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Research article

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

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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 known about how mutualistic interactions shape population spread, and what role outrunning mutualistic partners plays. Here, I examine host–symbiont interactions specifically; 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, integrodifference equation, invasion speed, mutualism, pathogen

Introduction

Species ranges are dynamic, and understanding why, how and when they change is critical to controlling the spread of invasive species (Sakai et al. 2001), facilitating species reintroductions (Ziółkowska et al. 2016) and understanding climate-induced range shifts and expansions (Weiss-Lehman and Shaw 2020). Yet, predicting which species (or populations) will spread and how fast remains a challenge (Kolar and Lodge 2001, Fournier et al. 2019), in part because spatial spread is governed by the processes acting at the low-density population edge (Lockwood et al. 2005), which may be different than the processes acting at high population density. For example, a population's rate of spread can be estimated by how fast individuals reproduce and disperse at low densities

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(first formulated by Skellam 1951 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; Taylor and Hastings 2005), and stochastic processes (Keller and Taylor 2008). Successful population spread is also shaped by species traits, interspecific interactions and the environmental context (Kolar and Lodge 2001, Shea and Chesson 2002, Svenning et al. 2014).

Population spread rate can be shaped by interspecific interactions. Biocontrol modeling studies (Owen and Lewis 2001, Fagan et al. 2002) have demonstrated 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 lupins; Owen and Lewis 2001). Similarly, generalized host–pathogen models show that pathogens can slow or reverse the spread of their host population (Hilker et al. 2005, Ducrot and Langlais 2008). 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 favor reduced dispersal, leading to slower rates of population spread compared to species that are spreading in isolation (Kubisch et al. 2014). 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; Torchin et al. 2002, 2003, Mitchell and Power 2003). The flip side of enemy release is that invasive species may also escape their mutualistic partners (Dickie et al. 2010); many invasive species, particularly plants, are constrained by a lack of mutualistic partners (e.g. mycorrhizal fungi, insect pollinators and vertebrate seed dispersers; Richardson et al. 2000, Pringle et al. 2009, Traveset and Richardson 2014). 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 (Drake 2003, Prenter et al. 2004) where a population effectively outruns enemies or mutualists, although this is not well studied (but see Phillips et al. 2010 for an example from lungworm parasites in cane toads). 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 (de Bary 1879). In host–symbiont interactions, 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 (Johnson et al. 1997), animals and gut microbes (Moran et al. 2019, Levin et al. 2021), coral and zooxanthellae (Day et al. 2008), bacteria and conjugative plasmids (Grohmann et al. 2003), plants and endophytes (Schardl et al. 2004), animals and chemosynthetic symbionts (Dubilier et al. 2008), cyanobacteria and their hosts (Usher et al. 2007). 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 (Johnson et al. 1997), with the magnitude and sign (positive, neutral, negative) of their interaction often depending on context (Chamberlain et al. 2014). For example, gut microbes in *Daphnia* can act as mutualists when resources are abundant and parasites when resources are rate (Rogalski et al. 2021). Thus, host–symbiont relationships provide 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 (Nelson and May 2017).

One challenge 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), or asymptotic transmission which falls in between these extremes (McCallum et al. 2001). Frequency-dependent transmission is best used to describe sexually transmitted or vector-borne pathogens; and density-dependent for most other pathogens (Keeling and Rohani 2008).

In contrast, for mutualistic symbionts, a key division is vertical (symbionts passed from parent to offspring) versus horizontal (symbionts acquired after birth) transmission (Ebert 2013). Although the terms density-dependent and frequency-dependent are rarely used to describe different cases of horizontal transmission of mutualists, mathematical functions capturing these differences have been used in symbiont models: frequency-dependent for fungal symbionts of plants (Nelson and May 2017) and density-dependent for zooxanthellae symbionts of corals (Day et al. 2008). 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; Selosse et al. 2006) or socially (e.g. baboon gut microbes; Tung et al. 2015), transmission will be best described by frequency-dependent, while transmission driven by unstructured contacts (e.g. corals; Day et al. 2008) will be best described by density-dependent. Taken together, this suggests that a single modeling framework could capture parasitic and mutualistic symbionts as ends of a spectrum (Nelson and May 2017).

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 (Antonovics 2009), 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 (McCallum et al. 2001), 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 (Bruns et al. 2019).

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 able 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 Fig. 1a for this annual cycle). I only consider a single host and a single symbiont species. See Shaw (2022) for model code and simulation output.

Symbiont transmission

Transmission of symbionts between hosts occurs locally at each point in space (x) and continuously during the first period of each year (Fig. 1a, thick arrow). I consider three modes of symbiont transmission: density-dependent, frequency-dependent and type II (also called asymptotic or saturating). The core distinctions among these modes are whether and how contact rates between hosts (and thus transmission rates) vary with host density (Begon et al. 2002, Fig. 1b). When contact rate and per capita transmission rate increase linearly with host density, this is density-dependent transmission (i.e. the per capita rate that an unpartnered host becomes partnered increases as partnered host density, P, increases; Fig. 1b) and has dynamics given by

$$\frac{dU}{dt} = -\beta UP \tag{1a}$$

$$\frac{dP}{dt} = \beta UP \tag{1b}$$

where β is the transmission rate between unpartnered (U) and partnered (P) hosts. Alternatively, when per capita transmission rate increases and then saturates with host density, this is a type II functional response, behaving like density-dependent transmission at low density and frequency-dependent transmission at high density, and has dynamics given by

Table 1. Model notation (parameters and functions), their meaning and the values considered.

	Meaning	Default value	Varied value(s)
a	Allee threshold	0	0, 0.05, 0.1 (Fig. 4)
b	Density-dependence parameter	1	–
g	Density-dependence function [Eq. 6]	–	–
h	Half-saturation constant (type II transmission)	–	0.05, 0.1, 0.2, 0.5, 1, 10
k	Dispersal kernel function [Eq. 8]	–	–
n	Net effect of symbiont on host	–	$-0.25 \leq n \leq 0.25$
t	Time (years)	150	1, 50, 100 (Fig. 2)
v_P	Variance of dispersal kernel for partnered hosts	0.25	0.025, 0.125, 0.25 (Fig. 4)
v_U	Variance of dispersal kernel for unpartnered hosts	0.25	–
β	Transmission rate	2	0.5, 1, 2 (Fig. 4)
τ_1	Transmission period	0.5	–
τ_2	Time point post-survival	–	–
τ_3	Time point post-fecundity	–	–
σ_P	Survival of partnered hosts	$\sigma_P=\sigma_U$ (when symbionts affect fecundity)	$\sigma_P=\sigma_U+n$ (when symbionts affect survival)
σ_U	Survival of unpartnered hosts	0.7	–
ϕ_P	Fecundity of partnered hosts	$\phi_P=\phi_U$ (when symbionts affect survival)	$\phi_P=\phi_U+n$ (when symbionts affect fecundity)
ϕ_U	Fecundity of unpartnered hosts	0.8	–

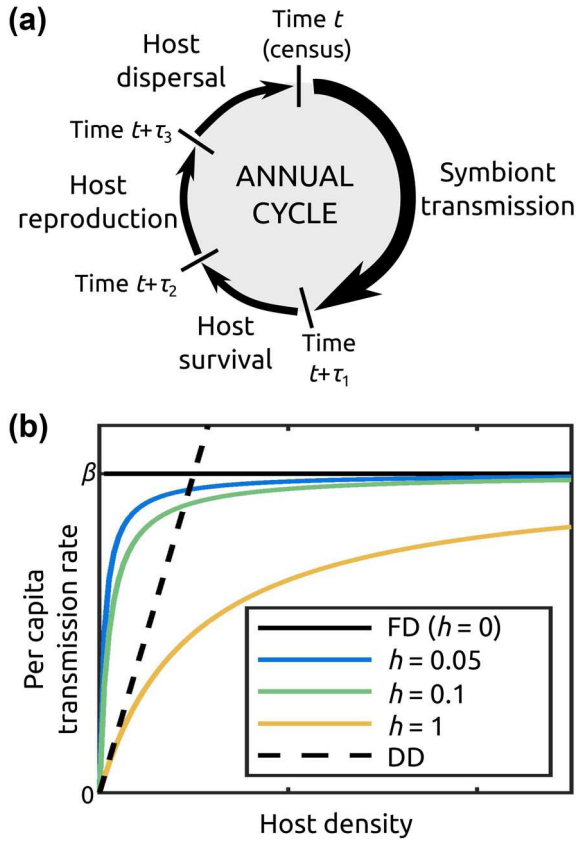

Figure 1. Schematic of the population's annual cycle and per capita transmission. (a) During a single year (starting with the census at time t, top of the circle), four processes occur sequentially. First, symbiont transmission occurs (thick arrow) for length of time τ_1 (from time t to $t + \tau_1$), 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 Eq. 5. Third, host reproduction occurs, and the population size at time $t + \tau_3$ is given by Eq. 7. Finally, host dispersal occurs at which point the population size is given by Eq. 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. (b) Per capita transmission rate as a function of host density for the different transmission types considered here. Transmission increases with density for density-dependent transmission. Transmission is constant with density for frequency-dependent transmission, and equal to β. Transmission increases and then asymptotes at β with density for type II transmission, and reaches half of the maximum transition rate when the host density is equal to the half-saturation constant (h).

$$\frac{dU}{dt} = -\beta U \frac{P}{h+U+P} \tag{2a}$$

$$\frac{dP}{dt} = \beta U \frac{P}{h+U+P} \tag{2b}$$

where h is the half-saturation constant. When per capita transmission is constant with host density, this is frequency-dependent transmission (i.e. the per capita rate that an unpartnered host becomes partnered increases as the frequency of partnered hosts within the population, $P/(U+P)$ increases; Fig. 1b), and can be captured by letting h go to 0 above. Transmission occurs continuously during a fraction τ_1 of the year ($0 \le \tau_1 \le 1$). Thus, at the end of the transmission period, the population size of unpartnered and partnered hosts can be found by integrating Eq. 1 and 2 and is given by

$$U_{t+\tau_1}(x) = \frac{U_t(x) N_t(x) e^{-\beta \tau_1 N_t(x)}}{P_t(x) + U_t(x) e^{-\beta \tau_1 N_t(x)}} \tag{3a}$$

$$P_{t+\tau_1}(x) = \frac{P_t(x) N_t(x)}{P_t(x) + U_t(x) e^{-\beta \tau_1 N_t(x)}} \tag{3b}$$

in the case of density-dependent transmission (from Eq. 1), where $N_t(x) = U_t(x) + P_t(x)$, and

$$U_{t+\tau_1}(x) = \frac{U_t(x) N_t(x)}{U_t(x) + P_t(x) e^{\frac{\beta \tau_1 N_t(x)}{h+N_t(x)}}} \tag{4a}$$

$$P_{t+\tau_1}(x) = \frac{P_t(x) N_t(x)}{P_t(x) + U_t(x) e^{\frac{-\beta \tau_1 N_t(x)}{h+N_t(x)}}} \tag{4b}$$

in the case of type II transmission (from Eq. 2).

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

$$U_{t+\tau_2}(x) = \sigma_U U_{t+\tau_1}(x) \tag{5a}$$

$$P_{t+\tau_2}(x) = \sigma_P P_{t+\tau_1}(x). \tag{5b}$$

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

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

with Allee threshold a, and density dependence parameter b. A population is not viable wherever its size falls below the Allee threshold (a), whereas the value of b is more arbitrary and sets the population carrying capacity. I chose to include an Allee effect because doing so can alter the dynamics of population spread across space (Stokes 1976, Kot et al. 1996, Taylor and Hastings 2005). 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 (Skellam 1951). 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 densities above the Allee threshold a, behind the population edge are pushed forward spilling past the population edge (Stokes 1976). 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 (Kot et al. 1996). This growth function (Eq. 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

$$U_{t+\tau_3}\left(x\right)=U_{t+\tau_2}\left(x\right)+\left[\phi_U U_{t+\tau_2}\left(x\right)+\phi_P P_{t+\tau_2}\left(x\right)\right]g_{t+\tau_2}\left(x\right) \quad (7a)$$

$$P_{t+\tau_3}\left(x\right)=P_{t+\tau_2}\left(x\right) \quad (7b)$$

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 (generations are overlapping).

Host dispersal

Finally, all hosts disperse, according to a dispersal kernel, $k(x–y)$, 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

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

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 (Venable et al. 2008, D'Aloia et al. 2015). 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

$$U_{t+1}\left(x\right)=\int_{-\infty}^{\infty}k\left(x-y;v_U\right)U_{t+\tau_3}\left(y\right)dy \quad (9a)$$

$$P_{t+1}\left(x\right)=\int_{-\infty}^{\infty}k\left(x-y;v_P\right)P_{t+\tau_3}\left(y\right)dy \quad (9b)$$

where the difference in the unpartnered and partnered hosts population density from one point ($t+\tau3$) 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 simulated the equations describing population size (Eq. 3–5, 7, 9). I initialized each simulation with a high density of unpartnered and partnered hosts in the center of space ($U_0(x)=6$; $P_0(x)=2$ for $|x|<0.1$; Fig. 2a, b). I iterated the model forward 150 times ($t=150$, long enough to move past any transient dynamics from initial conditions), recording the density of U and P hosts at each year (t) over space (x). To quantify how fast the population spread (i.e. the 'spread rate'), I first found the edge of the population (defined as the farthest point where the host density U_t+P_t exceeded a threshold of 0.001; beyond this edge the population was considered to be too low to be detectable) for each year t. Next, I calculated how far the population edge had moved from one year (t) to the next ($t+1$) and called this the annual spread rate. To quantify the impact of the symbiont on host spread, I also simulated a population with no symbionts (with initial conditions of $U_0(x)=8$; $P_0(x)=0$ for $|x|<0.1$). Then to calculate the net host population spread rate with symbionts, I took the spread rate with symbionts minus the spread rate without symbionts, reported as 'net host spread rate' below.

Scenarios

I explored the effect of transmission mode by simulating density-dependent transmission, frequency-dependent transmission (type II with $h=0$) and type II transmission with 6 other values of h ($h=0.05$, 0.1, 0.2, 0.5, 1, 10). I also explored cases where the symbiont had a positive or negative effect,

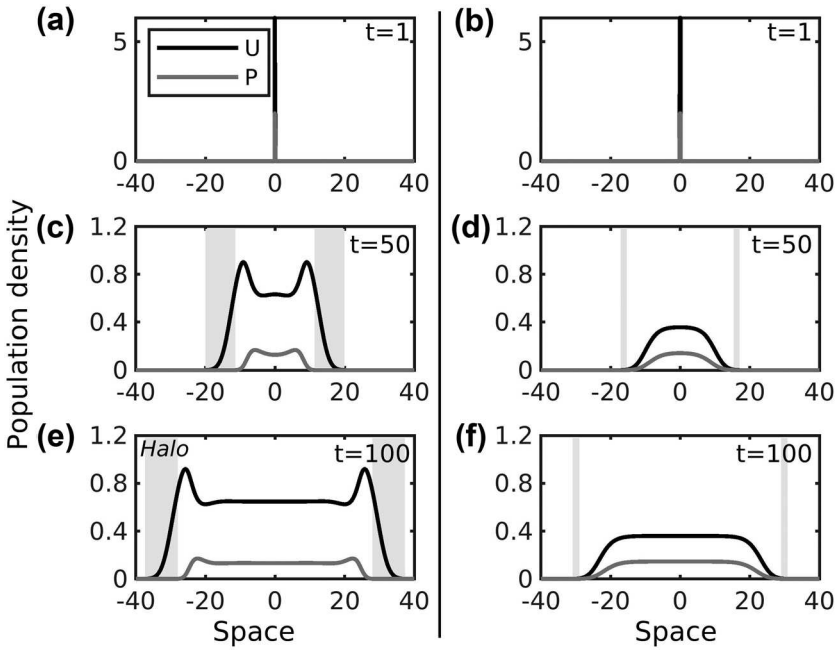

Figure 2. Hosts can either (a, c, e) outrun the symbiont spatially or (b, d, f) not (symbiont is present across the population). Each panel shows the host population density across space for unpartnered (black) and partnered (grey) hosts at a different time (t) during the simulation. Symbiont-free halos (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: default values from Table 1, $n=-0.15$ (parasite), symbiont affects survival. Transmission is (a, c, e) density-dependent and (b, d, f) frequency dependent ($h=0$).

and 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 $0 \leq \phi_P$).

As my aim was to develop a system-agnostic model, I chose a default set of parameter values generically rather than from particular a system, and then varied parameters around these values (Table 1). I considered a moderate life history pace for hosts with annual survival $\sigma=0.7$ and $\phi=0.8$ and moderate symbiont transmission with rate $\beta=2$ and occurring during $\tau_1=0.5$ of each annual cycle. The relative values for these four parameters determine what fraction of the host population is partnered, so I explored the effect of decreasing β (which should have the same effect as increasing σ and/or ϕ, or decreasing τ_1). I chose a default of no Allee effect ($a=0$) and explored the effect of adding an Allee effect. I set the default dispersal variance of all hosts as $v_P=v_U=0.25$; I expect that changing this would just change the spread rate quantitatively but not the qualitative pattern. However I did vary the difference in dispersal variance between partnered and unpartnered hosts (varying v_U, holding v_P constant).

Results

Within each simulation, the introduced host population initially increased in size, then spread out across space (Fig. 2). In some cases, the host population spatially outran the symbiont. This result occurred either when the symbiont was absent from the edge of the host population in a large 'symbiont-free halo' (per Bruns et al. 2019), i.e. a region of the population with only unpartnered hosts (Fig. 2a, c, e, pale grey regions), or when the symbiont was completely absent from the host population (Supporting information). In other cases, the host population did not outrun its symbiont and the symbiont was present close to the population edge (Fig. 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, either faster or slower (Fig. 3, Supporting information).

The mode of symbiont transmission was critical in determining whether hosts escaped their symbionts. With density-dependent transmission, the host population outran the symbiont and thus its spread rate was independent of the symbiont effect on the host (Fig. 3, open circles). However, with frequency-dependent transmission, the host did not outrun the symbiont and the net effect of the symbiont on the host affected the population spread rate (Fig. 3, black closed circles). With type II transmission and a small half-saturation constant (h), hosts could not outrun the symbiont and the magnitude of the symbiont's effect of population

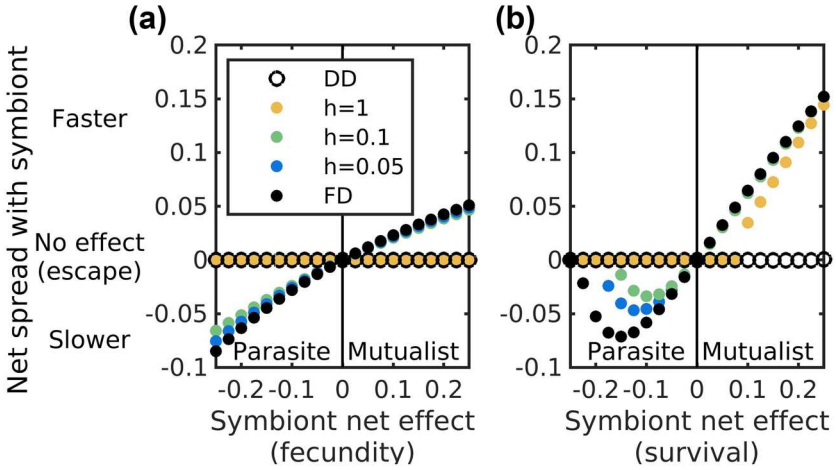

Figure 3. Hosts outrun the symbiont when transmission is density-dependent (DD; host spread is independent of the symbiont net effect, x-axis) but do not when transmission is frequency-dependent (FD; host spread shaped by symbiont). Net host population spread rate (rate of spread with symbiont minus rate of spread without symbiont) as a function of the symbiont net effect (n) on the host for density-dependent (open black circles), type II transmission (closed colored circles) frequency-dependent ($h=0$; closed black circles) when the symbiont affects 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: default values from Table 1, $-0.25 \leq n \leq 0.25$, $h=\{0.05, 0.1, 1\}$.

spread depended on the value of h (Fig. 3, closed colored circles). However, when h was large, the symbiont went extinct (Supporting information) and did not affect the spread rate (Fig. 3, closed colored 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 (Fig. 3, Supporting information).

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 (Fig. 4a and b). For low enough transmission rate values (β), hosts were able to outrun the symbiont even with frequency-dependent transmission (Fig. 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 (Fig. 4c, d), and in the extreme, the host population was able to outrun the symbiont (Fig. 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 (Fig. 4e, f). Each of these shifts occurred sharply as the corresponding parameter (β, v_P, a) changed (Supporting information).

The currency of the symbiont's effect (whether it affected host fecundity or survival) influenced the outcome. Symbionts that affect host survival shape symbiont persistence: symbionts that kill their hosts remove themselves whereas symbionts that boost host survival increase their own longevity. In contrast, symbionts that affect host fecundity do not have this effect. As a result, parasitic symbionts that greatly reduced host survival (i.e. high virulence) quickly killed off partnered hosts, enabling the host population to effectively escape the symbiont, creating a U-shaped pattern (Fig. 3b), whereas parasitic symbionts that greatly reduced host fecundity allowed partnered hosts to persist, preventing the host population from escaping the symbiont, creating a linear pattern (Fig. 3a). Similarly, mutualistic symbionts that boosted host survival were harder to escape than those that boosted host fecundity (Fig. 3, yellow circles; $h=1$). Symbiont currency also shapes the threshold effects described above. For symbionts that affect host fecundity, the threshold between when symbionts shape host spread rate versus not is independent of symbiont net effect (Supporting information). In contrast for symbionts that affect host survival, the threshold varies with symbiont net effect (n); since this value shapes symbiont persistence in the host population.

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. This intuitive result stems from the idea that positive impacts on host growth (mutualists) will lead to faster population spread while negative impacts (parasites) will slow population spread. An exception occurred for parasitic symbionts that kill their hosts (instead of reduce host fecundity); more virulent parasites killed their hosts, inadvertently driving the symbiont extinct, and thus did not affect host population spread rate. The mode of symbiont transmission between hosts determines whether symbionts affect host spread at all: with density-dependent (instead of

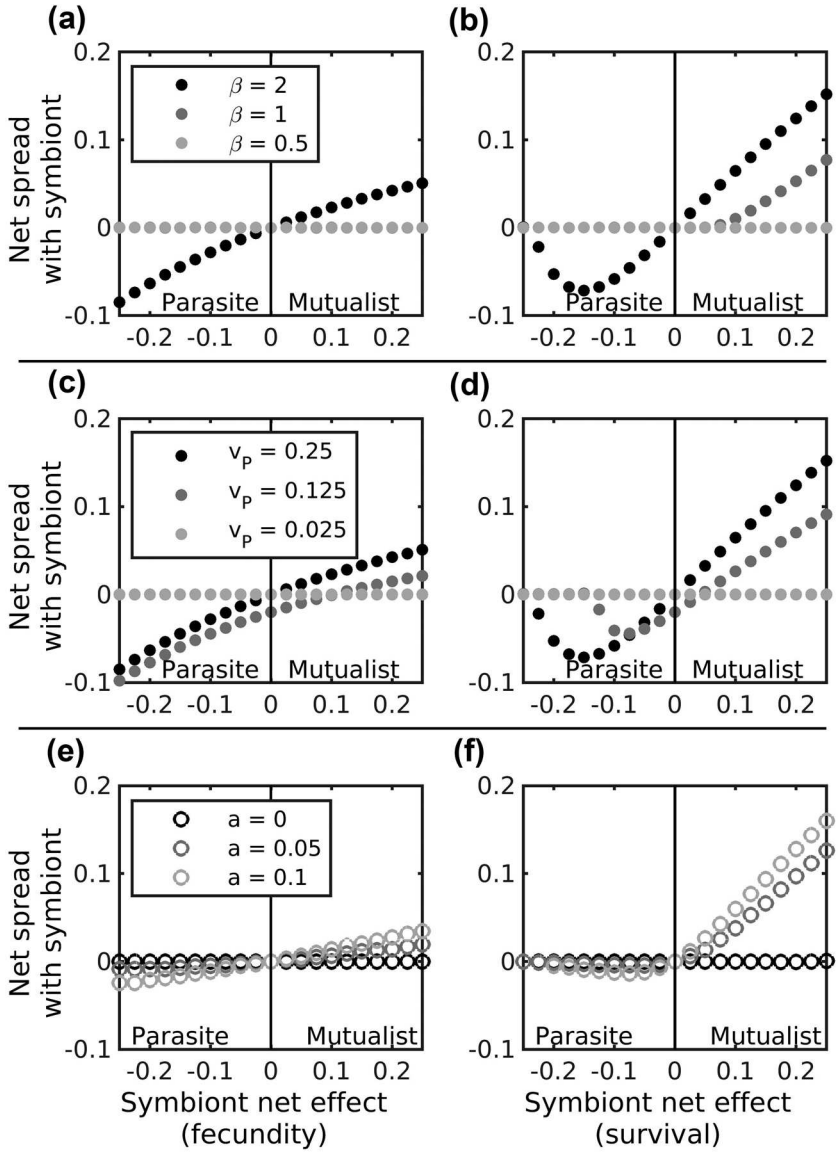

Figure 4. Choosing extreme model parameters values can break the general patterns in Fig. 3. Reducing (a, b) transmission rate ($\beta = \{0.5, 1, 2\}$) or (c, d) dispersal of partnered hosts ($v_p = \{0.25, 0.125, 0.025\}$) can lead to hosts escaping the symbiont even with frequency-dependent transmission. Increasing (e, f) the Allee threshold ($a = \{0, 0.05, 0.1\}$) can lead to hosts not escaping the symbiont even with density-dependent transmission. Net host population spread rate as a function of the symbiont net effect (n) when the 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 same parameter value as Fig. 3 and increasingly lighter grey show increasing changes. Parameters: default values from Table 1 unless specified otherwise, $-0.25 \leq n \leq 0.25$; density-dependent (open circles) or frequency-dependent (closed circles) transmission.

frequency-dependent or saturating) 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 our understanding of how mutualisms shape population spread (Svenning et al. 2014). Second, I show that hosts can indeed outrun enemies (here, parasitic symbionts or pathogens) during the spread phase of an invasion, confirming a previously suggested idea (Drake 2003). Furthermore, although past theory on pathogens shaping host spread has modeled frequency-dependent (Hilker et al. 2005, Ducrot and Langlais 2008), or density-dependent (Phillips et al. 2010) transmission, to my knowledge, the contrast between these transmission modes or the exploration of type II transmission has not been made explicitly before in this context.

My results build on past theory. First, they contrast with a finding that obligate mutualisms can slow down invasions, when mutualism favors the evolution of reduced dispersal (Kubisch et al. 2014). This discrepancy can be resolved by realizing the model presented here describes a facultative relationship for the host (but symbionts cannot exist outside their hosts; their dispersal is tied to host dispersal) and my model does not include evolution (and thus lacks the same eco-evolutionary feedback). Furthermore, whether evolution favors increased or reduced dispersal in the presence of a mutualist will depend on the relative benefit of keeping pace with a mutualistic partner versus escaping intraspecific competition. Second, prior theory has described a stochastic mechanism by which an organism can escape its pathogens during spread: if spread occurs via a series of low-density founder events, pathogens will be lost through chance events (Phillips et al. 2010). The results here provide a deterministic mechanism by which organisms escape parasites. Both mechanisms may act jointly in empirical systems; an interaction that could be explore in future work. Third, previous theory has quantified how hosts could escape parasitic symbionts during the introduction phase of invasion (Drake 2003); here I show that even if hosts do not escape symbionts during this first phase, they may be able to escape them during the subsequent phase of spread.

The work presented here also unites ideas from related biological fields. First, I show that the large 'symbiont-free halos' (per Bruns et al. 2019) found at the edge of stationary ranges should be expected at the edge of spreading ranges (and have been found in cane toads with lungworms; Brown et al. 2016), for symbionts with density-dependent but not frequency-dependent transmission. Thus, pathogens and symbionts with frequency-dependent transmission 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 breaks down in the presence of an Allee effect, enabling symbionts with density-dependent transmission to shape host population spread rate. In the presence of an Allee effect, a 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 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 determined by the high density core population) (Stokes 1976, Kot et al. 1996). 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 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 population has an Allee effect.

My model could be expanded in a number of future directions. First, one could explore additional transmission modes. 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 (Ebert 2013). I expect that symbionts with vertical transmission will shape their host population spread rates, since they will be present across the host population range. One could also 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 (Chamberlain et al. 2014), 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 (Svenning et al. 2014). 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 (Daversa et al. 2017) and moving is one of the ways unpartnered organisms can locate mutualistic partners (Shaw et al. 2021). Fifth, one could explore the role of evolution (and eco-evolutionary feedback loops) during spread, as mentioned above.

Finally, despite the population-level focus of this work, my findings have intriguing implications in terms of individual-level behavior. In particular, the mode of symbiont transmission is driven by individual contact behaviors 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 (Begon et al. 2002). This mode includes species where individuals are sedentary or territorial and symbionts are passed to nearest neighbors (as for plant mycorrhizal networks; Selosse et al. 2006), or where individuals pass symbionts along social networks (Tung et al. 2015). In contrast, density-dependent transmission describes scenarios where contacts between individuals increase with density. This mode applies to corals (Day et al. 2008) as well as many species with microbial symbionts (Shapiro and Turner 2014). Thus, individual-level contact behaviors 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 behavior 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 availability statement

Data are available from the Zenodo Digital Repository: <https://doi.org/10.5281/zenodo.6914074> (Shaw 2022).

Supporting information

The Supporting information associated with this article is available with the online version.

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