Migratory Recovery from Infection as a Selective Pressure for the Evolution of Migration

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abstract: Migration, a widespread animal behavior, can influence how individuals acquire and transmit pathogens. Past work has demonstrated that migration can reduce the costs of pathogen or parasite infection through two processes: migratory escape from infected areas or individuals and migratory culling of infected individuals. Here, we propose a third process: migratory recovery, where infected individuals lose their parasites and recover from infection during migration. Recovery can occur when parasites and/or their intermediate hosts cannot support changes in the migratory host's internal or external environment during migration. Thus, parasite mortality increases with migration. Although migratory recovery is likely widespread across species, it remains challenging to empirically test it as a selective force promoting migration. We develop a model and determine the conditions under which migratory recovery theoretically favors the evolution of migration. We show that incorporating migratory recovery into a model of migratory escape increases the range of biologically realistic conditions favoring migration and leads to scenarios where partial migration can evolve. Motivated by empirical estimates of infection costs, our model shows how recovery from infection could drive the evolution of migration. We suggest a number of future directions for both theoretical and empirical research in this area.

Keywords: host-parasite interaction, population dynamics, environmental gradient, evolutionarily stable strategy, partial migration, pathogen infection.

Introduction

Migration, the seasonal predictable movement of organisms across multiple locations, is one of several adaptive behaviors for maximizing fitness in spatially and temporally variable environments (Dingle 1980). Benefits of migration include escaping seasonally unfavorable climate or predators, facilitating the location of mates and breeding sites, and tracking changing food distributions (Northcote 1978; Avgar et al. 2014). Although less often considered, migration may also lower the risk of infection by pathogens and parasites (Altizer et al. 2011; Poulin et al. 2012). However, ecological factors covary, making it difficult to identify which ones contribute most to favoring migration (Poulin et al. 2012; Avgar et al. 2014). It is also extremely challenging to experimentally determine how infection risk might influence a host's migratory strategy. However, theoretical studies can more easily compare systems with different infection risks (controlling for other factors), offering powerful insights into the evolution of migration (Altizer et al. 2011). Two mechanisms have been proposed by which migration can reduce the risk of infection by parasites: migratory escape and migratory culling.

Hosts can enjoy benefits from seasonal migration by escaping parasites in time or space. This strategy can be particularly effective if individuals congregate for breeding or other activities (Loehle 1995; Altizer et al. 2011). A variant on migratory escape occurs when migration leads to the spatial separation of susceptible juveniles from infected adults, known as migratory allopatry (Krkošek et al. 2007). Escape from parasites has been suggested as a benefit of migration in mammals (Folstad et al. 1991; Qviller et al. 2013), birds (Zuk 1991; Piersma 1997), and fish (Poulin et al. 2012). Recent theoretical work also provides support for migratory escape as an added benefit of migration in systems where seasonal climate drives migration (Hall et al. 2014).

Alternatively, sick or parasitized individuals may simply not be able to survive the migratory journey. If infected individuals suffer higher mortality during migration, this can reduce the infection risk for noninfected individuals when parasites are transmitted between hosts. Migratory culling has been described as a likely benefit for migratory insects (Bartel et al. 2011). Parasites can also slow movement abilities of fish (Wagner et al. 2003; Binning et al. 2013), reptiles (Oppliger et al. 1996), and frogs (Goodman and Johnson 2011), suggesting that migratory culling could be a benefit in these taxonomic groups as well. Theoretically, migratory culling can reduce disease prevalence (Johns and Shaw 2016). Therefore, migratory culling could poten-

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tially favor the evolution of migration in the absence of any other migratory benefits.

Here, we propose that migratory recovery provides a third mechanism that enables hosts to reduce infection-related costs. Many migratory species move between different habitat types or across strong environmental gradients (e.g., temperature, humidity, salinity, altitude, dissolved oxygen). Parasites are often picked up and lost at different rates in these habitats (Dartnall 1972; Moyer et al. 2002; Li et al. 2011). For example, in the pet and aquaculture industries, fish and crustaceans are commonly dipped in water of different salinities to remove ectoparasites or reduce skin infections (Landsberg et al. 1991; Speare et al. 1996; Parsons et al. 2001). Similarly, some ectotherms combat pathogen infection by seeking out warm microclimates, a phenomenon known as behavioral fever (Kluger et al. 1975; Covert and Reynolds 1977). Likewise, sunning behavior in birds can dramatically increase feather surface temperature and has been attributed to ectoparasite control (Moyer and Wagenbach 1995). However, parasite loss via changes in the host's external or internal environment has not yet been considered explicitly as a potential infection-related benefit of migration (but see Gjelland et al. 2014).

We develop a mathematical model to explore migratory recovery as a potential benefit of migration. We assume that migration incurs a survival cost and that the primary benefit of migration is that infected individuals can recover, becoming uninfected, while nonmigrants cannot. We derive the evolutionarily stable probability of migration and show that there are biologically realistic conditions under which the best strategy is (*a*) to always migrate, (*b*) to never migrate, or (*c*) for part of the population to migrate ("partial migration"; Chapman et al. 2011). We show that even if migration decreases the survival of infected and uninfected individuals compared with not migrating, it can still be favored as long as migrating individuals are sufficiently likely to recover from infection. Overall, our results suggest that migratory recovery is a third infection-related benefit of migration that should be considered alongside migratory escape and migratory culling.

Model

We assume the hosts' annual cycle consists of two time periods, of length T_1 and T_2 respectively, where $T_1 + T_2 = 1$ (see fig. 1 and table 1 for model parameters). The first period is spent in one environment (where individuals reproduce), and the second period is spent either in a second environment (for migrating individuals) or also in the first environment (for nonmigrating individuals).

While in the first (breeding) environment, parasite-free (susceptible) individuals (S) become infected (I) at a constant rate β, such that the infection dynamics are given by

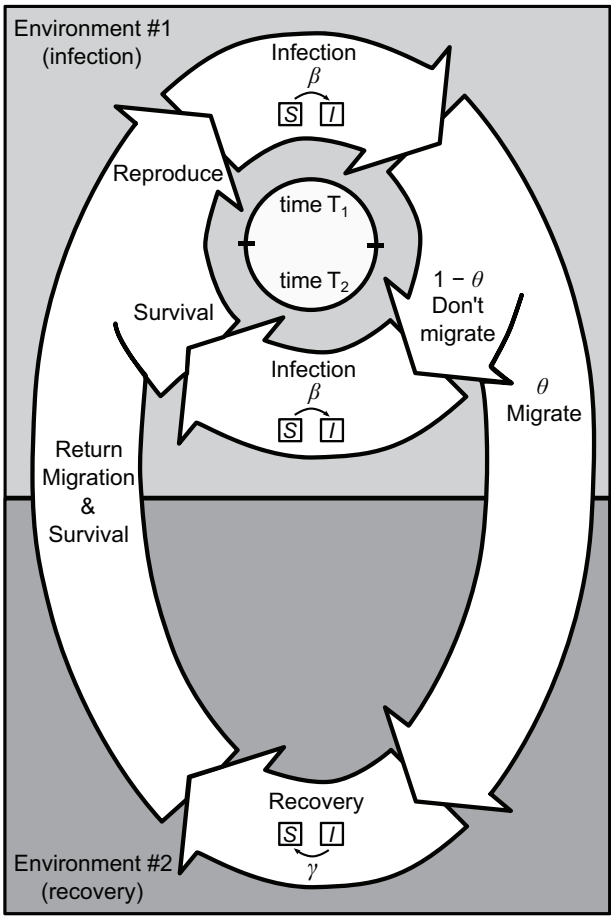

Figure 1: Model schematic of the annual cycle. A fraction $1 - \theta$ of individuals do not migrate, staying in environment 1 year-round, and are infected at a constant rate β throughout the entire year. The other θ individuals leave, migrating to environment 2, and both escape further infection and recover at rate γ. We assume that reproduction happens at the end of the year and that all infection- and migration-induced mortality occurs right before reproduction.

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

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

We assume that parasites are abundant and transmitted to hosts from the environment (βS) instead of directly from other parasitized individuals (βSI). The number of susceptible and infected individuals at the end of the first period is

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

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

Table 1: Model parameters, meaning, units (if applicable), and default values used in figures 2 and 3

Symbol	Meaning (units)	Default value
S	Number of susceptibles (individual)	...
I	Number of infecteds (individual)	...
b_{max}	Maximum total number of offspring born (individual)	...
a_1	Density-independent fecundity coefficient	NA
a_2	Density-dependent fecundity coefficient (individual^{-1})	NA
T_1	Time migrants spend in breeding environment (years)	.5
T_2	Time migrants spend in nonbreeding environment (years)	.5
β	Rate of infection in breeding environment (year^{-1})	Varied
γ	Rate of recovery (parasite loss) in nonbreeding environment (year^{-1})	Varied
σ	Annual survival probability of susceptible residents	Varied
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_F	Fecundity cost of infection ($0 \le c_F \le 1$)	Varied
ϕ	Maximum per capita fecundity of susceptibles (individual)	2
θ	Probability that an individual migrates ($0 \le \theta \le 1$)	Evolved

Note: NA = not applicable.

where T_1 is the length of the first time period and S_0 and I_0 are the initial number of susceptible and infected individuals. The second term of the infected host equation (eq. [2b]), $S_0(1 - e^{-\beta T_1})$, represents new infections (i.e., those hosts not remaining susceptible [eq. (2a)] leave that class exponentially).

Next, a fraction θ of the population migrates away, while the rest $(1 - \theta)$ remain. The second time period passes, and the migrants return. The number of nonmigratory individuals (residents) at the end of the second period is

$$S_R = (1 - \theta)\sigma[S(T_1)e^{-\beta T_2}], \tag{3a}$$

$$I_R = (1 - \theta)(1 - c_I)\sigma[I(T_1) + S(T_1)(1 - e^{-\beta T_2})], \tag{3b}$$

where T_2 is the length of the second period, σ is the survival probability of susceptible residents, and c_I is the survival cost of infection. Here, the number of susceptible residents (S_R) at the end of the year is the survival (σ) of the nonmigrating $(1 - \theta)$ hosts at the end of the first time period ($S(T_1)$) times the probability that they remain uninfected during the second period ($e^{-\beta T_2}$). Similarly, the number of infected residents (I_R) at the end of the year is the survival ($(1 - c_I)\sigma$) of the nonmigrating $(1 - \theta)$ infected hosts at the end of the first time period ($I(T_1)$) plus the number of susceptible hosts that became infected during the second period ($S(T_1)(1 - e^{-\beta T_2})$). We assume that parasites can be transmitted only in the breeding environment (i.e., no new infections occur during migration). We also assume that infected individuals "recover" at a constant rate γ as their parasites fall off, are expelled, and/or die at some point during their migration route. However, migration itself is costly. The number of migratory individuals at the end of the second period is then

$$S_M = \theta(1 - c_M)\sigma[S(T_1) + I(T_1)(1 - e^{-\gamma T_2})], \tag{4a}$$

$$I_M = \theta(1 - c_M)(1 - c_I)\sigma[I(T_1)e^{-\gamma T_2}], \tag{4b}$$

where c_M is the survival cost of migration. Here, the number of susceptible migrants (S_M) at the end of the year is the survival ($(1 - c_M)\sigma$) of the susceptible individuals that migrated ($\theta S(T_1)$) plus the infected individuals that migrated ($\theta I(T_1)$) and recovered from infection ($1 - e^{-\gamma T_2}$). Similarly, the number of infected migrants (I_M) is the number of initially infected individuals ($I(T_1)$) that migrated (θ), remained infected during the second period ($e^{-\gamma T_2}$), and survived despite both migration- and infection-induced survival costs ($(1 - c_M)(1 - c_I)\sigma$). Here, we assume that these costs are independent, but see the appendix (available online).

Finally, reproduction occurs at the end of the year when individuals return to the breeding habitat. The maximum total number of offspring born in the population is

$$b_{max} = \phi(S_R + S_M) + \phi(1 - c_F)(I_R + I_M), \tag{5a}$$

where ϕ is the maximum per capita fecundity of each susceptible individual and $\phi(1 - c_F)$ is the maximum per capita fecundity of each infected individual (c_F is the fecundity cost of infection). The actual number of offspring born is

$$b = a_1 b_{max} e^{-a_2 b_{max}}, \tag{5b}$$

where a_1 and a_2 are the density-independent and density-dependent fecundity coefficients, respectively (Ricker 1975). We assume that transmission of pathogens is horizontal (i.e., that all newborn individuals are susceptible). Our results are not influenced by the specific values of a_1 and a_2 (they drop out during analysis) or even by the form of den-

sity dependence used as long as the nontrivial equilibrium is stable (i.e., the population does not display stable oscillations or chaotic behavior; see the appendix).

We can combine equations (2)–(5) to get the full population model,

$$S(T_1 + T_2) = S_R + S_M + b, \tag{6a}$$

$$I(T_1 + T_2) = I_R + I_M, \tag{6b}$$

which allows us to relate the number of susceptible and infected individuals at the start of next year to the number of each at the start of this year.

Next, we determine the evolutionarily stable probability of migrating (see the appendix). To do this, we first find the model equilibria and stability. Then we consider a population at the nontrivial equilibrium where all individuals use the resident strategy $\bar{\theta}$ and introduce a mutant individual with strategy θ'. Finally, we determine what values of $\bar{\theta}$ are an evolutionarily stable strategy (ESS), that is, they prevent a mutant with any θ' from growing in number.

Interpretation

We have derived the ESS for our model analytically (eq. [A11]). However, the formulation is not straightforward to interpret. We first consider a simplified extreme case of the model and then explore how changing parameters influence the results.

Effect of Infection Rates

Consider the extreme case with very high rates of infection (large β) and recovery (large γ). Here, infection in the first environment is essentially guaranteed ($e^{-\beta T_1}$ in eq. [2] is very small), as is recovery in the second environment. In this case, the evolutionarily stable probability of migrating is

$$\theta_{ESS} = \begin{cases} 0 \text{ if } \dfrac{(1 - c_M)\sigma\phi}{1 - (1 - c_M)\sigma} < \dfrac{(1 - c_I)\sigma(1 - c_F)\phi}{1 - (1 - c_I)\sigma}, \\ 1 \text{ if } \dfrac{(1 - c_M)\sigma\phi}{1 - (1 - c_M)\sigma} > \dfrac{(1 - c_I)\sigma(1 - c_F)\phi}{1 - (1 - c_I)\sigma}. \end{cases} \tag{7}$$

The left-hand side of the inequality represents the ratio of maximum per capita gains and losses for individuals that migrate (and subsequently are susceptible). Here, the "gain" is the product of survival $(1 - c_M)\sigma$ and fecundity ϕ, and the "loss" is 1 minus survival. Similarly, the right-hand side is the same ratio but for individuals that do not migrate (and are infected). The best strategy is to never migrate ($\theta_{ESS} = 0$) when residents have a higher gain-to-loss ratio than migrants and to always migrate ($\theta_{ESS} = 1$) if the reverse is true. In the extreme case where there is no fecundity difference between susceptible and infected individuals ($c_F = 0$), the ESS is to never migrate if migration has a higher survival cost than infection does (i.e., $c_I < c_M$) and to always migrate if the reverse is true (fig. 2*A*).

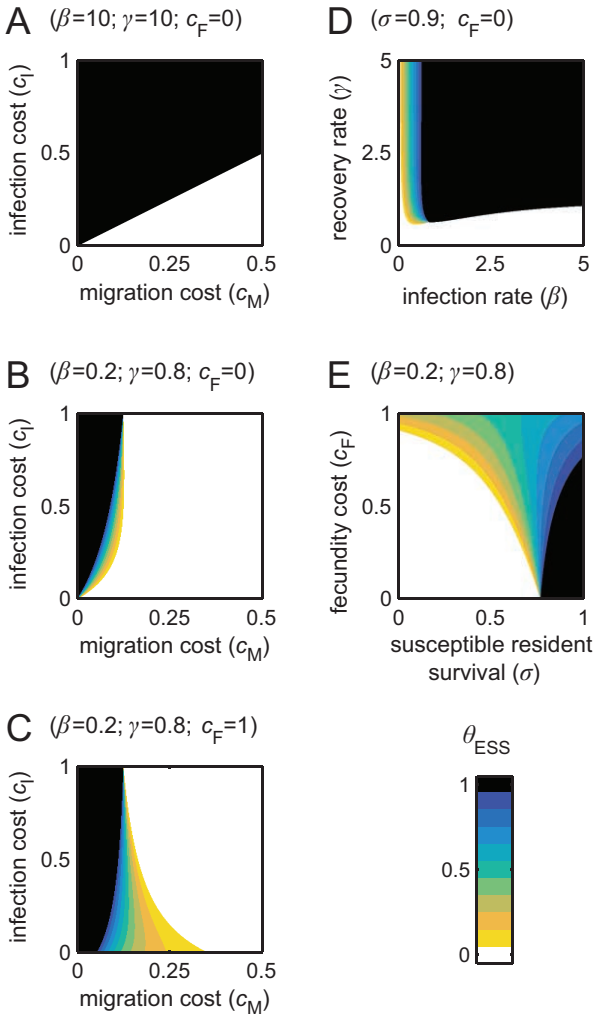

Figure 2: Evolutionarily stable probability of migrating (θ_{ESS}) as a function of parameters corresponding to mortality (*A*–*C*), infection (*D*), and survival and fecundity (*E*). Outcomes include always migrate (black; $\theta_{ESS} = 1$), never migrate (white; $\theta_{ESS} = 0$), and sometimes migrate (colored; $0 < \theta_{ESS} < 1$). *A*–*C* assume high annual survival of susceptible residents ($\sigma = 0.9$), that is, long-lived hosts. *A* shows very high infection (β) and recovery (γ) rates with no fecundity cost of infection (c_F). In contrast, *B* greatly reduces both infection and recovery. *C* imposes castration of infected hosts ($c_F = 1$, assuming full fecundity is restored once hosts clear infection). For *D* and *E*, the survival cost of infection ($c_I = 0.2$) exceeds that of migration ($c_M = 0.1$). All other parameters follow default values in table 1. ESS = evolutionarily stable strategy.

Of course, it is rare for an environment to promote either universal infection with parasites or universal recovery from infection. Thus, for smaller values of β and γ, not all residents will become infected, and not all infected migrants will recover. Here, the survival cost of infection must be much higher than the survival cost of migration before migration is favored (fig. 2*B*). Generally, the lower the rates of infection and recovery, the higher the costs of infection must be before migration is favored. Infection and recovery rates have different effects on the ESS: for low recovery migration is never favored, whereas for low infection the transition between nonmigratory and migratory strategies is more gradual (fig. 2*D*).

Partial Migration

Migration need not be an all-or-nothing approach to the problem of parasites. Although often the best strategy is to always or never migrate, there are conditions under which a mixed strategy, where only a fraction of the population migrates (partial migration), is favored (fig. 2, colored regions). Partial migration can be interpreted as a mixed strategy at the level of an individual. Suppose an uninfected individual that never migrates has a low probability of becoming infected in a single year. Across several years, the probability that this individual eventually becomes infected increases. Once infected, an individual stays infected (infection status across years is nonindependent) and cannot recover without migrating. Thus, a mixed strategy of migrating, for example, one year out of every five ($\theta_{ESS} = 0.2$) can arise. However, this strategy is favored only when individuals are relatively long-lived (high values of the baseline survival σ; fig. 2*E*). Mixed strategies are also more common when recovery is high and infection is low (fig. 2*D*).

Fecundity Costs of Infection

Parasites can affect more than just an individual's chance of survival. What happens when we consider these nonlethal costs of infection? Incorporating an infection-induced fecundity cost ($c_F > 0$) slightly increases the range of conditions favoring migration over residency and broadens the range favoring partial migration (fig. 2*B* vs. 2*C*). This increase is intuitive, given the additional cost of infection. However, it occurs only when the year-to-year survival of infected individuals is high enough for the occasional migration to be worthwhile (low c_I, high σ). Overall, this leads to a seemingly counterintuitive result: increasing the survival cost of infection can favor strategies where individuals migrate less (fig. 2*C*, high c_M values). This result can also be seen in figure 2*E*: for high susceptible resident survival, increasing the fecundity cost of infection can favor strategies where individuals migrate less. However, this decreased probability of migrating for increased infection cost occurs only when fecundity costs of infection (c_F) are high.

Migratory Escape versus Recovery

Our model includes a twofold benefit to migration. Migrants have the opportunity to lose their existing parasites. Additionally, our model assumes that migrants are not being infected with new parasites during this time. This second condition is technically migratory escape. Just how does our full model with migratory recovery differ from that including only migratory escape? Considering the case where $\gamma = 0$ (no recovery), we see that migratory escape alone is sufficient to favor migration in some cases, although generally partial migration is never adaptive when the only benefit to migration is escape (fig. 3). Adding migratory recovery on top of migratory escape has two effects: it increases

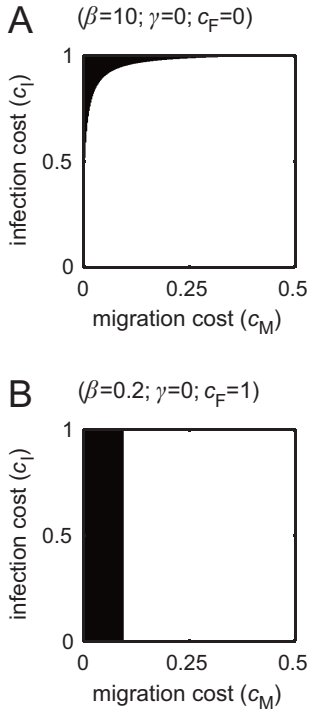

Figure 3: Evolutionarily stable probability of migrating (θ_{ESS}) as a function of the survival costs of migration (c_M) and infection (c_I), in the case of no recovery from infection in environment 2 ($\gamma = 0$). Outcomes include always migrate (black; $\theta_{ESS} = 1$) and never migrate (white; $\theta_{ESS} = 0$). *A* shows a high infection rate and no fecundity cost (like fig. 2*A*), and *B* shows a lower infection rate with parasitic castration (like fig. 2*C*). For both panels, $\sigma = 0.9$ (baseline survival). All other parameters follow default values in table 1. ESS = evolutionarily stable strategy.

the range of parameter space across which migration is favored (fig. 3*A* vs. 2*A*), and it favors partial migration in some cases (fig. 3*B* vs. 2*C*).

Discussion

Researchers increasingly recognize that parasites can drive the large-scale movement of animals, which, in turn, influences host life-history evolution. For instance, the dispersal patterns and maintained asexuality in bdelloid rotifers has been attributed to a loss of parasites due to desiccation stress (Wilson and Sherman 2010). Furthermore, escape from reef-based parasites is hypothesized as a selective force in the evolution of the pelagic larval phase experienced by many reef fishes (Strathmann et al. 2002). Nevertheless, escape or recovery from parasites is rarely considered to be the driving force behind seasonal migration. More often, research focuses on the negative consequences of seasonal migrations for disease spread (e.g., Waldenström et al. 2007; Elfving et al. 2010). Migrants may also be more susceptible to pathogens than residents since migrants encounter new parasites along their migratory route, often congregate in massive numbers at stopover points, and arrive from their migration in a weakened condition (Figuerola and Green 2000; Morgan et al. 2007; Altizer et al. 2011). Greater exposure, spread, and susceptibility to parasites may indeed represent migration-related costs in some species. However, these costs may be offset by several pathogen-related benefits to migration, which may ultimately lower infection risk in migrants.

Two parasite-related migration benefits have previously been identified. Migration can allow temporary escape from parasite-infested conspecifics or environments (migratory escape) or culling of infected individuals (migratory culling). Here, we propose a third benefit, which we term "migratory recovery": migration (especially between ecologically distinct areas) can help hosts seasonally lose their parasites and recover. We developed a model with migratory recovery and derive the conditions under which parasite-related costs are high enough to favor the evolution of seasonal migration.

Here, we report three primary results. First, we show that migration is favored over residency when the risk and costs of infection sufficiently outweigh the cost of migration—that is, to a first approximation, an individual should choose the behavior (migrate or not) that is least costly for any given year. Second, the best migratory behavior for long-lived hosts becomes more complicated. This complication arises because hosts can remain infected year to year unless they recover. In these scenarios, mixed strategies of migration and residency can evolve. Third, counterintuitive results arise when infection incurs both survival and fecundity costs. We might imagine that with two costs to infection, hosts should definitely migrate. However, increased mortality (from either migrating or becoming infected) reduces a host's lifespan. Short-lived hosts do not necessarily live long enough for the occasional migration strategy to be advantageous. Accordingly, we find that increasing infection mortality can sometimes actually decrease migration tendency.

Empirical Comparisons

Direct empirical support for migratory recovery is lacking. However, seasonal migration between two distinct environments may promote recovery from infection for a variety of aquatic and terrestrial species (table 2). Documented examples of environmental gradients leading to parasite loss include movement between habitats of different salinity, temperature, and/or humidity. Although changes to the host's external environment promote the loss of ectoparasites, endoparasite loss also occurs as hosts face internal physiological changes during seasonal migration. For instance, dietary changes throughout migration can make the chemical environment of the host's blood and/or digestive tract unsuitable for some parasite species (table 2; Buscher 1965; Wallace and Pence 1986). It is also possible that changes in host body temperature during migration across thermally distinct habitats leads to the loss of endoparasites in ectotherms. Recovery from infection may also occur if intermediate hosts are restricted in the breeding habitat (table 2; Möller 1978). These overlapping processes can lead to a predictable distance decay of both ecto- and endoparasites along a host's migration route (Thieltges et al. 2010).

Our model requires sufficiently high infection-related costs to favor the evolution of migration. Is this realistic? In nature, parasite-imposed costs range from moderate effects on host locomotion and energetics to high risk of mortality, as in the case of the chytrid fungus *Batrachochytrium dendrobatidis* (Skerratt et al. 2007). Parasites can also negatively impact host behavior in ways that ensure the parasite's own reproductive success (i.e., parasite-increased trophic transmission; Lafferty 1999; Barber et al. 2000). For instance, parasitized fish sometimes display altered movement patterns, habitat use, and risk-taking behaviors that increase predation by avian definitive hosts (for a review, see Barber et al. 2000). Systems where parasites impose high survival costs on hosts may experience increased selection pressure on strategies such as migration that will increase their chance of survival despite high migration costs.

Theoretical Comparisons

Migratory recovery can now join several other mechanisms that promote the evolution of partial migration. Uncertainty in survival can favor partial migration, where some individuals seasonally leave the area while others stay (Cohen 1967). Alternatively, separate density-dependent regulation

Table 2: Host species with documented potential for migratory recovery from their parasite(s)

Host	Parasite	Parasite type	Environmental factor	Estimated infection cost	Cost type	Reference(s)
Anatid ducks (pintails, shovelers, gadwells, teals)	Various trematodes and helminth worms, including Acanthocephala	Endo	Changed internal chemistry, intermediate host range	Unknown	Unknown	Buscher 1965
Carcharhinus leucas (bull shark)	*Dermophthirius maccallumi* (metazoan)	Ecto	↓Salinity	Unknown, but congeneric parasites have been implicated in the deaths of captive sharks	Energetic, mortality?	Watson and Thorson 1976; Cheung et al. 1982; Bullard et al. 2004
Gasterosteus aculeatus (three-spine stickleback)	Various ecto- and endoparasites, including *Schistocephalus solidus*	Ecto, endo	↑Salinity, ↑temperature, intermediate host range	Cost of *S. solidus*: increased metabolic rate, slower swimming speeds, increased mortality during food scarcity, reduced body condition	Energetic, mortality	Dartnall 1972; Barber et al. 2008; Karvonen et al. 2013
Ichthyosaura alpestris (alpine newt)	*Batrachochytrium dendrobatidis* (chytrid fungus)	Ecto	↓Humidity	Implicated in the decline and/or extinction of some 200 species	Mortality	Skerratt et al. 2007; A. Manica, personal communication
Mugil cephalus, *M. capito* (mullet)	Trematodes, monogeneans, and copepods, including *Caligus* species	Ecto, endo	↓Salinity	Copepods implicated in disease and mortality in aquaculture farms		Moravec and Libosvarsky 1975; Johnson et al. 2004; Baker et al. 2008
Platichthys flesus (European flounder), *Gadhus morhua* (Atlantic cod)	Various trematodes and copepods	Ecto, endo	↓Salinity	Unknown	Unknown	Möller 1978; Thieltges et al. 2010
Trichechus manatus manatus (Florida manatee)	Epibionts: barnacles and copepods	Ecto	↓Salinity	Unknown	Energetic	Ripple 1999
Zenaida macroura (mourning dove)	Various species of Ischnocera lice, including the genus *Columbicola*	Ecto	↓Humidity	Costs in host *Columba livia*: reduced male mating success, body and feather weight loss, reduced survival in high infestations, higher metabolic rate	Fecundity, energetic, mortality	Clayton 1990; Booth et al. 1993; Clayton et al. 1999; Moyer et al. 2002; Malenke et al. 2011

Note: Arrows indicate the direction of the environmental gradient promoting host recovery (parasite loss).

of migrant and resident populations can allow the two strategies to coexist (Kaitala et al. 1993). Finally, the ability to store breeding resources across years can lead to individuals migrating every few years to breed (Shaw and Levin 2011). Here, we demonstrate that nonindependence of infection status across years can also lead to partial migration. In other words, we expect partial migration to evolve in systems where infected individuals remain infected the next year unless they migrate and recover.

Some aspects of our model resemble the enemy release hypothesis (ERH), a key mechanism enabling invasiveness. The ERH postulates that invaders benefit from introduction to novel habitats because they escape the negative effects of coevolved predators and parasites (e.g., Torchin et al. 2003). The ERH resembles migratory recovery in several ways: (1) parasites are lost because intermediate hosts do not or cannot follow the invader/migrant to the new habitat, (2) conditions in the new habitat are not conducive to parasite growth and reproduction, and (3) the invader/migrant increases its own fitness by recovering from parasite-induced costs. Similar to our findings (fig. 2*D*), higher recovery in a new environment should promote invasion. Gradually, as parasites are acquired in the new range, some of the early advantage is reduced (e.g., Roche et al. 2010). Nevertheless, the initial advantage experienced by enemy release may be enough for invaders to establish in their introduced range and, similarly, for migration to evolve as a stable strategy in a population.

Future Directions

A number of the assumptions we made, particularly about the details of infection, could be relaxed in future models. First, we could change transmission dynamics. Here, we assume that individuals are infected at a constant rate, which is most suitable for parasites moving between multiple host species (complex lifecycle) or for parasites that have a free-living stage in the environment (indirect transmission). Alternatively, one could consider parasites that are transmitted directly from host to host by changing infection to be proportional to both the number of susceptible and the number of infected individuals (βSI). Such a model could be used to explore the scenario where migration to a new area promotes recovery from infection while not allowing individuals to avoid infected conspecifics (i.e., migratory recovery without migratory escape). Second, we could model host migration among communities of parasites. Here, we consider one parasite species and assume that migration always decreases infection. Since many species increase their exposure to new parasites during migration, future models could consider several parasite species and derive conditions under which host migration balances the benefit of recovery from one species with the cost of potential increased exposure to another. For example, repeated migrations between freshwater and saltwater may enable some salmonids to balance infection costs imposed by marine and freshwater parasites (Bailey et al. 1989; Higgins et al. 1993; Birkeland 1996). Indeed, the timing of sea trout (*Salmo trutta*) migration back to freshwater seems to be related to infestation by sea lice (*Lepeophtheirus salmoni*; Birkeland and Jakobsen 1997; Gjelland et al. 2014). Third, we could explicitly consider infection intensity. Here, we assume no cost difference between infections with one versus multiple parasites. This simplification means that our specific model predictions may be better suited to micro- rather than macroparasites. In the case of macroparasites, infestation intensity rather than occurrence influences the cost to hosts (Thompson et al. 1998). Expanding the model to include infection intensity that builds up through time would reveal the benefits of migration as a means of controlling parasite load rather than complete recovery from infection. Some seal-sucking lice systems may provide interesting empirical examples of this phenomenon (e.g., Leidenberger et al. 2007). Such a model could also include conditional strategies where movement depends on infection status (e.g., Gjelland et al. 2014). Fourth, we could explicitly consider the location of infection, which may impact both the costs to hosts and the benefits of migration as a recovery strategy. For instance, infestation in energetically costly tissues such as the brain, eyes, gonadal organs, or respiratory organs may be more likely to select for migration if the chance of recovery is high.

Animals experience many internal and external environmental changes during migration. We found documented potential for migratory recovery in systems where migrants experience a change in temperature, humidity, salinity, or internal chemistry (table 2). Changes in environmental factors such as oxygen levels, UV exposure, or flow conditions could hypothetically also facilitate recovery from parasites during migration, but these scenarios remain to be examined empirically. Furthermore, even if conditions at the hosts' breeding and wintering grounds are similar, parasite loss can occur at any point along the migration route. For instance, skin and surface pathogens such as chytrid fungus may be at risk of desiccation when amphibians migrate over land between ponds (A. Manica, personal communication). Thus, the range of conditions experienced by hosts along the entire migratory route should be considered when exploring migratory recovery as a selective force. It remains incredibly difficult to experimentally test the evolution of migration after exposure to parasites.

Individuals may not have to go far to benefit from migratory recovery. In fact, many nonmigratory species also face seasonally changing environments, which can slow pathogen transmission and promote recovery (Altizer et al. 2006). For instance, seasonal fluctuations in water temperature cause annual cycles in the parasite fauna of many

freshwater fishes (Dartnall 1972). Furthermore, behavioral changes that make use of contrasting environmental conditions between microhabitats may preclude the need for long-distance migration to achieve parasite loss (Kluger et al. 1975; Covert and Reynolds 1977; Moyer and Wagenbach 1995). These are valid considerations for researchers interested in exploring migratory recovery in their system. Although we have interpreted our model for the evolution of long-distance migration, we do not include distance explicitly. Thus, our results could extend to any system where individuals seek out habitats that promote recovery and return to reproduce. Our specific model could be modified to include strategies such as behavioral fever in order to understand the conditions under which such behaviors can evolve. We encourage researchers to build on this theoretical framework and consider loss of parasites as a potential explanation for animal movement patterns more broadly.

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Top, northern pintail (*Anas acuta*) female and male. Jackson Zoological Park, Jackson, Mississippi. (Photo © 2005 Jeff Whitlock, The Online Zoo, used with permission.) *Bottom*, Florida manatees (*Trichechus manatus manatus*) rooting for food in bottom sand of Crystal River National Wildlife Refuge. (Photo credit: Jim P. Reid, United States Fish and Wildlife Services.)

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Appendix from A. K. Shaw and S. A. Binning, "Migratory Recovery from Infection as a Selective Pressure for the Evolution of Migration" (Am. Nat., vol. 187, no. 4, p. 000)

Additional Model Detail

Model Development

We start by rewriting the full population model (eq. [6]) in matrix form and grouping terms by susceptible (S) and infected (I) individuals, by which individuals migrate (θ) or stay resident ($1 - \theta$), and by terms with and without density dependence (DD):

$$\begin{bmatrix} S \\ I \end{bmatrix}_{\tau+1} = \begin{bmatrix} A\theta + B(1-\theta) + \mathrm{DD}[J\theta + K(1-\theta)] & C\theta + \mathrm{DD}[L\theta + M(1-\theta)] \\ E\theta + F(1-\theta) & G\theta + H(1-\theta) \end{bmatrix} = \begin{bmatrix} S \\ I \end{bmatrix}_{\tau}, \tag{A1a}$$

where

$$A = \sigma_{\mathrm{SM}}[e^{-\beta T_1} + (1 - e^{-\beta T_1})(1 - e^{-\gamma T_2})], \tag{A1b}$$

$$B = \sigma_{\mathrm{SR}} e^{-\beta(T_1+T_2)}, \tag{A1c}$$

$$C = \sigma_{\mathrm{SM}}(1 - e^{-\gamma T_2}), \tag{A1d}$$

$$E = \sigma_{\mathrm{IM}} e^{-\gamma T_2}(1 - e^{-\beta T_1}), \tag{A1e}$$

$$F = \sigma_{\mathrm{IR}}[(1 - e^{-\beta T_1}) + (1 - e^{-\beta T_2})e^{-\beta T_1}], \tag{A1f}$$

$$G = \sigma_{\mathrm{IM}} e^{-\gamma T_2}, \tag{A1g}$$

$$H = \sigma_{\mathrm{IR}}, \tag{A1h}$$

$$J = \phi_{\mathrm{S}} A + \phi_{\mathrm{I}} \mathrm{E}, \tag{A1i}$$

$$K = \phi_{\mathrm{S}} B + \phi_{\mathrm{I}} \mathrm{F}, \tag{A1j}$$

$$L = \phi_{\mathrm{S}} C + \phi_{\mathrm{I}} G, \tag{A1k}$$

$$M = \phi_{\mathrm{I}} H, \tag{A1l}$$

where the coefficients A through M are all positive. This gives the number of susceptible and infected individuals at the start of next year ($\tau + 1$) as a function of those at the start of this year (τ).

As in the main text, θ is the probability of migrating; β and γ are the constant rates of infection and recovery, respectively; and T_1 and T_2 are the fraction of year that migrants spend in breeding and nonbreeding environments, respectively. Note that although in the main text we expressed the annual survival of infected and migratory individuals in

terms of the survival costs of infection and migration and similarly expressed the fecundity of infected individuals in terms of fecundity cost of infection, here we have left them in a more general form. This generality allows us to derive model results without making any assumptions about the relative values of survival and fecundity for each class. The annual survival probabilities of susceptible and infected residents and of susceptible and infected migrants are given by σ_{SR}, σ_{IR}, σ_{SM}, and σ_{IM}, respectively, and the fecundity of susceptible and infected individuals is given by ϕ_S and ϕ_I, respectively.

Similarly, we have left the density dependence (DD) in a general form. Our results are not influenced by the specific form of density dependence, as long as

$$\mathrm{DD}(S = 0,\ I = 0) = 1, \tag{A2a}$$

$$\frac{\partial \mathrm{DD}}{\partial S} < 0, \tag{A2b}$$

$$\frac{\partial \mathrm{DD}}{\partial I} < 0, \tag{A2c}$$

$$\mathrm{DD} \geq 0. \tag{A2d}$$

Model Analysis

For a model that was written explicitly in terms of S and I (with a specific form of density dependence, DD), we would analyze the model by solving for S^* and I^* explicitly to find the equilibrium population size. The next step would be to calculate the evolutionarily stable migration strategy, assuming the population was at the above equilibrium size. However, in our case DD is an unspecified function of S and I. It turns out that we can do the evolutionary analysis for the model without ever having to specify the exact form of the density dependence. To do so, we first find the nontrivial equilibrium by solving for DD^* in the same way we would solve for S^* and I^* in a traditional analysis. The equilibria of model (A1) are given by $S^* = I^* = 0$ (the trivial equilibrium) and

$$\mathrm{DD}^* = \frac{[1 - A\theta - B(1-\theta)][1 - G\theta - H(1-\theta)] - C\theta[E\theta + F(1-\theta)]}{[J\theta + K(1-\theta)][1 - G\theta - H(1-\theta)] + [L\theta + M(1-\theta)][E\theta + F(1-\theta)]}, \tag{A3a}$$

$$I^* = S^*\left[\frac{E\theta + F(1-\theta)}{1 - G\theta - H(1-\theta)}\right] \tag{A3b}$$

(the nontrivial equilibrium). If we specified how DD depends on S and I, equation (A3a) could be rewritten in terms of S^* and I^*. In this model, the population is viable (the trivial equilibrium is unstable) only if $\mathrm{DD}^* < 1$.

Next, we calculate the evolutionarily stable migration strategy, assuming the population is at the stable nontrivial equilibrium given by equation (A3) where all individuals use resident strategy $\overline{\theta}$. We denote the equilibrium of a population of individuals with strategy $\overline{\theta}$ as $\overline{\mathrm{DD}}$. A mutant individual with migration strategy θ' introduced into this population will grow in number according to

$$\begin{bmatrix} S' \\ I' \end{bmatrix}_{\tau+1} = \mathbf{J}\begin{bmatrix} S' \\ I' \end{bmatrix}_{\tau}, \tag{A4a}$$

where the Jacobian is given by

$$\mathbf{J} = \begin{bmatrix} A\theta' + B(1-\theta') + \overline{\mathrm{DD}}[J\theta' + K(1-\theta')] & C\theta' + \overline{\mathrm{DD}}[L\theta' + (1-\theta')M] \\ E\theta' + F(1-\theta') & G\theta' + H(1-\theta') \end{bmatrix}. \tag{A4b}$$

For $\overline{\theta}$ to be an ESS, we require that any mutant θ' is not able to grow in number, that is, that the dominant eigenvalue of **J** is less than 1 in magnitude. Since **J** is nonnegative, irreducible, and primitive (for the range of biologically realistic parameter values), there is one dominant eigenvalue that is real and positive. To determine when this eigenvalue (λ) is less than 1, we note that the characteristic equation of **J** is

$$\lambda^2 - (\mathrm{trJ})\lambda + \mathrm{detJ} = 0, \tag{A5a}$$

where trJ and detJ are the trace and determinant, respectively, of **J**, given by

$$\mathrm{trJ}(\theta') = \theta'\left[A - B + G - H + \overline{\mathrm{DD}}(J - K)\right] + B + H + K\overline{\mathrm{DD}}, \tag{A5b}$$

$$\begin{aligned}\mathrm{detJ}(\theta') = &\left[\theta'(A - B) + \theta'\overline{\mathrm{DD}}(J - K) + B + \overline{\mathrm{DD}}K\right]\left[\theta'(G - H) + H\right] \\ &- \left[\theta'(E - F) + F\right]\left[\theta' C + \theta'\overline{\mathrm{DD}}(L - M) + M\overline{\mathrm{DD}}\right].\end{aligned} \tag{A5c}$$

When the mutant and resident strategies are equal ($\theta' = \overline{\theta}$), the dominant eigenvalue is 1 (the population is at a stable equilibrium), and the characteristic equation is given by

$$(\lambda - 1)(\lambda - p) = 0. \tag{A6}$$

Using equations (A5a) and (A6), we know that

$$\mathrm{trJ}(\overline{\theta}) - \mathrm{detJ}(\overline{\theta}) = 1. \tag{A7}$$

When the mutant and resident strategies are not equal ($\theta' \neq \overline{\theta}$), we require that

$$\mathrm{trJ}(\theta') - \mathrm{detJ}(\theta') < 1 \tag{A8}$$

to ensure that the dominant eigenvalue is less than 1. Therefore, a migration strategy $\overline{\theta}$ is evolutionarily stable to invasions by a mutant with any other strategy θ' if

$$\mathrm{trJ}(\theta') - \mathrm{detJ}(\theta') < \mathrm{trJ}(\overline{\theta}) - \mathrm{detJ}(\overline{\theta}). \tag{A9}$$

Plugging in equations (A5b) and (A5c) and grouping by θ terms, this can be written as

$$x\theta'^2 + y\theta' < x\overline{\theta}^2 + y\overline{\theta} \tag{A10a}$$

for $\theta' \neq \overline{\theta}$, where

$$x = \left[C(E - F) - (A - B)(G - H)\right]\left[1 + \overline{\mathrm{DD}}\phi_S\right], \tag{A10b}$$

$$y = \left[(A - B)(1 - H) - B(G - H) + CF\right]\left[1 + \overline{\mathrm{DD}}\phi_S\right] + (G - H) + \overline{\mathrm{DD}}\phi_I(E - F). \tag{A10c}$$

Solving for the ESS here is then equivalent to maximizing $x\theta^2 + y\theta$ on the interval $0 \leq \theta \leq 1$. The evolutionarily stable probability of migrating can be summarized as

$$\theta_{\mathrm{ESS}} = \begin{cases} -y/(2x) & \text{if } 0 < y < -2x, \\ \text{either 0 or 1} & \text{if } -2x < y < 0, \\ 1 & \text{if } 0 < y,\ -2x < y, \\ 0 & \text{if } y < 0,\ y < -2x. \end{cases} \tag{A11}$$

This result demonstrates that a variety of outcomes are possible, including a pure ESS, where the entire population migrates ($\theta_{\text{ESS}} = 1$); a pure ESS, where the entire population does not migrate ($\theta_{\text{ESS}} = 0$); and a mixed ESS, where only a fraction of the population migrates each year ($0 < \theta_{\text{ESS}} < 1$; partial migration). Although this result suggests that a bistable ESS (both $\theta_{\text{ESS}} = 0$ and $\theta_{\text{ESS}} = 1$ are stable, and the end result depends on initial conditions) is possible, we have never observed this for any of the biologically realistic sets of parameter values we consider.

Extreme Case

To gain some intuition into the ESS (eq. [A11]), we consider the extreme case where infection in environment 1 is essentially guaranteed (β very large) and where recovery in environment 2 is also guaranteed (γ very large). Here, $e^{-\beta T} \to 0$ and $e^{-\gamma T} \to 0$. The full model (eq. [A1]) reduces to

$$\begin{bmatrix} S \\ I \end{bmatrix}_{\tau+1} = \begin{bmatrix} A\theta + \text{DD}[J\theta + K(1-\theta)] & A\theta + \text{DD}[J\theta + K(1-\theta)] \\ F(1-\theta) & F(1-\theta) \end{bmatrix} = \begin{bmatrix} S \\ I \end{bmatrix}_{\tau}, \tag{A12a}$$

where

$$A = \sigma_{\text{SM}}, \tag{A12b}$$

$$F = \sigma_{\text{IR,}} \tag{A12c}$$

$$J = \phi_{\text{S}} A, \tag{A12d}$$

$$K = \phi_{\text{I}} F. \tag{A12e}$$

The ESS (eq. [A11]) reduces to

$$\theta_{\text{ESS}} = \begin{cases} 1 & \text{if } y > 0, \\ 0 & \text{if } y < 0, \end{cases} \tag{A13a}$$

where

$$x = 0, \tag{A13b}$$

$$y = A - F + \overline{\text{DD}}(J - K). \tag{A13c}$$

Using the above ESS condition along with the equilibrium given by

$$\text{DD}^{*} = \frac{[1 - A\theta - F(1-\theta)]}{[J\theta + K(1-\theta)]}, \tag{A14a}$$

$$I^{*} = S^{*}\left[\frac{F(1-\theta)}{1 - F(1-\theta)}\right], \tag{A14b}$$

we can calculate the ESS in terms of our basic model parameters as

$$\theta_{\mathrm{ESS}} = \begin{cases} 0 & \text{if } \dfrac{\sigma_{\mathrm{SM}}\phi_{\mathrm{S}}}{1-\sigma_{\mathrm{SM}}} < \dfrac{\sigma_{\mathrm{IR}}\phi_{\mathrm{I}}}{1-\sigma_{\mathrm{IR}}}, \\ 1 & \text{if } \dfrac{\sigma_{\mathrm{SM}}\phi_{\mathrm{S}}}{1-\sigma_{\mathrm{SM}}} > \dfrac{\sigma_{\mathrm{IR}}\phi_{\mathrm{I}}}{1-\sigma_{\mathrm{IR}}}. \end{cases} \tag{A15}$$