How to outrun your parasites (or mutualists): symbiont transmission mode is key

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Abstract

Interspecific interactions shape how and when species (and population) ranges change. Natural enemies (like parasites) can slow population spread, or, conversely, a population can 'outrun' its enemies and spread uninhibited. Yet, less is know about how mutualistic interactions shape population spread, and what role 'outrunning' mutualistic partners plays. Here, I examine host-symbiont interactions specifically (where a symbiont species lives in/on a host species); common across animals and plants, and spanning the spectrum from parasitism to mutualism. I develop a model to determine when a symbiont shapes its host's population spread versus when the host outruns its symbiont. I find that symbiont transmission mode is key. For density-dependent transmission, symbionts cannot be sustained at the low-density population edge and the host outruns its symbiont, whereas frequency-dependent transmission leads to symbionts affecting host spread. However, this pattern breaks down in the presence of a host Allee effect; spread dynamics switch from 'pulled' to 'pushed', enabling a symbiont to influence population spread from behind the range edge. Overall, mutualistic symbionts speed up (and parasitic symbionts slow down) host population spread. These findings indicate that contact structures within a population (which shape symbiont transmission) are critical for determining whether host-symbiont interactions influence population spread.

Title

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Abstract

Interspecific interactions shape how and when species (and population) ranges change. Natural enemies (like parasites) can slow population spread, or, conversely, a population can ‘outrun’ its enemies and spread uninhibited. Yet, less is know about how mutualistic interactions shape population spread, and what role ‘outrunning’ mutualistic partners plays. Here, I examine host-symbiont interactions specifically (where a symbiont species lives in/on a host species); common across animals and plants, and spanning the spectrum from parasitism to mutualism. I develop a model to determine when a symbiont shapes its host’s population spread versus when the host outruns its symbiont. I find that symbiont transmission mode is key. For density-dependent transmission, symbionts cannot be sustained at the low-density population edge and the host outruns its symbiont, whereas frequency-dependent transmission leads to symbionts affecting host spread. However, this pattern breaks down in the presence of a host Allee effect; spread dynamics switch from ‘pulled’ to ‘pushed’, enabling a symbiont to influence population spread from behind the range edge. Overall, mutualistic symbionts speed up (and parasitic symbionts slow down) host population spread. These findings indicate that contact structures within a population (which shape symbiont transmission) are critical for determining whether host-symbiont interactions influence population spread.

Keywords

Allee effect, dispersal kernel, expansion rate, frequency-dependent transmission, integrodifference equation, invasion speed

Introduction

Species ranges are dynamic, and understanding why, how, and when they change is critical to controlling the spread of invasive species [1], facilitating the success of species reintroductions [2], and understanding the dynamics of climate-induced range shifts and expansions [3]. Yet, predicting which species (or populations) will spread and how fast remains a challenge [4,5]. One reason why is because spatial spread is governed by the processes acting at the low-density population edge [6], which may be different than the processes acting at higher population density, once equilibrium is reached. For example, a population's rate of spread can be estimated by how fast individuals reproduce and disperse at low density (first formulated by Skellam [7] for population spread, building on work by R.A. Fisher to model allele spread in the context of eugenics). However, the relationship between reproduction, dispersal, and spread can be complicated by other factors acting at low density such as Allee effects (e.g., due to mate-finding difficulties) [8], as well as stochastic processes [9]. Successful population spread is also shaped by species traits, interspecific interactions, and the environmental context [4,10,11].

Interspecific interactions in particular can influence the rate of population spread. Biocontrol modelling studies [12,13] have demonstrated that one species can indeed change how fast another spreads: namely that a species invasion can be slowed (or reversed) by the subsequent introduction of a predator that feeds on the invader (e.g., Lepidopterans feeding on lupin plants [12]). Similarly, generalized host-pathogen models show that pathogens can slow or reverse the spread of their host population [14,15]. In contrast, less is known about how positive

interspecific interactions (like mutualism and facilitation) shape rates of population spread. One model demonstrated that interspecific interactions (including mutualism) may favour reduced dispersal, leading to slower rates of population spread compared to species that are spreading in isolation [16]. However, it is unclear how sensitive these results are to model assumptions and parameter values. For invasive species in particular, interspecific interactions are thought to be important during the initial establishment period (prior to spread). The enemy release hypothesis postulates that species 'escape' their enemies (predators, pathogens, parasites, herbivores) when they colonize a new area; an idea supported from a range of empirical systems (plants and animals, in marine and terrestrial environments [17–19]). The flip side of enemy release is that invasive species may also 'escape' their mutualistic partners [20]; many invasive species, particularly plants, are constrained by a lack of mutualistic partners (e.g. mycorrhizal fungi, insect pollinators and vertebrate seed dispersers [21–23]). Intriguingly, although escape from enemies (and mutualists) is typically described during the introduction stage of an invasion, it may also be important in shaping subsequent population spread [24,25] where a population effectively 'outruns' enemies or mutualists, although this is not well studied (but see [26]). If so, the potential for a population to outrun its parasites (or mutualists) could affect spread dynamics not only for invasive species, but also for species that are spreading/shifting outwards from established ranges, as in the case of range shifts and reintroductions.

A good case study for exploring these research gaps are host-symbiont species pairs (which form an intimate association whether positive, neutral, or negative; [27]), where the movement of the smaller partner (the 'symbiont') depends largely on the movement of the larger partner (the

'host'). Host-symbiont interactions are widespread across organisms, encompassing plants and mycorrhizal fungi [28], animals and their gut microbes [29,30], coral and zooxanthellae [31], even bacteria and conjugative plasmids [32]. Host-symbiont pairs are likely to be introduced together (given their close proximity and dependency) and thus have the potential to spread jointly. These relationships can also be mutualistic, commensal, or parasitic [28] (with the magnitude and sign of their interaction often depending on context; [33]), thus providing an opportunity for a broad spectrum of interaction types to be explored. However, theory on host-symbiont interactions has, for the most part, developed separately for parasitic and mutualistic symbionts [34]. One challenging in bridging this divide is terminology differences in how symbiont transmission is described. For parasitic symbionts (e.g., pathogens), a key division in transmission is frequency-dependent (host contact rate leading to transmission does not scale with population abundance) versus density-dependent (host contact rate increases as host abundance increases) [35]. Frequency-dependent transmission is best used to describe sexually transmitted or vector-borne pathogens; and density-dependent for most other pathogens [36]. For mutualistic symbionts, a key division is vertical (symbionts passed from parent to offspring) versus horizontal (symbionts acquired after birth) transmission [37]. Although the terms 'density-dependent' and 'frequency-dependent' are rarely used to describe different cases of horizontal transmission, mathematical functions capturing these differences have been used in symbiont models: frequency-dependent for fungal symbionts of plants [34] and density-dependent for zooxanthellae symbionts of corals [31]. Furthermore, there is good justification for considering both transmission modes. Transmission of mutualistic symbionts depends on host contact structure (just like for parasitic symbionts); thus, when host contacts are structured either

spatially (e.g., plant mycorrhizal networks; [38]) or socially (e.g., baboon gut microbes [39]), transmission will be best described by frequency-dependent, while transmission driven by unstructured contacts (e.g., corals [31]) will be best described by density-dependent. Taken together, this suggests that a single modelling framework could capture parasitic and mutualistic symbionts as ends of a spectrum [34].

One clue to filling the above knowledge gaps (under what conditions hosts outrun their parasitic and mutualistic symbionts versus not) comes from the literature on species' range limits. General theory predicts that a host species' range can be limited by a pathogen that is present at the host population edge [40], i.e., pathogens that persist even at low population density (assuming that host abundance decreases at the population range edge). Pathogens with frequency-dependent transmission can persist at low density (since contact rate does not decrease as host density decreases); in contrast, pathogens with density-dependent transmission (where contact rate decreases as host abundance decreases) have a threshold host density below which they cannot persist [35], thus would be absent from a host population's range edge and thus cannot influence host range limits. Indeed, a pathogen with frequency-dependent transmission (anther-smut disease, caused by a pollinator-transmitted fungus) was present all the way to range edge in several alpine plant host species [41].

Here, I draw on ideas from species range limits, to study population spread in host-symbiont pairs. I develop a general modeling framework that encompasses both parasitic and mutualistic symbionts, and consider symbiont effects on each host reproduction and survival. I determine

under what conditions hosts are about to outrun their symbionts and, conversely, under what conditions the host population spread rate is influenced by its symbiont. Overall, I find that the mode of transmission is key: symbionts with frequency-dependent transmission influence host spread rate, while those with density-dependent transmission fall behind the population edge, thus mirroring the findings from species range limits.

Methods

I built a spatially-explicit population-based model that tracks hosts explicitly and symbionts implicitly (see Table 1 for model parameters). I track the density (per unit area) of unpartnered (U; without a symbiont) and partnered (P; carrying a symbiont) hosts in the population across space (x, one-dimensional) at each discrete time point (t) as $U_t(x)$ and $P_t(x)$. During each year (t) the processes of transmission, survival, reproduction, and dispersal occur, sequentially (see Figure 1 for this annual cycle).

Symbiont Transmission

Transmission of symbionts between hosts occurs locally at each point in space (x) and continuously during the first period of each year (Figure 1, thick arrow). Transmission can be density-dependent (i.e., the per capita rate that an unpartnered host becomes partnered increases as partnered host density, P, increases) with dynamics given by

$$\begin{aligned} \frac{dU}{dt} &= -\beta UP \\ \frac{dP}{dt} &= \beta UP \end{aligned} \tag{1}$$

where β is the transmission rate between unpartnered (U) and partnered (P) hosts. Alternatively,
144 transmission can be frequency-dependent (i.e., the per capita rate that an unpartnered host
becomes partnered increases as the frequency of partnered hosts within the population, $P/(U+P)$
146 increases) with dynamics given by

$$\frac{dU}{dt} = -\beta U \left(\frac{P}{U+P}\right)$$
$$\frac{dP}{dt} = \beta U \left(\frac{P}{U+P}\right) \quad . \tag{2}$$

150 Transmission occurs continuously during a fraction τ_1 of the year ($0 \leq \tau_1 \leq 1$). Thus, at the end of
the transmission period, the population size of unpartnered and partnered hosts can be found by
152 integrating eqn (1) and (2) and is given by

$$U_{t+\tau_1}(x) = \frac{U_t(x)[U_t(x)+P_t(x)]e^{-\beta\tau_1[U_t(x)+P_t(x)]}}{U_t(x)e^{-\beta\tau_1[U_t(x)+P_t(x)]}+P_t(x)}$$
$$P_{t+\tau_1}(x) = \frac{P_t(x)(U_t(x)+P_t(x))}{U_t(x)e^{-\beta\tau_1[U_t(x)+P_t(x)]}+P_t(x)} \tag{3}$$
154

156 in the case of density-dependent transmission (from eqn. 1) and

158
$$U_{t+\tau_1}(x) = \frac{U_t(x)[U_t(x)+P_t(x)]}{U_t(x)+P_t(x)e^{\beta\tau_1}}$$
$$P_{t+\tau_1}(x) = \frac{P_t(x)[U_t(x)+P_t(x)]}{P_t(x)+U_t(x)e^{-\beta\tau_1}} \tag{4}$$

160 in the case of frequency-dependent transmission (from eqn. 2).

162 ***Host Survival and Reproduction***

Next, I account for host demography. A fraction σ_U of unpartnered hosts and fraction σ_P of partnered hosts survive; now the host population at this point ($t + \tau_2$) is given by

$$\begin{aligned} U_{t+\tau_2}(x) &= \sigma_U U_{t+\tau_1}(x) \\ P_{t+\tau_2}(x) &= \sigma_P P_{t+\tau_1}(x) \end{aligned} . \quad (5)$$

Surviving hosts produce ϕ_U offspring per unpartnered host and ϕ_P offspring per partnered host. Reproduction is density-dependent where the strength of density-dependence is given by

$$g_{t+\tau_2}(x) = \begin{cases} 0, & \text{if } N_{t+\tau_2}(x) < a \\ \frac{b}{b+N_{t+\tau_2}(x)}, & \text{otherwise} \end{cases} \quad (6)$$

with Allee threshold a, density dependence parameter b and where $N_{t+\tau2}(x) = U_{t+\tau2}(x) + P_{t+\tau2}(x)$ is the total current local host population size. A population is not viable wherever its size falls below the Allee threshold (a). I chose to include an Allee effect because doing so can alter the dynamics of population spread across space [42,43]. Namely, without an Allee effect, a population's spread rate is determined by how fast hosts at the low-density population edge reproduce and disperse [7]. However, in the presence of an Allee effect, a population goes extinct anywhere it is below the Allee threshold a (i.e., on the population edge). In this case, the population spread rate is instead determined by how fast hosts at higher density (above the Allee threshold a) behind the population edge are 'pushed' forward spilling past the population edge. Overall this means that without an Allee effect, spread rate is determined by factors that affect populations at low density only, whereas with an Allee effect spread rate can be determined by factors that affect populations at higher density. This growth function (eqn. 6) enables me to explore population dynamics either in the presence (setting $a > 0$) or absence (setting $a = 0$) of an

Allee effect. I assume that all offspring are born unpartnered (i.e., no vertical transmission). The population size of unpartnered and partnered hosts at time $t + \tau_3$ is

$$\begin{aligned} U_{t+\tau_3}(x) &= U_{t+\tau_2}(x) + [\phi_U U_{t+\tau_2}(x) + \phi_P P_{t+\tau_2}(x)] g_{t+\tau_2}(x) \\ P_{t+\tau_3}(x) &= P_{t+\tau_2}(x). \end{aligned} \tag{7}$$

where the first term of each equations accounts for hosts surviving from one year to the next, while the other terms in the *U* equation account for newborn hosts (i.e., generations are overlapping).

Host Dispersal

Finally, all hosts disperse, according to a dispersal kernel, $k(x\text{-}y)$, that gives the proportion of hosts starting at a location *y* that disperse to each other location *x*. In particular, I use the Laplace dispersal kernel

$$k(x-y;v) = \frac{1}{\sqrt{2v}} \exp\left[-\sqrt{\frac{2(x-y)^2}{v}}\right] \tag{8}$$

where *v* is the variance (v_U for unpartnered hosts, v_P for partnered hosts). The Laplace kernel is effectively a negative exponential distribution in two directions (positive and negative along the x-axis); one of the most commonly used functions to describe dispersal, and which has been found to be a good fit to empirical data from plants and animals [44,45]. Note that symbionts are only able to move when carried by a host. Finally, the population size after dispersal is given by the pair of integrodifference equations

$$\begin{aligned} U_{t+1}(x) &= \int_{-\infty}^{\infty} k(x-y;v_U) U_{t+\tau_3}(y)\, dy \\ P_{t+1}(x) &= \int_{-\infty}^{\infty} k(x-y;v_P) P_{t+\tau_3}(y)\, dy \end{aligned} \tag{9}$$

where the difference in the unpartnered and partnered hosts population density from one point (t+τ3) to the next (t+1) is found by summing up (integrating) the hosts across all possible starting points (y) in space that end up at each end point in space (x).

Simulations

To run the model, I numerically simulate the equations describing population size (eqns. 3, 4, 5, 7, 9). I initialized each simulation with a high density of unpartnered and partnered hosts in the centre of space ($U_0(x) = 6$; $P_0(x) = 2$ for $|x| < 0.1$; Figure 2a, b). I iterated the model forward 150 times ($t = 150$), recording the local density of U and P hosts at each year (t) over space (x). To quantify the host population spread rate, I determined the location of the population edge (the farthest point where the host density $U_t + P_t$ exceeded a threshold of 0.001) for each t and I took the difference in population edge location from one time to the next as the spread rate.

Scenarios

In addition to exploring the effect of transmission mode (frequency-dependent, density-dependent), I also explored different symbiont effects. Namely, I considered cases where the symbiont had a positive or negative effect, I varied the magnitude of the effect on hosts (n between -0.25 and 0.25), as well as the 'currency' (affecting survival or fecundity). When symbionts affect survival, the demographic rates of partnered hosts are given by $\sigma_P = \sigma_U + n$ and $\phi_P = \phi_U$, and when symbionts affect fecundity, they are given by $\sigma_P = \sigma_U$ and $\phi_P = \phi_U + n$, where n is the symbiont's net effect on the host. In other words, mutualistic symbionts ($n > 0$) increase either host survival or fecundity, while parasitic symbionts ($n < 0$) decrease either host survival

or fecundity. Note that I do not consider values of n that would lead to biologically unreasonable survival or fecundity (i.e., I restrict n such that $0 \leq \sigma_P \leq 1$ and $0 \leq \phi_P$).

Results

Within each simulation, the introduced host population initially increased in size, then spread out across space (Figure 2). In some cases, the host population spatially 'outran' the symbiont; that is the symbiont was absent from the edge of the host population in a 'halo' (Figure 2a,c,e, pale grey regions). In other cases, the host population did not outrun and the symbiont was present close to the population edge (Figure 2b,d,f). Whenever the symbiont was outrun by the host population, it had no effect on the rate of population spread, whereas when the symbiont persisted at the host population edge, it influenced the rate of population spread (Figure 3).

The mode of symbiont transmission was critical in determining which of these two outcomes occurred. When transmission was density-dependent, the host population outran the symbiont and thus its spread rate was independent of the symbiont effect on the host (Figure 3, open circles). However, when transmission was frequency-dependent, the host did not outrun the symbiont and the net effect of the symbiont on the host affected the population spread rate (Figure 3, closed circles). Mutualists (symbionts with a positive net effect) increased the host population spread rate while parasites (symbionts with a negative net effect) decreased the host population spread rate (Figure 3).

The ‘currency’ of the symbiont’s effect (whether it affected host fecundity or survival) influenced the outcome in two ways in the case of frequency-dependent transmission. First, when the symbiont was parasitic with a strongly negative effect on host survival (i.e., highly virulent), partnered hosts were quick to die and the remaining host population outran the symbiont (Figure 3b far left). Second, when the symbiont was mutualistic, host population spread much faster when the symbiont increased survival than when it increased fecundity (Figure 3a vs 3b).

Finally, the overall results pattern (symbionts with frequency-dependent transmission shape host spread while those with density-dependent transmission do not) could be disrupted by choosing extreme values for some model parameters. For example, as transmission rate (β) decreases, fewer hosts acquire the symbiont and the overall effect of the symbiont on host population spread was diminished (Figure 4a-b). For low enough transmission rate values (β), hosts were able to outrun the symbiont even with frequency-dependent transmission (Figure 4a-b, pale grey). Similarly, if hosts carrying a symbiont (partnered hosts) dispersed shorter distances (with dispersal variance given by v_P) than hosts without a symbiont (unpartnered hosts; dispersal variance given by v_U), where $v_P < v_U$), the symbiont had less of an effect on host spread (Figure 4c-d), and in the extreme, the host population was able to outrun the symbiont (Figure 4c-d, pale grey). Finally, if the host population was subject to a strong Allee effect (with Allee threshold a), even if the threshold was quite small, hosts were not able to outrun the symbiont even with density-dependent transmission (Figure 4e-f).

Discussion

Here, I developed a host-symbiont model to understand whether and how symbionts shape the spread rate of their host population. I find that mutualistic symbionts typically speed up host population spread rate while parasitic symbionts slow it down. However, the mode of symbiont transmission between hosts is key to whether symbionts affect host spread at all: with density-dependent (instead of frequency-dependent) transmission, hosts outrun the symbiont spatially (symbionts are absent from the host population edge), and thus, symbionts have no effect on host population spread rate.

My findings fill two gaps in the literature on interspecific interactions and population spread. First, I demonstrate that mutualistic interactions (here, mutualistic symbionts) can substantially increase spread rate, a step towards improving out understanding of how mutualisms shape population spread [11] and providing a counter-perspective to prior theory that showed mutualisms can slow down invasions [16]. Second, I show that hosts can indeed outrun enemies (here, parasitic symbionts or pathogens) during the spread phase of an invasion. This result confirms a previously suggested idea [25], and provides a deterministic mechanism, complementing a previously described stochastic mechanism (if spread occurs via a series of low-density founder events, pathogens will be lost through chance events; [26]). Past theory demonstrating that pathogens can slow host spread have explored either frequency-dependent [14,15], or density-dependent [26] transmission, not both; to my knowledge, the contrast between density-dependent and frequency-dependent transmission has not been made explicitly before in this context.

The work presented here also unites ideas from related biological fields. First, I show that the
296 ‘symbiont-free halos’ (per [41]) found at the edge of stationary ranges are also found at the edge of spreading ranges, and I confirm that transmission mode is the critical driver of when this
298 occurs [40]: density-dependent transmission creates a halo while frequency-dependent transmission does not. Thus, pathogens and symbionts with frequency-dependent transmission
300 have the potential to shape spreading population as well as stationary range limits, while those with density-dependent transmission do not. Second, I show that this transmission-based pattern
302 breaks down in the presence of an Allee effect, where even symbionts with density-dependent transmission can shape host population spread rate. In the presence of an Allee effect, a
304 spreading population transitions from what is called a ‘pulled wave’ (individuals at the edge ‘pull’ the population forward, thus spread rate is determined by details of the low density
306 population edge) to a ‘pushed wave’ (there are insufficient individuals at the population edge so individuals at higher density behind the edge ‘push’ the population forward, and spread rate is
308 determined by the high density core population) [42,43]. Thus, if a symbiont is present in the host population at high density (even if it is absent from the population edge), it can still shape
310 the rate of population spread when the host has an Allee effect. Circling back to stationary ranges, my results suggest that density-dependent pathogens may shape range limits if the host
312 population has an Allee effect.

314 My model could be expanded in a number of future directions. First, one could explore additional transmission modes. For example, the transmission of most pathogens is likely to be
316 somewhere between density-dependent and frequency-dependent; a type II functional response

behaves like density-dependent at low density and frequency-dependent at high density [46]. I predict that this transmission mode will produce results similar to those with density-dependent transmission: host populations will typically outrun pathogens with this transmission. In addition to being transmitted horizontally (the mode explored here), symbionts can be transmitted vertically (transmission from parents to offspring) or with a mixture of horizontal and vertical [37]. I expect that symbionts with vertical transmission will shape their host population spread rates, since they will be present across the host population range. As a final transmission example, one could consider environmental transmission (equivalent to a generalist symbiont that is present in another host species across the full environment); I predict that these symbionts will also shape population spread. As a second direction, given how often the sign and magnitude of species interactions depend on context [33], one could explore the consequences of interactions varying as a function of host population density during population spread. Third, one could explore how other types of mutualism shape spread rate. In particular, dispersive mutualisms where one partner is physically transporting the other is likely a critical interaction for population spread [11]. Fourth, one could explore what happens when symbiont partnering happens during the dispersal process itself. The transient phase of movement can expose organisms to novel parasites and pathogens [47] and moving is one of the ways unpartnered organisms can locate mutualistic partners [48].

Finally, despite the population-level focus of this work, my findings have intriguing implications in terms of individual-level behaviour. In particular, the mode of symbiont transmission (density- or frequency- dependent) is driven by individual contact behaviours and how they scale with

population density. Frequency-dependent transmission describes scenarios where contacts between individuals are structured and not shaped by local population density [49]. This mode includes species where individuals are sedentary (or territorial) and symbionts are passed to nearest neighbours (as for plant mycorrhizal networks; [38]), or where individuals pass symbionts along social networks [39]. In contrast, density-dependent transmission describes scenarios where contacts between individuals increase with density. This mode applies to corals [31] as well as many species with microbial symbionts [50]. Thus, individual-level contact behaviours determine whether symbionts can shape the population-level outcome of spread rate. This may in turn drive selective pressures on individuals (e.g., selection for increased dispersal to outrun parasites). My findings also demonstrate that strategies for managing population spread (in either biocontrol or reintroduction scenarios) should account for how the host population's behaviour shapes contacts and thus transmission. Species with frequency-dependent contact structure will be easier to manipulate with symbionts. In contrast, it will be harder to control spread rates for species with density-dependent contact structure, unless they are subject to an Allee effect.

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Data, code and materials

No new data was collected for this study. The model code and results generated for this study are available via Github (https://github.com/allisonkshaw/symbiontspread/) and will be deposited in Data Dryad upon manuscript acceptance.

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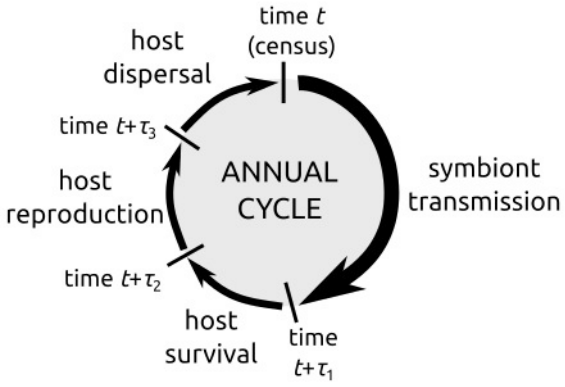

Figure 1. Schematic of the population's annual cycle. During a single year (starting with the census at time t, top of the circle), four processes occur sequentially. First, symbiont transmission occurs (thick arrow) for length of time τ_1 (from time t to $t+\tau_1$), at which point the population size is given by eqn. 3 or 4 (depending on transmission mode). Second, host survival occurs and the population size at time $t+\tau_2$ is given by eqn. 5. Third, host reproduction occurs, and the population size at time $t+\tau_3$ is given by eqn. 7. Finally, host dispersal occurs at which point the population size is given by eqn. 9, and the census for the following year (t+1) occurs. (Note that transmission is the only continuous process (while survival, reproduction and dispersal are discrete), so although τ_1 is meaningful, the values of τ_2 and τ_3 are not; they are just indicated to help explain how each process affects the population size.)

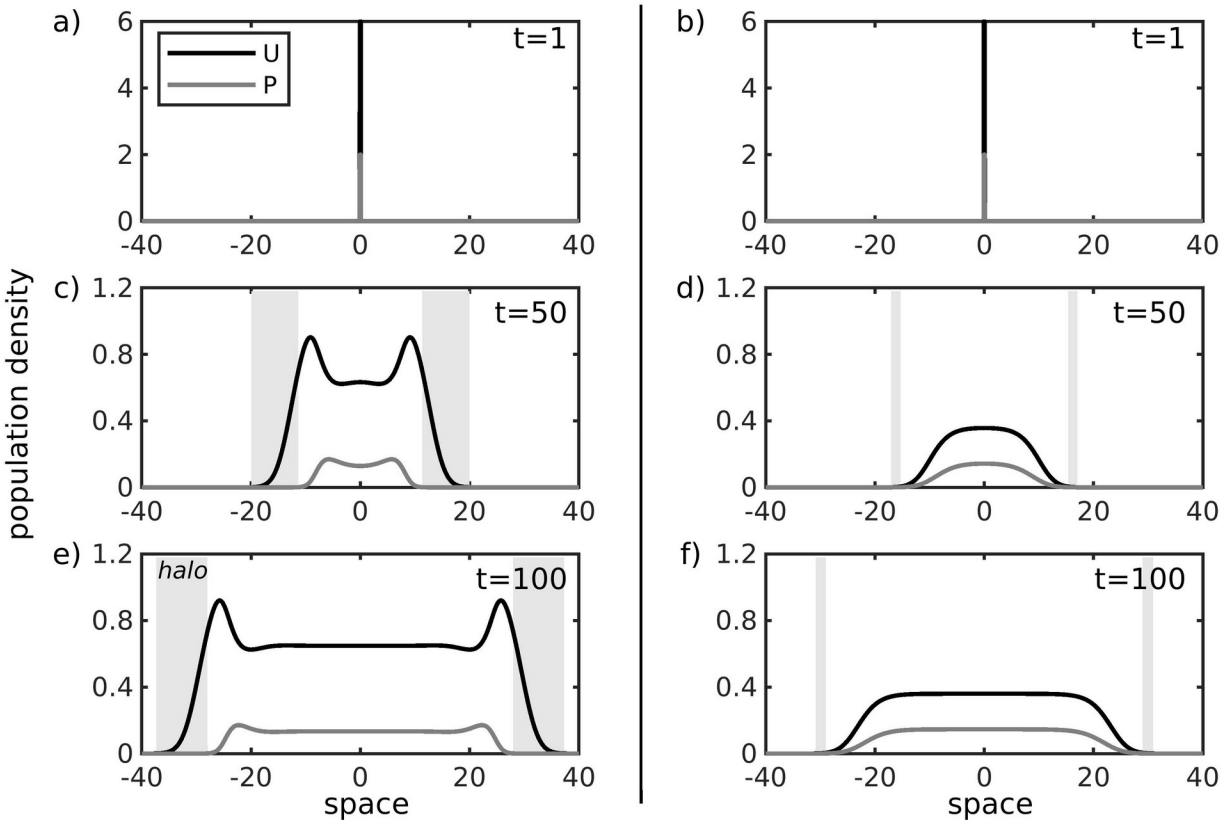

Figure 2. Hosts can either (a,c,e) 'outrun' the symbiont spatially or (b,d,f) not (symbiont is present across the population). Each panel shows the host population density across space for unpartnered (black) and partnered (grey) hosts at a different time (t) during the simulation. Symbiont-free 'halos' (where there are unpartnered hosts but not partnered ones) are shown in pale grey, and are much wider in the left panels than in the right ones. Parameters: $\beta = 2$, $\tau_1 = 0.5$, $\sigma_U = 0.7$, $\phi_U = 0.8$, $a = 0$, $b = 1$, $v_U = v_P = 0.25$, $n = -0.15$. Transmission is (ac,e) density-dependent and (b,d,f) frequency dependent.

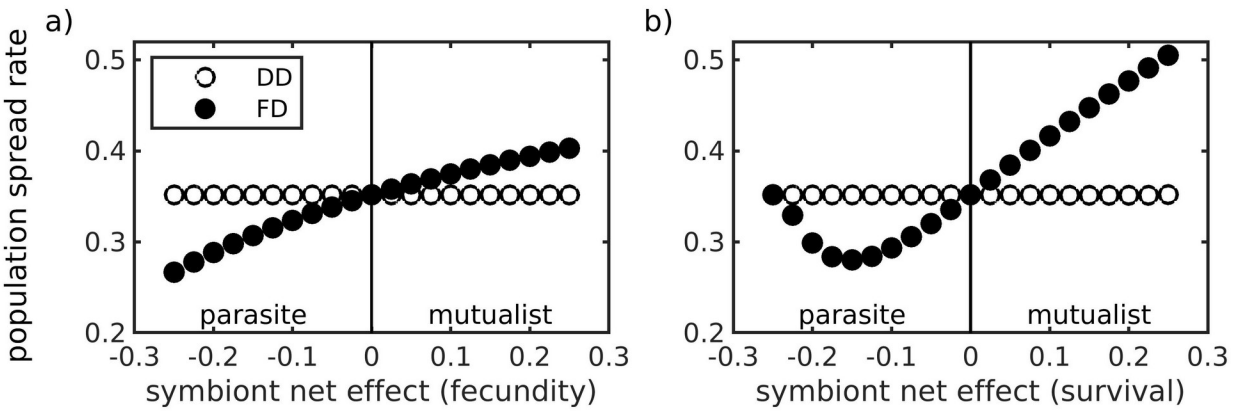

Figure 3. Hosts outrun the symbiont when transmission is density-dependent (DD: host spread is independent of the symbiont net effect, x-axis) but do not when transmission is frequency-dependent (FD; host spread shaped by symbiont). Host population spread rate as a function of the symbiont net effect (n) on the host for density-dependent (open black circles) and frequency-dependent (closed grey circles) when the symbiont affect host (a) fecundity and (b) survival. Symbionts are considered parasites when they have a net negative effect ($n < 0$) and mutualists when they have a net positive effect ($n > 0$). Parameters: the same as Figure 2 except with t = 150 and varying $-0.25 \leq n \leq 0.25$.

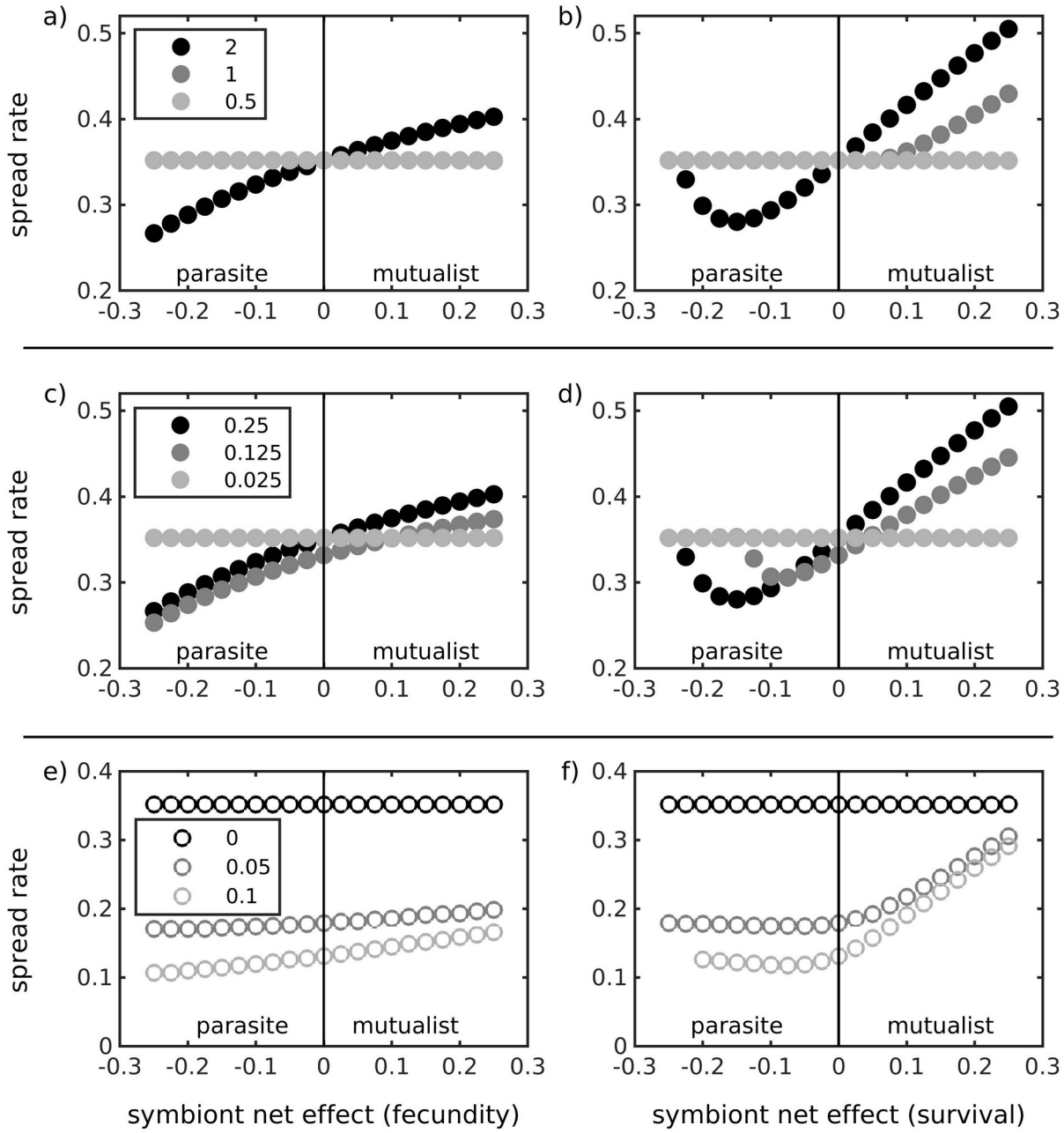

Figure 4. Choosing extreme model parameters values can 'break' the general patterns in Figure 3. Reducing (a-b) transmission rate (β) or (c-d) dispersal of partnered hosts (ν_P) can lead to hosts escaping the symbiont even with frequency-dependent transmission. Increasing (e-f) the Allee

404 threshold (a) can lead to hosts not escaping the symbiont even with density-dependent transmission. Host population spread rate as a function of the symbiont net effect (n) when the
406 symbiont affect host fecundity (left panels) and survival (right panels). Transmission is density-dependent (open circles) or frequency-dependent (closed circles), where results in black use the
408 same parameter value as Figure 3 and increasingly lighter grey show increasing changes. Parameters: $b = 1$, $\tau_1 = 0.5$, $\sigma_U = 0.7$, $\phi_U = 0.8$, $v_U = 0.25$, $t = 150$, $-0.25 \leq n \leq 0.25$ for all panels;
410 either have density-dependent (open circles) or frequency-dependent (closed circles), (a-b) $\beta = \{0, 5, 1, 2\}$, $a = 0$, $v_P = 0.25$, (c-d) $\beta = 2$, $a = 0$, $v_P = \{0.25, 0.125, 0.025\}$, and (e-f) $\beta = 2$, $a = \{0,$
412 $0.05, 0.1\}$, $v_P = 0.25$.

Table 1. Model parameters and variables, meaning and default values if applicable.

	Meaning	Default value
a	Allee threshold	0
b	Density-dependence parameter	1
g	Density-dependence function [eqn. 6]	–
k	Dispersal kernel function [eqn. 8]	–
n	Net effect of symbiont on host	varied
t	Time (year)	–
v_P	Variance of dispersal kernel for partnered hosts	0.25
v_U	Variance of dispersal kernel for unpartnered hosts	0.25
x	Space	–
y	Space	–
P	Density of partnered hosts	–
U	Density of unpartnered hosts	–
N	Overall population density (U+P)	–
β	Transmission rate	2
τ_1	Transmission period	0.5
τ_2	Time point post-survival	NA
τ_3	Time point post-fecundity	NA
σ_P	Survival of partnered hosts	varied
σ_U	Survival of unpartnered hosts	0.7
ϕ_P	Fecundity of partnered hosts	varied
ϕ_U	Fecundity of unpartnered hosts	0.8

414