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Parasite prevalence is determined by infection state- and risk-dependent dispersal of the host

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The spread of parasites and the emergence of disease are currently threatening global biodiversity and human welfare. To address this threat, we need to better understand those factors that determine parasite persistence and prevalence. It is known that dispersal is central to the spatial dynamics of host–parasite systems. Yet past studies have typically assumed that dispersal is a species-level constant, despite a growing body of empirical evidence that dispersal varies with ecological context, including the risk of infection and aspects of host state such as infection status (parasite-dependent dispersal; PDD). Here, we develop a metapopulation model to understand how different forms of PDD shape the prevalence of a directly transmitted parasite. We show that increasing host dispersal rate can increase, decrease or cause a non-monotonic change in regional parasite prevalence, depending on the type of PDD and characteristics of the host–parasite system (transmission rate, virulence, and dispersal mortality). This result contrasts with previous studies with parasite-independent dispersal which concluded that prevalence increases with host dispersal rate. We argue that accounting for host dispersal responses to parasites is necessary for a complete understanding of host–parasite dynamics and for predicting how parasite prevalence will respond to changes such as human alteration of landscape connectivity.

This article is part of the theme issue 'Diversity-dependence of dispersal: interspecific interactions determine spatial dynamics'.

1. Introduction

The increasing incidence of parasites affecting human, livestock and wildlife populations has created an urgent need to understand the factors that determine parasite persistence and prevalence, with the goal of mitigating the impact on human welfare and biodiversity [1,2]. Groups including the United Nations Environment Programme have listed the impacts of emerging parasites as an issue of global concern [3]. Many of these pressing questions and broad challenges in the study of host–parasite dynamics are inherently spatial: the movement of hosts and parasites drives transmission and can lead to the invasion of parasites into new ranges [4]. In recognition of the importance of space to host–parasite dynamics, much of the relevant ecological and epidemiological research has been set within a metacommunity framework [5], which explicitly accounts for the role of dispersal in linking parasites and host populations.

Although past metacommunity studies have provided valuable insights, most have taken an over-simplified view of the dispersal process, where dispersal is assumed to be a species-level characteristic, invariable among individuals and sites. It is now clear that this assumption is unrealistic; behavioural and physiological ecologists have demonstrated that host

dispersal varies with local ecological conditions, including parasitism risk, and with the impact of parasitism on the morphological, physiological and behavioural traits that mediate host movement [6–8]. We broadly group the results of these parasite-mediated factors as 'parasite-dependent dispersal' (PDD). The consequences of PDD for the ecological dynamics of host–parasite systems (e.g. parasite and host extinction rate, regional parasite prevalence) have rarely been considered and the full effects are still unknown. Yet, PDD has the potential to alter our understanding of the role of dispersal in host–parasite interactions.

Parasites influence host dispersal through a variety of mechanisms. Hosts have been shown to alter their dispersal behaviour in response to their perceived risk of gaining an infection (context-dependent dispersal), or to their own infection status (state-dependent dispersal). For each of these cases, dependency may be positive (increase with risk/infection) or negative (decrease with risk/infection). Positive context dependency is expected to occur when the costs of parasitism to the self or to offspring are greater than the costs of dispersal [9] and there is positive temporal autocorrelation in parasitism risk experienced locally in a patch [10,11]. This type of context dependency has been observed in empirical studies and can be manifested as either increased propensity to emigrate from habitat patches with parasites [8,12] or increased investment in dispersal traits for the organism or their offspring [13]. Negative context dependence is hypothesized to occur under several scenarios including when hosts exposed to parasites invest in defense traits, thereby reducing the amount of energy available for dispersal [14], when there is negative temporal autocorrelation in parasitism risk [11] or when parasites are more abundant in high-quality patches (i.e. patches with abiotic conditions that are favourable to both the host and the parasite and/or that confer high host population growth leading to high parasite abundance). Empirical studies of PDD are still rare, however, and we could only find one empirical example of negative context-dependent dispersal in which context was not conflated with infection state [15].

There are also many examples of dispersal that is dependent on host infection status. Infected hosts often disperse more than uninfected hosts (*I*-biased dispersal; [16,17]). This is hypothesized to occur for multiple non-exclusive reasons including when hosts use infection as a cue that their natal patch is risky and disperse to new patches to reduce risk for themselves and/or offspring [18], infected hosts disperse to avoid infecting kin [11,15,19] or reduce competition for kin [19], or when dispersal increases parasite clearance rates [19–21]. *I*-biased dispersal may also occur if the parasite manipulates infected hosts to increase dispersal to promote the spread of the parasite to unexploited host populations [22,23]. Alternatively, in some systems, uninfected (hereafter, 'susceptible') hosts disperse more than infected hosts (*S*-biased dispersal) when infected hosts have lower dispersal ability [15,24]. There are several mechanisms through which infection can reduce host dispersal ability: parasites may consume or damage tissues involved in movement [25,26], interfere with oxygen exchange [24,27] or morphological development [28], and deplete energy reserves needed for movement [14]. *S*-biased dispersal may also occur if the parasite manipulates infected hosts into reducing dispersal, for example, in order to divert resources away from dispersal to make them available to the parasite [23,29,30]. Host dispersal behaviour in natural systems can be simultaneously context dependent and state dependent. For example, the backswimmer *Notonecta undulata* increases emigration when it perceives cues of ectoparasitic Hydrachnidia mites (positive context dependency), and backswimmers infected with mites have reduced dispersal ability because mites damage their wings (*S*-biased dispersal; [8]).

The prevailing prediction in the theoretical literature holds that regional parasite prevalence (the proportion of a host metapopulation infected by the parasite) increases with increasing rates of dispersal of the parasite, or of the host carrying the parasite, because movement can introduce the parasite to unexploited groups of susceptible hosts in spatially structured populations [31,32]. A small number of empirical studies have supported this prediction [33]. However, few studies have considered that host dispersal behaviour may be altered by exposure to, or infection with, parasites. We hypothesize that compared to host dispersal that is independent of parasites, PDD will influence the rate at which parasites encounter susceptible hosts and thereby alter the outcome of host–parasite interactions at a regional scale (i.e. regional parasite prevalence). Moreover, if different host–parasite systems exhibit different forms of PDD (e.g. positive context dependency versus negative context dependency), this will introduce variation across systems in the relationship between dispersal and regional parasite prevalence. The effects of PDD may also depend on other characteristics of the system including local transmission rate, virulence, the risk of dispersal mortality and whether dispersal mortality depends on infection state. For example, virulent parasites that kill their hosts before they can disperse will have lower prevalence than those that do not immediately reduce host vigour. Similarly, when infected individuals experience high dispersal mortality due to impeded mobility or low energy reserves, their parasites should have lower regional prevalence.

Here, we develop a model to understand how varied forms of PDD shape parasite prevalence. We compare both context- and state-dependent dispersal alone and in combination. We also investigate how biological characteristics of the host–parasite system (dispersal mortality, transmission rate and virulence) influence the relationship between host dispersal and parasite prevalence. Ours is one of the first studies to consider the consequences of PDD and is the most comprehensive model, to date, of the effects of PDD on host–parasite dynamics. We find that incorporating PDD results in a complex picture of the relationship between dispersal and parasite prevalence. Our model demonstrates that this relationship can vary widely in strength and even direction: prevalence can be an increasing, decreasing, flat or non-monotonic function of host dispersal rate, depending on the form of PDD and biological characteristics of the host–parasite system.

2. Methods

We modelled a simple host–parasite system in which the parasite is directly transmitted. Host population growth is logistic; host populations can be controlled by the parasite (the parasite reduces the survival, but not the reproduction, of the host) as well as self-regulate (hosts do not produce offspring once a population reaches its carrying capacity). We built a spatially

implicit *SI* metapopulation model with P patches, inhabited by susceptible (i.e. non-infected; S) and infected (I) individuals (see table 1 for all model parameters). The model is equation-based; we track the number of susceptible and infected individuals present in each patch x (x = 1, …, P) and year t as $S_x(t)$ and $I_x(t)$. Each year, the processes of transmission, dispersal (with possible mortality), survival and reproduction occur sequentially, and generations are overlapping (figure 1). Note in particular, transmission must occur before dispersal since the only mechanism by which parasites spread among patches in our model is host dispersal. All processes are stochastic; for each one, we generate binomial random variables based on the number of individuals and probabilities of each process.

(a) Transmission

Transmission of parasites occurs locally within a patch. Susceptible individuals can become infected by direct contact with infected individuals with transmission parameter β. After transmission in year t, the number of susceptible and infected individuals in each patch x is given by:

$$S_x(t)' = S_x(t) - \mathrm{Bin}\left(S_x(t),\ 1 - e^{-\beta I_x(t)}\right), \tag{2.1}$$

$$I_x(t)' = I_x(t) + \mathrm{Bin}\left(S_x(t),\ 1 - e^{-\beta I_x(t)}\right), \tag{2.2}$$

where Bin() indicates a binomial random variable, and $1 - e^{-\beta I_x(t)}$ is the probability that each susceptible individual becomes infected [34]. Note that with this discrete-time transmission, all transmission events come from individuals who were infected at the start of the transmission period ($I_x(t)$), and none from any new infections that occur during the transmission period. Infection probability for a susceptible individual therefore increases with the number of infected individuals in their patch. Infected individuals never recover.

(b) Dispersal

Dispersal occurs once per year and is global (dispersing individuals from patch x settle in any patch in the world that is not x at random). Any living individual can disperse every year. Dispersal strategies can take a number of different forms; in particular, we focus on PDD where dispersal depends on an individual's state (whether it is susceptible or infected) and/or an individual's context (the infection prevalence in its patch, defined as the proportion of the local host population infected with the parasite). Dispersal can come with added mortality that can depend on state (dispersing susceptible and infected individuals die with probability μ_{ds} and μ_{di}, respectively). After dispersal in year t, the number of susceptible and infected individuals in each patch x is given by:

$$S_x(t)'' = \mathrm{Bin}(S_x(t)', 1 - d) + M_{sx}(t), \tag{2.3}$$

$$I_x(t)'' = \mathrm{Bin}(I_x(t)', 1 - d) + M_{ix}(t), \tag{2.4}$$

where the first term of each equation describes the number of individuals (a binomial random variable) that do not disperse (stay in their patch), and the second term describes the number of individuals that arrive from other patches. Susceptible and infected immigrants into patch x in year t come from the pool of immigrants with size:

$$M_{sx}(t) = \sum_{j \neq x} \mathrm{Bin}\left(S_j(t)', d\right)(1 - \mu_{ds}), \tag{2.5}$$

$$M_{ix}(t) = \sum_{j \neq x} \mathrm{Bin}\left(I_j(t)', d\right)(1 - \mu_{di}). \tag{2.6}$$

These equations sum up the number of individuals that disperse and survive dispersal across each other starting patch j. Individuals in the immigrant pool land in a new patch with uniform probability.

For the dispersal probability d, we modelled multiple PDD scenarios, described below.

(i) Null

Dispersal is the same for all individuals (no PDD); each individual disperses with probability d_{lo}, regardless of their state or context.

(ii) State dependent

Dispersal depends on an individual's infection state (figure 2). If dispersal increases with infection (I-biased dispersal), S individuals disperse with probability d_{lo} and I individuals disperse with probability $d_{\mathrm{lo}} + \delta$, where δ is the state-dependent dispersal increase parameter. In contrast, if dispersal decreases with infection (S-biased dispersal), S individuals disperse with probability $d_{\mathrm{lo}} + \delta$ and I individuals disperse with probability d_{lo}.

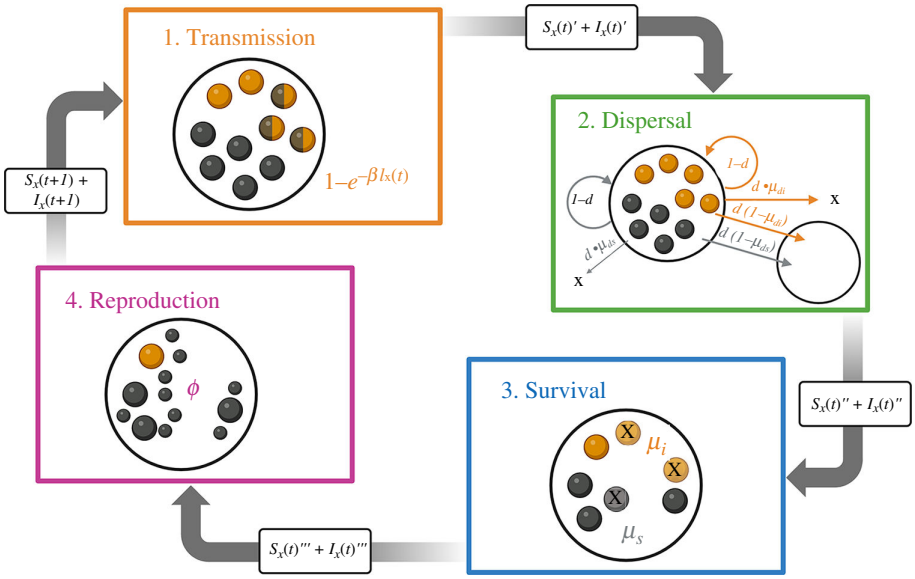

Figure 1. Life cycle of the host. The life cycle begins with $S_x(t)$ susceptible (represented as grey spheres) and $I_x(t)$ infected (represented as orange spheres) individuals in each patch x in year t. The number of susceptible and infected individuals is tracked through each stage of the life cycle. 1. Transmission occurs locally in the natal patch. Susceptible individuals are infected with probability $1 - e[-\beta I_x(t)]$. After transmission, there are $S_x(t)$` and $I_x(t)$` individuals in patch x. In this example, three susceptible individuals become newly infected. 2. Individuals disperse with probability d. Depending on the form of PDD, d is a function of infection state, local parasite prevalence, both or neither (see §2 for detailed equations). Dispersers survive and colonize a new patch with probability $1 - \mu_{ds}$ if they are susceptible, and with probability $1 - \mu_{di}$ if they are infected. The 'X' symbol represents mortality. Dispersal is global; individuals immigrate into new patches at random. The sum of emigrants and immigrants results in $S_x(t)$`` and $I_x(t)$`` individuals in patch x. 3. Susceptible individuals die with probability μ_s and infected individuals die with probability μ_i , resulting in $S_x(t)$``` and $I_x(t)$``` individuals in patch x. 4. All susceptible and infected individuals produce a Poisson random number of susceptible offspring with mean ☒ until the patch capacity is met, resulting in $S_x(t+1)$ and $I_x(t+1)$ individuals in patch x in the year $t+1$. The life cycle then repeats.

Table 1. Model parameters, meaning and default values.

parameter	meaning	values
P	number of patches	50
K	patch-level carrying capacity (for reproduction)	20
β	transmission parameter	0.3 (high transmission) or 0.1 (low transmission)
d_{lo}	lower bound probability of dispersing	0.05
d_{hi}	higher bound probability of dispersing	0.5
δ	dispersal increase (state dependent)	0.2
τ	dispersal midpoint (context dependent)	varied ($\tau = 0.05, 0.1, \ldots, 0.9, 0.95$)
λ	dispersal slope (context dependent)	10
μ_s	mortality probability for susceptible individuals (annual)	0.5
μ_i	mortality probability for infected individuals (annual)	$\mu_s + 0.02$
μ_{ds}	mortality probability for dispersing susceptible individuals (annual)	0 or 0.1
μ_{di}	mortality probability for dispersing infected individuals (annual)	0 or 0.1 or 0.2 or 0.3
ϕ	maximum per capita fecundity (annual)	2

(iii) Context dependent

Dispersal probability is determined by a logistic function of prevalence (p) in an individual's patch (figure 2). We chose a logistic function because models of the evolution of host dispersal in response to parasite prevalence have predicted non-monotonic relationships, with suggestions of a 'threshold' prevalence at which hosts switch from being non-dispersive to dispersive (or vice versa) [11,19]. This is part of a more general trend of evolutionary models predicting such threshold responses (e.g. population density and body condition; [35,36]). Research on the evolution of density-dependent dispersal suggests that the shape, including the steepness, of these threshold responses depends on factors including dispersal mortality, the intensity of environmental fluctuations and the accuracy of information that individuals have about the environment [36–38]. Of course, these factors will vary across host–parasite systems since they inhabit a wide variety of environments with different amounts of environmental variation and risks for dispersers. Moreover, parasites vary in the effects that they have on host dispersal

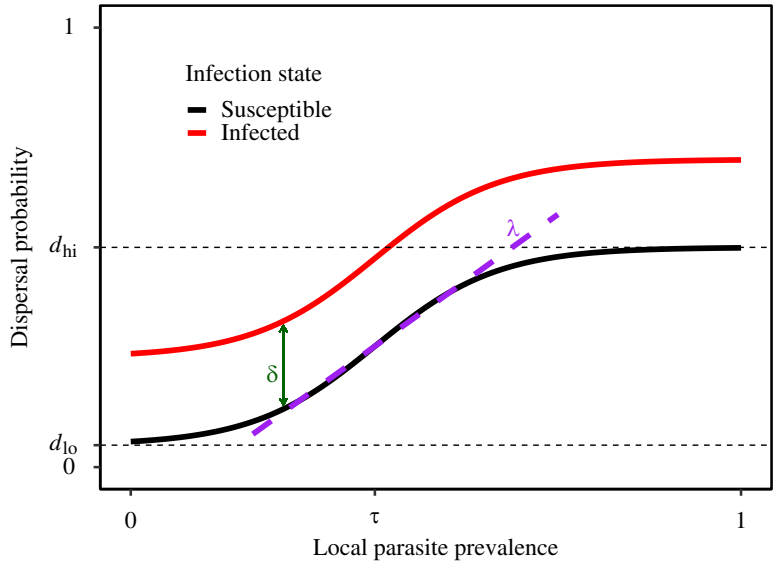

Figure 2. Model schematic showing state- and context-dependent PDD parameters. One form of PDD is represented here: positive context-dependent (dispersal probability increases with increasing local parasite prevalence) and *I*-biased dispersal (infected individuals are more likely to disperse than susceptible individuals). In our model, we crossed all possible forms of state-dependent (unbiased, *I*-biased and *S*-biased) and context-dependent (independent, positive and negative) dispersal.

mortality [6]. The level and accuracy of the information that hosts have about parasite prevalence will also vary across systems, as parasites vary greatly in conspicuousness [39] and hosts vary in the extent to which they can detect parasites [40]. The logistic function is flexible enough to model the variability in the shape of context-dependent dispersal that likely occurs in real host–parasite systems; it can take forms from roughly linear to stepped.

If dispersal increases with infection risk (i.e. prevalence; positive context dependence), individuals disperse with probability:

$$d = d_{lo} + \frac{d_{hi} - d_{lo}}{1 + e^{\lambda(\tau - p)}}, \tag{2.7}$$

where d_{lo} is the lower bound on the probability of dispersal, d_{hi} is the upper bound on the probability of dispersal, τ is the context-dependent dispersal midpoint parameter (the prevalence value where dispersal has increased by half between d_{lo} and d_{hi}) and λ is the context-dependent dispersal slope (the slope of the dispersal function at τ). Here, individuals in low-prevalence patches tend to disperse less than those in high-prevalence patches.

In contrast, if dispersal decreases with infection risk (negative context dependence), individuals disperse with probability:

$$d = d_{hi} + \frac{d_{lo} - d_{hi}}{1 + e^{\lambda(\tau - p)}}. \tag{2.8}$$

Here, individuals in low-prevalence patches tend to disperse more than those in high-prevalence patches. We focus on positive context dependence because it is apparently more common in empirical systems but we also explore negative context dependence for completeness and because there is at least some evidence that it occurs in empirical systems.

(iv) State dependent and context dependent

Finally, dispersal can be both state dependent and context dependent with additive effects, where changing state would change an individual's dispersal probability by δ and changing context would change an individual's dispersal probability between d_{lo} and d_{hi} (figure 2; equations (2.7) and (2.8)). For example, with *I*-biased and positive context-dependent dispersal, *S* individuals disperse with probability given by equation(2.7) above and *I* individuals disperse with probability:

$$d = \delta + d_{lo} + \frac{d_{hi} - d_{lo}}{1 + e^{\lambda(\tau - p)}}. \tag{2.9}$$

In addition, we consider three other possible additive combinations: *I*-biased and negative context-dependent dispersal, *S*-biased and positive context-dependent dispersal, and *S*-biased and negative context-dependent dispersal. Note that we do not consider cases where susceptible and infected individuals have different shapes or signs of context dependency (e.g. *S*-biased, positive context dependence/*I*-biased negative context dependence).

(c) Survival

Following dispersal, individuals survive or die. After survival in year t, the number of susceptible and infected individuals in each patch x is given by:

$$S_x(t)''' = S_x(t)'' - \mathrm{Bin}(S_x(t)'', \mu_s), \tag{2.10}$$

$$I_x(t)''' = I_x(t)'' - \mathrm{Bin}(I_x(t)'', \mu_i), \tag{2.11}$$

where μ_s is the probability that each susceptible individual dies and μ_i is the probability that each infected individual dies.

(d) Reproduction

Reproduction is density dependent, with a capacity of K individuals in each patch. If there are already K or more surviving individuals present, reproduction does not occur; we describe the available capacity in each patch as:

$$k = K - S_x(t)''' - I_x(t)''' . \tag{2.12}$$

After reproduction, the life cycle resets. At the start of the following year, $t+1$, the number of susceptible individuals in each patch x is given by:

$$S_x(t+1) = S_x(t)''' + \min[k, \mathrm{Pois}(\phi)(S_x(t)''' + I_x(t)''')], \tag{2.13}$$

where Pois() indicates a Poisson random variable, the first term represents the existing susceptible individuals and the second term is the newborn offspring (all offspring are born susceptible). For the second term, each of the surviving $S_x(t)''' + I_x(t)'''$ individuals can produce a Poisson random number of offspring with mean ϕ, within the remaining k patch capacity. The number of infected individuals remains unchanged by reproduction, so

$$I_x(t+1) = I_x(t)''' . \tag{2.14}$$

(e) Simulations

We initialized each simulation with K susceptible individuals in each of the P patches. We then introduced infection into one individual in the metapopulation at random. As each simulation progressed (by iterating the model forward via equations 2.1–2.14), we tracked the number of S and I individuals in each year (figure 1) and calculated the regional parasite prevalence (overall proportion of individuals in the metapopulation that were infected). Each simulation was run until either the parasite disappeared from the population, or until the regional parasite prevalence was no longer changing systematically. Specifically, we calculated the average regional parasite prevalence over the past 10 years and compared it to the average regional prevalence for the 10 years prior to that. If these two values differed by less than 0.02, we said that equilibrium had been reached. In some cases, a dynamic equilibrium was reached, with regular oscillations in population size and parasite prevalence. At the end of each simulation, we quantified the regional prevalence (averaged over the last 10 years). We also quantified the metapopulation dispersal fraction (averaged over the last 10 years of the simulation) as the number of individuals departing for dispersal divided by the number of individuals in the population at that time (i.e. censusing dispersal at departure and not settlement). For the parameter values we chose, the host metapopulation never went extinct. We ran 1000 replicate simulations for each set of parameter values (see below). Finally, we calculated the average and standard deviation of prevalence across all the replicates where the parasite persisted, for each set of parameter values.

(f) Simulation scenarios

We ran four sets of simulations. First, we ran null simulations where dispersal was not state or context dependent, where all individuals dispersed with probability d_{lo}. We ran simulations for 20 values of d_{lo} between 0.05 and 1. Second, we ran simulations where dispersal was only state dependent and either S-biased or I-biased. For each of these two cases, we ran simulations for 19 values of δ (the state-dependent dispersal increase parameter) between 0.05 and $1 - d_{\mathrm{lo}}$. Third, we ran simulations where dispersal was context dependent with and without being state dependent: positive and negative context-dependent dispersal, crossed with state-independent, S-biased or I-biased dispersal. For each of these six cases, we ran simulations for 19 values of τ (the context-dependent dispersal midpoint parameter) between 0.05 and 0.95. Fourth, we ran a set of sensitivity simulations for context-dependent dispersal where we varied λ (5 and 20 instead of 10), d_{lo} (0.025 and 0.1 instead of 0.05) and d_{hi} (0.25 and 0.8 instead of 0.5).

Across these sets, we considered four different host–parasite characteristics: high transmission ($\beta = 0.3$) and high virulence ($\mu_i = \mu_s + 0.2$), low transmission ($\beta = 0.1$) and low virulence ($\mu_i = \mu_s + 0.02$), high transmission and low virulence, and low transmission and high virulence. We also considered four different dispersal cost scenarios: no dispersal mortality ($\mu_{\mathrm{ds}} = \mu_{\mathrm{di}} = 0$), fixed dispersal mortality ($\mu_{\mathrm{ds}} = \mu_{\mathrm{di}} = 0.1$), low state-dependent dispersal mortality ($\mu_{\mathrm{ds}} = 0.1$ $\mu_{\mathrm{di}} = 0.2$) and high state-dependent dispersal mortality ($\mu_{\mathrm{ds}} = 0.1$ $\mu_{\mathrm{di}} = 0.3$). We ran 1000 replicate simulations for each of the above sets of parameter values.

3. Results

In the results we show below, the dispersal parameters being varied are d_{lo} (unconditional dispersal simulations), δ (state-dependent dispersal simulations), τ (context-dependent dispersal simulations), or δ and τ (state- and context-dependent dispersal simulations). Varying these parameters resulted in variation in the fraction of the host metapopulation that disperses.

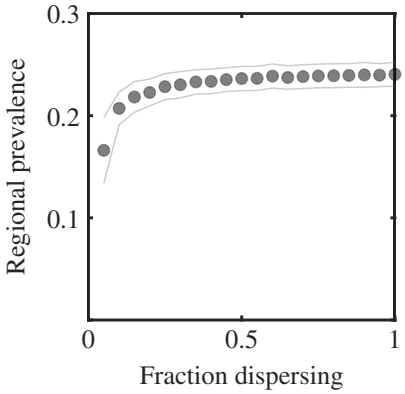

Figure 3. Unconditional dispersal. Regional parasite prevalence as a function of the fraction of individuals in the host metapopulation that disperse when host dispersal is independent of both context and infection state. Parameter values: low transmission ($\beta = 0.1$), low virulence ($\mu_i = \mu_s$s0.02) and no dispersal mortality ($\mu_{ds} = \mu_{di} = 0$). Other parameter values are as given in table 1.

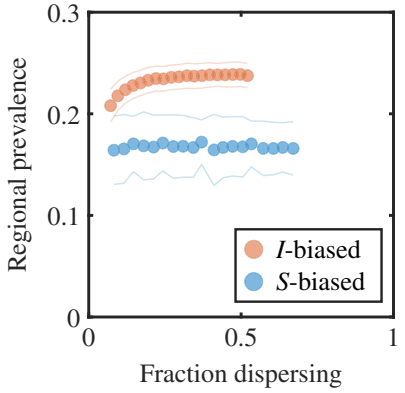

Figure 4. State-dependent dispersal. Regional parasite prevalence as a function of the fraction of individuals in the host metapopulation that disperse, with *I*-biased and *S*-biased state dependence. Host dispersal is independent of context. Parameter values: low transmission ($\beta = 0.1$), low virulence ($\mu_i = \mu_s$s0.02), no dispersal mortality ($\mu_{ds} = \mu_{di} = 0$). Other parameter values are as given in table 1.

To facilitate the interpretation of the results and comparisons across forms of PDD, we present observed regional parasite prevalence as a function of this dispersal fraction, rather than as a function of model parameters (i.e. the form of PDD described by parameters d_{lo}, δ, τ, etc.). First, we consider only scenarios where parasite transmission rate and virulence are both low and there is no dispersal mortality. In later sections, we consider scenarios varying these parameters.

(a) Unconditional dispersal

We start with our null model, meaning host dispersal is unconditional (independent of both context—local parasite prevalence—and infection state). In this case, we find that regional parasite prevalence increases asymptotically with the fraction of the host population that disperses (figure 3).

(b) State-dependent dispersal

I-biased dispersal results in higher regional parasite prevalence than *S*-biased dispersal overall (figure 4). When host dispersal is *I*-biased, regional parasite prevalence increases asymptotically with dispersal fraction (figure 4). Surprisingly, when host dispersal is *S*-biased, changing the fraction of the host population that disperses does not change regional parasite prevalence (figure 4).

(c) State- and context-dependent dispersal

When host dispersal exhibits positive context dependence (host dispersal increases with increasing local parasite prevalence), the relationship between regional parasite prevalence and dispersal fraction is very similar to the unconditional case (figure 5*a*). The value of the asymptote is very similar, but the form of state dependence determines the dispersal fraction at which this asymptote is reached. Interestingly, the effects of state dependence on overall regional parasite prevalence are minimal when host dispersal is positively context dependent.

In contrast, when hosts exhibit negative context-dependent dispersal (hosts dispersal decreases with increasing local parasite prevalence), we find that the relationship between the fraction dispersing and regional parasite prevalence becomes U-shaped

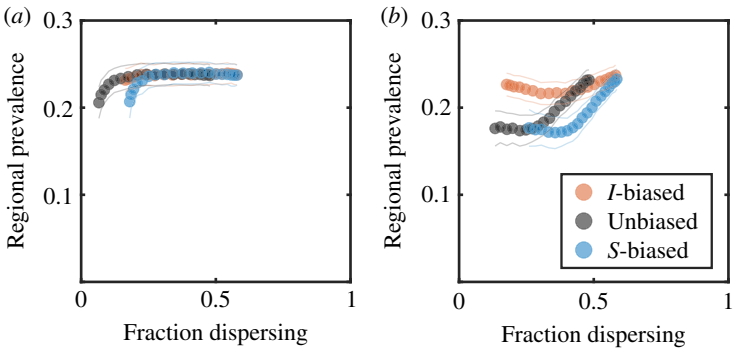

Figure 5. State- and context-dependent dispersal. Regional parasite prevalence as a function of the fraction of individuals in the host metapopulation that disperse for (*a*) positive and (*b*) negative context-dependent dispersal, with and without state dependence. Parameter values: low transmission ($\beta = 0.1$), low virulence ($\mu_i = \mu_s + 0.02$) and no dispersal mortality ($\mu_{ds} = \mu_{di} = 0$). Other parameter values are as given in table 1.

(figure 5*b*). The exact shape of this relationship varies with state dependence. The U-shape is dampened when dispersal is *I*-biased, compared to the *S*-biased and unbiased cases (figure 5*b*).

(d) Effects of dispersal mortality

Adding dispersal mortality that affects infected and susceptible hosts equally changes the shape of the relationship between regional parasite prevalence and dispersal fraction. When host dispersal is unconditional, state or positive context dependent, this relationship changes from generally positive to hump-shaped (figure 6*a–c*). The exception is when dispersal is *S*-biased and context independent—this relationship remains flat (figure 6*b*). Intriguingly, when dispersal exhibits positive context dependence, the effect of state dependence is flipped compared to cases with no dispersal mortality: *I*-biased dispersal leads to lower infection prevalence than unbiased dispersal, and *S*-biased dispersal generally has the highest infection prevalence (figure 6*c*).

When host dispersal exhibits negative context-dependent dispersal, adding dispersal mortality only changes the relationship between fraction dispersing and regional parasite prevalence slightly (figure 6*d*) compared to the case with no dispersal mortality. When there is no dispersal mortality, these relationships have a U shape (figure 5*b*). Adding dispersal mortality makes the positive slopes of the U-shapes less steep (figure 6*d*).

In simulations where infected individuals were more likely to die during dispersal than susceptible individuals, the relationship between dispersal rate and regional prevalence becomes more negative (electronic supplementary material, figure S1*a–c*). Additionally, when dispersal exhibits positive context dependence, the overall difference in regional parasite prevalence between *S*- and *I*-biased dispersal is larger, with the *S*-biased case having higher prevalence (electronic supplementary material, figure S1*c*). When host dispersal exhibits negative context dependence, higher dispersal mortality in infected individuals alters the shape of the relationship between dispersal fraction and regional parasite prevalence. The relationship changes from U-shaped to monotonically decreasing (electronic supplementary material, figure S1*d*). Interestingly, there is an interaction between dispersal fraction and state dependence: the negative slope is steeper when dispersal is *I*-biased compared with the unbiased and *S*-biased cases (electronic supplementary material, figure S1*d*). The result is that *I*-biased dispersal can cause higher or lower regional parasite prevalence than unbiased and *S*-biased dispersal, depending on the dispersal fraction.

(e) Characteristics of the host–parasite system

The relationship between regional parasite prevalence and the fraction of the metapopulation that disperses depends on the characteristics of the host–parasite system. We go back to the simplest case—host dispersal is unconditional (independent of both context—local parasite prevalence—and infection state), and there is no dispersal mortality. Surprisingly, when the transmission rate was high and virulence low, the relationship between the fraction dispersing and regional parasite prevalence was flat (figure 7*a*). When the transmission rate and virulence are both high, regional parasite prevalence increases with the fraction of the host population that disperses (figure 7*b*). This is similar to the pattern when transmission rate and virulence are both low (figure 3), but in this case, regional parasite prevalence does not reach an asymptote. Finally, parasites with low transmission and high virulence always went extinct (not shown) because these parasites kill off their hosts faster than they can spread.

Transmission rate and virulence also change the interaction between form of PDD and fraction dispersing on regional parasite prevalence (electronic supplementary material, figure S2). In the cases shown above (transmission rate and virulence low), the relationship between regional parasite prevalence and dispersal was positive asymptotic when dispersal exhibits positive context dependence and U-shaped when dispersal exhibits negative context dependence (figure 5). When we change the transmission rate to high (keeping virulence low), these relationships in both the positive and negative context-dependent cases become flat (electronic supplementary material, figure S2*a*,*c*). When

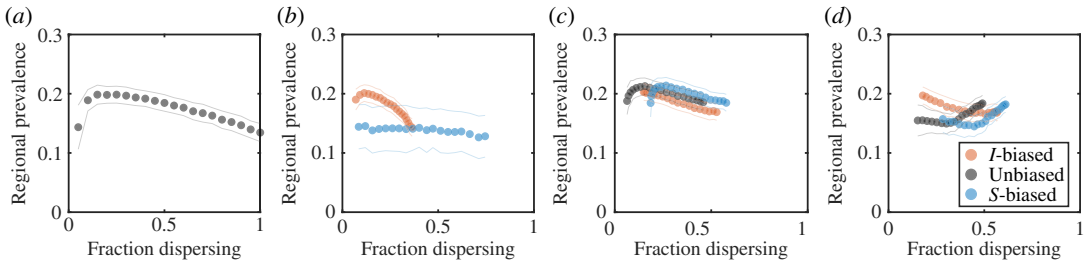

Figure 6. Effects of dispersal mortality. Regional parasite prevalence as a function of the fraction of individuals in the host metapopulation that disperse for (*a*) null dispersal, (*b*) state-dependent dispersal, (*c*) positive context-dependent dispersal, with and without state dependence, and (*d*) negative context-dependent dispersal, with and without state dependence. Parameter values: low transmission ($\beta = 0.1$), low virulence ($\mu_i = \mu_s + 0.02$) and equal dispersal mortality ($\mu_{ds} = \mu_{di} = 0.1$). Other parameter values are as given in table 1.

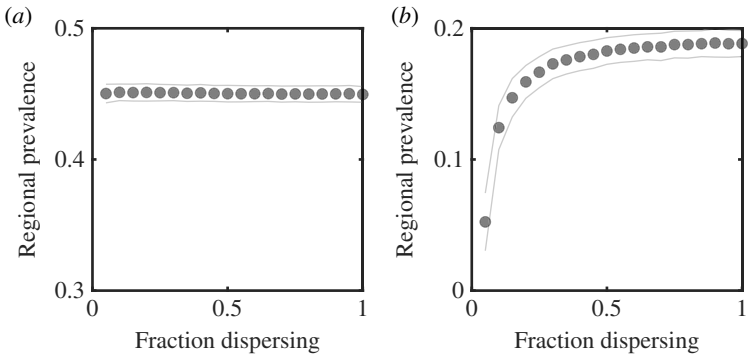

Figure 7. Varied transmission rate and virulence. Regional parasite prevalence as a function of the fraction of individuals in the host metapopulation that disperse for (*a*) high transmission ($\beta = 0.3$)/low virulence ($\mu_i = \mu_s + 0.02$) and (*b*) high transmission ($\beta = 0.3$)/high virulence ($\mu_i = \mu_s + 0.2$), in populations that exhibit unconditional dispersal. Parameter values: no dispersal mortality ($\mu_{ds} = \mu_{di} = 0$). Other parameter values are as given in table 1. Note that simulations with low transmission and high virulence are not shown because these parasites always went extinct.

transmission rate and virulence are both high, these relationships in both the positive and negative context-dependent cases become positive (no asymptote; electronic supplementary material, figure S2*b*,*d*).

(f) Sensitivity to dispersal parameters

The strength of the relationship between regional prevalence and the fraction of the population dispersing depended on the details of the underlying context-dependent dispersal function, but the direction of this relationship was generally consistent (electronic supplementary material, figure S3). Namely, the relationship was most pronounced when there was a stark difference in dispersal between low-prevalence and high-prevalence patches (small d_{lo}, large d_{hi}, large λ; electronic supplementary material, figure S3*f*), and least pronounced when there was little difference in dispersal between low-prevalence and high-prevalence patches (large d_{lo}, small d_{hi}, small λ; electronic supplementary material, figure S3*c*).

4. Discussion

The main finding of our model is that the relationship between dispersal and parasite prevalence varies with PDD, dispersal mortality and characteristics of the host–parasite system, rather than being monotonically and universally positive. We find that regional parasite prevalence can increase, decrease or remain constant as the fraction of the host metapopulation that disperses increases. This contrasts with previous studies which have found positive relationships between dispersal and regional parasite prevalence [32,33] (but see [41] for more nuanced results on parasite occupancy). The key innovation of our model that accounts for this novel prediction is that we incorporate state and context dependency in host dispersal. We demonstrate that the effect of dispersal on parasite prevalence cannot be understood without consideration of how hosts modulate dispersal in response to parasites (context dependency and/or state dependency), and that the effects of PDD interact strongly with the degree of dispersal mortality and the characteristics of the host–parasite system (i.e. transmission rate and virulence).

(a) Mechanisms of dispersal shaping regional parasite prevalence

There are two primary mechanisms through which PDD influences regional parasite prevalence. First, regional prevalence depends on the encounter rates of susceptible and infected hosts [42]. The dispersal of infected hosts can carry the parasite to underexploited populations of susceptible hosts, leading to an increase in regional prevalence. This is the mechanism underlying the positive dispersal–prevalence relationship observed in previous studies [32,33]. This effect is apparent in our study in scenarios with no dispersal mortality and where infected hosts have moderate to high dispersal probability. The dispersal of susceptible individuals into patches with parasites can enhance this effect but has a negligible impact in the absence

of the movement of infected individuals. This can be seen in scenarios with *S*-biased and negative context-dependent dispersal. In this case, infected individuals tend to stay in patches with parasites, rather than move the parasites to new sites. The result is that regional parasite prevalence stays low until the fraction dispersing gets high (>0.5). When more than half the host metapopulation disperses, the parasite is able to spread enough that prevalence increases rapidly.

The second mechanism through which PDD influences parasite prevalence is through effects on dispersal mortality. Mortality of susceptible hosts during dispersal has short-term impacts on parasite prevalence in the sense that it reduces the proportion of susceptible hosts relative to infected ones. Susceptible hosts are also more likely to survive to reproduce, meaning that their deaths lead to a reduction in the birth rate of new, susceptible individuals, further reducing the *S*:*I* ratio. However, mortality of infected hosts has greater and longer-lasting effects on prevalence as each death both removes a single infected host and eliminates the potential for that individual to transmit the parasite to susceptible hosts in the future. This mechanism is similar to the concept of migratory culling, in which infected hosts fail to complete migration, resulting in reduced parasite prevalence in migratory compared to resident populations [43,44]. The differential effects of the deaths of susceptible versus infected hosts mean that adding dispersal mortality to the system can change the effect of PDD on regional parasite prevalence. This is true even when susceptible and infected hosts have the same mortality risk during dispersal, but the effect is magnified when dispersal is *I*-biased because the number of infected hosts dispersing, and therefore experiencing dispersal-related mortality, increases. The unintuitive result is that scenarios in which infected individuals are more likely to disperse (*I*-biased) can have lower parasite prevalence than *S*-biased scenarios with the same dispersal fraction.

(b) Forms of parasite-dependent dispersal

Both state- and context-dependent host dispersal acting independently influence regional parasite prevalence and interact in interesting and non-intuitive ways. When dispersal is state-dependent only, *I*-biased dispersal leads to higher regional parasite prevalence than *S*-biased dispersal. However, *I*-biased PDD and positive context-dependent PDD have similar and redundant effects because both result in the dispersal of infected individuals (who tend to be in high-prevalence patches). When there is no dispersal mortality, regional parasite prevalence as a function of dispersal fraction is nearly identical for the *I*-biased/context-independent, unbiased/positive context-dependent and *I*-biased/positive context-dependent cases. Overall, our results suggest that regional parasite prevalence reaches its maximum value for a given host–parasite system as long as the number of infected dispersers is sufficiently high.

Positive context-dependent dispersal appears to be more common in natural systems than negative context-dependent dispersal. However, negative context dependence may occur in systems in which hosts have evolved to invest in defence traits rather than avoidance traits like dispersal [14], when there is negative temporal autocorrelation in local parasitism risk [11], or when parasite abundance is positively correlated with patch quality, leading hosts to accept parasitism as the cost of remaining in a patch which has other fitness-promoting characteristics. The relationship between dispersal fraction and regional parasite prevalence when hosts exhibit negative context dependency is close to the inverse of the relationship when hosts exhibit positive context dependency. Narrowing into the range of dispersal fractions that are most commonly seen in nature (<0.5), switching from positive to negative context dependency would convert this relationship from strongly positive to flat or slightly negative. This indicates that the particular way that host dispersal behaviour responds to local parasitism risk strongly impacts the regional dynamics of the host–parasite system.

(c) Role of characteristics of the host–parasite system

Another major finding of our study is that the relationship between dispersal fraction and parasite prevalence depends on characteristics of the host–parasite system, specifically transmission rate and virulence. In general, regional prevalence increases with the transmission rate and decreases with increasing virulence. In scenarios with high transmission/low virulence (and no dispersal mortality), regional parasite prevalence can reach its maximum at even low host dispersal. The effect of increasing host dispersal is negligible because parasites are present in most habitat patches even at low host dispersal, and therefore, infected dispersers often move to patches already occupied by the parasite. This contrasts with scenarios with high transmission rate/high virulence (and no dispersal mortality). Here, regional parasite prevalence and the proportion of patches occupied by the parasite tend to be lower than the preceding case. Increasing the movement of infected individuals (by increasing host dispersal rate) into unexploited patches therefore has a large positive effect on prevalence.

Characteristics of the host–parasite system influence the context on which PDD acts and, ultimately, change the consequences of PDD. The effects of PDD on the dispersal–prevalence relationship change substantially in strength and direction (e.g. shifting from positive to flat) as the transmission rate and virulence change. The strong effects of host–parasite characteristics are highly important because they indicate that the relationship between PDD and parasite prevalence will vary widely across host–parasite systems, and even within the same system through time as transmission rate and virulence evolve. This makes the empirical characterization of the PDD–prevalence relationship difficult. Future empirical studies of this relationship should carefully consider the biology of the host–parasite system when interpreting results.

(d) Perspectives and future directions

The interaction between ecological context (dispersal mortality, virulence and transmission rate) and PDD on prevalence is especially relevant in the context of human alterations of landscape connectivity. Practically, the existence of this interaction means that changes to natural systems that alter dispersal rates such as habitat fragmentation, human-vectored dispersal or the creation of natural corridors will have different effects on different classes of parasites. Restoring landscape connectivity in fragmented habitats is a method being used worldwide to protect biodiversity [45]. It has been suggested that increasing connectivity may have a negative impact on the host because of the classical view that dispersal increases the spread and prevalence of parasites [31,32]. This prediction has been supported by some empirical studies. For example, increasing connectivity increases the incidence of biotically dispersed parasites of plants [46]. This is what we expect in this system given that plant hosts have limited capacity for PDD. However, for hosts that exhibit PDD, increasing connectivity may actually reduce regional parasite prevalence in some cases, depending on the form of host PDD and the characteristics of the host–parasite system. This prediction should be tested empirically in a system in which the host exhibits PDD.

Our model is one of the first to consider the consequences of PDD. Our goal was to explore the effects of host dispersal on parasite dynamics. For simplicity, we limited the scope of our model to consider only parasite-dependent emigration decisions, rather than incorporating parasite dependency in all three stages of dispersal (including dispersal distance and settlement [47]). If susceptible individuals can choose to settle in patches with low parasite prevalence, then increasing dispersal should decrease the number of susceptible hosts available to parasites and lead to lower parasite prevalence. A similar effect has been predicted for plant pathogens carried by animal vectors: a theoretical study showed that pathogen spread was slowed when vectors preferred to settle on host plants whose infection status matched their own (i.e. uninfected vectors choosing uninfected host plants and vice versa [48]). There is some evidence that animal hosts may be able to avoid settling in patches with high parasite density [7,49]. However, this remains an open question. It is unclear whether the ability to both efficiently detect parasites in a potential settlement site and make settlement decisions based on this information is common across hosts. This ability likely depends on many factors including habitat type (e.g. chemical signals of natural enemies tend to be more detectable in aquatic than terrestrial systems [40]) and host mobility (e.g. some species are able to sample multiple patches when making settlement decisions, whereas others find it more difficult/risky to sample multiple patches). Interesting avenues of future study would investigate parasite dependency in habitat choice, and whether the shape of the dispersal kernel is context dependent (e.g. do hosts disperse further after being exposed to parasites?).

Here, we make the simplifying assumption that host dispersal is global. This assumption is likely representative for some systems and spatial scales, for example, where dispersal kernels are not monotonically decreasing [50]. However, in many species, immigration probability will not be constant across habitats in a region but will decline with distance [51]. Theory predicts that shifting dispersal from global to more localized reduces metapopulation persistence times, metapopulation size and patch occupancy [52]. Thrall & Burdon [53] show a similar pattern in the context of a plant–pathogen system in which the pathogen disperses independently of the host. Their model demonstrates that local dispersal of the pathogen results in a lower proportion of diseased sites and lower disease load than (near) global pathogen dispersal [53]. In our current model, since the parasite only moves with the host, we expect that changing host dispersal from global to local would have a similar effect of reducing parasite occupancy and prevalence overall.

However, it is unclear whether altering the assumption of global dispersal would change the patterns we observed in our model between PDD and parasite prevalence. Dispersal probability that declines with distance produces spatial autocorrelation in population size in single-species systems [54]. A similar effect may occur in host–parasite systems: localized host dispersal resulting in spatial autocorrelation in the host population as well as the parasites they carry. Autocorrelation could alter the consequences of PDD as it would create a context where hosts would typically move between patches with parasites under positive prevalence-dependent dispersal, and hosts would typically move between patches without parasites under negative prevalence-dependent dispersal. The effects of dispersal on parasite prevalence may therefore be dampened when dispersal is local, but this question should be investigated further.

In our model, we explored a wide range of parameter space on multiple axes including dispersal, dispersal mortality, transmission rate and virulence, to determine how their interactions influence host–parasite systems. However, natural systems do not fill the entirety of this parameter space; traits relevant to host–parasite interactions are often correlated because they evolve in hosts and parasites in concert with, and influenced by, other traits. For example, increasing dispersal mortality tends to cause the evolution of reduced dispersal rates [55], and parasite evolution can be shaped by trade-offs between virulence and transmission rate, such that parasite fitness is maximized at intermediate levels of those traits [56]. We do not comment here on which combination of parameter values in our model may be most commonly found in nature because this becomes a very complicated question when we consider more than two traits. Moreover, our parameter values were chosen to explore parameter space rather than to mimic any natural system(s). However, an interesting avenue of future research would be to estimate the form of PDD and the relationship between dispersal rate and regional parasite prevalence in multiple systems which vary in traits including dispersal mortality, virulence and transmission rate, to test whether the general patterns we observe here match empirical patterns.

Consideration of the evolution of the traits in our model (dispersal rate, dispersal mortality, virulence, etc., as well as other traits relevant to host–parasite interactions such as host resistance) was outside the scope of our model. However, in natural systems, these traits will evolve, and their evolution will depend on the values of other traits. If hosts are dispersing in response to their parasites, this will change the context in which traits like virulence and transmission evolve and potentially change the range of values of these traits expected in natural systems and/or the relationship between traits. Our model shows that characteristics of the host–parasite system including virulence influence the consequences of PDD. These traits will also shape

the evolution of PDD strategies themselves [11]. Many interesting open questions present themselves when PDD is considered in host–parasite evolution. These include whether eco-evolutionary dynamics result in correlations across systems between parasite traits such as virulence and the form of PDD exhibited by hosts, and whether evolutionary conflicts across traits emerge as the result of PDD (e.g. hosts evolving a form of PDD that reduces infection risk for individuals but results in the evolution of increased virulence). Our study highlights that PDD has the potential to change our understanding of host–parasite interactions in space, and so should be integrated into future studies of this subject.

5. Conclusion

In this study, we used a metapopulation model to explore the relationship between PDD and parasite prevalence. Host dispersal in our model is linked to both parasite infection and parasitism risk, which generates variation in dispersal between individuals and among sites. We find that this PDD impacts the relationship between dispersal and regional parasite prevalence; increasing dispersal can either increase or decrease regional prevalence, depending on the form of PDD exhibited by the host, the amount of dispersal mortality and the characteristics of the host–parasite system. Future studies should test these predictions empirically. A fruitful next step would be to conduct an experimental test in a system that our model predicts will exhibit a non-positive relationship between dispersal rate and parasite prevalence (e.g. a system with low transmission rate, low virulence and some dispersal mortality) since positive relationships have already been observed empirically [33]. Our results demonstrate that conditional dispersal behaviour must be considered in order to understand the spatial dynamics of host–parasite systems. Practically, our findings have implications for controlling parasites in the context of human alteration of natural landscapes.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. Model code and results are provided as electronic supplementary material [57].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. C.B.B.: conceptualization, funding acquisition, methodology, writing—original draft, writing—review and editing; A.K.S.: conceptualization, data curation, formal analysis, funding acquisition, methodology, validation, visualization, writing—review and editing.

Both authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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