Title: Migration timing depends on balancing the risks of exposure and escape from infection

Short title: Migratory timing and infection risk

Authors: Allison K. Shaw[1], Raine MacArthur-Waltz[2], Sandra A. Binning[3]

[1] Department of Ecology, Evolution, and Behavior, University of Minnesota, St. Paul, Minnesota, USA, ashaw@umn.edu
[2] Department of Plant and Microbial Biology, University of Minnesota, St. Paul, Minnesota, USA
[3] Département de sciences biologiques, Université de Montréal, 1375 Ave. Thérèse-Lavoie-Roux, Montréal, QC, H2V 0B3, Canada

Abstract

As anthropogenic changes alter animal migration routes, understanding the consequences of these changes is critical for conservation. One consequence of altered migratory routes is exposure to infection by new parasites (i.e., migratory exposure). Theoretical models aiming to understand migration-infection dynamics typically have focused on infection-related benefits (i.e., migratory escape) to migration and have not included migratory exposure as a cost, which limits our ability to understand how changes to migration might be constrained by infection-related costs. Here, we explore one way animals cope with altered migratory routes: by adjusting the amount of time spent in different environments to minimize overall intensity of parasite infection. We develop a model of one host moving between two environments (each with an environmentally-specific parasite). We find that if the function describing parasite infection accumulation over time is accelerating, the optimal strategy is for hosts to split their time between environments. However, if both parasite accumulation functions are saturating, it is best for hosts to stick to a single environment. This model generates novel predictions about how time spent by migratory species in different environments is shaped by coinfection with multiple parasites.

Keywords: bet-hedging, host-parasite interactions, migratory behavior, movement ecology, parasite abundance, parasite intensity

Introduction

Animal migration, the predictable, round-trip movement of individuals between distinct habitats, is a widespread behavior that occurs in many organisms from insects to large mammals (Dingle and Drake 2007, Dingle 2014). Yet, this behavior is increasingly threatened by anthropogenic change (Cooke et al. 2024). Habitat loss, climatic change and barriers (e.g., dams, fences, wind farms) are among the most worrisome pressures faced by animals trying to get from one habitat to the next (Wilcove and Wikelski 2008, Xu et al. 2021, Tolvanen et al. 2023, Cooke et al. 2024, Lisovski et al. 2024). Luckily for some species, migration is a plastic behavior allowing individuals to alter aspects of their migration to mitigate the challenges presented by local changes (Xu et al. 2021, Lewin et al. 2024, Laforge et al. 2025). For example, elk (*Cervus canadensis*) have been known to alter their migration routes, timing, duration and distance in response to changes in predation risk, precipitation regimes, land use and forage availability (White and Garrott 2005, Jones et al. 2014, Middleton et al. 2018, Rickbeil et al. 2019, Laforge et al. 2025). Climate-related shifts in the timing and/or location of bird and marine vertebrate migrations is also increasingly reported (van Weelden et al. 2021, Lewin et al. 2024, Kuletz et al. 2024, Puleo et al. 2025, López-Ramírez et al. 2025). However, changes to migration timing and traditional migration routes may introduce new challenges for migrants including the need to locate new stopover or foraging sites, moving through unsuitable or suboptimal habitats, and navigating a new landscape of natural enemies such as predators and pathogens (Hall et al. 2016, Gallagher et al. 2017, Daversa et al. 2017, Weinstein et al. 2018, Lisovski et al. 2024). Exploring how migrants optimize their migration strategies to minimize these pressures is key to understanding the resilience of migratory behaviors in the face of anthropogenic change.

While migratory behaviors have traditionally been studied in the context of understanding how migrants track resources including food, shelter and mates or favourable climates (Dingle and Drake 2007), the implications of the predictable movements of individuals across distinct landscapes in terms of

interactions with natural enemies is of increasing interest to researchers. In particular, the idea that infection can both shape and be shaped by animal migration is now an established concept in the literature (Altizer et al. 2011, Peacock et al. 2020, Poulin and de Angeli Dutra 2021, Binning et al. 2022). Conceptual and theoretical models backed by a growing body of empirical evidence have shown that individuals can gain infection-related benefits from migration via processes including environmental and social escape from parasites (migratory escape), recovery from existing infections (migratory recovery) and a reduction in the number of infected conspecifics in a population during migration (migratory culling) (Binning et al. 2022). Alternatively, migrants may also be at a greater risk of becoming infected by parasites and pathogens than non-migratory individuals since moving across greater ranges and diverse landscapes increases the encounter probability with infectious agents (migratory exposure) (Altizer et al. 2011, Binning et al. 2022). For example, some species of migratory ungulates, fishes and birds harbor greater prevalence, richness and/or abundance of parasites than residents (Figuerola and Green 2000, Koprivnikar and Leung 2015, Teitelbaum et al. 2018, Shaw et al. 2018). Although this risk of migration is well known and acknowledged in many systems, particularly migratory birds (Figuerola and Green 2000, Koprivnikar and Leung 2015), it is not often explicitly accounted for in theoretical models (but see Shaw et al. 2018), which more commonly assume that moving away from the breeding environment benefits migrants through social and environmental migratory escape (Shaw and Binning 2020, Binning et al. 2022).

As many migratory species are being forced to migrate along non-traditional routes and/or during altered times to reach their destination, they may encounter novel circumstances (e.g., new habitats, new aggregations of con- and heterospecifics) that enhance opportunities for parasite transmission (Altizer et al. 2011, Cooke et al. 2024). As a result, migratory exposure may fundamentally alter the costs and benefits of migration from an infection standpoint. Indeed, migrants may need to balance the risk of exposure to new infections as a result of migration with the risk of accumulating high infection intensity (i.e., number of parasites in an infected host (Reiczigel et al. 2019)) if they remain. A similar situation

arises in existing systems including sea trout (*Salmo trutta*), which migrate from marine to freshwater environments to breed. Migration timing in sea trout appears to be influenced by the prevalence of parasitic sea lice in the marine environment as well as individual infection status: higher infections drive earlier migration back to freshwater rivers (Birkeland and Jakobsen 1997, Serra-Llinares et al. 2020). However, once in freshwater, trout can be infected with trematodes and monogeneans, setting up a potential trade-off in terms of the optimal time individuals should spend in each habitat in order to minimize the accumulation of parasites (Mo 1997). How migrants might balance parasite escape and exposure as they move between environments is an increasingly relevant question as threats to traditional migratory routes and patterns increase.

Here, we build a theoretical model to understand optimal migration timing in the face of migratory escape and exposure. We focus on the case where migrants move between two environments, accumulating a different parasite in each one, and use it to ask three questions. First, what is the best (optimal) migration timing strategy (including the possibility of not migrating) for minimizing overall infection? Second, how big is the infection benefit of the optimal strategy compared to the baseline of not migrating? Third, how sensitive is infection intensity to migration timing (i.e., how bad is it to be slightly not optimal)? By explicitly incorporating migratory exposure into models (something that has rarely been done), we are able to better understand the role this phenomenon plays in shaping migration timing.

Methods

Model

We developed a model with minimal parameters and complexity: one host, two environments (A and B), and two environment-specific parasites (A and B). See Table S1 for all model parameters. This model

describes a scenario in which a host starts with no vertically transmitted infection intensity and completes one migratory cycle as follows. Hosts start the year in environment A, accumulating infection by parasite A, resulting in infection intensity I_A. Next, hosts move to a second environment B, where they accumulate infection by parasite B, at infection intensity I_B. Finally, hosts return to environment A at the end of the year. Overall, hosts spend a proportion τ of the year in environment A and the remainder $(1 - \tau)$ in environment B (Figure S1).

We describe the accumulation of infection as a function of the time spent in a given environment. For example, spending τ time in environment A yields an infection intensity for parasite A given by

$$I_A = \tau^{1/\delta_A} \quad [1]$$

where δ_A is the accumulation for parasite A (in environment A). The value of δ_A determines the shape of the parasite accumulation curve. Values of $\delta_A > 1$ correspond to saturating infection accumulation (see (Balstad et al. 2020), $\delta_A = 1$ indicate a linear curve, and values of $\delta_A < 1$ correspond to accelerating curves (Figure 1). Parasite accumulation curves have previously been modeled in these ways based on existing empirical evidence (Duerr et al. 2003, Benesh 2011, Shaw et al. 2023, Albery et al. 2024). Note that all curves end up at the same point at the end of the year. In other words, individuals become maximally infected ($I_A = 1$) if they spend the full year in a single environment ($\tau = 1$), regardless of the value of δ_A. Overall, bigger values of δ_A lead to a faster initial accumulation of infection intensity before leveling off, while lower values of δ_A cause an initially slower accumulation before speeding up. Infection with parasite B accumulates similarly while in environment B: a host spending $1 - \tau$ time in environment B will have an infection intensity for parasite B given by

$$I_B = (1-\tau)^{1/\delta_B} \quad [2]$$

where δ_B is the accumulation for parasite B (in environment B). Using a single exponential form for both parasites gives the model flexible performance without unneeded complication. The δ parameterization allows us to describe different possible trajectories for increase in infection intensity, but also biologically

indicates that the parasites cause the same overall cost to fitness if a complete year is spent in its environment.

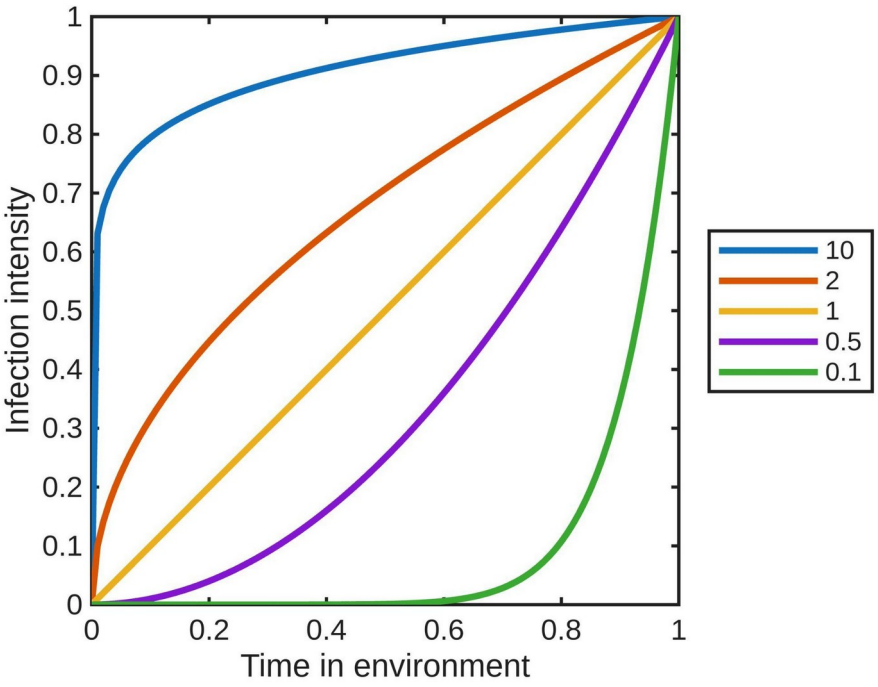

Figure 1. Intensity of parasite infection (y-axis; I_A for parasite A and I_B for parasite B) in hosts as a function of time spent in that parasite's environment (x-axis; τ in environment A, 1–τ in environment B) for 5 different values of the parasite accumulation parameter (different colored lines; δ_A for parasite A, δ_B for parasite B). Values of $\delta > 1$ correspond to saturating infection accumulation, $\delta = 1$ indicate a linear accumulation, and values of $\delta < 1$ correspond to accelerating.

To focus our model on the core mechanisms of interest, we make a number of simplifying assumptions. We assume that both parasites are environment specific (e.g., there is no lag between leaving an environment, and the ending of exposure to its corresponding parasite), we ignore interactions between parasites (e.g., being infected with parasite A does not influence subsequent infection with parasite B), and assume there is no recovery from infection. We also assume that the cost of infection is lower host fitness, and that both parasites reduce host fitness in a comparable manner. In other words, it is appropriate to add the effects as a net infection intensity given by

$$I_\tau = \tau^{1/\delta_A} + (1-\tau)^{1/\delta_B} \quad [3]$$

(i.e., adding equations 1 and 2 together). These simplifying assumptions are reasonable since they do not

inhibit our ability to address the primary question of interest (although they may provide interesting avenues for further exploration).

Analysis

Our aim is to determine what host migration timing strategy (value of τ) should be favored to minimize infection intensity. In other words, what proportion of the year should hosts spend in Environment A versus in Environment B to minimize their overall infection intensity (given by equation 3), which will maximize host fitness? We look for the optimal migration timing, which we call τ^*. Possible outcomes could include: migrating immediately ($\tau^* = 0$), splitting the year between the two environments ($0 < \tau^* < 1$), or never migrating ($\tau^* = 1$). We describe the infection intensity that corresponds to τ^* as I_τ^* (the minimal possible infection intensity).

To find τ^*, we look for the critical point(s) of equation 3. We could do this by taking the partial derivative with respect to τ, setting it to zero, and solving for τ. However, this approach does not have a straightforward solution and overlooks the possibility that infection might be minimized by one of the end point cases, i.e., we seek the value of τ^* on the interval [0,1] that minimizes equation 3. Thus, we instead use a numeric approach to find any critical points and calculate their corresponding I_τ value to determine which produced the global minimum value of I_τ.

Next, we look at the consequences for a host of using a migration strategy other than the optimal one. Our first approach is to quantify how much more infection intensity a host would experience by not migrating at all (i.e., if it used $\tau = 1$) compared to migrating optimally. In our model, hosts become fully infected if they never migrate. In other words, $I_\tau = 1$ for $\tau = 1$, giving us a non-migratory (resident) baseline to compare optimal migration strategies against. Thus, the difference in infection intensity experienced by a resident host compared to migrating optimally is

$$D_R = 1 - I_\tau^* \quad . \qquad [4]$$

Given how we have set up the model, $I_\tau = 1$ also for $\tau = 0$. In other words, never migrating (spending all time in environment A) and migrating immediately (spending no time in environment A) will always have the same infection cost.

As a second way to measure the consequences of using a non-optimal strategy, we describe the cost (in terms of increased infection intensity) to hosts of mistiming migration (i.e. migrating too early or too late with respect to the optimal timing in terms of minimizing infection). The amount of increased infection intensity a host that migrates early with error ϵ experiences is given by

$$D_E(\epsilon) = I(\tau^* - \epsilon) - I_\tau^* \qquad [5]$$

and similarly the cost to a host of migrating late with error ϵ is given by

$$D_L(\epsilon) = I(\tau^* + \epsilon) - I_\tau^* \quad . \qquad [6]$$

Here, ϵ describes the size of the migration timing error in terms of the proportion of the year. We used $\epsilon = 0.05$ as a default (i.e., 5% of the year, equivalent to an 18 day shift in migration timing as can be observed in some migratory birds and mammals; Bischof et al. 2012, Dossman et al. 2023), and also looked at $\epsilon = 0.1$ (i.e. 10% of the year or a 36 day shift) for comparison. A host migrating early means it spends less time in environment A than optimal (i.e., $\tau^* - \epsilon$) whereas a host migrating late means it spends more time in environment A than optimal (i.e., $\tau^* + \epsilon$).

Results

Across the parameter values that we considered, we found a range of possible optimal migration strategies (Figure 2A). The behavior of the model can be described in four quadrants in terms of the accumulation

parameter values for parasite A (δ_A) and parasite B (δ_B). When the parasite accumulation curves of both parasites A and B follow an accelerating profile (i.e., δ_A, $\delta_B < 1$; see Figure 1), there is always a migration strategy that minimizes infection intensity (Figure 2A, lower left quadrant). This strategy is always more favorable than not migrating and involves splitting time relatively evenly between the two environments (intermediate τ^* values). If parasite B instead accumulates following a saturating curve ($\delta_B > 1$), while parasite A is accelerating ($\delta_A < 1$), hosts benefit by spending less time in environment A as δ_A increases (smaller τ^* values; Figure 2A, upper left quadrant). If instead it is parasite A that accumulates following a saturating curve ($\delta_A > 1$), while parasite B is accelerating ($\delta_B < 1$), hosts benefit if they spend more time in environment A (larger τ^* values) and in some cases not migrating is the optimal strategy (when δ_B approaches 1, grey regions) (Figure 2A, lower right quadrant). Lastly, if both parasites accumulate following saturating curves (δ_A, $\delta_B > 1$), migration is never favorable in terms of parasite accumulation compared to not migrating (Figure 2A, upper right quadrant).

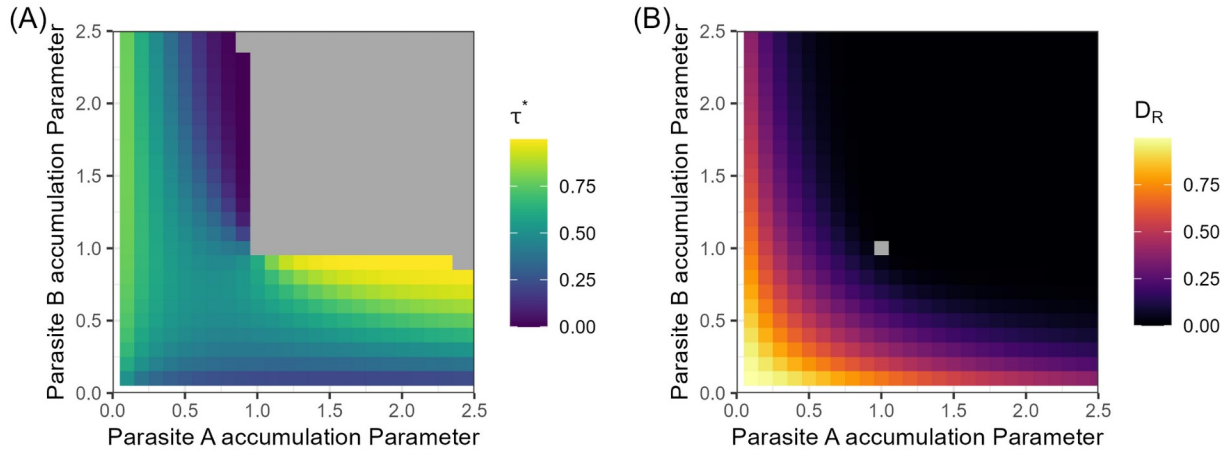

Figure 2. A: The optimal host migration strategy (colors, τ^*) as a function of the infection accumulation parameter for parasite A (x-axis, δ_A) and parasite B (y-axis, δ_B), ranging from 0 to 2.5 in 0.1 increments. B: The amount of infection avoided by migrating optimally relative to not migrating (D_R, equation 4). Gray represents cases where there are multiple optional strategies.

We found that the benefit to migrating (i.e., how much infection can be avoided) compared to not migrating also varies across parasite accumulation parameters (Figure 2B). The largest migration benefit is found in cases where both parasite accumulation curves are accelerating (Figure 2B, lower left), where

it is possible to have nearly zero infection intensity at the end of the season with well timed migration. This scenario is in contrast to the other quadrants, where the infection-related benefit, if any, to migrating is small.

These results can be unpacked by looking at the within-year dynamics of a few example cases (Figure 3). We again start in the lower left quadrant, when both parasites accumulate following accelerating curves (Figure 3C). Here, parasite infection accumulates increasingly quickly the longer a host stays in an environment, so it is best for hosts to split their time between environment A and environment B. During the first part of the year, hosts accumulate infection following the accumulation curve for parasite A in environment A (blue line in Figure 3C). If hosts were to spend the full year in environment A, they would follow the accelerating blue curve and end up with an infection of I=1. However, by switching to environment B partway through the year, the host switches to an initially lower rate of infection accumulation described by the accumulation curve for parasite B in environment B (purple line in Figure 3C), resulting in an overall lower rate of infection intensity at the end of the year (dashed line in Figure 3C) compared to not migrating. Next, we move to the second quadrant where parasite B accumulates following a saturating curve (Figure 3A). Because the parasite B curve is saturating, most of the cost is paid early on and remaining in environment B longer leads to slower accumulation of infection intensity. In contrast for parasite A (accelerating), the infection cost increases the longer the host stays in environment A. Thus, the optimal strategy is to move from environment A to environment B relatively early on (Figure 3A). In the third quadrant, where parasite B accelerates and parasite A saturates (Figure 3D), we see the reverse. Here, the cost for parasite A is high early on and slows over time, whereas the cost for parasite B starts slow and increases. However, switching to environment B before the end of the year still often leads to an overall lower infection intensity than not migrating, although the switch typically comes relatively late in the year (Figure 3D). In the final quadrant, where both parasites accumulate following saturating curves, most of the cost of infection is paid early on for both environments (Figure 3B). This scenario means that switching environments leads to a higher cost than

staying put. Thus, migration is never favored compared to not migrating (Figure 3B).

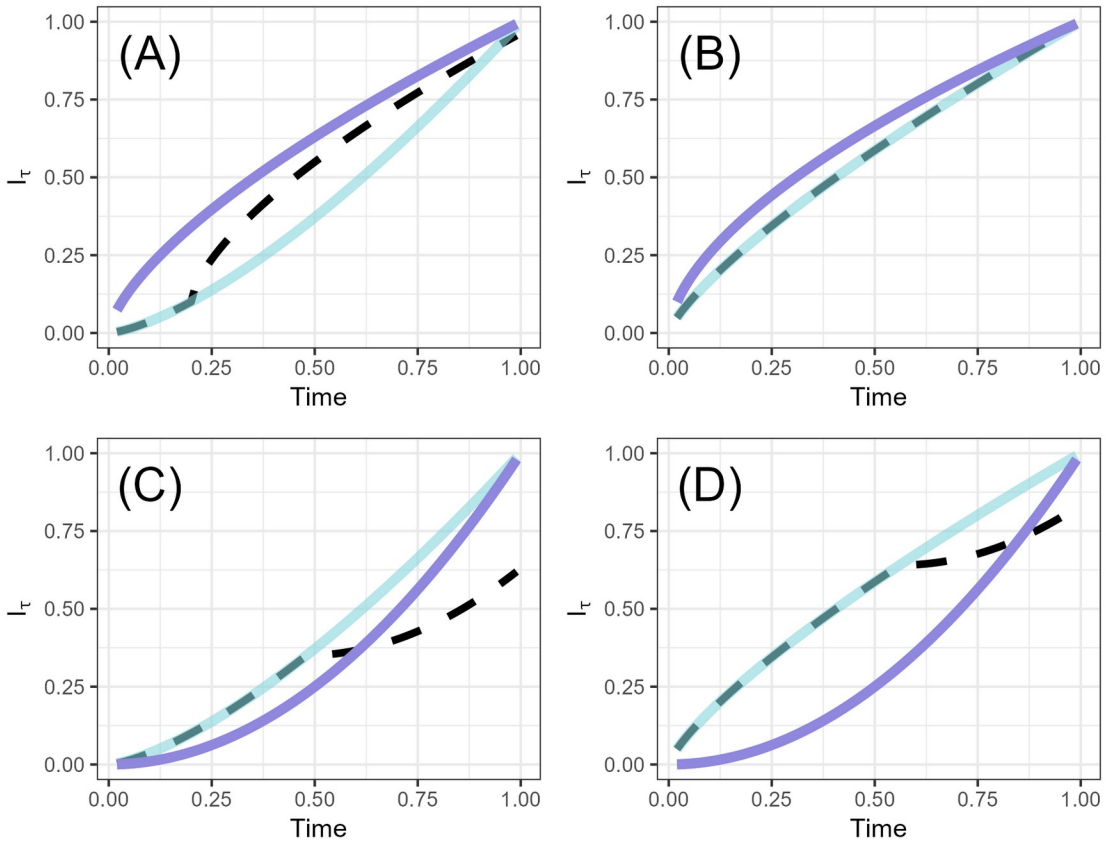

Figure 3. Four examples (corresponding to the 4 quadrants of Figure 2A) of infection intensity over time for hosts that spend all of their time in environment A (blue solid lines), all of their time in environment B (purple solid lines), or use the optimal migration strategy (black dashed lines), for three different sets of parasite accumulation parameters: (A) $\delta_A = 0.7$ and $\delta_B = 1.5$, (B) $\delta_A = 1.3$ and $\delta_B = 1.5$. (C) $\delta_A = 0.7$ and $\delta_B = 0.5$, and (D) $\delta_A = 1.3$ and $\delta_B = 0.5$.

Intriguingly, the optimal migration strategy is not monotonic with respect to either parasite parameter. Instead, when $\delta_A<1$, gradually increasing δ_B (the parasite B accumulation parameter), leads to hosts spending increasingly less time in environment B (larger τ^*) before switching to spending increasing more time there (smaller τ^*) (moving vertically across Figure 2A). When both parasites follow accelerating curves where parasite B initially accumulates more slowly ($\delta_B < \delta_A < 1$), hosts switch from environment A to B relatively early on (Figure S2A). As δ_B increases, getting closer to δ_A, the relative benefit of being in environment B decreases and hosts are favored to spend equal time in both environments (Figure S2B).

As δ_B increases further, passing δ_A and 1 in value, the parasite B curve switches to be saturating. Now, the cost of being in environment B decreases the longer hosts stay there while the cost of being in environment A increases the longer the host stays there. Thus, hosts are now favored to spend less time in environment A (Figure S2C). A parallel result can be seen by gradually increasing δ_A (the parasite accumulation parameter for parasite A), which leads to hosts spending increasingly less time in environment A, before switching to spending increasingly more time there (moving horizontally across Figure 2A). These patterns stand in contrast to patterns in the amount of infection benefit that hosts gain by migrating, which *does* change monotonically with respect to each of the two parasite parameters (Figure 2B).

Given that migratory species can display flexibility in the start date of their migrations, we can look at how important it is for hosts to be precise with their migration timing. Specifically, we ask what the cost to hosts is (in terms of increased infection) of migrating 5% earlier or later than the optimal migration timing. Over the course of a 365 day annual cycle, a 5% change in migration timing corresponds to an approximately 18 day difference in migration timing, which can occur in nature (Bischof et al. 2012, Dossman et al. 2023). We find that migrating early (i.e. leaving environment A earlier than what is optimal) is most costly when δ_B is very small (0.1) and $\delta_A > 0.5$ (Figure 4A). In this region of parameter space, the optimal strategy ($\tau^* \sim 0.2$) is to spend ~20% of the year in environment A and then move to environment B for the remaining ~80% (Figure 2A lower part). The accumulation curve for parasite B increases dramatically starting around 0.8 (Figure 1 green line), so spending too much time in environment B (i.e., leaving environment A too early) comes at a steep cost. Migrating early is also somewhat costly when δ_A is very small and $\delta_B > 0.5$, as well as when $\delta_A > 2$ and δ_B is close to 1 (Figure 4A). Mirroring the results for migrating too early, we find that migrating late (i.e. staying in environment A longer than is optimal) is mostly costly when δ_A is very small (0.1) and $\delta_B > 0.5$ (Figure 4B). In this region of parameter space, the optimal strategy is to spend ~80% of the year in environment A and only 20% in environment B (Figure 2A far left). The logic here mirrors the case of migrating too early above.

Similar to above, migrating late is also somewhat costly when δ_B is very small and $\delta_A > 0.5$, as well as when δ_A is close to 1 and δ_B is very large. These results are qualitatively true under a larger error to migrating (10% or approximately 36 days, shown in Figure S3). Overall, we see that when δ_A and δ_B are different, there is an asymmetry in cost where migrating earlier is worse (or better) than migrating late (Figure S4).

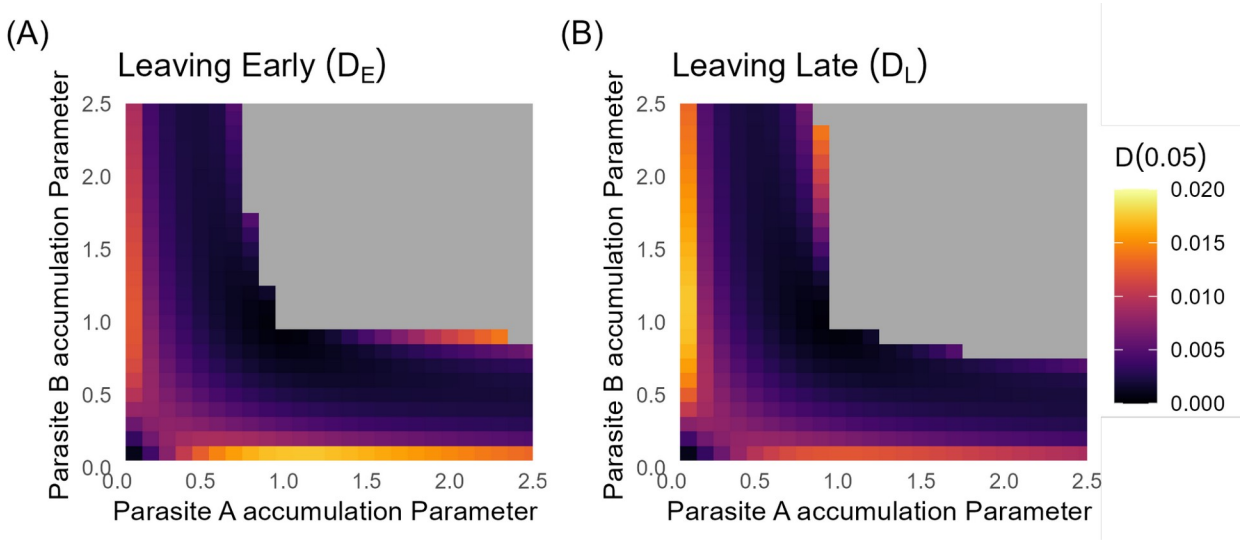

Figure 4. How bad is leaving environment A early versus leaving environment A late for hosts from an infection perspective? The cost (in terms of increased infection intensity) of migrating (A) 5% of the year too early, $D_E(0.05)$ or (B) 5% of the year too late $D_L(0.05)$, compared to migrating with optimal (τ^*) as a function of the infection accumulation parameter for parasite A (x-axis, δ_A) and parasite B (y-axis, δ_B), ranging from 0 to 2.5 in 0.1 increments. Gray represents cases where there are multiple optional strategies. See Figure S3 with a 10% error for comparison.

Discussion

In the face of a new barrier to a common migratory route, when might you expect a population of ungulates to forge a new path or to remain year-round as residents? If weather patterns become more unpredictable, how much should a migratory bird advance or delay the start of their spring migration and what are the consequences of not adjusting? Answering these questions empirically is challenging,

especially when trying to assess the impact of changing migration patterns on wildlife disease spread.

Theoretical models can help bridge gaps in our understanding by allowing us to explore different outcomes across a broader range of scenarios than most data sets allow, providing a valuable complement to data-driven research questions (Servedio et al. 2014). We developed a model to understand how the conflicting pressures of avoiding environment-specific parasites shapes host migration strategies between two environments. In terms of minimizing parasite infection, we found that migration was often a better strategy than not migrating despite the cost of migratory exposure. However, the details of how much time hosts should spend in each environment and how much infection could be avoided by migrating depends on the parasite accumulation functions. The shape of the parasite accumulation functions also influences the extent to which hosts benefit under the optimal strategy compared to not migrating. Furthermore, mistiming migration can have different consequences when hosts migrate too early or too late.

Biological interpretation

The rate at which hosts accumulate parasites in nature depends on a number of factors including parasite life-history traits and transmission stages, host immunocompetence and condition, and environmental factors such as temperature, humidity and habitat structure (e.g. Mignatti et al. 2016, Cable et al. 2017, Davenport et al. 2024, Silva et al. 2025). Hosts may accumulate parasites at an accelerating rate (convex function) when existing infections suppress immunity thereby making a host more susceptible to infection by the same parasite. For example, In West Africa, human infection with the roundworm *Onchocerca volvulus* increases with parasite burden due to immunosuppression (Duerr et al. 2003). Alternatively, parasites accumulating at a saturating (concave) rate can occur when hosts can develop acquired immunity against a parasite that makes them more able to defend against future infections. For instance, rainbow trout (*Oncorhynchus mykiss*) in experimental mesocosms accumulated infection with *Diplostomum spathaceum* trematodes more slowly the longer they were in the cages suggesting that their

immunity to infection built up with increasing exposure (Karvonen et al. 2004). In our model we found
342 that if both parasites accumulated according to a saturating function (concave), it was better for hosts to never migrate. In contrast, if one or both parasites accumulated according to an accelerating function,
344 migration was always a better strategy than remaining. Thus our model predicts that the details of how parasites and hosts interact will shape whether or not migration is an effective strategy to minimize
346 parasite infection.

348 Our model did not account for costs of migration that are independent of infection. In reality, hosts can incur substantial direct costs as a result of migration in terms of increased energy expenditure or
350 mortality (Wikelski et al. 2003, Rittenhouse et al. 2009). Despite not explicitly including such costs in our model, we can interpret our results in a way that speaks to these costs. For example, under some
352 parameter combinations, the benefit to migrating is quite small in terms of the amount of infection avoided. In these cases, we expect that migration would only be favored if the direct costs (e.g., mortality)
354 were quite low. However, under other parameter combinations such as when both parasites have strongly accelerating accumulation functions, the benefit to migrating is quite large (Figure 2B). In this case, we
356 expect that migration would be favored more easily (i.e., under a wider range of direct costs to migration).

358 We explored cases where hosts mistime their migration with respect to the optimal time for minimizing parasite infection. When, biologically, might we expect this outcome to happen? First, migrants may be
360 optimizing the timing of their migration based on factors other than infection. For example, to maximize reproductive success, many birds face selection pressure to migrate early in order to secure breeding sites
362 (Kokko 1999), whereas birds that experience poorer overwintering conditions may need to migrate later in the year (Dossman et al. 2023). Either of these cases might come with an infection cost if birds miss the
364 optimal timing in terms of infection. Second, changing conditions can lead to a shift in optimal migration timing that migrants may not be able to match, depending on what cues they use to time their migration
366 (Torstenson and Shaw 2025). Plasticity in migration timing is common in many migratory species, and

may be an important mechanism for buffering against perturbations such as climatic or physical barriers to migration as well as altered parasite exposure (Bischof et al. 2012, Serra-Llinares et al. 2020, Dossman et al. 2023, Chauveau et al. 2025, Smith et al. 2026).

Connections to past theory

Our result regarding the shape of the parasite accumulation curve is effectively an example of Jensen's inequality. This inequality states that for concave functions, the average value of a function evaluated at each of two points will be smaller than if one were to first take the average of the two points and evaluate that same function at that new point (Jensen 1906, Ruel and Ayres 1999). So, for example, when our parasite accumulation curve is concave (saturating) (e.g., $\delta=2$ in Figure 1), a host that never migrates would have an infection intensity of 0 for parasite B and 1 for parasite A (black dots in Figure S5), for a total infection intensity of 1. In contrast, splitting time between both environments (say 0.5 in each one or $\tau=0.5$) would yield an infection intensity of 0.7 for each parasite (red dot in Figure S5A), for a total infection intensity of 1.4, which is greater than 1. Thus, splitting time between two environments results in a higher infection intensity than only visiting a single environment. In contrast if both parasite accumulation functions are convex (accelerating) (e.g., $\delta=0.5$ in Figure 1), splitting time between both environments (again say 0.5 in each one) would yield an infection intensity of 0.25 for each parasite (red dot in Figure S5B), for a total infection intensity of 0.5, which is less than 1. Thus, splitting time between two environments results in a lower infection intensity than only visiting a single environment and migration is favored.

Our model also builds on past theoretical work on host migration and parasite infection. Shaw et al. (2018) found that migration can be beneficial even when it increases host exposure to novel parasites. However, Shaw et al. (2018) considered infection as a binary (susceptible vs infected) whereas we consider infection intensity (i.e., number of parasites in an infected host). Balstad et al. (2020) also found

that whether or not hosts are favored to migrate depends on the timing of migration and on the rate of infection accumulation. However, they only considered saturating parasite accumulation functions and parasites in a single environment (Balstad et al. 2020). We expand on this work by considering different parasite accumulation functions and parasites in multiple environments. In doing so, we can better capture nuances in migratory decisions that animals must make in light of increasing anthropogenic pressures.

Future directions

The modelling framework described here could be expanded in future studies in three different ways. First, one could test the robustness of our results by changing an assumption to see whether the results stay the same or change, thus identifying a factor that mediates our findings. To start, one could use a different infection accumulation function where the maximal infection differs between parasites (rather than assumed to be equal as we did). Along similar lines, one could consider differential effects of parasites on host fitness. Indeed, in nature, different parasites likely have distinct transmission rates and virulence in their hosts. In both of these cases, one could look at tradeoffs, e.g., how much of a fitness difference between parasites is needed to counterbalance a difference in rate of accumulation between parasites? Second, one could explore the effects of adding different biological concepts to the model. For instance, interactions between parasite species can influence patterns of parasite establishment in hosts similar to the way priority effects influence succession dynamics in a habitat (Dallas et al. 2019). For example, Hoverman et al. (2013) found reduced infection success of the trematode *Ribeiroia ondatrae* in larval Pacific chorus frog *Pseudacris regilla* when hosts were first infected with a competing trematode species *Echinostoma trivolvis*. In contrast, Halliday et al. (2020) found that Ribwort plantain (*Plantago lanceolata)* infected with one strain of a fungal parasite *Podosphaera plantaginis* were more likely to be infected with other strains suggesting facilitation among the fungal parasites. Furthermore, additional mechanisms for how host migration and infection interact such as migratory recovery or migratory

418 stalling (i.e. interrupted migration due to parasite-induced costs; Peacock et al. 2018) could be included in future models to explore how changes to infection accumulation curves impact migratory
420 behaviors. Third, one could ask different but related questions. For example, while we focused on dynamics during a single year, future work could extend this model to explore how migration timing
422 plays out across multiple years, especially if parasite abundance in the environment or host tolerance to infection varies from year to year (Marcogliese 2016, McNew et al. 2019). Furthermore, one could
424 explore how organisms obtain the information used to make their migratory decisions. For example, what is the optimal piece of information needed to inform migratory timing decisions? One could also ask how
426 much certainty hosts should acquire about environment B before deciding to migrate there. Alternatively, it might be interesting to investigate whether there are cases where environment A is so costly in terms of
428 infection that hosts should always migrate to environment B regardless of the conditions there. Future work along these three different avenues would improve our ability to understand and predict the
430 consequences of altered migration behavior under ongoing anthropogenic changes.

References

Albery, G. F., A. Z. Hasik, S. Morris, A. Morris, F. Kenyon, D. McBean, J. M. Pemberton, D. H. Nussey, and J. A. Firth. 2024. Divergent age-related changes in parasite infection occur independently of behaviour and demography in a wild ungulate. Philosophical Transactions of the Royal Society B: Biological Sciences 379:20230508.

Altizer, S., R. Bartel, and B. A. Han. 2011. Animal migration and infectious disease risk. Science 331:296–302.

Balstad, L. J., S. A. Binning, M. E. Craft, M. Zuk, and A. K. Shaw. 2020. Parasite intensity and the evolution of migratory behavior. Ecology 102:e03229.

Benesh, D. P. 2011. Intensity-dependent host mortality: what can it tell us about larval growth strategies in complex life cycle helminths? Parasitology 138:913–925.

Binning, S. A., M. E. Craft, M. Zuk, and A. K. Shaw. 2022. How to study parasites and host migration: a roadmap for empiricists. Biological Reviews 97:1161–1178.

Birkeland, K., and P. J. Jakobsen. 1997. Salmon lice, *Lepeophtheirus salmonis*, infestation as a causal agent of premature return to rivers and estuaries by sea trout, *Salmo trutta*, juveniles. Environmental Biology of Fishes 49:129–137.

Bischof, R., L. E. Loe, E. L. Meisingset, B. Zimmermann, B. Van Moorter, and A. Mysterud. 2012. A migratory northern ungulate in the pursuit of spring: jumping or surfing the green wave? The American Naturalist 180:407–424.

Cable, J., I. Barber, B. Boag, A. R. Ellison, E. R. Morgan, K. Murray, E. L. Pascoe, S. M. Sait, A. J. Wilson, and M. Booth. 2017. Global change, parasite transmission and disease control: lessons from ecology. Philosophical Transactions of the Royal Society B: Biological Sciences 372:20160088.

Chauveau, V., M. Garel, C. Toïgo, P. Anderwald, M. Apollonio, B. Bassano, M. Beurier, M. Bouche, A. Brambilla, F. Brivio, Y. Bunz, F. Cagnacci, M. Canut, J. Cavailhes, I. Champly, F.

Filli, A. Frey-Roos, G. Gressmann, S. Grignolio, I. Herfindal, F. Jurgeit, L. Martinelli, L. Maurino, R. Papet, E. Petit, M. Ramanzin, P. Semenzato, E. Vannard, A. Coulon, A. Loison, and P. Marchand. 2025. It's time to go—Drivers and plasticity of migration phenology in a short-distance migratory ungulate. Journal of Animal Ecology 94:1204–1220.

Cooke, S. J., M. L. Piczak, N. J. Singh, S. Åkesson, A. T. Ford, S. Chowdhury, G. W. Mitchell, D. R. Norris, M. Hardesty-Moore, D. McCauley, N. Hammerschlag, M. A. Tucker, J. J. Horns, R. R. Reisinger, V. Kubelka, and R. J. Lennox. 2024. Animal migration in the Anthropocene: threats and mitigation options. Biological Reviews 99:1242–1260.

Dallas, T. A., A.-L. Laine, and O. Ovaskainen. 2019. Detecting parasite associations within multi-species host and parasite communities. Proceedings of the Royal Society B: Biological Sciences 286:20191109.

Davenport, E. S., M. K. Dziuba, L. E. Jacobson, S. K. Calhoun, K. J. Monell, and M. A. Duffy. 2024. How does parasite environmental transmission stage concentration change before, during, and after disease outbreaks? Ecology 105:e4235.

Daversa, D. R., A. Fenton, A. I. Dell, T. W. J. Garner, and A. Manica. 2017. Infections on the move: how transient phases of host movement influence disease spread. Proceedings of the Royal Society B: Biological Sciences 284:20171807.

Dingle, H. 2014. Migration: The Biology of Life on the Move. Oxford University Press.

Dingle, H., and V. A. Drake. 2007. What Is Migration? BioScience 57:113–121.

Dossman, B. C., A. D. Rodewald, C. E. Studds, and P. P. Marra. 2023. Migratory birds with delayed spring departure migrate faster but pay the costs. Ecology 104:e3938.

Duerr, H. P., K. Dietz, and M. Eichner. 2003. On the interpretation of age–intensity profiles and dispersion patterns in parasitological surveys. Parasitology 126:87–101.

Figuerola, J., and A. J. Green. 2000. Haematozoan parasites and migratory behaviour in waterfowl. Evolutionary Ecology 14:143–153.

Gallagher, A. J., S. Creel, R. P. Wilson, and S. J. Cooke. 2017. Energy landscapes and the

landscape of fear. Trends in Ecology & Evolution 32:88–96.

Hall, R. J., L. M. Brown, and S. Altizer. 2016. Modeling vector-borne disease risk in migratory animals under climate change. Integrative and Comparative Biology 56:353–364.

Halliday, F. W., R. M. Penczykowski, B. Barrès, J. L. Eck, E. Numminen, and A.-L. Laine. 2020. Facilitative priority effects drive parasite assembly under coinfection. Nature Ecology & Evolution 4:1510–1521.

Hoverman, J. T., B. J. Hoye, and P. T. J. Johnson. 2013. Does timing matter? How priority effects influence the outcome of parasite interactions within hosts. Oecologia 173:1471–1480.

Jensen, J. L. W. V. 1906. Sur les fonctions convexes et les inégalités entre les valeurs moyennes. Acta mathematica 30:175–193.

Jones, J. D., M. J. Kauffman, K. L. Monteith, B. M. Scurlock, S. E. Albeke, and P. C. Cross. 2014. Supplemental feeding alters migration of a temperate ungulate. Ecological Applications 24:1769–1779.

Karvonen, A., P. J. Hudson, O. Seppälä, and E. T. Valtonen. 2004. Transmission dynamics of a trematode parasite: exposure, acquired resistance and parasite aggregation. Parasitology Research 92:183–188.

Kokko, H. 1999. Competition for early arrival in migratory birds. Journal of Animal Ecology 68:940–950.

Koprivnikar, J., and T. L. F. Leung. 2015. Flying with diverse passengers: greater richness of parasitic nematodes in migratory birds. Oikos 124:399–405.

Kuletz, K. J., S. H. Ferguson, M. Frederiksen, C. P. Gallagher, D. D. W. Hauser, H. Hop, K. M. Kovacs, C. Lydersen, A. Mosbech, and A. C. Seitz. 2024. A review of climate change impacts on migration patterns of marine vertebrates in Arctic and Subarctic ecosystems. Frontiers in Environmental Science 12:1434549.

Laforge, M. P., E. Vander Wal, Q. M. R. Webber, C. Geremia, M. J. Kauffman, D. E. McWhirter, A. Middleton, T. W. Mong, K. L. Monteith, A. C. Ortega, H. Sawyer, and J. A. Merkle. 2025.

Consistent individual differences and plasticity in migration behaviour of three North American ungulates. Ecology Letters 28:e70101.

Lewin, P. J., J. Wynn, J. M. Arcos, R. E. Austin, J. Blagrove, S. Bond, G. Carrasco, K. Delord, L. Fisher-Reeves, D. García, N. Gillies, T. Guilford, I. Hawkins, P. Jaggers, C. Kirk, M. Louzao, L. Maurice, M. McMinn, T. Micol, J. Morford, G. Morgan, J. Moss, E. M. Riera, A. Rodriguez, K. Siddiqi-Davies, H. Weimerskirch, R. B. Wynn, and O. Padget. 2024. Climate change drives migratory range shift via individual plasticity in shearwaters. Proceedings of the National Academy of Sciences 121:e2312438121.

Lisovski, S., B. J. Hoye, J. R. Conklin, P. F. Battley, R. A. Fuller, K. B. Gosbell, M. Klaassen, C. Benjamin Lee, N. J. Murray, and S. Bauer. 2024. Predicting resilience of migratory birds to environmental change. Proceedings of the National Academy of Sciences 121:e2311146121.

López-Ramírez, S., J.-M. García-Carrasco, R. Real, and A.-R. Muñoz. 2025. Climate-driven shifts in trans-Saharan bird migration illustrated by the spotted flycatcher (*Muscicapa striata*). Scientific Reports 15:35201.

Marcogliese, D. J. 2016. The Distribution and Abundance of Parasites in Aquatic Ecosystems in a Changing Climate: More than Just Temperature. Integrative and Comparative Biology 56:611–619.

McNew, S. M., S. A. Knutie, G. B. Goodman, A. Theodosopoulos, A. Saulsberry, J. Yépez R., S. E. Bush, and D. H. Clayton. 2019. Annual environmental variation influences host tolerance to parasites. Proceedings of the Royal Society B: Biological Sciences 286:20190049.

Middleton, A. D., J. A. Merkle, D. E. McWhirter, J. G. Cook, R. C. Cook, P. J. White, and M. J. Kauffman. 2018. Green-wave surfing increases fat gain in a migratory ungulate. Oikos 127:1060–1068.

Mignatti, A., B. Boag, and I. M. Cattadori. 2016. Host immunity shapes the impact of climate changes on the dynamics of parasite infections. Proceedings of the National Academy of Sciences 113:2970–2975.

Mo, T. A. 1997. Seasonal occurrence of *Gyrodactylus derjavini* (Monogenea) on brown trout, *Salmo trutta*, and Atlantic salmon, *S. salar*, in the Sandvikselva river, Norway. The Journal of Parasitology 83:1025–1029.

Peacock, S. J., J. Bouhours, M. A. Lewis, and P. K. Molnár. 2018. Macroparasite dynamics of migratory host populations. Theoretical Population Biology 120:29–41.

Peacock, S. J., M. Krkošek, M. A. Lewis, and P. K. Molnár. 2020. A unifying framework for the transient parasite dynamics of migratory hosts. Proceedings of the National Academy of Sciences 117:10897–10903.

Poulin, R., and D. de Angeli Dutra. 2021. Animal migrations and parasitism: reciprocal effects within a unified framework. Biological Reviews 96:1331–1348.

Puleo, L., F. Huang, M. Stager, and N. R. Senner. 2025. Flexibility in the face of climate change? A rapid and dramatic shift towards later spring migration in Hudsonian godwits (*Limosa haemastica*). Proceedings of the Royal Society B: Biological Sciences 292:20250982.

Reiczigel, J., M. Marozzi, I. Fábián, and L. Rózsa. 2019. Biostatistics for Parasitologists – A Primer to Quantitative Parasitology. Trends in Parasitology 35:277–281.

Rickbeil, G. J. M., J. A. Merkle, G. Anderson, M. P. Atwood, J. P. Beckmann, E. K. Cole, A. B. Courtemanch, S. Dewey, D. D. Gustine, M. J. Kauffman, D. E. McWhirter, T. Mong, K. Proffitt, P. J. White, and A. D. Middleton. 2019. Plasticity in elk migration timing is a response to changing environmental conditions. Global Change Biology 25:2368–2381.

Rittenhouse, T. A. G., R. D. Semlitsch, and F. R. Thompson. 2009. Survival costs associated with wood frog breeding migrations: effects of timber harvest and drought. Ecology 90:1620–1630.

Ruel, J. J., and M. P. Ayres. 1999. Jensen's inequality predicts effects of environmental variation. Trends in Ecology & Evolution 14:361–366.

Serra-Llinares, R. M., T. Bøhn, Ø. Karlsen, R. Nilsen, C. Freitas, J. Albretsen, T. Haraldstad, E. Thorstad, K. Elvik, and P. Bjørn. 2020. Impacts of salmon lice on mortality, marine migration distance and premature return in sea trout. Marine Ecology Progress Series 635:151–168.

Servedio, M. R., Y. Brandvain, S. Dhole, C. L. Fitzpatrick, E. E. Goldberg, C. A. Stern, J. V. Cleve, and D. J. Yeh. 2014. Not just a theory—The utility of mathematical models in evolutionary biology. PLOS Biology 12:e1002017.

Shaw, A. K., and S. A. Binning. 2020. Recovery from infection is more likely to favour the evolution of migration than social escape from infection. Journal of Animal Ecology 89:1448–1457.

Shaw, A. K., M. Levet, and S. A. Binning. 2023. A unified evolutionary framework for understanding parasite infection and host migratory behaviour. Ecology Letters 26:1987–2002.

Shaw, A. K., J. Sherman, F. K. Barker, and M. Zuk. 2018. Metrics matter: the effect of parasite richness, intensity and prevalence on the evolution of host migration. Proceedings of the Royal Society B: Biological Sciences 285:20182147.

Silva, L. M., K. C. King, and J. C. Koella. 2025. Dissecting transmission to understand parasite evolution. PLOS Pathogens 21:e1012964.

Smith, R. J., J. M. Graham, M. I. Hatch, E. Lasek-Nesselquist, and A. M. Royer. 2026. Multi-year change and annual variation in fall migratory timing of songbirds captured across a 19-year time span in northeastern Pennsylvania, USA. Journal of Field Ornithology 97:6.

Teitelbaum, C. S., S. Huang, R. J. Hall, and S. Altizer. 2018. Migratory behaviour predicts greater parasite diversity in ungulates. Proceedings of the Royal Society B: Biological Sciences 285:20180089.

Tolvanen, A., H. Routavaara, M. Jokikokko, and P. Rana. 2023. How far are birds, bats, and terrestrial mammals displaced from onshore wind power development? – A systematic review. Biological Conservation 288:110382.

Torstenson, M., and A. K. Shaw. 2025. Strength of seasonality and type of migratory cue determine the fitness consequences of changing phenology for migratory animals. Oikos 2025:e10862.

van Weelden, C., J. R. Towers, and T. Bosker. 2021. Impacts of climate change on cetacean

distribution, habitat and migration. Climate Change Ecology 1:100009.

Weinstein, S. B., J. C. Buck, and H. S. Young. 2018. A landscape of disgust. Science 359:1213–1214.

White, P. J., and R. A. Garrott. 2005. Northern Yellowstone elk after wolf restoration. Wildlife Society Bulletin 33:942–955.

Wikelski, M., E. M. Tarlow, A. Raim, R. H. Diehl, R. P. Larkin, and G. H. Visser. 2003. Costs of migration in free-flying songbirds. Nature 423:704–704.

Wilcove, D. S., and M. Wikelski. 2008. Going, going, gone: Is animal migration disappearing? PLOS Biology 6:e188.

Xu, W., K. Barker, A. Shawler, A. Van Scoyoc, J. A. Smith, T. Mueller, H. Sawyer, C. Andreozzi, O. R. Bidder, H. Karandikar, S. Mumme, E. Templin, and A. D. Middleton. 2021. The plasticity of ungulate migration in a changing world. Ecology 102:e03293.

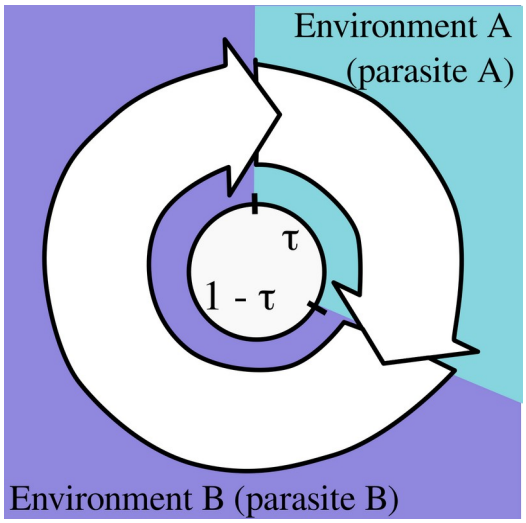

Figure S1. Hosts spend the first part of the year (of length τ) in Environment A where they can potentially accumulate Parasite A. Hosts then migrate to Environment B for the remainder of the year (1 – τ), where they potentially accumulate Parasite B. The model determines what the best (optimal) value of τ is.

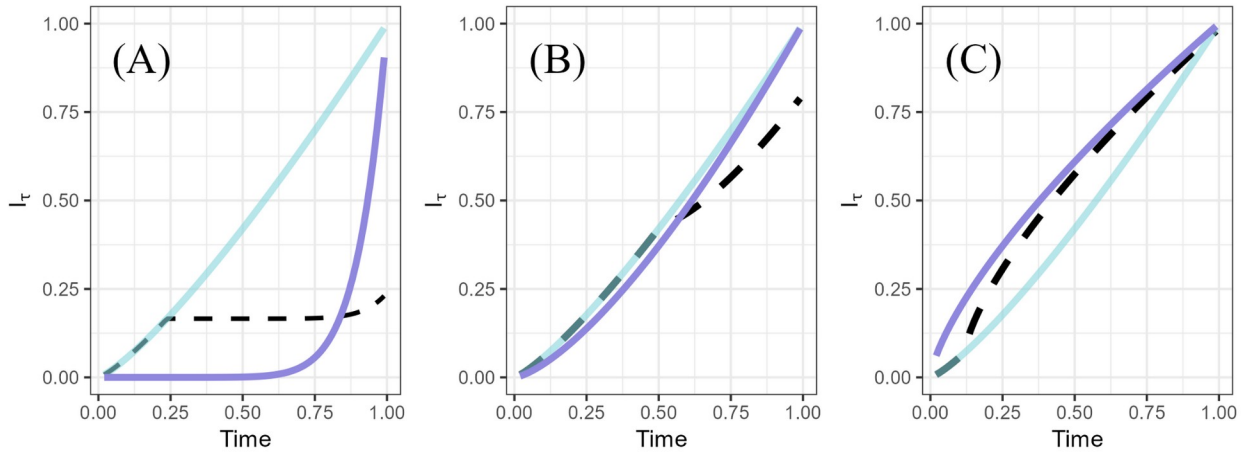

Figure S2. Infection intensity over time for hosts that spend all of their time in environment A (blue solid lines), all of their time in environment B (purple solid lines), or use the optimal migration strategy (black dashed lines), for $\delta_A = 0.8$ and three different sets of B parasite accumulation parameters: (A) $\delta_B = 0.1$, (b) $\delta_B = 0.7$, and (C) $\delta_B = 1.4$.

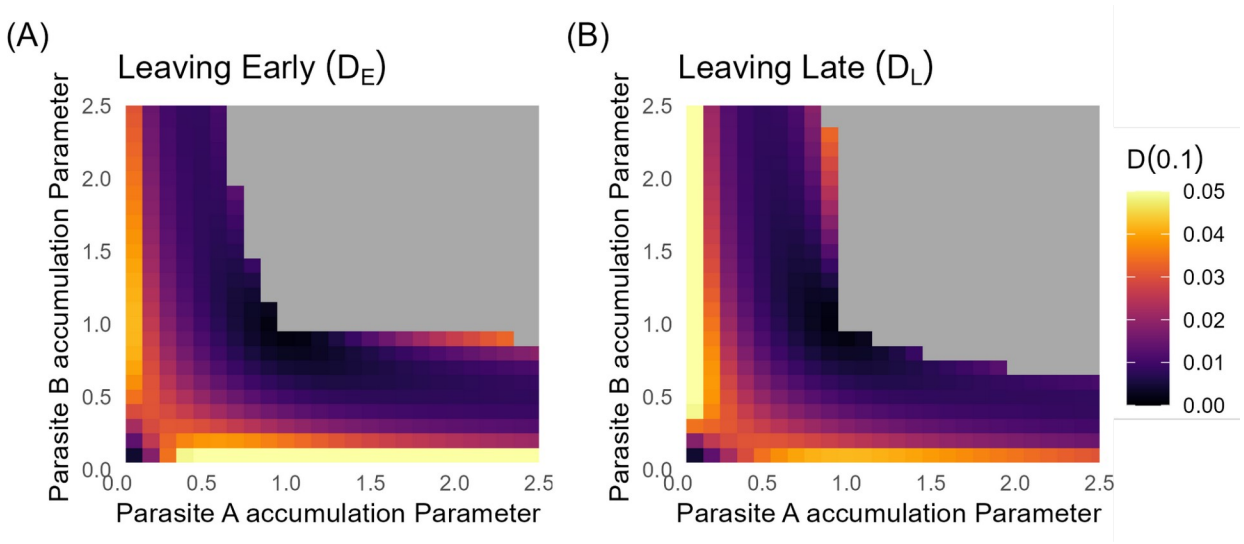

Figure S3. How bad is leaving environment A early versus leaving environment A late from an infection perspective? The cost (in terms of increased infection intensity) of migrating (A) 10% of the year too early, $D_E(0.1)$ or (B) 10% of the year too late $D_L(0.1)$, compared to migrating with optimal (τ^*) as a function of the infection accumulation parameter for parasite A (x-axis, δ_A) and parasite B (y-axis, δ_B), ranging from 0 to 2.5 in 0.1 increments. Gray represents cases where there are multiple optional strategies. See Figure 4 with a 5% error for comparison.

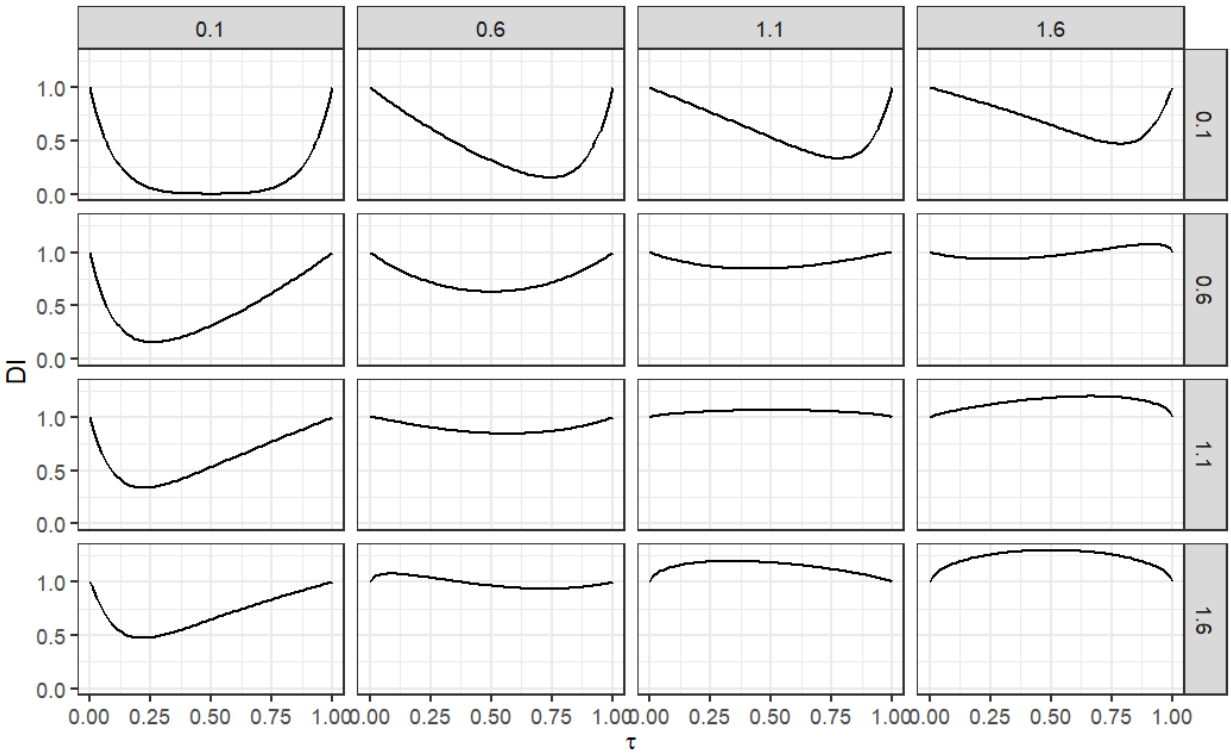

Figure S4. The cost (in terms of increased infection intensity; y-axis) of migrating at a different time (τ; x-axis) than the optimal τ^* (the lowest point of each graph) as a function of the infection accumulation parameter for parasite A (horizontally across panels, δ_A) and parasite B (vertically across panels, δ_B).

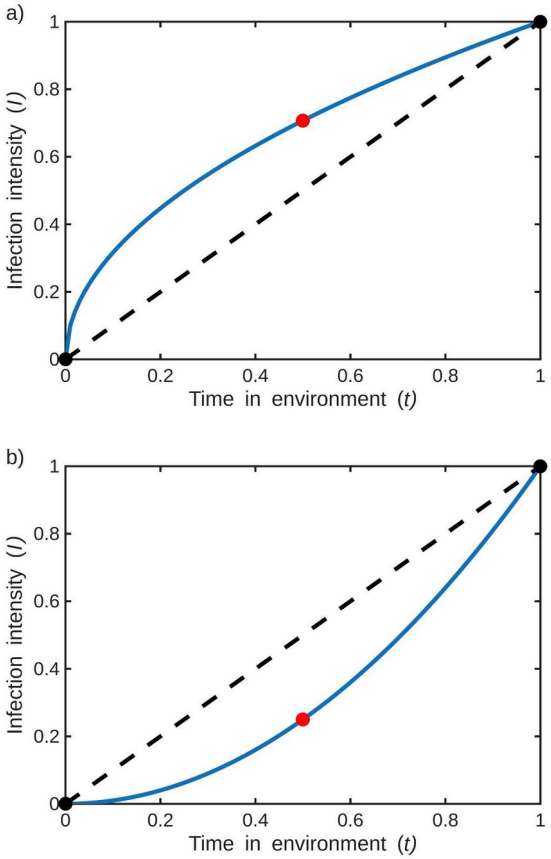

Figure S5. Examples of a parasite accumulation functions that is (a) concave (saturating) and (b) convex (accelerating), In each panel the blue line shows the parasite accumulation function, the black dots show infection intensity resulting from a non-migrating strategy (with the dashed line showing the average between the two), and the red dot shows the infection intensity resulting from a migratory strategy with τ=0.5.

Table S1. Model parameters – their notation, meaning, and values considered.

Notation	**Meaning**	**Possible value(s)**
τ	Fraction of the year spent in environment A (breeding)	$0 \leq \tau \leq 1$
τ^*	Optimal value of τ	$0 \leq \tau^* \leq 1$
δ_A	Accumulation parameter for parasite *A*	$0.1 \leq \delta_A \leq 2.5$
δ_B	Accumulation parameter for parasite *B*	$0.1 \leq \delta_B \leq 2.5$
I_A	Infection intensity for parasite *A* [eqn. 1]	$0 \leq I_A \leq 1$
I_B	Infection intensity for parasite *B* [eqn. 2]	$0 \leq I_B \leq 1$
I_τ	Total infection intensity (both parasites summed) for an individual using strategy τ [eqn. 3]	$0 \leq I_\tau \leq 2$
I_τ^*	Minimal value of I_τ (the one corresponding to τ^*)	$0 \leq I_\tau^* \leq 1$
D_R	Difference in infection intensity for a resident (non-migrating host) compared to a host migrating with τ^* [eqn. 4]	$0 \leq D_R \leq 1$
ε	Error in migration timing of hosts (in terms of proportion of the year)	0.05, 0.1
$D_E(x)$	Difference in infection intensity for host migrating ε early compared to a host migrating with τ^* [eqn. 5]	$0 \leq D_E(x) \leq 1$
$D_L(x)$	Difference in infection intensity for host migrating ε late compared to a host migrating with τ^* [eqn. 6]	$0 \leq D_L(x) \leq 1$