Responses of migratory species and their pathogens to supplemental feeding

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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...
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.)

<table>
<thead>
<tr>
<th>species</th>
<th>provisioned resource</th>
<th>changes in migration</th>
<th>references</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anna's hummingbirds <em>Calypte anna</em></td>
<td>bird feeders</td>
<td>Anna's hummingbirds have become more abundant in winter at northern latitudes, suggesting a reduced migratory propensity associated with human landscapes</td>
<td>Greig et al. 2017 [28]</td>
</tr>
<tr>
<td>monarch butterfly <em>Danaus plexippus</em></td>
<td>exotic milkweed plants</td>
<td>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</td>
<td>Satterfield et al. [23,46]; Batalden et al. [47]</td>
</tr>
<tr>
<td>elk <em>Cervus elaphus</em></td>
<td>winter supplements</td>
<td>elk fed winter supplements in Wyoming were found to delay migration, reduce migratory duration on the summer range and form dense aggregations</td>
<td>Cross et al. [48]; Jones et al. [49]; Hebblewhite et al. 2006 [50]</td>
</tr>
<tr>
<td>Eurasian blackcap <em>Sylvia atricapilla</em></td>
<td>bird feeders</td>
<td>typically migrants to the Mediterranean, blackcaps increasingly overwinter in Britain, associated with bird feeders</td>
<td>Plummer et al. [51]</td>
</tr>
<tr>
<td>European blackbirds <em>Turdus merula</em></td>
<td>possibly fruits, earthworms, seeds, feeders in gardens and on lawns</td>
<td>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</td>
<td>Berthold [39]; Partecke and Gwinner 2007 [52]</td>
</tr>
<tr>
<td>grey-headed flying foxes <em>Pteropus poliocephalus</em></td>
<td>fruit in gardens</td>
<td>traditionally nomadic migrants, these fruit bats have established resident camps in urban Australia in response to supplemental fruit in gardens</td>
<td>Van der Ree et al. 2006 [29]</td>
</tr>
<tr>
<td>trumpeter swans <em>Cygnus buccinator</em></td>
<td>wheat (intentionally fed)</td>
<td>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</td>
<td>Baskin 1993 [53]</td>
</tr>
<tr>
<td>white storks <em>Ciconia ciconia</em></td>
<td>garbage</td>
<td>while the birds typically migrate from Europe to Africa, a resident population formed in Spain, where the birds feed in landfills year-round</td>
<td>Tortosa et al. [25,26]</td>
</tr>
</tbody>
</table>
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 caurulescens) 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, (iii) 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 allopatriy). Evidence for migratory allopatriy 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]).
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 presence for parasites transmitted during the breeding season. This was tested in another similar transmission model that also demonstrated migratory escape, such that infection presence was lower when hosts spent more time migrating [11]. Resident populations in both models, represented by no time spent migrating, experienced higher 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 allopatri

Newly resident animals could experience higher host–parasite contact rates due to the loss of migratory allopatri, 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
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—whenver 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.

References


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