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

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ABSTRACT

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.

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 et al., 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 dynamics, but it is subject to 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 and May, 1977; McPeek and Holt, 1992; Gandon, 1999; Gandon and 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 and 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, 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 differential costs of dispersal 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 and Brown, 1992; 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* Knowles et al., 2013), if the infection (or indirect transmission) of parasites occurs during dispersal, then susceptible individuals experience a higher cost than do 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 and Iwakuma, 2009; Terui et al., 2014). These biased dispersal propensities are important for

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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 and Chapman, 2010; Karvonen and Seehausen, 2012). One scenario, 'selection against infected emigrants', suggests that parasite-imposed natural selection acts on the initiation of 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 and Altizer, 2006). Hence, host species and their respective societies have developed various mechanisms to resist 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.

In this study, we develop mathematical models to provide 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; Maynard Smith, 1993) 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 neighbor-modulated approach in inclusive fitness theory (Taylor and Frank, 1996; Frank, 1998; Rousset, 2004). 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. Model

Hereafter, by 'infection' or 'get infected', we mean the transition of 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 Fig. 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; $n_d \to +\infty$), each of which fosters an equal number of adults (N). Each adult asexually

Table 1
Notation summary.

n_d	Number of subpopulations in the whole population
N	Capacity of each subpopulation
$J\ (\to +\infty)$	Fecundity of each adult individual
R	Prevalence within a subpopulation
z_S	Dispersal rate of susceptible individual
z_I	Dispersal rate of infected individual
ε_{Emig}	Intensity of selection against infected emigrant
p	Basic probability of dispersal success
α^D	Probability of infection during dispersal
β^D	Probability of recovery during dispersal
$\mathbf{P}^D$	Transition matrix expressed by α^D and β^D
ε_{Immig}	Intensity of selection against infected immigrant
α^A	Probability of infection after dispersal
β^A	Probability of recovery after dispersal
$\mathbf{P}^A$	Transition matrix expressed by α^A and β^A
δ	Virulence on infected individuals at the competition stage
$\mathbf{x}^\bullet = (x_S^\bullet, x_I^\bullet)$	Dispersal strategy of the focal adult
$\mathbf{x}^{OR} = (x_S^{OR}, x_I^{OR})$	Averaged dispersal strategy within the focal subpopulation
$\mathbf{x}^1 = (x_S^1, x_I^1)$	Averaged dispersal strategy over the whole population
$\mathbf{z}^* = (z_S^*, z_I^*)$	ES-dispersal strategy
w	Fitness of the focal adult.
D_S,D_I.	Selection gradients for x_S (or x_I, respectively)
(ξ_S,ξ_I)	Deviations of the focal adult from the population mean
r or F_{ST}^R	Relatedness coefficient within the focal subpopulation
Δ	Bias predictor
$G_{pI/pS}$	Relative competitive ability of philopatric I-individuals compared to that of philopatric S-individuals
C_S(or C_I)	Costs of dispersal for S- (or I-) individuals, respectively
Auto^A	Autocorrelation coefficient of transition matrix $\mathbf{P}^A$
R_E	Effective prevalence
$[S]_{tot}^{iC}$ (or $[I]_{tot}^{iC}$)	Number of S- (or I-) individuals just before competition in subpopulation $i=0$ (home) or $i=1$ (away)
$[S]_{tot}^{iA}$ (or $[I]_{tot}^{iA}$)	Number of S- (or I-) individuals immediately after dispersal in subpopulation $i=0$ (home) or $i=1$ (away)
$[S]_m^{iA}$ (or $[I]_m^{iA}$)	Number of S- (or I-) migrant individuals immediately after dispersal, that originates from subpopulation $i=0$ (home) or $i=1$ (away)
$[S]_p^{iA}$ (or $[I]_p^{iA}$)	Number of S- (or I-) philopatric individuals immediately after dispersal in subpopulation $i=0$ (home) or $i=1$ (away)
$[S]_m^{iB}$ (or $[I]_m^{iB}$)	Number of S- (or I-) migrant individuals just before dispersal from subpopulation $i=0$ (home) or $i=1$ (away)
$[S]_p^{iB}$ (or $[I]_p^{iB}$)	Number of S- (or I-) philopatric individuals immediately after dispersal in subpopulation $i=0$ (home) or $i=1$ (away)
$\mathbf{X}_{Immig}$	Two-dimensional diagonal matrix with the entries 1 and $1-\varepsilon_{Immig}$, representing the selection against infected immigrants
$\mathbf{X}_{Emig}$	Two-dimensional diagonal matrix with the entries 1 and $1-\varepsilon_{Emig}$, representing the selection against infected emigrants
u (or v)	Mean emigration (or immigration) rate, respectively