# Toward a unified dilution effect theory: How competence, competition, and the transmission mechanism influence disease prevalence

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**ABSTRACT:** Biodiversity in communities is changing globally, including the gain and loss of host species in host-pathogen communities. The dilution effect argues for a mechanistic link between increased host diversity and decreased disease in a focal host. However, we currently have a limited understanding of how the pathogen transmission mechanism and between-host interactions influence whether increased host diversity leads to increased (amplification) or decreased (dilution) infection prevalence. We use a two-host-one-pathogen model to unify theory for pathogens with environmental transmission and density-dependent and frequency-dependent direct transmission. We then identify general rules governing how the pathogen transmission mechanism and characteristics of the introduced host (disease competence and competitive ability) influence whether the introduction of a second host species increases or decreases disease prevalence in a focal host. We discuss how our results yield insight into how specific biological mechanisms shape host biodiversity-disease patterns.

# 1 Introduction

Biodiversity in communities is changing across the globe, with species introductions in some and extirpation in others. Altered biodiversity can influence patterns of infectious diseases because most pathogens can infect multiple host species and most communities are made up of multiple host species (Cleaveland et al., 2001; Pedersen et al., 2005; Rigaud et al., 2010). These effects of biodiversity on disease levels can occur as a result of the ways each host species interacts with the pathogen (e.g., within-species transmission) and interspecific interactions between host species (e.g., resource competition and between-species transmission).

The dilution effect argues that increased host biodiversity decreases disease prevalence (i.e., the proportion of infected hosts in a population) (Keesing et al., 2006). However, when and whether increased host biodiversity reduces disease prevalence (dilution) or increases disease prevalence (amplification) in a focal host population has been debated in the literature (e.g., Lafferty and Wood 2013; Ostfeld and Keesing 2013; Wood and Lafferty 2013 and reviewed in Rohr et al. 2019). Empirical evidence is mixed: a recent meta-analysis found general empirical support for dilution (Civitello et al., 2015), but amplification also occurs (Wood et al., 2014; Venesky et al., 2014; Searle et al., 2016). This implies that increased host biodiversity likely has context-dependent effects (Salkeld et al., 2013), motivating calls for theory that identifies specific biological mechanisms promoting amplification versus dilution (Buhnerkempe et al., 2015; Halsey, 2019; Rohr et al., 2019).

Current theory (Keesing et al., 2006, 2010) predicts that amplification versus dilution generally depends on how host biodiversity affects host-pathogen encounter rates, susceptible host densities, and transmission, mortality and recovery rates. Specific mechanisms affecting these factors include host competence (i.e., the host's ability to transmit infection), intraspecific and interspecific host competition, and the pathogen transmission mechanism. For the latter, some studies have focused on pathogens with frequency-dependent direct transmission (transmission via host-host contact that depends on the frequency of infected hosts),

density-dependent direct transmission (transmission via host-host contact that depends on the density of infected hosts), or environmental transmission (transmission via contact with infectious propagules, e.g., spores, excreted by infected hosts). Many of those studies suggest that frequency-dependent direct transmission promotes dilution whereas density-dependent direct transmission and environmental transmission promote amplification (Begon et al., 1992; Begon and Bowers, 1994; Dobson, 2004; Rudolf and Antonovics, 2005; Hatcher et al., 2006; Mihaljevic et al., 2014; Faust et al., 2017; Roberts and Heesterbeek, 2018). However, theoretical studies also show that accounting for interspecific host competition (Ogden and Tsao, 2009; Strauss et al., 2015; O'Regan et al., 2015; Searle et al., 2016) and host competence (Rudolf and Antonovics, 2005; O'Regan et al., 2015; Roberts and Heesterbeek, 2018) can qualitatively alter predictions. For example, introduction of a high competence host can cause dilution (i.e., *lower* prevalence) in a focal host, even for density-dependent direct transmission pathogens (O'Regan et al., 2015; Roberts and Heesterbeek, 2018) or environmental transmission (Searle et al., 2016).

Current theory is limited in two key ways. First, it is unclear what general rules govern the context dependent effects the pathogen transmission mode and host species characteristics (e.g., competence and competitive ability) have on amplification and dilution. Second, the theories for pathogens with different transmission modes have developed largely independently. This makes it difficult to compare predictions across models and isolate how the pathogen transmission mode influences disease prevalence. Overall, there is a clear need for new theory that can unify existing theory and provide general predictions about how specific mechanisms shape host biodiversity-disease relationships.

To begin to address these limitations, we develop a framework using a two-host-one-pathogen model that unifies the theories for environmental transmission, density-dependent direct transmission and frequency-dependent direct transmission. We use that framework to explore how the pathogen transmission mode and characteristics of an introduced host (specifically, disease competence and interspecific and intraspecific competitive abilities) in-

fluence whether introduction of a second host increases or decreases infection prevalence in a focal host. We discuss how our conditions based on the addition of a second host species yield insight into mechanisms driving host biodiversity-disease relationships. Our approach and results point the way forward for developing a unified theory for amplification and dilution of disease.

# 2 Models and Methods

## 2.1 Two-host-one-pathogen model with environmental transmission

We consider a system with two host species and an environmentally transmitted pathogen, where new infections arise when susceptible hosts come in contact with infectious propagules that were released by infected individuals. To simplify the presentation and analysis, we assume there is no recovery from infection (i.e., infection is always lethal). However, all of our results hold for models with recovery from infection; see appendix S2. We refer to host species 1 and 2 as the 'focal' and 'introduced' hosts, respectively.

The two-host-one-pathogen model describes the changes in the densities of susceptible ($S_i$) and infected ($I_i$) hosts in each population ($i = 1, 2$) and the density of infectious propagules ($P$) in the environment,

$$\begin{aligned}
\frac{dS_i}{dt} &= \underbrace{\Big[f_i(S_1, S_2, I_1, I_2)\Big]}_{\text{growth \& competition}} - \underbrace{\beta_i S_i P}_{\text{infection}} \\
\frac{dI_i}{dt} &= \underbrace{\beta_i S_i P}_{\text{infection}} \underbrace{-m_i I_i}_{\text{mortality}} \\
\frac{dP}{dt} &= \underbrace{\chi_1 I_1 + \chi_2 I_2}_{\text{propagule excretion}} - \underbrace{(u_{11}S_1 - u_{12}I_1 - u_{21}S_2 - u_{22}I_2)P}_{\text{propagule uptake}} \underbrace{-\mu P}_{\text{degradation}} .
\end{aligned} \tag{1}$$

In the model, susceptible hosts increase due to reproduction at rate $f_i(S_1, S_2, I_1, I_2)$ and become infected when they come in contact with infectious propagules ($\beta_i S_i P$); infected hosts excrete infectious propagules into the environment ($\chi_i I_i$) and die ($m_i I_i$); and infectious propagules are lost due to uptake by all hosts ($u_{i1} S_i P$ and $u_{i2} I_i P$ terms) and degradation ($\mu P$). The total population size for each host is $N_i = S_i + I_i$. We assume model (1) has a stable endemic equilibrium, $p^* = (S_1^*, S_2^*, I_1^*, I_2^*, P^*)$, where both hosts coexist with the pathogen (hereafter, the 'multi-species equilibrium'). We also assume model (1) has a stable endemic equilibrium, $\hat{p} = (\hat{S}_1, 0, \hat{I}_1, 0, \hat{P})$, where only the focal host and pathogen coexist (hereafter, the 'single-species equilibrium').

The reproduction rates ($f_i$) account for reproductive output from both susceptible and infected individuals and the possibility of interspecific host competition. We use the general functions in order to develop theory that applies across systems. When presenting specific numerical examples we use the Lotka-Volterra competition functions,

$$f_i = r_i(S_i + c_i I_i)[1 - \alpha_{i1}(S_1 + e_{i1} I_1) - \alpha_{i2}(S_2 + e_{i2} I_2)] \tag{2}$$

where $r_i$ and $c_i r_i$ are the maximum exponential growth rates of susceptible and infected individuals of species $i$, $\alpha_{ij}$ is the per capita competitive effect of host $j$ on host $i$, and $e_{ij}$ determines whether infected individuals of host $j$ have weaker ($e_{ij} < 1$), equal ($e_{ij} = 1$), or stronger ($e_{ij} > 1$) competitive effects on host $i$ than susceptible individuals of host $j$. In general, infected hosts are unlikely to be stronger competitors, however it could occur for pathogens that cause gigantism, provided infection does not alter feeding rates.

Our metric of disease is the infection prevalence in the focal host ($I_1^*/N_1^*$) at the multi-species equilibrium. Our approach is to compute how the parameters defining the competence and competitive ability of the introduced host influence infection prevalence of the focal host at the multi-species equilibrium. This is done using the Jacobian-based theory in Yodzis (1988); see Box 1.

## Box 1: Computing equilibrium density dependencies on model parameters

Infection prevalence of the focal host at the multi-species equilibrium ($p^*$) depends on the characteristics of the introduced host that define competence ($\chi_2$, $\beta_2$, $m_2$, and $u_{21}$), sink/source ($\chi_2$, $u_{22}$), intraspecific competitive ability (e.g., $\alpha_{22}$ in a Lotka-Volterra model), and interspecific competitive ability (e.g., $\alpha_{12}$ in a Lotka-Volterra model). We compute this dependence based on how a small change in one parameter affects the infection prevalence of the focal host at the multi-species equilibrium. Mathematically, this is done using partial derivatives. For example, the effect of the introduced host having a higher infection coefficient ($\beta_2$) is computed using the derivative $\partial(I_1^*/N_1^*)/\partial\beta_2$; positive and negative values mean larger infection coefficients lead to higher or lower prevalence in the focal host, respectively. Due to their large size, all derivative equations are relegated to the appendices.

The derivatives are computed using the Jacobian-based theory developed in Bender et al. (1984), Yodzis (1988), Novak et al. (2011), and Cortez and Abrams (2016); see appendix S1.2 for additional details. The Jacobian is a matrix of derivatives that accounts for all of the intraspecific and interspecific interactions of the system. The Jacobian for model (1) is

$$
\begin{aligned}
J\Big|_{p^*} &= \begin{pmatrix} J_{11} & J_{12} & J_{13} & J_{14} & J_{15} \\ J_{21} & J_{22} & J_{23} & J_{24} & J_{25} \\ J_{31} & 0 & J_{33} & 0 & J_{35} \\ 0 & J_{41} & 0 & J_{44} & J_{45} \\ J_{51} & J_{52} & J_{53} & J_{54} & J_{55} \end{pmatrix} \\
&= \begin{pmatrix} \frac{\partial f_1}{\partial S_1} - \beta_1 P & \frac{\partial f_1}{\partial S_2} & \frac{\partial f_1}{\partial I_1} & \frac{\partial f_1}{\partial I_2} & -\beta_1 S_1 \\ \frac{\partial f_2}{\partial S_1} & \frac{\partial f_2}{\partial S_2} - \beta_2 P & \frac{\partial f_2}{\partial I_1} & \frac{\partial f_2}{\partial I_2} & -\beta_2 S_2 \\ \beta_1 P & 0 & -m_1 & 0 & \beta_1 S_1 \\ 0 & \beta_2 P & 0 & -m_2 & \beta_2 S_2 \\ -u_{11} P & -u_{21} P & \chi_1 - u_{12} P & \chi_2 - u_{22} P & -U - \mu \end{pmatrix}\Bigg|_{p^*} .
\end{aligned}
\tag{3}
$$

where $U = u_{11}S_1 + u_{12}I_1 + u_{21}S_1 + u_{22}S_2$ is the total per spore uptake rate at equilibrium.

The entries in the first and second rows represent combinations of effects of interspecific competition ($J_{12}, J_{14}, J_{21}, J_{23}$), infection ($J_{11}, J_{15}, J_{22}, J_{25}$), and intraspecific competition and reproduction ($J_{11}, J_{13}, J_{22}, J_{24}$); the entries in the third and fourth rows represent the effects of infection ($J_{31},\ J_{35},\ J_{42},\ J_{45}$) and mortality ($J_{33},\ J_{44}$); and the entries in the fifth row represent the negative effects due to uptake by susceptible hosts ($J_{51},\ J_{52}$) and degradation ($J_{55}$) and the combined effects of propagule release and uptake by infected hosts ($J_{53},\ J_{54}$). With the Jacobian, we can predict how introduced hosts with higher or lower competence or competitive ability influence infection prevalence in the focal host.

Roberts and Heesterbeek (2018) used a similar approach to explore how changes in introduced host density affected disease levels in a focal host. Our approach is distinct because we focus on how the characteristics (not the density) of the introduced host affect infection prevalence in the focal host. Some studies have also used the pathogen basic reproductive number ($R_0$) as a metric for levels of disease (Dobson, 2004; Joseph et al., 2013; Mihaljevic et al., 2014; O'Regan et al., 2015). That metric measures how changes in host biodiversity affect the ability of pathogen to invade a completely susceptible community. While analytically simpler, we do not use that metric because there is no *a priori* reason to expect that invasion potential and equilibrium prevalence are affected by the pathogen transmission mechanism and the host characteristics in the same way.

There are two key advantages to our approach. First, it allows us to identify which specific characteristics of the introduced host promote higher versus lower multi-species prevalence in the focal host and if there are interactions between the characteristics (e.g., the effect of increased competence in the introduced host may depend on its interspecific competitive ability). Second, determining the factors that promote higher or lower multi-species prevalence allows us to make predictions about the factors that promote amplification (i.e., higher prevalence in the focal host at the multi-species equilibrium than the single-species equilibrium; $I_1^*/N_1^* > \hat{I}_1/\hat{N}_1$) versus dilution (i.e., lower prevalence in the focal host at the multi-species equilibrium than the single-species equilibrium; $I_1^*/N_1^* < \hat{I}_1/\hat{N}_1$), respectively.

The major limitation of this approach is that it is based on a linear approximation (the derivative). Consequently, it may not accurately capture how infection prevalence depends on a particular parameter if the dependence is sufficiently nonlinear. Nonetheless, because our approach only depends on the signs of the Jacobian entries, our general predictions will hold so long as the nonlinear dependence does not cause the Jacobian entries to change sign.

### 2.2 High and low competence hosts, sinks, and sources

Throughout, we describe host species as being higher or lower competence and sinks or sources of infectious propagules. Host competence is defined by $\beta_i\chi_i/m_iu_{i1}$, which is the number of new infections produced by a single infected host when placed in a completely susceptible population of infinite size (i.e, $R_0$ when host density is arbitrarily large). Higher competence hosts produce more new infections per infected individual because they have a combination of larger infection coefficients ($\beta_i$), larger propagule release rates ($\chi_i$), smaller mortality rates ($m_i$), and smaller uptake rates by susceptible hosts ($u_{i1}$).

Sink and source hosts are defined by the excretion ($\chi_i$) and uptake ($u_{i2}$) rates of infected hosts. Source and sink hosts excrete infectious propagules at rates faster or slower, respectively, than they take them up. A host species is a larger source or a smaller sink if it has a larger propagule release rate ($\chi_i$) and smaller uptake rates ($u_{i2}$).

Competence and sink/source are not identical. For example, a low competence host can be a large source if its transmission parameter ($\beta_i$) is small and its excretion rate ($\chi_i$) is large.

## 3 Results

### 3.1 Unifying environmental and direct transmission models

We first show that by studying the environmental transmission model (1), we can identify how the characteristics of the introduced host influence amplification and dilution for environ-

mental transmission (ET), density-dependent direct transmission (DDDT), and frequency-dependent direct transmission (FDDT) pathogens. Our work builds on Lafferty et al. (2015), which showed that ET, DDDT, and FDDT models are all special cases of a general-consumer resource model. Following previous studies (Li et al., 2009; Eisenberg et al., 2013; Cortez and Weitz, 2013), we use an alternative approach that involves identifying specific conditions under which our ET model reduces to a DDDT or FDDT model. This allows us to directly study how the pathogen transmission mechanism affects disease prevalence.

In general, the ET model reduces to a model with direct transmission when the host excretion rates ($\chi_i$) are large and the infectious propagule uptake ($u_{ij}$) or degradation ($\mu$) rates are large. If the loss of infectious propagules due to uptake by hosts is negligible compared to loss due to degradation ($u_{ij} = 0$), then the ET model reduces to a DDDT model. Alternatively, if there is no degradation of infectious propagules ($\mu = 0$), then the ET model reduces to a FDDT model.

The intuition is the following. Infectious propagules persist in the environment for short periods of time when degradation or uptake rates are large. Consequently, susceptible hosts can only encounter infectious propagules immediately after the infectious propagules are excreted by an infectious host. This requires the susceptible hosts to be in close proximity to an infected individual, in effect implying infection only occurs when there are direct contacts between hosts. When loss due to uptake is negligible ($u_{ij} \approx 0$) compared to degradation, the rate of contact between susceptible hosts and infectious propagules is proportional to the density of infected hosts. In this case, the dynamics of the ET pathogen are essentially identical to those of a DDDT pathogen. In contrast, when there is no degradation ($\mu = 0$), the rate of contact between susceptible hosts and infectious propagules is proportional to the weighted frequency of susceptible hosts in the community, where the weights are the uptake rates of each host class. In this case, the dynamics of the ET pathogen are essentially identical to those of a FDDT pathogen.

To mathematically justify the above, we assume the changes in infectious propagule

density are much faster than changes in the host densities, i.e., the host excretion rates ($\chi_i$) and infectious propagule degradation ($\mu$) or uptake ($u_{ij}$) rates are large. Under this condition, the infectious propagule densities reach a quasi-steady state defined by $dP/dt = 0$. Solving for the quasi-steady density and substituting into the infected host equations yields

$$\frac{dI_i}{dt} = \underbrace{(\beta_i\chi_1 I_1 + \beta_i\chi_2 I_2)\frac{S_i}{U+\mu}}_{\text{infection}} \underbrace{-m_i I_i}_{\text{mortality}} . \tag{4}$$

where $U = u_{11}S_1 - u_{12}I_1 - u_{21}S_2 - u_{22}I_2$ is the total uptake of infectious propagules by all host classes. When loss due to uptake is negligible relative to degradation ($U + \mu \approx \mu$), the infection rates simplify to the infection rates of a DDDT model, $\beta_i\chi_j I_j/\mu = \bar{\beta}_{ij} I_j S_i$. When there is no degradation ($\mu = 0$), the infection rates simplify to the infection rates of a FDDT model with weighted frequencies, $\beta_i\chi_j I_j S_i/U = \bar{\beta}_i I_j S_i/(u_{11}S_1 - u_{12}I_1 - u_{21}S_2 - u_{22}I_2)$. While fast infectious propagule dynamics are necessary for the dynamics of the environmental and direct transmission models to be identical, our results about equilibrium infection prevalence apply for any speed of the infectious propagule dynamics. This is because the equilibria of the ET model are always identical to those of a DDDT or FDDT model when $U = 0$ or $\mu = 0$, respectively.

This unified framework shows that the three transmission modes sit in a two-dimensional space defined by the total uptake ($U$) and degradation ($\mu$) rates of the infectious propagules, with ET lying intermediate between DDDT and FDDT (see Figure 1A). In particular, the equilibrium densities of the ET model are identical to those of a FDDT model when the degradation rate is zero ($\mu = 0$; blue vertical axis) and identical to those of a DDDT model when the uptake rates are negligible ($U = 0$; red horizontal axis). When the uptake and degradation rates are both nonzero ($\mu > 0$, $U > 0$; white), ET behaves like a combination of DDDT and FDDT, determined by the magnitudes of the uptake and degradation rates.

## 3.2 How transmission mode affects amplification and dilution

Having shown that environmental transmission sits intermediate between density-dependent and frequency-dependent direct transmission, we now identify how the pathogen transmission mode influences prevalence in the focal host.

Our approach involves using a change of parameters to convert the ET model from a form that behaves like a DDDT model ($U = 0$) to a form that behaves like a FDDT model ($\mu = 0$) (gray line in Figure 1A). To make a fair comparison between models, our change of parameters holds all parameters constant except the uptake ($u_{ij}$) and degradation ($\mu$) rates, which must necessarily differ between the models. The uptake and degradation rates are varied such that we hold constant the per capita total loss rate of infectious propagules at the single-species equilibrium ($\hat{U} + \mu = u_{11}\hat{S}_1 - u_{12}\hat{I}_1 - u_{21}\hat{S}_2 - u_{22}\hat{I}_2 + \mu$); see appendix S1.5.4 for details. This particular change of parameters results in the single-species equilibrium densities being the same across models and only the multi-species equilibrium densities differing across models. This allows us to isolate how the transmission mechanism influences whether introduction of a second host increases or decreases disease prevalence in the focal host. The biological way to think about the transformation is that the endemic prevalence in the focal host at the single-species equilibrium is known, the pathogen transmission mechanism is unknown, and we want to predict how prevalence at the multi-species equilibrium depends the transmission mechanism. We note that our results are unchanged if we use a change of parameters that instead holds the multi-species equilibrium densities constant and causes the single-species equilibrium densities to change; see appendix S1.5.3 for details. Biologically, this alternative approach would be akin to knowing endemic prevalence in the focal host at the multi-species equilibrium and predicting how endemic prevalence at the single-species equilibrium depends on the pathogen transmission mechanism.

As shown in appendix S1.5.4, lower focal host prevalence under FDDT is promoted by (i) weaker interspecific host competition, (ii) weak intraspecific competition in the introduced host, and (iii) lower competence in the introduced host. Conversely, lower focal host preva-

lence under DDDT is promoted by (i) stronger interspecific host competition, (ii) stronger intraspecific competition in the introduced host, and (iii) higher competence in the introduced host. Prevalence in the focal host under ET always sits between FDDT and DDDT.

For example, in the absence of interspecific competition (Figure 1B), focal host prevalence is typically lower under FDDT than DDDT, but the opposite can occur if the introduced host is a strong intraspecific competitor and a high competence host (purple curve). When interspecific host competition is stronger (Figure 1C), lower focal host prevalence under density-dependent direct transmission is more common. Moreover, increased interspecific competition can reverse the relationship between transmission mode and focal host prevalence. For example, in the absence of interspecific competition, focal host prevalence is lower under FDDT for introduced hosts in Figure 1B that are low competence, strong intraspecific competitors (vermilion "Low, Strong" curve) and high competence, weak intraspecific competitors (blue-green "High, Weak" curve). However, the pattern reverses when interspecific competition is sufficiently strong (vermilion and blue-green curves are decreasing in Figure 1C). In our numerical simulations, transmission mode only had a modest effect on focal host prevalence in all cases where the introduced host was a high competence, weak intraspecific competitor and increased interspecific competition reversed the relationship between transmission mode and focal host prevalence (blue-green curves in Figure 1 have small slopes).

## 3.3 How host competence and competitive ability affect amplification and dilution

We now explore how the competence and intraspecific and interspecific competitive abilities of the introduced host affect prevalence in the focal host. Details are provided in appendix S1.4.

**Competence of the introduced host:** Intuition suggests that a higher competence host (larger $\beta_i\chi_i/m_iu_{1i}$) will cause greater prevalence than a lower competence host. This

pattern holds under many conditions. For example, prevalence declines with increased host mortality in Figure 2A (red and magenta curves) and prevalence increases with increased infection coefficients in Figure 2B (left side of red, magenta, and green curves). The mechanism is that higher competence hosts produce more infectious propagules per infectious propagule they are exposed to, which leads to more infectious propagules and consequently, higher prevalence in the focal host.

However, higher competence can decrease focal host prevalence if the introduced host is a large sink (i.e., the introduced host has low excretion or high uptake rates). Specifically, focal host prevalence can increase with higher introduced host mortality rates ($m_2$; blue curve in 2A) or decrease with higher infection coefficients ($\beta_2$; left side of cyan and blue curves in Figure 2B). The mechanism is that increasing the infection rate or decreasing the mortality rate of a sink host increases the number of infected hosts in the sink population. This results in greater rates of uptake of infectious propagules, decreased infectious propagule density, and consequently, lower prevalence in the focal host.

**Intraspecific competitive ability of the introduced host:** Stronger intraspecific competition in the introduced host leads to increased focal host prevalence, unless the introduced host is a sufficiently large source (i.e., the introduced host has very high excretion or very low uptake rates). In addition, the threshold for being a sufficiently large source increases with increased interspecific competition between the hosts. For example, in the absence of interspecific competition (Figure 3A), stronger intraspecific competition leads to greater focal host prevalence when the introduced hosts are sinks (blue curve) and lower prevalence when the introduced hosts are sources (cyan and red curves). However, when interspecific competition is higher (Figure 3B), stronger intraspecific competition causes lower prevalence only if the introduced host is a sufficiently strong source (cyan curve switches from decreasing in Figure 3A to increasing in Figure 3B).

The mechanism is that increased intraspecific competitive ability causes the density of the introduced host to decrease. If the introduced host is a sink, the decrease in density

results in more infectious propagules and greater prevalence in the focal host. In contrast, if the introduced host is a source, the decrease in density results in fewer infectious propagules and lower prevalence in the focal host.

**Interspecific competitive ability of the introduced host:** Stronger interspecific competitive ability of the introduced host causes a decrease in focal host prevalence (blue and cyan curves in Figure 3C), unless the introduced host is a large source (magenta and red curves in Figure 3C).

The mechanism is that increased interspecific competitive ability of the introduced host decreases susceptible focal host density, which decreases the focal host transmission rate. However, the decrease in focal host density also causes an increase in introduced host density (through reduced interspecific competition from the focal host). This leads to increased densities of infected introduced hosts and infectious propagules, which increases the focal host transmission rate. If the introduced host is not a large source of infectious propagules, then the decrease in focal host transmission rates is greater than the increase, resulting in an overall decrease in focal host prevalence. However, if the introduced host is a large source of infectious propagules, then the increase in focal host transmission rates is greater, resulting in an overall increase in focal host prevalence.

## 3.4 Predictions for factors promoting amplification versus dilution

Our results focus on how prevalence in a focal host responds to the addition of a second host. While this does not directly address the effects of biodiversity on disease, it does provide some insight about factors promoting amplification or dilution in the focal host. Our predictions are summarized in Table 1.

We predict higher competence introduced hosts promote amplification, unless the introduced host is a large sink; introduced hosts that are stronger intraspecific competitors promote amplification, unless the introduced host is a large source; and introduced hosts that are stronger interspecific competitors promote dilution, unless the introduced host is a

large source. We also predict that greater dilution and less amplification will occur under frequency-dependent direct transmission (FDDT) when compared to density-dependent direct transmission (DDDT) when interspecific host competition is weak, the introduced host has lower competence, and the introduced host experiences weaker intraspecific competition. Greater dilution and less amplification occurs under DDDT under the opposite conditions.

These predictions focus on which factors promote amplification versus dilution, but do not necessarily indicate which one will occur in a given system. In some cases, we can place restrictions on which outcome can occur. Specifically, for any level of interspecific competition, it is possible for dilution to occur under FDDT and amplification to occur under DDDT (Figure 4A). In contrast, only when interspecific competition is sufficiently high can dilution occur under DDDT and amplification occur under FDDT. For example, in Figure 4B, amplification occurs for both transmission mechanisms when interspecific competition is absent or low (dashed curves are above dotted line) whereas dilution can occur for DDDT if interspecific competition is sufficiently strong (solid line passes through dotted line).

There are three conditions under which some or all of our predictions can be reversed. First, every prediction can be reversed if interspecific host competition is greater than intraspecific host competition. This can occur, e.g., in systems where host coexistence is pathogen-mediated. Second, every prediction can be reversed if one or both hosts are experiencing sufficiently large positive density dependence (at equilibrium). This occurs when the pathogen reduces the density of one host to the point where the growth rate of that host is an increasing function of its own density. This is analogous to positive density dependence of a prey species in a predator-prey system, which occurs when the predator reduces the prey density to levels below the hump in the predator nullcline. The filled circles in Figure 2BC denote the minimum parameter values at which one host is experiencing positive density dependence. When the positive density dependence is sufficiently large, all of the curves reverse direction. Third, the predictions about host competence can be reversed if infected hosts are sufficiently stronger interspecific competitors than susceptible hosts. For example,

if infected hosts are stronger interspecific competitors, then higher competence hosts can amplify disease less (decreasing portions of magenta and red curves left of the filled circles in Figure 2C). This can occur, e.g., in systems where pathogens cause gigantism in the host, provided infection does not also decrease feeding rates.

# 4 Discussion

Whether increased host biodiversity leads to greater or less disease has been contested in the literature (Lafferty and Wood, 2013; Ostfeld and Keesing, 2013; Wood and Lafferty, 2013), leading to calls for new theory explaining how particular mechanisms influence amplification and dilution (Buhnerkempe et al., 2015; Halsey, 2019; Rohr et al., 2019). We developed a framework that unifies environmental transmission and direct transmission models and shows environmental transmission lies intermediate between density-dependent and frequency-dependent direct transmission (Figure 1A). We used that framework to identify the context dependent rules defining when the pathogen transmission mode and the competence and competitive ability of an introduced host promote higher versus lower infection prevalence in a focal host (Table 1). These results provide insight about the mechanisms influencing host biodiversity-disease relationships and point the way forward for developing a unified theory for amplification and dilution of disease.

Our general rules and their context dependencies (Table 1) align with the results from previous theoretical studies, suggesting that our results may help unify and explain different observed patterns. For example, in many studies, the addition of one more or more host species causes levels of disease to always increase under density-dependent direct transmission (Begon et al., 1992; Dobson, 2004; Faust et al., 2017) or environmental transmission (Begon and Bowers, 1994) and always decrease under frequency-dependent direct transmission (Rudolf and Antonovics, 2005; Dobson, 2004; Mihaljevic et al., 2014; Roberts and Heesterbeek, 2018). All of these studies assume the introduced host is competent (i.e., can

spread the pathogen) and most assume no interspecific competition between species. However, other studies have shown that interspecific host competition (Rudolf and Antonovics, 2005; Ogden and Tsao, 2009; Strauss et al., 2015; O'Regan et al., 2015; Searle et al., 2016) or a non-competent host (Cáceres et al., 2014; Dallas et al., 2016; Strauss et al., 2015) can lead to lower levels of disease under density-dependent direct transmission or environmental transmission. All of these patterns align with our predictions that (i) interspecific competition promotes dilution (unless the introduced host is a large source), (ii) dilution is more likely under frequency-dependent direct transmission than density-dependent direct transmission in the absence of interspecific host competition, and (iii) lower competence of the introduced host promotes dilution (unless the host is a sufficiently large source).

Our results also help explain how effects of host competence vary with the pathogen transmission mechanism. Many studies predict that introduced hosts with higher competence increase levels of disease more than lower competence hosts (Begon et al., 1992; Begon and Bowers, 1994; Joseph et al., 2013; Roberts and Heesterbeek, 2018). However, this does not always hold for frequency-dependent direct transmission models (Dobson, 2004; Rudolf and Antonovics, 2005; Mihaljevic et al., 2014; O'Regan et al., 2015). Our results can explain this difference. Specifically, under density-dependent direct transmission ($u_{ij} = 0$), all hosts are necessarily sources because all uptake rates are zero. Consequently, we predict higher levels of disease with higher competence of an introduced host whenever there is density-dependent-direct transmission. In contrast, under frequency-dependent direct transmission ($\mu = 0$), a host can be a sink or a source. An introduced host is more likely to be sink in a frequency-dependent direct transmission model if (i) the introduced host has lower transmission coefficients; (ii) the introduced host has low density, which can arise via the introduced host being a strong intraspecific competitor or the focal host be a strong interspecific competitor; and (iii) the focal host spends more time per encounter interacting with heterospecifics than conspecifics (e.g., focal hosts spend more time defending territory against heterospecifics than conspecifics). We predict that lower levels of disease with higher

competence of an introduced host arises under frequency-dependent direct transmission because the introduced host is a sink.

While our results align with and help explain many patterns observed in previous studies, there are limitations to our predictions. One limitation is that our results focus on a single metric of disease levels (prevalence). Two other commonly used metric are infected host density and the pathogen basic reproductive number ($R_0$). Studies using different metrics have reported qualitatively similar results. For example, as we found, increased interspecific competition decreases infection prevalence (Rudolf and Antonovics, 2005) and $R_0$ (O'Regan et al., 2015) for density-dependent and frequency-dependent direct transmission pathogens. However, other studies have shown that predictions can disagree between metrics (Roche et al., 2012; Roberts and Heesterbeek, 2018). This disagreement is unsurprising since there is no *a priori* reason to expect that pathogen invasion ($R_0$) and endemic infected density or prevalence will be affected in the same way. An important area of future work is understanding when and why the metrics disagree or agree. Such work would highlight the potentially different ways in which host biodiversity affects disease dynamics. In addition, because analytical calculations for the $R_0$-based metric are often much simpler than the calculations for the other two metrics, that future work would also identify when predictions from the simpler method will suffice or be insufficient.

A second limitation is that our results can only provide insight about host biodiversity-disease relationships because we focus on the introduction of second host species. In communities with more than two host species, host biodiversity-disease relationships are influenced by correlations between abundance, species richness, host competence, and the order in which species are lost (Dobson, 2004; Roche et al., 2012; Joseph et al., 2013; Mihaljevic et al., 2014; Faust et al., 2017). Also, correlations between species traits could potentially affect predictions. For example, propagule uptake and resource consumption rates of *Daphnia* species are affected by the host filtering rate (Hall et al., 2007; Dallas et al., 2016). Similar correlations may be present in insects (Evans and Entwistle, 1987; Naug, 2014), snails (Lafferty,

1993; Miura et al., 2006), and grazing mammals (Williams and Barker, 2008; Wobeser, 2013) that consume their environmentally transmitted pathogens or encounter them while foraging (Hall et al., 2007). While our results can provide some insight about the effects of these correlations, a direct study of host biodiversity-disease relationships will require applying our approach to multi-host communities.

Finally, an important area of future work is exploring if our unified framework or the framework in Lafferty et al. (2015) can unify dilution effect theory for environmental, direct, and vector-borne transmission. Vector transmission and frequency-dependent direct transmission are thought to be similar (Rudolf and Antonovics, 2005), suggesting that our predictions may also apply to vector-born pathogens. However, patterns of amplification and dilution are affected by how the abundance and biting behavior of the vector respond to host biodiversity (Miller and Huppert, 2013; Normal et al., 1999). Nonetheless, we think the approach used in this study may provide a way to develop a unified theory for all three transmission modes.

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# References

Begon, M., and R. G. Bowers. 1994. Host-host-pathogen models and microbial pest control: the effect of host self regulation. Journal of Theoretical Biology 169:275–287.

Begon, M., R. G. Bowers, N. Kadianakis, and D. E. Hodgkinson. 1992. Disease and com-

munity structure: the importance of host self-regulation in a host-host-pathogen model. American Naturalist 139:1131–1150.

Bender, E. A., T. J. Case, and M. E. Gilpin. 1984. Perturbation experiments in community ecology: theory and practice. Ecology 65:1–13.

Buhnerkempe, M. G., M. G. Roberts, A. P. Dobson, H. Heesterbeek, P. J. Hudson, and J. O. Lloyd-Smith. 2015. Eight challenges in modelling disease ecology in multi-host, multi-agent systems. Epidemics 10:26–30.

Cáceres, C. E., G. Davis, S. Duple, S. R. Hall, A. Koss, P. Lee, and Z. Rapti. 2014. Complex *Daphnia* interactions with parasites and competitors. Mathematical Biosciences 258:148–161.

Civitello, D. J., J. Cohen, H. Fatima, N. T. Halstead, J. Liriano, T. A. McMahon, C. N. Ortega, E. L. Sauer, T. Sehgal, S. Young, and J. R. Rohr. 2015. Biodiversity inhibits parasites: Broad evidence for the dilution effect. PNAS 112:8667–8671.

Cleaveland, S., M. K. Laurenson, and L. H. Taylor. 2001. Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. Philosophical Transactions of the Royal Society of London B 356:991–999.

Cortez, M. H., and P. A. Abrams. 2016. Hydra effects in stable communities and their implications for system dynamics. Ecology 97:1135–1145.

Cortez, M. H., and J. S. Weitz. 2013. Distinguishing between indirect and direct modes of transmission using epidemiological time series. American Naturalist 181:E43–E52.

Dallas, T., R. J. Hall, and J. M. Drake. 2016. Competition-mediated feedbacks in experimental multispecies epizootics. Ecology 97:661–670.

Dobson, A. 2004. Population dynamics of pathogens with multiple host species. American Naturalist 164:S64–S78.

Eisenberg, M. C., S. L. Robertson, and J. H. Tien. 2013. Identifiability and estimation of multiple transmission pathways in cholera and waterborne disease. Journal of Theoretical Biology 324:84–102.

Evans, H. F., and P. F. Entwistle. 1987. Viral diseases. *In* J. R. Fuxa and T. Tanada, eds., Epizootiology of Insect Diseases. Wiley, New York.

Faust, C. L., A. P. Dobson, N. Gottdenker, L. S. Bloomfield, H. I. McCallum, T. R. Gillespie, M. Diuk-Wasser, and R. K. Plowright. 2017. Null expectations for disease dynamics in shrinking habitat: dilution or amplification? Philosophical Transactions of the Royal Society B: Biological Sciences 372:20160173.

Hall, S. R., L. Sivars-Becker, C. Becker, M. A. Duffy, A. J. Tessier, and C. E. Cáceres. 2007. Eating yourself sick: transmission of disease as a function of foraging ecology. Ecology Letters 10:207–218.

Halsey, S. 2019. Defuse the dilution effect debate. Nature Ecology & Evolution 3:145.

Hatcher, M. J., J. T. A. Dick, and A. M. Dunn. 2006. How parasites affect interactions between competitors and predators. Ecology Letters 9:1253–1271.

Joseph, M. B., J. R. Mihaljevic, S. A. Orlofske, and S. H. Paull. 2013. Does life history mediate changing disease risk when communities disassemble? Ecology letters 16:1405–1412.

Keesing, F., L. K. Belden, P. Daszak, A. Dobson, and C. D. Harvell et al. 2010. Impacts of biodiversity on the emergence and transmission of infectious diseases. Nature 468:647–652.

Keesing, F., R. D. Holt, and R. S. Ostfeld. 2006. Effects of species diversity on disease risk. Ecology Letters 9:485–498.

Lafferty, K. D. 1993. Effects of parasitic castration on growth, reproduction and population

dynamics of the marine snail cerithidea californica. Marine Ecology-Progress Series 96:229–229.

Lafferty, K. D., G. DeLeo, C. J. Briggs, A. P. Dobson, T. Gross, and A. M. Kuris. 2015. A general consumer-resource population model. Science 349:854–857.

Lafferty, K. D., and C. L. Wood. 2013. It's a myth that protection against disease is a strong and general service of biodiversity conservation: Response to Ostfeld and Keesing. Trends in Ecology and Evolution 28:503–504.

Li, S., J. N. Eisenberg, I. H. Spicknall, and J. S. Koopman. 2009. Dynamics and control of infections transmitted from person to person through the environment. American Journal of Epidemiology 170:257–265.

Mihaljevic, J. R., M. B. Joseph, S. A. Orlofske, and S. H. Paull. 2014. The scaling of host density with richness affects the direction, shape, and detectability of diversity-disease relationships. PloS one 9:e97812.

Miller, E., and A. Huppert. 2013. The effects of host diversity on vector-borne disease: the conditions under which diversity will amplify or dilute the disease risk. PLoS One 8:e80279.

Miura, O., A. M. Kuris, M. E. Torchin, R. F. Hechinger, and S. Chiba. 2006. Parasites alter host phenotype and may create a new ecological niche for snail hosts. Proceedings of the Royal Society B: Biological Sciences 273:1323–1328.

Naug, D. 2014. Infected honeybee foragers incur a higher loss in efficiency than in the rate of energetic gain. Biology Letters 10:20140731.

Normal, R., R. G. Bowers, M. Begon, and P. J. Hudson. 1999. Persistence of tick-borne virus in the presence of multiple host species: Tick reservoirs and parasite mediated competition. Journal of Theoretical Biology 200:111–118.

Novak, M., J. T. Wootton, D. F. Doak, M. Emmerson, J. A. Estes, and M. T. Tinker. 2011. Predicting community responses to perturbations in the face of imperfect knowledge and network complexity. Ecology 92:836–846.

Ogden, N. H., and J. I. Tsao. 2009. Biodiversity and lyme disease: Dilution or amplification? Epidemics 1:196–206.

O'Regan, S. M., J. E. Vinson, and A. W. Park. 2015. Interspecific contact and competition may affect the strength and direction of disease-diversity relationships for directly transmitted microparasites. The American Naturalist 186:480–494.

Ostfeld, R. S., and F. Keesing. 2013. Straw men don't get lyme disease: response to Wood and Lafferty. Trends in Ecology and Evolution 28:502–503.

Pedersen, A. B., S. Altizer, M. Poss, A. A. Cunningham, and C. L. Nunn. 2005. Patterns of host specificity and transmission among parasites of wild primates. International Journal of Parasitology 35:647–657.

Rigaud, T., M. Perrot-Minnot, and R. J. F. Brown. 2010. Parasite and host assemblages: embracing the reality will improve our knowledge of parasite transmission and virulence. Proceeding of the Royal Society B 277:3693–3702.

Roberts, M., and J. Heesterbeek. 2018. Quantifying the dilution effect for models in ecological epidemiology. Journal of The Royal Society Interface 15:20170791.

Roche, B., A. P. Dobson, J. F. Guegan, and P. Rohani. 2012. Linking community and disease ecology: the impact of biodiversity on pathogen transmission. Philosophical Transactions of the Royal Society B 367:2807–2813.

Rohr, J. R., D. J. Civitello, F. W. Halliday, P. J. Hudson, K. D. Lafferty, C. L. Wood, and E. A. Mordecai. 2019. Towards common ground in the biodiversity–disease debate. Nature Ecology & Evolution pages 1–10.

Rudolf, V. H. W., and J. Antonovics. 2005. Species coexistence and pathogens with frequency-dependent transmission. American Naturalist 166:112–118.

Salkeld, D. J., K. A. Padgett, and J. H. Jones. 2013. A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. Ecology Letters 16:679–686.

Searle, C. L., M. H. Cortez, K. K. Hunsberger, D. C. Grippi, I. A. Oleksy, C. L. Shaw, S. B. de la Serna, C. L. Lash, K. L. Dhir, and M. A. Duffy. 2016. Population density, not host competence, drives patterns of disease in an invaded community. American Naturalist 188:554–566.

Strauss, A. T., D. J. Civitello, C. E. Cáceres, and S. R. Hall. 2015. Success, failure and ambiguity of the dilution effect among competitors. Ecology letters 18:916–926.

Venesky, M. D., X. Liu, E. L. Sauer, and J. R. Rohr. 2014. Linking manipulative experiments to field data to test the dilution effect. Journal of Animal Ecology 83:557–565.

Williams, E. S., and I. K. Barker. 2008. Infectious diseases of wild mammals. John Wiley & Sons.

Wobeser, G. A. 2013. Essentials of disease in wild animals. John Wiley & Sons.

Wood, C. L., and K. D. Lafferty. 2013. Biodiversity and disease: a synthesis of ecological perspectives on lyme disease transmission. Trends in Ecology and Evolution 28:239–247.

Wood, C. L., K. D. Lafferty, G. De Leo, H. S. Young, P. J. Hudson, and A. M. Kuris. 2014. Does biodiversity protect humans against infectious disease? Ecology 95:817–832.

Yodzis, P. 1988. The indeterminacy of ecological interactions. Ecology 69:508–515.

# 6 Tables & Figures

**Table 1:** Predictions for how the characteristics of an introduced host and the pathogen transmission mode affect amplification and dilution in a focal host

| Characteristic | Predicted effects |
|---|---|
| Competence | Higher competence promotes amplification, unless the host is a sufficiently large sink for infectious propagules |
| Competitive ability | Stronger intraspecific competition promotes amplification, unless the host is a sufficiently large source of infectious propagules |
| | Stronger interspecific competitors promotes dilution, unless the host is a sufficiently large source of infectious propagules |
| Transmission mode | Frequency-dependent direct transmission promotes dilution more than density-dependent direct transmission when<br>(i) weak interspecific host competition<br>(ii) introduced host is a weaker intraspecific competitor<br>(iii) introduced host is a lower competence host |
| | Density-dependent direct transmission promotes dilution more than frequency-dependent direct transmission when<br>(i) strong interspecific host competition<br>(ii) introduced host is a stronger intraspecific competitor<br>(iii) introduced host is a higher competence host |
| Conditions that can reverse predictions | Sufficiently strong positive density dependence in either host |
| | Interspecific competition greater than intraspecific competition |
| | Infected hosts are stronger interspecific competitors than susceptible hosts |

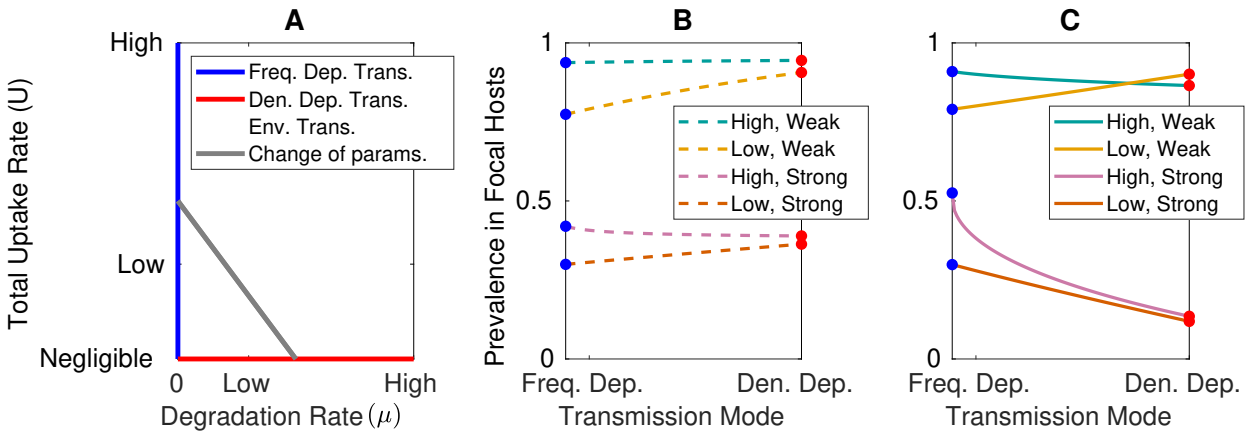


Figure 1: Environmental transmission models and density-dependent and frequency-dependent direct transmission models can be unified, which helps identify how the transmission mechanism influences infection prevalence in a focal host. (A) Environmental transmission sits intermediate between density-dependent and frequency-dependent direct transmission, with environmental transmission models (white) being identical to density-dependent direct transmission models when loss of infectious propagules due to uptake by hosts is negligible ($U = 0$, red) and identical to frequency-dependent direct transmission models when there is no infectious propagule degradation ($\mu = 0$, blue). Gray line illustrates a change of parameters that transforms a particular parameterization of the environmental transmission model from a frequency-dependent form to a density-dependent form while holding the single-species equilibrium densities constant; see text for details. Effect of transmission mode on focal host prevalence in the (B; dashed) absence and (C; solid) presence of interspecific host competition for introduced hosts that are low or high competence and weak or strong intraspecific competitors. Panels show equilibrium prevalence in the focal host as the environmental transmission model is transformed from a frequency-dependent form (blue dots) to a density-dependent form (red dots) while holding the single-species equilibrium densities constant. See appendix S1.6 for models and parameters.

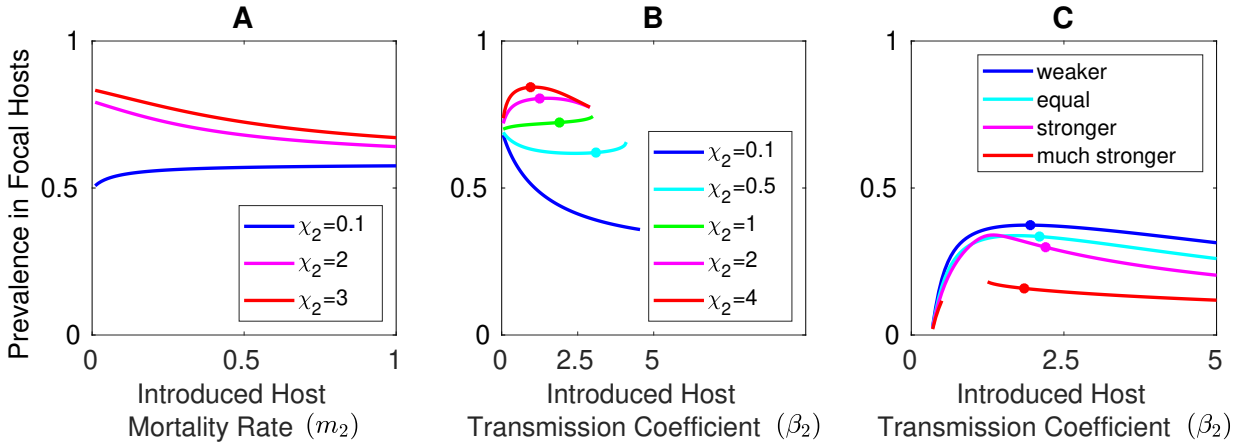


Figure 2: Increased competence of an introduced host leads to greater infection prevalence in a focal host, unless the introduced host is a sufficiently large sink for infectious propagules, one or both hosts experience strong positive density dependence at equilibrium, or infected hosts are stronger interspecific competitors than susceptible hosts. All panels show equilibrium prevalence in the focal host as components defining the competence of the introduced host are varied; filled circles in panels B and C denote parameter values above which at least one host experiences positive density dependence. (A) Response to increased disease-induced mortality when the introduced host is a (blue) large sink, (magenta) small source, or (red) large source. (B) Response to increased transmission rates when the introduced host is a (blue) large sink, (cyan) small sink, (green) equal source, (magenta) large source, or (red) very large source. (C) Response to increased transmission rates when infected hosts are (blue) weaker, (cyan) equal, (magenta) stronger, or (red) much stronger interspecific competitors than susceptible hosts. Break in red curve is due to coexistence being impossible for intermediate transmission coefficients. See appendix S1.6 for equations and parameters.

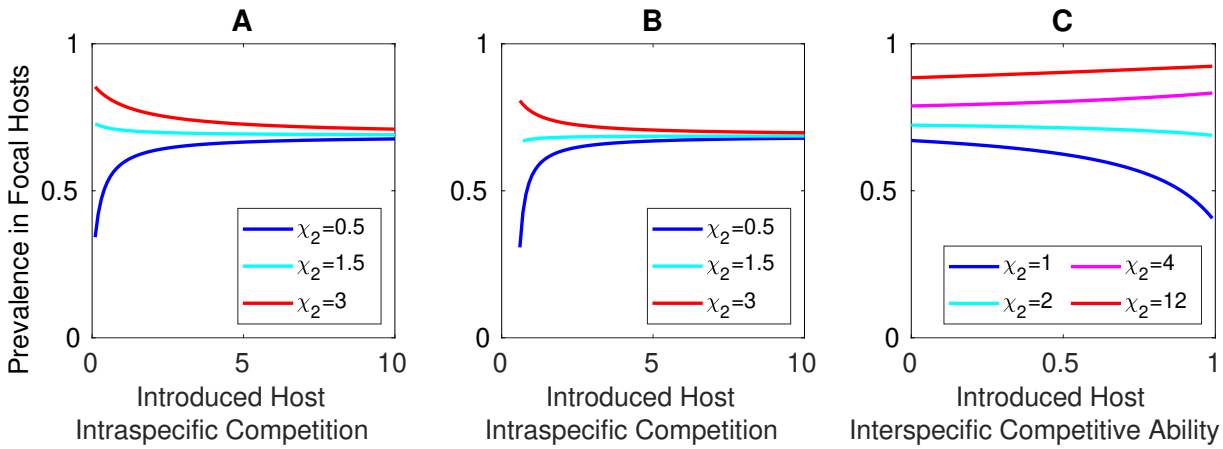


Figure 3: Increased intraspecific competitive ability of the introduced host leads to greater infection prevalence in the focal host and increased interspecific competitive ability of the introduced host leads to lower infection prevalence in a focal host, unless the introduced host is a sufficiently large source of infectious propagules. All panels show equilibrium infection prevalence in the focal host as the (A,B) intraspecific or (C) interspecific competitive ability of the introduced host is varied. Response to increased intraspecific competitive ability of the introduced host in the (A) absence and (B) presence of interspecific competition when the introduced host is a (blue) large sink, (cyan) small source, or (red) large source. (C) Response to increased interspecific competitive ability of the introduced host when the introduced host that is a (blue) large sink, (cyan) equal source, (magenta) large source, or (red) very large source. See appendix S1.6 for equations and parameters.

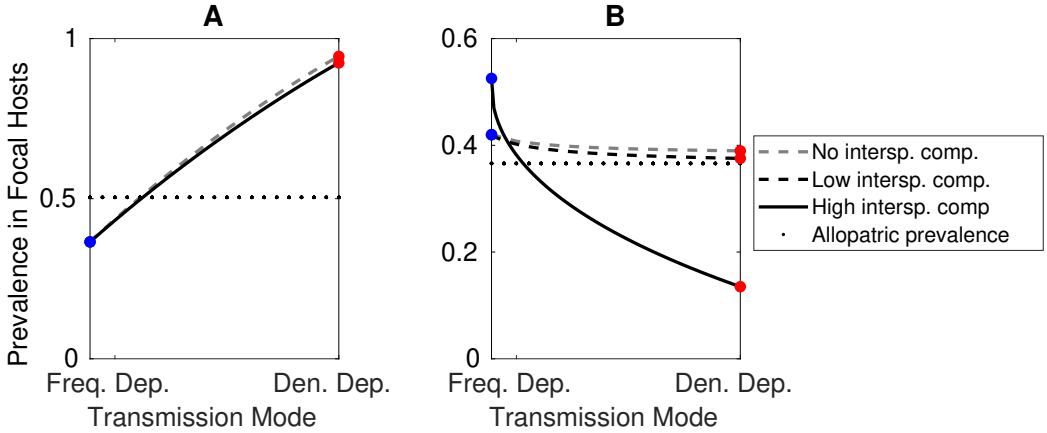


Figure 4: Interspecific host competition influences whether frequency-dependent and density-dependent direct transmission lead to different predictions about amplification and dilution in a focal host. (A) Frequency-dependent direct transmission can cause dilution when density-dependent direct transmission causes amplification in the (dashed gray) absence or (solid black) presence of interspecific competition. (B) Less amplification can occur under density-dependent direct transmission than frequency-dependent direct transmission when interspecific host competition is absent (dashed gray) or low (dashed black). However, density-dependent direct transmission can cause dilution when frequency-dependent direct transmission causes amplification only if (solid black) interspecific host competition is sufficiently strong. In both panels, dotted horizontal lines denote the prevalence in the focal host in allopatry. Dashed and solid curves show multi-species equilibrium prevalence in the focal host as the environmental transmission model is transformed from a frequency-dependent form (blue dots) to a density-dependent form (red dots) while holding the single-species equilibrium densities for the focal host constant; see text for details. See appendix S1.6 for models and parameters.