}$), with u_3's 95% CI not overlapping the other genotypes (Strauss et al., 2015). All host genotypes significantly depressed their foraging in the presence of parasites (Figure 1a,b). Foraging depression was more than twice as strong for genotype 1 ($\alpha = 3.5 \times 10^{-7}$ L/spore) as for genotypes 2 or 3 (1.4×10^{-7} and 7.6×10^{-8}; Figure 1b). Given this coefficient, the foraging rate of genotype 1 decreased fourfold over the range of spore doses in the trait assay (roughly corresponding to the range of spore densities in the mesocosm experiment, see Appendix Section 2d).

3.2 | Resource–host–parasite model

Susceptibility (u) modulates host mortality while foraging depression ($\alpha > 0$) enables the parasite-driven hydra effect. Higher susceptibility (u) of host populations leads to higher prevalence of infection (p^*) and thus higher mortality ($d + vp^*$). Higher mortality then leads to larger release of resources and suppression of host density, hence a stronger trophic cascade. Thus, susceptibility acts analogously to attack rate of predators (see Appendix Section 1a and Shurin & Seabloom, 2005). Yet, with lower susceptibility (hence lower mortality), foraging depression can drive a hydra effect. Insight arises from the numerator and denominator of host density (Schröder et al., 2014) derived from the resource equation (Equation 1a). Host density ($H^* = S^* + I^*$) is the ratio of primary [resource] productivity, $PP = rR^*(1 - R^*/K)$, to per host food consumption, $FC = f(Z^*)R^*$. Resource density, R^*, in turn, is host death rate [$d + vp^*$, with prevalence $p^* = I^*/H^*$] divided by fecundity per resource available [$cf(Z^*)$]. Thus, host density depends on productivity and consumption of resources, without ($Z-$) or with disease ($Z+$):

$$\text{Resources without disease: } R^*_{Z-} = \frac{d}{cf_0}, \tag{2a}$$

$$\text{Resources with disease: } R^*_{Z+} = \frac{d + vp^*}{cf(Z^*)}, \tag{2b}$$

$$\text{Hosts without disease: } H^*_{Z-} = \frac{PP_{Z-}}{FC_{Z-}} = \frac{rR^*_{Z-}(1 - R^*_{Z-}/K)}{f_0 R^*_{Z-}}, \tag{2c}$$

$$\text{Hosts with disease: } H^*_{Z+} = \frac{PP_{Z+}}{FC_{Z+}} = \frac{rR^*_{Z+}(1 - R^*_{Z+}/K)}{f(Z^*)R^*_{Z+}}. \tag{2d}$$

Because disease raises mortality, it still must increase the minimal resource requirement of hosts (compare Equation 2a, b; $R^*_{Z+} > R^*_{Z-}$ since foraging rate can drop, $f(Z) \leq f_0$, while mortality increases, $d + vp^* > d$). Released resources would lower primary productivity if hosts have an overly high minimal requirement without disease (i.e. $R^*_{Z-} > K/2$; PP is maximized at $K/2$, so higher R^*_{Z+} would

lower *PP*). Lower primary productivity, in turn, necessarily decreases host density (permitting only a trophic cascade). In contrast, if hosts more strongly controlled resources without disease ($R^*_{Z-} < K/2$), resource release can increase primary productivity (if R^*_{Z+} is closer to $K/2$ than R^*_{Z-}). However, increased primary productivity alone may not suffice: Hydra effects only emerge when increased productivity ($PP_{Z+} > PP_{Z-}$) outweighs increased food consumption per host ($FC_{Z+} > FC_{Z-}$; Equation 2c,d).

Hence, food consumption places an additional constraint on the hydra effect. Food consumption per host (*FC*) is the product of foraging rate, $f(Z^*)$, and the density of resources, R^*. Higher mortality from infection increases both R^* and food consumption when foraging rates stay constant [$\alpha = 0$, so $f(Z^*) = f_0$]. If parasites only kill hosts ($v > 0$, $\alpha = 0$), food consumption increases more with R^* than primary productivity can [since H^*_{Z+} simplifies to $r(1 - R^*/K)/f_0$, a decreasing function of R^*]. So, parasites that only kill must decrease host density. If parasites only depress host foraging rate ($v = 0$, $\alpha > 0$), resource density with disease simplifies to $R^*_{Z+} = d/[cf(Z^*)]$ (from Equation 2b). This minimal resource requirement, R^*_{Z+}, compensates for foraging depression completely; hence, food consumption remains constant ($FC_{Z+} = FC_{Z-}$ when $v = 0$; Equation 2c,d). Thus, a parasite that only depresses foraging rate of hosts (but does not kill) will increase host density if primary productivity increases with disease. If parasites increase mortality and depress host foraging rate ($v > 0$, $\alpha > 0$), they can drive a hydra effect if the increase in *PP* outweighs the simultaneous increase in food consumption per host. Thus, a tension between foraging depression and mortality emerges because they differentially influence the production and consumption of resources.

These duelling effects of production and consumption explain why hydra effects become more likely with higher carrying capacity of the resource (*K*). Higher *K* elevates the density of parasite propagules in the environment (*Z*) in both the mortality-only case ($v > 0$, $\alpha = 0$; dashed red) and the foraging-depression case ($v > 0$, $\alpha > 0$; solid red curve; Figure 2a). More propagules, in turn, decrease foraging rate (when $\alpha > 0$; Figure 2b). Resources (R^*) also increase with carrying capacity, but more so if hosts depress foraging (Figure 2c). That larger release of resources can create a stronger increase in primary productivity and more so at higher *K* (Figure 2d). At the same time, food consumption per host (Figure 2e) increases with *K* but less so with foraging depression than without. Hence, epidemics more easily drive a hydra effect at higher *K* (Figure 2f).

This framework predicts how carrying capacity (*K*), susceptibility (*u*) and foraging depression (α) jointly determine the range from strong cascades to hydra effects during epidemics. Foraging rate drops with α, releasing resources (higher resource ratio); however, as parameterized, *K* has a stronger effect on resource release (Figure 3a). That resource release can then increase primary productivity (*PP*). Foraging depression (high α) also decreases food consumption (*FC*) per host. Hence, the host ratio (H^*_{Z+}/H^*_{Z-}) increases

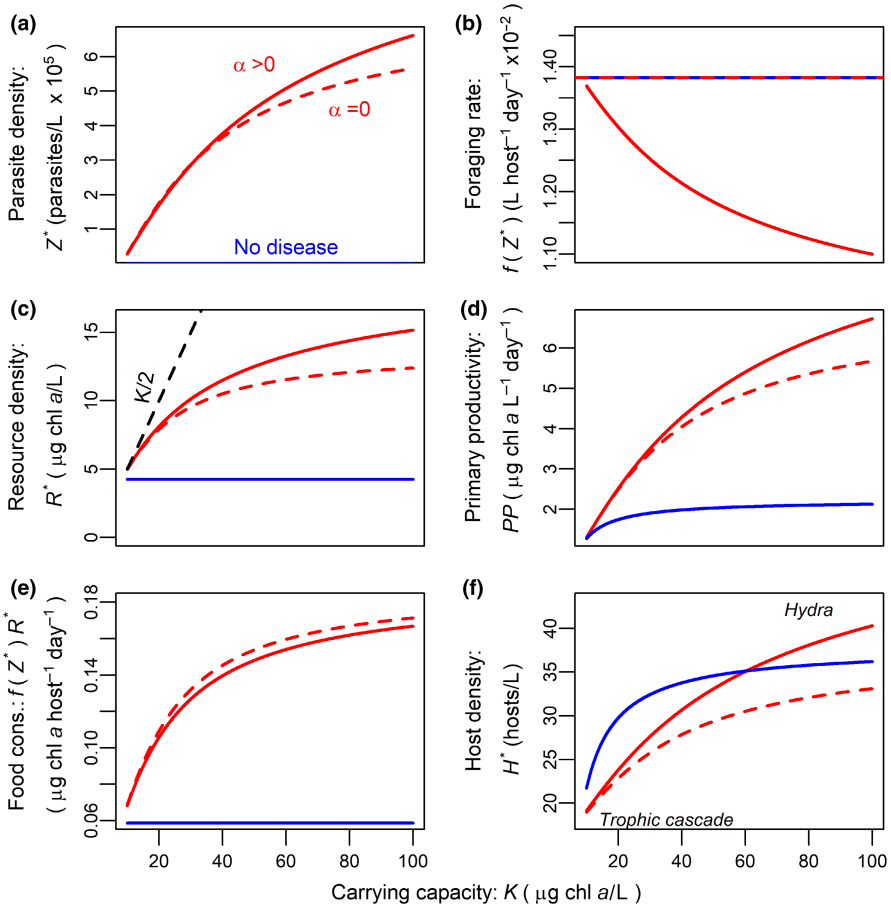

FIGURE 2 Foraging depression produces a hydra effect at high carrying capacity (see Figure 3; Equation 2). Equilibrium quantities shown for three cases: no-disease (blue, *Z*–), mortality-only (dashed red, *Z*+; $\alpha = 0$, $v > 0$) and foraging depression with mortality (solid red, *Z*+; $\alpha > 0$, $v > 0$). (a) Higher carrying capacity of the resource (*K*) increases propagule density (Z^*), dropping (b) foraging rate if $\alpha > 0$. (c) Parasites release resources ($R^*_{Z+} > R^*_{Z-}$). R^*_{Z+} is closer to $K/2$ (dashed black) with foraging depression. (d) Hence, primary productivity (*PP*) is highest when $\alpha > 0$ and lowest without disease. (e) Food consumption, [$FC = f(Z^*)R^*$], is higher without foraging depression. (f) Given these responses of *PP* and *FC*, epidemics cause trophic cascades ($H^*_{Z-} > H^*_{Z+}$ always) without foraging depression but can cause hydra effects when $\alpha > 0$ at higher *K* (parameters follow Table 1)

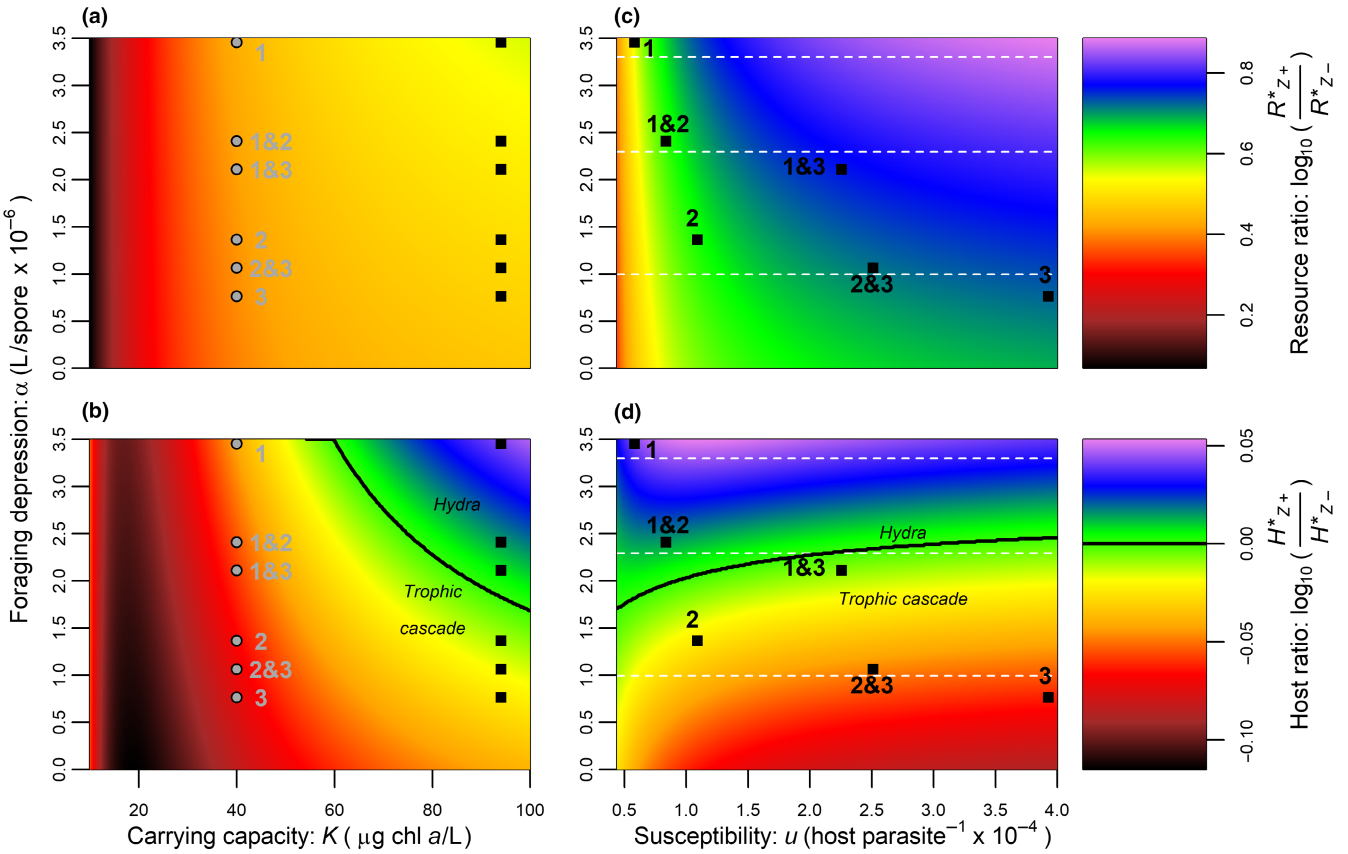

FIGURE 3 Combinations of carrying capacity (*K*, left column) or susceptibility (*u*, right) with foraging depression (α) create cascades or hydra effects. Points indicate traits of numbered genotypes at low (light grey) or high (black) nutrient supply (note this comparison is qualitative). Colours indicate $\log_{10}$ density ratios of resource (top row: $\log_{10}[R^*_{Z+}/R^*_{Z-}]$) or hosts (bottom: $\log_{10}[H^*_{Z+}/H^*_{Z-}]$). Black curves note host ratio = 0 (above: hydra effects). *K*–α space: (intermediate $u = 2.22 \times 10^{-4}$) (a) Resource ratio increases with α but especially *K*. (b) Both *K* and α jointly increase host ratio, yielding a hydra effect at high enough *K*–α (see Figure 2f). *u*–α parameter space: (high $K = 94.3$) slices at dotted white lines shown in Figure S2. (c) Higher *u* and α both increase resource ratio. (d) Higher *u* increases parasite-driven mortality, reducing the region of hydra effects in *u*–α space. (Parameters follow Table 1)

with higher *K* (due to enhanced *PP*) and higher α (via lower *FC*). In fact, it can cross from trophic cascade into hydra effect (past the black line [$\log_{10}$ ratio = 0] in Figure 3b). That hydra effect becomes less likely when hosts are more susceptible. Higher *u* leads to higher mortality. That mortality effect releases resources strongly (Figure 3c) and boosts primary production. However, higher mortality also increases food consumption per host, decreasing the likelihood of hydra effects (Figure 3d; see Figure S2 for an exception at low *u* and high α). Similarly, hydra effects become less likely as parasites lower survival more per individual host (higher *v*; see Figure S3 for small exceptions). Therefore, hydra effects arise at higher *K* and when hosts experience more foraging depression and less mortality.

These modelling results predict a qualitative pattern for the mesocosm results. More specifically, trait values (see Figure 3) indicate where genotype treatments should fall along the spectrum from strong cascades to hydra effects. These patterns calculated from equilibria are qualitatively similar when the system is not yet in equilibrium in simulations or mesocosms (see Figure 4; Figures S4–S6). Similarly, the presence of two host clones does not alter the predicted impact of host traits (Figures S4 and S6). Therefore, the model predicts patterns linking traits to the cascade–hydra effect spectrum for the experiment to test.

3.3 | Mesocosm test of model predictions

Most genotype treatments produced trophic cascades (released resources and suppressed host density). In populations with disease, higher susceptibility (*u*) increased the prevalence of fungal infection (found from beta regression; $p = 0.0067$; see Figure S7a and Appendix Section 2d). Higher nutrients also increased the prevalence of disease ($p = 0.0198$), as predicted (see Figure S7a). The model predicts parasites will increase resources and more so at high nutrients or high susceptibility (see Figure 3a,c; Figures S5a,c,e and S6a,c,e); the model also predicts parasites will depress hosts overall and more so at high susceptibility (see Figures 3d and 4a,c,e; Figure S4a,c,e). Relative to disease-free populations, disease released algal resources overall ($p = 0.00126$) and suppressed host density ($p = 0.00137$; see Table S1 for more model-data comparison). Susceptibility increased the strength of these trophic cascades, as predicted. Treatments with higher susceptibility displayed stronger release of algal resources

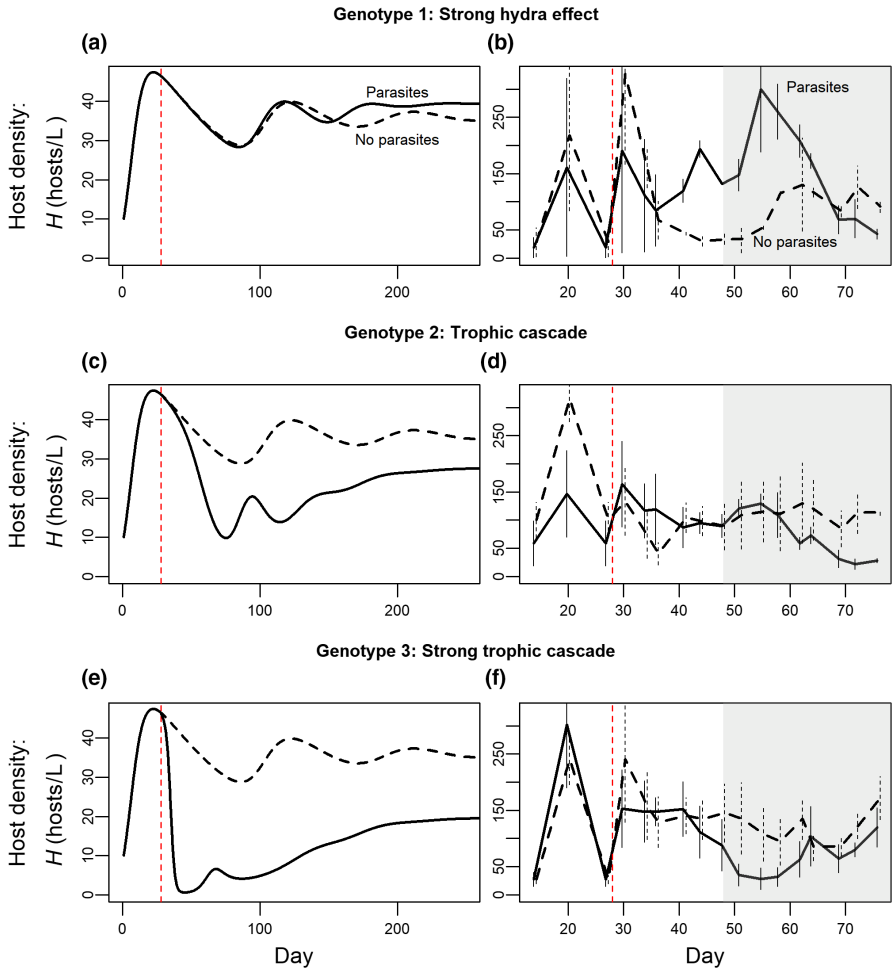

FIGURE 4 Simulated and experimental time series at high nutrients (K = 94.3 in simulations or 50 μg/L P in mesocosms) produce a spectrum ranging from hydra effects to trophic cascades. In both simulations and the experiment, hosts and parasites are added on days 1 and 28 (red line) respectively. (a) With genotype 1's traits, the hydra effect emerges given sufficient time as host density with parasites (solid) becomes higher than without (dashed). (b) Mesocosms of genotype 1 experienced a hydra effect [mean density across replicates with parasites (solid) or without (dashed); bars are standard error at each time point]. (c) With genotype 2's traits, a trophic cascade (host suppression and resource release [Figure S5]) occurs in simulations and (d) the mesocosm. (e–f) This cascade is larger for genotype 3 (the most susceptible). (Parameters follow Table 1). For analyses, average mesocosm density was taken from day 48 to 76 (grey region, see Appendix Section 2c). Experimental time series shifted slightly horizontally for clarity. Compare simulations to Figure 3's equilibrium outcomes and mesocosm time series to Figure 5's mesocosm averages

(positive $u \times Z$ interaction, $p = 0.0277$; see Figure 5a, Figures S5b,d,f, S6b,d,f and S8b). Susceptibility also strengthened host suppression (negative $u \times Z$ interaction, $p < 0.001$; Figures 4b,d,f and 5d; Figures S4b,d,f and S8d). However, the mesocosm did not match model predictions well for the interaction of nutrients and cascade strength. Unlike model predictions from equilibrium, higher nutrients increased resources without disease (p = <0.001, see Appendix Section 2e for possible explanations); this trend likely contributed to the lack of a significant trend for nutrients and resource ratio. The model predicted that nutrients would amplify resource ratio, but this amplification was non-significant ($p = 0.841$). For host ratio, the model does not make a simple prediction for the effect of nutrients, and none was found ($p = 0.241$). Thus, the model-data match was clearer for the impact of susceptibility on trophic cascades than for nutrients on them.

Two sets of treatments displayed a hydra effect. At high nutrient supply, host density was higher with disease than without it for genotype 1 alone (strong hydra: 79% higher with disease, $p = 0.007$; Figures 3d and 4a and square labelled '1' in Figure 5b) and for the mixture of genotypes 1 & 2 (weaker hydra: 34% higher, $p = 0.020$; Figure 3d; Figure S4a and square '1&2' in Figure 5b). Since genotype 1 has low susceptibility (u) and high coefficient of foraging depression (α; Figure 1b), these results follow the model prediction for hydra effects at high K, lower u and higher α (see Figures 3b,d and 4a; Figure S4a).

4 | DISCUSSION

Parasites can drive trophic cascades in a variety of disease systems (Buck & Ripple, 2017). More specifically, disease epidemics can depress host density while releasing resources of hosts. Here, we offer a framework to predict strength of those trophic cascades as

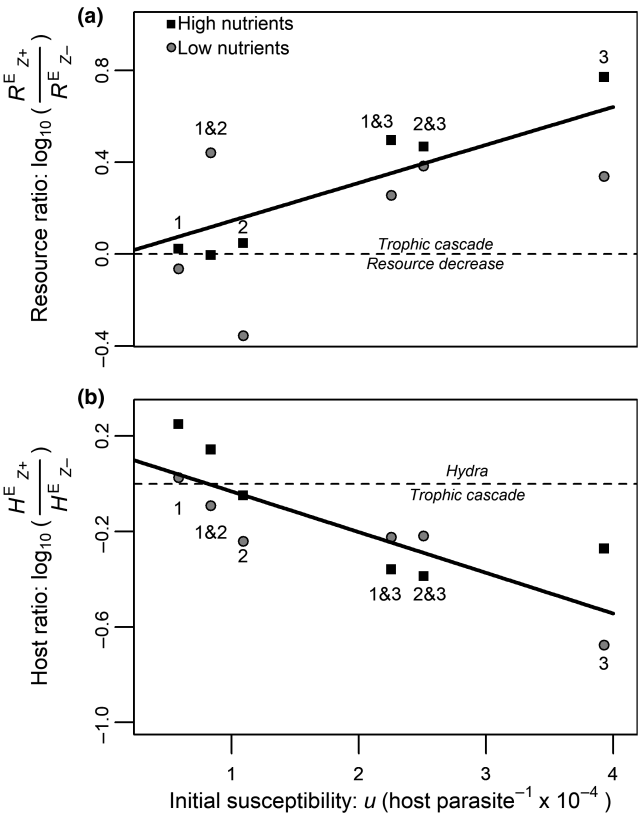

FIGURE 5 In experimental epidemics, parasites drove cascades or hydra effects depending on susceptibility (u), foraging depression (α) and nutrient supply (K). Each point represents the average resource or host ratio. Algal resources: R^E (E for 'experiment'). (a) Higher susceptibility amplifies resource release (increasing $\log_{10}$ ratio of resources). (Error bars for ratios not shown: they are non-trivial to compute). Plankton hosts: H^E. (b) Higher susceptibility amplifies trophic cascades (stronger host suppression; $\log_{10}$ host ratios become more negative with u). However, two genotype treatments with low susceptibility and strong foraging depression (1 and 1&2 together) displayed disease-driven hydra effects ($\log_{10}$ host ratios >0) at high nutrient supply

a function of traits and nutrient supply. Higher transmission rate elevates infection prevalence. Higher prevalence, in turn, raises mortality, strengthening parasite-driven cascades. This mapping from a direct density effect (higher mortality) to an indirect density effect (resource release) closely resembles how higher attack rate leads to larger trophic cascades caused by predators (Shurin & Seabloom, 2005). Hence, this framework also applies to predator-driven cascades (Hall et al., 2008; Raffel et al., 2008 and see Appendix Section 1a for more details). Mortality imposed by either enemy—parasite, or predator—can cause trophic cascades.

The combination of model and experiment also revealed another possibility: Epidemics of virulent parasites can produce hydra effects. These hydra effects were caused by foraging depression, a trait-mediated effect. Foraging depression arises commonly in this host–parasite system (Hite et al., 2017; Strauss et al., 2019), in other host–parasite systems (Hite et al., 2020) and in predator–prey systems (Laundré et al., 2010; Morgan, 1988). Hence, the trait-mediated mechanism may apply quite broadly, to a variety of victim–enemy systems. Furthermore, the experimental demonstration of a parasite-driven hydra effect contributes a unique perspective. It was created by multigenerational feedbacks among players rather than by experimentally fixed mortality (Schröder et al., 2014). Additionally, it shows higher density of a whole population, not just that of a particular life stage (Preston & Sauer, 2020). Finally, this experimentally demonstrated hydra effect accompanies the observation that host density can increase during large fungal epidemics in *Daphnia* (Penczykowski et al., 2022). Hence, this parasite-driven hydra effect is relevant in nature.

Given its emergence here, how can we anticipate parasite-driven hydra effects in other systems? What factors might constrain or amplify their possibility? First, the relationship between susceptibility (u) and foraging depression (α) traits may make hydra effects more or less likely. In the set of genotypes here, susceptibility and foraging depression were negatively correlated, yielding strong trophic cascades (high u, low α; lower right Figure 3d) or strong hydra effects (low u, high α; upper left Figure 3d). For another set of genotypes of this host, these traits did not correlate negatively (Strauss et al., 2019). A positive correlation might reduce the probability of strong cascades or strong hydra effects. Second, hosts may evolve weaker or stronger foraging depression as selection weighs lowered fecundity against lowered exposure to parasites. Further modelling efforts would clarify these eco-evolutionary possibilities. Third, hydra effects should become less likely when parasites harm host fitness more. Similarly, parasites that impose high virulence on mortality and on fecundity should dampen hydra effects (see Appendix Sections 1e, g; but see some counter examples there). Future work could clarify how each factor influences hydra effects in a variety of host–parasite–resource systems.

Here, using models and experiments, we delineate when parasites should cause trophic cascades versus when they should trigger hydra effects. Parasite-driven trophic cascades have emerged in various systems. To predict the strength of these cascades, we developed a trait-based model framework and tested it experimentally. Yet, the parasite-mediated hydra effect that arose revealed an even newer possibility: Parasites that kill hosts could increase host density if they depress foraging via a 'prudent resource exploitation' mechanism. Hydra effects arise here due to particular feedbacks between hosts and resources. Now, disease ecologists can ask: are disease-mediated hydra effects rare? Or could they arise more commonly—if we just know where and when to look for them? Here, the mathematical model guides us. First, hosts must depress their foraging rate in the presence of parasites (a trait-mediated effect). Second, parasites must not increase host mortality too greatly (i.e. the density-mediated effect cannot become too strong). Either ensures that the density of a dynamic resource increases during epidemics. Third, higher resource density must increase resource productivity—and increase it more than per host food consumption. Without these features, a hydra effect cannot occur via this foraging mechanism. Overall, we show how these three factors (foraging depression, mortality, productivity) govern the range from strong parasite-driven trophic cascades to hydra effects.

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CONFLICT OF INTEREST

The authors have no conflicts of interest.

AUTHORS' CONTRIBUTIONS

J.C.W. and S.R.H. designed the mesocosm experiment; A.T.S. and S.R.H. designed the trait measurements; A.T.S. performed the trait measurements; J.C.W. performed the mesocosm experiment, mathematical modelling, data analysis and wrote the first draft of the manuscript while all authors contributed substantially to revisions.

DATA AVAILABILITY STATEMENT

All data and theoretical code required to produce the results are available in Dryad Digital Repository https://doi.org/10.5061/dryad.mw6m905zg (Walsman et al., 2022).

ORCID

Jason Cosens Walsman https://orcid.org/0000-0001-8141-0340
Alexander Thomas Strauss https://orcid.org/0000-0003-0633-8443

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