OIKOS

Research

Infection state can affect host migratory decisions

Naven Narayanan, Sandra Ann Binning and Allison K. Shaw

Naven Narayanan (https://orcid.org/0000-0002-3855-5656) ✉ (venka210@umn.edu) and A. K. Shaw, Ecology, Evolution and Behavior, Ecology Building, Univ. of Minnesota Twin Cities- E, 1987 Upper Buford Cir, Saint Paul, MN 55108, USA. – S. A. Binning, Dept de Sciences Biologiques, Univ. of Montreal, Montreal, QC, Canada.

Oikos
129: 1493–1503, 2020
doi: 10.1111/oik.07188

Subject Editor: Calvin Dytham
Editor-in-Chief: Dries Bonte
Accepted 3 June 2020

Organisms across a wide range of taxa use migration as a strategy to avoid, reduce or recover from parasitic infection. Previous work has identified three different processes by which migration can help reduce infection risk and/or costs: migratory escape from infection, migratory culling of infected individuals and migratory recovery from infection. However, most theoretical modelling of host migration in response to infection assumes that individuals have a single strategy during both infected and susceptible states, meaning an individual's state (susceptible or infected) is irrelevant to its decision to migrate. Here, we construct a model with two independent strategies of migration for an individual based on whether they are infected or susceptible, to study when the decision to migrate is favourable. We show that the best strategy for individuals of a given infection state is to either always migrate or never migrate. When infected and susceptible individuals differ in their migration strategy, this leads to partial migration at the population level (only some individuals migrate). Whereas previous theory showed that partial migration can be optimal in the face of infection, our work further parses out the contributors to partial or complete migration by determining which individuals in the population are involved in migration for different sets of conditions.

Keywords: evolutionarily stable strategy, host–parasite interaction, infection, migratory escape, migratory recovery, population dynamics

Introduction

Migration can be defined as the roundtrip movement of organisms across different habitats (Aidley 1981, Dingle 2014). This widespread phenomenon occurs across a vast range of taxa and scales, including miniscule copepods moving diurnally through the water column (Hays et al. 1995), monarch butterflies travelling across North America (Brower 1996), loggerhead sea turtles swimming the North Pacific ocean (Lohmann 2007) and arctic shorebirds flying annually across the Americas (Alerstam et al. 2001). Migratory behaviour is an adaptive response for survival in ephemeral and patchy habitats, is believed to have evolved in response to seasonal changes in resource availability and/or climate (Dingle 1980) and has numerous benefits including increased forage quality and quantity for migrants, increased mating opportunities and reduced predation (Avgar et al. 2014). Although increased exposure to novel parasites is often considered a cost of migratory behavior (Koprivnikar and Leung 2015), recent

NORDIC SOCIETY OIKOS

www.oikosjournal.org

theoretical and empirical research suggests that migration can also confer a number of benefits to migrants in terms of escape and/or recovery from infection (Folstad et al. 1991, Shaw and Binning 2016).

Migration can help prevent or reduce infection and its spread in populations via three non-mutually exclusive mechanisms. Firstly, via the process of migratory culling, parasite prevalence in a population is reduced as a result of increased mortality of infected individuals along the migratory route (Bradley and Altizer 2005, van Gils et al. 2007, Bartel et al. 2011). For instance, monarch butterflies *Danaus plexippus* infected with protozoa *Ophryocystis elektroscirrha* have reduced flight endurance compared to healthy conspecifics leading to reduced migratory success of infected individuals (Bradley and Altizer 2005). This effectively reduces the percentage of infected individuals in a population and, thus, reduces disease transmission within the group. Secondly, migratory escape suggests that migrating hosts benefit by physically separating themselves from contaminated habitats (environmental migratory escape) and/or infected conspecifics (social migratory escape) for some period of time (Folstad et al. 1991, Loehle 1995, Altizer et al. 2011, Shaw and Binning 2020). For example, reindeer *Rangifer tarandus tarandus* experience lower intensities of warble fly *Hypoderma tarandi* larvae infection the further they migrate from their calving grounds (Folstad et al. 1991). Thirdly, migratory recovery posits that migration across an environmental gradient promotes host recovery from infection as parasites die and fall off along the migration route as conditions become untenable for parasite survival (Shaw and Binning 2016, Daversa et al. 2018a, b, Shaw et al. 2019). For instance, spiny common toads *Bufo spinosus* recover from the fungal parasite *Batrachochytrium dendrobatidis* during their fall migration from ponds to land burrows as chytrid fungus is prone to desiccation during land exposure (Daversa et al. 2018a). This body of theoretical and empirical work has been instrumental in enhancing our understanding of migration-related dynamics in the context of disease spread and recovery, especially in helping to quantify the infection-related benefits gained by individuals and populations in terms of offsetting other costs of migration.

Although theoretical models can be used to elucidate general patterns and uncover counter-intuitive predictions that help guide future empirical research, they necessarily make simplifying assumptions, which may not accurately reflect ecological reality. Indeed, theoretical models exploring the infection-related benefits of migration have typically assumed that decisions to migrate or not are independent of an individual's infection status (susceptible or infected) (Hall et al. 2014, 2016, Shaw and Binning 2016, Shaw et al. 2019). In reality, infection can profoundly change both an individual's movement behaviours (decisions about when and where to move) and locomotor performance (ability to execute a movement behaviour), thereby influencing their likelihood to migrate (McElroy and de Buron 2014, Binning et al. 2017, Risely et al. 2018).

In some cases, parasites can increase the movement behaviours of their hosts by manipulating them to move to habitats more suitable for their own growth and/or reproduction (Curtis 1993, Lion et al. 2006, Binning et al. 2017). For example, when infected with the trematode, *Gynaecotyla adunca*, estuarine snails *Tritia obsoleta* (formerly *Ilyanassa obsolete*) make vertical migrations to the higher intertidal zone, where the parasite cercaria are more likely to encounter semi terrestrial crustaceans, the parasite's next host (Curtis 1993). Therapeutic behaviours such as behavioural chill or behavioural fever, whereby infected animals actively seek out cooler or warmer microhabitats in order to slow, prevent or recover from disease progression, also lead to diverging movement decisions depending on infection status (Covert and Reynolds 1977, Ouedraogo et al. 2004, Richards-Zawacki 2010, Mohammed et al. 2016, Arnold et al. 2019, Truitt et al. 2019). On the other hand, infected individuals may also move less than healthy conspecifics due to the physiological and mechanical damage caused by parasites, or to avoid extra energetic costs associated with movement (Main and Bull 2000, Mouritsen 2002, Debeffe et al. 2014, McElroy and de Buron 2014, Welicky and Sikkel 2015, van Dijk et al. 2015, Risely et al. 2018). French grunt *Haemulon flavolineatum* infected with the parasitic isopod *Anilocra haemuli* are almost three times less likely to embark on diel migrations off the coral reef than uninfected conspecifics (Welicky and Sikkel 2015). Indeed, parasitic infection can also induce sickness behaviours such as lethargy as an adaptive host immune response, which consequently reduce host movement (Hart 1988, Lopes 2014, 2017).

Healthy individuals may also alter their movement patterns as a strategy to avoid infection. For instance, susceptible individuals can engage in avoidance behaviours such as reducing contact with infected conspecifics (Kavaliers et al. 2004, Curtis 2014, Stephenson et al. 2018). For instance, the normally gregarious Caribbean spiny lobster *Panulirus argus* will actively avoid den sharing with conspecifics infected with a lethal virus (Behringer et al. 2006). These examples all suggest that infection state – whether an individual is susceptible or infected – could be important determinants of host movement strategy. Indeed, theoretical dispersal studies have shown higher rates of dispersal can evolve for either susceptible individuals (S-biased dispersal) or infected individuals (I-biased dispersal) based on the strength of virulence and rates of parasite release during dispersal (Iritani and Iwasa 2014). Thus, for some empirical systems, it appears ecologically relevant to model migration decisions separately based on whether an individual is infected or not.

Although there exist other drivers for migration including resource acquisition, breeding and competition, here, we use a theoretical approach by setting up a model and asking under what conditions migration evolves in response to infection. The goal of the model is to understand the evolution of migration in the context of infection by parasites and pathogens independent of other extrinsic biotic or environmental factors. We answer our question by determining the

evolutionarily stable strategy (ESS) of migration of organisms in a scenario where the probability of migration is dependent on the organism's infected state. We define infection in this study as one caused by parasites which have an obligatory impact on the fitness of an individual (although the degree of infection cost is variable). We take into account several costs associated with migration and infection and consider different rates of transmission and recovery of infection while defining a basal cost to residency in the first environment. We show that complete, partial or no migration may arise based on the relative costs of infection and migration and the rates of transmission and recovery of the infection. Thus our work adds to the literature (Chapman et al. 2011, Hegemann et al. 2019) another possible mechanism that favours partial migration. Whether susceptible and infected individuals have the same or different migration strategy depends on how important it is to escape or recover from infection as opposed to remaining a resident and avoiding migration costs. Furthermore we show that although partial migration could arise at the level of the population, the migration strategy at the level of individuals of a given infected state is binary i.e. either all the individuals of an infected state or none of them move and there is no partial migration strategy that arises.

Methods and results

Model setup

The model captures the phenomena of migratory escape and recovery (Fig. 1). See Table 1 for all parameters. In this model, the annual cycle is divided into two periods ($T_1 + T_2 = 1$) where individuals spend the first part (T_1) in an environment where there is a likelihood of transmission of the parasite to susceptible individuals (S) resulting in them becoming infected individuals (I). The change in number of infected and susceptible individuals is given by

$$\frac{dS}{dt} = -\beta S \tag{1a}$$

$$\frac{dI}{dt} = \beta S \tag{1b}$$

where β is defined as the transmission rate. There is no direct transmission of infection from infected to susceptible individuals (i.e. transmission is via the environment).

After T_1, the number of susceptible and infected individuals are given by

$$S(T_1) = S_0 e^{-\beta T_1} \tag{2a}$$

$$I(T_1) = I_0 + S_0\left(1 - e^{-\beta T_1}\right) \tag{2b}$$

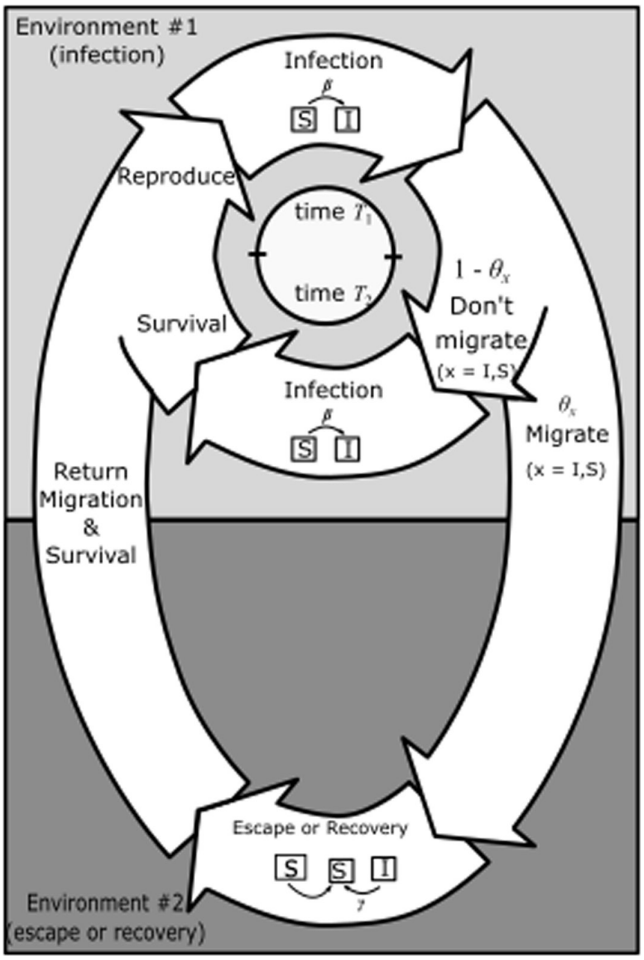

Figure 1. Model setup of the annual cycle. A fraction of individuals θ_x [where $x = I$ (infected), S (susceptible)] migrate after the first time period T_1 to environment 2 from environment 1 in order to recover (with some recovery rate γ) or escape infection. Infection in environment 1 occurs with a rate of transmission of β. The remaining fraction $(1-\theta_x)$ [where $x = I$, S] remain in environment 1 for the duration of the second time period T_2. Following this, reproduction occurs at the end of the year, after time $(T_1 + T_2)$ and it is assumed that all mortality arising due to costs of infection or migration occurs before reproduction.

where S_0 and I_0 are initial numbers of susceptible and infected individuals and $S_0 e^{-\beta T_1}$ is the fraction of individuals that remain uninfected after time period T_1 i.e. the first part of the year. T_1 is the length of the first time interval and does not convey any information on what happens within that interval.

The second part of the annual cycle (T_2) is either spent in another environment for migrating individuals or in the same environment (for resident individuals). Infected individuals that move benefit by recovering from infection at rate γ while susceptible individuals that move benefit by escaping from infection for the duration of the second time period i.e. T_2. The proportion of infected individuals that migrate is given by θ_I and the proportion of susceptible individuals that migrate by θ_S. We assume that residency, migration

Table 1. List of model parameters, meaning, units (if applicable) and values used in figures.

Symbol	Meaning (units)	Values
S	Number of susceptibles (individuals)	…
I	Number of infected (individuals)	…
b_{max}	Maximum total offspring born (individuals)	…
a_1	Density-independent fecundity coefficient	N/A
a_2	Density-dependent fecundity coefficient (individuals^{-1})	N/A
T_1	Time spent by individuals in breeding environment (years)	0.5
T_2	Time spent by individuals in non-breeding environment (years)	0.5
β	Rate of infection in breeding environment (years^{-1})	Varied
γ	Rate of recovery (parasite loss) in nonbreeding environment (years^{-1})	Varied
σ	Annual survival probability of susceptible residents	1.0
c_M	Survival cost of migration ($0 \le c_M \le 1$)	Varied
c_I	Survival cost of infection ($0 \le c_I \le 1$)	Varied
c_R	Survival cost of residency ($0 \le c_R \le 1$)	0.05
ϕ_S	Maximum per capita fecundity of susceptible individuals	2.0
ϕ_I	Per capita fecundity of infected individuals	1.6
θ_S	Probability that a susceptible individual migrates ($0 \le \theta_s \le 1$)	Evolved
θ_I	Probability that an infected individual migrates ($0 \le \theta_I \le 1$)	Evolved

and infection are costly and reduce survival. The fraction of susceptible non-migrants that survive is given by $\sigma(1-c_R)$, the baseline value of survival times the cost of residency. We assigned c_R a small value (0.05) in order to prevent it from becoming a confounding factor while determining the effect of infection state on migration strategy. The costs of migrating and being infected are given by c_M and c_I, respectively. Hence the fraction of susceptible migrants surviving is given by the product of $(1-c_M)$ and σ, and the fraction of infected non-migrants surviving by $(1-c_I)(1-c_R)\sigma$. We assume that infected migrants pay both costs and thus the fraction surviving is $(1-c_I)(1-c_M)\sigma$.

The number of non-migrating individuals which are susceptible (S_R) is given by

$$S_R = (1-\theta_S)\sigma(1-c_R)\left[S(T_1)e^{-\beta T_2}\right] \tag{3}$$

where the first (and only) term of S_R tells us the fraction of susceptible individuals that chose not to migrate $(1-\theta S_S)$ times the survival probability $\sigma(1-c_R)$ and fraction of individuals that did not get infected in the second time period of the year i.e. $S(T_1)e^{-\beta T_2}$.

The number of non-migrating infected individuals (I_R) at the end of T_2 is therefore

$$I_R = (1-\theta_I)\sigma(1-c_I)(1-c_R)[I(T_1)] + (1-\theta_S)\sigma(1-c_I)(1-c_R)\left[S(T_1)(1-e^{-\beta T_2})\right] \tag{4}$$

Here, the first term of the expression for I_R tells us the number of individuals that were already infected after T_1 that did not migrate and the second term informs us of the number of individuals that were susceptible after T_1 and chose not to migrate and as a result getting infected in the second time period of the year i.e. T_2.

The number of migrating individuals which are susceptible (S_M) after T_2 is given by

$$S_M = \theta_S\sigma(1-c_M)[S(T_1)] + \theta_I\sigma(1-c_M)I(T_1)\left(1-e^{-\gamma T_2}\right) \tag{5}$$

where the first term gives us the number of susceptible individuals after T_1 that migrated to the second environment and remained susceptible. The second term gives us the number of individuals that were infected after T_1 but migrated to the second environment and recovered from the infection.

The number of infected individuals that migrate (I_M) after T_2 is given by

$$I_M = \theta_I\sigma(1-c_I)(1-c_M)\left[I(T_1)e^{-\gamma T_2}\right] \tag{6}$$

The first (and only) term in the expression for I_M gives us the number of individuals that were infected at T_1, migrated to the second environment and remained infected even after T_2.

At the end of a year (i.e. after $T_1 + T_2$), all individuals reproduce. The number of offspring each susceptible or infected individual produces is denoted by ϕ_S and ϕ_I respectively. We assume that infection potentially reduces fecundity so $\phi_I \le \phi_S$. All offspring produced are susceptible individuals regardless of their parents' infected state as infection is not transmitted across generations. The maximum possible number of offspring is equal to

$$b_{max} = \phi_S(S_R + S_M) + \phi_I(I_R + I_M) \tag{7}$$

We assume that the number of offspring produced is density dependent. While deriving the analytical results of the model, the density-dependent function (Δ) was considered to be any function that was strictly decreasing as a function of population size and was equal to a maximal value of 1 when the size of the population was zero.

Analytical model

We combined the equations above into a matrix χ to link the number of susceptible and infected individuals at some time (τ) to the next time step ($\tau+1$) as given below. The scale of (τ) and ($\tau+1$) is in years and is different than the scale of T_1 and T_2 (fraction of years), i.e. $(\tau+1)=(\tau+T_1+T_2)$.

$$\begin{bmatrix} S \\ I \end{bmatrix}_{\tau+1} = \begin{bmatrix} (1+\Delta\phi_S)\left[(A(1-\theta_S)+B\theta_S+C\theta_I)\right] & (1+\Delta\phi_S)D\theta_I \\ +\Delta\phi_I\left(G\theta_I+E(1-\theta_I)+F(1-\theta_S)\right) & +\Delta\phi_I\left(J\theta_I+H(1-\theta_I)\right) \\ G\theta_I+E(1-\theta_I)+F(1-\theta_S) & J\theta_I+H(1-\theta_I) \end{bmatrix} \begin{bmatrix} S \\ I \end{bmatrix}_{\tau}$$

where

$$A=\sigma(1-c_R)e^{-\beta(T_1+T_2)}$$

$$B=\sigma(1-c_M)e^{-\beta T_1}$$

$$C=\sigma(1-c_M)(1-e^{-\beta T_1})(1-e^{-\gamma T_2})$$

$$D=\sigma(1-c_M)(1-e^{-\gamma T_2})$$

$$E=\sigma(1-c_I)(1-e^{-\beta T_1})$$

$$F=\sigma(1-c_I)(1-e^{-\beta T_2})e^{-\beta T_1}$$

$$G=\sigma(1-c_M)(1-c_I)(1-e^{-\beta T_1})e^{-\gamma T_2}$$

$$H=\sigma(1-c_I)$$

$$J=\sigma(1-c_M)(1-c_I)e^{-\gamma T_2}$$

We calculated the equilibria and their stability from the above equation by setting the left hand side equal to the right hand side to determine the equilibrium points. Once the equilibria were determined, each equilibrium point's stability was calculated by checking if the Jacobian of the χ matrix at the point fulfilled the stability criteria. The stability of the system was quantified using the Jury criteria. This allowed us to explore the range of parameters for which the system was stable or unstable at both equilibria.

The next step was to calculate the evolutionarily stable strategy (ESS) of migration in the population. We considered a stable population at the non-trivial equilibrium in which all the individuals had a single resident migration strategy ($\bar{\theta}_S, \bar{\theta}_I$). We checked if there existed a mutant strategy (θ_S', θ_I') that could invade the resident strategy. This approach yielded a function in two variables

$$f(\theta_S,\theta_I)=x\theta_S+y\theta_I+z\theta_S\theta_I \quad (8)$$

where x, y and z are real numbers which are comprised of coefficients previously defined in the matrix χ (see Supplementary material Appendix 1 for complete expression). We then maximized $f(\theta_S,\theta_I)$ over both θ_S and θ_I in order to determine the best strategies for given values of all the variables in the function.

We found that $f(\theta_S,\theta_I)$ was always maximized by one of four possible pairs of (θ_S,θ_I):(0,0),(0,1),(1,0) or (1,1) (Fig. 2). Thus the ESS migration strategy (for a given infection status) was always either complete migration or complete residency. This means that in a given scenario, all susceptible individuals either completely migrate i.e. $\theta_S=1$ or no one migrates i.e. $\theta_S=0$. Similarly, for a given case either all the infected individuals migrate i.e. $\theta_I=1$ or none of them migrate i.e. $\theta_I=0$ (Fig. 2). There is no ESS in this system wherein only some infected individuals or some susceptible individuals move in one annual cycle. Thus, partial migration (alternate

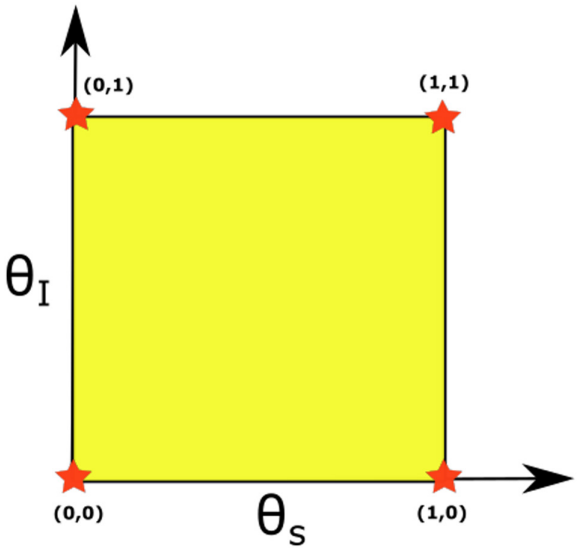

Figure 2. Graphical representation of entire space of potential migration strategies (θ_S, θ_I) for a population depicted by the yellow area in the square. $0 \le (\theta_S, \theta_I) \le 1$ where 0 implies no migration and 1 implies complete migration. The ESS migration strategy was always one of four possibilities (these possibilities are depicted by red stars along with their values) for any scenario depending on the values of x, y and z.

migratory strategies adopted by individuals of the same infection state) is never a stable strategy. The specific parameter values (and thus values of x, y and z) determined which of these four pairs was the ESS in each situation. Thus a quick version for determining the ESS is considering the four values of $f(x)$ at each potential ESS:

$$f(0,0) = 0$$

$$f(0,1) = y$$

$$f(1,0) = x$$

$$f(1,1) = x + y + z$$

and determining which (θ_S,θ_I) pair gives the maximal value of $f(\theta_S,\theta_I)$.

Each ordered pair is a different strategy which, biologically, are the different approaches to migration taken by the population of susceptible and infected individuals in order to escape or recover from the infection. The first strategy is where both susceptible and infected individuals do not migrate i.e. $\theta_S = \theta_I = 0$ (strategy 1). The second strategy is where susceptible individuals migrate but infected ones do not i.e. $\theta_S = 1$ and $\theta_I = 0$ (strategy 2). Biologically, this strategy represents movement in order to escape infection, but not migrating for recovery from it. The third strategy, on the other hand, is where susceptible individuals do not migrate but infected ones do i.e. $\theta_S = 1$ and $\theta_I = 0$ (strategy 3). Biologically, this means that migration is used only for the process of recovery and not escape. The final strategy is the one where both susceptible and infected individuals migrate i.e. $\theta_S = 1$ and $\theta_I = 1$ (strategy 4). Here, migration is utilized by individuals in the population in order to escape and/or recover from infection they may have encountered in the first environment in the first time period (T_1).

Simulations

The simulations carried out attempted to illustrate both graphically and biologically for the results derived in the analytical model. While ESS analysis provided the set of best strategies of migration as well as the conditions when each of them would arise, the sheer number of variables that existed made it complicated to interpret it in terms of what was happening biologically. Simulations were done in order to determine the best strategy for a broad spectrum of meaningful variables including cost of infection, cost of migration, rate of transmission and rate of recovery and obtain a biologically meaningful interpretation of it. While working on the analytical model we considered a very general model with a few biologically realistic constraints. However, in the simulations, an arbitrary density dependent function (Δ) in agreement with said constraints was chosen. This was of the form:

$$\Delta = a_1 e^{-a_2 b_{\max}} \tag{9}$$

where $b_{\max} = \phi_S(S_R + S_M) + \phi_I(I_R + I_M)$. This means that the total number of offspring born in the population in a year is

$$b = b_{\max} a_1 e^{-a_2 b_{\max}} \tag{10}$$

where a_1 and a_2 are density independent and density dependent coefficients of fecundity, respectively. These coefficients did not have any effect on the results either qualitatively or quantitatively.

We initially defined one of the strategies (1, 2, 3 or 4) as our resident strategy and allowed an initial population of arbitrary size to grow until it approached an equilibrium value (at around 100 years). At equilibrium, we introduced the other three possible strategies, one at a time and in separate scenarios, to observe if each mutant strategy was able to invade the resident strategy. This process was repeated for different values of β, γ, c_I and c_M. The transmission and recovery rates varied from 0 to 1 with step sizes of 0.05. Three regimes of cost of infection and migration were considered: low (0.2), medium (0.5) and high (0.8), for a total of nine possible combinations of c_I and c_M regimes. Over the entire course of simulations, the fecundity coefficients were kept constant $\phi_S = 2.0$ and $\phi_I = 1.6$, where infected individuals had lower fecundity due to the fecundity cost of infection we included. Further, we introduced a nominal cost to residency ($c_R = 0.05$) thereby preventing the susceptible, residents from surviving indefinitely.

We quantified the growth rate of the mutant as the leading eigenvalue of the χ matrix when it is introduced into a population at equilibrium where there is an existing resident strategy. The population was simulated to 200 years to attain this non-trivial equilibrium. If the dominant eigenvalues of all the mutant strategies while invading a resident strategy are < 1, then the resident strategy is defined as an ESS. Mutant strategies have growth rates > 1 are counted as strategies that can invade a resident strategy. This implies that we could hypothetically get more than one ESS in the system. We calculated this by analyzing pairwise invasive plots (PIPs) where we compared all pairs of possible ESS strategies in a pairwise fashion.

The simulation results built upon and lent further credence to the results obtained by analytical evaluations of our model. Each one of the entire set of four potential ESSs is observed in different regions of the parameter space (Fig. 3a–i). We considered different regimes of infection and migration costs (low, medium and high for each of the costs and all the resulting possible combinations).

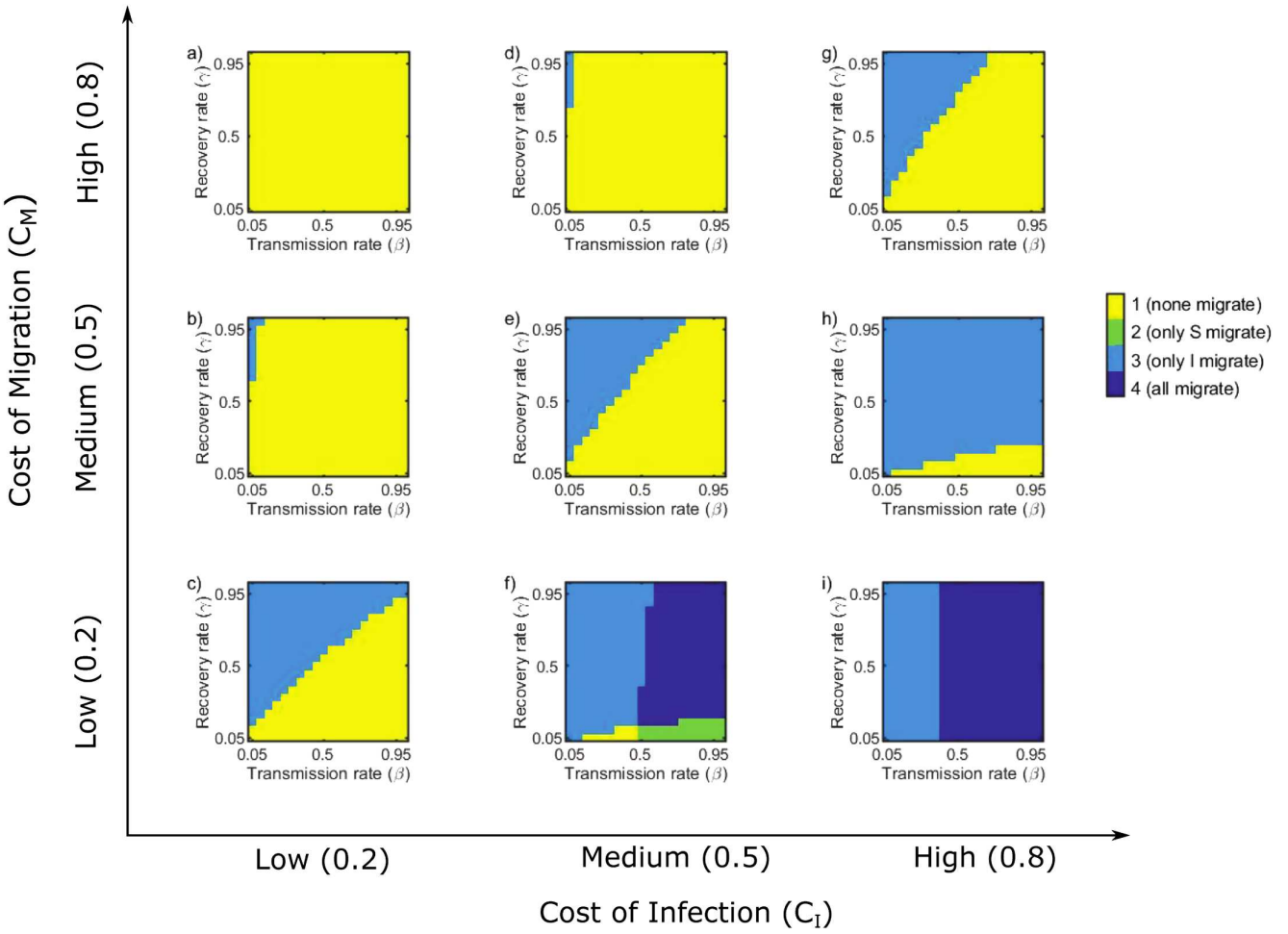

Figure 3. Migration strategy for population given varying combinations of rate of transmission of infection (β) and rate of recovery from infection (γ) with increasing levels of cost of infection (c_I) along rows of figure (from left to right) and increasing levels of cost of migration (c_M) along the columns (from bottom to top) with (a) low c_I (0.2) and high c_M (0.8), (b) low c_I (0.2) and medium c_M (0.5), (c) low c_I (0.2) and low c_M (0.2), (d) medium c_I (0.5) and high c_M (0.8), (e) medium c_I (0.5) and medium c_M (0.5), (f) medium c_I (0.5) and low c_M (0.2), (g) high c_I (0.8) and high c_M (0.8), (h) high c_I (0.8) and medium c_M (0.5) and (i) high c_I (0.8) and low c_M (0.2). In these scenarios we see different levels of complete, partial and no migration in the different cases.

For a given regime of infection and migration costs, the ESS depended on the rates of transmission and recovery. When migration and infection costs are low (Fig. 3c), migration of infected individuals only (i.e. strategy 3, or migratory recovery) is favoured for low infection rates (β) and a large range of recovery rates (γ). As β increases, strategy 3 is the ESS only when the recovery rate is extremely high, while strategy 1 (no one migrates) becomes the ESS when recovery rates are low. A similar pattern is observed with a medium migration cost (for all infection costs) (Fig. 3b, e, h) where no migration is favoured when β is high and γ is relatively lower. Strategy 3 arises in these scenarios when β is low and γ is relatively higher. Furthermore, this pattern is also observed in cases where the cost of migration is high, and when the cost of infection is either in the middle (Fig. 3d) or high (Fig. 3g) regime. However, when the infection cost is low and the migration cost is high, strategy 1 (no migration) is favoured regardless of the β and γ values (Fig. 3a).

Finally, the other two strategies (strategy 2 and 4) are only ever the ESS when migration cost is low and infection cost is medium (Fig. 3f). Here, only susceptible individuals are favoured to migrate (strategy 2, or migratory escape) when the infection rate is high and the recovery rate is low. When infection and recovery rates are both high, both infected and susceptible individuals migrate and hence both phenomena, migratory escape and recovery, are observed in these cases (strategy 4). No migration is only observed in the population when the recovery rate is low and infection rate is low or intermediate (strategy 1, Fig. 3f). For the rest of the parameter space (of β and γ) we observe only the migration of infected individuals (strategy 3). We observed that in the trivial case where there was no infection (β = 0), both strategies 1 and 3 were observed as equally good strategies for the population.

A result we find consistently across all our simulations is that when there is no infection transmission (β = 0) in the population, two strategies (1 and 3) invade equally well when

they are mutant strategies in the presence of the other as the resident strategy. In fact, their growth rate as invasive mutants is exactly the same as their growth rate when they are abundant in the population as the resident strategy. This suggests that they are both equally good ESS and cannot invade each other. This result arises due to the lack of infected individuals in the equilibrium population. In these cases, susceptible individuals should never migrate ($\theta_S = 1$). But since there are no infected individuals, both $\theta_I = 0$ (thus implying strategy 1) or $\theta_I = 1$ (thus implying strategy 3) would be an ESS.

Discussion

When does complete migration arise?

Complete migration (strategy 4) arose when the cost of infection was relatively higher ($c_I = 0.5$, 0.8) than the cost of migration ($c_M = 0.2$) and when both the transmission rate (β) (>0.5) and recovery rate (γ) were relatively large (>0 when c_I was 0.8 and >0.2 when c_I was 0.5) (Fig. 3f, i). This suggests that migratory escape and recovery can work in parallel such that the population as a whole avoids costly infections that are transmitted from the environment. Spiny common toads *Bufo spinus* migrate out of ponds, which act as reservoirs for chytrid fungus spores, to terrestrial burrows to hibernate. This seasonal migration not only allows susceptible toads to avoid contracting chytrid through water exposure, but also promotes recovery in infected individuals (Daversa et al. 2018a). However, migration, especially over long distances, may be challenging for infected individuals leading to differences in the timing or distance covered by infected versus healthy individuals rather than different migratory decisions themselves (Møller et al. 2004, Weber and Stilianakis 2007). Bewick's swans *Cygnus columbianus bewickii* infected with avian influenza migrate later and travel shorter distances than uninfected swans (van Gils et al. 2007). On the other hand, changes in the timing of migration due to infection also occurs in sea trout *Salmo trutta* infected with salmon lice *Lepeophtheirus salmonis*. When lice infection is high, sea trout spend less time in the ocean and migrate back to freshwater earlier than uninfected fish, or during years when infection prevalence is low (Birkeland and Jakobsen 1997, Gjelland et al. 2014, Halttunen et al. 2018). Future models could explore how early migratory return/migratory delay based on infection status affects population dynamics in fully migratory populations.

When does no migration arise?

No migration (strategy 1) occurs when the cost of migration is high ($c_M = 0.8$) and the cost of infection is low ($c_I = 0.2$) regardless of transmission (β) and recovery (γ) rates (Fig. 3a). This suggests that for low-virulence parasites, it is best for individuals to stay in their breeding habitat and risk getting infected rather than paying a high cost of migration ($c_I \ll c_M$).

Non-migratory species have evolved a variety of behavioural and/or physiological strategies for dealing with persistent parasite infection in their habitat (Wisenden et al. 2009, Binning et al. 2017, Hart and Hart 2018). This includes investment into immune defenses as well as parasite avoidance behaviours, cleaning interactions and therapeutic behaviours (e.g. behavioural fever) (Wisenden et al. 2009, Hart 2011, Hart and Hart 2018). For instance, frequent switching of sleeping sites is a common parasite avoidance strategy in mammals (Hausfater and Jean Meade 1982, Butler and Roper 1996, Reckardt and Kerth 2007). Investment into energetically-costly immune defenses may also be more possible for non-migratory organisms. For instance, migration causes immune suppression in thrushes (Owen and Moore 2006, 2008). Migration is an energetically costly, time consuming and risky behaviour that exposes individuals to new habitats, and potentially new risks of infection (Altizer et al. 2011). Therefore, in certain scenarios, the best choice for individuals is to remain at home and tolerate known parasite costs rather than to migrate away and risk incurring unforeseen costs along the way.

When do only susceptible individuals migrate?

Only one scenario led to migratory escape i.e. where only susceptible individuals migrated (strategy 2): when there was a medium ($c_I = 0.5$) to high ($c_I = 0.8$) cost of infection and where the cost of migration was low ($c_M = 0.2$) (Fig. 3f). This form of partial migration also only arose when the transmission rate (β) was significantly large (>0.5) and when the recovery rate was very small or absent . This suggests that migratory escape is only favoured when the benefit of migratory recovery is not large enough to balance out the cost of migration for infected individuals.

Given that parasites can impose significant performance costs on hosts (Bradley and Altizer 2005, McElroy and de Buron 2014, Binning et al. 2017), it is not difficult to imagine cases where infected individuals move less than susceptible ones. Australian sleepy lizards *Tiliqua rugosa* infected with ticks have smaller home ranges, move shorter distances in a day, and generally move less often than individuals which had their ticks experimentally removed (Main and Bull 2000). Similarly, dispersal distances of great tits *Parus major* leaving from nests infected with hen fleas *Ceratophyllus gallinae*, are shorter than those leaving from uninfected nests (Heeb et al. 1999). Some species of *Galaxiid* fishes in New Zealand migrate from freshwater streams to marine habitats to escape trematode infection: juvenile migrants returning back to streams are completely free of parasites, and migratory fishes in general have fewer parasites than resident species suggesting that migration may have evolved in some species as an escape from parasite infection (Poulin et al. 2012). Indeed, prophylactic movement behaviours, such as nest switching and avoidance of infected habitats that many hosts engage in to reduce their risk of infection (Perrot-Minnot and Cézilly 2009, Weinstein et al. 2018), could be a precursor to evolving

longer-distance migration behaviour as a means of parasite escape.

When do only infected individuals migrate?

We observe migration of only infected individuals (strategy 3) in all our simulations barring the scenario where cost of migration is high ($c_M = 0.8$) and cost of infection is low ($c_I = 0.2$) (Fig. 3b–i). We observe that infected individuals migrate for a vast range of transmission (β) and recovery (γ) rates ($0.05 \leq \beta \leq 1$, $0.05 \leq \gamma \leq 1$) based on the costs of migration and infection. We also observed that migration of infected individuals was favoured in scenarios with high transmission rates β only when recovery rates γ were concomitantly high. In other words, unless there was a high chance of recovery, infected individuals would rather suffer the costs of infection than risk migrating and not recovering.

Clear cases where migration occurs exclusively in infected hosts is lacking. Furthermore, examples where only infected individuals migrate in a population are believed to be behavioural manipulations used by the parasite to increase its own transmission rather than an adaptive host strategy (Curtis 1993). For example, estuarine snails *Tritia obsoleta* (formerly *Ilyanassa obsoleta*) will repeatedly migrate to intertidal habitats only when infected with the trematode *Gynaecotyla adunca*. This behaviour puts them into greater contact, and thus greater risk of being consumed by, the trematode's second intermediate crustaceans hosts (Curtis 1993). Regardless, a growing body of evidence suggests that increased movement behaviour to enhance recovery in infected hosts does, indeed, occur (Shaw and Binning 2016). Small-scale changes in the activity patterns and microhabitat preference of infected individuals such as behavioural chill or behavioural fever, frequently occur as a therapeutic response to infection (Moore 2013, Rakus et al. 2017). Migratory locusts *Locusta migratoria migratoriodes* will thermoregulate through the selection of high-temperature microhabitats when infected with the pathogenic fungus *Metarhizium anisopliae*, which increases their survival (Ouedraogo et al. 2004). Similarly, Trinidadian guppies *Poecilia reticulata* infected with ectoparasitic *Gyrodactylus turnbulli* preferentially select warmer habitats compared to when uninfected, allowing them to recover from infection (Mohammed et al. 2016). Differential movement decisions between infected and susceptible hosts could serve as a precursor to evolving infection-based migratory behaviour. This, in turn, could alter patterns of partial migration seen in populations. This calls for further investigation into infection driven movement preference in the real world.

Future directions and model caveats

Our model can be adapted to better capture the dynamics of specific empirical systems. For instance, a potential increase in the degree of resolution of migration strategies can be obtained by constructing a model where there is individual level variation in terms of costs suffered due to infection and migration instead of broadly classifying individuals as susceptible or infected. This is akin to taking infection intensity into account in terms of migration decisions. For instance, in sea trout, migratory decisions appear to be related to the intensity of sea lice infection (Halttunen et al. 2018). Also, the assumptions made regarding parasite transmission type (assumed in our study to be indirect transmission from the environment to individuals in the breeding environment exclusively) could be modified, which might lead to further unintuitive results regarding migration patterns of organisms. Indeed, transmission type does affect the scenarios under which partial migration is likely to evolve (Shaw et al. 2019). Consideration of directly transmitted infections which follow density or frequency-dependent transmission dynamics is a potentially worthwhile area of future investigation. Next, we have sidestepped complexities that arise due to vertical transmission of infection from parents to offspring. This implies that in our model, we assume all newborn individuals are susceptible at birth. In reality, vertical transmission from parents to offspring is very common in animals (Fine 1975), and our model could be adapted accordingly. There exist migrations that do not follow the annual breeding/non breeding cycle we consider in our model and perform these round trips at different timescales. Diel vertical migrations (DVMs), for instance, would of course not have reproduction every cycle (as defined in our model). In such a scenario, there require to be slight modifications to our model. For instance, modifying the model to allow for reproduction only after a fixed number of migratory cycles (i.e. days in this case) could serve as a more accurate model of infection transmission and migration for a different timescale. Corresponding modifications can be made to the parameter space that could more realistically depict costs of infection, costs of movement etc. for the particular type of migration. Since the model scales all our costs to values between 0 and 1 (0 implying no cost and 1 implying maximal cost leading to death of individuals in a population) the entire gamut of costs for different types of migrations can be incorporated. We believe that the major results regarding migration (partial, complete, no migration) will still be observed. However, the parameter space (c_I, c_M, β, and γ) where we observe them will be different when the model is modified to study different types of migration at different timescales. This is a potential avenue for further exploration as it can help confirm whether migration strategies remain the same across spatio-temporal scales. Finally, we do not consider infections for which individuals can recover and gain immunity (i.e. SIR model). In this scenario, we might expect reduced migration to escape infection as there is a non-zero probability of a subset of individuals gaining immunity that protects them from getting infected and allows them to escape the cost of migration. This scenario should be explored further.

Finally, one caveat of our model is that we do not model the phenomenon of migratory culling. We did not include it for two reasons. Firstly, in our model, we count the number of individuals in each class (susceptible and resident, susceptible

and migratory, infected and migratory, infected and resident) discretely (i.e. only at the end of T_1 time period and T_2 time period once they move back to the first environment). Migratory culling, as has been described in monarch butterflies, involves infected individuals dying during the migration journey (Bradley and Altizer 2005, Altizer et al. 2011). This implies differences in the number of individuals that leave the first environment and then reach the second environment. Our method of counting does not allow for this degree of resolution as we only look at the number of individuals in each class at the end of every year. Secondly, migratory culling causes increased mortality of infected individuals during migration (Altizer et al. 2011). In our model, we would expect this to lead to migration evolving as a strategy only when the cost of infection is extremely high ($c_I \gg c_M$). This would lead to a difference in degree and not kind of strategy adopted by the individuals and hence we expect the qualitative aspects to remain the same.

Our results add to the existing set of hypotheses explaining how partial migration arises in a population. Partial migration has previously been attributed to intraspecific and intrasexual competition (Ketterson and Nolan 1976, Gauthreaux 1982), limited foraging opportunities (Jahn et al. 2010), intermittent breeding to escape migration costs (Shaw and Levin 2011) and sexual conflict (Grayson et al. 2011). Our study suggests that infection could also drive partial migration. Although other theoretical and empirical studies have documented partial migration in response to infection (Shaw and Binning 2016, Hegemann et al. 2019, Shaw et al. 2019), they do not consider infection status in individual decisions to migrate. Thus, our results build on this growing body of literature and suggest that partial migration can arise as an intermediate strategy with individuals changing their migration tendency as they pass between infection classes.

Acknowledgements – We thank members of the Shaw lab and Marlene Zuk, and the Subject Editor for helpful comments on the manuscript.
Funding – NV is supported in part by Startup Funds from the University of Minnesota (to AKS), SAB is supported by the Natural Sciences and Engineering Research Council of Canada (NSERC) and the Canada Research Chair program, and AKS is supported in part by the National Science Foundation under grant no. DEB-1654609 (to AKS and SAB).

References

Aidley, D. J. 1981. Animal migration. – CUP Archive.
Alerstam, T. et al. 2001. Migration along orthodromic sun compass routes by arctic birds. – Science 291: 300–303.
Altizer, S. et al. 2011. Animal migration and infectious disease risk. – Science 331: 296–302.
Arnold, P. A. et al. 2019. *Drosophila melanogaster* infected with wolbachia strain WMelCS prefer cooler temperatures. – Ecol. Entomol. 44: 287–290.
Avgar, T. et al. 2014. On the adaptive benefits of mammal migration. – Can. J. Zool. 92: 481–490.
Bartel, R. A. et al. 2011. Monarch butterfly migration and parasite transmission in eastern North America. – Ecology 92: 342–351.
Behringer, D. C. et al. 2006. Avoidance of disease by social lobsters. – Nature 441: 421–421.
Binning, S. A. et al. 2017. Parasites and host performance: incorporating infection into our understanding of animal movement. – Integr. Comp. Biol. 57: 267–280.
Birkeland, K. and Jakobsen, P. J. 1997. Salmon lice, lepeophtheirus salmonis, infestation as a causal agent of premature return to rivers and estuaries by sea trout, *Salmo trutta*, juveniles. – Environ. Biol. Fish. 49: 129–137.
Bradley, C. A. and Altizer, S. 2005. Parasites hinder monarch butterfly flight: implications for disease spread in migratory hosts: parasites and monarch flight. – Ecol. Lett. 8: 290–300.
Brower, L. P. 1996. Monarch butterfly orientation: missing pieces of a magnificent puzzle. – J. Exp. Biol. 199: 93–103.
Butler, J. M. and Roper, T. J. 1996. Ectoparasites and sett use in european badgers. – Anim. Behav. 52: 621–629.
Chapman, B. B. et al. 2011. The ecology and evolution of partial migration. – Oikos 120: 1764–1775.
Covert, J. B. and Reynolds, W. W. 1977. Survival value of fever in fish. – Nature 267: 43–45.
Curtis, L. A. 1993. Parasite transmission in the intertidal zone: vertical migrations, infective stages and snail trails. – J. Exp. Mar. Biol. Ecol. 173: 197–209.
Curtis, V. A. 2014. Infection-avoidance behaviour in humans and other animals. – Trends Immunol. 35: 457–464.
Daversa, D. R. et al. 2018a. Routine habitat switching alters the likelihood and persistence of infection with a pathogenic parasite. – Funct. Ecol. 32: 1262–1270.
Daversa, D. R. et al. 2018b. Seasonal migrations, body temperature fluctuations and infection dynamics in adult amphibians. – PeerJ 6: e4698.
Debeffe, L. et al. 2014. Parasite abundance contributes to condition-dependent dispersal in a wild population of large herbivore. – Oikos 123: 1121–1125.
Dingle, H. 1980. 1 – Ecology and evolution of migration. – In: Gauthreaux, S. A. (ed.), Animal migration, orientation and navigation. Academic Press, pp. 1–101.
Dingle, H. 2014. Migration: the biology of life on the move. – Oxford Univ. Press, <www.oxfordscholarship.com/view/10.1093/acprof:oso/9780199640386.001.0001/acprof-9780199640386>.
Fine, P. E. M. 1975. Vectors and vertical transmission: an epidemiologic perspective. – Ann. N. Y. Acad. Sci. 266: 173–194.
Folstad, I. et al. 1991. Parasite avoidance: the cause of post-calving migrations in rangifer? – Can. J. Zool. 69: 2423–2429.
Gauthreaux, S. A. 1982. The ecology and evolution of avian migration systems, – <www.sciencedirect.com/science/article/pii/B9780122494062500113>.
Gjelland, K. et al. 2014. Effects of salmon lice infection on the behaviour of sea trout in the marine phase. – Aquacult. Environ. Interact. 5: 221–233.
Grayson, K. L. et al. 2011. Life history benefits of residency in a partially migrating pond-breeding amphibian. – Ecology 92: 1236–1246.
Hall, R. J. et al. 2014. Greater migratory propensity in hosts lowers pathogen transmission and impacts. – J. Anim. Ecol. 83: 1068–1077.
Hall, R. J. et al. 2016. Modeling vector-borne disease risk in migratory animals under climate change. – Integr. Comp. Biol. 56: 353–364.

Halttunen, E. et al. 2018. Sea trout adapt their migratory behaviour in response to high salmon lice concentrations. – J. Fish Dis. 41: 953–967.
Hart, B. L. 1988. Biological basis of the behavior of sick animals. – Neurosci. Biobehav. Rev. 12: 123–137.
Hart, B. L. 2011. Behavioural defences in animals against pathogens and parasites: parallels with the pillars of medicine in humans. – Phil. Trans. R. Soc. B 366: 3406–3417.
Hart, B. L. and Hart, L. A. 2018. How mammals stay healthy in nature: the evolution of behaviours to avoid parasites and pathogens. – Phil. Trans. R. Soc. B 373: 20170205.
Hausfater, G. and Jean Meade, B. 1982. Alternation of sleeping groves by yellow baboons (*Papio cynocephalus*) as a strategy for parasite avoidance. – Primates 23: 287–297.
Hays, G. C. et al. 1995. Spatio–temporal patterns in the diel vertical migration of the copepod metridia lucens in the northeast atlantic derived from the continuous plankton recorder survey. – Limnol. Oceanogr. 40: 469–475.
Heeb, P. et al. 1999. Ectoparasite infestation and sex-biased local recruitment of hosts. – Nature 400: 63–65.
Hegemann, A. et al. 2019. A physiological perspective on the ecology and evolution of partial migration. – J. Ornithol. 160: 893–905.
Iritani, R. and Iwasa, Y. 2014. Parasite infection drives the evolution of state-dependent dispersal of the host. – Theor. Popul. Biol. 92: 1–13.
Jahn, A. E. et al. 2010. Determinants of partial bird migration in the amazon basin. – J. Anim. Ecol. 79: 983–992.
Kavaliers, M. et al. 2004. Olfactory-mediated parasite recognition and avoidance: linking genes to behavior. – Horm. Behav. Olfact. Sex Behav. 46: 272–283.
Ketterson, E. D. and Nolan, V. 1976. Geographic variation and its climatic correlates in the sex ratio of eastern-wintering dark-eyed juncos (*Junco hyemalis hyemalis*). – Ecology 57: 679–693.
Koprivnikar, J. and Leung, T. L. F. 2015. Flying with diverse passengers: greater richness of parasitic nematodes in migratory birds. – Oikos 124: 399–405.
Lion, S. et al. 2006. The evolution of parasite manipulation of host dispersal. – Proc. R. Soc. B 273: 1063–1071.
Loehle, C. 1995. Social barriers to pathogen transmission in wild animal populations. – Ecology 76: 326–335.
Lohmann, K. J. 2007. Sea turtles: navigating with magnetism. – Curr. Biol. 17: R102–R104.
Lopes, P. C. 2014. When is it socially acceptable to feel sick? – Proc. R. Soc. B 281: 20140218.
Lopes, P. C. 2017. Why are behavioral and immune traits linked? – Horm. Behav. 88: 52–59.
Main, A. R. and Bull, C. M. 2000. The impact of tick parasites on the behaviour of the lizard *Tiliqua rugosa*. – Oecologia 122: 574–581.
McElroy, E. J. and de Buron, I. 2014. Host performance as a target of manipulation by parasites: a meta-analysis. – J. Parasitol. 100: 399–410.
Mohammed, R. S. et al. 2016. Getting into hot water: sick guppies frequent warmer thermal conditions. – Oecologia 181: 911–917.
Møller, A. P. et al. 2004. Parasitism, immunity, and arrival date in a migratory bird, the barn swallow. – Ecology 85: 206–219.
Moore, J. 2013. An overview of parasite-induced behavioral alterations – and some lessons from bats. – J. Exp. Biol. 216: 11–17.
Mouritsen, K. N. 2002. The parasite-induced surfacing behaviour in the cockle austrovenus stutchburyi: a test of an alternative hypothesis and identification of potential mechanisms. – Parasitology 124: 521–528.
Ouedraogo, R. M. et al. 2004. Behavioral thermoregulation in the migratory locust: a therapy to overcome fungal infection. – Oecologia 138: 312–319.
Owen, J. C. and Moore, F. R. 2008. Swainson's thrushes in migratory disposition exhibit reduced immune function. – J. Ethol. 26: 383–388.
Perrot-Minnot, M. J. and Cézilly, F. 2009. Parasites and behaviour, pp. 49–68. – In: Thomas, F. et al. (eds), Ecology and evolution of parasitism. Oxford Univ. Press.
Poulin, R. et al. 2012. Migration as an escape from parasitism in new zealand galaxiid fishes. – Oecologia 169: 955–963.
Rakus, K. et al. 2017. Behavioral fever in ectothermic vertebrates. – Dev. Comp. Immunol. 66: 84–91.
Reckardt, K. and Kerth, G. 2007. Roost selection and roost switching of female Bechstein's bats (*Myotis bechsteinii*) as a strategy of parasite avoidance. – Oecologia 154: 581–588.
Richards-Zawacki, C. L. 2010. Thermoregulatory behaviour affects prevalence of chytrid fungal infection in a wild population of panamanian golden frogs. – Proc. R. Soc. B 277: 519–528.
Risely, A. et al. 2018. Migratory animals feel the cost of getting sick: a meta-analysis across species. – J. Anim. Ecol. 87: 301–14.
Shaw, A. K. and Binning, S. A. 2016. Migratory recovery from infection as a selective pressure for the evolution of migration. – Am. Nat. 187: 491–501.
Shaw, A. K. and Binning, S. A. 2020. Recovery from infection is more likely to favour the evolution of migration than social escape from infection. – J. Anim. Ecol. 89: 1448–1457.
Shaw, A. K. and Levin, S. A. 2011. To breed or not to breed: a model of partial migration. – Oikos 120: 1871–1879.
Shaw, A. K. et al. 2019. Host migration strategy is shaped by forms of parasite transmission and infection cost. – J. Anim. Ecol. 88: 1601–1612.
Stephenson, J. F. et al. 2018. Transmission risk predicts avoidance of infected conspecifics in trinidadian guppies. – J. Anim. Ecol. 87: 1525–1533.
Truitt, A. M. et al. 2019. Wolbachia modifies thermal preference in drosophila melanogaster. – Environ. Microbiol. 21: 3259–3268.
van Dijk, J. G. B. et al. 2015. Weak negative associations between avian influenza virus infection and movement behaviour in a key host species, the mallard *Anas platyrhynchos*. – Oikos 124: 1293–1303.
van Gils, J. A. et al. 2007. Hampered foraging and migratory performance in swans infected with low-pathogenic avian influenza a virus. – PLoS One 2: e184.
Weber, T. P. and Stilianakis, N. I. 2007. Ecologic immunology of avian influenza (H5N1) in migratory birds. – Emerg. Infect. Dis. 13: 1139–1143.
Weinstein, S. B. et al. 2018. Fear of Feces? Tradeoffs between disease risk and foraging drive animal activity around raccoon latrines. – Oikos 127: 927–934.
Welicky, R. L. and Sikkel, P. C. 2015. Decreased movement related to parasite infection in a diel migratory coral reef fish. – Behav. Ecol. Sociobiol. 69: 1437–1446.
Wisenden, B. et al. 2009. Behavioral defenses against parasites and pathogens. - In: Zaccone, G. (ed.), Fish defences Vol. 2. Pathogens, parasites and predators. Science Publishers, pp. 151–168.

Supplementary material (available online as Appendix oik-07188 at <www.oikosjournal.org/appendix/oik-07188>).
Appendix 1