Evolution of dispersal of social animals under threat of parasitism

入谷, 亮介

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Evolution of dispersal of social animals under threat of parasitism.

Ryosuke Iritani

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Preface

Dispersal is defined as any movement that induces spatial gene flow [1]. Dispersal is central to population ecology because gene flow can contribute to the genetic makeup of metapopulations. In particular, limited dispersal, which attenuates population well-mixing, would result in the situation where localized interactions (including social behaviors, resource competition, and mate competition) occur among closely related individuals, which is called as population viscosity. Viscosity, in turn, has profound effects on the evolution of dispersal. For example, inbreeding avoidance [2] and strong kin competition [3, 4] can drive the evolution of dispersal. In this respect, inclusive fitness theory has been one of the most successful paradigms for explaining dispersal evolution. Inclusive fitness theory predicts that seemingly costly social behaviours, including sex ratio [5], altruism and spite [6, 7] and dispersal [3], can be favored by kin selection in viscous populations. Consequently, personal cost can be partially compensated by indirect benefits from kin, as predicted by Hamilton's rule [8, 9]. Thus, social evolution can be driven by and maintained by viscosity.

On the other hand, sociality is faced with severe menaces of parasitism, because parasite transmission occurs locally thereby inhibiting social evolution. As is widely accepted, in the contexts of host-parasite interactions, dispersal again plays an important role, because the relative level of dispersal for each species can have dramatic impacts on host-parasite coevolutionary processes [10]. As patterns of parasite dispersal can be governed by that of host species (simultaneous migration [11]), understanding the evolutionary causes and consequences for dispersal of host species is of pivotal importance in population biology. Hence, dispersal and sociality are tightly linked in the studies of host-parasite interaction.

It is 'condition-dependent dispersal' that has recently begun to gather broad attentions from empirical, experimental, and theoretical researchers. Condition-dependent dispersal refers to the response in dispersal propensity on any conditions experienced by individuals (*sensu lato*; for more broad definitions on conditional dispersal, see [12]). Some experimental and/or empirical studies demonstrated that host species can respond to own disease-status by modifying disper-

sal propensity; i.e., disease state-dependent dispersal is reported. In particular, dispersal rate of sick individuals and that of healthy individuals were different: dispersal rate was biased towards infected individuals (I-biased dispersal) or towards uninfected individuals (U-biased dispersal).

These differences can generate spatial patterns of distributions for both species. For example, if dispersal rate is biased towards infected individuals, then parasites can also disperse carried by their hosts, leading to spread over spaces. On the other hand, if dispersal rate is biased towards uninfected individuals, then parasites are spatially localized. Hence, explaining the causes and consequences for disease state-dependent dispersal would add greatly to the understandings of spatial dynamics of host-parasite interactions. Unfortunately, however, there are few theoretical studies available that provides predictions on the evolution of dispersal of host species.

In the series of this thesis, I clarify the conditions for disease state-dependent dispersal. Specifically, in Chapter1, I develop a new model of dispersal evolution built on Wright's islands model [13] and Hamilton & May's (1977) seminal model [3], showing the approach to analyze the evolutionary dynamics of disease state-dependent dispersal rates. Also, I show that the genetic structure of host species is modified by disease state-dependent dispersal. In Chapter2, I extend the previous model to more general models such that the timing of parasite infections can modify 'effective' values of costs of dispersal with subsequences of disease state-dependent dispersal.

In the first and second chapters, however, one the most important features that characterizes parasites is neglected: transmission. Various modes of transmission are possible, and we would restrict ourselves to considering horizontal transmission only. Horizontal transmission is fundamental to understand parasite evolution, because (1) life-history constraints on virulence and horizontal transmission are at work so that negative associations between these two traits can develop, and (2) how parasites and infectious diseases spread over space strongly depends on the propensity of horizontal transmission. Therefore, in Chapter3 I incorporate horizontal transmission occurring at a local scale and aim to evaluate the effects of horizontal transmission on the evolution of disease state-dependent dispersal. Consequently, when horizontal transmission occurs after dispersal, there are two equilibria available such that U-biased dispersal and I-biased dispersal are both stable as an evolutionary outcome. On the other hand, when assuming that horizontal transmission occurs before dispersal, no disease state-dependent dispersal is expected, i.e., dispersal rates are equal for infected and uninfected individuals. I can explain these results from altruism-spite aspects of social evolution theory ([6, 7]).

Chapter1

Dispersal plays a fundamental role in shaping the ecological processes such as host—parasite interactions, and the understanding of host dispersal tendency leads to that of parasites. Here, we present the result of our study on how the evolutionarily stable dispersal of a host would depend on parasite infection, considering kin competition among neighbours. We show that the evolving dispersal rate might be higher for susceptible than for infected individuals (S-biased dispersal) or vice versa (I-biased dispersal). S-biased dispersal is favoured by strong virulence affecting competitive ability, by high rate of parasite release during dispersal, and by low virulence for infected emigrants (i.e. low virulence affecting dispersal ability), whereas I-biased dispersal is favoured in the opposite situation. We also discuss population structure or between-deme genetic differentiation of the host measured with Wright's F_{ST}. In I-biased dispersal, between-deme genetic differentiation decreases with the infection rate, while in S-biased dispersal, genetic differentiation increases with infection rate.

Chapter2

The process of dispersal is central to population biology and evolutionary ecology. Because of negative impacts on host fitness, parasite infection generates potential costs of dispersal. However, theoretical predictions that address this issue are lacking. Here, we develop a mathematical model to demonstrate how the dispersal rate of hosts evolves under the influence of parasites in ecological scenarios incorporating pre-, during-, and post-dispersal infection/recovery events. We show that (1) the dispersal tendency is strongly biased towards either infected individuals or susceptible individuals, (2) the bias is inherently determined by the parasite-mediated relative cost of dispersal, and (3) the dispersal costs are determined by the autocorrelation of disease states (susceptible and infected) between pre- and post-dispersal. Our results suggest that parasite virulence in concert with the timing of infection drive the evolution of disease state-biased dispersal. To understand the evolutionary processes in spatial host-parasite systems, the parasite-induced costs of dispersal need to be taken into account.

Chapter3

In viscous populations, horizontal transmission can occur among closely related individuals, which incurs considerable costs to sociality. Disease-state dependent dispersal, whereby individuals dis-

perse conditionally on their own disease-state, may solve this: infected individuals can lower the risk of transmission to siblings if they disperse, but at the same time, uninfected individuals can escape from such a risk by dispersing. Empirical studies have found that either pattern for disease-state dependent dispersal is relevant so that dispersal propensity is biased towards infected individuals or uninfected individuals. No theoretical study is available, however, that investigates the effect of horizontal transmission among kin on the evolution of dispersal. Here, we develop kin selection models and aim to show that either pattern of disease-state dispersal can evolve by natural selection. We found that before-dispersal transmission has no impact, while after-dispersal transmission has a dramatic impact such that disease-state dependent dispersal exhibits two stable equilibria, where dispersal biases extremely towards uninfected or infected individuals, indicating that transmission avoidance can evolve in viscous populations. We discuss the mechanisms that produce such extreme endpoints of evolution in terms of altruism.

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Chapter 1.

Parasite infection drives the evolution of state-dependent dispersal of the host.

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1. Introduction

Dispersal is defined as any movement of individuals or propagules from the natal or breeding area to another area (Greenwood & Harvey 1982; Ronce 2007). It is a widespread phenomenon and has broad consequences for the genetic makeup of populations. The mode and rate of the dispersal of organisms has been studied intensively in both community ecology and evolutionary ecology (Clobert 2001).

Mixed or partial dispersal (i.e. dispersal heteromorphism) is commonly observed in wild species (Frank 1986), and mathematical models have been developed to predict the evolutionary outcome of the dispersal rate. High dispersal might be favoured under diverse selective pressures, such as inbreeding depression (Bengtsson 1978; May 1979; Motro 1991; Perrin & Mazalov 1999, 2000; Roze & Rousset 2005), local kin competition (Hamitlton & May 1977; Motro 1982a, 1982b, 1983; Frank 1986; Taylor 1988; Gandon 1999; Gandon & Michalakis 1999) and asynchronous temporal variability in local fitness and/or spatio-temporal resource heterogeneity (Gadgil 1971; van Valen 1971; Levin *et al.* 1984; Cohen & Levin 1991; McPeek & Holt 1992; Holt & McPeek 1996; Travis 2001); however, in other situations, philopatry (i.e. lack of dispersal) might be favoured, and these theoretical predictions above were tested empirically to be found feasible (reviewed in Ronce 2007). For example, rapid evolution of reduced seed dispersal in urban patchy environments has been reported in *Crepis Sancta* (Cheptou *et al*. 2008). A reduction in dispersal potential in anemochorous plants on oceanic islands has evolved over as little as 10 years (Cody & Overton 1996).

The level of dispersal determines the level of gene flow across metapopulations, and modifies the genetic structure (for example measured with *F*-statistics; Wright 1943) of the population; therefore, understanding evolutionary forces acting on dispersal-related traits is important in the study of population biology. Dispersal affects not only population structure of the focal species but also its interactions with other species such as predators and parasites in subdivided populations. In turn, heterospecific interactions could have profound effects on the evolution of dispersal (Clobert 2001).

Parasite load is ubiquitous in living organisms (Combes 1998; Morand & Krasnov 2001), and it may incur a large cost to socially group-forming animals (Alexander 1974). Parasites affect various life history traits of their hosts (Thomas *et al*. 2007; Débarre *et al*. 2011), and host–parasite co-migration among subpopulations influences metapopulation dynamics (e.g. May & Southwood 1990). Because hosts undergo dispersal carrying their parasites attached, host dispersal tendency is important in determining the parasite dispersal, the abundance in local habitats, and the spatial distribution of both species (e.g. Galbreath & Hoberg 2012). Theoretical studies of the host–parasite coevolutionary process derived many interesting predictions. For examples, host dispersal or spatial structure drives the evolution of parasite virulence and transmission ability (Gandon *et al.* 1996; Gandon & Michalakis 2000; Boots & Sasaki 1999, 2001; Morgan *et al*. 2005). The evolution of parasites in a spatially structured population is formed by the interplay of genetic and epidemiological structures, which are in turn characterized by the feedbacks of ecological dynamics (Lion & Boots 2010). Limited parasite dispersal favours weaker virulence because of kin selection caused by a high relatedness among parasites infecting the same host individual (Wild *et al.* 2009). If hosts show higher migration rates than that of parasites, then hosts might diversify their genes faster than parasites, and parasites might become locally maladapted in host–parasite metapopulations (Gandon *et al.* 1996). Oppliger *et al.* (1999) tested the theoretical prediction that were supported in a lizard species.

In these theoretical models, however, host migration rates are often assumed to be the same between infected and susceptible individuals (where, by 'susceptible', we mean 'uninfected' henceforth). Because parasites have negative impacts on hosts (Lehmann 1993; Fitze *et al*. 2004), hosts have evolved mechanisms to avoid infection, and one example is dispersal (Clobert 2001, pp.169; Folstad *et al*. 1991; Brown & Brown 1992; Chaianunporn & Havestadt 2012); however, only a few empirical studies focus on the change in host dispersal tendency caused by parasitism. Particularly, in some species, 'S-biased dispersal' is reported; i.e. susceptible hosts are more dispersive

than those infected with parasites in a great tit *Parus major* (Heeb *et al*. 1999), in a ciliate *Paramecium caudatum* (Fellous *et al.* 2011), and in a money spider *Erigone atra* (Goodacre *et al*. 2009). In contrast, several studies showed that infected hosts disperse more frequently than susceptible hosts (called 'I-biased dispersal'), in a yellow-bellied marmot *Marmota flaviventris* (van Vulen 1996) and in a cliff swallow *Hirundo pyrrhonota* (Brown & Brown 1992). In a more complex case, the maternal parasite load of ticks in a common lizard (*Zootoca vivipara*) altered offspring natal dispersal depending on offspring sexes (Sorci *et al*. 1994). These dispersal trends might have important effects on the community genetic structure, because they could greatly affect spatial distribution of hosts and parasites, in terms of not only the number of individuals but also allele frequencies. Indeed, several studies have argued that parasite population structure is determined by host movement or migration, which is tested and confirmed in several nematodes infecting mammals (Blounin *et al.* 1995), seabird ticks infecting several birds (McCoy *et al.* 2003), trematodes infecting several freshwater hosts (Blasco–Costa, *et al.* 2012), and freshwater pearl mussels larvae parasitizing trout or salmon (Karlsson *et al.* 2013). In contrast, how the host population structure is shaped by parasite-affected dispersal is still unknown.

Here, we study the evolutionary outcomes of host dispersal that might differ conditionally to the state (infected or susceptible), using the direct fitness approach (Taylor & Frank 1996). We find that dispersal bias toward susceptible or infected individual can evolve by natural selection, which is determined by a single quantity mainly characterized by virulence parameters: virulence affecting host's competitive ability favors S-biased dispersal, whereas virulence affecting host's disperesal ability favors S-biased dispersal. We also discuss the genetic structure of the host population in the same local population, measured with Wright's F_{ST} (Wright, 1943). F_{ST} is shown to be strongly affected by several parasitic and demographic factors.

2. Methods

We consider a host population that has a structure similar to that studied by Hamilton &

May (1977) and other studies (e.g. Frank 1986, Taylor 1988). We consider an asexually reproducing host with haploid genetics and discrete non-overlapping generations (i.e. Wright-Fisher demography). Following is the life history of the host:

(1) Start of the generation: the population consists of infinitely many demes, each of which supports exactly *N* adults. That is, *N* represents the deme size.

(2) Reproduction: each adult produces a very large number, *M*, of offspring.

(3) Infection: in each deme, a fraction *R* of offspring are infected, but fraction 1–*R* are uninfected, i.e. they remain susceptible.

(4) State-dependent dispersal: in a resident population, susceptible individuals disperse at rate z_S, whereas infected individuals disperse at rate z_I.

(5) Mortality and release during dispersal: dispersal incurs a cost so that susceptible (or infected) individual survives with probability p_S (or p_I, respectively). We assume $p_S > p_I$ with $p_I = (1-\varepsilon)p_S$, which implies that infected individuals suffer a greater mortality in the dispersal stage than do susceptible individuals because of selection against infected emigrants, ε. Also, during dispersal, a fraction γ of infected individuals who survive the dispersal stage become released from the parasite (the rate of parasite maladaptation to the novel environment).

(6) Mortality event: after dispersal, infected individual dies with rate *d* (parasite virulence affecting competitive ability) before competition, i.e. infected individuals show weaker competitive ability than susceptible individuals by factor $(1-d)$.

(7) Competition: individuals compete for *N*-breeding opportunity to be parents in the next generation.

We consider the fitness of a rare mutant (focal individual) whose dispersal rate, (x_S, x_I), are slightly different from those of the resident, (z_S, z_I). The fitness of the focal individual depends on dispersal rates of deme mates (neighbours). The neighbours have some chance to be sibs of the focal individual, and are likely to be genetically similar to the focal individual compared with individuals randomly sampled from the entire population. We define y_S and y_I to be dispersal rates averaged over

all the other individuals in the same deme (neighbour-mean dispersal rates).

The fitness, w, of the focal individual is given as $w = w_{\mathrm{HOME}} + w_{\mathrm{AWAY}}$, where w_{HOME} is the expected number of adult offspring of the focal individual in the natal deme (HOME deme). It is given as follows:

$$w_{\mathrm{HOME}} = \frac{M(1-R)(1-x_S) + MR(1-d)(1-x_I)}{M\{(1-R)(1-y_S + p_S z_S) + R(1-d)(1-y_I + (1-\gamma)p_I z_I) + R\gamma p_I z_I\}} \qquad (1a)$$

The numerator indicates the number of copies of the focal individual, and the denominator indicates the total number of individuals in the HOME deme.

In a similar manner, w_{AWAY} is the expected number of demes occupied by a copy of the focal individual in AWAY demes (i.e. non-HOME demes) and is given as follows:

$$w_{\mathrm{AWAY}} = \frac{M(1-R)p_S x_S + MR(1-d)(1-\gamma)p_I x_I + R\gamma p_I x_I}{M\{(1-R)(1-z_S + p_S z_S) + R(1-d)(1-z_I + (1-\gamma)p_I z_I) + R\gamma p_I z_I\}}. \qquad (1b)$$

After some arithmetic, these can be reduced to

$$w_{\mathrm{HOME}} = \frac{(1-R_{\mathrm{E}})(1-x_S) + R_{\mathrm{E}}(1-x_I)}{(1-R_{\mathrm{E}})(1-y_S + p_S z_S) + R_{\mathrm{E}}(1-y_I + (1+\frac{\gamma d}{1-d})p_I z_I)} \qquad (2a)$$

and

$$w_{\mathrm{AWAY}} = \frac{(1-R_{\mathrm{E}})p_S x_S + R_{\mathrm{E}} p_I (1+\frac{\gamma d}{1-d}) z_I}{(1-R_{\mathrm{E}})(1-z_S + p_S z_S) + R_{\mathrm{E}}(1-z_I + (1+\frac{\gamma d}{1-d})p_I z_I)}, \qquad (2b)$$

where R_{E} is 'effective infection rate', defined as $R_{\mathrm{E}} = \frac{(1-d)R}{(1-R)+R(1-d)} = \frac{(1-d)R}{1-dR}$. This value indicates the expected number of individuals infected before dispersal considering parasite-induced mortality. We can see that R_{E} is a monotonically increasing function of R, and it increases from 0 to 1, as R increases from 0 to 1, because $d < 1$.

We need to note some assumptions of this model. First, we have assumed that infection rate R and virulence d are constant both spatially and temporally. To justify

this restrictive assumption, we additionally presume that the infection event (before dispersal) occurs independently and identically across space and time, in infinite demes and over sufficiently large generations, so that we can regard the infection rate *R* and *d* as the "expected infection rate" and "expected virulence". Accordingly, our model gives explanation for the host evolution, rather than coevolution. Second, we do not explicitly consider horizontal transmission dynamics (Anderson & May 1979), but we can take it into account if we presume that the transmission occurs before dispersal; thus we can view the infection rate *R* as the prevalence after horizontal transmission. Finally, we do restrict ourselves to consider no population dynamics, although there are some studies that work on the evolution of altruistic behavior incorporating demographic factors (e.g. Lehmann *et al*. 2006; Alizon & Taylor 2008). Our assumptions here may, however, provide the insights into the aspect of how parasite infection operates on dispersal rates of the hosts.

2.1 Relatedness, genetic structure, and evolutionary equilibrium

Each individual has a pair of traits, (x_S, x_I). In general, let x_i be the phenotype of an individual, and y_i be the neighbour-mean phenotype. y_i is positively correlated with phenotype of the focal individual x_i. The degree to which this is correlated is represented by relatedness among neighbours, $F_{\mathrm{ST}}^{\mathrm{R}}$ (Hamilton 1964a, 1964b). To clarify this, we adopt the direct fitness approach (Taylor & Frank 1996). Let g_S (or g_I) be the breeding value of the trait x_S (or x_I, respectively). The phenotype is the sum of the breeding value and the environmental value, $x_i = g_i + e_i$ and hence, $\mathrm{d}x_i / \mathrm{d}g_i = 1$ (for $i = S, I$). On the other hand neighbour-mean phenotype, y_i, is less strongly correlated with the breeding value of the focal individual g_i. We set $\mathrm{d}y_i / \mathrm{d}g_i = F_{\mathrm{ST}}^{\mathrm{R}} \le 1$ (for $i = S, I$). Since we consider no pleiotropy or linkage disequilibirum, we have $\mathrm{d}x_i / \mathrm{d}g_j = \mathrm{d}y_i / \mathrm{d}g_j = 0$ for $i \neq j$. A similar formalism has been adopted by Perrin & Mazalov (1999, 2000) that discusses the evolution of

sex-biased dispersal. Throughout, we assume that the selection is weak, i.e., the deviation $\delta_i := x_i - z_i$ is small for $i = S, I$.

At an interior evolutionary equilibrium, the selection gradients with respect to the breeding value should be zero, which implies $\frac{\mathrm{d}w}{\mathrm{d}g_S} = \frac{\mathrm{d}w}{\mathrm{d}g_I} = 0$ (Taylor & Frank 1996; Frank 1998). Because the fitness w is a function of the phenotypes of the focal individual (x_S, x_I) and of its neighbours (y_S, y_I), we have the following equations:

$$\frac{\mathrm{d}w}{\mathrm{d}g_S} = \frac{\partial w}{\partial x_S} + F_{\mathrm{ST}}^{\mathrm{R}} \frac{\partial w}{\partial y_S} = 0, \tag{3a}$$

$$\frac{\mathrm{d}w}{\mathrm{d}g_I} = \frac{\partial w}{\partial x_I} + F_{\mathrm{ST}}^{\mathrm{R}} \frac{\partial w}{\partial y_I} = 0 \tag{3b}$$

Both derivatives are evaluated at $x_i = y_i = z_i = z_i^*$ for $i = S, I$. Eq. (3) provides the evolutionary isoclines, and are rewritten as follows (Appendix A) :

$$L_S : F_{\mathrm{ST}}^{\mathrm{R}}(1-u^*) = (1-p_S)(1-u^*+v^*), \tag{4a}$$

$$L_I : F_{\mathrm{ST}}^{\mathrm{R}}(1-u^*) = (1-p_I)(1-u^*+v^*), \tag{4b}$$

where $u^* = (1-R_{\mathrm{E}})z_S^* + R_{\mathrm{E}} z_I^*$ is the expected emigration rate and $v^* = (1-R_{\mathrm{E}})p_S z_S^* + R_{\mathrm{E}} p_I \left(1 + \frac{rd}{1-d}\right) z_I^*$ is the expected immigration rate. The two isoclines L_S and L_I form lines through the state space (the unit square) for $0 \le z_S^* \le 1$ and $0 \le z_I^* \le 1$ (see Section 3). Note that we set the coefficient of relatedness, $F_{\mathrm{ST}}^{\mathrm{R}}$, the same for susceptible and infected individuals, because the non-heritable class division into susceptible and infected individuals occurs randomly after reproduction.

Relatedness is not a given constant, but is determined dynamically by several factors, such as dispersal rates, dispersal cost, or breeding opportunity. Taylor (1988) discussed the evolutionary outcome of relatedness and dispersal rate at equilibrium, and we employed this method of analysis. As derived in Appendix B, the stationary value of $F_{\mathrm{ST}}^{\mathrm{R}}$ is

$$F_{\mathrm{ST}}^{\mathrm{R}} = \frac{1}{N - (N-1)(1-m)^2}, \tag{5a}$$

where m is the backward migration rate (Vuilleumier *et al.* 2010), which is defined as

$$m = \frac{v^*}{1-u^*+v^*}. \tag{5b}$$

Here we discuss the evolution of dispersal rate that is affected by relatedness $F_{\mathrm{ST}}^{\mathrm{R}}$, and the relatedness $F_{\mathrm{ST}}^{\mathrm{R}}$, in turn, is determined by demographic parameters, including dispersal rates.

To study population genetic structure, we calculate Wright's F_{ST} (see Appendix C). F_{ST} measures spatial differentiation of alleles, i.e. the extent to which neutral markers within a deme are more alike than genes sampled from different demes. In the infinite islands model (Wright 1943; Rousset 2004), F_{ST} is written as

$$F_{\mathrm{ST}} = \frac{(1-m)^2}{N-(N-1)(1-m)^2} = F_{\mathrm{ST}}^{\mathrm{R}}(1-m)^2, \tag{6}$$

which is derived under a common assumption that those born in the different demes are not related to each other in the infinite islands population (e.g. Ohtsuki 2010).

3. Results

In Appendix D, we show that the evolutionarily stable conditions (Maynard Smith 1982), expressed in two equations given by Eq. (4), are not satisfied simultaneously within the unit square for $0<z_S<1$ and $0<z_I<1$. Hence we search for the ES-dispersal rate (z_S^*, z_I^*) on the boundary of the unit square (i.e. either z_S or z_I is 0 or 1). We also investigate the convergence stability of the calculated ESS by depicting evolutionary vector field.

As illustrated in Figure 1, under some conditions the ES-dispersal rate of susceptible individuals is larger than that of infected individuals ($z_S^* > z_I^*$), which implies that S-biased dispersal should evolve; however, under other conditions, the ES-dispersal rate for susceptible individual is smaller than that of the infected ($z_S^* < z_I^*$), which implies I-biased dispersal.

The derivation is explained in Appendix E, and the results are summarized as follows:

If $\dfrac{\gamma d}{1-d} > \dfrac{p_S - p_I}{p_I} = \dfrac{\varepsilon}{1-\varepsilon}$, dispersal rate is higher for infected individuals. (7a)

If $\frac{\gamma d}{1-d} < \frac{p_S - p_I}{p_I} = \frac{\varepsilon}{1-\varepsilon}$, dispersal rate is higher for susceptible individuals. (7b)

The left hand sides of the inequalities are the product of the parasite release rate multiplied by the virulence of the parasite expressed in competitive ability. The right hand sides are the selection intensity on infected emigrants. Both competitive ability and dispersal survival are lower for infected hosts than for susceptible hosts. Both expressions are the difference in these quantities divided by the quantity for infected hosts. Note that if the equality of (7) is satisfied, then the system is neutrally stable because the evolutionary isoclines defined by the two equations (4) are identical. Thus we exclude the equality of (7) to consider the generic parameter sets.

3.1. When $\frac{\gamma d}{1-d} > \frac{\varepsilon}{1-\varepsilon}$ holds true.

Figure 1 illustrates the vector field of evolutionary dynamics. Horizontal (or vertical) axis is dispersal rate for susceptible individual, z_S , (or dispersal rate for infected individual, z_I , respectively). We can calculate the ESS, on the boundary of the unit square for $0 < z_S < 1$ and $0 < z_I < 1$ by analyzing the sign of selection gradients, such as $\frac{\mathrm{d}w}{\mathrm{d}g_S}$ and $\frac{\mathrm{d}w}{\mathrm{d}g_I}$ evaluated at $x_i = y_i = z_i = z_i^*$ for $i = S, I$ (Figure 1).

In Appendix A, we derive the ESS solution and relatedness $F_{\mathrm{ST}}^{\mathrm{R}}$ at evolutionary equilibrium. Figure 2 illustrates the ES-dispersal rates for susceptible and infected individuals, plotted against infection rate *R* for varying *d*. The ES-dispersal rate for infected individual, z_I , is a non-decreasing function of the reduction in competitive ability or parasite-induced mortality due to infection, *d*. A large *d* indicates a strong incentive for infected individuals to disperse and to acquire the opportunity for parasite release. In the island model with a homogeneous population, the dispersal rate should evolve to an intermediate value (Taylor 1988; Rousset 2004). In the population of both susceptible and infected individuals, their ES-dispersal rate (z_S^*, z_I^*) might be either very large or zero, but their average (emigration rate) $u = (1 - R_{\mathrm{E}})z_S + R_{\mathrm{E}} z_I$ takes an

intermediate value.

The left panels (a) of Figure 3 illustrate how the between-deme genetic differentiation F_{ST} changes with infection rate R for varying deme size N and virulence affecting competitive ability d. This figure shows that genetic differentiation F_{ST} is a decreasing (or more precisely, non-increasing step-wise) function of infection rate R. Virulence, d, also affects the differentiation; when R is small d has no effect on it, whereas when R is large, large d greatly reduces F_{ST}. This is because a strong virulence makes more infected individuals emigrate from their natal deme. The step-wise behaviour of F_{ST} can be deduced from analytical expression shown in Appendix E. F_{ST} has three phases: for small R, $dw/dg_S = 0 < dw/dg_I$ (the first phase) holds at (z_S^*, z_I^*), so that an intermediate value of z_S^* is favoured while high z_I^* is favoured to be unity (i.e. $z_I^* = 1$). Intuitively, "$dw/dg_I > 0$" implies "for infected individuals, the more dispersive, the better". As R increases, both dw/dg_S and dw/dg_I decrease, resulting in $dw/dg_S < 0 < dw/dg_I$ (the second phase) with $(z_S^*, z_I^*) = (0,1)$. In the second phase, F_{ST} decreases with R, because the obligate dispersal for infected individuals (i.e. $z_I = 1$) causes high level of gene flow (as R increases). Moreover, increased R causes $dw/dg_S < 0 = dw/dg_I$ (the third phase) and an intermediate value of $z_I^*(<1)$ is favoured while $z_S^* = 0$. A large deme size N reduces the critical value(s) of three phases of F_{ST}. This is because large N reduces z_S^* for small R so that z_S^* is rapidly reduced to be null, which makes F_{ST} enter on a decreasing phase (the second phase).

3.2. When $\frac{\gamma d}{1-d} < \frac{\varepsilon}{1-\varepsilon}$ holds true.

By the analysis similar to that in the last section, we can derive the ESS solution of dispersal rates for susceptible and infected hosts as explained in Appendix E. In contrast to the previous discussion, the ES-dispersal rate is smaller for infected hosts.

Figure 2 illustrates how the ES-dispersal rates depend on infection rate R, for varying virulence d. Since the dispersal rate of infected individuals stays low, the dispersal rate of susceptive hosts tends to increase to keep the average dispersal rate in an intermediate value. When d is small, infected individuals have weak incentive for

dispersal to stay in the natal deme. The right panels (b) of Figure 3 illustrate the between-deme genetic differentiation F_{ST} for varying R, d, and N. We can see that F_{ST} increase with infection rate R, because z_I is very small. Also, F_{ST} increases with decreased d and increased N. This is because, when d is small, the level of gene flow among demes is high due to high dispersal rates (z_S, z_I) (Figure 2). Large deme size N causes the critical value of three phases

The dispersal bias is determined by the sign of $\gamma d/(1-d)-\varepsilon/(1-\varepsilon)$, that in turn is determined by the balance between virulence affecting competition d and that affecting dispersal success ε. Hence we analyze the dependence of ES-dispersal rate (z_S, z_I) (and thus the dispersal bias) on relative intensity of virulence ε / d, with d fixed, in Figure 4. We find dispersal bias is strongly affected by infection rate R. Also we can see that the dispersal bias changes at a particular value (see Discussion).

4. Discussion

We studied the evolution of the dispersal rate of a host species, focusing, in particular, on the possible difference of the ES-dispersal rate between a host infected by parasites and a host that is not infected.

4.1 evolution of dispersal bias

Our analyses showed that the dispersal rate might evolve to be higher for either the infected hosts (I-biased dispersal), or for susceptible hosts (S-biased dispersal), depending on the parameters. I-biased dispersal evolves if the virulence in competitive ability multiplied by the parasite release rate is greater than the level of selection against infected emigtants. In contrast the S-biased dispersal evolves if the opposite inequality holds true. A high relatedness F_{ST}^{R} within a deme (i.e. among neighbours) favours the evolution of high dispersal, as has been pointed out by Frank (1986). As shown in Figure 2, when $\gamma d/(1-d) > \varepsilon/(1-\varepsilon)$ holds true, a higher infection rate R favours a low dispersal rate for susceptible individuals and a high dispersal rate for infected

individuals.

Infected individuals have a strong incentive for dispersal if dispersal provides an opportunity that occurs with probability γ for being released from virulent parasites. This increase in dispersal for infected individuals leads to philopatry of susceptible individuals, because natural selection favours the average rate of dispersal at an intermediate value, as studied in Frank (1986), Taylor (1988), Frank (1998), and Rousset (2004).

The sign of $\gamma\left(d/(1-d)\right)-\varepsilon/(1-\varepsilon)$ is determined by the interplay of ε and d for $\gamma>0$. Figure 4 illustrates the dependence of ES-(z_S,z_I) on the relative value of virulence, ε/d. After some arithmetics, we have

$$\begin{aligned} \frac{\gamma d}{1-d} > \frac{\varepsilon}{1-\varepsilon} \quad &\Leftrightarrow \quad \varepsilon < \varepsilon_0 \\ \frac{\gamma d}{1-d} < \frac{\varepsilon}{1-\varepsilon} \quad &\Leftrightarrow \quad \varepsilon > \varepsilon_0 \end{aligned} , \tag{8}$$

where $\varepsilon_0 := \gamma d/(1-d+\gamma d)$, which leads to the threshold value of $\varepsilon/d=\varepsilon_0/d$. As ε/d increases with d fixed, quantity $\gamma d/(1-d)-\varepsilon/(1-\varepsilon)$ changes its sign from positive to negative at $\varepsilon/d=\varepsilon_0/d$. Hence the ESS dispersal bias changes discontinuously (from I-bias to S-bias). Also note that $0\le\varepsilon_0=\gamma d/(1-d+\gamma d)\le 1$, as long as $0<d<1$; thus, the ESS dispersal bias always changes at an intermediate value of ε/d. This suggests us that the relative intensity of parasite virulence (on dispersal stage or on competition stage) may drastically change host's adaptive dispersal tendency.

In some sytems, parasites are reported to manipulate host dispersal behaviours to facillitate their dispersal (Thomas *et al.* 2007). Lion *et al*. (2006) discussed the evolution of parasite manipulation of host dispersal, considering differential migration patterns for infected and susceptible individual. They incorporate dispersal rates as a given parameters to analyze the consequences of the dispersal of infected individuals for the spatial dynamical aspects in a host–parasite system at (non-trivial) steady states

(see the section 3 of their article: Ecological Dynamics). From our results, dispersal bias towards S or I is quantitative and it is determined by parasite virulence (virulence). Combining our results and theirs, we provide the possibility of more detailed analysis of spatial host–parasite interactions, e.g. clustering of susceptible hosts and global density of infected hosts.

We leave two additional notes. First, substituting $R_{\mathrm{E}} = 0$ into (E-7) (i.e. assuming no infection), we can recover the following result obtained by previous studies (e.g. Taylor 1988; Rousset 2004; Massol *et al*. 2011 for continuous-time model):

$$x_S = \frac{F_{\mathrm{ST}}^{\mathrm{R}} - c_S}{F_{\mathrm{ST}}^{\mathrm{R}} - c_S^2}, \tag{9}$$

with $F_{\mathrm{ST}}^{\mathrm{R}} = r_S := \left(1 + \sqrt{1 + 4N(N-1)c_S^2}\right) / (2N)$. Eq. (8) leads to $x_S > 0$: this implies that in the presence of kin competition, some level of dispersal is always favoured as an evolutionary outcome in the infinite islands model.

Second, our model is applicable to the prediction of the evolution of dispersal in a class-structured population, particularly when there are two classes, say, "weaker" and "stronger" (but see Ronce *et al*. (2000) for age-structured populations, and Gyllenberg *et al*. (2008, 2011), and McNamara & Dall for the evolution of unconditional philopatry). In some natural populations, socially weaker individuals tend to disperse ('ideal despotic distribution', Fretwell (1972)), whereas in some cases stronger individuals are reported to disperse more frequently, e.g. in roe deer (Wahlström & Liberg (1995)), and in common lizard (Galliard *et al*. 2005). Also, our formulation describes the evolution of state-dependent dispersal, i.e. conditional dispersal, which is well studied in an amount of previous studies (e.g. density- and/or patch-size- dependence, Travis 1999, Poethke & Hovestadt 2002; age structure, Ronce *et al*. 2000, Bonte & de la Pena 2009; Cotto *et al.* 2013; local predation risk, Poethke *et al*. 2010; response to prey dynamics under predation, Travis *et al.* 2013). Our analytical results show that dispersal bias might evolve in a class-structured population, and the bias is determined by the relative magnitude of costs of dispersal. Note that, however,

the ‘costs of dispersal’ may include varieties of biological scenarios (Bonte *et al*. 2012). In our model, the cost of dispersal is characterized by the following scenarios: (I) mortality during dispersal, (II) parasites release during dispersal, and (III) parasite-induced mortality after dispersal (see eq.(D-3) in Appendix D); our result here is somehow general and provides testable predictions on how dispersal cost variation affects the evolution of dispersal in spatially structured populations. There is a limitation when applying our method to the evolution dispersal depending on more than two states (see Appendix F), and overcoming the difficulty requires us to specify ecological scenarios in more detail.

4.2 Association of host dispersal with parasite traits

Boots *et al*. (2004) have analyzed the evolutionary association between virulence d and host dispersal z_I, and concluded that host mobility enhances the success of parasite transmission. In addition, using moment equations, Lion & Boots (2010) predicted that whether parasites are harmful to their hosts is dependent on the balance between the level of horizontal transmission and the possibility of newly-infection, and their results are supported by an experimental test (Boots & Mealor 2007). In our analyses, the condition for I-biased dispersal to evolve is given by Eq. (7a). This indicates that a strong parasite virulence affecting competitive ability favours I-biased host dispersal, which might lead to a spatial spread of infected individuals or infectious disease (Fellous *et al.* 2011). In this scenario, we expect that parasites evolve to be virulent when spatial spread is advantageous for parasites. In contrast, if the virulence appearing in the reduction of competitive ability d is very weak, then S-biased dispersal is favoured and adaptive z_I becomes small by natural selection. In this case, parasites may fail to spread out spatially and form spatial clusterings.

From our analyses and previous studies (e.g. Boots & Sasaki 1999), we can conclude that, when we consider host–parasite coevolution, we need to take into account of the following life history components: (I) host migration tendency, x_S and x_I;

(II) parasite migration tendency (or parasite transmission), which is not considered in this study; (III) parasite maladaptation or release during dispersal, γ; (IV) parasite virulence affecting host competitive ability, *d*, and (V) the selection against infected emigrants, *ε*.

As far as we know, few or no empirical studies exist in which the evolution of host dispersal and parasite traits is investigated simultaneously. If the cost of host dispersal and parasite traits can be evaluated for the same system, the inequality conditions in Eqs. (7a), and (7b) would serve as an important target of the field study. Indeed, we can see that our results support the empirical tests of dispersal in host–parasite system. As discussed above, strong virulence affecting competitive ability, *d*, promotes I-biased dispersal, and this pattern of dispersal bias is observed in yellow-bellied marmot (*Marmota flaviventris*) (van Vuren 1996). Indeed, this marmot is infected with several ectoparasites, including mites. Mites are generally considered to have strong negative impacts on the hosts, because they are capable of horizontal transmission (Eward 1983; Anderson & May 1982; Clayton & Tompkins 1995; Frank 1996). Hence, our model predicts that the marmot shows I-biased dispersal because of the strong virulence by mites. This might also lead to an understanding of the spatial spread of infectious disease and host–parasite geographic range (Duncan *et al.* 2011; Fellous *et al.* 2012). Our model predicts the possibility that if parasites' spatial spread or migration is selected for, then high virulence are favoured because it induces the high level of dispersal by their infected host.

4.3 Parasite release γ and selection against emigrants ε

As stated in section 4.1, dispersal costs may strongly depend on parasite release rate *γ*, which is reported in endangered parasitic species of freshwater pearl mussel (e.g. Morales *et al.* 2006; Akiyama & Iwakuma 2009; Terui *et al.* 2013). The species have a completely host-dependent dispersal during their early life stages: larvae (Glochidia) attach to fish gills (Meyers & Millemann, 1977; Schwalb *et al.*, 2011) and they leave

from their host during and/or after host dispersal to the upstream (Terui *et al.* 2013). It is also reported that they harm their hosts during their parasitic stages (Thomas *et al.* 2013). Indeed, their population structure is strongly affected by their hosts' migration tendency, depending on which host (trout or salmon) to use (Karlsson *et al.* 2013). In the system of freshwater pearl mussels and fish species, host's dispersal bias toward susceptible or infected individuals has not been reported to our knowledge. However, our theoretical results suggest that host fish may have evolved I-biased dispersal in their life stages, because parasite release during dispersal is obligate. Hence our model may be able to predict host's migration tendency (disperse toward upstream or stay downstream), which leads to better understanding of the life history of these endangered mussels.

Also, we considered selection against emigrants, ε . For socially group-forming animals, 'social barriers to pathogen' is hypothyzed (Loehle 1995), which postulate that philopatric individuals or group members reject immigrants to reduce the risk of infection between groups. Our model incorporates such social barrier effect, which is here ε, to predict S-biased dispersal. Estimating whether such barriers act toward 'infected' emigrants or not, and wehether biased dispersal occurs or not is a future challenging.

4.4 Host genetic structure shaped by gene flow, and coevolutionary story

In this study, we showed that host gene flow, and thus the parasite gene flow, can be influenced by parasite traits. Hence, we conjecture that the geographic variation of the host–parasite coevolutionary process is strongly affected by the host adaptive migration, depending on whether it is susceptible or infected. Because dispersal determines gene flow across space, S-biased dispersal and I-biased dispersal result in different outcomes of spatially structure of host–parasite populations, which is called 'geographic mosaic theory' (Thompson 2005). Combining our analysis with this theory, we can conclude that if parasitic effects, such as infection rate or parasite virulence, vary spatially among

populations, then the level of gene flow determined by adaptive host migration can drastically modify host–parasite spatial genetic structure.

4.5 concluding remarks: problems and future works

In this manuscript, we have proposed simple assumptions that a population is homogeneous with respect to local infection rate, R; virulence, d; selection against infected emigrants, ε, and parasite release rate, r, although several papers have reported that there is a spatial variation in host–parasite interaction (e.g. Altizer *et al.* 2000). Further consideration of spatially structured population dynamics (e.g. lattice model, Boots & Sasaki 2001), genetically explicit dynamics (e.g. gene-for-gene model, Flor 1971; Gandon *et al.* 1996, Sasaki 2000; matching-alleles model, Seger 1988), or their combination (e.g. Frank 1991) is required. Also, as one of fundamental characteristics of host–parasite interactions, horizontal and/or vertical transmission dynamics (Anderson & May 1979) needs to be considered. In addition, our results may lead to predict the avoidance of infecting relatives, which is of great interest in the theory of evolution of dispersal from altruistic aspects in spatially structured populations (Rousset 2004; Lehmann *et al*. 2006).

Appendix

For simplicity, we drop the asterisk (*) for symbols at equilibrium throughout the Appendix.

(A) Evolutionary isoclines: L_S and L_I

We derive the evolutionary isoclines by differentiation using the direct fitness approach (Taylor & Frank 1996). Remember that g_S (or g_I) is the breeding value of x_S (or x_I, respectively), i.e. $x_S = g_S + e_S$ and $x_I = g_I + e_I$, where e–terms are errors, and that $F_{\mathrm{ST}}^{\mathrm{R}}$ denotes the slope of neighbour-mean dispersal rate y_S (or y_I) on the focal breeding value g_S (or g_I, respectively). The total derivative of w with respect to g_S evaluated at $x_i = y_i = z_i$ for $i = S, I$ yields

$$\frac{\mathrm{d}w}{\mathrm{d}g_S} = \frac{\mathrm{d}x_S}{\mathrm{d}g_S}\frac{\partial w}{\partial x_S}\bigg|_{(z_S, z_I)} + \frac{\mathrm{d}y_S}{\mathrm{d}g_S}\frac{\partial w}{\partial y_S}\bigg|_{(z_S, z_I)}$$

$$= \frac{(1-R_{\mathrm{E}})F_{\mathrm{ST}}^{\mathrm{R}}\{(1-R_{\mathrm{E}})(1-z_S) + R_{\mathrm{E}}(1-z_I)\}}{\left\{(1-R_{\mathrm{E}})(1-c_S z_S) + R_{\mathrm{E}}(1-c_I z_I)\right\}^2} + \frac{-(1-R_{\mathrm{E}})c_S}{(1-R_{\mathrm{E}})(1-c_S z_S) + R_{\mathrm{E}}(1-c_I z_I)}$$

$$= (1-R_{\mathrm{E}})\left\{\frac{F_{\mathrm{ST}}^{\mathrm{R}}(1-u)}{(1-u+v)^2} + \frac{-c_S}{(1-u+v)}\right\} \qquad \text{(A-1)}$$

Here we introduce the cost of dispersal: $c_S = 1 - p_S$ and $c_I = 1 - p_I(1 + \frac{\gamma d}{1-d})$, hereafter. Setting the derivative 0 and multiplying both sides by the positive term $(1-u+v)^2$, we have the equation of L_S in Eq. (4a). In a similar manner, we have L_I in Eq. (4b).

(B) Computation for relatedness coefficient

Previous studies have derived the stationary value of relatedness $F_{\mathrm{ST}}^{\mathrm{R}}$ from a recurrence equation (e.g. Taylor 1988; Gandon & Michalakis 1999; Rousset 2004; Ohtsuki 2010). We adopt the same method as theirs, in the following steps. First, we can write the backward migration rate as

$$m = \frac{v}{1-u+v}, \text{ (B-1)}$$

where u (and v) is the expected emigration rate (and immigration rate, respectively) defined as

$$u = (1-R_{\mathrm{E}})z_S + R_{\mathrm{E}}z_I, \text{ (B-2)}$$

$$v = (1-R_{\mathrm{E}})(1-c_S)z_S + R_{\mathrm{E}}(1-c_I)z_I. \text{ (B-3)}$$

Second, let $F_{\mathrm{ST}}^{\mathrm{R}}$ be the probability that 2 individuals sampled with replacement within a focal deme have a common ancestor within the deme (and this definition gives the coefficient of relatedness). It is well known that, under the assumption of asexual haploidy, this value obeys the following recursion at a particular time t :

$$F_{\mathrm{ST}}^{\mathrm{R}}[t+1] = \frac{1}{N} + \frac{N-1}{N}(1-m)^2 F_{\mathrm{ST}}^{\mathrm{R}}[t] \quad \text{(B-4)}.$$

Here, the first term $1/N$ is the probability that the same individual is sampled twice (and this can occur when sampling is done with replacement). The second has three components; $(N-1)/N$ is the probability that different 2 individual is sampled, $(1-m)^2$ is the probability that 2 different individuals are both philopatric, and $F_{\mathrm{ST}}^{\mathrm{R}}[t]$ is the probability that such sampled individual(s) comes or come from the focal deme. Then, solving $F_{\mathrm{ST}}^{\mathrm{R}}[t+1] = F_{\mathrm{ST}}^{\mathrm{R}}[t]$, we have the following stationary value

$$F_{\mathrm{ST}}^{\mathrm{R}} = \frac{1}{N-(N-1)(1-m)^2}. \quad \text{(B-5)}$$

Although in this form m is implicit (which is determined by ES-(z_S, z_I)), we derive its explicit form after calculating (z_S, z_I).

(C) Computation for F_{ST}

Wright (1943) derived F_{ST} under diffusion approximation in the infinite islands model, but we can derive the same result (Rousset 2004, Ohtsuki 2010). Let m be the backward migration rate defined in Eq. (5b). Then, because $(1-m)^2$ is the probability that randomly chosen 2 individuals without replacement both come from the parents that are

native to the deme, the probability of IBD (denoted by Q) follows the recurrence equation (Malécot 1970):

$$Q[t+1]=\frac{1}{N}(1-m)^2+\frac{N-1}{N}(1-m)^2Q[t]. \quad \text{(C-1)}$$

Setting $Q=Q'=Q^*$, we have the following equilibrium condition

$$F_{\mathrm{ST}}=\frac{(1-m)^2}{N-(N-1)(1-m)^2}=(1-m)^2F_{\mathrm{ST}}^{\mathrm{R}}, \quad \text{(C-2)}$$

since $F_{\mathrm{ST}}=Q^*$ in the infinite island model (Rousset 2004). We investigate explicit form of F_{ST} after calculating that of $F_{\mathrm{ST}}^{\mathrm{R}}$, and thus after calculating ES-(z_S,z_I) in Appendix E.

(D) ESS located on the boundary

The definition of L_S and L_I are given as

$$L_S: F_{\mathrm{ST}}^{\mathrm{R}}(1-u)=c_S(1-u+v), \quad \text{(D-1)}$$

$$L_S: F_{\mathrm{ST}}^{\mathrm{R}}(1-u)=c_I(1-u+v), \quad \text{(D-2)}$$

where, remember that

$$c_S:=1-p_S$$
$$c_I:=1-p_S(1-\varepsilon)(1+\frac{\gamma d}{1-d}) \quad \text{. (D-3)}$$

Suppose that Eqs. (D-3) hold simultaneously; then, we subtract both sides to obtain

$$(c_S-c_I)(1-u+v)=0. \quad \text{(D-4)}$$

For generic parameters with $c_S\neq c_I$, we necessitate $1-u+v=0$. Because $1-u\geq 0$ and $v\geq 0$ hold true, we conclude $v=0$ and $1-u=0$. Here, note that $u=1$ implies $z_S=z_I=1$, since $0<R_E<1$. Substituting $z_S=z_I=1$ into $v=(1-R_{\mathrm{E}})(1-c_S)z_S+R_{\mathrm{E}}(1-c_I)z_I$, we can immediately see that $v=(1-R_{\mathrm{E}})(1-c_S)+R_{\mathrm{E}}(1-c_I)\neq 0$; this is contradictory. Hence, we have proven that L_S and L_I never intersect each other on the rectangle, and ES (z_S,z_I) does not lie in the interior of the unit rectangle.

(E) The explicit solution of ESS

To compute ESS, we need some steps. First, we derive backward migration rate m in terms of costs and relatedness. Suppose that ES-(z_S, z_I) lies on L_S. Then the equation

$L_S : F_{\mathrm{ST}}^{\mathrm{R}}(1-u) = c_S(1-u+v)$ (E-1)

needs to be satisfied. Dividing both sides by the positive term $(1-u+v)$ yields

$L_S : F_{\mathrm{ST}}^{\mathrm{R}}(1-m) = c_S$. (E-2)

In case $c_S > c_I$ (or $c_S < c_I$), the inequality $dw/dg_I \propto F_{\mathrm{ST}}^{\mathrm{R}}(1-m) - c_I > 0$ (or $dw/dg_I < 0$) always holds so that high (or low) z_I is favoured, which causes I-biased (or S-biased, respectively) dispersal. In a similar manner, when (z_S, z_I) lies on L_I, we have

$L_I : F_{\mathrm{ST}}^{\mathrm{R}}(1-m) = c_I$. (E-3)

Here, suppose that ES-(z_S, z_I) does not lie on neither L_S or L_I. In case of I-biased (or S-biased) dispersal, the inequaities $F_{\mathrm{ST}}^{\mathrm{R}}(1-m) - c_S > 0$ and $F_{\mathrm{ST}}^{\mathrm{R}}(1-m) - c_I < 0$ (or $F_{\mathrm{ST}}^{\mathrm{R}}(1-m) - c_S < 0$ and $F_{\mathrm{ST}}(1-m) - c_I > 0$, respectively) can hold simultaneously so that ES-dispersal rate is $(z_S, z_I) = (0,1)$ (or $(z_S, z_I) = (1,0)$, respectively). In such cases, we can calculate m to obtain

$$(z_S, z_I) = (1,0) \Rightarrow m = \frac{(1-R_{\mathrm{E}})(1-c_S)}{1-(1-R_{\mathrm{E}})c_S}, \text{ (E-4)}$$

$$(z_S, z_I) = (0,1) \Rightarrow m = \frac{R_{\mathrm{E}}(1-c_I)}{1-R_{\mathrm{E}}c_I}. \text{ (E-5)}$$

Second, we need 2 case analyses depending on the sign of $\frac{\gamma d}{1-d} - \frac{\varepsilon}{1-\varepsilon}$.

(E-i) ***When*** $\frac{\gamma d}{1-d} > \frac{\varepsilon}{1-\varepsilon}$ ***holds***

We can depict L_S and L_I on the unit rectangle, and partition it into 3 regions. Here are examples as shown in Figure 2 (i). On the left region, the selective pressure acts on both z_S and z_I towards large values (i.e., dw/dg_S > 0 and dw/dg_I > 0), and *vice versa* on the right region. On the central region between L_S and L_I, large z_I and small z_S are favoured: dw/dg_S < 0 and dw/dg_I > 0. Depicting the vector fields and substituting either z_I=1 or

z_S=0 into L_S, L_I, Eq. (5) and Eq. (6) yields the following explicit forms of (z_S, z_I) and $F_{\mathrm{ST}}^{\mathrm{R}}$:

$$z_S = \begin{cases} \dfrac{r_S - c_S - R_{\mathrm{E}}(r_S - c_S c_I)}{(1-R_{\mathrm{E}})(r_S - c_S^2)} & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_S - c_S}{r_S - c_S c_I} \\ 0 & \text{if} \quad \dfrac{r_S - c_S}{r_S - c_S c_I} < R_{\mathrm{E}} < 1 \end{cases}, \tag{E-6}$$

$$z_I = \begin{cases} 1 & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_I - c_I}{r_I - c_I^2} \\ \dfrac{r_I - c_I}{R_{\mathrm{E}}(r_I - c_I^2)} & \text{if} \quad \dfrac{r_I - c_I}{r_I - c_I^2} < R_{\mathrm{E}} < 1 \end{cases}, \tag{E-7}$$

and

$$F_{\mathrm{ST}}^{\mathrm{R}} = \begin{cases} r_S & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_S - c_S}{r_S - c_S c_I} \\ \dfrac{(1-c_I R_{\mathrm{E}})^2}{N(1-c_I R_{\mathrm{E}})^2 - (N-1)(1-R_{\mathrm{E}})^2} & \text{if} \quad \dfrac{r_S - c_S}{r_S - c_S c_I} < R_{\mathrm{E}} < \dfrac{r_I - c_I}{r_I - c_I^2} \\ r_I & \text{if} \quad \dfrac{r_I - c_I}{r_I - c_I^2} < R_{\mathrm{E}} < 1 \end{cases}, \tag{E-8}$$

where

$$r_S := \frac{1 + \sqrt{1 + 4N(N-1)c_S^2}}{2N} \tag{E-9}$$

and

$$r_I := \frac{1 + \sqrt{1 + 4N(N-1)c_I^2}}{2N}. \tag{E-10}$$

Note that c_I can be negative. Especially in this case, these results are greatly simplified as follows:

$$z_S = \begin{cases} \dfrac{(r_S - c_S) - R_{\mathrm{E}} c_S (1 - c_I)}{(1 - R_{\mathrm{E}})(r_S - c_S^2)} & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_S - c_S}{r_S - c_S c_I} \\ 0 & \text{if} \quad \dfrac{r_S - c_S}{r_S - c_S c_I} < R_{\mathrm{E}} < 1 \end{cases}, \quad \text{(E-11)}$$

while the ES-dispersal rate for infected is always unity (i.e. $z_I \equiv 1$). Relatedness coefficient $F_{\mathrm{ST}}^{\mathrm{R}}$ is given as

$$F_{\mathrm{ST}}^{\mathrm{R}} = \begin{cases} r_S & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_S - c_S}{r_S - c_S c_I} \\ \dfrac{(1 - c_I R_{\mathrm{E}})^2}{N(1 - c_I R_{\mathrm{E}})^2 - (N-1)(1 - R_{\mathrm{E}})^2} & \text{if} \quad \dfrac{r_S - c_S}{r_S - c_S c_I} < R_{\mathrm{E}} < 1 \end{cases}. \quad \text{(E-12)}$$

Thus there are only two phases (compare with the case of three phases; see Figure 3).

(E-ii) ***When*** $\dfrac{\gamma d}{1-d} < \dfrac{\varepsilon}{1-\varepsilon}$ ***holds***

Similar analysis yields the solution:

$$z_S = \begin{cases} \dfrac{1}{1 - R_{\mathrm{E}}} \dfrac{r_S - c_S}{r_S - c_S^2} & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{c_S (1 - c_S)}{r_S - c_S^2} \\ 1 & \text{if} \quad \dfrac{c_S (1 - c_S)}{r_S - c_S^2} < R_{\mathrm{E}} < 1 \end{cases}, \quad \text{(E-13)}$$

$$z_I = \begin{cases} 0 & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{(1 - c_S) c_I}{r_I - c_S c_I} \\ \dfrac{r_I - c_I - (1 - R_{\mathrm{E}})(r_I - c_S c_I)}{R_{\mathrm{E}}(r_I - c_I^2)} & \text{if} \quad \dfrac{(1 - c_S) c_I}{r_I - c_S c_I} < R_{\mathrm{E}} < 1 \end{cases}, \quad \text{(E-14)}$$

and

$$F_{\mathrm{ST}}^{\mathrm{R}}=\begin{cases} r_S & \text{if} \quad 0<R_{\mathrm{E}}<\dfrac{c_S(1-c_S)}{r_S-c_S^2} \\ \dfrac{(1-c_S+c_SR_{\mathrm{E}})^2}{N(1-c_S+c_SR_{\mathrm{E}})^2-(N-1)R_{\mathrm{E}}^2} & \text{if} \quad \dfrac{c_S(1-c_S)}{r_S-c_S^2}<R_{\mathrm{E}}<\dfrac{c_I(1-c_S)}{r_I-c_Sc_I} \\ r_I & \text{if} \quad \dfrac{c_I(1-c_S)}{r_I-c_Sc_I}<R_{\mathrm{E}}<1 \end{cases} . \quad \text{(E-15)}$$

We have found a singular strategy on the boundary of phenotypic space for $0\le z_S\le 1$ and $0\le z_I\le 1$, and we still need to investigate whether it is really ESS. To do so, we calculate the second derivatives of fitness $W(x_S,x_I)$ with respect to small deviations (δ_S,δ_I) from resident dispersal rates (z_S,z_I), i.e., we compute the Hessian matrix of W with respect to (δ_S,δ_I). This computation is somehow tedious; to avoid carrying it out directly, we transform mutant (or resident) dispersal rates (x_S,x_I) (or (z_S,z_I), respectively) to emigration and immigration rate, $(u^\bullet,v^\bullet)$ (or (u,v), respectively), defined in Eq. (B-2, 3). After some arithemtics, we have

$$W(u^\bullet,v^\bullet):=w(x_S,x_I)=\frac{1-u^\bullet}{1-F_{\mathrm{ST}}^{\mathrm{R}}u^\bullet-(1-F_{\mathrm{ST}}^{\mathrm{R}})u+v}+\frac{v^\bullet}{1-u+v}, \quad \text{(E-16)}$$

where we changed the notation of fitness for $w(x_S,x_I)$ to $W(u^\bullet,v^\bullet)$ to distinguish the variable dependence. Note that we explicitly write the neighbour-mean strategy (the denominator in the first fitness component) as $(1-R_{\mathrm{E}})y_S+R_{\mathrm{E}}y_I=F_{\mathrm{ST}}^{\mathrm{R}}u^\bullet+(1-F_{\mathrm{ST}}^{\mathrm{R}})u$ Additionally, we write the small deviation of the mutant from the resident as

$$\begin{aligned} \delta_u &:= u^\bullet-u=(1-R_{\mathrm{E}})\delta_S+R_{\mathrm{E}}\delta_I \\ \delta_v &:= v^\bullet-v=(1-R_{\mathrm{E}})(1-c_S)\delta_S+R_{\mathrm{E}}(1-c_I)\delta_I \end{aligned} \quad \text{(E-17)}$$

with the following Jacobian matrix

$$J:=\frac{\partial(\delta_u,\delta_v)}{\partial(\delta_S,\delta_I)}=\begin{pmatrix} 1-R_{\mathrm{E}} & R_{\mathrm{E}} \\ (1-R_{\mathrm{E}})(1-c_S) & R_{\mathrm{E}}(1-c_I) \end{pmatrix}. \quad \text{(E-18)}$$

For generic parameters, this determinant is not null; in particular, $\det J>0$ implies

I-biased dispersal to evolve whereas $\det J < 0$ implies S-biased dispersal. Then W can be rewritten as

$$W(u+\delta_u, v+\delta_v) = \frac{1-u-\delta_u}{1-F_{\mathrm{ST}}^{\mathrm{R}}\delta_u - u + v} + \frac{v+\delta_v}{1-u+v}. \quad \text{(E-19)}$$

On this setup, we have the first partial derivatives of W

$$\begin{pmatrix} \frac{\partial W}{\partial \delta_u} \\ \frac{\partial W}{\partial \delta_v} \end{pmatrix} = \begin{pmatrix} \frac{F_{\mathrm{ST}}^{\mathrm{R}}(1-u)-(1-u+v)}{(1-F_{\mathrm{ST}}^{\mathrm{R}}\delta_u - u + v)^2} \\ \frac{1}{1-u+v} \end{pmatrix} \quad \text{(E-20)}$$

and the second derivatives

$$\begin{aligned} \frac{\partial^2 W}{\partial \delta_u^2} &= \frac{2F_{\mathrm{ST}}^{\mathrm{R}}\{F_{\mathrm{ST}}^{\mathrm{R}}(1-u)-(1-u+v)\}}{(1-F_{\mathrm{ST}}^{\mathrm{R}}\delta_u - u + v)^3} \\ \frac{\partial^2 W}{\partial \delta_u \partial \delta_v} &= 0 \qquad \text{(E-21)} \\ \frac{\partial^2 W}{\partial \delta_v^2} &= 0 \end{aligned}$$

for any small deviations (δ_u, δ_v). Thus we obtain the Hessian matrix of W as

$$H[W(u+\delta_u, v+\delta_v)] = \begin{pmatrix} \frac{2F_{\mathrm{ST}}^{\mathrm{R}}\{F_{\mathrm{ST}}^{\mathrm{R}}(1-u)-(1-u+v)\}}{(1-F_{\mathrm{ST}}^{\mathrm{R}}\delta_u - u + v)^3} & 0 \\ 0 & 0 \end{pmatrix}. \quad \text{(E-22)}$$

Obviously, $\det H[W(u+\delta_u, v+\delta_v)] \equiv 0$ (i.e. $\det H[W]$ is always null irrespectively of (δ_u, δ_v)). By the chain-rule,

$$H[w(z_S+\delta_S, z_I+\delta_I)] = J^{\mathrm{T}} H[W(u+\delta_u, v+\delta_v)] J \quad \text{(E-23)}$$

holds, where the superscript $^{\mathrm{T}}$ denotes transpose, and we can see that $\det H[w]$ is also always null, because

$$\det H[w] = (\det J)^2 \det H[W]. \quad \text{(E-24)}$$

Thus we call this situation as ‘weak ESS’ such that the singular strategy is surely convergence stable, but is not a branching point (Lundberg 2013). This means that the singular strategy (z_S, z_I) is optimal when invading a resident population. After the

occupation of the system by (z_S, z_I), any rare mutant can invade, but the mutant may not increase in its frequency in the population. This 'neutral stability' occurs in Fisher's sex ratio game (Maynard Smith 1989) or in the evolution of partial migration (Lundberg 2013). For the same reason, the approach to analyze disruptive or stabilizing selection in structured population, which is developed by Chesson (1984), Metz & Gyllenberg (2001) and Ajar (2003), does not work here to investigate evolutionary stability, although it provides a powerful method for analyzing disruptive selection in subdivided populations.

(F) Evolution of states-dependent dispersal in more than 2 classes.

We divided the juvenile offspring into 2 classes (i.e. susceptible state and infected state), but we are also interested in whether it is possible to apply our methods to the case where there are more than 2 infection states. As a result, when there are more than two states, we find it is impossible to investigate evolutionary nor convergence stability of state-dependent dispersal rates using our present method. To demonstrate this, we introduce three-states population as an example. First, let us denote (R_S, R_I; 0, d) the situation that there are only 2 infection states like in our present model, where '0' indicates no virulence on susceptible individual, d indicates virulence on infected individual, R_I is infection rate, and R_S=1–R_I; we used the subscripts S and I to clearly distinguish the states. Here, remember that effective infection rate R_E is calculated by the following scaling

$$R_\mathrm{E} = \frac{(1-d)R_I}{1-dR_I}. \quad \text{(F-1)}$$

Effective infection rate measures the extent to 'how many individuals are infected after parasite-induced mortality event'. Suppose the state-dependent dispersal rate (of the focal individual), (x_S, x_I). Using this factor R_E, we can define the following linear transformation

$$\begin{pmatrix} u \\ v \end{pmatrix} = \begin{pmatrix} (1-R_{\mathrm{E}}) & R_{\mathrm{E}} \\ (1-R_{\mathrm{E}})(1-c_S) & R_{\mathrm{E}}(1-c_I) \end{pmatrix} \begin{pmatrix} x_S \\ x_I \end{pmatrix}, \quad \text{(F-2)}$$

where u is the expected emigration rate and v is the expected immigration rate.

Second, let us consider the following three states: (R_S, R_L, R_H ; 0, d_L, d_H) with state-dependent dispersal rate (x_S, x_L, x_H) , where $R_S + R_L + R_H = 1$ and $0 < d_L < d_H < 1$; the subscript L (or H) is used to indicate the state of 'lightly infected' individual (or 'heavily infected' individual, respectively). Note that the virulence on L-individual (or H-individual) is d_L (or d_H, respectively). Suppose that they suffer different dispersal costs, c_S, c_L and c_I. The effective value of infection can be written as

$$\begin{aligned} R_{L;\mathrm{E}} &\coloneqq \frac{(1-d_L)R_L}{R_S + (1-d_L)R_L + (1-d_H)R_H} \\ R_{H;\mathrm{E}} &\coloneqq \frac{(1-d_H)R_H}{R_S + (1-d_L)R_L + (1-d_H)R_H} \\ R_{L;\mathrm{E}} &\coloneqq 1 - R_{L;\mathrm{E}} - R_{H;\mathrm{E}} \end{aligned}, \quad \text{(F-3)}$$

analogously to the case of two-states. Then, we can write the fitness function $w = w_{\mathrm{HOME}} + w_{\mathrm{AWAY}}$ as

$$w_{\mathrm{HOME}} = \frac{R_{S;\mathrm{E}}(1-x_S) + R_{L;\mathrm{E}}(1-x_L) + R_{H;\mathrm{E}}(1-x_H)}{R_{S;\mathrm{E}}(1-y_S+(1-c_S)z_S) + R_{L;\mathrm{E}}(1-y_L+(1-c_L)z_L) + R_{H;\mathrm{E}}(1-y_H+(1-c_H)z_H)}$$

(F-4)

and

$$w_{\mathrm{AWAY}} = \frac{R_{S;\mathrm{E}}(1-c_S)x_S + R_{L;\mathrm{E}}(1-c_L)x_L + R_{H;\mathrm{E}}(1-c_H)x_H}{R_{S;\mathrm{E}}(1-c_S z_S) + R_{L;\mathrm{E}}(1-c_L z_L) + R_{H;\mathrm{E}}(1-c_H z_H)}, \quad \text{(F-5)}$$

where y-terms indicate neighbour-meand dispersal rates for each state, and z-terms indicate resident dispersal rates for each state. Here we introduce the following linear transformation

$$\begin{pmatrix} u^{\bullet} \\ v^{\bullet} \end{pmatrix} = \begin{pmatrix} R_{S;\mathrm{E}} & R_{L;\mathrm{E}} & R_{H;\mathrm{E}} \\ R_{S;\mathrm{E}}(1-c_S) & R_{L;\mathrm{E}}(1-c_L) & R_{H;\mathrm{E}}(1-c_H) \end{pmatrix} \begin{pmatrix} x_S \\ x_L \\ x_H \end{pmatrix}, \quad \text{(F-6)}$$

$$\begin{pmatrix} u^{0\mathrm{R}} \\ v^{0\mathrm{R}} \end{pmatrix} = \begin{pmatrix} R_{S;\mathrm{E}} & R_{L;\mathrm{E}} & R_{H;\mathrm{E}} \\ R_{S;\mathrm{E}}(1-c_S) & R_{L;\mathrm{E}}(1-c_L) & R_{H;\mathrm{E}}(1-c_H) \end{pmatrix} \begin{pmatrix} y_S \\ y_L \\ y_H \end{pmatrix}, \text{(F-7)}$$

and

$$\begin{pmatrix} u \\ v \end{pmatrix} = \begin{pmatrix} R_{S;\mathrm{E}} & R_{L;\mathrm{E}} & R_{H;\mathrm{E}} \\ R_{S;\mathrm{E}}(1-c_S) & R_{L;\mathrm{E}}(1-c_L) & R_{H;\mathrm{E}}(1-c_H) \end{pmatrix} \begin{pmatrix} z_S \\ z_L \\ z_H \end{pmatrix}. \text{(F-8)}$$

It is apparent that this linear map has rank 2, i.e. the dimension of the image of this lenear map is 2. Then fitness *w* can be rewritten as

$$w(x_S, x_L, x_H) = W(u^\bullet, v^\bullet) = \frac{1-u^\bullet}{1-u^{0\mathrm{R}}+v} + \frac{v^\bullet}{1-u+v}, \text{(F-9)}$$

where we use *W* instead of *w* to explicitly distinguish the variable dependence; hence we can see that *W* is characterized by just two variables *u* and *v*, and that the evolutionary stability of (*u*, *v*) corresponds the evolutionary stability of (x_S, x_L, x_H). In other words,

$$\begin{pmatrix} \dfrac{\partial W}{\partial u^\bullet} \\ \dfrac{\partial W}{\partial v^\bullet} \end{pmatrix} = \begin{pmatrix} R_{S;\mathrm{E}} & R_{L;\mathrm{E}} & R_{H;\mathrm{E}} \\ R_{S;\mathrm{E}}(1-c_S) & R_{L;\mathrm{E}}(1-c_L) & R_{H;\mathrm{E}}(1-c_H) \end{pmatrix} \begin{pmatrix} \dfrac{\partial w}{\partial x_S} \\ \dfrac{\partial w}{\partial x_H} \\ \dfrac{\partial w}{\partial x_H} \end{pmatrix}. \text{(F-10)}$$

However, even if we solve the evolutionary satbility condition of (*u*, *v*) on LHS, we have just two equations on (*u*, *v*) so that immediately we find it impossible to solve it with respect to (x_S, x_L, x_H) and we find the system neutrally stable. In a general *n*-states cases for $n \geq 3$, we can conclude that our model is not capable of searching for a singular strategy.

Our result here is, surely, not interesting mathematically, but it states that, when considering 3-(or more) states-dependent dispersal evolution, we need to take into account more specific scenario(s); here in our model we assume 2 fitness components enjoyed by either philopatric or migrant offspring, and thus we can characterize fitness

w just by two migrant variables (u and v). If natural selection acting on dispersal emerges not only during dispersal, then we may incorporate more than two states; we leave this problem as a future study.

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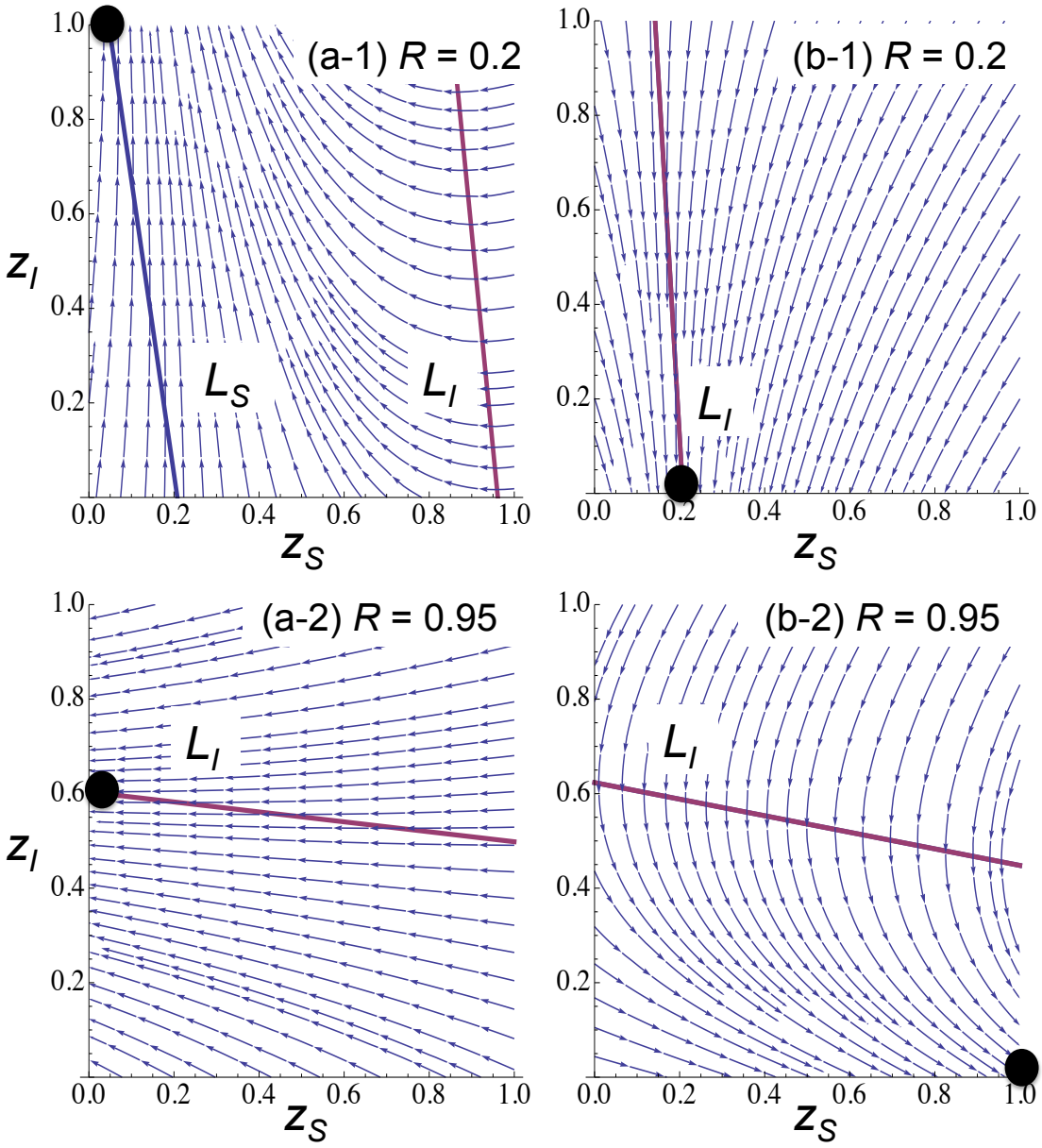

Figure 1

Evolutionary vector fields of dispersal rate (z_s, z_l). Two lines indicate L_s and L_l in each of (a) and (b). The ESS indicated by a solid circle is located on the boundary. Parameters: (a) $\varepsilon = 0.1$, $d = 0.6$, $\gamma = 0.5$, $p_s = 0.5$, and $N = 5$; $\gamma d/(1-d) > \varepsilon/(1-\varepsilon)$ holds true. Infection rate is set as R=0.2 in (a-1), and R=0.95 in (a-2). (b) $\varepsilon = 0.5$, $d = 0.6$, $\gamma = 0.1$, $p_s = 0.5$, and $N = 5$; $\gamma d/(1-d) < \varepsilon/(1-\varepsilon)$ holds true. Infection rate is set as R=0.2 in (b-1), and R=0.95 in (b-2).

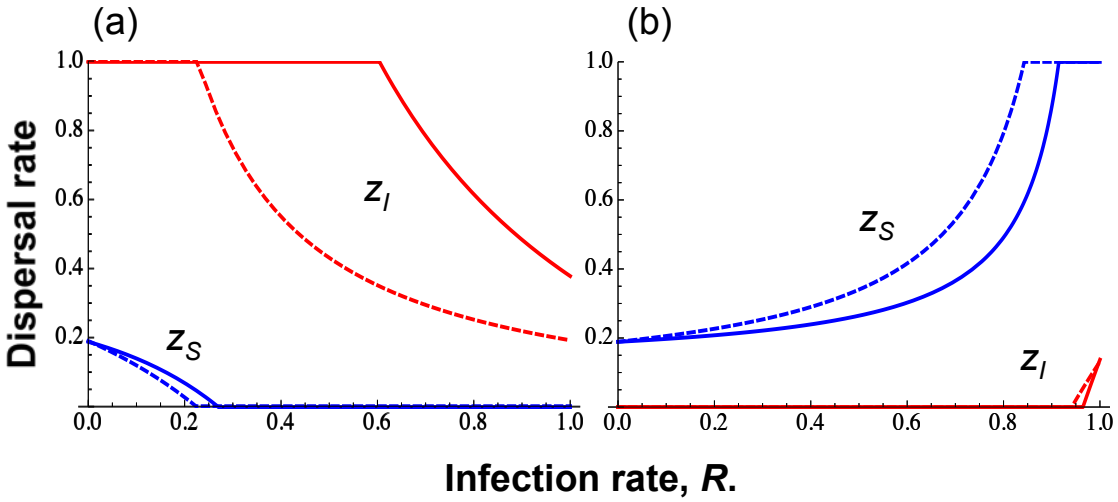

Figure 2

The ES-dispersal rate (z_S, z_I), plotted against infection rate, R. (a) I-biased dispersal. Parameter values: $\varepsilon = 0.1$, $\gamma = 0.5$, $p_S = 0.5$, and $N = 5$, for $d = 0.6$ (solid) and $d = 0.2$ (dashed) ; $\gamma d/(1-d) > \varepsilon/(1-\varepsilon)$ holds true. (b) S-biased dispersal. Parameter values are: $\varepsilon = 0.5$, $\gamma = 0.1$, $p_S = 0.5$, and $N = 5$, for $d = 0.6$ (solid) and d = 0.2 (dashed); $\gamma d/(1-d) < \varepsilon/(1-\varepsilon)$ holds true. The sign of $\gamma d/(1-d)-\varepsilon/(1-\varepsilon)$ changes from negative to positive value as d increases. Large d can facilitate I-biased dispersal, whereas small d can facilitate S-biased dispersal. In case of (a) $\gamma d/(1-d) > \varepsilon/(1-\varepsilon)$, ES-dispersal rate for either susceptible or infected individuals is non-increasing with R, whereas, in case of (b) $\gamma d/(1-d) < \varepsilon/(1-\varepsilon)$, we can see the trend which is opposite to that of (a).

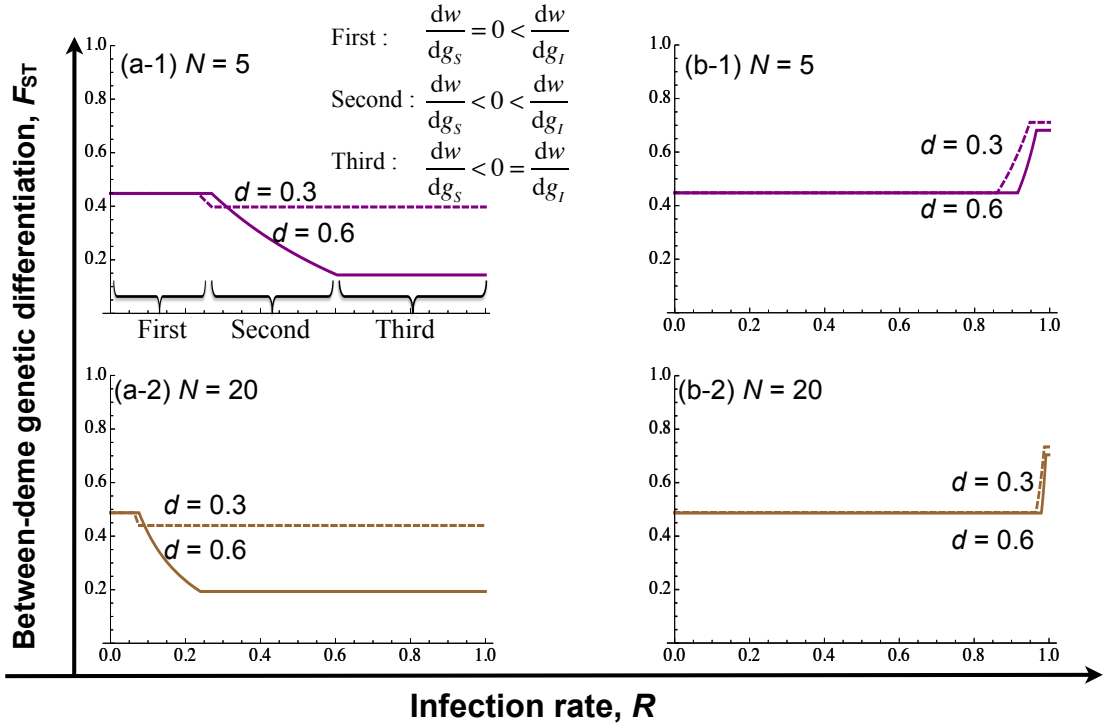

Figure 3

Between-deme genetic differentiation, F_{ST}. Horizontal axis is for the infection rate, R. Parameters are (i) $\varepsilon = 0.1$, $\gamma = 0.5$, and $p_S = 0.5$, for $N = 5$ (a-1) or 20 (a-2), and for $d =$ 0.6 (solid) or 0.3 (dashed); $\gamma d/(1-d) > \varepsilon/(1-\varepsilon)$ holds true. In each case, F_{ST} is decreased with increased R, increased d, and decreased N. In (a-1), "First (Second, or Third)" indicates the phase, in which $dw/dg_S = 0 < dw/dg_I$ ($dw/dg_S < 0 < dw/dg_I$, or $dw/dg_S < 0 = dw/dg_I$, respectively) holds. Similar phases can be seen in other panels. (b) $\varepsilon = 0.5$, $\gamma = 0.1$, and $p_S = 0.5$, for $N = 5$ (b-1) or 20 (b-2), and for $d = 0.6$ (solid) and 0.3 (dashed); $\gamma d/(1-d) < \varepsilon/(1-\varepsilon)$ holds true. An oppostite trend is observed compared to (a).

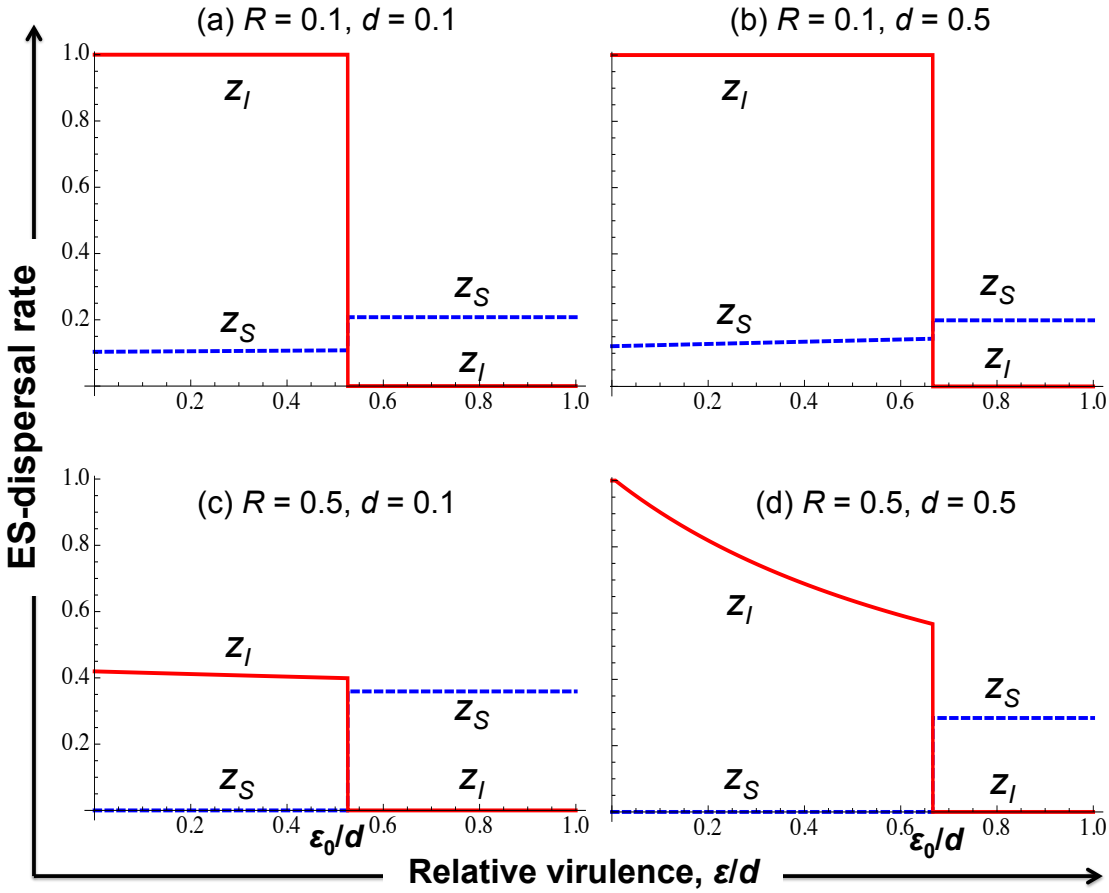

Figure 4

The dependence of ES-dispersal rates on relative virulence, (ε/d) with d fixed. (either d = 0.1 or 0.5) Parameter values : p = 0.5, γ = 0.5, and N = 5. The sign of $\gamma d/(1-d)-\varepsilon/(1-\varepsilon)$ changes at an intermediate value ε_0/d (= $\gamma/(1-d+d\gamma)$), so that $\varepsilon/d < \varepsilon_0/d$ indicates I-biased dispersal, whereas $\varepsilon/d > \varepsilon_0/d$ indicates S-biased dispersal.

Chapter 2.

How parasite-mediated costs drive the evolution of disease state-dependent dispersal.

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1. Introduction

Dispersal, defined as any movement of individuals and/or propagules causing gene flow across space, is central to evolutionary ecology and population biology (Ronce 2007). Dispersal affects various ecological aspects, including interspecific interactions such as host–parasite systems (Clobert 2001; Chaianunporn and Hovestadt (2012). Knowledge of dispersal tendency directly leads to an understanding of species distribution, population genetic structure, and biodiversity. This topic has been intensively studied both theoretically and empirically. Dispersal not only influences evolutionary and/or ecological conditions, but it also affects various selective pressures. For instance, conventional wisdom holds that the interplay between the benefits and costs due to kin competition, spatio-temporal fluctuations in the environment, and inbreeding avoidance drive the evolution of dispersal (Hamilton & May 1977; McPeek & Holt 1992; Gandon 1999; Gandon & Michalakis 1999). Initiation, travel, and settlement are three processes associated with dispersal; therefore, cost payment can take place before, during, and after dispersal (Bonte *et al*. 2012). For example, dispersers are subject to natural selection at each stage of initiation (selection against emigration), travel (selection during transportation of dispersing units), and settlement (selection against immigration). Thus, the selective forces affecting dispersal are closely associated with the costs of the entire dispersal process. Iritani & Iwasa (2014) examined the evolution of host dispersal when affected by parasites, and showed that parasite infection is a strong selective force acting on the dispersal rate; therefore, concluding that the dispersal bias towards susceptible individuals (S-biased dispersal) or infected individuals (I-biased dispersal) is determined by the differentiated dispersal costs between disease states. Their results indicate that the cost variations within subpopulations play a critical role in the evolution of dispersal, even in a homogeneous population, and that life history events can give rise to cost variations.

The results from previous empirical studies indicate either S-biased (Heeb *et al.* 1999; Goodacre *et al*. 2009; Fellous *et al*. 2011) or I-biased dispersal (Brown & Brown

1996; van Vuren 1996) in host–parasite systems. In a host–parasite system, parasite life history can greatly modify the dispersal costs of their hosts. For example, in the case of the blue tit (*Cyanistes caeruleus*), if the infection (or indirect transmission) of parasites occurs during dispersal, then susceptible individuals are subject to a higher cost than that by infected individuals. Similarly, if infected hosts have a chance of recovery during dispersal (e.g. salmon that is parasitised by the larvae of freshwater pearl-mussels), infected individuals are expected to have strong incentives for dispersing (Morales *et al*. 2006; Akiyama & Iwakuma 2009; Terui *et al*. 2014). These biased dispersal tendencies are important for population biology, because if S-biased dispersal is observed in a metapopulation, parasites fail to spread over space. Consequently, parasites are locally clustered, and host subpopulations may suffer endemic infectious diseases. In contrast, if I-biased dispersal is realised, then parasites may spread. In this sense, understanding host dispersal bias is central to spatial epidemiology.

Recently, several studies have reported that parasites represent important agents in the diversification of host fish species through the selection against migrants, and the underlying mechanisms may include diverse scenarios (MacColl & Chapman 2010; Karvonen & Seehausen 2012). One scenario, 'selection against infected emigrants', suggests that parasite-imposed natural selection acts on the initial dispersal of infected individuals; and another scenario refers to 'selection against infected immigrants'. These selection mechanisms are well studied in the context of social evolution in that parasite and/or disease infection is costly to social organisation or group living (Alexander 1974; Altizer *et al*. 2003, Nunn & Altizer 2006). Hence, host species and their respective societies have developed various mechanisms to combat infection (e.g. 'social barrier'; Loehle 1995). Therefore, determining how parasites mediate the costs of host dispersal involves diverse ecological scenarios. Unfortunately, due to the substantial complexity of the environment, direct estimates of the dispersal cost and/or dispersal bias between disease states are often very difficult to determine.

In this study, we develop mathematical models to resolve the quantitative measures for dispersal bias that are associated with parasite infection and host life history, while also taking ecological dispersal cost variations (among disease states) that emerge in host–parasite systems into account. We incorporate the following factors: local infection before dispersal, infection during dispersal, recovery during dispersal, recovery after dispersal, post-dispersal parasite-induced additional mortality (virulence), and selection against infected immigrants or emigrants (Table 1). We also analyse the evolutionary stability (ES; Smith 1982) and convergence stability (CS; Eshel 1983) for host dispersal strategies that depend on the disease state (S or I). We assume an island model population structure and employ the direct fitness approach in inclusive fitness theory (Taylor & Frank 1996; Frank 1998; Rousset 2004). We also provide proof of evolutionary stability. Lastly, we show that natural selection favours dispersal bias towards susceptible or infected individuals and that the bias is determined by the relative cost of dispersal for each disease state.

2. Methods

Hereafter, by 'infection' or 'get infected', we mean the transition to a disease state from S to I. On the other hand, by 'recovery', we mean the transition from I to S. We illustrate the entire life history of the host in Figure 1(a), following Bonte *et al*. (2012).

2.1 Life history: before departure

Assume that the host population follows Wright–Fisher demography with non-overlapping generations, and is composed of a sufficiently large number of subpopulations (n_{d}; $n_{\mathrm{d}} \to +\infty$), each of which fosters an equal number of adults (N). Each adult asexually reproduces equal, large numbers of offspring (fecundity as J; $J \to +\infty$) under haploid genetics, which is followed by the death of all adults. In each subpopulation, local parasite infection occurs at random with prevalence (R), among offspring, and infection occurs immediately before offspring dispersal. Note that after

infection, there are two host states: susceptible individuals (i.e. not infected; S-individuals) and infected individuals (i.e. infected; I-individuals). Specifically, after the infection stage, a randomly sampled individual (from the entire population) is an I-individual with the probability R. State-dependent host dispersal (i.e. dispersal is conditional on either the S or I disease states) follows the infection stage; and S-individuals disperse with the probability z_S, while I-individuals disperse with the probability z_I. In the 'selection against infected emigrants' scenario, we assume a fraction ($\varepsilon_{\text{Emig}}$) of infected emigrants suffer mortality until the departure phase.

2.2 Life history: during dispersal, before immigration

During travel between subpopulations, each individual succeeds in transfer with the probability p. In other words, the remained ratio (1–p) of individual die during the travel, which represents 'selection during transportation' (e.g. due to predation). In addition, we incorporate parasite infection and recovery during dispersal, a discrete time transition that is represented by linear recurrence equations. Therefore, during travel, S-individuals are infected with parasites at the rate α^{D}, and I-individuals recover from parasitic infection with the rate β^{D} (where the superscript $^{\text{D}}$ denotes 'during' dispersal; Figure 1 (b)). In the 'selection against infected immigrants' scenario, we assume I-individuals fail to settle in other subpopulations at the additional rate $\varepsilon_{\text{Immig}}$.

2.3 Life history: after immigration

We assume that, immediately after dispersal, infection and recovery takes place in each subpopulation. Moreover, a fraction, α^{A}, of S-individuals are infected and a fraction, β^{A}, of I-individuals recover from infection in each population (where the superscript $^{\text{A}}$ denotes 'after' dispersal; Figure 1). Lastly, local competition occurs, so N-individuals within subpopulations are chosen to form the next generation; and at the competition stage, I-individuals suffer from parasitic damage ('virulence', δ), which

makes them competitively weaker than S-individuals by the factor $(1-\delta)$. Thus, competition is asymmetric between disease states (see below and Appendix (a) for the fitness function).

2.4 Fitness

Let $\mathbf{x}^{\bullet} := (x_S^{\bullet}, x_I^{\bullet})$ denote the deviant strategy of a rare mutant ('focal adult'), whose components indicate a state-dependent dispersal rate. In this situation, the offspring of the focal adult disperse with rate $x_S^{\bullet}$ when susceptible and the rate $x_I^{\bullet}$ when infected. However, note that the strategy of the offspring is equivalent to that of the parent because asexual haploidy is assumed. Therefore, the offspring of each focal adult are exact copies the parent; hence, it is not necessary to distinguish the strategy of the adult individual and its offspring. Henceforth, we use 'individual' to indicate 'juvenile offspring'. Let $\mathbf{x}^{0\mathrm{R}} := (x_S^{0\mathrm{R}}, x_I^{0\mathrm{R}})$ be the average strategy of adults within the 'home subpopulation' (i.e. the native subpopulation of the focal adult), and note that we calculate the average value $\mathbf{x}^{0\mathrm{R}}$, among *N*-members, including the focal adult. Let $\mathbf{x}^{1} := (x_S^{1}, x_I^{1})$ be the mean strategy for the entire population. The fitness measure, *w*, is the expected number of adult offspring of the focal adult, and it can be partitioned into

$$w[\mathbf{x}^{\bullet}, \mathbf{x}^{0\mathrm{R}}, \mathbf{x}^{1}] := w_{\mathrm{HOME}} + w_{\mathrm{AWAY}}, \tag{1}$$

where the first term represents the fitness component in the home subpopulation, and the second term represents the sum of the fitness components in other subpopulations ('away' subpopulation). Appendix (a) is devoted to deriving an explicit form, and we summarise the notation in Table 1.

3. Results

In order to calculate the convergence stability condition, we adopt the direct fitness approach, or specifically, the neighbour-modulated approach in kin selection theory (Taylor & Frank 1996; Frank 1998; Rousset 2004). We can write the fitness gradient $\nabla w := (D_S, D_I)^{\mathrm{T}}$, as

$$D_S = \frac{\partial w}{\partial \xi_S} = \frac{\partial w}{\partial x_S^\bullet} + F_{\mathrm{ST}}^{\mathrm{R}} \frac{\partial w}{\partial x_S^{0\mathrm{R}}}, \tag{2-a}$$

and

$$D_I = \frac{\partial w}{\partial \xi_I} = \frac{\partial w}{\partial x_I^\bullet} + F_{\mathrm{ST}}^{\mathrm{R}} \frac{\partial w}{\partial x_I^{0\mathrm{R}}}, \tag{2-b}$$

where both of the derivatives are evaluated at $\mathbf{x}^\bullet = \mathbf{x}^{0\mathrm{R}} = \mathbf{x}^1 = \mathbf{z}^* = (z_S^*, z_I^*)$. Here, (ξ_S, ξ_I) represents a pair of small deviations of the focal adult $(x_S^\bullet, x_I^\bullet)$ from residents (x_S^1, x_I^1) with $(\xi_S, \xi_I) \coloneqq (x_S^\bullet, x_I^\bullet) - (x_S^1, x_I^1)$, and $F_{\mathrm{ST}}^{\mathrm{R}}$ is the coefficient of relatedness (Hamilton 1964a, b; see Appendix (b) and (c) for the detailed expansion).

The singular strategy (SS; Metz *et al.* 1997), (z_S^*, z_I^*), is found by analysing the vector-field on the unit square for $0 \le z_S^* \le 1$ and $0 \le z_I^* \le 1$ (Figure 2). Because Eqs. (2) do not hold simultaneously (fundamental algebra was used to prove the relationship; see Appendix D in Iritani & Iwasa 2014), the SS lies on the boundary of the unit square for $0 \le z_S^* \le 1$ and $0 \le z_I^* \le 1$. The analytical form of SS is shown in Appendix (c), and its dependence on R is illustrated in Figure 3. We use the Kuhn–Tucker theorem to prove that the SS is surely an evolutionarily stable strategy (ESS; Smith 1982; see Appendix (d)).

Our analysis showed that either S-biased dispersal or I-biased dispersal might evolve, and we derived a critical quantity (the 'bias predictor') to determine the dispersal bias (I-bias or S-bias). The bias predictor (Δ), derived in Appendix (b), gives a qualitative measure for the evolutionary consequences of host dispersal. Explicitly, this bias is determined by the sign of the bias predictor (Δ):

$$\Delta \coloneqq \underbrace{\left(\frac{\beta^{\mathrm{D}}}{G_{\mathrm{p}I/\mathrm{p}S}} + (1-\beta^{\mathrm{D}})(1-\varepsilon_{\mathrm{Immig}}) \right)(1-\varepsilon_{\mathrm{Emig}})}_{=:(1-C_I)/p} - \underbrace{\left((1-\alpha^{\mathrm{D}}) + \alpha^{\mathrm{D}}(1-\varepsilon_{\mathrm{Immig}}) G_{\mathrm{p}I/\mathrm{p}S} \right)}_{=:(1-C_S)/p}, \tag{3-a}$$

where

$$G_{\mathrm{p}I/\mathrm{p}S} \coloneqq \{1-(1-\beta^{\mathrm{A}})\delta\} \big/ (1-\alpha^{\mathrm{A}}\delta) > 0 \tag{3-b}$$

is the 'competition asymmetry' between S- and I-individuals. This accounts for the

relative competitive ability of philopatric I-individuals as compared to that of philopatric S-individuals, because philopatric S-individuals are subject to virulence with the probability α^{A}, whereas philopatric I-individuals suffer virulence with the probability $(1-\beta^{A})$. When Δ is positive, I-biased dispersal is favoured, and when the value is negative S-biased dispersal is favoured. The greater this value is, the stronger the evolutionary forces that act on I-biased dispersal. The first term in Eq. (3-a) is the relative competitive ability of migrant I-individuals compared to that of philopatric S-individuals (divided by *p*), and the second term is that of migrant S-individuals compared to that of philopatric S-individuals, (also divided by *p*). Consequently, Δ is the difference between the value of dispersal costs, denoted by C_S and C_I, (for S- and I-individuals, respectively) divided by the basic dispersal success *p* (i.e. $\Delta = (C_S - C_I)/p$, see Appendix (b)). In Figure 4, we illustrate the dependence of Δ on several parameters.

Summarising the results, we derived the evolutionary outcome of dispersal bias:

$$\begin{array}{lllll} \Delta > 0 & \Leftrightarrow & C_S > C_I & \Leftrightarrow & z_S < z_I \\ \Delta < 0 & \Leftrightarrow & C_S < C_I & \Leftrightarrow & z_S > z_I \end{array} \quad . (4)$$

Thus, we conclude that populations with a lower cost of dispersal should disperse particularly when the costs are affected by ecological scenarios associated with the life history of the host, and that dispersal costs are estimated from epidemiological dynamics (disease state transition) and from traits of parasites (virulence).

4. Discussion

Our analysis showed that (1) dispersal bias can evolve depending on the difference in the costs of dispersal, and (2) such differentiated costs (represented by Δ in Eq. 3-a) arise through several ecological factors. We can reduce Δ to a simplified measure as needed (see below). In addition, to clarify the dispersal bias by Δ, there is no need to directly estimate the 'pure' virulence value, δ. Instead, only the negative impacts (e.g. $\alpha^{A}\delta$, which quantifies the virulence multiplied by the probability of infection after

dispersal) need to be estimated. Thus, the bias predictor (Δ) provides a target parameter for the prediction of dispersal bias, and consequently, the spread of parasites.

4.1 Generality of the bias predictor

Our results can be generally interpreted in three ways. First, we can recover a classical result (Frank 1986; Taylor 1988; Frank 1998; Rousset 2004) by setting $R=0$ and $\alpha^{\mathrm{A}}=\alpha^{\mathrm{D}}=0$ (i.e. assuming no infection):

$$z_S^*=\frac{r_S-C_S}{r_S-C_S^2}, \quad (5)$$

where r_S represents relatedness (see Appendix (c)). This means that the effect of kin-competition is at play, along with dispersal costs. Second, we can recover our previous result on the evolution of host dispersal bias. Setting $\alpha^{\mathrm{A}}=\alpha^{\mathrm{D}}=\beta^{\mathrm{A}}=\varepsilon_{\mathrm{Immig}}=0$, and following with some algebra, yields

$$\Delta \propto \delta\frac{\beta^{\mathrm{D}}}{1-\delta}-\frac{\varepsilon_{\mathrm{Emig}}}{1-\varepsilon_{\mathrm{Emig}}}, \quad (6)$$

where '$\propto$' represents 'positively proportional to', henceforth. This index is equivalent to that obtained by Iritani & Iwasa (2014), and note that their term 'release rate during dispersal', γ, corresponds to β^{D} in our present model.

In a third sense, the bias predictor (Δ) serves as a good estimator for the prediction of dispersal bias between states, so it can be applied depending on the context. For example, setting $\varepsilon_{\mathrm{Emig}}=\varepsilon_{\mathrm{Immig}}=0$, we can reduce the bias predictor (Δ) to

$$\Delta=\frac{(1-G_{\mathrm{p}I/\mathrm{p}S})(\alpha^{\mathrm{D}}G_{\mathrm{p}I/\mathrm{p}S}+\beta^{\mathrm{D}})}{G_{\mathrm{p}I/\mathrm{p}S}} \quad (7)$$

To investigate the dependence of Δ on δ, the partial differentiation of Δ, with respect to δ, by chain-rule, yields

$$\frac{\partial \Delta}{\partial \delta} = \frac{\partial \Delta}{\partial G_{\mathrm{p}I/\mathrm{p}S}} \cdot \frac{\partial G_{\mathrm{p}I/\mathrm{p}S}}{\partial \delta} = \underbrace{\left(\frac{\alpha^{\mathrm{D}}}{(1-\alpha^{\mathrm{A}}\delta)^2} + \frac{\beta^{\mathrm{D}}}{\{1-(1-\beta^{\mathrm{A}})\delta\}^2} \right)}_{>0} \mathrm{Auto}^{\mathrm{A}}, \quad (8)$$

where $\mathrm{Auto}^{\mathrm{A}} := 1-\beta^{\mathrm{A}}-\alpha^{\mathrm{A}}$ is the autocorrelation of disease state transitions, calculated between the two stages: 'immediately after dispersal' and 'just before competition' (for the derivation, see Appendix (e)). Thus, I-biased dispersal can be either promoted or inhibited by parasite virulence depending on the sign of $\mathrm{Auto}^{\mathrm{A}}$, since all other factors are positive (Figure 4). Therefore, if I-individuals are more likely to be in state-I than S-individuals (because of some ecological scenarios), then strong virulence, δ, favours I-biased dispersal. Most notably, we can find $\Delta \propto \mathrm{Auto}^{\mathrm{A}}$, and it implies that when measuring dispersal bias between disease states, only the autocorrelation of disease states before and after dispersal need to be estimated—as long as disease state transitions can occur during dispersal (i.e. either α^{D} or β^{D} is positive). Some may ask why the autocorrelation matters. The dependence of the bias predictor (Δ) on the autocorrelation ($\mathrm{Auto}^{\mathrm{A}}$) can be explained by noting that, from the definition of competition asymmetry, $G_{\mathrm{p}I/\mathrm{p}S}$ in Eq. (3), we have

$$\begin{aligned} G_{\mathrm{p}I/\mathrm{p}S} < 1 \quad &\Leftrightarrow \quad 1-(1-\beta^{\mathrm{A}})\delta < 1-\alpha^{\mathrm{A}}\delta \\ &\Leftrightarrow \qquad 0 < \mathrm{Auto}^{\mathrm{A}} \end{aligned} \quad . (9)$$

Therefore, $G_{\mathrm{p}I/\mathrm{p}S} < 1$ implies that infected philopatric individuals that were infected after settlement (with competitive ability $p(1-\alpha^{\mathrm{A}}\delta)$) are competitively stronger than those that were infected before the dispersal stage (with competitive ability $p\{1-(1-\beta^{\mathrm{A}})\delta\}$); and this gives I-individuals a strong incentive for dispersing (i.e. I-biased dispersal is advantageous). Thus, we can conclude that when $\mathrm{Auto}^{\mathrm{A}}$ is positive, higher virulence favours I-biased dispersal; and when it is negative, S-biased dispersal is favoured. The practical method to estimate the autocorrelation of disease states requires individual-level tracking rather than tracking at the population-level.

The bias predictor (Δ) has an outstanding merit that it is independent of prevalence

(R). The prevalence in local subpopulations or groups is usually difficult to measure in natural populations. Using the bias predictor (Δ) allows the prediction of the dispersal bias among disease states without directly estimating the local prevalence. However, it should be noted that Δ may be dependent on prevalence (R) if density-dependent horizontal transmission occurs. This is because the 'transition' between states corresponds to horizontal transmission and recovery processes, which are neglected in the present model. This issue is challenging and will be addressed in a future study.

4.2 Ecological scenarios modify dispersal costs

We have derived the dispersal costs and their differentiation by several factors, and we discuss the ecological scenarios that determine Δ.

4.2.1 Transition during dispersal, α^{D} and β^{D}

Intuitively, α^{D} measures the 'infection risk during dispersal' for S-individuals, whereas β^{D} measures the 'recovery rate during dispersal' for I-individuals. Hence, it is expected that both factors enhance I-biased dispersal, but this depends on the autocorrelation (as discussed in Discussion 4.1). Indeed, infection during (and after) dispersal is observed in a bird species (Knowles *et al.* 2013), and recovery during dispersal is also observed in salmon that are infected with the larvae of freshwater pearl-mussels (e.g. Morales *et al.* 2006; Akiyama & Iwakuma 2009; Terui *et al.* 2013). Thus, the bias predictor presented here plays an essential role in understanding the distribution of hosts and parasites in these systems.

Specifically, 'selection against immigrant pathogens' is observed, where pathogens have lower performance in the novel environments that they encounter after being carried via their hosts' dispersal (e.g. Giraud 2006a, b; Giraud *et al.* 2006; Le Gac & Giraud 2004). If such a mechanism is substantial, then the host species may recover from parasites after the dispersal stage (i.e. recovery during dispersal). Our current results shed light on Giraud's (2006a, b) system for the study of host mobility and its

effect on parasite local adaptation and diversification.

We can verify that Δ increases monotonically with increased α^{D} and β^{D} when $\mathrm{Auto}^{\mathrm{A}} > 0$ (Appendix (b)); and this occurs when the philopatric individuals that are infected after dispersal with competitive ability $\left(1-\alpha^{\mathrm{A}}\delta\right)p$ are competitively stronger than continuously infected individuals with competitive ability $\left(1-(1-\beta^{\mathrm{A}})\delta\right)p$ (see Discussion 4.1). Thus, dispersal bias is promoted by transitions that occur during dispersal. This is because, when $\mathrm{Auto}^{\mathrm{A}} > 0$, I-individuals have an incentive for recovery during dispersal, and the dispersal of S-individuals is disadvantageous because they risk infection during dispersal.

4.2.2 Transition after dispersal, α^{A} *and* β^{A}

Hosts can exhibit class-transition after dispersal, because of a variety of parasite life histories. For example, Knowles *et al.* (2013) reported that infection occurred after dispersal, and potentially during dispersal, in the blue tit (*C. caeruleus*). In the study, they examined the location and timing of infections from two malaria parasites (*Plasmodium circumflexum* and *P. relictum*), and determined that the infection with *P. circumflexum* occurred after birds settled into breeding territories. On the other hand, they demonstrated that the infection with *P. relictum* occurred during major dispersal events, because they found a positive correlation between the dispersal distance and the probability of infection with *P. relictum*. Our present models exclude dispersal distance, while they lack the foci on dispersal bias. Thus, our results shed light on the epidemiologically important issues of the spread of malarial parasites.

Also, for the sake of generality, we considered post-dispersal recovery, because it can occur when the focal host is not definitive, but intermediate. Therefore, if parasites are transmitted to another host species by hitchhiking on an intermediate host species, then parasite clearance can occur after dispersal. In this context, however, we need to take into account the possibility that the dispersal of I-individuals may be the

consequence of parasite manipulation (see Discussion 4.5 below).

The dependence of Δ on α^{A} and β^{A} is complex due to additional parameters, but deserves discussion. Indeed, from the definition of $G_{\mathrm{p}I/\mathrm{p}S}$ in Eq. (3), we obtain

$$\frac{\partial G_{\mathrm{p}I/\mathrm{p}S}}{\partial \alpha^{\mathrm{A}}} > 0 \quad \text{and} \quad \frac{\partial G_{\mathrm{p}I/\mathrm{p}S}}{\partial \beta^{\mathrm{A}}} > 0 \,, \quad (10)$$

so that

$$\frac{\partial \Delta}{\partial \alpha^{\mathrm{A}}} = \frac{\partial \Delta}{\partial G_{\mathrm{p}I/\mathrm{p}S}} \cdot \frac{\partial G_{\mathrm{p}I/\mathrm{p}S}}{\partial \alpha^{\mathrm{A}}} < 0 \quad \text{and} \quad \frac{\partial \Delta}{\partial \beta^{\mathrm{A}}} = \frac{\partial \Delta}{\partial G_{\mathrm{p}I/\mathrm{p}S}} \cdot \frac{\partial G_{\mathrm{p}I/\mathrm{p}S}}{\partial \beta^{\mathrm{A}}} < 0 \,. \quad (11)$$

Thus, the sign of Δ can change from positive to negative as α^{A} and β^{A} increase (indeed, we can easily solve $\Delta = 0$ with respect to α^{A} or β^{A} to verify whether $\Delta = 0$ occurs at an intermediate value for $0 \le \alpha^{\mathrm{A}} \le 1$ and $0 \le \beta^{\mathrm{A}} \le 1$, but the calculation is tedious due to multiple parameters).

Horizontal transmission can occur following the dispersal stage in many systems (Anderson & May 1979). In the model, the post-dispersal transition event can be regarded as a horizontal transmission event, whereby parasites are transferred from one host individual to another of the same species and generation when the transmission is frequency independent. It is often suggested, however, that host–parasite dynamics are heavily influenced by the density and/or frequency of I-individuals (Hochachka & Dhondt 2000; Begon *et al*. 2002; but see Oli *et al.* 2006 for frequency-independent dynamics). We will address this issue in a future study.

4.2.3 Selection against infected emigrants and immigrants, $\varepsilon_{\mathrm{Emig}}$ *and* $\varepsilon_{\mathrm{Immig}}$

Parasites can harm their hosts at each stage of the hosts' life history (Barrett *et al.* 2008). Since the cost of dispersal is partially paid before dispersal (Bonte *et al.* 2012), selection against infected emigrants can emerge as a result of early-stage infection and imposed parasite damage. Indeed, as reviewed in Zuk & Stoehr (2002), parasites can harm their hosts during the developmental stage, which may possibly result in the incomplete

development of dispersal-related traits (e.g. wings in insects).

Selection against infected immigrants can act in the context of the evolution of 'social barrier' mechanisms (Loehle 1995), in which group-living organisms are adapted to pathogen and/or parasite transmission. Selection against infected immigrants may act in animals with the ability to distinguish between infected and uninfected individuals, and between philopatric and migrant individuals. Loehle (1995) proposed the hypothesis of social avoidance, in which strange and sick individuals fail to join the other group members. This phenomenon is observed in wild populations of guppies (avoidance of infected mates; Kennedy *et al.* 1987) and chimpanzees (avoidance of strangers; Goodall 1986). Combinations of such mechanisms can potentially result in selection against infected immigrants.

It is obvious that Δ decreases as $\varepsilon_{\mathrm{Emig}}$ increases, but the dependence of Δ on $\varepsilon_{\mathrm{Immig}}$ is not as apparent. In Figure 4b, we illustrate the dependence of Δ on $\varepsilon_{\mathrm{Immig}}$. $\mathrm{Auto}^{\mathrm{A}} > 0$ implies that a large $\varepsilon_{\mathrm{Immig}}$ is likely to favour I-biased dispersal (see Discussion 4.1). Thus, we can conclude that the autocorrelation ($\mathrm{Auto}^{\mathrm{A}}$) plays a key role in determining the evolution of biased dispersal.

4.3 ES-dispersal strategy

As shown in Figure 3, ES-dispersal rate z_S^* and z_I^* is either 0 or 1 when R varies. This result is similar (or mathematically equivalent) to that of Iritani & Iwasa (2014); however, our result is more general (see Discussion 4.1). Because we consider the finite capacity of subpopulations, we must also take the intensity of kin competition into account. In our result of ES-dispersal strategy (described in Appendix (c)), the dispersal rate that evolves is balanced between the kin competition effect (which is represented by the relatedness coefficient) and the cost of dispersal (e.g. C_S). Such a drastic dispersal bias can be understood from the kin competition perspective. For example,

when the bias predictor Δ is positive, I-individuals have a strong incentive for dispersing. However, from the S-individuals' perspective, their infected 'sibs' will emigrate so that the mean dispersal tendency among sibs (i.e. the mean dispersal rate of individuals bearing the same gene) can be balanced to enhance their inclusive fitness; and this occurs because kin selection favours an intermediate value of dispersal (Hamilton & May 1977; Frank 1986; Taylor 1988). As the prevalence (R) increases, the mean dispersal rate among sibs increases, so that S-individuals have a lower dispersal tendency due to kin selection.

As already examined, some species express either I-biased dispersal (Brown & Brown 1992; van Vuren 1996) or S-biased dispersal (Heeb *et al.* 1999; Goodacre *et al.* 2009; Fellous *et al.* 2011). Our analysis provides feedback for these systems that can be used for further studies of the evolutionary consequences of host migration, and thus, parasite spread.

4.4 Future study

4.4.1 Co-evolutionary dynamics

Previous studies have argued the possibility that local parasite adaptation is affected by host dispersal (Gandon *et al*. 1996; Boots & Sasaki 1999, 2002), and that host dispersal tendency is strongly dependent on parasite virulence (Iritani & Iwasa 2014). In ecological scenarios, dispersal bias plays an important role in that the gene flow of parasites is either regulated or promoted across space because of the difference between host dispersal tendencies. The geographical distribution of parasites is heavily influenced by host dispersal and distribution. For instance, when the bias is towards infected individuals, parasites succeed in migrating across space. Conventional wisdom suggests that coevolution exhibits spatial mosaics (i.e. the 'geographic mosaic theory of coevolution'; Thompson 2005). Previous studies combining parasite adaptation and host dispersal have assumed, however, that the host migration tendency among patches is constant, but our results suggest that is not always feasible. Demonstrating that

between-subpopulation genetic differentiation is either reduced or enhanced by parasites, we can conclude that spatial genetic diversity can occur not only through geographically diverged host–parasite interactions, but also through state-dependent host dispersals. Thus, the model described here could be extended to allow for such coevolution, but only by accounting for variation in the levels of host–parasite co-migration.

4.4.2. Host manipulation by parasites

Our results provide interesting aspects regarding host manipulation by parasites (Thomas *et al.* 2007; Hughes *et al.* 2012), as some parasites manipulate their hosts to promote the transmission probability to another (or next) host. Lion *et al.* (2006) studied how parasite-induced host dispersal modifies the dynamic spatial structure of the host–parasite community. In their model, they assumed that the dispersal of infected hosts was completely determined by the parasite (i.e. it was a fixed parasite trait); however, we set the dispersal of I-individuals as an evolving host trait. Thus, combining our results with those of Lion *et al.* (2006) would permit a more specific analysis of the host–parasite structuring process.

4.4.3. Role of horizontal transmission

We constructed a host–parasite model assuming no horizontal transmission. It is often suggested, however, that host–parasite dynamics are heavily influenced by host density and prevalence (Hochachka & Dhondt 2000; Begon *et al*. 2002). Therefore, an investigation of the effect of horizontal transmission on the evolution of host dispersal (or its bias) is needed. In particular, in the model, we conclude that dispersal bias is independent of the initial prevalence (R). In the event of frequency- or density-dependent transmission, the infection factor β^{A} might be dependent on R, because the risk of horizontal transmission is thought to increase as the density of infected hosts or the prevalence of the infection increases (Hochachka & Dhondt 2000; Begon *et al*. 2002). Consequently, our present analysis of the dispersal-bias predictor is

insufficient when frequency-dependent transmission cannot be neglected (though we can apply this model when the transmission is not frequency-dependent). By incorporating horizontal transmission, we can predict the spatial dynamics of host–parasite coevolution, and explain how geographical biodiversity is shaped.

We have restricted ourselves to assuming the prevalence (R) is constant over space and generations, although this is not the case in host–parasite systems (i.e. prevalence varies both spatially and temporally). Our present results provide a predictable hypothesis regarding dispersal bias that is independent of prevalence, and in the future, we aim to develop a more applicable estimator under increasingly relaxed assumptions regarding spatial heterogeneity.

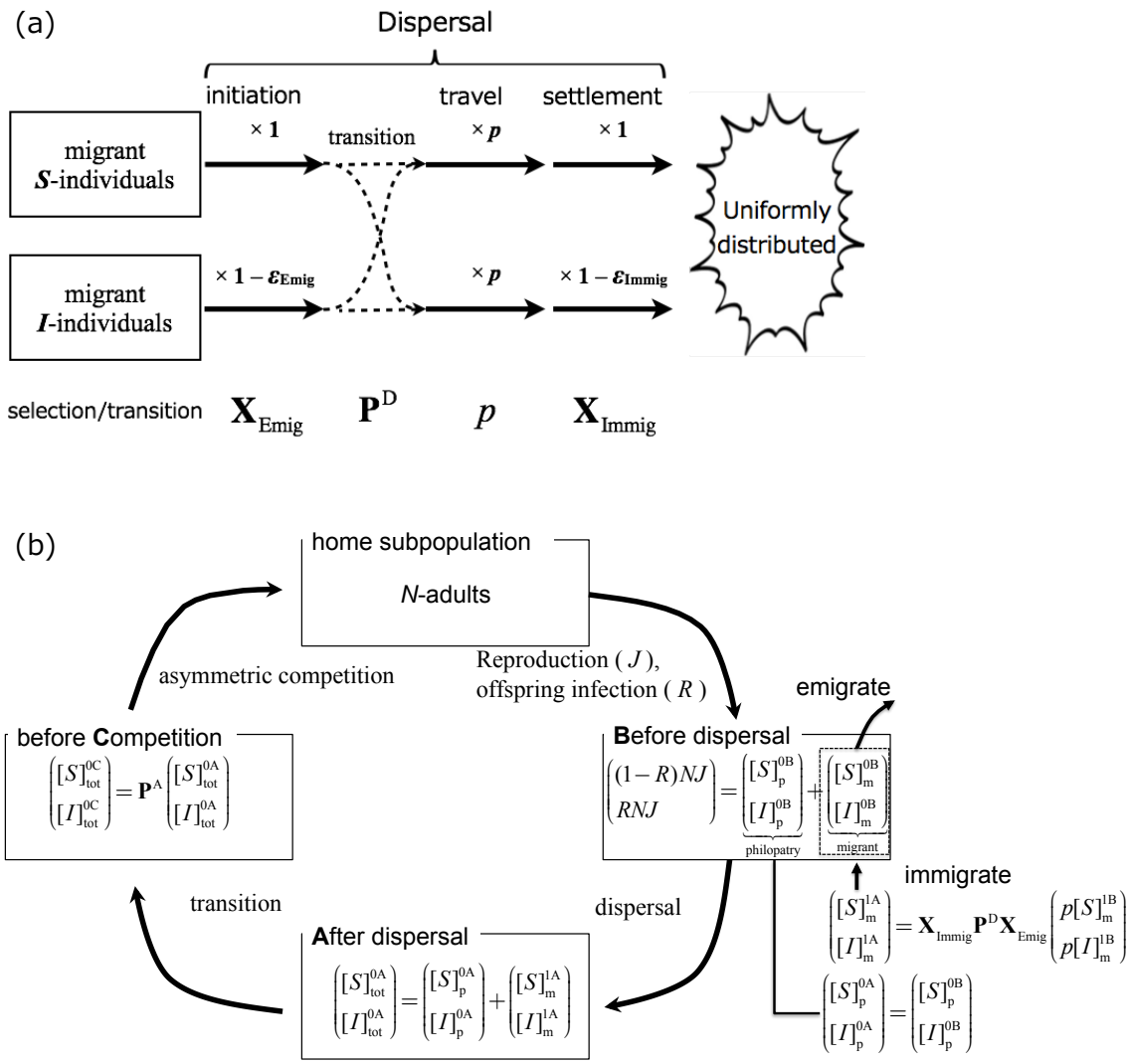

Figure 1

The schematics of the host life cycle under study: reproduction, infection, dispersal, after dispersal, and competition. (a) Processes of dispersal (initiation, travel, and settlement) and life cycle event that migrants experience. Each solid arrow represents the probability of success at each stage: i.e. selection strength at each stage. Dashed, crossed arrows represent transition during dispersal ($\mathbf{P}^{\mathrm{D}}$). Note that the travel success enjoyed by S- and I-individuals is denoted by a common factor p, which we allow various interpretations (e.g. the probability of successful initiation, travel, settlement, and/or their product). (b) The whole life cycle. Here we concentrate on the focal subpopulation (i.e. 'home' subpopulation), and changing the superscript ^{0} to ^{1} yields the

full life history in the non-focal subpopulation (i.e. ‘away’ subpopulation). The notation is used in accordance with that shown in Table 1.

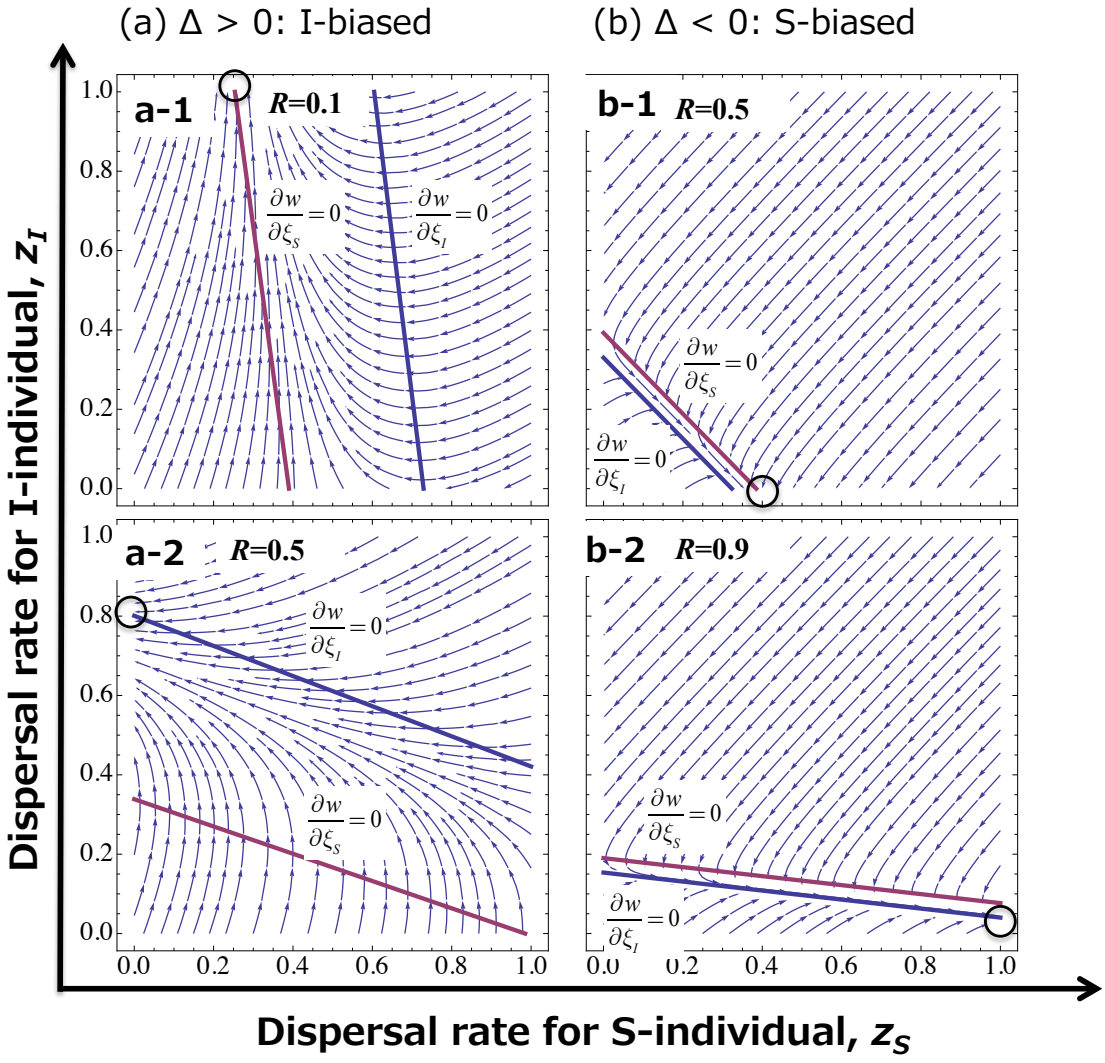

Figure 2

Evolutionary vector fields of the dispersal rate (z_S, z_I). Two lines indicate $\partial w/\partial \xi_S\big|_{(\xi_S,\xi_I)=(0,0)} = 0$ and $\partial w/\partial \xi_I\big|_{(\xi_S,\xi_I)=(0,0)} = 0$ in both (a) and (b). We can observe that the candidate evolutionarily stable strategy (ESS), indicated by a solid circle, is located on the boundary. Parameters: (a) $\alpha^{\mathrm{A}} = \beta^{\mathrm{A}} = 0.4$ so that $\Delta > 0$ holds, and (b) $\alpha^{\mathrm{A}} = \beta^{\mathrm{A}} = 0.6$ so that $\Delta < 0$ holds. Other parameters are shared in (a) and (b): $\varepsilon_{\mathrm{Emig}} = 0.5$, $\varepsilon_{\mathrm{Immig}} = 0.1$, $p = 0.6$, $\delta = 0.2$, $N = 5$, and $\alpha^{\mathrm{D}} = \beta^{\mathrm{D}} = 0.6$. The prevalence R is varied, and is indicated in each panel.

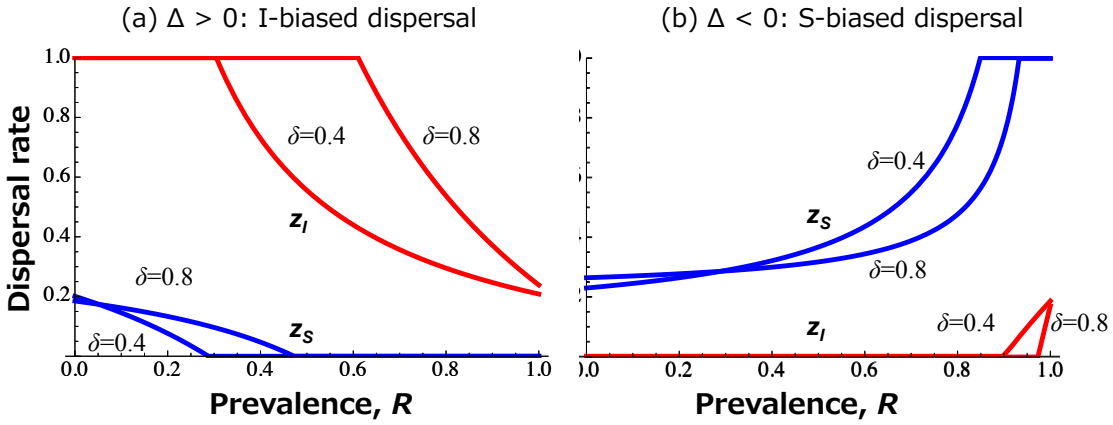

Figure 3

ES-dispersal strategy, (z_S, z_I), plotted against prevalence, R. We vary the virulence, δ = 0.4 and 0.8, as shown in the panel. Parameters: (a) $\alpha^A = \beta^A = 0.4$ so that $\text{Auto}^A < 0$ and $\Delta > 0$ holds, and (b) $\alpha^A = \beta^A = 0.6$ so that $\text{Auto}^A > 0$ and $\Delta < 0$ holds. Other parameters are shared between (a) and (b): $\varepsilon_{\text{Emig}} = 0.1$, $\varepsilon_{\text{Immig}} = 0.1$, $p = 0.6$, $N = 5$, and $\alpha^D = \beta^D = 0.6$.

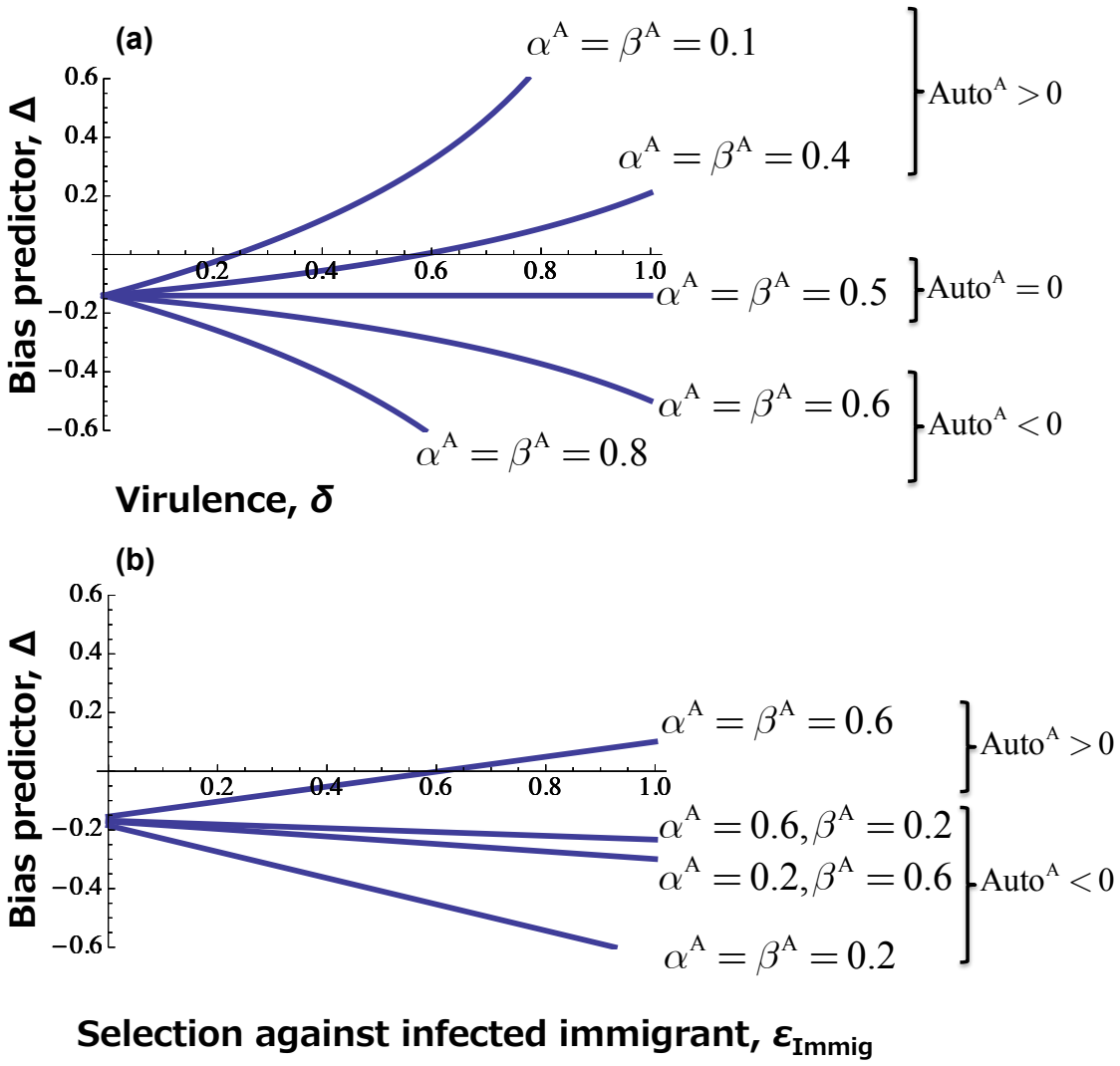

Figure 4

(a) The dependence of Δ on δ, α^A, and β^A. When Auto^A is positive, strong virulence indicates a large Δ, and I-biased dispersal is favoured; when Auto^A is negative, weak virulence indicates a small Δ, and S-biased dispersal is favoured. When $\text{Auto}^A = 0$, Δ is no longer dependent on δ. (b) The dependence of Δ on $\varepsilon_{\text{Immig}}$, α^A, and β^A. When Auto^A is positive, a large $\varepsilon_{\text{Immig}}$ indicates a large Δ, and I-biased dispersal is favoured; when Auto^A is negative, a small $\varepsilon_{\text{Immig}}$ indicates a small Δ, and S-biased dispersal is favoured.

Appendix

(a) Fitness measure

The fitness measure under study, w, is defined as the expected number of adult offspring. We partitioned w into two components as follows:

$$w[\mathbf{x}^{\bullet},\mathbf{x}^{0\mathrm{R}},\mathbf{x}^{1}] = w_{\mathrm{HOME}}[\mathbf{x}^{\bullet},\mathbf{x}^{0\mathrm{R}},\mathbf{x}^{1}] + w_{\mathrm{AWAY}}[\mathbf{x}^{\bullet},\mathbf{x}^{0\mathrm{R}},\mathbf{x}^{1}], \qquad \text{(a-1)}$$

where the first component indicates the probability that the offspring of focal adult can become the adult in the next generation of the home subpopulation, and the second component does so in away subpopulation. To obtain this fitness measure, we need to calculate the transition dynamics as shown in Figure 1. We calculate the state-transitions in a backwards manner.

(a-1) Transition after dispersal

First, we describe the transition dynamics after dispersal, but before competition, as

$$\begin{pmatrix} [S]_{\mathrm{tot}}^{i\mathrm{C}} \\ [I]_{\mathrm{tot}}^{i\mathrm{C}} \end{pmatrix} = \underbrace{\begin{pmatrix} 1-\alpha^{\mathrm{A}} & \beta^{\mathrm{A}} \\ \alpha^{\mathrm{A}} & 1-\beta^{\mathrm{A}} \end{pmatrix}}_{=\mathbf{P}^{\mathrm{A}}} \begin{pmatrix} [S]_{\mathrm{tot}}^{i\mathrm{A}} \\ [I]_{\mathrm{tot}}^{i\mathrm{A}} \end{pmatrix}, \qquad \text{(a-2)}$$

where $[S]_{\mathrm{tot}}^{i\mathrm{C}}$ (or $[I]_{\mathrm{tot}}^{i\mathrm{C}}$) represents the number of S-individuals (or I-individuals, respectively) just before competition, for $i=0$ (home subpopulation) or $i=1$ (away subpopulation). The super script, $^{\mathrm{C}}$, is used to emphasise 'before competition', and the subscript $_{\mathrm{tot}}$ indicates that the dynamics occur for all individuals (including both philopatric individuals and migrants) in each subpopulation. In the components of transition matrix, the superscript $^{\mathrm{A}}$ represents 'after dispersal'. $[S]_{\mathrm{tot}}^{i\mathrm{A}}$ (or $[I]_{\mathrm{tot}}^{i\mathrm{A}}$) represents the number of S-individuals (or I-individuals) immediately after dispersal.

$[S]_{\mathrm{tot}}^{i\mathrm{A}}$ and $[I]_{\mathrm{tot}}^{i\mathrm{A}}$ can be calculated by standard algebra. In the home subpopulation, it is calculated as

$$\begin{pmatrix} [S]_{\mathrm{tot}}^{0\mathrm{A}} \\ [I]_{\mathrm{tot}}^{0\mathrm{A}} \end{pmatrix} = \begin{pmatrix} RNJ(1-x_S^{0\mathrm{R}}) + [S]_{\mathrm{m}}^{1\mathrm{A}} \\ (1-R)NJ(1-x_I^{0\mathrm{R}}) + [I]_{\mathrm{m}}^{1\mathrm{A}} \end{pmatrix} \qquad \text{(a-3)}$$

and in another subpopulation,

$$\begin{pmatrix} [S]_{\text{tot}}^{1\text{A}} \\ [I]_{\text{tot}}^{1\text{A}} \end{pmatrix} = \begin{pmatrix} RNJ(1-x_S^1)+[S]_{\text{m}}^{1\text{A}} \\ (1-R)NJ(1-x_I^1)+[I]_{\text{m}}^{1\text{A}} \end{pmatrix}. \quad \text{(a-4)}$$

(a-2) Transition and selection during dispersal

During-dispersal dynamics for migrants can be written as follows:

$$\begin{pmatrix} [S]_{\text{m}}^{i\text{A}} \\ [I]_{\text{m}}^{i\text{A}} \end{pmatrix} = \underbrace{\begin{pmatrix} 1 & 0 \\ 0 & 1-\varepsilon_{\text{Immig}} \end{pmatrix}}_{=:\mathbf{X}_{\text{Immig}}} \underbrace{\begin{pmatrix} 1-\alpha^{\text{D}} & \beta^{\text{D}} \\ \alpha^{\text{D}} & 1-\beta^{\text{D}} \end{pmatrix}}_{=:\mathbf{P}^{\text{D}}} \underbrace{\begin{pmatrix} 1 & 0 \\ 0 & 1-\varepsilon_{\text{Emig}} \end{pmatrix}}_{=:\mathbf{X}_{\text{Emig}}} \begin{pmatrix} p[S]_{\text{m}}^{i\text{B}} \\ p[I]_{\text{m}}^{i\text{B}} \end{pmatrix}, \quad \text{(a-5)}$$

where $[S]_{\text{m}}^{i\text{B}}$ (or $[I]_{\text{m}}^{i\text{B}}$) represents the number of susceptible (or infected, respectively) migrants before dispersal, $[S]_{\text{m}}^{\text{B}}$; the superscript $^{\text{B}}$ indicates 'before dispersal'. The subscript $_{\text{m}}$ emphasises that they are calculated only for migrants (excluding philopatric individuals). Note that both of the migrant components are multiplied by the basic dispersal success probability, p. The first matrix, $\mathbf{X}_{\text{Immig}}$, denotes the selection against infected immigrants. In the second matrix, $\mathbf{P}^{\text{D}}$, the components α^{D} and β^{D} indicate the transition probabilities from one state to the other. For example, β^{D} represents the probability that dispersed individuals become susceptible during dispersal given that they are infected before dispersal. The third matrix, $\mathbf{X}_{\text{Emig}}$, denotes the selection against infected emigrants.

From (a-2)–(a-5), we can write the overall state transitions in the home subpopulation in a matrix form as

$$\begin{pmatrix} [S]_{\text{tot}}^{0\text{C}} \\ [I]_{\text{tot}}^{0\text{C}} \end{pmatrix} = \mathbf{P}^{\text{A}} \left\{ \underbrace{\begin{pmatrix} (1-R)NJ(1-x_S^{0\text{R}}) \\ RNJ(1-x_I^{0\text{R}}) \end{pmatrix}}_{\text{philopatry}} + \underbrace{\mathbf{X}_{\text{Immig}}\mathbf{P}^{\text{D}}\mathbf{X}_{\text{Emig}} \begin{pmatrix} (1-R)pNJx_S^1 \\ pRNJx_I^1 \end{pmatrix}}_{\text{migrant}} \right\}, \quad \text{(a-6)}$$

where

$$\mathbf{X}_{\text{Emig}} := \begin{pmatrix} 1 & 0 \\ 0 & 1-\varepsilon_{\text{Emig}} \end{pmatrix} \quad \text{(a-7)}$$

denotes initial probability. The dynamics in another subpopulation can be written in the same way (by changing both superscripts $^{0\text{R}}$ and ^{0} to ^{1}):

$$\begin{pmatrix} [S]^{1\text{C}}_{\text{tot}} \\ [I]^{1\text{C}}_{\text{tot}} \end{pmatrix} = \mathbf{P}^{\text{A}} \left\{ \underbrace{\begin{pmatrix} (1-R)NJ(1-x_S^1) \\ RNJ(1-x_I^1) \end{pmatrix}}_{\text{philopatry}} + \underbrace{\mathbf{X}_{\text{Immig}}\mathbf{P}^{\text{D}}\mathbf{X}_{\text{Emig}} \begin{pmatrix} (1-R)pNJx_S^1 \\ pRNJx_I^1 \end{pmatrix}}_{\text{migrant}} \right\} \quad \text{(a-8)}$$

(a-3) Fitness measure, w

In this setup, we have the fitness function *w*:

$$w = \frac{[S]^{\bullet\text{C}}_{\text{p}} + (1-\delta)[I]^{\bullet\text{C}}_{\text{p}}}{[S]^{0\text{C}}_{\text{tot}} + (1-\delta)[I]^{0\text{C}}_{\text{tot}}} + \frac{[S]^{\bullet\text{C}}_{\text{m}} + (1-\delta)[I]^{\bullet\text{C}}_{\text{m}}}{[S]^{1\text{C}}_{\text{tot}} + (1-\delta)[I]^{1\text{C}}_{\text{tot}}}, \text{ (a-9)}$$

where the superscript $^{\bullet}$ indicates the 'focal adult' with

$$[S]^{\bullet\text{C}}_{\text{p}} + (1-\delta)[I]^{\bullet\text{C}}_{\text{p}} = (1-R)J(1-\delta\alpha^{\text{A}})(1-x_S^{\bullet}) + RJ\{1-\delta(1-\beta^{\text{A}})\}(1-x_I^{\bullet}) \quad \text{(a-10)}$$

and

$$[S]^{\bullet\text{C}}_{\text{m}} + (1-\delta)[I]^{\bullet\text{C}}_{\text{m}} = (1-R)J(1-\delta\alpha^{\text{A}})(1-C_S)x_S^{\bullet} + RJ\{1-\delta(1-\beta^{\text{A}})\}(1-C_I)x_S^{\bullet},$$

where we used the notations C_S and C_I:

$$C_S = 1 - p\left(1-\alpha^{\text{D}} + \alpha^{\text{D}}(1-\varepsilon_{\text{Immig}})G_{\text{p}I/\text{p}S}\right) \quad \text{(a-11)}$$

and

$$C_I = 1 - p\left(\frac{\beta^{\text{D}}}{G_{\text{p}I/\text{p}S}} + (1-\beta^{\text{D}})(1-\varepsilon_{\text{Immig}})\right)(1-\varepsilon_{\text{Emig}}) \text{ . (a-12)}$$

These can be regarded as effective values of *S* for dispersal associated with impact of parasite infection. In the denominator of the first (or second) component of *w*, we express the total number of individuals in the home (or away) subpopulation, with the

asymmetric competition described by (1–δ). We set a conventional assumption for $0 \le C_S$ and $0 \le C_I$, which is not always the case, particularly when $G_{\mathrm{p}I/\mathrm{p}S}$ takes an extreme value. For example, permitting $G_{\mathrm{p}I/\mathrm{p}S} \to +0$, we can see that C_I diverges to –∞, as long as $\beta^{\mathrm{D}} > 0$. We can see that the extremity of $G_{\mathrm{p}I/\mathrm{p}S} \to +0$ means $\delta \approx 1$ (obligate mortality by parasite) with $\beta^{\mathrm{A}} \approx 1$ (obligate recovery after dispersal), which is unfeasible. Similarly, $G_{\mathrm{p}I/\mathrm{p}S} \to +\infty$ means $\delta \approx 1$ with $\alpha^{\mathrm{A}} \approx 1$ (obligate infection after dispersal), which is again unfeasible. Therefore, we restrict ourselves to considering a 'moderate' value of costs of dispersal, although the extension for negative *C*'s is possible.

In an explicit form, w_{HOME} can be written as

$$\frac{1}{N}\cdot\frac{(1-R)(1-\delta\alpha^{\mathrm{A}})(1-x_S^{\bullet})+R\{1-\delta(1-\beta^{\mathrm{A}})\}(1-x_I^{\bullet})}{(1-R)(1-\delta\alpha^{\mathrm{A}})\{1-x_S^{0\mathrm{R}}+(1-C_S)x_S^{1}\}+R\{1-\delta(1-\beta^{\mathrm{A}})\}\{1-x_I^{0\mathrm{R}}+(1-C_I)x_S^{1}\}} \quad \text{(a-13)}$$

and w_{AWAY} can be written as

$$\frac{1}{N}\cdot\frac{(1-R)(1-\delta\alpha^{\mathrm{A}})(1-C_S)x_S^{\bullet}+R\{1-\delta(1-\beta^{\mathrm{A}})\}(1-C_I)x_S^{\bullet}}{(1-R)(1-\delta\alpha^{\mathrm{A}})\{1-x_S^{0\mathrm{R}}+(1-C_S)x_S^{1}\}+R\{1-\delta(1-\beta^{\mathrm{A}})\}\{1-x_I^{0\mathrm{R}}+(1-C_I)x_S^{1}\}}$$

(a-14)

To scale the prevalence, we define the effective prevalence as

$$R_{\mathrm{E}} := \frac{R\{1-\delta(1-\beta^{\mathrm{A}})\}}{(1-R)\{1-\delta\alpha^{\mathrm{A}}\}+R\{1-\delta(1-\beta^{\mathrm{A}})\}} = \frac{RG_{\mathrm{p}I/\mathrm{p}S}}{(1-R)+RG_{\mathrm{p}I/\mathrm{p}S}}. \quad \text{(a-15)}$$

In the denominator, the fraction $(1-R)$ of individuals is susceptible before dispersal, but goes dead with the fraction $\delta\alpha^{\mathrm{A}}$ because of parasite-induced mortality (parasite virulence) after transition, but before competition. On the other hand, the second term in the denominator (which is equivalent to the numerator) indicates the fraction *R* of individuals that are infected before dispersal, but die before competition due to virulence after transition at rate $\delta(1-\beta^{\mathrm{A}})$. From the second expression, we can

interpret the effective prevalence as the ratio of individuals that are infected just before dispersal, taking into account the effect of relative competition ability, t. It is obvious that R_{E} is an increasing function of t, and thus, that of α^{A} and β^{A}. This scaling reduces the fitness function to

$$w_{\mathrm{HOME}} = \frac{(1-R_{\mathrm{E}})(1-x_S^{\bullet}) + R_{\mathrm{E}}(1-x_I^{\bullet})}{(1-R_{\mathrm{E}})\{1-x_S^{0\mathrm{R}} + (1-C_S)x_S^1\} + R_{\mathrm{E}}\{1-x_I^{0\mathrm{R}} + (1-C_I)x_S^1\}} \quad \text{(a-16)}$$

and

$$w_{\mathrm{AWAY}} = \frac{(1-R_{\mathrm{E}})(1-C_S)x_S^{\bullet} + R_{\mathrm{E}}(1-C_I)x_S^{\bullet}}{(1-R_{\mathrm{E}})\{1-x_S^{0\mathrm{R}} + (1-C_S)x_S^1\} + R_{\mathrm{E}}\{1-x_I^{0\mathrm{R}} + (1-C_I)x_S^1\}}. \quad \text{(a-17)}$$

(b) Calculation of bias predictor, Δ

Our analysis showed that the dispersal bias is determined by the sign of the predictor

$$\Delta := \left(\frac{\beta^{\mathrm{D}}}{G_{\mathrm{p}I/\mathrm{p}S}} + (1-\beta^{\mathrm{D}})(1-\varepsilon_{\mathrm{Emig}})\right)(1-\varepsilon_{\mathrm{Immig}}) - \left((1-\alpha^{\mathrm{D}}) + \alpha^{\mathrm{D}}(1-\varepsilon_{\mathrm{Emig}})G_{\mathrm{p}I/\mathrm{p}S}\right). \quad \text{(b-1)}$$

The derivation is obtained from the definitions of C_S and C_I:

$$\begin{aligned} C_S - C_I &= 1 - p\left(1-\alpha^{\mathrm{D}} + \alpha^{\mathrm{D}}(1-\varepsilon_{\mathrm{Immig}})t\right) - 1 + p\left(\frac{\beta^{\mathrm{D}}}{t} + (1-\beta^{\mathrm{D}})(1-\varepsilon_{\mathrm{Immig}})\right)(1-\varepsilon_{\mathrm{Emig}}) \\ &= p\left(\frac{\beta^{\mathrm{D}}}{t} + (1-\beta^{\mathrm{D}})(1-\varepsilon_{\mathrm{Immig}})\right)(1-\varepsilon_{\mathrm{Emig}}) - p\left(1-\alpha^{\mathrm{D}} + \alpha^{\mathrm{D}}(1-\varepsilon_{\mathrm{Immig}})t\right) \\ &= p\Delta \end{aligned}$$

(b-2)

The proof that this value predicts the dispersal bias is shown in Appendix (d) below. Note that the equality $\Delta = 0$ (i.e. $C_S = C_I$) implies the neutrality of evolutionary dynamics for (z_S, z_I), but it does not hold for generic parameters; therefore, we exclude the equality $\Delta = 0$.

Δ varies depending on each factor. Here we demonstrate its dependence on α^{D} and β^{D} (as discussed in the main text). The differentiation of Δ at $\varepsilon_{\mathrm{Emig}} = \varepsilon_{\mathrm{Immig}} = 0$

with respect to α^{D} or β^{D} yields

$$\frac{\partial \Delta}{\partial \alpha^{\mathrm{D}}} = 1 - G_{\mathrm{p}I/\mathrm{p}S} \quad \text{(b-3)}$$

and

$$\frac{\partial \Delta}{\partial \beta^{\mathrm{D}}} = \frac{1}{G_{\mathrm{p}I/\mathrm{p}S}} - 1 = \frac{1}{G_{\mathrm{p}I/\mathrm{p}S}}(1 - G_{\mathrm{p}I/\mathrm{p}S}) . \text{(b-4)}$$

Thus, $\mathrm{Auto}^{\mathrm{A}} > 0$ indicates Δ is an increasing function of α^{D} or β^{D} since $(1 - G_{\mathrm{p}I/\mathrm{p}S}) = \mathrm{Auto}^{\mathrm{A}}$.

(c) ES-dispersal strategy and relatedness coefficient : direct fitness expansion

In order to analyse convergence stability, we employ the direct fitness approach (Taylor & Frank 1996; Frank 1998; Rousset 2004). First, we partially differentiate *w* with respect to the deviations (ξ_S, ξ_I):

$$\frac{\partial w}{\partial \xi_i} = \frac{\partial w}{\partial x_i^{\bullet}} + r\frac{\partial w}{\partial x_i^{0\mathrm{R}}} = r\frac{1-u^*}{\{1-u^*+v^*\}^2} - \frac{C_i}{\{1-u^*+v^*\}} \quad \text{(c-1)}$$

for $i = S, I$, evaluated at $\mathbf{x}^{\bullet} = \mathbf{x}^{\mathbf{0R}} = \mathbf{x}^{\mathbf{1}} = \mathbf{z}^*$. Here, u^* (or v^*) is the average emigration rate (or immigration rate, respectively):

$$u^* = (1 - R_{\mathrm{E}})z_S^* + R_{\mathrm{E}}z_I^* , \text{(c-2)}$$

$$v^* = (1 - R_{\mathrm{E}})(1 - C_S)z_S^* + R_{\mathrm{E}}(1 - C_I)z_I^* . \text{(c-3)}$$

Note that the within-subpopulation average is calculated with respect to the effective prevalence $1 - R_{\mathrm{E}}$ and R_{E}. The asterisk (*) indicates 'at equilibrium', but hereafter, we drop it for the simplicity of expressions. The backwards migration rate, defined as the probability that a randomly sampled individual after dispersal (but before competition) within subpopulation has its parent in another subpopulation, can be written as

$$m = \frac{v}{1 - u + v} . \text{(c-4)}$$

In other words, $1 - m$ is the probability that a randomly sampled individual within a subpopulation is native. The coefficient of relatedness *r* (Hamilton 1964a, b) is

described as

$$r := F_{\mathrm{ST}}^{\mathrm{R}} := \frac{\mathrm{d}x_S^{0\mathrm{R}}}{\mathrm{d}\xi_S} = \frac{\mathrm{d}x_I^{0\mathrm{R}}}{\mathrm{d}\xi_I}, \text{ (c-5)}$$

with $F_{\mathrm{ST}}^{\mathrm{R}} = \{1+(N-1)F_{\mathrm{ST}}\}/N$ and $F_{\mathrm{ST}} = (1-m)^2 F_{\mathrm{ST}}^{\mathrm{R}}$ (Rousset 2004), which suggests that there are infinitely many subpopulations. Therefore, the allelic correlation is null between different subpopulations. The notation $^{\mathrm{R}}$ is again used to emphasise that two individuals are sampled from the home subpopulation with 'replacement'. Relatedness r is the same for S-individuals and I-individuals, because in this model the class-division into S and I is not genetically inherent, but occurs at random despite genetic backgrounds. Note that the coefficient of relatedness, r, needs to be evaluated at the resident strategy $\mathbf{x}^{\bullet} = \mathbf{x}^{0\mathrm{R}} = \mathbf{x}^{1}$ when we produce the evolutionary vector field (see Figure 2).

(c-1) When Δ is positive

When Δ is positive, the ES-dispersal strategy is biased towards infected individuals (I-biased). The solution (z_S^*, z_I^*) is

$$z_S^* = \begin{cases} \dfrac{r_S - C_S - R_{\mathrm{E}}(r_S - C_S C_I)}{(1-R_{\mathrm{E}})(r_S - C_S^2)} & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_S - C_S}{r_S - C_S C_I} \\ 0 & \text{if} \quad \dfrac{r_S - C_S}{r_S - C_S C_I} < R_{\mathrm{E}} < 1 \end{cases} \text{ (c-6)}$$

and

$$z_I^* = \begin{cases} 1 & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_I - C_I}{r_I - C_I^2} \\ \dfrac{r_I - C_I}{R_{\mathrm{E}}(r_I - C_I^2)} & \text{if} \quad \dfrac{r_I - C_I}{r_I - C_I^2} < R_{\mathrm{E}} < 1 \end{cases}, \text{ (c-7)}$$

where the coefficient of relatedness r_S and r_I are defined as

$$r_S := \frac{1+\sqrt{1+4N(N-1)C_S^2}}{2N} \quad \text{(c-8)}$$

and

$$r_I := \frac{1+\sqrt{1+4N(N-1)C_I^2}}{2N} . \quad \text{(c-9)}$$

The coefficient of relatedness also varies as R_{E} changes:

$$F_{\mathrm{ST}}^{\mathrm{R}} = r = \begin{cases} r_S & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{r_S - C_S}{r_S - C_S C_I} \\ \dfrac{1}{N-(N-1)\left(\dfrac{1-R_{\mathrm{E}}}{1-R_{\mathrm{E}}C_I}\right)^2} & \text{if} \quad \dfrac{r_S - C_S}{r_S - C_S C_I} < R_{\mathrm{E}} < \dfrac{r_I - C_I}{r_I - C_I^2} \\ r_I & \text{if} \quad \dfrac{r_I - C_I}{r_I - C_I^2} < R_{\mathrm{E}} < 1 \end{cases} \quad \text{(c-10)}$$

In particular, when $N = 1$, the coefficient of relatedness is always unity.

(c-2) When Δ is negative

Similar to (c-1), we have

$$z_S^* = \begin{cases} \dfrac{1}{1-R_{\mathrm{E}}} \dfrac{r_S - C_S}{r_S - C_S^2} & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{C_S(1-C_S)}{r_S - C_S^2} \\ 1 & \text{if} \quad \dfrac{C_S(1-C_S)}{r_S - C_S^2} < R_{\mathrm{E}} < 1 \end{cases} , \quad \text{(c-11)}$$

$$z_I^* = \begin{cases} 0 & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{(1-C_S)C_I}{r_I - C_S C_I} \\ \dfrac{r_I - C_I - (1-R_{\mathrm{E}})(r_I - C_S C_I)}{R_{\mathrm{E}}(r_I - C_I^2)} & \text{if} \quad \dfrac{(1-C_S)C_I}{r_I - C_S C_I} < R_{\mathrm{E}} < 1 \end{cases} , \quad \text{(c-12)}$$

and

$$F_{\mathrm{ST}}^{\mathrm{R}} = r = \begin{cases} r_S & \text{if} \quad 0 < R_{\mathrm{E}} < \dfrac{C_S - C_S^2}{r_S - C_S^2} \\ \dfrac{1}{N-(N-1)(\dfrac{R_{\mathrm{E}}}{1-C_S+R_{\mathrm{E}}C_S})^2} & \text{if} \quad \dfrac{C_S - C_S^2}{r_S - C_S^2} < R_{\mathrm{E}} < \dfrac{C_I(1-C_S)}{r_I - C_I C_S} \\ r_I & \text{if} \quad \dfrac{C_I(1-C_S)}{r_I - C_I C_S} < R_{\mathrm{E}} < 1 \end{cases} \quad \text{. (c-13)}$$

(d) Derivation of the solution and proof of the evolutionary stability

We need to prove the maximality of the solution given in Appendix (c), which is left unsolved in Iritani & Iwasa (2014). For the proof, we use the theorem given by Kuhn & Tucker (1951). The direct calculation is tedious, and thus we operate the following linear transformation beforehand:

$$\mathbf{y}^{\bullet} = \begin{pmatrix} u^{\bullet} \\ v^{\bullet} \end{pmatrix} := \begin{pmatrix} 1-R_{\mathrm{E}} & R_{\mathrm{E}} \\ (1-R_{\mathrm{E}})(1-C_S) & R_{\mathrm{E}}(1-C_I) \end{pmatrix} \begin{pmatrix} x_S^{\bullet} \\ x_I^{\bullet} \end{pmatrix}$$
$$\mathbf{y}^{*} = \begin{pmatrix} u^{*} \\ v^{*} \end{pmatrix} := \begin{pmatrix} 1-R_{\mathrm{E}} & R_{\mathrm{E}} \\ (1-R_{\mathrm{E}})(1-C_S) & R_{\mathrm{E}}(1-C_I) \end{pmatrix} \begin{pmatrix} z_S^{*} \\ z_I^{*} \end{pmatrix} \quad \text{(d-1)}$$

where the notations for u and v are the emigration rate and immigration rate, respectively (see Eqs. (c-2)). We write the square matrix on the right hand side as **T**. In order to incorporate the kin selection effect, we calculate the subpopulation mean strategy and get

$$u^{0\mathrm{R}} = ru^{\bullet} + (1-r)u^{*}$$
$$v^{0\mathrm{R}} = rv^{\bullet} + (1-r)v^{*} \quad \text{(d-2)}$$

where the notation $^{0\mathrm{R}}$ is used in the same way as above, and r is the coefficient of relatedness. This can be justified by assuming weak selection. Then the fitness function of the focal individual can be rewritten as

$$w[\mathbf{x}^{\bullet},\mathbf{x}^{0\mathrm{R}}]=W(u^{\bullet},v^{\bullet},u^{0\mathrm{R}},v^{0\mathrm{R}})=W[\mathbf{y}^{\bullet},\mathbf{y}^{0\mathrm{R}}] = \frac{1-u^{\bullet}}{1-u^{0\mathrm{R}}+v^{*}}+\frac{v^{\bullet}}{1-u^{*}+v^{*}}$$
$$= \frac{1-u^{\bullet}}{1-ru^{\bullet}-(1-r)u^{*}+v^{*}}+\frac{v^{\bullet}}{1-u^{*}+v^{*}}$$

(d-3)

where we write the fitness as W rather than w, to clarify the difference between the variables (which are **y**'s rather than **x**'s). Here, because we are interested in evolutionary stability rather than convergence stability, we formulate it as if u^{*} and v^{*} are not variables but parameters; therefore, we write $W(u^{\bullet},v^{\bullet},u^{0\mathrm{R}},v^{0\mathrm{R}})=W[\mathbf{y}^{\bullet},\mathbf{y}^{0\mathrm{R}}]$ simply as $W(u^{\bullet},v^{\bullet})=W[\mathbf{y}^{\bullet}]$. Also note that the constraint or the feasible region, which is a unit square for $0\le x_S\le 1$ and $0\le x_I\le 1$, needs to be written in terms of u and v (see below). Calculation of the fitness gradient ∇W of the fitness $W(u,v)$ evaluated at $\mathbf{y}^{*}$ yields

$$\nabla W=\begin{pmatrix} \dfrac{-(1-u^{*}+v^{*})+r_S(1-u^{*})}{(1-u^{*}+v^{*})^2} \\ \dfrac{1}{(1-u^{*}+v^{*})} \end{pmatrix}. \quad \text{(d-4)}$$

Apparently, ∇W is never null because the second component is always positive.

Second, to depict the domain of $W(u,v)$, we calculate the orientation, i.e. the determinant of **T**:

$$\det\mathbf{T}=R_{\mathrm{E}}(1-R_{\mathrm{E}})(C_S-C_I)\propto\Delta, \quad \text{(d-5)}$$

where $\propto$ is used to indicate 'positively proportional to'. This estimation means that state-biased dispersal is determined by whether the orientation of **T** is preserved or not. For the sake of simplicity, we restrict ourselves to assuming Δ is positive.

When Δ is positive, we can write the constraints as

$$
\begin{aligned}
g_1 &:= -(1-C_S)u + v \geq 0 \\
g_2 &:= -(1-C_I)(u-1) + v - \{1-(1-R_E)C_S - R_E C_I\} \geq 0 \\
g_3 &:= (1-C_S)(u-1) + \{1-(1-R_E)C_S - R_E C_I\} - v \geq 0 \\
g_4 &:= (1-C_I)u - v \geq 0
\end{aligned} \tag{d-6}
$$

Let $\mathcal{Y}$ be the parallelogram represented by the above constraints (d-6). Obviously, $\mathcal{Y}$ is convex in $\mathbf{R}^2$. Because **T** is a linear (and thus continuous) mapping, the image of the boundary of a unit square corresponds to the boundary of $\mathcal{Y}$. The gradient vectors are given as

$$
\nabla g_1 = \begin{pmatrix} -(1-C_S) \\ 1 \end{pmatrix}, \nabla g_2 = \begin{pmatrix} -(1-C_I) \\ 1 \end{pmatrix}, \nabla g_3 = \begin{pmatrix} 1-C_S \\ -1 \end{pmatrix}, \nabla g_4 = \begin{pmatrix} 1-C_I \\ -1 \end{pmatrix}. \tag{d-7}
$$

(d-1) Deviation of the candidate ESS

Because the gradient ∇W is never null, we can see that the ESS is on the boundary if it exists. To derive the ES solution, the following theorem by Kuhn & Tucker (1951) is applied. We rewrite the theorem in terms of our system under study:

Theorem 1 [Kuhn–Tucker: necessary condition for maximality]

Let $K := \{1 \leq i \leq 4 ; g_i(\mathbf{y}^*) = 0\}$ *be a set of indices for the constraints* $g_1, g_2, g_3, g_4 \geq 0$. *Assume that* $W[\mathbf{y}]$ *and each of* $\{g_k ; k \in K\}$ *are differentiable, and that each* $\{g_k ; k \notin K\}$ *is continuous. If* $\nabla g_k[\mathbf{y}^*]$ *are linearly independent for* $k \in K$ *and if* $\mathbf{y}^*$ *is evolutionarily stable, then there exists Lagrange multipliers* $\lambda_k \geq 0$ *for* $k \in K$ *such that*

$$
\nabla W[\mathbf{y}^*] + \sum_{k \in K} \lambda_k \nabla g_k[\mathbf{y}^*] = \mathbf{0}. \tag{d-8}
$$

Using this theorem, we can analytically investigate the candidate ESS $\mathbf{y} = \mathbf{y}^*$. As an example, we derive the solution in the case where (z_S^*, z_I^*) lies on the boundary for $z_S^* = 0$ (i.e. $g_4 = 0$). After substitution for gradients and $k = 4$, the Eq. (d-8) yields

$$\begin{pmatrix} \dfrac{-(1-u^*+v^*)+r(1-u^*)}{(1-u^*+v^*)^2} \\ \dfrac{1}{1-u^*+v^*} \end{pmatrix} + \lambda_4 \begin{pmatrix} 1-C_I \\ -1 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}. \text{ (d-9)}$$

Here, the equality $g_4 = (1-C_I)u - v = 0$ yields $1-u+v = 1-C_I u$, and hence, we have

$$\begin{pmatrix} \dfrac{-(1-C_I u^*)+r(1-u^*)}{(1-C_I u^*)^2} \\ \dfrac{1}{1-C_I u^*} \end{pmatrix} + \lambda_4 \begin{pmatrix} 1-C_I \\ -1 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}. \text{ (d-10)}$$

By a standard method of linear algebra, we have a desired result:

$$u^* = \frac{r-C_I}{r-C_I^2}, \text{ (d-11)}$$

where $r = r_I$. Note that $\lambda_4 = 1/(1-C_I u^*) > 0$ holds. In addition, we need to check whether this candidate solution satisfies other constraints. The satisfaction of $g_1, g_2 \geq 0$ is obvious; as for $g_3 \geq 0$, the substitution of Eq. (d-11) into (d-6) yields

$$\begin{aligned} (1-C_S)(u^*-1) - v^* + 1 - (1-R_{\mathrm{E}})C_S - R_{\mathrm{E}}C_I \geq 0 \quad &\Leftrightarrow \quad (C_S - C_I)R_{\mathrm{E}} \geq (C_S - C_I)u^* \\ &\Leftrightarrow \qquad R_{\mathrm{E}} \geq u^* = \frac{r_I - C_I}{r_I - C_I^2} \end{aligned} \quad \text{(d-12)}$$

since $C_S - C_I > 0$. Thus, in terms of (z_S^*, z_I^*), the (unique) candidate ESS is

$$(z_S^*, z_I^*) = \left(0, \frac{r_I - C_I}{R_{\mathrm{E}}(r_I - C_I^2)}\right) \quad \text{(d-13)}$$

for $R_{\mathrm{E}} \geq (r_I - C_I)/(r_I - C_I^2)$.

(d-2) Proof of the evolutionary stability (maximality)

Next, we introduce a theorem asserting the maximality of $\mathbf{y}^*$, by Kuhn & Tucker

(1951):

Theorem 2 [Kuhn–Tucker: sufficient condition for maximality]

Let $\mathbf{y}^* = (u^*, v^*) \in \mathcal{Y}$ *be a candidate ESS (i.e. SS) for the fitness function* $W : \mathcal{Y} \to \mathbf{R}$, *which is semi-concave at* $\mathbf{y}^*$. *Consider the constraints for* **g**, *defined as Eqs. (d-7). In addition, assume that the equality holds at* $k \in K := \{1 \le i \le 4 ; g_i(t^*) = 0\}$. *Then* $\mathbf{y}^*$ *is the ESS if it satisfies the following property:*

There are $\lambda_i \ge 0$ $(i = 1,2,3,4)$ *such that*

$$\nabla W[\mathbf{y}^*] + \sum_{k \in K} \lambda_k \nabla g_k[\mathbf{y}^*] = \mathbf{0} . \text{(d-14)}$$

The quasi-concavity of W is easy to prove; more precisely, W is concave on $\mathcal{Y}$ (concavity of W implies its quasi-concavity). In order to demonstrate an example, we show that $(z_S^*, z_I^*) = \left(\frac{r_S - C_S - R_E(r_S - C_S C_I)}{(1 - R_E)(r_S - C_S^2)}, 1 \right)$ is evolutionarily stable for $R_E \le (r_S - C_S) / (r_S - C_S C_I)$.

First, we rewrite the candidate ESS in terms of u and v:

$$\begin{aligned} u^* &= (1 - R_E) x_S + R_E x_I = \frac{r_S - C_S + R_E C_S (C_I - C_S)}{r_S - C_S^2} \\ v^* &= (1 - R_E)(1 - C_S) x_S + R_E (1 - C_I) x_I = \frac{r_S - C_S}{r_S - C_S^2} \{1 - (1 - R_E) C_S - R_E C_I\} \end{aligned} \quad \text{(d-15)}$$

Substitution of $\left(u^*, v^* \right)$ into Eq. (d-4) yields

$$\nabla W = \begin{pmatrix} \dfrac{-C_S + C_S^2}{r_S (1 - u^*)} \\ \dfrac{C_S}{r_S (1 - u^*)} \end{pmatrix} . \text{(d-16)}$$

Second, because $\mathbf{y}^*$ meets the equality of the constraints for $g_3 = 0$, and from the theorem, we have

$$\begin{pmatrix} \dfrac{-C_S + C_S^2}{r_S(1-u^*)} \\ \dfrac{C_S}{r_S(1-u^*)} \end{pmatrix} + \lambda_3 \begin{pmatrix} (1-C_S) \\ -1 \end{pmatrix} = \mathbf{0}, \text{ (d-17)}$$

which yields $\lambda_3 = C_S / \{r_S(1-u^*)\} > 0$ under the assumption that $C_S > 0$ and $1-u^* > 0$. We also need to check that this solution satisfies the other constraints, $g_1, g_2, g_4 \geq 0$. Obviously, $g_1 \geq 0$ and $g_2 \geq 0$ hold. To see the condition for $g_4[\mathbf{y}^*] \geq 0$, we have:

$$(1-C_I)u^* - v^* \quad = \quad (1-R_{\mathrm{E}})(C_S - C_I)z_S^* \quad \geq 0 \text{ . (d-18)}$$

Because $(1-R_{\mathrm{E}})(C_S - C_I) \geq 0$ by the assumption that $\Delta > 0$, solving $z_S^* \geq 0$ yields

$$R_{\mathrm{E}} \leq \frac{r_S - C_S}{r_S - C_S^2}, \text{ (d-19)}$$

which is consistent with our case.

In other cases, e.g. where $(z_S^*, z_I^*) = (1,0)$, we can employ the same method as above; the condition

$$\frac{r_S - C_S}{r_S - C_S C_I} < R_{\mathrm{E}} < \frac{r_I - C_I}{r_I - C_I^2} \quad \text{(d-20)}$$

can be given by solving the condition that Lagrange multipliers λ_3 and λ_4 are both positive.

(e) Calculation for autocorrelation

We begin with the general formulation of the calculation of autocorrelation for the two-dimensional dynamics described by a transition matrix **P**:

$$\mathbf{P} := \begin{pmatrix} 1-\alpha & \beta \\ \alpha & 1-\beta \end{pmatrix}. \text{ (e-1)}$$

Let Q_τ denote the state (*S* or *I*) at time τ so that $Q_\tau = 1$ for state-S and $Q_\tau = 0$ for

state-I, and let H_S (and H'_S) denote the probability that a randomly sampled individual is in state-S at time τ (and $\tau+1$, respectively). The correlation coefficient of statistical association between disease state at time τ and $\tau+1$ is given by

$$\begin{aligned} \mathrm{Auto}[\mathbf{P}] &= \frac{\mathrm{Cov}[Q_\tau, Q_{\tau+1}]}{\sqrt{\mathrm{Var}[Q_\tau]\cdot\mathrm{Var}[Q_{\tau+1}]}} \\ &= \left(1-\alpha-\beta\right)\sqrt{\frac{H'_S(1-H'_S)}{H_S(1-H_S)}} \quad \text{(e-2)} \\ &\propto \left(1-\alpha-\beta\right) \end{aligned}$$

Hence, applying Eq. (e-2) for $\mathbf{P}=\mathbf{P}^{\mathrm{A}}$, immediately we have

$$\mathrm{Auto}[\mathbf{P}^{\mathrm{A}}] \propto 1-\alpha^{\mathrm{A}}-\beta^{\mathrm{A}}. \quad \text{(e-3)}$$

In the main text, we defined the right hand side in Eq. (e-3) as the autocorrelation of disease states, because $1-\alpha^{\mathrm{A}}-\beta^{\mathrm{A}}$ is sufficient for our purpose of predicting the dispersal bias; thus, all that matters in our analyses is the sign of $\mathrm{Auto}^{\mathrm{A}}$.

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Chapter 3.

Disease state-dependent dispersal and the avoidance of transmission among kin.

This study is submitted by Ryosuke Iritani as a single author.

1 Introduction

Sociality considerably increases the parasite burden and risk of parasite infection (Alexander 1974 [2]). Because parasite infection has major impacts on diverse aspects of host life history, host species have developed mechanisms to avoid and/or resist infection, including dispersal (Loehle 1995 [38]; also see pp.169-179 in Clobert (2001) [9]). Dispersal plays a fundamental role in the establishment of sociality, because it affects the genetic composition of social groups as well as the pandemics of infectious diseases within and/or across groups. In turn, it is also widely accepted that population genetic structure greatly affects the evolution of dispersal by interaction among kin, including competition (Hamilton & May 1977 [26], Frank 1986 [17], Taylor 1988 [55]), and mating (Perrin & Mazalov 2000 [48]). Thus, dispersal and parasite-induced selection are closely linked in the context of sociality.

Social evolution is primarily driven by kin selection (Hamilton 1964a, b [23, 24] Frank 1998[19], Marshall 2015[41]). In support of the predictions of inclusive fitness theory, ample evidence suggests that kin selection drives the evolution of a variety of social behaviours that can incur potential costs, e.g., dispersal (Frank 1986 [17], Taylor 1988 [55], Frank 1998 [19], Rousset 2004 [50]), helping (altruism) and harming (spite) (Lehmann et al. 2006 [36], West & Gardner 2010 [61]), sex ratio (Hamilton 1967 [25]), and the virulence-resistance coevolution in host-parasite interactions (Frank 1996 [18]). One of the key assumptions underlying inclusive fitness theory is that social behaviours occur among kin due to limited dispersal. Hence, viscosity is crucial to understanding the evolution of sociality.

Viscosity also has a profound effect on sociality in the presence of horizontally transmissible parasites, because horizontal transmission can occur between closely related individuals in social organisms. This can impose large costs, because transmission of parasites among closely related individuals is disadvantageous in terms of inclusive fitness. In this vein, we can expect that condition-dependent dispersal (‘disease state-dependent dispersal’) can play a central role: for example, if infected individuals disperse from their natal group, then the risk of transmission among kin may be mitigated; however, the opposite may also be the case, because uninfected individuals can ‘escape’ from the risk of transmission from their siblings by dispersal. Hence, it is not intuitively obvious what specific patterns of disease state-dependent dispersal are likely to emerge by natural selection.

Indeed, there is empirical evidence for the emergence of disease state-dependent dispersal in

animals. Heinze & Walter (2010) [28] have suggested that in *Temnothorax* ants, infected workers have fewer social interactions and tend to leave their colony if they are infected with an entomopathogenic fungus. In addition, Kralj & Fuchs (2006) [35] have shown that honeybee workers tend to abandon their hives when infected with parasitic varroa mites. Rueppell et al. (2010) [52] performed computer simulations to demonstrate that altruistic self-removal of infected individuals from social groups can impede the spread of diseases, which is supported by their own empirical tests with honey bee populations (*Apis mellifera L.*). From a more general empirical avenue, several studies have reported two contrasting patterns for disease state-dependent dispersal: dispersal propensity is either biased towards infected individuals (I-biased dispersal) or uninfected individuals (U-biased dispersal). I-biased dispersal has been reported in a yellow-bellied marmot (*Marmota flaviventris*) population infected with several parasites, including fleas (*Oropsylla stanfordi*), lice (*Linognathoides marmotae*), and mites (family Dermanyssidae) (Van Vuren 1996), as well as in a cliff swallow (*Hirundo pyrrhonota*) population infected with hematophagous fleas (*Ceratophyllus celsus*) and swallow bugs (*Oeciacus vicarius*). In contrast, U-biased dispersal has been reported in a great tit (*Parus major*) population infected with hen fleas (*Ceratophyllus gallinae*; Heeb et al. 1999 [27]), in a money spider (*Erigone atra*) population infected with an endosymbiont (*Rickettsia*; Goodacre et al. 2009 [21]), in a chub (*Squalius cephalus*)) population infected with the larvae of duck mussels (*Anodonta anatina*; Horký et al. 2014 [29]), in a ciliate (*Paramecium caudatum*) population infected with a bacterium (*Holospora undulata*; Fellous et al. 2011 [15]), and in a cichlid (*Tropheus moorii*) population infected with flatworms *Cichlidogyrus* (Grégoir et al. 2015 [22]). There is also some evidence of the direct negative impacts of parasites on host mobility (e.g. Luong et al. 2015 [39], Bradley & Altizer 2005 [6]). In addition, the potential contributions of parasites to host dispersal or migratory behaviours have been reported (Tschirren et al. 2007 [59], Debeffe et al. 2014 [12], van Dijk et al. 2015 [60]) and the effects of parasite-driven selection on migration (MacCol & Chapman 2010 [40]). The host migration-driven genetic structure in the context of host-parasite interactions has been studied in avian species (e.g. McCoy et al. 2003 [43], Knowles et al. 2014 [33], Koprivnikar & Leung 2015 [34]; for review, see Altizer 2011 [4]). Furthermore, parasites may be used as tags for host migration behaviours (Galbreath & Hoberg 2012[20], Terui & Miyazaki 2015[57]). In general, disease state-dependent dispersal may contribute to the spatial genetic makeup of host-parasite interactions, because dispersal of the infected host will result in dispersal of the parasites ("simultaneous migration", Morgan et al. 2007 [45]). Thus, obtaining a detailed understanding of disease state-dependent dispersal is cen-

tral to the evolutionary ecology of host-parasite interactions; accordingly, the role of host-dispersal ecology in host-parasite interactions has received broad research attention (Poulin 2011, [49]).

Some theoretical studies have tackled the problem involving the evolution of host dispersal in the context of parasitism. For example, Chaianunporn & Hovestadt (2012) [7] have studied the effect of interspecific interactions on dispersal in a metacommunity context, showing that parasitism could promote dispersal in both the hosts and parasites. However, they have not considered disease state-dependent dispersal. Iritani & Iwasa (2014) and Iritani (2015) ([31], [30]) have studied the evolution of disease state-dependent dispersal, showing that differences in the timing of infection (before, during, and/or after dispersal) could modify the cost of dispersal for juveniles in each disease state, resulting in either pattern for disease state-dependent dispersal. However, they have not evaluated the effects of horizontal transmission on disease state-dependent dispersal.

In the present paper, I address the question, "how does the risk of parasite transmission among kin drive or inhibit the evolution of dispersal?" In particular, I aim to clarify which members of a natal group should disperse when the risk of infection or horizontal transmission is high: infected or uninfected individuals? To answer this question, I use the neighbour-modulated approach (Taylor & Frank 1996, [56]) on the basis of inclusive fitness theory. Toward this end, I aim to capture the effect of horizontal transmission on the evolutionary endpoints for the dispersal rates of uninfected and infected juveniles. I here consider two possibilities for the timing of horizontal transmission, either before dispersal or after dispersal (but before competition) to see how the different timing of horizontal transmission affect evolutionary endopoint of dispersal, i.e., convergently stable dispersal rate(s) (Eshel 1983[13], Rousset & Billiard 2000 [51], Rousset 2004 [50]).

2 Methods

2.1 Life cycle

Here, I demonstrate the basal model structure in the absence of horizontal transmission. My models are built on Wright's island model of dispersal (Wright 1931 [62], Hamilton & May 1977 [26]), where the entire population consists of an infinite number of islands ("social groups"). Host organism is asexual haploid, following the birth-death Moran demography. At the beginning of adulthood, N adult individuals are supported in each social group, each reproducing an infinite number (J) of offspring ($J \to \infty$) equally. Immediately after reproduction, one adult is randomly

chosen to die in each group, and juveniles become infected with probability P and remain uninfected with probability $1 - P$, wherein I assume that each juvenile randomly becomes infected from an external environmental source (e.g. via infection-borne vectors, or during foraging). Juveniles are thus categorized into two disease-states: 'uninfected' or 'infected'. Juveniles disperse conditionally according to their own disease state (right before the dispersal stage), where the dispersal rate for uninfected juveniles (or infected juveniles) is generically denoted by d_{U} (or d_{I}). Dispersal also incurs some extrinsic costs (c) so that only the fraction $1 - c$ of dispersed juveniles survive to settle in an alternative group. Finally, competition for a breeding spot (which is one out of N per group) follows to form the next generation. In the competition, infected juveniles suffer a disadvantage such that infected juvenile suffer a dicrease in competitive ability by the factor v compared to uninfected juveniles. Thus, the competitive ability for uninfected juveniles is unity, while that for infected juveniles is $1 - v$. I refer to v as parasite virulence ($0 \leq v \leq 1$). I do not consider parasite evolution.

In the baseline life cycle described above, horizontal transmission occurs either before or after dispersal (Figure 1 summarizes the life cycle). Let P' be the ratio of infected juveniles right before dispersal (i.e., the probability that a randomly sampled juvenile is infected right before dispersal). Similarly, let P'' be the ratio of infected juveniles immediately after dispersal (i.e., the probability that a randomly sampled juvenile is infected (in state-I) immediately after dispersal), and let P''' be the ratio of infected juveniles right before competition (i.e., the probability that a randomly sampled jveniles is infected right before competition). Both P'' and P''' may or may not be the functions of the dispersal rate (depending on the models under study). Therefore, the values of P'' and P''' in the mutant-native group ('focal group') are functions of the group-average dispersal rates, which I clarify below.

2.2 Dynamics of horizontal transmission

Here, I describe the dynamics of horizontal transmission. Let q_{U} (or $q_{\mathrm{I}} = 1 - q_{\mathrm{U}}$) be a generic symbol for the ratio of uninfected (or infected) juveniles in a group. I assume that such contact occurs in a one-shot, pairwise manner, and has no immediate effect on fecundity or survival. I use a discrete SI-model (Kermack & McKendrick 1927 [32]):

$$\begin{aligned} q'_{\mathrm{U}} &= q_{\mathrm{U}} - \kappa q_{\mathrm{U}} q_{\mathrm{I}}, \\ q'_{\mathrm{I}} &= q_{\mathrm{I}} + \kappa q_{\mathrm{U}} q_{\mathrm{I}}, \end{aligned} \tag{2-1}$$

where κ is a generic symbol for the probability of horizontal transmission ('transmission rate', $0 \leq \kappa \leq 1$) given that a single uninfected juvenile had physical contact with an infected juvenile (which occurs at rate q_{I}).

For horizontal transmission before dispersal, we presume $\alpha = \kappa$ with $q_{\mathrm{I}} = P$ so that:

$$P' = P + \alpha P(1 - P), \tag{2-2}$$

whereas for horizontal transmission after dispersal, we presume $\beta = \kappa$ with $q_{\mathrm{I}} = P''$ so that:

$$P''' = P'' + \beta P''(1 - P''). \tag{2-3}$$

Given the setup, we can derive the fitness W, the expected number of adult offspring of the mutant, as:

$$W := \frac{(1 - \beta v P_0'')(1 - P')(1 - d_{\mathrm{U}}^{\bullet}) + (1 - v)P'(1 - d_{\mathrm{I}}^{\bullet})}{(1 - \beta v P_0'')(1 - P')(1 - d_{\mathrm{U}}^{0} + (1 - c)d_{\mathrm{U}}) + (1 - v)P'(1 - d_{\mathrm{I}}^{0} + (1 - c)d_{\mathrm{I}})} \tag{2-4}$$

$$+ \frac{(1 - \beta v P'')(1 - P')(1 - c)d_{\mathrm{U}}^{\bullet} + (1 - v)P'(1 - c)d_{\mathrm{I}}^{\bullet}}{(1 - \beta v P'')(1 - P')(1 - c d_{\mathrm{U}}) + (1 - v)P'(1 - c d_{\mathrm{I}})}. \tag{2-5}$$

Note that the discounting factor $1 - v$ is multiplied with P', accounting fot the reduced competitive ability of infected juveniles. Also, P_0'' (defined as the probability that a randomly sampled juvenile is infected right after dispersal in the focal group) is a function of $(d_{\mathrm{U}}^{0}, d_{\mathrm{I}}^{0})$, and the factor $1 - v\beta P_0''$ represents the competitive ability of juveniles (in the focal group) that were uninfected right before dispersal; for derivation, see Appendix A, wherein the reproductive values are also taken into account.

In order to investigate the emergence of disease state-dependence in dispersal, I first present the case in which dispersal rates are the same between disease states; i.e., the dispersal rate exhibits state-independence: $d_{\mathrm{U}} = d_{\mathrm{I}} =: d$ (Model 1). Secondly, I assume the disease state-dependent case in which the phenotype is represented by a pair of dispersal rates $(d_{\mathrm{U}}, d_{\mathrm{I}})$ that evolve jointly (Model 2).

2.3 Analyses

Because the main focus of this study is to capture the effect of horizontal transmission, I employ the neighbour-modulated approach (Taylor & Frank 1996 [56], Frank 1998 [19], and Taylor et al. 2007 [54]). To analyse the evolutionary endpoints for the phenotype of dispersal rates $(d_{\mathrm{U}}, d_{\mathrm{I}})$, I

assume a vanishingly rare mutant that has a slightly different phenotype, $d_{\mathrm{U}}^{\bullet} = d_{\mathrm{U}} + \delta_{\mathrm{U}}$ and $d_{\mathrm{I}}^{\bullet} = d_{\mathrm{I}} + \delta_{\mathrm{I}}$, where the δ-terms are small deviations of mutant dispersal rates (denoted by $(d_{\mathrm{U}}^{\bullet}, d_{\mathrm{I}}^{\bullet})$) from wild-type dispersal rates (denoted by $(d_{\mathrm{U}}, d_{\mathrm{I}})$). Because of the population viscosity, phenotypic correlations can develop between phenotypes of the mutant juveniles and the mean phenotype of juveniles in the focal group, where the latter is written as $(d_{\mathrm{U}}^{0}, d_{\mathrm{I}}^{0})$. Then, we can analyse the direction of selection by the partial derivative of W with respect to the deviation δ:

$$\begin{aligned} D(d) &:= \left.\frac{\mathrm{d}W}{\mathrm{d}\delta}\right|_{\delta=0} \\ &= \left.\frac{\partial W}{\partial d^{\bullet}}\right|_{d^{\bullet}=d^{0}=d} + R\left.\frac{\partial W}{\partial d^{0}}\right|_{d^{\bullet}=d^{0}=d} \end{aligned} \tag{2-6}$$

in the disease state-independent case. If $D > 0$ is satisfied, then higher dispersal rate is favoured.

Similarly, in the disease state-dependent case, the direction of selection on $(d_{\mathrm{U}}, d_{\mathrm{I}})$ can be analysed by:

$$\begin{aligned} D_{\mathrm{U}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &:= \left.\frac{\partial W}{\partial \delta_{\mathrm{U}}}\right|_{\delta_{\mathrm{U}}=\delta_{\mathrm{I}}=0} = \frac{\partial W}{\partial d_{\mathrm{U}}^{\bullet}} + R\frac{\partial W}{\partial d_{\mathrm{U}}^{0}}, \\ D_{\mathrm{I}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &:= \left.\frac{\partial W}{\partial \delta_{\mathrm{I}}}\right|_{\delta_{\mathrm{U}}=\delta_{\mathrm{I}}=0} = \frac{\partial W}{\partial d_{\mathrm{I}}^{\bullet}} + R\frac{\partial W}{\partial d_{\mathrm{I}}^{0}}, \end{aligned} \tag{2-7}$$

where $D_{\mathrm{U}}(d_{\mathrm{U}}, d_{\mathrm{I}}) > 0$ (or $D_{\mathrm{I}}(d_{\mathrm{U}}, d_{\mathrm{I}}) > 0$) indicates that a higher value of dispersal for uninfected juveniles (or that for infected juveniles) is likely to be favoured by natural selection. In the above expressions, each derivative is evaluated at $\delta_{\mathrm{U}} = \delta_{\mathrm{I}} = 0$.

Here, R is the relatedness coefficient; i.e., the probability that a pair of randomly sampled juveniles immediately after reproduction are identical-by-descent (namely, "whole-member relatedness", Pepper, 2000 [47]). R is given by:

$$R = \left.\frac{\mathrm{d}d^{0}}{\mathrm{d}\delta}\right|_{\delta=0}, \tag{2-8}$$

in the disease state-independent case, and

$$R = \left.\frac{\mathrm{d}d_{\mathrm{U}}^{0}}{\mathrm{d}\delta_{\mathrm{U}}}\right|_{\delta_{\mathrm{U}}=\delta_{\mathrm{I}}=0} = \left.\frac{\mathrm{d}d_{\mathrm{I}}^{0}}{\mathrm{d}\delta_{\mathrm{I}}}\right|_{\delta_{\mathrm{U}}=\delta_{\mathrm{I}}=0}, \tag{2-9}$$

in the disease state-dependent case. The coefficient of relatedness, R, is dependent on the population-average dispersal rates and thus needs the evaluation of stationary values. Under the Moran pro-

cess, a stationary value for R is such that solves:

$$R = \frac{1}{N} + \frac{N-1}{N} hR, \tag{2-10}$$

where h is the probability that a randomly sampled adult is philopatric ('backward philopatric rate'), and is a function of dispersal rate(s). For the deviation of Equation (5-33), see Mullon & Lehmann (2014) [46] and Appendix B.

The results are categorized according to the following steady-state relations: U-biased dispersal given $d_U > d_I$, I-biased dispersal given $d_U > d_I$, or no disease state-dependence given $d_U = d_I$.

3 Results

3.1 Model 1: Horizontal transmission before dispersal

In this section, I incorporate horizontal transmission occurring before dispersal only; then, it holds that $P'' = P'''$ (the ratio of infected juveniles right after dispersal is equal to that right before competition), while P is updated to be P' following Equation (2-2).

3.1.1 Disease state-independent dispersal

When the dispersal rate is disease state-independent, fitness W is much simplified:

$$W = \frac{1 - d^\bullet}{1 - d^0 + (1-c)d} + \frac{(1-c)d^\bullet}{1 - cd}, \tag{3-1}$$

which is exactly the same as Taylor's (1988) formulation [55]. By the neighbor-motulated expansion, the condition for the evolution of increased dispersal rate $D(d) > 0$ reads:

$$D(d) = -C_\emptyset + B_\emptyset R > 0. \tag{3-2}$$

The first term, which is defined by $-C_\emptyset := -c/(1-cd)$, is the fitness cost to dispersal; i.e., the cost of dispersal ($-c$) divided by one minus the total loss from the gene pool due to the costly dispersal ($1 - cd$). The second term, with $B_\emptyset := (1-d)/(1-cd)^2$, is the "indirect" fitness benefit from kin competition avoidance, multiplied by the relatedness coefficient (R). This is a classical result that the balance between the dispersal cost and benefit of kin competition avoidance can determine the evolutionarily stable dispersal rate (Maynard Smith & Price 1973[53], Hamilton & May 1977[26], Taylor 1988[55]). These terms necessarily appear in the present models throughoutly (see below).

A convergently stable phenotype $d = d^*$ would solve $D(d) = 0$ [13]. After some algebra with Equation (5-33) and backward philopatric rate $h = (1 - d)/(1 - cd)$, we obtain:

$$d^* = \frac{R - c}{R - c^2} = \frac{1}{1 + Nc}. \tag{3-3}$$

Thus, the convergently stable dispersal rate is equivalent to the well-known results demonstrated by Hamilton & May (1977) [26], Frank (1986) [17], and Taylor (1988) [55], and any parameters involving with parasite traits (β, v, and P-values) have no effect. This d^* is evolutionarily stable (proof follows from Ajar 2003, [1] and Massol et al. 2013 [42]; available upon request for the author). Hereafter, I write $d_\emptyset := 1/(1 + Nc)$, which represents a benchmark result ('Taylor's (1988) result'; Taylor 1988, [55]).

3.1.2 Disease state-dependent dispersal

Here, I investigate the case in which dispersal is conditional on the disease state, such that a 'phenotype' is a pair of dispersal rates, $(d_\mathrm{U}, d_\mathrm{I})$. If it holds generically that $d_\mathrm{U} \neq d_\mathrm{I}$, then the conclustion is that disease state-dependent dispersal can evolve. The direction of selection can be captured by $D_\mathrm{U}(d_\mathrm{U}, d_\mathrm{I})$ and $D_\mathrm{I}(d_\mathrm{U}, d_\mathrm{I})$; however, as a result, the convergently stable dispersal rates read:

$$d^*_\mathrm{U} = d^*_\mathrm{I} = \frac{1}{1 + Nc} = d_\emptyset. \tag{3-4}$$

Thus, disease state-dependent dispersal is not evoked (but see Appendix C for a more precise interpretation of this result in terms of structural stability), and Taylor's (1988) result holds [55].

3.2 Model 2: Horizontal transmission after dispersal

In this section, I investigate the case in which horizontal transmission occurs after dispersal.

3.2.1 Disease state-independent model

The condition for increased dispersal rate in the form of Hamilton's rule is equivalent to the previous model of disease state-independent dispersal (Equation (3-1)) and thus the condition for the evolution of increased dispersal rate reads (Equation (3-2)):

$$-C_\emptyset + B_\emptyset R > 0. \tag{3-5}$$

Consequently, evolutionarily stable dispersal rate reads $d^* = 1/(1+Nc) = d_\emptyset$. Thus, horizontal transmission does not generate additional selective pressure for dispersal rate, and Taylor's (1988) result holds [55].

3.2.2 Disease state-dependent model

In this case, we are to analyse the joint evolution of a pair of dispersal rates, $d_\mathrm{U}, d_\mathrm{I}$. The condition for the evolution of increased dispersal rate for uninfected or infected juveniles is, respectively, give by:

$$\begin{aligned} D_\mathrm{U} &\propto -C + RB + (d_\mathrm{U} - d_\mathrm{I})RK_\mathrm{U} > 0, \\ D_\mathrm{I} &\propto -C + RB - (d_\mathrm{U} - d_\mathrm{I})RK_\mathrm{I} > 0 \end{aligned} \tag{3-6}$$

where C is the fitness cost to dispersal defined by:

$$-C := \frac{-c}{(1-v\beta P'')(1-P)(1-cd_\mathrm{U}) + (1-v)P'(1-cd_\mathrm{I})}, \tag{3-7}$$

while B is the fitness benefit for reduced kin competition:

$$B := \frac{(1-v\beta P'')(1-P)(1-d_\mathrm{U}) + (1-v)P'(1-d_\mathrm{I})}{\left((1-v\beta P'')(1-P)(1-cd_\mathrm{U}) + (1-v)P'(1-cd_\mathrm{I})\right)^2}. \tag{3-8}$$

Additional terms involving K_U and K_I have appeared, that are positive-valued functions of $(d_\mathrm{U}, d_\mathrm{I})$:

$$\begin{aligned} K_\mathrm{U} &:= \frac{v\beta(1-v)P'(1-P')(1-c)}{(1-v\beta P'')\left((1-v\beta P'')(1-P')(1-cd_\mathrm{U}) + (1-v)P'(1-cd_\mathrm{I})\right)^2} \cdot \frac{P'(1-cd_\mathrm{I})}{\left((1-P')(1-cd_\mathrm{U}) + P'(1-cd_\mathrm{I})\right)^2} \\ K_\mathrm{I} &:= \frac{v\beta P'(1-P')(1-c)}{\left((1-\beta vP'')(1-P')(1-cd_\mathrm{U}) + (1-v)P'(1-cd_\mathrm{I})\right)^2} \cdot \frac{(1-P')(1-cd_\mathrm{U})}{\left((1-P')(1-cd_\mathrm{U}) + P'(1-cd_\mathrm{I})\right)^2} \end{aligned} \tag{3-9}$$

The K-terms clearly isolate the effects of horizontal transmission on the evolutionary dynamics of disease state-dependent dispersal. It can be shown that $d_\mathrm{U} = d_\mathrm{I} = d_\emptyset$ (which solves $D_\mathrm{U} = D_\mathrm{I} = 0$) is convergently unstable; more precisely, $d_\mathrm{U} = d_\mathrm{I} = d_\emptyset$ is an interior unstable equilibrium (saddle) in the phenotypic space for $0 \le d_\mathrm{U} \le 1$ and $0 \le d_\mathrm{I} \le 1$. As a result, the evolutionary dynamics exhibit bistability, where the convergently stable phenotype is located on the boundary of the phenotypic space (Figure 2).

This trend is generic, so that any change in the given parameters does not erode the (structural) stability of these evolutionary dynamics. Therefore, disease state-dependent dispersal (either $d_\mathrm{U}^* >$

d_{I}^{*} or $d_{\mathrm{U}}^{*} < d_{\mathrm{I}}^{*}$) is convergently stable. In Figure 3, the convergently stable dispersal rate for uninfected individuals is plotted for $d_{\mathrm{I}} = 1$ (left) or $d_{\mathrm{I}} = 0$ (right) as a function of transmission rate (β, top panels: A and B) and as a function of virulence (v, bottom panels: C and D).

To summarize, the present models show that disease state-dependent dispersal can evolve by avoiding transmission among kin, but its evolutionary endpoint depends on the timing of horizontal transmission: if horizontal transmission occurs only before dispersal, disease state-dependent dispersal is not evoked. However, if horizontal transmission occurs after dispersal, then disease state-dependent dispersal is favoured. One of two opposite patterns for disease state-biased dispersal can emerge according to the selective forces that reinforce the trend of dispersal bias, and the evolutionary forces that produce such two patterns are balanced.

4 Discussion

4.1 Emergence of disease state-dependent dispersal

Some well-established explanations for dispersal evolution have been put forward, including that dispersal aids kin competition avoidance (Hamilton & May 1977 [26]) and inbreeding avoidance (Perrin & Mazalov 2000 [48]), but is counterselected by the multiple costs of dispersal (Bonte et al. 2012 [5]). The present results highlight the pivotal importance of transmission avoidance among kin, which would drive the evolution of disease state-dependent dispersal. Also, it is suggested that evolutionary forces are balanced so that the two extremes of dispersal bias are, to some extent, fairly likely to evolve when horizontal transmission occurs after dispersal. This indicates that the selective pressures of transmission avoidance among kin by U-biased and I-biased dispersal are generically of the same magnitude. This has not been intuitively obvious, and my theoretical challenge has answered the question addressed above: the intensity of selection for U-biased or I-biased dispersal are balanced, and we can expect both patterns depending on the evolutionary history of dispersal.

In the case of disease state-independent dispersal ($d_{\mathrm{U}} = d_{\mathrm{I}}$) irrespectively of when horizontal transmission occur (before and/or after dispersal), we recover the classical result obtained by Taylor (1988) [55] such that benefit of reduced kin competition and cost of dispersal are balanced. This can be understood from the expression of P'', where the dependence of P'' on dispersal rate vanishes. In words, disease state-independent dispersal does not modify the local density of infected juveniles, and thus disease state-independent does not confer any additional cost or benefit

due to the avoidance of horizontal transmission among kin.

The most striking result is that, on the other hand, when horizontal transmission occurs after dispersal, there are two convergently stable states, where either I-biased or U-biased dispersal is expected. Why do such extreme patterns occur? This effect can also be seen from Equation (3-6), because either D_{U} or D_{I} contains a term proportional to $d_{\mathrm{U}} - d_{\mathrm{I}}$ (see the third terms in Equation (3-6)), although these terms are of opposite signs. Thus, a small difference in dispersal rates would push the state from the point $d_{\mathrm{U}} = d_{\mathrm{I}} = d_{\emptyset}$ to the boundary. For example, when the population is at $d_{\mathrm{U}} = d_{\mathrm{I}} = d_{\emptyset}$ (i.e. population is at the evolutionarily stable states without state-dependent dispersal), a small mutation that differentiates $d_{\mathrm{U}} < d_{\mathrm{I}}$ would have, on average, a higher fixation probability than expected for a selectively neutral mutation, and would eventually leads to $d_{\mathrm{I}} = 1$ by recurrent allelic substitutions under weak selection. The other extreme can be reasoned using the same logic. Biologically, this evolutionary process and its endpoint strongly depend on the genetic architecture as well as on the history of the emergence of phenotypic plasticity. Once phenotypic plasticity is genetically established by e.g. genetic accommodation, this trend of biased dispersal is self-reinforced. The aim of this study was not, however, to describe the whole process of adaptation, but rather my present results clarify that disease state-dependent dispersal aids the horizontal transmission among kin, isolated by the K-terms in Equation (3-6).

The convergently stable dispersal rate for uninfected individuals is plotted for $d_{\mathrm{I}} = 1$ (left) or $d_{\mathrm{I}} = 0$ (right) as a function of transmission rate (β, top panels: A and B) and as a function of virulence (v, bottom panels: C and D). In the left panels (A, C), a convergently stable dispersal rate for infected juveniles is complete, $d_{\mathrm{I}} = 1$, and thus horizontal transmission does not occur among kin. In this scenario, when the transmission rate (β) or virulence (v) is small, parasites have low impacts, and the dispersal rate for uninfected juveniles is small so that the average group dispersal rate is balanced around $d_{\emptyset}$, as neutral stability predicts. However, as β and v are increased, the inclusive fitness effect via the change in competitive ability of philopatric uninfected juveniles is negative; thus, a higher dispersal rate is likely to be favoured, leading to a higher dispersal rate for uninfected juveniles. In contrast, when $d_{\mathrm{I}}^{*} = 0$ (Figure 3B, D), the transmission risk among kin is severest. Then, increasing β would escalate the risk of transmission among kin, thereby favouring higher dispersal for uninfected juveniles to escape from it. In contrast, as v is increased, the negative impact of horizontal transmission becomes severer, and the relative number of infected juveniles is decreased.

Our results show that virulence has dramatic impacts on the evolutionary endpoints for disease

state-dependent dispersal, which can be deduced from the expression of K_{U} and K_{I}. One extreme represents $v = 0$ (i.e., parasites have no impact on host fitness), evolutionary dynamics is reduced so that $d_{\emptyset}$ is the unique, stable equilbrium. This is because $v = 0$ indicates that "there is no parasite" and thus juveniles show no individual variation.

In other extreme, the impact of parasite is the severest when $v = 1$, i.e., when infected juveniles are bound to be mortal at the stage of competition; in this case, there is no selection upon dispersal rate for infected juveniles because they are incapable of reproducing offspring irrespectively of the dispersal rate. Thus, dispersal of uninfected juveniles confers no additional benefit involving transmission avoidance among kin, thereby leading to $d_{\mathrm{U}} \to d_{\emptyset}$. That is, kin competition avoidance vs. cost of dispersal are the sole driving forces, which recovers Taylor's (1988) result [55] (namely $K_{\mathrm{U}} = 0$ when $v = 1$; see Figure 3D). This trend is generally sound: as higher virulence reduces the strength of kin competition by killing infected juveniles, lower dispersal rate for uninfected juveniles is favoured (Figure 3). Hence, while infected juveniles have the incentive to reducing the possibility of disease transmission to their siblings, selection upon dispersal of uninfected juveniles is predominated by the evolutionary dynamics of dispersal rate for infected juveniles.

In empirical studies of disease state-dependent dispersal, parasite species are identified and thus more or less information of parasite traits is available. How can one clarify the patterns of evolution of disease state-dependent/independent dispersal based on such information? I would point out two promising avenues. Firstly, the timing of transmission may matter for coevolutionary processes of host-parasite interactions (Day 2003 [11]): the present results predict that pre-dispersal transmission does not impose any additional selective pressure, while post-dispersal transmission has dramatic impacts. As far as we know, there is no study that investigates the timing of transmission in the context of metapopulations. Hence, there are more scope for assessing the timing of transmission and its effect on disease state-dependent dispersal.

A second point concerns how to classify the petterns of disease state-dependent dispersal; the classification of such patterns are not necessarily within the scope of our results (because there are two candidates of steady states). Nevertheless, further investigations are possible on the basis of virulence-transmission tradeoff theory (Ewald 1993[14]), which represents one of the best established paradigm that horizontally transmissible parasites are more virulent and exploit the hosts, while vertically transmissible parasites tend to be benign. (Ewald 1993[14], Clayton & Tompkins 1994[8]) This mechanism is explained by the "tragedy of the commons" (Alizon et al.

2009[3]), because the parasites that inflict severe damage on their hosts may have lower reproductive success through overexploitations. For example, ticks generally show strong virulence and possess high abilities of horizontal transmission (Clayton & Tompkins 1994[8], Møller et al. 2009[44]), while lice are able to vertically transmit with relatively low virulence (Clayton & Tompkins 1994[8]). According to on our models, for low-virulence parasites (say $v \to 0$ and thus $K_{\mathrm{U}}, K_{\mathrm{I}} \to 0$), dispersal bias is not pronounced (Figure 2, A, C, D). The present results demonstrate that more remarkable parasitic traits (higher virulence and higher transmission rate) would produce clearer tendency for disease state-dependence in dispersal (Figure 2, B). Therefore, if the virulence-transmission tradeoff hypothesis is taken into account, where horizontal transmissibility is positively correlated with virulence, then the substantial trend both for host and parasite can show pronounced variations of parasite traits and dispersal bias over taxa. This may explain the reason for the limited availability of empirical or eperimental evidence for disease state-dependent dispersal: for some cases, where parasite traits are outstanding (strong virulence with high horizontal transmissibility), host develops disease state-dependent dispersal, while for other cases, where parasite traits are benign (low virulence and low horizontal transmissibility), host shows relatively mild or no responses to parasites in the dispersal propensities.

4.2 Social aspects of transmission avoidance

Empirical studies have revealed that either U-biased or I-biased dispersal can emerge. Examples of some outstanding systems that are consistent with the present results can be found in Heinze & Walter (2010) [28], Kralj & Fuchs (2006) [35], and Rueppell et al. (2010) [52]. Self-removal is a well-appreciated concept, especially in bees, and is considered to be an extreme form of altruistic dispersal. The present results suggest that the timing of horizontal transmission matter in such a way that produces the bistability of evolutionary dynamics. This is simply because dispersal does not affect the magnitude of the pre-dispersal infection risk. In other words, P' (the probability that a randomly sampled juvenile is infected right before dispersal, which thus represents the risk of horizontal transmission before dispersal) is independent of dispersal rates. Disease state-independent dispersal does not, however, facilitate transmission avoidance. Thus, these results suggest that "altruistic" disease state-dependent dispersal by infected individuals can evolve by natural selection, which is consistent with self-removal in bee. From the point of view of the infected individuals, dispersal can lower the risk of transmission to their siblings, which can be interpreted as altruism towards related individuals. In contrast, from the point of view of the

uninfected dispersers, dispersal would aid in escaping from disease transmission, but is associated with the same cost that it would otherwise incur from transmission. Thus, the dispersal of infected and uninfected juveniles may represent altruism (rather than spite), and these forces are balanced at the evolutionarily stable strategy for disease state-dependent models ($d_\emptyset$). In general, "spite and altruism are two sides of the same coin" (Lehmann et al. 2006 [36]) in the following sense: spiteful behaviour oriented towards unrelated individuals can in turn increase the fitness of closely related individuals. Similarly, altruistic acts oriented preferentially towards related individuals can consequently decrease the fitness of unrelated individuals. This conceptual issue was pointed out by Lehmann et al. (2006) [36] and West & Gardner (2010) [61], who highlighted the importance of clarifying the differences. For example, I-biased populations would exhibit, on average, lowered possibilities of transmission among kin. In this case, I-biased dispersal can be interpretted as altruism. At the same time, however, I-biased dispersal potentiates the transmission of parasites to unrelated juveniles, which can be interpreted as spiteful behaviour to unrelated individuals as third party. Thus, the altrusim-spite interpretation based on our modeling approach completely matches with those formulated in Lehmann et al. (2006) [36] and West & Gardner (2010) [61].

The present models are conceptually similar with those of Perrin & Mazalov (2000) [48], who studied the effect of inbreeding risk on the evolution of sex-biased dispersal. In particular, they showed that when the modes of social competition (local resource competition or local mate competition) are fairly acting, no sex-biased dispersal is evoked. On the other hand, if sex-specific competition is substantial, then male-biased dispersal can be favoured. The distinction from the present models is that, in their models, encoutering with kin (with subsequent inbreeding) has direct deleterious effects on fitness (namely, inbreeding depression, which reduces the fecundity of a pair). In light of Perrin & Mazalov's (2000) [48] seminal study, I speculate that parasite transmission in a social group may represent one mode of social interaction (including competition), which conforms with my interpretation of disease state-dependent dispersal as a spiteful/altruistic behaviour. Indeed, the fact is well appreciated that social interaction (e.g. allo-grooming) mediates horizontal transmission among group members (Theis et al. 2015; [58]). Hence, further empirical studies are required to study the social behaviours with different consequences for disease state-dependent dispersal.

4.3 Future extensions

The present modelling approach was simplified to maintain tractability, which inevitably results in certain limitations. For example, I have not considered the vertical transmission of parasites (and subsequent dynamics of epidemiology), group size variations (e.g. Cote & Poullin 1995 [10]), the possibility of host manipulation by parasites (Lion et al. 2006 [37]), genetic dynamics ("infection genetics"; e.g. gene-for-gene model (Flor 1971 [16]), spatial variations in parasite prevalence (which is here P), or parasite evolution (virulence, transmissibility, and/or infectivity). However, pointing out these limitations highlights that further implementations on the basis of this modelling framework should be possible, regardless of the choice of whether or not to use inclusive fitness theory.

4.4 Conclusion

Our study represents the first attempt to analyse the effect of horizontal parasite transmission on the evolution of dispersal, revealing that disease state-dependent dispersal can evolve. The altruistic aspect of disease state-dependent dispersal is disclosed in social animals facing parasitism. Further studies are required to fully understand the maintenance of sociality in the face of diseases, i.e., evolution of social immunity.

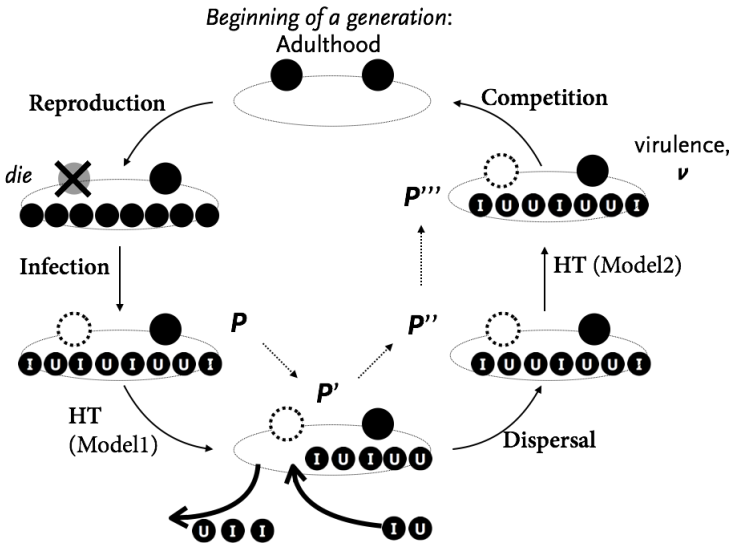

Figure 1: The assumed life cycle is depicted for group size $N = 2$, where HT represents 'horizontal transmission'. Each individual produces an infinite number of juveniles. After reproduction, one individual is chosen to die at random (the birth-death Moran demography). Juveniles are infected at random with probability P. In Model 1, HT occurs before dispersal, resulting in the ratio of infected juveniles to become P'. Juveniles disperse with success probability $(1 - c)$. The ratio of infected juveniles after dispersal is denoted by P''. In Model 2, HT follows dispersal, which updates the ratio of infected juveniles from P'' to P'''. Finally, juveniles compete for the single vacant spot to form the next generaion, and infected juveniles have a lower competitive ability $(1-\nu)$. Note that 'parasite-induced mortality (ν) right before competition' is equivalent to 'reduced competitive ability'.

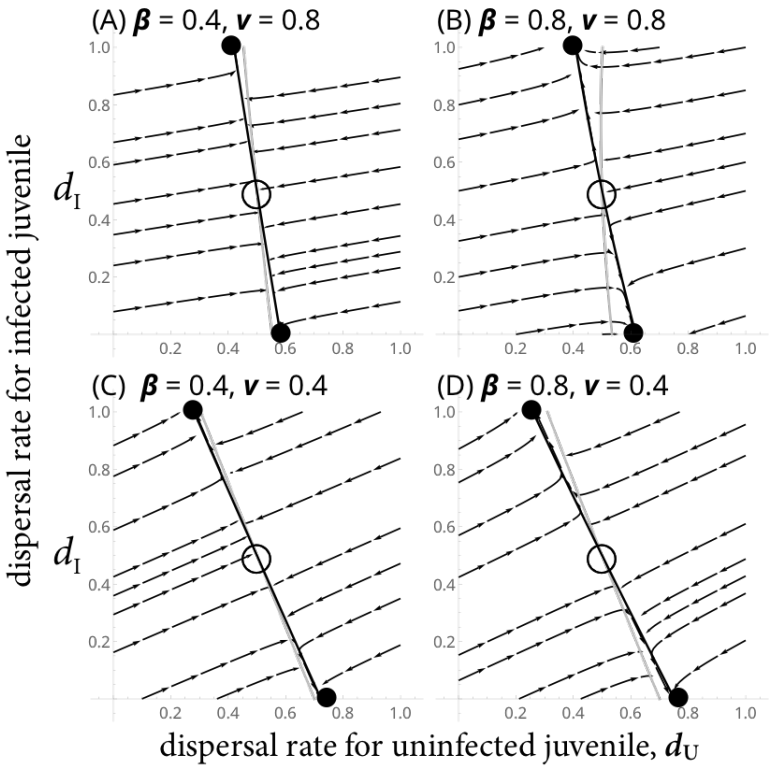

Figure 2: The evolutionary trajectory based on D_U (the isocline is coloured in black), D_I (the isocline is coloured in grey) is depicted for $N = 5$, $P = 0.4$, and $\alpha = 0$ (no horizontal transmission before dispersal). An interior unstable equilibrium occurs at $d_U = d_I = 1/(1 + Nc)$ (open circle). As a result, two boundary equilibria occur (closed circles).

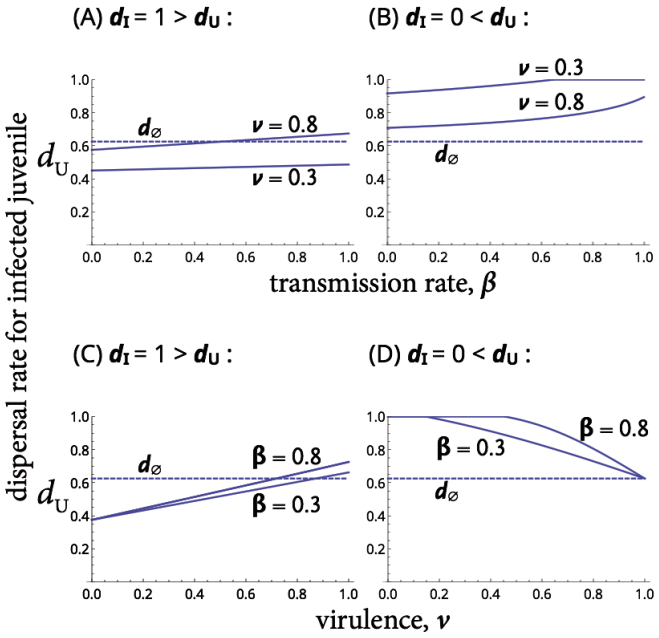

Figure 3: The convergently stable dispersal rate for uninfected individuals is plotted for $d_I = 1$ (left) or $d_I = 0$ (right) as a function of transmission rate (β, top panels: A and B) and virulence (v, bottom panels: C and D). The results for $d_\emptyset = 1/(1 + Nc)$ are also plotted for comparison (dashed line). (A, C): When $d_I^* = 1$, there is no transmission among kin. Increased β and v would reduce the relative number of uninfected juveniles (because they are infected and suffer the virulence), thereby increasing the intensity of kin competition. Thus, a higher dispersal rate for uninfected juveniles is favoured. (B, D) When $d_I^* = 0$, the transmission risk among kin is the severest. Then, increased β would escalate the risk of transmission among kin, favouring higher dispersal for uninfected juveniles to escape from it. In contrast, as v is increased, the negative impact of horizontal transmission becomes severer, and the relative number of infected juveniles is decreased.

5 Appendix

5.1 Invasion fitness and selection gradient

In the context of evolutionary dynamics, invasion fitness characterizes whether a vanishingly rare mutant showing up in the population can increase its frequency. In a class structured population, however, relative contribution of individuals in different classes affect the total gene frequency and its asymptotic growth rate. The method proposed by Rousset (2004) [50] allows one to compute them automatically. Let $\mathcal{W}$ be a reproductive success matrix , whose elements read:

$$\mathcal{W} := \begin{pmatrix} W_{\mathrm{U|U}} & W_{\mathrm{U|I}} \\ W_{\mathrm{I|U}} & W_{\mathrm{I|I}} \end{pmatrix}. \tag{5-1}$$

Here, the element $W_{\mathrm{Y|X}}$ denotes the expected number of successful juveniles in disease-state Y (right before competition) born to the parent in disease-state X (right before competition). juvenile succesfulness is characterized by winning a breeding spot.

Suppose that the population is monomoprhic; then, the elements of $\mathcal{W}$ (evaluated at $\delta = 0$) can be computed as:

$$W_{\mathrm{U|U}} = \frac{1}{1 - vP'''}, \tag{5-2}$$

$$W_{\mathrm{U|I}} = \frac{1}{1 - vP'''}, \tag{5-3}$$

$$W_{\mathrm{I|U}} = \frac{1 - v}{1 - vP'''}, \tag{5-4}$$

$$W_{\mathrm{I|I}} = \frac{1 - v}{1 - vP'''}. \tag{5-5}$$

It is easy to show that the dominant eigenvalue is 1, associated left eigenvector $\mathbf{q}$ and right eigenvector $\mathbf{p}$ reading:

$$\mathbf{q}^{\mathrm{T}} = (1, 1), \tag{5-6}$$

$$\mathbf{p} = \begin{pmatrix} 1 - P''' \\ P''' \end{pmatrix}. \tag{5-7}$$

Thus, we can simply sum up the fitness gains via infected and uninfected juveniles right before competition. Hereafter, we call P-values simply as 'prevalence', which defines the probabilities that a randomly sampled juveniles at specific stages is infected.

When we compute the reproductive values, we have used P-values; of our interest is, however,

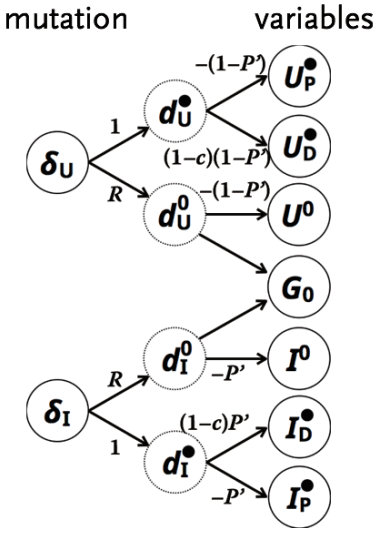

Figure 4: A path diagram is shown to compute the variable dependence of invasion fitness W. Any dispersal rates and neighbor-dispersal rates (shown in the middle row: $d_{\rm U}^\bullet, d_{\rm U}^0, d_{\rm I}^0, d_{\rm I}^0$) are dependent on the mutation effects, $\delta_{\rm U}, \delta_{\rm I}$. These dispersal rates mediate the change in the variables of fitness in the right row.

to compute the selective pressures, and thus below we use the following notations:

$$
\begin{aligned}
U_{\rm P}^\bullet &:= (1-P')(1-d_{\rm U}^\bullet), \\
I_{\rm P}^\bullet &:= P'(1-d_{\rm I}^\bullet), \\
U_{\rm D}^\bullet &:= (1-P')(1-c)d_{\rm U}^\bullet, \\
I_{\rm D}^\bullet &:= P'(1-c)d_{\rm I}^\bullet, \\
U_{\rm P}^0 &:= (1-P')(1-d_{\rm U}^0), \\
I_{\rm P}^0 &:= P'(1-d_{\rm I}^0), \\
U_{\rm P} &:= (1-P')(1-d_{\rm U}), \\
I_{\rm P} &:= P'(1-d_{\rm I}), \\
U_{\rm D} &:= (1-P')(1-c)d_{\rm U}, \\
I_{\rm D} &:= P'(1-c)d_{\rm I}, \\
U^0 &:= U_{\rm P}^0 + U_{\rm D}, \\
I^0 &:= I_{\rm P}^0 + I_{\rm D}, \\
U &:= U_{\rm P} + U_{\rm D}, \\
I &:= I_{\rm P} + I_{\rm D}, \qquad (5\text{-}8)
\end{aligned}
$$

These quantities define the relative number of juveniles; for example, $U^{\bullet}_{\mathrm{D}}$ denotes the relative number of dispersed uninfected juveniles of the focal adult, I^{0}_{P} denotes the relative number of philopatric infected juveniles of the focal adult, and U^{0} reads the relative number of uninfected individuals in the focal group immediately after dispersal (before horizontal transmission). The other symbols can be read in a similar manner. Also note that the backward philopatric rate, namely a probability that a randomly sampled adult individual was philopatric, reads:

$$h = \frac{(1-\beta v P'')U_{\mathrm{P}} + (1-v)I_{\mathrm{P}}}{(1-\beta v P'')U + (1-v)I}. \tag{5-9}$$

From Equation (5-8) we can translate:

$$\begin{aligned}
P'''_0 &= \frac{I^0 + \beta U^0 \frac{I^0}{U^0+I^0}}{U^0+I^0} && (5\text{-}10)\\
P''' &= \frac{I + \beta U \frac{I}{U+I}}{U+I}, && (5\text{-}11)\\
P''_0 &= \frac{I^0}{U^0+I^0}, && (5\text{-}12)\\
P'' &= \frac{I}{U+I}, && (5\text{-}13)\\
P' &= P + \alpha P(1-P). && (5\text{-}14)
\end{aligned}$$

Invasion fitness of a mutant adult reads:

$$\begin{aligned}
W &= \frac{(1-\frac{\beta I^0}{U^0+I^0})U^{\bullet}_{\mathrm{P}} + \frac{\beta I^0}{U^0+I^0}(1-v)U^{\bullet}_{\mathrm{P}} + (1-v)I^{\bullet}_{\mathrm{P}}}{(1-\frac{\beta I^0}{U^0+I^0})U^0 + \frac{\beta I^0}{U^0+I^0}(1-v)U^0 + (1-v)I^0}\\
&+ \frac{(1-\frac{\beta I}{U+I})U^{\bullet}_{\mathrm{D}} + \frac{\beta I}{U+I}(1-v)U^{\bullet}_{\mathrm{D}} + (1-v)I^{\bullet}_{\mathrm{D}}}{(1-\frac{\beta I}{U+I})U + \frac{\beta I}{U+I}(1-v)U + (1-v)I}
\end{aligned} \tag{5-15}$$

where the first term (or second term) accounts for the sum of fitness gains via philopatric juveniles, (or that via dispersed juveniles, respectively). Partially using P''_0 and P'' (prevalence immediately after dispersal), W can be writte as:

$$\begin{aligned}
W &= \frac{(1-\beta v P''_0)U^{\bullet}_{\mathrm{P}} + (1-v)I^{\bullet}_{\mathrm{P}}}{(1-\beta v P''_0)U^0 + (1-v)I^0} + \frac{(1-\beta v P'')U^{\bullet}_{\mathrm{D}} + (1-v)I^{\bullet}_{\mathrm{D}}}{(1-\beta v P'')U + (1-v)I}\\
&= \frac{G^0 U^{\bullet}_{\mathrm{P}} + (1-v)I^{\bullet}_{\mathrm{P}}}{G^0 U^0 + (1-v)I^0} + \frac{GU^{\bullet}_{\mathrm{D}} + (1-v)I^{\bullet}_{\mathrm{D}}}{GU + (1-v)I}
\end{aligned} \tag{5-16}$$

The term $G^0 := 1-\beta v P''_0 = 1 - P''_0 + P''_0(1-\beta) + P''_0\beta(1-v)$ accounts for the reduced competitive

ability due to horizontal transmission; with a probability P_0'', an uninfected juvenile encounter with an infected juveniles (after dispersal), in which case, with a probability β he/she gets infected and thus suffers the virulence. G^0 represents the competitive ability of uninfected individuals in the focal group ($G = 1 - \beta v P''$), while G represents that in other groups. G takes into account the effect of horizontal transmission.

Hereafter, we prefer to use Equation (5-16) over Equation (5-15) simply for convenience. Hence, we regard W as a function of G^0, $U_{\mathrm{P}}^{\bullet}$, $U_{\mathrm{D}}^{\bullet}$, $I_{\mathrm{P}}^{\bullet}$, $I_{\mathrm{D}}^{\bullet}$, U^0, and I^0, thereby expanding the selective pressures in the neighbor-modulated way. This computation is successful in the point that we can completely purify the effect of horizontal transmission, as we see below.

Selection gradient are computed from chain-rules. Under weak selection, selection gradient reads:

$$\begin{aligned} D_{\mathrm{U}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &= \frac{\partial W}{\partial \delta_{\mathrm{U}}}, \\ D_{\mathrm{I}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &= \frac{\partial W}{\partial \delta_{\mathrm{I}}} \end{aligned} \tag{5-17}$$

where all the partial derivatives are evaluated at $\delta_{\mathrm{U}} = \delta_{\mathrm{I}} = 0$. Neighbor-modulated expansion yields

$$\begin{aligned} D_{\mathrm{U}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &= \frac{\partial W}{\partial d_{\mathrm{U}}^{\bullet}} + R\frac{\partial W}{\partial d_{\mathrm{U}}^{0}}, \\ D_{\mathrm{I}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &= \frac{\partial W}{\partial d_{\mathrm{I}}^{\bullet}} + R\frac{\partial W}{\partial d_{\mathrm{I}}^{0}}. \end{aligned} \tag{5-18}$$

Here, R is the coefficient of relatedness, defined as the probability that a pair of randomly sampled juveniles immediately after reproduction are identical by descent. R is independent of disease-state (U vs. I), because infection occurs at random after reproduction irrespectively of whether a juvenile is mutant or wild. Moreover, we can expand Equation (5-18) by recalling the variable dependences (see the diagram in Figure 5.1):

$$\begin{aligned} D_{\mathrm{U}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &= \frac{\partial W}{\partial U_{\mathrm{P}}^{\bullet}}\frac{\partial U_{\mathrm{P}}^{\bullet}}{\partial d_{\mathrm{U}}^{\bullet}} + \frac{\partial W}{\partial U_{\mathrm{D}}^{\bullet}}\frac{\partial U_{\mathrm{D}}^{\bullet}}{\partial d_{\mathrm{U}}^{\bullet}} + R\frac{\partial W}{\partial U^{0}}\frac{\partial U^{0}}{\partial d_{\mathrm{U}}^{0}} + R\frac{\partial W}{\partial G^{0}}\frac{\partial G^{0}}{\partial d_{\mathrm{U}}^{0}}, \\ D_{\mathrm{I}}(d_{\mathrm{U}}, d_{\mathrm{I}}) &= \frac{\partial W}{\partial I_{\mathrm{P}}^{\bullet}}\frac{\partial I_{\mathrm{P}}^{\bullet}}{\partial d_{\mathrm{I}}^{\bullet}} + \frac{\partial W}{\partial I_{\mathrm{D}}^{\bullet}}\frac{\partial I_{\mathrm{D}}^{\bullet}}{\partial d_{\mathrm{I}}^{\bullet}} + R\frac{\partial W}{\partial I^{0}}\frac{\partial I^{0}}{\partial d_{\mathrm{I}}^{0}} + R\frac{\partial W}{\partial G^{0}}\frac{\partial G^{0}}{\partial d_{\mathrm{I}}^{0}}. \end{aligned} \tag{5-19}$$

In each equation of Equation (5-19), the sum of first and second term corresponds to the fitness

cost of dispersal, while the third term accounts for the avoidance of kin competition; the final term accounts for the inclusive fitness effect of horizontal transmission (after dispersal), because β (horizontal transmission rate after dispersal) is isolated in the expression. Coefficient of relatedness is multiplied because transmission occurs locally.

Below, we evaluate each partial derivative (evaluated at $\delta_{\mathrm{U}} = \delta_{\mathrm{I}} = 0$):

$$\begin{aligned}
\frac{\partial W}{\partial U_{\mathrm{P}}^{\bullet}}\frac{\partial U_{\mathrm{P}}^{\bullet}}{\partial d_{\mathrm{U}}^{\bullet}} &= G(1-P')\frac{-1}{GU+(1-v)I} \\
\frac{\partial W}{\partial U_{\mathrm{D}}^{\bullet}}\frac{\partial U_{\mathrm{D}}^{\bullet}}{\partial d_{\mathrm{U}}^{\bullet}} &= G(1-P')\frac{1-c}{GU+(1-v)I} \\
\frac{\partial W}{\partial U^{0}}\frac{\partial U^{0}}{\partial d_{\mathrm{U}}^{0}} &= G(1-P')\frac{GU_{\mathrm{P}}+(1-v)I_{\mathrm{P}}}{\{GU+(1-v)I\}^{2}} \\
\frac{\partial W}{\partial G^{0}}\frac{\partial G^{0}}{\partial d_{\mathrm{U}}^{0}} &= (1-P')\frac{\beta v(1-v)(UI_{\mathrm{P}}-IU_{\mathrm{P}})}{\{GU+(1-v)I\}^{2}}\cdot\frac{I}{(U+I)^{2}} \\
\frac{\partial W}{\partial U_{\mathrm{P}}^{\bullet}}\frac{\partial U_{\mathrm{P}}^{\bullet}}{\partial d_{\mathrm{I}}^{\bullet}} &= (1-v)P'\frac{-1}{GU+(1-v)I} \\
\frac{\partial W}{\partial I_{\mathrm{D}}^{\bullet}}\frac{\partial I_{\mathrm{D}}^{\bullet}}{\partial d_{\mathrm{I}}^{\bullet}} &= (1-v)P'\frac{1-c}{GU+(1-v)I} \\
\frac{\partial W}{\partial I^{0}}\frac{\partial I^{0}}{\partial d_{\mathrm{I}}^{0}} &= (1-v)P'\frac{GU_{\mathrm{P}}+(1-v)I_{\mathrm{P}}}{\{GU+(1-v)I\}^{2}} \\
\frac{\partial W}{\partial G^{0}}\frac{\partial G^{0}}{\partial d_{\mathrm{I}}^{0}} &= -P'\frac{\beta v(1-v)(UI_{\mathrm{P}}-IU_{\mathrm{P}})}{\{GU+(1-v)I\}^{2}}\cdot\frac{U}{(U+I)^{2}}
\end{aligned} \tag{5-20}$$

Some algebra yields:

$$UI_{\mathrm{P}} - IU_{\mathrm{P}} = P'(1-P')(1-c)(d_{\mathrm{U}} - d_{\mathrm{I}}). \tag{5-21}$$

It is worthy noting that

$$\frac{\partial h}{\partial G} = \frac{\partial W}{\partial G^{0}} \tag{5-22}$$

when the latter is evaluated $\delta_{\mathrm{U}} = \delta_{\mathrm{I}} = 0$; this partial derivative measures the effect of transmission on the fitness via philopatric juveniles. Finally, we obtain the selective pressures by substituting

Equation (5-20) into Equation (5-19) to obtain:

$$\begin{aligned}\frac{D_{\mathrm{U}}}{G(1-P')} &= -C+RB+R\frac{\beta v(1-v)P'(1-P')(1-c)(d_{\mathrm{U}}-d_{\mathrm{I}})}{G\{GU+(1-v)I\}^2}\cdot\frac{I}{(U+I)^2}\\ \frac{D_{\mathrm{I}}}{(1-v)P'} &= -C+RB-R\frac{\beta vP'(1-P')(1-c)(d_{\mathrm{U}}-d_{\mathrm{I}})}{\{GU+(1-v)I\}^2}\cdot\frac{U}{(U+I)^2},\end{aligned} \tag{5-23}$$

where we use the notation for

$$-C := \frac{-c}{GU+(1-v)I} \tag{5-24}$$

$$B := \frac{GU_{\mathrm{P}}+(1-v)I_{\mathrm{P}}}{\{GU+(1-v)I\}^2}. \tag{5-25}$$

The third term in Equation (5-23) tells us the conditions for neutrality: evolutionary dynamics exhibits neutrality $D_{\mathrm{U}}/\{G(1-P')\} = D_{\mathrm{I}}/\{(1-v)P'\} = RB-C$, whenver one of the equalities meets: $\beta = 0$ (transmission never occurs), $v = 0$ (no impact of parasites), $P' = 0$ (there is no infected juvenile at the stage of dispersal), $P' = 1$ (there is no uninfected juvenile at the stage of dispersal), $c = 1$ (disperser can never win a spot), or $d_{\mathrm{U}} = d_{\mathrm{I}}$ (there is no dispersal bias); otherwise, evolutionary dynamics exhibits an interior saddle. Because α does not appear in the expression (but implicitly affects $P' = P+\alpha P(1-P)$, which is always larger than 0 and smaller than 1 as long as $P \neq 0, 1$), α does not contribute to disease state-dependent dispersal. We would write

$$\frac{\beta v(1-v)P'(1-P')(1-c)}{G\{GU+(1-v)I\}^2}\cdot\frac{I}{(U+I)^2} =: K_{\mathrm{U}}, \tag{5-26}$$

$$\frac{\beta vP'(1-P')(1-c)}{\{GU+(1-v)I\}^2}\cdot\frac{U}{(U+I)^2} =: K_{\mathrm{I}} \tag{5-27}$$

5.2 Fitness in disease-independent dispersal

When dispersal rate exhibits unconditionality ($d_{\mathrm{U}} = d_{\mathrm{I}}$), fitness is rather simplified. Invasion fitness reads:

$$W = \frac{G^0U_{\mathrm{P}}^{\bullet}+(1-v)I_{\mathrm{P}}^{\bullet}}{G^0U^0+(1-v)I^0}+\frac{GU_{\mathrm{D}}^{\bullet}+(1-v)I_{\mathrm{D}}^{\bullet}}{GU+(1-v)I} \tag{5-28}$$

$$= \frac{1-d^{\bullet}}{1-d^0+(1-c)d}+\frac{(1-c)d^{\bullet}}{1-cd}, \tag{5-29}$$

which is the same as the fitness function when there is no horizontal transmission. Backward philopatric rate h is simply $h = (1 - d)/(1 - cd)$. Convergently stable dispersal rate d^* is such that solves $\mathrm{d}W/\mathrm{d}\delta = 0$ evaluated at $d = d^*$. This condition yields

$$d^* = \frac{R - c}{R - c^2}. \tag{5-30}$$

Since we are interested in studying steady states of the population, we ought to evaluate the stationary value for R. Let F be the probability that a pair of disinct adults are identical by descent, namely the "others-only" coefficient of relatedness (Pepper 2000 [47]); In any generation, R and F satisfy:

$$R = \frac{1}{N} + \frac{N - 1}{N} F, \tag{5-31}$$

which is a well known result: with a probability $1/N$, a single individual is sampled twice; otherwise, two distinct individuals in the same group are identical by descent with a probability F. Over two succesive generations, F obeys the recursion:

$$F_{\mathrm{Next}} = \frac{2}{N} hR + \frac{N - 2}{N} F. \tag{5-32}$$

With the probability $2/N$, (only) one of the pair of distinct adults is a new breeder such that has filled a single vacant breeding-spot which was available because of the death of an adult in the previous generation; in which case, they are identical by descent with the probability R given that the new breeder was philopatric (which occurs with a probability h). On the other hand, with probability $(N-2)/N$, two distinct individuals are both "survivors" from the previous generation(s), in which case they are identical by descent with a probability F. After some arrangement with Equation (5-31) and Equation (5-32), we obtain the stationary value of R that would satisfy:

$$R = \frac{1}{N} + \frac{N - 1}{N} Rh. \tag{5-33}$$

Finally, h reads simply:

$$h = \frac{1 - d}{1 - cd} \tag{5-34}$$

in a monomorpchi population. The direct substitution of Equation (5-33) into Equation (5-30) is possible, but we prefer to compute as implicitly as possible. Looking to the evolutionary stability condition $RB_\emptyset - C_\emptyset = 0$, we multiply $1 - cd$ to obtain $Rh = c$ with $F = Rh$; thus $F = c$ at equilbrium. Substituting $R = 1/N + (N-1)F/N$ (Equation (5-31)) into Equation (5-30) yields:

$$\begin{aligned} d^* &= \frac{R-c}{R-c^2} && (5\text{-}35)\\ &= \frac{\frac{1}{N}+\frac{N-1}{N}F-c}{\frac{1}{N}+\frac{N-1}{N}F-c^2} && (5\text{-}36)\\ &= \frac{\frac{1}{N}+\frac{N-1}{N}c-c}{\frac{1}{N}+\frac{N-1}{N}c-c^2} && (5\text{-}37)\\ &= \frac{1}{1+Nc}, && (5\text{-}38) \end{aligned}$$

as desired. Thus, disease state-independent dispersal model gives exactly the same result as that obtained by Taylor (1988) [55].

5.3 No horizontal transmission after dispersal

When we consider no transmission after dispersal, we can simply assume $\beta = 0$ and hence $G = 1$. Then, fitness function is simply

$$\begin{aligned} W &= \frac{(1-P')(1-d_{\mathrm{U}}^\bullet)+(1-v)P'(1-d_{\mathrm{I}}^\bullet)}{(1-P')(1-d_{\mathrm{U}}^0+(1-c)d_{\mathrm{U}})+(1-v)P'(1-d_{\mathrm{I}}^0+(1-c)d_{\mathrm{I}})} && (5\text{-}39)\\ &+ \frac{(1-P')(1-c)d_{\mathrm{U}}^\bullet+(1-v)P'(1-c)d_{\mathrm{I}}^\bullet}{(1-P')(1-cd_{\mathrm{U}})+(1-v)P'(1-cd_{\mathrm{I}})}. && (5\text{-}40) \end{aligned}$$

Recall that P' is the prevalence immediately before dispersal. Here, we define the quantities of immigration rate (given the survival during dispersal):

$$\begin{aligned} \overline{d^\bullet} &:= \frac{(1-P')d_{\mathrm{U}}^\bullet+(1-v)P'd_{\mathrm{I}}^\bullet}{1-vP'}, && (5\text{-}41)\\ \overline{d^0} &:= \frac{(1-P')d_{\mathrm{U}}^0+(1-v)P'd_{\mathrm{I}}^0}{1-vP'}, && (5\text{-}42)\\ \overline{d} &:= \frac{(1-P')d_{\mathrm{U}}+(1-v)P'd_{\mathrm{I}}}{1-vP'}. && (5\text{-}43) \end{aligned}$$

Then, invasion fitness $w(\overline{d^\bullet}, \overline{d^0}) := W(d_{\mathrm{U}}^\bullet, d_{\mathrm{I}}^\bullet, d_{\mathrm{U}}^0, d_{\mathrm{I}}^0)$ can be reduced to:

$$w = \frac{1-\overline{d^\bullet}}{1-\overline{d^0}+(1-c)\overline{d}} + \frac{(1-c)\overline{d^\bullet}}{1-c\overline{d}}. \quad (5\text{-}44)$$

This indicates that the evolutionary dynamics exhibits neutrality not only in the first order (selection gradient) but in the full order (fitness function), and thus disease state-dependent dispersal is not evoked.

Neutral stability is, in this system, characterized by two properties. Firstly, invasion fitness W is a constant ($W \equiv 1$) on some set L. Namely,

$$w(\overline{d^\bullet}, \overline{d^0})\big|_{\overline{d^\bullet}=\overline{d^0}=d_\emptyset} \equiv W\big|_{(d_\mathrm{U}^\bullet, d_\mathrm{I}^\bullet)\in L} \equiv 1, \tag{5-45}$$

where

$$L := \left\{(d_\mathrm{U}, d_\mathrm{I})\middle| \frac{(1-P')d_\mathrm{U} + (1-v)P'd_\mathrm{I}}{1-P'v} = d_\emptyset\right\} \tag{5-46}$$

Secondly, any point on L is convergently stable for each fixed values of parameters. L always passes through $\overline{d} = d_\emptyset$ as long as N and c are both fixed and $\beta = 0$; in words, α, v, P changes the "slope" of L, but never affects the value of $d_\emptyset = 1/(1+Nc)$. Thus, any points on $L\backslash\{d_\emptyset\}$ would become unstable even with only slight changes in α, v, and/or P, while $d_\emptyset$ is always stable independently of such parameters of parasitic impacts. Therefore, $L/\{d_\emptyset\}$ is structurally unstable, and we can conclude that convergently stable dispersal rate is $d_\emptyset$.

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