A unifying framework for the transient parasite dynamics of migratory hosts

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Migrations allow animals to track seasonal changes in resources, find mates, and avoid harsh climates, but these regular, long-distance movements also have implications for parasite dynamics and animal health. Migratory animals have been dubbed "super-spreaders" of infection, but migration can also reduce parasite burdens within host populations via migratory escape from contaminated habitats and transmission hotspots, migratory recovery due to parasite mortality, and migratory culling of infected individuals. Here, we show that a single migratory host–macroparasite model can give rise to these different phenomena under different parametrizations, providing a unifying framework for a mechanistic understanding of the parasite dynamics of migratory animals. Importantly, our model includes the impact of parasite burden on host movement capability during migration, which can lead to "parasite-induced migratory stalling" due to a positive feedback between increasing parasite burdens and reduced movement. Our results provide general insight into the conditions leading to different health outcomes in migratory wildlife. Our approach lays the foundation for tactical models that can help understand, predict, and mitigate future changes of disease risk in migratory wildlife that may arise from shifting migratory patterns, loss of migratory behavior, or climate effects on parasite development, mortality, and transmission.

migration | wildlife health | host–parasite | population dynamics

The interactions between animals and their parasites can be profoundly affected by host movement. The mass movement of entire populations that occurs in migratory animals, in particular, can have substantial consequences on parasitism levels and the health of the hosts (1, 2), but the underlying dynamics are complex and difficult to disentangle. Multiple, nonexclusive mechanisms are simultaneously at play—of which some can amplify the impacts of parasitism (3) while others can benefit host health (1, 4). Migrations can compromise host immune systems (5, 6), expose hosts to new pathogens, increase host densities and transmission rates, and spread parasites to uninfected populations (3). Conversely, "migratory escape" from parasitism can occur when hosts move away from habitats where parasites have accumulated, such as breeding and overwintering grounds (7–10). Migratory escape should result in declining parasite burdens with migration so long as the hosts are able to "outrun" their parasites before reinfection occurs, a process that depends both on migration speed of the host and transmission rate of the parasites. The rate of within-host parasite mortality will affect how quickly migratory hosts "recover" from infection after escape (11). Migratory culling (12) and migratory stalling (13) may also reduce mean parasite burdens throughout the migration, but the mechanisms and population outcomes differ from those for migratory escape: The most heavily parasitized individuals, often at the trailing end of the migration, may not complete the migration (stalling) or die trying to do so (culling), thus resulting in smaller, but overall healthier, populations at the end of the migration. The relative impact of migratory culling and stalling will depend on the strength of nonlethal and lethal effects of parasitism on host mortality and movement, respectively.

Understanding the parameters that give rise to these different parasite-related outcomes in migratory hosts is essential for predicting and managing wildlife health in the face of climate-associated changes in parasite dynamics (14) and shifting migration patterns (15). Disentangling the different mechanisms is difficult in practice because 1) migratory animals often cover vast distances, making it challenging to obtain appropriate data (16); 2) different mechanisms can lead to the same observed patterns in parasite burdens toward the end of a migration (9); and 3) appropriate modeling frameworks for studying spatially dynamic host–parasite interactions have only become available recently (2, 13, 17–19). The development of models and theory to describe the interactions between migratory host and parasite populations is an alternative that can offer deep and generalizable insights into conditions under which we might expect migration to reduce disease risk via escape or culling, or contribute to pathogen spread based on characteristics of the host, parasite, and environment (20). Despite increasing recognition of the diverse effects of host movement on disease dynamics (1) and accumulating empirical examples (9, 10, 21–23), theoretical frameworks for studying disease dynamics during long-distance movement have been lagging (20).

Macroparasites (e.g., helminths, arthropods) are key players in the lives of most animals (24) and are ideally suited for

Significance

When animals migrate, they take their parasites with them—or not. Understanding infectious disease in migratory animals is challenging because the vast distances covered result in variable host densities and infection pressure and make it difficult to collect data. Empirical studies show that migrants may have higher, lower, or the same infection intensity as residents. We present a model that produces different infection patterns and migration outcomes under different parameters, laying a theoretical foundation for exploring what may be driving observed diverse patterns in nature. Our model can help guide empirical studies, suggesting when and where data need to be collected in order to distinguish mechanisms, and inform targeted management and conservation efforts.

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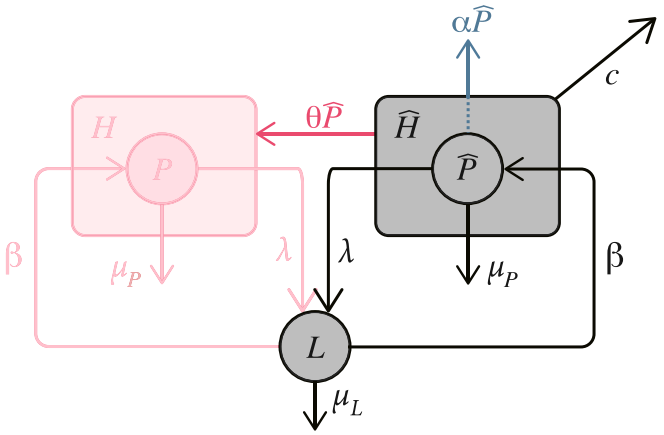

Fig. 1. Schematic of the model used to describe the host–parasite dynamics of migratory wildlife. State variables include the host density $H(x,t)$, mean parasite burden $P(x,t)$, and density of stationary parasite larvae $L(x,t)$, with the hat denoting migrating hosts and their parasites. The basic model ignores host mortality and stopping but includes host movement at speed c (black arrows and gray boxes), and allowed us to focus on migratory escape and recovery from parasitism. We added complexity to this basic model in two ways: 1) including parasite-induced host mortality at per-parasite rate α (blue arrow), to capture the dynamics of migratory culling, and 2) including stationary hosts and parasite-induced stopping at per-parasite rate θ (pink), which led to parasite-induced migratory stalling. Description of other parameters and base values are in Table 1. Model equations are given in *SI Appendix*.

investigating the parasite dynamics of migratory animals: 1) most macroparasites have clearly structured life cycles, often with a free-living stage that slows reinfection to hosts; 2) the parasite burdens of hosts can usually be understood as an emergent property of host and parasite traits, such as the length of the free-living stage and the movement speed of hosts (13); and 3) host performance (22), including survival, movement speed (12), endurance (25), and stamina (26), tends to decline with increasing parasite burden, which represents the intensity of infection. The interactions of migratory animals with their macroparasites have, however, largely been neglected in the literature to date, with most existing models focusing on individual-based or metapopulation models for microparasites (20). In these existing models, susceptible and infected hosts are tracked without consideration of parasite burdens (e.g., refs. 2 and 17–19). Moreover, most models do not explicitly incorporate the movement of animals during migrations, and those that do (i.e., ref. 2) do not consider how parasite burdens and resultant impacts on host survival and movement may vary among individuals within a population.

Host–macroparasite models that consider parasite burdens of migratory animals are more complex than susceptible-infected models because parasite burdens may vary dynamically in both space, even within a migrating population, and time, within individuals and populations. For example, animals at the trailing edge of the migration may be more parasitized than those at the leading edge because followers are exposed to parasites shed by the leaders. This pattern of higher parasite burdens in late migrators has been observed in several species of migratory birds (27, 28), and may be exacerbated if healthy individuals tend to depart earlier and/or parasitism has negative effects on the movement capacity of hosts. The pattern of infection intensity within a host population can affect the host–parasite dynamics and be informative of the mechanisms (e.g., migratory escape or culling) at play.

To better understand the diversity of mechanisms by which parasitism and host movement interact to affect host health, we refined a recently developed partial-differential-equation (PDE) model of spatiotemporal host–macroparasite dynamics (ref. 13 and Fig. 1) to explore conditions under which we might expect migratory escape, migratory culling, and migratory stalling to occur. Unlike previous modeling studies, our approach considers environmentally transmitted macroparasites and how the number of parasites per host (i.e., parasite burden) affects mortality and transmission rates of parasites as well as host movement and survival. We show that the same model can give rise to migratory escape, culling, or stalling depending on the parametrization, thus providing a unifying framework for migratory host and macroparasite dynamics. Our model and results are strategic, providing general insight, but could be adapted and made tactical, for example to address questions regarding the impact of changing parameters on specific host–parasite systems.

Results

We employed variants of a spatial host–parasite model (13) to identify conditions under which wildlife populations are likely to experience migratory escape, migratory culling, or migratory stalling. The base model (Fig. 1, black) considers a migratory host population, $\hat{H}(x,t)$, moving along a one-dimensional migration corridor at a constant speed, c. The parasite burden of migrating hosts, $\hat{P}(x,t)$, decreases as parasites die at per capita rate μ_P, and increases as hosts take up stationary free-living parasite larvae from the environment, $L(x,t)$, at rate β. Infected hosts produce larvae at per-parasite rate λ, and larvae die at per capita rate μ_L. We added complexity to this model in two ways: first, we considered parasite-induced host mortality at per-parasite rate α to understand the dynamics of migratory culling (Fig. 1, blue). Second, we considered the possibility for migrating hosts to stop moving at per-parasite rate θ to understand the dynamics of migratory stalling (Fig. 1, pink). Higher values of α and θ reflect a greater impact of parasitism on host survival and movement, respectively, which may relate to the strenuousness of the migration and thus susceptibility of the host and/or virulence of the parasite.

The model focuses on host–parasite dynamics during migration and ignores host birth, which occurs during a nonmigratory breeding season for many species. In the absence of host birth, nonzero equilibria for parasite burdens and larvae do not exist (29) regardless of the migration speed of hosts (c), transmission

Table 1. Base parameter values used in simulations of the host–parasite model (Fig. 1; see *SI Appendix* for equations)

Symbol	Description	Value in simulations: Escape/Recovery	Culling	Stalling
λ	Parasite production, d^{-1}	0.03	0.03	0.03
μ_L	Free-living parasite larvae death, d^{-1}	0.015	0.015	0.015
β	Uptake of parasite larvae by hosts, d^{-1}	**0.0001–0.022**	**0.0001–0.05**	**0.0001–0.05**
c	Speed of migrating hosts, km d^{-1}	**1–100**	25	25
μ_P	Within-host parasite natural death, d^{-1}	**0.001–0.05**	0.01	0.01
α	Parasite-induced host death, d^{-1}	0	**0–0.003**	0
θ	Per-parasite increase in stopping, d^{-1}	0	0	**0–0.004**

The parameter ranges explored when considering escape, culling, and stalling are shown in bold.

rate of parasites (β), mortality rate of parasites (μ_P), or host mortality due to parasitism (α) (see *SI Appendix* for model equations and analysis). However, the time it takes to reach the parasite-free equilibrium and peak parasite burdens will depend on these parameters, and these transient dynamics may be more relevant for host populations that have a limited migration duration. We considered the transient dynamics over a 365-d migration period, although this timeframe is arbitrary and does not affect the results (i.e., the parameters can be rescaled to any timeframe to produce the same results).

Simulations generally show initial increases in parasite burdens at the location of peak host density (Fig. 2*A*, day 1). As the host population migrates, parasite burdens decline at the leading edge of the migrating host population but increase for hosts at the trailing end of the migrating host population, such that the spatial distribution of parasite burdens within the migrating host population forms a slowly varying traveling wave (Fig. 2*A*, day 80). The front of this wave gradually shifts backward within the host population as individuals at the leading edge are not exposed to new infections. Eventually, the parasite burden at the trailing edge will also decline (Fig. 2*A*, day 360). We summarized these dynamics using three metrics: 1) the time until mean parasite burdens peaked, $t(\overline{P}_{\max})$; 2) the time until parasite burdens declined to initial levels, $t(\overline{P}_0)$; and 3) the peak mean parasite burden among migrating hosts, $\overline{P}_{\max}$ (Fig. 2*B*). These three metrics are all positively related (it will take longer to reach a higher peak mean parasite burden) but have different practical implications and thus we discuss them all. For models that included host mortality or stopping, we also included the fraction of hosts that were alive and migrating at the time when parasite burdens have declined again to the initial conditions, $\hat{f}(t(\overline{P}_0)) = \int \hat{H}(x,\ t(\overline{P}_0))\,dx / \int \left\{\hat{H}(x,\ t(\overline{P}_0)) + H(x,\ t(\overline{P}_0))\right\} dx$.

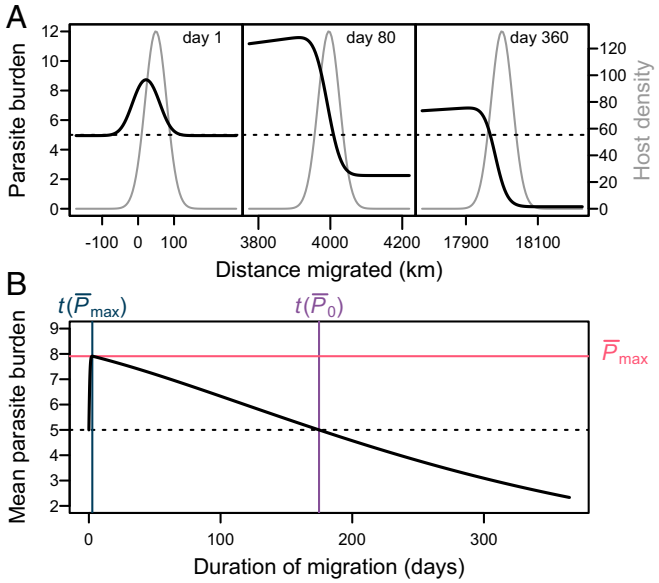

Fig. 2. (*A*) Simulation of migratory escape using the basic model (Fig. 1), where the host population (gray line) is initially Gaussian distributed around $x = 0$ at $t = 0$ d and migrates at $c = 50$ km d^{-1}. The parasite burden (black line) is initially $P(x,t_0) = 5$ parasites host^{-1} (horizontal dotted line). The mean parasite burden across all migrating hosts, P, is the convolution of the parasite burden (black line) and the host density (normalized to integrate to 1). (*B*) The mean parasite burden initially increased but then declined with increasing duration of migration. We capture these dynamics using three metrics: 1) the time to peak mean parasite burden, $t(P_{\max})$ (vertical blue line), 2) time until parasite burdens declined to initial, $t(P_0)$ (vertical purple line), and the peak mean parasite burden, $P_{\max}$ (horizontal pink line). Other parameters in this simulation were $\lambda = 0.03$ d^{-1}, $\mu_L = 0.015$ d^{-1}, $\mu_P = 0.01$ d^{-1}, and $\beta = 0.004$ d^{-1}.

Migratory Escape. Under the basic model, it took longer to reach peak parasite burdens when the transmission rate was high, migration speed was low, and/or within-host parasite mortality was low (Fig. 3*A*). At low parasite mortality ($\mu_P = 0.001$ d^{-1} in our simulations), migratory escape was impossible within a year for all but the lowest transmission rates ($\beta = 0.0001$ d^{-1}, Fig. 3 *A* and *B*). At high parasite mortality ($\mu_P = 0.05$ d^{-1}), parasite burdens began to decline within 10 d (Fig. 3*A*) and declined below initial burdens within 54 d (Fig. 3*B*) for all values of transmission rate and migration speed that we investigated. Lower migration speeds and/or faster transmission rates also led to higher peak mean parasite burdens (Fig. 3*C*).

Migratory Culling. The addition of parasite-induced host mortality (α) to the model greatly reduced the migration time required for parasite burdens to decline and resulted in much lower peak parasite burdens among migrating hosts. The number of days until parasite burdens declined or reached initial values was more sensitive to changes in α than to changes in β (Fig. 4 *A* and *C*), except at very low transmission rates. At low rates of parasite-induced host mortality, the time needed to escape increased with increasing transmission rates up to $\beta = 0.01$ d^{-1}, consistent with the general pattern of higher transmission leading to higher parasite burdens and thus longer times to escape. However, as transmission rates increased from $\beta = 0.01$ d^{-1} to 0.05 d^{-1}, it took less time for parasite burdens to decline to initial levels (Fig. 4*C*) because relatively high parasite burdens (Fig. 4*E*) accelerated the mortality of heavily parasitized hosts (Fig. 4*G*). The proportion of hosts alive at any given point in time decreased with increasing parasite-induced mortality (*SI Appendix*, Fig. S2 *B–G*). However, because parasite burdens declined to initial levels faster under high parasite-induced mortality (Fig. 4*C*), the proportion of hosts alive (i.e., migrating) at that time actually increased with increasing parasite-induced mortality at moderate to high transmission rates (Fig. 4*G*).

Migratory Stalling. When we incorporated parasite-induced stopping of migrating hosts (θ), the time until parasite burdens declined was also reduced, as with parasite-induced mortality (Fig. 4 *B* and *D*). However, even at low values of θ, a small fraction of hosts was still migrating when their parasite burdens declined below initial values (Fig. 4*H*) when compared to the fraction still migrating (i.e., still alive) under the migratory culling scenario (Fig. 4*G*). At the same θ, a larger fraction of the host population ceased migrating as β increased because the peak mean parasite burden was higher (Fig. 5). For hosts that stopped migrating, parasite burdens increased through the 365-d simulation for all but the lowest values of β (Fig. 5*A*).

Discussion

Multiple simultaneously acting mechanisms make it difficult to unravel the host–parasite dynamics of migrating wildlife. Empirical examples exist for migrants with higher (23, 30, 31), lower (9, 10, 16), and similar (32) parasite burdens as resident hosts, and our analyses suggest that such seemingly idiosyncratic patterns could in fact reflect systematic variation of how key parameters determining the host–parasite dynamics balance against one another. In this paper, we present a unifying modeling framework for describing the multiple mechanisms by which migration can affect macroparasite transmission and vice versa. Our framework includes the spatial infection dynamics during migration and the impact of infection intensity on host survival and movement. This allowed us to show that migratory escape, culling, and stalling may all be different sides of the same

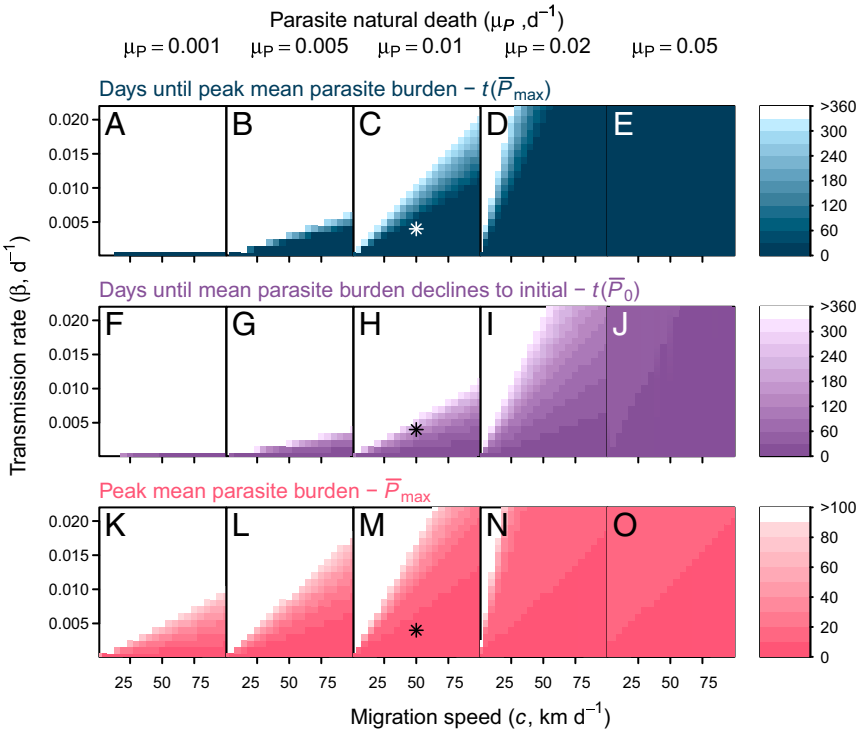

Fig. 3. The transient dynamics under the base model and "escape" parametrization (Table 1) over a 365-d period, summarized as the days until peak parasite burdens (blue), the days until parasite burdens decline to initial (purple), and the peak mean parasite burden (pink, Fig. 2). Each metric is shown over increasing migration speed of hosts (x axis), transmission rate of parasites (y axis), and mortality rate of parasites (left to right panels). The asterisk indicates parameter values for the simulation shown in Fig. 2.

coin, arising from the same model depending on characteristics of the host, the parasite, and the environment.

We have shown that, in theory, complete migratory escape from parasites (i.e., zero parasite burden for all hosts) will always occur if a migrating population traverses uninfested habitats for a long enough time, because the leading individuals will not be exposed to new infections. With nonzero parasite mortality, these leading individuals will lose their infections and cease to shed parasites to infect the individuals behind. Thus, the parasite front will gradually shift backward in the host population until all hosts are parasite-free. In mathematical terms, the long-term equilibrium of our model is unequivocally a mean parasite burden of zero. In nomadic species or those with long and variable migration routes, complete escape as such may indeed occur [although nomadic animals are more likely to encounter a diversity of parasites and thus may have higher parasite richness due to "environmental sampling" (4, 23)]. However, our simulations suggest that for many species that undergo seasonal migration, complete escape from parasites would likely take much longer than the duration of migration. For seasonal migrants, it is more relevant to consider the transient dynamics over the course of the migration, such as the peak parasite burden and the time that it takes for parasite burdens to drop below the initial level (i.e., the "time to escape"). Our simulations showed that both these metrics increase with increasing parasite transmission rate but decrease with faster migration speeds and increasing parasite mortality rates. In practical terms, this means it is more difficult for hosts to outrun parasites that have long-lived adult parasite stages within the host and short environmental transmission stages relative to the time it takes for trailing individuals to pass larvae laid down by the leading individuals of a host population (which in turn is determined by the movement speed and spatial spread of the hosts).

Perhaps counterintuitively, migratory escape becomes easier for the host population when high parasite burdens are lethal or have sublethal effects on host movement. When heavily parasitized hosts cease to migrate (either due to stalling or death), their parasites are also removed from the migrating host population, thus reducing transmission and reinfection. As such, both migratory culling and migratory stalling can improve overall host population health, but this comes at the expense of smaller and/or more fragmented populations. For individual hosts that are left behind, separation from the migratory group may undermine other benefits of group living, increasing the susceptibility of hosts to other forms of mortality such as predation (33) and decreasing the benefits that migration itself conferred such as mating opportunities and favorable environmental conditions for growth and reproduction. Whether or not culling and stalling benefit the host population as a whole thus depends on the strengths of these processes (i.e., the magnitude of parameters for parasite-induced mortality and stalling), as well as on whether host numbers and cohesive populations are more valuable than overall population health (which may, for example, be the case for long-lived species with slow population growth rates). Indeed, if the impact of parasites on movement capacity is high enough, strong feedback loops between parasitism-induced slow movement and increased parasite exposure due to slow movement, can result in entire host populations stalling in infection hotspots, with cascading implications for both host population health and ecosystems missing the migrants.

The potential for parasite-induced migratory stalling may be greatest among species that rely on group cohesion for foraging benefits, predator evasion, or navigational accuracy during long-distance migrations (34). For one, group cohesion may increase host and parasite densities, and thus infection rates, leading to higher parasite burdens. Stalling may occur abruptly if healthy individuals choose to maintain group cohesion with infected

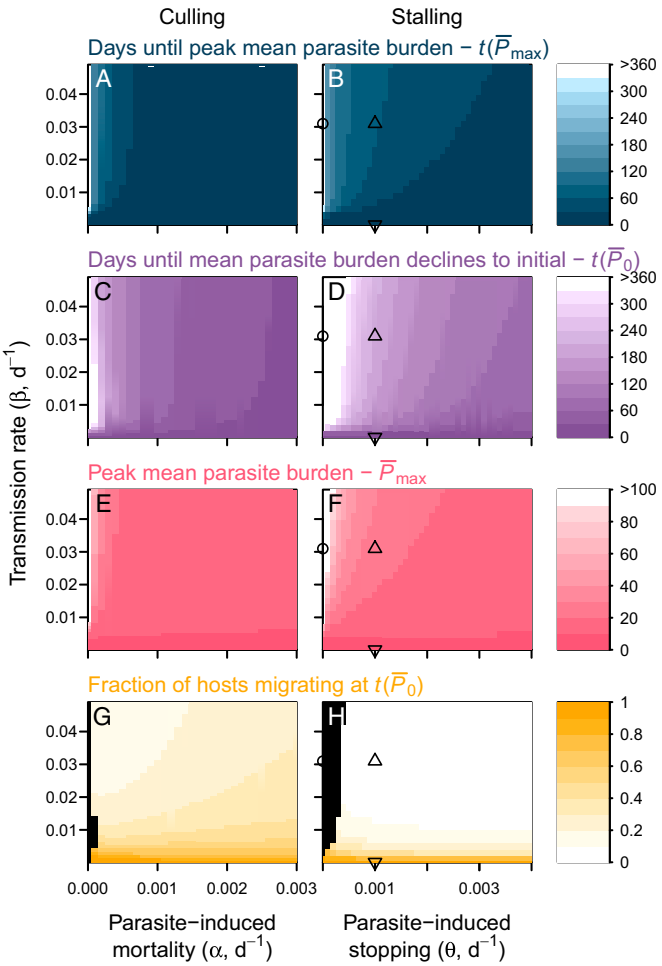

Fig. 4. Transient dynamics of the model over a 365-d period with parasite transmission rates from β = 0–0.05 d^{-1} (*y* axis) and per-parasite rates of host mortality from α = 0–0.003 d^{-1} (*x* axis) in the "migratory culling" scenario (*Left*) or per-parasite rates of host stopping from θ = 0–0.004 d^{-1} in the "migratory stalling" scenario (*Right*). Dynamics are summarized as the days until peak parasite burdens (*A* and *B*, blue), the days until parasite burdens decline to initial (*C* and *D*, purple), the peak mean parasite burden (*E* and *F*, pink), and the fraction of hosts alive and migrating at the time when parasite burdens have declined to initial (*G* and *H*, yellow). Black regions in *G*, *H* are parameter combinations for which the fraction of hosts migrating at $t(\overline{P}_0)$ could not be calculated as parasite burdens did not reach $\overline{P}_0$ within the 365-d simulation (*C* and *D*). Host–parasite dynamics over the 365-d migration are shown in Fig. 5 for parameter combinations indicated by open points in the stalling panels (*B*, *D*, *F*, and *H*).

hosts that have reduced migratory ability in order to reap the benefits of group living. In such cases, there may be a threshold prevalence of infection within the host population, above which stalling would be expected due to the strong behavioral tendency to maintain the flock, herd, or school. Alternately, healthy individuals may avoid contact with parasitized conspecifics (7), in which case fragmentation of the host population may occur at lower parasite burdens than we would expect based on declining movement rates alone. Such avoidance behavior may therefore accelerate migratory stalling, to the benefit of those hosts that escape. The current model does not consider individual movement decisions that may lead to this type of collective behavior, but future research might consider individual-based models in which movement decisions are based on both conspecific distance (35) and parasite burden.

Our analyses indicate that distinguishing between escape, stalling, and culling may not be possible given data from the beginning and end of a migration only, as all mechanisms suggest a declining parasite burden once the peak has been passed. Distinguishing between stalling and culling may be particularly difficult because both mechanisms result in fewer and healthier hosts reaching their destination. Nevertheless, such distinctions can be important because the slowing of movement without actual parasite-induced mortality may have very different consequences for the conservation of host populations, as well as for the persistence of parasites, than parasite-induced culling. Migratory stalling could potentially lead to nonmigratory subpopulations that persist through time, and there are examples of ungulate species that undergo partial migration for which the sedentary groups experience higher parasite burdens than the migratory groups (36, 37). Our framework suggests that in order to distinguish between the various mechanisms affecting migratory host–parasite dynamics, it would be critical to collect data along the migration route, including on host densities, host spread, and parasite burdens (this may be easier for terrestrial migrants than, for example, birds). Linking such data with model predictions for the shifting traveling wave of parasite burdens during a migration, in particular, could help estimate key model parameters and driving mechanisms.

Our models are strategic rather than tactical: intending to illuminate general mechanisms rather than describing any system specifically. Species-specific parameters and additional population dynamics mechanisms are, however, easily incorporated. Adaptations of the model could, for example, be used to explore the role of age–structure in migratory host–parasite dynamics (e.g., newborn caribou calves having lower movement speeds but also leaving the breeding grounds with relatively low parasite burdens, refs. 8 and 14), the role of differing migration strategies (e.g., circular migrations reducing reinfection risks compared to migration routes that are simply reversed seasonally), the role of population dynamics processes that usually occur between migrations (e.g., births), as well as different parasite life cycles and infection strategies (e.g., a requirement of an intermediate host for larval development slowing reinfection and facilitating escape). We have only considered a single host–single parasite system, but the model could be expanded to include multiple parasites, multiple host populations, and/or generalist parasites, allowing for investigations into how migration may affect parasite diversity as well as intensity (4, 23). We also call for the development of microparasite models that explicitly capture the dynamics throughout the migratory period (or transient phase, as described in ref. 2) and impacts of the parasites during the migration, but suspect that similar results would be obtained in that case, albeit for slightly different reasons. For example, we modeled parasite transmission via a free-living larval stage, whereas for many microparasites the direct contact between susceptible and infected individuals is necessary. This could be approximated via an infinitely short free-living stage in our system, or directly modeled in an susceptible-infectious-recovered (SIR) framework. Either way, we suspect that lagging infection waves, with low and high infections at the leading and trailing ends, respectively, would also arise, but now because of the reduced chance of encountering an infected conspecific at the lower density, leading end of a migration.

Conclusions

Wildlife migrations have been on the decline (15, 38) due to a number of factors including anthropogenic resource subsidies that encourage sedentary life histories [e.g., milkweed planting for butterflies (39)] and industrial developments that directly impede migratory pathways [e.g., the building of hydroelectric dams on major salmon-bearing rivers in the US in the 1960s (40)]. The loss of migratory behavior may have dramatic

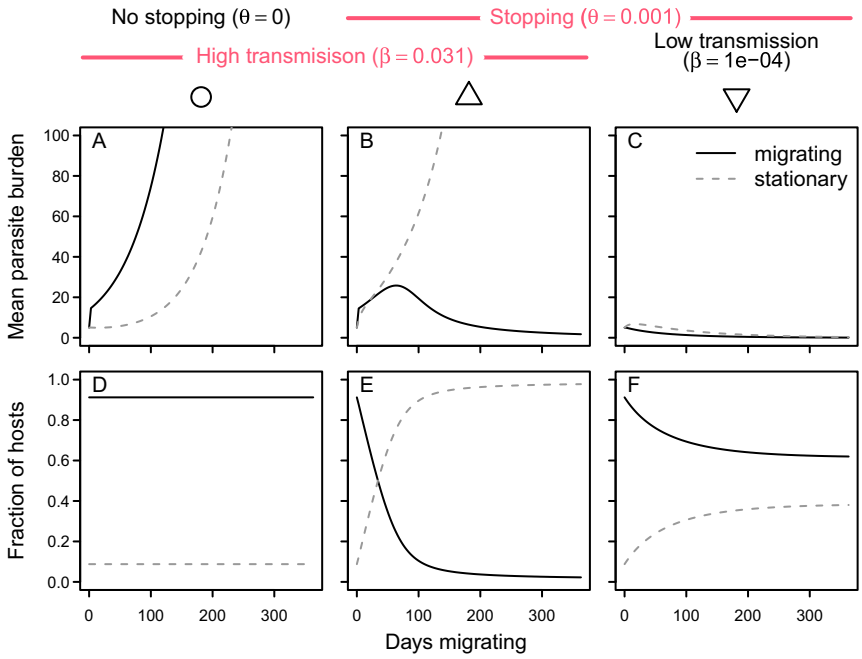

Fig. 5. (*A–C*) The mean parasite burden of migrating (solid black lines) and stationary hosts (dotted gray lines) over a 365-d simulation for three different combinations of parasite-induced stopping (θ) and transmission rate (β) (Fig. 4, *Right*): 1) no stopping and high transmission (circle), 2) stopping and high transmission (up triangle), 3) stopping and low transmission (down triangle). Other parameters are at the "stalling" values in Table 1. (*D–F*) The corresponding fraction of hosts that are migrating (solid black lines) and stationary (dotted gray lines).

consequences for the transmission of parasitic diseases among wildlife, but disentangling the interactions between migration and parasitism has been hindered by the lack of a unifying framework to describe the diverse outcomes observed in nature (1). We have presented a spatial model for migratory host–macroparasite dynamics that incorporates the impact of parasite burdens on host mortality and migratory capabilities and can describe the mechanisms that lead to parasite spread, migratory escape, migratory culling, or migratory stalling. These general insights may help guide empirical studies to differentiate the potential health outcomes for migratory wildlife, and help understand how parasitic diseases will change in the Anthropocene when migration patterns are changing (15), emerging infectious diseases are on the rise (41, 42), and climate change is altering host–parasite dynamics (43, 44).

Materials and Methods

Model. We refined a previously described model of migratory host–macroparasite dynamics (13) to focus on the mechanisms of migratory escape, migratory culling, and migratory stalling. The model consists of seven coupled PDEs (see *SI Appendix* for equations) that track spatial and temporal changes in the density of moving and stationary host populations, the mean parasite burdens of both moving and stationary hosts, the variance-to-mean ratios of the distribution of parasites among both stationary and moving hosts, and the density of stationary parasite larvae in the environment (Fig. 1). Unlike most host–macroparasite models, we modeled the variance-to-mean ratios as dynamic variables because host processes that affect the mean parasite burden (e.g., parasite-induced mortality and parasite-mediated movement capacity) can also affect the spatial distribution of parasites among hosts (29). This is particularly important when considering migratory hosts because spatial variability in the aggregation of parasites can interact with host movement to affect host–parasite dynamics (13).

Parasites were distributed among hosts according to the negative binomial, consistent with previous models and empirical data (45). We ignored host birth and natural host mortality in order to focus on the parasite-mediated processes during migration.

We numerically simulated the system of equations on a discrete space-time grid. The time step was adjusted depending on migration speed in the simulation so that $\delta t = \eta \delta x / c$, where η is a whole number and c is the speed of moving hosts. At each time step, we first applied host movement and then applied parasite-induced host mortality, parasite attachment, parasite mortality, and host stopping, using the operator-splitting method (46). We assumed Neumann boundary conditions where the derivative across the boundary is zero.

We began all simulations with a Gaussian spatial distribution of migrating hosts centered at $x = 0$ km with the same parasite burden through space (*SI Appendix*, Fig. S1). The initial distribution of stationary parasite larvae in the environment mirrored the distribution of hosts, with a peak density of 10× the peak host density to represent the build-up of parasite larvae in overwintering or breeding habitats prior to migration. For the migratory stalling case, we started with a small fraction of stationary hosts at all points in space to avoid numerical problems because the stationary host density appears in the denominator within model equations. These stationary hosts had the same, constant parasite burdens as their migratory counterparts. Further details on the model can be found in *SI Appendix*, and R code to reproduce simulations is available at https://github.com/sjpeacock/Parasit-mig-patterns.

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