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SYMPOSIUM

Are Migratory Animals Superspreaders of Infection?

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Synopsis Migratory animals are simultaneously challenged by the physiological demands of long-distance movements and the need to avoid natural enemies including parasites and pathogens. The potential for animal migrations to disperse pathogens across large geographic areas has prompted a growing body of research investigating the interactions between migration and infection. However, the phenomenon of animal migration is yet to be incorporated into broader theories in disease ecology. Because migrations may expose animals to a greater number and diversity of pathogens, increase contact rates between hosts, and render them more susceptible to infection via changes to immune function, migration has the potential to generate both "superspreader species" and infection "hotspots". However, migration has also been shown to reduce transmission in some species, by facilitating parasite avoidance ("migratory escape") and weeding out infected individuals ("migratory culling"). This symposium was convened in an effort to characterize more broadly the role that animal migrations play in the dynamics of infectious disease, by integrating a range of approaches and scales across host taxa. We began with questions related to within-host processes, focusing on the consequences of nutritional constraints and strenuous movement for individual immune capability, and of parasite infection for movement capacity. We then scaled-up to between-host processes to identify what types, distances, or patterns of host movements are associated with the spread of infectious agents. Finally, we discussed landscape-scale relationships between migration and infectious disease, and how these may be altered as a result of anthropogenic changes to climate and land use. We are just beginning to scratch the surface of the interactions between infection and animal migrations; yet, with so many migrations now under threat, there is an urgent need to develop a holistic understanding of the potential for migrations to both increase and reduce infection risk.

Introduction

The migratory behavior of animals has fascinated humans for centuries. With the advent of increasingly sophisticated tagging and tracking technologies, we now know that billions of migratory animals traverse the globe each year in pursuit of food, safety, and reproductive opportunities (Bowlin et al. 2010; Bauer and Hoye 2014). Research on animal migrations—broadly defined as consistent, directional movements from one destination to another, undistracted by intervening resources—has historically focused on when, where, how, and why animals migrate (Dingle 2014). However, it is increasingly recognized that animal migrations have the potential to alter ecosystem structure, dynamics, and function in the communities they visit (Bauer and Hoye 2014). In particular, there is growing interest in the role of these predictable, directed, mass movements in the transmission and evolution of parasites (Altizer et al. 2011).

Migrations form unique links between disparate locations, involve large numbers of individuals, and may increase parasite exposure through the use of multiple different habitats and increased interspecies interactions. As a result, animal migrations are widely assumed to enhance the cross-species transmission and global spread of parasites. For instance, migrations are suspected to have facilitated the rapid emergence and spread of neotropical ticks (Cohen et al. 2015), Phocine Distemper Virus (Harding et al. 2002), *Mycoplasma gallisepticum* (Hochachka and Dhondt 2000), and finch trichomoniasis (Lawson et

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al. 2011), as well as zoonotic pathogens including Ebola virus (Ogawa et al. 2015), avian influenza viruses (Takekawa et al. 2010; Prosser et al. 2013; Verhagen et al. 2015), and West Nile virus (Dusek et al. 2009). Yet, our understanding of the involvement of migrants is often quite superficial. For instance, finding active infection (or antibodies) in migratory species (Leblebicioglu et al. 2014), and demonstrating spatio-temporal and phylogenetic correlations between outbreaks and broad migration patterns (Verhagen et al. 2015) can only provide coarse indications of the involvement of migrants. Assessing the importance of animal migrations to infectious disease dynamics requires the integration of migration, as an ecological phenomenon, into formal theories of disease ecology and applications of physiological ecology.

Migration as a superspreading process

The dynamics of infectious diseases, in both humans and wildlife, are profoundly altered by so-called "superspreader" hosts (Lloyd-Smith et al. 2005; Paull et al. 2012). Superspreaders are defined as those individuals, species, or habitat patches that are responsible for more secondary cases than an upper percentile (e.g., 99th) of the expected distribution of transmission events assuming homogeneous transmission (Lloyd-Smith et al. 2005; Paull et al. 2012). These individuals, species, and habitat patches are typically characterized by increased susceptibility, competence (intensity and duration of infectiousness), and contact rates (Galvani and May 2005; Hawley et al. 2011; Paull et al. 2012; Streicker et al. 2013; Barron et al. 2015), and have been shown to play pivotal roles in shaping the speed, distance, and overall magnitude of epidemics, as well as pathogen maintenance and local extinction (Lloyd-Smith et al. 2005). Critically, although migration has yet to be considered in the context of superspreader theory, migration has been hypothesized to alter the susceptibility, competence, and contact rates of species undertaking migration and the locations they visit *en route* (Fig. 1). Migration therefore has the potential to generate both superspreader hosts and infection "hotspots" in time and space.

In formulating this symposium, we sought to investigate the potential for migratory animals to act as superspreaders, based on evidence that migration may increase contact rates, exposure, susceptibility, and competence (Fig. 1). Migrations form unparalleled links between otherwise disconnected geographic locations, involve large numbers of individuals, and frequently result in unique inter-species interactions (Altizer et al. 2011; Bauer and Hoye 2014), each of which are likely to increase the number of contacts with other potential hosts compared to resident species. In addition, hosts that move between regions are naturally exposed to parasites in each of the regions they visit, resulting in migratory species experiencing a higher diversity of parasite exposure and infection (Figuerola and Green 2000; Jenkins et al. 2012; Koprivnikar and Leung 2015; Hannon et al. 2016). Finally, long-distance migration can be incredibly physiologically demanding, entailing repeated cycles of strenuous physical exertion and high metabolic rates interspersed with periods of frantic energy acquisition and physical recovery (Weber and Stilianakis 2007; Piersma and van Gils 2011). These intense physiological demands have been correlated with reduced immune responses (Owen and Moore 2006, 2008; Dolan et al. 2016), either directly, or via resource redistribution prior to the onset of migration (reviewed by Buehler et al. 2010 and Altizer et al. 2011). Such changes to host resistance have been suggested to render migrants more susceptible to infection (van Dijk et al. 2014) or reinfection (Gylfe et al. 2000). Collectively, these traits suggest migration has the potential to act as a superspreading process, both in terms of novel introductions to and amplification within resident communities (Fig. 1).

Migration as a transmission–reduction process

Critically, traits that heighten migrants' exposure, susceptibility, and contact rates may simultaneously generate mechanisms that curtail parasite transmission. First, migrants may have co-evolved with the parasites throughout their entire migratory range, resulting in adaptive immune responses (Moller and Erritzoe 1998). Migrants may also upregulate immune function in response to increased densities of conspecifics (Srygley and Lorch 2011). Moreover, parasites may impose constraints on the foraging, fueling, breeding, and survival of their hosts, particularly in the face of additional physiological stressors (Ostfeld 2008). The increased susceptibility and competence of migrants (due to increased exposure and/or lowered resistance) can therefore be expected to simultaneously decrease capacity for migration (Altizer et al. 2011). Theoretical and empirical work has shown that when infection renders hosts incapable of migrating successfully, this "culling" process reduces transmission potential (Bradley and Altizer 2005; Bartel et al. 2011; Altizer et al. 2015). Cycles of parasite transmission can be interrupted when migrants depart habitats that have accumulated infectious propagules, resulting in reduced transmission through migratory "escape"

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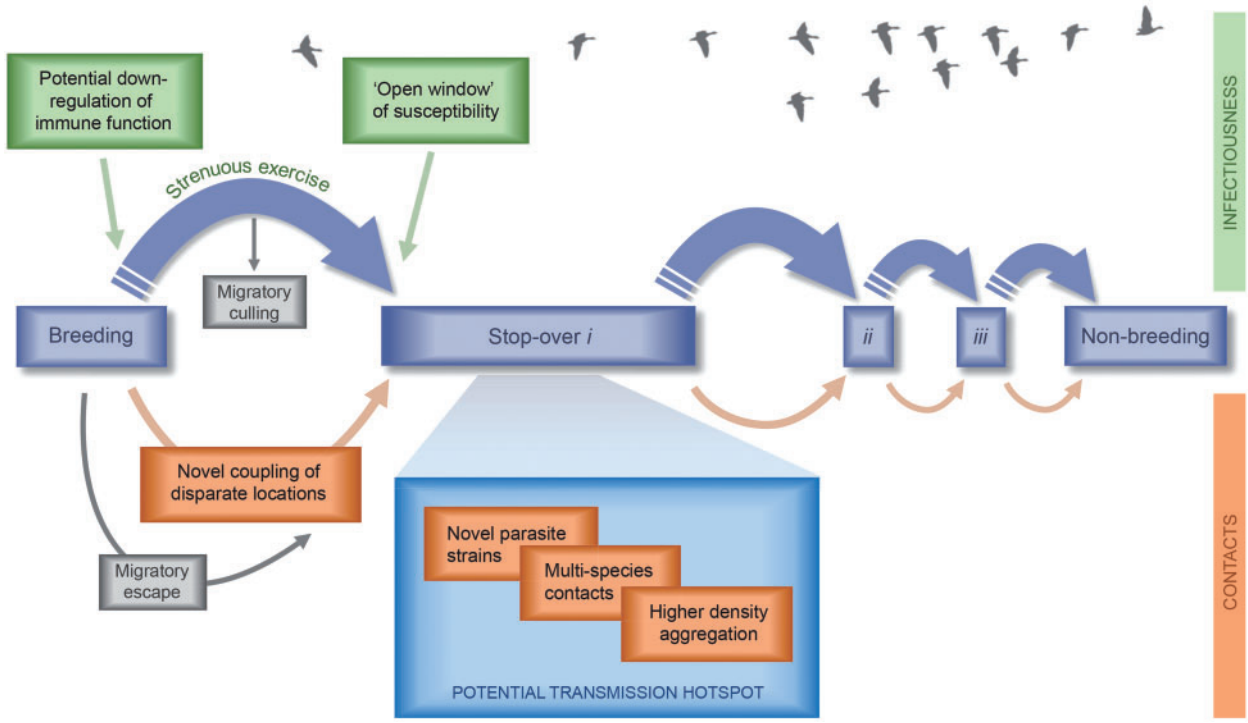

Fig. 1. Migratory animals have the potential to act as superspreading species (sensu Paull et al. 2012) through several interacting mechanisms. Migrants move between multiple sites throughout the annual cycle, including breeding, non-breeding, and intervening stop-over sites (*i, ii, iii,*. . .). In doing so, migrants often experience higher rates of contact with both novel pathogens and susceptible hosts than resident species at each of these sites. Migrants may also experience increased susceptibility and hence infectiousness through repeated cycles of migratory preparation and strenuous exercise. However, migration may also reduce transmission by removing infected individuals from the population ("culling") and removing susceptible individuals from habitat patches where infectious stages have accumulated ("escape") .

(Bartel et al. 2011). Migratory escape may be particularly effective when parasite transmission is restricted to a single stage of the migratory cycle and when unfavorable conditions (such as harsh winters or a lack of hosts) reduce propagule abundance between intervals of migrant occupancy—(Hall et al. 2014). The concepts of migratory culling and migratory escape have been well-documented in monarch butterflies (Altizer et al. 2011; Satterfield et al. 2015), but further evidence exists in tick-infested red deer (Mysterud et al. 2016), benthic fish infected by skin-piercing trematodes (Poulin et al. 2012), reindeer infected by warble fly larvae (Folstad et al. 1991), and the seroprevalence of antibodies to avian influenza viruses in lesser black-backed gulls (Arriero et al. 2015). Collectively, these findings demonstrate that migratory behavior can operate as a mechanism to reduce infection in host populations, with consequences for pathogen transmission, pathogen evolution, and the evolution of migratory strategies (Altizer et al. 2011; Hall et al. 2014; Johns and Shaw 2016).

Ultimately, any broad assessment of the role of migrants in the transmission and evolution of pathogens within host communities is predicated on a detailed understanding of the infection process, including the host's ability to either resist infection, or tolerate and successfully migrate in spite of their infection burden. Our symposium and the associated papers in this issue therefore sought to investigate the interactions between migration and infection using a range of integrative approaches spanning several levels of biological organization. In the symposium, "Are migratory animals superspreaders of infection?" speakers explored: (1) effects of strenuous activity on host resistance, and potential consequences for individual infection; (2) interactions between immune responses and other stressors, such as nutrition and reproduction; (3) links between parasite spread and long-distance movements, and the spatial limits to these interactions; (4) performance of infected animals relative to uninfected conspecifics; and (5) infection dynamics and pathogen evolution in the context of environmental change.

Migration, physical exertion, and constraints on host immune defenses

Migrations are generally thought to be an evolutionary adaptation that allows animals to track seasonal

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changes in the availability of limiting resources, such that migratory strategies may be fundamentally driven by dietary needs (Dusek et al. 2009; Jachowski and Singh 2015). However, in addition to gross calorific intake, migrations may be driven by more specific nutritional needs or dietary components. Srygley (2016, this issue) reviews evidence from Mormon crickets (*Anabrus simplex*) and migratory locusts (*Locusta migratoria)* to reveal that deprivation of either carbohydrates or protein not only initiates collective movements in these hosts, but also increases susceptibility to bacterial and fungal infection. While the expenditure of such limited resources during strenuous movement is widely expected to come at the expense of immune defenses, identifying these trade-offs is challenging and, as a consequence, rarely done in free-living animals. van Dijk and Matson (2016, this issue) therefore discuss insights into the consequences of physical activity for immunity drawn from human exercise physiology. Exercise immunology proposes the existence of finite "open windows" of susceptibility following exercise (van Dijk and Matson 2016). Taken in the context of migratory movements, the timing of immunosuppression following bouts of movement may therefore coincide with exposure to (novel) parasites, highlighting the potential for complex temporal interactions between physical activity and immune responses. Such conflicts between higher exposure to parasites (especially owing to increased contact with conspecifics) and simultaneous constraints on the ability to defend against them likely occurs in other migratory species (Moller and Erritzoe 1998; Koprivnikar and Leung 2015) and highlights one of the challenges in predicting "optimal" levels of parasite defense for migrants.

Because the precise causal links between movement or migration and disease susceptibility are very difficult to establish, experimental approaches are likely to be beneficial in this endeavor. One experimental approach for investigating potential costs of migration for individual immunity is the use of forced flight in captive animals. To this end, Fritzsche McKay and colleagues (2016, this issue) forced monarch butterflies (*Danaus plexippus*) to fly for moderate durations of time across several days and measured subsequent changes in three invertebrate immune measures. Because being reproductively active can also constrain resources available for immune defenses, this study compared effects of flight on immune responses in groups that differed in reproductive status: reproductively active or in diapause, the non-reproductive physiological condition associated with fall migration in the wild (Fritzsche McKay et al. 2016). Forced flight did not impair any of the measured immune responses and reproductive status did not modify the costs of flight for immunity. However, monarchs in diapause (the migratory condition) were more efficient fliers than reproductive monarchs, suggesting migration-adapted animals may deploy limited resources more efficiently than non-migrants, potentially rendering any immune costs of flight less than previously assumed (Weber 2009). Moreover, reconciling the relationship between immune response measures and susceptibility to primary infection remains an outstanding challenge in both human exercise immunology and wildlife eco-immunology (van Dijk and Matson 2016). This link is essential for scaling-up individual-level processes to population-level disease dynamics.

Contributions of migrants to parasite transmission and spread

Epidemiological theory has long established that movement of infected individuals between patches can enhance parasite colonization (Hess et al. 2002), and that such movements may play a pivotal role in local adaptation and coevolution between hosts and parasites (Gandon and Michalakis 2002; Lion and Gandon 2015). As Hill and Runstadler (2016, this issue) discuss in the context of avian influenza viruses, a burgeoning number of phylogenetic analyses imply that animal migration can govern the structure of transmission networks and maintenance of pathogen diversity across broad spatial scales. Yet, this approach is highly sensitive to sampling bias, with the majority of genetic information obtained through "reactionary surveillance" targeted at migratory birds following outbreaks of zoonotic disease, rather than long-term surveillance representative of all hosts. Hill and Runstadler therefore suggest phylogeographics need to be tempered with detailed information on host physiology, migratory status, and pathology of infection, in order to assess the relative contribution of migrants (Newman et al. 2009 and Verhagen et al. 2014). In particular, because sub-lethal effects of parasites can be intensified through periods of increased physiological stress (Pedersen and Greives 2008), long-distance migrants may be especially susceptible to negative effects of parasitic infection. Given the potential for sub-lethal effects of infection on individual behavior to alter the timing, location, and total burden of infection (Galsworthy et al. 2011; Bauer et al. 2015), Hoye et al. (2016, this issue) add to a limited number of empirical studies assessing the potential for infection to alter the ecological performance of hosts. Bewick's swans (*Cygnus columbianus bewickii*) that were naturally infected with avian influenza virus but lacked antibodies indicative of prior infection showed decreased foraging

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rates and were unlikely to be re-sighted one year after infection (Hoye et al. 2016). However, swans that were infected but had survived a previous infection were indistinguishable from uninfected birds in each of the organismal performance metrics. Critically, movements were only studied during the overwintering period and studies examining the migratory behavior of infected and uninfected animals remain sorely needed. Ideally, these would be combined with explicit consideration of the duration of infection (Gaidet et al. 2010) and individual contact rates (Boulinier et al. 2016) in order to assess the dispersal potential of migrants.

Hill and Runstadler (2016) also urge that viral flow may follow more complex patterns of animal movement than the broad flyway-based assumptions used to date. Movements classified as migration (predictable, directed, often inter-seasonal), and dispersal (once-off, permanent relocation from natal site), have received the majority of attention with respect to parasite transmission and maintenance. Yet, Boulinier et al (2016, this issue) demonstrate that ''prospecting''—within season movements from the site of breeding to other potential breeding sites—can influence the spatial ecology, evolution, and epidemiology of infectious diseases. Within their seabird study system, several lines of evidence suggest that within-season prospecting movements maintain gene-flow and alter parasite pressure among breeding colonies (Boulinier et al. 2016). However, this influence has a distinct spatial boundary, defined by the scale of the prospecting movements of the host species. Critically, these movements tend to be on the scale of tens to a few hundred kilometers, and may lack many of the physiological challenges faced by migrants that result in ''culling''. Finally, because there also appear to be heterogeneities in transmission potential within migratory populations—as a function of other life history events (e.g., juveniles or failed breeders—Boulinier et al. 2016) or as a function of infection history (e.g., those that have survived previous infection but lack protective immunity (Hoye et al. 2016)—understanding of the connection between host, species, temporal, and spatial heterogeneities in infectious diseases is needed (Paull et al. 2012).

Anthropogenic change and the shifting landscape of migration–parasite interactions

Global change processes such as climate warming (Robinson et al. 2009; Tian et al. 2015), and habitat loss or alteration (Garamszegi 2011; James and Abbott 2014; Martin and Fahrig 2016) are actively altering (and endangering) some animal migrations (Wilcove and Wikelski 2008; Robinson et al. 2009; Bowlin et al. 2010). Such changes in climate or habitat availability not only affect migration behavior, but may also profoundly alter infection dynamics. For instance, high densities of captive salmon farmed in coastal pens act as transmission foci that increase sea lice infection pressure for wild salmon by as much as four orders of magnitude (Krkošek et al. 2007), eroding the traditional benefits of migratory escape. Similarly, increased planting of invasive tropical milkweed in southern regions of the United States has facilitated winter breeding —a more sedentary behavior—in monarch butterflies (Satterfield et al. 2015). Satterfield and colleagues (2016, this issue) show a striking increase in prevalence of the protozoan parasite, *Ophryocytsis elektroscirrha* in year-round breeding monarchs in California, demonstrating the impacts of the loss of migratory behavior for infectious disease dynamics.

Migratory patterns have also shifted in elk, with a reduction in the number of long-distance versus short-distance migrants leading to higher burdens of parasites such as *Ixodes* ticks (Mysterud et al. 2016). In the western United States, the practice of subsidized feeding grounds for elk not only substantially shortens historical migration distances, but also creates hotspots for transmission of an abortion-inducing pathogen *(Brucella abortus)* through increased aggregation densities (Merkle et al., unpublished data). Importantly, changes to the local climate, particularly snow cover, interact with the food subsidies to have a profound effect on both animal movements and transmission risk.

Climate-related changes to migration phenology appear widespread (Saino et al. 2011; James and Abbott 2014) and are likely to interact with vector emergence timing, activity levels, and distributions to alter contact rates and transmission dynamics. Hall and colleagues (2016, this issue) modeled the potential influence of several climate change-related scenarios on a vector-borne disease in a hypothetical songbird population exhibiting long-distance migration and in which transmission is limited to the breeding season. They found that if vectors advanced their phenology more quickly than hosts, pathogen prevalence was reduced as a result of a temporal mismatch between host and vector (Hall et al. 2016). However, if both host and vector advance their phenology at the same pace then migration distances became shorter, and pathogen transmission escalated because more infected migrants survived the journey and re-initiated infection cycles at the breeding site (Hall et al. 2016). As more animals shift their migratory patterns in response to

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environmental change (Robinson et al. 2009; Gilroy et al. 2016), there is a pressing need to better understand the role of animal movement in the transmission of infections, including zoonotic pathogens.

Conclusions

Our understanding of the epidemiological importance of migratory animals is in its infancy. These predictable, directed movements, undertaken by billions of animals each year, are increasingly recognized for their capacity to profoundly alter ecosystem dynamics (Bauer and Hoye 2014). However, migration is yet to be integrated within disease ecology more broadly. Several lines of evidence suggest that migratory movements may precipitate key changes to host physiology and behavior, producing the hallmark characteristics of so-called superspreader species (Paull et al. 2012). Overwhelmingly, our symposium highlighted the need to temper widely held assumptions, such as migration-induced immunosuppression and long-distance transmission events, with the physiological and nutritional demands of migration and anthropogenic changes to habitat and climate. Critically, migration may reduce transmission and infection in host populations in certain circumstances, with cascading consequences for the evolution and maintenance of migratory strategies (Clark et al. 2016; Johns and Shaw 2016; Shaw and Binning 2016). Collectively, our symposium demonstrated that migratory animals are dynamic entities, adaptively modified by movement, infection, and the (changing) environments they encounter. Their role in pathogen spread is likely to be complex, and therefore deserves concerted research efforts going forward.

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