

Toward a unified dilution effect theory: How competence, competition, and the transmission mechanism influence disease-diversity relationships

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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 dilution effect theory for pathogens with environmental transmission and density-dependent and frequency-dependent direct transmission. We use the unified framework to identify how the pathogen transmission mechanism and characteristics of an introduced host (disease competence and competitive ability) influence infection prevalence in a focal host and under what conditions amplification or dilution is promoted. Our approach identifies general rules governing how specific biological mechanisms shape host biodiversity-disease patterns.

1 Introduction

2 Biodiversity in communities is changing across the globe, with species introductions in some
3 and extirpation in others. Altered biodiversity can influence patterns of infectious diseases
4 because most pathogens can infect multiple host species and most communities are made
5 up of multiple host species (Cleaveland et al., 2001; Pedersen et al., 2005; Rigaud et al.,
6 2010). These effects of biodiversity on disease levels can occur both via the ways each
7 host species interacts with the pathogen (e.g., within-species transmission) and as a result
8 of interspecific interactions between host species (e.g., resource competition and between-
9 species transmission).

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

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

28 density-dependent direct transmission (transmission via host-host contact that depends on
29 the density of infected hosts), or environmental transmission (transmission via contact with
30 infectious propagules, e.g., spores, excreted by infected hosts). Many of those studies suggest
31 that frequency-dependent direct transmission promotes dilution whereas density-dependent
32 direct transmission and environmental transmission promote amplification (Begon et al.,
33 1992; Begon and Bowers, 1994; Dobson, 2004; Rudolf and Antonovics, 2005; Hatcher et al.,
34 2006; Mihaljevic et al., 2014; Faust et al., 2017; Roberts and Heesterbeek, 2018). However,
35 theoretical studies also show that accounting for interspecific host competition (Ogden and
36 Tsao, 2009; Strauss et al., 2015; O'Regan et al., 2015; Searle et al., 2016) and host compe-
37 tence (Rudolf and Antonovics, 2005; O'Regan et al., 2015; Roberts and Heesterbeek, 2018)
38 can qualitatively alter predictions. For example, introduction of a high competence host
39 can cause dilution (i.e., *lower* prevalence) in a focal host, even when the pathogen utilizes
40 density-dependent direct transmission (O'Regan et al., 2015; Roberts and Heesterbeek, 2018)
41 or environmental transmission (Searle et al., 2016).

42 Current theory is limited in two key ways. First, while it shows that transmission mode
43 and host species characteristics (e.g., competence and competitive ability) have context
44 dependent effects on amplification and dilution, it is currently unclear what general rules
45 govern these context dependencies and which biological mechanisms promote amplification
46 versus dilution. Second, the theory for pathogens with different transmission modes has
47 developed largely independently. This makes it difficult to compare predictions across models
48 and to isolate the effects of pathogen transmission mode on disease prevalence. Overall, there
49 is a clear need for new theory that can unify the existing bodies of theory and provide general
50 predictions about how specific mechanisms shape host biodiversity-disease relationships.

51 To begin to address these limitations, we use a two-host-one-pathogen model to explore
52 how the pathogen transmission mode and characteristics of the introduced host (specifically,
53 disease competence and interspecific and intraspecific competitive abilities) influence infec-
54 tion prevalence in a focal host. We do this by developing and applying a framework that

55 unifies the theories for environmental transmission, density-dependent direct transmission
 56 and frequency-dependent direct transmission. We then interpret our conditions in terms of
 57 mechanisms that promote amplification and dilution. Our approach and results point the
 58 way forward for developing a unified theory for amplification and dilution of disease.

59 2 Models and Methods

60 2.1 Two-host-one-pathogen model with environmental transmis- 61 sion

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

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

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

71 In the model, susceptible hosts increase due to reproduction at rate $f_i(S_1, S_2, I_1, I_2)$ and

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

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

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

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

90 Our metric of disease is the infection prevalence of the focal host (I_1^*/N_1^*) at the multi-
91 species equilibrium. Our approach is to compute how the parameters defining the compe-
92 tence and competitive ability of the introduced host influence infection prevalence of the
93 focal host at the multi-species equilibrium. This is done using the Jacobian-based theory in
94 Yodzis (1988); see Box 1 for additional details.

95 **Box 1: Computing equilibrium density dependencies on model parameters**
96

97 Infection prevalence of the focal host at the multi-species equilibrium (p^*) depends
 98 on the characteristics of the introduced host that define competence (χ_2 , β_2 , m_2 , and u_{21}),
 99 sink/source (χ_2 , u_{22}), intraspecific competitive ability (e.g., α_{22} in a Lotka-Volterra model),
 100 and interspecific competitive ability (e.g., α_{12} in a Lotka-Volterra model). We compute this
 101 dependence based on how a small change in one parameter affects the infection prevalence
 102 of the focal host at the multi-species equilibrium. Mathematically, this is done using partial
 103 derivatives. For example, the effect of the introduced host having a higher infection coefficient
 104 (β_2) is computed using the derivative $\partial(I_1^*/N_1^*)/\partial\beta_2$; positive and negative values mean larger
 105 infection coefficients lead to higher or lower prevalence in the focal host, respectively. Due
 106 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

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

107 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.
 108 The entries in the first and second rows represent combinations of effects of interspecific
 109 competition (J_{12} , J_{14} , J_{21} , J_{23}), infection (J_{11} , J_{15} , J_{22} , J_{25}), and intraspecific competition and

110 reproduction ($J_{11}, J_{13}, J_{22}, J_{24}$); the entries in the third and fourth rows represent the effects
111 of infection ($J_{31}, J_{35}, J_{42}, J_{45}$) and mortality (J_{33}, J_{44}); and the entries in the fifth row
112 represent the negative effects due to uptake by susceptible hosts (J_{51}, J_{52}) and degradation
113 (J_{55}) and the combined effects of propagule release and uptake by infected hosts (J_{53}, J_{54}).
114 With the Jacobian, we can predict how introduced hosts with higher or lower competence or
115 competitive ability influence infection prevalence in the focal host. Roberts and Heesterbeek
116 (2018) used a similar approach to explore how changes in introduced host density affected
117 disease levels in a focal host. Our approach is distinct because we focus on how the char-
118 acteristics (not the density) of the introduced host affects infected prevalence in the focal
119 host.

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

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

134 **2.2 High and low competence hosts, sinks, and sources**

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

142 Sink and source hosts are defined by the excretion (χ_i) and uptake (u_{i2}) rates of infected
143 hosts. Source hosts excrete infectious propagules at rates faster than they take them up
144 whereas sink hosts excrete infectious propagules at rates slower than they take them up. A
145 host species is a larger source or a smaller sink if it has a larger propagule release rate (χ_i)
146 and smaller uptake rates (u_{i2}).

147 Competence and sink/source are not identical because competence depends on the uptake
148 rates of susceptible hosts whereas sink/source depends on the uptake rates of infected hosts.
149 Thus, a high competence host can be a large sink if u_{i1} is small and u_{i2} is large.

150 **3 Results**

151 **3.1 Unifying environmental and direct transmission models**

152 We first show that by studying the environmental transmission model (1), we can iden-
153 tify how the characteristics of the introduced host influence patterns of amplification and
154 dilution for both environmentally and directly transmitted pathogens. We do this by unify-
155 ing environmental transmission (ET), density-dependent direct transmission (DDDT), and
156 frequency-dependent direct transmission (FDDT) models with two host species under a sin-
157 gle framework. This greatly extends prior work on single-host-single-pathogen models (Li

158 et al., 2009; Eisenberg et al., 2013; Cortez and Weitz, 2013).

159 Our approach involves identifying specific conditions under which our ET model reduces
160 to a DDDT or FDDT model. In general, the ET model reduces to a model with direct
161 transmission when the host excretion rates (χ_i) are large and the infectious propagule uptake
162 (u_{ij}) or degradation (μ) rates are large. If the loss of infectious propagules due to uptake by
163 hosts is negligible compared to loss due to degradation ($u_{ij} = 0$), then the ET model reduces
164 to a DDDT model. Alternatively, if there is no degradation of infectious propagules ($\mu = 0$),
165 then the ET model reduces to a FDDT model.

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

179 To mathematically justify the above, we assume the changes in infectious propagule
180 density are much faster than changes in the host densities, i.e., the host excretion rates (χ_i)
181 and infectious propagule degradation (μ) or uptake (u_{ij}) rates are large. Under this condition,
182 the infectious propagule densities reach a quasi-steady state defined by $dP/dt = 0$. Solving

183 for the quasi-steady density and substituting into the infected host equations yields

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

184 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
 185 host classes. When loss due to uptake is negligible relative to degradation ($U + \mu \approx \mu$), the
 186 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
 187 there is no degradation ($\mu = 0$), the infection rates simplify to the infection rates of a FDDT
 188 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
 189 fast infectious propagule dynamics are necessary for the dynamics of the environmental and
 190 direct transmission models to be identical, our results about equilibrium infection prevalence
 191 apply for any speed of the infectious propagule dynamics. This is because the equilibria of
 192 the ET model are always identical to those of a DDDT or FDDT model when $U = 0$ or
 193 $\mu = 0$, respectively.

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

202 **3.2 How transmission mode affects amplification and dilution**

203 The previous section showed that environmental transmission sits intermediate between
 204 density-dependent and frequency-dependent direct transmission. Here, we use that to iden-
 205 tify how the pathogen transmission mode influences prevalence in the focal host.

206 Our approach involves using a change of parameters to convert the ET model from a
207 form that behaves like a DDDT model ($U = 0$) to a form that behaves like a FDDT model
208 ($\mu = 0$) (gray line in Figure 1A). To make a fair comparison between models, our change
209 of parameters satisfies two constraints. First, all parameters are kept constant except the
210 uptake (u_{ij}) and degradation (μ) rates, which must necessarily differ between the models.
211 Second, we hold constant the per capita total loss rate of infectious propagules at the single-
212 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.
213 This results in the single-species equilibrium densities being the same across models and only
214 the multi-species equilibrium densities changing as the environmental transmission model is
215 converted between forms. Thus, by identifying host densities at the multi-species equilibrium
216 change with the transformation, we can determine how the pathogen transmission mode
217 affects infection prevalence in the focal host. We note that our results are nearly identical
218 if we use a change of parameters that instead holds the multi-species equilibrium densities
219 constant and causes the single-species equilibrium densities to change; see appendix S1.5.3
220 for details.

221 As shown in appendix S1.5.4, lower focal host prevalence under FDDT is promoted by (i)
222 weaker interspecific host competition, (ii) weak intraspecific competition in the introduced
223 host, and (iii) lower competence in the introduced host. Conversely, lower focal host preva-
224 lence under DDDT is promoted by (i) stronger interspecific host competition, (ii) stronger
225 intraspecific competition in the introduced host, and (iii) higher competence in the intro-
226 duced host. Prevalence in the focal host under ET always sits between FDDT and DDDT.

227 For example, in the absence of interspecific competition (Figure 1B), focal host prevalence
228 is typically lower under FDDT than DDDT, but the opposite can occur if the introduced
229 host is a strong intraspecific competitor and a high competence host (purple curve). When
230 interspecific host competition is stronger (Figure 1C), lower focal host prevalence under
231 density-dependent direct transmission is more common. Moreover, increased interspecific
232 competition can reverse the relationship between transmission mode and focal host preva-

233 lence. For example, in the absence of interspecific competition, focal host prevalence is lower
234 under FDDT for introduced hosts in Figure 1B that are low competence, strong intraspe-
235 cific competitors (vermilion “Low, Strong” curve) and high competence, weak intraspecific
236 competitors (blue-green “High, Weak” curve). However, the pattern reverses when inter-
237 specific competition is sufficiently strong (vermilion and blue-green curves are decreasing in
238 Figure 1C). In our numerical simulations, transmission mode only had a modest effect on
239 focal host prevalence in all cases where the introduced host was a high competence, weak
240 intraspecific competitor and increased interspecific competition reversed the relationship be-
241 tween transmission mode and focal host prevalence (blue-green curves in Figure 1 have small
242 slopes).

243 **3.3 How host competence and competitive ability affect amplifi-** 244 **cation and dilution**

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

248 **Competence of the introduced host:** Intuition suggests that a higher competence
249 host (larger $\beta_i \chi_i / m_i u_{1i}$) will cause greater prevalence than a lower competence host. This
250 pattern holds under many conditions. For example, prevalence declines with increased host
251 mortality in Figure 2A (red and magenta curves) and prevalence increases with increased
252 infection coefficients in Figure 2B (left side of red, magenta, and green curves). The mech-
253 anism is that higher competence hosts produce more infectious propagules per infectious
254 propagule they are exposed to, which leads to more infectious propagules and consequently,
255 higher prevalence in the focal host.

256 However, higher competence can decrease focal host prevalence in two instances. First,
257 focal host prevalence can increase with higher introduced host mortality rates (m_2) (blue
258 curve in 2A) if the introduced host is a large sink (i.e., the introduced host has low excretion

259 or high uptake rates). Second, focal host prevalence can decrease with higher infection
260 coefficients (β_2) if the introduced host is a large sink (left side of cyan and blue curves in
261 Figure 2B). The mechanism is that increasing the infection rate or decreasing the mortality
262 rate of a sink host increases the number of infected hosts in the sink population. This results
263 in greater rates of uptake of infectious propagules, decreased infectious propagule density,
264 and consequently, lower prevalence in the focal host.

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

276 The mechanism is the following. In the absence of interspecific competition, increased
277 intraspecific competitive ability causes the density of the introduced host to decrease. If
278 the introduced host is a sink, the decrease in density results in more infectious propagules
279 and greater prevalence in the focal host. In contrast, if the introduced host is a source, the
280 decrease in density results in fewer infectious propagules and lower prevalence in the focal
281 host. In the presence of interspecific competition, the decrease in density of the introduced
282 host also reduces competition with the focal host. This causes an increase in the number
283 of susceptible hosts in the focal population, which leads to more infections and greater
284 prevalence in the focal host. Because of this positive effect on focal host prevalence, the
285 introduced host must be a very large source of infectious propagules in order for increases

286 in its intraspecific competitive ability to have an overall negative effect on prevalence in the
287 focal host.

288 **Interspecific competitive ability of the introduced host:** Stronger interspecific
289 competitive ability of the introduced host causes a decrease in focal host prevalence, unless
290 the introduced host is a large source. Specifically, when the introduced host is an equal or
291 smaller source than the focal host, stronger interspecific competition leads to decreased focal
292 host prevalence (blue and cyan curves in Figure 3C). In contrast, when the introduced host
293 is a sufficiently larger source than the focal host, stronger interspecific competition leads to
294 greater prevalence (magenta and red curves in Figure 3C).

295 The mechanism is that increased interspecific competitive ability of the introduced host
296 has two effects. First, increased interspecific competitive ability decreases susceptible focal
297 host density, which in turn decreases the focal host transmission rate. Second, the decrease in
298 focal host density causes an increase in introduced host density (through reduced interspecific
299 competition from the focal host). This results in an increased density of infected introduced
300 hosts, which leads to greater infectious propagule densities and an increase in the focal host
301 transmission rate. If the introduced host is not a large source of infectious propagules, then
302 the decrease in focal host infection rates (effect 1) is greater than the increase (effect 2),
303 resulting in a decrease in focal host prevalence. However, if the introduced host is a large
304 source of infectious propagules, then the increase in focal host infection rates (effect 1) is
305 greater, resulting in increased focal host prevalence.

306 **3.4 Predictions for factors promoting amplification versus dilution**

307 Here, we interpret the conditions for increased and decreased infection prevalence in terms of
308 factors promoting amplification or dilution in the focal host. Our predictions are summarized
309 in Table 1.

310 We predict higher competence introduced hosts promote amplification, unless the in-
311 troduced host is a large sink; introduced hosts that are stronger intraspecific competitors

312 promote amplification, unless the introduced host is a large source; and introduced hosts
313 that are stronger interspecific competitors promote dilution, unless the introduced host is a
314 large source. We also predict that greater dilution and less amplification will occur under
315 frequency-dependent direct transmission (FDDT) when compared to density-dependent di-
316 rect transmission (DDDT) when interspecific host competition is weak, the introduced host
317 has lower competence, and the introduced host experiences weaker intraspecific competition.
318 Greater dilution and less amplification occurs under DDDT under the opposite conditions.

319 Our predictions focus on which factors promote amplification versus dilution, but do not
320 necessarily indicate which one will occur in a given system. However, in some cases, we
321 can place restrictions on which outcome can occur. Specifically, for any level of interspecific
322 competition, it is possible for dilution to occur under FDDT and amplification to occur under
323 DDDT (Figure 4A). In contrast, only when interspecific competition is sufficiently high can
324 dilution occur under DDDT and amplification occur under FDDT. For example, in Figure
325 4B, amplification occurs for both transmission mechanisms when interspecific competition
326 is absent or low (dashed curves are above dotted line) whereas dilution can occur for DDDT
327 if interspecific competition is sufficiently strong (solid line passes through dotted line).

328 There are three conditions under which some or all of our predictions can be reversed.
329 First, all of the predictions can be reversed if the effects of interspecific host competition are
330 greater than the effects of intraspecific competition. This can occur, e.g., in systems where
331 there is pathogen-mediated coexistence of the two host species.

332 Second, all of the predictions can be reversed if one or both hosts are experiencing suf-
333 ficiently large positive density dependence (at equilibrium). This occurs when the pathogen
334 reduces the density of one host to the point where the growth rate of that host is an in-
335 creasing function of its own density. This is analogous to positive density dependence of a
336 prey species in a predator-prey system, which occurs when the predator reduces the prey
337 density to levels below the hump in the predator nullcline. The filled circles in Figure 2BC
338 denote the minimum parameter values at which one host is experiencing positive density

339 dependence. When the positive density dependence is sufficiently large, all of the curves
340 reverse direction.

341 Third, the predictions about host competence can be reversed if infected hosts are suf-
342 ficiently stronger interspecific competitors than susceptible hosts. For example, if infected
343 hosts are stronger interspecific competitors, then higher competence hosts can amplify dis-
344 ease less (decreasing portions of magenta and red curves left of the filled circles in Figure 2C).
345 We do not expect this scenario to arise frequently, but it can occur, e.g., in systems where
346 pathogens cause gigantism in the host, provided infection does not also decrease feeding
347 rates.

348 4 Discussion

349 Whether increased host biodiversity leads to greater or less disease has been contested in the
350 literature (Lafferty and Wood, 2013; Ostfeld and Keesing, 2013; Wood and Lafferty, 2013),
351 leading to calls for new theory explaining how particular mechanisms influence amplification
352 and dilution (Buhnerkempe et al., 2015; Halsey, 2019; Rohr et al., 2019). To begin to
353 address this need, we developed a framework that unifies environmental transmission and
354 direct transmission models and used that framework to identify general rules about how host
355 competence and competitive ability and the pathogen transmission mode promote higher
356 versus lower prevalence in a focal host. Our resulting predictions about factors promoting
357 amplification and dilution (Table 1) help unify and extend the existing bodies of dilution
358 theory and point the way forward for developing a unified theory for amplification and
359 dilution of disease.

360 Our approach shows that there are general rules governing how specific biological mech-
361 anisms shape biodiversity-disease patterns, but the rules have context dependencies (Table
362 1). This in turn helps explain some of the differing predictions made in previous stud-
363 ies. For example, in agreement with previous studies that did not include interspecific host

364 competition (Dobson 2004; Rudolf and Antonovics 2005; Hatcher et al. 2006; Faust et al.
365 2017), in the absence of interspecific competition, dilution occurs more frequently under
366 frequency-dependent direct transmission and amplification occurs more frequently under
367 density-dependent direct transmission and environmental transmission (Figure 4). However,
368 as found in other studies (Ogden and Tsao, 2009; Strauss et al., 2015; O'Regan et al., 2015;
369 Searle et al., 2016), incorporating interspecific host competition can alter predictions, in-
370 cluding the possibility that dilution occurs under density-dependent direct transmission, but
371 not frequency-dependent direct transmission (Figure 4B). Our results show that in general
372 increased interspecific competitive ability of the introduced host promotes dilution in a focal
373 host, unless the introduced host is a large source of infectious propagules.

374 Our unified framework helps explain how differences in the transmission mechanism in-
375 fluence amplification and dilution. First, our framework shows that environmental transmis-
376 sion lies intermediate between density-dependent and frequency-dependent direct transmis-
377 sion, with the relative rates of infectious propagule degradation and uptake by hosts dictat-
378 ing whether an environmental transmission system behaves more like a density-dependent
379 or frequency-dependent direct transmission system (Figure 1A). Second, while our gener-
380 eral rules (Table 1) hold for all three transmission types, their implications can differ for
381 density-dependent and frequency-dependent direct transmission pathogen. For example,
382 under density-dependent direct transmission, all hosts are necessarily sources of infectious
383 propagules because the uptake rates are zero. This means that, all else being equal, a higher
384 competence of the introduced host promotes amplification when there is density-dependent-
385 direct transmission. In contrast, under frequency-dependent direct transmission, a host can
386 be a sink or a source. An introduced host is more likely to be sink if (i) the introduced host
387 has a lower transmission coefficient; (ii) the introduced host has lower density, which can arise
388 via the introduced host being a strong intraspecific competitor or the focal host be a strong
389 interspecific competitor; and (iii) the focal host spends more time per encounter interacting
390 with heterospecifics than conspecifics (e.g., focal hosts spend more time defending territory

391 against heterospecifics than conspecifics). Because hosts can be sinks, higher competence
392 of an introduced host does not necessarily promote amplification for frequency-dependent
393 direct transmission pathogens.

394 Our framework identifies additional areas where new theory is needed. First, our frame-
395 work does not address correlations between traits, which could potentially affect predictions
396 about biodiversity-disease relationships. For example, the diluting effects of *Daphnia* species
397 are influenced by propagule uptake rates and resource consumption rates, both of which are
398 affected by the host filtering rate (Hall et al., 2007; Dallas et al., 2016). Similar correlations
399 may also be present in insects (Evans and Entwistle, 1987; Naug, 2014), snails (Lafferty,
400 1993; Miura et al., 2006), and grazing mammals (Williams and Barker, 2008; Wobeser,
401 2013) that consume their environmentally transmitted pathogens or encounter them while
402 foraging (Hall et al., 2007).

403 Second, new theory is needed to understand if our predictions also hold for vector-borne
404 pathogens. Vector transmission and frequency-dependent direct transmission are thought to
405 be similar (Rudolf and Antonovics, 2005). However, patterns of amplification and dilution
406 can be influenced by how host biodiversity affects the abundance and biting behavior of
407 the vector (Miller and Huppert, 2013; Normal et al., 1999). An important area of future
408 work is exploring if our unified framework for environmental and direct transmission can be
409 extended to include vector-borne transmission.

410 Finally, previous studies have used three metrics to study biodiversity-disease relation-
411 ships: the proportion of infected hosts (prevalence), the density of infected hosts, and
412 the pathogen basic reproductive number (R_0). Predictions can disagree between metrics
413 (Roberts and Heesterbeek, 2018). For our model, all of our general predictions about focal
414 host prevalence (Table 1) also hold for focal host infected density; see appendices for de-
415 tails. However, this does not preclude disagreement between the metrics. For example, in
416 Figure 3B, increased intraspecific competitive ability of the introduced host causes higher
417 infected density in all cases, even though prevalence decreases when the introduced host

418 has the largest excretion rate (red curve). The disagreement is due to the introduced host
419 being a sufficiently large source to cause prevalence to decrease with increased intraspecific
420 competitive ability, but an insufficiently large source to also cause the infected host density
421 to decrease. Similar disagreements can occur with other host characteristics or the pathogen
422 transmission mode. Thus, new theory is needed to determine when and why predictions dif-
423 fer between the three metrics and how that affects our understanding of biodiversity-disease
424 relationships.

425 Overall, our work helps unify dilution theory for pathogens with environmental or direct
426 transmission and provides a way forward toward the development of a general unified dilution
427 theory.

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541 6 Tables & Figures

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

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

545

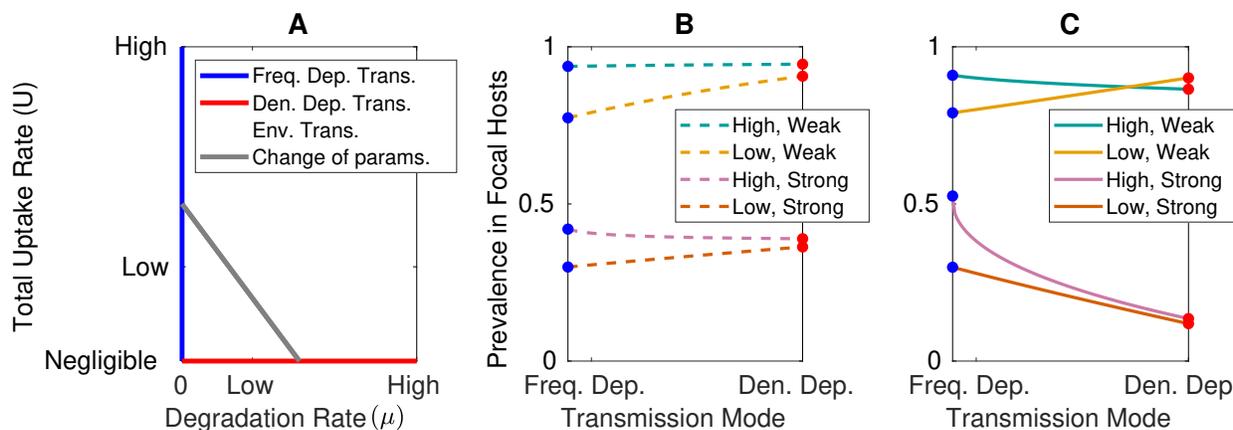


Figure 1: Environmental transmission models and density-dependent and frequency-dependent direct transmission models can be unified, which helps identify how the transmission mechanism influences infection prevalence in a focal host. (A) Environmental transmission sits intermediate between density-dependent 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.

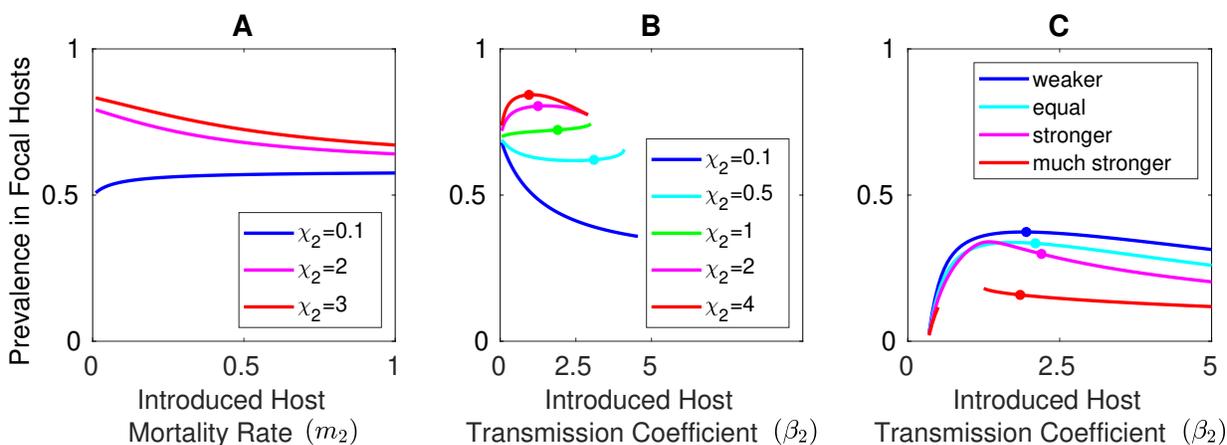


Figure 2: Increased competence of an introduced host leads to greater infection prevalence in a focal host, unless the introduced host is a sufficiently large sink for infectious propagules, one or both hosts experience strong positive density dependence at equilibrium, or infected hosts are stronger 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.

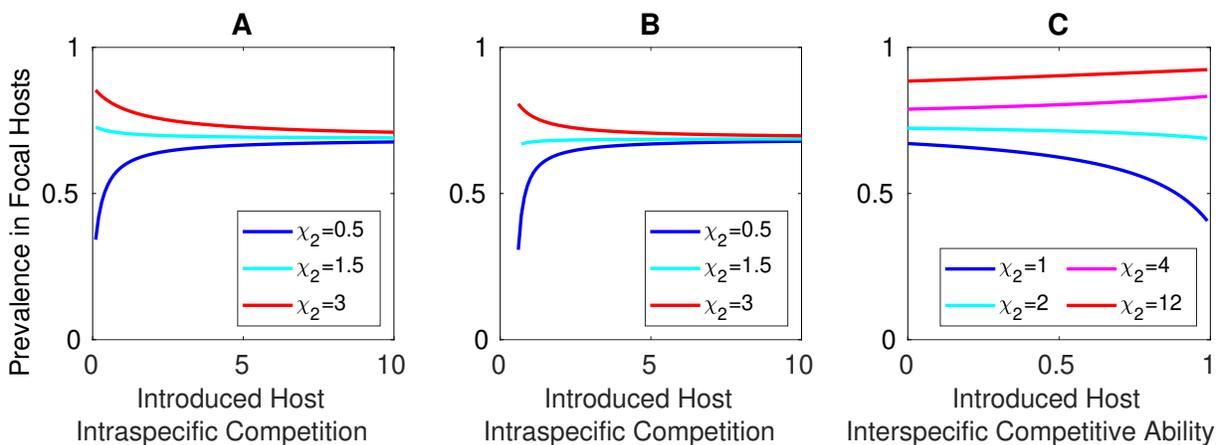


Figure 3: Increased intraspecific competitive ability of the introduced host leads to greater infection prevalence in the focal host and increased interspecific competitive ability of the introduced host leads to lower infection prevalence in a focal host, unless the introduced host is a sufficiently 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.

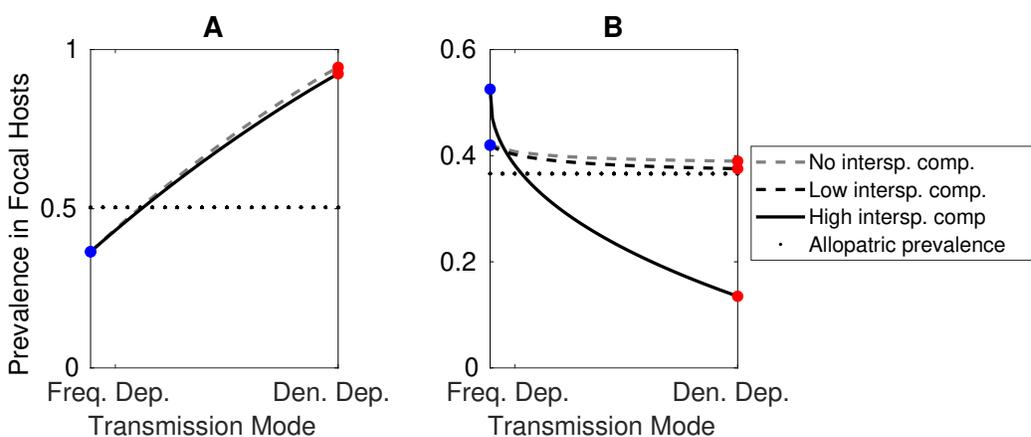


Figure 4: Interspecific host competition influences whether frequency-dependent and density-dependent direct transmission lead to different predictions about amplification and dilution in a focal host. (A) Frequency-dependent direct transmission can cause dilution when density-dependent direct transmission 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.