Supporting Information. Clay, P.A., M.H. Cortez, and M.A. Duffy. 2021. Dose relationships can exacerbate, mute, or reverse the impact of heterospecific host density on infection prevalence. Ecology.

Appendix S4: Analysis of model with dose-dependent rates

In this appendix, we analytically compute how disease prevalence in the focal host de-pends on the density of a second host. These analytical calculations to provide additional support for our predictions and conclusions about the signs and magnitudes of the slopes of the curves we computed with our numerical simulations. The first outlines our approach, assumptions, and some terminology. In the following sections we apply the method to the different models.

Section S1 Methods, assumptions, and terminology

Approach & Assumptions: Let $(S_1^*, S_2^*, I_1^*, I_2^*, P^*)$ denote the equilibrium host and infectious propagule densities. Mathematically, we compute the derivative $d(I_1^*/N_1^*)/dN_2^*$, which defines how equilibrium infection prevalence in the focal host (I_1^*/N_1^*) changes with a small change in the density of the second host (N_2^*). Disease prevalence in the focal host increases with increased density of the second host when $d(I_1^*/N_1^*)/dN_2^* > 0$ and decreases with increased density of the second host when $d(I_1^*/N_1^*)/dN_2^* < 0$. The derivative is computed by taking the ratio of the derivatives $\partial(I_1^*/N_1^*)/\partial r_2$ and $\partial N_2^*/\partial r_2$ (Abrams and Cortez, 2015; Roberts and Heesterbeek, 2018), which are in turn computed using the Jacobian-based approach in Bender et al. (1984), Yodzis (1988), Novak et al. (2011), and Cortez and Abrams (2016). The specific equations defining each derivative are

$$\frac{\partial S_1^*}{\partial r_2} = -(-1)^{2+1} N_2^* |M_{21}|/|J| \tag{S1}$$

$$\frac{\partial S_2^*}{\partial r_2} = -(-1)^{2+2} N_2^* |M_{22}|/|J| \tag{S2}$$

$$\frac{\partial I_1^*}{\partial r_2} = -(-1)^{2+3} N_2^* |M_{23}|/|J| \tag{S3}$$

$$\frac{\partial I_2^*}{\partial r_2} = -(-1)^{2+4} N_2^* |M_{24}|/|J| \tag{S4}$$

$$\frac{\partial(I_1^*/N_1^*)}{\partial r_2} = \frac{1}{(N_1^*)^2}\left(S_1^* \frac{\partial I_1^*}{\partial r_2} - I_1^* \frac{\partial S_1^*}{\partial r_2}\right) \tag{S5}$$

$$\frac{\partial N_2^*}{\partial r_2} = \frac{\partial S_2^*}{\partial r_2} + \frac{\partial I_2^*}{\partial r_2} \tag{S6}$$

$$\frac{d(I_1^*/N_1^*)}{dN_2^*} = \left(\frac{\partial(I_1^*/N_1^*)}{\partial r_2}\right) \Big/ \left(\frac{\partial N_2^*}{\partial r_2}\right). \tag{S7}$$

where J is the Jacobian, M_{ij} is the submatrix of the Jacobian where the ith row and jth column have been removed, $|A|$ denotes the determinant of matrix A, and equation (S5) was simplified using the quotient rule for differentiation.

Throughout this appendix we assume the following. First, because we focus on stable equilibria, we assume the determinant of the Jacobian is negative when evaluated at an equilibrium, i.e., $|J| < 0$. Second, we assume host 1 experiences negative density dependence at equilibrium due to intraspecific competition. Mathematically, this means that the growth rate of population one at equilibrium decreases with increased conspecific density, i.e., $(\partial/\partial S_1)(dS_1/dt) < 0$ and $(\partial/\partial I_1)(dS_1/dt) < 0$. This assumption is expected to hold in most empirical systems. It is violated only in systems where the pathogen reduces the density of host 1 to very low values. Third, we assume that increasing the exponential growth rate of host 2 causes its total density to increase, i.e., $\frac{\partial N_2^*}{\partial r_2} > 0$. We expect this assumption to hold in most empirical systems. The only exceptions are those where the population is experiencing a hydra effect, i.e., decreased mortality (higher r_2) causes lower density, $\frac{\partial N_2^*}{\partial r_2} < 0$ (Abrams, 2009). Hydra effects are possible in our model, but we did not observe them in any of our numerical simulations.

Fourth, while our approach only focuses on small changes in the density of the second host, we will interpret our results in terms of the relationship between infection prevalence in host 1 and host 2 density for large changes in host density. This interpretation is reasonable because (i) under the assumption that $\partial N_2^*/\partial r_2$ has fixed sign, the sign of $d(I_1^*/N_1^*)/dN_2^*$ will only depend on the sign of $\partial(I_1^*/N_1^*)/\partial r_2$; (ii) the sign of $\partial(I_1^*/N_1^*)/\partial r_2$ changes smoothly with increases in r_2 because it only depends on the product of Jacobian entries, all of which change smoothly with increases in r_2; and (iii) the sign of $\partial(I_1^*/N_1^*)/\partial r_2$ will only change a small number of times as r_2 is increased because most of the Jacobian entries have fixed signs.

Terminology: We will describe infected hosts of each population in terms of whether they are sinks or sources of infections propagules. Intuitively, a source host has larger values of x_i and smaller values of f_i and a sink has smaller values of x_i and larger values of f_i. Mathematically, sink and source are defined by the net per capita rate of production of infectious propagules by infected hosts at equilibrium, i.e., $X_i = x_i - P^* f_i$. A host is a sink (at equilibrium) if infected individuals produce fewer infectious propagules than they uptake ($X_i = x_i - P^* f_i < 0$). A host is a source (at equilibrium) if infected individuals produce more infectious propagules than they uptake ($X_i = x_i - P^* f_i > 0$). We say that host i is a larger or smaller source than host j if $X_i > X_j$ and $X_i < X_j$, respectively. A similar definition applies to smaller and larger sinks. We note two things: (i) if a host does not excrete infectious propagules ($x_i = 0$), then that host is necessarily a sink for infectious propagules and (ii) for any equilibrium with positive infectious propagule density, at least one host species must be a source.

Section S2 Analysis of model with dose-dependent infection rates

The model with dose-dependent infection rates is

$$\begin{aligned}\frac{dS_i}{dt} &= N_i(r_i - \alpha_{i1}N_1 - \alpha_{i2}N_2) - \beta_i(f_iP)^{k_i}S_i \\ \frac{dI_i}{dt} &= \beta_i(f_iP)^{k_i}S_i - m_iI_i \\ \frac{dP}{dt} &= x_1I_1 + x_2I_2 - \mu P - f_1N_1P - f_2N_2P\end{aligned} \tag{S8}$$

where $N_i = S_i + I_i$. Let $(S_1^*, S_2^*, I_1^*, I_2^*, P^*)$ be a stable equilibrium point of model (S8).

The Jacobian of model (S8) evaluated at the equilibrium is

$$J = \begin{pmatrix} J_{11} & -\alpha_{12}N_1^* & J_{13} & -\alpha_{12}N_1^* & -\beta_1(f_1P)^{k_1}k_1S_1^*/P^* \\ -\alpha_{21}N_2^* & J_{22} & -\alpha_{21}N_2^* & J_{24} & -\beta_2(f_2P^*)^{k_2}k_2S_2^*/P^* \\ \beta_1(f_1P^*)^{k_1} & 0 & -m_1 & 0 & \beta_1(f_1P^*)^{k_1}k_1S_1^*/P^* \\ 0 & \beta_2(f_2P^*)^{k_2} & 0 & -m_2 & \beta_2(f_2P^*)^{k_2}k_2S_2^*/P^* \\ -f_1P^* & -f_2P^* & x_1 - P^*f_1 & x_2 - P^*f_2 & -U - \mu \end{pmatrix} \tag{S9}$$

where $J_{11} = r_1 - 2\alpha_{11}N_1^* - \alpha_{12}N_2^* - \beta_1(f_1P^*)^{k_1}$, $J_{13} = r_1 - 2\alpha_{11}N_1^* - \alpha_{12}N_2^*$, $J_{22} = r_2 - \alpha_{21}N_1^* - 2\alpha_{22}N_2^* - \beta_2(f_2P^*)^{k_2}$, and $J_{24} = r_2 - \alpha_{21}N_1^* - 2\alpha_{22}N_2^*$, and $U = N_1^*f_1 + N_2^*f_2$ is the total per infectious propagule uptake rate by hosts at equilibrium. Our assumption that host 1 experiences negative density dependence due to intraspecific competition implies that J_{11} and J_{13} are negative. We simplify some of the Jacobian entries using the fact that $dI_i/dt = 0$ at equilibrium means $\beta_i(f_iP^*)^{k_i}S_i^* = m_iI_i^*$. This yields,

$$J|_\rho = \begin{pmatrix} J_{11} & -\alpha_{12}N_1^* & J_{13} & -\alpha_{12}N_1^* & -k_1m_1I_1^*/P^* \\ -\alpha_{21}N_2^* & J_{22} & -\alpha_{21}N_2^* & J_{24} & -k_2m_2I_2^*/P^* \\ m_1I_1^*/S_1^* & 0 & -m_1 & 0 & k_1m_1I_1^*/P^* \\ 0 & m_2I_2^*/S_2^* & 0 & -m_2 & k_2m_2I_2^*/P^* \\ -I_1^*P^* & -I_2^*P^* & X_1 & X_2 & -U - \mu \end{pmatrix} \tag{S10}$$

where $X_i = x_i - f_iP^*$ is the net per capita rate of production of infectious propagules by host i.

Evaluating equation (S5) yields

$$\frac{\partial(I_1^*/N_1^*)}{\partial r_2} = k_1 \underbrace{\frac{N_2^*m_1I_1^*m_2}{(N_1^*)^2S_2^*P^*|J|}}_{-}\left[\alpha_{12}N_1^*N_2^*(X_1I_1^* - f_1P^*S_1^*) + \underbrace{(I_1^*J_{13} + J_{11}S_1^*)}_{-}(X_2I_2^* - f_2P^*S_2^*)\right] \tag{S11}$$

The equation defining $\partial N_2^*/\partial r_2$ is much longer and not included here; the full equation is given in the accompanying Maple worksheets. The main thing to know is that its magnitude is not proportional to k_1.

Equation (S11) shows the following. First, the magnitude of $\partial(I_1^*/N_1^*)/\partial r_2$ is proportional to k_1 and the sign of $\partial(I_1^*/N_1^*)/\partial r_2$ is not directly affected by the value of k_1. Second, the sign of $\partial(I_1^*/N_1^*)/\partial r_2$ depends on the terms $X_i I_i^* - f_i S_i^* P^*$. The sign of $\partial(I_1^*/N_1^*)/\partial r_2$ is more positive when X_1 is more negative (i.e., host 1 is a smaller source or larger sink) and X_2 is more positive (i.e., host 2 is large source). In addition, in the special case where $X_1 I_1^* - f_1 S_1^* P^* = X_2 I_2^* - f_2 S_2^* P^*$ (which must necessarily be positive), the terms in brackets simplify to

$$(X_1 I_1^* - f_2 S_2^* P^*)[-N_1^*(r_1 - \alpha_{12} N_2^*) - \alpha_{11}(N_1^*)^2 - \beta_1 (f_1 P)^{k_1} S_1^*] < 0 \tag{S12}$$

In total, we expect $\partial(I_1^*/N_1^*)/\partial r_2$ will be negative when host 2 is a sink or much smaller source of infectious propagules than host 1 (i.e., X_2 much smaller than X_1) and positive otherwise.

After recalling that $d(I_1^*/N_1^*)/dN_2^*$ is the ratio of $\partial(I_1^*/N_1^*)/\partial r_2$ and $\partial N_2^*/\partial r_2 > 0$ and combining the above, we predict:

- A negative relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* < 0$) when host 2 is a sink or a sufficiently smaller source of infectious propagules than host 1.

- A positive relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* > 0$) when host 2 is a sufficiently larger source of infectious propagules. In particular, we expect a positive relationship whenever host 2 is an equal or larger source of infectious propagules than host 1.

- The slope of the relationship between infection prevalence in host 1 and host 2 density (i.e., the magnitude of $d(I_1^*/N_1^*)/dN_2^*$) will be greater for larger values of k. The slope will also be larger in magnitude if host 1 is a much larger or much smaller source than host 2, and vice versa (i.e., X_1 and X_2 of opposite signs or X_1 and X_2 both positive with one much larger than the other).

These predictions qualitatively match the results from our simulations in the middle column of figure 4.

Section S3 Analysis of model with dose-dependent excretion rates

The model with dose-dependent infection and excretion rates is

$$\begin{aligned}
\frac{dS_i}{dt} &= N_i(r_i - \alpha_{i1}N_1 - \alpha_{i2}N_2) - \beta_i(f_iP)^{k_i}S_i \\
\frac{dI_i}{dt} &= \beta_i(f_iP)^{k_i}S_i - m_iI_i \\
\frac{dP}{dt} &= x_1\left(\frac{1}{2} + \frac{f_1P}{2f_1P_1}\right)^{\gamma_1} I_1 + x_2\left(\frac{1}{2} + \frac{f_2P}{2f_2P_2}\right)^{\gamma_2} I_2 - \mu P - f_1N_1P - f_2N_2P
\end{aligned} \tag{S13}$$

where $N_i = S_i + I_i$. Let $(S_1^*, S_2^*, I_1^*, I_2^*, P^*)$ be a stable equilibrium point of model (S13).

The Jacobian of model (S13) evaluated at the equilibrium is

$$J = \begin{pmatrix}
J_{11} & -\alpha_{12}N_1^* & J_{13} & -\alpha_{12}N_1^* & -\beta_1(f_1P)^{k_1}k_1S_1^*/P^* \\
-\alpha_{21}N_2^* & J_{22} & -\alpha_{21}N_2^* & J_{24} & -\beta_2(f_2P^*)^{k_2}k_2S_2^*/P^* \\
\beta_1(f_1P^*)^{k_1} & 0 & -m_1 & 0 & \beta_1(f_1P^*)^{k_1}k_1S_1^*/P^* \\
0 & \beta_2(f_2P^*)^{k_2} & 0 & -m_2 & \beta_2(f_2P^*)^{k_2}k_2S_2^*/P^* \\
-f_1P^* & -f_2P^* & X_1 & X_2 & Q - U - \mu
\end{pmatrix} \tag{S14}$$

where $J_{11} = r_1 - 2\alpha_{11}N_1^* - \alpha_{12}N_2^* - \beta_1(f_1P^*)^{k_1}$, $J_{13} = r_1 - 2\alpha_{11}N_1^* - \alpha_{12}N_2^*$, $J_{22} = r_2 - \alpha_{21}N_1^* - 2\alpha_{22}N_2^* - \beta_2(f_2P^*)^{k_2}$, $J_{24} = r_2 - \alpha_{21}N_1^* - 2\alpha_{22}N_2^*$, $U = N_1^*f_1 + N_2^*f_2$ is the total per infectious propagule uptake rate by hosts at equilibrium,

$$Q = \frac{x_1\gamma_1I_1^*}{2P_1}\left(\frac{1}{2} + \frac{f_1P^*}{2f_1P_1}\right)^{\gamma_1-1} + \frac{x_2\gamma_2I_2^*}{2P_2}\left(\frac{1}{2} + \frac{f_2P^*}{2f_2P_2^*}\right)^{\gamma_2-1} \tag{S15}$$

and we redefine X_i as

$$X_i = x_i\left(\frac{1}{2} + \frac{f_iP^*}{2f_iP_i}\right)^{\gamma_i} - P^*f_i \tag{S16}$$

which is still interpreted as the net production (at equilibrium) of infectious propagules by infected hosts in population i. Our assumption that host 1 experiences negative density dependence due to intraspecific competition implies that J_{11} and J_{13} are negative. Simplifying the Jacobian entries using $\beta_i(f_iP^*)^{k_i}S_i^* = m_iI_i^*$ produces

$$J|_\rho = \begin{pmatrix}
J_{11} & -\alpha_{12}N_1^* & J_{13} & -\alpha_{12}N_1^* & -k_1m_1I_1^*/P^* \\
-\alpha_{21}N_2^* & J_{22} & -\alpha_{21}N_2^* & J_{24} & -k_2m_2I_2^*/P^* \\
m_1I_1^*/S_1^* & 0 & -m_1 & 0 & k_1m_1I_1^*/P^* \\
0 & m_2I_2^*/S_2^* & 0 & -m_2 & k_2m_2I_2^*/P^* \\
-I_1^*P^* & -I_2^*P^* & X_1 & X_2 & Q - U - \mu
\end{pmatrix} \tag{S17}$$

Notice that matrix (S17) is nearly identical in form to the Jacobian (S10) for model (S8). The only differences are that X_1 and X_2 are defined by equation (S16) and the J_{55} entry of matrix (S17) contains the extra term Q.

Because of the similarity of the Jacobians for models (S8) and (S13), the equations defining $\partial(I^*/N_1^*)/\partial r_2$ and $\partial N_2^*/\partial r_2$ are identical except that X_1 and X_2 are defined by equation (S16) and all instances of $U + \mu$ are replaced with $-Q + U + \mu$. As a consequence, all of our predictions for model (S13) are the same as those for model (S8). Specifically, we predict:

- A negative relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* < 0$) when host 2 is a sink or a sufficiently smaller source of infectious propagules than host 1.

- A positive relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* > 0$) when host 2 is a sufficiently larger source of infectious propagules. In particular, we expect a positive relationship whenever host 2 is an equal or larger source of infectious propagules than host 1.

- The slope of the relationship between infection prevalence in host 1 and host 2 density (i.e., the magnitude of $d(I_1^*/N_1^*)/dN_2^*$) will be greater for larger values of k. The slope will also be larger in magnitude if host 1 is a much larger or much smaller source than host 2, and vice versa (i.e., X_1 and X_2 of opposite signs or X_1 and X_2 both positive with one much larger than the other).

These predictions qualitatively match the results from our simulations in figure 4.

Section S4 Analysis of model with dose-dependent mortality rates

The model with dose-dependent infection and mortality rates is

$$\begin{aligned}\frac{dS_i}{dt} &= N_i(r_i - \alpha_{i1}N_1 - \alpha_{i2}N_2) - \beta_i(f_iP)^{k_i}S_i \\ \frac{dI_i}{dt} &= \beta_i(f_iP)^{k_i}S_i - m_{min}I_i - m_{i,dose}I_i \\ \frac{dP}{dt} &= x_1I_1 + x_2I_2 - \mu P - f_1N_1P - f_2N_2P\end{aligned} \tag{S18}$$

where $N_i = S_i + I_i$, $m_{i,dose} = (m_i - m_{min})(f_iP/f_iP_i)^{\rho_i}$ is the dose-dependent mortality rate. Let $(S_1^*, S_2^*, I_1^*, I_2^*, P^*)$ be a stable equilibrium point of model (S8).

The Jacobian of model (S18) evaluated at the equilibrium is

$$J = \begin{pmatrix} J_{11} & -\alpha_{12}N_1^* & J_{13} & -\alpha_{12}N_1^* & -\beta_1(f_1P)^{k_1}k_1S_1^*/P^* \\ -\alpha_{21}N_2^* & J_{22} & -\alpha_{21}N_2^* & J_{24} & -\beta_2(f_2P^*)^{k_2}k_2S_2^*/P^* \\ \beta_1(f_1P^*)^{k_1} & 0 & -m_{min} - m_{1,dose} & 0 & \frac{\beta_1(f_1P^*)^{k_1}k_1S_1^*}{P^*} - \frac{\rho_1m_{1,dose}I_1^*}{P^*} \\ 0 & \beta_2(f_2P^*)^{k_2} & 0 & -m_{min} - m_{2,dose} & \frac{\beta_2(f_2P^*)^{k_2}k_2S_2^*}{P^*} - \frac{\rho_2m_{2,dose}I_2^*}{P^*} \\ -f_1P^* & -f_2P^* & x_1 - P^*f_1 & x_2 - P^*f_2 & -U - \mu \end{pmatrix} \tag{S19}$$

where $J_{11} = r_1 - 2\alpha_{11}N_1^* - \alpha_{12}N_2^* - \beta_1(f_1P^*)^{k_1}$, $J_{13} = r_1 - 2\alpha_{11}N_1^* - \alpha_{12}N_2^*$, $J_{22} = r_2 - \alpha_{21}N_1^* - 2\alpha_{22}N_2^* - \beta_2(f_2P^*)^{k_2}$, and $J_{24} = r_2 - \alpha_{21}N_1^* - 2\alpha_{22}N_2^*$, and $U = N_1^*f_1 + N_2^*f_2$ is the total per infectious propagule uptake rate by hosts at equilibrium. Our assumption that host 1 experiences negative density dependence due to intraspecific competition implies that J_{11} and J_{13} are negative. We simplify some of the Jacobian entries using the fact that $dI_i/dt = 0$ at equilibrium means $\beta_i(f_iP^*)^{k_i}S_i^* = m_{min}I_i^* + m_{i,dose}I_i^*$. This yields,

$$J = \begin{pmatrix} J_{11} & -\alpha_{12}N_1^* & J_{13} & -\alpha_{12}N_1^* & -\frac{kI_1^*}{P^*}(m_{min} + m_{1,dose}) \\ -\alpha_{21}N_2^* & J_{22} & -\alpha_{21}N_2^* & J_{24} & -\frac{kI_2^*}{P^*}(m_{min} + m_{2,dose}) \\ (m_{min} + m_{1,dose})I_1^*/S_1^* & 0 & -m_{min} - m_{1,dose} & 0 & \frac{k_1I_1^*}{P^*}(m_{min} + m_{1,dose}) - \frac{\rho_1I_1^*m_{1,dose}}{P} \\ 0 & (m_{min} + m_{2,dose})I_2^*/S_2^* & 0 & -m_{min} - m_{2,dose} & \frac{k_2I_2^*}{P^*}(m_{min} + m_{2,dose}) - \frac{\rho_2I_2^*m_{2,dose}}{P} \\ -I_1^*P^* & -I_2^*P^* & X_1 & X_2 & -U - \mu \end{pmatrix} \tag{S20}$$

where $X_i = x_i - f_iP^*$ is the net per capita rate of production of infectious propagules by host i.

Evaluating equation (S5) yields

$$\frac{\partial(I_1^*/N_1^*)}{\partial r_2} = \Big((k_1-\rho_1)m_{1,dose}+k_1 m_{min}\Big)\underbrace{\frac{I_1^* N_2^*(m_{min}+m_{2,dose})}{(N_1^*)^2 S_2^* P^* |J|}}_{-}\Big[\alpha_{12}N_1^*N_2^*(X_1 I_1^* - f_1 P^* S_1^*) + \underbrace{(I_1^* J_{13} + J_{11} S_1^*)}_{-}(X_2 I_2^* - f_2 P^* S_2^*)\Big]. \tag{S21}$$

The equation defining $\frac{\partial N_2^*}{\partial r_2}$ is very long and not included here; the full equation is given in the accompanying Maple worksheets. The main thing to know is that its magnitude is not proportional to $(k_1-\rho_1)m_{1,dose}+k_1 m_{min}$.

Equation (S21) shows the following. First, the magnitude of $\partial(I_1^*/N_1^*)/\partial r_2$ is (i) proportional to $(k_1-\rho_1)m_{1,dose}+k_1 m_{min}$ and (ii) depends on the magnitudes and signs of the $X_i I_i^* - f_i P^* S_i^*$. Second, the sign of $\partial(I_1^*/N_1^*)/\partial r_2$ depends on (i) the sign of $(k_1-\rho_1)m_{1,dose}+k_1 m_{min}$ and (ii) the terms in brackets in equation (S21). The sign of $(k_1-\rho_1)m_{1,dose}+k_1 m_{min}$ is negative when ρ_1 is sufficiently larger than k_1 and positive otherwise. Using the same calculation from appendix S2.1, the terms in brackets are positive when host 2 is a sink or much smaller source of host 1 (i.e., X_2 much smaller than X_1) and negative otherwise.

Recalling that $d(I_1^*/N_1^*)/dN_2^*$ is the ratio of $\partial(I_1^*/N_1^*)/\partial r_2$ and $\partial N_2^*/\partial r_2 > 0$ and combining the above produces the following predictions.

- If k greater than or equal to ρ, or ρ is insufficiently larger than k, then we predict:
  - A negative relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* < 0$) when host 2 is a sink or a sufficiently smaller source of infectious propagules than host 1.
  - A positive relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* > 0$) when host 2 is a sufficiently larger source of infectious propagules. In particular, we expect a positive relationship whenever host 2 is an equal or larger source of infectious propagules than host 1.
- If ρ is sufficiently larger than k, then we predict:
  - A positive relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* < 0$) when host 2 is a sink or a sufficiently smaller source of infectious propagules than host 1.
  - A negative relationship between infection prevalence in host 1 and host 2 density ($d(I_1^*/N_1^*)/dN_2^* > 0$) when host 2 is a sufficiently larger source of infectious propagules. In particular, we expect a positive relationship whenever host 2 is an equal or larger source of infectious propagules than host 1.

- The slope of the relationship between infection prevalence in host 1 and host 2 density (i.e., the magnitude of $d(I_1^*/N_1^*)/dN_2^*$) will be greater when (i) ρ is much larger than k or vice versa and (ii) host 1 is a much larger or much smaller source than host 2, and vice versa (i.e., X_1 and X_2 of opposite signs or X_1 and X_2 both positive with one much larger than the other).

These predictions qualitatively match the results from our simulations in figure 5.

References

Abrams, P. A. 2009. When does greater mortality increase population size? The long history and diverse mechanisms underlying the hydra effect. Ecology Letters 12:462–474.

Abrams, P. A., and M. H. Cortez. 2015. The many potential interactions between predators that share competing prey. Ecological Monographs 85:625–641.

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.

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

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