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 S3

In this document, we (i) rerun our simulations while varying one parameter at a time to explore whether our qualitative results are sensitive to changes in parameter values (Section S1 – Section S9), (ii) run a scenario in which we examine the impact of inter-specific host density on infection prevalence in a host that cannot sustain transmission on its own (Section S10), and (iii) run a scenario with dose-infectivity, dose-mortality, and dose-excretion relationships simultaneously.

Section S1- Non-focal host is a superior competitor to focal host. $(\boldsymbol{\alpha_{12} = 0.75})$

Increasing α_{12} does not qualitatively change our results. If the competing host has stronger interspecific effects on the focal host than the focal host has on the competing host, then changes to infection prevalence due to competing host density observed when the hosts are equal competitors (main text) occur over a smaller range of competing host densities. Note that in Figure S1 C and F, compounded positive feedback loops from dose-excretion relationships and dose-infectivity relationships mean that infection prevalence and propagule density are both 0 for any density of a low-competence competitor.

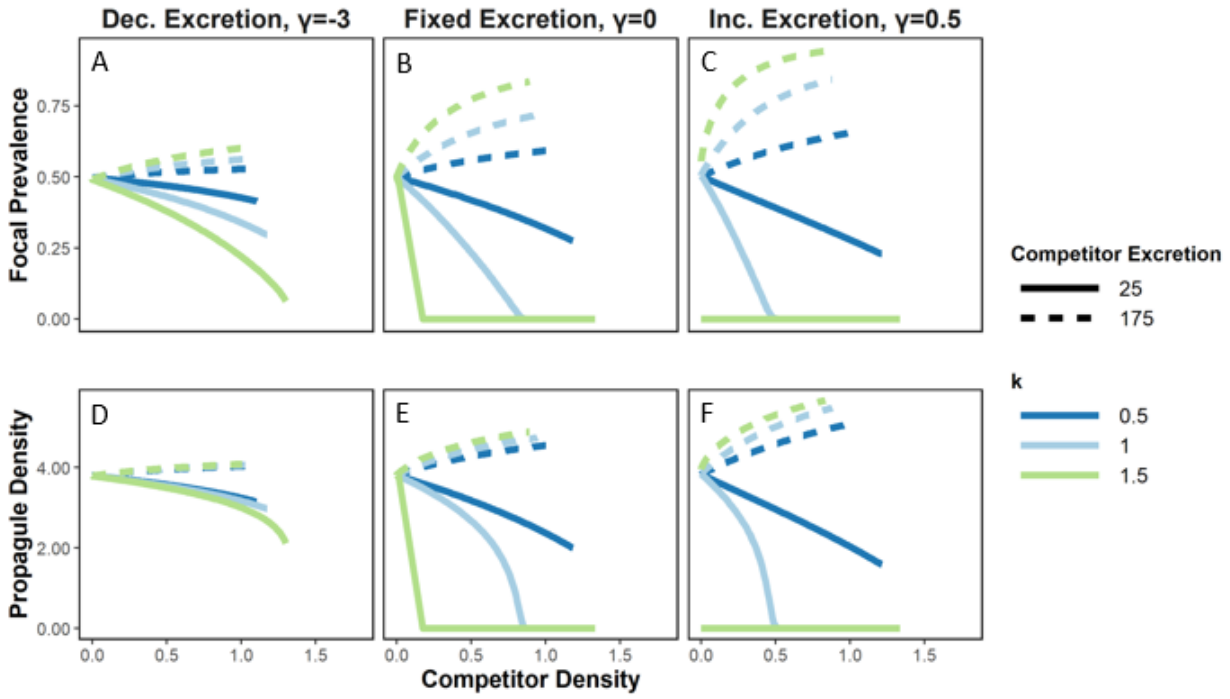

Figure S1- Figure 4 in main text, except that $\alpha_{12} = 0.75$

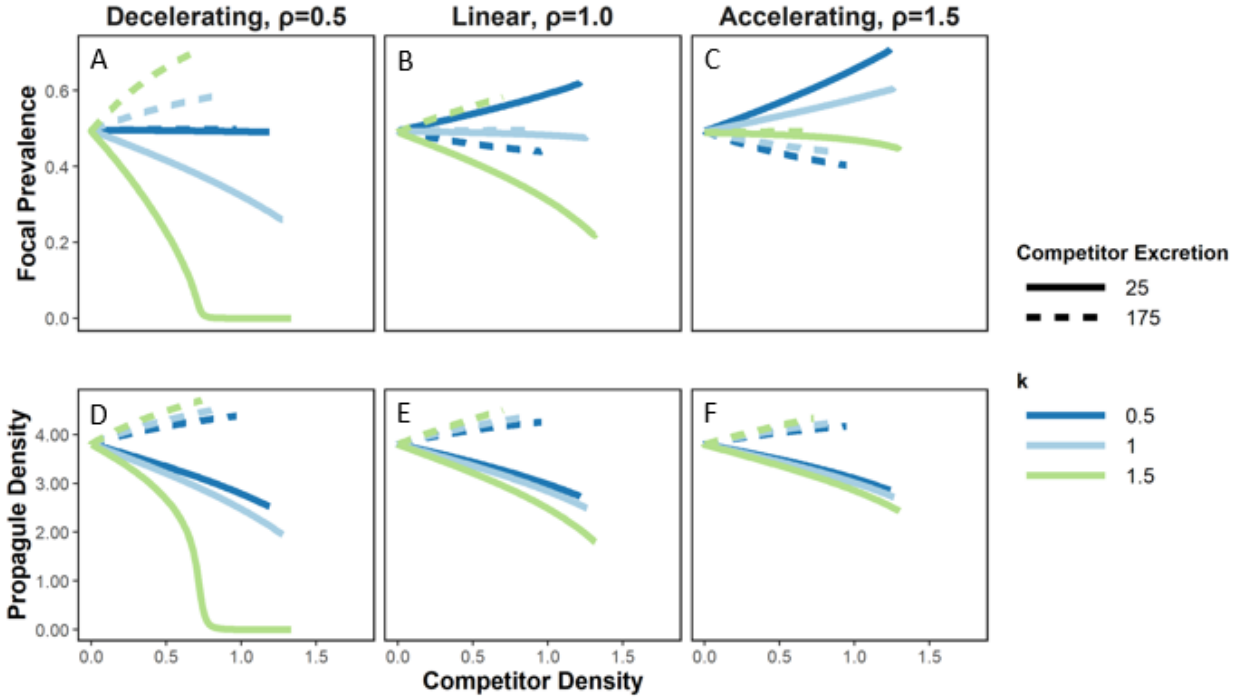

Figure S2- Figure 5 in main text, except that $\alpha_{12} = 0.75$

Section S2- Non-focal host is an inferior competitor to focal host. $(\alpha_{12} = 0.25)$

Decreasing α_{12} does not qualitatively change our results. If the competing host has weaker interspecific effects on the focal host than the focal host has on the competing host, then changes to infection prevalence due to competing host density observed when the hosts are equal competitors (main text) occur over a larger range of competing host densities.

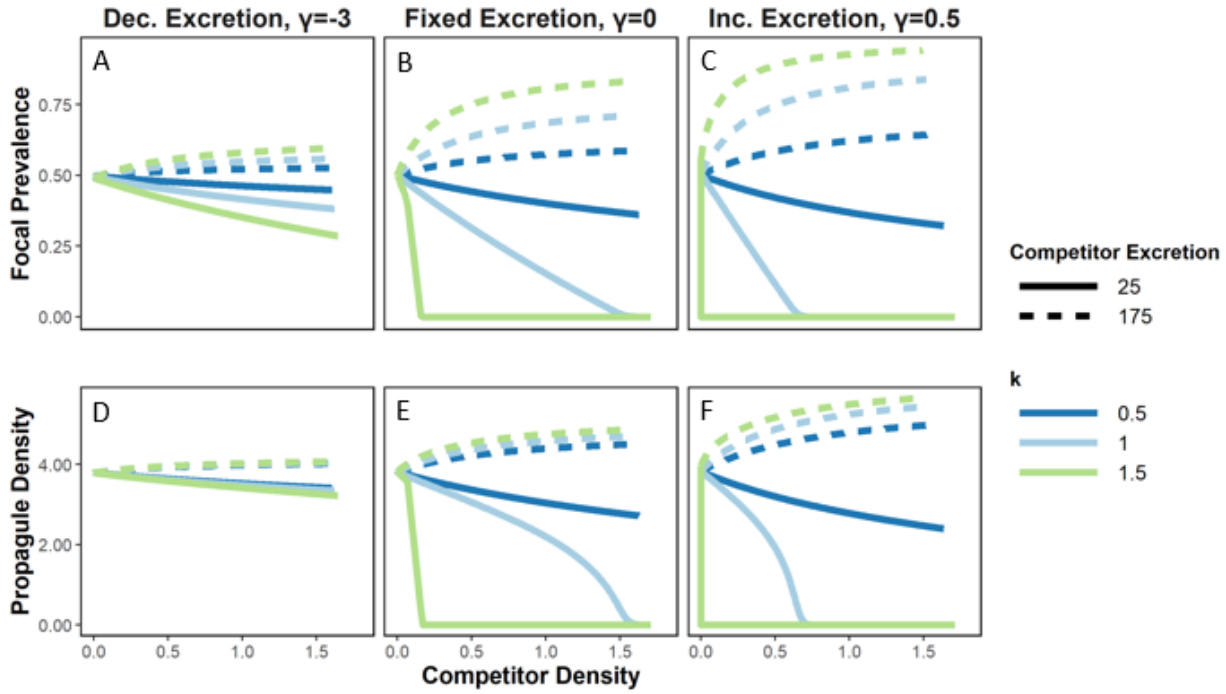

Figure S3- Figure 4 in main text, except that $\alpha_{12} = 0.25$

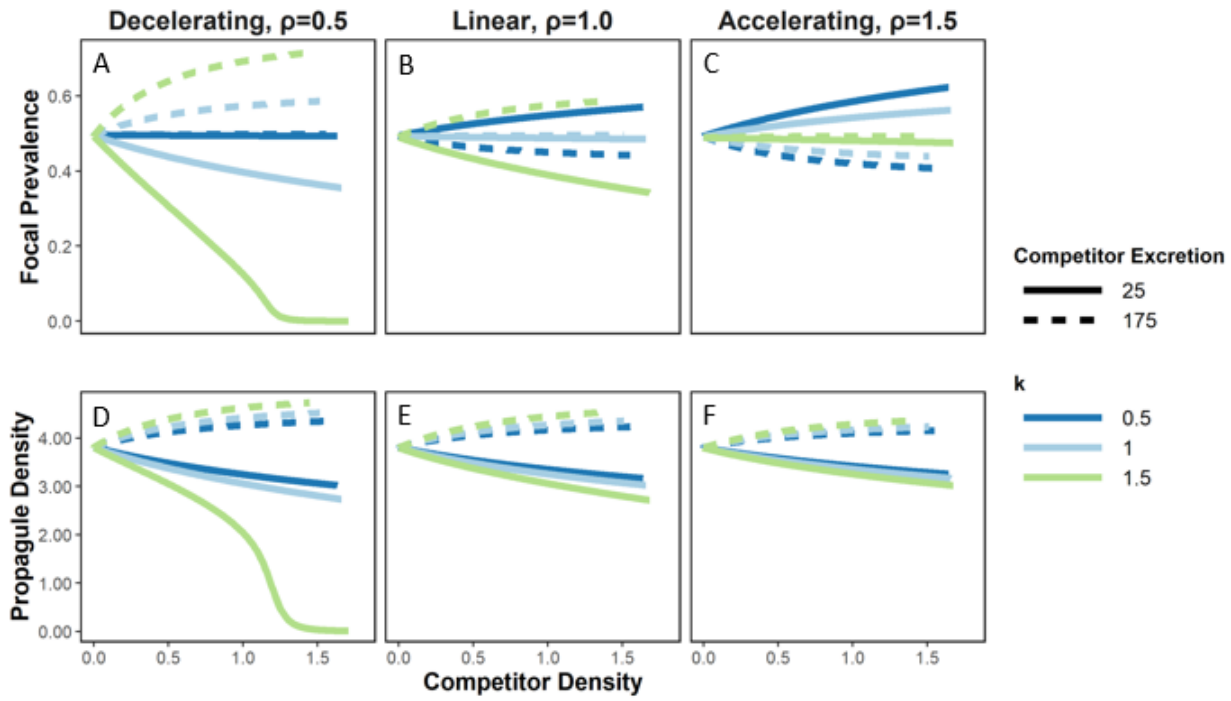

Figure S4- Figure 5 in main text, except that $\alpha_{12} = 0.25$

Section S3- Non-focal host has a higher contact rate than focal host. $(\boldsymbol{f_2 = 1.5})$

Increasing f_2 does not qualitatively change our results. If the competing host has a higher contact rate than the focal host, then the competing host is more likely to decrease infection prevalence in the focal host, or increase infection prevalence under strong host-mortality relationships, as it is removing more infectious spores from the environment (compare to Figure 4 and 5 in main text).

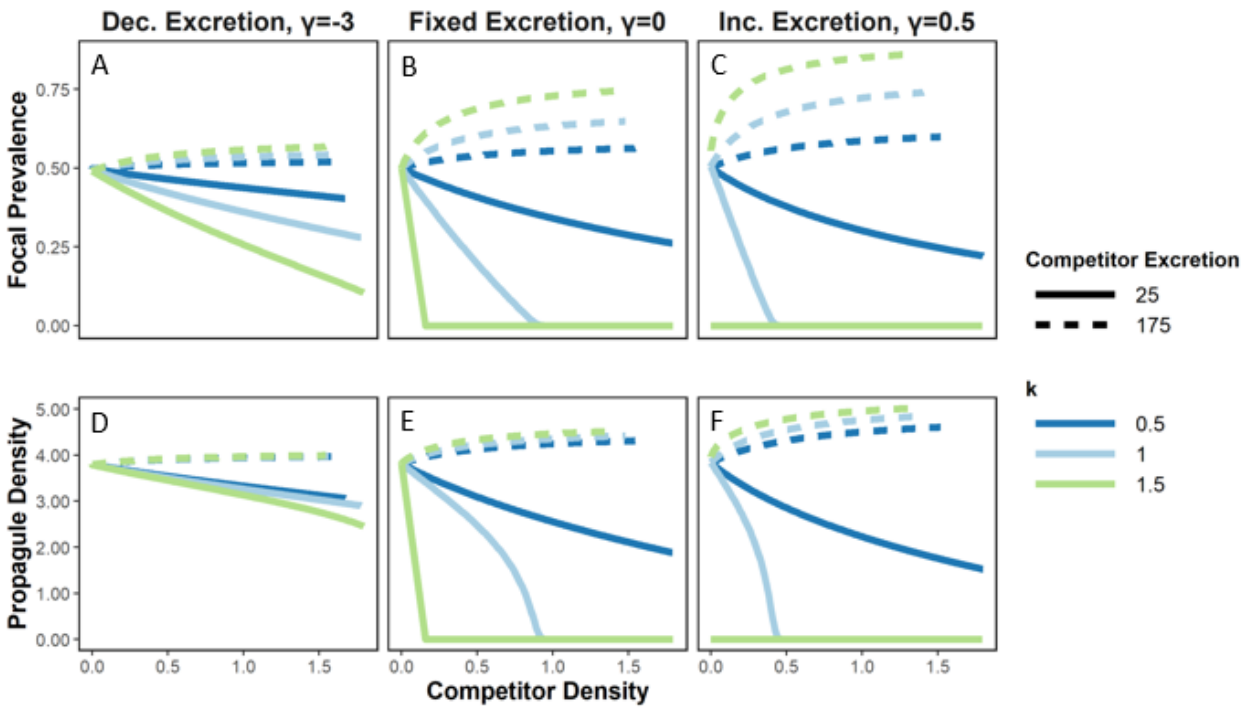

Figure S5- Figure 4 in main text, except that $f_2 = 1.5$

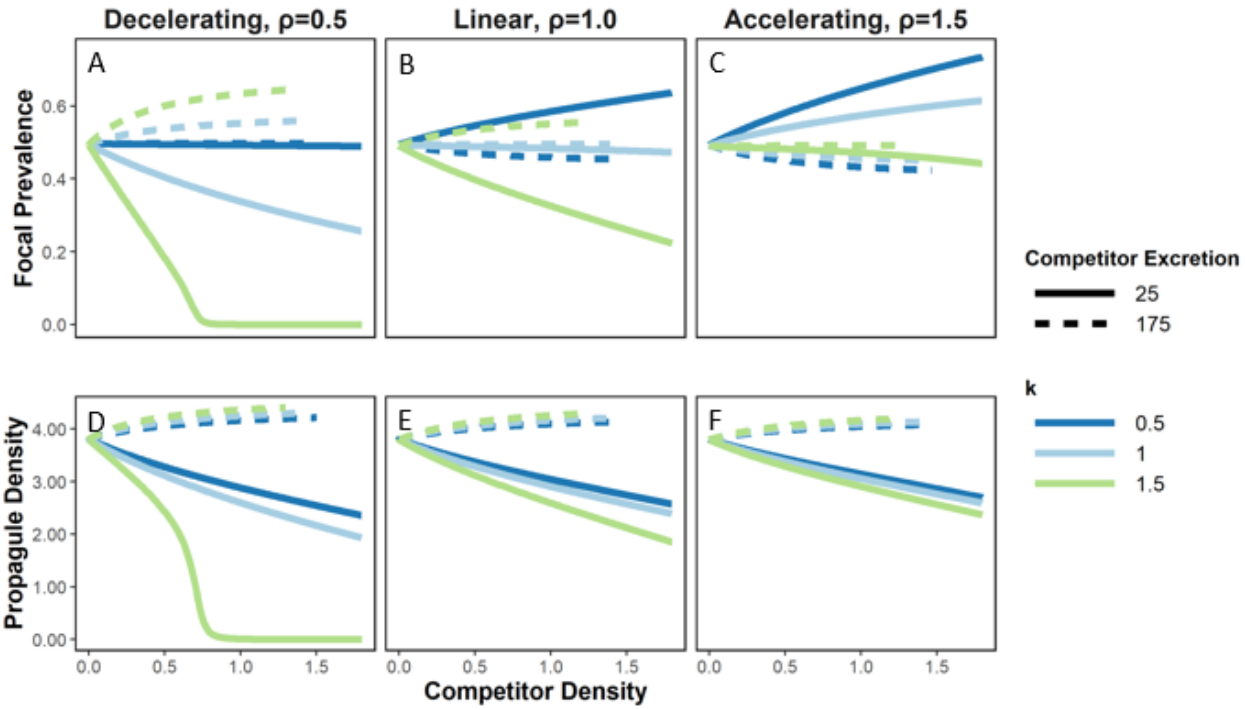

Figure S6- Figure 5 in main text, except that $f_2 = 1.5$

Section S4- Non-focal host has a lower contact rate than focal host. $(f_2 = 0.5)$

Decreasing f_2 does not qualitatively change our results. If the competing host has a lower contact rate than the focal host, then the competing host is more likely to increase infection prevalence in the focal host, or decrease infection prevalence under strong dose-mortality relationships, as it is removing less infectious spores from the environment. (compare "Equal Excretion" scenario in Figure S2.4.1 to Figure 3 in main text).

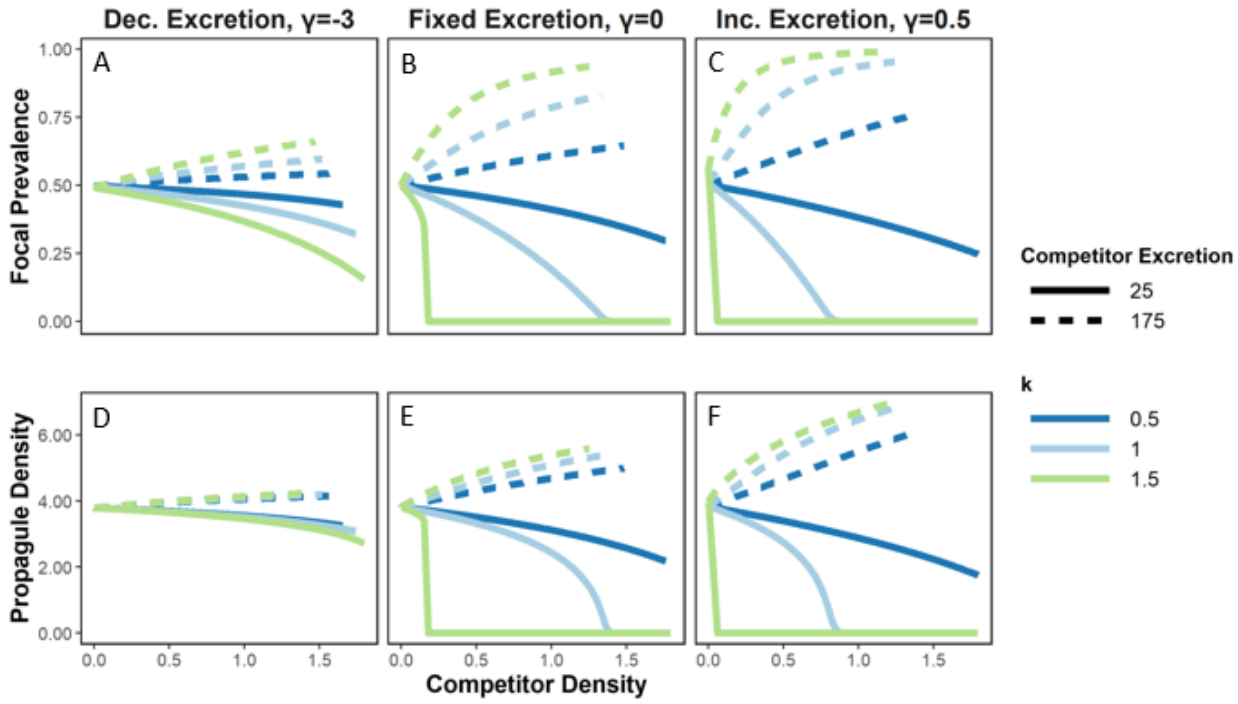

Figure S7- Figure 4 in main text, except that $f_2 = 0.5$

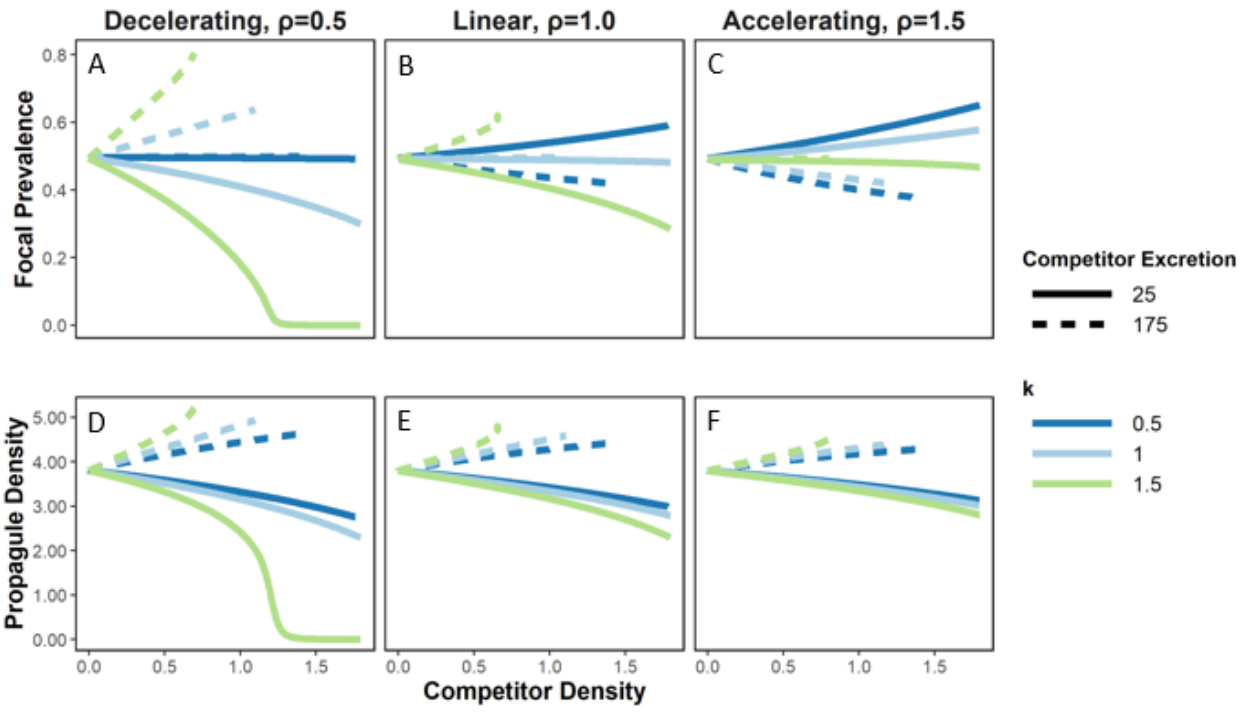

Figure S8- Figure 5 in main text, except that $f_2 = 0.5$

Section S5- Non-focal host has a higher susceptibility than focal host. $(\boldsymbol{\beta_2 = 1.5\beta_1})$

Increasing β_2 does not qualitatively change our results. If the competing host has a higher susceptibility than the focal host, then the competing host is less likely to decrease infection prevalence in the focal host, as it less likely to remove spores from the environment without becoming infected (though the opposite occurs under strong dose-mortality relationships).

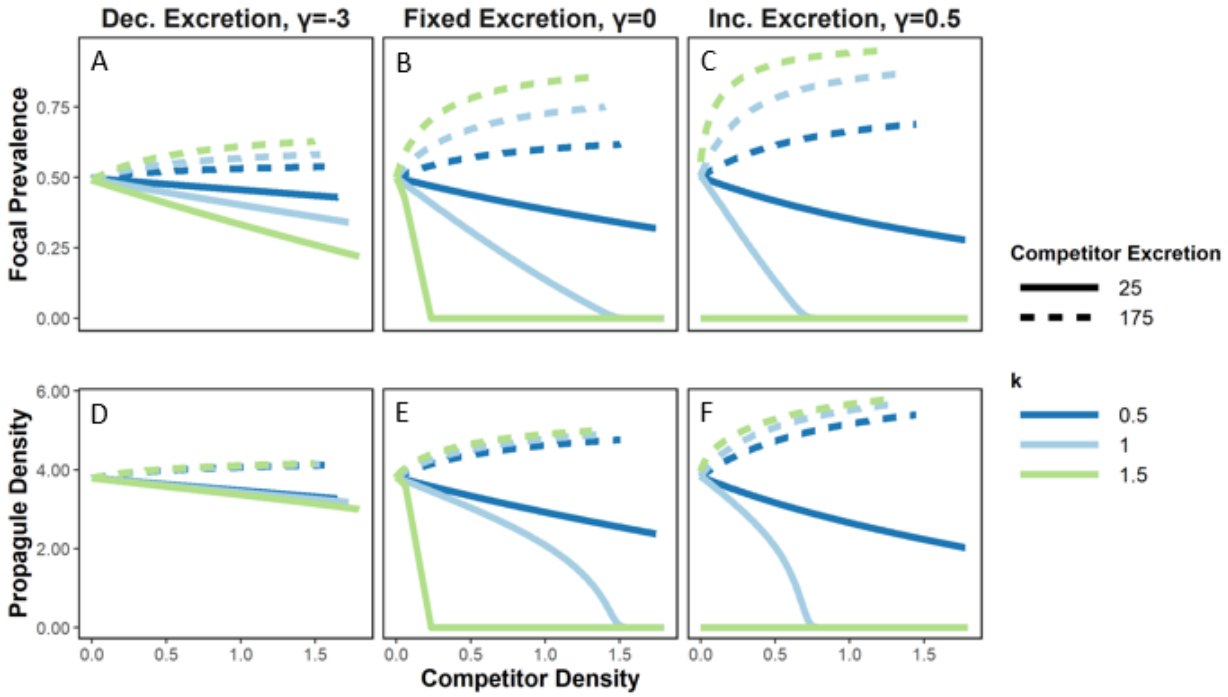

Figure S9- Figure 4 in main text, except that $\beta_2 = 1.5\beta_1$

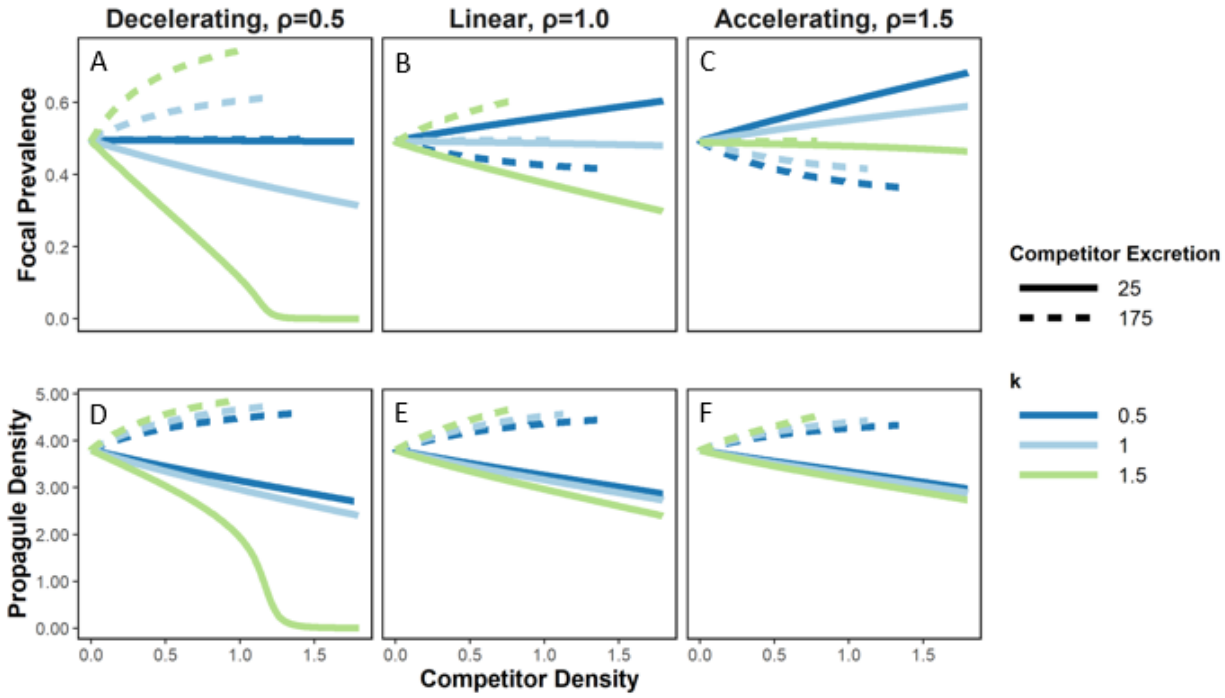

Figure S10- Figure 5 in main text, except that $\beta_2 = 1.5\beta_1$

Section S6- Non-focal host has a lower susceptibility than focal host. $(\boldsymbol{\beta_2 = 0.5\beta_2})$

Decreasing β_2 does not qualitatively change our results. If the competing host has a lower susceptibility than the focal host, then the competing host is more likely to decrease infection prevalence in the focal host, as it more likely to remove spores from the environment without becoming infected (though the opposite occurs under strong dose-mortality relationships).

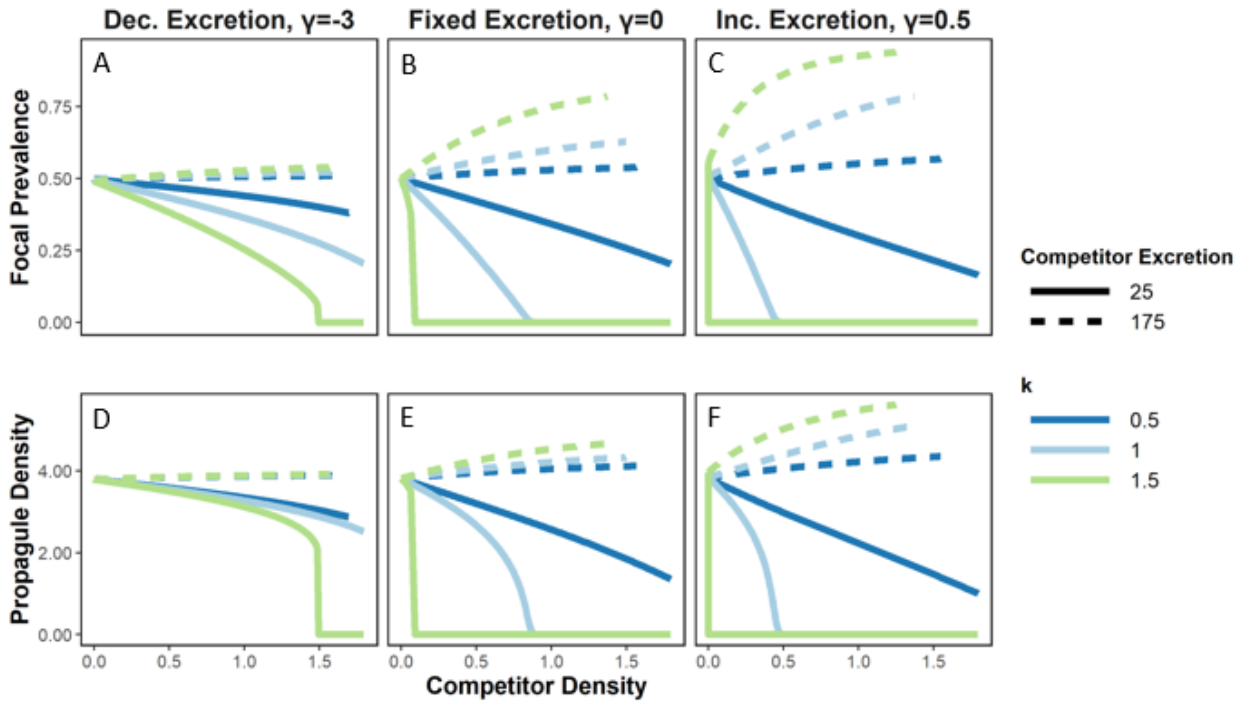

Figure S11- Figure 4 in main text, except that $\beta_2 = 0.5\beta_1$

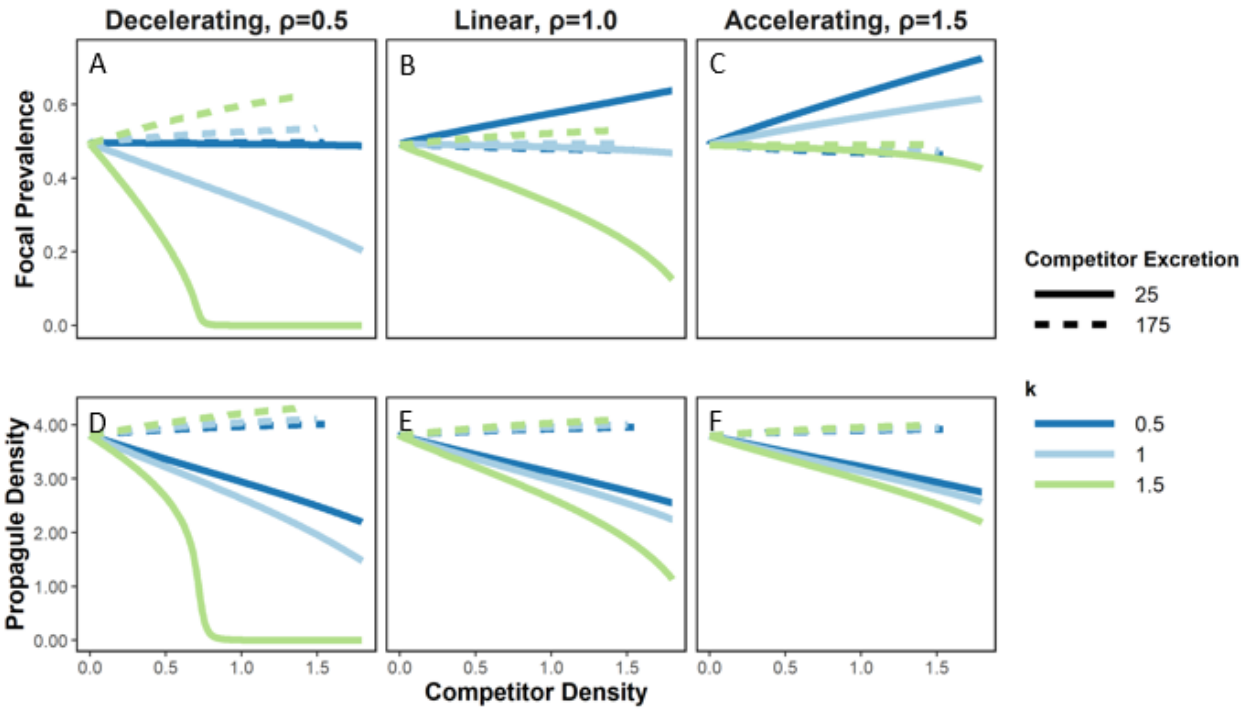

Figure S12- Figure 5 in main text, except that $\beta_2 = 0.5\beta_1$

Section S7- Non-focal host has a higher mortality rate than focal host. $(\boldsymbol{m_2 = 0.6})$

Increasing m_2 does not qualitatively change our results.

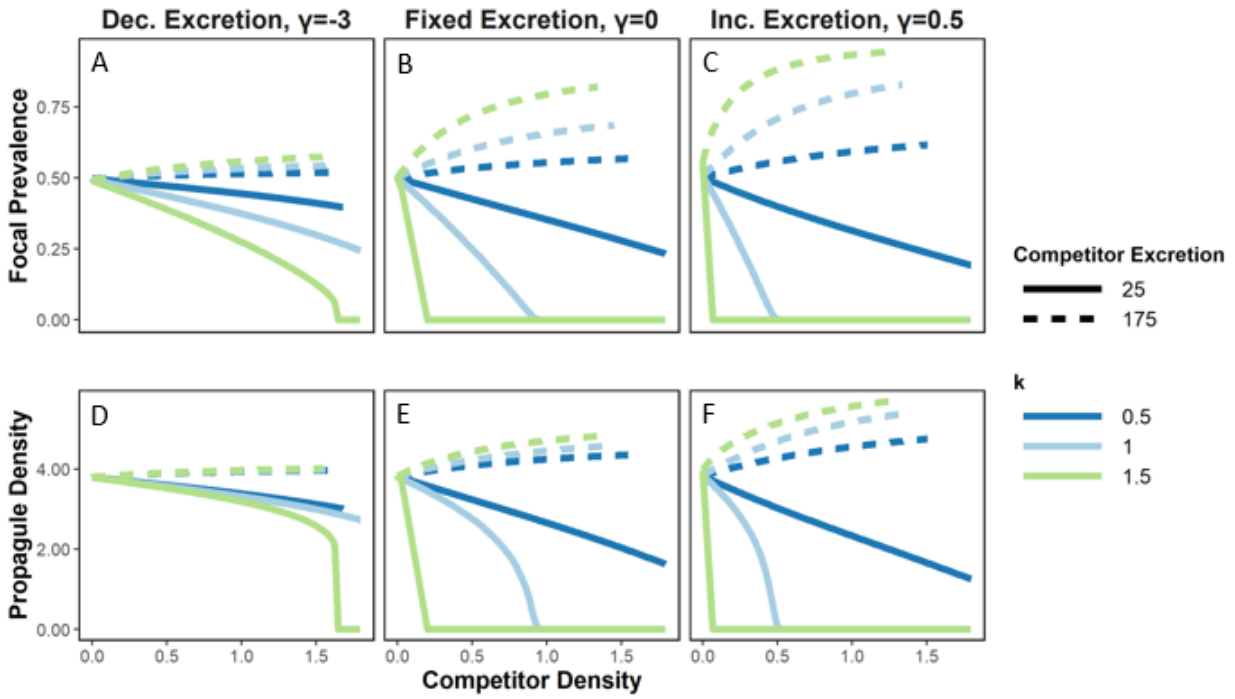

Figure S13- Figure 4 in main text, except that $m_2 = 0.6$

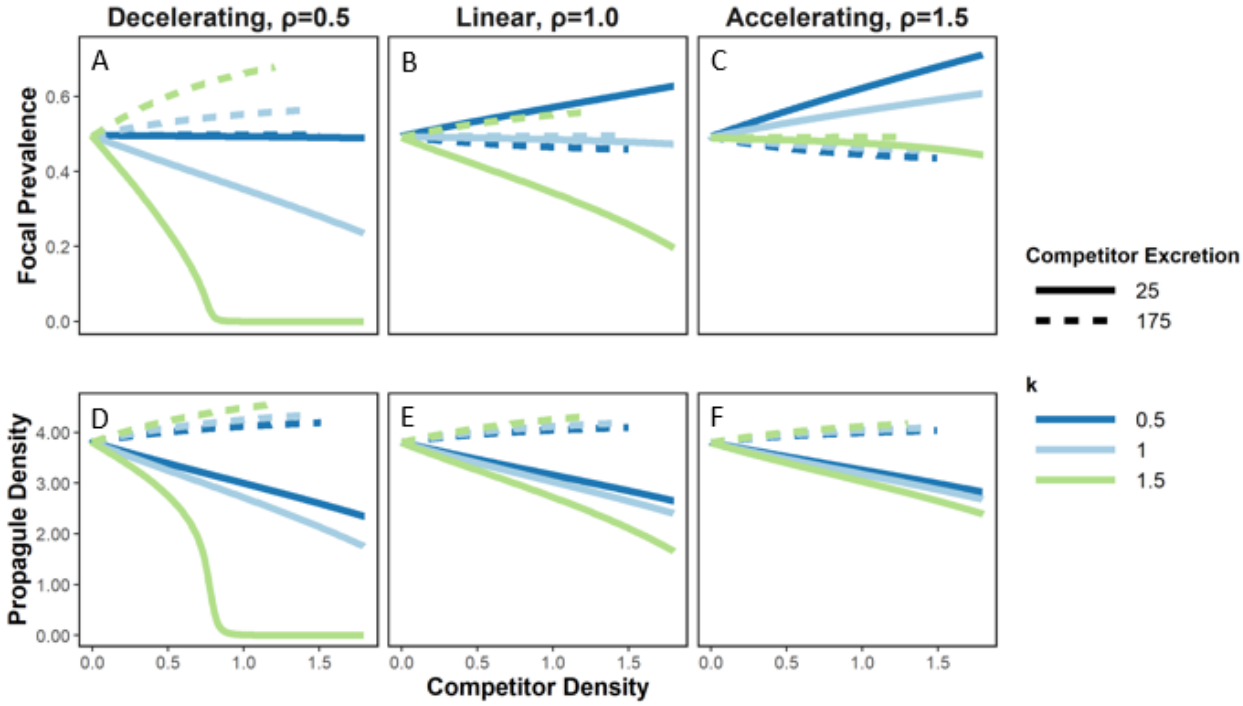

Figure S14- Figure 5 in main text, except that $m_2 = 0.6$

Section S8- Non-focal host has a lower mortality rate than focal host. ($m_2 = 0.2$)

Decreasing m_2 does not qualitatively change our results.

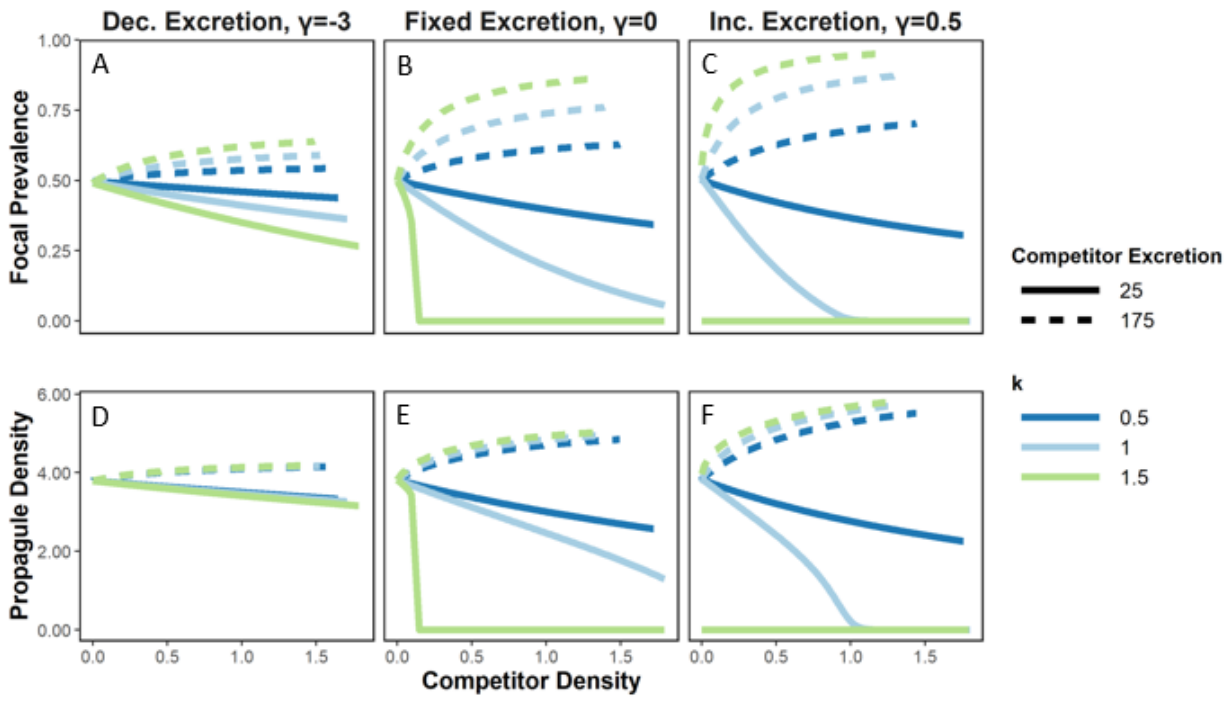

Figure S15- Figure 4 in main text, except that $m_2 = 0.2$

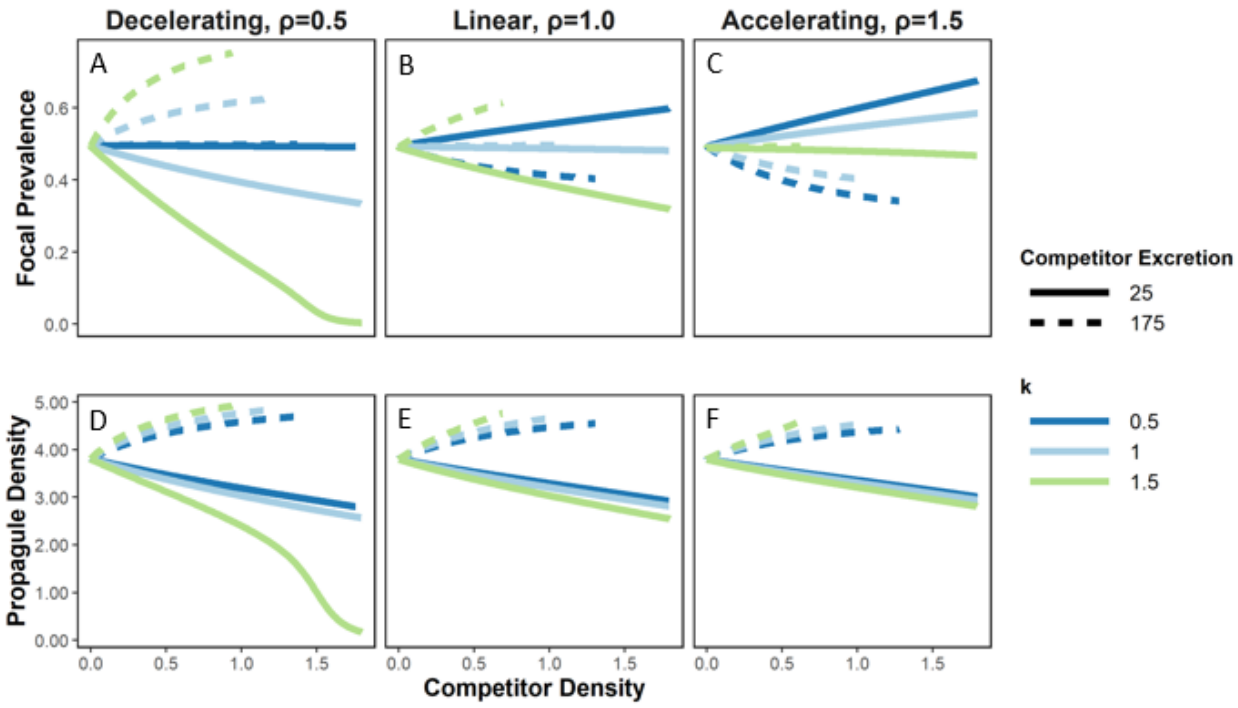

Figure S16- Figure 5 in main text, except that $m_2 = 0.2$

Section S9- Non-focal hosts have different dose-infectivity relationships than focal hosts. ($k_2 \neq k_1$)

To test asymmetric dose-infectivity relationships, we also needed asymmetric values of β, as β is a function of k. Thus, we calculated β_2 using Eq. 8, substituting k_2 for k_1. In Figures S17 and S18, k_2=0.5, in Figures S19 and S20, k_2=1.0, and in Figures S21 and S22, k_2=1.5. Thus, propagule density converges to the same value at the point when the competitor drives the focal host extinct within each figure regardless of the value of k_1, as k_1 does not impact disease dynamics in the absence of the focal host.

Our results do not qualitatively change for the most part if $k_2 \neq k_1$. The only qualitative change we find is that if k_2=0, k_1=1.5, the dose-excretion relationship is static or positive, and the competitor has low competence (excretion = 25), increasing competitor density first

decreases focal host infection prevalence and propagule density, before increasing them again (Figure S17). This is because initially, increasing low-competence competitor density creates a large decrease in propagule density due to positive feedback loops (positive dose-excretion, accelerating dose-infectivity). However, as the competitor density increases further and eventually drives the focal host to extinction, the propagule density must increase to converge upon the propagule density at equilibrium in the absence of the focal host (Figure S17).

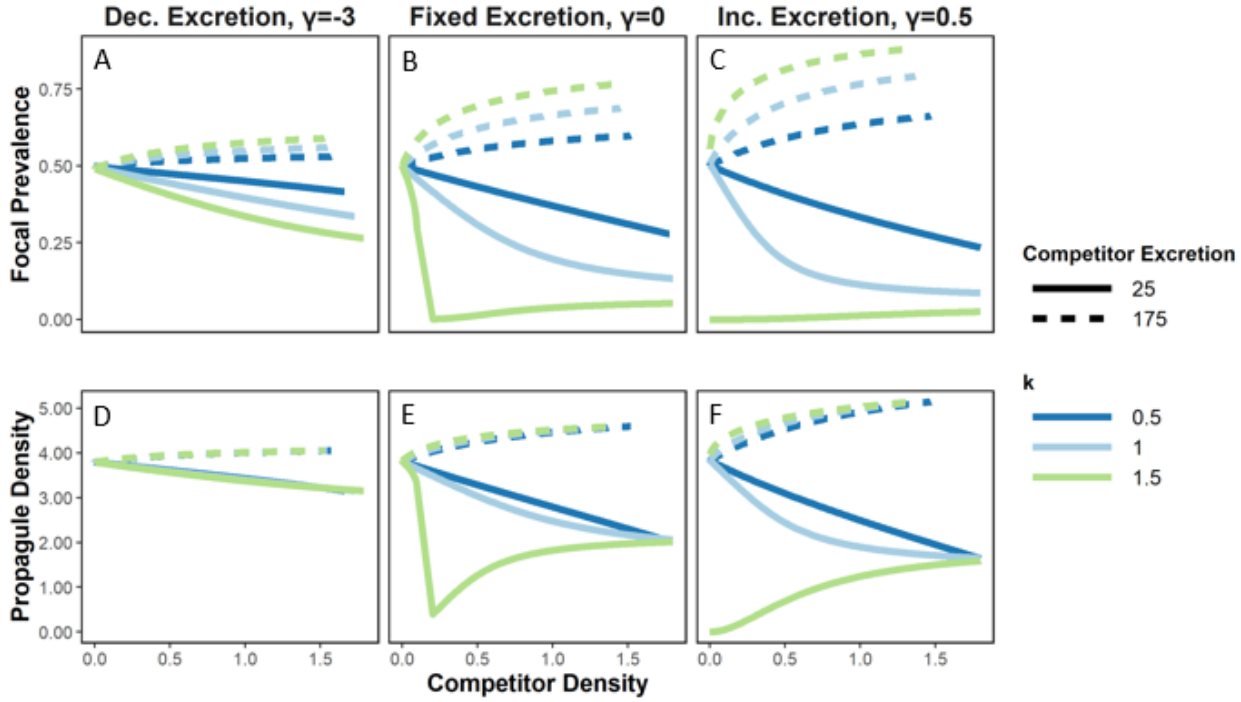

Figure S17- Figure 4 in main text, except that $k_2 = 0.5$

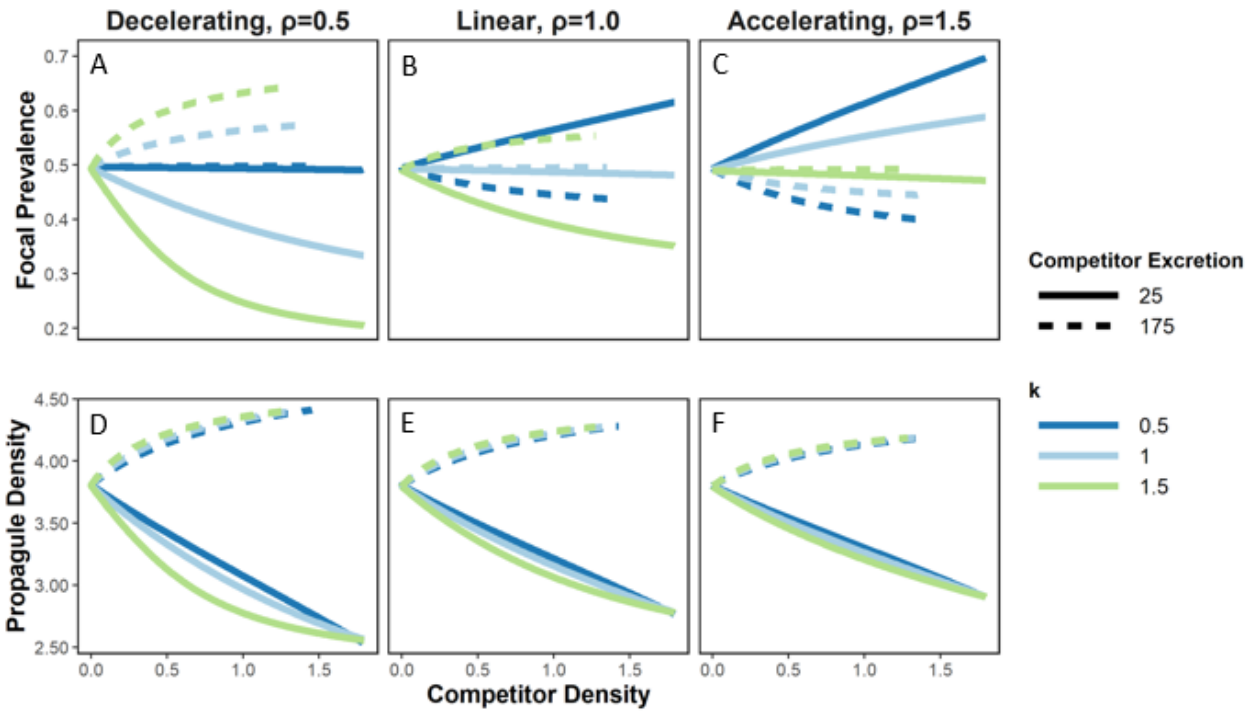

Figure S18- Figure 5 in main text, except that $k_2 = 0.5$

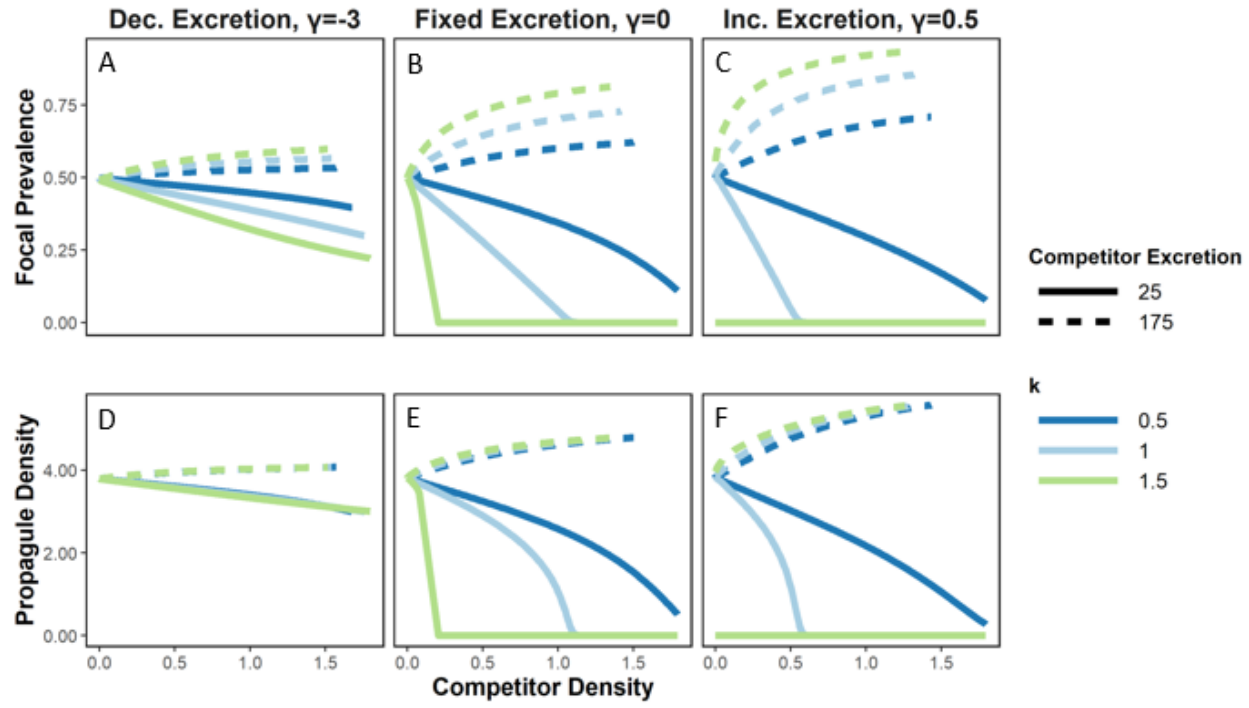

Figure S19- Figure 4 in main text, except that $k_2 = 1.0$

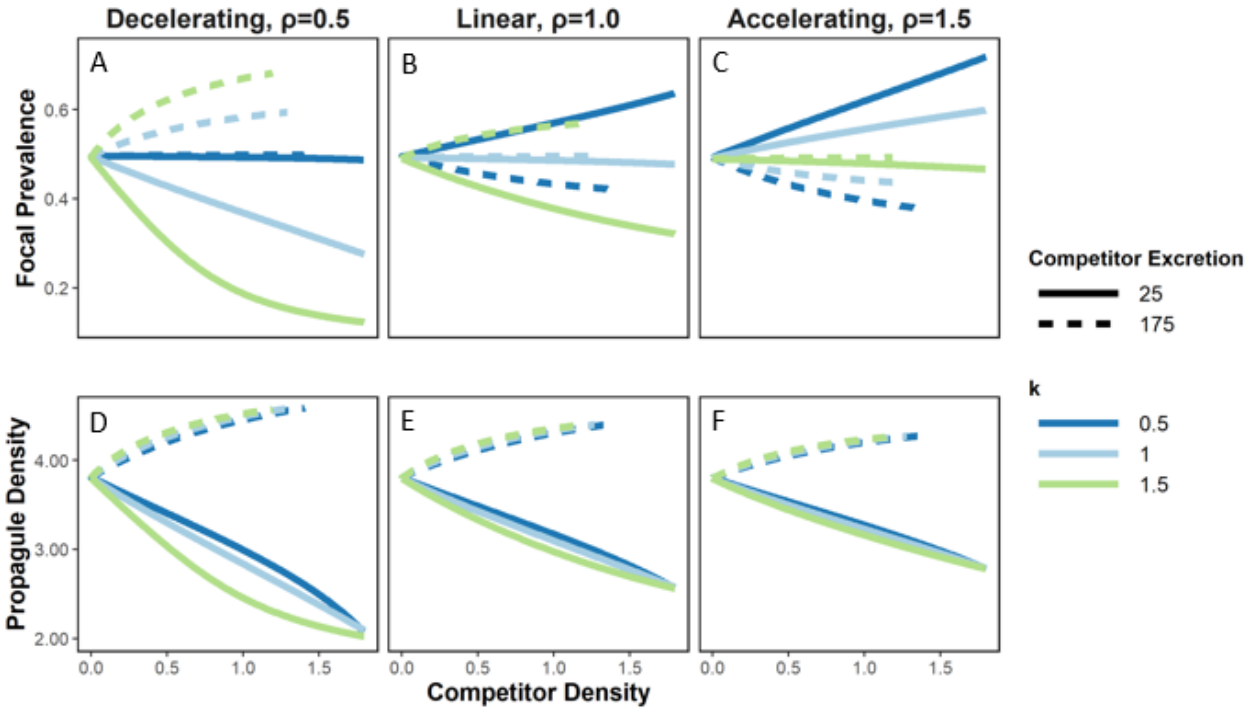

Figure S20- Figure 5 in main text, except that $k_2 = 1.0$

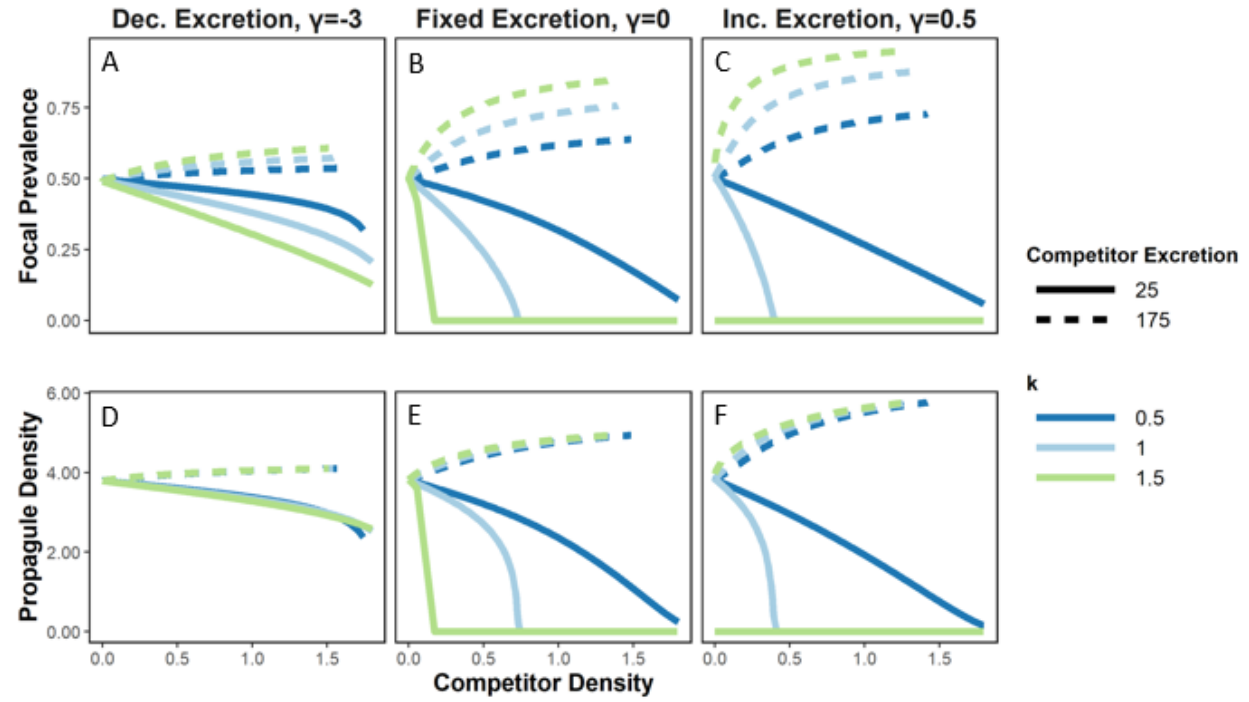

Figure S21- Figure 4 in main text, except that $k_2 = 1.5$

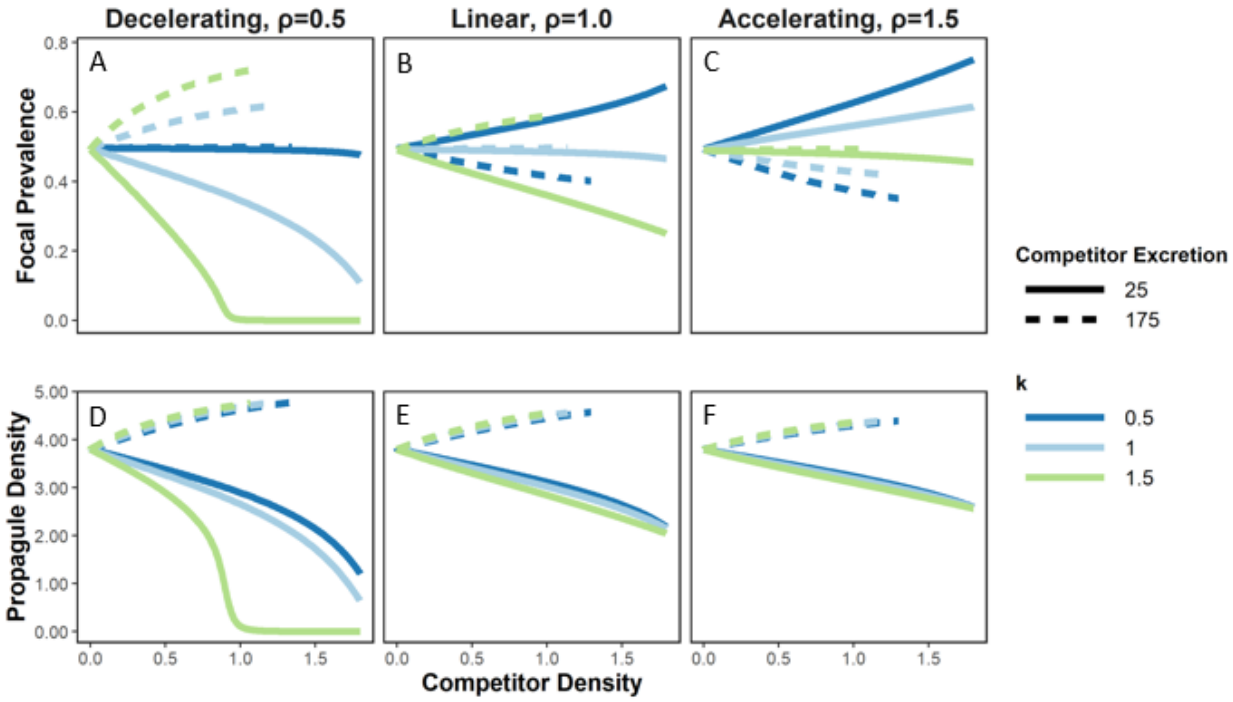

Figure S22- Figure 5 in main text, except that $k_2 = 1.5$

Section S10- When the focal host cannot sustain transmission.

In this scenario we examine the impact of inter-specific host density on infection prevalence in a focal host when the focal host cannot maintain the parasite as an endemic infection without the competing host. Thus, we model a scenario where $x_1 = 0$ and $x_2 = 100$, and examine the infection prevalence in N_1 as we increase r_2. In other words, how does the density of a competent reservoir host impact spillover into a non-competent focal host? We find that dose relationships alter the minimum competitor density needed to sustain infection in the focal host.

Decelerating dose-infectivity relationships promote outbreaks at lower competitor densities than accelerating dose-infectivity relationships (Figure S23). This pattern emerges because low propagule doses create a higher infection rate under decelerating dose-infectivity relationships than under accelerating dose-infectivity relationships (Figure 1A). This mirrors past work that accelerating dose-infectivity relationships create Allee effects during parasite invasion (Regoes et al., 2002). However, accelerating dose-infectivity relationships only facilitate Allee effects if the infection rate of linear, decelerating, and accelerating dose-infectivity relationships intercept at a dose greater than 1 (as in Figure 1A). If dose-infectivity relationships intercept at a dose of 1 infectious propagule (i.e. β not changing with k in our model), then increasing k can only increase infection rate, and thus reduce Allee effects.

Dose-excretion and dose-mortality relationships can also alter the minimum competitor density needed for parasite persistence in the focal host, depending on whether these relationships increase or decrease transmission with dose. When propagule excretion increases with dose, then a higher dose is needed for hosts to excrete enough propagules to maintain transmission, thus increasing the strength of the Allee effect (Figure S23,S24). On the other hand, when propagule excretion decreases or host mortality increases with dose, this increases the transmission potential of propagules at low doses, thus weakening the Allee effect . Once again, these patterns hold true only if various dose-excretion and dose-mortality relationships intercept at a dose greater than 1 (as in Figure 1 C,D). If the dose-excretion or dose-mortality relationships intercept at a dose of 1 infectious propagule (i.e. $f_i P_1 = 1$ in our model), then increasing γ or decreasing ρ can only increase infection rate, and thus reduce Allee effects.

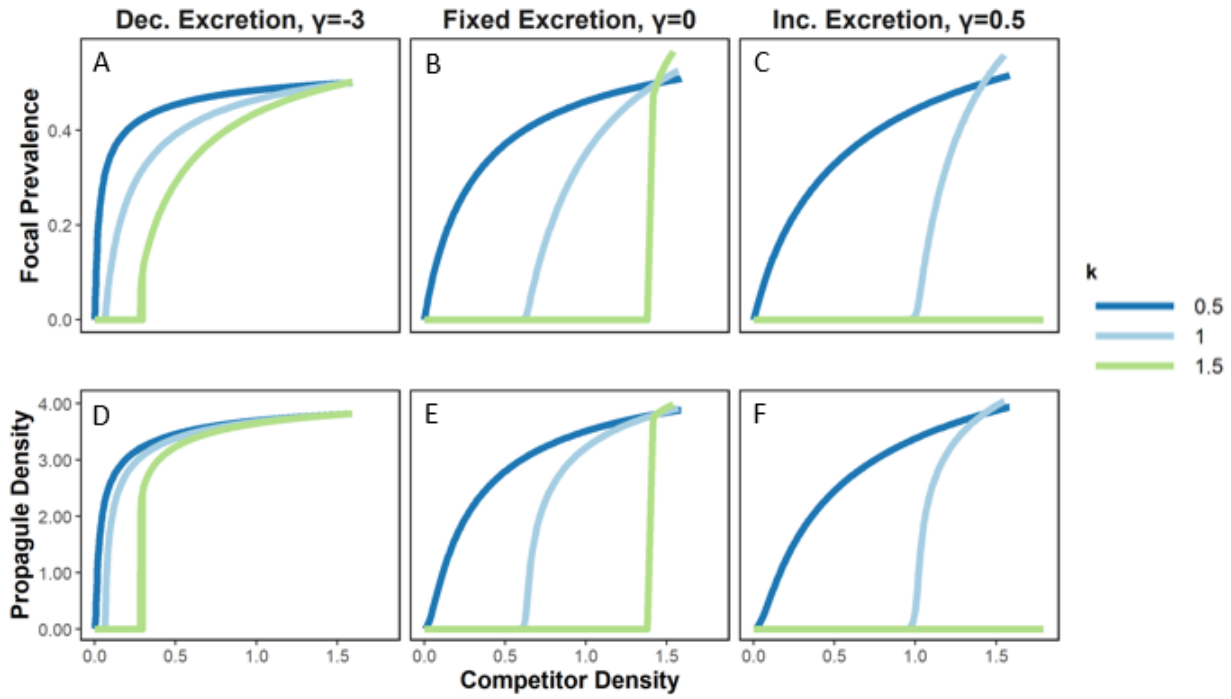

Figure S23- Figure 4 in main text, except that $x_1 = 0$ and $x_2 = 100$. β_1 and β_2 are equal and calculated so that infection prevalence is 0.5 in the competing host when the focal host is absent and $r_2 = 1$ based on Eq. 8. Note that in C and F, compounded positive feedback loops from dose-excretion relationships and dose-infectivity relationships mean that infection prevalence and propagule density are both 0 as long as the non-competent focal host has a density of greater than 1.

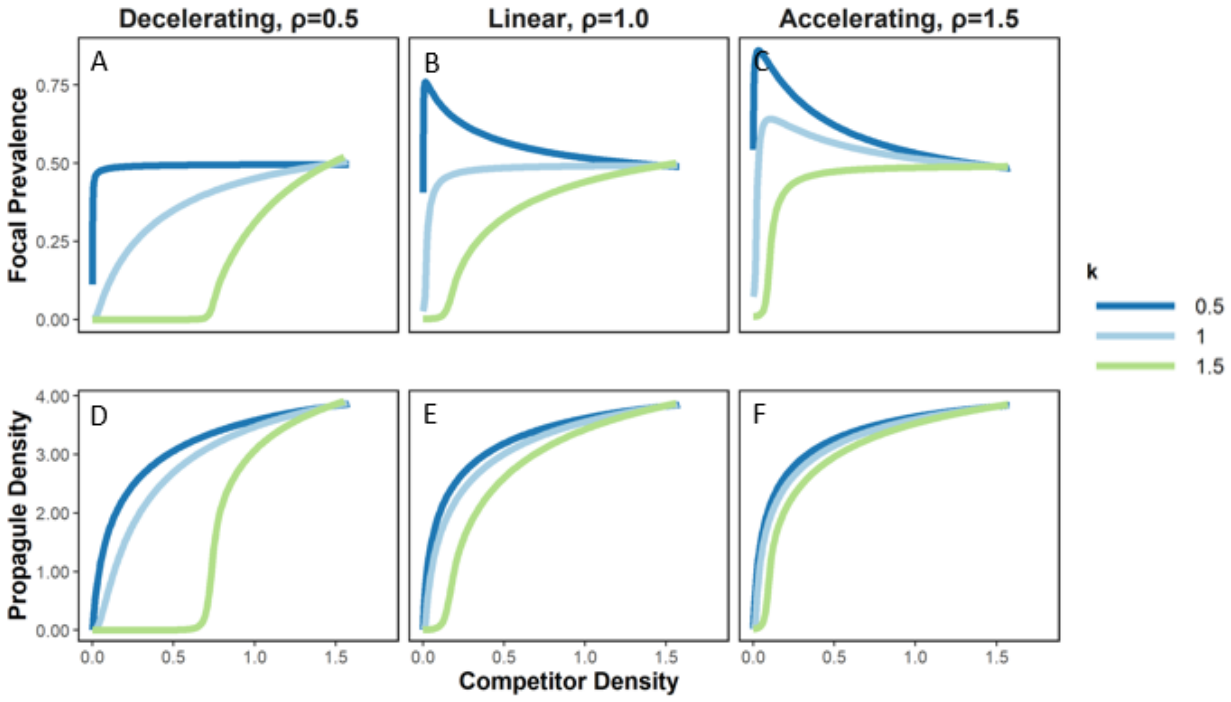

Figure S24- Figure 5 in main text, except that $x_1 = 0$ and $x_2 = 100$. β_1 and β_2 are equal and calculated so that infection prevalence is 0.5 in the competing host when the focal host is absent and $r_2 = 1$ based on Eq. 8. As in figure 5, increases in competitor density can have opposite effects on infection prevalence in the focal host and propagule density, depending on whether infected host mortality or the infection rate change faster with dose.

Section S11- Stacked dose-excretion and dose-mortality relationships

In our meta-analysis, we found several studies that documented positive dose-excretion relationships in addition to dose-mortality relationships. In our main text, we show the impact of the cumulative effects of positive dose-excretion and dose-mortality on friendly competition. Here, we show the simultaneous impacts of both dose-relationships on the impact of competitor host density on infection prevalence in a focal host and infectious propagule density. We found that the patterns were qualitatively no different than if we had dose-mortality relationships alone.

However, since positive dose-excretion relationships create positive feedback loops, these changes happen over a smaller range of competitor host densities (Figure S25).

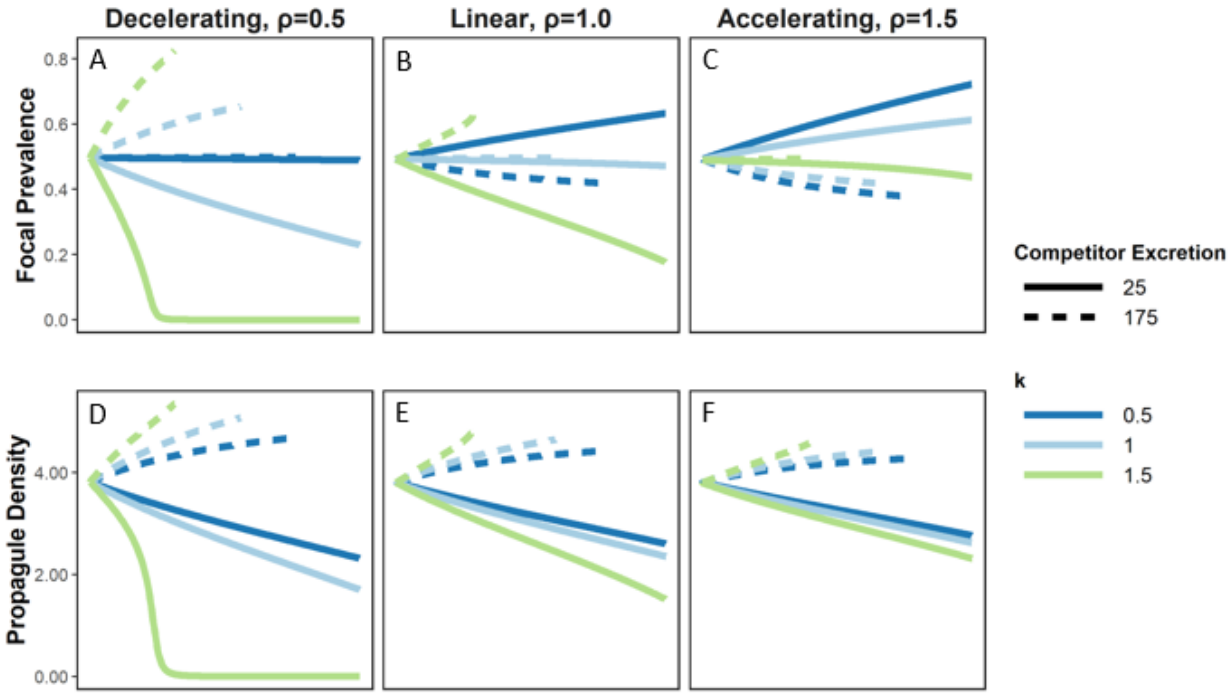

Figure S25- Figure 5 in main text, except that $\gamma = 0.5$.