

Research



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Gaps in modelling animal migration with evolutionary game theory: infection can favour the loss of migration

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Ongoing environmental changes alter how natural selection shapes animal migration. Understanding how these changes play out theoretically can be done using evolutionary game theoretic (EGT) approaches, such as looking for evolutionarily stable strategies. Here, we first describe historical patterns of how EGT models have explored different drivers of migration. We find that there are substantial gaps in both the taxa (mammals, amphibians, reptiles, insects) and mechanisms (mutualism, interspecific competition) included in past EGT models of migration. Although enemy interactions, including parasites, are increasingly considered in models of animal migration, they remain the least studied of factors for migration considered to date. Furthermore, few papers look at changes in migration in response to perturbations (e.g. climate change, new species interactions). To address this gap, we present a new EGT model to understand how infection with a novel parasite changes host migration. We find three possible outcomes when migrants encounter novel parasites: maintenance of migration (despite the added infection cost), loss of migration (evolutionary shift to residency) or population collapse, depending on the risk and cost of getting infected, and the cost currency. Our work demonstrates how emerging infection can alter animal behaviour such as migration.

This article is part of the theme issue 'Half a century of evolutionary games: a synthesis of theory, application and future directions'.

1. Introduction

Evolutionary game theory (EGT) is a tool for understanding what behaviours are expected to emerge in populations of organisms [1,2], by accounting for the population context (see this issue). Movement behaviours, such as dispersal or migration (predictable, and often round-trip, movement of animals through different habitats), are well-suited to studying with EGT since organismal movement is driven in part by the presence (or absence) of other individuals. As individuals move, the spatial structure of populations emerges, which in turn shapes individual movement strategies, leading to an eco-evolutionary feedback loop [3].

Organisms that travel long distances (e.g. migratory animals) are likely to be impacted by ongoing ecological transformation [4–7], i.e. land use change, urbanization, habitat encroachment, invasive species, climate change [8–11]. Migration has evolved in many species as a way for individuals to maximize their fitness in seasonal or otherwise variable environments, allowing individuals to track favourable climates, resources and/or minimize negative biotic interactions (such as predation, competition and parasitism [12,13]). Unfortunately, animal migrations are declining globally due to anthropogenic changes such as habitat destruction and barriers [4]. Changes to biotic interactions can also lead to loss of migratory behaviour. For instance, the introduction of non-native perennial milkweed (*Asclepias curassavica*) in southern parts of the United States has promoted a shift to residency in formerly migratory populations of monarch butterflies

(*Danaus plexippus*; [14,15]). These drivers of migratory loss are less studied and understood, perhaps because there are inherent challenges to studying both species interactions and migratory behaviours in natural populations [13].

EGT can help us understand not only how migration evolves, but how it might shift in response to changing conditions. The history of EGT approaches to migration goes back several decades [16], yet novel models are still being developed. However, we lack a good quantitative understanding of where gaps lie and what remains to be understood. EGT models of migration span both analytic [17,18] and numerical [19,20] techniques and have invoked a number of selective pressures (mechanisms) for the evolution of migration including dependence on an individual's state or status (energy, dominance, body size, age; [21–25]), competition through density dependence [26,27], predation ([28,29], including harvesting [30]), pathogens and parasites [31,32] and abiotic factors (climate, temperature, latitude; [28,33,34]). Here, we first conduct a systematic review of the historical context for EGT in studies on seasonal migration to identify knowledge gaps, and then, based on an identified gap, we develop a model to understand how infection with novel parasites, a phenomenon likely to increase due to ecological transformation, can influence migratory behaviours.

2. Historical background of the evolutionarily stable strategy in migration behaviour

To quantify historical patterns, we searched Web of Science for papers that use EGT in models of animal migration. We conducted six searches on 30 November 2021 using the following term combinations: evolutionary stable* and migrat*; evolutionary game theory and migrat*; 'invasion analysis' (with quotes) and migrat*; migrat* and citing Maynard Smith & Price 1973 [1]; migrat* and citing Maynard Smith 1974 [35]. Together, these searches yielded 300 papers. We skimmed each paper title and abstract and removed papers that were not relevant (e.g. cellular migration, organismal dispersal, empirical work), which left us with 40 papers (electronic supplementary material, table S1). We categorized these papers according to the modelling method used (analytical, numerical, both), focal taxon (birds, fish, zooplankton, system-agnostic) and mechanisms (selective pressure on migration) considered (state dependence, density dependence, predation, parasites/pathogens, abiotic factors). To categorize papers, we read the abstract, searched the main text for keywords relating to the categories, and read the text surrounding the keywords. Finally, we determined which papers modelled changes in migration strategy in response to an environmental perturbation. This last criteria was more challenging than the others to identify with certainty since some papers discussed perturbations without modelling them explicitly.

Of the 40 relevant papers, 11 used analytical methods, 15 numerical, and 14 used a combination of both methods (figure 1a). Analytic solutions, exact answers that can be obtained without the use of a computer (i.e. with pencil and paper), were used in the oldest three papers in our search (all from the 1980s [16,36,37]). By contrast, numeric solutions, approximate answers that require a computer, were used in the 1990s [21,30,38–41] and onwards, reflecting the increased availability of computational resources in science. It was not until 2002 that another purely analytical paper was published [34]. Since then, there has been a mix of analytical and

numerical approaches with the vast majority of papers incorporating at least some numerical methods. Since there is typically a trade-off between analytical approaches (which offer exact solutions to biologically simpler questions) and numerical approaches (which offer approximate solutions to biologically more complex questions), a combination of approaches is often seen as the best approach.

In terms of taxa, 10 papers focused on birds, 13 on fish, eight on zooplankton and nine were general (system-agnostic) (figure 1b). The oldest papers (from the 1980s and early 1990s) focused on zooplankton [16,37,38] and birds [21,36]. The mid-1990s brought the first system-agnostic paper [39] and the first fish-focused paper [30] in our search. The subsequent decades included papers on a mix of systems, although no zooplankton-focused papers were published between 1999 and 2019. The most notable pattern, however, is what is missing: there were no models specifically for terrestrial migrants: mammals, amphibians, reptiles or insects. We propose four explanations for these absences. First, perhaps existing EGT models for these taxa were not picked up by our search. For example, Fryxell *et al.*'s evolutionary ungulate model does not explicitly use the word 'evolution' [42]. Second, EGT models for other taxa may be sufficient to describe migration in these taxa. For example, Shaw and Levin's system-agnostic model draws examples from invertebrates, amphibians, reptiles and mammals [24]. Third, EGT models may not be the most appropriate tool for studying migration in these taxa. For example, Hays models sea turtle migration frequency (remigration intervals) as a condition-dependent process without an evolutionary framing [43]. Finally, movements in some taxa (e.g. insects [44]) are not considered migration by all researchers and thus might be overlooked by theorists developing EGT models.

We identified several interactions between model mechanism driving migratory behaviour and focal taxon (figure 1c). Most papers, across taxa, consider density dependence and abiotic factors (31 papers each). Many (16) papers also considered state dependence. Papers considering interspecific interactions were less common. Completely absent were the consideration of mutualists or interspecific competitors as mechanisms driving migratory behaviour. By contrast, predators/parasites/pathogens were considered, although less frequently than state dependence, density dependence or abiotic factors. Predation (including harvesting) was only considered as a mechanism in fish and zooplankton papers (12 papers). Pathogens and parasites were least often included, and typically considered a mechanism in papers that were system agnostic (five papers). We also found very few papers (7 of 40, marked with asterisk in electronic supplementary material, table S1) that modelled changes in migration strategy in response to a perturbation (climate change, harvesting, habitat loss). Overall, interspecific interactions have been less of a focus in EGT models of migratory behaviour. In particular, the role of a perturbation such as the introduction of novel parasites in leading to migratory loss has not been explored, despite a pressing need to understand how emerging infectious diseases influence animal movement patterns more broadly.

3. Migratory loss in response to an emerging infection

Parasites can affect host physiology and behaviour in ways that impact their movement abilities [45]. For example, new

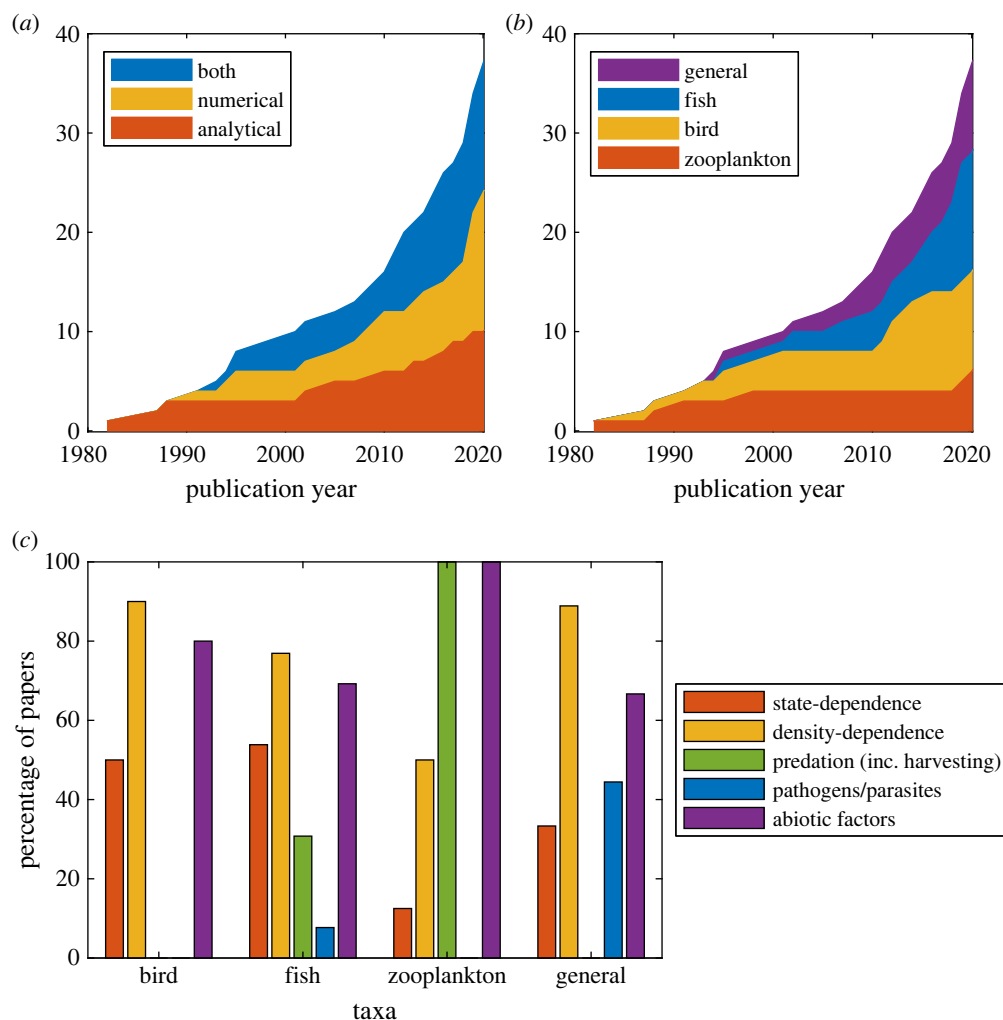


Figure 1. Literature search results. The cumulative number of papers ($N = 40$) over time (a) using each methodological approach (analytical: orange; numerical: yellow; both: blue), and (b) on each taxonomic group (zooplankton: orange; bird: yellow; fish: blue; general: purple). (c) The distribution of mechanisms driving migratory behaviour included in each model by taxa (state dependence: orange - 16 papers; density dependence: yellow - 31 papers; predation/harvesting: green - 12 papers; pathogens/parasites: blue - 5 papers; abiotic factors: purple - 31 papers; papers can include more than one factor).

parasites may divert resources and energy away from the host, increasing transportation costs and altering host energy allocation to movement [45]. As such, a migrant exposed to a new parasite may experience an increased cost of migratory behaviour without a corresponding increase in benefit. If year-long residency in the migrant's breeding habitat becomes increasingly possible because of altered climates and food availability, infection with a new parasite may lead to loss of migration. Alternatively, if residency is not possible, loss of migration may lead to population collapse or extinction.

Migrants, vectors and parasites are all affected by ecological transformation. In response, migrants may change their migration routes and travel further, less far and/or across different habitats than they would have historically. Ecological transformation can also lead animals to change their migration timing [6]. Similarly, ecological transformation may allow reservoir hosts to expand their range. Vectors of parasites such as ticks and mosquitoes can also shift geographical ranges as a result of changes in land use and climate [46,47]. All of these changes can expose migrants to new parasites, vectors and reservoir hosts. This is of concern as changes to either host or pathogen ecology, or both, is associated with disease emergence [48]. Disease emergence can lead to spillover (and spillback) of generalist parasites between species [49], including those from invasive species (e.g. novel weapons hypothesis [8]). A recent study suggests that land

use and climate change will increase cross-species transmission, particularly of viruses, and that future hotspots for viral transmission will be in biodiverse areas at high elevations with a high population density of humans [50].

Ecological transformation can also lead parasites themselves to better infect migrant hosts. For example, the persistence of some parasites in the environment is increasing due to climate change [9]. Vectors and their pathogens generally replicate better in warmer conditions [51]; more frequent extreme weather events can lead to outbreaks of environmentally transmitted pathogens (e.g. more anthrax introduction in grazers [52]), and longer summers/warmer winters can increase the active infection window in trematodes, helminths and ticks [53–55]. Thus, in this era of the anthropocene and emerging infectious diseases, a migrant could encounter a new parasite in a new migration location, or in the same migration location that now has a new parasite introduced to this area.

Any of these mechanisms could bring a migratory species into contact with a novel parasite. Here we ask, under what conditions does this new contact, and subsequent infection, lead a migratory species to evolve a shift to residency? That is, when is migration still worth it, despite the increased chance of parasite infection? And what host–parasite system characteristics are most likely to lead to loss of migration? We

build a mathematical model to answer these questions. In particular, we explore how cost currency (whether residency and infection each reduce survival or fecundity) shapes the model outcomes.

(a) Model methods

To understand how infection with a novel parasite can affect a migratory population, we built a population-level model that tracks host behaviour (in terms of migration) and host status (in terms of infection). We track the number of susceptible (S) and infected (I) individuals in a host population both throughout an annual cycle and across years (see electronic supplementary material, table S2 for all model parameters and variables). We assume the annual cycle is split into two 'seasons' of length τ_1 each (set equal; $\tau_1 = \tau_2 = 0.5$). Hosts are characterized by a migration strategy (θ , the fraction of the population migrating). We assume that residency and infection are costly and that the 'currency' of these costs are either reduced survival or fecundity. In this section, we develop the general model (allowing for any combination of infection and migration), and then in the results section we explain how we analysed the model to ask our question (i.e. starting with a migratory population and introducing a novel parasite).

(i) Annual cycle

During the annual cycle, the processes of migration and infection occur followed by mortality and then reproduction. We call the initial number of susceptible and infected individuals S_0 and I_0 , respectively. During the first season (of length τ_1), all individuals inhabit environment A where there is no transmission, i.e. the population dynamics are constant. Thus, the number of susceptible and infected individuals at the end of the first season is

$$S(\tau_1) = S_0, \quad (3.1a)$$

and

$$I(\tau_1) = I_0. \quad (3.1b)$$

Next, a fraction θ of the population migrates to environment B for the second season (of length τ_2) while the rest remain resident in environment A. In environment B, susceptible individuals can become infected with the novel parasite (if it is present) indirectly from the environment at rate β ; the population dynamics here are given by

$$\frac{dS}{dt} = -\beta S \quad (3.2a)$$

and

$$\frac{dI}{dt} = \beta S. \quad (3.2b)$$

At the end of the second season, migrants return to environment A and we account for mortality. This assumption allows us to derive results analytically; accounting for mortality throughout the year would lead to fluctuating population sizes and make our analytical approach intractable. Our intuition from past work is that this logistical assumption (per [56]) would not qualitatively change the outcome. Thus, the number of surviving susceptible and infected migrant individuals at the end of the second season (found by integrating equations ((3.2a) and (3.2b)) is

$$S_M(\tau_1 + \tau_2) = \theta[S_0 e^{-\beta\tau_2}] \sigma \quad (3.3a)$$

and

$$I_M(\tau_1 + \tau_2) = \theta[I_0 + S_0(1 - e^{-\beta\tau_2})](1 - \mu_I)\sigma \quad (3.3b)$$

where σ is the survival of susceptible migrants (set to 0.9) and μ_I describes the survival cost of infection. We also account for mortality of residents; the number of surviving susceptible and infected resident individuals at the end of the second season is

$$S_R(\tau_1 + \tau_2) = (1 - \theta)S_0(1 - \mu_R)\sigma \quad (3.4a)$$

and

$$I_R(\tau_1 + \tau_2) = (1 - \theta)I_0(1 - \mu_I)(1 - \mu_R)\sigma \quad (3.4b)$$

where μ_R describes the survival cost of residency. Since we are considering a population that has evolved to migrate before being exposed to the novel parasite, migration must confer some benefit compared to residency, such as access to seasonal food, escape from enemies or seasonally harsh conditions [57–60]. We assume that the costs of residency and infection are multiplicative. For example, an individual that migrated one year, became infected, then stayed resident the following year would be an infected resident and pay both costs.

Finally, reproduction occurs, where surviving individuals compete among themselves for access to breeding resources. The number of offspring produced is

$$b = [\phi S_M(\tau_1 + \tau_2) + (1 - \psi_R)\phi S_R(\tau_1 + \tau_2) + (1 - \psi_I)\phi I_M(\tau_1 + \tau_2) + (1 - \psi_I)(1 - \psi_R)\phi I_R(\tau_1 + \tau_2)]\Delta(N) \quad (3.5)$$

where ϕ is the fecundity of susceptible migrants (set to 2), ψ_R describes the fecundity cost of residency, and ψ_I describes the fecundity cost of infection, and $\Delta(N)$ is a density-dependent function that captures how much competition among individuals reduces fecundity, where N is the total population size. To derive our results, we do not specify the form of $\Delta(N)$, but just require that there is no competition when no individuals are present ($\Delta(0) = 1$) and increasing population size always increases competition (Δ is a strictly decreasing function of N). As for survival, we assume that the fecundity costs of residency and infection are multiplicative. Finally, we assume that all offspring are born susceptible (and thus get added to the count of S individuals). Doing so, we can finally write down an expression relating the number of susceptible and infected individuals in one year to the number in the next (see electronic supplementary material, appendix S2 for full model equations).

(ii) Model analysis

Next, we use an evolutionary game theory approach to analyse the model. Specifically, we use adaptive dynamics to analytically determine the evolutionarily stable strategy (ESS; [1]) of migratory tendency as a function of all the model parameters (see electronic supplementary material, appendix S2 for details). Although we can express the ESS mathematically (electronic supplementary material, equation (S11)), it is easier to interpret through figures showing the ESS plotted as a function of key model parameters for a number of scenarios (below). Once we found the ESS migration strategy, we determined what impact it had on parasite infection prevalence (see electronic supplementary material, appendix S2 for derivation of prevalence).

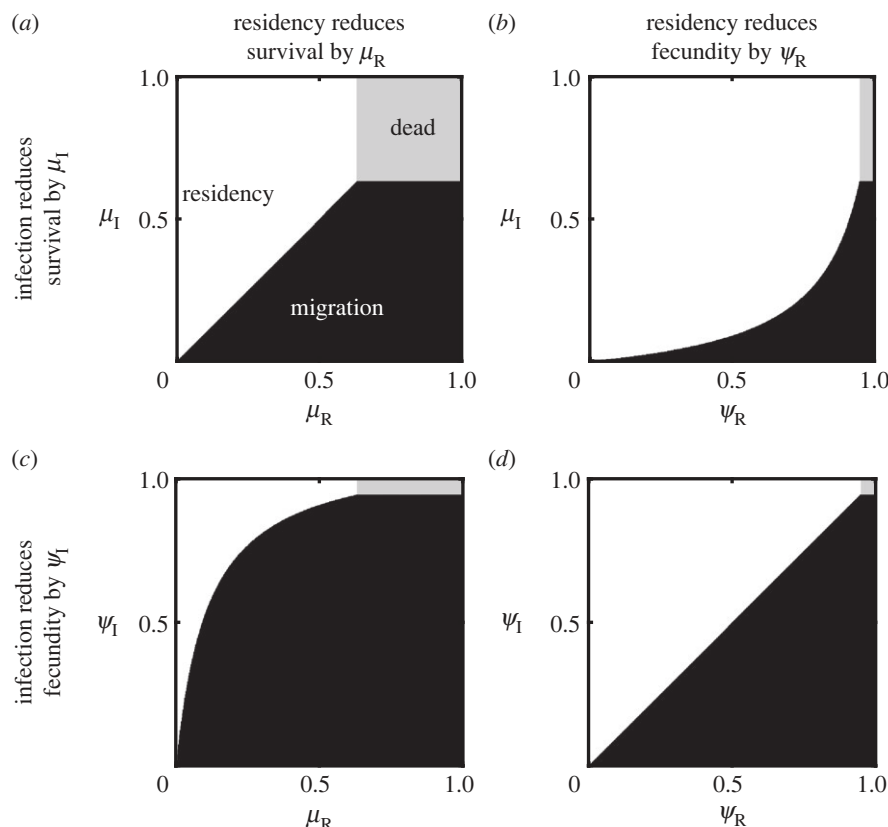


Figure 2. Migration strategies. The evolutionarily stable migration strategy (fraction of the population migrating) for four cost-currency scenarios: (a) infection and residency both reduce survival; (b) infection reduces survival, residency reduces fecundity; (c) infection reduces fecundity, residency reduces survival; and (d) infection and residency both reduce fecundity. Parameters: $\beta = 10$; costs are zero ($\mu_R = \psi_R = \mu_I = \psi_I = 0$) unless varied along x- and y-axes. White areas indicate where the population evolved full residency, black areas indicate where the population maintained migration, and grey areas indicate where the population went extinct.

(b) Model results

(i) Evolutionarily stable strategy in the absence of infection

To start, we consider a population in the absence of the novel parasite ($I_0 = 0$), and determine under what conditions migration is favoured. For this scenario, the evolutionarily stable migration strategy (the fraction of the population migrating) is simply

$$\theta = \begin{cases} 0, & \text{if } \sigma[1 + \phi\Delta(N)] > (1 - \mu_R)\sigma[1 + (1 - \psi_R)\phi\Delta(N)] \\ 1, & \text{if } \sigma[1 + \phi\Delta(N)] < (1 - \mu_R)\sigma[1 + (1 - \psi_R)\phi\Delta(N)] \end{cases} \quad (3.6)$$

(see electronic supplementary material, appendix S2 for derivation). In other words, full migration ($\theta = 1$) is best when the expected growth rate of migrants (survival plus reproduction) exceeds the expected growth rate of residents, and full residency ($\theta = 0$) is best when the reverse is true. Intuitively, if we assume that there is some cost to residency (μ_R or ψ_R greater than 0), we find that full migration is always favoured. Thus, in the absence of any infection, this population evolves to migrate.

(ii) Evolutionarily stable strategy in the presence of parasite infection

Adding a parasite to environment B leads to three possible outcomes. First, migration can persist despite the added cost of infection, if the infection cost is low relative to the cost of residency (figure 2, black regions). Second, hosts can switch to residency, if the cost of infection is too high relative

(figure 2, white regions). Third, the host population can go extinct, if both costs (infection, residency) are too high (figure 2, grey regions). Intriguingly, we never see an outcome of partial migration ($0 < \theta < 1$), where only a subset of the population migrates each year (an outcome we have seen in many of our earlier models [61–64]).

The currency (reduced survival or reduced fecundity) of each cost shapes the outcome. Sustained migration is favoured across the broadest range of cost values when infection reduces fecundity and residency reduces survival (figure 2c). Conversely, hosts switch to residency across the broadest range of cost values when infection reduces survival and residency reduces fecundity (figure 2b). Overall then, reduced survival is a stronger motivator than reduced fecundity. This makes intuitive sense. A fecundity cost lowers fitness for a single year. By contrast, a survival cost lowers fitness for the current year and all future years by decreasing the probability that an individual survives to reproduce again. Thus, parasites that reduce host survival should more often drive their hosts to residency than parasites that reduce host fecundity. We see a similar outcome by comparing different baseline host survival rates (electronic supplementary material, figure S2). In our model we assume costs are proportional, so as the baseline survival (σ) increases, the survival cost increases as well. Thus, we see a shift from residency to migration as σ increases if doing so increases the survival cost of residency (electronic supplementary material, figure S2c). And we see the opposite (a shift from migration to residency) as σ increases if doing so increases the survival cost of infection (electronic supplementary material, figure S2b). By contrast, changing

host fecundity, and the fecundity costs of infection and residency have little effect on migratory behaviour (electronic supplementary material, figure S2 y-axes).

The cost and currency of infection along with host behaviour (migration or residency) shape the proportion of the population infected (infection prevalence). A parasite with higher survival cost (i.e. more likely to kill its host) intuitively leads to lower infection prevalence in the host population, by removing infected hosts through death (electronic supplementary material, figure S1a, orange line). Conversely, parasites that reduce host fecundity instead of killing their host lead to higher infection prevalence (electronic supplementary material, figure S1a). When the infection cost is so high that hosts switch to residency, the parasite is eradicated from the host population and prevalence drops to zero (electronic supplementary material, figure S1a). Parasites with higher transmission rates have higher infection prevalence (electronic supplementary material, figure S1b). For sufficiently high parasite transmission (and thus risk of infection), hosts switch to residency, again eradicating the parasite.

(c) Model discussion

Overall, introduction of a novel parasite has the potential to lead to three outcomes: maintenance of migration (despite the added infection cost), loss of migration, or population collapse. Migration is typically maintained when the cost and risk of getting infected by the new parasite are relatively low, and when the alternative of residency has a cost in currency of survival. Conversely, migration is often lost in response to novel parasites that reduce host fecundity (instead of survival). Population collapse is most likely when both parasites and residency reduce survival and least likely when both reduce fecundity (figure 2). These results parallel those found in Shaw *et al.* [62], which explored the conditions under which migratory behaviour evolves following the introduction of a novel parasite. Both here and in that study, that cost currency of movement and infection are key factors shaping the migratory outcome. Intriguingly, here we never saw a response of reduced migration (i.e. shift from full to partial migration); a result that may be different under different transmission scenarios (i.e. direct transmission). Indeed, Shaw *et al.* [62], found that parasite transmission mode was an important determinant of the evolved migration strategy. Both Shaw *et al.* [62] and this paper explore how novel parasite introduction can fundamentally alter movement decisions of hosts. However, while Shaw *et al.* [62] were interested in understanding how historic selection pressures may have influenced contemporary migratory behaviours, this paper addresses a more pressing conservation issue in the context of ongoing ecological transformation: how will novel parasite infection affect current migratory behaviours in the future? By generating predictions around when we expect to see fundamental shifts in migratory behaviours following pathogen introductions (i.e. when infections have important fecundity costs or when increased residency and infection have significant survival costs for hosts), we hope to provide useful insights for assessing which populations may be most at risk of migration loss or collapse following parasite spillover.

Our model assumes parasites are acquired indirectly from the environment, and so most closely fits parasites that are vector-transmitted (i.e. some blood-borne infections), have a long free-living stage in the environment (i.e. arthropods

including ticks), or that have complex life cycles (i.e. many species of helminth worms). There are empirical examples of novel parasites threatening host migratory behaviours in indirectly transmitted infections. For example, the invasive nematode *Anguillicoloides crassus* threatens populations of European eels, *Anguilla anguilla*, by damaging their swim bladders and swimming abilities, which can lead to death and migration failure [65,66]. This novel infection is acquired by eels through ingestion of the nematode's intermediate hosts, which can be copepods or other crustaceans. Our model could be expanded to directly transmitted infections as well. Empirical examples of directly transmitted novel parasites affecting host movement patterns include sea lice in salmonids, chytridiomycosis in amphibians and *Ophryocystis elektroscirrha* infection in monarch butterflies [15,67,68]. Similarly, our findings complement previous empirical work exploring how migration disrupted for another reason might impact infection dynamics. For example, recreational feeding during winter months has disrupted the migration patterns of numerous species of birds, insects, mammals and fishes (reviewed in [69]). Elk (*Cervus elaphus*) supplemented with food in winter migrate shorter distances, spend longer at stop-over sites and arrive at summer ranges later than unfed elk [70]. Hay supplementation in the western USA is related to higher rates of brucellosis infection in elk [71]. Similarly, bird feeders can be a source of disease transmission, especially when birds congregate around them in winter [72–74]. Other forms of resource supplementation, which may incentivize residency, include the provisioning of habitat [73]. Efforts to combat population declines of monarch butterflies (*Danaus plexippus*) in parts of the southern USA include planting non-native tropical milkweed (*Asclepias curassavica*) in gardens, which do not enter dormancy in autumn and allow butterflies to breed year-round [69]. Unfortunately, this increased availability of breeding sites has led to the formation of fully resident monarch populations, which experience severe infection with the protozoa *O. elektroscirrha* [69].

Most interactions between potential novel hosts and parasites do not result in pathogen spillover [75–77]. When pathogen spillover does occur, it is most frequently between two closely related hosts [78]. In these cases, pathogens typically do not exact a high mortality cost on novel hosts [79]. For example, brucellosis, which can be transmitted from elk to cattle, causes reproductive failure, but rarely death in adults [79]. However, spillover events between distantly related hosts, although rarer, are more likely to lead to higher mortality costs [79]. Ongoing environmental changes that bring new sets of species into contact are increasing the opportunities for spillover between more distantly related hosts. Thus, we should expect that an increasingly larger proportion of spillover events will cause high host mortality. Combined with our model results, this suggests that loss of migration will become an increasingly common phenomenon in the future.

4. Conclusion

Despite several decades of researchers using evolutionary game theory (EGT) approaches to study migration, there are still gaps in our knowledge. First, given the heterogeneity in taxa covered by current theory, we should ask how important it is to have taxa-specific models. Can migrations in the

missing taxa (mammals, amphibians, reptiles, insects) be sufficiently understood by existing models, or should we develop EGT models for these groups? Second, we have effectively no theory about how mutualistic interactions or interspecific competition shape the evolution of migration. Future work should determine whether this gap is due to an unintended oversight on the part of theorists or lack of empirical support to justify developing models. Finally, the majority of models on parasites as a mechanism for migration have aimed to understand increased migration in response to infection. Here we consider the opposite, which has rarely been studied: when does novel infection lead to migration loss? Future theoretical work using EGT approaches should expand our approach to determine how robust our findings are to our specific assumptions. For example, we never observed partial migration, but our past work suggests that a reduction in migration (i.e. shift from full migration to partial migration) should be a possible outcome when parasites have density-dependent transmission [62], when migration decisions depend on infection [63,64], and when recovery from infection is possible [61]. Our work is in line with the broader idea that parasites are critical, if overlooked, drivers

of host behaviour and life history and that, 50 years on, EGT remains a topical and relevant way of studying patterns of animal behaviour, including migration.

Data accessibility. The model code is provided on Zenodo: <https://doi.org/10.5281/zenodo.7139870> [80].

The data are provided in the electronic supplementary material [81].

Authors' contributions. A.K.S.: conceptualization, funding acquisition, investigation, methodology, software, supervision, visualization, writing—original draft, writing—review and editing, M.T.: data curation, investigation, visualization, writing—original draft, writing—review and editing, M.E.C.: conceptualization funding acquisition, writing—original draft, writing—review and editing, S.A.B.: conceptualization, funding acquisition, writing—original draft, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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