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Title: Competition-mediated feedbacks in experimental multi-species epizootics

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Abstract

Competition structures ecological communities and alters host-pathogen interactions. In environmentally-transmitted pathogens, an infection-resistant competitor may influence infection dynamics in a susceptible species through the negative impacts of competition (e.g., by reducing host density or causing nutritional stress that increases susceptibility to infection) and/or the positive impacts of reducing transmission efficiency (e.g., by removing environmental pathogen stages). Thus, a non-susceptible competi-

tor may enhance, reduce, or have no net effect on susceptible host density and infection prevalence. Here, we couple an epidemiological model with experimental epidemics to test how resource competition with a non-susceptible competitor (*Daphnia pulicaria*) influences fungal microparasite (*Metschnikowia bicuspidata*) infection dynamics in a susceptible host species (*D. dentifera*). Our model and experiments suggest that competitor density can mediate the direction and magnitude of the effect of competition on infection dynamics, with a peak in infection prevalence occurring at intermediate competitor densities. At low densities, the non-susceptible competitor *D. pulicaria* may reduce infection prevalence in the susceptible host by removing fungal spores from the environment through feeding. However, when competitor density is increased and resources become limiting, *D. pulicaria* negatively impacts the susceptible host by increasing susceptible host feeding rates, and therefore fungal spore intake, and further by reducing susceptible host population size as it is driven towards competitive exclusion. In conclusion, these results show that a tradeoff between the competitor as a consumer of pathogen, which serves to reduce epidemic size, and as a modifier of susceptible host foraging ecology, which influences infection rates, may alternately enhance

or dampen the magnitude of local epidemics.

Keywords

Dilution effect, Diversity-disease, Competition, Environmentally transmitted pathogen, Multi-host pathogen, Resource limitation

Introduction

Competition is a major structuring force of ecological communities, and the strength of interactions between competitors can determine whether coexistence or competitive exclusion occurs (Armstrong and McGehee, 1976; Wang et al., 2009). Further, environmental conditions can influence the outcome of competition, as competitive outcomes can be shaped by predators (Wollrab et al., 2013), temperature (Carmona-Catot et al., 2013), resource availability (Riebesell, 1974), or natural enemies (Decaestecker et al., 2015). Studies of competition typically focus on the long-term or equilibrium dynamics of competing species (Wedin and Tilman, 1993), but many interesting competitive interactions occur when populations are not at equilibrium, or are in the transient pe-

riod before competitive coexistence or exclusion (Pickett, 1980). Epidemic pathogens or parasites are a prime example of this, as they disrupt host population dynamics, and can influence transient population dynamics and competitive outcomes. Pathogens may mediate the interactions between competing species by disproportionately affecting one of the competitors (Price et al., 1988), or by altering aspects of host life history such as development time or dispersal (Thomas et al., 2000). While the influence of parasites on competitive interactions has received ample attention (Park, 1948; Price et al., 1988; Preston and Johnson, 2010; Hatcher et al., 2012), there have been few studies into the role of competitors on parasite populations (Hall et al., 2009).

Competitors may differ in competence (Kilpatrick et al., 2006) and susceptibility to pathogen infection (Hall et al., 2007), such that the addition of a competitor can reduce, enhance, or have no net effect on infection dynamics in another competing host. For instance, a competitor that is a more competent host species could increase parasite population size, which would potentially elevate infection rates of the other competing host. However, if the competitor is a less competent host, or if the effect of competition reduces potential pathogen transmission events to the other competitor, infec-

tion risk may be reduced. The effect of competition therefore may offer a mechanistic explanation for some cases of the relationship between host diversity and disease risk (the "diversity-disease relationship").

The diversity-disease relationship proposes that increases in host diversity may reduce (i.e. a dilution effect) or enhance (i.e. an amplification effect) infection risk in a focal host species (Ostfeld and Keesing, 2012; Orlofske et al., 2012). In theory, dilution effects may arise for many different reasons, but generally, the non-focal species are considered to be subject to "wasted" transmission events, so that pathogen fitness is reduced by infecting a less suitable host (Keesing et al., 2006). Studies of the dilution effect are typically phenomenological (Salkeld et al., 2013), and do not incorporate ecological interactions among species in the community. The inclusion of ecological interactions (e.g., competition) into studies of multi-host pathogen dynamics may inform a general theory for when we expect host diversity to reduce or enhance disease. Here, we use a combination of modeling and experiments to provide a link between diversity-disease relationships and parasite-mediated competition. Specifically, we investigate the impact of the addition of a non-susceptible superior competitor that consumes en-

vironmental pathogen on the infection and population dynamics of a susceptible host species.

To do this, we use a model system comprised of two sympatric zooplankton competitors, *Daphnia dentifera* and *Daphnia pulicaria*. These two species have been found to co-occur in the north temperate lakes of the United States (Duffy et al., 2010). *Daphnia dentifera* is susceptible to infection by an environmentally-transmitted yeast pathogen (*Metschnikowia bicuspidata*), and is also an inferior competitor to *D. pulicaria*, as *D. pulicaria* has larger body size, reproductive rate (*unpublished data*), and foraging rate (Gliwicz, 2004). Further, *D. pulicaria* has been found to outcompete *D. lumholtzi*, a formidable invasive competitor (Engel and Tollrian, 2011). While *D. pulicaria* does not become infected, it does consume pathogen spores during foraging, potentially reducing pathogen transmission to susceptible hosts (so-called "friendly competition" ; Hall et al. (2009)). Reduced resources can nutritionally stress susceptible hosts, which can result in enhanced pathogen transmission (Pulkkinen and Ebert, 2004) as a result of increased filtering rate (Hall et al., 2007), providing a mechanistic link between host foraging ecology and pathogen transmission. Therefore, the impact of competition on

infection dynamics will depend on the tradeoff between the role of the competitor as a consumer-of-pathogen and as a consumer-of-resources.

Previously, Hall et al. (2009) performed an experiment in which *D. pulicaria* were allowed to graze on pathogen spores, and then this media was exposed to susceptible *D. dentifera* to see if *D. pulicaria* grazing could reduce transmission by depleting environmental pathogen spores. However, this study did not account for the role of the competitor (*D. pulicaria*) as a consumer. By reducing algal resources, the competitor may indirectly influence susceptible host foraging rate, which is intrinsically linked to pathogen transmission in this system. A theory for this complex of interactions was recently developed by Cáceres et al. (2014), who examined the equilibrium outcomes of competition between a susceptible and a non-susceptible competitor, finding competitive exclusion of the susceptible host species over long time scales when the non-susceptible species is a superior competitor. While previous studies of parasite-mediated competition have focused on directly transmitted pathogens, and superior competitors that are also susceptible to parasitism (Price et al., 1986, 1988), we focus on an environmentally-transmitted pathogen, and the interaction between a dominant com-

petitor that does not become infected and an inferior susceptible competitor. Theory predicts competitive exclusion of the inferior competitor in the long-term, although these species coexist in natural systems, most likely through niche partitioning or complex community interactions. We focus on the transient period where both species coexist, and examine infection dynamics as a result of competition for a limiting resource altering exposure to an environmentally transmitted pathogen. These transient dynamics are important, and ecologically relevant, given that the seasonal fluctuations in both zooplankton population sizes and infection dynamics may preclude zooplankton populations from achieving equilibrium dynamics (Hutchinson, 1961; Scheffer et al., 2003).

Using a modified version of Cáceres et al. (2014) model that more closely matches our experimental system (see Supplementary Materials for a comparison of our model to Cáceres et al. (2014)), we extend this theory by examining the influence of competitor density on epidemic and population dynamics under non-equilibrium conditions. By examining the transient dynamics of our theoretical model, we generate several testable model predictions, and experimentally test these predictions using the same zooplankton-pathogen system examined by Hall et al. (2007). First, we predict that

the extent to which the competitor enhances or reduces infection prevalence in the susceptible host will depend on the initial density of the competitor and the availability of algal resources. When resources are limiting, we predict that the competitor will enhance infection prevalence in the susceptible host species by enhancing susceptible host foraging rate, and subsequent pathogen transmission. Second, we predict that susceptible host population size will decrease as a function of competitor density, since the susceptible host will be excluded more rapidly at higher densities of the superior competitor. This reduction in susceptible host population size may reduce infection prevalence if contact with pathogen (and therefore transmission) is reduced, or increase infection prevalence if susceptible host filtering rate is increased as a response to reduced resources. To test these hypotheses, we parameterized our epidemiological model, and compared model outputs with experimental epidemics. Experimental epidemics were initiated at three competitor densities, as increased competitor density serves to reduce resource availability through exploitative competition. We found that competition rarely benefited the susceptible host species, either enhancing infection prevalence at intermediate competitor densities, or competitively excluding the susceptible host at

high competitor densities. This work highlights the importance of competitive interactions in evaluating the direction of diversity-disease relationships.

Methods

Study system Clonal lines of two sympatric freshwater cladocerans were used in this study, *D. dentifera* (provided by M. Duffy) and *D. pulicaria* (originally isolated from Oneida Lake, New York, and provided by N. Hairston Jr.). *Metschnikowia bicuspidata* is a fungal pathogen that infects *D. dentifera*, but not *D. pulicaria*. Pathogen transmission can occur when the host ingests the pathogen, allowing the pathogen to pierce the gut wall and grow within the host. Parasite-induced mortality causes the release of a multitude of infectious spores (see Table 1), which are then filtered and ingested by other hosts. Recent studies have found essentially no genetic variation in the fungal pathogen, reducing the likelihood of genotype × genotype interactions (Searle et al., 2015). However, genetic variation within natural *D. dentifera* populations could influence pathogen transmission dynamics through heterogeneity in resistance, or spore production per host (Auld et al., 2013; Carius et al., 2001). We acknowledge this as

an interesting avenue for further research. However, our focus is to elucidate patterns in infection dynamics due to competitor density. Therefore, we selected a single, well-studied *D. dentifera* clone with moderate susceptibility to infection (Dallas and Drake, 2014) for our experiments. Model sensitivity analyses (Supplementary Material) further suggest that our qualitative results are robust to variation in plausible ranges of host infection parameters.

Epidemiological model To examine the impact of a competitor on susceptible host infection dynamics, we used a two-host compartmental model, where the susceptible host species may be uninfected (S) or infected (I) by an environmentally transmitted fungal pathogen (with free-living spore population size P). This susceptible host species competes for resources (R) with a non-susceptible competitor (C). The model was formulated to correspond directly to the experimental treatments, allowing for the testing of model predictions with experimental data. The demographic and epidemic dynamics are described by the following system of differential equations:

$$\dot{S} = e_S f_S(R) R(S + I\phi) - \mu_S S - u f_S(R) SP \quad (1)$$

$$\dot{I} = uf_S(R)SP - \mu_I I \tag{2}$$

$$\dot{P} = \theta\mu_I I - \mu_P P - z_S f_S(R)(S+I)P - z_C f_C(R)CP \tag{3}$$

$$\dot{R} = \pi - \mu_R R - f_S(R)(S+I)R - f_C(R)CR \tag{4}$$

$$\dot{C} = e_C f_C(R)RC - \mu_C C \tag{5}$$

Susceptible (S) and competitor (C) populations grow proportionally to the rate at which individuals can acquire ($f_j(R)$, $j = S, C$) and assimilate (e_j, $j = S, C$) resources, die at rate μ_S (susceptible) or μ_C (competitor), and become infected at a rate determined by their filtering rate ($f_j(R)$, $j = S, C$) and a per spore infectivity parameter (u). Filtering rates depend on the density of algal resources (R) (Hall et al., 2010; Cáceres et al., 2014) and filtering alters the rate of environmental pathogen and resource loss from the system, as well as the transmission of pathogen. Infected (I)

individuals still produce susceptible offspring, but at a rate reduced by ϕ. Infected individuals die at rate $\mu_I > \mu_S$ to account for pathogen-induced mortality (i.e. virulence). Upon death, hosts release a burst of pathogen spores (θ) to the environmental pathogen bank. Environmental pathogen (P) decays at a constant rate μ_P, and is also ingested by susceptible (S), infected (I), and competitor (C) individuals at rates determined by their corresponding filtering rates ($f_j(R)$, $j = S, C$), and a parameter which determines the fraction of spores ingested that are rendered non-infectious after passage through the host gut (z_j, $j = S, C$); this matches observations of spore survival after bluegill feeding (Duffy, 2009), and *Daphnia* hosts exposed to *Pasteuria ramosa*, a bacterial parasite (King et al., 2013). Resource (R) is introduced at a constant rate (π), and decays at a per capita rate μ_R plus additional decay as a function of host foraging ($f_j(R)$, $j = S, C$).

While the exact relationship between algal resource concentration and *Daphnia* filtering rate is unclear, evidence suggests that clearance rate is negatively related to algal resource quantity, such that it is highest when algal resources are limiting (Porter et al., 1982; Hall et al., 2007, 2010; Sarnelle and Wilson, 2008). Therefore, we use a

type II functional response for filtering rates (Eq. 6 and 7). To establish the competitor as dominant, we increased the competitor's assimilation coefficient (e) and maximum filtering rate (f_{C0}) relative to the susceptible host species, reflecting the biology of the system, as the competitor is a larger-bodied grazer with an elevated filtering rate, larger clutch sizes, and faster growth. The equations for host species and competitor filtering rates are provided below, where f_{S0} and f_{C0} are the maximum filtering rates at low resource availability for susceptible and competitor species respectively, and f_{S1} and f_{C1} determine how rapidly their foraging rates decline in response to increasing resource availability.

$$f_S(R) = \frac{f_{S0}}{1 + f_{S1}R} = \frac{0.02}{1 + 4R} \tag{6}$$

$$f_C(R) = \frac{f_{C0}}{1 + f_{C1}R} = \frac{0.025}{1 + 4R} \tag{7}$$

The pathogen basic reproduction number (R_0) is a threshold quantity determining pathogen invasion. We provide it to highlight the effect of the opposing forces of spore removal through foraging (f_S and z_S), and spore creation through pathogen transmission (u) and infected host death (θ). For our system, this can be expressed as

$$R_0 = \frac{\theta u f_S(R_S) S^*}{\mu_P + z_S f_S(R_S) S^*}$$

with the derivation outlined in the Supplementary Materials.

Parameter definitions, units, and details of the parametrization are provided in Table 1; parameter values were obtained largely from the published literature. To account for uncertainty in some parameter estimates, and to investigate the generality of the simulation results, we performed a sensitivity analysis (Supplementary Material). We solved this model numerically for a range of initial competitor densities (0 - 100 hosts L^{-1}). Simulations were initiated with 30 susceptible hosts, no infected hosts, and 10000 pathogen spores. Simulated epidemics were run for 70 days, corresponding to condi-

tions in experimental epidemics. From epidemic simulations, we calculated mean infection prevalence (i.e., average infection prevalence over 70 day time series), and mean susceptible host density as our response variables to changes in competitor density.

Experimental epidemics To test our model predictions, we devised a mesocosm experiment where we manipulated competitor density as a means to modify resource availability, and therefore the effects of competition. Experimental populations were formed by dividing five gallon glass aquaria (16" x 8" x 10") in half, separating the two sides of the aquaria with partitions of 210 μ m Nitex mesh, and filling the tank with 6 L of media; a combination of 2 L filtered pondwater (30 micron filter) and 4 L deionized water. Species were separated by this mesh partition, which allowed for the flow of resources and pathogen spores, but restricted movement of individuals, thereby isolating the effects of resource competition (i.e. exploitative competition) from any direct interaction (i.e. interference competition), and removing any confusion identifying *Daphnia* neonates to species.

Resource competition was produced by altering the density of *D. pulicaria* and restricting algal resources. Every day, each half of experimental mesocosms was fed 1 mL of a

solution of 200 mg freeze-dried, pulverized *Spirulina* sp. suspended in 100 mL deionized water. We fed both partitions of the aquaria the same amount to ensure that resources were well-mixed between halves of each tank, and that the resource concentration throughout the aquaria was approximately 0.67 mg algal dry weight L^{-1}. Five mesocosms were formed for each of three initial *D. pulicaria* densities (0, 30, and 100 individuals L^{-1}) for a total of fifteen aquaria. Populations of *D. dentifera* were established in each of the fifteen experimental aquaria at a density of 30 individuals L^{-1} at the start of the experiment. Competitor densities were chosen based on our susceptible host density, where the 30 individual L^{-1} treatment corresponds to both species starting at equal densities, and the 100 competitors L^{-1} corresponding to a case where the competitor dominates the community. Both sides of the aquaria were inoculated with 10 *Metschnikowia* spores mL^{-1} one day after populations were established.

Mesocosms were sampled every 3-4 days until infection was no longer observed, which was after 70 days. We assessed infection prevalence and host density by stirring tanks and taking a 1 L water sample from each partition of each aquarium. Infection was assessed by visual inspection using a dissecting microscope (10× - 40×) under low light

and keeping hosts in a minimal amount of water to reduce host mortality. Hosts are translucent, and opaque pathogen clusters are present in host heart or gills approximately one week after pathogen transmission. Hosts were returned to their respective aquarium. Sampling with replacement is ideal in this experiment, as spores that infected hosts liberate upon death drive subsequent infections in natural systems; removal of infected individuals would artificially reduce epidemic size or duration.

We analyzed the influence of competition on epidemic dynamics and host density. To examine epidemic dynamics in *D. dentifera* in response to competition with *D. pulicaria*, we calculated two quantities meant to capture aspects of epidemic size and duration: mean infection prevalence, and epidemic duration. Mean infection prevalence was quantified as the fraction of *D. dentifera* infected averaged over the total number of sampling points in which the susceptible host population persisted. Epidemic duration was defined as the number of days epidemics had non-zero prevalence. These measures were compared among initial competitor density treatments using Kruskal-Wallis tests. These tests addressed the influence of competitor density on infection dynamics and epidemic duration. Kruskal-Wallis tests were also used to investigate the relation-

ship between the time until *D. dentifera* population extinction and initial competitor density, which addressed the influence of competitor density on susceptible host demography and extinction dynamics. While it is possible that very small populations would not be detected in our 1 L sample, population extinction was noted only when a sample contained no hosts, and a visual inspection of the tank confirmed no living *D. dentifera* hosts.

Results

Comparison of model and experiments Equilibrium analysis of the model (see Supplementary Material) demonstrated that in the long term, *D. dentifera* would be excluded by *D. pulicaria*, and indeed our experimental populations went extinct within 70 days. Our epidemiological model revealed some outcomes that were not observed in our experimental epidemics. For instance, by examining numerous algal resource input values (Figure 1 and 2), we found that the theoretical hump-shaped relationship between initial competitor density and infection prevalence in the susceptible host species was not strongly influenced by resource availability. Consideration of the pathogen ba-

sic reproductive number in the absence of the competitor suggested that the addition of a competitor could enhance or reduce epidemic risk through antagonistic effects of increasing the filtering rate (and the chance of pathogen exposure) while simultaneously reducing the number of susceptible hosts and infectious propagules. Further exploration of the conditions where competition could reduce or enhance epidemic risk is outlined in the sensitivty analysis section of the Supplementary Materials. Overall, this effort suggested that the hump-shaped relationship between competitor density and infection prevalence observed in both our experimental epidemics and epidemiological model is robust to a range of parameter values. The range of parameters in which competitor density strictly reduces infection prevalence is small, and corresponds to situations in which the competitor digests a much larger proportion of spores than the susceptible host, or when susceptible hosts produce too few infectious spores to result in sustained transmission. Infection prevalence in the susceptible host species increased when competitors were first added to the system until a threshold was reached, and then declined. When resources were less limiting, competitors were able to reduce infection prevalence in the susceptible host more strongly, and mean susceptible host population

sizes were larger (Figure 2).

Competitor density and susceptible host epidemic dynamics Experimental epidemics were qualitatively similar to predictions derived from our epidemiological model (Figure 1), despite independent parameterization of the epidemiological model. The first testable prediction from our theoretical model was that prevalence has a hump-shaped relationship with the initial density of the competitor species. In our experimental epidemics, competitor density had a strong effect on susceptible host species infection dynamics (Figure 3 and 4). Mean infection prevalence (Kruskal-Wallis test; $\chi^2 = 6.74$, df = 2, p = 0.034) and epidemic duration (Kruskal-Wallis test; $\chi^2 = 6.31$, df = 2, p = 0.043) both increased at intermediate levels of competition (30 *Daphnia* L^{-1} for both species). Further, it is interesting to note that at the early stages of epidemics, after *Daphnia* populations were exposed to free-living pathogen spores, infection prevalence increased monotonically with competitor density (Figure 3), suggesting that the competitor presence increased infection prevalence over very short timescales.

Competitor density reduces susceptible host population size Our model predicts that mean susceptible host population size should decline with increasing initial competitor density (Figure 2). In our experiments, epidemics were smaller when *D. pulicaria* densities were at their highest (100 *D. pulicaria* L^{-1}), driven not by the removal of pathogen from the environment, but by the competitive exclusion of the inferior competitor (Figure 4b). The time until *D. dentifera* extinction was reduced by increasing *D. pulicaria* density, though not significantly (Kruskal-Wallis test; $\chi^2 = 4.92$, df = 2, p = 0.085). The resulting termination of epidemics with competitive exclusion is evident when examining the infection time series (Figure 3). However, experimental epidemics also resulted in susceptible host extinction in the absence of competition, a phenomenon not predicted in our model. This is likely an experimental artifact; a result of detriorating water quality, limited food supply, and enhanced mortality as a result of bi-weekly sampling.

Discussion

This study investigates support for the "friendly competition" concept (Hall et al., 2009) over ecologically relevant timescales, using a theoretical model and experiments. We found that when resources are limiting, competition with a superior competitor may be entirely "unfriendly" to susceptible host populations in two different ways: by increasing infection prevalence in susceptible hosts (through increased filtering induced by nutritional stress), and by reducing resources to levels below which susceptible host populations cannot maintain themselves. Prevalence increases through increased pathogen intake occurred at intermediate competitor densities; although prevalence declined at high competitor densities (often interpreted as evidence for friendly competition), the net effect of competition was negative, reducing susceptible host populations towards competitive exclusion. Consistent with classic theory (Gause, 1934), our model predicts that eventual competitive exclusion of the inferior competitor is inevitable, barring niche partitioning or other coexistence mechanisms such as refugia. Overall, our findings suggest that the competitive effects of a dominant competitor are usually negative, and that any positive effect of the competitor removing pathogen

from the environment may be overwhelmed by the effect of reduced resource availability on inferior competitor feeding behavior and persistence.

Our model and experimental design make several simplifying assumptions that could influence competitor effects on infection dynamics in natural systems. For instance, our model allowed filtering rate, which is crucial to pathogen transmission, to vary only with resource quantity while other factors (e.g. pesticides; Fernández-Casalderrey et al. (1994)) may influence foraging ecology and therefore affect transmission independently of resource depletion by competitors. Within hosts, gut residence time may correspond to changes in the probability that a pathogen spore will pierce the gut wall and cause infection. This might explain the findings of previous studies in which both biotic and abiotic stressors decreased filtering rate and also increased pathogen infection success (Day and Kaushik, 1987; Fernández-Casalderrey et al., 1994; Coors and De Meester, 2008; Coors et al., 2008; Jansen et al., 2011). Hosts experiencing stress, either through starvation (Pulkkinen and Ebert, 2004) or from the presence of secondary compounds from competitors, may experience higher transmission success due to the inability to resist pathogen infection (Lafferty and Holt, 2003); in this case, prevalence may con-

tinue to increase at higher competitor densities than predicted by our model. Finally, *Daphnia* feeding selectivity (DeMott, 1982; Knisely and Geller, 1986), and spatial aggregation of pathogen (given that pathogen spores settle quickly after host mortality) may reduce spore encounter rates and host infection independently of resource and competitor density.

Previous studies in the *Daphnia*-microparasite system have suggested that competition with a non-susceptible host should reduce infection prevalence, resulting in so-called "friendly competition" (Hall et al., 2009). Further, Civitello et al. (2013) argued that increasing susceptible host density could inhibit disease spread as a result of pathogen consumption and host foraging interference, suggesting another instance of foraging influencing infection prevalence. Lastly, Cáceres et al. (2014) examined the conditions under which "friendly competition" could result in long term persistence of the inferior competitor, using an epidemiological model nearly identical to ours. We arrived at some conclusions also supported by Cáceres et al. (2014), including the fact that two hosts competing for a limiting resource are unlikely to coexist indefinitely. However, our study also considers the transient dynamics before the susceptible host was

362 excluded. These transient dynamics are ecologically relevant (Hastings, 2004), both
363 to zooplankton specifically and to studies of host-parasite interactions more generally
364 (Dobson, 2004). Zooplankton populations are unlikely to have equilibrium population
365 densities (McCauley and Murdoch, 1987) due to seasonal and stochastic changes in
366 resource availability and environmental conditions, which influence host demographic
367 rates. This means that models examining equilibrium conditions may not correspond
368 to experimental data, making comparisons of models to experiments difficult (Hastings,
369 2004). Our analysis suggests that friendly competition is unlikely to occur over shorter,
370 biologically relevant timescales.

371 There are many ways that parasites can influence interactions between hosts (Hatcher
372 et al., 2012). Many studies focus on how a parasite can handicap the superior com-
373 petitor, leading to parasite-mediated coexistence (Freeland, 1983; Schall, 1992; Hatcher
374 et al., 2006; Schmitz and Nudds, 1994). However, these studies typically do not con-
375 sider how pathogen uptake is influenced by changes to foraging rates due to basal re-
376 source availability. Our study suggests that competition-mediated foraging rates could
377 increase prevalence in a pathogen-susceptible, superior resource competitor, reducing

its abundance relative to the inferior competitor below that expected when pathogen transmission is assumed to be independent of resource availability. Further theoretical and empirical work in this area is warranted.

Given its importance for transmission potential of zoonoses such as Lyme Disease (Ostfeld and Keesing, 2012), there has been much recent interest in the role of host diversity in either diluting or amplifying pathogen transmission (Civitello et al., 2015). Many studies of diversity-disease relationships tend not to incorporate ecological interactions, most notably competition for basal resources. In simple systems where a host and non-host diluter acquire environmental pathogen stages, prevalence is predicted to decline monotonically with non-host density. Our results show that at least initially, intermediate diluter density maximizes epidemic size by increasing host acquisition of the pathogen. The situation in which the pathogen can become a food resource introduces complexity into the study of infectious disease in ecological communities, but is not specific to our study system. Many pathogens are environmentally transmitted, and are subject to incidental predation by hosts, and non-hosts alike (Thieltges et al., 2008; Parker et al., 2010). Therefore, our results suggest that evidence for dilution or

394 amplification may be influenced by the time scale of observation, resource availability,
395 as well as the relative abundance or richness of lower-competency hosts.

396 Much like Strauss et al. (2015), this study attempts to unify two concepts in disease
397 ecology by relating dilution theory to parasite-mediated competition. We highlight the
398 importance of ecological context (resource availability) to competitive interactions be-
399 tween hosts, and how this influences infection dynamics in the susceptible host through
400 a mechanism related to host foraging ecology. Studies of diversity-disease relationships
401 and parasite-mediated competition often do not incorporate the potentially strong ef-
402 fect of environmental regulation, specifically with regards to resource availability. The
403 incorporation of resource-mediated species interactions (direct and indirect) into stud-
404 ies of diversity-disease relationships may yield a more mechanistic view of diversity-
405 disease relationships and other areas of disease ecology.

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Supplementary Materials

Appendix A: Equilibrium analysis, non-dimensionalization, and sensitivity analysis of epidemiological model

Tables

Table 1: Parameters, definitions, and units used in our epidemiological model. We chose plausible estimates for parameters for which data were not readily available (denoted by an empty citation column). Our values of assimilation efficacy were estimated using information on *Daphnia* population growth rates (Civitello et al., 2013; Smith, 1963).

Variable	Units	Definition	Value	Citation
e_S	–	Assimilation efficiency (Susceptible)	26	
e_C	–	Assimilation efficiency (Competitor)	28	
μ_S	day^{-1}	Death rate (Susceptible)	0.10	1
μ_I	day^{-1}	Death rate (Infected)	0.15	2
μ_C	day^{-1}	Death rate (Competitor)	0.10	
μ_P	day^{-1}	Death rate (Pathogen)	0.25	3
μ_R	day^{-1}	Death rate (Resource)	0.25	
z_S	–	Fraction spores digested (Susceptible)	0.30	
z_C	–	Fraction spores digested (Competitor)	0.30	
ϕ	–	Fecundity reduction by infection	0.75	2
f_{S0}, f_{S1}	ml day^{-1}	Host filtering rate (Susceptible)	0.020, 4	4, 5
f_{C0}, f_{C1}	ml day^{-1}	Host filtering rate (Competitor)	0.025, 4	4, 5
u	–	Per spore infectivity	2.03 x 10^{-4}	4
θ	#	Mean spore load per infected host	2 x 10^4	3
π	mg L^{-1} day^{-1}	Resource supply rate	0.005 – 4	6

1: Stich and Maier (2007); 2: Duffy and Hall (2008); 3: Dallas and Drake (2014);

4: Hall et al. (2010); 5: DeMott (1982); 6: Tessier and Woodruff (2002)

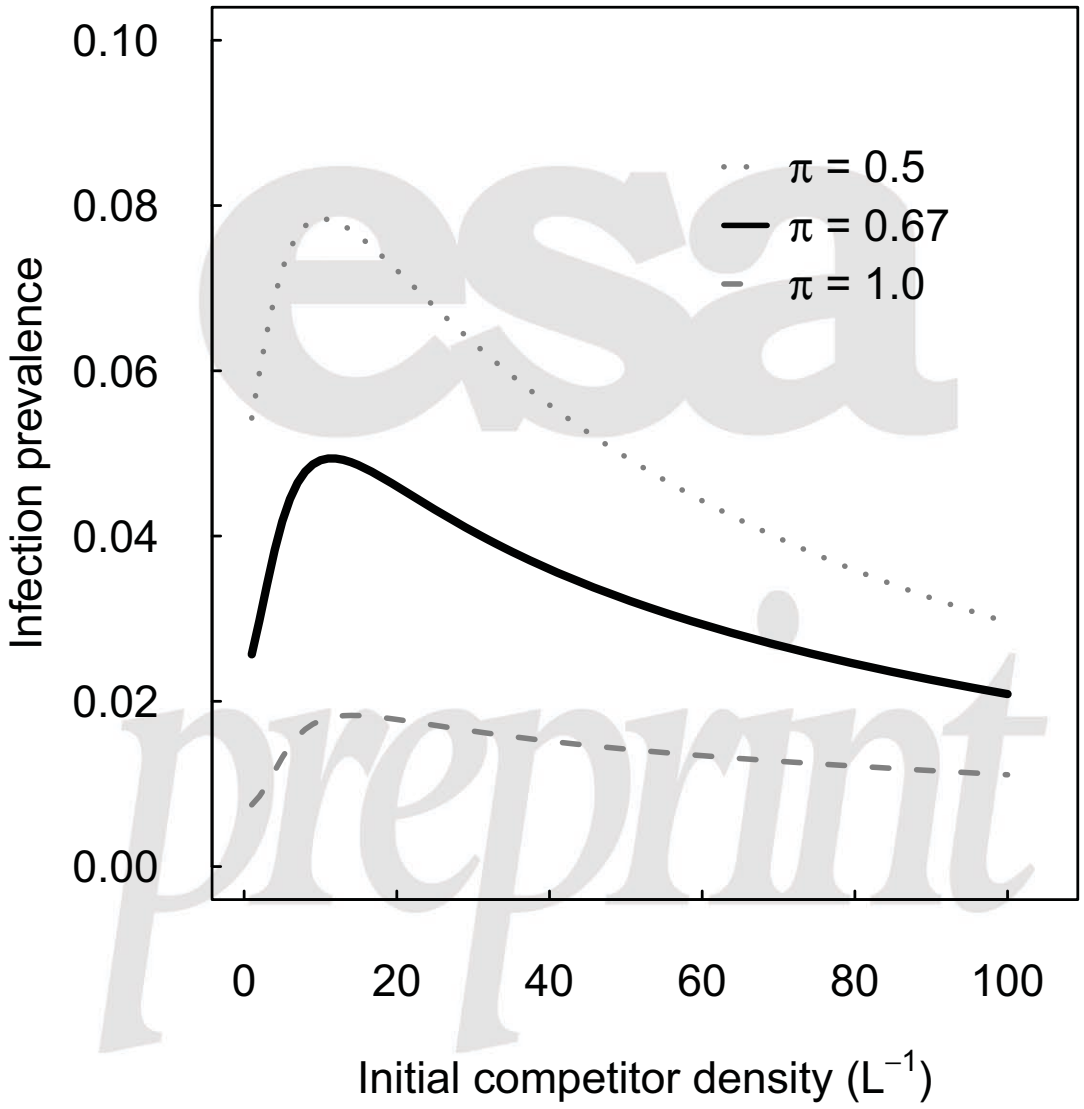
Figures

Figure 1: Non-monotonic relationship between mean infection prevalence and initial competitor density (x-axis) for three potential algal resource input levels (π; y-axis). Enhanced resource input rates reduce infection prevalence by decreasing filtering rates (and hence pathogen exposure). The reduction in infection prevalence at larger initial competitor densities is a result of reduced susceptible host population sizes, and not a positive effect of the competitor removing environmental pathogen.

Figure 2: Mean population size of the susceptible host species ($S + I$) as a function of initial competitor density for three different algal resource input levels. $\pi = 0.5$ and $\pi = 1$ are the lower and upper dashed lines, respectively, and the solid black line corresponds to $\pi = 0.67$, the rate of algal resource supply in our experimental epidemics.

Figure 3: Mean infection prevalence (with standard error bars) for the epidemic time series. The inset barplot compares the infection prevalence at day 10, which corresponds to the first wave of infection, as the pathogen typically takes between 7 and 12 days to be readily identifiable, suggesting that competition intitially increased infection prevalence proportional to *D. pulicaria* density.

Figure 4: Experimental epidemics at three competitor densities revealed that intermediate levels of competition significantly increased mean infection prevalence (a) and epidemic duration (c)). There was no difference in epidemic measures between no competitor and high *D. pulicaria* competitor density treatments, driven by truncated epidemics at high competitor densities as a result of the competitive exclusion of the susceptible host (*D. dentifera*; b).



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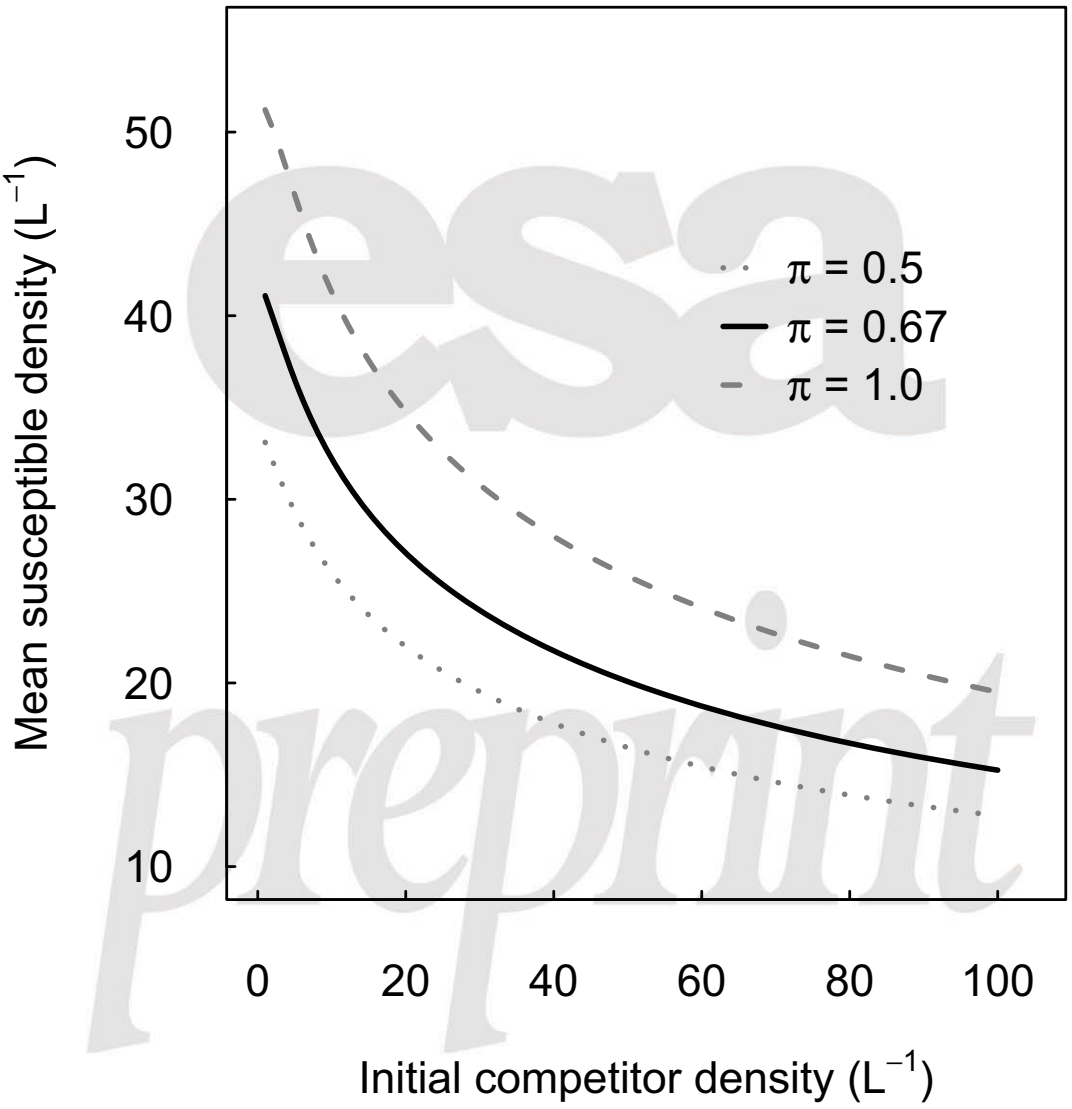
576 Figure 1



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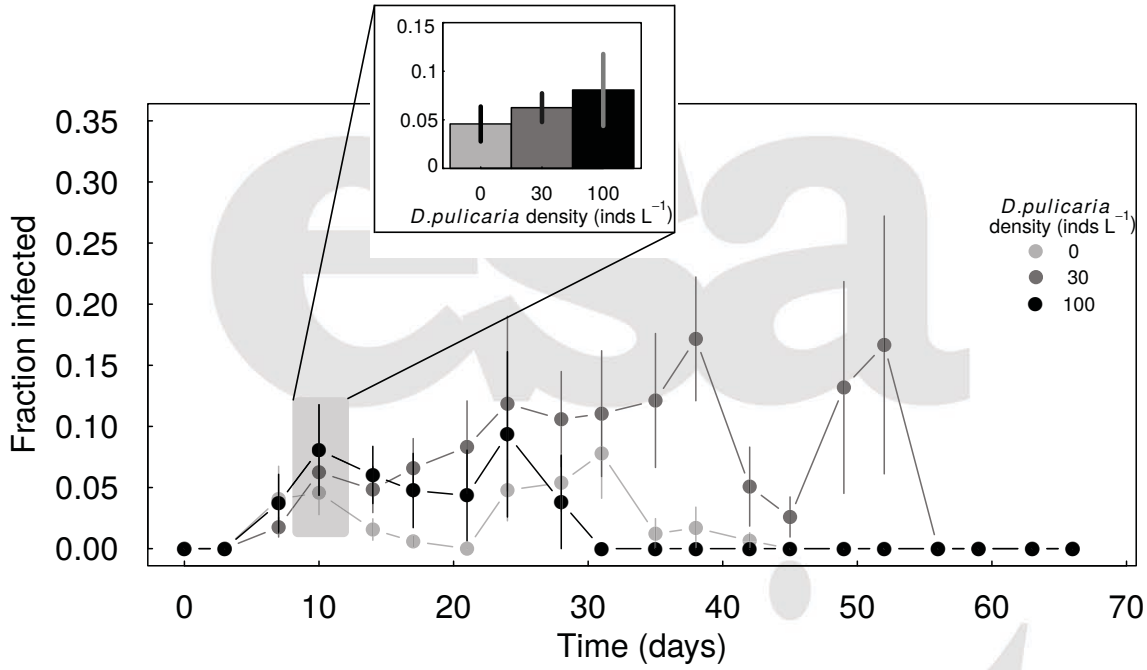

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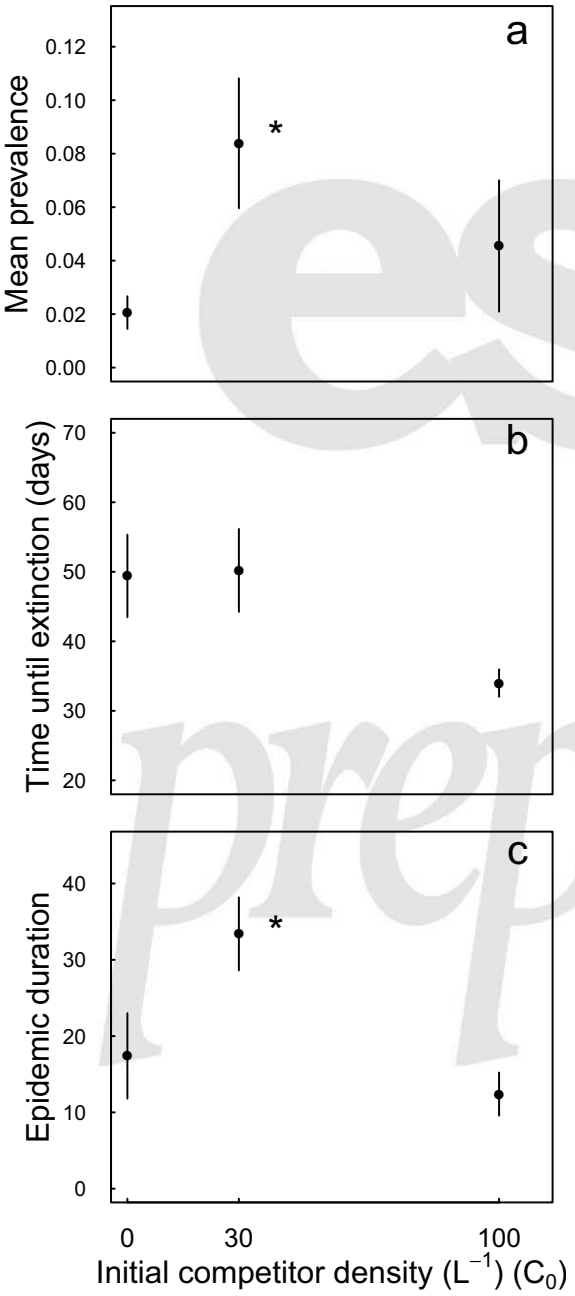
580 Figure 3



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582 Figure 4