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Animal migrations and parasitism: reciprocal effects within a unified framework

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ABSTRACT

Migrations, i.e. the recurring, roundtrip movement of animals between distant and distinct habitats, occur among diverse metazoan taxa. Although traditionally linked to avoidance of food shortages, predators or harsh abiotic conditions, there is increasing evidence that parasites may have played a role in the evolution of migration. On the one hand, selective pressures from parasites can favour migratory strategies that allow either avoidance of infections or recovery from them. On the other hand, infected animals incur physiological costs that may limit their migratory abilities, affecting their speed, the timing of their departure or arrival, and/or their condition upon reaching their destination. During migration, reduced immunocompetence as well as exposure to different external conditions and parasite infective stages can influence infection dynamics. Here, we first explore whether parasites represent extra costs for their hosts during migration. We then review how infection dynamics and infection risk are affected by host migration, thereby considering parasites as both causes and consequences of migration. We also evaluate the comparative evidence testing the hypothesis that migratory species harbour a richer parasite fauna than their closest free-living relatives, finding general support for the hypothesis. Then we consider the implications of host migratory behaviour for parasite ecology and evolution, which have received much less attention. Parasites of migratory hosts may achieve much greater spatial dispersal than those of non-migratory hosts, expanding their geographical range, and providing more opportunities for host-switching. Exploiting migratory hosts also exerts pressures on the parasite to adapt its phenology and life-cycle duration, including the timing of major developmental, reproduction and transmission events. Natural selection may even favour parasites that manipulate their host's migratory strategy in ways that can enhance parasite transmission. Finally, we propose a simple integrated framework based on eco-evolutionary feedbacks to consider the reciprocal selection pressures acting on migratory hosts and their parasites. Host migratory strategies and parasite traits evolve in tandem, each acting on the other along two-way causal paths and feedback loops. Their likely adjustments to predicted climate change will be understood best from this coevolutionary perspective.

Key words: evolutionary feedback, costs, migratory escape, migratory recovery, parasite species richness, pathogen dispersal, phenology, life-history strategies, host manipulation

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I. INTRODUCTION

Animal migrations rank as one of the most spectacular and intriguing phenomena in nature (Milner-Gulland, Fryxell & Sinclair, 2011; Dingle, 2014). Although salmon and shorebirds immediately come to mind as obvious examples, migratory taxa range from insects (Satterfield *et al.*, 2020) to mammals (Gnanadesikan, Pearse & Shaw, 2017). Migrations consist of relatively long-distance, more-or-less synchronised movement of individual animals, following a seasonal or other clear temporal cycle. They usually take the animals back and forth between two distinct areas with different environmental conditions, one in which they reproduce and one in which they spend the rest of the year. In certain species, only some individuals, or some populations, undergo migrations, whereas the others reside in a single area year-round; in other species, all individuals migrate, but not necessarily for the same distance or to the same destination (Milner-Gulland *et al.*, 2011; Dingle, 2014). Migratory behaviour is triggered by particular cues resulting from changes in local conditions, and is characterised by persistent and directional movement requiring both the use of mechanisms of orientation, and the reallocation of resources to support the energy demands of continuous movement (Dingle, 2014). The most widely invoked ultimate explanations for animal migrations are that animals move to avoid (*i*) seasonal food shortages and to take advantage of abundant resources currently available elsewhere, (*ii*) challenging climatic conditions during winter at high latitudes, and/or (*iii*) seasonal peaks in predation risk by moving into enemy-free space (Alerstam, Hedenström & Åkesson, 2003; Milner-Gulland *et al.*, 2011; Avgar, Street & Fryxell, 2014; Dingle, 2014; Chapman, Reynolds & Wilson, 2015; Gnanadesikan *et al.*, 2017). All these benefits of migrating are usually tied with reproduction, bringing the animals to more suitable breeding grounds, or to a better post-reproduction area, than the habitat they leave behind. Other evolutionary factors are sometimes hypothesised as having played a role in shaping migratory strategies, but the above three are put forward most frequently.

Parasitic species carried by migrants have long been used as markers to identify their area of origin or reconstruct the route followed during migration in fish (e.g. Criscione, Cooper & Blouin, 2006; Alarcos & Timi, 2013; Jacobson *et al.*, 2019), birds (Durrant *et al.*, 2008; Sheehan *et al.*, 2016) and mammals (Iwasa-Arai *et al.*, 2018). However, parasites are not just passive passengers that convey useful information: they are increasingly considered as potential drivers of animal migrations, since the diseases they cause represent another environmental pressure, like food scarcity or high predation risk, that animals may avoid by moving to a different habitat (Piersma, 1997; Altizer, Bartel & Han, 2011; Møller & Szép, 2011; Shaw & Binning, 2016). Yet the interactions between parasites and migration go well beyond the possible role of parasites as a force behind the evolution of migration. Even if animals migrate for completely different reasons, their risk of parasite infections may be higher than that of non-migrants due to a trade-off between the energy invested in migratory requirements *versus* immune function (Eikenaar & Hegemann, 2016). Infected individuals may experience higher mortality during migration, arrive later at their destination, be in lower condition at the end of migration, or not migrate at all (Altizer *et al.*, 2011). Migratory species, or migratory individuals in species where migration is not universal, can be exposed to a greater diversity of parasites by spending time in two or more distinct habitats every year, and in various stopover areas in between (Leung & Koprivnikar, 2016). Thus, even if they have not driven the evolution of migration, parasites can still exert selective pressures on migrating animals.

At the same time, host migration can exert strong selective pressures on the parasites themselves. Inevitably, parasites are incidental co-migrants with their hosts (Cohen & Satterfield, 2020). On the one hand, the obligatory journey with their migrating hosts favours adaptations in developmental rates, phenology, and/or life-cycle patterns in order to survive changing conditions and long periods with no opportunity for transmission. Given how often the ability to modify host phenotype has evolved among parasites (Poulin, 2010), some parasites may even be able to induce their host to abort their migration, or adopt slightly different migration patterns that improve the parasite's own transmission. On the other hand, long-distance host migrations allow large-scale parasite dispersal. This not only affects the parasite's geographical range and the genetic structure of its populations across its range, but also exposes the parasite to a broader array of potential host species, therefore increasing opportunities for host switching.

The ecological and evolutionary influence of parasites on host migration, and the corresponding influence of host migration on parasites, are generally considered separately. Yet a full understanding of the interactions between behaviour and parasitism requires the careful and explicit consideration of the feedbacks between the two (Ezenwa *et al.*, 2016). The overarching aim of this review is to unify these reciprocal effects into an integrated overview of the coevolutionary processes in which animal migrations and parasites feed back on each other. At a time when the timing and spatial extent of animal migrations are changing rapidly in response to global climate change (Bussière, Underhill & Altwegg, 2015; Kristensen *et al.*, 2015; Cohen, Lajeunesse & Rohr, 2018), it is imperative to understand and anticipate the consequences

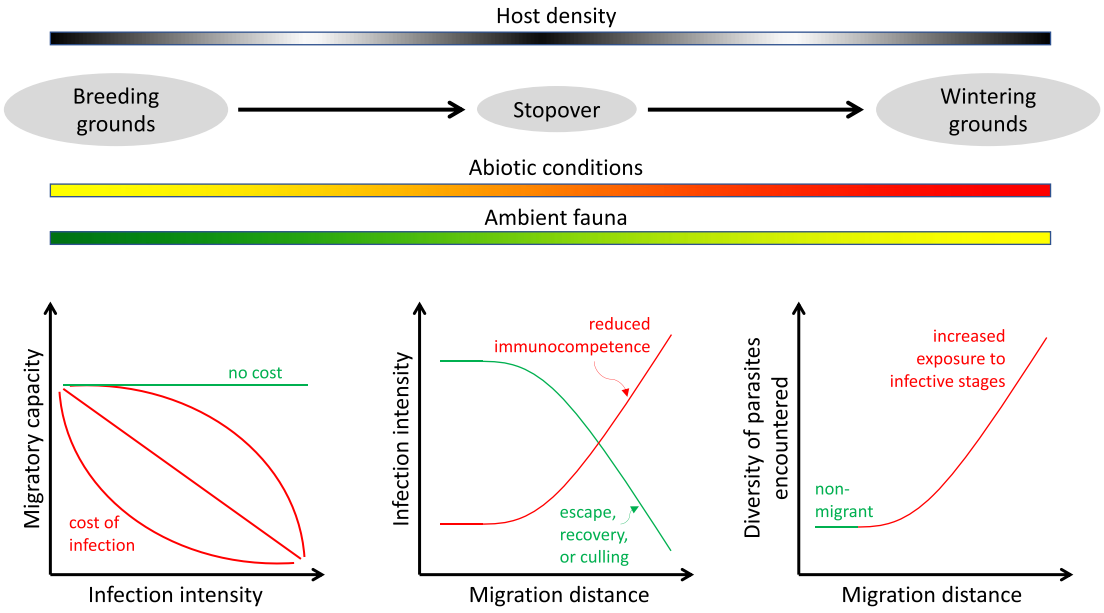

Fig 1. Graphical summary of the ways in which parasites can represent costs, causes or consequences of migration. Over one half of the migratory cycle, e.g. from the breeding to the wintering grounds, the context for host–parasite interactions can change dramatically (the same would apply for the return phase from the wintering to the breeding grounds, not shown here). Host population density can fluctuate widely, peaking at the start and the end, as well as at stopover sites. By contrast, abiotic conditions and the composition of the ambient fauna (vectors and intermediate hosts of pathogens) are, in most cases, likely to change along a unidirectional gradient. Migratory capacity (i.e. speed, stamina, distance achieved) may be unaffected by parasite infections, or it may decrease linearly or non-linearly with increasing infection intensity, depending on specific circumstances. Infections by particular parasite species may decrease during migration, if migratory behaviour allows hosts to escape or recover from infection, or if severely infected individuals are culled in transit; alternatively, the severity of infections may increase if the demands of migration cause immune defences to be compromised. Migratory host species, especially long-distance migrants, may be exploited by a greater diversity of parasites than resident species, as a consequence of the greater variety of habitats, vectors and intermediate hosts they encounter.

for the ecology of diseases in migratory species (e.g. Garamszegi, 2011). The scope of this review extends to all animals (except humans) and parasites in the broadest sense, including pathogenic microorganisms to helminths and arthropods. Here, we consider as parasites any symbionts generally known or assumed to impose a fitness cost to their host, even if this cost is not manifested during migratory events but at other times in the host's life. We first begin by reviewing the many implications and influences of parasites on the evolution and ecology of animal migrations. We also use a systematic review to test whether migrants are exploited by more parasite species than non-migrants. We then flip over the causality arrow and discuss the evolutionary pressures placed on parasites by the migratory behaviour of their hosts. Finally, we bring together the two branches of the reciprocal parasite–migration coevolutionary process and consider the feedbacks between them in a unified framework.

II. PARASITES AS A COST DURING MIGRATION

In this and the next three sections, we explore the implications of parasites for their hosts as costs, causes and consequences of migration (summarised in Fig. 1). We begin by considering the additional costs they may impose on migratory hosts. By their very nature, parasites divert energy and resources from their host, and generally induce some reduction in host fitness. All else being equal, an infected individual should experience greater physiological stress during periods of high energy demands and sustained locomotion, such as migration, than an uninfected conspecific (Binning, Shaw & Roche, 2017). There is indeed some evidence that parasite infections negatively impact host migration; however, not all parasites do so, and in some cases infected animals may overcome the effects of parasites and complete their migration as successfully as uninfected individuals by fine-tuning their movement strategy or adjusting their investments in immune defence (Buehler, Tieleman & Piersma, 2010; Binning *et al.*, 2017). In extreme cases, infected individuals undertaking migration end up succumbing in transit. Migratory culling (*sensu* Bradley & Altizer, 2005) can reduce prevalence of infection in a migratory population relative to a non-migratory one (e.g. Slowinski *et al.*, 2018), and has been interpreted as evidence of the selective pressures exerted by parasites on migrants (Shaw *et al.*, 2018). From the perspective of the individual, however, death during migration represents the

ultimate cost of infection. Empirical assessments of the costs of parasitism during migration at both individual and population levels have been conducted in animals ranging from insects to mammals, and taken together reveal much variation and context dependence.

Monarch butterflies, *Danaus plexippus*, from eastern North America are famous for their long-distance migration that takes them to their wintering range in the mountains of central Mexico. Monarchs are hosts to the gregarine *Ophryocystis elektroscirrha*, a protozoan that can be transmitted vertically during oviposition, or horizontally *via* spores ingested by feeding caterpillars. Under controlled conditions, experimentally infected monarchs from eastern North America flew at lower speeds, achieved shorter distances, and lost more body mass from flight than uninfected controls (Bradley & Altizer, 2005). The mechanisms by which the parasite lowers flight performance do not involve depletion of energy reserves, and are yet to be fully understood (Satterfield, Wright & Altizer, 2013). Whatever they are, the reduced endurance of infected individuals during powered flight was interpreted as contributing to migratory culling, i.e. the removal of infected individuals from the population, which may explain why prevalence of infection by *O. elektroscirrha* is much lower in this migratory population than in non-migratory monarch populations (Altizer, 2001; Bradley & Altizer, 2005).

Parasitism has also been linked with reduced migration performance in salmonid fishes. Sea lice, i.e. the ectoparasitic copepods *Lepeophtheirus salmonis*, infect salmonids when they leave freshwater habitats and migrate out to sea. In anadromous populations of brown trout, *Salmo trutta*, that migrate to coastal waters to feed for a few months before returning to river habitats, experimental infection with sea lice caused increased mortality, while infected fish that survived stayed closer to estuaries and returned prematurely to fresh waters (Serra-Llinares *et al.*, 2020). Lice-infected pink salmon, *Oncorhynchus gorbuscha*, had reduced endurance for prolonged swimming compared to uninfected control fish, but only when infected by large female lice (Mages & Dill, 2010). In this case, the effect of infection was also intensity dependent, i.e. the more lice per fish, the lower the swimming ability. Atlantic salmon, *Salmo salar*, infected with lice also showed intensity-dependent reductions in lipid reserves and body condition (Susdorf *et al.*, 2018). The most convincing demonstration of the culling effect of sea lice on migrating salmon comes from a meta-analysis of field experiments replicated across multiple rivers in two countries (Krkošek *et al.*, 2013). The study estimated that return rates of Atlantic salmon were 39% lower due to mortality induced by sea lice at natural intensities of infection. A later meta-analysis of a larger number of field trials conducted in Norway supported this general finding, although it also emphasised the modulating effect of other risk factors (Vollset *et al.*, 2016). Apart from sea lice, a range of viral, bacterial and protozoan parasites have also been associated with decreased swimming performance and survival in migrating salmonids (e.g. Margolis, McDonald & Whitaker, 1992; Kocan *et al.*, 2006; Jeffries *et al.*, 2014; Bass *et al.*, 2019). There is even evidence that intestinal helminth parasites can interfere with the ability of salmon to orientate on their way out to sea (Garnick & Margolis, 1990), possibly due to their general debilitating effects, with consequences for survival and return rates.

Apart from salmonids, the other fish whose long-distance migration has been studied in the context of parasitism is the European eel, *Anguilla anguilla*. The parasitic nematode *Anguillicoloides crassus* has spread across European eel populations since its introduction from eastern Asia, and raised concerns about its health impact on the fish (Kirk, 2003). The blood-feeding adult stage infecting eels causes tissue damage in the swim-bladder wall and impairs its buoyancy function. Many studies conducted in water flumes, artificial stream channels, or other forms of enclosures have found that infection with *A. crassus* significantly reduces the eels' maximum speed, cruising speed, swimming endurance, or the distance they cover in a given time period (Sprengel & Lüchtenberg, 1991; Palstra *et al.*, 2007; Sjöberg *et al.*, 2009; Newbold *et al.*, 2015). Not all studies have reported such negative effects, however (Münderle, Sures & Taraschewski, 2004). Nevertheless, there is general agreement that infection may affect the ability of eels to control their vertical position in the water column, and that infected fish may either experience total migration failure or arrive too late in the Sargasso Sea spawning grounds to take part in reproduction.

Multiple studies on various species of migrating birds have focused on the potential influence of vector-borne haemosporidian parasites that cause avian malaria. Under experimental conditions, haemosporidian infection can decrease body condition, survival and fitness of birds (Marzal *et al.*, 2005; Palinauskas *et al.*, 2008; Schoenle *et al.*, 2017). However, many studies on long-range migratory passerine birds report that infections by the widespread haemosporidian *Haemoproteus* spp. (and to a lesser extent *Plasmodium* spp. and *Leucocytozoon* spp.) are not associated with any detectable cost based on various measures related to migration success, ranging from fat reserves prior to departure, white blood cell counts or body condition during migration, to arrival time or return rates to breeding grounds (Ashford, 1971; Davidar & Morton, 1993; Rätti, Dufva & Alatalo, 1993; Cornelius, Davis & Altizer, 2014; Hahn *et al.*, 2018; Sorensen *et al.*, 2016, 2019). By contrast, a few other studies report that haemosporidian infections are associated with lower fat reserves during migration, longer stopover duration, reduced body mass at arrival, and/or delayed arrival at the breeding grounds in passerines (Møller, de Lope & Saino, 2004; Garvin, Szell & Moore, 2006; Emmenegger *et al.*, 2018*b*; Hegemann *et al.*, 2018; Ágh *et al.*, 2019), raptors (Ishak *et al.*, 2010), and waterfowl (Merrill *et al.*, 2018). Not all conspecifics cover the same distance during migration, and there is also no consistency among results of intraspecific correlations between distance travelled and prevalence or intensity of infection by haemosporidians (Smith, Greiner & Wolf, 2004; Kelly *et al.*, 2016). The above contrasting findings suggest effects that depend on the parasite and/or host taxon, rather than a general debilitating impact of avian malaria. Far fewer

studies have been conducted on the influence of other parasite taxa on bird migrations. Their results indicate that parasites as diverse as avian influenza, vector-borne trypanosomes, lice and mites can reduce the body condition of migrating birds and/or delay their arrival at their destination (Rätti *et al.*, 1993; Latta, 2003; Møller *et al.*, 2004; Latorre-Margalef *et al.*, 2009); these effects are inconsistent, however, ranging in magnitude from very small to dramatic, and are often sex dependent, possibly due to the different costs of reproduction between males and females.

Finally, only one study on the cost of parasitism in migrating mammals could be found. It reported that based on GPS (global positioning system) tracking, there was a negative correlation between the number of ticks per individual red deer, *Cervus elaphus*, and the distance they covered between their winter range and their chosen high-elevation summer range (Mysterud *et al.*, 2016). Having ruled out the possible loss of ticks by individuals that migrated the greatest distance, the authors concluded a cost of parasitism.

Overall, there is considerable evidence that parasitic infections reduce the ability of many animals to complete their migration successfully. Practically all studies on insects, fish and mammals that undergo long-distance migrations have observed costs of parasitism, by a wide range of parasite taxa, manifested as decreases in locomotory speed, distance travelled, survival, etc. Of course, this conclusion may reflect a publication bias: studies on costs of parasitism for migratory hosts may only be conducted and/or published when negative effects are expected and/or found. By contrast, studies on migratory birds often fail to uncover any impact of parasites, or find only small and context-dependent effects. This may be due to the focus on avian malaria, with the infection having reached a sublethal chronic phase in many studied populations prior to migration. A meta-analysis across all animal taxa confirms that parasite infection is generally strongly associated with reduced movement, and weakly associated with reduced energy reserves, delayed migration and lower survival, with infection intensity also linked with these effects (Risely, Klaassen & Hoye, 2018). Most of the available field studies are correlational or observational; experimental studies are generally limited to those conducted under controlled conditions in wind tunnels or water flumes. Recent technological advances, such as the miniaturisation of electronic tracking devices and satellite-based telemetry infrastructure, provide valuable tools for the experimental study of animal migrations in the field (Birnie-Gauvin *et al.*, 2020). These methodological developments will allow a more accurate assessment of the true costs of parasitism during migration in natural contexts.

III. DYNAMICS OF INFECTION DURING MIGRATION

Animals can in some cases migrate to control the abundance of their parasites if these have imposed strong selective pressures (see Section IV). In many other cases, however, animal migrations have completely different evolutionary causes, with parasite populations experiencing fluctuations as a coincidental side-effect. A reduction in infection levels and the ensuing relaxation of parasite pressure can provide a secondary benefit of migration, whereas enhanced infections resulting from migrations represent an additional cost.

Patterns of infection by particular parasite species may change during migration for multiple reasons (Altizer *et al.*, 2011). In some cases, parasites may thrive in response to reduced investments in immune defence because of the energetic demands of migration. Recruitment of new parasite individuals may vary widely through the migratory cycle since exposure to, and acquisition of, infective stages change as hosts experience different habitats by leaving one area, visiting one or many stopover sites, and settling in a new area. Survival of ectoparasites may also vary in response to the changing environmental conditions encountered during the host's migratory cycle. At the host population level, some studies have reported a reduction in parasite prevalence over the course of migration, providing evidence of migratory culling of infected hosts (e.g. Bartel *et al.*, 2011) or host recovery from infection. Migrants that stop over at locations with dense populations of resident, non-migrating conspecifics may experience a surge in infections (Satterfield *et al.*, 2018). Asynchrony in departure time among migrating individuals may also separate infected from uninfected individuals, with consequences for prevalence and transmission over time during migration and at stopover sites and the final destination (Bauer, Lisovski & Hahn, 2016). However, few studies have actually tested the above scenarios by investigating the infection levels by particular parasites in host individuals that survive through the migratory cycle.

Helminth parasites often have sufficiently long lifespans and low virulence to ensure that both they and their avian hosts survive migrations. In a study of common eiders, *Somateria mollissima*, which migrate between wintering grounds in eastern Canada and southern Greenland to breeding grounds further north, some differences in infection levels by certain species of helminths were observed between pre- and post-migratory birds, however these were ascribed to the habitats visited by the birds during migration, rather than to migration in itself (Vestbo *et al.*, 2019). Similar findings have been reported for other migratory bird species (e.g. Yanez & Canaris, 1988). In catadromous and anadromous fishes, which migrate between fresh water and the sea, availability of suitable intermediate hosts in the two habitats determines whether the recruitment of trophically transmitted endoparasitic helminths will vary through the migratory cycle, while tolerance of salinity changes will determine whether infections by ectoparasites show fluctuations (e.g. Aprahamian, 1985). Similarly, whitefish, *Coregonus lavaretus*, migrating up rivers experience a reduction in infection levels by cestodes compared to their conspecifics that permanently reside in lake habitats, since copepods, which are the intermediate hosts of cestodes, are absent from rivers and therefore recruitment of new parasites stops during

migration (Balling & Pfeiffer, 1997). In these cases, the characteristics of the external habitats occupied by the host through the migratory cycle appear to matter more to infection dynamics than any decline in immunocompetence resulting from the energetic demands of migration.

Among the few other attempts to track infection levels through a migratory cycle, serological and genomic data have been used to characterise changes in the incidence of viral infections in migrating birds (e.g. Pearce *et al.*, 2009; Hoye *et al.*, 2011). For most parasites, the ideal approach to monitor changes in infection levels by particular species would be to follow and sample the same marked host individuals, or at least the same cohort, at various stages of migration (before, during, after) in a longitudinal study. For example, Daversa *et al.* (2018) used radio-tracking of toads, *Bufo spinosus*, to show that infections by the fungal parasite *Batrachochytrium dendrobatidis* acquired in the aquatic breeding habitat are inevitably lost during the post-breeding migration into terrestrial habitats. In the absence of further studies providing this kind of information, we can only conclude that infection dynamics over the migratory cycle are likely to depend on the parasite's life cycle, transmission mode, lifespan, and other species-level properties.

IV. MIGRATING TO ESCAPE OR RECOVER FROM PARASITES

In theory, migration can reduce the prevalence of given parasites in the population, and thus the infection risk faced by individuals. All else being equal and if the only benefits of migrating are infection related, individuals in a migratory population could suffer less from parasitism (i.e. lower probability of infection) than those in an identical but non-migratory population. The basic conditions under which migration could reduce infection prevalence are: (*i*) the existence of a parasite-free area accessible from the area of origin, (*ii*) the absence of parasite transmission during migration and at stopover sites, and (*iii*) a sufficient amount of time spent either migrating or residing in the parasite-free area (Johns & Shaw, 2016). Although one mechanism through which migration reduces parasite prevalence is migratory culling, which has no individual-level benefit, selection can still favour migration as an anti-parasite strategy if on average the risk or intensity of infection of migratory individuals is lower than that of non-migrants, and if the cost of infection outweighs the cost of migration. Parasite-mediated selection for migration could even play a role in maintaining migratory divides, i.e. the separation between bird populations that migrate in different directions and winter in different locations, if they are exploited by different parasite faunas (Møller & Szép, 2011).

The notion that animal migrations may have in part evolved, or are maintained by selection, in order to avoid or eliminate parasites has spawned two related hypotheses, migratory escape and migratory recovery. First, the migratory escape hypothesis postulates that natural selection has favoured migratory patterns in which animals migrate towards a refuge with low infection risk, leaving behind an area with a seasonally high risk of infection by debilitating parasites (Loehle, 1995; Piersma, 1997; Altizer *et al.*, 2011). The existence of a safe area to migrate to is necessary for escape. For example, moving to higher latitudes and/or coastal habitats during the non-breeding season allows shorebirds to escape from vector-borne haemosporidian parasites (Mendes *et al.*, 2005; Clark, Clegg & Klaassen, 2016). Migratory escape applies not only in situations where a particular habitat is associated with infection, but also where parasites are transmitted directly by social contacts, in which case moving away from high-density aggregations of conspecifics lowers infection risk (Shaw & Binning, 2020). This can in some cases be intimately linked with reproduction, if migration leads to the spatial segregation, or allopatry, of susceptible juveniles from infected adults, providing an escape from infection for the former (Krkošek *et al.*, 2007). Modelling results indicate that if a pathogen is transmitted only in the breeding area, moving to a different area and staying away for longer lowers both the pathogen's transmission success (R_0, its basic reproductive number) and the likelihood that an individual becomes infected (Hall, Altizer & Bartel, 2014).

Several studies provide empirical support for migratory escape. In the painted lady butterfly, *Vanessa cardui*, which migrates between Europe and northern Africa, infections by parasitoids build up with increasing time spent in both areas until departure, consistent with mounting pressures to migrate towards enemy-free space (Stefanescu *et al.*, 2012). Conversely, populations of monarch butterflies, *D. plexippus*, in the southern USA which benefit from the year-long availability of suitable plants for larval development and no longer migrate to reproduce in central Mexico, incur greater infection prevalence by the protozoan *O. elektroscirrha* than their migratory conspecifics (Satterfield, Maerz & Altizer, 2015). Juvenile galaxiid fishes that migrate out of rivers and into coastal seas avoid infections by freshwater trematodes at a critical period of their life, whereas congeners that do not migrate incur severe trematode-induced developmental malformations and mortality (Poulin *et al.*, 2012). Finally, large-scale migrations of ungulates have also been linked with escape from areas of high infection risk and movement towards parasite-free areas (Folstad *et al.*, 1991; Mijele *et al.*, 2016).

An indirect line of evidence also provides strong support for a role of migratory escape from parasites in the evolution of Afro-Palaearctic passerine birds. Based on the generally well-supported assumption that individuals are exposed to a greater diversity of pathogens in more humid, tropical environments than in temperate ones, and if parasitic diseases are the main drivers of bird migration, we might hypothesise that evolution would favour more strongly the evolution of annual migration towards higher latitudes in tropical birds, than that of migration toward the tropics in temperate birds. In support of this prediction, a phylogenetic reconstruction of evolutionary events among Afro-Palaearctic passerines indicates that northward migration has evolved three times more frequently among African-based species than southward migration

among Palaearctic species (O'Connor *et al.*, 2018). Furthermore, Palaearctic resident species, and those that migrate between Africa and the Palaearctic, have evolved a lower diversity of genes in the major histocompatibility complex class I, than African resident species (O'Connor *et al.*, 2018, 2020). These are key immune genes involved in parasite recognition. Escape from parasite-rich tropical areas, even if only for part of the year, appears to have relaxed selection for broad parasite detection and thus reduced investments in defence.

Secondly, the migratory recovery hypothesis proposes that migration allows infected individuals to lose their parasites and recover from infection (Shaw & Binning, 2016). This will most likely happen if migration occurs along an environmental gradient with increasingly unfavourable conditions for parasite survival or transmission. For example, both correlative (Halttunen *et al.*, 2018) and experimental (Birkeland & Jakobsen, 1997) studies indicate that anadromous brown trout, *S. trutta*, leave coastal waters and return to river habitats sooner when heavily infected by sea lice *L. salmonis*; the fish then rapidly lose their parasites as sea lice cannot tolerate fresh water. Other putative examples of migratory recovery from infection all involve movement along gradients in abiotic factors (see Shaw & Binning, 2016). Since this benefit only applies to infected individuals, migratory recovery could generate plastic migratory strategies within a population. Whether an individual migrates or not, or the timing and distance of migration, may all be infection dependent if recovery from infection is the main benefit. Thus, migratory recovery may explain why some species are partial migrants, with only some individuals migrating and the rest residing in the same area year-round (see also Narayanan, Binning & Shaw, 2020).

In practice, migratory escape and migratory recovery are not totally distinct hypotheses. An infected animal can initiate migration both to *escape* further infections and *recover* from current ones. In simulation models involving a density-dependent, contact-transmitted parasite, migratory recovery emerged as a stronger selective force driving the evolution of migration, compared to migratory escape (Shaw & Binning, 2020). Similar models also reveal that whether or not parasitism can drive the evolution of host migration depends on the parasite's transmission mode and on which fitness components are affected by infection and migration costs (Shaw *et al.*, 2019). Across the broad parameter space considered in these theoretical studies, a wide range of conditions lead to the evolution of migration in response to parasitism, whether the driving benefits are to escape or recover from infection. Given the empirical evidence which also supports a role for parasites, it is therefore likely that they have contributed to shaping the migratory patterns of many extant animal species.

V. PARASITISM IN MIGRANTS *VERSUS* NON-MIGRANTS

Even if some animal species migrate to avoid or get rid of particular parasite species, there are several reasons to expect that, all else being equal, migratory species will be exploited by a greater diversity of parasite species than non-migratory species. Also, within species where not all individuals migrate, we might expect migrants to harbour more parasite species than conspecific residents (e.g. Dick & Belosevic, 1981). First, the physiological costs of migration may lead to a trade-off between investments into long-distance movement *versus* immunocompetence (Altizer *et al.*, 2011; Eikenaar & Hegemann, 2016), making migrants easier resources to exploit than non-migrants. Second, migrants visit and occupy a greater range of habitats than non-migrants, exposing themselves to a wider range of vector-borne pathogens and food items from which they can acquire trophically transmitted parasites. For example, migratory songbirds breeding in Europe and wintering in Africa are parasitised by both European and African lineages of haemosporidian blood parasites, whereas the resident bird species they encounter in each area are only infected by the local parasite lineages (Waldenström *et al.*, 2002). How universal is this pattern? Are migrants consistently parasitised by more species than non-migrants? Here, we use a systematic review approach to address this question using data from comparative studies across related host species. Although several such studies have now been conducted, their findings are yet to be synthesised and evaluated together.

(1) Data collection

We conducted a literature search of the *Web of Science* (WoS) database on 5 October 2020, using the search terms: migrat* AND (parasit* OR pathogen* OR disease* OR infect*), and restricting the records to those falling in the following WoS categories: Parasitology, Infectious diseases, Ecology, Zoology, Fisheries, Entomology, Marine freshwater biology, Evolutionary biology, Biodiversity conservation, Ornithology, and Behavioral sciences. The broad and non-specific search terms made it less likely relevant studies would be missed, and also served to find pertinent literature for other sections of this article. The search returned 8852 records, whose titles and abstracts were checked individually to retrieve comparative studies of parasite species richness *versus* host migration. We retained all studies that considered at least 10 host species and provided a quantitative test of either (*i*) the difference in parasite species richness between migratory and non-migratory animal species within the same higher taxonomic group ($N = 12$), or (*ii*) the relationship between migratory distance and parasite species richness ($N = 4$). Each of these studies provided one independent statistical assessment of the effect of migratory behaviour on parasite species richness. In addition to the outcome of the key statistical test, we recorded whether the study had accounted for three important factors in comparative analyses of parasite species richness: the phylogenetic relatedness among the host species tested (Morand & Poulin, 2003), the unequal study effort among host species (Walther *et al.*, 1995), and the effect of at least one of the host traits, like body size, known to influence parasite diversity (Kamiya

Table 1. Comparative analyses contrasting parasite species richness between migratory (mig) and non-migratory (non-mig) species, or relating parasite species richness to migratory distance. The outcome of the analysis is shown, as well as the confounding variables (if any) that were considered in testing for a difference

Host taxon	Parasite taxa	Number of mig and non-mig host species	Confounding variables*	Migration *versus* parasite richness?	References
Parasite species richness in migratory *versus* non-migratory species					
Marine fishes	Monogeneans	44 mig, 116 non-mig	—	mig > non-mig	Caro *et al.* (1997)
Marine sparid fishes	Monogeneans	5 mig, 9 non-mig	P, T	mig > non-mig	Desdevises (2006)
Marine fishes	Copepods	73 mig, 109 non-mig	—	mig > non-mig	Raibaut *et al.* (1998)
Fishes	Metazoan parasites	Total = 906	P, S, T	mig > non-mig	Shaw *et al.* (2018)
Passerine birds	Feather mites	38 mig, 32 non-mig	P, S, T	mig = non-mig	Figuerola (2000)
Birds	Haemosporidians and trypanosomes	Total = 263	P, S, T	mig = non-mig	Arriero & Møller (2008)
Birds	Haemosporidians	18 mig, 35 non-mig	P, S, T	mig > non-mig	Jenkins *et al.* (2012)
Birds	Nematodes	Total = 188	P, S, T	mig > non-mig	Koprivnikar & Leung (2015)
Birds	Nematodes	Total = 153	P, S, T	mig > non-mig	Leung & Koprivnikar (2016)
Birds	Helminths	Total = 21	P, T	mig > non-mig	Hannon *et al.* (2016)
Bats	Viruses	12 mig, 21 non-mig	P, S, T	mig = non-mig	Turmelle & Olival (2009)
Ungulates	All parasites	35 mig, 48 non-mig	P, S, T	mig > non-mig	Teitelbaum *et al.* (2018)
Parasite species richness *versus* host migratory distance					
Anseriform birds	Haemosporidians	Total = 108	P, S	Positive distance effect	Figuerola & Green (2000)
Birds	Haemosporidians	Total = 145	P, S, T	No distance effect	Gutiérrez *et al.* (2019)
Charadriiform birds	Helminths	Total = 106	P, S, T	No distance effect	Gutiérrez *et al.* (2017)
Birds	Helminths	Total = 327	P, S, T	Positive distance effect	Gutiérrez *et al.* (2019)

* P, host phylogenetic relationships; S, sampling effort per host species; T, any potentially influential host trait, like body size.

et al., 2014). When separate results were provided with and without correcting for phylogenetic influences, we used the former as the more conservative estimate. Finally, because the number of published studies was small, and because effect sizes could not be extracted from all studies, no attempt was made to conduct a formal meta-analysis. Instead, we were limited to a vote-counting approach.

(2) Results and discussion

Nine of the 12 studies testing the difference in parasite species richness between migratory and non-migratory host species found that migratory species harboured more parasite species (or lineages in the case of haemosporidians), whereas none of them reported the opposite pattern (Table 1). All studies on fish supported greater parasite richness in migratory species. Among studies on birds and mammals, there was no association between particular types of parasites and whether or not a study found greater parasite richness in migratory species. Of the four studies testing the relationship between migratory distance and parasite species richness, only two found a significant correlation, whereas the other two found no effect at all (Table 1). Most studies accounted for the potentially confounding effects of host phylogeny, uneven sampling effort and other host traits. Overall, these findings provide some support for an association between migratory behaviour and parasite species richness, with migrants hosting more parasite species than non-migrants. However, the number of comparative tests remains limited, and evaluating them through a qualitative vote-counting approach is far from ideal (see Koricheva, Gurevitch & Mengersen, 2013). For instance, vote-counting does not take the sample size of each study into account (Friedman, 2001). Although sample sizes are generally high in the studies considered here (see Table 1), the apparent support for a link between migratory behaviour and parasite species richness must be interpreted with caution.

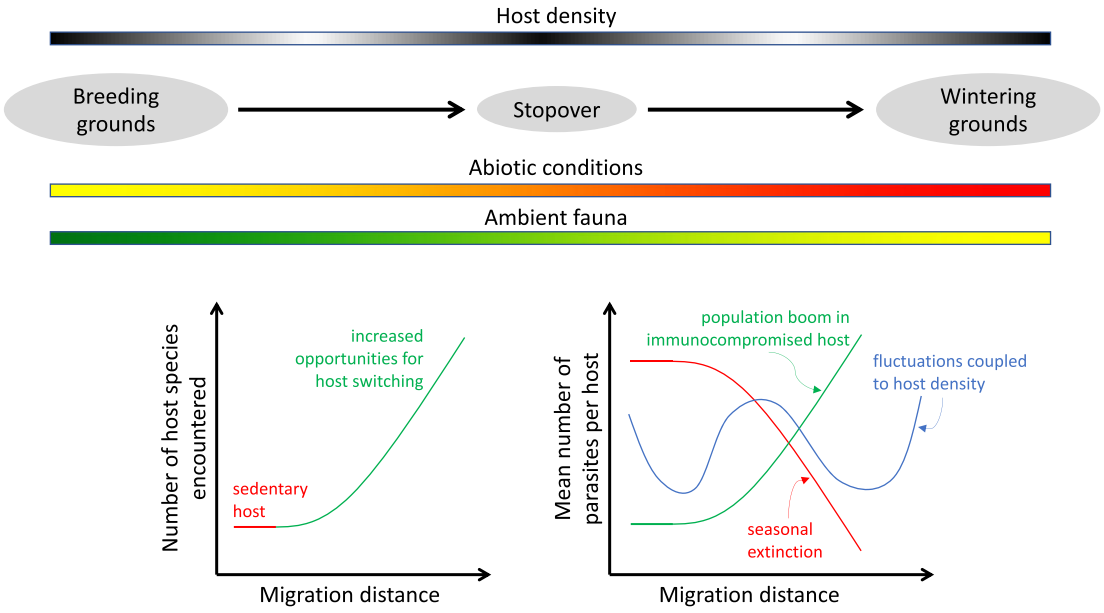

Fig 2. Graphical summary of the immediate consequences for parasites of host migration from the breeding to the wintering grounds (the same would apply for the return phase from the wintering to the breeding grounds, not shown here). The changing ambient fauna over the course of host migration exposes the parasite to an increasing diversity of other potential host species, creating opportunities for host switching. At the same time, the population dynamics of the parasites during host migration will depend on a range of system-specific circumstances. Low tolerance to changing abiotic conditions may cause the parasites to disappear from the migrating host cohort. By contrast, tolerance of changing conditions coupled with compromised host immunity due to the energetic demands of migration may allow the parasites to proliferate. Alternatively, if their transmission is density-dependent and they have short generation times, they may fluctuate in abundance over the course of migration.

The seemingly higher diversity of parasites in migratory host species than in their non-migratory relatives may well arise as a simple consequence of migration taking individuals to a broader range of habitats, where they encounter various food items and vectors. Several comparative studies reporting more parasite species per migratory host species have also highlighted the important role that utilizing multiple habitat types plays as a driver of parasite diversity (e.g. Leung & Koprivnikar, 2016; Gutiérrez *et al.*, 2017). In turn, the expanded parasite fauna of migratory species can impose additional fitness costs and select for host counter-adaptations. Comparative evidence of a different nature also suggests that migratory species may be exposed to greater selection pressures from parasites. Migratory passerines have relatively larger organs associated with defence against parasites, i.e. the spleen and bursa of Fabricius, than their closest, non-migratory relatives (Møller & Erritzøe, 1998). They sometimes also have higher lymphocyte counts, another measure of immune condition (Carbó-Ramirez & Zuria, 2015). However, differences in immunocompetence between migratory and non-migratory species may not apply more generally, as no difference in relative spleen size was seen between migrants and non-migrants in non-passerine birds (Koprivnikar & Leung, 2015). Furthermore, selective pressures favouring investments into immune defence may be driven not by parasite species richness, but by the prevalence of certain parasite species with higher virulence. Comparisons of the prevalence of particular parasite taxa between migratory and non-migratory bird species have yielded inconsistent results (Garamszegi & Møller, 2007; Fecchio *et al.*, 2013; Emmenegger *et al.*, 2018*a*), but these studies remain too few for any conclusion.

VI. MIGRATORY HOSTS AS PARASITE DISPERSAL AGENTS

In this and the next section, we now turn to the implications of host migratory behaviour for parasite ecology and evolution (Fig. 2). Perhaps the most obvious impact of host migration is its potential to spread parasites over broad spatial scales (Altizer *et al.*, 2011). Host migration can transport parasites across latitudes and environments, bring them into contact with new host populations and possibly new suitable host species, and contribute to the expansion of the parasites' geographical range (Boulinier *et al.*, 2016). Migratory hosts connect distant localities in a meta-population context, and therefore can act as 'superspreaders' of infectious agents across populations, in the same way as socially active individuals can act as superspreaders within local populations (Fritzche McKay & Hoye, 2016). The efficacy of host migration to spread parasites depends on the dynamics of infection during the transient phases of migration, i.e. during

Table 2. Non-exhaustive list of tick species, and (if reported) the tick-borne pathogens they harboured, identified as having been dispersed by birds to European or North American regions, from wintering grounds at lower latitudes

Tick species	Pathogen	Bird carrier	Location	References
Haemaphysalis concinna, *Ixodes arboricola*, *I. redikorzevi*, *I. ricinus*	*Rickettsia* spp.	*Turdus merula*, *Turdus philomelos*, others	Romania	Marcutan *et al.* (2016)
Ixodes ricinus, *I. frontalis*, *Hyalomma marginatum*	*Rickettsia* spp., *Borrelia* spp., '*Candidatus* Neoehrlichia mikurensis', *Babesia venatorum*, tick-borne encephalitis virus	*Turdus merula*, *Turdus philomelos*, *Erithacus rubecula*, others	Western Russia	Movila *et al.* (2013)
Ixodes ricinus	*Borrelia* spp.	*Erithacus rubecula*, *Turdus merula*, others	Sweden	Comstedt *et al.* (2006)
Ixodes ricinus, *Hyalomma rufipes*, *Dermacentor* sp.	N/A	*Turdus merula*, *Turdus philomelos*, *Erithacus rubecula*, others	Southern Norway	Hasle *et al.* (2009)
Ixodes ricinus	*Borrelia* spp., *Rickettsia* spp., '*Candidatus* Neoehrlichia mikurensis'	*Erithacus rubecula*, *Turdus merula*, *Phoenicurus phoenicurus*, others	Denmark	Klitgaard *et al.* (2019)
Ixodes ricinus	*Rickettsia* spp., *Babesia* spp., *Anaplasma phagocytophilum*	*Erithacus rubecula*, *Turdus iliacus*, others	Northern Germany	Hildebrandt *et al.* (2010)
Ixodes scapularis, *I. baergi*, *I. muris* *Amblyomma* spp.	*Borrelia burgdorferi*	*Geothlypis trichas*, *Catharus ustulatus*, others	Canada (multiple locations)	Scott *et al.* (2001)
Ixodes scapularis, *I. dentatus*, *I. muris*, *I. pacificus*, *I. auritulus*, *Amblyomma* spp.	*Borrelia burgdorferi*	*Catharus ustulatus*, *Wilsonia pusilla*, *Turdus migratorius*, others	Canada (multiple locations)	Morshed *et al.* (2005)
Ixodes scapularis, *Haemaphysalis leporispaluatris*	N/A	*Catharus ustulatus*, *Geothlypis trichas*, *Turdus migratorius*, others	Ontario (Canada)	Klich *et al.* (1996)
Ixodes dammini, *I. brunneus*, *I. muris*, *Amblyomma maculatum*, *Haemaphysalis leporispaluatris*	*Borrelia burgdorferi*	*Geothlypis trichas*, *Seiurus noveboracensis*, *Melospiza georgiana*, others	Maine, New Hampshire	Smith *et al.* (1996)

movement or at stopover sites; migratory culling and stopover sites unfavourable for parasite transmission can limit the spatial spread of parasites, whereas migration-induced immunosuppression and favourable stopover sites can facilitate it (Daversa *et al.*, 2017). Importantly, migratory animals may be spreading parasites that infect their own species, or they may instead act as vehicles spreading parasites of other, non-migratory species; indeed, migratory animals can even disperse plant parasites (Sugiura & Yamazaki, 2007; Menning *et al.*, 2020).

The most studied parasites in the context of dispersal *via* host migration are those carried by migratory birds that are infective to humans. This simply reflects a study bias, and should not be interpreted as evidence that other types of migratory animals are not important vehicles of parasite dispersal. Research in this area is increasingly driven by concerns that climate change will modify avian migratory patterns, possibly introducing pathogens to new areas and causing emerging infectious diseases (Fuller *et al.*, 2012). For instance, many species of ticks have been recorded on birds returning to northern latitudes from their wintering grounds (see Table 2 for examples). In many reported cases, ticks recovered from migrating birds harbour low prevalence of pathogens capable of infecting humans and causing serious diseases, including viral encephalitis, Lyme disease (*Borrelia burgdorferi*), spotted fever (*Rickettsia* spp.), and neoehrlichiosis (*Neoehrlichia* sp.) (see references in Table 2). Nevertheless, although migrants may carry ticks and tick-borne pathogens across space, a synthesis of North American studies indicates that non-migratory, ground-foraging passerines are mostly responsible for maintaining tick populations locally (Loss *et al.*, 2016). Thus, migrants probably act as occasional dispersal agents, whereas resident birds are the true disease reservoirs.

Multiple lines of evidence also point to migratory birds as critical long-distance transport agents for a range of other human diseases, including mosquito-borne diseases such as West Nile virus (Malkinson *et al.*, 2002; Peterson, Vieglais & Andreasen, 2003; Owen *et al.*, 2006) and St. Louis encephalitis virus (Auguste, Pybus & Carrington, 2009). Multiple

avian influenza viruses, including the highly pathogenic strain H5N1 which can cause severe respiratory illness and even death in some people, are circulated across large areas by migrating waterfowl (Gilbert *et al.*, 2006; Hill *et al.*, 2012; Kwon *et al.*, 2016; van Toor *et al.*, 2018). However, the extent to which migrating waterfowl can spread most strains of avian influenza viruses on intercontinental scales remains controversial (Krauss *et al.*, 2007; Koehler *et al.*, 2008; Latorre-Margalef *et al.*, 2009; Miller *et al.*, 2015). Short viral shedding periods suggest that the efficacy of migratory birds as dispersers of influenza viruses decreases with migratory distance (Lam *et al.*, 2012), such that the poultry trade probably plays a greater role on multiple spatial scales than natural avian migrations (Gauthier-Clerc, Lebarbenchon & Thomas, 2007).

Migratory birds can also disperse avian protozoan parasites. Finches are believed to have introduced the pathogenic *Trichomonas gallinae* from Great Britain to Scandinavia through migration (Lawson *et al.*, 2011). Multiple studies have concluded that migratory birds spread various lineages of haemosporidian blood parasites, which cause avian malaria, between Europe and Africa, and between the Americas (Smith & Ramey, 2015; von Rönn *et al.*, 2015; Ricklefs *et al.*, 2017; but see Pulgarin-R *et al.*, 2019). In addition, it is likely that migratory birds have transmitted *Plasmodium relictum* to endemic Hawaiian honeycreepers (subfamily Drepanidinae) after the human-mediated introduction of suitable mosquito vectors to the islands in the 19th century (Atkinson & LaPointe, 2009). This does not necessarily mean that parasite lineages taken to a new area by migratory birds will successfully infect local birds. For example, *Plasmodium* lineages apparently introduced to the Galapagos Islands by migrating bobolinks, *Dolichonyx oryzivorus*, cannot complete their development in endemic Galapagos birds (Levin *et al.*, 2013). Similarly, resident birds in the Dominican Republic and migratory birds wintering there harbour distinct haemosporidian parasite species, with very few in common (Soares, Latta & Ricklefs, 2019). Nevertheless, transportation to new areas and exposure to a community of potentially new hosts creates ecological and evolutionary opportunities for parasites that are unavailable to species not infecting migrants.

VII. HOST MIGRATION SHAPING PARASITE LIFE-HISTORY STRATEGIES

Dispersal is an important form of animal movement related to migration that also serves to connect spatially separate populations or localities. It differs from migration, however, in that it involves the passive or active movement of individuals away from where they were produced, it is not necessarily seasonal or directional (i.e. there is no single destination for all individuals), and it does not necessarily involve a return to the point of origin. Inevitably, host dispersal is an important driver of parasite population genetic structure, although it is not the only one (Mazé-Guilmo *et al.*, 2016). All else being equal, however, dispersing hosts allow for greater gene flow among parasite populations than do resident hosts, with implications for local adaptation and the spread of beneficial alleles (Gandon & Michalakis, 2002). Host dispersal may also shape the evolution of parasite virulence, pushing it to higher levels than in purely resident host populations (Lion & Gandon, 2015). Therefore, host migratory behaviour should also impose selective pressures on parasites that may lead to adaptations different from those favoured in parasites of non-migratory hosts. Unlike other large-scale host displacements, such as anthropogenic species introductions to new areas, host migrations represent repeated, predictable, and directional transport of parasites, exerting strong selective pressures on parasites to adapt. The influence of host migration on parasite evolution has received much less attention than the effects of parasite infections on host migratory behaviour. There are no broad comparative analyses available that contrast the life-history traits of parasites of migratory hosts *versus* those of closely related parasite species exploiting non-migratory hosts. We may nonetheless expect certain traits to be favoured in parasites of migratory hosts.

First, just as selection may, under some circumstances, favour higher levels of virulence in parasites exploiting host populations that undergo dispersal than those that do not (Lion & Gandon, 2015), it may also drive the evolution of virulence in parasites of migratory host species. There is much theoretical support (Cressler *et al.*, 2016) and some empirical evidence (Eshelman *et al.*, 2010) that this is the case. Virulence is generally considered as a parasite trait, although it is measured through host phenotype (e.g. increased host mortality or reduced host fecundity). Therefore, it should perhaps be seen as a property of the host–parasite interaction, determined by both parasite exploitation strategies and host tolerance and compensation mechanisms (Poulin & Combes, 1999). Nevertheless, the different transmission landscape associated with host migration, such as the dense aggregations of host individuals on breeding grounds occurring repeatedly each year, could select for more aggressive exploitation of host resources, and thus higher virulence.

Second, host-mediated dispersal across large geographic scales means that parasites of migrants should on average be exposed to a broader range of alternative host species than parasites of non-migrants, which should favour a relaxation of host specificity and facilitate host-switching. Host migration provides repeated re-introduction to an area and sustained propagule pressure, increasing the likelihood that parasites successfully colonise new hosts and expand their niche (Mestre, Poulin & Hortal, 2020). For example, multiple lineages of vector-borne haemosporidians infecting migratory birds have independently evolved year-round transmission, seemingly by adopting a wide range of blood-feeding insects that may act as suitable vectors in both their host's breeding and wintering grounds (Pérez-Tris & Bensch, 2005). Migratory birds also show weaker coevolutionary relationships with their haemosporidian parasites than non-migratory birds, because of more frequent host-switching (Jenkins *et al.*, 2012). Indeed, host migration

may be a key driver of haemosporidian diversification (Fecchio *et al.*, 2019). Similarly, trophically transmitted helminths would benefit from increased transmission opportunities if they could use the various species of potential intermediate hosts available at both ends of their host's migratory route. Therefore, we might predict that parasites using migratory hosts are closer to the generalist end of the specialisation spectrum than related parasites strictly using resident hosts.

Third, the parasite's phenology and life-cycle duration, including the timing of major developmental, reproduction and transmission events, are also under strong selection imposed by host migrations. In fact, parasite adaptations would be expected particularly in response to host strategies that rely on migration to escape from infection (see Section IV). Assuming some level of synchrony among migratory individuals and inter-annual consistency in migratory patterns (Bauer *et al.*, 2016), selection might favour either accelerated life cycles to allow completion during limited temporal windows for transmission, or periods of dormancy or extended lifespans in intermediate hosts to await the return of migratory definitive hosts. Other adjustments to life cycle and developmental patterns are possible. For instance, some haemosporidian parasites of migratory birds have synchronised the production of gametocytes (the stages transmissible to mosquito vectors) to match the return of their hosts to breeding areas (Soares, Young & Ricklefs, 2020). Both the transmission dynamics and incidence of reassortment (horizontal genetic exchanges) in avian influenza viruses coincide with the period spent by their migratory hosts at the breeding grounds (van Dijk *et al.*, 2014; Hill *et al.*, 2016). Some helminths can time their egg release to coincide with the brief annual breeding visits of their hosts to particular habitats, using osmotic or endocrine changes in the host as cues to synchronise their reproduction with that of their host (Shostak & Dick, 1989; Tinsley, 1999). There is no reason why parasites of long-distance migratory hosts could not use similar mechanisms to match their reproduction with arrival in a suitable habitat. Finally, host migration can even favour certain modes of transmission over others, i.e. vertical rather than horizontal transmission to enable persistence across the migratory cycle (Vilaplana *et al.*, 2010).

Fourth, the changing environmental conditions experienced by parasites over the long-distance migration of their hosts should select for increased tolerance and the ability to survive and/or transmit under a wider range of abiotic conditions. There are exceptions: in some migratory species, niche tracking allows migrants to experience lower climatic variation than residents (Eyres *et al.*, 2020), which could provide stable environmental conditions for their parasites. In most cases, however, long-distance host migration is associated with changes in environmental conditions. Migratory animals may actually move to abiotically different habitats in order to recover from infections (see Section IV). In response, variable conditions will select for parasite adaptations. For instance, the ectoparasitic monogenean *Gyrodactylus salaris*, a parasite of Atlantic salmon, *Salmo salar*, demonstrates greater tolerance to salinity than other related monogeneans whose hosts complete their entire life cycles in fresh waters (Soleng & Bakke, 1997). Although the parasite cannot survive in sea water, it can tolerate brackish water, which may explain how it spreads among different river systems with migrating salmon (Soleng & Bakke, 1997; Soleng, Bakke & Hansen, 1998). Endoparasites can also benefit from adapting to changing external conditions during host migration. The freshwater myxosporean *Myxidium salvelini*, for example, initiates arrested development after its sockeye salmon, *Oncorhynchus nerka*, host enters sea water, and resumes spore production once the host returns to fresh water (Higgins, Margolis & Kent, 1993). This strategy allows the parasite to survive during its host sojourn in an unfavourable environment.

Yet another possible evolutionary path exists for parasites of migratory hosts: they could modify the migratory behaviour of their hosts to their own advantage. The ability to manipulate host phenotypic traits has evolved repeatedly across multiple parasite taxa (Poulin, 2010). From a theoretical perspective, targeted manipulation of host spatial dispersal should be favoured under a wide range of conditions (Lion, van Baalen & Wilson, 2006). Empirical evidence confirms that a wide range of parasites reduce the locomotory performance of their hosts, affecting their speed and stamina (McElroy & de Buron, 2014). Parasites are known to modify small-scale migratory patterns of invertebrate hosts, with possible advantages for their own transmission; examples range from trematodes altering the upshore–downshore migrations of their intertidal snail hosts (Lambert & Farley, 1968; Curtis, 1993), to parasitic dinoflagellates changing the diel vertical migration of bloom-forming dinoflagellates (Park *et al.*, 2002). There is no reason why long-distance host migration should not also be the target of parasite manipulation. A simulation model of parasite dynamics in long-distance migratory hosts reveals that 'migratory stalling', i.e. the parasite-induced halting of host migration, has positive feedback for parasite transmission (Peacock *et al.*, 2018, 2020), providing support for the hypothesis that parasites can benefit from manipulating host migratory behaviour. As with the other parasite adaptations mentioned above, robust comparative studies between parasite taxa exploiting migrant hosts and their relatives exploiting resident hosts are needed to confirm whether host migration has left its evolutionary signature on parasite life-history strategies.

VIII. PARASITES AND MIGRATION: A UNIFIED FRAMEWORK

Research into the interaction between animal migration and parasitism has been mostly one-sided: much more is known regarding the implications of parasitic diseases for animal migrations than regarding the consequences of host

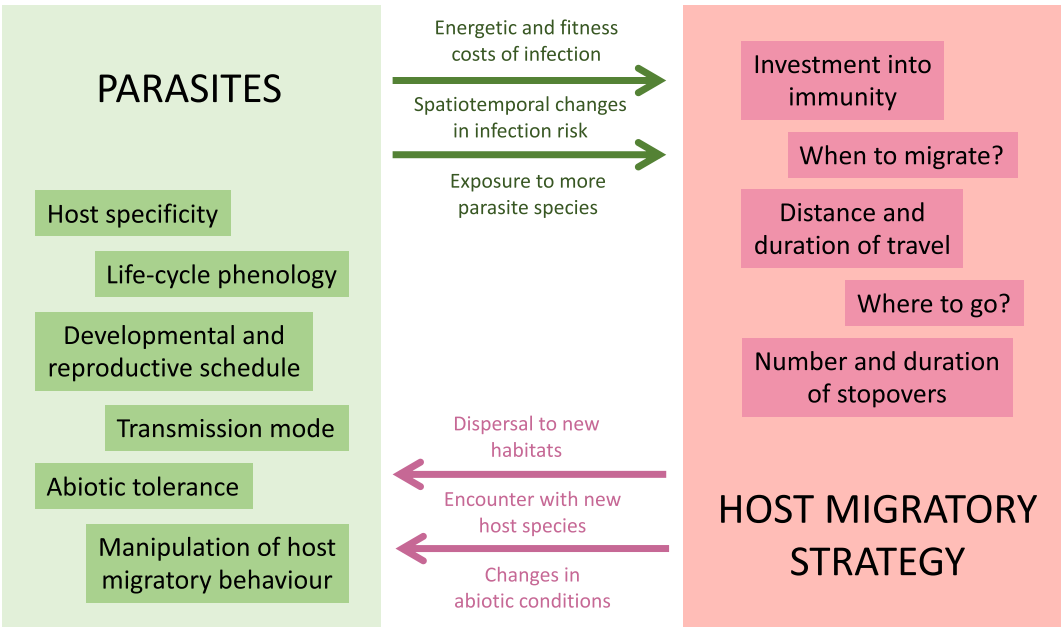

Fig 3. Feedback between parasitism and host migration, highlighting the main reciprocal selective pressures they each impose on the other. Only some of the parasite traits likely influenced by host migration, and only some aspects of host migratory behaviour likely to be shaped by parasitism, are shown.

migration for parasite ecology and evolution. Practically all studies of the interaction between parasitism and migration have used a correlational approach, making it difficult to distinguish cause from consequence. To a large extent, we have relied on the interpretations of the authors of the original studies to assign a direction to the cause-and-effect pathway in each case. Experimental approaches, in which variables relating to migration (e.g. distance covered, flight or swimming speed, resources available in transit) or parasitism (infection levels, timing of infection, etc.) are manipulated, will be needed to confirm the outcomes of correlational studies. Nevertheless, feedbacks do exist between parasitism and host behaviour, and understanding them is essential to resolve better how they have coevolved (Ezenwa *et al.*, 2016) and how they may evolve further in response to environmental change (Garamszegi, 2011; Cohen *et al.*, 2018).

The feedbacks between animal migration and parasitism are best visualised as reciprocal selective pressures acting simultaneously in both directions (Fig. 3). Whether parasites have been important forces driving the evolution of migration (e.g. the migratory escape or migratory recovery hypotheses), or whether they merely impose an additional energetic cost during migration, inherent plasticity in animal behaviour combined with spatiotemporal changes in infection risk and/or parasite diversity/virulence should select for adjustments in migratory behaviour. Where and when animals leave an area, how long and how far they travel, and how many stops they make during their route, are all traits shaped to some extent by patterns of parasite distributions across time and seasons. Recent observations of changes in migration phenology (Bussière *et al.*, 2015; Kristensen *et al.*, 2015; Cohen *et al.*, 2018), for instance, cannot be understood fully independently of changes in the disease landscape. Changes in parasite pressures will also continue to shape investments in immune defence, as they have in the past based on their genomic signature in migratory passerines (O'Connor *et al.*, 2018, 2020).

At the same time that they exert pressures on their hosts, parasites face reciprocal pressures to adapt to the migratory behaviour of their hosts (Fig. 3). Fundamental characters such as virulence, transmission mode, developmental schedules and tolerance of infective stages to abiotic conditions are likely under strong selection from the habitat shifts and fluctuations in host population density that are associated with migratory behaviour. Comparative analyses are lacking, however there are good reasons to assume that parasites of migratory hosts possess a different suite of traits from those of related species exploiting sedentary hosts.

Positive feedbacks can link traits in two species and have major effects on their evolution (Crespi, 2004). They can be viewed as instances of self-reinforcing antagonistic coevolution. For instance, positive feedback between parasitism and host migration can lead to causal loops across generations, with increases in a host trait (e.g. greater migratory distance) driving increases in a parasite trait (e.g. greater tolerance to changing abiotic conditions), and *vice versa*. Negative feedbacks, on the other hand, can dampen the strength of reciprocal selective pressures; for example, a decrease in parasite virulence may weaken the pressures on the hosts to migrate, and *vice versa*. In either case, feedbacks between parasitism and host migration act both within or across generations: what begins as adjustments through phenotypic

plasticity on short timescales can lead to adaptive shifts in gene frequencies over evolutionary time (Pelletier, Garant & Hendry, 2009). The asymmetry in generation times between parasites and hosts may lead to evolutionary changes occurring at different speeds in hosts and parasites, with phenotypic plasticity playing a more important role for the hosts. Regardless, the feedback framework allows the simultaneous consideration of both causal pathways (parasites shape host migrations, host migrations affect parasites; Fig. 3), and provides a better platform to study dynamic interactions between migratory animals and their parasites in a changing environment. Migratory behaviour has not evolved in a biotic vacuum; beyond their predators and their food resources, migratory animals have coevolved with parasites and the diseases they cause. Biotic interactions are increasingly recognised as important modulators of the eco-evolutionary dynamics of animal dispersal and range expansion (Miller *et al.*, 2020) – processes related to migration. Considering the feedback between parasitism and migration is urgently needed for an integrated, holistic perspective of animal migrations, and to anticipate their future evolution in a changing world.

IX. CONCLUSIONS

(1) Migratory behaviour is ubiquitous across animal taxa ranging from invertebrates to vertebrates. Although migrations are usually examined in the context of avoidance of predators, food scarcity or harsh conditions to maximise reproductive success, animal movements on large spatial scales are also linked with parasitism. Parasitic infections can be both causes and consequences of migrations, whereas host migration presents opportunities and challenges for parasites.

(2) Parasites impose costs on their migrating hosts, which can result in decreased performance manifested as reduced speed, longer stopovers, delayed arrival at the destination, or even disorientation during migration. The trade-off between the energetic demands of migration and resistance against infections, combined with changing exposure to new parasites along the migratory route, can result in fluctuating infection levels. In some cases, heavily infected individuals may be culled from the host population during migration.

(3) Under the right conditions, parasites can act as selective forces favouring the evolution of migration, or acting to maintain it. By moving to a parasite-free area, animals can avoid further infections (migratory escape), eliminate current infections (migratory recovery), or both. Past selective pressures from parasites may reveal themselves in the particular areas used by animals as breeding or wintering grounds, and even in genomic differences between migratory and non-migratory species.

(4) The compromised immune resistance of migrating animals due to the demands of sustained movement, and the greater variety of habitats they traverse, may expose them to infections by a greater diversity of parasites. A systematic review of published comparative studies linking parasite species richness to migratory behaviour reveals that, in the majority of taxa investigated, migratory species harbour a richer parasite fauna than closely related but non-migratory species.

(5) From the parasites' perspective, host migration serves as an efficient long-distance dispersal mechanism. Migratory birds, in particular, have been implicated as dispersal agents for a broad range of human diseases, from influenza viruses to several tick- or mosquito-borne protozoan pathogens. However, dispersal *via* migratory hosts also provides greater opportunities for parasites to encounter potential new host species and expand their host range through host-switching, relative to what is available to parasites of non-migratory hosts.

(6) Host migration also imposes strong selective pressures on parasites: compared to what they would experience in non-migratory hosts, parasites of migrants undergo pronounced changes in host density, external abiotic conditions, and the locally available fauna they may use as intermediate hosts or vectors for transmission. These pressures may select for adjustments in the phenology (timing or duration) of key life-cycle, developmental and/or reproductive events, virulence, host specificity, or tolerance of a broader range of abiotic conditions. Host migration may even favour parasites capable of manipulating the migratory behaviour of their hosts to their own advantage, inducing any modification in host migration that may improve parasite transmission.

(7) Finally, we knit together these various causal strands into a unified framework based on reciprocal, coevolutionary feedbacks between hosts and parasites. Parasites can shape host migratory strategy over evolutionary time, while host migration simultaneously selects for life-history adaptations in parasites. For instance, positive feedback loops can accelerate and reinforce connections between host and parasite traits, while negative feedback can dampen them. We encourage future studies to adopt this integrated approach in any attempt to predict how climatic and environmental changes will affect long-distance animal movements.

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