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Habitat selection under the risk of infectious disease

Suzanne L. Robertson[1] and Ian M. Hamilton[2]

[1]*Mathematical Biosciences Institute, The Ohio State University, Columbus, Ohio, USA and*
[2]*Department of Mathematics and Department of Ecology, Evolution and Organismal Biology, The Ohio State University, Columbus, Ohio, USA*

ABSTRACT

Question: How does the risk of infectious disease transmission affect individual habitat selection decisions and the resulting spatial distributions of populations?

Mathematical method: We use a differential equation model to describe disease dynamics in two habitats coupled by natal dispersal and use an evolutionary game theoretical approach to calculate the evolutionarily stable strategy for habitat choice.

Key assumptions: Natal dispersal by offspring with ideal knowledge of habitats. Habitats differ only in resource quality. Fecundity is proportional to intake rate, which decreases with density. We assume density-dependent disease transmission, with infection reducing fecundity or lifespan. Disease may be present in both habitats or the high-quality habitat only.

Conclusions: In the absence of disease, our model predicts input matching (i.e. the distribution of individuals matches the distribution of resource inputs). The negative fitness consequences of infection can result in undermatching (underuse of the high-quality habitat compared with input matching), but stable overmatching (overuse of the high-quality habitat) is never predicted. Increasing the risk of transmission increases the degree of observed undermatching when only the high-quality habitat is infected but reduces undermatching when both habitats present a risk of disease. Increasing the cost of infection by reducing fecundity reduces use of the high-quality habitat (undermatching) in both cases. Increasing the cost of infection by increasing mortality rates also reduces the use of the high-quality habitat when both habitats are infected; if only the high-quality habitat is infected, undermatching may initially increase with mortality but eventually decreases.

Keywords: habitat selection, ideal free distribution, infectious disease transmission, undermatching.

INTRODUCTION

Animals must consider many factors when choosing where to spend their time, including the properties of potential habitats as well as the location of others in the population. Individuals may benefit from choosing habitats that offer greater availability of food or resources; however, if others in the population make the same choice, foragers in habitats of

Correspondence: S.L. Robertson, Mathematical Biosciences Institute, The Ohio State University, Columbus, OH 43210, USA. e-mail: srobertson@mbi.osu.edu

high intrinsic quality may experience greater competition for the same resource. Therefore, the payoff to each individual for using a habitat depends both on the habitat itself as well as the strategy of others in the population. If individuals act to maximize their own fitness, the resulting equilibrium spatial distribution is one where fitness is equal in all habitats – an ideal free distribution (Fretwell and Lucas, 1969). The equilibrium strategy is a Nash equilibrium (Nash, 1951) of a habitat selection game against the field and is stable if no individual can increase its fitness by changing strategy and modifying its habitat usage. The equilibrium can correspond to a fraction of the population using each habitat exclusively, or each individual spending a fraction of its time in each habitat (Hamilton, 2010).

Ideal free distribution (IFD) theory assumes that all individuals have complete knowledge about the distribution of resources among habitats, are free to move between habitats with no cost, are equal competitors, and competition occurs through resource depletion only. If fitness is determined by intake rate of a resource that continuously enters each habitat and is immediately consumed, the spatial distribution of a population at equilibrium is predicted to match the distribution of resource inputs [habitat matching or input matching (Parker, 1978)]. Habitat matching can also occur if fitness is any monotonically increasing function of per capita resource use (Fagen, 1987), not only when fitness is proportional to intake rate.

The IFD maximizes individual fitness; given our assumptions and an IFD, if any individual tries to change habitats, or spend more time in a habitat, the per capita intake rate of that individual will decrease. The densities in each habitat yielding equal expected fitness can be found by plotting the habitat isodar (Morris, 1988). For fixed population sizes, the IFD is also an evolutionarily stable strategy [ESS (Maynard Smith, 1982)] provided fitness decreases with density in a habitat, i.e. the equilibrium cannot be invaded by mutants using another strategy (Cressman and Křivan, 2006). Cressman and Křivan (2010) generalized this result to density-dependent population games. An IFD can be achieved through conditional dispersal between habitats (Morris *et al.*, 2004), as well as through population dynamics alone with no dispersal, as fitness is equal in all habitats at equilibrium (Cressman and Křivan, 2006).

While many theoretical and experimental studies on habitat selection support the prediction of input matching (Milinski, 1979, 1994; Harper, 1982; Godin and Keenleyside, 1984; Parker and Sutherland, 1986; Abrahams, 1989; Kacelnik *et al.*, 1992), an undermatching effect is often observed (Kennedy and Gray, 1993; Earn and Johnstone, 1997; Baum and Kraft, 1998), where the density in the high-quality habitat is not as high as expected under input matching. This may be due to violation of one or more of the assumptions of IFD theory, such as differences in competitive weight between individuals (Grand, 1997), competition resulting from mechanisms other than depletion of resources or other forms of interference, cost of movement between habitats, or perceptual constraints (Abrahams, 1986; Tregenza, 1995; Tregenza *et al.*, 1996).

Undermatching may also be caused by increased risk in higher quality habitats associated with higher population sizes. Aside from starvation and competition, individuals can face additional risks such as predation and food theft by kleptoparasites, both of which can influence the spatial distribution of a population. Several models predict that predators (Hugie and Dill, 1994; Sih, 2005) and kleptoparasites (Parker and Sutherland, 1986; Hamilton, 2002) will congregate in habitats with higher resource availability for their prey or hosts. When predation risk is high, habitat riskiness can become a more important determinant for habitat use than resource levels (Gilliam and Fraser, 1987; Luttbeg and Sih, 2004; Dupuch *et al.*, 2009).

Infection by a pathogen or parasite may also be a risk individuals consider when choosing a habitat. The risk of infection increases with density (and number of contacts) for many

diseases (Anderson and May, 1979; Dwyer, 1991; McCallum *et al.*, 2001; Elliot and Hart, 2010). Since habitats with more resources can support a larger population than poor-quality habitats, they may present a greater risk of disease transmission along with associated physiological or behavioural costs. Infection can lower fitness via a direct impact on fecundity or mortality. Physical malformations or behavioural changes of infected individuals may render them more susceptible to predation (Bakker *et al.*, 1997; Goodman and Johnson, 2011), and reduced activity levels of infected individuals can result in decreased intake rates and fecundity (Hart, 1988). Parasites can also induce behavioural changes in their host (Moore, 1984; Lafferty and Morris, 1996), which increase their own fitness at the expense of their host.

Several species appear to adjust habitat selection when faced with the risk of exposure to infectious disease (Hart, 1994). The giving-up densities of white-tailed deer *Odocoileus virginianus* increase with the density of larval lone star ticks *Amblyomma americanum*, but not predators (Allan *et al.*, 2010). Many species also appear to reduce parasitism rates by using non-foraging sites for defecation (Gilbert, 1997). The eastern grey kangaroo *Marcropus giganteus* and sheep *Ovis aries* avoid foraging from contaminated sites (Hutchings *et al.*, 2001; Garnick *et al.*, 2010), and the bat *Myotis bechsteinii* and the great tit *Parus major* choose uninfected roosts and nesting sites (Christe *et al.*, 1994; Reckardt and Kerth, 2007). The grey treefrog *Hyla versicolor* lays fewer eggs in pools containing snails infected with the trematode *Pseudosuccinea columella* (Kiesecker and Skelly, 2000), and bullfrog (*Rana catesbeiana*) tadpoles avoid conspecifics infected by *Candida humicola* (Kiesecker *et al.*, 1999).

In this study, we consider how predictions about habitat selection may be altered by incorporating the risk of infection by pathogens or parasites, and investigate whether a change in behaviour in response to this risk can lead to undermatching as observed in field studies and experiments. We use a two-habitat susceptible-infected differential equation model to calculate the optimal strategy for habitat choice when disease is present in either one or both habitats. We compare our model predictions to input matching and investigate how factors influencing the risk of infectious disease and cost of infection affect equilibrium habitat choice and the corresponding observed spatial distribution. Specifically, we consider the effect of infected fecundity, infected mortality, transmission rate of the disease, resource input rates, recovery rate, and the probability of vertical transmission or inherited immunity.

THE MODEL

We consider habitat selection by a single population with a choice between two habitats differing only in resource input. Let Q_1 denote the input rate of resources available in habitat 1 and Q_2 denote the input rate of resources in habitat 2. Without loss of generality, we assume habitat 1 is the habitat of higher quality (i.e. $Q_1 > Q_2$). We also assume resources are divisible and are consumed immediately upon entering the habitat [continuous input (Parker and Sutherland, 1986)], and birth rates are directly proportional to the intake rate, $R_i = Q_i/N_i$, where N_i is the density in habitat i ($i = 1, 2$).

We model a system characterized by natal dispersal only; individuals disperse at birth and then choose a habitat and remain there for life. We note that at this scale, we may expect to see only the high-quality habitat occupied at low densities, if resource input rates exceed the maximum intake rate of single individuals (Morris, 1994). However, here we are interested in the case where equilibrium densities are large enough so both habitats are occupied, and disease is supported in the population.

We let the trait p denote the probability that newborns choose to settle in habitat 1 and $1 - p$ denote the probability of choosing habitat 2. We assume the strategy p is genetically determined, with heritability equal to 1. All newborns are susceptible to infection, and individuals become infected through contact with infectious individuals. We consider a disease where the rate of transmission is proportional to the number of infected individuals in a habitat [density-dependent transmission (McCallum *et al.*, 2001)], rather than the proportion of the population that is infected [frequency-dependent transmission (Hethcote, 2000)]. We write a differential equation model for the change in the number of susceptible and infected individuals over time:

$$\begin{aligned}
\dot{S}_1 &= p\left(\frac{b_S Q_1}{S_1 + I_1} S_1 + \frac{b_S Q_2}{S_2 + I_2} S_2 + \frac{b_I Q_1}{S_1 + I_1} I_1 + \frac{b_I Q_2}{S_2 + I_2} I_2\right) - \beta I_1 S_1 - \mu_S S_1 \\
\dot{I}_1 &= \beta I_1 S_1 - \mu_I I_1 \\
\dot{S}_2 &= (1-p)\left(\frac{b_S Q_1}{S_1 + I_1} S_1 + \frac{b_S Q_2}{S_2 + I_2} S_2 + \frac{b_I Q_1}{S_1 + I_1} I_1 + \frac{b_I Q_2}{S_2 + I_2} I_2\right) - \beta I_2 S_2 - \mu_S S_2 \\
\dot{I}_2 &= \beta I_2 S_2 - \mu_I I_2
\end{aligned} \tag{1}$$

where b_S and b_I are the birth rate constants for conversion of resources into offspring for susceptible and infected individuals, respectively. The mortality rates of susceptible and infected individuals are represented by μ_S and μ_I, respectively, and the transmission rate of the disease is given by β. We assume infection by the disease may lower the birth rates of infectious individuals ($b_S \geq b_I$), or it may shorten their lifespan by increasing mortality rates ($\mu_S \leq \mu_I$). A list of parameters is given in Table 1.

Note that total population size is not constant for this model, but rather depends upon parameters such as the quality of the habitats, the probability of infection, as well as the consequences of becoming infected. When there is no disease in the population, the model equilibrates to the following disease-free equilibrium (DFE):

$$(S_1^*, I_1^*, S_2^*, I_2^*) = \left(p\,\frac{b_S(Q_1 + Q_2)}{\mu_S}, 0, (1-p)\,\frac{b_S(Q_1 + Q_2)}{\mu_S}, 0\right). \tag{2}$$

Table 1. Parameters for models (1) and (8)

Parameter	Description
b_S	Susceptible birth rate constant
b_I	Infected birth rate constant
b_R	Recovered birth rate constant
μ_S	Susceptible mortality rate
μ_I	Infected mortality rate
μ_R	Recovered mortality rate
β	Transmission rate of disease
Q_1	Resource level in habitat 1
Q_2	Resource level in habitat 2
τ	Probability of vertical transmission
γ	Recovery rate
δ	Probability of inheriting immunity (from recovered parent)

The DFE is locally asymptotically stable for values of the basic reproductive number $R_0 \doteq \frac{\max(p, 1-p)\,\beta b_S(Q_1+Q_2)}{\mu_I \mu_S} < 1$ and unstable for $R_0 > 1$, where R_0 represents the expected number of new infections produced by introducing one infected individual into a completely susceptible population. As R_0 increases past 1, the disease can first be supported in only one habitat, but eventually the disease is able to persist in both habitats.

We assume individuals choose habitats to maximize their fitness. We define the fitness of an individual choosing habitat i to be the expected number of offspring produced over the course of its lifetime, given that habitat i was chosen at birth. Note the habitat choice of the offspring has no direct impact on the fitness of the parent, as we assume the population is at equilibrium and next-generation offspring do not contribute to fitness. If all individuals are born susceptible, the expected number of offspring produced by an individual in habitat i, denoted $E(O_i)$, is found by multiplying the susceptible birth rate $\left(\frac{b_S Q_i}{N_i^*}\right)$ by the expected amount of time spent susceptible $\left(\frac{1}{\mu_S + \beta I_i^*}\right)$, and adding it to the probability of becoming infected $\left(\frac{\beta I_i^*}{\mu_S + \beta I_i^*}\right)$ times the infected birth rate $\left(\frac{b_I Q_i}{N_i^*}\right)$ multiplied by the expected amount of time spent infected $\left(\frac{1}{\mu_I}\right)$. The susceptible and infected birth rates in each habitat depend on the total density in that habitat (N_i^*), and the probability of infection in each habitat depends on the number of infected individuals in that habitat (I_i^*). We assume the evolution of the strategy p occurs on a longer timescale than the population dynamics. Therefore, the fitness of an individual using each habitat is a function of the equilibrium density of susceptible and infected residents; these densities depend on initial conditions and parameter values, including p. The fitness for using habitat i is given by:

$$F_i = \frac{b_S Q_i}{N_i^*}\frac{1}{\mu_S + \beta I_i^*} + \frac{b_I Q_i}{N_i^*}\frac{1}{\mu_I}\frac{\beta I_i^*}{\mu_S + \beta I_i^*}. \tag{3}$$

The equilibrium p^* can be found by solving $F_1 = F_2$ for p. Individual fitness is maximized in the sense that if all individuals are using the strategy p^*, no individual can increase its fitness by changing strategies. The equilibrium p^* is evolutionarily and convergent stable (see Appendix 1). The value of p^*, or the fraction of the population choosing habitat 1 at birth for which individual fitness is maximized, does not necessarily correspond to the fraction of the population observed in habitat 1 at equilibrium $\left(n^* = \frac{N_1^*}{N_1^* + N_2^*}\right)$, as birth and death rates may differ with the level of disease in the two habitats.

When there is no disease in the population, we can substitute the DFE (2) into equation (3) and solve $F_1 = F_2$, finding input matching: $p^* = n^* = \frac{Q_1}{Q_1 + Q_2}$. In the following sections, we consider three possible ways in which a disease may affect a population, always with a negative impact on fitness, and investigate how p^* and n^* deviate from the disease-free input matching prediction. First, infection may increase mortality rates. Second, the disease may lower fecundity or sterilize infected individuals. We also

Table 2. Effect of increasing model parameters on equilibria p_b^*, p_0^*, n_b^* and n_0^*

Parameter		p_b^*	p_0^*	n_b^*	n_0^*
Infected mortality rate ($b_I = 1$)	μ_I	0	0	−	− then +
Infected mortality	μ_I	− then +	+	−	− then +
Infected birth rate ($\mu_I = 0.1$)	b_I	+	+	+	+
Infected birth rate	b_I	+	+	+	+
Transmission rate	β	+	−	+	−
Total resource level	$Q_1 + Q_2$	+	−	+	−
Recovery rate	γ	+	+	+	+
Probability of vertical transmission	τ	+	N.A.	+	N.A.
Probability of inheriting immunity	δ	+	+	+	+

Note: Unless otherwise indicated, parameter values used are: $b_S = 1$, $b_I = 0.5$, $Q_1 = 10$, $Q_2 = 5$, $b_R = 1$, $\mu_S = 0.1$, $\mu_I = 0.2$, $\mu_R = 0.1$, $\beta = 0.01$, $\gamma = 0$, $\tau = 0$, $\delta = 0$. To calculate the equilibrium strategy, we use MATLAB (version R2011b) to find the value of p for which $F_1 = F_2$, obtaining S_1^*, I_1^*, S_2^* and I_2^* as a function of p by numerically solving the differential equations (1) (or (8) if $\gamma > 0$) and observing the limiting behaviour. To find p_b^* we use initial conditions of $S_1(0) = S_2(0) = 100$, $I_1(0) = I_2(0) = 1$. To find p_0^* we use initial conditions of $S_1(0) = 100$, $S_2(0) = 101$, $I_1(0) = 1$, $I_2(0) = 0$. The observed proportion of the population in habitat 1 at equilibrium n_b^* (n_0^*) is then $\frac{S_1^* + I_1^*}{S_1^* + I_1^* + S_2^* + I_2^*}$, with equilibrium values calculated from the respective initial conditions.

consider the case where the disease affects both birth and death rates. For each, we consider the case where both habitats are infected (denoting equilibria p_b^*, n_b^*) as well as when only the higher quality habitat is infected (denoting equilibria p_0^*, n_0^*). Results are summarized in Table 2.

Case 1: Disease affects only host mortality

In general, we cannot find a mathematically tractable closed form solution for the endemic equilibrium of model (1). However, if infection by disease has no effect on host fecundity, then we let $b_S = b_I = b$, and we can solve for the equilibrium number of susceptible and infected individuals in each habitat (denoted by asterisks) when the disease is present in both habitats:

$$(S_1^*, I_1^*, S_2^*, I_2^*) = \left(\frac{\mu_I}{\beta}, \frac{pb(Q_1 + Q_2)}{\mu_I} - \frac{\mu_S}{\beta}, \frac{\mu_I}{\beta}, \frac{(1-p)b(Q_1 + Q_2)}{\mu_I} - \frac{\mu_S}{\beta} \right). \tag{4}$$

This endemic equilibrium (4) is stable for parameter values resulting in $\frac{\min(p, 1-p)\,\beta b_S (Q_1 + Q_2)}{\mu_I \mu_S} > 1$, or equivalently, parameter values for which (4) is positive. There are two additional equilibria corresponding to the disease being present in only one habitat. We are interested in the case where choosing the high-quality habitat, habitat 1, is associated with a risk of disease transmission and the low-quality habitat is disease free:

$$(S_1^*, I_1^*, S_2^*, I_2^*) = \left(\frac{\mu_I}{\beta}, \frac{pb(Q_1 + Q_2)}{\mu_I} - \frac{\mu_S}{\beta}, (1-p)\frac{b(Q_1 + Q_2)}{\mu_S}, 0 \right). \tag{5}$$

Equilibrium (5) is stable for parameter values resulting in $\frac{p\beta b_S(Q_1+Q_2)}{\mu_I\mu_S}>1$ and $\frac{(1-p)\beta b_S(Q_1+Q_2)}{\mu_I\mu_S}<1$, or equivalently, parameter values for which equilibrium (5) is non-negative but (4) is not; it becomes unstable when $\frac{(1-p)\beta b_S(Q_1+Q_2)}{\mu_I\mu_S}>1$, but for this parameter range we can consider the situation where one habitat is disease free as long as no infected individuals are introduced into that habitat (or the habitat is kept disease free via management). Recall that with the model we are considering, the disease cannot spread between habitats, since residency is determined at birth and all individuals are born susceptible. When $\frac{p\beta b_S(Q_1+Q_2)}{\mu_I\mu_S}<1$, the disease-free equilibrium (2) is stable and the disease cannot persist in either habitat.

We first consider habitat selection when both habitats are infected. Substituting the endemic equilibrium (4) into equation (3), we find both habitats yield equal fitness when $p_b^*=\frac{Q_1}{Q_1+Q_2}$. Recall the probability of a newborn individual choosing to reside in habitat 1 is not necessarily equal to the fraction of the population in habitat 1 at equilibrium. When both habitats are infected,

$$\frac{N_1^*}{N_2^*}=\frac{b\beta Q_1+\mu_I(\mu_I-\mu_S)}{b\beta Q_2+\mu_I(\mu_I-\mu_S)}. \tag{6}$$

We see $\frac{N_1^*}{N_2^*}<\frac{Q_1}{Q_2}$ and thus $n_b^*<\frac{Q_1}{Q_1+Q_2}$ if $\mu_I>\mu_S$. Therefore, habitat 1 is underused (undermatched) compared with the input matching expected in the absence of disease, provided infection increases mortality rates. As the infected mortality rate μ_I increases from the susceptible mortality rate μ_S, the equilibrium strategy p_b^* does not change, whereas the observed fraction of the population in habitat 1 at equilibrium, n_b^*, decreases (Fig. 1). The same fraction of offspring are choosing habitat 1 at birth, but fewer are present in habitat 1 at equilibrium compared with the disease-free case. Fitness is equal in the two habitats as individuals trade off shorter lifespan for a higher reproductive rate in habitat 1.

A larger infected mortality rate also implies a shortened infectious period, reducing the amount of time available to spread the disease. As μ_I increases, the equilibrium densities of susceptibles in both habitats (S_1^* and S_2^*) increase at the same constant rate, whereas the density of infecteds in habitat 1 (I_1^*) decreases faster than those in habitat 2 (I_2^*). Therefore, while we do not see a change in habitat selection in response to the risk of infectious disease (p_b^* remains the input matching prediction), undermatching is still observed because infection depends on density.

If the disease is only present in the high-quality habitat (habitat 1), the value of p that yields equal fitness is $p_0^*=\frac{Q_1}{Q_1+Q_2}$. If we look at the population sizes in the two habitats at equilibrium, we see an observed ratio of

$$\frac{N_1^*}{N_2^*}=\frac{\mu_S}{bQ_2}\left(\frac{bQ_1}{\mu_I}+\frac{\mu_I-\mu_S}{\beta}\right). \tag{7}$$

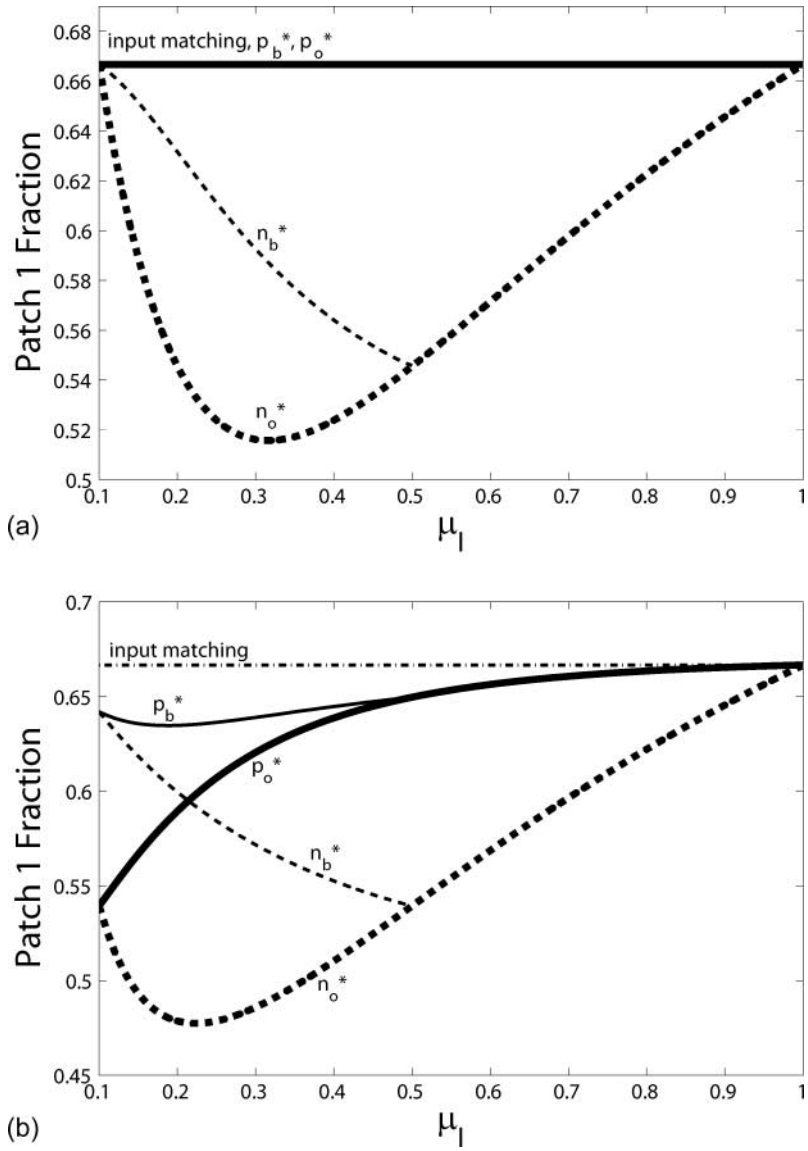

Fig. 1. The effect of infected mortality (μ_I) on the equilibrium probability of choosing habitat 1 at birth (p_b^* when both habitats are infected, p_0^* when only habitat 1 is infected) and the fraction of the population observed in habitat 1 at equilibrium (n_b^* when both habitats are infected, n_0^* when only habitat 1 is infected). In (a) disease affects host mortality only ($b_I = b_S = 1$), whereas in (b) disease affects both mortality and fecundity ($b_I = 0.5$). Other parameter values: $b_S = 1$, $\mu_S = 0.1$, $\beta = 0.01$, $Q_1 = 10$, $Q_2 = 5$, $\gamma = 0$, $\tau = 0$, $\delta = 0$.

Assuming $\mu_I > \mu_S$, this fraction is less than the input matching fraction of $\frac{Q_1}{Q_2}$, so $n_0^* < \frac{Q_1}{Q_1 + Q_2}$, for $\frac{\beta b Q_1}{\mu_S \mu_I} > 1$. We note this condition is equivalent to $R_0 > 1$ with $p = \frac{Q_1}{Q_1 + Q_2}$.

Thus the equilibrium proportion of the population choosing habitat 1 at birth again remains the same as input matching predictions, yet undermatching is again observed. As μ_I increases from μ_S, the population size in disease-free habitat 2 remains constant at N_2^*, and undermatching first increases and then decreases along with the population size in habitat 1, N_1^*. Furthermore, for a fixed set of parameter values, the proportion of the population in habitat 1 is much lower when the disease is only present in habitat 1 than when both habitats are infected (Fig. 1a).

Case 2: Disease affects only host fecundity

If the disease does not affect mortality, so $\mu_I = \mu_S$, and instead lowers birth rates, so $b_I < b_S$, then the value of p for which individual fitness is maximized ($F_1 = F_2$) is no longer necessarily the input matching prediction of $\frac{Q_1}{Q_1 + Q_2}$. To calculate the equilibrium strategy without an analytical expression for the population dynamics equilibrium, we use MATLAB (version R2011b) to find the value of p for which $F_1 = F_2$, obtaining S_1^*, I_1^*, S_2^* and I_2^* as a function of p by numerically solving the differential equations (1) and observing the limiting behaviour. As the birth rate of infected individuals decreases (relative to susceptible birth rate), we see a decrease in relative use of the high-quality habitat. Both p_b^* and p_0^* are less than the input matching prediction, with $p_0^* < p_b^*$ (Fig. 2a; Table 2). However, since mortality rates are not affected by infection, the observed fraction of the total population in habitat 1 at equilibrium is now equal to the fraction choosing habitat 1 at birth ($n_b^* = p_b^*$ and $n_0^* = p_0^*$). In this case, undermatching is caused by a behavioural change in response to the risk of infectious disease, rather than direct density effects of infection.

Case 3: Disease affects both mortality and fecundity

In the case where both mortality and birth rates are negatively affected by pathogen or parasite infection, we see a combination of the density and behavioural effects for cases 1 and 2 (Figs. 1b and 2b). The equilibrium fraction of the population choosing habitat 1 at birth may be lower than the input matching prediction, and the observed fraction of the population inhabiting habitat 1 at equilibrium may be even less $\left(n_0^* \le p_0^* \le \frac{Q_1}{Q_1 + Q_2}, n_b^* \le p_b^* \le \frac{Q_1}{Q_1 + Q_2}\right)$. Again, undermatching is greatest when habitat 2 is disease free ($n_0^* \le n_b^*$, $p_0^* \le p_b^*$).

Role of transmission rate and resource input rate

The parameters b_I and μ_I determine the severity of fitness consequences for an individual who becomes infected with the disease. However, other model parameters may also affect the overall risk or cost of infection and can influence how an animal changes its habitat selection strategy to avoid becoming infected. In this section, we explore the effect of changes in transmission rate and resource input rates on the equilibrium strategy and distribution of the population.

Increasing the transmission rate of the disease (β) increases the probability an individual will become infected, either by increasing the contact rate or chance of transmission per

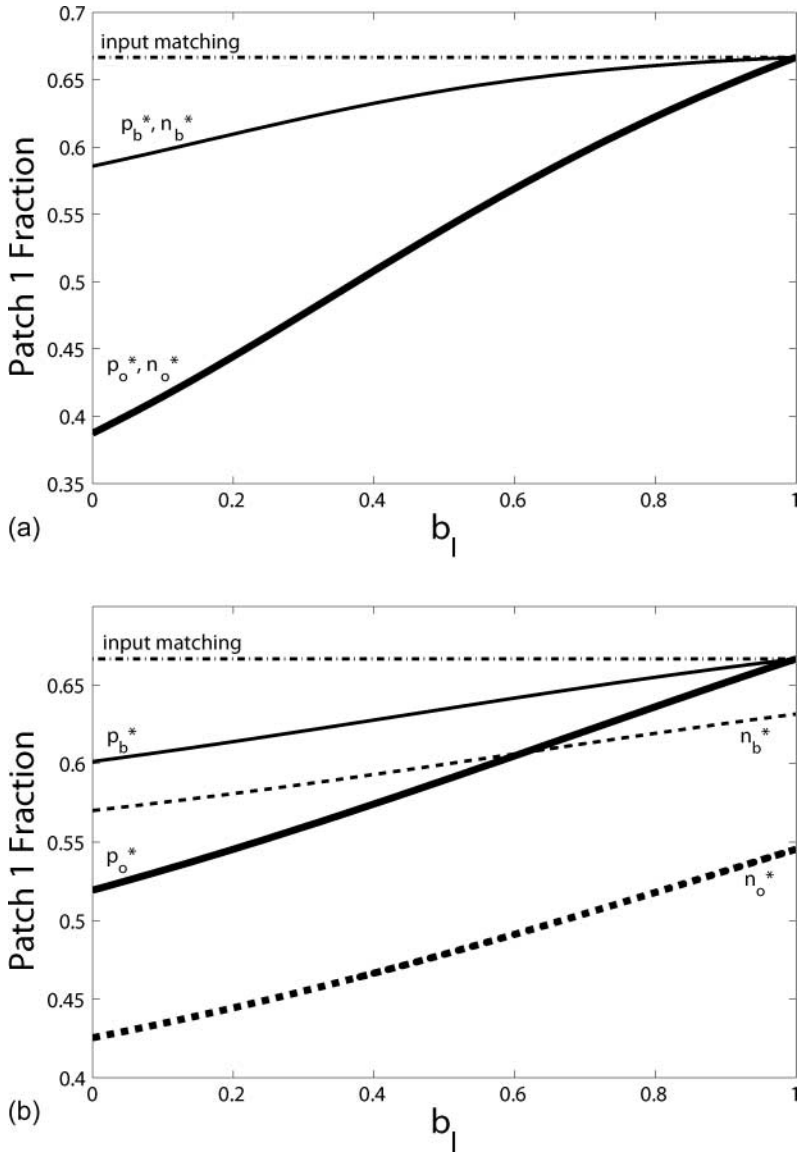

Fig. 2. The effect of infected birth rate on the equilibrium probability of choosing habitat 1 at birth (p_b^* when both habitats are infected, p_0^* when only habitat 1 is infected) and the fraction of the population observed in habitat 1 at equilibrium (n_b^* when both habitats are infected, n_0^* when only habitat 1 is infected). In (a) disease affects fecundity only ($\mu_I = \mu_S = 0.1$), whereas in (b) disease affects both mortality and fecundity ($\mu_I = 0.2$). Other parameter values: $b_S = 1$, $\mu_S = 0.1$, $\beta = 0.01$, $Q_1 = 10$, $Q_2 = 5$, $\gamma = 0$, $\tau = 0$, $\delta = 0$.

contact, and thus increases the level of disease that can be sustained in the population. If both habitats are infected, increasing the transmission rate leads to an increase in the relative use of the high-quality habitat (increased p_b^* and n_b^*; Fig. 3; Table 2). We note that for higher values of μ_I, there may be an initial decrease in the proportion of individuals

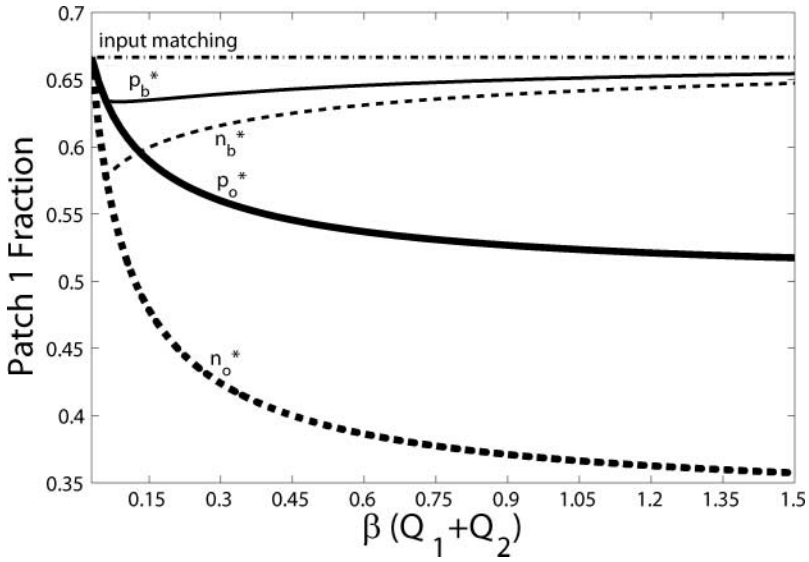

Fig. 3. The effect of transmission rate (β) and total resource input ($Q_1 + Q_2$) on the equilibrium probability of choosing habitat 1 at birth (p_b^* when both habitats are infected, p_0^* when only habitat 1 is infected) and the fraction of the population observed in habitat 1 at equilibrium (n_b^* when both habitats are infected, n_0^* when only habitat 1 is infected). Resource inputs are fixed at a 2:1 ratio between habitats 1 and 2. Figure generated by fixing $\beta = 0.01$ and varying $Q_1 + Q_2$ from 3 to 150, and by fixing $Q_1 + Q_2 = 15$ and varying β from 0.002 to 0.1. Other parameter values: $b_S = 1$, $b_I = 0.5$, $\mu_S = 0.1$, $\mu_I = 0.2$, $\gamma = 0$, $\tau = 0$, $\delta = 0$.

choosing habitat 1 (p_b^*) for low β; when β is large enough, p_b^* increases with β. If only habitat 1 is infected, increasing the transmission rate causes a decrease in both p_0^* and n_0^* (Fig. 3; Table 2), resulting in a decrease in the use of habitat 1.

Increasing the total input level of resources ($Q_1 + Q_2$) increases fecundity for both susceptible and infected individuals, along with the total population size and level of disease that can be sustained in both habitats. An increase in density, and incidence of disease, leads to an increased use of the high-quality habitat when both habitats are infected (but a decreased use of the high-quality habitat when it is the only habitat infected). Increasing the overall resource input level (while maintaining a 2:1 ratio) affects p_b^*, p_0^*, n_b^* and n_0^* in the same way as increasing β (Fig. 3; Table 2).

When both habitats are infected, increasing β results in a lower total population size in each infected habitat by reducing the equilibrium number of susceptibles and increasing the equilibrium number of infecteds. Increasing $Q_1 + Q_2$ increases total population size by increasing the equilibrium number of infected individuals, and has no effect on susceptible density. In both cases, I_1^* increases more quickly than I_2^*, leading to an increase in n_b^* and reducing observed undermatching. At low values of $Q_1 + Q_2$ and β, the prevalence of disease, and corresponding risk of infection, may increase more quickly in habitat 1 than habitat 2 when μ_I is large, causing an initial decrease in p_b^* despite the increase in n_b^*. We still observe an increase in the use of habitat 1, even though a small proportion of newborns are choosing to settle there. As $Q_1 + Q_2$ or β increases, the proportion of the population infected at equilibrium increases, approaching 1 in both habitats; p_b^* increases towards the input

matching prediction as the risk associated with choosing the high-quality habitat over the low-quality habitat is reduced.

When only habitat 1 is infected, both p_0^* and n_0^* decrease with $Q_1 + Q_2$ or β. As disease prevalence rises in habitat 1 while habitat 2 remains disease free, the difference in risk between the two habitats increases and the relative use of habitat 1 decreases. The degree of observed undermatching is again much greater when infection is only in habitat 1 compared with both habitats.

Extended model

Up to this point, we have assumed that all individuals are born into the susceptible class, with the only means of disease transmission via contact with an infected individual. Also, once individuals are infected by the disease, they are infected for life. In this section, we relax these assumptions by incorporating vertical as well as horizontal transmission of the disease, allowing recovery from the disease, and including the possibility of inherited immunity from a recovered parent.

To investigate the effect on habitat choice if the disease can be transmitted from parent to offspring at birth (Anderson and May, 1979; Jones *et al.*, 2011), we assume individuals inherit the disease from an infected parent with probability τ. We also assume infection status does not affect habitat choice. That is, newborns choose a habitat at birth to reside in for life, with p again representing the probability of choosing habitat 1 for all newborns.

We allow infected individuals to recover from the disease at a rate γ. We assume that when individuals recover they enter a recovered class, subscripted by R, and thereafter have

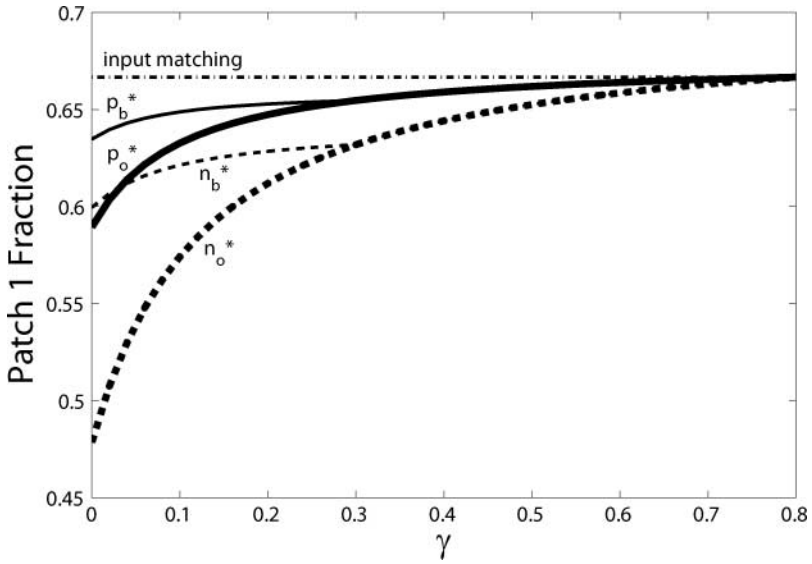

Fig. 4. The effect of recovery rate (γ) on the equilibrium probability of choosing habitat 1 at birth (p_b^* when both habitats are infected, p_0^* when only habitat 1 is infected) and the fraction of the population observed in habitat 1 at equilibrium (n_b^* when both habitats are infected, n_0^* when only habitat 1 is infected). Parameter values: $b_S = 1$, $b_I = 0.5$, $b_R = 1$, $\mu_S = 0.1$, $\mu_I = 0.2$, $\mu_R = 0.1$, $\beta = 0.01$, $\tau = 0$, $\delta = 0$.

lifelong immunity. Recovered individuals have a birth rate constant b_R and a death rate μ_R, which we will assume to be equal to the analogous parameters for susceptible individuals.

If parents are in a high-risk environment, they may invest some of their resources into immunity for their offspring (Tschirren *et al.*, 2004). We consider the case where offspring have a non-zero probability of inheriting immunity from a parent in the recovered class. Let the parameter δ denote the fraction of offspring of recovered individuals born with immunity to the disease (joining the recovered class). Then $1-\delta$ is the probability these offspring are born susceptible.

Model (1) with these modifications is given by the following set of differential equations:

$$\begin{aligned}
\dot{S}_1 &= p\left(\frac{b_S Q_1}{N_1} S_1 + \frac{b_S Q_2}{N_2} S_2 + (1-\delta)\left(\frac{b_R Q_1}{N_1} R_1 + \frac{b_R Q_2}{N_2} R_2\right) + \right. \\
&\qquad \left. (1-\tau)\left(\frac{b_I Q_1}{N_1} I_1 + \frac{b_I Q_2}{N_2} I_2\right)\right) - \beta I_1 S_1 - \mu_S S_1 \\
\dot{I}_1 &= \tau p\left(\frac{b_I Q_1}{N_1} I_1 + \frac{b_I Q_2}{N_2} I_2\right) + \beta I_1 S_1 - \gamma I_1 - \mu_I I_1 \\
\dot{R}_1 &= p\delta\left(\frac{b_R Q_1}{N_1} R_1 + \frac{b_R Q_2}{N_2} R_2\right) + \gamma I_1 - \mu_R R_1 \\
\dot{S}_2 &= (1-p)\left(\frac{b_S Q_1}{N_1} S_1 + \frac{b_S Q_2}{N_2} S_2 + (1-\delta)\left(\frac{b_R Q_1}{N_1} R_1 + \frac{b_R Q_2}{N_2} R_2\right) + \right. \\
&\qquad \left. (1-\tau)\left(\frac{b_I Q_1}{N_1} I_1 + \frac{b_I Q_2}{N_2} I_2\right)\right) - \beta I_2 S_2 - \mu_S S_2 \\
\dot{I}_2 &= \tau(1-p)\left(\frac{b_I Q_1}{N_1} I_1 + \frac{b_I Q_2}{N_2} I_2\right) + \beta I_2 S_2 - \gamma I_2 - \mu_I I_2 \\
\dot{R}_2 &= (1-p)\delta\left(\frac{b_R Q_1}{N_1} R_1 + \frac{b_R Q_2}{N_2} R_2\right) + \gamma I_2 - \mu_R R_2
\end{aligned} \tag{8}$$

where $N_i = S_i + I_i + R_i$, $i = 1, 2$. We note that by setting $\tau = 0$, $\gamma = 0$, and $\delta = 0$, the model reduces to equations (1) provided $R_1(0) = R_2(0) = 0$.

Our measure of fitness is again lifetime reproductive success, and the fitness function (3) can be modified to take into account these changes to the model (see Appendix 2). We can again numerically determine the value of p for which the fitness functions in the two habitats are equal, and compute the corresponding proportion of the equilibrium population in habitat 1. Figures 4–6 illustrate how p_b^*, p_0^*, n_b^* and n_0^* change with the length of the infectious period, probability of vertical transmission, and the probability of inheriting immunity from a recovered parent.

As the recovery rate (γ) increases, the time spent infectious decreases; this results in an increase in both p^* and n^* regardless of whether one or both habitats are infected (Fig. 4; Table 2). A faster recovery rate reduces the negative impact on fitness from becoming infected, as well as the amount of time infectious individuals are able to transmit the disease, reducing the risk of infection to others as well. Thus increasing γ reduces the

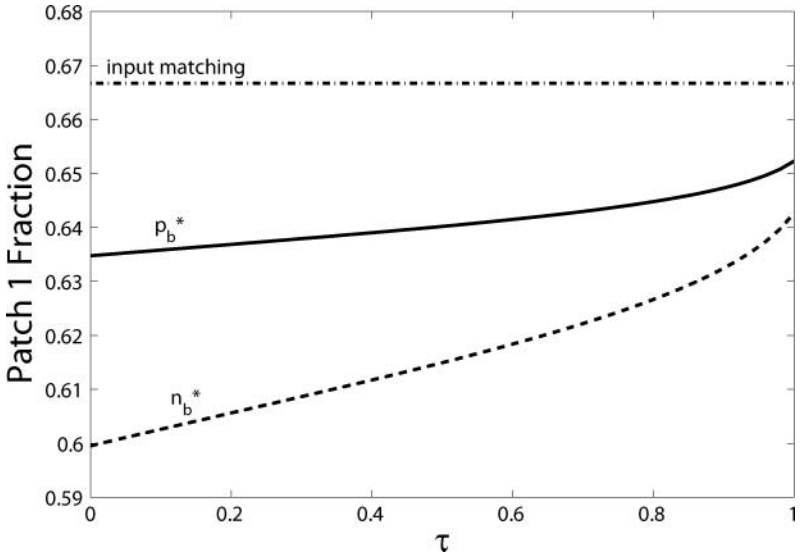

Fig. 5. The effect of the probability of vertical transmission (τ) on the equilibrium probability of choosing habitat 1 at birth (p_b^*) and the fraction of the population observed in habitat 1 at equilibrium (n_b^*). Parameter values: $b_S = 1$, $b_I = 0.5$, $\mu_S = 0.1$, $\mu_I = 0.2$, $\beta = 0.01$, $\tau = 0$, $\delta = 0$.

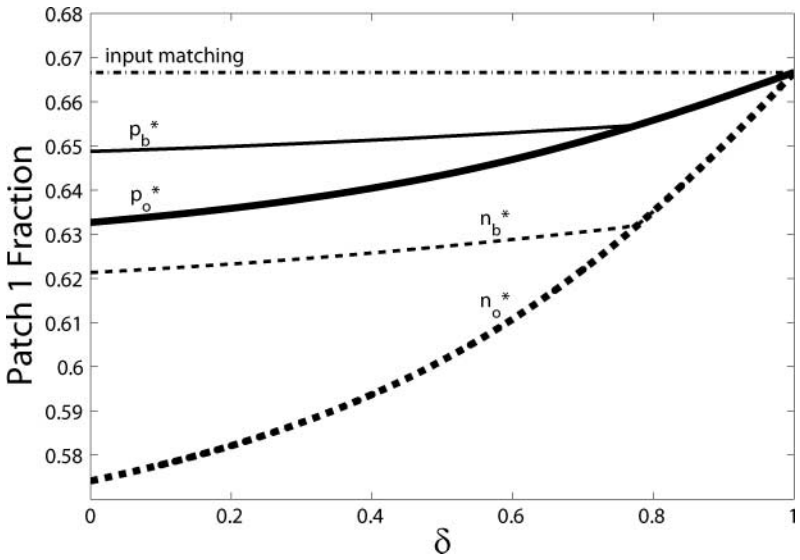

Fig. 6. The effect of the probability of inheriting immunity (δ) on the equilibrium probability of offspring choosing habitat 1 at birth (p_b^* when both habitats are infected, p_0^* when only habitat 1 is infected) and the fraction of the population observed in habitat 1 at equilibrium (n_b^* when both habitats are infected, n_0^* when only habitat 1 is infected). Parameter values: $b_S = 1$, $b_I = 0.5$, $b_R = 1$, $\mu_S = 0.1$, $\mu_I = 0.2$, $\mu_R = 0.1$, $\beta = 0.01$, $\tau = 0$, $\gamma = 0.1$.

relative cost of choosing habitat 1 over habitat 2 and leads to a distribution closer to input matching. If γ is sufficiently high, the disease can be eliminated from the low-quality habitat or even both habitats.

Certain pathogens and parasites can be passed from parent to offspring at birth (Knell and Webberley, 2004). Increasing the probability of vertical transmission of the disease (τ) increases the overall prevalence of the disease, causing an increase in p_b^* and n_b^* (Fig. 5; Table 2). Only the results for both habitats infected are shown, as habitat 2 cannot be kept disease free when newborns can be born infected. While the risk of infection increases in both habitats with τ, the difference in risk between the two habitats decreases, causing an increase in the relative use of habitat 1. For the extreme case when $\tau = 1$, or offspring of infected individuals are always born infected, susceptible levels may be reduced to zero if infected mortality levels are low enough. When the entire population is infected, the risk of disease no longer influences habitat selection and there are no differences in mortality rates between habitats, so the equilibrium distribution returns to input matching. If infected mortality rates are high enough to maintain a positive level of susceptibles, then undermatching is still observed for p_b^* and n_b^* when $\tau = 1$.

Finally, we consider the effect of recovered individuals transferring immunity to their offspring. Increasing the probability of acquiring immunity at birth from a recovered parent (δ) leads to an increase in p_b^*, p_0^*, n_b^* and n_0^* (Fig. 6; Table 2). For individuals born immune, disease poses no risk and should not influence their habitat selection. Increasing the probability of inheriting immunity (when there is a positive recovery rate) increases the amount of otherwise susceptible individuals removed from the at-risk population and decreases overall levels of disease.

DISCUSSION

Our results show that the risk of infectious disease can have a substantial impact on habitat selection and the resulting spatial distribution of populations across habitats differing only in resource quality (and thus in potential risk of disease). When infection by a pathogen or parasite has a negative impact on fitness, our model predicts decreased use of the high-quality habitat compared with input matching. Overmatching, or overuse of the high-quality habitat, is never predicted by our model, as we assume disease has only negative fitness consequences. The undermatching observed at equilibrium may be due to a change in optimal habitat selection or direct density effects of infection occurring after natal dispersal. The degree to which the high-quality habitat is underused varies with the fitness consequences of becoming infected with the disease as well as the risk of infection associated with each habitat.

The degree of undermatching increases with the difference in risk between the habitats. An increase in disease prevalence in both habitats can reduce undermatching if it reduces the relative cost of choosing the higher quality habitat. The implication is that the risk of disease plays less of a role in habitat selection as equilibrium density increases, when all potential habitats harbour disease. Some other models predict that undermatching becomes more pronounced at high densities (where populations are not necessarily at equilibrium). For example, in a model of habitat selection under perceptual constraints, individuals are less able to gather accurate information about their choices for habitat selection at high densities and become more likely to choose randomly (Abrahams, 1986), a result confirmed experimentally in zebra fish by Gillis and Kramer (1987).

If only the high-quality habitat is infected, then an increase in disease prevalence increases the difference in risk between the habitats, causing a decrease in the use of the infected habitat. For a given set of parameter values resulting in a stable endemic

equilibrium, we see the largest difference in risk between the two habitats when one habitat is disease free. Disease-free habitats can result in extreme undermatching due to the diseased habitat being unable to maintain large population sizes. Recall the disease-free equilibrium for the low-quality habitat is often unstable, and any introduction of disease would result in an outbreak of infected individuals.

In our model, undermatching is caused by decreased use of the high-quality habitat rather than overuse of the low-quality habitat. Therefore, the individuals using habitat 1 have a higher intake rate over a shorter lifespan. If a similar, competing species (or mutant of the original species) is immune to the disease, it would have an advantage in the high-quality habitat. The species susceptible to infection might then use the low-quality habitat, with the immune species alone using the high-quality habitat. Habitat selection could thus cause extinction of the disease from the high-quality habitat, but also the differential susceptibility of the competing species may provide a mechanism to allow for co-existence (Holt, 2010).

Our findings indicate that infectious disease can affect the spatial distributions of populations through more than just direct effects on fecundity and mortality. Disease can also influence density through habitat selection that affects the ability of the disease to transmit itself. Avoidance of a diseased habitat may lower susceptible density and reduce R_0 for the disease in that habitat with implications for the evolution of pathogen virulence, and the co-evolution of host and pathogen. Also, when there is no mixing between habitats, and all dispersing newborns are susceptible, the transmissibility and virulence of the disease is likely to evolve differently in each habitat.

There are many parallels between predators and parasites/pathogens, and recently there have been efforts to describe their ecological similarities and differences (Hatcher *et al.*, 2006; Borer *et al.*, 2007; Johnson *et al.*, 2008; Raffel *et al.*, 2008; Rohr *et al.*, 2009). The threat of predation is known to affect habitat choice (Hugie and Dill, 1994; Grand and Dill, 1999; Luttbeg and Sih, 2004) and can also lead to undermatching. If predators as well as prey are free to move between habitats, a 'leapfrogging' effect is predicted, where predators distribute themselves in proportion to the prey's resource levels (Hugie and Dill, 1994; Sih, 2005). Thus larger numbers of predators gather in high-resource habitats, resulting in fewer numbers of prey in those habitats, relative to input matching (Hammond *et al.*, 2007). Our results show that animals may also adjust habitat selection in response to the risk of infectious disease in a similar manner, and one must consider the possibility that undermatching observed in nature is caused by the presence of parasites or pathogens as well as predators, especially when potential habitats differ in their risk of infection.

The risks of parasitism and predation are not always independent, and may simultaneously affect habitat selection. Infection often results in behavioural changes (Moore, 1984; Hart, 1988; Levri, 1999) that influence the risk of predation. Reduced activity may minimize predation risk (Hart, 1988), while parasitic infections may induce behavioural or physiological changes in their hosts that facilitate further transmission of the parasite (Bakker *et al.*, 1997; Levri, 1999; Johnson *et al.*, 2006; Goodman and Johnson, 2011), often by harming the host and increasing the probability of predation. The behavioural choices individuals make to avoid predation risk can also affect their vulnerability to infection or the probability of becoming infected (Decaestecker *et al.*, 2002). When individuals experience both the risks of predation and infection, defences against one type of enemy may result in increased susceptibility to the other (Rigby and Jokela, 2000). Furthermore, if parasites can pass from prey to predators, diseased prey might potentially be easier to capture while posing a threat of infection to the predator

(Lafferty, 1992). The interaction between the risks of predation and parasites/pathogens and the behavioural changes in response to these risks likely all play a role in habitat selection, and present many possibilities for future research.

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APPENDIX 1: STABILITY OF p^*

Evolutionary stability

In this appendix, we show evolutionary stability of the equilibrium p^* found by setting $F_1 = F_2$, where F_i is as in equation (3). We let p^* be the resident strategy and $\tilde{p}$ denote the strategy of a potential invader ($0 \le p^*$, $\tilde{p} \ge 1$). Assuming a small fraction δ of the population is using the invader strategy and $1 - \delta$ is using the resident strategy, the state of the population is given by

$$\sigma = \sigma(\tilde{p}, p^*) = \delta\tilde{p} + (1 - \delta)p^*. \tag{A1}$$

The strategy p^* is an evolutionarily stable strategy (ESS) if the relative fitness of an invader is maximized at $\tilde{p} = p^*$ in a resident population with strategy p^*. We need to check that

$$\frac{\partial}{\partial\tilde{p}}\left(W(\tilde{p}, \sigma) - W(p^*, \sigma)\right)\Big|_{\tilde{p}=p^*} = 0 \tag{A2}$$

and

$$\frac{\partial^2}{\partial\tilde{p}^2}\left(W(\tilde{p}, \sigma) - W(p^*, \sigma)\right)\Big|_{\tilde{p}=p^*} < 0 \tag{A3}$$

where

$$W(\tilde{p}, \sigma) = \tilde{p}F_1(\sigma) + (1 - \tilde{p})F_2(\sigma) \tag{A4}$$

and $W(p^*, \sigma)$ is defined similarly. Taking the derivative of $W(\tilde{p}, \sigma) - W(p^*, \sigma)$ with respect to $\tilde{p}$, we have

$$\begin{aligned}\frac{\partial}{\partial\tilde{p}}\left(W(\tilde{p}, \sigma) - W(p^*, \sigma)\right) = \\ F_1(\sigma) - F_2(\sigma) + \tilde{p}\frac{\partial F_1}{\partial\sigma}\delta + (1 - \tilde{p})\frac{\partial F_2}{\partial\sigma}\delta - p^*\frac{\partial F_1}{\partial\sigma}\delta - (1 - p^*)\frac{\partial F_2}{\partial\sigma}\delta.\end{aligned} \tag{A5}$$

Evaluating at $\tilde{p} = p^*$, (A5) is equal to 0, since $F_1(\sigma) = F_2(\sigma)$. Therefore, (A2) holds.

To show (A3) holds, we take the derivative of (A5) with respect to $\tilde{p}$ and evaluate at $\tilde{p} = p^*$:

$$\frac{\partial}{\partial\tilde{p}}\left(F_1(\sigma) - F_2(\sigma)\right)\Big|_{\tilde{p}=p^*} + \delta\left(\frac{\partial F_1}{\partial\sigma} - \frac{\partial F_2}{\partial\sigma}\right)\Big|_{\tilde{p}=p^*} < 0 \tag{A6}$$

since $\frac{\partial F_1}{\partial N_1} < 0$, $\frac{\partial N_1}{\partial\sigma} > 0$, $\frac{\partial F_2}{\partial N_2} < 0$, and $\frac{\partial N_2}{\partial\sigma} > 0$ implies $\frac{\partial F_1}{\partial\sigma} = \frac{\partial F_1}{\partial N_1}\frac{\partial N_1}{\partial\sigma} < 0$ and $\frac{\partial F_2}{\partial\sigma} = \frac{\partial F_2}{\partial N_2}\frac{\partial N_2}{\partial\sigma} > 0$, where N_i is the equilibrium population size in habitat i for the population distribution given by (A1).

Convergence stability

Knowing that p^* is an ESS is not enough to guarantee that the population approaches this equilibrium. To show that p^* is convergent stable (that is, if the population is using a nearby strategy, it will move towards p^*), we must also check that the following expression (McGill and Brown, 2007)

$$\frac{\partial^2}{\partial \tilde{p}^2}\left(W(\tilde{p},\sigma)-W(p^*,\sigma)\right)+\frac{\partial^2}{\partial \tilde{p}\partial p^*}\left(W(\tilde{p},\sigma)-W(p^*,\sigma)\right)+ \frac{\partial^2}{\partial \tilde{p}\partial N}\left(W(\tilde{p},\sigma)-W(p^*,\sigma)\right)\frac{\partial N^*}{\partial p^*} \quad \text{(A7)}$$

is less than zero when evaluated at $\tilde{p}=p^*$. The first term of (A7) is equal to (A6), which is negative. To compute the second term, we take the derivative of (A5) with respect to p^* and evaluate at $\tilde{p}=p^*$:

$$\frac{\partial}{\partial p^*}\left(F_1(\sigma)-F_2(\sigma)\right)|_{\tilde{p}=p^*}+\delta\left(\frac{\partial F_1}{\partial \sigma}-\frac{\partial F_2}{\partial \sigma}\right)|_{\tilde{p}=p^*}<0 \quad \text{(A8)}$$

similarly to the calculation for (A6). The last term of (A7) is zero if a change in strategy has no effect on population size. However, for our model, an increase in the fraction of the population choosing habitat 1 at birth can affect the population size in each habitat. Numerical results show an increase in population strategy p^* results in a lower equilibrium total population size:

$$\frac{\partial N^*}{\partial p^*}|_{\tilde{p}=p^*}<0. \quad \text{(A9)}$$

Also, assuming an increase of size ΔN corresponds to an increase of $n^*\Delta N$ in habitat 1 and $(1-n^*)\Delta N$ in habitat 2, numerical results show

$$\frac{\partial^2}{\partial \tilde{p}\partial N}\left(W(\tilde{p},\sigma)-W(p^*,\sigma)\right)|_{\tilde{p}=p^*}=\frac{\partial}{\partial N}\left(F_1(\sigma)-F_2(\sigma)\right)|_{\tilde{p}=p^*}>0. \quad \text{(A10)}$$

Therefore, (A7) is negative and p^* is convergent stable.

APPENDIX 2: EXTENDED FITNESS FUNCTIONS

The fitness functions for each habitat must be modified for the extended model (8). We again measure fitness for an individual using habitat i by the expected number of offspring ($E(O_i)$) produced over the course of the individual's lifetime. Individuals are born susceptible (BS), born infected (BI), or born recovered (BR), so

$$F_i=E(O_i)=P(BS)E(O_i|BS)+P(BI)E(O_i|BI)+P(BR)E(O_i|BR). \quad \text{(A11)}$$

An individual can be born susceptible if its parent is susceptible (PS), if the parent is infected (PI) and offspring do not inherit the disease (DN), or if the parent has recovered (PR) and offspring do not inherit immunity (IN). Then

$$P(BS)=P(PS)+P(PI\cap DN)+P(PR\cap IN)= P(PS)+P(DN|PI)\,P(PI)+P(IN|PR)\,P(PR) \quad \text{(A12)}$$

where $P(DN|PI) = 1 - \tau$ and $P(IN|PR) = 1 - \delta$. An individual is born infected only if the parent is infected (PI) and if offspring inherit the disease (DI). Thus

$$P(BI) = P(PI \cap DI) = P(DI|PI)\,P(PI) \tag{A13}$$

where $P(DI|PI) = \tau$. An individual is born recovered if the parent is recovered (PR) and the offspring inherit immunity (II). Then

$$P(BR) = P(PR \cap II) = P(II|PR)\,P(PR) \tag{A14}$$

where $P(II|PR) = \delta$. We will need to calculate the probability that a parent was susceptible (PS), infected (PI), or recovered (PR) for a randomly chosen offspring. The probability that an individual in habitat 1 was born to a susceptible (infected, recovered, respectively) parent is equal to the total number of offspring produced by susceptible (infected, recovered, respectively) individuals that choose habitat 1 divided by the total number of offspring choosing habitat 1. Then

$$P(PS) = \frac{\dfrac{b_S Q_1}{N_1^*} S_1^* + \dfrac{b_S Q_2}{N_2^*} S_2^*}{\dfrac{b_S Q_1}{N_1^*} S_1^* + \dfrac{b_S Q_2}{N_2^*} S_2^* + \dfrac{b_I Q_1}{N_1^*} I_1^* + \dfrac{b_I Q_2}{N_2^*} I_2^* + \dfrac{b_R Q_1}{N_1^*} R_1^* + \dfrac{b_R Q_2}{N_2^*} R_2^*} \tag{A15}$$

and the equations for $P(PI)$ and $P(PR)$ are similar. If the probability of choosing a habitat at birth is independent of infection, the probability of having a susceptible, infected, or recovered parent will be the same in both habitats.

Finally, we need to calculate the expected number of offspring given that an individual is born into each class. For an individual born susceptible, we must add the expected number of offspring produced while susceptible (susceptible birth rate times the expected amount of time spent susceptible), the probability of becoming infected times the expected number of offspring produced while infected (infected birth rate times expected time spent infected), and the probability of reaching the recovered stage times the expected amount of offspring produced while recovered (recovered birth rate times expected time spent recovered). For an individual born infected, we add the expected number of offspring produced while infected to the probability of reaching the recovered stage times the expected amount of offspring produced while recovered. For an individual born recovered, the expected number of offspring is simply the expected number of offspring produced while recovered:

$$\begin{aligned}
E(O_i|BS) &= \frac{b_S Q_i}{N_i^*}\frac{1}{\mu_S + \beta I_i^*} + \frac{b_I Q_i}{N_i^*}\frac{\beta I_i^*}{\mu_S + \beta I_i^*}\frac{1}{\mu_I + \gamma} + \frac{b_R Q_i}{N_i^*}\frac{\beta I_i^*}{\mu_S + \beta I_i^*}\frac{\gamma}{\mu_I + \gamma}\frac{1}{\mu_R}, \\
E(O_i|BI) &= \frac{b_I Q_i}{N_i^*}\frac{1}{\mu_I + \gamma} + \frac{b_R Q_i}{N_i^*}\frac{\gamma}{\mu_I + \gamma}\frac{1}{\mu_R}, \\
E(O_i|BR) &= \frac{b_R Q_i}{N_i^*}\frac{1}{\mu_R}.
\end{aligned} \tag{A16}$$