Interspecific Contact and Competition May Affect the Strength and Direction of Disease-Diversity Relationships for Directly Transmitted Microparasites

Suzanne M. O'Regan,[1,2,*] John E. Vinson,[1] and Andrew W. Park[1,3]

1. Odum School of Ecology, University of Georgia, Athens, Georgia 30602; 2. National Institute for Mathematical and Biological Synthesis, University of Tennessee, Knoxville, Tennessee 37996; 3. Department of Infectious Diseases, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602

Submitted July 22, 2014; Accepted June 3, 2015; Electronically published September 4, 2015

Online enhancements: appendix, scripts.

abstract: The frequency of opportunities for transmission is key to the severity of directly transmitted disease outbreaks in multihost communities. Transmission opportunities for generalist microparasites often arise from competitive and trophic interactions. Additionally, contact heterogeneities within and between species either hinder or promote transmission. General theory incorporating competition and contact heterogeneities for disease-diversity relationships is underdeveloped. Here, we present a formal framework to explore disease-diversity relationships for directly transmitted parasites that infect multiple host species, including influenza viruses, rabies virus, distemper viruses, and hantaviruses. We explicitly include host regulation via intra- and interspecific competition, where the latter can be dependent on or independent of interspecific contact rates (covering resource utilization overlap, habitat selection preferences, and temporal niche partitioning). We examine how these factors interact with frequency- and density-dependent transmission along with traits of the hosts in the assemblage, culminating in the derivation of a relationship describing the propensity for parasite fitness to decrease in species assemblages relative to that in single-host species. This relationship reveals that increases in biodiversity do not necessarily suppress frequency-dependent parasite transmission and that regulation of hosts via interspecific competition does not always lead to a reduction in parasite fitness. Our approach explicitly shows that species identity and ecological interactions between hosts together determine microparasite transmission outcomes in multispecies communities.

Keywords: biodiversity, dilution effect, disease ecology, epidemiology, host-parasite interactions, transmission.

Introduction

Directly transmitted microparasites often infect multiple host species, and consequently the ecology of species interactions is crucial for determining parasite establishment and persistence in multihost communities. The frequency and magnitude of disease outbreaks is often determined by the rate of transmission opportunities in the assemblage. Cross-species contact—achieved, for example, through feeding on or utilizing a common resource—may facilitate parasite spillover from one host species to another. Alternatively, ecological interactions that regulate susceptible host species, such as intra- and interspecific competition, can control or possibly promote parasite transmission in multi-host-species communities. For example, reduced host species abundances resulting from interspecific competition may lead to a decreased number of cross-species transmission events. Additionally, community structures that result in contact heterogeneities within and between species will affect disease outcomes (Dalziel et al. 2014). Given that interspecific competition and contact may be either correlated or independent of one another according to community contexts (fig. 1), it is unclear under what conditions competitive interactions and contact between multiple hosts combine to enhance or, alternatively, reduce the risk of outbreaks of microparasitic infections in multispecies communities.

To quantify how community structure and ecological context, including competition and contact heterogeneities, may affect outbreak tendency, it is possible to calculate the fitness of a parasite in a community of host species. Parasite fitness is typically measured by the basic reproduction number R_0, the average number of secondary infections caused by one infectious individual (Anderson and May 1991). Although this was originally defined to describe transmission in a susceptible single-species host population, the basic reproduction number of the parasite in a multihost community can be readily derived through calculation of the dominant eigenvalue of the next-generation matrix obtained from linearizing the infected-host subsystem at the disease-free equilibrium (Diekmann et al.

* Corresponding author; e-mail: soregan@nimbios.org.

Am. Nat. 2015. Vol. 186, pp. 480–494.
DOI: 10.1086/682721

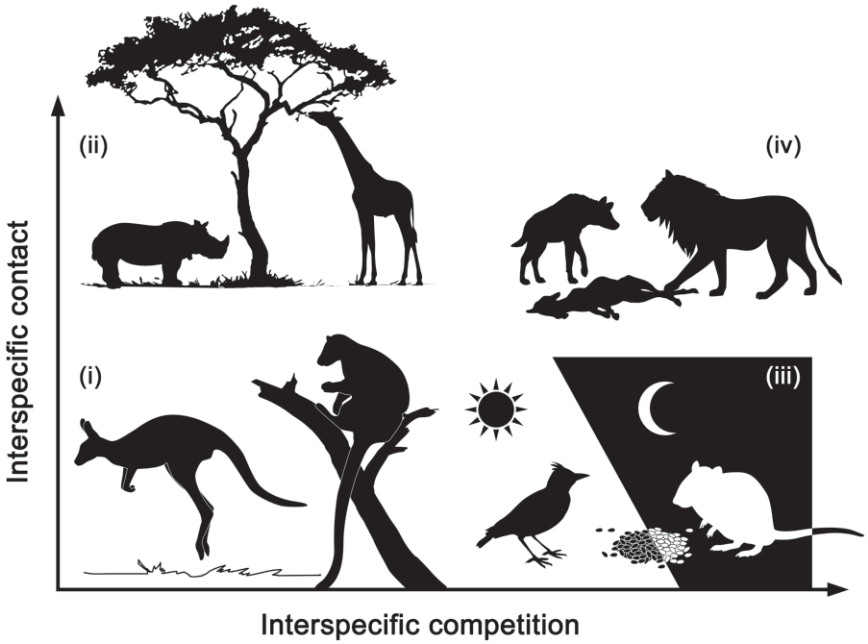

Figure 1: Ecological contexts encompassing competition and interspecific contact structure that may affect microparasite transmission in multispecies communities. Four separate cases are considered: (i) low interspecific contact and weak interspecific competition, (ii) high interspecific contact and weak interspecific competition, (iii) low interspecific contact and strong interspecific competition, and (iv) high interspecific contact and strong interspecific competition. The degree of susceptible host regulation that arises from interspecific competition strengthens along the interspecific competition axis. We use this framework to explore how susceptible host regulation through competition and cross-species contact interact to drive parasite transmission.

2010). Theoretical studies have considered how parasite fitness in a multihost community (hereafter, community R_0) is impacted by the composition and abundance of the host community (Norman et al. 1999; Roche et al. 2012; Mihaljevic et al. 2014). Dobson (2004) explored the conditions under which species richness, in combination with transmission mode, increases or decreases the propensity for outbreaks, which he quantified by calculating the basic reproduction number across multispecies communities. Rudolf and Antonovics (2005) described how reduction in parasite incidence might arise from different assumptions about transmission in combination with regulation mechanisms, but their study investigated the effects of intraspecific competition and frequency-dependent transmission only. Neither of the latter models included interspecific competition. While models for interspecific competition and disease transmission are well developed, they are rarely examined jointly (but see Bowers and Turner 1997; Peixoto and Abramson 2006). Recently, community epidemiology has made promising advances in the study of parasite transmission in multispecies host communities by invoking phenomenological assumptions linking species richness, abundance, and evenness (Roche et al. 2012; Joseph et al. 2013; Mihaljevic et al. 2014), but these studies do not include interspecific contact heterogeneities and host competition. Our approach introduces these scenarios and asks under what conditions is parasite fitness in a multi-host-species community reduced relative to parasite fitness in a community consisting of a single focal host species. This question represents a fundamental knowledge gap concerning how broad assumptions about transmission mechanisms and strength of competition contribute to the spread of directly transmitted parasites in multispecies communities. This "missing piece" includes the potential independence of contact rates and strength of competition mediated by variable overlap in resource-utilization functions, habitat selection, temporal niche partitioning, and behavioral avoidance.

Accordingly, we consider simple epidemiological models encompassing intra- and interspecific competition along with intra- and interspecific contact. We compare parasite fitness across communities of a single host species (the resident or focal host) and communities composed of a focal and alternative host species. Analytical results of the effect of host species richness on parasite fitness are achieved via a one- and two-host species comparison, and the main results are shown to hold in larger host species assemblages by means of numerical simulations (appendix, available online). The models are flexible in their assumptions about the correlation between contact rates and strength of competition (fig. 1). Contrasting contact and regulatory regimes

include (i) low interspecific contact and weak interspecific competition (e.g., when host species are spatially separated or select different habitats); (ii) high interspecific contact and weak interspecific competition (e.g., host species use different resources, but the resources co-occur); (iii) low interspecific contact and strong interspecific competition (e.g., host species compete for a limiting resource but have limited contact with each other, such as through temporal partitioning or behavioral avoidance); and (iv) high contact and strong interspecific competition (e.g., when species are competing for a common resource).

In addition to regulatory forces, the transmission mode also influences parasite fitness in a community of host species. Per capita contact rates may increase with host density, for example, if the parasite is transmitted primarily through random contact between individuals (density-dependent transmission). Alternatively, contact rates may remain approximately constant across a range of densities, for example, if parasite transmission occurs through sexual contact or between members of populations that are strongly socially structured (frequency-dependent transmission). We incorporate mechanistic host competition into susceptible-infectious-susceptible (SIS) mathematical models spanning frequency- and density-dependent transmission. Using these models, we derive analytical relationships that describe the propensity for amplification and reduction of disease transmission as a function of host species richness by calculating basic reproduction numbers, which measure parasite fitness (Dobson 2004; Roche et al. 2012). These expressions for the disease-diversity relationship can be represented graphically in ecologically relevant parameter space. Our results demonstrate that disease-diversity relationships are more complex than is often recognized, including the potential for disease amplification in host-parasite systems with frequency-dependent transmission and decreased outbreak potential in communities exhibiting density-dependent transmission. Additionally, we demonstrate that elements of community composition, such as host species traits and contact patterns between species, are key components of disease-diversity relationships that should not be neglected by predictive models. In "Discussion," we summarize caveats linked to our modeling approach. Finally, we place our findings in the context of the dilution-effect hypothesis, which broadly posits that the net effect of increasing biodiversity is reduction in parasite transmission (quantified in this article as the propensity for decreases in parasite fitness as species richness increases).

Quantifying Parasite Fitness across Communities

Here, we extend theoretical studies that have examined the effects of increasing biodiversity—for example, through changing species richness or evenness—on parasite dynamics (Norman et al. 1999; Holt et al. 2003; Dobson 2004; Rudolf and Antonovics 2005). To quantify parasite fitness across host communities, we use the basic reproduction number of the parasite in a community of hosts, R_0, which has been used to identify conditions for increased and decreased parasite transmission in multihost disease systems (Norman et al. 1999; Dobson 2004; Roche et al. 2012; Joseph et al. 2013; Mihaljevic et al. 2014). To calibrate the effect of increasing host species richness, we additionally calculate the basic reproduction number of the parasite in a single host species, R_0^j. The advantage of using basic reproduction numbers to quantify parasite fitness is that they are epidemiological properties correlated with parasite incidence and prevalence and may be expressed analytically for single- and two-species communities. Moreover, the basic reproduction number describes the initial growth rate of the parasite population in an entirely susceptible mono- or heterospecific host community, and consequently it is a measure of the propensity for outbreaks across different communities.

Model Formulation and Analysis

We consider a focal host species (species 1) that is a permanent community resident and the introduction of a second alternative host species (species 2) to the community such that parasite transmission occurs between the resident and additional host species. We use the SIS framework as a general model for parasite transmission. By setting parameters to 0, it is easy to generalize this model by noting that the form of the next-generation matrix used to calculate community R_0 is the same for models that make different assumptions regarding immunity and reinfection, such as SI, SIR, and SIRS systems (see the appendix). The SIS model for a two-species community can be written as

$$\begin{aligned}
\frac{dS_1}{dt} &= [b_{01} - b_{11}(N_1 + \alpha_{12}N_2)]N_1 - \sum_{j=1}^{2} \beta_{1j} f(.) I_j S_1 - \mu_1 S_1 + \gamma_1 I_1, \\
\frac{dI_1}{dt} &= \sum_{j=1}^{2} \beta_{1j} f(.) I_j S_1 - \Gamma_1 I_1, \\
\frac{dS_2}{dt} &= [b_{02} - b_{12}(N_2 + \alpha_{21}N_1)]N_2 - \sum_{j=1}^{2} \beta_{2j} f(.) I_j S_2 - \mu_2 S_2 + \gamma_2 I_2, \\
\frac{dI_2}{dt} &= \sum_{j=1}^{2} \beta_{2j} f(.) I_j S_2 - \Gamma_2 I_2.
\end{aligned} \tag{1}$$

Parameters and variables of the model are listed in table 1. The model encompasses single- and two-species communities, with the single-species model being achieved by

Table 1: Model parameters and formulas

Symbol/formula	Meaning
S_j	Number of susceptible individuals of species j
I_j	Number of infectious individuals of species j
$N_j = S_j + I_j$	Population size of species j
m	Number of species in the community
b_{0j}	Per capita natural birth rate of species j
b_{1j}	Per capita density-dependent reduction in birth rate of species j
μ_j	Per capita natural mortality rate of species j
$r_j = b_{0j} - \mu_j$	Per capita natural growth rate of species j
$K_j = r_j/b_{1j}$	Carrying capacity of species j
s_j	Susceptibility of species j
i_j	Infectiousness of species j
$p_{jk} = s_j i_k$	Transmissibility of the parasite from species k to species j
c_{jk}	Per capita contact rate between members of species j and k
$\beta_{jk} = p_{jk} c_{jk}$	Per capita transmission rate from a member of species k to a member of species j
γ_j	Per capita recovery rate of species j
δ_j	Per capita disease-induced mortality rate of species j
$\Gamma_j = \gamma_j + \delta_j + \mu_j$	Per capita removal rate from the I_j class
α_{jk}	Competition coefficient (relative competitive effect of species k on species j)
$\alpha_{jk} = 0$	Interspecific competition absent
$0 < \alpha_{jk} < 1$	Interspecific competition leading to species coexistence
$f(.) = 1$	Density-dependent transmission
$f(.) = 1/\sum_{j=1}^{m} N_j$	Frequency-dependent transmission
$R_0^j = \beta_{jj} K_j/\Gamma_j$	Single-species basic reproduction number (density-dependent transmission)
$R_0^j = \beta_{jj}/\Gamma_j$	Single-species basic reproduction number (frequency-dependent transmission)
$b = \beta_{12}\beta_{21}/\beta_{11}\beta_{22}$	Ratio of per capita interspecific transmission rates to per capita intraspecific transmission rates
$\sigma = R_0^1/R_0^2$	Ratio of the resident species basic reproduction number to that of the additional host species
$R_0 = \frac{1}{2}\left(R_0^1 + R_0^2 + \sqrt{(R_0^1 + R_0^2)^2 - 4R_0^1 R_0^2(1-b)}\right)$	Two-species basic reproduction number (density-dependent transmission, no interspecific competition, $R_0^j = \beta_{jj} K_j/\Gamma_j$)
$R_0 = \frac{1}{2}\left(\frac{R_0^1(1-\alpha_{12})}{1-\alpha_{12}\alpha_{21}} + \frac{R_0^2(1-\alpha_{21})}{1-\alpha_{12}\alpha_{21}} + \sqrt{\left(\frac{R_0^1(1-\alpha_{12})}{1-\alpha_{12}\alpha_{21}} + \frac{R_0^2(1-\alpha_{21})}{1-\alpha_{12}\alpha_{21}}\right)^2 - 4\frac{R_0^1 R_0^2(1-\alpha_{12})(1-\alpha_{21})}{(1-\alpha_{12}\alpha_{21})^2}(1-b)}\right)$	Two-species basic reproduction number (density-dependent transmission, interspecific competition, $R_0^j = \beta_{jj} K_j/\Gamma_j$)
$R_0 = \frac{1}{2}\left(\frac{R_0^1 K_1}{K_1+K_2} + \frac{R_0^2 K_2}{K_1+K_2} + \sqrt{\left(\frac{R_0^1 K_1}{K_1+K_2} + \frac{R_0^2 K_2}{K_1+K_2}\right)^2 - 4\frac{R_0^1 R_0^2 K_1 K_2}{(K_1+K_2)^2}(1-b)}\right)$	Two-species basic reproduction number (frequency-dependent transmission, intraspecific competition, $R_0^j = \beta_{jj}/\Gamma_j$)

setting symbols with subscript 2 to 0. For analytical tractability and to allow ease of comparison of this model with other published models (e.g., Getz and Pickering 1983; Holt and Pickering 1985; Dobson 2004; Rudolf and Antonovics 2005; McCormack and Allen 2007), we assume that density dependence arises via reduction in birth rates with increasing host density. Density- and frequency-dependent transmission modes are characterized by the function $f(.)$. We assume that both intra- and interspecific transmission occur in the two-species system. The per capita transmission rate β_{jk} from species k to species j is the product of transmissibility of the parasite p_{jk} and the number of contacts per unit of time per infected host k with susceptible host j, c_{jk}. If transmission is density dependent, the per capita contact rate between hosts of species j and k scales linearly with community size,

$$c_{jk}(N_j, N_k) = c_{jk}(N_j + N_k),$$

and we assume that the force of infection exerted by species k hosts on species j hosts is $\beta_{jk}I_k$. If transmission is frequency dependent, the per capita contact rate between hosts of species j and k is constant,

$$c_{jk}(N_j, N_k) = c_{jk},$$

and the force of infection is $\beta_{jk}I_k/(N_j + N_k)$. Thus, the force of infection is proportional to the frequency of infectious individuals of species k relative to the total community abundance. Intuitively, the proportion of infectious individuals of species j (probability of contacting species j hosts) is reduced when an alternative host species k is added to the assemblage, which may reduce disease transmission (encounter reduction, sensu Keesing et al. 2006). Our framework can be adapted to flexibly account for different encounter probabilities (appendix).

Heterogeneities in Host Contact Rates and Transmissibility

Contact patterns—for example, through foraging, sexual, or antagonistic encounters—are key to the transmission process. To describe the interspecific mixing patterns in figure 1 mathematically, we use the who-acquires-infection-from-whom (WAIFW) matrix (Anderson and May 1991), whose entries are composed of the per capita transmission rates β_{jk},

$$\mathbf{W} = \begin{bmatrix} \beta_{11} & \beta_{12} \\ \beta_{21} & \beta_{22} \end{bmatrix} = \begin{bmatrix} p_{11}c_{11} & p_{12}c_{12} \\ p_{21}c_{21} & p_{22}c_{22} \end{bmatrix},$$

noting that the per capita contact rate c_{jk} in each entry is constant for frequency-dependent transmission and equal to $c_{jk}(N_1 + N_2)$ for density-dependent transmission. In addition to mixing between species, the WAIFW matrix conveniently describes elements of host competence. Competence is central to studies of parasite transmission in multispecies host communities and embodies the ways in which different host species contribute unequally to parasite fitness. Components of competence include behavioral exposure (Hawley et al. 2011), grooming of ectoparasites and vectors (Keesing et al. 2009), and parasite replication, shedding, and immune activation (Komar et al. 2003). We allow variation in competence to be manifested in susceptibility (s_j), infectiousness (i_j), or both (fig. 2*A*). Here, we assume that transmissibility p_{jk} is composed of the product of susceptibility of host j

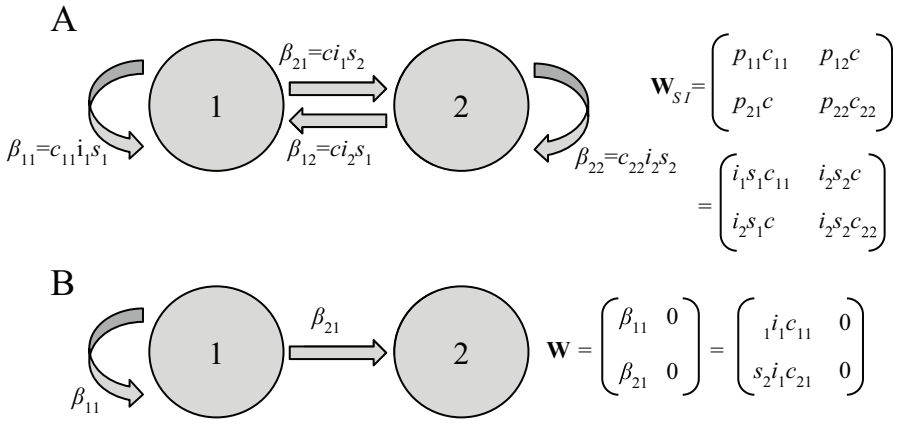

Figure 2: Schematics representing transmission networks for species 1 and 2. Circles represent species 1 (resident host) and 2 (alternative host species), and arrows represent transmission rates within and between species. The corresponding who-acquires-infection-from-whom matrix is shown for each network. *A*, Transmission rates are composed of host susceptibility, infectiousness, and per capita contact rates. *B*, Spillover from the resident host (circle 1) to an alternative dead-end host for transmission (circle 2). Arrows indicate transmission rates within and between members of each population. Since species 2 is a dead-end host, no transmission occurs between members of this species.

and the infectiousness of host k. For example, if competence is driven by susceptibility and the infectiousness of each host species in the assemblage is equal, then each entry of the WAIFW matrix is

$$W_{jk} = s_j i c_{jk}.$$

On the other hand, if transmission is driven by infectiousness i_k and the susceptibility of each host is equal to s, then each entry of the WAIFW matrix is

$$W_{jk} = s i_k c_{jk}.$$

To summarize the contributions of inter- and specific transmission to parasite fitness, we introduce the ratio of inter- to intraspecific transmission coefficients,

$$b = \frac{\beta_{12}\beta_{21}}{\beta_{11}\beta_{22}}.$$

Interestingly, b depends only on the ratio of interspecific ($c_{12} = c_{21} = c$) to intraspecific (c_{jj}) contact rates,

$$b = \frac{s_1 i_2 c \times s_2 i_1 c}{s_1 i_1 c_{11} \times s_2 i_2 c_{22}} = \frac{c^2}{c_{11}c_{22}}.$$

Thus, under this framework, differences in host contact rates are the key determinant of the interspecific-intraspecific transmission ratio, not parasite transmissibility heterogeneities. Consequently, we assume that b is a function of contact only and hereafter refer to it as a contact ratio. If interspecific contact is weak ($b < 1$), then spillover of the parasite between species may occur infrequently, whereas strong interspecific contact ($b > 1$) will facilitate spillover. The ratio of inter- to intraspecific transmission rates has been shown to determine parasite establishment and persistence in some contexts (e.g., Bowers and Turner 1997; Holt et al. 2003; Begon et al. 2008). Since b summarizes the relative degree of mixing between species, we consider the following subcases for the contact ratio representing different contact networks: (1) $b = 1$ (e.g., high degree of mixing due to shared resource utilization; panel ii in fig. 1); (2) $b > 1$ (e.g., species are highly territorial and encounter other species more frequently than members of their own species); and (3) $b < 1$ (e.g., species with high sociality within groups). We will show that b is a key contributor to predicted parasite fitness outcomes in multispecies communities.

Criterion for Comparing Parasite Fitness across Mono- and Heterospecific Communities

To compare parasite fitness across mono- and heterospecific communities, we compare the basic reproduction number of the parasite in a resident host (R_0^1) to community R_0. We use equation (1) to calculate single-host- and two-species-community basic reproduction numbers (see the appendix for details and table 1 for expressions). In a two-species assemblage, community R_0 is expressed analytically in terms of the resident and alternative hosts' basic reproduction numbers (R_0^1 and R_0^2; see table 1). For example, assuming that a second host species is added to an assemblage at the same abundance as the resident host, with the two species having equal interspecific competitive effects on one another, and additionally assuming that transmission is density dependent, the community R_0 for the two-species assemblage is expressed as

$$R_0 = \frac{1}{2}\left(\frac{R_0^1}{1+\alpha} + \frac{R_0^2}{1+\alpha} + \sqrt{\left(\frac{R_0^1}{1+\alpha} + \frac{R_0^2}{1+\alpha}\right)^2 - 4\frac{R_0^1 R_0^2}{(1+\alpha)^2}(1-b)}\right),$$

where α is the symmetric interspecific competition coefficient, b is the ratio of inter- to intraspecific transmission, and

$$R_0^j = \frac{\beta_{jj} K_j}{\Gamma_j},$$

$j = 1, 2$, is single-species R_0. This expression is derived in the section titled "Density-Dependent Transmission and Intra- and Interspecific Competition" in the appendix, whereas the assumption of symmetric competition is relaxed in the section titled "Density-Dependent Transmission and Intraspecific and Asymmetric Interspecific Competition" in the appendix. If competition is asymmetric, the community R_0 expression is different (table 1).

We posit that the net outcome of community interactions and increase in biodiversity is reduction of fitness of the parasite in a heterospecific community relative to its fitness in a monospecific resident host community (a dilution effect) if the following inequality is satisfied:

$$R_0 < R_0^1. \tag{2}$$

We refer to the satisfaction of the inequality $R_0 > R_0^1$ as an amplification effect.

Results

Table 2 summarizes the analytical conditions on the model parameters for which relation (2) will hold for a two-species community and predicted outcomes for parasite fitness (see the appendix for mathematical details). A dilution effect is not a ubiquitous outcome (fig. 3). In all combinations of transmission mode and competition that we examined, increasing species richness through adding a second host species to the assemblage may result in enhanced parasite fitness (i.e., $R_0 > R_0^1$). Since the addition of a second host increases overall population abundance, the combination of density-dependent transmission and regulation by intra-

Table 2: Predicted parasite transmission outcomes for directly transmitted microparasites in a two-host species assemblage

	Density-dependent transmission	Dilution-effect criterion	Frequency-dependent transmission	Dilution-effect criterion
Additive (regulation by intraspecific competition)	Amplification/no change	…	Amplification/dilution	$\sigma > \frac{K_1 b}{K_1 + K_2} + \frac{K_2}{K_1 + K_2}$
Substitutive (regulation by interspecific competition)	Amplification/dilution	$\sigma > \frac{(1-\alpha_{12})b}{\alpha_{12}(1-\alpha_{12}\alpha_{21})} + \frac{1-\alpha_{21}}{1-\alpha_{12}\alpha_{21}}$	Amplification/dilution	$\sigma > \frac{K_1(1-\alpha_{12})b + K_2(1-\alpha_{21})}{K_1(1-\alpha_{12}) + K_2(1-\alpha_{21})}$

Note: Rows describe the means of regulation, and columns represent transmission mode. A row-column combination represents the predicted outcomes for models with those combinations (eq. [1]). Each relationship is linear in $\sigma = R_0^1/R_0^2$, and $b = \beta_{12}\beta_{21}/\beta_{11}\beta_{22}$. The analytical relationships arise from the criterion $R_0 < R_0^1$. If the inequality is satisfied by a parameter set, a dilution effect has been realized.

specific competition cannot lead to a reduction in parasite fitness (e.g., as shown in Dobson 2004), but a dilution effect may manifest through all other transmission-competition combinations. Importantly, neither frequency-dependent transmission nor susceptible host regulation via interspecific competition guarantees reduction of parasite transmission.

The analytical conditions in table 2 for reduction of parasite fitness in a two-host-species community relative to parasite fitness in a community consisting of a single focal host species are characterized by the degree of contact between species and host traits. Specifically, conditions for $R_0 = R_0^1$ are represented by a linear configuration of the ratio of the single-host basic reproduction numbers (σ) and the ratio of inter- to intraspecific contact coefficients (b). In addition to intraspecific contact rates, the ratio of basic reproduction numbers σ captures species-level properties, such as carrying capacity, life span, recovery, and disease-induced mortality rates, thereby quantifying relative differences in life-history traits (carrying capacity, life span, transmission competence) as well as effects of parasites on host survival and morbidity (virulence, infectious period). On the other hand, the interspecific-intraspecific contact ratio b characterizes the relative contact rates between and within species only. For brevity, we refer to trait ratio and trait effects to emphasize that the ratio of the single-host basic reproduction numbers σ includes intrinsic host-species properties and effects of parasites on hosts, whereas b simply refers to ecological contact rates.

The linear configurations in table 2 divide contact-trait parameter space into disjoint regions for which dilution and amplification of parasite fitness manifest (fig. 3), with the slope and intercept of each line determining the orientation. Above and to the left of each line in figure 3 are parameter pairs for which a dilution effect occurs, whereas below and to the right of each line are parameter pairs for which amplification in parasite fitness is the outcome in a two-species community.

When transmission is density dependent and populations of each species are regulated by interspecific competition, resulting in fewer available susceptible hosts, the expression arising from the dilution-effect criterion is

$$\sigma > \frac{(1-\alpha_{12})b}{\alpha_{12}(1-\alpha_{12}\alpha_{21})} + \frac{1-\alpha_{21}}{1-\alpha_{12}\alpha_{21}}. \quad (3)$$

The relative competitive effect of the alternative host (species 2) on the resident host (species 1), α_{12}, determines the parameter space for which a dilution effect manifests because it is the dominating parameter in the slope term. As α_{12} increases, the magnitude of the slope declines (as does, to a lesser extent, the intercept), and the parameter combinations for which an amplification effect may manifest declines in area (compare fig. 3*C* with 3*D*). This suggests that as competition strengthens, a dilution effect may manifest even if the additional host species possesses properties that make it a more competent host for the parasite ($R_0^2 > R_0^1$). On the other hand, high interspecific contact ($b > 1$) is predicted to overwhelm the diluting effect of adding a less competent host to the assemblage, and weak competition facilitates this amplification effect. The analytical results generalize to multispecies communities exhibiting density-dependent transmission and interspecific competition (appendix, figs. A2, A3; figs. A1–A3 are available online). In conclusion, interspecific competition, manifested as an increase in the competitive effect of the additional host species on the resident, facilitates a reduction in parasite transmission for the resident host species.

In contrast, if transmission is frequency dependent and species are regulated through intraspecific competition, the relative abundances of each species at carrying capacity feature in the relationship:

$$\sigma > \frac{K_1 b}{K_1 + K_2} + \frac{K_2}{K_1 + K_2}. \quad (4)$$

The slope depends on the relative abundance of the resident host species, and the intercept is a function of the relative abundance of the additional host species. Since the relative abundances of each host species sum to 1, there

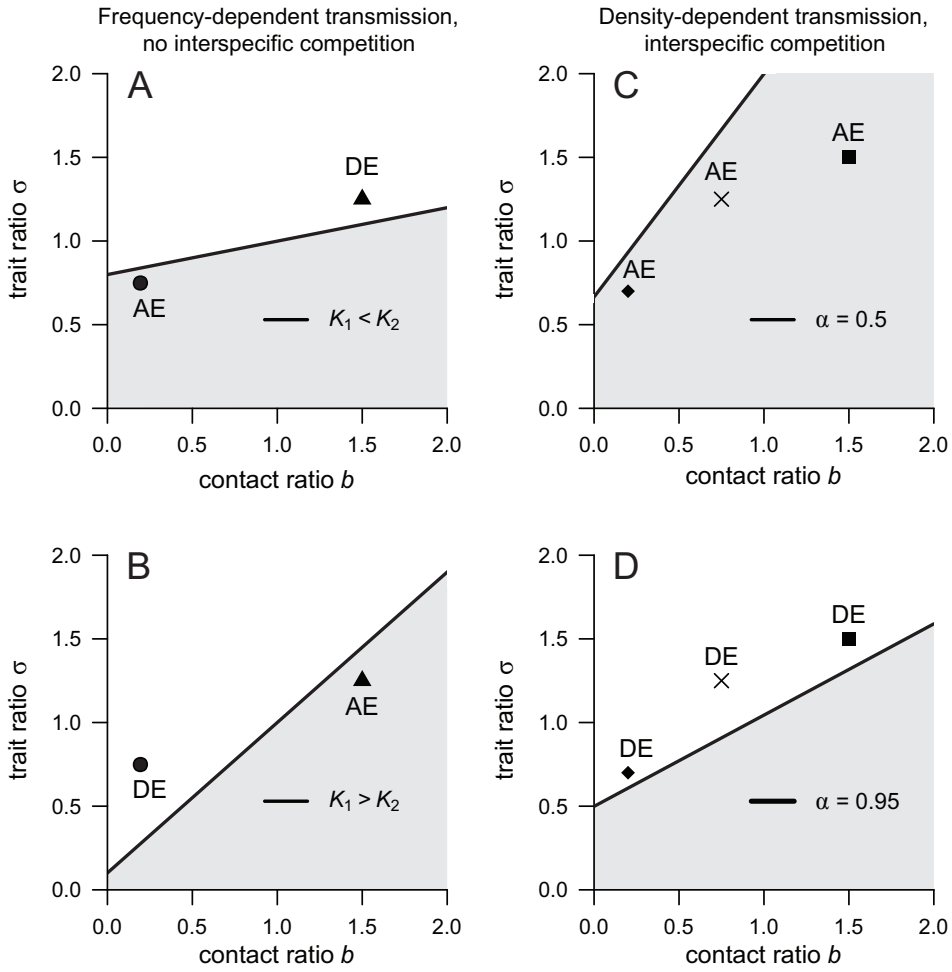

Figure 3: Analytical relationships for combinations of frequency-dependent transmission and intraspecific competition and density-dependent transmission and interspecific competition. The horizontal axis in each figure represents the ratio of inter- to intraspecific transmission (contact ratio, b), and the vertical axis describes the ratio of R_0^1 to R_0^2 (trait ratio, σ). The black lines represent parameter sets (b, σ) for which community R_0 equals the basic reproduction number of the parasite in the resident host, $R_0 = R_0^1$. These relationships separate parameter pairs for which a dilution effect manifests ($R_0 < R_0^1$; white region above each line in *A*–*D*) from pairs that result in an amplification effect ($R_0 > R_0^1$; gray region below each line). Symbols representing parameter pairs that result in dilution (DE) and amplification (AE) are discussed in detail in the text. *A*, The black line separating parameter sets that result in amplification or dilution is a linear configuration resulting from frequency-dependent transmission, intraspecific competition, and no interspecific competition (eq. [4]) when $K_1 < K_2$. Here, $K_1 = 20$ and $K_2 = 80$. *B*, A linear configuration resulting from frequency-dependent transmission, intraspecific competition, and no interspecific competition (eq. [4]) when $K_1 > K_2$. Here, $K_1 = 90$ and $K_2 = 10$. *C*, The black line delineating parameter pairs that lead to amplification or dilution is a linear configuration arising from density-dependent transmission and symmetric interspecific competition, $\sigma = [1/(1 + \alpha)][(b/\alpha) + 1]$. Here, $\alpha = 0.5$ (moderate competition). *D*, A linear configuration arising from density-dependent transmission and strong symmetric interspecific competition. Here, $\alpha = 0.95$.

is an inverse relationship between the slope and the intercept (compare fig. 3*A* with fig. 3*B*). Notably, an amplification effect is possible if a host species with a single-host reproduction number that is less than that of the resident (hereafter, a diluting host species) and a lower carrying capacity than the resident host species is added to the assemblage, provided cross-species contact is sufficiently high ($R_0^1 > R_0^2$, $K_1 > K_2$; fig. 3*A*). On the other hand, an amplification effect is predicted if the additional host species has properties that enhance parasite transmission and its carrying capacity is greater than that of the resident, even if interspecific contact is low ($b < 1$, $R_0^2 > R_0^1$, $K_2 > K_1$; fig. 3*B*). The relative disease-free equilibrium abundances additionally feature in the relationship for frequency-dependent transmission and interspecific competition between host species (table 2). Finally, we note that the predictions for frequency-dependent transmission result from the assumption that the force of infection depends on the frequency of infec-

tious hosts relative to the community abundance $I_k/(N_j + N_k)$. We show in the section titled "Generalizing the Disease-Diversity Relationship for Frequency-Dependent Transmission" in the appendix that reduction of parasite transmission for the focal host will not arise unless the frequency of infectious hosts is reduced when a second species is added.

Relating the Analytical Conditions Back to the Ecological Context (Fig. 1): Interspecific Contact, Host Traits, and Parasite Effects as Drivers of Transmission Outcomes in Communities

The derived relationships shown in table 2 indicate that the ratio of single-species reproduction numbers, σ (which encompasses host traits such as reservoir competence and life span), and b (which determines the degree of parasite spillover mediated by interspecific contact) are both key to manifestation of a dilution effect for a focal host when a parasite is directly transmitted. Parameter pairs with $\sigma > 1$ represent communities with a resident species that is a more competent host for the parasite than the species added to the assemblage by providing a more optimal environment for the parasite to grow and spread in the absence of alternative host species (since R_0^j represents parasite fitness in a population consisting of a single host species). Intrinsic host traits that lead to enhanced R_0^1 relative to R_0^2 include longer life span or infectious period of the resident than the additional host species and greater carrying capacity if transmission is density dependent. Parasites with relatively low virulence and resident hosts with high transmission competence also lead to larger R_0^1 relative to R_0^2. Alternatively, parameter sets with $\sigma < 1$ indicate that the additional host species has properties that make it a more competent host for the parasite than the focal species. In particular, we are interested in the effect of introducing a second host species to the assemblage that is either a diluting host (i.e., $\sigma > 1$) or an amplifying host ($\sigma < 1$) on parasite fitness in a community.

Examining where the relationships shown in table 2 lie in parameter space enables us to distinguish between trait-based (species-specific intrinsic properties that determine parasite fitness in a single host) and contact-based (ecological) mechanisms that determine parasite transmission outcomes in different contexts. Figure 4 is a schematic representing the possible outcomes for community R_0 depending on combinations of b and σ. The slope and intercept of the line, which are determined by transmission mode and the influence of interspecific competition, affect the range of parameters for which each outcome can manifest. Vertical movement away from the horizontal $\sigma = 1$ line represents host species becoming less alike in terms of carrying capacity, life span, competence, infectious period, and virulence. Using figure 4 as a guide, we will describe what outcomes occur for low and high interspecific contact cases separately (under ecological contexts depicted in panels i and iii and in panels ii and iv, respectively, of fig. 1). We summarize the suite of outcomes for frequency-dependent transmission in an assemblage where each species is regulated by intraspecific competition in table 3 and the outcomes for density-dependent transmission where each species is regulated by interspecific competition in table 4.

Model Predictions Assuming Low Interspecific Contact ($b \leq 1$; Panels i and iii in Fig. 1)

A dilution effect is predicted if interspecific contact is weak ($b \ll 1$), the transmission competence of the additional host species is weak relative to the resident ($\sigma > 1$), and transmission is frequency dependent (figs. 3, 4). We term this dilution effect a "trait- and contact-driven dilution effect" because host and parasite traits, along with low contact between species, combine to reduce community R_0 relative to R_0^1. Counterintuitively, if the additional host provides higher parasite fitness than the resident ($R_0^1 < R_0^2$), a contact-driven dilution effect may arise provided the weak interspecific contact between host species overrides the amplifying effect of adding a more competent species to the community on parasite transmission.

For frequency-dependent transmission, contact-driven dilution effects depend on the relative abundance of the additional host species. Depending on the proportion of species 2 at equilibrium, the outcomes for the parameter sets indicated by the circles in figure 3*A* and 3*B* may be either contact-mediated dilution (fig. 3*B*) or, surprisingly, trait-driven amplification (fig. 3*A*). Here, an amplification effect is mediated by host life-history traits and/or the effects of the parasite on each host species. Increasing the relative abundance of the alternative species to that of the resident (amounting to increasing the intercept of the linear relationship) causes the parameter space corresponding to contact-driven dilution effects to contract (compare where the line lies in fig. 3*A* with that in fig. 3*B*).

Contact-driven dilution effects and trait-mediated amplification effects are also predicted if transmission is density dependent and there is competition between species. As the relative competitive effect of the alternative host species on the focal host increases, the parameter space for contact-driven dilution effects increases in area (compare the outcomes of the systems represented by diamonds in fig. 3*C* [amplification] with those in fig. 3*D* [dilution]). Notably, adding a diluting host species is a not a sufficient condition for a dilution effect (e.g., the system represented by the cross symbol in fig. 3*C*). A dilution effect additionally requires that either little cross-species contact occurs or the additional host must have a strong relative competitive effect on the

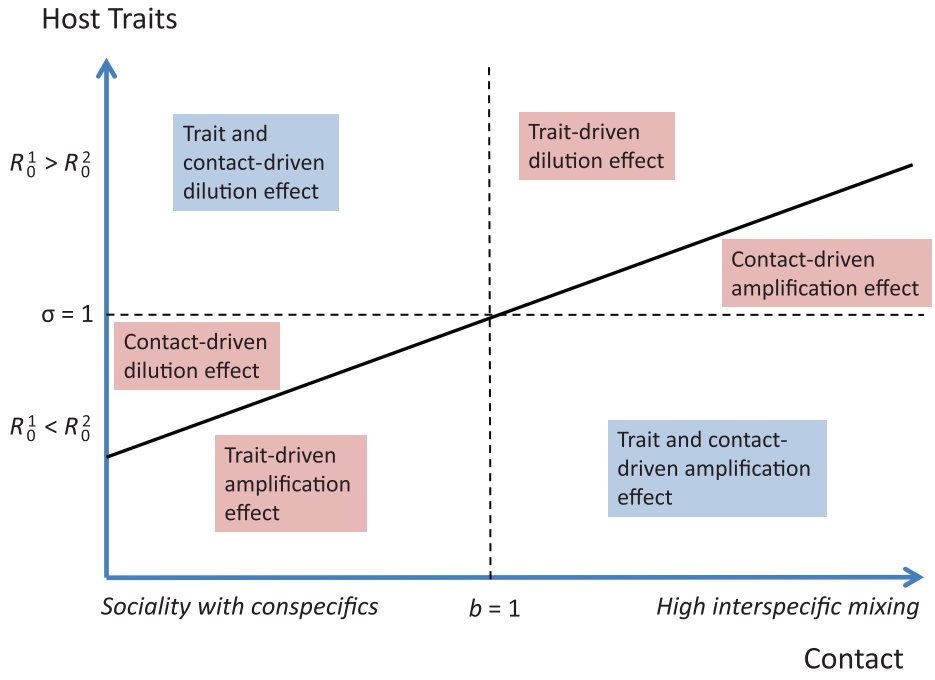

Figure 4: Schematic based on linear relationship for frequency-dependent transmission and intraspecific competition (bold line). The dashed vertical line at $b = 1$ indicates the parameter pairs for which per capita interspecific contact rates equal per capita intraspecific contact rates. The dashed horizontal line at $\sigma = 1$ indicates the parameter pairs for which parasite fitness as propagated by the resident species equals that propagated by the additional host species ($R_0^1 = R_0^2$). The schematic is relevant for frequency-dependent transmission (since $\sigma = 1$ at $b = 1$; eq. [4]) but is also representative of transmission outcomes when interspecific competition is a component of community interactions. The bold and dashed lines divide parameter space into regions for which different kinds of amplification and dilution effects can manifest. Blue regions indicate intuitive outcomes for the disease-diversity relationship, and red regions suggest counterintuitive outcomes. For example, introducing an alternative host species with low transmission competence of the parasite relative to that of the focal host, together with high sociality within species and a low interspecific contact rate, leads to a dilution effect, as expected (trait- and contact-driven dilution effect, in blue), but this type of social regime, combined with an additional species with transmission competence higher than that of the resident, could also lead to dilution (contact-driven dilution effect, in red), contrary to expectations.

resident (compare the cross symbol in fig. 3*C* with its counterpart in fig. 3*D*).

Model Predictions Assuming High Interspecific Contact ($b \geq 1$; Panels ii and iv of Fig. 1)

In cases of high interspecific contact (e.g., panels ii and iv of fig. 1) and assuming frequency-dependent transmission, the addition of a second host species will result in contact- and trait-driven amplification effects if $R_0^1 < R_0^2$ (fig. 4), as the high degree of interspecific contact and the addition of amplifying hosts to the assemblage combine to increase community R_0. For a dilution effect to occur, the additional host species must have diluting traits such that $R_0^1 > R_0^2$, although this does not guarantee the effect.

Table 3: Summary of conditions and potential transmission outcomes in different interspecific contact contexts (fig. 1) given that transmission is frequency dependent and species are regulated by intraspecific competition

Contact ratio	Regulation regime	Analytical result	Potential outcomes
$b > 1$	ii	$R_0^1 > R_0^2$ is not a sufficient condition for dilution	Trait-driven dilution effect Contact-driven amplification effect
$b = 1$	ii	$R_0^1 > R_0^2$ is a necessary and sufficient condition for dilution	Trait- and contact-driven amplification effect
$0 < b < 1$	i	$R_0^1 > R_0^2$ is a sufficient condition for dilution	Contact-driven dilution effect Trait-driven amplification effect Trait- and contact-driven dilution effect

Table 4: Summary of conditions and potential transmission outcomes in different interspecific contact contexts (fig. 1) given that transmission is density dependent and species are regulated by interspecific competition

Contact ratio	Regulation regime	Analytical result	Potential outcomes
$b = 1$	iv	$R_0^1/R_0^2 > 1/\alpha_{12}$ (necessary and sufficient)	Trait-driven dilution effect Contact-driven amplification effect
$b > 1$	iv: weak competitive effect of species 2 on species 1 ($\alpha_{12} \to 0$)	$R_0^1/R_0^2 \to \infty \Rightarrow R_0^1 \gg R_0^2$	Contact-driven amplification effect Trait- and contact-driven amplification effect
	iv: strong competitive effect of species 2 on species 1 ($\alpha_{12} \to 1$)	$R_0^1/R_0^2 \to 1 \Rightarrow R_0^1 > R_0^2$	Trait-driven dilution effect Contact-driven amplification effect Trait- and contact-driven amplification effect
$0 < b < 1$	iii: weak competitive effect of species 2 on species 1 ($\alpha_{12} \to 0$)	$R_0^1 > R_0^2$ is not a sufficient condition for the dilution effect	Trait-driven amplification effect Contact-driven amplification effect Trait- and contact-driven amplification effect
	iii: strong competitive effect of species 2 on species 1 ($\alpha_{12} \to 1$)	$R_0^1 > R_0^2$ is not a sufficient condition for the dilution effect	Contact-driven dilution effect Trait-driven amplification effect Trait- and contact-driven dilution effect

Again, the relative abundance of the alternative host species is important to transmission outcomes. Greater abundances of diluting alternative hosts may counteract the amplifying effect of strong interspecific contact, thereby driving a net dilution effect mediated by traits of the host and parasite (triangle in fig. 3*A*). Less obviously, if the abundance of these additional hosts is less than the resident abundance, a contact-driven amplification effect is predicted (triangle in fig. 3*B*) because of the similarity of parasite fitness in each host species (R_0^1 is only slightly greater than R_0^2 in this scenario). As the intercept of the line (eq. [4]) increases, the parameter space for trait-driven dilution effects increases in area (e.g., triangle in fig. 3*A*; fig. 4).

Additionally, trait-driven dilution and contact-driven amplification are possible outcomes if transmission is density dependent and species are regulated by interspecific competition. In this context, the competitive effect of the additional species on the resident, along with relatively poor competency of the additional species, combine to overcome the potential for spillover via high contact between species and thereby drive a dilution effect (compare the outcomes for the systems represented by the squares in fig. 3*D* [dilution] with those in fig. 3*C* [amplification]). In the appendix, we show that contact-driven amplification effects generalize to multispecies communities exhibiting interspecific competition (fig. A2).

Special Case: Spillover of the Parasite to Dead-End Hosts

Many species novel to a parasite are capable of becoming infected but do not play a role in onward transmission (e.g., humans are "dead-end" hosts for directly transmitted diseases such as rabies and hantavirus). Figure 2*B* shows a transmission regime between the focal host and an additional dead-end host species. Assuming that the additional host is not infectious (i.e., $i_2 = 0$), the WAIFW matrix is

$$\mathbf{W} = \begin{bmatrix} \beta_{11} & 0 \\ \beta_{21} & 0 \end{bmatrix} = \begin{bmatrix} s_1 i_1 c_{11} & 0 \\ s_2 i_1 c_{21} & 0 \end{bmatrix}.$$

To investigate whether the dilution effect occurs, we compared the expression for community R_0 derived from the next-generation matrix to the basic reproduction number of the parasite in the resident species R_0^1 for all transmission-competition combinations previously considered (table 5). A dilution effect occurs if transmission is frequency dependent and if hosts are regulated by interspecific competition when transmission is density dependent. Addition of a dead-end species to the community does not lead to amplification effects in these contexts since the expressions for community R_0 are a fraction of R_0^1 (table 5).

Discussion

We have presented a simple tractable model to link host-species diversity, abundance, and parasite transmission to compare the propensity for infectious disease outbreaks across mono- and multihost communities. Adding more species can either increase or decrease parasite fitness, depending on ecological context and the traits of the species in an assemblage. We demonstrate that disease transmission outcomes in multihost communities are more complex than expected, including the potential for disease amplification in communities exhibiting frequency-dependent transmission and dilution of outbreak risk in density-dependent transmission systems. Our approach offers important insights to other studies that have exploited robust empirical or phenomeno-

Table 5: Conditions that guarantee a dilution effect when a dead-end host for the infection is added to the assemblage, where $K_1/(K_1 + K_2)$ is the abundance of susceptible resident hosts relative to community abundance

Transmission mode and regulation regime	Community R_0	Result
Density dependent, intraspecific competition	R_0^1	No change
Frequency dependent, intraspecific competition	$\frac{K_1 R_0^1}{K_1 + K_2}$	Dilution effect
Density dependent, interspecific competition	$\frac{(1-\alpha_{12})R_0^1}{1-\alpha_{12}\alpha_{21}}$	Dilution effect

logical patterns, including the predictability of host community changes (Johnson et al. 2013) and relationships between species richness and abundance (Roche et al. 2012; Mihaljevic et al. 2014). Our findings emphasize the importance of contact rates, competition, and relative interspecies differences in parasite fitness in developing theory describing the relationship between diversity and microparasitic disease outbreak propensity, thus corroborating empirical studies that have shown these elements to be important for disease-diversity relationships (e.g., Clay et al. 2009; Hall et al. 2009; Johnson et al. 2013). Incorporating competition and contact heterogeneities into simple models changes our predictions of how increasing species richness alters community R_0 (e.g., Dobson 2004; Rudolf and Antonovics 2005).

Our findings may be interpreted in the context of the dilution-effect hypothesis, which is broadly stated as the reduction of parasite transmission in increasingly diverse host communities, acknowledging that some researchers sometimes refer to the particular risk of infection in one species, for example, Lyme disease in humans (Ostfeld and Keesing 2000*a*, 2000*b*; Schmidt and Ostfeld 2001). Many empirical studies have indicated that there is an association between high biodiversity and reduced infectious disease risk, for example, Lyme borreliosis in small mammals and ticks (LoGiudice et al. 2003), West Nile virus in wild birds (Ezenwa et al. 2006) and mosquito vectors (Allan et al. 2009), hantavirus in rodents (Suzán et al. 2009), trematodes in amphibians (Johnson et al. 2013), fungal pathogens of rice crops (Zhu et al. 2000), and yellow barley virus in plants (Lacroix et al. 2014). However, reduction of infectious disease risk in diverse communities may be idiosyncratic and more likely determined by ecological interactions between host species (Salkeld et al. 2013). Simple mathematical models have been used to describe hypothetical mechanisms for a dilution effect, such as encounter reduction and susceptible host regulation mediated by nonhost species (Keesing et al. 2006), but not to study how diluting mechanisms might reduce parasite transmission in a multihost community under a general framework with flexibility in transmission mode and dominant competitive forces. Our tractable models bridge this gap in theory, in particular by providing a theoretical basis for the effects of community composition on the propensity for disease outbreaks in multihost assemblages. Our results suggest that reduction of parasite transmission in multispecies communities will occur if interspecific contact rates are sufficiently low and species in the assemblage differ substantially in their parasite transmission potential. Importantly, the propensity for disease outbreaks may be enhanced on adding additional host species when transmission is either frequency or density dependent, and this propensity is host, parasite, and ecological context dependent.

We have shown that reduction of parasite transmission in multihost communities may manifest for all contact and competition regimes considered. However, the predicted outcomes for parasite transmission are due to different mechanisms. Dilution effects driven by life-history traits such as transmission competence tend to manifest in high interspecific contact scenarios, for example, if there is high overlap in species' resource acquisition functions or if species interact with each other through antagonistic encounters. In general, hosts in an assemblage that compete with each other for a limiting resource must differ substantially in their parasite fitness for a dilution effect to manifest. On the other hand, dilution effects driven by low contact rates between species tend to occur if species exploit different niches or are competitors that avoid one another. These are new theoretical lines of inquiry that could potentially be tested empirically.

Our work challenges some of the key assumptions of the dilution-effect hypothesis (Ostfeld and Keesing 2012); we show that adding a less competent host to the assemblage does not inevitably lead to a dilution effect and that adding a more competent host does not invariably lead to amplification of disease risk. Our analysis suggests that it is possible for host traits to negate the effect of contact on transmission outcomes and for interspecific contact to neutralize the influence of relative parasite fitness afforded by different host species. Our findings lend theoretical support to empirical studies that have shown that species identity is key to the disease-diversity relationship (LoGiudice et al. 2008; Salkeld and Lane 2010; Venesky et al. 2014). For example, the presence of a noncompetent host has been shown to drive a dilution effect in many systems (Johnson et al. 2008; Hall et al. 2009; Keesing et al. 2009). On the other hand, the presence of competent hosts may enhance parasite transmission in some cases (Power and Mitchell 2004; Hamer et al. 2011). Our findings suggest potential new avenues for empirical ex-

ploration that may clarify mechanisms behind dilution and amplification of parasite transmission and may help refine the predictions of the effect of species identity on disease risk in communities.

The generality of the hypothesis that biodiversity is protective against disease has been questioned (Randolph and Dobson 2012; Wood and Lafferty 2013). Our results elucidate conditions under which dilution and amplification of disease transmission with increased species richness can be expected in simple models. We demonstrate that both dilution and amplification effects are possible if a diluting host is added to the assemblage (i.e., the single-host reproduction number of the additional species is less than that of the resident). Whether dilution or amplification is observed depends on ecological context (degree of contact and strength of competition). For example, a greater relative abundance of less competent additional hosts is not sufficient to guarantee a dilution effect in frequency-dependent transmission systems if the degree of interspecific contact is high (e.g., in marine or freshwater assemblages). Ecological dynamics as well as traits of the host species and parasite effects all combine to determine the fate of parasite transmission, even in simple models of two-host-species assemblages.

Naturally, the simple analytical model developed here has some limitations. We compare parasite transmission across multihost communities that are monospecific and heterospecific rather than comparing how increasing diversity affects parasite transmission in a focal host. Parasite fitness in a community is measured using R_0, a quantity capturing parasite dynamics in single and multispecies communities that is correlated with parasite prevalence. Infectious disease risk has been measured by various means in empirical studies, including, for example, prevalence in the focal host, rate of change of infected hosts, prevalence in the community, density of infected vectors, and prevalence of infected vectors (e.g., Mitchell et al. 2002; Johnson et al. 2008, 2013; LoGiudice et al. 2008; Clay et al. 2009; Salkeld and Lane 2010; Searle et al. 2011). We use R_0 as a measure of disease risk across communities, acknowledging that it may not directly measure disease risk in the focal host when a second species is added to the assemblage and that it measures outbreak potential in naive host populations only.

Community ecology is more complicated than the simple Lotka-Volterra models that we use here to calculate community R_0. We ignore trophic levels above and below the level of the host species, which may control host species abundance via top-down and/or bottom-up effects (Keesing et al. 2006). Additionally, the structure of contact networks, host age structure, and the effects of demographic stochasticity are elements that affect the propensity for outbreaks (Newman 2002; Meyers et al. 2005; Dalziel et al. 2014) that are not included in our simple models. We do not examine the impact of correlations between life-history traits and the effects of parasites on hosts (Keesing et al. 2010). All of these complexities will impact transmission outcomes, underscoring the importance of initially studying simple models, which here lead to more nuanced predictions for dilution and amplification effects.

In conclusion, predictions for the level of microparasite transmission in communities cannot be made simply from knowledge of parasite transmission mode or host regulatory mechanism. Metrics of biodiversity such as species richness and evenness may fail to capture predictable outcomes of community assembly and disassembly on parasite fitness. We demonstrate the importance of contact rates, competition, and relative interspecies differences in R_0 on the propensity for disease outbreaks in multispecies communities. Moreover, our analysis demonstrates that even simple canonical models predict that directly transmitted parasite transmission outcomes in community settings are likely to depend on the context in which the ecological dynamics play out. We recommend that these elements be included as components of more complicated predictive models of disease-diversity relationships.

Acknowledgments

We thank T. Dallas, B. Dalziel, J. Haven, the associate editor, and three anonymous reviewers for valuable comments on the manuscript. We also thank K. Carter for assistance in preparing figure 1. This research was funded by grant 220020193 from the James S. McDonnell Foundation. S.M.O. completed this work while a postdoctoral fellow at the National Institute for Mathematical and Biological Synthesis, an institute sponsored by the National Science Foundation (NSF) through NSF award DBI-1300426, with additional support from the University of Tennessee, Knoxville.

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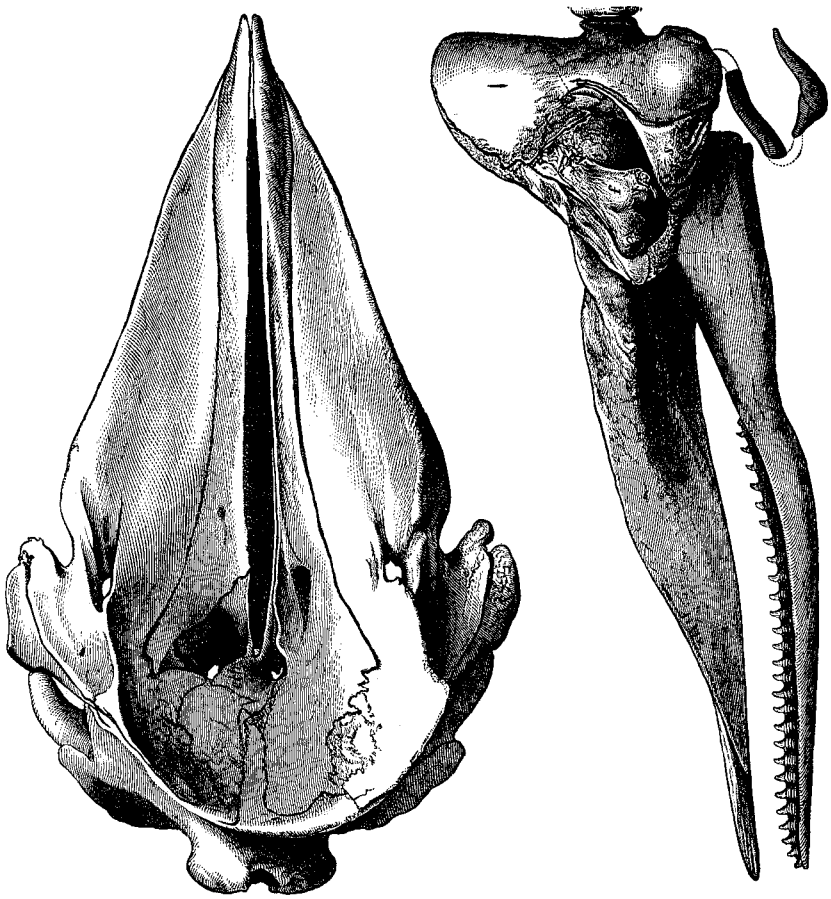
Associate Editor: C. Jessica E. Metcalf
Editor: Susan Kalisz



"Vastness of size is so generally, and it may almost be conceded, so naturally associated in the popular idea with the whales, that some may scarcely be able to realize at first the fact that there are species no larger than ordinary porpoises; and yet which agree so closely in all the more essential elements of structure with some of the whales, that it is impossible, in a natural system, to separate them from their gigantic relatives." Pictured: Two views of the skull of an adult *Physeter macrocephalus*. From "The Sperm Whales, Giant and Pygmy" by Theodore Gill (*The American Naturalist*, 1871, 4:725–743).