PHILOSOPHICAL TRANSACTIONS B

rstb.royalsocietypublishing.org

Review

Cite this article: Satterfield DA, Marra PP, Sillett TS, Altizer S. 2018 Responses of migratory species and their pathogens to supplemental feeding. *Phil. Trans. R. Soc. B* **373**: 20170094.
http://dx.doi.org/10.1098/rstb.2017.0094

Accepted: 9 October 2017

One contribution of 14 to a theme issue 'Anthropogenic resource subsidies and host–parasite dynamics in wildlife'.

Subject Areas:
ecology, health and disease and epidemiology, behaviour

Keywords:
host–parasite interactions, supplemental feeding, pathogen, *Danaus plexippus*, seasonal migration, resident

Author for correspondence:
Dara A. Satterfield
e-mail: dara.satterfield@gmail.com

Responses of migratory species and their pathogens to supplemental feeding

Dara A. Satterfield[1], Peter P. Marra[1], T. Scott Sillett[1] and Sonia Altizer[2]

[1]Migratory Bird Center, Smithsonian Conservation Biology Institute, National Zoological Park, Washington, DC, USA
[2]Odum School of Ecology, University of Georgia, Athens, GA, USA

DAS, 0000-0003-3036-5580; SA, 0000-0001-9966-2773

Migratory animals undergo seasonal and often spectacular movements and perform crucial ecosystem services. In response to anthropogenic changes, including food subsidies, some migratory animals are now migrating shorter distances or halting migration altogether and forming resident populations. Recent studies suggest that shifts in migratory behaviour can alter the risk of infection for wildlife. Although migration is commonly assumed to enhance pathogen spread, for many species, migration has the opposite effect of lowering infection risk, if animals escape from habitats where pathogen stages have accumulated or if strenuous journeys cull infected hosts. Here, we summarize responses of migratory species to supplemental feeding and review modelling and empirical work that provides support for mechanisms through which resource-induced changes in migration can alter pathogen transmission. In particular, we focus on the well-studied example of monarch butterflies and their protozoan parasites in North America. We also identify areas for future research, including combining new technologies for tracking animal movements with pathogen surveillance and exploring potential evolutionary responses of hosts and pathogens to changing movement patterns. Given that many migratory animals harbour pathogens of conservation concern and zoonotic potential, studies that document ongoing shifts in migratory behaviour and infection risk are vitally needed.

This article is part of the theme issue 'Anthropogenic resource subsidies and host–parasite dynamics in wildlife'.

1. Introduction

Animal migrations are widespread, spectacular and have important ecological consequences for processes ranging from pollination to nutrient transfer and trophic cascades [1–4]. Seasonal migration can also profoundly affect interactions between hosts and pathogens [5–7]. On the one hand, migration is commonly assumed to enhance the geographical spread of pathogens and can expose animals to diverse parasites as they move annually between breeding and wintering grounds (e.g. [8–10]). On the other hand, migration can lower infection risk for many animals, in part, by removing infected individuals during strenuous journeys and by interrupting pathogen transmission for part of the year (reviewed in [5]). These processes have been demonstrated in theoretical work and empirical studies on diverse systems, across insect, fish, bird and mammal hosts and their protozoan, viral, bacterial and macroparasites (reviewed in [5,6,11]).

Animal migrations worldwide are changing in response to human activities including climate change, habitat alteration (e.g. [12–15,16]) and, interestingly, food subsidies. Humans can intentionally or unintentionally provide food for wildlife, in the context of urbanization, agriculture, recreation, or wildlife management, with food sources including bird feeders, wildlife feeding stations, dumpsters and agricultural fields [17]. Shifts in food availability can spur shifts in animal migrations [18], most of which probably evolved due to natural variation in food resources [1]. Some animals have responded to supplemental feeding by halting migration altogether, forming resident populations that live

THE ROYAL SOCIETY PUBLISHING

year-round in the same location (e.g. [19–21]). Other populations have responded by migrating shorter distances (short-stopping) [22]. Rapid transitions towards residency and short-stopping behaviours are now observed in numerous populations of migratory insects, birds and mammals (e.g. [23,24]). For example, Spanish white storks (*Ciconia ciconia*) typically migrate to Africa each winter, but now forego migration and subsist on city landfills in Spain year-round [25,26]. European blackbirds (*Turdus merula*), Anna's hummingbirds (*Calypte anna*), grey-headed flying foxes (*Pteropus poliocephalus*) and other species are showing similar behavioural shifts tied to supplemental feeding [27–29].

Evidence that seasonal migration lowers the transmission of some pathogens emphasizes the importance of studying how shifts to shorter migrations and year-round residency alter the ecology of infectious diseases [5,6,30]. One concern is that resident populations could support greater parasite burdens due to persistently high host densities, longer breeding seasons and the absence of mechanisms that reduce parasitism during migration [5,30,31]. Populations showing short-stopping behaviours might also experience increased infection prevalence, e.g. if shorter migratory journeys allow infected migrants to survive longer. Moreover, a crucial need exists to investigate how resident animals affect the population dynamics of migratory populations, including whether greater disease risk in residents affects pathogen transmission to migrants and under which conditions resident populations might rescue migratory species from extinction.

Greater pathogen transmission arising from reduced migratory behaviour can have dire impacts on wildlife conservation and human health [23,32–35]. In one prominent case study, permanent 'camps' of historically nomadic flying foxes have formed in cities along the Australian east coast, where they feed on fruiting trees in suburban gardens [29]. This change in bat movement and ecology from nomadism to year-round residency has triggered deadly Hendra virus outbreaks in horses and humans [35,36]. Threats to species of conservation concern arise when transmission occurs from infected residents to migratory conspecifics. For example, high densities of sedentary farmed salmon can support heavy loads of parasitic sea lice in outdoor pens near river mouths on the Pacific Coast, increasing transmission to wild juvenile salmon (many of which have declining populations) migrating along adjacent routes [37,38].

Here, we synthesize current knowledge about changes in migratory behaviours associated with supplemental food resources and outline mechanisms by which the loss or reduction in migration could alter host–parasite interactions. To illustrate changes in infection outcomes, we highlight case studies on migratory fish, birds and mammals and their parasites. In particular, we focus on monarch butterflies (*Danaus plexippus*) and a debilitating protozoan parasite as a case study for which recent increases in resident behaviour have facilitated high infection prevalence that could ultimately pose risks for remaining migrants. Our synthesis underscores the need for additional work to (i) assess the extent to which other animals are undergoing changes in migration in response to human activities, (ii) test hypotheses about how changes in migration alter infectious disease dynamics, (iii) develop predictions about the types of pathogens and hosts that will be most affected by shifts in migration and (iv) evaluate if greater pathogen transmission in resident populations poses a risk to other species and populations. Understanding the links between changes in migratory behaviour and infection risk is an essential research task to address concerns for public health and wildlife conservation, particularly as residency behaviours of migrants are expected to become more common [39].

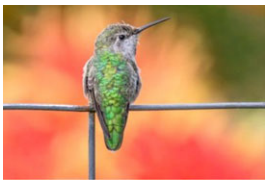
2. How are animal migrations changing in response to resource subsidies?

Anthropogenic forces have changed the timing and extent of many seasonal migrations (e.g. [14,40–42]). Climate warming, in particular, has been linked to more limited migrations of several bird species in Europe, with higher frequencies of birds remaining closer to breeding grounds during winter [43,44]. Food subsidies from intentional or accidental sources can cause similar changes in migratory movements, by reducing the seasonal fluctuations in resources that often drive migration [17,45]. These resources (e.g. provided through bird feeders, urban waste or crops) can be more locally abundant or more continuously available than natural foods [17], and in many cases have enabled migratory animals to halt, shorten or delay migrations. Reports of populations increasing their tendency to become resident in response to human-provided resources are accumulating in the literature (representative examples in table 1). These shifts in migratory behaviours can happen over relatively short timescales [19,54,55] and are taxonomically and geographically widespread (table 1). The implications of resource-driven changes in movement for the population biology and ecology of migratory species were recently identified as a key emerging issue in wildlife conservation, with potentially global effects [56].

Some migratory species are fed by wildlife managers to support populations or limit human–wildlife conflicts. For example, trumpeter swans (*Cygnus buccinator*) in North America traditionally migrate from breeding grounds in Canada to wintering grounds in Idaho and Oregon. In the 1930s, wildlife managers began feeding swans during winter owing to declines driven by overharvesting. The swan population rebounded, but many birds ceased to migrate and formed resident sub-populations in areas as far north as Montana [53,57]. In a similar example (reviewed in this issue, [58]), elk (*Cervus canadensis*) herds in the Greater Yellowstone Ecosystem are fed hay and alfalfa during the winter to prevent elk from entering private lands. Supplemental feeding of elk can delay the onset of migration, shorten migratory journeys and cause animals to aggregate around feeding stations, elevating their density and contact rates [49]. Likewise, supplemental feeding of both white-tailed deer (*Odocoileus virginianus*) and mule deer (*O. hemionus*) has been found to delay spring migration, thus prolonging duration of stay on the winter range [59,60].

Several types of migratory animals, including butterflies, sharks, birds and bats have responded to recreational feeding. As one example, evening grosbeaks (*Coccothraustes vespertinus*) began wintering farther north and shortening their migration in Europe, probably in response to backyard feeding stations [61]. Eurasian blackcaps (*Sylvia atricapilla*) have altered their migration to travel northwest from Germany and Austria to Britain, rather than southwest to Spain—a change driven by both warming temperatures and supplemental feeding at bird feeders [51]. In western North America, Anna's hummingbirds (*Calypte anna*) traditionally migrate to the south for the winter, but citizen science data indicated that in recent years

Table 1. Examples of case studies in which changes to animal migration occurred in response to human-provided resource subsidy. (Monarch photo: Pat Davis; Other images: Public domain). (Online version in colour.)

species		provisioned resource	changes in migration	references
Anna's hummingbirds *Calypte anna*		bird feeders	Anna's hummingbirds have become more abundant in winter at northern latitudes, suggesting a reduced migratory propensity associated with human landscapes	Greig *et al.* 2017 [28]
monarch butterfly *Danaus plexippus*		exotic milkweed plants	the planting of exotic milkweed plants that grow year-round in the southern USA enables some monarchs to forego migration to breed during the winter, leading to high protozoan parasite burdens	Satterfield *et al.* [23,46]; Batalden *et al.* [47]
elk *Cervus elaphus*		winter supplements	elk fed winter supplements in Wyoming were found to delay migration, reduce migratory duration on the summer range and form dense aggregations	Cross *et al.* [48]; Jones *et al.* [49]; Hebblewhite *et al.* 2006 [50]
Eurasian blackcap *Sylvia atricapilla*		bird feeders	typically migrants to the Mediterranean, blackcaps increasingly overwinter in Britain, associated with bird feeders	Plummer *et al.* [51]
European blackbirds *Turdus merula*		*possibly* fruits, earthworms, seeds, feeders in gardens and on lawns	blackbirds used to be migratory between central Europe and the Mediterranean. Now 50% of birds stay near breeding grounds all year, possibly driven by foods and climate	Berthold [39]; Partecke and Gwinner 2007 [52]
grey-headed flying foxes *Pteropus poliocephalus*		fruit in gardens	traditionally nomadic migrants, these fruit bats have established resident camps in urban Australia in response to supplemental fruit in gardens	Van der Ree *et al.* 2006 [29]
trumpeter swans *Cygnus buccinator*		wheat (intentionally fed)	after a severe decline, swans were fed wheat at a wildlife refuge in Montana, leading to a population increase but also a non-migratory population vulnerable to harsh winters	Baskin 1993 [53]
white storks *Ciconia ciconia*		garbage	while the birds typically migrate from Europe to Africa, a resident population formed in Spain, where the birds feed in landfills year-round	Tortosa *et al.* [25,26]

the hummingbirds have become more abundant at northern latitudes during winter. This change, associated with a concomitant increase in artificial nectar feeders over the same time span, suggests a reduction in the proportion of birds that are migratory [28]. Ruby-throated hummingbirds (*Archilochus colubris*) have shown similar changes, with some now found overwintering along the Gulf coast instead of migrating further south [62]. Recreational feeding extends also to oceans. Impacts on migration are suspected in some cases but not well understood. For instance, bull sharks (*Carcharhinus leucas*) adjusted local movement patterns at a food provisioning site for tourists in Fiji [63], and effects of feeding on the long-distance migration of bull sharks have been hypothesized but remain untested [64]. Similarly, non-migratory sicklefin lemon sharks (*Negaprion acutidens*) and Southern stingrays (*Dasyatis americana*) demonstrated higher site fidelity associated with provisioning [65,66], suggesting a need to assess whether supplemental food sources also alter longer-distance movements in marine animals.

Other migratory populations can use foods that are unintentionally provided. Lesser snow geese (*Chen caerulescens*) in North America, for instance, have shortened their migrations to forage in rice fields in the Midwest and Great Basin areas during the winter, whereas the geese previously wintered along the Gulf coast or in Mexico [67]. Endangered black storks (*Ciconia nigra*) in western Europe have also responded to rice fields and other land use changes, with an increasing number of birds wintering in the Iberian Peninsula since 1990, rather than migrating to northern Africa to overwinter [68,69]. Whooping cranes (*Grus americana*), which historically migrated as far south as South Carolina (and later, Florida), have shortened their migratory distances since being reintroduced 14 years ago and now often winter in the Midwest, significantly closer to their breeding grounds. This change, first implemented by older and more experienced birds, has allowed cranes to use new overwintering areas characterized by high grain cover and warmer temperatures associated with climate change [22].

As reports of residency and shorter migrations have become more common, migration itself has become increasingly challenging for animals. Migrants encounter geographical barriers (e.g. fences), habitat alteration and other threats (e.g. mortality due to cats, buildings, wind turbines; [14,70–72]). In these cases, residency might confer a fitness advantage over migration, enhancing survival or reproduction [73–75]. Importantly, evolutionary losses of traits important for migration might occur in newly formed resident populations and reinforce the degradation of long-distance movement [76–78], although this remains an important question for future work. We also need to understand whether and how animals that curtail or abandon migrations interact with remaining long-distance migrants and whether these interactions are harmful for migratory populations more broadly.

3. Mechanisms by which animal migrations alter host–pathogen interactions

Long-distance migration can have profound consequences for species interactions, including for the ecology of infectious disease [3,5]. On the one hand, animal migration might increase infection risk, if considering the simple *abundance and distribution* of hosts and parasites across the migratory pathway. This can occur when migration facilitates parasite dispersal over long distances or increases host exposure to a greater diversity of parasites as migrants encounter different habitats across a broad geographical range [79–81]. On the other hand, a growing body of evidence from across taxonomic groups indicates that migration often does the opposite and reduces infection risk [5,82]. This body of work considers not only host–parasite distributions and ranges but also *processes* during migration, such as host survival, host interactions with conspecifics, and parasite persistence and decay in the environment. Underlying these host–parasite interactions is the complex and continually changing status of immune function, and migration can be a time when animals experience greater susceptibility owing to the energetic demands of a long-distance journey [83–86].

Long-distance migration can decrease infection risk through several mechanisms (figure 1), including by affecting (i) transmission opportunities, (ii) survival of infected hosts, (iiii) spatial separation between susceptible and infected hosts or age classes, and (iv) recovery of infected hosts, reviewed in [5,6,87]. First, prolonged use of habitats allows parasite infectious stages to accumulate in the environment over time. Consequently, migration can enable animals to periodically escape from contaminated habitats (i.e. *migratory escape*; [31,88]). Between intervals of habitat use, harsh winters and long absences of hosts could eliminate most parasites, resulting in nearly disease-free conditions by the time hosts return. Support for migratory escape has been observed for migratory ungulates, including caribou (*Rangifer tarandus*), red deer (*Cervus elaphus*) and elk, which annually escape infestation from warble flies and roundworms, ticks and liver flukes (respectively; [89–93]). Second, long-distance migration can lower parasite prevalence by removing infected animals from the population (i.e. *migratory culling*). This happens when diseased animals delay the onset of migration or are unable to complete the journey, owing to the combined energetic costs of migration and infection [31]. Evidence in support of migratory culling has been reported in fall armyworm moths (*Spodoptera frugiperda*) affected by parasitic nematodes [94] and in Bewick's swans (*C. columbianus*) affected by low-parasitic avian influenza viruses [95,96].

Third, some migratory populations travel to spawning grounds, where adults die or depart after depositing offspring. This strategy results in a spatial separation between juveniles that are highly vulnerable to infection and adults that harbour disease-causing agents (*migratory allopatry*). Evidence for migratory allopatry in lowering infection risk comes from long-term studies of sea lice in Pacific salmon [38,97]. Such ontogenetic migration, in which juveniles and adults separate, is common among benthic invertebrates and anadromous fishes [98,99], and decreased parasite transmission may be a selective force for such migrations [100]. Fourth, migration can also decrease infection risk by shortening the period of infectiousness. In *migratory recovery*, individuals recover from infection during migration, such as when environmental conditions *en route* are unfavourable to parasites [101]. This could shorten infectious periods and thus reduce the window of opportunity for pathogen transmission. Although direct evidence in support of this mechanism is lacking to date, many species that migrate seasonally between distinct environments show potential for migratory recovery to operate (summarized in [101]).

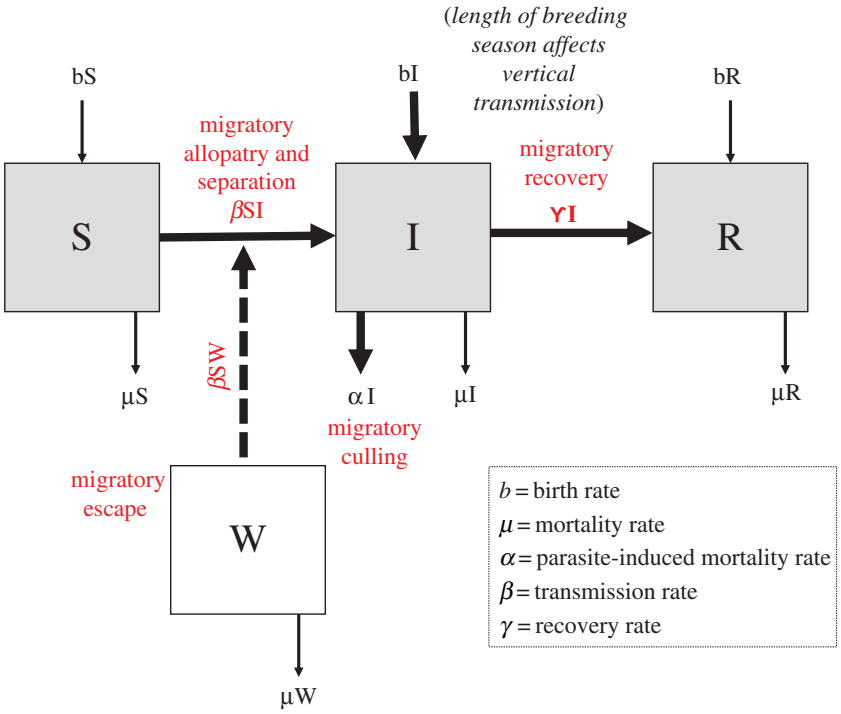


Figure 1. Mechanisms (red text) through which migration lowers infection risk, illustrated with a transmission model for susceptible (S), infected (I) and recovered (R) hosts, and allowing for pathogen transmission through direct contact or via encounters with parasite infectious stages shed into the environment (W). (i) Migratory escape allows susceptible hosts (S) to escape environments where infectious stages accumulate over time, limiting both shedding (λ, not shown) and transmission rate (βSW) for environmentally transmitted parasites. (ii) *Migratory culling* lowers the number of infected individuals (I) by increasing infection-induced mortality (α) during strenuous migration. (iii) Migratory allopatry and separation reduce transmission by separating more susceptible (S) and more infected (I) individuals in different age classes during migration, thus limiting contact rates. (iv) Migratory recovery reduces the number of infected individuals (I), as infected animals recover (at rate Υ) due to conditions during migration.(Online version in colour.)

4. Changing migrations can alter infection risk: hypotheses, theory, and empirical support

The loss or degradation of migrations in response to anthropogenic resource subsidies could have a wide range of effects on disease occurrence (figure 2), with outcomes probably depending on host life history, modes of pathogen transmission and the strength of trade-offs between resources and costly immune defence. Emerging syntheses have underscored the need to examine how food provisioning alters infection risk for migratory species [5,102]. Most recently, a meta-analysis showed that the relationship between supplemental feeding and higher risk from microparasites was stronger for dietary generalist species and, to some small degree, for migratory species; however, the association with migratory status was relatively weak [103]. Additional work is needed to understand interactions between supplemental feeding, disease risk and animal migrations—and how the deterioration of these migrations affects populations.

In some cases, the loss of migration might reduce pathogen burdens, if less mobile hosts encounter fewer parasite species or if reduced host movement frees up resources for defence against infection. However, we predict that shorter migrations or shifts from migratory to resident behaviour will be more likely to increase disease risk, owing to the breakdown of mechanisms that lower pathogen transmission [6]. Empirical support for mechanisms by which altered migrations are changing infection risk is extremely limited. However, the few case studies available demonstrate strong disease responses to the loss of animal migration. Below, we outline several hypotheses, examine theoretical and empirical support to date, and propose a framework to guide future work in this area.

(a) Loss of migratory escape, culling and recovery

The most immediate effect of diminished movement behaviour could occur by the loss of migratory escape. If migrants shift to year-round residency or longer breeding seasons, they might be exposed to higher numbers of infectious stages that accumulate in the environment (loss of migratory escape). The build-up of environmentally transmitted pathogens in even high-quality habitats can create ecological traps for animals, as recent modelling work showed [104]. Migratory escape and consequences for its loss would probably be most pronounced for specialist parasites that do not use alternative hosts during the absence of migrants [5], causing parasites to die in the environment by the time their obligate hosts return. Importantly, the loss of migratory escape could alter the relative importance of different modes of pathogen transmission. Some pathogens such as baculovirus in African armyworms and neogregarines in monarchs (discussed below) use two modes of transmission, with environmental (or horizontal) transfer operating during one part of the annual cycle and vertical transmission occurring at other times of the year. Transitions to more resident behaviours could increase environmental transmission (due to pathogen accumulation) relative to other modes of transmission, potentially providing an opportunity for parasites to evolve higher virulence [23].

Recent theoretical work has explored disease outcomes when migratory escape is lost. A transmission model (by Hall *et al.* [6]) for pathogens in a two-way migratory host population found strong support for migratory escape in lowering infection risk; that is, infection prevalence was lower for populations that left the breeding grounds sooner. Results from the model suggested that environmental changes

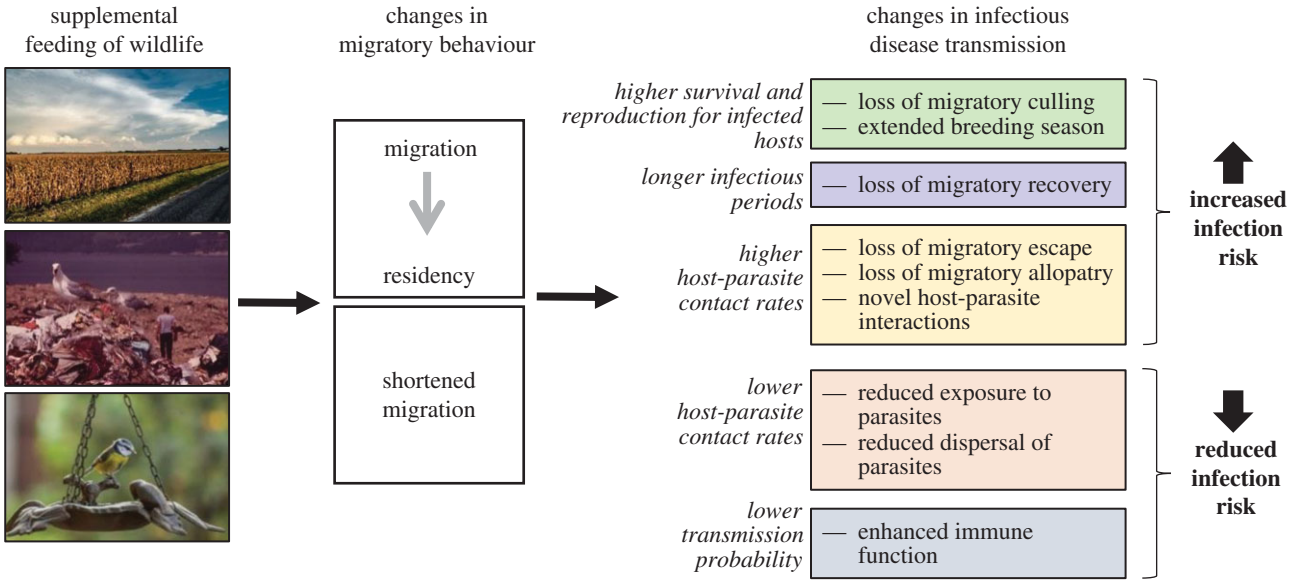


Figure 2. Conceptual diagram illustrating how supplemental resources from unintentional (e.g. crops, landfills) and intentional sources (e.g. bird feeders) can cause changes in migratory behaviour, with consequences that raise or reduce infection risk. Diminished migrations, including shifts to residency and short-stopping behaviours, can result in some processes that increase transmission, such as the loss of migratory culling and migratory escape. Other mechanisms may reduce transmission, such as if resident animals experience higher immunity. *(Image credits: Public domain).*

reducing migratory propensity would probably cause higher infection prevalence for parasites transmitted during the breeding season. This was tested in another similar transmission model that also demonstrated migratory escape, such that infection prevalence was lower when hosts spent more time migrating [11]. Resident populations in both models, represented by no time spent migrating, experienced high infection prevalence owing to the loss of migratory culling and migratory escape [6,11]. Future empirical work could examine the loss of migratory escape in wild populations undergoing rapid behavioural change. Investigators could measure the abundance and viability of parasite infectious stages in the environment at sites where parasites might accumulate and compare these measures with those from migratory sites at both seasonal breeding and wintering habitats.

In addition to the loss of migratory escape, if hosts stop migrating or travel shorter distances, the relative survival of infected animals could increase in the absence of the physiological and energetic demands of migration (loss of migratory culling). Hosts undergoing the most strenuous migrations (measured, for example, in terms of distance travelled or energy allocated during pre-migration) would be expected to experience the strongest migratory culling [105] and, as a result, the greatest responses to its loss. For species like silver Y moths (*Autographa gamma*) that take advantage of directional winds and use minimal self-propulsion [106], migratory culling (or its loss) might be less important. Similarly, parasite species most likely to benefit from diminished migrations are those that typically impair host mobility or cause severe reductions in energy reserves needed to undertake long journeys.

Modelling work predicts that the loss of migratory culling would increase pathogen prevalence: a mathematical model for vector-borne pathogens showed that shorter migration distances (keeping the period of breeding and transmission constant) led to higher infection prevalence, due to weakened migratory culling [105]. Other transmission models (for directly transmitted pathogens) also showed evidence for migratory culling [6,11], which held for both density-dependent and frequency-dependent transmission. However, infection prevalence declined more rapidly in response to migratory culling when parasites exhibited density-dependent transmission [11]. Empirical work to examine the loss of migratory culling has been hampered by the difficulty in tracking both healthy and infected individuals throughout their long-distance migrations, although new tracking technologies and molecular tools for estimating natal origins and migration distances make this work more feasible. The most direct tests of migratory culling in the field would compare survival during migration (or migratory distances travelled) of healthy versus infected individuals and scale these against survival estimates for resident animals that do not migrate. Alternatively, movement or flight performance of healthy and infected individuals could be measured experimentally in captivity [107,108].

In addition to migratory culling and escape, some hosts that stop migrating due to supplemental feeding could undergo a loss of migratory recovery, which occurs when infected hosts recover from infections during migration due to changes in environmental conditions that are unsuitable to the parasite [101]. Parasites that might be affected by migratory recovery could include, for example, ectoparasites such as copepods or barnacles that are sensitive to salinity changes and infect migratory flounders, manatees (*Trichechus*) and other hosts [101]. For newly resident populations, if migration no longer affords time for animals to recover between cycles of transmission, longer infectious periods could produce greater opportunities for parasite transmission. Despite theoretical support for migratory recovery [101], this mechanism has not been examined in the wild, and future work is needed to monitor the infection status of animals over time during the course of seasonal migrations.

(b) Loss of migratory allopatry

Newly resident animals could experience higher host–parasite contact rates due to the loss of migratory allopatry, which separates infected and susceptible individuals during migration. The loss of migration may place susceptible host life stages

and infected individuals in closer proximity or more frequent contact. This has occurred in Pacific Northwest salmon, for which migration normally separates adult salmon from susceptible juvenile salmon and truncates the transmission potential of sea lice between these groups (migratory allopatry; [109]). The introduction of fish farms in outdoor semi-enclosed sea pens, however, has altered this relationship. High densities of farmed salmon (acting, essentially, as year-round resident salmon) can support heavy loads of parasitic sea lice. When pens are located near river mouths, sea lice from adult farmed salmon can transmit to wild juvenile salmon migrating along adjacent routes [32,38]. The farms have been linked to high lice-induced mortality of wild chum and pink salmon and were found to raise infection risk along an 80 km stretch of the wild fish route, in part due to the breakdown in migratory allopatry [32].

(c) Increased host density and contact rates

Newly formed resident populations could reach higher local densities than migrants and, as a result, experience greater contact rates, host susceptibility and pathogen transmission. Higher density following shifts towards residency could be due to longer breeding seasons, greater recruitment of susceptible animals and aggregation around supplemental resources (as discussed elsewhere [102,110,111]). Implications of higher host density around resource subsidies for pathogen transmission have been examined in host–pathogen models (e.g. [111]) and in several host–parasite systems. For example, elk in Wyoming given supplemental resources have formed high-density, sedentary winter populations with higher brucellosis transmission compared with free-ranging elk [48].

Animals living at high densities might experience more intense competition for resources, leading to physiological or nutritional stress. Crowding and stress might increase susceptibility to infection and allow for larger outbreaks and more severe individual fitness consequences. In insects, this hypothesis was initially examined (by Steinhaus [112]) in studies of caterpillars and their natural pathogens and is now supported by work from insects, birds, fish, mammals and amphibians (e.g. [113,114]). Whether newly resident populations supported by human-provided resources experience crowding, stress and greater susceptibility to infection remains an area open for future work.

(d) Less frequent encounters with parasites and limited dispersal

Although resident behaviours are expected to promote parasitism through several mechanisms, residency could reduce infection risk in other ways. In particular, residents could encounter a less diverse community of pathogens, compared to migrants, by remaining in the same environment year-round [79,115]. Cross-species comparisons of residents versus migrants provide some support for this hypothesis, with resident bird species harbouring lower parasite richness of nematodes [116] and helminths in general [117], compared with migratory species. However, other work found macroparasite richness to be determined by habitat type rather than migratory behaviour [118]. Within-species comparisons of parasite richness among residents versus migrants remain rare. However, at least two case studies within species suggest that changes in migratory propensity reduce infection risk for urban populations. European blackbirds, as one example, were once fully migratory; however, a significant proportion of blackbirds are now residents living in urban areas [39,119]. Driving this behaviour could be a combination of climate change and supplemental feeding at garden feeders, in orchards and on lawns (where the birds forage for earthworms; [39,120]). Evans *et al*. [27] showed that blackbirds had lower tick prevalence and intensity and lower avian malaria prevalence at some urban sites. A study of mallards in Sweden similarly demonstrated lower infection prevalence of avian influenza and avian coronavirus in resident ducks, frequently fed by humans in an urban pond, relative to wild ducks [121].

In addition to lowering host–parasite contact rates, diminished migration might lower pathogen dispersal rates, in cases where migrants frequently act as 'superspreaders' of infection that disperse pathogens long distances [86]. This could occur for avian influenza, for example, for which genetic evidence suggests migrants can transport pathogens from north to south in the autumn [122]. Thus, higher residency and lower migration rates among water birds might, in some cases, limit the dispersal of some pathogens or pathogen subtypes. However, higher residency might also provide more opportunities for pathogens to accumulate in environmental reservoirs and potentially for higher virulence to evolve (see following section; [5,123]). Additional work using transmission models to explore consequences of increased residency for avian influenza dynamics is needed.

(e) Changes to host resistance or pathogen virulence

Reliable resources and reduced movement might allow for greater host resistance to infection and lower prevalence in resident populations. In particular, energy available for immune defence can depend on trade-offs and interactions between immunity, migration and infection and might vary throughout annual migratory cycles [7]. If demanding migrations typically act to lower immune function (e.g. [124]), then shorter migrations or transitions to residency could enhance immunity, offsetting mechanisms that increase exposure to infection. In addition, resident hosts might be able to locally adapt to sympatric parasites which they continually encounter [125], an advantage through which parasitism could actually select against migrants [126].

Increased year-round transmission and the loss of migratory culling—a process that might normally remove animals infected with highly virulent pathogens—could allow more virulent forms of pathogens to invade populations that experience reduced migrations. Migration has been hypothesized to select for decreased parasite virulence [23,127,128], because successful parasite transmission might hinge upon an infected host's ability to survive strenuous journeys. By contrast, residency might release such evolutionary constraints and allow higher parasite virulence to evolve.

(f) Resident populations as reservoirs of parasites

In some cases, the formation of resident populations will establish new interactions within and between species, introducing novel opportunities for transmission. In particular, unique infection dynamics emerge when migrant and resident hosts interact, as suggested in multiple studies of waterfowl. Residents can act as reservoirs of pathogens and expose migrants to infection, as has been suggested for avian influenza subtypes that are maintained in year-round mallard populations and can

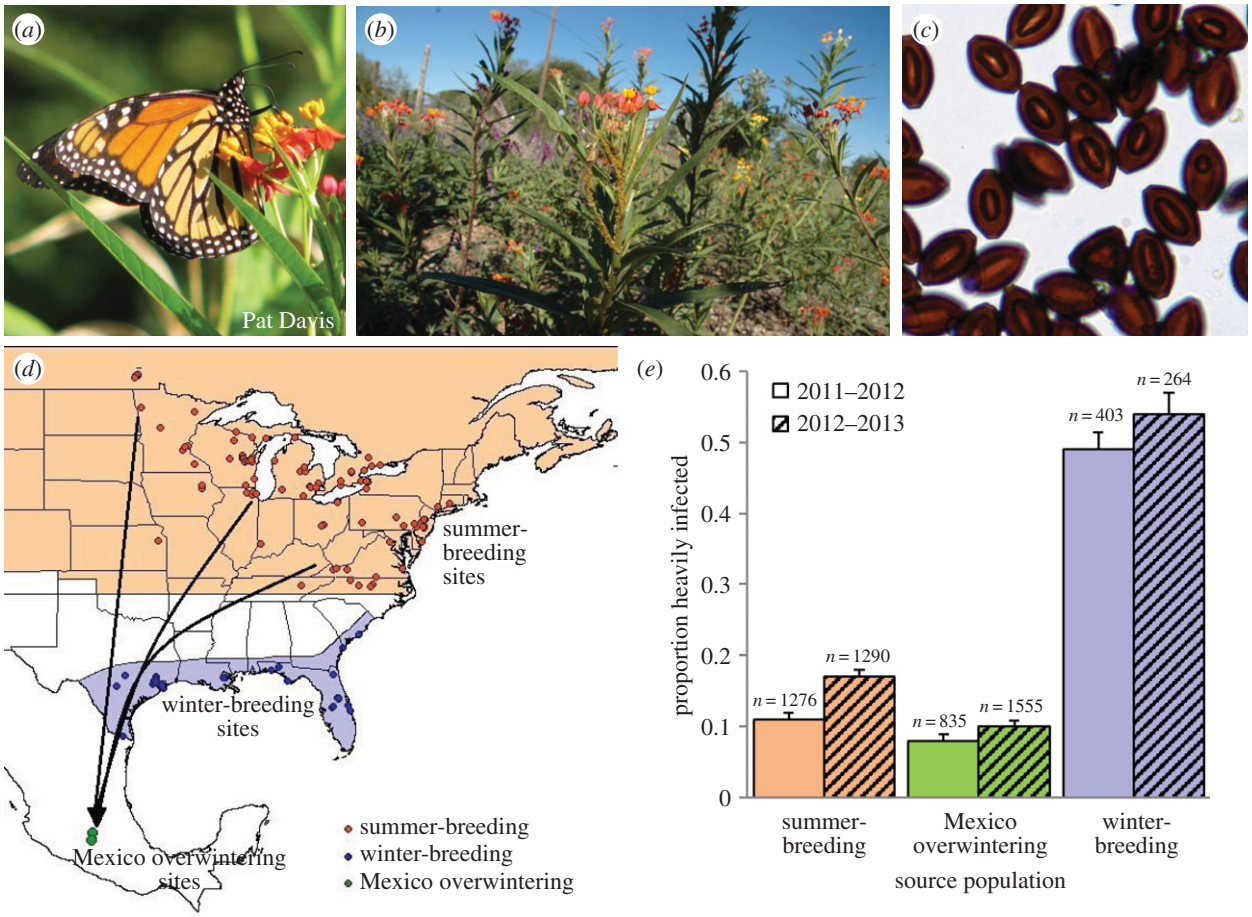


Figure 3. (*a*) Some monarch butterflies are responding to (*b*) the widespread availability of tropical milkweed (*Asclepias curassavica*) by breeding year-round in the same locations of the southern USA, rather than migrating, raising infection risk from (*c*) the protozoan parasite *Ophryocystis elektroscirrha*. A study involving citizen scientists [23] showed that resident monarchs at (*d*) winter-breeding sites (blue points) compared to migratory sites (orange points for summer breeding; green points for overwintering) show (*e*) five times higher infection prevalence on average. Image credits: (*a*) Pat Davis, (*b*) Dara Satterfield, (*c*) Sonia Altizer.

be transmitted to migratory birds [129–131]. In fact, several studies that emphasized the role of migratory birds in epizootics demonstrate that it was specifically migrants *interacting with residents* that determined infection dynamics for avian influenza or blood parasites [130–132]. As some animals shift towards non-migratory behaviours, one concern is that newly resident populations will become sources of infection.

Residents can act as reservoirs of pathogens not only within species but also across species (e.g. [133]), as with blood parasites in resident birds that can be transmitted to migratory species on wintering grounds in Africa [134]. Phylogenetic methods examining host specificity suggest that bird species migrating from Europe acquire parasite lineages of *Haemoproteus* and *Plasmodium* from resident bird species in tropical Africa during the non-breeding season [132]. In some cases, pathogen transmission from resident populations could drastically affect human health. As one example, resident colonies of fruit bats persisting on food in suburban gardens in Australia (rather than eating wild foods during nomadic movements) have led to deadly Hendra virus outbreaks in horses and humans [35]. Another case in point occurred for avian influenza transmission. In southern China, paddy fields constructed for raising high densities of domestic ducks are visited seasonally by migratory waterfowl, allowing for a 'complex spatio-temporal interplay' of transmission of highly pathogenic avian influenza between resident domestic ducks and wild birds [135]. Recent evidence suggests that, in 2008, resident–migrant interactions contributed to pandemic spread of AIV, with domestic ducks (numbering over 14 million annually) in the Poyang Lake area of China transmitting H5N1 to migratory birds that then transported the virus to other areas [135].

5. Monarch butterflies and their debilitating parasites: a case study illustrating how shifts from migratory to resident behaviour alter host–parasite interactions

The interaction between monarch butterflies and a debilitating protozoan parasite, *Ophryocystis elektroscirrha* (OE), serves as a model host–parasite system to investigate how resource subsidy-driven changes in migratory behaviour influence the spread and impacts of infectious disease (figure 3). Monarchs in eastern North America are iconic insects famous for their annual migration, during which hundreds of millions of butterflies travel from as far north as Canada to overwinter in the oyamel forests of central Mexico [136,137]. A shorter migration persists in the western USA, where monarchs winter along the California coast [138]. Monarchs also form genetically distinct, non-migratory populations around the globe, in areas such as the Caribbean and Pacific [139,140]. Throughout their range, monarchs are commonly infected with OE [141], which is transmitted from adults to larvae via spores scattered onto eggs and milkweed; spores must be

ingested by a larva to cause a new infection [141–143]. Adults parasitized with OE emerge covered with up to millions of dormant parasite spores on the outside of their bodies (figure 3). Transmission can be vertical (from adult females to the surface of their eggs) or environmental (when larvae consume spores deposited onto milkweed from unrelated infected adults; [142,144]). Infections lower adult monarch lifespan, size and flight ability [107,145,146].

Previous work showed that, across multiple migratory and non-migratory monarch populations, OE prevalence decreases with migratory propensity and annual distance flown [31,82,147], with the lowest infection prevalence occurring among eastern North American monarchs, which migrate the farthest distances [146]. Field and experimental studies provided evidence for both migratory culling and migratory escape in contributing to this pattern. Consistent with migratory culling, experiments with captive monarchs showed that infection lowers flight performance [107], and field studies showed that infection prevalence declines during the monarchs' annual autumn migration [31]. Furthermore, healthy monarchs wintering in Mexico originate, on average, from locations more distant from the wintering sites relative to infected monarchs [82]. Evidence for migratory escape comes from studies showing that prevalence increases during the monarch's breeding season in the northern USA and Canada, reaches a peak just prior to migration, and is lowest in the early spring, when the monarchs return from their wintering sites to breeding grounds with newly sprouted, parasite-free milkweed plants [31]. Thus, multiple lines of evidence demonstrate that seasonal migration protects monarchs from infection by OE parasites.

Like many other migrants, monarchs have experienced population declines coincident with the loss of breeding and overwintering habitats in both eastern and western North America [148–150]. Recent attention has focused on the loss of common milkweed (*Asclepias syriaca*) in agriculturally intense regions of the Midwestern USA [151–153]. Efforts to replace monarch breeding habitat have included planting milkweed in gardens and yards. One of the most popular milkweed in gardens is a non-native species, tropical milkweed (*A. curassavica*; figure 3), which is attractive, easy to grow and commonly sold in nurseries [47,154]. Unlike most native milkweeds that enter dormancy in the autumn, tropical milkweed persists throughout the year in mild climates [23,46,47]. Tropical milkweed also has high concentrations of secondary compounds called cardenolides that are attractive to monarchs, provide a defence against many predators, and prolong lifespan for infected individuals [155–157]. In the southern USA, especially along the Gulf Coast, newly formed resident monarch populations have become common [158], enabled by the year-round breeding habitat afforded by *A. curassavica* [23,46,47].

Starting in 2009, volunteers for the citizen science programme *Monarch Health* (www.monarchparasites.org) began reporting that winter-breeding monarchs in the southern USA showed signs of severe OE infections (S. Altizer 2009, unpublished data). A recent analysis of citizen scientist monitoring data from the eastern USA and Canada showed that non-migratory monarch populations experience, on average, five times higher OE prevalence than their migratory counterparts (figure 3; [23]). In some locations, infection prevalence reached 100%, and volunteer reports showed that monarch winter-breeding occurs exclusively at sites with tropical milkweed. A strikingly similar pattern was observed in the western USA: Resident monarchs breeding year-round in southern California face nine-times higher infection risk compared with migratory monarchs at coastal overwintering sites [46]. High infection prevalence has been attributed to the loss of both migratory escape and culling.

One concern is that heavily parasitized resident monarchs and OE-contaminated tropical milkweed plants could act as a source of infection for migratory monarchs or could cause migrants to abandon their journeys upon encountering sites with resident conspecifics. This is most likely to happen during autumn and spring, as migrants pass through sites occupied by resident monarchs. Exposure to tropical milkweed in the autumn might induce migrants to break reproductive diapause (a pre-migratory physiological state associated with the delay of reproductive development; [159]) and to stop migrating. Infected monarchs in poor condition, and hence less able to migrate successfully, might be especially likely to join resident monarchs. Furthermore, in the spring, tropical milkweed sites might expose migrants' offspring to high levels of OE infection risk. Migratory monarchs typically lay the first generation of eggs in the spring on milkweed in Gulf coastal states, where tropical milkweed and resident monarchs with heavy parasite loads are concentrated. Thus, important questions remain about whether resident monarchs supported by human-planted tropical milkweed pose infection risks for migratory monarchs and present an additional threat to monarch migration.

Collectively, work on the monarch–OE interaction suggests that limiting the availability of milkweed to be seasonal rather than year-round would better support monarch health and migration. Native milkweeds, which typically die back each autumn, do not allow for the year-round breeding and resident strategies associated with high levels of OE infection. In response to these results, many gardeners and citizen scientists—some of whom helped to uncover the link between disease and year-round tropical milkweed—are planting native milkweeds and promoting their availability throughout North America. Some gardeners are also removing or cutting back tropical milkweed in the southern USA during the autumn and winter.

6. Broader implications and directions for future work

Greater pathogen transmission arising from non-migratory behaviours can impair wildlife conservation and human health, especially when resident populations act as infection sources [23,33–35,37,46]. Most responses of supplemental feeding have been recorded among birds and in terrestrial environments, but changes in migration are probably occurring among other vertebrates, among insects and in aquatic environments—wherever humans provide resources. Findings summarized here call attention to an urgent need to assess the extent to which other animals are undergoing changes in migration and disease, and to identify strategies to preserve migrations when they are protective. As a first step, parasite surveillance is needed for populations showing changes in migratory behaviour. Such surveillance should focus on both zoonotic pathogens and the micro- and macroparasites exclusive to wildlife. Whole-genome sequencing will enable rapid detection of emerging pathogens with high fidelity and could aid in surveillance of wildlife diseases [160]. As additional

case studies become available, meta-analyses could uncover patterns and predictions about the circumstances, pathogen types and host taxa that determine how disease dynamics respond to changes in migration.

Focused laboratory and field studies would provide resolution about mechanisms underlying the interaction between disease and migration. The use of new tracking technologies (e.g. GPS transmitters, geolocators) and stable isotopes will enable comparisons of migration distances and flight performance *en route* among infected and uninfected individuals (i.e. directly examining migratory culling in the wild), and assessment of infection risk, immunity and physiological parameters among residents versus migrants. Laboratory experiments should examine whether animals that enter a pre-migratory state or invest in the energetic demands of migration have lower immune defences than resident animals.

New empirical work could inform mathematical models that explore the loss of mechanisms during shifts from migratory to resident behaviours. Models provide the opportunity to observe net outcomes of the loss of migration on disease, when there are co-occurring and opposing influences; for instance, a model could simultaneously explore in a population the loss of migratory culling (which could increase disease risk) and the more consistent availability of human-provided food at resident sites (which could lower host susceptibility to infection; this issue, [161]). More work is needed to understand how migratory and newly resident populations interact and to predict when residents might serve as sources of infection. Models that explore the dynamics of linked migrant-resident populations will be important to that end.

Finally, research is needed to investigate how supplemental feeding and changes in animal migration will alter parasite evolution. The loss of migration might release constraints on parasite virulence and allow more highly virulent parasites to thrive once hosts no longer undergo strenuous journeys. Reduced host movement behaviours might also allow local adaptation of parasites or changes in host breadth for some pathogens. Changes in host movement might further affect the population genetic structure and genetic diversity of pathogens. Except for some work with monarch butterflies, these questions remain almost entirely unexplored.

The case studies in this paper highlight implications for management, conservation and public health. For some animal populations, supplemental feeding has had beneficial effects that must be weighed against any altered risks. For ungulates and large birds, supplemental feeding is often used as a management tool that has helped populations rebound or stabilize following population crashes. When supplemental feeding alters migration, however, this practice should be assessed for effects on infectious disease dynamics. Other intentional feeding is recreational, such as at bird feeders, and has allowed opportunities for the public to connect with wildlife. Restricting the availability of intentionally provided resources to certain seasons or only during the non-migration period (if first deemed safe in analyses and models) could be one way to reduce effects on migratory behaviours and disease, while still supporting these populations and engaging the public. For instance, gardeners could provide critical habitat to help monarch butterflies in North America recover from their severe decline, and providing native milkweeds that grow seasonally will be more beneficial than exotic milkweeds that allow year-round monarch breeding. In other cases, resident populations supported by supplemental feeding are now so well established that they will probably persist far into the future. Future work is needed to evaluate the role of these populations in interacting with migratory animals. For example, residents could provide a rescue effect to shrinking migratory populations but also amplify transmission and act as a source of parasites for these animals. Some resident populations pose a risk not only to wildlife but also to humans. A better understanding of this phenomenon could help predict where and when spillover is most likely. Finally, the studies reviewed here suggest that protecting migratory behaviour is an essential part of protecting wildlife health.

Data accessibility. This article has no additional data.

Authors' contributions. D.S. and S.A. wrote the manuscript. All authors contributed to concept development and revisions.

Competing interests. The authors declare no competing interests.

Funding. This work was supported by funding to DS from the Smithsonian Institution National Board Fellowship through the James Smithson Fellowship Program; from a National Science Foundation Dissertation Improvement Grant (no. 1406862); and from a Monarch Joint Venture research grant.

Acknowledgements. We thank Daniel J. Becker, Sonia M. Hernandez and Richard J. Hall for co-organizing the symposium that inspired this work ('Resource Provisioning and Wildlife–Pathogen Interactions in Human-Altered Landscapes' at the 101st meeting of the Ecological Society of America in 2016).

References

1. Dingle H. 2014 *Migration: The biology of life on the move*, 2nd edn. Oxford, UK: Oxford University Press.
2. Holdo R, Holt RD, Sinclair ARE, Godley BJ, Thirgood S. 2011 Migration impacts on communities and ecosystems: empirical evidence and theoretical insights. In *Animal migration: A synthesis* (eds EJ Milner-Gulland, JM Fryxell, ARE Sinclair), pp. 131–142. Oxford, UK: Oxford University Press.
3. Bauer S, Hoye BJ. 2014 Migratory animals couple biodiversity and ecosystem functioning worldwide. *Science* **344**, 1242552. (doi:10.1126/science.1242552)
4. López-Hoffman L, Chester CC, Semmens DJ, Thogmartin WE, McGoffin MSR, Merideth R, Diffendorfer JE. 2017 Ecosystem services from transborder migratory species: implications for conservation governance. *Ann. Rev. Envir. Resour.* **42**, 509–539. (doi:10.1146/annurev-environ-110615-090119)
5. Altizer S, Bartel R, Han BA. 2011 Animal migration and infectious disease risk. *Science* **331**, 296–302. (doi:10.1126/science.1194694)
6. Hall RJ, Altizer S, Bartel RA. 2014 Greater migratory propensity in hosts lowers pathogen transmission and impacts. *J. Anim. Ecol.* **83**, 1068–1077. (doi:10.1111/1365-2656.12204)
7. Buehler DM, Tieleman BI, Piersma T. 2010 How do migratory species stay healthy over the annual cycle? A conceptual model for immune function and for resistance to disease. *Integr. Comp. Biol.* **50**, 346–357. (doi:10.1093/icb/icq055)
8. Dwyer G, Elkinton JS. 1995 Host dispersal and the spatial spread of insect pathogens. *Ecology* **76**, 1262–1275. (doi:10.2307/1940933)
9. Rappole JH, Derrickson SR, Hubálek Z. 2000 Migratory birds and spread of West Nile virus in the Western Hemisphere. *Emerg. Infect. Dis.* **6**, 319–328. (doi:10.3201/eid0604.000401)
10. Figuerola J, Green A. 2000 Haematozoan parasites and migratory behaviour in waterfowl. *Evol. Ecol.* **14**, 143–153. (doi:10.1023/A:1011009419264)

11. Johns S, Shaw AK. 2016 Theoretical insight into three disease-related benefits of migration. *Popul. Ecol.* **58**, 213–221. (doi:10.1007/s10144-015-0518-x)
12. Visser ME, Both C. 2005 Shifts in phenology due to global climate change: the need for a yardstick. *Proc. R. Soc. Lond. B* **272**, 2561–2569. (doi:10.1098/rspb.2005.3356)
13. Both C, Bouwhuis S, Lessells CM, Visser ME. 2006 Climate change and population declines in a long-distance migratory bird. *Nature* **441**, 81–83. (doi:10.1038/nature04539)
14. Wilcove DS, Wikelski M. 2008 Going, going, gone: is animal migration disappearing? *PLoS Biol.* **6**, e188. (doi:10.1371/journal.pbio.0060188)
15. Sims DW, Genner MJ, Southward AJ, Hawkins SJ. 2001 Timing of squid migration reflects North Atlantic climate variability. *Proc. R. Soc. Lond. B* **268**, 2607–2611. (doi:10.1098/rspb.2001.1847)
16. Palacín C, Alonso JC, Martín CA, Alonso JA. 2017 Changes in bird-migration patterns associated with human-induced mortality. *Conservation Biology* **31**, 106–115.
17. Oro D, Genovart M, Tavecchia G, Fowler MS, Martínez-Abraín A. 2013 Ecological and evolutionary implications of food subsidies from humans. *Ecol. Lett.* **16**, 1501–1514. (doi:10.1111/ele.12187)
18. Plaza PI, Lambertucci SA. 2017 How are garbage dumps impacting vertebrate demography, health, and conservation? *Global Ecology and Conservation* **12**, 9–20. (doi:10.1016/j.gecco.2017.08.002)
19. Fiedler W. 2003 Recent changes in migratory behavior of birds: A compilation of field observations and ringing data. In *Avian migration* (eds P Berthold, E Gwinner, E Sonnenschein), pp. 21–38. Berlin, Germany: Springer.
20. Feige N, van der Jeugd HP, van der Graaf AJ, Larsson K, Leito A, Stahl J. 2008 Newly established breeding sites of the Barnacle Goose (*Branta leucopsis*) in North-western Europe–an overview of breeding habitats and colony development. *Vogelwelt* **129**, 244–252.
21. Gilbert NI, Correia RA, Silva JP, Pacheco C, Catry I, Atkinson PW, Gill JA, Franco AMA. 2016 Are white storks addicted to junk food? Impacts of landfill use on the movement and behaviour of resident white storks (*Ciconia ciconia*) from a partially migratory population. *Mov. Ecol.* **4**, 7. (doi:10.1186/s40462-016-0070-0)
22. Teitelbaum CS, Converse SJ, Fagan WF, Böhning-Gaese K, O'Hara RB, Lacy AE, Mueller T. 2016 Experience drives innovation of new migration patterns of whooping cranes in response to global change. *Nat. Comm.* **7**, 12793. (doi:10.1038/ncomms12793)
23. Satterfield DA, Maerz JC, Altizer S. 2015 Loss of migratory behaviour increases infection risk for a butterfly host. *Proc. R. Soc. B* **282**, 20141734. (doi:10.1098/rspb.2014.1734)
24. Flack A *et al.* 2016 Costs of migratory decisions: a comparison across eight white stork populations. *Sci. Adv.* **2**, e1500931. (doi:10.1126/sciadv.1500931)
25. Tortosa FS, Máñez M, Barcell M. 1995 Wintering White Storks (*Ciconia ciconia*) in South West Spain in the years 1991 and 1992. *Die Vogelwarte* **38**, 41–45.
26. Tortosa FS, Caballero JM, Reyes-López J. 2002 Effect of rubbish dumps on breeding success in the White Stork in Southern Spain. *Waterbirds* **25**, 39–43. (doi:10.1675/1524-4695(2002)025[0039:EORDOB]2.0.CO;2)
27. Evans KL, Gaston KJ, Sharp SP, McGowan A, Simeoni M, Hatchwell BJ. 2009 Effects of urbanisation on disease prevalence and age structure in blackbird *Turdus merula* populations. *Oikos* **118**, 774–782. (doi:10.1111/j.1600-0706.2008.17226.x)
28. Greig EI, Wood EM, Bonter DN. 2017 Winter range expansion of a hummingbird is associated with urbanization and supplementary feeding. *Proc. R. Soc. B* **284**, 20170256. (doi:10.1098/rspb.2017.0256)
29. Van Der Ree R, McDonnell MJ, Temby I, Nelson J, Whittingham E. 2006 The establishment and dynamics of a recently established urban camp of flying foxes (*Pteropus poliocephalus*) outside their geographic range. *J. Zool.* **268**, 177–185. (doi:10.1111/j.1469-7998.2005.00005.x)
30. Altizer S, Ostfeld RS, Johnson PTJ, Kutz S, Harvell CD. 2013 Climate change and infectious diseases: from evidence to a predictive framework. *Science* **341**, 514–519. (doi:10.1126/science.1239401)
31. Bartel RA, Oberhauser KS, de Roode JC, Altizer SM. 2011 Monarch butterfly migration and parasite transmission in eastern North America. *Ecology* **92**, 342–351. (doi:10.1890/10-0489.1)
32. Krkosek M, Lewis MA, Morton A, Frazer LN, Volpe JP. 2006 Epizootics of wild fish induced by farm fish. *Proc. Natl Acad. Sci. USA* **103**, 15 506–15 510. (doi:10.1073/pnas.0603525103)
33. Epstein JH, McKee J, Shaw P, Hicks V, Micalizzi G, Daszak P, Marm Kilpatrick A, Kaufman G. 2006 The Australian White Ibis (*Threskiornis molucca*) as a reservoir of zoonotic and livestock pathogens. *EcoHealth* **3**, 290–298. (doi:10.1007/s10393-006-0064-2)
34. Cross PC, Edwards WH, Scurlock BM, Maichak EJ, Rogerson JD. 2007 Effects of management and climate on elk brucellosis in the Greater Yellowstone Ecosystem. *Ecol. Appl.* **17**, 957–964. (doi:10.1890/06-1603)
35. Plowright RK, Foley P, Field HE, Dobson AP, Foley JE, Eby P, Daszak P. 2011 Urban habituation, ecological connectivity and epidemic dampening: the emergence of Hendra virus from flying foxes (*Pteropus* spp.). *Proc. R. Soc. B* **278**, 3703–3712. (doi:10.1098/rspb.2011.0522)
36. Plowright RK *et al.* 2015 Ecological dynamics of emerging bat virus spillover. *Proc. R. Soc. B* **282**, 20142124. (doi:10.1098/rspb.2014.2124)
37. Krkošek M, Lewis MA, Volpe JP. 2005 Transmission dynamics of parasitic sea lice from farm to wild salmon. *Proc. R. Soc. B* **272**, 689–696. (doi:10.1098/rspb.2004.3027)
38. Krkošek M, Ford JS, Morton A, Lele S, Myers RA, Lewis MA. 2007 Declining wild salmon populations in relation to parasites from farm salmon. *Science* **318**, 1772–1775. (doi:10.1126/science.1148744)
39. Berthold P. 2001 *Bird migration: a general survey*, 276 p. Oxford, UK: Oxford University Press.
40. Hurlbert AH, Liang Z. 2012 Spatiotemporal variation in avian migration phenology: citizen science reveals effects of climate change. *PLoS ONE* **7**, e31662. (doi:10.1371/journal.pone.0031662)
41. Németh Z. 2017 Partial migration and decreasing migration distance in the Hungarian population of the Common Blackbird (*Turdus merula* Linnaeus, 1758): Analysis of 85 years of ring recovery data. *Ornis Hungarica.* **25**, 101–108. (doi:10.1515/orhu-2017-0007)
42. Bailleul F, Lesage V, Power M, Doidge DW, Hammill MO. 2012 Migration phenology of beluga whales in a changing Arctic. *Climat. Resear.* **53**, 169–178. (doi:10.3354/cr01104)
43. Doswald N, Willis SG, Collingham YC, Pain DJ, Green RE, Huntley B. 2009 Potential impacts of climatic change on the breeding and non-breeding ranges and migration distance of European Sylvia warblers. *J. Biogeogr.* **36**, 1194–1208. (doi:10.1111/j.1365-2699.2009.02086.x)
44. Visser ME, Perdeck AC, Van Balen JH, Both C. 2009 Climate change leads to decreasing bird migration distances. *Glob. Chang. Biol.* **15**, 1859–1865. (doi:10.1111/j.1365-2486.2009.01865.x)
45. Newton I. 2008 *Migration ecology of birds*. London, UK: Academic Press.
46. Satterfield DA, Villablanca FX, Maerz JC, Altizer S. 2016 Migratory monarchs wintering in California experience low infection risk compared to monarchs breeding year-round on non-native milkweed. *Integr. Comp. Biol.* **56**, 343–352. (doi:10.1093/icb/icw030)
47. Batalden R, Oberhauser K. 2015 Potential changes in eastern North American monarch migration in response to an introduced milkweed, *Asclepias curassavica*. In *Monarchs in a changing world: biology and conservation of an iconic insect* (eds KS Oberhauser, KR Nail, S Altizer), pp. 215–224. Ithaca, NY: Cornell University Press.
48. Cross PC, Cole EK, Dobson AP, Edwards WH, Hamlin KL, Luikart G, Middleton AD, Scurlock BM, White PJ. 2010 Probable causes of increasing brucellosis in free-ranging elk of the Greater Yellowstone Ecosystem. *Ecol. Appl.* **20**, 278–288. (doi:10.1890/08-2062.1)
49. Jones JD, Kauffman MJ, Monteith KL, Scurlock BM, Albeke SE, Cross PC. 2014 Supplemental feeding alters migration of a temperate ungulate. *Ecol. Appl.* **24**, 1769–1779. (doi:10.1890/13-2092.1)
50. Hebblewhite M *et al.* 2006 Is the migratory behavior of montane elk herds in peril? The case of Alberta's Ya Ha Tinda elk herd. *Wildlife Society Bulletin* **34**, 1280–1294. (doi:10.2193/0091-7648(2006)34[1280:ITMBOM]2.0.CO;2)
51. Plummer KE, Siriwardena GM, Conway GJ, Risely K, Toms MP. 2015 Is supplementary feeding in gardens a driver of evolutionary change in a migratory bird species? *Glob. Chang. Biol.* **21**, 4353–4363. (doi:10.1111/gcb.13070)

52. Partecke J, Gwinner E. 2007 Increased sedentariness in European Blackbirds following urbanization: A consequence of local adaptation? *Ecology* **88**, 882–890. (doi:10.1890/06-1105)
53. Baskin Y. 1993 Trumpeter swans relearn migration. *BioSci.* **43**, 76–79. (doi:10.2307/1311967)
54. Sutherland WJ. 1998 Evidence for flexibility and constraint in migration systems. *J. Avian Biol.* **29**, 441–446. (doi:10.2307/3677163)
55. Pulido F, Berthold P. 2010 Current selection for lower migratory activity will drive the evolution of residency in a migratory bird population. *Proc. Natl Acad. Sci. USA* **107**, 7341–7346. (doi:10.1073/pnas.0910361107)
56. Sutherland WJ *et al.* 2017 A 2017 horizon scan of emerging issues for global conservation and biological diversity. *TREE* **32**, 31–40. (doi:10.1016/j.tree.2016.11.005)
57. Handrigan SA, Schummer ML, Petrie SA, Norris DR. 2016 Range expansion and migration of Trumpeter Swans *Cygnus buccinator* re-introduced in southwest and central Ontario. *Wildfowl* **66**, 60–74.
58. Cotterill GG, Cross PC, Cole EK, Fuda RK, Rogerson JD, Scurlock BM, du Toit JT. 2018 Winter feeding of elk in the Greater Yellowstone Ecosystem and its effects on disease dynamics. *Phil. Trans. R. Soc. B* **373**, 20170093. (doi:10.1098/rstb.2017.0093)
59. Lewis TL, Rongstad OJ. 1992 Effects of supplemental feeding on white-tailed deer migration in wisconsin. In *The biology of deer* (ed. RD Brown), pp. 458–458. New York, NY: Springer.
60. Peterson C, Messmer TA. 2007 Effects of winter-feeding on mule deer in northern Utah. *J. Wildl. Manag.* **71**, 1440–1445. (doi:10.2193/2006-202)
61. Feare CJ. 1984 *The starling*. Oxford, UK: Oxford University Press.
62. Cubie D. 2014 Site fidelity, residency, and sex ratios of wintering ruby-throated hummingbirds (*Archilochus colubris*) on the southeastern U.S. Atlantic Coast. *Wilson J. Ornithol.* **126**, 775–778. (doi:10.1676/14-005.1)
63. Brunnschweiler JM, Barnett A. 2013 Opportunistic visitors: long-term behavioural response of bull sharks to food provisioning in Fiji. *PLoS ONE* **8**, e58522. (doi:10.1371/journal.pone.0058522)
64. Espinoza M, Heupel MR, Tobin AJ, Simpfendorfer CA. 2016 Evidence of partial migration in a large coastal predator: opportunistic foraging and reproduction as key drivers? *PLoS ONE* **11**, e0147608. (doi:10.1371/journal.pone.0147608)
65. Clua E, Buray N, Legendre P, Mourier J, Planes S. 2010 Behavioural response of sicklefin lemon sharks *Negaprion acutidens* to underwater feeding for ecotourism purposes. *Mar. Ecol. Progr. Series* **414**, 257–266. (doi:10.3354/meps08746)
66. Corcoran MJ, Wetherbee BM, Shivji MS, Potenski MD, Chapman DD, Harvey GM. 2013 Supplemental feeding for ecotourism reverses diel activity and alters movement patterns and spatial distribution of the southern stingray, *Dasyatis americana*. *PLoS ONE.* **8**, e59235. (doi:10.1371/journal.pone.0059235)
67. Jefferies RL, Rockwell RF, Abraham KF. 2004 Agricultural food subsidies, migratory connectivity and large-scale disturbance in arctic coastal systems: a case study. *Integr. Comp. Biol.* **44**, 130–139. (doi:10.1093/icb/44.2.130)
68. Cano L. 2006 An approach to wintering of black stork *Ciconia nigra* in the Iberian Peninsula. *Biota* **7**, 7–13.
69. Cano LS, Pacheco C, Refoyo P, Tellería JL. 2014 Geographical and environmental factors affecting the distribution of wintering black storks *Ciconia nigra* in the Iberian Peninsula. *J. Avian Biol.* **45**, 514–521. (doi:10.1111/jav.00391)
70. Harris G, Thirgood S, Hopcraft JGC, Cromsigt JPGM, Berger J. 2009 Global decline in aggregated migrations of large terrestrial mammals. *Endang. Spec. Resear.* **7**, 55–76. (doi:10.3354/esr00173)
71. Loss SR, Will T, Marra PP. 2013 The impact of free-ranging domestic cats on wildlife of the United States. *Nat. Comm.* **4**, 1396. (doi:10.1038/ncomms2380)
72. Loss SR, Will T, Marra PP. 2015 Direct mortality of birds from anthropogenic causes. *Ann. Rev. Ecol. Evol. Sys.* **46**, 99–120. (doi:10.1146/annurev-ecolsys-112414-054133)
73. Yeh PJ, Price TD. 2004 Adaptive phenotypic plasticity and the successful colonization of a novel environment. *Am. Nat.* **164**, 531–542. (doi:10.1086/423825)
74. Berger J. 2007 Fear, human shields and the redistribution of prey and predators in protected areas. *Biol. Lett.* **3**, 620–623. (doi:10.1098/rsbl.2007.0415)
75. Rotics S *et al.* 2017 Wintering in Europe instead of Africa enhances juvenile survival in a long-distance migrant. *Anim. Behav.* **126**, 79–88. (doi:10.1016/j.anbehav.2017.01.016)
76. Pulido F, Berthold P, van Noordwijk AJ. 1996 Frequency of migrants and migratory activity are genetically correlated in a bird population: evolutionary implications. *Proc. Natl Acad. Sci. USA* **93**, 14 642–14 647. (doi:10.1073/pnas.93.25.14642)
77. Pérez-Tris J, Tellería JL. 2002 Migratory and sedentary blackcaps in sympatric non-breeding grounds: implications for the evolution of avian migration. *J. Anim. Ecol.* **71**, 211–224. (doi:10.1046/j.1365-2656.2002.00590.x)
78. Pulido F, Widmer M. 2005 Are long-distance migrants constrained in their evolutionary response to environmental change? Causes of variation in the timing of autumn migration in a Blackcap (*S. atricapilla*) and two Garden Warbler (*Sylvia borin*) populations. *Ann. N. Y. Acad. Sci.* **1046**, 228–241.
79. Møller AP, Erritzøe J. 1998 Host immune defence and migration in birds. *Evol. Ecol.* **12**, 945–953. (doi:10.1023/A:1006516222343)
80. Kilpatrick AM, Chmura AA, Gibbons DW, Fleischer RC, Marra PP, Daszak P. 2006 Predicting the global spread of H5N1 avian influenza. *Proc. Natl Acad. Sci. USA* **103**, 19 368–19 373. (doi:10.1073/pnas.0609227103)
81. Dusek RJ, McLean RG, Kramer LD, Ubico SR, Ii APD, Ebel GD, Guptill SC. 2009 Prevalence of West Nile Virus in migratory birds during spring and fall migration. *Am. J. Trop. Med. Hyg.* **81**, 1151–1158. (doi:10.4269/ajtmh.2009.09-0106)
82. Altizer S, Hobson KA, Davis AK, De Roode JC, Wassenaar LI. 2015 Do healthy monarchs migrate farther? Tracking natal origins of parasitized vs. uninfected monarch butterflies overwintering in Mexico. *PLoS ONE* **10**, e0141371. (doi:10.1371/journal.pone.0141371)
83. Sheldon BC, Verhulst S. 1996 Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. *TREE* **11**, 317–321.
84. Owen JC, Moore FR. 2006 Seasonal differences in immunological condition of three species of thrushes. *Condor* **108**, 389–398. (doi:10.1650/0010-5422(2006)108[389:SDIIC0]2.0.CO;2)
85. Buehler DM, Piersma T. 2008 Travelling on a budget: predictions and ecological evidence for bottlenecks in the annual cycle of long-distance migrants. *Phil. Trans. R. Soc. B* **363**, 247–266. (doi:10.1098/rstb.2007.2138)
86. Fritzsche McKay A, Hoye BJ. 2016 Are migratory animals superspreaders of infection? *Integr. Comp. Biol.* **56**, 260–267. (doi:10.1093/icb/icw054)
87. Hoverman JT, Searle CL. 2016 Behavioural influences on disease risk: implications for conservation and management. *Anim. Behav.* **120**, 263–271. (doi:10.1016/j.anbehav.2016.05.013)
88. Loehle C. 1995 Social barriers to pathogen transmission in wild animal populations. *Ecology* **76**, 326–335. (doi:10.2307/1941192)
89. Folstad I, Nilssen AC, Halvorsen O, Andersen J. 1991 Parasite avoidance: the cause of post-calving migrations in *Rangifer*? *Can. J. Zool.* **69**, 2423–2429. (doi:10.1139/z91-340)
90. Hoar BM, Ruckstuhl K, Kutz S. 2012 Development and availability of the free-living stages of *Ostertagia gruehneri*, an abomasal parasite of barrenground caribou (*Rangifer tarandus groenlandicus*), on the Canadian tundra. *Parasitology* **139**, 1093–1100. (doi:10.1017/S003118201200042X)
91. Qviller L, Risnes-Olsen N, Bærum KM, Meisingset EL, Loe LE, Ytrehus B, Viljugrein H, Mysterud A. 2013 Landscape level variation in tick abundance relative to seasonal migration in red deer. *PLoS ONE* **8**, e71299. (doi:10.1371/journal.pone.0071299)
92. Mysterud A, Qviller L, Meisingset EL, Viljugrein H. 2016 Parasite load and seasonal migration in red deer. *Oecologia* **180**, 401–407. (doi:10.1007/s00442-015-3465-5)
93. Pruvot M, Lejeune M, Kutz S, Hutchins W, Musiani M, Massolo A, Orsel K. 2016 Better alone or in ill company? the effect of migration and inter-species comingling on *Fascioloides magna* infection in elk. *PLoS ONE* **11**, e0159319. (doi:10.1371/journal.pone.0159319)
94. Simmons AM, Rogers CE. 1991 Dispersal and Seasonal occurrence of *Noctuidonema guyanense*, an ectoparasitic nematode of adult fall armyworm (Lepidoptera: Noctuidae), in the United States. *J. Entomol. Sci.* **26**, 136–148. (doi:10.18474/0749-8004-26.1.136)

95. van Gils JA, Munster VJ, Radersma R, Liefhebber D, Fouchier RAM, Klaassen M. 2007 Hampered foraging and migratory performance in swans infected with low-pathogenic avian influenza A virus. *PLoS ONE* **2**, e184. (doi:10.1371/journal.pone.0000184)
96. Hoye BJ, Munster VJ, Huig N, Vries Pd, Oosterbeek K, Tijsen W, Klaassen M, Fouchier RA, van Gils JA. 2016 Hampered performance of migratory swans: intra- and inter-seasonal effects of avian influenza virus. *Integr. Comp. Biol.* **56**, 317–329.
97. Costello MJ. 2009 How sea lice from salmon farms may cause wild salmonid declines in Europe and North America and be a threat to fishes elsewhere. *Proc. R. Soc. B.* **276**, 3385–3394. (doi:10.1098/rspb.2009.0771)
98. Groot C, Margolis L. 1991 *Pacific salmon life histories*. Vancouver, Canada: UBC Press.
99. Quinn TP. 2004 *The behavior and ecology of pacific salmon and trout*. Seattle, WA: University of Washington Press.
100. Strathmann RR, Hughes TP, Kuris AM, Lindeman KC, Morgan SG, Pandolfi JM, Warner RR. 2002 Evolution of local recruitment and its consequences for marine populations. *Bull. Mar. Sci.* **70**, 377–396.
101. Shaw AK, Binning SA. 2016 Migratory recovery from infection as a selective pressure for the evolution of migration. *Am. Nat.* **187**, 491–501. (doi:10.1086/685386)
102. Becker DJ, Streicker DG, Altizer S. 2015 Linking anthropogenic resources to wildlife–pathogen dynamics: a review and meta-analysis. *Ecol. Lett.* **18**, 483–495. (doi:10.1111/ele.12428)
103. Becker DJ, Streicker DG, Altizer S. Using host species traits to understand the consequences of resource provisioning for host–parasite interactions. *J. Anim. Ecol.* 1–16. (doi:10.1111/1365-2656.12765)
104. Leach CB, Webb CT, Cross PC. 2016 When environmentally persistent pathogens transform good habitat into ecological traps. *Open Sci.* **3**, 160051.
105. Hall RJ, Brown LM, Altizer S. 2016 Modeling vector-borne disease risk in migratory animals under climate change. *Integr. Comp. Biol.* **56**, 353–364. (doi:10.1093/icb/icw049)
106. Chapman JW, Reynolds DR, Wilson K. 2015 Long-range seasonal migration in insects: mechanisms, evolutionary drivers and ecological consequences. *Ecol. Lett.* **18**, 287–302. (doi:10.1111/ele.12407)
107. Bradley CA, Altizer S. 2005 Parasites hinder monarch butterfly flight: implications for disease spread in migratory hosts. *Ecol. Lett.* **8**, 290–300. (doi:10.1111/j.1461-0248.2005.00722.x)
108. McKay AF, Ezenwa VO, Altizer S. 2016 Unravelling the costs of flight for immune defenses in the migratory monarch butterfly. *Integr. Comp. Biol.* **56**, 278–289. (doi:10.1093/icb/icw056)
109. Krkošek M. 2017 Population biology of infectious diseases shared by wild and farmed fish. *Can. J. Fish. Aquat. Sci.* **74**, 620–628. (doi:10.1139/cjfas-2016-0379)
110. Bradley CA, Altizer S. 2007 Urbanization and the ecology of wildlife diseases. *TREE* **22**, 95–102.
111. Becker DJ, Hall RJ. 2014 Too much of a good thing: resource provisioning alters infectious disease dynamics in wildlife. *Biol. Lett.* **10**, 20140309. (doi:10.1098/rsbl.2014.0309)
112. Steinhaus EA. 1958 Crowding as a possible stress factor in insect disease. *Ecology* **39**, 503–514. (doi:10.2307/1931761)
113. Iguchi K, Ogawa K, Nagae M, Ito F. 2003 The influence of rearing density on stress response and disease susceptibility of ayu (*Plecoglossus altivelis*). *Aquaculture* **220**, 515–523. (doi:10.1016/S0044-8486(02)00626-9)
114. Padgett DA, Glaser R. 2003 How stress influences the immune response. *Trends Immunol.* **24**, 444–448. (doi:10.1016/S1471-4906(03)00173-X)
115. Piersma T. 1997 Do global patterns of habitat use and migration strategies co-evolve with relative investments in immunocompetence due to spatial variation in parasite pressure? *Oikos* **80**, 623–631.
116. Koprivnikar J, Leung TLF. 2015 Flying with diverse passengers: greater richness of parasitic nematodes in migratory birds. *Oikos* **124**, 399–405. (doi:10.1111/oik.01799)
117. Hannon ER, Kinsella JM, Calhoun DM, Joseph MB, Johnson PTJ. 2016 Endohelminths in bird hosts from northern california and an analysis of the role of life history traits on parasite richness. *J. Parasitol.* **102**, 199–207. (doi:10.1645/15-867)
118. Gutiérrez JS, Rakhimberdiev E, Piersma T, Thieltges DW. 2017 Migration and parasitism: habitat use, not migration distance, influences helminth species richness in Charadriiform birds. *J. Biogeogr.* **44**, 1137–1147.
119. Evans KL, Newton J, Gaston KJ, Sharp SP, McGowan A, Hatchwell BJ. 2012 Colonisation of urban environments is associated with reduced migratory behaviour, facilitating divergence from ancestral populations. *Oikos* **121**, 634–640. (doi:10.1111/j.1600-0706.2011.19722.x)
120. Van Vliet J, Musters CJM, Ter Keurs WJ. 2009 Changes in migration behaviour of Blackbirds Turdus merula from the Netherlands. *Bird Study* **56**, 276–281. (doi:10.1080/00063650902792148)
121. Wille M, Lindqvist K, Muradrasoli S, Olsen B, Järhult JD. 2017 Urbanization and the dynamics of RNA viruses in Mallards (*Anas platyrhynchos*). *Infect. Gen. Evol.* **51**, 89–97. (doi:10.1016/j.meegid.2017.03.019)
122. Hill NJ, Ma EJ, Meixell BW, Lindberg MS, Boyce WM, Runstadler JA. 2016 Transmission of influenza reflects seasonality of wild birds across the annual cycle. *Ecol. Lett.* **19**, 915–925. (doi:10.1111/ele.12629)
123. Hill NJ, Runstadler JA. 2016 A bird's eye view of influenza a virus transmission: challenges with characterizing both sides of a co-evolutionary dynamic. *Integr. Comp. Biol.* **56**, 304–316. (doi:10.1093/icb/icw055)
124. Eikenaar C, Hegemann A. 2016 Migratory common blackbirds have lower innate immune function during autumn migration than resident conspecifics. *Biol. Lett.* **12**, 20160078. (doi:10.1098/rsbl.2016.0078)
125. Lemoine M, Doligez B, Richner H. 2011 On the equivalence of host local adaptation and parasite maladaptation: an experimental test. *Am. Nat.* **179**, 270–281. (doi:10.1086/663699)
126. MacColl AD, Chapman SM. 2010 Parasites can cause selection against migrants following dispersal between environments. *Funct. Ecol.* **24**, 847–856. (doi:10.1111/j.1365-2435.2010.01691.x)
127. Altizer SA. 2001 Migratory behaviour and host-parasite co-evolution in natural populations of monarch butterflies infected with a protozoan parasite. *Evol. Ecol. Res.* **3**, 611–632.
128. de Roode JC, Yates AJ, Altizer S. 2008 Virulence-transmission trade-offs and population divergence in virulence in a naturally occurring butterfly parasite. *Proc. Natl Acad. Sci. USA* **105**, 7489–7494. (doi:10.1073/pnas.0710909105)
129. Hill NJ, Takekawa JY, Ackerman JT, Hobson KA, Herring G, Cardona CJ, Runstadler JA, Boyce WM. 2012 Migration strategy affects avian influenza dynamics in mallards (*Anas platyrhynchos*). *Mol. Ecol.* **21**, 5986–5999. (doi:10.1111/j.1365-294X.2012.05735.x)
130. Dijk JG, Hoye BJ, Verhagen JH, Nolet BA, Fouchier RA, Klaassen M. 2014 Juveniles and migrants as drivers for seasonal epizootics of avian influenza virus. *J. Anim. Ecol.* **83**, 266–275. (doi:10.1111/1365-2656.12131)
131. Verhagen JH, Dijk Jv, Vuong O, Bestebroer T, Lexmond P, Klaassen M, Fouchier RAM. 2014 Migratory birds reinforce local circulation of avian influenza viruses. *PLoS ONE* **9**, e112366. (doi:10.1371/journal.pone.0112366)
132. Clark NJ, Clegg SM, Klaassen M. 2016 Migration strategy and pathogen risk: non-breeding distribution drives malaria prevalence in migratory waders. *Oikos* **125**, 1358–1368. (doi:10.1111/oik.03220)
133. Valkiunas G. 1993 The role of seasonal migrations in the distribution of *Haemosporidia* of birds in North Palaearctic. *Ekologija* **2**, 57–67.
134. Waldenström J, Bensch S, Kiboi S, Hasselquist D, Ottosson U. 2002 Cross-species infection of blood parasites between resident and migratory songbirds in Africa. *Mol. Ecol.* **11**, 1545–1554. (doi:10.1046/j.1365-294X.2002.01523.x)
135. Cappelle J *et al.* 2014 Risks of avian influenza transmission in areas of intensive free-ranging duck production with wild waterfowl. *Ecohealth* **11**, 109–119. (doi:10.1007/s10393-014-0914-2)
136. Urquhart FA, Urquhart NR. 1976 The overwintering site of the eastern population of the monarch butterfly (*Danaus p. plexippus*; Danaidae) in southern Mexico. *J. Lepid. Soc.* **30**, 153–158.
137. Brower LP. 1995 Understanding and misunderstanding the migration of the monarch butterfly (Nymphalidae) in North America: 1857–1995. *J. Lepid. Soc.* **49**, 304–385.
138. Nagano C, Sakai W, Malcolm S, Cockrell BJ, Brower LP. 1993 Spring migration of monarch butterflies in California. In *Biology and conservation of the*

monarch butterfly (eds SB Malcolm, MP Zalucki), pp. 219–232. Los Angeles, CA: Natural History Museum of Los Angeles County. (Science Series).

139. Ackery PR, Vane-Wright RI. 1984 *Milkweed butterflies: their cladistics and biology*. Ithaca, NY: Cornell University Press.
140. Zhan S *et al.* 2014 The genetics of monarch butterfly migration and warning colouration. *Nature* **514**, 317. (doi:10.1038/nature13812)
141. McLaughlin RE, Myers J. 1970 Ophryocystis elektroscirrha sp. n., a neogregarine pathogen of the monarch butterfly *Danaus plexippus* (L.) and the Florida queen butterfly *D. gilippus berenice* Cramer1. *J. Eukary. Microbiol.* **17**, 300–305.
142. Altizer S, Oberhauser K, Geurts K. 2004 Transmission of the protozoan parasite *Ophryocystis elektroscirrha* in monarch butterfly populations: implications for prevalence and population-level impacts. In *The monarch butterfly: biology and conservation* (eds KS Oberhauser, MJ Solensky), pp. 203–218. Ithaca, NY: Cornell University Press.
143. Altizer S, de Roode JC. 2015 Monarchs and their debilitating parasites: immunity, migration and medicinal plant use. In *Monarchs in a changing world: biology and conservation of an iconic insect* (eds K Oberhauser, S Altizer, K Nail), pp. 83–93. Ithaca, NY: Cornell University Press.
144. de Roode JC, Chi J, Rarick RM, Altizer S. 2009 Strength in numbers: high parasite burdens increase transmission of a protozoan parasite of monarch butterflies (*Danaus plexippus*). *Oecologia* **161**, 67–75. (doi:10.1007/s00442-009-1361-6)
145. Altizer SM, Oberhauser KS. 1999 Effects of the protozoan parasite *Ophryocystis elektroscirrha* on the Fitness of Monarch Butterflies (Danaus plexippus). *J. Invert. Pathol.* **74**, 76–88. (doi:10.1006/jipa.1999.4853)
146. de Roode JC, Gold LR, Altizer S. 2007 Virulence determinants in a natural butterfly-parasite system. *Parasitology* **134**, 657–668. (doi:10.1017/S0031182006002009)
147. Altizer SM, Oberhauser KS, Brower LP. 2000 Associations between host migration and the prevalence of a protozoan parasite in natural populations of adult monarch butterflies. *Ecol. Entomol.* **25**, 125–139. (doi:10.1046/j.1365-2311.2000.00246.x)
148. Brower LP, Taylor OR, Williams EH, Slayback DA, Zubieta RR, Ramírez MI. 2012 Decline of monarch butterflies overwintering in Mexico: is the migratory phenomenon at risk? *Insect. Conserv. Divers.* **5**, 95–100.
149. Vidal O, Rendon-Salinas E. 2014 Dynamics and trends of overwintering colonies of the monarch butterfly in Mexico. *Biol. Conserv.* **180**, 165–175. (doi:10.1016/j.biocon.2014.09.041)
150. Jepsen S, Hoffman Black S. 2015 Understanding and conserving the western North American monarch population. In *Monarchs in a changing world: biology and conservation of an iconic insect* (eds KS Oberhauser, KR Nail, S Altizer), pp. 147–156. Ithaca, NY: Cornell University Press.
151. Flockhart DTT, Pichancourt J-B, Norris DR, Martin TG. 2015 Unravelling the annual cycle in a migratory animal: breeding-season habitat loss drives population declines of monarch butterflies. *J. Anim. Ecol.* **84**, 155–165. (doi:10.1111/1365-2656.12253)
152. Pleasants J. 2017 Milkweed restoration in the Midwest for monarch butterfly recovery: estimates of milkweeds lost, milkweeds remaining and milkweeds that must be added to increase the monarch population. *Insect Conser. Divers.* **10**, 42–53. (doi:10.1111/icad.12198)
153. Thogmartin WE *et al.* 2017 Monarch butterfly population decline in North America: identifying the threatening processes. *R. Soc. open sci.* **4**, 170760. (doi:10.1098/rsos.170760)
154. Woodson REJ. 1954 The North American species of *Asclepias L. Ann. Missouri. Bot. Garden* **41**, 1–211. (doi:10.2307/2394652)
155. de Roode JC, Pedersen AB, Hunter MD, Altizer S. 2008 Host plant species affects virulence in monarch butterfly parasites. *J. Anim. Ecol.* **77**, 120–126. (doi:10.1111/j.1365-2656.2007.01305.x)
156. Lefevre T *et al.* 2012 Behavioural resistance against a protozoan parasite in the monarch butterfly. *J. Anim. Ecol.* **81**, 70–79. (doi:10.1111/j.1365-2656.2011.01901.x)
157. Sternberg ED, Lefèvre T, Li J, de Castillejo CLF, Li H, Hunter MD, De Roode JC. 2012 Food plant derived disease tolerance and resistance in a natural butterfly-plant-parasite interactions. *Evolution* **66**, 3367–3376. (doi:10.1111/j.1558-5646.2012.01693.x)
158. Howard E, Aschen H, Davis AK. 2010 Citizen science observations of monarch butterfly overwintering in the southern United States. *Psyche: J. Entomol.* **2010**, 1–6. (doi:10.1155/2010/689301)
159. Goehring L, Oberhauser KS. 2002 Effects of photoperiod, temperature, and host plant age on induction of reproductive diapause and development time in *Danaus plexippus*. *Ecol. Entomol.* **27**, 674–685. (doi:10.1046/j.1365-2311.2002.00454.x)
160. Gardy J, Loman NJ, Rambaut A. 2015 Real-time digital pathogen surveillance—the time is now. *Genome Biol.* **16**, 155. (doi:10.1186/s13059-015-0726-x)
161. Brown LM, Hall RJ. 2018 Consequences of resource supplementation for disease risk in a partially migratory population. *Phil. Trans. R. Soc. B* **373**, 20170095. (doi:10.1098/rstb.2017.0095)