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# Animal Migration and Infectious Disease Risk

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Animal migrations are often spectacular, and migratory species harbor zoonotic pathogens of importance to humans. Animal migrations are expected to enhance the global spread of pathogens and facilitate cross-species transmission. This does happen, but new research has also shown that migration allows hosts to escape from infected habitats, reduces disease levels when infected animals do not migrate successfully, and may lead to the evolution of less-virulent pathogens. Migratory demands can also reduce immune function, with consequences for host susceptibility and mortality. Studies of pathogen dynamics in migratory species and how these will respond to global change are urgently needed to predict future disease risks for wildlife and humans alike.

Billions of animals from groups as diverse as mammals, birds, fish, and insects undertake regular long-distance movements each year to track seasonal changes in resources and habitats (1). The most dramatic migrations, such as those by monarch butterflies (Fig. 1), gray whales, and some shorebirds and dragonflies (Fig. 2), span entire continents or hemispheres, can take several months to complete, and are accompanied by high energetic demands and extreme physiological changes. The ultimate cause of these seasonal migrations remains debated; most hypotheses focus on avoidance of food scarcity, seeking physiologically optimal climates, and avoiding predation during periods of reproduction [e.g., (2)]. Contemporary studies of migration have uncovered mechanisms of animal navigation, energy budgets, resource use, and phenological responses to environmental change; migratory species have also been recognized for their potential to connect geographically distant habitats and transfer large amounts of biomass and nutrients between ecosystems [reviewed in (3)]. These studies illustrate the profound ecological and evolutionary consequences of migratory journeys for animal populations on a global scale.

Owing to their long-distance movements and exposure to diverse habitats, migratory animals have far-reaching implications for the emergence and spread of infectious diseases. Importantly, most previous work on the role of host movement in infectious disease dynamics has focused on spatially localized or random dispersal. For example, dispersal events give rise to traveling waves of infection in pathogens such as raccoon rabies (4), influenza in humans (5), and nuclear polyhedrosis viruses in insects (6). In the context of metapopulations, limited amounts of host movement could actually prevent host extinction

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**Fig. 1.** Monarch butterflies (*Danaus plexippus*), shown here at a wintering site in central Mexico, undertake one of the longest distance two-way migrations of any insect species worldwide. Monarchs are commonly infected by a debilitating protozoan parasite that can lower the flight ability of migrating butterflies.

in the face of a debilitating pathogen and allow host resistance genes to spread (7, 8). From a different perspective, case studies of species invasions demonstrate that one-time transfers of even a few individuals can transport pathogens long distances and introduce them to novel habitats (9). Yet relatively few studies have examined how regular, directed mass movements that characterize seasonal migration affect the transmission and evolution of pathogens within host

populations and the response of migratory species to infection risks.

In this article, we review the consequences of long-distance migrations for the ecological dynamics of host-pathogen interactions and outline key challenges for future work. Ecological processes linked with migration can increase or decrease the between-host transmission of pathogens, depending on host migratory behavior and pathogen traits (Fig. 3). Moreover, new work shows that for some species, the energetic demands of migration compromise host immunity, possibly increasing susceptibility to infection and intensifying the impacts of disease. Importantly, many migratory species are at risk of future declines because of habitat loss and exploitation, and animal migrations are shifting with ongoing anthropogenic change (10). Thus, understanding how human

activities that alter migratory patterns influence wildlife-pathogen dynamics is urgently needed to help guide conservation and management of migratory species and mitigate future risks from infectious disease.

## What Goes Around Comes Around: Pathogen Exposure and Spatial Spread

An oft-cited but poorly supported assumption is that long-distance movements of migrating animals can enhance the geographic spread of pathogens, including zoonotic pathogens important for human health such as Ebola virus in bats, avian influenza viruses in waterfowl and shorebirds, and Lyme disease and West Nile virus (WNV) in songbirds. For example, WNV initially spread in North America along a major corridor for migrating birds and rapidly expanded from its point of origin in New York City along the Atlantic seaboard from 1999 to 2000 (11). Although experimental work concluded that passerine birds in migratory condition were competent hosts for WNV and could serve as effective dispersal agents (12), evidence to show that this expansion resulted from movements of migratory birds remains

equivocal. For the zoonotic pathogen Ebola virus, a recent study points to the coincident timing of an annual influx of migratory fruit bats in the Democratic Republic of Congo and the start of human Ebola outbreaks in local villages during 2007 (13). In central Kazakhstan, saiga antelopes (*Saiga tatarica*) become infected with gastrointestinal nematodes (*Marshallagia*) during the course of seasonal migration by grazing on pastures used by domesticated sheep earlier in

Animal	Locations and distance traveled	Major infectious diseases	Major threats to migration	
	<b>Chinook salmon</b> ( <i>Oncorhynchus tshawytscha</i> )	3- to 4-year-old adults migrate up to 1500 km from the Pacific Ocean upriver to freshwater spawning sites in the Pacific Northwestern U.S.	Sea lice ( <i>Lepeophtheirus</i> sp.); Myxozoan ( <i>Henneguya</i> sp.)	Dam construction; Human-modified water flow; Deforestation; Fish hatcheries
	<b>Green sea turtle</b> ( <i>Chelonia mydas</i> )	Adults migrate over 2300 km to nesting locations in tropical to subtropical areas of the Atlantic Ocean, Gulf of Mexico, Mediterranean Sea, and the Indo-Pacific	Tumor-forming herpesvirus (fibropapillomatosis); Spirorchid cardiovascular flukes	Hunting and egg poaching; Bycatch; Nesting and foraging habitat destruction
	<b>Western toad</b> ( <i>Anaxyrus boreas</i> )	Annual breeding migration up to 6 km from hibernating sites (likely underground) to breeding ponds in high-elevation habitats in the Western U.S.	Chytrid fungus ( <i>Batrachochytrium</i> sp.); Parasitic trematode ( <i>Ribeiroia</i> sp.); Oomycete ( <i>Saprolegnia</i> sp.)	Building of roads; Loss of breeding habitat through deforestation
	<b>Ruddy turnstone</b> ( <i>Arenaria interpres</i> )	Annual migration up to 27,000 km from Arctic nesting grounds to overwintering sites along the coastlines of all continents except Antarctica	Avian influenza virus; West Nile virus; Multiple endoparasitic worms	Habitat loss (due to dams, freshwater extraction); Overharvesting of food resources at stopover sites
	<b>Flying foxes</b> ( <i>Pteropus</i> spp.)	Unknown maximum migratory distances for many species; can range between 50-1000 km across Southeast Asia and Australia	Paramyxoviruses such as Nipah virus and Hendra virus	Loss of feeding grounds through deforestation; Habitat loss through land conversion
	<b>Green darner</b> ( <i>Anax junius</i> )	Exact distances unknown, but adults travel 700 km or more annually from southern Canada and northern U.S. to Central America	Eugregarine protozoan ( <i>Geneiorhynchus</i> sp.)	Unknown; possibly destruction of freshwater breeding habitats
	<b>Wildebeest</b> ( <i>Connochaetes taurinus</i> )	In the Serengeti, animals move between wet and dry seasons across an area of 30,000 km <sup>2</sup>	Rinderpest ( <i>Morbillivirus</i> sp.); Brucellosis ( <i>Brucella</i> ); Foot-and-mouth disease ( <i>Aphtaepizooticae</i> )	Landcover change (reduction in tree cover); Fire frequency; Exposure to infected domestic livestock
	<b>Swainson's thrush</b> ( <i>Catharus ustulatus</i> )	Migrate up to 10,000 km annually between breeding grounds in Canada/northern U.S. to overwintering sites in Central and South America	West Nile virus; Lyme disease; Blood parasites ( <i>Haemoproteus</i> and <i>Plasmodium</i> )	Habitat loss on breeding and wintering grounds; Building strikes during migration
	<b>Gray whale</b> ( <i>Eschrichtius robustus</i> )	Annual migrations of over 18,000 km from feeding sites in the Bering Sea to winter breeding grounds along the coast of Baja California	Whale lice (cyamid amphipods, <i>Cyamus</i> spp.); Barnacles ( <i>Cryptolepas</i> ); Multiple endoparasitic worms	Industrial activity near calving lagoons; Oil exploration along migration routes; Vessel harassment

**Fig. 2.** Representative migratory species, including migration distances and routes, known parasites and pathogens, and major threats to species persistence. Infectious diseases have been examined in the context of

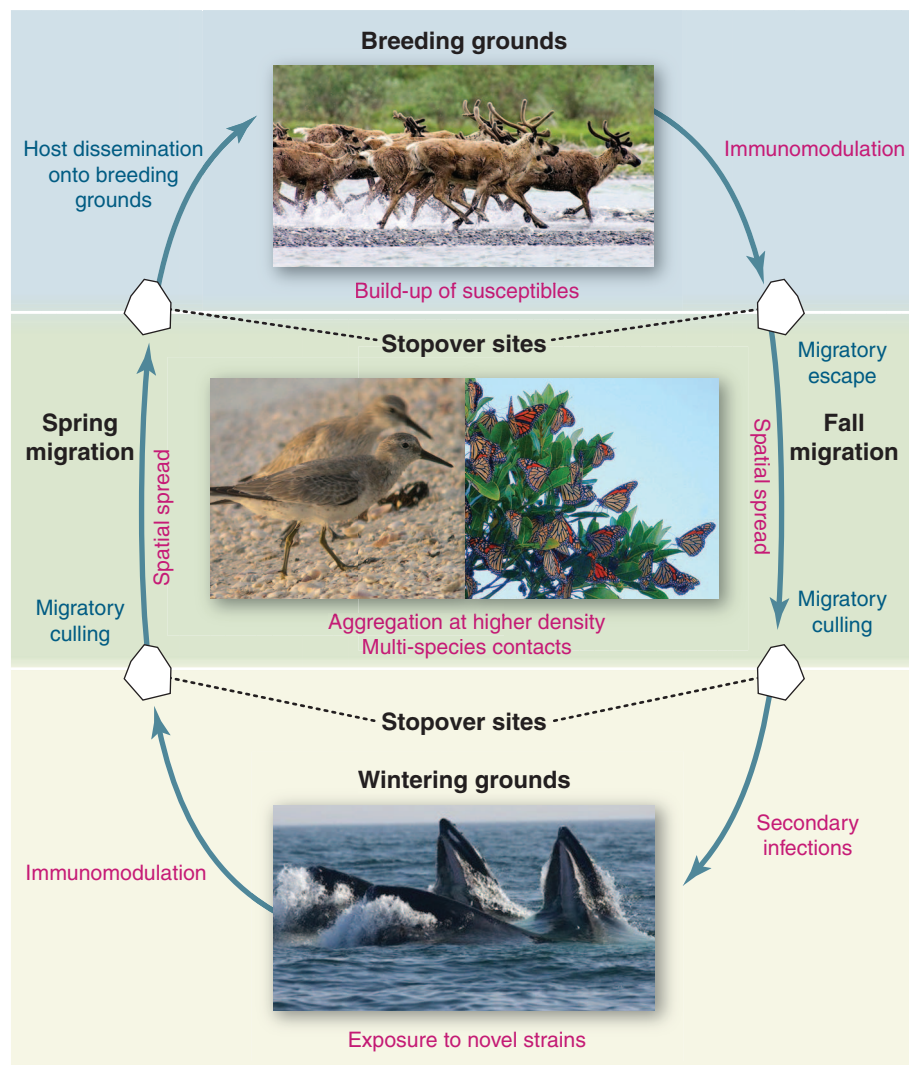
migration for some, but not all, of these species. Supporting references and photo credits are provided in the supporting online material (SOM) text.

the season. As migration continues, saiga carry and transmit *Marshallagia* to northern sheep populations, leading to pulses of infection that coincide with annual saiga migrations (14).

The potential for serious disease risks for human and livestock health has raised alarm about the role of migratory species in moving infectious agents to distant locations. Yet surprisingly few examples of long-distance pathogen dispersal by migrating animals have been clearly documented in the published literature, and some studies indicate that migrants might be unfairly blamed for transporting pathogens. As a case in point, wild waterfowl (Anseriformes) and shorebirds (Charadriiformes) represent the major natural reservoirs for diverse strains of avian influenza virus (AIV) worldwide, including the highly pathogenic (HP) H5N1 subtype that can lethally infect humans (15). Although many of these migratory birds can become infected by HP H5N1, recent work incorporating what is known about viral shedding period, host migration phenology, and the geographical distribution of viral subtypes suggests that most wild birds are unlikely to spread HP H5N1 long distances (e.g., between Asia and the Americas) as previously suspected [e.g., (16, 17)]. Central to the question of how far any host species can transport a pathogen are the concepts of pathogen virulence and host tolerance to infection. Specifically, virulence refers to the damage that parasites inflict on their hosts, and tolerance refers to the host's ability to withstand infection without suffering major fitness costs. Thus, host-parasite species or genotype combinations associated with very low virulence or high tolerance should be the most promising candidates for long-distance movement of pathogen strains, a simple prediction that could be explored within migratory species or using cross-species comparisons.

Beyond their potential role in pathogen spatial spread, a handful of studies suggest that migratory species themselves encounter a broader range of pathogens from diverse environments throughout their annual cycle compared with species residing in the same area year-round (Fig. 3). One field study showed that songbird species migrating from Europe became infected by strains of vector-borne blood parasites originating from tropical bird species at overwintering sites in Africa (18), in addition to the suite of parasite strains transmitted at their summer breeding grounds. The authors posited that winter exposure to parasites in tropical locations is a significant cost of migration, because resident species wintering in northern latitudes encounter fewer parasite strains and do not experience year-round transmission. Similarly, the number of parasite species per host was positively related to distances flown by migratory waterfowl (19), indicating that migrating animals could become exposed to parasites through encounters with different host species and habitat types.

Although some animals undertake nonstop migrations, most migratory species use stop-



**Fig. 3.** Points along a general annual migratory cycle where key processes can increase (red text) or decrease (blue text) pathogen exposure or transmission. Behavioral mechanisms such as migratory escape and migratory culling could reduce overall pathogen prevalence. As animals travel to distant geographic locations, the use of multiple habitat types including stopover sites, breeding areas, and wintering grounds can increase transmission as a result of host aggregations and exposure to multihost pathogens. This might be especially true for migratory staging areas where animals stop to rest and refuel. High energetic demands of spring and fall migration can also result in immunomodulation, possibly leading to immune suppression and secondary infections. [Photo credits (clockwise): J. Goldstein, B. McCord, A. Friedlaender, and R. Hall]

over points along the migration route to rest and feed. These stopover points usually occur frequently along a journey, although some species like shorebirds fly thousands of kilometers between only a handful of staging areas (1). Refueling locations are often shared by multiple species, and the high local densities and high species diversity can increase both within- and between-species transmission of pathogens. In one of the most striking examples of this phenomenon, shorebirds such as sanderlings (*Calidris alba*), ruddy turnstones (*Arenaria interpres*; Fig. 2), and red knots (*Calidris canutus*), which migrate annually between Arctic breeding grounds and South American wintering sites, congregate to feed in massive numbers

in the Delaware Bay and the Bay of Fundy to rebuild fat reserves, leading to upwards of 1.5 million birds intermingling, at densities of over 200 birds per square meter (20). This phenomenon creates an ecological hotspot at Delaware Bay, where the prevalence of AIV is 17 times greater than at any other surveillance site worldwide (20).

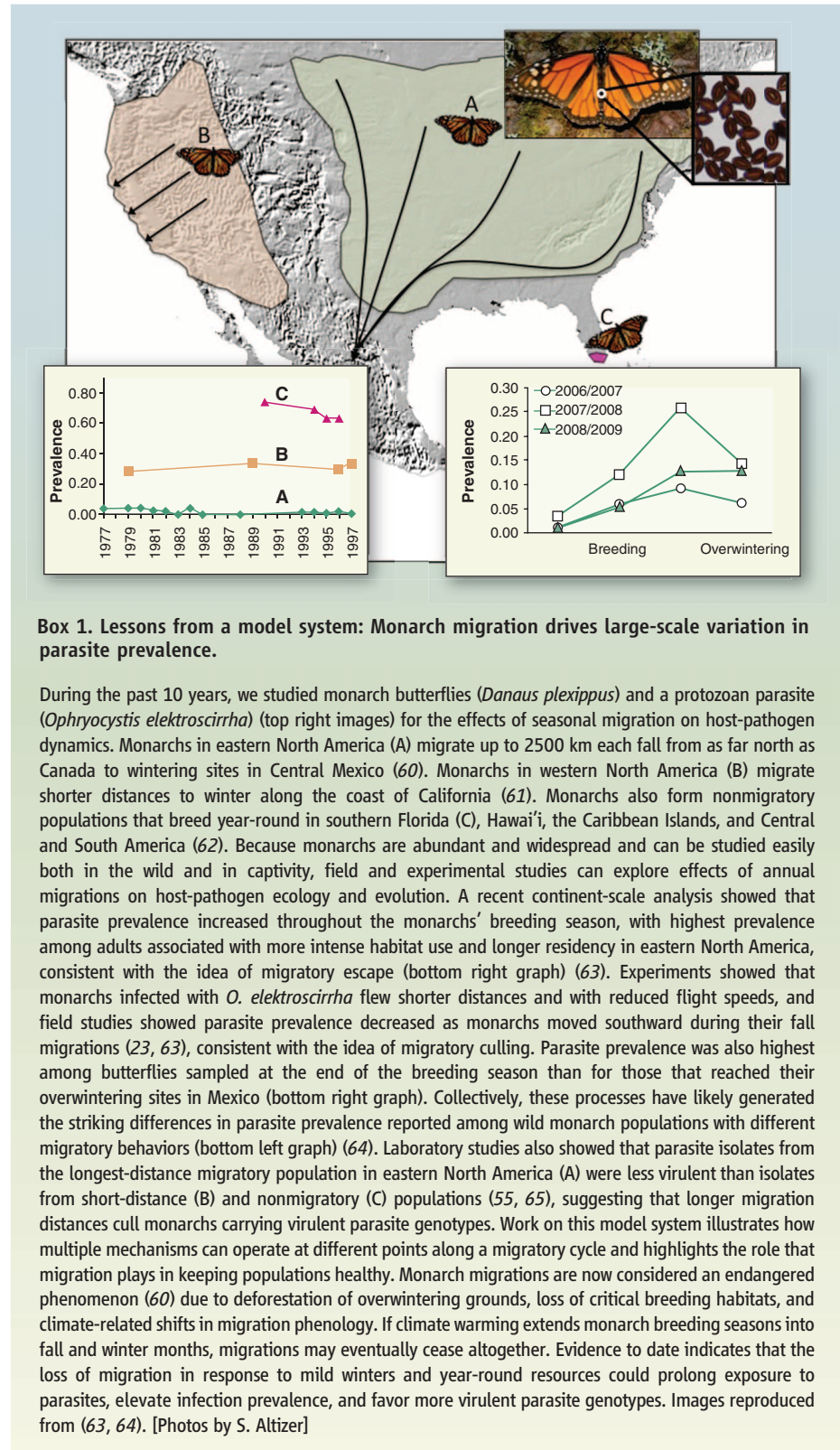
#### Leaving Parasites Behind: Migration as a Way of Lowering Infection Risk

Although greater exposure to parasites and pathogens can pose a significant cost of long-distance migration, for some animal species, long-distance migration will reduce infection risk by at least two nonexclusive processes (Fig. 3). First, if pro-

longed use of habitats allows parasites with environmental transmission modes to accumulate (i.e., those parasites with infectious stages that can persist outside of hosts, such as many helminths, ectoparasites, and microbial pathogens with fecal-oral transmission), migration will allow animals to escape from contaminated habitats [i.e., “migratory escape” (21)]. Between intervals of habitat use, unfavorable conditions (such as harsh winters and a lack of hosts) could eliminate most parasites, resulting in hosts returning to these habitats after a long absence to encounter largely disease-free conditions (21). Empirical support for migratory escape comes from a few well-studied host-parasite interactions, including research on reindeer (*Rangifer tarandus*), which showed that the abundance of warble flies (*Hypoderma tarandi*) was negatively correlated with the distance migrated to summer pastures from reindeer calving grounds (the main larval shedding area in early spring) (22). This observation prompted researchers to suggest that the reindeers’ annual postcalving migration reduces warble fly transmission by allowing animals to leave behind areas where large numbers of larvae have been shed (and where adult flies will later emerge). It is worth noting that escape will be less successful from pathogens with long-lived infectious stages that persist between periods of host absence or pathogens that cause chronic or life-long infections.

Long-distance migration can also lower pathogen prevalence by removing infected animals from the population [i.e., “migratory culling” (23)]. In this scenario, diseased animals suffering from the negative consequences of infection are less likely to migrate long distances owing to the combined physiological demands of migration and infection. Work on the migratory fall armyworm moth (*Spodoptera frugiperda*) suggested that insects infected by an ectoparasitic nematode (*Noctuidonema guyanense*) had reduced migratory ability because few to no parasites were detected in moths recolonizing sites as they returned north (24). More recent work on Bewick’s swans (*Cygnus columbianus bewickii*) showed that infection by low-pathogenic avian influenza (LPAI) viruses delayed migration over a month and reduced the travel distances of infected birds compared with those of healthy individuals (25). However, a study of AIV in white fronted geese did not find any difference in distances migrated between infected and uninfected birds (26), suggesting that, not surprisingly, some species are better able to tolerate infections during long journeys and raising the possibility that migration could select for greater tolerance to infections in some hosts due to the high fitness costs of attempting migration with a debilitating pathogen.

Whether the net effects of migration will increase or decrease prevalence depends in large part on the mode of parasite transmission and the level of host specificity, both of which will affect opportunities for cross-species transmission at



**Box 1. Lessons from a model system: Monarch migration drives large-scale variation in parasite prevalence.**

During the past 10 years, we studied monarch butterflies (*Danaus plexippus*) and a protozoan parasite (*Ophryocystis elektroscirra*) (top right images) for the effects of seasonal migration on host-pathogen dynamics. Monarchs in eastern North America (A) migrate up to 2500 km each fall from as far north as Canada to wintering sites in Central Mexico (60). Monarchs in western North America (B) migrate shorter distances to winter along the coast of California (61). Monarchs also form nonmigratory populations that breed year-round in southern Florida (C), Hawai’i, the Caribbean Islands, and Central and South America (62). Because monarchs are abundant and widespread and can be studied easily both in the wild and in captivity, field and experimental studies can explore effects of annual migrations on host-pathogen ecology and evolution. A recent continent-scale analysis showed that parasite prevalence increased throughout the monarchs’ breeding season, with highest prevalence among adults associated with more intense habitat use and longer residency in eastern North America, consistent with the idea of migratory escape (bottom right graph) (63). Experiments showed that monarchs infected with *O. elektroscirra* flew shorter distances and with reduced flight speeds, and field studies showed parasite prevalence decreased as monarchs moved southward during their fall migrations (23, 63), consistent with the idea of migratory culling. Parasite prevalence was also highest among butterflies sampled at the end of the breeding season than for those that reached their overwintering sites in Mexico (bottom right graph). Collectively, these processes have likely generated the striking differences in parasite prevalence reported among wild monarch populations with different migratory behaviors (bottom left graph) (64). Laboratory studies also showed that parasite isolates from the longest-distance migratory population in eastern North America (A) were less virulent than isolates from short-distance (B) and nonmigratory (C) populations (55, 65), suggesting that longer migration distances cull monarchs carrying virulent parasite genotypes. Work on this model system illustrates how multiple mechanisms can operate at different points along a migratory cycle and highlights the role that migration plays in keeping populations healthy. Monarch migrations are now considered an endangered phenomenon (60) due to deforestation of overwintering grounds, loss of critical breeding habitats, and climate-related shifts in migration phenology. If climate warming extends monarch breeding seasons into fall and winter months, migrations may eventually cease altogether. Evidence to date indicates that the loss of migration in response to mild winters and year-round resources could prolong exposure to parasites, elevate infection prevalence, and favor more virulent parasite genotypes. Images reproduced from (63, 64). [Photos by S. Altizer]

staging and stopover sites. Parasites that decline in response to host migration may include specialist pathogens, as well as those with transmission stages that can build up in the environment, pathogens transmitted by biting vectors or intermediate hosts, or for which transmis-

sion occurs mainly from adults to juveniles during the breeding season (e.g., Box 1). Conversely, migrating hosts could experience higher pressure from generalist parasites if opportunities for cross-species transmission are high at stopover areas or wintering grounds or from special-

ist pathogens if transmission increases with dense host aggregations that accompany mass migrations. Importantly, effects of migration on pathogen dynamics within host populations should translate to large differences in prevalence across host populations with different migratory strategies. Over the past few years, we have focused on monarch butterflies (*Danaus plexippus*) as a model system to study the effects of migration on host-pathogen interactions (Box 1) and found that both migratory culling and migratory escape can cause spatio-temporal variation in prevalence within populations and extreme differences in prevalence among populations with different migratory strategies. However, we are not aware of intraspecific comparisons of prevalence between migratory and nonmigratory populations for other animal species.

### Immune Defense Balanced Against the Demands of Migration

In addition to ecological mechanisms affecting between-host transmission, the physiological stress and energetic demands of migration can alter the outcome of infection within individuals through interactions with the host's immune system (Fig. 2). More generally, because several immune pathways in both vertebrates and invertebrates are known to be costly (27, 28), seasonal demands such as premigratory fattening or strenuous activity will likely lower the resource pool available for mounting an immune response (29). In anticipation of migration, for example, some animals accrue up to 50% of their lean body mass in fat, increase muscle mass, and atrophy organs that are not essential during migration (1). Thus, before migration, animals might adjust components of their immune response to a desired level (i.e., immunomodulation), or the energetic demands of migration could reduce the efficacy of some immune pathways (i.e., immunosuppression).

To date, the effects of long-distance migration on immune defenses have been best studied in birds. In a rare study of immune changes in wild individuals during migration, field observations of three species of thrushes showed that migrating birds had lower baseline measures for several components of innate immunity (including leukocyte and lymphocyte counts), and exhibited lower fat reserves and higher energetic stress, relative to individuals measured outside of the migratory season (30). Captive experiments with Swainson's thrushes (*Catharus ustulatus*; Fig. 2) later demonstrated that cell-mediated immunity was suppressed with the onset of migratory restlessness (the agitated behavior of birds that would normally precede their migratory departure) (31), suggesting that predictable changes in immunity occur in preparation for long-distance flight. In this species, the energetic costs of migration can intensify seasonal immune changes: Migrating thrushes that arrived at stopover sites in poorest condition had the lowest counts of immune cells (32).

The extent of altered immunity before and during migration is likely to be both species- and resource-dependent and will further depend on the specific immune pathway measured. Red knots, for example, exhibited no change in either antibody production or cell-mediated immunity after long flights in a wind tunnel, a result that argues against migration-mediated immunosuppression (33). Another study of captive red knots revealed no declines in costly immune defenses during the annual periods of mass gain (34); however, animals in this study had constant access to high-quality food, which might have negated energetic trade-offs between immune investment and weight gain. Interestingly, barn swallows (*Hirundo rustica*) in better physical condition showed greater measures of cellular immunity during migration, cleared ectoparasites and blood parasites more effectively, and arrived earlier at breeding grounds than birds with poor energy reserves (35). These studies suggest that animals in robust condition or with access to resources might tolerate long journeys without significant immunocompromise. Studies of migratory species to date also emphasize the need for a more detailed understanding of the mechanisms linking nutrient intake and metabolic activity to innate and adaptive immune measures, a step that is essential to predicting how different immune pathways will respond to physiological changes that occur before and during long-distance migrations.

Perhaps most importantly, immune changes that accompany long-distance migration could lead to a relapse of prior infections and more severe disease following exposure to new pathogens, increasing the likelihood of migratory culling and lowering the probability of spatial spread. This possibility was investigated for Lyme disease in redwings (*Turdus iliacus*) (36). Consistent with results showing negative effects of migratory status on immunity, migratory restlessness alone was sufficient to reactivate latent *Borrelia* infections in captive birds. Thus, the demands of migration could ultimately lead to more severe infections and greater removal of infected hosts. Together, these results point to a role for migration-mediated immune changes in the dynamics of other wildlife pathogens, including zoonotic agents such as WNV (12) and bat-transmitted corona and rabies viruses (37, 38).

### Effects of Anthropogenic Change and Climate

Changes to the ecology of migratory species in the past century (Fig. 2) could have enormous impacts on pathogen spread in wildlife and livestock, as well as altering human exposure to zoonotic infections. As one example, habitat loss caused by urbanization or agricultural expansion can eliminate stopover sites and result in higher densities of animals that use fewer remaining sites along the migration route (10). Although the resulting impacts on infectious diseases remain speculative, dense aggregations of animals at dwindling stopover sites might create ecological hot spots for pathogen transmission among wildlife species, as illustrated in the case of AIV in

migrating shorebirds at Delaware Bay (20). Moreover, continuing human encroachment on stopover habitats increases the likelihood of contact and spillover infection from wildlife reservoir hosts to humans and domesticated species.

For some animal species, physical barriers such as fences (terrestrial species) or hydroelectric dams (aquatic species) impede migration (39), leaving animals to choose between navigating a narrow migratory corridor or forming nonmigratory populations. Consequently, pathogen prevalence could increase when animals stop migrating and become confined to smaller habitats, if parasite infectious stages build up with more intense use of a given habitat. Attempts to control cattle exposure to brucellosis from bison (*Bison bison*) and elk (*Cervus elaphus*) in the Greater Yellowstone Ecosystem illustrate these risks. Due to the potential threat of *Brucella* transmission from bison to cattle, bison are routinely culled if they leave the confines of Yellowstone National Park (40). Elk migration is less restricted, but there is evidence that supplemental feeding areas encourage the formation of dense nonmigratory populations that support higher prevalence of brucellosis, with 10 to 30% seroprevalence in animals at the feeding grounds compared with 2 to 3% seroprevalence in unfed elk ranging the park (41). High population densities in elk also correlate with higher gastrointestinal parasite loads at feeding grounds (42), suggesting that high densities of nonmigrating hosts lead to increasing intraspecific transmission of multiple parasites.

More generally, human activities that discourage long-distance animal movements and encourage the formation of local year-round populations can cause the emergence of zoonotic pathogens in humans. For example, human-mediated environmental changes facilitated outbreaks of two zoonotic paramyxoviruses carried by flying foxes (*Pteropus* fruit bats; Fig. 2). These animals are highly mobile and seasonally nomadic in response to local food availability (43). Anthropogenic changes such as deforestation and agricultural production likely influenced the emergence of lethal Nipah and Hendra virus outbreaks in humans in Australia and Malaysia in two key ways: by resource supplementation and habitat alteration limiting migratory behaviors of fruit bats and by facilitating close contact with domesticated virus-amplifying hosts (pigs and horses). In Malaysia, resident flying foxes foraging on fruit trees on or near pig farms transmitted Nipah virus to pigs, probably via urine or partially consumed fruit with subsequent spread from pigs to humans [(43) and references therein]. Human activities are also thought to increase the risk of Hendra virus outbreaks in Australia by driving flying foxes from formerly forested areas into urban and suburban areas (44), where they form dense nonmigratory colonies that aggregate in public gardens containing abundant food sources.

In marine systems, aquaculture increases exposure to parasites in wild fish species, partic-

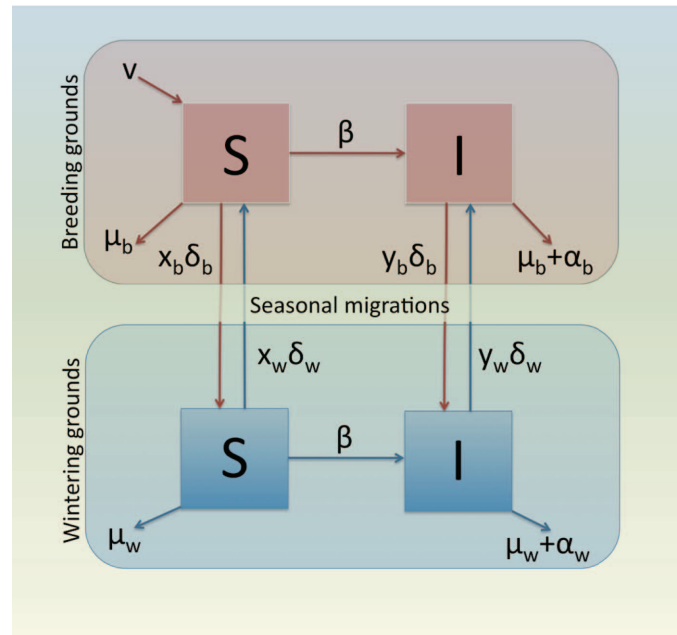
ularly in salmonids. Migration normally protects wild juvenile salmon from marine parasites in coastal waters by spatially separating them from infected wild adults offshore (45), but densely populated salmon farms place farmed fish enclosures adjacent to wild salmon migratory corridors, increasing the transmission of parasitic sea lice (*Lepeophtheirus salmonis*) to wild juveniles returning to sea (45).

Finally, climate change will alter infectious disease dynamics in some migratory species (46). To survive, many migratory species must respond to climate changes by shifting migratory routes and phenology in response to temperature and the availability of key resources (i.e., flowering plants, insects) [e.g., (47)]. It is possible that changes in the timing of migration could disrupt the synchronicity of host and parasite life cycles, much in the way that ecological mismatch in migration timing or altered migratory routes could impact whether suitable food and habitat are available when migrants arrive. For example, the spawning periodicity of whale barnacles in calving lagoons of gray whales is a classic example of a parasite synchronizing its reproduction to overlap with a host's migratory cycle (48). If the timing of whale migrations and barnacle reproduction shift in response to different environmental cues, this could result in reduced infections over time. On the other hand, altered migration routes might facilitate contact between otherwise geographically separated host species, leading to novel pathogen introductions and increasing disease risks for some wildlife species (46). One example of this phenomenon involves outbreaks of phocine distemper virus in harbor seals (*Phoca vitulina*) in the North Sea, which was likely introduced by harp seals (*Pagophilus groenlandicus*) migrating beyond their normal range and contacting harbor seal populations (49). Moreover, if climate warming extends hosts' breeding seasons, migrations may cease altogether, with year-round resident populations replacing migratory ones (Box 1), leading to greater pathogen prevalence through a loss of migratory culling and escape.

### Outlook and Future Challenges

Understanding the mechanisms by which long-distance movements affect host-pathogen systems offers exciting challenges for future work, especially in the context of global change and evolutionary dynamics. In terms of basic research, there remains a great need to identify conditions under which migration will increase host exposure

to infectious agents versus cases where migration can reduce transmission, with the ultimate goal of predicting the net outcomes for host species where multiple mechanisms operate on the same or different pathogens (e.g., Box 1). To that end, mechanistic models are needed to examine how migration affects infectious disease dynamics and to explore the relevance of possible mechanisms. Such models must combine within-season processes (including host reproduction, overwintering survival, and pathogen transmission) with between-season migration (Fig. 4). For example, to examine the importance of environ-



**Fig. 4.** A compartmental model illustrating infectious disease dynamics (S-I model) in a migratory host population moving between geographically distinct breeding and overwintering habitats. Susceptible hosts (S) in the breeding grounds are born ( $v$ ), die ( $\mu_b$ ) because of background mortality, and become infected at a rate,  $\beta$ . Infected hosts (I) suffer disease-induced mortality ( $\alpha_b$ ). Different fractions of susceptible ( $x_b$ ) and infected hosts ( $y_b$ ) survive migration from the breeding habitat and arrive successfully at an overwintering habitat at some rate ( $\delta_b$ ). Here, natural ( $\mu_w$ ) and disease-induced mortality ( $\alpha_w$ ) are both influenced by a different set of environmental conditions that characterize wintering grounds. The fraction of hosts surviving the spring migration the following year ( $x_w \delta_w$ ,  $y_w \delta_w$ ) will return to the breeding grounds to reproduce. A simple model like this can be readily modified to accommodate different parasite species and their transmission modes, host recovery, host age structure, and cross-species transmission.

mental transmission for the dynamics of LP AI in North American birds, Breban *et al.* (50) modeled a waterfowl population migrating between two geographically distant sites, with transmission dynamics occurring at both breeding and wintering grounds. Similarly, models describing interconnected networks of metapopulations could be useful in investigating disease dynamics between habitats linked through seasonal migrations (51). Although currently uncommon in the literature, epidemiological models can also be extended to capture mechanisms such as migratory culling and migratory escape and to include

multiple infectious agents to explore questions of coinfection and multihost transmission dynamics (Fig. 4).

One outstanding question is whether parasites can increase the migratory propensity of their hosts by favoring the evolution of migratory behaviors. Long-distance migration has previously been hypothesized to reduce predation risks for ungulates and birds, with the general rationale being that the survival costs of migration should be outweighed by fitness benefits associated with reproduction. In support of this idea, field studies of wolf predation on North American elk at their summer breeding grounds (52) and nest predation on migrating songbirds (2) showed that animals traveling farthest experienced the lowest predation risk. Similar observational studies could ask how the prevalence, intensity, virulence, and diversity of key parasites change with migratory distances traveled. To that end, comparing infection dynamics between migratory and nonmigratory populations of the same species offers a powerful test of both pattern and process (e.g., Box 1), although researchers will need to keep in mind that climate differences (e.g. milder climates for habitats used by nonmigratory populations) could confound some comparisons. Modeling approaches are also needed to explore how seasonal migration might respond evolutionarily to parasite-driven pressures, similar to other studies that examined effects of within-site competition, costs of dispersal, and variation in habitat quality on random dispersal strategies (53).

Another question related to host evolution is whether the combined demands of migration and disease risk could select for greater or lower investment in resistance or immunity. Field and laboratory studies have already documented between-season changes in immune investment, suggesting that some migratory species suppress specific immune responses before or during migration (30). The reduction in investment in immune defense could be an adaptive response to lower risks from certain parasites in migratory species (beyond issues related to energetic trade-offs) and might affect adaptive immunity (shown to be costly for many vertebrate species) more strongly than innate defenses. Over longer time scales, long-distance migration could select for greater levels of innate immunity in migratory species or populations, especially if migrating animals encounter more diverse parasite assemblages (54). With this in mind, comparisons of adaptive and innate immune defense and re-

sistance to specific pathogens between migratory and nonmigratory populations represent a challenge for future work that could be especially tractable with invertebrate systems (55).

Pathogens might also respond to migration-mediated selection, with ecological pressures arising from migration leading to divergence in virulence. There is some evidence to show that less-virulent strains circulate in migratory populations than in resident populations. The negative correlation between virulence and host migration distance, illustrated in the monarch system (Box 1), highlights the troubling possibility that pathogens infecting other migratory species could become more virulent if migrations decline. Moreover, dwindling migrations might affect host life history by altering pathogen virulence in once-migratory hosts. For example, a theoretical study showed that even moderate increases in virulence can change host breeding phenology to stimulate hosts to develop more quickly and breed earlier before they have a chance to become heavily infected (56). The recent facial tumor disease devastating Tasmanian devil populations provides a striking empirical example of high disease-induced mortality shifting host reproductive strategy from an iteroparous to a semelparous pattern through precocious sexual maturity in young devils (57). Although the hosts in this example are nonmigratory, they illustrate how virulent pathogens can generate longer-term fecundity costs beyond their direct impacts on host survival.

Studying the migratory process in any wild-life species poses exceptional logistical challenges, in part because distances separating multiple habitats can sometimes span thousands of kilometers, making sampling for infection or immunity intractable for field researchers. One problem is that historically, large numbers of animals have been sampled and marked at migratory staging areas, but for many species their subsequent whereabouts remain unknown (58). Tracking animals over long time periods and across vast distances has become more feasible with technological innovations such as radar and satellite telemetry for larger animals and ultra-light geolocators, stable isotopes, and radio tags to record or infer the movements of smaller animals (59). Furthermore, physiological measurements such as heart rate, wing beat frequency, and blood metabolites can be obtained remotely for some species, enabling scientists to examine how infection status influences movement rates and the energetic costs of migration (59).

Interdisciplinary studies to connect the fields of migration biology and infectious disease ecology are still in the early stages, and there are many exciting research opportunities to examine how infection dynamics relate to animal physiology, evolution, behavior, and environmental variation across the annual migratory cycle. Most

evidence comes from studies of avian-pathogen systems, especially viruses. Although this is not surprising given the relevance of pathogens such as avian influenza and WNV to human health, there remains a great need to explore other systems. Good places to start would be to make connections between disease and migration for species such as sea turtles, wildebeest, bats, dragonflies, and whales (Fig. 2). Parasite infections and movement ecology in species in each of these groups have been well studied separately but not yet bridged. Taking a broad view of diverse host life histories and parasite transmission modes will allow future studies to identify ecological generalities and system-specific complexities that govern the mechanistic relationships between host movement behavior and infectious disease dynamics.

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References

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