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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

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increases infection prevalence in definitive hosts only up to a 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

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

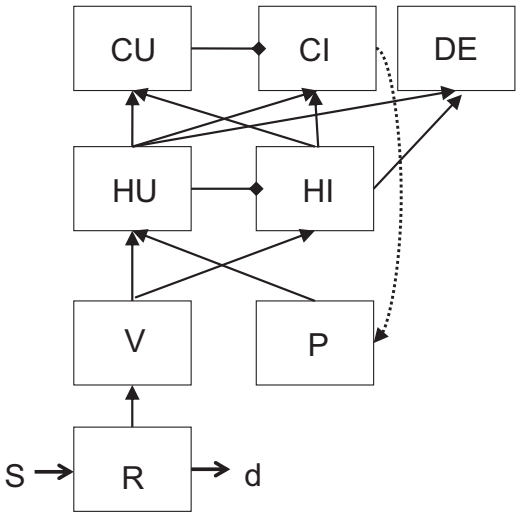

Fig. 1. The structure of the complete food web. Resources (R) enter the system at rate S and leave at rate d. Resources are taken up by vegetation (V) which is consumed by both uninfected and infected herbivores (*HU* and *HI*, respectively). Infection of herbivores occurs when they consume parasite 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