Supplementary Material for 'Toward the unification of dilution effect theory for environmental and direct transmission pathogens'

Michael H. Cortez & Meghan A. Duffy

Contents

S1 Analysis of SI Environmental Transmission Model **2**
S1.1 Model, assumptions, and definitions 2
S1.1.1 Allopatric equilibrium 2
S1.1.2 Sympatric equilibrium 3
S1.2 Method for computing equilibrium dependence on parameters 4
S1.3 Allopatric equilibrium dependence on parameters 5
S1.4 Sympatric equilibrium dependence on parameters 6
S1.4.1 Response to increased excretion or removal of infectious propagules . 6
S1.4.2 Response to increased mortality of host 2 7
S1.4.3 Response to host 1 experiencing increased interspecific competition . 8
S1.4.4 Response to host 2 experiencing increased competition 9
S1.4.5 Response to increased infection rates of host 2 11
S1.5 Relationships between environmental and direct transmission models 13
S1.5.1 Model equivalences in the limit of fast infectious propagule dynamics 13
S1.5.2 Factors affecting the sign of $U^* - \hat{U}$ 14
S1.5.3 Parameter transformation that holds the sympatric equilibrium constant 16
S1.5.4 Parameter transformation that holds the allopatric equilibrium constant 19
S1.6 Figure equations and parameters 23

S1 Analysis of SI Environmental Transmission Model

S1.1 Model, assumptions, and definitions

The sympatric model (1) in the main text describes the changes in the densities of susceptible (S_i) and infected (I_i) hosts in each population and the density of infectious propagules (P). The total population size for host i is $N_i = S_i + I_i$. Let $p^* = (S_1^*, S_2^*, I_1^*, I_2^*, P^*)$ be a stable endemic coexistence equilibrium of the sympatric model. Let $U^* = u_{11}S_1^* + u_{12}I_1^* + u_{21}S_2^* + u_{22}I_2^*$ denote the total per spore uptake rate at the sympatric equilibrium.

Fixing $S_i = I_i = 0$ for either $i = 1$ or $i = 2$ reduces the sympatric model with two host species to an allopatric model with one host species. We focus on the allopatric model with host species 1, but all of our results apply to the allopatric model with host species 2 after a change of indices. Let $\hat{p} = (\hat{S}_1, \hat{I}_1, \hat{P})$ be a stable endemic coexistence equilibrium of the allopatric model. The pathogen's basic reproductive number for the allopatric system is $\mathcal{R}_0 = \beta_1\chi_1\bar{S}_1/m_1(\mu + u_{11}\bar{S}_1)$ where $\bar{S}_1$ is the susceptible density at the allopatric disease free equilibrium. The total per spore uptake rates at the allopatric equilibrium is $\hat{U} = u_{11}\hat{S}_1 + u_{12}\hat{I}_1$.

Throughout we describe hosts as being higher or lower competence and small or large sinks or sources for infectious propagules. Here, higher competence means higher values of β_2 and χ_2 and lower values of m_2 and u_{2i}. A host is a sink if $\chi_i - u_{i2}P^* < 0$, i.e., the per capita excretion of infectious propagules for infected hosts in population i is lower than the per capita uptake rate at the sympatric equilibrium. A host is a source if $\chi_i - u_{i2}P^* > 0$, i.e., the per capita excretion of infectious propagules for infected hosts in population i is higher than the per capita uptake rate at the sympatric equilibrium. Large sinks (or equivalently, smaller sources) have smaller chi_i and larger u_{ij} values.

Our analysis primarily focuses on systems where both host species experience negative density dependence at the allopatric and sympatric equilibria. Mathematically, we assume the growth rate of host i at equilibrium decreases with an increase in the density of any host class, i.e., $\partial f_i/\partial S_i < 0$ and $\partial f_i/\partial I_i < 0$ for $i = 1, 2$ when evaluated at $\hat{p}$ or p^*. We expect this assumption to be met in most systems. We then discuss how our results can differ if one or both hosts experiences positive density dependence. Mathematically, positive density dependence is defined by $\partial f_i/\partial X_i > 0$ for some host class X_i. Positive density dependence can arise if the pathogen suppresses host densities to very low values.

S1.1.1 Allopatric equilibrium

The Jacobian evaluated at the allopatric equilibrium, $\hat{p}$, is

$$\hat{J} = \left(\begin{array}{ccc} \frac{\partial f_1}{\partial S_1} - \beta_1 P & \frac{\partial f_1}{\partial I_1} & -\beta_1 S_1 \\ \beta_1 P & -m_1 & \beta_1 S_1 \\ -u_{11}P & \chi_1 - u_{12}P & -U - \mu \end{array} \right) \Bigg|_{\hat{p}} \tag{S1}$$

with sign structure

$$\left(\begin{array}{ccc} - & \pm & - \\ + & - & + \\ - & + & - \end{array} \right) \tag{S2}$$

where $\pm$ means the entry can have a positive or negative sign. The entries in the first row represent the combined effects of competition and reproduction of each host class ($\hat{J}_{11}, \hat{J}_{12}$) and infection ($\hat{J}_{11}, \hat{J}_{13}$); the entries in the second row represent the effects of infection ($\hat{J}_{21}$, $\hat{J}_{23}$) and mortality ($\hat{J}_{22}$); and the entries in the third row represent the negative effects due to uptake by susceptible hosts ($\hat{J}_{31}$) and degradation ($\hat{J}_{33}$) and the combined effect of propagule release and uptake by infected hosts ($\hat{J}_{32}$). Stability of $\hat{p}$ implies $|\hat{J}| < 0$.

The entry $\hat{J}_{11}$ is assumed to be negative for the following reason. We write $dS_1/dt = S_1\bar{f}_{1S}(\cdot) + I_1\bar{f}_{1I}(\cdot) - \beta_1 S_1 P$ where $X_1\bar{f}_{1X}$ is the reproductive rate of hosts in class X_1. We assume $\partial\bar{f}_{1X}/\partial Y_1 < 0$ for any host classes X_1 and Y_1 because of intraspecific competition. Computing the $\hat{J}_{11}$ entry yields

$$\hat{J}_{11} = \bar{f}_{1S} - \beta_1 P + S_1\frac{\partial\bar{f}_{1S}}{\partial S_1} + I_1\frac{\partial\bar{f}_{1I}}{\partial S_1}\bigg|_{\hat{p}} = -\frac{I_1\bar{f}_{1I}(\cdot)}{S_1} + S_1\frac{\partial\bar{f}_{1S}}{\partial S_1} + I_1\frac{\partial\bar{f}_{1I}}{\partial S_1}\bigg|_{\hat{p}} < 0 \tag{S3}$$

where the last equality follows from the fact that at equilibrium $dS_1/dt = 0$, which means $\bar{f}_{1S}(\cdot) - \beta_1\hat{P} = -\hat{I}_1\bar{f}_{1I}(\cdot)/\hat{S}_1$. While $\hat{J}_{11}$ is negative, entry $\hat{J}_{12}$ can have either sign because $\partial(X_1\bar{f}_{1X})/\partial X_1$ can be positive or negative depending on the densities of the host classes.

S1.1.2 Sympatric equilibrium

The Jacobian evaluated at the sympatric equilibrium, p^*, is

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S_1} - \beta_1 P & \frac{\partial f_1}{\partial S_2} & \frac{\partial f_1}{\partial I_1} & \frac{\partial f_1}{\partial I_2} & -\beta_1 S_1 \\ \frac{\partial f_2}{\partial S_1} & \frac{\partial f_2}{\partial S_2} - \beta_2 P & \frac{\partial f_2}{\partial I_1} & \frac{\partial f_2}{\partial I_2} & -\beta_2 S_2 \\ \beta_1 P & 0 & -m_1 & 0 & \beta_1 S_1 \\ 0 & \beta_2 P & 0 & -m_2 & \beta_2 S_2 \\ -u_{11}P & -u_{21}P & \chi_1 - u_{12}P & \chi_2 - u_{22}P & -U - \mu \end{pmatrix}\bigg|_{p^*} \tag{S4}$$

with sign structure

$$\begin{pmatrix} - & - & \pm & - & - \\ - & - & - & \pm & - \\ + & 0 & - & 0 & + \\ 0 & + & 0 & - & + \\ - & - & \pm & \pm & - \end{pmatrix} \tag{S5}$$

where $\pm$ means the entry can have a positive or negative sign. The entries in the first and second rows represent the combined effects of intraspecific competition and reproduction of each host class ($J_{11}, J_{13}, J_{22}, J_{24}$), interspecific competition ($J_{12}, J_{14}, J_{21}, J_{23}$), and infection ($J_{11}, J_{15}, J_{22}, J_{25}$); 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}). Stability of p^* implies $|J| < 0$.

The reason why entries J_{11} and J_{22} are negative and entries J_{13} and J_{24} can be either sign is the same as for the allopatric model; see the last paragraph of the previous subsection.

S1.2 Method for computing equilibrium dependence on parameters

We use the Jacobian-based theory in Bender et al. (1984), Yodzis (1988), Novak et al. (2011) and Cortez and Abrams (2016) to compute how changes in a parameter affect equilibrium densities. Let X_i $(i = 1, 2, ...)$ be the variables of the model, $dX_i/dt = F_i(\cdot)$. Let J be the Jacobian of the model evaluated at an equilibrium point $(X_1^*, X_2^*, ...)$.

If a is a parameter of the model that only affects the equation for X_j, i.e., only $F_j(\cdot)$ depends on a, then the change in X_i^* with a small change in the parameter a is defined by the derivative,

$$\frac{\partial X_i^*}{\partial a} = -\frac{\partial F_j}{\partial a}(J^{-1})_{ji} = -\frac{\partial F_j}{\partial a}\frac{(-1)^{i+j}M_{ji}}{|J|} \tag{S6}$$

where M_{ji} is the j, i minor of the Jacobian (i.e., the determinant of the submatrix of J where row j and column i are removed). When we are only interested in the sign of the derivative, we write

$$\frac{\partial X_i^*}{\partial a} \propto (-1)^k M_{ji}/|J| \tag{S7}$$

where "$\propto$" means "proportional to", $(-1)^k = \text{sgn}(-\frac{\partial F_j}{\partial a})(-1)^{1+i+j}$, and $\text{sgn}(\cdot)$ is the sign function.

If a is a parameter that affects the equations for a set Q of the variables, i.e., $F_j(\cdot)$ depends on a if $j \in Q$, then the change in X_i^* with a small change in the parameter a is defined by the sum of derivatives,

$$\frac{\partial X_i^*}{\partial a} = \sum_{j\in Q} -\frac{\partial F_j}{\partial a}(J^{-1})_{ji} = \sum_{j\in Q} -\frac{\partial F_j}{\partial a}\frac{(-1)^{i+j}M_{ji}}{|J|}. \tag{S8}$$

S1.3 Allopatric equilibrium dependence on parameters

All terms and derivatives in this section are evaluated at $\hat{p}$. The response in the density of infected hosts to increased degradation of infectious propagules is

$$\frac{\partial \hat{I}_1}{\partial \mu} = (-1)^{2+3}\hat{P}\frac{M_{32}}{|\hat{J}|} = \frac{-\hat{P}}{|\hat{J}|}\begin{vmatrix} \frac{\partial f_1}{\partial S_1} - \beta_1\hat{P} & -\beta_1\hat{S}_1 \\ \beta_1\hat{P} & \beta_1\hat{S}_1 \end{vmatrix} = \frac{-\beta_1\hat{S}_1\hat{P}}{|\hat{J}|}\frac{\partial f_1}{\partial S_1}. \tag{S9}$$

Because $|\hat{J}| < 0$, the sign of the derivative is determined by $\partial f_1/\partial S_1$. The response in the density of susceptible hosts is

$$\frac{\partial \hat{S}_1}{\partial \mu} = (-1)^{1+3}\hat{P}\frac{M_{31}}{|\hat{J}|} = \frac{\hat{P}}{|\hat{J}|}\begin{vmatrix} \frac{\partial f_1}{\partial I_1} & -\beta_1\hat{S}_1 \\ 0 & \beta_1 S_1 \end{vmatrix} = \frac{\beta_1\hat{S}_1\hat{P}}{|\hat{J}|}\frac{\partial f_1}{\partial I_1} \tag{S10}$$

The sign of this derivative is determined by $-\partial f_1/\partial S_1$. The response in the proportion of infected hosts is determined using the chain rule,

$$\frac{\partial}{\partial \mu}\left(\frac{\hat{I}_1}{\hat{N}_1}\right) = \frac{1}{\hat{N}_1^2}\left[\hat{S}_1\frac{\partial \hat{I}_1}{\partial \mu} - \hat{I}_1\frac{\partial \hat{S}_1}{\partial \mu}\right] = -\frac{\beta_1\hat{S}_1\hat{P}}{\hat{N}_1^2|\hat{J}|}\left[\hat{S}_1\frac{\partial f_1}{\partial S_1} + \hat{I}_1\frac{\partial f_1}{\partial I_1}\right]. \tag{S11}$$

S1.4 Sympatric equilibrium dependence on parameters

All terms and derivatives in the following subsections are evaluated at p^*. In the following equations, $U^* + \mu - u_{11}S_1^* - u_{21}S_2^* > 0$, the effects of interspecific competition show up through the terms $\frac{\partial f_i}{\partial X_j}$ for $i \neq j$ and $X \in \{S, I\}$, and interspecific competition is absent when $\partial f_i/\partial X_j = 0$ for all host classes X and $i \neq j$. To compute how the proportion of infected individuals ($I_1/N_1 = I_1/[S_1 + I_1]$) responds to a small change in parameter a, we use the chain rule,

$$\begin{aligned}\frac{\partial}{\partial a}\left(\frac{I_1^*}{N_1^*}\right) &= \frac{1}{(N_1^*)^2}\left[(S_1^* + I_1^*)\frac{\partial I_1^*}{\partial a} - I_1^*\left(\frac{\partial S_1^*}{\partial a} + \frac{\partial I_1^*}{\partial a}\right)\right] = \frac{1}{(N_1^*)^2}\left[S_1^*\frac{\partial I_1^*}{\partial a} - I_1^*\frac{\partial S_1^*}{\partial a}\right] \\ &\propto \left[\frac{\partial I_1^*}{\partial a} - \frac{\beta_1 P^*}{m_1}\frac{\partial S_1^*}{\partial a}\right]\end{aligned} \tag{S12}$$

where the last line uses the equilibrium condition $0 = dI_i/dt|_{p^*} = \beta_1 S_1^* P^* - m_1 I_1^*$ to substitute for I_1^*.

S1.4.1 Response to increased excretion or removal of infectious propagules

Response to increased degradation rate: The responses to increases in the degradation rate of the infectious propagules are defined by

$$\begin{aligned}\frac{\partial I_1^*}{\partial \mu} \propto (-1)^{3+5}\frac{M_{53}}{|J|} &= \frac{\beta_1 S_1}{|J|}\left(m_2\frac{\partial f_1}{\partial S_1}\frac{\partial f_2}{\partial S_2} + \beta_2 P\frac{\partial f_1}{\partial S_1}\frac{\partial f_2}{\partial I_2} - m_2\beta_2 P\frac{\partial f_1}{\partial S_1}\right) \\ &+ \frac{\beta_1\beta_2 P}{|J|}\left[\frac{\partial f_2}{\partial I_2}S_2\frac{\partial f_1}{\partial S_2} - \frac{\partial f_1}{\partial I_2}\left(S_1\frac{\partial f_2}{\partial S_1} + S_2\frac{\partial f_2}{\partial S_2}\right)\right] - \frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial S_1}\frac{\beta_1 S_1 m_2}{|J|},\end{aligned} \tag{S13}$$

$$\begin{aligned}\frac{\partial S_1^*}{\partial \mu} \propto (-1)^{1+5}\frac{M_{13}}{|J|} &= \frac{1}{|J|}\left(\frac{\partial f_2}{\partial S_2} - \beta_2 P\right)\left(m_1 m_2 - \beta_1 S_1 m_2\frac{\partial f_1}{\partial I_1}\right) - \frac{\beta_1 S_1\beta_2 P}{|J|}\left(\frac{\partial f_1}{\partial I_1}\frac{\partial f_2}{\partial I_2} - m_1\frac{\partial f_2}{\partial I_2}\right) \\ &+ \frac{m_1\beta_2 S_2}{|J|}\left(\frac{\partial f_2}{\partial S_2} - \beta_2 P\right)\frac{\partial f_1}{\partial I_2} + \frac{\beta_2 P}{|J|}\frac{\partial f_1}{\partial I_2}\left(\frac{\partial f_2}{\partial I_1}\beta_1 S_1 - m_1\beta_2 S_2\right) \\ &+ \frac{1}{|J|}\frac{\partial f_1}{\partial S_2}\left(\frac{\partial f_2}{\partial I_1}m_2\beta_1 S_1 + \frac{\partial f_2}{\partial I_2}m_1\beta_2 S_2 - \beta_2 S_2 m_1 m_2\right),\end{aligned} \tag{S14}$$

and

$$\begin{aligned}\frac{\partial}{\partial \mu}\left(\frac{I_1^*}{N_1^*}\right) \propto &\frac{P\beta_1^2 S_1^2}{m_1|J|}\frac{\partial f_1}{\partial I_1}\left(P\beta_2\frac{\partial f_2}{\partial I_2} - P\beta_2 m_2 + m_2\frac{\partial f_2}{\partial S_2}\right) \\ &+ \frac{\beta_1\beta_2 S_1^2 P}{|J|}\frac{\partial f_2}{\partial I_2}\left(\frac{\partial f_1}{\partial S_1} - \beta_1 P\right) + \frac{\beta_1 m_2 S_1^2}{|J|}\left(\frac{\partial f_1}{\partial S_1} - \beta_1 P\right)\left(\frac{\partial f_2}{\partial S_2} - \beta_2 P\right) \\ &- \frac{\beta_1 S_1^2}{m_1|J|}\left(P\beta_2\frac{\partial f_1}{\partial I_2} + m_2\frac{\partial f_1}{\partial S_2}\right)\left(P\beta_1\frac{\partial f_2}{\partial I_1} + m_1\frac{\partial f_2}{\partial S_1}\right).\end{aligned} \tag{S15}$$

Negative density dependence: In the absence of interspecific competition, equations (S13) and (S15) are negative. Thus, increased removal of infectious propagules causes the number and proportion of infected hosts to decrease. With increased interspecific competition, equations (S13) and (S15) become more positive.

We expect equations (S13) and (S15) will be negative for most systems. Equations (S13) and (S15) can be positive under two scenarios: (1) interspecific competition is greater than intraspecific competition ($\partial f_i/\partial X_j$ much larger than $\partial f_i/\partial X_i$ for all host classes X and $i \neq j$) and (2) interspecific competition between infected and susceptible hosts is greater than both intraspecific competition between infected and susceptible hosts ($\partial f_i/\partial I_j$ larger than $\partial f_i/\partial I_i$ for $i \neq j$) and interspecific competition between susceptible hosts ($\partial f_i/\partial I_j$ larger than $\partial f_i/\partial S_j$ for $i \neq j$).

Positive density dependence: If the positive density dependence is sufficiently weak, then the above results apply. If the positive density dependence is sufficiently strong, then any of the above predictions can be reversed. For example, if there is no interspecific competition and positive density dependence in host 1, then equations (S13) and (S15) can be positive.

Response to increased uptake: Because $\partial(dP/dt)/\partial\mu$ and $\partial(dP/dt)/\partial\beta_{ij}$ have the same sign, the signs of $\partial I_1^*/\partial\beta_{ij}$ and $\partial(I_1^*/N_1^*)/\partial\beta_{ij}$ are the same as the signs of $\partial I_1^*/\partial\mu$ and $\partial(I_1^*/N_1^*)/\partial\mu$, respectively.

Response to increased excretion rates: Because $\partial(dP/dt)/\partial\mu$ and $\partial(dP/dt)/\partial\chi_i$ have opposite signs, the signs of $\partial I_1^*/\partial\chi_i$ and $\partial(I_1^*/N_1^*)/\partial\chi_i$ are the opposite of the signs of $\partial I_1^*/\partial\mu$ and $\partial(I_1^*/N_1^*)/\partial\mu$, respectively.

S1.4.2 Response to increased mortality of host 2

The responses to increases in the mortality rate of I_2 are defined by

$$\begin{aligned}\frac{\partial I_1^*}{\partial m_2} \propto (-1)^{4+3}\frac{M_{43}}{|J|} &= (\chi_2 - u_{22}P)\frac{\beta_1 S_1}{|J|}\frac{\partial f_1}{\partial S_1}\left(\frac{\partial f_2}{\partial S_2} + \beta_2 P\right) + \frac{\beta_1 u_{22} S_1 P}{|J|}\frac{\partial f_1}{\partial S_1}\frac{\partial f_2}{\partial I_2} \\ &- (\chi_2 - u_{22}P)\frac{\beta_1}{|J|}\frac{\partial f_1}{\partial S_2}\left(P S_2\beta_2 + S_1\frac{\partial f_2}{\partial S_1}\right) - \frac{\beta_1 u_{22} S_1 P}{|J|}\frac{\partial f_1}{\partial I_2}\frac{\partial f_2}{\partial S_1} \\ &+ \frac{\beta_1 P}{|J|}\frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial I_2}(U^* + \mu - S_1 u_{11}) \\ &+ \frac{\beta_1\beta_2 P^2}{|J|}\frac{\partial f_1}{\partial I_2}(U^* + \mu - S_1 u_{11} - S_2 u_{22}) - \frac{\beta_1 P}{|J|}\frac{\partial f_1}{\partial I_2}\frac{\partial f_2}{\partial S_2}(U^* + \mu - S_1 u_{11}),\end{aligned} \tag{S16}$$

$$\begin{aligned}\frac{\partial S_1^*}{\partial m_2} \propto (-1)^{4+1}\frac{M_{41}}{|J|} &= (\chi_2 - u_{22}P)\frac{\beta_1 S_1}{|J|}\left(\frac{\partial f_2}{\partial S_2} - \beta_2 P\right)\left(m_1 - \frac{\partial f_1}{\partial S_1}\right) + \frac{u_{21} P\beta_1 S_1}{|J|}\frac{\partial f_2}{\partial I_2}\left(m_1 - \frac{\partial f_1}{\partial I_1}\right) \\ &+ \frac{(\chi_2 - u_{22}P)}{|J|}\frac{\partial f_1}{\partial S_2}\left(\beta_1 S_1\frac{\partial f_2}{\partial I_1} - m_1\beta_2 S_2\right) - \frac{u_{21}P}{|J|}\frac{\partial f_1}{\partial I_2}\left(m_1\beta_2 S_2 - \beta_1 S_1\frac{\partial f_2}{\partial I_1}\right) \\ &+ \frac{m_1(U^* + \mu) - (\chi_1 - u_{12}P)\beta_1 S_1}{|J|}\left[\frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial I_2} - \left(\frac{\partial f_2}{\partial I_2} - \beta_2 P\right)\frac{\partial f_1}{\partial I_2}\right],\end{aligned} \tag{S17}$$

and

$$\begin{aligned}\frac{\partial}{\partial m_2}\left(\frac{I_1^*}{N_1^*}\right) \propto & \frac{\beta_1 S_1^2}{m_1|J|}(\chi_2 - u_{21}P)\left(\frac{\partial f_2}{\partial S_2} - \beta_2 P\right)\left(P\beta_1\frac{\partial f_1}{\partial I_1} + m_1\frac{\partial f_1}{\partial S_1} - \beta_1 m_1 P\right) \\ & + \frac{\beta_1 u_{21} S_1^2 P}{m_1|J|}\frac{\partial f_2}{\partial I_2}\left(P\beta_1\frac{\partial f_1}{\partial I_1} + m_1\frac{\partial f_1}{\partial S_1} - \beta_1 m_1 P\right) \\ & - \frac{\beta_1 S_1^2}{m_1|J|}(\chi_2 - u_{21}P)\frac{\partial f_1}{\partial S_2}\left(\beta_1 P\frac{\partial f_2}{\partial I_1} + m_1\frac{\partial f_2}{\partial S_1}\right) - \frac{\beta_1 u_{21} S_1^2 P}{m_1|J|}\frac{\partial f_1}{\partial I_2}\left(\beta_1 P\frac{\partial f_2}{\partial I_1} + m_1\frac{\partial f_2}{\partial S_1}\right) \\ & + \frac{S_1 P}{|J|}\left(\beta_2 P\frac{\partial f_1}{\partial I_2} - \frac{\partial f_1}{\partial I_2}\frac{\partial f_2}{\partial S_2} + \frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial I_2}\right)(\chi_1 I_1 - u_{12} I_1 P - u_{11} S_1 P)\,.\end{aligned} \tag{S18}$$

Negative density dependence: In the absence of interspecific competition, only the first line of equation (S16) is nonzero and only the first two lines of equation (S18) are nonzero. The signs of the first term in each equation are $-(\chi_2 - u_{22}P)$ and the remaining nonzero terms are negative. This means increased removal of I_2 causes I_1 and I_1/N_1 to decrease unless infected hosts in population 2 are large sinks for infectious propagules, i.e., $\chi_2 - u_{22}P$ negative and large in magnitude. Increased interspecific host competition causes the signs of equations (S16) and (S18) to switch from negative to positive when host 2 is a small sink or source and from positive to negative when host 2 is a large sink.

We expect equations (S16) to be negative for most systems where host 2 is not a large sink and positive for most systems where host 2 is a large sink. Equations (S16) and (S18) have the opposite signs under two scenarios: (1) interspecific competition is greater than intraspecific competition ($\partial f_i/\partial X_j$ much larger than $\partial f_i/\partial X_i$ for all host classes X and $i \neq j$) and (2) interspecific competition between infected and susceptible hosts is greater than both intraspecific competition between infected and susceptible hosts ($\partial f_i/\partial I_j$ larger than $\partial f_i/\partial I_i$ for $i \neq j$) and interspecific competition between susceptible hosts ($\partial f_i/\partial I_j$ larger than $\partial f_i/\partial S_j$ for $i \neq j$).

Positive density dependence: If the positive density dependence is sufficiently weak, then the above results apply. If the positive density dependence is sufficiently strong, then any of the above predictions can be reversed.

S1.4.3 Response to host 1 experiencing increased interspecific competition

Let α_{12} be any parameter that negatively affects the reproduction rate of host 1. Mathematically, we assume $\partial f_1/\partial\alpha_{12} < 0$, which implies $\partial(dS_1/dt)/\partial\alpha_{12} < 0$. For example, a model with Lotka-Volterra competition, i.e., $f_1 = r_1(S_1 + b_1 I_1)[1 - \alpha_{11}(S_1 + c_1 I_1) - \alpha_{12}(S_2 + c_2 I_2)]$, satisfies these conditions. Note that our results apply to any parameter that negatively affects the growth rate of host 1. For the Lotka-Volterra example, our results apply to changes in α_{12} as well as changes in α_{11}, c_1, and c_2.

The responses to host 1 experiencing increased interspecific competition are defined by

$$\begin{aligned}\frac{\partial I_1^*}{\partial\alpha_{12}} \propto & \frac{(-1)^{1+3}M_{13}}{|J|} = (\chi_2 I_2 - u_{21}S_2 P - u_{22}I_2 P)\frac{\beta_1 m_2 P}{\beta_2 S_2|J|}\left(S_2\frac{\partial f_2}{\partial S_2} + S_1\frac{\partial f_2}{\partial S_1}\right) \\ & - \frac{\beta_1 P}{|J|}\left[\left(\frac{\partial f_2}{\partial S_2} - \beta_2 P\right)m_2 + \frac{\partial f_2}{\partial I_2}\beta_2 P\right](U^* + \mu - u_{11}S_1 - u_{21}S_2)\,,\end{aligned} \tag{S19}$$

$$\frac{\partial S_1^*}{\partial \alpha_{12}} \propto \frac{(-1)^{1+1}M_{11}}{|J|} = (\chi_2 - u_{22}P)\frac{m_1\beta_2 S_2}{|J|}\frac{\partial f_2}{\partial S_2} + (\chi_1 - u_{12}P)\frac{\beta_1 S_1}{|J|}\left(m_2\frac{\partial f_2}{\partial S_2} + \beta_2 S_2\frac{\partial f_2}{\partial I_2} - \beta_2 m_2 P\right)$$
$$- \frac{m_1}{|J|}(U^* + \mu)\left(m_2\frac{\partial f_2}{\partial S_2} + \beta_2 P\frac{\partial f_2}{\partial I_2}\right) + \frac{m_1 m_2}{|J|}(U^* + \mu - u_{21}S_2)$$
$$- (\chi_2 - u_{22}P)\frac{\beta_1 S_1 \beta_2 P}{|J|}\frac{\partial f_2}{\partial I_1} + \frac{m_2 u_{21} P \beta_1 S_1}{|J|}\frac{\partial f_2}{\partial I_1}, \tag{S20}$$

and

$$\frac{\partial}{\partial \alpha_{12}}\left(\frac{I_1^*}{N_1^*}\right) \propto \frac{-S_1 P}{|J|}\left[\chi_1 I_1 - u_{12}I_1 P - u_{11}S_1 P\right]\left(\beta_2 P\frac{\partial f_2}{\partial I_2} + m_2\frac{\partial f_2}{\partial S_2} - m_2\beta_2 P\right)$$
$$+ \frac{\beta_1 m_2 S_1^2 P}{\beta_2 m_1 S_2 |J|}\left[\chi_2 I_2 - u_{22}I_2 P - u_{21}S_2 P\right]\left(\beta_1 P\frac{\partial f_2}{\partial I_1} + m_1\frac{\partial f_2}{\partial S_1}\right). \tag{S21}$$

Negative density dependence: The second line of equation (S19) is negative and the sign of the first line of equation (S19) is determined by $\chi_2 I_2 - u_{21}S_2 P - u_{22}I_2 P$. This means that increased interspecific competition causes the number of infected individuals to decrease, unless host 2 is a large source ($\chi_2 - u_{22}P$ positive and large in magnitude).

The sign of the first line of equation (S21) is determined by $-(\chi_1 I_1 - u_{12}I_1 P - u_{11}S_1 P)$ and the sign of the second line of equation (S21) is determined by $\chi_2 I_2 - u_{22}I_2 P - u_{21}S_2 P$. Equation (S21) is negative when $\chi_1 I_1 - u_{12}I_1 P - u_{11}S_1 P$ is positive and sufficiently large or $\chi_2 I_2 - u_{22}I_2 P - u_{21}S_2 P$ is negative and sufficiently large. Biologically, this means that increased interspecific competition will cause the proportion of infected host to decrease unless the host 2 is a sufficiently large source (i.e., $\chi_2 - u_{22}P$ is positive and sufficiently large in magnitude).

Positive density dependence: Sufficiently strong positive density dependence in host 2 can reverse the above predictions.

S1.4.4 Response to host 2 experiencing increased competition

Let α_{22} be any parameter that negatively affects the reproduction rate of host 2. Mathematically, we assume $\partial f_2/\partial \alpha_{22} < 0$, which implies $\partial(dS_2/dt)/\partial \alpha_{22} < 0$. For example, a model with Lotka-Volterra competition, $f_2 = r_2(S_2 + b_2 I_2)[1 - \alpha_{22}(S_1 + c_1 I_1) - \alpha_{22}(S_2 + c_2 I_2)]$, satisfies these conditions. Note that our results apply to changes in any parameter that negatively affects the growth rate of host 2. For the Lotka-Volterra example, our results apply to changes in α_{22} as well as changes in α_{21}, c_1, and c_2.

THe responses to host 2 experiencing increased competition are defined by

$$\frac{\partial I_1^*}{\partial \alpha_{22}} \propto \frac{(-1)^{2+3} M_{23}}{|J|} = -\frac{\beta_1 m_2}{|J| S_2}\left(S_1 \frac{\partial f_1}{\partial S_1} + S_2 \frac{\partial f_1}{\partial S_2}\right)(\chi_2 I_2 - u_{21} S_2 P - u_{22} I_2 P) + \frac{\beta_1 P}{|J|}\left[m_2 \frac{\partial f_1}{\partial S_2} + \frac{\partial f_1}{\partial I_2}\beta_2 P\right](U^* + \mu - u_{11} S_1 - u_{21} S_2) \tag{S22}$$

$$= \frac{\beta_1 \beta_2 P^2}{|J|}\frac{\partial f_1}{\partial I_2}(U^* + \mu - u_{11} S_1 - u_{21} S_2) + \frac{\beta_2 m_2}{|J|}\frac{\partial f_1}{\partial S_2}(u_{22} P I_2 + \chi_1 I_1 - u_{11} S_1 P) - \frac{\beta_2 m_2 S_1}{S_2 |J|}\frac{\partial f_1}{\partial I_1}(\chi_2 I_2 - u_{21} S_2 P - u_{22} S_2 P), \tag{S23}$$

$$\frac{\partial S_1^*}{\partial \alpha_{22}} \propto \frac{(-1)^{2+1} M_{21}}{|J|} = (\chi_2 - u_{22} P)\frac{\beta_1 S_1 \beta_2 P}{|J|}\left(\frac{\partial f_1}{\partial I_1} - m_1\right) - \frac{u_{21} P m_2 \beta_1 S_1}{|J|}\left(\frac{\partial f_1}{\partial I_1} - m_1\right) + \frac{m_1 \beta_2 P}{|J|}\frac{\partial f_1}{\partial I_2}(U^* + \mu - u_{21} S_2) + \frac{m_1 m_2}{|J|}\frac{\partial f_1}{\partial S_2}(U^* + \mu) - \frac{\partial f_1}{\partial S_2}\left[(\chi_1 - u_{12} P) m_2 \beta_1 S_1 + (\chi_2 - u_{22} P) m_1 \beta_2 S_2\right], \tag{S24}$$

and

$$\frac{\partial}{\partial \alpha_{22}}\left(\frac{I_1^*}{N_1^*}\right) \propto -\frac{\beta_1 m_2 S_1^2 P}{\beta_2 S_2 m_1 |J|}\left[\chi_2 I_2 - u_{22} I_2 P - u_{21} S_2 P\right]\left(\beta_1 P \frac{\partial f_1}{\partial I_1} + m_1 \frac{\partial f_1}{\partial S_1} - m_1 \beta_1 P\right) + \frac{S_1 P}{|J|}\left[\chi_1 I_1 - u_{12} I_1 P - u_{11} S_1 P\right]\left(\beta_2 P \frac{\partial f_1}{\partial I_2} + m_2 \frac{\partial f_1}{\partial S_2}\right). \tag{S25}$$

Negative density dependence: The sign of the first line of equation (S22) is $-(\chi_2 I_2 - u_{22} I_2 P - u_{21} S_2 P)$ and the second line is positive. This means that increased intraspecific competition in host 2 causes an increase in the number of infected individuals in population 1 unless host 2 is a sufficiently large source. In addition, host 2 needs to be a larger source when interspecific host competition is stronger. That is, when interspecific competition is low, host 2 only needs to be a small source for equation (S22) to be negative, but when interspecific competition is high, host 2 needs to be a large source.

The sign of the first line of equation (S25) is $-(\chi_2 I_2 - u_{22} I_2 P - u_{21} S_2 P)$ and the sign of the second line is $\chi_1 I_1 - u_{12} I_1 P - u_{11} S_1 P$. Equation (S25) is negative when $\chi_1 - u_{12} P$ is negative and sufficiently large or $\chi_2 - u_{22} P$ is positive and sufficiently large. Biologically, this means that increased intraspecific competition will cause the proportion of infected hosts in population 1 to increase unless the host 2 is a sufficiently large source of infectious propagules.

Positive density dependence: The above predictions can be reversed if host 1 has sufficiently strong positive density dependence at equilibrium.

S1.4.5 Response to increased infection rates of host 2

The responses to increases in the infection rate of host 2 are defined by

$$\begin{aligned}\frac{\partial I_1^*}{\partial \beta_2} = &\frac{-\beta_1 u_{22} S_1 P}{|J|}\frac{\partial f_1}{\partial S_1}\left(\frac{\partial f_2}{\partial I_2} - m_2\right) - (\chi_2 - u_{22}P)\frac{\beta_1 S_1}{|J|}\left(\frac{\partial f_1}{\partial S_1}\frac{\partial f_2}{\partial S_2} - \frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial S_1}\right)\\ &+ \frac{\beta_1 P}{|J|}(U^* + \mu - u_{11}S_1)\left(\frac{\partial f_1}{\partial I_2}\frac{\partial f_2}{\partial S_2} - \frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial I_2} + m_2\frac{\partial f_1}{\partial S_2}\right) + \frac{\beta_1 u_{22} S_1 P}{|J|}\frac{\partial f_1}{\partial I_2}\frac{\partial f_2}{\partial S_1},\end{aligned} \tag{S26}$$

$$\frac{\partial S_1^*}{\partial \beta_2} = -\frac{\partial}{\partial \beta_2}\left(\frac{dS_2}{dt}\right)\frac{(-1)^{2+1}M_{21}}{|J|} - \frac{\partial}{\partial \beta_2}\left(\frac{dI_2}{dt}\right)\frac{(-1)^{4+1}M_{41}}{|J|} = \frac{S_2 P}{|J|}(M_{41} - M_{21}), \tag{S27}$$

where M_{ij} is the i, j minor of J, and

$$\begin{aligned}\frac{\partial}{\partial \beta_2}\left(\frac{I_1^*}{N_1^*}\right) \propto &-\frac{(\chi_2 - u_{22}P)}{|J|}\frac{\partial f_2}{\partial S_2}\left(\beta_1 P\frac{\partial f_1}{\partial I_1} + m_1\frac{\partial f_1}{\partial S_1} - \beta_1 m_1 P\right)\\ &- \frac{u_{21}P}{|J|}\left(\frac{\partial f_2}{\partial I_2} - m_2\right)\left(\beta_1 P\frac{\partial f_1}{\partial I_1} + m_1\frac{\partial f_1}{\partial S_1} - \beta_1 m_1 P\right)\\ &+ \frac{(\chi_2 - u_{22}P)}{|J|}\frac{\partial f_1}{\partial S_2}\left(\beta_1 P\frac{\partial f_2}{\partial I_1} + m_2\frac{\partial f_2}{\partial S_1}\right) - (\chi_1 - u_{12}P)\frac{\beta_1 P}{|J|}\frac{\partial f_1}{\partial S_2}\left(\frac{\partial f_2}{\partial I_2} - m_2\right)\\ &+ \frac{m_1 u_{11}P}{|J|}\frac{\partial f_1}{\partial S_2}\left(\frac{\partial f_2}{\partial I_2} - m_2\right) + \frac{u_{21}P}{|J|}\frac{\partial f_1}{\partial I_2}\left(\beta_1 P\frac{\partial f_2}{\partial I_1} + m_1\frac{\partial f_2}{\partial S_1}\right)\\ &+ (\chi_1 I_1 - u_{12}I_1 P - u_{11}S_1 P)\frac{m_1 P}{\beta_1 S_1 |J|}\frac{\partial f_1}{\partial I_2}\frac{\partial f_2}{\partial S_2}.\end{aligned} \tag{S28}$$

Negative density dependence: In the absence of interspecific competition, equations (S26) and (S28) are positive unless $\chi_2 - u_{22}P$ is negative and large in magnitude. This means that increasing β_2 causes the number and proportion of infections in population 1 to increase unless host 2 is a large sink for infectious propagules.

Increased interspecific competition makes equation (S26) more negative unless $\chi_2 - u_{22}P$ is negative and large in magnitude. Increased interspecific competition makes equation (S28) more negative unless $\chi_2 - u_{22}P$ or $\chi_1 - u_{12}P$ are negative and large in magnitude. Increased interspecific competition can cause equations (S26) and (S28) to change sign when (1) interspecific competition is much greater than intraspecific competition ($\partial f_i/\partial X_j$ much larger than $\partial f_i/\partial X_i$ for all host classes X and $i \neq j$), (2) interspecific competition between infected and susceptible hosts is greater than both intraspecific competition between infected and susceptible hosts ($\partial f_i/\partial I_j$ larger than $\partial f_i/\partial I_i$ for $i \neq j$) and interspecific competition between susceptible hosts ($\partial f_i/\partial I_j$ larger than $\partial f_i/\partial S_j$ for $i \neq j$), or (3) host 2 is a large source and host 1 is a large sink ($\chi_1 - u_{11}P$ negative and large in magnitude).

Overall, we expect increased transmission rates of host 2 to (i) increase the number and proportion of infected hosts in population 1 when interspecific competition is low unless host 2 is a large sink, (ii) decrease the number of infected hosts in population 1 when interspecific

competition is high unless host 2 is a large sink, and (iii) decrease the proportion of infected hosts in population 1 when interspecific competition is high unless either host is a sink.

Positive density dependence: As expected, all of the above predictions can be reversed if one or both hosts have sufficiently strong positive density dependence at equilibrium.

S1.5 Relationships between environmental and direct transmission models

The first subsection identifies conditions under which the environmental transmission model (1) reduces to a density dependent or frequency dependent direct transmission model. The second subsection provides interpretation for an important quantity in the calculations. The remaining subsections present the changes in parameter values that hold either the sympatric equilibrium densities (p^*) constant or the allopatric equilibrium densities ($\hat{p}$) constant.

S1.5.1 Model equivalences in the limit of fast infectious propagule dynamics

To reduce the environmental transmission model (1) to a density dependent direct transmission model, we assume there is no uptake of infectious propagules by any host class ($u_{ij} = 0$ for all i and j) and that χ_i and μ are sufficiently large that there is a separation of time scales between the dynamics of the infectious propagules and the dynamics of the host classes. Under these assumptions, the dynamics of the infectious propagules reach a quasi-steady state equilibrium density defined by $P = \chi_1 I_1/\mu + \chi_2 I_2/\mu$. Substitution into the equations for the host dynamics yields a density dependent direct transmission model

$$\begin{aligned} \frac{dS_i}{dt} &= \underbrace{f_i(S_1, S_2, I_1, I_2)}_{\text{growth \& competition}} - \underbrace{\bar{\beta}_{i1} I_1 S_i + \bar{\beta}_{i2} I_2 S_i}_{\text{infection}} \\ \frac{dI_i}{dt} &= \underbrace{\bar{\beta}_{i1} I_1 S_i + \bar{\beta}_{i2} I_2 S_i}_{\text{infection}} \underbrace{-m_i I_i}_{\text{mortality}} \end{aligned} \tag{S29}$$

where the direct transmission coefficients are $\bar{\beta}_{ji} = \beta_i \chi_j/\mu$. The equilibria of model (S29) are identical to those of model (1) when $u_{ij} = 0$. Thus, all of our equilibrium-based results apply to density dependent direct transmission SI models.

To reduce the environmental transmission model (1) to a frequency dependent direct transmission model, we assume there is no degradation of infectious propagules ($\mu = 0$) and that χ_i and u_{ij} are sufficiently large such that there is a separation of time scales between the dynamics of the infectious propagules and the dynamics of the host classes. Under these assumptions, the dynamics of the infectious propagules reach a quasi-steady state equilibrium density defined by $P = (\chi_1 I_1 + \chi_2 I_2)/(u_{11} S_1 + u_{12} I_1 + u_{21} S_2 + u_{22} I_2)$. Substitution into the equations for the host dynamics yields a frequency dependent direct transmission model

$$\begin{aligned} \frac{dS_i}{dt} &= \underbrace{f_i(S_1, S_2, I_1, I_2)}_{\text{growth \& competition}} - \underbrace{\frac{\beta_i(\chi_1 I_1 + \chi_2 I_2) S_i}{(u_{11} S_1 + u_{12} I_1 + u_{21} S_2 + u_{22} I_2)}}_{\text{infection}} \\ \frac{dI_i}{dt} &= \underbrace{\frac{\beta_i(\chi_1 I_1 + \chi_2 I_2) S_i}{(u_{11} S_1 + u_{12} I_1 + u_{21} S_2 + u_{22} I_2)}}_{\text{infection}} \underbrace{-m_i I_i}_{\text{mortality}} \end{aligned} \tag{S30}$$

where the transmission rates depend on the weighted frequency of susceptible hosts in the community. The equilibria of model (S30) and (1) are identical when $\mu = 0$. Thus, all of our equilibrium-based results apply to frequency dependent direct transmission SI models.

S1.5.2 Factors affecting the sign of $U^* - \hat{U}$

Here, we focus on determining factors that effect the sign of $U^* - \hat{U}$. This is needed because for both changes of parameters presented in the following subsections, the signs of effects of the transformation on the host densities are influenced by the sign of $U^* - \hat{U}$. Using the equilibrium conditions $dP/dt = 0$ and $dI_1/dt = 0$ for the allopatric and sympatric models, we can rewrite $U^* - \hat{U}$ as

$$U^* - \hat{U} = \frac{\chi_1 I_1^*}{P^*} + \frac{\chi_2 I_2^*}{P^*} - \frac{\chi_1 \hat{I}_1}{\hat{P}} \tag{S31}$$

$$= \frac{\chi_1 \beta_1}{m_1}(S_1^* - \hat{S}_1) + \frac{\chi_2 \beta_2}{m_2} S_2^*. \tag{S32}$$

Before giving general conditions, we start with a few special cases.

Special case 1: No interspecific host competition: In this case, the magnitudes of S_1^* and S_2^* are determined by the competence and intraspecific competitive ability of each host. $U^* - \hat{U}$ is more likely to be positive when host 2 has lower competence and lower intraspecific competitive ability, because both of these make S_1^* and S_2^* larger. Conversely, $U^* - \hat{U}$ is more likely to be negative when host 2 has higher competence and higher intraspecific competitive ability, because both of these make S_1^* and S_2^* smaller.

Special case 2: Symmetric uptake: Assuming $u_{ij} = u_i$ results in

$$U^* - \hat{U} = u_1(N_1^* - \hat{N}_1) + u_2 N_2^* \tag{S33}$$

where $N_i = S_i + I_i$. The total population size for each host at the sympatric equilibrium is determined solely by the levels of intraspecific and interspecific host competition and the amount of disease induced mortality. In particular, N_i^* is lower with increased interspecific competition, increased intraspecific competitive ability of host i, and increased numbers of infected individuals in host i. $U^* - \hat{U}$ is more likely to be positive when interspecific competition is weak, host 2 is a weaker intraspecific competitor, and host 2 is a lower competence host. Conversely, $U^* - \hat{U}$ is more likely to be negative when interspecific competition is strong, and host 2 is a strong intraspecific competitor, and a higher competence host.

Special case 3: No uptake by infected hosts: Assuming $u_{i2} = 0$ yields

$$u_{11}(S_1^* - \hat{S}_1) + u_{21} S_2 = U^* - \hat{U} = \frac{\chi_1 \beta_1}{m_1}(S_1^* - \hat{S}_1) + \frac{\chi_2 \beta_2}{m_2} S_2^*. \tag{S34}$$

Setting the left and right hand sides equal to each other, solving for $S_1^* - \hat{S}_1$, substituting into equation (S32), and some algebraic manipulation yields

$$U^* - \hat{U} = u_{21} S_2^* \left(1 - \frac{\frac{\chi_2 \beta_2}{m_2 u_{21}} - 1}{\frac{\chi_1 \beta_1}{m_1 u_{11}} - 1} \right) = u_{21} S_2^* \left(1 - \frac{\mathcal{R}_2(\infty) - 1}{\mathcal{R}_1(\infty) - 1} \right). \tag{S35}$$

Here, $\mathcal{R}_i(\infty) = \chi_i \beta_i / m_i u_{i1}$ is the basic reproductive number for the pathogen in an allopatric system where the density of host i is infinite. It determines if the pathogen can invade the

allopatric system in the limit where there are an infinite number of susceptible hosts. We interpret more positive values of $\mathcal{R}_i(\infty)$ to mean that host i is a higher competence host for the pathogen. If $\mathcal{R}_i(\infty) < 1$, then the pathogen cannot invade a completely susceptible population of host i of any size. We assume $\mathcal{R}_1(\infty) > 1$ because this is a necessary condition to apply our either of our change of parameters.

Equation (S35) is positive whenever $\mathcal{R}_1(\infty) > \mathcal{R}_2(\infty)$ and equation (S35) is negative whenever $\mathcal{R}_1(\infty) < \mathcal{R}_2(\infty)$. Thus, $U^* - \hat{U}$ is positive when host 2 is a lower competence host than host 1 and $U^* - \hat{U}$ is negative when host 2 is a higher competence host than host 1.

General Case: In general, S_i^* will be smaller in magnitude when host j is a stronger interspecific competitor, host i is a stronger intraspecific competitor, and host j is a higher competence host. Thus, $U^* - \hat{U}$ is more likely to be positive when (i) interspecific competition is weaker, (ii) host 2 is a weaker intraspecific competitor, and (iii) host 2 is a lower competence host. In contrast, $U^* - \hat{U}$ is more likely to be negative when (i) interspecific competition is stronger, (ii) host 2 is a stronger intraspecific competitor, and (iii) host 2 is a higher competence host.

S1.5.3 Parameter transformation that holds the sympatric equilibrium constant

Here we present a continuous change of parameters between $\mu = 0$ and $u_{ij} = 0$ that holds the sympatric equilibrium (p^*) constant. This change of variables converts the environmental transmission model from a form that behaves like a frequency dependent direct transmission model (at equilibrium) to a form that behaves like a density dependent direct transmission model (at equilibrium), while leaving the host and infectious propagule densities at the sympatric equilibrium unchanged.

Change of parameters: We assume model (1) is parameterized such that $u_{ij} \neq 0$ for at least one i, j pair; our approach cannot be applied to models where $u_{ij} = 0$ for all i, j. We rewrite the infectious propagule equation for the sympatric system as

$$\frac{dP}{dt} = \chi_1 I_1 - f(q)(u_{11}S_1 + u_{12}I_1)P - q\mu P \tag{S36}$$

and similarly we rewrite the infectious propagule equation for the sympatric system as

$$\frac{dP}{dt} = \chi_1 I_1 + \chi_2 I_2 - f(q)(u_{11}S_1 + u_{12}I_1 + u_{21}S_2 + u_{22}I_2)P - q\mu P \tag{S37}$$

where

$$f(q) = 1 + \frac{\mu}{U^*} - \frac{\mu}{U^*}q, \qquad \text{for } 0 \leq q \leq 1 + U^*/\mu \tag{S38}$$

and U^* is the total per infectious propagule uptake rate at the sympatric equilibrium. We assume the sympatric and allopatric equilibria exist with positive densities for $q \in [0, 1 + U^*/\mu]$.

At $q = 0$, there is no degradation and uptake rates are increased by the factor $f(0) = 1 + \mu/U^*$. In this case the allopatric and sympatric equilibria of the environmental transmission model (1) are identical to those of the frequency dependent direct transmission model (S30) with uptake rates $u_{ij}(1 + \mu/U^*)$. At $q = 1$, $f(1) = 1$ and the environmental transmission model is unchanged. At $q = 1 + U^*/\mu$, there is no uptake and the degradation rate is $\mu + U^*$. In this case, the allopatric and sympatric equilibria of the environmental transmission model (1) are identical to those of a density dependent direct transmission model (S29) with degradation rate $\mu + U^*$.

The densities at the sympatric equilibrium (p^*) are independent of the value of q. This is because the sum of the degradation and total per capita uptake rates at equilibrium in the sympatric model is held fixed at $U^* + \mu$ as q is varied.

Effect on loss rate at allopatric equilibrium: Varying q changes the infectious propagule growth rate (dP/dt) in the allopatric model through its effect on the total per capita loss rate of infectious propagules ($U + \mu$). To see how the loss rate changes in the allopatric model, we can compute the difference in the loss rates at equilibrium for $q = 1$ and $q = 1 + \hat{U}/\mu$,

$$\text{[loss rate for q=1 + } U^*/\mu\text{]} - \text{[loss rate for q=1]} = U^* + \mu - (\hat{U} + \mu) = U^* - \hat{U}. \tag{S39}$$

Similarly, we can compute the difference in the loss rates at equilibrium for $q = 0$ and $q = 1$,

$$[\text{loss rate for q=1}] - [\text{loss rate for q=0}] = \hat{U} + \mu - \left(1 + \frac{\mu}{U^*}\right)\hat{U} = \frac{\mu}{U^*}(U^* - \hat{U}). \tag{S40}$$

In both cases, the effect of increasing q on the total per capita loss rate of infectious propagules depends on the sign of $U^* - \hat{U}$.

Another way to see how varying q affects the per capita infectious propagule loss rate is to compute the partial derivative,

$$\frac{\partial}{\partial q}\frac{dP}{dt} = \frac{\partial}{\partial q}\left[\chi_1 I_1 - f(q)\overbrace{(u_{11}S_1 + u_{12}I_1 + u_{13}R_1)}^{\text{total per capita uptake},U} - q\mu P\right] \tag{S41}$$

$$= \frac{\mu}{U^*}UP - \mu P = -P\mu(U^* - U)/U^*. \tag{S42}$$

When evaluated at the allopatric equilibrium, $\hat{p}$, the above shows that increasing q decreases the infectious propagule growth rate (i.e., increases the total infectious propagule per capita loss rate) of the allopatric model when $U^* - \hat{U} > 0$.

In total, if $U^* - \hat{U}$ is positive, then the total per capita loss rate will increase as the environmental transmission model is changed from a form that behaves like a frequency dependent direct transmission model ($q = 0$) into a form that behaves like a density dependent direct transmission model ($q = 1+\hat{U}/\mu$). If $U^*-\hat{U}$ is negative, then the opposite is true.

Effect on allopatric equilibrium densities: Because varying q changes the total loss rate of infectious propagules, varying q changes the densities at the allopatric equilibrium, $\hat{p}$. The effects of varying q are determined by

$$\frac{\partial \hat{I}_1}{\partial q} = -\frac{\partial}{\partial q}\left(\frac{dP}{dt}\right)\frac{(-1)^{2+3}M_{32}}{|\hat{J}|} \propto -(U^* - \hat{U})\frac{M_{32}}{|J|} = -(U^* - \hat{U})\frac{\beta_1 S_1}{|\hat{J}|}\frac{\partial f_1}{\partial S_1}, \tag{S43}$$

$$\frac{\partial \hat{S}_1}{\partial q} = -\frac{\partial}{\partial q}\left(\frac{dP}{dt}\right)\frac{(-1)^{1+3}M_{31}}{|\hat{J}|} \propto (U^* - \hat{U})\frac{M_{31}}{|\hat{J}|} = (U^* - \hat{U})\frac{\beta_1 S_1}{|\hat{J}|}\frac{\partial f_1}{\partial S_1} \tag{S44}$$

and

$$\frac{\partial}{\partial q}\left(\frac{\hat{I}_1}{\hat{N}_1}\right) = \frac{1}{\hat{N}_1^2}\left[\hat{S}_1\frac{\partial \hat{I}_1}{\partial q} - \hat{I}_1\frac{\partial \hat{S}_1}{\partial q}\right] \propto -(U^* - \hat{U})\frac{1}{|\hat{J}|}\left[\hat{S}_1\frac{\partial f_1}{\partial S_1} + \hat{I}_1\frac{\partial f_1}{\partial I_1}\right] \tag{S45}$$

where $|\hat{J}| < 0$, M_{31} and M_{32} are submatrices of matrix (S1), and all terms are evaluated at $\hat{p}$.

Negative density dependence of host 1 at equilibrium: We assume that host 1 has negative density dependence at the allopatric equilibrium, i.e., $\partial f_1/\partial X_1 < 0$ for $X \in \{S, I\}$.

If $U^* - \hat{U} > 0$, then equations (S43) and (S45) are negative. This implies $I_1^* - \hat{I}_1$ and $(I_1^*/N_1^*) - (\hat{I}_1/\hat{N}_1)$ are more positive under frequency dependent direct transmission than density dependent direct transmission, i.e., greater dilution (or less amplification) in host 1

occurs under frequency dependent direct transmission than under density dependent direct transmission. If $U^* - \hat{U} < 0$, then equations (S43) and (S45) are positive. In this case, $I_1^* - \hat{I}_1$ and $(I_1^*/N_1^*) - (\hat{I}_1/\hat{N}_1)$ are more negative under frequency dependent direct transmission than density dependent direct transmission, i.e., greater dilution (or less amplification) in host 1 occurs under density dependent direct transmission than under frequency dependent direct transmission.

In total, we predict that frequency dependent direct transmission leads to more dilution and a greater reduction in infected density in host 1 than density dependent direct transmission when:

- (D1) Interspecific competition is weaker, (D2) host 2 is a weaker intraspecific competitor, and (D3) host 2 a lower competence host.

We also predict that density dependent direct transmission leads to more dilution and a greater reduction in infected density in host 1 than frequency dependent direct transmission when:

- (D4) Interspecific competition is stronger, (D5) host 2 is a stronger intraspecific competitor, and (D6) host 2 a higher competence host.

Positive density dependence of host 1 at equilibrium: If host 1 has sufficiently strong positive density dependence in either class or positive density dependence in both classes at the allopatric equilibrium, then the above predictions are reversed.

S1.5.4 Parameter transformation that holds the allopatric equilibrium constant

Here we present a continuous change of parameters between $\mu = 0$ and $u_{ij} = 0$ that holds the allopatric equilibrium ($\hat{p}$) constant. This change of variables converts the environmental transmission model from a form that behaves like a frequency dependent direct transmission model (at equilibrium) to a form that behaves like a density dependent direct transmission model (at equilibrium), while leaving the densities at the allopatric equilibrium for host 1 unchanged.

Change of Parameters: We assume model (1) is parameterized such that $u_{ij} \neq 0$ for at least one i, j pair; our approach cannot be applied to models where $u_{ij} = 0$ for all i, j. We rewrite the infectious propagule equation for the allopatric system as

$$\frac{dP}{dt} = \chi_1 I_1 - f(q)(u_{11}S_1 + u_{12}I_1)P - q\mu P \tag{S46}$$

and similarly we rewrite the infectious propagule equation for the sympatric system as

$$\frac{dP}{dt} = \chi_1 I_1 + \chi_2 I_2 - f(q)(u_{11}S_1 + u_{12}I_1 + u_{21}S_2 + u_{22}I_2)P - q\mu P \tag{S47}$$

where

$$f(q) = \frac{\hat{U} + \mu}{\hat{U}} - \left(\frac{\hat{U} + \mu}{\hat{U}} - 1\right) q = 1 + \frac{\mu}{\hat{U}} - \frac{\mu}{\hat{U}} q, \qquad 0 \leq q \leq 1 + \hat{U}/\mu \tag{S48}$$

and $\hat{U} = u_{11}\hat{S}_1 + u_{12}\hat{I}_1$ is the total per infectious propagule uptake rate at the allopatric equilibrium for host 1. We assume the sympatric and allopatric equilibria exist with positive densities for $q \in [0, 1 + \hat{U}/\mu]$.

At $q = 0$, there is no degradation and the uptake rates are increased by the factor $f(0) = 1 + \mu/\hat{U}$. In this case the allopatric and sympatric equilibria of the environmental transmission model (1) are identical to those of a frequency dependent direct transmission model (S30) with uptake rates $u_{ij}(1+\mu/\hat{U})$. At $q = 1$, the environmental transmission model is unchanged. At $q = 1 + \hat{U}/\mu$, there is no uptake and the degradation rate is $\mu + \hat{U}$. In this case the allopatric and sympatric equilibria of the environmental transmission model (1) are identical to those of a density dependent direct transmission model (S29) with degradation rate $\mu + \hat{U}$.

The densities at the allopatric equilibrium ($\hat{p}$) are independent of the value of q. This is because the sum of the degradation and total per capita uptake rates at equilibrium in the allopatric model is held fixed at $\hat{U} + \mu$ as q is varied.

Effect on loss rate at sympatric equilibrium: Varying q changes the infectious propagule growth rate (dP/dt) in the sympatric model (1) through its effect on the total per capita loss rate of infectious propagules ($U + \mu$). To see how it changes, we compare the loss rates at equilibrium when $q = 1$ and when $q = 1 + \hat{U}/\mu$. This yields

$$[\text{loss rate for q}{=}1 + \hat{U}/\mu] - [\text{loss rate for q}{=}1] = \hat{U} + \mu - (U^* + \mu) = \hat{U} - U^*. \tag{S49}$$

Similarly, we can compute difference in the loss rate at equilibrium for $q = 0$ and $q = 1$. This yields

$$[\text{loss rate for q=1}] - [\text{loss rate for q=0}] = U^* + \mu - \left(1 + \frac{\mu}{\hat{U}}\right) U^* = \frac{\mu}{\hat{U}}(\hat{U} - U^*). \quad \text{(S50)}$$

In both cases, the effect of increasing q on the total per capita loss rate of infectious propagules depends on the sign of $\hat{U} - U^*$.

Another way to see how varying q affects the per capita infectious propagule loss rate is to compute the partial derivative,

$$\frac{\partial}{\partial q}\frac{dP}{dt} = \frac{\partial}{\partial q}\left[\chi_1 I_1 + \chi_2 I_2 - f(q)\overbrace{(u_{11}S_1 + u_{12}I_1 + u_{13}R_1 + u_{21}S_2 + u_{22}I_2 + u_{23}R_2)}^{\text{total per capita uptake},U} - q\mu P\right] \quad \text{(S51)}$$

$$= \frac{\mu}{\hat{U}}UP - \mu P = P\mu\hat{U}(U - \hat{U}). \quad \text{(S52)}$$

When evaluated at the sympatric equilibrium, p^*, the above shows that increasing q decreases the infectious propagule growth rate (i.e., increases the total infectious propagule per capita loss rate) of the allopatric model when $U^* - \hat{U} < 0$.

In total, if $U^* - \hat{U}$ is negative, then the total per capita loss rate will increase as the environmental transmission model is continuously changed from a form that behaves like a frequency dependent direct transmission model ($q = 0$) into a form that behaves like a density dependent direct transmission model ($q = 1 + \hat{U}/\mu$). If $U^* - \hat{U}$ is positive, then the opposite is true.

Effect on sympatric equilibrium densities: Because varying q changes the total loss rate of infectious propagules in the sympatric model, varying q changes the densities at the sympatric equilibrium, p^*. The effects of varying q are determined by

$$\begin{aligned}\frac{\partial I_1^*}{\partial q} &= -\frac{\partial}{\partial q}\left(\frac{dP}{dt}\right)\frac{(-1)^{3+5}M_{53}}{|J|} \\ &\propto -(U^* - \hat{U})\left[\frac{\beta_1 S_1}{|J|}\left(m_2\frac{\partial f_1}{\partial S_1}\frac{\partial f_2}{\partial S_2} + \beta_2 P\frac{\partial f_1}{\partial S_1}\frac{\partial f_2}{\partial I_2} - m_2\beta_2 P\frac{\partial f_1}{\partial S_1}\right)\right. \\ &\quad \left. + \frac{\beta_1\beta_2 P}{|J|}\left(\frac{\partial f_2}{\partial I_2}S_2\frac{\partial f_1}{\partial S_2} - \frac{\partial f_1}{\partial I_2}\left(S_1\frac{\partial f_2}{\partial S_1} + S_2\frac{\partial f_2}{\partial S_2}\right)\right) - \frac{\beta_1 S_1 m_2}{|J|}\frac{\partial f_1}{\partial S_2}\frac{\partial f_2}{\partial S_1}\right],\end{aligned} \quad \text{(S53)}$$

$$\frac{\partial S_1^*}{\partial q} = -\frac{\partial}{\partial q}\left(\frac{dP}{dt}\right)\frac{(-1)^{1+5}M_{51}}{|J|} \propto -(U^* - \hat{U})\frac{M_{51}}{|J|}, \quad \text{(S54)}$$

and

$$\frac{\partial}{\partial q}\left(\frac{I_1^*}{N_1^*}\right) \propto -(U^*-\hat{U})\Bigg[\frac{P\beta_1^2 S_1^2}{m_1|J|}\frac{\partial f_1}{\partial I_1}\left(P\beta_2\frac{\partial f_2}{\partial I_2}-P\beta_2 m_2+m_2\frac{\partial f_2}{\partial I_2}\right) + \frac{\beta_1\beta_2 S_1^2 P}{|J|}\frac{\partial f_2}{\partial I_2}\left(\frac{\partial f_1}{\partial I_1}-\beta_2\right)+\frac{\beta_1 m_2 S_1^2}{|J|}\left(\frac{\partial f_1}{\partial S_1}-\beta_2 P\right)\left(\frac{\partial f_2}{\partial S_2}-\beta_2 P\right) - \frac{\beta_1 S_1^2}{m_1|J|}\left(P\beta_2\frac{\partial f_1}{\partial I_2}+m_2\frac{\partial f_1}{\partial S_2}\right)\left(P\beta_2\frac{\partial f_2}{\partial I_1}+m_1\frac{\partial f_2}{\partial S_1}\right)\Bigg] \quad \text{(S55)}$$

where all terms are evaluated at p^* and $M_{51}/|J|$ is given in equation (S14). Note that the conditions defining the signs of equations (S53) and (S55) are combinations of the conditions listed in section S1.4.1 and S1.5.2 because $\frac{\partial X_1^*}{\partial q} \propto -(U^*-\hat{U})\frac{\partial X_1^*}{\partial \mu}$ for $X = S, I$.

Negative density dependence at equilibrium: We assume both hosts have negative density dependence at equilibrium, i.e., $\partial f_i/\partial X_i < 0$ for $X \in \{S, I, R\}$. Equations (S53) and (S55) are positive when either (i) $U^* - \hat{U} > 0$ and the terms in brackets are negative or (ii) $U^* - \hat{U} < 0$ and the terms in brackets are positive. In these cases, $I_1^* - \hat{I}_1$ and $I_1^*/N_1^* - \hat{I}_1/\hat{N}_1$ are more negative under frequency dependent direct transmission than density dependent direct transmission. This means frequency dependent direct transmission results in a smaller density and proportion of infected individuals than density dependent direct transmission. Conversely, equations (S53) and (S55) are negative when either (i) $U^* - \hat{U} < 0$ and the terms in brackets are negative or (ii) $U^* - \hat{U} > 0$ and terms in brackets are positive. In these cases, $I_1^* - \hat{I}_1$ and $I_1^*/N_1^* - \hat{I}_1/\hat{N}_1$ are more positive under frequency dependent direct transmission than density dependent direct transmission. This means density dependent direct transmission results in a smaller density and proportion of infected individuals than frequency dependent direct transmission.

Recall from subsection S1.4.1 that the terms in brackets in equations (S53) and (S55) are negative when interspecific host competition is sufficiently low and positive when (1) interspecific competition is greater than intraspecific competition or (2) competition between infected and susceptible hosts is stronger than competition between susceptible hosts. Also recall from subsection S1.5.2 that positive values of $U^* - \hat{U}$ are promoted when (a) interspecific competition is weak, and host 2 is (b) a low competence host and (c) a weak intraspecific competitor. Combining this yields the following predictions.

- Assume interspecific competition between hosts is less than intraspecific competition and infected hosts are weaker competitors than susceptible hosts
  - Frequency dependent direct transmission is more likely to reduce the number and proportion of infected individuals in host 1 compared to density dependent direct transmission when (D1) interspecific competition is weaker, (D2) host 2 is a weaker intraspecific competitor, and (D3) host 2 is a lower competence host.
  - Density dependent direct transmission is more likely to reduce the number and proportion of infected individuals in host 1 compared to frequency dependent direct transmission when (D4) interspecific competition is stronger, (D5) host 2 is a stronger intraspecific competitor, and (D6) host 2 is a higher competence host.

- Assume interspecific competition between hosts is greater than intraspecific competition or infected hosts are stronger competitors than susceptible hosts

  - Frequency dependent direct transmission is more likely to reduce the number and proportion of infected individuals in host 1 compared to density dependent direct transmission when (F1) host 2 is a stronger intraspecific competitor and (F2) host 2 is a higher competence host.
  - Density dependent direct transmission is more likely to reduce the number and proportion of infected individuals in host 1 compared to frequency dependent direct transmission when (F3) host 2 is a weaker intraspecific competitor and (F4) host 2 is a lower competence host.

We note three things about the above. First, conditions D1-D6 in this section are identical to the conditions with the same labels in section S1.5.3 (hence the duplicate labeling). This agreement is expected because in both cases increased removal of infectious propagules causes the number and proportion of infected individuals in the focal host to decrease (i.e, $\partial \hat{I}_1/\partial\mu$, $\partial(\hat{I}_1/\hat{N}_1)/\partial\mu$, $\partial I_1^*/\partial\mu$, and $\partial(I_1^*/N_1^*)/\partial\mu$ are all negative). Second, conditions F1-F4 differ from conditions D1-D6 because increased removal of infectious propagules causes the number and proportion of infected individuals in host 1 at the sympatric equilibrium to increase (i.e, $\partial I_1^*/\partial\mu$ and $\partial(I_1^*/N_1^*)/\partial\mu$ positive). This disagreement is also expected because interspecific host competition does not influence the response to increased removal of infectious propagules in the allopatric model; $\partial \hat{I}_1/\partial\mu$ is independent of interspecific competition because there is only one host species in the allopatric model.

Third, if interspecific competition is absent and there is negative density dependence in host 1, it is not possible for host 1 to experience amplification at $q = 0$ and dilution at $q = 1+\hat{U}/\mu$. The proof by contradiction is the following. Under the assumed negative density dependence, amplification at $q = 0$ and dilution at $q = 1+\hat{U}/\mu$ is only possible if $U^* - \hat{U} < 0$, which implies $S_1^* - \hat{S}_1 < 0$ and sufficiently large for $0 \leq q \leq 1 + \hat{U}/\mu$. Because interspecific competition is absent, the equilibrium host densities satisfy $0 = f_1(S_1^*, I_1^*) - m_1 I_1^*$. The dependence of S_1^* on I_1^* can be computed using implicit differentiation,

$$0 = \frac{\partial f_1}{\partial S_1}\frac{\partial S_1^*}{\partial I_1^*} + \frac{\partial f_1}{\partial I_1} - m_1\bigg|_{p^*} \quad \Rightarrow \quad \frac{\partial S_1^*}{\partial I_1^*} = -\left(\frac{\partial f_1}{\partial I_1} - m_1\right)\Big/\frac{\partial f_1}{\partial S_1}\bigg|_{p^*} \tag{S56}$$

The assumed negative density dependence implies $\frac{\partial S_1^*}{\partial I_1^*}$ is negative. Thus, increases in I_1^* imply decreases in S_1^* and vice versa. Combining $\frac{\partial S_1^*}{\partial I_1^*} < 0$ and $S_1^* - \hat{S}_1 < 0$ yields $S_1^* - \hat{S}_1 < 0$ and $I_1^* - \hat{I}_1 > 0$ for all $0 \leq q \leq 1 + \hat{U}/\mu$, which implies $I_1^*/(S_1^* + I_1^*) > \hat{I}_1/(\hat{S}_1 + \hat{I}_1)$ for $0 \leq q \leq 1+\hat{U}/\mu$. However, this means that the proportion of infected hosts at the sympatric equilibrium is higher than the proportion of infected host at the allopatric equilibrium for all values of q, which contradicts our assumption that dilution occurs at $q = 1 + \hat{U}/\mu$.

Positive density dependence at equilibrium: If either host has positive density dependence at equilibrium and the positive density dependence is sufficiently large, then the terms in brackets in equations (S53) and (S55) change signs and the sign of $U^* - \hat{U}$ is unchanged. In this case, all of the above predictions are reversed.

S1.6 Figure equations and parameters

For all figures, the model equations are

$$\begin{aligned}
\frac{dS_i}{dt} &= \underbrace{\left[r_i(S_i + c_i I_i)[1 - \alpha_{i1}(S_1 + e_{i1}I_1) - \alpha_{i2}(S_2 + e_{i2}I_2)]\right]}_{\text{growth \& competition}} - \underbrace{\beta_i P S_i}_{\text{infection}} \\
\frac{dI_i}{dt} &= \underbrace{\beta_i S_i P}_{\text{infection}} \underbrace{-m_i I_i}_{\text{mortality}} \\
\frac{dP}{dt} &= \underbrace{\chi_1 I_1 + \chi_2 I_2}_{\text{propagule excretion}} - \underbrace{(u_{11}S_1 - u_{12}I_1 - u_{21}S_2 - u_{22}I_2)P}_{\text{propagule uptake}} \underbrace{-\mu P}_{\text{degradation}} .
\end{aligned} \tag{S57}$$

where r_i and $c_i r_i$ (with $c_i \leq 1$) are the maximum exponential growth rates of susceptible and infected individuals of species i, α_{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; all other parameters are defined as in model (1) of the main text.

For Figures 1 and 4, the infectious propagule equation is

$$\frac{dP}{dt} = \underbrace{\chi_1 I_1 + \chi_2 I_2}_{\text{propagule excretion}} - \underbrace{f(q)(u_{11}S_1 - u_{12}I_1 - u_{21}S_2 - u_{22}I_2)P}_{\text{propagule uptake}} \underbrace{-q\mu P}_{\text{degradation}} \tag{S58}$$

where $f(q) = 1 + \frac{\mu}{\hat{U}} - \frac{\mu}{\hat{U}}q$ is a change of parameters defined on $0 \leq q \leq 1 + \hat{U}/\mu$ that transforms the environmental transmission model from a form that behaves like a frequency dependent direct transmission model to a form that behaves like a density dependent direct transmission models, while leaving the densities at the allopatric equilibrium for host 1 unchanged. See appendix S1.5.4 for details.

Figure 1D,E:

High, Weak (blue-green): $r_1 = 3$, $r_2 = 3$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{22} = 0.1$, $a_{ij} = 1$, $\beta_1 = 1.5$, $\beta_2 = 1.7$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 1$, $\chi_2 = 2$, $u_{11} = 8$, $u_{12} = 1$, $u_{21} = 0.5$, $u_{22} = 0.5$, and $\mu = 6$. For the dashed curve $\alpha_{21} = 0$ and $\alpha_{12} = 0$. For the solid curve $\alpha_{21} = 0.9$ and $\alpha_{12} = 0.001$. $\hat{U} = 7.11$ in the change of parameters function.

Low, Weak (orange): $r_1 = 2$, $r_2 = 2$, $\alpha_{11} = 1$, $\alpha_{22} = 0.1$, $a_{ij} = 1$, $\beta_1 = 2$, $\beta_2 = 1/3$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $\chi_2 = 2$, $u_{ij} = 3$, and $\mu = 1$. For the dashed curve $\alpha_{21} = 0$ and $\alpha_{12} = 0$. For the solid curve $\alpha_{21} = 0.9$ and $\alpha_{12} = 0.05$. $\hat{U} = 1.85$ in the change of parameters function.

High, Strong (purple): $r_1 = 0.1$, $r_2 = 0.1$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{22} = 10$, $a_{ij} = 1$, $\beta_1 = 4$, $\beta_2 = 4$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $\chi_2 = 4$, $u_{ij} = 3$, and $\mu = 20$. For the dashed curve $\alpha_{21} = 0$ and $\alpha_{12} = 0$. For the solid curve $\alpha_{21} = 0.5$ and $\alpha_{12} = 9$. $\hat{U} = 1.26$ in the change of parameters function.

Low, Strong (vermillion): $r_1 = 2$, $r_2 = 2$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{22} = 10$, $a_{ij} = 1$, $\beta_1 = 1$, $\beta_2 = 1/3$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $\chi_2 = 2$, $u_{ij} = 3$, and $\mu = 10$. For the dashed curve $\alpha_{21} = 0$ and $\alpha_{12} = 0$. For the solid curve $\alpha_{21} = 0.4$ and $\alpha_{12} = 4$. $\hat{U} = 2.92$ in the

change of parameters function.

Figure 2A: $r_1 = 2$, $r_2 = 2$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{21} = 0$, $\alpha_{12} = 0$, $\alpha_{22} = 1$, $a_{ij} = 0.5$, $\beta_1 = 0.5$, $\beta_2 = 0.5$, $m_1 = 0.1$, $\chi_1 = 2$, $u_{ij} = 2$, and $\mu = 1$. The values of χ_2 are (blue) $\chi_2 = 0.1$, (cyan) $\chi_2 = 2$, and (red) $\chi_2 = 3$.
Figure 2B: $r_1 = 2$, $r_2 = 2$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{21} = 0$, $\alpha_{12} = 0$, $\alpha_{22} = 1$, $a_{ij} = 0$, $\beta_1 = 0.5$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $u_{ij} = 2$, and $\mu = 1$. The values of χ_2 are (blue) $\chi_2 = 0.1$, (cyan) $\chi_2 = 0.5$, (green) $\chi_2 = 1$, (magenta) $\chi_2 = 2$, and (red) $\chi_2 = 4$.
Figure 2C: $r_1 = 2$, $r_2 = 2$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{21} = 0.8$, $\alpha_{12} = 0.8$, $\alpha_{22} = 1$, $a_{11} = 1$, $a_{22} = 1$, $\beta_1 = 0.5$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $\chi_2 = 2$, $u_{ij} = 2$, and $\mu = 7$. The values of a_{12} and a_{21} are (blue) $a_{12} = a_{21} = 0.5$, (cyan) $a_{12} = a_{21} = 1$, (magenta) $a_{12} = a_{21} = 1.5$, and (red) $a_{12} = a_{21} = 2.5$.

Figure 3A: $r_1 = 2$, $r_2 = 2$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{21} = 0$, $\alpha_{12} = 0$, $a_{ij} = 1$, $\beta_1 = 0.5$, $\beta_2 = 0.5$, $m_1 = 0.1$, $\chi_1 = 2$, $u_{ij} = 2$, and $\mu = 1$. The values of χ_2 are (blue) $\chi_2 = 0.5$, (cyan) $\chi_2 = 1.5$, and (red) $\chi_2 = 3$.
Figure 3B: Same as Figure 3A except $\alpha_{21} = 0.5$ and $\alpha_{12} = 0.5$.
Figure 3C: $r_1 = 2$, $r_2 = 2$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{21} = 0.5$, $\alpha_{22} = 1$, $a_{ij} = 1$, $\beta_1 = 0.5$, $\beta_2 = 0.5$, $m_1 = 0.1$, $\chi_1 = 2$, $u_{ij} = 2$, and $\mu = 1$. The values of χ_2 are (blue) $\chi_2 = 1$, (cyan) $\chi_2 = 2$, (magenta) $\chi_2 = 4$, and (red) $\chi_2 = 3$.

Figure 4A: The parameters for both curves are $r_1 = 10$, $r_2 = 10$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{22} = 0.1$, $a_{11} = 0.9$, $a_{12} = 0.9$, $a_{21} = 0.9$, $a_{22} = 0.9$, $\beta_1 = 0.5$, $\beta_2 = 1/3$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $\chi_2 = 2$, $u_{ij} = 4$, and $\mu = 1$. For the dashed curve $\alpha_{21} = 0$ and $\alpha_{12} = 0$. For the solid curve $\alpha_{21} = 0.5$ and $\alpha_{12} = 0.05$. $\hat{U} = 4.17$ in the change of parameters function.
Figure 4B: The parameters for all three curves are $r_1 = 0.1$, $r_2 = 0.1$, $c_1 = 0$, $c_2 = 0$, $\alpha_{11} = 1$, $\alpha_{22} = 10$, $a_{ij} = 1$, $\beta_1 = 4$, $\beta_2 = 4$, $m_1 = 0.1$, $m_2 = 0.1$, $\chi_1 = 2$, $\chi_2 = 4$, $u_{ij} = 3$, and $\mu = 20$. For the dashed gray curve $\alpha_{21} = 0$ and $\alpha_{12} = 0$. For the dashed black curve $\alpha_{21} = 0.2$ and $\alpha_{12} = 2$. For the solid black curve $\alpha_{21} = 0.5$ and $\alpha_{12} = 9$. $\hat{U} = 1.26$ in the change of parameters function.

References

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

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

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.

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