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Macroparasite dynamics of migratory host populations

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HIGHLIGHTS

- Host migration affects parasite dynamics in many wildlife species.
- We develop a spatial model in which host movement depends on parasite burden.
- Positive feedbacks can lead to parasite-induced migratory stalling of host populations.
- The general model is adaptable for different migratory host–macroparasite systems.

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ABSTRACT

Spatial variability in host density is a key factor affecting disease dynamics of wildlife, and yet there are few spatially explicit models of host–macroparasite dynamics. This limits our understanding of parasitism in migratory hosts, whose densities change considerably in both space and time. In this paper, we develop a model for host–macroparasite dynamics that considers the directional movement of host populations and their associated parasites. We include spatiotemporal changes in the mean and variance in parasite burden per host, as well as parasite-mediated host mortality and parasite-mediated migratory ability. Reduced migratory ability with increasing parasitism results in heavily infested hosts halting their migration, and higher parasite burdens in stationary hosts than in moving hosts. Simulations reveal the potential for positive feedbacks between parasite-reduced migratory ability and increasing parasite burdens at infection hotspots, such as stopover sites, that may lead to parasite-induced migratory stalling. This framework could help understand how global change might influence wildlife disease via changes to migratory patterns and parasite demographic rates.

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1. Introduction

Many animals undergo arduous migrations to track seasonal changes in environmental conditions and resources. The resulting spatiotemporal changes in host density have profound and diverse consequences for the dynamical interactions between hosts and parasites (Altizer et al., 2011). For example, host migration may facilitate the spread of parasites into new areas where they might infect novel host species—an increasing concern in the face of warming temperatures that allow parasites to persist where they previously could not (e.g., Kutz et al., 2013). Alternately, migratory hosts may escape parasitism by moving away from infection hotspots where parasites have accumulated in the environment (Bartel et al., 2011). Such migratory escape has, for example, been proposed as a driver of post-calving migration in caribou (Folstad et al., 1991). Migratory lifecycles may also reduce transmission of parasites from adults to juveniles, termed migratory allopatry, as is the case for sea louse parasites of Pacific salmon (Krkošek et al., 2007). Mechanisms such as parasite spread and migratory escape may act simultaneously, with their relative importance depending on the life histories of both the parasite and the host. Further, changes in host–parasite dynamics due to, for example, climate change (Kutz et al., 2013) or the introduction of reservoir hosts (Krkošek et al., 2007; Morgan et al., 2007) may alter how migration influences host–parasite dynamics. These complexities make it difficult to understand and predict the how migration influences host–parasite dynamics.

Mathematical models describing the growth and spread of infectious pathogens through a host population have been integral to the understanding of disease dynamics in both human and wildlife populations (May and Anderson, 1991; Hudson et al., 2002). Two basic structures have been applied in modelling disease dynamics: (1) compartmental models typically used to describe microparasites and (2) macroparasite models. Compartmental models track

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the transition of hosts between susceptible (S) and infected (I) categories and thus describe the prevalence of infection within the host population. Sometimes immune or recovered (R) hosts are also considered, leading to the common designation as SIR models. These models are typically used to describe microparasites (e.g., viruses, bacteria) because the impact of the parasite is assumed to be independent of the number of parasites infecting a host (Anderson and May, 1979).

Several recent studies have used compartmental models to understand and predict parasite dynamics in migratory wildlife (e.g., Hall et al., 2014; Johns and Shaw, 2015; Hall et al., 2016). These models tracked the densities of susceptible and infected hosts at different stages in the annual cycle (e.g., breeding, migration, and overwintering). Hall et al. (2014) describe an SI model in which mortality of host populations during migration depends on their infection status at the end of the breeding or overwintering season. They found that migration lowered pathogen prevalence via culling of infected hosts, and thus host population health improved with earlier departure and longer-distance migrations. Johns and Shaw (2015) built upon that model to look at disease prevalence in migratory vs. non-migratory populations with similar results: host populations ended up healthier if they spent more time migrating and had higher mortality during migration due to disease or other factors. More recent work on vector-borne diseases has also considered how changing phenology associated with climate change might lead to "migratory mismatch" of host and vector densities (Hall et al., 2016).

Macroparasite dynamics require a different model structure than microparasites because the impact of macroparasites on hosts is often proportional to parasite burden, as is typical for many helminths (parasitic worms; e.g., tapeworms, flukes) or ectoparasites (e.g., ticks, lice). Macroparasites also tend to be aggregated among hosts (Shaw et al., 1998). Explicitly considering the intensity of infection and the degree of aggregation is important in macroparasite models because the mortality of heavily infected hosts will result in disproportionate mortality in the parasite population, which in turn feeds back on host population health (Anderson and May, 1978). A less-recognized complication is that the degree of aggregation will change with any process that tends to select heavily infested hosts, such as parasite-induced host mortality, with subsequent impacts on parasite population dynamics. This additional complexity has hindered the development of spatially explicit models for macroparasite dynamics (Riley et al., 2015). Spatial effects have been *implicitly* included in macroparasite models via spatial patchiness in infection pressure (Cornell et al., 2004; May, 1978) or discrete geographic areas (Morgan et al., 2007), but models that *explicitly* track the movement of hosts and their parasites have been lacking (but see Milner and Zhao, 2008 who consider passive flow of parasites in a river system).

Explicitly spatial macroparasite models are needed to understand and predict how host movement and parasitism might interact to affect wildlife health, which is especially important for migratory species. Existing models of parasite dynamics in migratory animals (e.g., Hall et al., 2014; Johns and Shaw, 2015; Hall et al., 2016; Morgan et al., 2007) do not consider how parasite burdens change dynamically over time and space or incorporate the dynamic processes occurring during movement that might influence parasite burdens, such as transmission and parasite-mediated migratory ability. These shortcomings not only limit our understanding for macroparasites, but ignore important aspects of host biology. Animals with high parasite burdens, for example, often show reduced migratory ability (Risely et al., 2017). Monarch butterflies infested with protozoan parasites are slower and fly shorter distances (Bradley and Altizer, 2005) and juvenile salmon infested with sea lice have reduced swimming performance (Nendick et al., 2011) and compromised schooling behaviour (Krkošek

et al., 2011). Parasite-mediated migratory ability may affect both the spatial distribution of hosts, reducing the distance migrated by parasitized individuals, and the spatial patterns in parasite burden, resulting in higher parasite burdens of stationary hosts left behind.

Here, we develop a new modelling framework for migratory-host and macroparasite population dynamics that considers dynamic changes in host abundance, parasite burden, and parasite aggregation. This extends previous host–macroparasite models (e.g., Anderson and May, 1978; Kretzschmar and Adler, 1993) to explicitly include spatial representation of a migration corridor. Parasite aggregation, as well as abundance, is allowed to change dynamically in space and time as a consequence of multiple interacting demographic, spatial, and epidemiological processes. First, we introduce the model and then we explore the model-predicted dynamics under a range of parameters. These simulation exercises provide new insights, such as the potential for parasite-mediated migratory stalling, and hint at the potential for broader application of the model in future studies.

2. Model

We develop a model that tracks changes in host abundance, parasite burden, and the aggregation of parasites along a one-dimensional migration corridor using a system of partial differential equations (PDEs). The model includes potential impacts of parasite burden on the migratory ability of hosts by dividing the host population into two categories: those that are moving at a constant speed and those that are stationary. We consider the rate at which hosts change from moving to stationary (i.e., stopping) to be a function of parasite burden. We also consider how the aggregation of parasites in the host population might change as the host population migrates (Adler and Kretzschmar, 1992; Kretzschmar and Adler, 1993). In the following section, we develop equations describing the spatiotemporal changes in host abundance, mean parasite burden, and the variance-to-mean ratio in the parasite distribution among hosts.

2.1. Birth, death, stopping, and starting

Following the approach of Anderson and May (1978) and Kretzschmar and Adler (1993), we begin with a system of differential equations that describe the number of hosts with i parasites, p_i. We extend the model of Kretzschmar and Adler (1993) to include a spatial component, and distinguish moving and stationary hosts, where $p_i(x, t)$ is the number of stationary hosts with i parasites at location x and time t, and $\hat{p}_i(x, t)$ is the number of moving hosts at location x and time t. For all variables, we use ˆ to denote the moving population. Moving hosts stop at parasite-dependent rate γ_i and stationary hosts start moving at constant rate ω. Other parameters in the model do not directly depend on whether hosts are moving or stationary. Hosts are born parasite-free and stationary at rate β; we assume the host birth is independent of parasite burden, although this assumption could be relaxed in future models (e.g., Dobson and Hudson, 1992). Hosts die at natural rate μ, with additive parasite-induced mortality at per-parasite rate α (Anderson and May, 1978). Parasites attach at rate ϕ (see Section 2.2), reproduce within the host at rate ρ, and die at rate σ. We assume that parasite demographic rates are density independent, except that the rate of parasite-induced host death depends on parasite burden. The basic model is described by four partial differential equations:

$$\frac{\partial p_0}{\partial t} = \beta \sum_{i=0}^{\infty}(p_i + \hat{p}_i) - (\mu + \phi)p_0 + \sigma p_1 + \gamma_0 \hat{p}_0 - \omega p_0$$

(1)