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# How intraguild predation affects the host diversity-disease relationship in a multihost community

Min Su[a,*], Yuanqi Yang[a], Cang Hui[b]

[a] School of Mathematics, Hefei University of Technology, Hefei 230009, China
[b] Centre for Invasion Biology, Department of Mathematical Sciences, Stellenbosch University, and African Institute for Mathematical Sciences, Matieland 7602, South Africa

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ABSTRACT

Broad evidence has shown that host diversity can impede disease invasion and reduce the eventual prevalence, but little is known on how species interactions play in shaping this host diversity-disease relationship. Previous work has illustrated that intraguild predation (IGP), combined with parasite-mediated indirect effects, can have strong influences on parasitic infection. Following this line of thinking, we here examine the role of predatory interactions in the disease transmission within a multihost community. Through varying fractions of IGP in a competitive community, we show that, dependent on the fraction of predatory interactions, species richness can switch from enhancing to inhibiting disease establishment/prevalence. Without IGP interactions, high host species richness can likely weaken the 'dilution effect' and in some cases even enhance the disease establishment (and/or prevalence) due to the existence of alternative sources for infection, whereas IGP can generally heighten the negative diversity-disease relationship due to the reduction of encounter rate between prospective hosts and parasites. Although trait-mediated interactions (captured as the infection-induced changes in predation rate) only weakly affect disease prevalence, density-mediated interactions (captured as the additional infection-induced mortality) can pose a relatively strong influence on disease transmission. Our results thus underline the importance of considering species interactions when investigating the host diversity-disease relationship.



## 1. Introduction

Species interactions can have profound influences on assemblage diversity and composition, thus dictating the disease dynamics of parasitic infection in multihost communities (Frainer et al., 2018; Hui and Richardson, 2017; Johnson et al., 2015; Lafferty et al., 2008; Wilson et al., 1996). Indeed, the risk of an infectious disease can greatly depend on how the multiple host species vary in their competence against the parasitic infection (Civitello et al., 2015; Johnson et al., 2013; Keesing et al., 2010). Recent research echoes that host diversity in an ecological community can generally impede the invasion and transmission of parasites; this supports a negative relationship between biodiversity and disease prevalence attributed to the dilution effect (Johnson et al., 2013; Keesing et al., 2006; Young et al., 2017).

The negative relationship between host diversity and disease risk can be achieved via several mechanisms, such as reducing the encounter rate, regulating susceptible hosts, and interfering with parasite transmission (Keesing et al., 2006; Chen et al., 2018; Chen and Zhou, 2015). In particular, host diversity can inhibit the risk of parasitic infection and suppress parasite prevalence by regulating the abundances of potential host species (Johnson et al., 2015; Keesing et al., 2006; Ostfeld and Keesing, 2000; Young et al., 2017). For example, the diversity of tick hosts can reduce the prevalence of Lyme disease through the dilution effect (LoGiudice et al., 2003; Ostfeld and Keesing, 2000).

However, a few studies have demonstrated a contrasting result that increased host diversity could aggravate disease risk if these added host species function as alternative sources of infection (Dobson, 2004; Holt and Pickering, 1985; Schmidt and Ostfeld, 2001). To this end, the role of host species diversity, whether it dilutes or amplifies disease risk, in parasite-mediated multihost ecosystems remains elusive, especially for zoonotic diseases in humans (Civitello et al., 2013; Keesing et al., 2010).

The effect of biodiversity on disease risk, arguably, depends on both the community/network structure and the traits of hosts and their parasites (Keesing et al., 2006; Mitchell et al., 2002; Roche et al., 2013; Rohr et al., 2015). Generalist parasites can emerge only in some host communities, implying that host community structure may influence parasite emergence and persis-

* Corresponding author.
*E-mail address:* sum04@163.com (M. Su).

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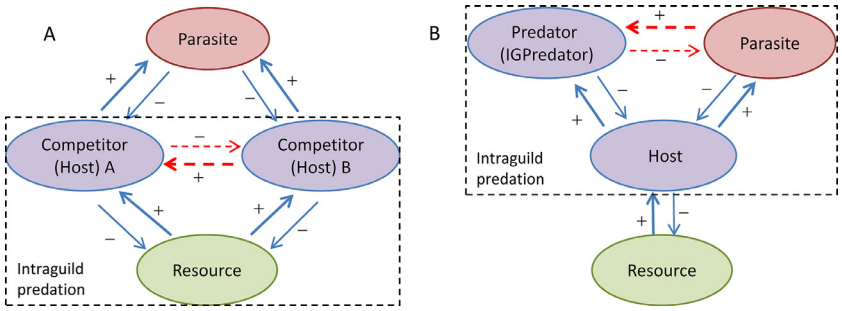


**Fig. 1.** The general modules of intraguild predation (IGP). (A) IGP between a predator and its prey that also shares a common resource; (B) IGP here represents a predator consuming both the parasite and its host population (i.e. infected and susceptible population).

tence (AI-Shorbaji et al., 2017). Evidence is accumulating to show that food-web structures can strongly affect community stability and disease transmission; of particular interest here is the role of predatory interactions that could potentially alter community structures and inhibit parasite transmission (Dobson, 2004; LoGiudice et al., 2003; McQuaid and Britton, 2015; Orlofske et al., 2012; Packer et al., 2003; Rohr et al., 2015; Su et al., 2009).

Adding cross-guild predatory interactions can drastically affect disease invasion and the eventual prevalence (Hudson et al., 1992; Packer et al., 2003; Rohr et al., 2015; Su et al., 2015). Intraguild predation (IGP) frequently occurs in natural communities (Arim and Marquet, 2004; Brodeur and Rosenheim, 2000; Holt and Polis, 1997); in many cases, intraguild predators and prey are closely related so that they may share the same parasites (Hatcher et al., 2006; Rohr et al., 2015; Sieber and Hilker, 2011). Using a two hosts-parasite model to explore the effect of IGP on disease dilution, Rohr et al. (2015) have shown that IGP can weaken the dilution effect, while hosts that do not engage IGP can reduce parasitic infection. However, how IGP interactions in a multihost community (host species engaging both predation and competition with other host species) affect disease prevalence and the host diversity-disease relationship, is yet to be investigated.

Parasite presence can indirectly modify the strength of predator-prey interactions through changing the life-history and behavioral strategies/traits of predators (Dunn et al., 2012; Hatcher et al., 2006; Raffel et al., 2010; Su and Boots, 2017). Such indirect effects induced by parasitism are composed of both density-mediated components (resulting from parasite-induced reduction in host reproduction and survival) and trait-mediated components (resulting from parasite-induced changes in host life history strategies and traits) (Dunn et al., 2012; Hatcher et al., 2014). Growing studies indicate that parasites can substantially alter host population dynamics and IGP community structures through these indirect effects (Hatcher et al., 2014). Consequently, exploring the impact of IGP mediated by indirect effects on the disease dynamics in a multiple host community can be insightful to understanding the relationship between host diversity and disease risk.

Taken together, we are still yet to elucidate: (i) the effect of intraguild predation on the well-established relationship between host diversity and disease prevalence; (ii) the parasite-induced indirect effects (i.e., density- and trait-mediated components) on the potential 'dilution' of disease risk in a multihost community. To this end, we here constructed a mathematical model for depicting disease dynamics in a multihost IGP community (Fig. 1). We first introduced IGP into a multiple host community with competition interactions between hosts, and then investigated the effect of IGP on the relationship between host diversity and disease prevalence. Following the common practice of network ecology that often investigates how the proportion of different types of biotic interactions affects network function (McQuaid and Britton, 2015; Mougi and Kondoh, 2012), we explored the impact of interaction complexity on disease prevalence by varying the proportion of IGP interactions that can cause trait-versus density-mediated indirect effects. In particular, following McQuaid and Britton (2015), we computed disease prevalence and community structures (captured by species abundances, community stability and diversity) at the long-term equilibrium for simulated communities (Appendix S1). We further calculated the threshold of disease invasion, represented by the basic reproduction number ($R_0$), using the next-generation matrix under different levels of host species diversity, and compared disease transmission in systems with versus without IGP (Appendix S2). As will be shown below, we have demonstrated that the relationship between host diversity and disease invasion/prevalence depends profoundly on the presence of IGP in multihost communities.

## 2. Material and methods

We first extended the two-host/one-parasite model of Hatcher et al. (2014) into a model describing a multihost community facing parasitic infection, and then explored the effect of adding IGP on disease transmission by varying the proportion of IGP interactions in the community. The model for this multihost IGP community reads:

$$\begin{aligned}\frac{dS_i}{dt} &= \underbrace{H_i\left(r_i-\delta_i H_i-\sum_{j=1,j\neq i}^{N}a_{ij}H_j\right)}_{density-dependent\ \ recruitment}-\underbrace{\sum_{j=1}^{N}\beta_{ij}S_iI_j}_{infection\ \ rate}\\ &-\underbrace{\sum_{j=1,j\neq i}^{N}\gamma_{ij}S_iS_j+\sum_{j=1,j\neq i}^{N}e\gamma_{ji}S_iS_j}_{natural\ \ predation\ \ rate}-\underbrace{kS_iH_i+ekH_i^2}_{cannibalism}\\ &-\underbrace{\sum_{j=1,j\neq i}^{N}\rho_j\gamma_{ij}S_iI_j+\sum_{j=1,j\neq i}^{N}ev_j\gamma_{ji}S_iI_j+\sum_{j=1,j\neq i}^{N}e\rho_i\gamma_{ji}I_iH_j}_{infection-induced\ \ predationrate}\end{aligned}\tag{2.1}$$

$$\begin{aligned}\frac{dI_i}{dt} &= \underbrace{\sum_{j=1}^{N}\beta_{ij}S_iI_j}_{infection\ \ rate}-\underbrace{\sum_{j=1,j\neq i}^{N}v_i\gamma_{ij}I_iS_j-\sum_{j=1,j\neq i}^{N}\rho_j\gamma_{ij}I_iI_j}_{infection-induced\ \ predation\ \ rate}\\ &-\underbrace{\Omega_iI_i}_{infection-induced\ \ mortality}-\underbrace{kI_iH_i}_{cannibalism}\end{aligned}\tag{2.2}$$

where $S_i, I_i, H_i (i = 1, \cdots, N)$ represent the abundance of susceptible, infected, and total host population, respectively, with $H_i = S_i + I_i$, and $N$ the number of host species (i.e. host diversity). Parameter $r_i$ is the intrinsic reproduction rate of species $i$ (randomly chosen from [0,1]); $\delta_i$ density-dependent self-regulation (randomly

chosen from [0,1]); $a_{ij}$ the competition coefficient between host $i$ and $j$; $k$ the rate of cannibalism. Note, we only presented results for $k = 0$ in the main text; additional results for systems with cannibalism ($k > 0$) largely conformed to the results without cannibalism and were thus only presented in the Supporting Information (Appendix S2, S3).

Previous studies have shown that the Who Acquires Infection From Whom (WAIFW) matrix is useful to describe parasite transmission in multihost community (Dobson, 1995, 2004; McCallum et al., 2001; McQuaid and Britton, 2015). Density-dependent transmission has already been incorporated into the WAIFW matrix which fits well with empirical data in some natural systems, such as *Pasteurella muris* epidemics in laboratory mice (Anderson and May, 1979; de Jong et al., 1995) and the epidemics of brucellosis in the bison (Dobson and Meagher, 1996). Note, we here only assumed, in the WAIFW matrix, horizontal transmission of parasitic infection (i.e. all newborns are susceptible) and also that the transmission follows a density-dependent form, with $\beta_{ij}$ representing the infection rate of transmitting parasites from host $j$ to $i$. We further assumed a lower inter-specific transmission rate than the intra-specific transmission rate, i.e., $\beta_{ii} > \beta_{ij}$, $i \neq j$ (Holt et al., 2003; Keesing et al., 2006; Rudolf and Antonovics, 2005).

In this model, $\gamma_{ij}$ represents the rate of intraguild predation of host $j$ on $i$. For mathematical tractability, we chose a linear functional response, which is nevertheless a reasonable assumption for freshwater amphipods (Dick et al., 1993). Parameter $e$ represents the conversion rate, $e \in [0, 1]$, and for simplicity we fixed the parameter $e = 0.3$ for all host species (Dick et al., 1993; Hatcher et al., 2008, 2014). Note, additional tests show that different conversion rates only quantitatively affect disease prevalence, with a higher rate of conversion resulting in slightly lower disease prevalence and higher diversity (Appendix S4). We further divided predation into two components: natural predation between susceptible hosts, and infection-mediated predation with at least one host species involved in the predation interaction also infected (reflecting the reduced appetite of infected predators $\rho_i \in [0, 1]$ and the increased vulnerability of infected prey $v_i \in [1, 2]$; Hatcher et al., 2014). Thus, $\rho_i\gamma_{ij}$ and $v_i\gamma_{ij}$ represent the attack rate of an infected predator on susceptible (and infected) prey and that of a susceptible predator on infected prey, respectively. Note, the infection-mediated predation captures infection-induced changes in predation behavior and will be hereafter referred as the trait-mediated effect of infection. Parameter $\Omega_i$ represents infection-induced mortality, and hereafter will be referred as the density-mediated effect of infection.

We generated the network structure of this multihost community from a cascade model. In particular, for each pair of host species $i, j = 1, \ldots, N$ with $i < j$, species $i$ cannot consume species $j$, whereas species $j$ may consume species $i$ with a probability $P{=}2CN/(N-1)$, where $C$ is the connectance of host network (Chen and Cohen, 2001). Because interaction strengths of each resource normally decrease with increasing diversity of resources due to foraging effort allocation (McQuaid and Britton, 2015; Mougi and Kondoh, 2012), we also set the interaction coefficients of species competition being inversely proportional to the number of competing species,

$$a_{ij} \propto A_{ij} / \sum_{k \in C_i, k \neq i} A_{ik},$$

where $A_{ij}$ is the potential competition ability of species $j$ on $i$, chosen from $U(0, 1)$, and $C_i$ is the set of all competitors for species $i$.

As IGP depicts the predation interaction between species also competing for other resources (Arim and Marquet, 2004; Holt and Polis, 1997; Hatcher et al., 2014; Sieber and Hilker, 2011), we here used $p_{\mathrm{IGP}}$ to denote the proportion of competition link between two host species that also engage in IGP (an antagonistic link) between the same pair of species (Fig. 1A). By varying the proportion $p_{\mathrm{IGP}}$ we could demonstrate the potential effect of IGP on the dynamics of parasitic infection. We generated the predation rate $\gamma_{ij}$ between two competitors using a similar approach,

$$\gamma_{ij} \propto \Gamma_{ij} / \sum_{k \in R_j, k \neq j} \Gamma_{kj},$$

where $\Gamma_{ij}$ is the potential preferenceof species $j$ on $i$, chosen from $U(0, 1)$, and $R_j$ is the set of all potential prey for predator species $j$.

Similarly, we assumed that the infection rate of a parasite species on each host species is inversely proportional to the number of host species it can infect,

$$\beta_{ij} \propto B_{ij} / \sum_{k \in P_j} B_{kj},$$

where $B_{ij}$ is the potential parasite transmission from host species $j$ to $i$, chosen from $U(0, 1)$ with $\beta_{ii}$ the maximum of $\beta_{ij} (j = 1, \cdots, N)$. $P_j$ is set of all potential hosts that host $j$ can transmit parasites; we assumed that the parasite is a generalist and can exploit all available host species.

The spectral radius of the next-generation matrix is a reliable estimator of the basic reproduction number (Hurford et al., 2010; Xue and Scoglio, 2013). For simplicity, only these model compartments that correspond to the already and prospective infected species were considered (Xue and Scoglio, 2013). The original ordinary differential Eq. (2.2) was decomposed into two column vectors $\mathcal{F} = (\mathcal{F}_i)$ and $\mathcal{V} = (\mathcal{V}_i)$, where $\mathcal{F}_i$ is the $i$th row of $\mathcal{F}$ representing the rate at which new infections appear in species $i$, and $\mathcal{V}_i$ is the $i$th row of $\mathcal{V}$ representing the rate at which infected host species $i$ declines from predation and infection-induced mortality. The Jacobian matrix of $F$ denotes transmission, and the Jacobian matrix of $V$ denotes transition:

$$F = \left[\partial \mathcal{F}_i(x^0)/\partial I_j\right] \text{and} V = \left[\partial \mathcal{V}_i(x^0)/\partial I_j\right], \tag{2.3}$$

where $x^0 = [S_1^0, S_2^0, \ldots, S_N^0]^T$ represents the disease free equilibrium, and $I_j$ is the abundance of infected individuals of species $j$, $j = 1, 2, ..., N$. Therefore, we can obtain the basic reproduction number for parasite transmission, $R_0 = \rho(FV^{-1})$, as the spectral radius of matrix $FV^{-1}$ (Appendix S2).

Community diversity is measured here using Hill numbers (Chao et al., 2014; Hill, 1973; Hui et al., 2018), defined as

$${}^qD = \left(\sum_{i=1}^{N} p_i^q\right)^{1/(1-q)}, \tag{2.4}$$

where $p_i (i = 1, \cdots, N)$ is the proportion of individuals of the $i$th host species in the entire community and $q$ is the sensitivity parameter. For $q = 0$, the diversity index represents the number of persistent species in community after transient dynamics. $q = 1$ represents the exponential of Shannon diversity index, and $q = 2$ can yield the Simpson diversity index. Using these Hill numbers, we can explore how host diversity in the community is affected by the initial proportion of IGP.

With such complex interactions in an ecological community, stability is not always ensured. However, with the complex community Jacobian, we have to resort to numerical simulations for stability analyses to avoid mathematical intractability (Appendix S1). These numerical simulations have nevertheless revealed that the probability of an assembled community being locally unstable is surprisingly low (Fig. 2), confirming the proposition of previous studies (Gibbs et al., 2018; Serván et al., 2018). To this end, we decided to only report results from 500 stable but randomly assembled communities; in particular, on how the community structures at the asymptotically stable equilibrium, in terms of basic reproduction number, disease prevalence, and community diversity, respond to changes in the level of intraguild predation.

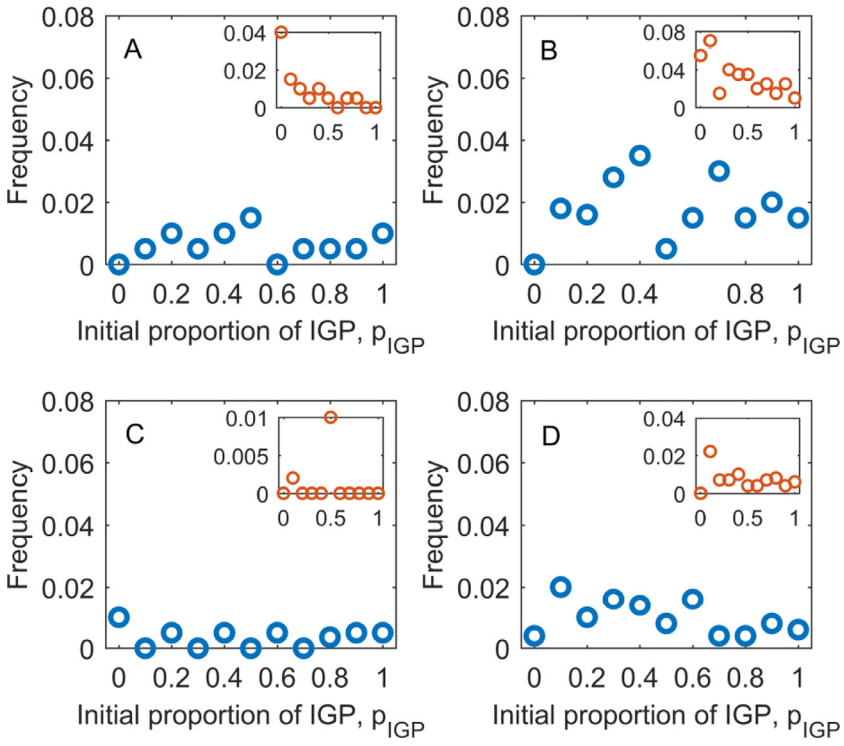


**Fig. 2.** The frequency of unstable dynamics in 500 simulated communities with a given of IGP interactions. (A) no-infection effect, (B) trait-mediated effect (infection-induced changes in predation), (C) density-mediated effect (infection-induced mortality), (D) concurrence of density- and trait-mediated effects. The blue circles represent system without cannibalism, $k = 0$ and red are cannibalism, $k = 0.01$. Parameters are $N = 50$, $C = 0.2$, $e = 0.3$. Other parameters $r_i$, $\delta_i$, $\rho_i$, $\Omega_i$ are chosen from $U(0, 1)$; $v_i$ from $U(1, 2)$.

## 3. Results

Results from the multihost IGP communities displayed a negative relationship between host diversity and disease prevalence, similar to the pattern found in reported empirical cases (Civitello et al., 2015; Johnson et al., 2013). In the presence of IGP, i.e., when $p_{IGP} > 0$, host species diversity can dilute disease risk among multiple host species, regardless of how infection has changed the predation behavior and/or induced mortality, leading to the obvious decline in total parasite prevalence with increasing host species richness (Fig. 3; Fig. S1). This could be explained by that there can be an even greater increment in the total host abundance than the increment in the total abundance of infected hosts, when the community harbors more host species (Appendix S5).

However, in the absence of IGP, i.e., when $p_{IGP} = 0$, with increasing host richness, the infection remained at full prevalence in systems without cannibalism ($k = 0$; Fig. 3A, B), although it dropped slightly in systems with cannibalism ($k > 0$; Fig. S1A, B). To describe the relationship between host richness and disease prevalence with $p_{IGP} = 0$ clearly, we designed a standard statistical analysis using the Akaike information criterion (AIC) and root mean square error (RMSE) to indicate the goodness of fit. According to the categories of simulated results (Fig. 3), we chose the monotonic and unimodal functions for fitting. Both fit models verified the disease prevalence with no density-mediated effect can keep invariant respect to species richness (Table 1). Interestingly, the prevalence of infection can even increase with increasing species richness (for low host richness communities) if there is infection induced additional mortality (Fig. 3C, D; Fig. S1C, D). Statistical analysis also showed that the unimodal polynomial (low AIC/RMSE) can have higher goodness of fit than the logarithmic model (high AIC/RMSE), confirming that a positive relationship between disease prevalence and host richness, albeit rare, can emerge in species-poor communities that face lethal diseases but also lack intraguild predation (Table 1). A negative relationship can be expected in all other cases.

**Table 1**
The parameters of fit models for Fig. 3 and model examination without IGP.

| Categories | $a$ | $b$ | $c$ | RMSE | AIC | Case |
|---|---|---|---|---|---|---|
| $y = a + b\log x$ | 1 | 0 | – | 0 | $-\infty$ | 1 |
| | 1 | 0 | – | 0 | $-\infty$ | 2 |
| | 0.5580 | 0.0026 | – | 0.0001 | −75.70 | 3 |
| | 0.5636 | −0.0032 | – | 0.0005 | −79.14 | 4 |
| $y = ax^2 + bx + c$ | 0.00000 | −0.0000 | 1.0000 | 0.0000 | −712.24 | 1 |
| | 0.00000 | −0.0000 | 1.0000 | 0.0000 | −712.24 | 2 |
| | −0.00002 | 0.0024 | 0.5245 | 0.0000 | −91.02 | 3 |
| | −0.00001 | 0.0012 | 0.5348 | 0.0000 | −84.27 | 4 |

Notes: a, b, c are model parameters. Cases 1 to 4 represent no-infection effect, trait-mediated, density-mediated and concurrence of density- and trait-mediated effects, respectively.

We further displayed the impact of IGP on the basic reproduction number, $R_0$, calculated as the spectral radius of the next-generation matrix ($\rho(FV^{-1})$). As infection-induced mortality (i.e., $\Omega_i > 0$) is necessary for calculating $V^{-1}$ (Appendix S2), we here only calculated the basic reproduction number accordingly (Fig. 4; Fig. S2, S10). With the presence of IGP ($p_{IGP} > 0$), $R_0$ declined with host species richness, suggesting that host diversity can impede the invasion of parasites in the multihost IGP community (Fig. 4; Fig. S2, S10). However, when IGP is absent ($p_{IGP} = 0$), host diversity can stimulate parasite invasion as shown by the increase of $R_0$ with species richness (Fig. 4; Fig. S2, S10). As such, we can conclude that, if infection is density mediated (either through infection-induced mortality or the concurrence of infection-induced changes in mortality and predation) IGP communities with rich host species can impede disease invasion and suppress disease prevalence; in

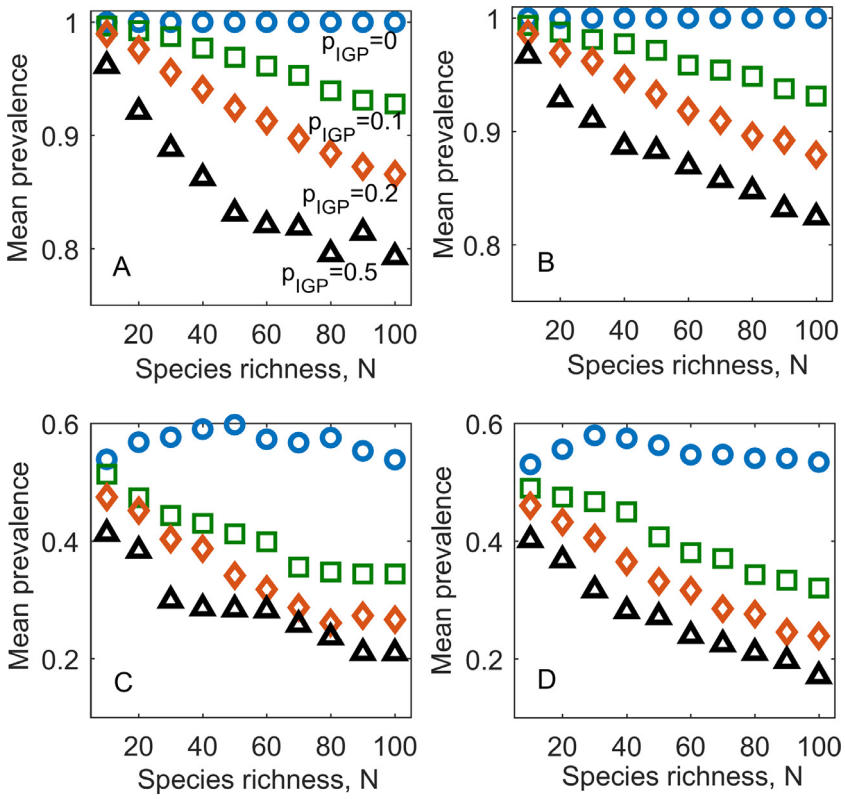


**Fig. 3.** Relationships between the mean disease prevalence and host species richness, evaluated at different proportions of IGP interactions for (A) no-infection effect, (B) trait-mediated effect, (C) density-mediated effect, (D) concurrence of density- and trait-mediated effects, respectively. Parameter $k = 0$ (without cannibalism) and other parameters are the same with Fig. 2.

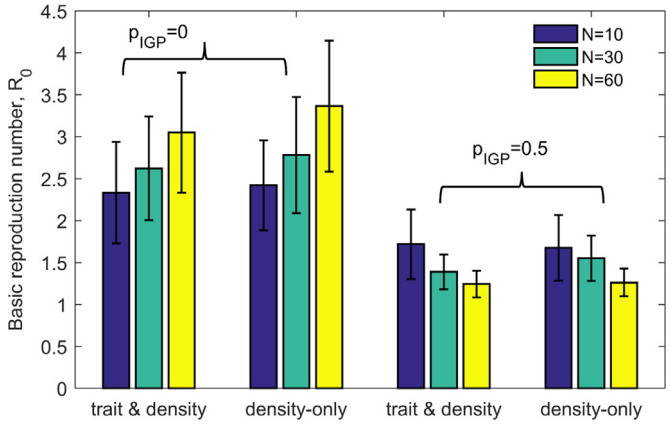


**Fig. 4.** Basic reproduction number, $R_0$, for with ($p_{IGP} = 0.5$) versus without IGP ($p_{IGP} = 0$) in the model considering density-mediated effect (denoted by 'density-only') or the concurrence of density- and trait-mediated effects (denoted by 'trait & density'), respectively. Error bars represent the standard deviation from 500 simulations. $N = 50$ and other parameters are the same as Fig. 3.

contrast, species-rich communities without IGP can facilitate disease invasion.

We further illustrated how changing the proportion of IGP interactions can affect the disease prevalence in the multihost community. Generally, increasing the proportion of IGP interactions will reduce the prevalence of parasitic infection, regardless whether there are infection-induced indirect effects (Fig. 5). This confirms that IGP in competitive communities can impede disease prevalence. Interestingly, comparing disease prevalence affected by infection-induced predation with the one without such indirect effect, we found such changes in infection-induced predation can aggravate disease prevalence (comparing Fig. 5A to B). Meanwhile, additional mortality for infected hosts can clearly reduce disease prevalence, for cases with or without infection-induced changes in predation (Fig. 5). Fig. 5 also indicated that the effect of IGP in reducing disease prevalence eventually levels off when the proportion of IGP becomes too high, especially if there is also infection-induced additional mortality (Fig. 5C, D). Although considering cannibalism cannot alter the results qualitatively, it can impede the disease prevalence quantitatively (compare results of $k = 0$ with $k$= 0.01 in Fig. 5), which because the offspring from cannibalism by infected hosts contributes to the susceptible class.

Importantly, the proportion of IGP can drastically affect the eventual diversity of the multihost communities (Fig. 6; Fig. S3). Although host diversity can become slightly higher when considering infection-induced changes in predation, the effect of increasing IGP proportion is clear–host diversity measured by Hill numbers of three different $q$ values declined monotonically with the increase of IGP proportion $p_{IGP}$ (Fig. 6A, B; Fig. S3A, B). However, when considering infection-induced morality, increasing the IGP proportion can have a more complicated effect on the resultant host diversity (Fig. 6C, D; Fig. S3C, D). Specifically, the diversity of communities with a small proportion of IGP interactions can increase with the IGP proportion but not for communities with a large proportion of IGP interactions. For evidence, we here chose $q = 1$ as an example to model examination (Table 2). The statistical analysis showed that quadratic polynomial (low AIC) provided a better fit to the diversity curve than the logarithmic model (high AIC) as considering infection-induced morality, but an inverse result was predicted with no infection-induced mortality (Table 2), confirming the prediction of scatter diagrams. The initial increase of host diversity when the IGP proportion was low could be due to that the low-level of predation and the infection-induced mortality have effectively controlled the abundance of infected hosts and in a net term

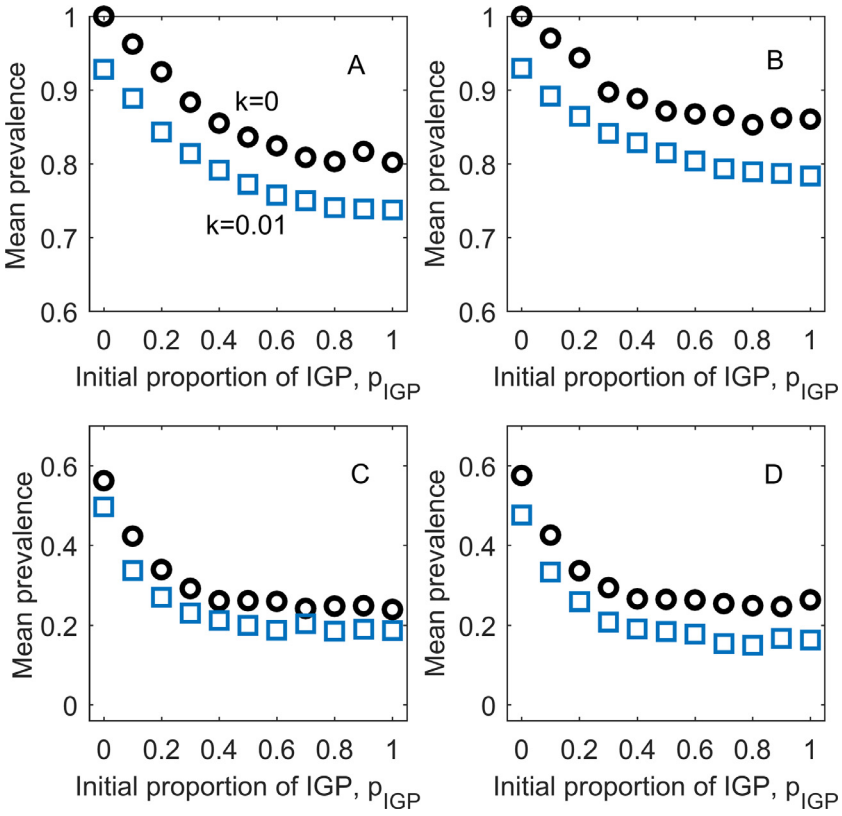


**Fig. 5.** Mean disease prevalence as a function of the initial proportion of IGP interactions for (A) no-infection effect, (B) trait-mediated effect, (C) density-mediated effect, (D) concurrence of density- and trait-mediated effects, respectively. Parameters are the same as Fig. 2.

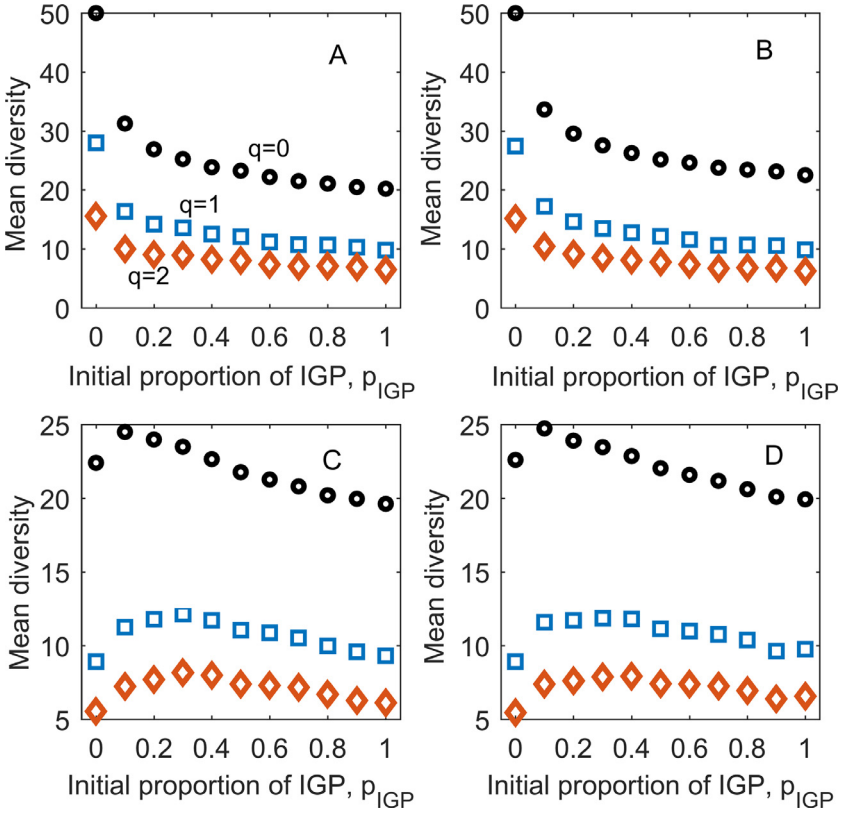


**Fig. 6.** Community diversity, ${}^{q}D(q = 0, 1, 2)$, as a function of the initial proportion of IGP interactions for (A) no-infection effect, (B) trait-mediated effect, (C) density-mediated effect, (D) concurrence of density- and trait-mediated effects, respectively. In each simulation, the species is considered extinct when its population size is below $10^{-4}$. $N = 50$ and other parameters are the same as Fig. 3.

**Table 2**
The parameters of fit models for Fig. 6 and model examination with $q = 1$.

| Categories | $a$ | $b$ | $c$ | RMSE | AIC | Case |
|---|---|---|---|---|---|---|
| $y = a + b\log x$ | 9.9217 | −2.7919 | – | 0.0000 | −31.84 | 1 |
| | 9.9085 | −3.0753 | – | 0.0000 | −27.82 | 2 |
| | 10.0519 | −0.9794 | – | 0.0000 | −5.65 | 3 |
| | 10.2617 | −0.8896 | – | 0.0000 | −10.81 | 4 |
| $y = ax^2 + bx + c$ | 7.0172 | −14.2026 | 17.2431 | 0.0000 | −20.72 | 1 |
| | 8.9714 | −16.8748 | 18.1717 | 0.0000 | −13.63 | 2 |
| | −3.9725 | 1.4771 | 11.5448 | 0.0000 | −20.47 | 3 |
| | −2.8699 | 0.6267 | 11.7266 | 0.0000 | −26.58 | 4 |

Notes: a, b, c are model parameters. Cases 1 to 4 represent no-infection effect, trait-mediated, density-mediated and concurrence of density- and trait-mediated effects, respectively.

have boosted host abundances and diversity. At the high-level of predation, the negative effect of predation on host abundances and diversity has exceeded its role to suppress infection spreading.

## 4. Discussion

Ecological communities with a rich host diversity can generally impede the invasion and transmission of parasites, leading to a negative relationship between host species richness and parasite risk due to the dilution effect (Dunn et al., 2012; Lafferty et al., 2008; Wilson et al., 1996; Young et al., 2017; Woolhouse et al., 2001). Although many studies have explored the effect of host diversity on disease risks, to our knowledge, very few has focused on role of interaction complexity in shaping the diversity-disease relationship. This is albeit strong evidence showing that the complexity of species interactions can indeed pose profound influences on community composition and stability (Duffy et al., 2007; Hui and Richardson, 2019; Landi et al., 2018; McQuaid and Britton, 2015; Mougi and Kondoh, 2012). We here highlighted the joint effects of host richness and the complexity of IGP interactions that can mediate infection-induced indirect effects on the disease risk in a multihost community. The complexity of species interactions, measured by the various levels of IGP interactions, has been shown to influence the relationship between host species richness and disease prevalence.

The introduction of IGP predators to a host community with shared parasitic diseases can increase the complexity of biotic interactions and alter host community structures (Duffy et al., 2007; Melián et al., 2009; Mitchell et al., 2002; Selakovic et al., 2014). Previous works on parasitic infection in multihost communities have shown that host community structures can profoundly affect the establishment and prevalence of infectious diseases in humans and animals (Hatcher et al., 2006; Holt et al., 2003; Mitchell et al., 2002; Roche et al., 2013). Here, we demonstrated that the predatory interaction can alter the diversity-disease relationship, with a hump shaped diversity-disease relationship emerged in systems without IGP if infection can cause additional mortality (Fig. 3C, D). For a specific parasite, the strength of inter-specific competition declines inversely with species richness, and consequently with more species the total host abundance and the source of infection increase (Fig. S9). This explains the firstly positive relationship between host diversity and disease prevalence in such competitive species-poor communities. In a host species-rich community, the risk of disease invasion is diluted among the host species, leading to a negative diversity-disease relationship (Fig. 3C, D). However, in a community mixed with IGP interactions, hosts that are strongly affected by infection will also suffer strongly (as easy prey) from predation, creating a sink for disease transmission (Johnson and Thieltges, 2010).

Whether host richness is an enhancer or inhibitor for disease establishment depends largely on the presence of predatory interactions (Fig. 4). We noted that the invasion threshold of the disease as described by the basic reproduction number ($R_0$) enhanced with increasing host richness but only for purely resource competition communities; in contrast, it declined with host richness when hosts were also engaging predatory interactions (Fig. 4; Fig. S10). The result also indicated that for communities only with competitive interactions, the addition of host species creates alternative sources for infection but reduces the fraction of susceptible hosts, consequently increasing the invasion risk of infectious diseases. Therefore, our results further emphasized that both the diversity-disease relationship and the disease establishment strongly depend on how the hosts interact with each other in the ecological community.

Many studies have shown that increase of predator abundance or attack rate in infection systems can be an effective mechanism for controlling infectious disease dynamics, and the removal of predatory interactions could increase the prevalence of parasitic infection and as a result weaken ecosystem functioning (Al-Shorbaji et al., 2017; Hudson et al., 1992; Packer et al., 2003; Raffel et al., 2010; Roy and Holt, 2008; Su and Hui, 2011). Here, we demonstrated that the more complex biotic interactions induced by the higher fraction of IGP interactions in a competitive community could result in lowering the prevalence of parasites (Fig. 5) and reducing host diversity when there are either no infection-mediated indirect effects or changes in predatory behavior (Fig. 6A, B). The result confirmed that predatory interactions can effectively reduce the prevalence of parasites and the diversity of host species, both of which can benefit the control of infectious diseases (Packer et al., 2003; Roy and Holt, 2008; Su and Hui, 2011).

IGP interactions can mediate many infection-induced indirect effects in natural systems (Brodeur and Rosenheim, 2000; Dunn et al., 2012; Hatcher et al., 2014; Holt and Polis, 1997; Rohr et al., 2015; Su et al., 2009). For example, Rohr et al. (2015) have demonstrated that IGP predators can increase infection risks, while non-IGP predators reduce the risk; this suggests that IGP can weaken the dilution effect and amplify parasitic loads (Fig. 1B). Our model has a different structure from this one (comparing Fig. 1A to B), and our results thus emphasize the role of interaction complexity in effectively controlling the disease prevalence. That is, the presence of IGP can strengthen the dilution effect, while the absence of IGP can weaken the dilution effect and even to a level that amplifies disease risk from increasing host diversity. Community/network structure and its complexity are thus the essential ingredients to consider when exploring the relationship between host richness and disease prevalence.

Infection induced changes in predation behavior and mortality can affect the coexistence of IGP hosts (Dunn et al., 2012; Hatcher et al., 2006). Indirect effects of parasitic infection are important, at a range of biological scales, to determining invasion success and species persistence (Dunn et al., 2012). Consistent with previous studies, our results also emphasized that indirect effects of parasitism can strongly affect disease transmission, where infection-induced mortality plays a more important role than infection-induced changes in predation behavior (Fig. 5) or host diversity (Fig. 6). Although trait-mediated effects such as infection-induced changes in predation behavior can increase disease prevalence by weakening the predatory interactions, density-mediated effects such as infection-induced mortality can reduce disease prevalence through directly depleting infected hosts.

Taken together, interaction complexity in a community can alter its response to disease invasion and transmission, which then affects the resulted diversity-disease relationship. Real ecosystems are complex, and to render analytic tractability we have ignored many biological processes that could potentially affect disease

risks. For example, density-dependent transmission of parasites can potentially generate a positive diversity-disease relationship; in contrast, frequent-dependent transmission could possibly sustain a high host diversity that suppresses the threshold of disease invasion, with both cases potentially applicable in a WAIFW matrix (Keesing et al., 2006; McCallum et al., 2001). Similarly, although the assumption of the linear functional response in feeding behavior is typical for filter feeders, but the feeding rate of insects and vertebrates often saturates (e.g., Type 2 or 3 Holling functional response; Hall et al., 2005); how the saturation in feeding rates affects the relationship between host richness and disease prevalence in multihost communities needs to be further investigated. In addition, theoretical models mostly focus on equal disease resistance among hosts, not considering host-specific susceptibility. Nonetheless, most have supported a negative relationship between host richness and parasite prevalence (Young et al., 2017). Clearly, whether host diversity can possibly dilute or amplify disease risks warrants further investigation, especially given the complex reality of multihost communities (Fontaine et al., 2011). Considering these complexities in future studies could be important to provide better understandings of the relationship between host diversity and disease prevalence in ecosystems.

## CRediT authorship contribution statement

**Min Su:** Conceptualization, Methodology, Writing - original draft, Writing - review & editing. **Yuanqi Yang:** Software, Methodology. **Cang Hui:** Methodology, Writing - review & editing.

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## Supplementary materials

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