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Interactive effects of nutrient enrichment and the manipulation of intermediate hosts by parasites on infection prevalence and food web structure

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ABSTRACT

Parasites with complex life cycles frequently increase their transmission to definitive hosts (where reproduction occurs) by increasing the susceptibility of intermediate hosts to predation by definitive hosts. While recent evidence finds that anthropogenic driven habitat alterations can alter host–parasite relationships, whether such alterations interact with intermediate host manipulation to influence infection prevalence and food web structure remains unknown. We develop a nutrient-limited food web model to investigate how manipulation of intermediate host susceptibility, nutrient supply, and predator diversity determine parasite abundance and infection prevalence in intermediate and definitive hosts. We show that the effects of intermediate host manipulation on parasite abundance and infection prevalence depend on nutrient supply while the coexistence of competing definitive hosts and "dead-ends" (where parasites cannot reproduce) depends primarily on intermediate host susceptibility to predation. Our results suggest that anthropogenic changes in nutrient supply will interact with host–parasite relationships to determine parasite abundance, infection prevalence, and food web structure.

1. Introduction

Parasitic and infectious diseases kill more humans worldwide than any other health risk (WHO, 2004) and pose a major threat to wild and domestic animals (Daszak et al., 2000). Current anthropogenic habitat alterations can alter the relationships between hosts and parasites and influence infection rates of both humans and wildlife (Daszak et al., 2000; Lafferty and Holt, 2003; McKenzie and Townsend, 2007). While much work has focused on how changes in land use and climate influence host–parasite systems (Daszak et al., 2000; McKenzie and Townsend, 2007; Patz et al., 2004), recent reviews suggest that human driven increases in nutrient supply (e.g., through fertilizer and subsequent runoff, sewage waste) may increase both the prevalence of parasites and the severity of infection (Johnson and Carpenter, 2007; Johnson et al., 2007; Lafferty, 1997; McKenzie and Townsend, 2007; Tylianakis et al., 2008). In the absence of direct tests, though, it is unclear whether such associations reflect a bias caused by the fact that only positive relationships are reported (McKenzie and Townsend, 2007).

To our knowledge, Johnson et al. (2007) provided the first experimental evidence that demonstrates that eutrophication can increase the prevalence of infection. In a system that consisted of a trematode parasite (*Ribeiroia ondatrae*), snails as first hosts, and amphibians as second hosts, they found that increasing resources led to an increase in the infection of amphibian hosts. The increase in infection occurred because algal production increased with resources, leading to an increase in snail production (and the density of infected snails) and per snail production of cercariae, the free swimming forms of the parasite, by infected snails.

R. ondatrae disrupts limb development in amphibians and while it was beyond the scope of their experiment, it is reasonable to assume that the malformation might cause increased predation of the amphibians by birds, the definitive host. Examples of parasites that increase their transmission to definitive hosts by inducing phenotypic changes in intermediate hosts to make them more susceptible to predation can be found in every major taxonomic grouping (Moore, 2002). While this phenomenon has long been studied by parasitologists, comparatively little attention has been paid to how these manipulations influence energy flow in food webs and ecosystem functioning (Lafferty et al., 2008; Lefevre et al., 2009; Loreau et al., 2005; Thomas et al., 2005) although there has been some work on how host manipulation influences food chains. One generality derived from Lotka–Volterra parasite models is that parasite-induced alteration in intermediate host phenotype increases infection prevalence in definitive hosts only up to a

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certain point (Fenton and Rands, 2006; Hadeler and Freedman, 1989; Lafferty, 1992). Specifically, in a predator–prey system with prey as an intermediate host and the predator as a definitive host for a parasite, theory suggests that an increase in the susceptibility of infected prey to predation (relative to uninfected prey) leads to an asymptotic increase in the prevalence of infection in predators, defined as the number of infected predators divided by the total number of predators, and a unimodal relationship in the prevalence of infection in prey. The decline in infection prevalence in prey with increased susceptibility occurs because infected prey are increasingly consumed, making them increasingly rare relative to uninfected prey (Fenton and Rands, 2006; Hadeler and Freedman, 1989; Lafferty, 1992).

The above theoretical studies and the results of Johnson et al. (2007) show that changes in resources and intermediate host susceptibility can affect the abundance of parasites and the infection prevalence in intermediate and definitive hosts. Johnson et al. (2007)'s work also underscores the basic and often overlooked fact that parasitism is a fundamental ecological interaction and offers a common research area to epidemiologists, parasitologists, ecologists, and conservation biologists (Lafferty et al., 2008; Lefevre et al., 2009). Parasites can determine species coexistence (Holt and Pickering, 1985; Lafferty et al., 2008; Price et al., 1988; Thompson et al., 2005; Yan et al., 1998), and can be influenced by the composition of the food web including predator diversity (Keesing et al., 2006; Ostfeld and Keesing, 2000; Stauffer et al., 1997, 2006). Seppala et al. (2008) examined a system where infection of an intermediate host (isopods) by a trophically transmitted parasite (*Acanthocephalus lucii*) leads to greater consumption by both perch and dragonfly larvae. Only perch, however, serve as a definitive host for the parasite; dragonfly larvae are a dead end where the parasite cannot reproduce. Despite the presence of the dead end, increased susceptibility of the isopods to predation by both the definitive host and dead end was still beneficial to the parasite because it ensured that some individuals would make it to the definitive host and reproduce. What remains unknown is whether the presence of the dead end decreased infection prevalence in the intermediate and definitive hosts.

In this study, we develop a nutrient-limited food web model and investigate how predator diversity, nutrient enrichment, and intermediate host modification determine parasite abundance and infection prevalence in intermediate and definitive hosts. Our model food web consists of basal resources (nutrients), vegetation, herbivores that serve as intermediate hosts, and predators that serve as definitive hosts of the parasite; reproduction of the parasite occurs only in the definitive host (Fig. 1). Infection increases the susceptibility of infected prey to predation. We first investigate how predator diversity affects coexistence. Specifically, we add an additional dead end predator that cannot serve as a host for the parasite (Seppala et al., 2008), and evaluate how competition between dead-ends and definitive hosts for infected and uninfected herbivores determines the persistence of both types of predators and parasites. Then, we investigate how enrichment and infected herbivore susceptibility affect infection prevalence and parasite abundance in food webs with and without dead-ends.

2. Methods

2.1. The model

The model is

$$\frac{dR}{dt} = S - R(d + c_R V)$$

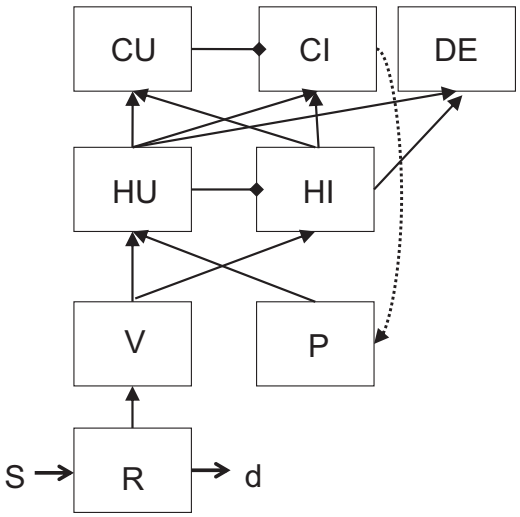

Fig. 1. The structure of the complete food web. Resources (R) enter the system at rate S and leave at rate d. Resources are taken up by vegetation (V) which is consumed by both uninfected and infected herbivores (HU and HI, respectively). Infection of herbivores occurs when they consume parasite propagules (indicated by the solid line with a diamond head between HU and HI). Herbivores are consumed by uninfected and infected definitive hosts (CU and CI, respectively) and dead ends (DE). Infection of the definitive host occurs when an uninfected definitive host consumes an infected herbivore (indicated by the solid line with a diamond head between CU and CI). Parasite reproduction occurs only in infected definitive hosts (indicated by the dotted line).

$$\frac{dV}{dt} = V(c_R e_R R - m_V - c_{HUV} H_U - c_{HIV} H_I)$$

$$\frac{dH_U}{dt} = H_U(c_{HUV} e_{HU} V - c_{CUHU} C_U - c_{CIHU} C_I - c_{DHU} D - c_{HUP} P - m_{HU}) + H_I c_{HIV} e_{HI} V$$

$$\frac{dH_I}{dt} = c_{HUP} H_U P - H_I(c_{CUHI} C_U + c_{CIHI} C_I + c_{DHI} D + m_{HI})$$

$$\frac{dC_U}{dt} = C_U(c_{CUHU} e_{CUHU} H_U - c_{CUHI} H_I - m_{CU}) + C_I(c_{CIHU} e_{CIHU} H_U + c_{CIHI} e_{CIHI} H_I)$$

$$\frac{dC_I}{dt} = c_{CUHI} C_U H_I - C_I m_{CI}$$

$$\frac{dD}{dt} = D(c_{DHU} e_{DHU} H_U + c_{DHI} e_{DHI} H_I - m_D)$$

$$\frac{dP}{dt} = rC_I - P(m_P + c_{HUP} H_U + c_{HIP} H_I)$$

where R is the basal resource (nutrient) pool with supply rate S and loss rate d. V is the population density of the plant. Plants consume resources at rate c_R and convert resources into new individuals at rate e_R. Plants die at rate m_V and are consumed by uninfected herbivores (H_U) at rate c_{HUV} and by infected herbivores (H_I) at rate c_{HIV}. The conversion rate of plants into uninfected herbivores by uninfected herbivores is e_{HU} and by infected herbivores is e_{HI}. Uninfected herbivores die at rate m_{HU}. Uninfected herbivores are consumed by uninfected predators (C_U) at rate c_{CUHU}, by infected predators (C_I) at rate c_{CIHU}, and by predators that cannot function as a final host (dead-ends: D) at rate c_{DHU} (note that the rate of consumption of infected herbivores is similar to α as described by Lafferty (1992) but here we chose to use c_{CUHU}, c_{CIHU}, and c_{DHU} to clearly delineate consumption by definitive hosts and dead ends). All functional responses are linear. We used linear functional responses because they are the simplest response

and allowed for the stable persistence of the entire food web. The main limitations of linear functional responses in our model are that they do not allow for interference or direct interactions between definitive and dead end hosts beyond consumption of herbivores or switching between consumption of uninfected and infected herbivores. Parasite propagules (P) are ingested at rate c_{HUP} and c_{HIP} by uninfected and infected herbivores, respectively. Infection of herbivores is assumed to be instantaneous; uninfected herbivores immediately convert to infected herbivores when parasitized. Infected herbivores are consumed by C_U at rate c_{CUHI}, by C_I at rate c_{CIHI} and by D at rate c_{DHI}, and die at rate m_{HI}. Infection of definitive hosts is also assumed to be instantaneous. The conversion coefficient of uninfected herbivores into newborn uninfected definitive host predators is e_{CUHU}. For infected predators, the conversion coefficients of uninfected herbivores and infected herbivores into newborn uninfected predators are e_{CIHU} and e_{CIHI}, respectively. Uninfected predators die at rate m_{CU} and infected predators die at rate m_{CI}. For dead-ends, the conversion coefficients of uninfected herbivores and infected herbivores into newborn dead-ends are e_{DHU} and e_{DHI}, respectively; dead-ends die at rate m_D. Parasites reproduce only in infected predators at rate r and are lost at rate m_P.

For all the results presented below, $c_R = 0.5$, $d = 0.1$, $e_R = e_{HU} = e_{HI} = 0.1$, $e_{CUHU} = e_{CIHU} = e_{CIHI} = e_{DHU} = e_{DHI} = 0.3$, $r = 0.5$, $c_{HUP} = c_{HIP} = 0.5$, all mortality rates (m values) were 0.05 except for parasite propagules ($m_p = 0.1$). All consumption rates (c values) were 0.3 except when listed differently below. When we varied the susceptibility of infected intermediates to predation by definitive hosts, c_{CUHI} and c_{CIHI} were always equivalent. We investigated many different values for the rates above to see if the model produced different behaviors in different regions of the parameter space. Changing the above rates did not affect the model behavior or our overall results. We use the above rates because they allow clear presentation of our results.

2.2. Parasites, nutrient enrichment, competition, and food web structure

We investigated how competition between definitive hosts and dead-ends for infected and uninfected herbivores determines coexistence by varying the dead-end's ability to consume infected (c_{DHI} between 0 and 1.0) and uninfected herbivores (c_{DHU} between 0 and 1.0) in nine different combinations of nutrient supply and predation rates of infected herbivores by definitive hosts. The nine combinations resulted from the complete cross of three different levels of enrichment ($S = 5$, 10, or 15) with three levels of herbivore susceptibility to predation by definitive hosts ($c_{CUHI} = c_{CIHI} = 0.3$, 0.45, or 0.6). The ability of the definitive host to consume uninfected herbivores was held constant ($c_{CUHU} = c_{CIHU} = 0.3$).

2.3. Effects of nutrient enrichment and intermediate host susceptibility to predation in simple food chains

We investigated how changes in nutrient supply (S) and the susceptibility of infected herbivores to predation influenced the prevalence of infection and abundances in the simple food web that consisted of every component except the dead-ends. We varied S from 4 to 10 and $c_{CUHU} = c_{CIHU}$ from 0.3 to 2.0. We defined the prevalence of infection for herbivores, H_{Inf}, as $H_{inf} = H_I/(H_I + H_U)$ and the prevalence of infection for predators, C_{Inf}, as $C_{inf} = C_I/(C_I + C_U)$.

2.4. Effects of nutrient enrichment and intermediate host susceptibility to predation in food webs with dead-ends

We used the results of the competition models to identify parameters (c_{DHU}, c_{DHI}) that allowed for the persistence of parasites with definitive and dead-end hosts. We then investigated whether dead-end hosts influenced the relationships between enrichment and intermediate host susceptibility found in the absence of dead-ends. The ranges of nutrient supply and host susceptibility were the same as those used in the food web without dead-ends. We investigated several different combinations of dead-end predation rates on infected and uninfected herbivores and found that the patterns were qualitatively identical. We present only one combination here for the sake of brevity ($c_{DHU} = 0.2$, $c_{DHI} = c_{CUHI} + 0.1$).

Our model is a simple representation of a nutrient-limited food web, which makes a number of simplifying assumptions in describing how parasites affect hosts. Although there is a tremendous diversity of ways in which parasites affect hosts, we present our model as a significant starting point in understanding the relationship between nutrient limitation and behavior modification.

We used package ODESOLVE in R version 2.9.2 to implement each numerical simulation for 4999 time steps to remove transient dynamics in our model. We verified that transient dynamics were removed by calculating the temporal standard deviation over the last 500 time steps for each species. We ran a subset of the above models using different initial abundances of each taxon and found that models with the same parameters would converge on the same solution regardless of initial densities, suggesting that our solutions were unique. Temporal standard deviations never exceeded 0.01, indicating that nearly stable conditions were reached. We report the results of our model from the 5000th simulation time step.

3. Results

3.1. Parasites, nutrient enrichment, competition, and food web structure

At all levels of nutrient supply, when dead ends were the better competitor for both uninfected and infected herbivores, the parasite could not persist and food webs consisted of dead ends and uninfected herbivores (upper right quadrat Fig. 2). The ability of uninfected carnivores to consume infected herbivores did not influence this result (the upper right quadrat of all panels in Fig. 2 shows no variation with c_{CUHI}). At the lowest level of nutrient supply, parasites could not persist when dead ends were the better competitor for uninfected herbivores, except when their consumption of infected herbivores was very low (lower right quadrat in $S = 5$). With greater nutrient supply ($S = 10$, 15), parasites could persist when dead ends were the better competitor for uninfected herbivores, but worse for infected herbivores.

At the higher levels of nutrient supply, when the definitive hosts were better competitors for uninfected herbivores than the dead ends, the persistence of the dead end depended on competition for infected herbivores. Dead-ends persisted if their ability to consume infected herbivores offset their disadvantage in ability to consume uninfected herbivores (left two quadrats for $S = 10$ and $S = 15$ panels, Fig. 2).

3.2. Effects of nutrient enrichment and intermediate host susceptibility to predation in simple food webs

The effects of increasing the susceptibility of infected herbivores to predation on parasite abundance and infection prevalence depended on enrichment (Fig. 3). At low levels of nutrient supply, the percent of infected herbivores increased asymptotically. With increasing nutrient supply, the relationship became unimodal. The peak occurred at lower levels of prey susceptibility with increasing nutrient supply and at the highest levels the percent of infected herbivores declined over almost the entire range of susceptibility. At all levels of prey susceptibility, infection prevalence increased

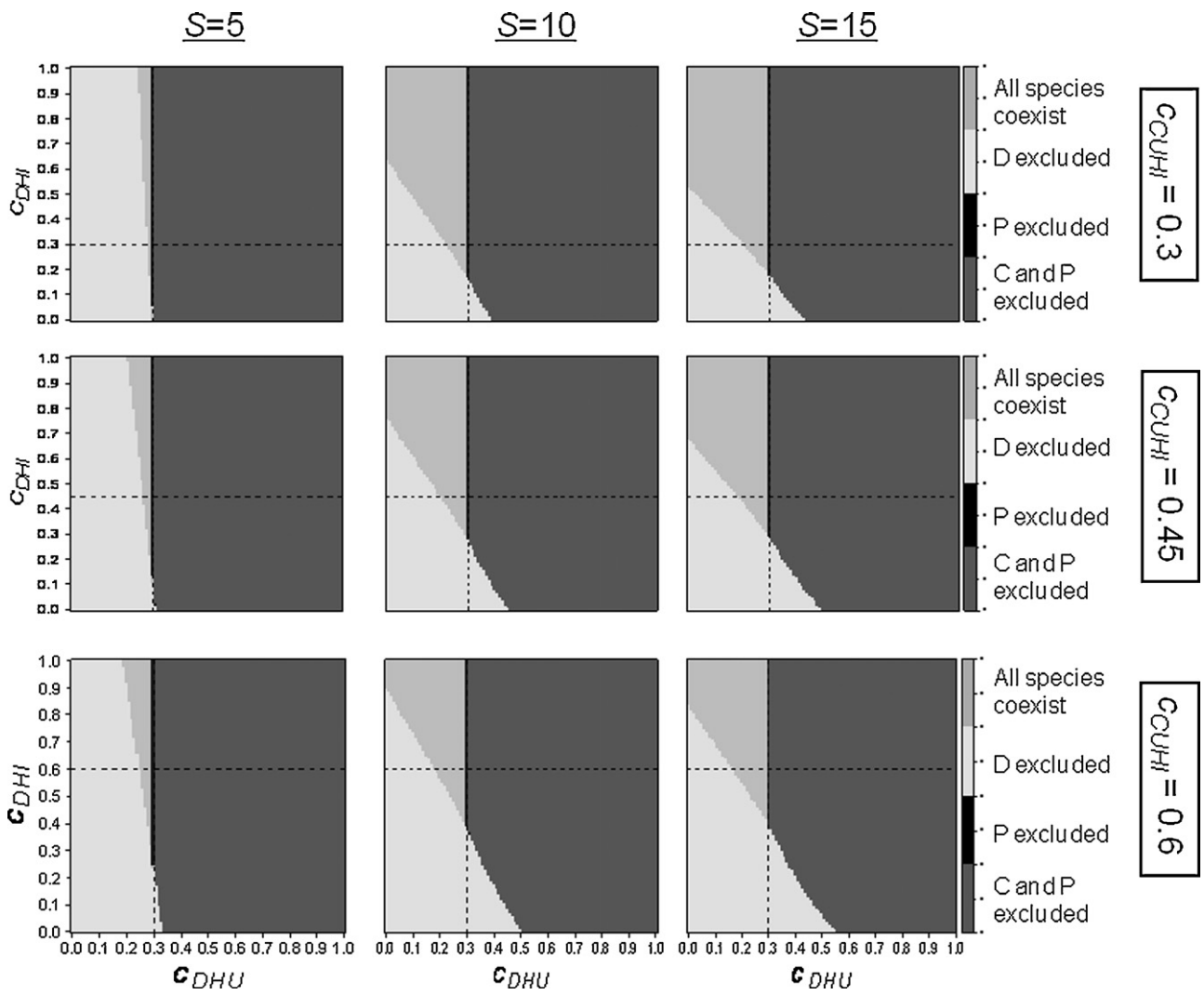

Fig. 2. Outcome of competition between dead-ends and definitive hosts for infected and uninfected herbivores at three levels of nutrient supply (S = 5, 10, or 15) and three levels of susceptibility of infected herbivores to predation by definitive hosts (c_{CUHI} = 0.3, 0.45, and 0.6). Different colors indicate which species coexist. The solid lines in each panel signify equivalent predation rates for dead-ends and definitive hosts on uninfected herbivores (vertical) or infected herbivores (horizontal). Note that the ability of uninfected and infected definitive hosts to consume infected herbivores was the same ($c_{CUHI} = c_{CIHI}$).

with nutrient supply. The increase in infection with nutrient supply was greater at lower levels of susceptibility (Fig. 3A). Infection prevalence in predators showed an asymptotic relationship with both increasing nutrient supply increasing prey susceptibility. The effect of increasing susceptibility on infection prevalence was more pronounced at lower levels of nutrient supply and the effect of increasing nutrient supply was more pronounced at low levels of prey susceptibility (Fig. 3A). Parasites increased with prey susceptibility and increased linearly with increasing nutrient supply (Fig. 3A).

3.3. *Effects of nutrient enrichment and intermediate host susceptibility to predation in food webs with dead-ends*

Dead ends could persist with increasing prey susceptibility and nutrient supply (dead end persistence is indicated by the gray color in Fig. 3B). When dead ends were present, they decreased herbivore infection and parasite abundance. At all levels of nutrient supply, increasing prey susceptibility asymptotically decreased infection prevalence in herbivores. At low levels of nutrient supply, increasing prey susceptibility had little effect on parasite abundance. At high levels of nutrient supply, increasing prey susceptibility slightly increased parasite abundance, but abundances were much less than the abundances of parasites found without dead ends at similar levels of α and nutrient supply. Dead ends slightly decreased infection prevalence in definitive hosts, but had little effect on the relationships between prey susceptibility, nutrient supply and infection prevalence.

4. Discussion

Anthropogenic processes are increasing nutrient availability in many ecosystems (Vitousek et al., 1997) and it is projected that this will drastically modify host–parasite relationships (Lafferty, 1997; McKenzie and Townsend, 2007). Our model suggests that enrichment will affect the structure of food webs with parasites as well as increase infection prevalence and overall parasite abundance. Competitive interactions between definitive hosts and dead-ends primarily depended on the relative abilities of each predator to consume uninfected and infected prey. Our results show in systems with dead ends, parasites cannot persist when dead-ends are present and are better competitors for uninfected intermediate hosts. When parasites can persist with dead-ends (due to the definitive host's ability to consume infected intermediates), their

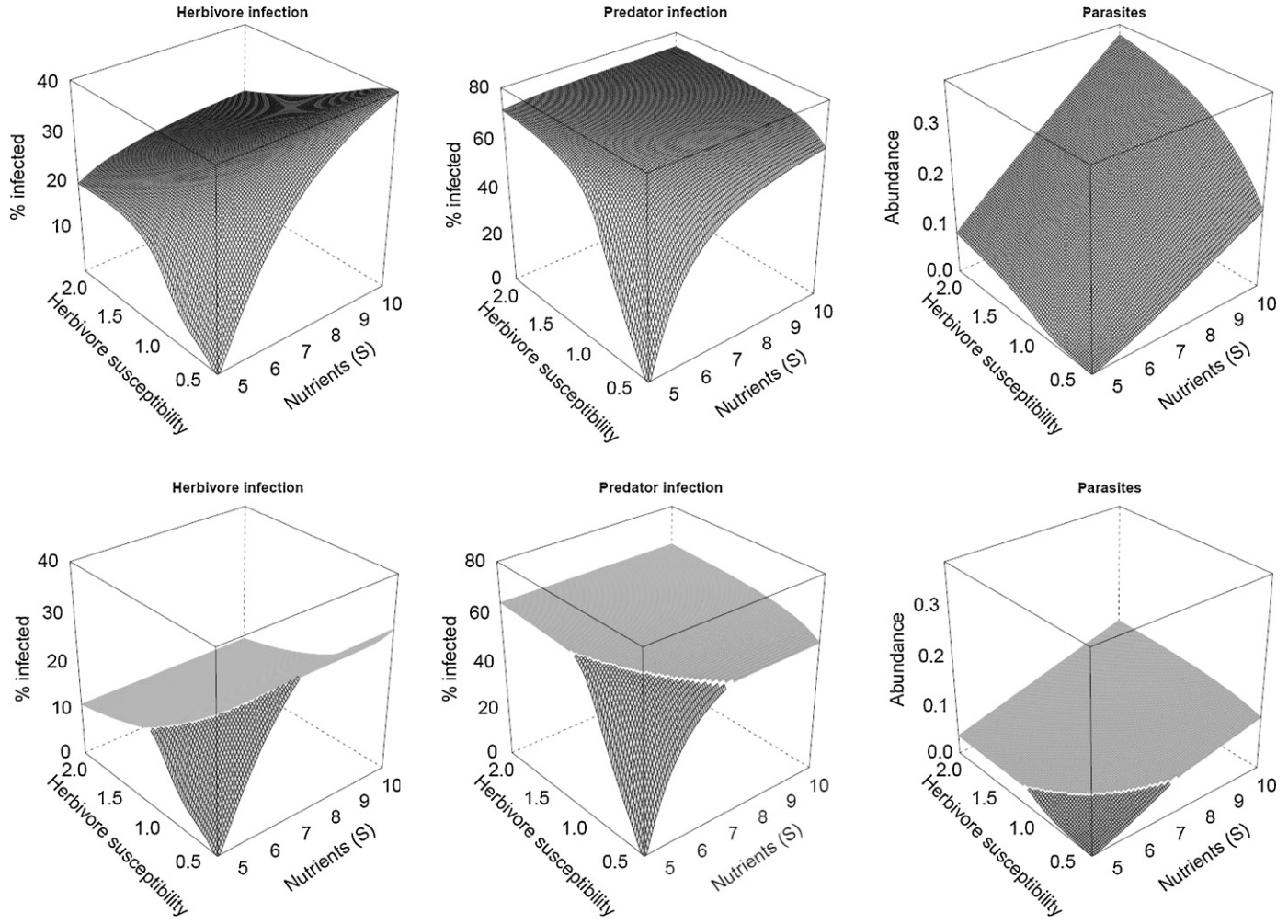

Fig. 3. Effects of nutrient supply (S) and herbivore susceptibility (c_{CUHU} and c_{CUHI}) on infection prevalence (Herb. infection and Pred. infection) and parasite abundance in the simple food webs (A) and with dead ends (Panel B). Gray shading in panel B indicate values of S and herbivore susceptibility that allowed the dead end to persist.

abundance was greatly reduced compared to food webs without dead-ends. Our model suggests that nutrient enrichment influences infection prevalence in intermediate and definitive hosts and parasite abundance non-linearly (Fig. 3).

4.1. Parasites, nutrient enrichment, competition, and food web structure

Previous work found that parasites can mediate the outcome of competition (Holt and Pickering, 1985; Price et al., 1988; Yan et al., 1998). This work, however, relies on infection generating differences in competitive ability among species. Here, we ignore any differences that infection may generate in competitive ability and other measures of performance in the definitive or dead-end hosts; in nature, definitive hosts can show little decline in performance due to parasitic infection (Anderson, 1972; Munger and Karasov, 1991). Our model shows that differences in preferences for uninfected and infected herbivores by definitive hosts and dead-ends in the absence of changes due to infection are sufficient to alter the outcome of competition between the two predators and determine food web structure. The parasite allows dead end and definitive hosts to partition the herbivore resource and specialize on infected and uninfected classes of herbivores and coexistence of dead ends and definitive hosts requires competitive equivalence between the two based on this partition. The better competitor for the uninfected herbivore typically competitively excludes the other predator. It is important to note that we only observed a negative effect on parasite persistence when the dead end was competitively dominant for the uninfected herbivores. Declines in disease persistence with predator diversity may require dead ends for the disease to be the most effective predators on uninfected intermediate hosts.

However, even in regions where the dead-end is a better competitor for uninfected intermediate hosts, parasites can persist if the definitive host consumes enough of the infected prey to compensate for differences in ability to consume uninfected prey. Therefore, the chances of parasite persistence increase when the modification in the susceptibility to predation are aimed at the definitive host. Many parasites are generalists for definitive hosts that are closely related. For a predator to be a dead end, it should not be closely related or forage similar to the definitive hosts. Such predator specific modifications of intermediate prey consumed by taxonomically distant predators occur in natural systems. Cezilly et al. (2000), for example, found that the modification of an intermediate host (*Gammarus pulex*) depended on the definitive host of the parasite. When infection was by an acanthocephalan parasite (*Pomphorhynchus laevis*) with fish as a definitive host, *G. pulex* showed a positive response to light but remained low in the water column. When infection was by a different acanthocephalan (*Polymorphus minutus*) with ducks as a definitive host, *G. pulex* swam higher

in the water column. Thus, each parasite modified their shared intermediate host to increase the probability of transmission to their definitive host while decreasing probability of transmission to dead-ends.

Our results demonstrate that if the dead-end is the more efficient predator for uninfected herbivores and infection increases transmission to dead-ends more than to definitive hosts, then the parasite will ultimately be excluded. This suggests that the harvesting of top trophic levels may increase the abundance of parasites when the harvested species is not a host for the parasite. Stauffer et al. (1997, 2006) provide an example of how top–down control of parasites may have contributed to the exclusion of trematodes in the genus *Schistosoma* that cause urinary schistosomiasis in humans in Lake Malawi in Africa. These trematodes infect snails as intermediate hosts (but do not modify behavior), and ultimately infect humans as definitive hosts. They found that the trematodes were not present in Lake Malawi prior to 1985, but overfishing of snail-eating fish allowed for an increase in snail density and schistosome infections in school-aged children. Further, the recovery of the fish near some villages led to a decline in infection in those villages (Stauffer et al., 1997, 2006).

Our model also demonstrates that an exotic top predator may reduce parasite abundances upon invasion if it is not a host for native parasites (see also Genner et al., 2008; Kopp and Jokela, 2007; Thieltges et al., 2009). Exotic animal species might not serve as definitive hosts for native parasites; exotics acquire only 25% of parasite species in their new range relative to the number of parasites experienced in their native range (Torchin et al., 2003; Torchin and Mitchell, 2004). Given the lack of an evolutionary relationship between exotic predators and native parasites, depression of native parasite abundances by exotic predators may occur frequently.

4.2. Effects of nutrient enrichment and intermediate host susceptibility to predation in simple food webs

Enrichment increased infection in intermediate and final hosts and parasite abundance. This result supports those of Johnson et al. (2007) who found evidence for two complementary mechanisms that led to an increase in transmission of parasites to higher trophic levels (note that in our model parasites have a 2 host life cycle and in Johnson et al. (2007) parasites have a 3 host life cycle). They found increased resources could: (1) increase the population growth rate of intermediate hosts and thus enhance transmission by directly increasing the density of infected intermediate hosts, and (2) increase the per capita production of parasite cercariae within individual intermediate hosts. While our model focuses solely on the first mechanism because parasite production depended only on trophic transmission by intermediate hosts (i.e., there was no loss or growth term for parasites in infected intermediate hosts or free living stages), we also found that nutrient enrichment can increase the trophic transmission of parasites. The experiment conducted by Johnson et al. (2007) varied resources at 2 levels, and therefore could not determine whether increases in infection with enrichment should be linear or more complex. Our model extends their results by showing that the relationship between enrichment and infection prevalence tends to asymptote at higher levels of nutrient supply. Relatively modest increases in nutrients initially led to rapid increases in infection of both intermediate and final hosts (within similar levels of intermediate host susceptibility). However, the prevalence of infection quickly reached a maximum which was relatively unaffected by further increases in resources.

Interestingly, the abundance of parasites increased linearly with enrichment across the entire range of nutrient supply. The lack of direct response of infection prevalence to parasite abundance at higher levels of nutrient supply suggests that regulation of intermediate host density by the definitive host can limit the spread of infection at higher levels of nutrient supply. Previous theory also found that a decrease in the abundance of predators will increase infection prevalence (Ostfeld and Holt, 2004; Packer et al., 2003). However, in these models reproduction of the parasite occurs in the prey. In systems such as the one studied here, infection prevalence reflects a balance between increases due to reproduction in the definitive host and decreases due to the removal of required intermediate hosts by predation. At low levels of nutrient supply, infection prevalence may be limited by the density of definitive hosts. With enrichment, the density of definitive hosts will increase and control the abundance of intermediate hosts, at which point transmission of the parasite to the top trophic level may remain constant despite further increases in nutrient supply.

Similar to previous phenomenological models (Fenton and Rands, 2006; Lafferty, 1992), our mechanistic model found an asymptotic increase in the prevalence of infection in definitive hosts with an increase in the susceptibility of intermediate prey. For infection prevalence in herbivores, our results only partially agreed with previous models. Earlier work found that the prevalence of infection of intermediate hosts initially increased due to increased transmission from prey to predator and subsequent increase in parasite abundance, but eventually decreased due to the increase in removal of infected prey by predation (Fenton and Rands, 2006; Lafferty, 1992). Our mechanistic model showed a unimodal response of intermediate host infection with intermediate host susceptibility only at mid ranges of nutrient supply (Fig. 3A). At low levels of nutrient supply, infection prevalence increased asymptotically and at high resource levels, infection prevalence declined with increased susceptibility to predation. Such changes in the relationship between herbivore modification and infection prevalence with nutrient supply suggest that anthropogenic changes in nutrient supply might have a greater effect on host–parasite dynamics than the biological consequences of infection.

4.3. Effects of nutrient enrichment and intermediate host susceptibility to predation in food webs with dead-ends

Generally, the overall patterns found between infection prevalence and parasite abundance and nutrient enrichment and host susceptibility are similar to those found in the absence of dead-ends. One difference is that parasite abundance was reduced by nearly 50%. The decline in parasite abundance when dead-ends are present is similar to the "dilution effect," where increasing the diversity of predators leads to a decline in the abundance of disease vectors (Keesing et al., 2006; Ostfeld and Keesing, 2000). Here, in a different infection paradigm, we also find that predator diversity may limit infection prevalence in intermediate hosts and parasite abundance. This finding, like the dilution effect, depends not only on differences in the reservoir capabilities of the hosts but also on differences in the competitive abilities of predators. Unlike the dilution effect, though, the decline in parasite abundance with predator diversity did not translate into a difference in the prevalence of infection in definitive hosts, particularly at high levels of nutrient supply. Even though the abundances of definitive hosts declined with the addition of a competitor, the proportion infected remained similar.

In sum, our results add to the growing list of ways in which increased nutrient availability (a precursor to eutrophication) can affect the structure and function of natural and managed ecosystems (Vitousek et al., 1997). We show that increased nutrient availability will modify tight biological relationships between hosts and parasites and increase infection prevalence in definitive hosts. This increase can occur despite the persistence of alternate predators that do not serve as hosts when alternate predators are weaker

competitors suggesting that predator diversity should be conserved as it offers the greatest probability of including effective predators that are not competent hosts for disease (Keesing et al., 2006; Ostfeld and Keesing, 2000). Our results suggest that the conservation of predator diversity will influence the overall health of ecosystems and humans (Keesing et al., 2006; Ostfeld and Keesing, 2000; Stauffer et al., 1997, 2006).

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