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Author for correspondence:
Allison K. Shaw
e-mail: ashaw@umn.edu

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Metrics matter: the effect of parasite richness, intensity and prevalence on the evolution of host migration

Allison K. Shaw[1], Julie Sherman[3], F. Keith Barker[1,2] and Marlene Zuk[1]

[1]Department of Ecology, Evolution, and Behavior, and [2]Bell Museum of Natural History, University of Minnesota, St Paul, MN 55108, USA
[3]Department of Mathematics, University of Minnesota, Minneapolis, MN 55455, USA

AKS, 0000-0001-7969-8365

Parasites have long been thought to influence the evolution of migration, but precisely determining the conditions under which this occurs by quantifying costs of infection remains a challenge. Here we developed a model that demonstrates how the metric used to describe infection (richness/diversity, prevalence or intensity) shapes the prediction of whether migration will evolve. The model shows that predictions based on minimizing richness yield opposite results compared to those based on minimizing prevalence, with migration only selected for when minimizing prevalence. Consistent with these findings, empirical studies that measure parasite diversity typically find that migrants are worse off than residents, while those measuring prevalence or intensity find the opposite. Our own empirical analysis of fish parasite data finds that migrants (of all types) have higher parasite richness than residents, but with no significant difference in either prevalence or intensity.

1. Background

Migration is a ubiquitous behaviour that spans a large range of temporal and spatial scales [1]. Although stereotypical images of migration often feature birds moving to the equator for the winter and ungulates travelling across African savannahs, migratory behaviour is a much broader phenomenon. Amphibians have small-scale migrations between aquatic and terrestrial habitats [2], as do many land crabs [3]; moths migrate altitudinally to escape seasonally hot conditions [4]; plankton migrate daily up and down in the water column [5]; and some sea lions migrate to breed every 17–18 months [6]. The uniqueness of migration comes from the predictable and directional nature of the movement. However, movement of all forms can be inherently costly in terms of depleting energy, reducing survival and increasing exposure to novel and uncertain conditions [7].

Why, then, do organisms migrate? Three broad sets of factors are thought to shape migration [8]. First, some species migrate between breeding/spawning grounds and areas that are better suited to adult survival or resource accumulation. Second, some species are constantly on the move tracking changing resource patterns. Third, some species spend part of the year in one area that is well suited for breeding and foraging, but migrate away to seek refuge (from cold, dry or stormy weather) during part of the year. Both early and modern conceptions of migration as a refuge behaviour focus on climate as a driving factor [8,9]. However, these seasonal movements may also be driven by predators [10,11], parasitoids [12], or parasites and pathogens [13].

The role of parasites in determining host ecology and behaviour is becoming increasingly recognized [14,15]. Parasites (which here we define broadly to include both macroparasites and microparasites, as per [16]) in particular can shape migration patterns in several distinct ways. By migrating, individuals can escape parasites that are restricted to certain habitats [17,18], or move to

Table 1. Summary of empirical studies that have compared parasite infection between migratory and non-migratory hosts. Metrics to quantify parasite infection include: diversity (typically richness, the number of species present), prevalence (the number or fraction of individuals infected) and intensity (the number of parasites per infected individual).

	migrants have less	no significant difference	migrants have more
diversity		*Plasmodium* and *Haemoproteus* spp. in passerine birds [25] haemosporidian parasites in junco birds [26]	haematozoa in Anseriform birds [21] haematozoa in birds [27] nematodes in Anseriform and Accipitriform birds [28,29] parasites in Artiodactyla and Perissodactyla mammals [30]
prevalence	protozoans in monarch butterflies [31,32] haemosporidian parasites in junco birds [26]	blood parasites in sparrows [33] intestinal parasites in European Passerine birds [34] parasites in Artiodactyla and Perissodactyla mammals [30]	
intensity	warble fly in reindeer [17] trematodes in galaxiid fish [13] ticks in red deer [35] isopods in French grunt fish [36]	blood parasites in sparrows [33] haemosporidian parasites in junco birds [26]	

or through new environments that facilitate recovery from infection [19,20]. Conversely, individuals may also encounter novel parasites as they migrate to or through new habitats [21–23]. However, determining the actual costs of parasite infection from exposure risk is challenging, and hence it has been difficult to understand the role of parasites in the evolution of host migration.

Often, the cost of parasite infection is inferred indirectly by measuring the types and abundances of parasites present in a host population. Three metrics are commonly used to quantify infection: diversity, prevalence and intensity [24]. Diversity describes the variety of parasite types (typically quantified as richness, the number of parasite species) either within a single host individual or within the host population. Prevalence describes the fraction of the host population infected with a parasite. Finally, intensity (or level) describes the average number of individual parasites of a given type present in an infected host. The majority of studies documenting a relationship between migration and infection use only a single metric (table 1), and only one study that we know of has measured all three simultaneously [26]. Importantly, the choice of infection metric can shape the perception of whether migration is costly or beneficial in terms of infection. Studies that quantify infection diversity typically find that migrants are worse off than non-migrants and conclude infection is a cost of migration [27,28]. By contrast, studies quantifying infection prevalence or intensity typically find that migrants are better off than residents and conclude that migration has infection-related benefits [13,17,31,37].

This apparent contradiction may be resolvable by considering several metrics of parasite infection simultaneously, using either theoretical or empirical approaches. A theoretical approach can provide a conceptual framework to determine how the different measures of infection shape evolutionary outcomes. Such a model can be used to understand under what conditions migration has infection-related costs and when it has infection-related benefits (measured by several infection metrics), and under what conditions the host is expected to evolve migration or residency. Theoretical results may then inform empirical studies that examine a set of taxa, comparing the degree of parasite infection of migrants versus residents using more than one metric of infection. It may be the case that most migrants have lower prevalence (or intensity) but higher richness of parasites. Conversely, in some clades, migrants may show both greater infection prevalence and richness (suggesting other factors drive the evolution of their migration) while in other clades migrants have both lower infection prevalence and richness (suggesting that infection-related benefits could favour migration).

Here, we first take a theoretical approach to resolve this difficulty in understanding how parasite infection shapes host migration. We develop a mathematical model with the built-in assumption that migratory individuals are exposed to a greater richness of parasites than non-migratory ones, and we determine under what conditions migrants have a higher or lower prevalence of infection than non-migrants. We use our model to determine when migration is a better strategy than residency, which enables us to conclude when prevalence versus richness is a stronger factor shaping the evolution of migration. Thus, in this model, infections (both prevalence and richness) are simultaneously causes and consequences of host movement patterns.

In the light of these theoretical insights, we next conduct a non-exhaustive empirical study as a step towards examining the importance of considering different infection metrics empirically. Since existing studies on the topic typically use a single metric, we wanted to make an initial attempt at examining three infection metrics simultaneously. We surveyed fish and their parasites, a group of species with well-documented life history and host–parasite interactions. We chose fish because datasets on this taxonomic group are

readily available in the literature and have been assembled without regard to any hypothesis about the relationship between parasitism and migration. Our empirical work is meant to complement our theoretical work (rather than test it directly), and offer guidelines and directions for future research.

2. Material and methods

(a) Model development

Our model plays out in two habitats, each with its own endemic parasite (figure 1). We think of these as two fairly similar parasites (e.g. two species of blood parasites or two species of intestinal parasites), although it could also describe two very different parasite species.

We track four types of individual hosts: susceptible (S, uninfected), infected with just parasite 1 (I_1), infected with just parasite 2 (I_2) and infected with both parasites 1 and 2 (I_3). See [38] for all model symbols and their meanings. While in environment 1, individuals that are not already infected by parasite 1 become infected at rate β_1. In other words, S individuals move to I_1 while I_2 individuals move to I_3, with dynamics given by

$$\frac{dS}{dt} = -\beta_1 S, \tag{2.1a}$$

$$\frac{dI_1}{dt} = \beta_1 S, \tag{2.1b}$$

$$\frac{dI_2}{dt} = -\beta_1 I_2 \tag{2.1c}$$

and

$$\frac{dI_3}{dt} = \beta_1 I_2. \tag{2.1d}$$

Similarly, in environment 2, individuals that are not already infected by parasite 2 become infected at rate β_2. Here, S individuals move to I_2 while I_1 individuals move to I_3, with dynamics given by

$$\frac{dS}{dt} = -\beta_2 S, \tag{2.2a}$$

$$\frac{dI_2}{dt} = \beta_2 S, \tag{2.2b}$$

$$\frac{dI_1}{dt} = -\beta_2 I_1 \tag{2.2c}$$

and

$$\frac{dI_3}{dt} = \beta_2 I_1. \tag{2.2d}$$

Hosts can move between these habitats throughout the year. All individuals start in environment 1 (the breeding habitat), spending the first part of the year (time T_1) there. Next, a fraction θ of individuals migrate to environment 2 and spend the second part of the year (time T_2) there, then return to environment 1 to reproduce. The remaining $1-\theta$ individuals (non-migratory residents) stay in environment 1.

Prior to reproduction, we assess survival. Susceptible residents have the highest annual survival probability ($\sigma_{SR} = 0.9$) because they avoid both the costs of infection and of migration. Resident individuals with either just parasite 1 or parasite 2 have lower survival probabilities (σ_{1R} and σ_{2R}, respectively), determined by the infection cost of each parasite. For individuals infected with both parasites 1 and 2, we assumed that the cost of both parasites was the higher of the cost of each parasite separately. In other words, their survival probability is $\sigma_{3R} = \min(\sigma_{1R}, \sigma_{2R})$. For comparison, we also considered parasite costs that were additive (the cost of both is the sum of each separate cost), subadditive (the cost of both is less than the sum of each separate cost) and superadditive (the cost of both is more than the sum of each separate cost). For these cases, the survival probability of individuals infected with both parasites is

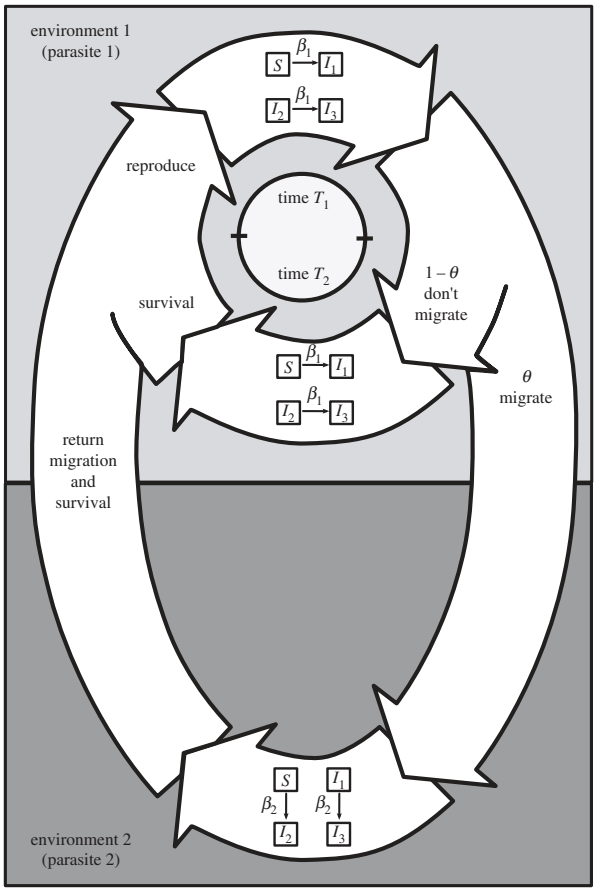

Figure 1. Model schematic. A fraction $(1-\theta)$ of individuals stay in environment 1 year-round (where they are infected by parasite 1 at rate β_1), the remaining individuals migrate to environment 2 for part of the year (where they are infected by parasite 2 at rate β_2) but return to environment 1 to reproduce.

$$\sigma_{3R} = \sigma_{SR} - (\sigma_{SR} - \sigma_{1R}) - (\sigma_{SR} - \sigma_{2R}) - \varepsilon, \tag{2.3}$$

where ε is the magnitude of interaction, with $\varepsilon = 0$ for additive, $\varepsilon > 0$ for superadditive and $\varepsilon < 0$ for subadditive. We assume that migrating incurs a survival cost, such that susceptible migrants have lower survival than susceptible residents ($\sigma_{SM} = \sigma_{SR} - 0.1$), and infected migrants similarly have lower survival than their resident counterparts ($\sigma_{1M} = \sigma_{1R} - 0.1$, $\sigma_{2M} = \sigma_{2R} - 0.1$ and $\sigma_{3M} = \sigma_{3R} - 0.1$).

At the end of the year, surviving individuals reproduce. Susceptible individuals have the highest fecundity ($\varphi_S = 3$), and individuals with either just parasite 1 or parasite 2 have lower fecundity (φ_1 and φ_2, respectively). As a baseline, we set $\varphi_1 = \varphi_2 = 1$ and varied their relative values in other simulations. For individuals infected with both parasites 1 and 2, we assumed a cost structure similar to survival with $\varphi_3 = \min(\varphi_1, \varphi_2)$ and additive, subadditive and superadditive costs for comparison, with

$$\varphi_3 = \varphi_3 - (\varphi_3 - \varphi_1) - (\varphi_3 - \varphi_2) - \varepsilon. \tag{2.4}$$

Births are density-dependent, where the total number of offspring born is

$$b = b_{max}\exp(-\delta b_{max}), \tag{2.5a}$$

where δ is the density-dependent fecundity coefficient and

$$b_{max} = S\varphi_S + I_1\varphi_1 + I_2\varphi_2 + I_3\varphi_3, \tag{2.5b}$$

is the maximum total number offspring produced (occurring at low density). See [38] for full model equations.

(b) Model simulations

We ran simulations of our model, focusing on three factors: the relative transmission rates (β_1 versus β_2), survival costs (σ_{1R} versus σ_{2R}) and fecundity costs (φ_1 versus φ_2) of the two parasites. We set the following as baseline parameter values: $\beta_1 = 1$, $\beta_2 = 1$, $\sigma_{1R} = 0.5$, $\sigma_{2R} = 0.5$, $\varphi_1 = 1$, $\varphi_2 = 1$. Then, to vary transmission rates we let $0 \leq \beta_2 \leq 2$, to vary survival costs we let $0 \leq \sigma_{2R} \leq \sigma_{SR}$, and to vary fecundity costs we let $0 \leq \varphi_2 \leq \varphi_S$. For each set of parameter values, we first simulated a population of only migrants ($\theta = 1$) and a separate population of only residents ($\theta = 0$), each run for 250 years (enough to ensure the population reached its ecological equilibrium). We quantified the parasite richness (number of parasites infecting any individual) and prevalence (fraction of the population infected with any parasite) for each population.

Next, we analysed our model, using pairwise-invasibility plots (PiP) [39] to determine the evolutionarily stable strategy (ESS) [40]; that is, the migration probability that is favoured by selection (θ_{ESS}). To construct a PiP, we first chose a dominant (most abundant) migration strategy (value of $\theta = \bar{\theta}$). We simulated a population with a strategy $\bar{\theta}$ for 250 years. Next, we introduced an individual with a mutant migration strategy (value of $\theta = \theta'$) and calculated its rate of growth analytically, as quantified by the dominant eigenvalue of the Jacobian matrix, $\lambda_{dom}(\bar{\theta}, \theta')$ (see [38] for details). A mutant strategy with $\lambda_{dom} > 1$ is counted as being able to invade the population. We repeated this process for all pairwise combinations of $\bar{\theta}$ and θ', for $\theta = 0, 0.01, 0.02, \ldots, 1$. Finally, the ESS is identified as the value of $\bar{\theta}$ for which no mutant strategies can invade, that is

$$\lambda_{dom}(\bar{\theta}, \theta') < 1 \forall \bar{\theta} \neq \theta'. \tag{2.6}$$

(c) Data collection

Next, we collected empirical data to examine the effect of different metric types, as a complementary approach to our model. We compiled the most comprehensive list of fish species with the parasite and life-history data that we could. We obtained the complete database provided with the Fish Parasite Ecology Software Tool (FishPEST [41]), giving us a list of 4650 distinct fish host species which were known to have at least one parasite. Next, we wrote a Python (https://www.python.org/) script that searched for each identified host species in FishBase, a database with taxonomic, life history and ecological details for over 33 000 fish species [42] and extracted migratory information (migratory or non-migratory) for each species. Migratory species were further classified as anadromous, amphidromous, catadromous, oceanodromous or potamodromous. We were able to find migratory data for 1290 host species. The Natural History Museum Host–Parasite Database [43] provided more comprehensive reporting of fish–parasite relationships than FishPEST; consequently, using a Python script we queried the NHM database for each of the 1290 host species with known migratory status, extracted all parasites which had been documented as infecting it, and the corresponding reference information. We found parasite information for 906 host species in the NHM database. Finally, because our database was primarily endoparasites, we supplemented it with ectoparasites by manually entering all the crustacean parasites listed in [44] that were found on any of the 906 host species in our data. Note that [44] only covers North American species, but it was the only robust source of ectoparasite data for fish that we could find; thus we erred on the side of including some ectoparasite data rather than excluding them entirely. Analyses of richness data (see below) were performed both with and without the ectoparasite data in order to assess their impact on the results (see the output in [38] for details). This dataset was the final dataset that we analysed (i.e. we did not analyse host–parasite data from FishPEST).

Using queries to an SQLite [45] database of the results described above, we quantified parasite richness as the total number of parasite species observed for each host species. Quantification of sampling effort is critical in evaluating parasite species richness [46]. As a first attempt to do so, we counted the distinct references (across all parasites) for each host species, as a potential metric of effort. However, previous studies have shown that the number of publications can be a misleading measure of parasite sampling effort, because of parasitological biases towards reporting new host associations [21] (see also Results below); consequently, we also quantified effort using the total number of specimens of host species in museum collections. This number gives a good approximation of the ease with which different host species are sampled, as well as a direct measurement of host individuals available for parasitologists to examine. Both the number of lots and the number of specimens were calculated by downloading the September 2016 snapshot of VertNet's (http://vertnet.org/index.html) fish data [47], then tabulating lots and individuals by the concatenated genus and species entries of each record in R [48].

Next, we calculated infection prevalence and intensity for a random subset of host–parasite pairs as follows. We generated a list of all the unique references in our database that had a known year of publication (2928 total) and chose a single host from each one. Next, for all hosts in this new list, we selected one reference at random, yielding 523 references. We selected 325 of these references to search for online. (We systematically searched for the first 250 references, which led to a dataset with primarily migratory hosts, and so for the remaining 273 references we only searched for the 68 that included at least one non-migratory host. This still yielded a dataset of mostly migratory hosts, so we added 7 more references with at least one non-migratory host, giving us 97 non-migratory species.) We were able to find 175 of the 325 references we searched for. We then skimmed these articles looking for prevalence and/or intensity data. Of these 175 articles, 61 had data on parasite prevalence (across 45 non-migratory and 59 migratory hosts) and 52 had data on parasite intensity (across 26 non-migratory and 41 migratory hosts). Using recorded sample sizes, prevalence data were transformed to binary categorical (uninfected/infected) response data, with one record per sampled individual, using a script in R. Similarly, intensity data were transformed to a total number of parasites per individual.

(d) Data analysis

Parasite richness, prevalence and intensity data were all analysed comparatively using generalized linear mixed models, incorporating phylogenetic relationships among sampled host species, as implemented in the R package *MCMCglmm* [49,50]. Phylogenetic relationships among host species and their estimated body sizes were obtained from the tree of 7824 fish species inferred by Rabosky *et al*. [51] and accompanying data. The datasets described above were filtered so that only host species included in Rabosky *et al*.'s tree were retained, except when non-overlapping congeners were included in the data and the tree, in which case one species with data was mapped to one species on the tree (i.e. multiple congeners were not mapped without intrageneric sampling). The richness data were analysed with ln(richness) a function of migratory type (coded either as non-migratory versus migratory or as a multi-category predictor, with non-migratory defined as the intercept), body size (ln [length]) and ln(number of host specimens) as fixed effects, and phylogeny as a random effect, using a Gaussian link function. Prevalence data were analysed as a categorical response with migratory type (both versions as described above), body size and parasite

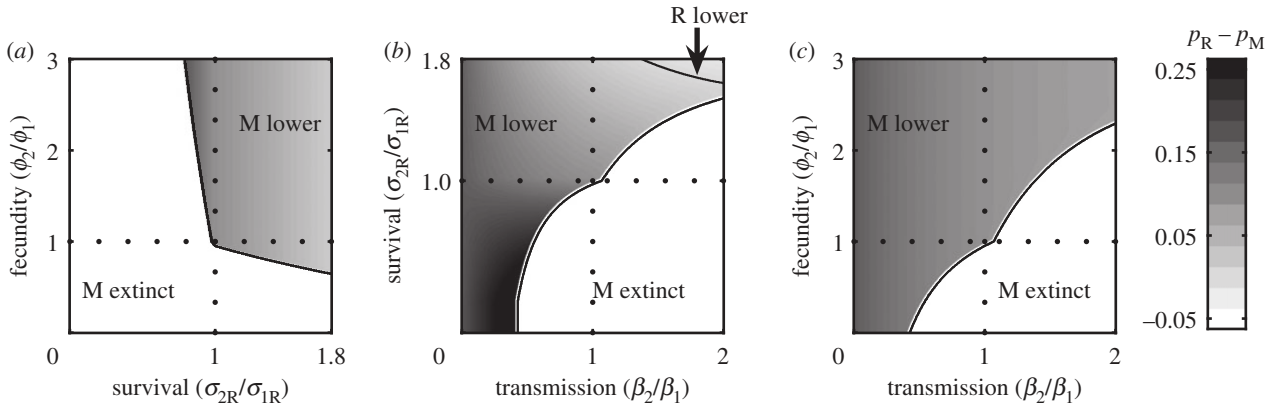

Figure 2. Difference in infection prevalence of residents (p_R) and migrants (p_M). Shown are cases where migrants have lower infection prevalence, residents have lower infection prevalence, and where migrants go extinct. Parameter values: $\beta_1 = 1$, $\beta_2 = 1$ (*a*) or $0 \leq \beta_2 \leq 2$ (*b*,*c*); $\varphi_1 = 1$, $\varphi_2 = 1$ (*b*) or $0 \leq \varphi_2 \leq 3$ (*a*,*c*); $\sigma_{1R} = 0.5$, $\sigma_{2R} = 0.5$ (*c*) or $0 \leq \sigma_{2R} \leq \sigma_{SR}$ (*a*,*b*), all other values are given in table S1 in [38]. The survival and fecundity of an individual with both parasites are the minimum survival and fecundity of having either one of the parasites.

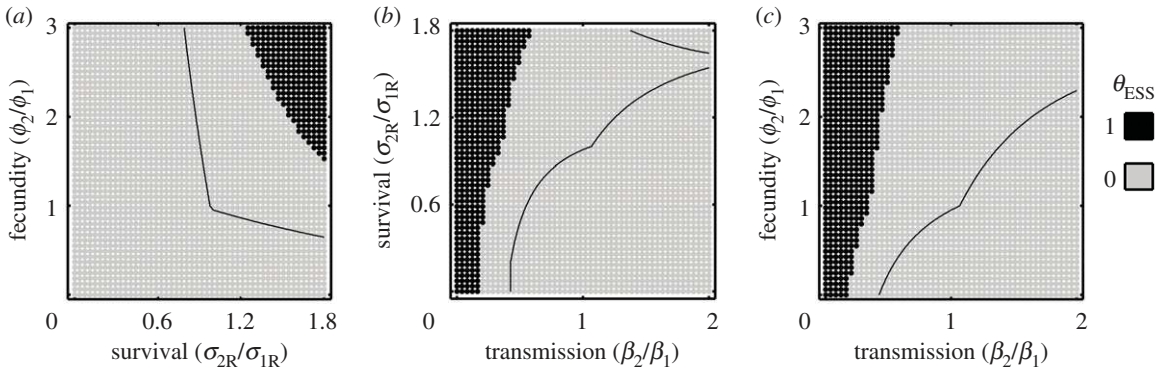

Figure 3. Evolutionarily stable probability of migration as a result of trade-offs between fecundity, survival and transmission rates. Parameter values are the same as figure 2 and lines from figure 2 are shown for comparison. Black points indicate a strategy of always migrating ($\theta_{ESS} = 1$), grey points indicate never migrating ($\theta_{ESS} = 0$) and intermediate values of θ_{ESS} were never observed.

clade (phylum + class) as fixed predictors, and phylogeny as a random effect, using a threshold model. Finally, intensity data were analysed as ln(number of parasites) with migratory type (both versions as described above), body size and parasite clade as fixed effects, and phylogeny as a random effect, using a Gaussian link function. All MCMC analyses were run for at least 1×10^5 post-burn-in generations, sampling every tenth generation, assessing burn-in by short initial runs.

3. Results

(a) Model results

The relative degree of infection of migrants and residents depended on the infection metric considered. For all parameter combinations we explored, migrant populations had higher parasite richness (an intuitive finding, given our model assumptions). However, migrants often had lower infection prevalence than residents (figure 2), although in other cases, the migratory population went extinct (figure 2 white regions). With equal transmission rates for both parasites, migrants had lower infection prevalence only when parasite 1 was sufficiently more costly than parasite 2 in terms of both survival and fecundity (figure 2*a*). By contrast, with different rates of transmission for the two parasites, migrants had lower infection prevalence than residents as long as parasite 2 had either a lower transmission rate than parasite 1 or a lower cost (in terms of survival or fecundity; figure 2*b*,*c*). Counterintuitively, migrants could have lower infection prevalence than residents even when parasite 2 was transmitted at a faster rate than parasite 1, as long as the cost of parasite 1 (in terms of either survival or fecundity) was sufficiently high.

We can interpret these results as generating predictions for whether migration or residency should evolve based on minimizing infection richness or prevalence. Thus, we would predict that if parasite richness is the primary mechanism by which infection influences migration we should never see migration evolve in this model, but if parasite prevalence is the primary mechanism, migration should evolve aligning with the regions of figure 2.

The results of our evolutionary analysis fell somewhere between these two extremes (figure 3), suggesting that both richness and prevalence shape the evolution of migration. Full migration ($\theta_{ESS} = 1$) only evolved when migrants had lower infection prevalence than residents. However, having lower infection prevalence was not sufficient for migration; there were some cases where migrants had a lower prevalence than residents, but full residency ($\theta_{ESS} = 0$) evolved. Particularly notable is that the evolution of migration was quite sensitive to transmission rate; migration never evolved when parasite 2 was transmitted at a faster rate than parasite 1, and migration evolved as long as parasite 2 was transmitted sufficiently slowly, regardless of the relative costs of

Table 2. Results of generalized linear mixed model analysis of fish parasite richness as a function of sampling effort, body size and migratory status, accounting for host phylogeny (as implemented in *MCMCglmm*; [49]). Two models were fitted, one treating host migration as present or absent (migration) and one breaking migratory types down into categories (migratory type). Shown are the deviance information criterion (DIC) [52], location effects (β_i) for the intercept (richness of non-migratory species) and migratory classes and the estimated probabilities of location effect posteriors including zero ($p[\beta = 0]$).

model	DIC	parameters	β_i	$p(\beta = 0)$
migration	1786.4	intercept (non-migratory richness)	−0.44	0.622
		migratory	0.45	0.010
		ln(length)	0.36	<0.001
		ln(# of individuals)	0.17	<0.001
migratory type	1789.4	intercept (non-migratory richness)	−0.42	0.642
		amphidromous	0.15	0.510
		anadromous	0.61	0.048
		catadromous	1.236	0.004
		oceanodromous	0.51	0.012
		potamodromous	0.47	0.067
		body size (length)	0.34	<0.001
		ln(# of individuals)	0.17	<0.001

the two parasites (figure 3*b*,*c*, contrasting with the predictions from figure 2*b*,*c*).

Finally, the cost structure for individuals infected with both parasites influenced when migration evolved. Migration was favoured under the broadest set of conditions when parasite costs were subadditive, that is, when the cost of both parasites was less than the sum of each parasite cost separately (electronic supplementary material, figure S1). When parasite costs were additive (the cost of both was equal to the sum of each separately), migration was favoured under a smaller set of conditions. Finally, when parasite costs were superadditive (the cost of both was greater than the cost of each separately), migration was very rarely favoured (electronic supplementary material, figure S1).

(b) Empirical results

We found a strong relationship between the number of parasites known to infect a given host and the number of publications reporting parasites for that host, with each publication adding approximately 3.5 parasite species for a given host (results not shown). However, as previously noted [21], the number of publications is not necessarily the best measure of sampling effort. Of 28 518 parasite–host combinations reported, fully 31% were unique (i.e. only one paper reported the combination), and individual parasite species were only reported an average of 1.5 times for a given host (range = 1.0–6.3 reports per species per host). This probably reflects a parasitological bias towards reporting novel parasite–host associations, meaning that publication number does not reflect sampling intensity. As described above, we instead used the number of museum specimens (either lots or individuals) of each host species as an estimate of parasite sampling effort. Multivariate analysis of parasite richness as a function of migration, number of host specimens (only results using individuals shown, but nearly identical results were obtained for lots) and body size strongly supports the significance of all three predictors (table 2), regardless of whether ectoparasite data were included (results not shown). Analyses using both migration (presence/absence) and migratory type (anadromous, etc.), show higher parasite richness in migratory than non-migratory species (table 2). Examining the results by migratory type demonstrates that this effect is positive for every migratory category, with the effect significantly positive in three out of five. The single largest effect size was for catadromous species, with an incremental addition to species richness more than twice that for the next highest category (anadromous). By contrast with the results obtained for parasite richness, we found no effect of migratory status of species on either parasite prevalence or intensity (table 3). Although we only report here the results of treating migration as a binary predictor, analyses using a multistate categorization yielded similarly negative results (not shown).

4. Discussion

Here we have shown, first, that migration can be driven by parasitism (although different infection metrics give rise to different predictions) and, second, that parasite richness (the number of different types of parasite), but not prevalence or intensity, is higher among migrant than non-migrant species of fish. To our knowledge, this is only the second study (see [26]) to examine all three metrics of infection in the context of migration using either theoretical or empirical approaches.

In our model, we find that migrants typically have greater parasite richness but lower infection prevalence than non-migrants (residents). Our finding of higher richness is, intuitively, due to our model assumptions—namely that migrants come into contact with two types of parasites while non-migrants only come into contact with a single one. Our finding of lower prevalence among migrants, although less intuitive, seems to be driven by the assumption that migration incurs a mortality cost. Simulating our modelling without any mortality (a biologically unreasonable assumption, but useful to make this point) results in migrants

Table 3. Results of generalized linear mixed model analysis of fish parasite prevalence and intensity as a function of migratory status, body size and parasite clade, accounting for host phylogeny (as implemented in *MCMCglmm* [49]). Shown are the deviance information criterion (DIC) [52], location effects (β_i) for the predictors and the estimated probabilities of location effect posteriors including zero ($p[\beta = 0]$). Parasite clade effects not relevant to hypotheses under test are not shown.

response	DIC	parameters	β_i	$p(\beta = 0)$
prevalence	15 072.2	intercept (non-migratory prevalence)	−2.06	0.164
		ln(length)	0.53	0.013
		migratory	−0.46	0.272
log intensity	575.0	intercept (non-migratory intensity)	0.61	0.596
		ln(length)	0.15	0.422
		migratory	−0.22	0.576

having a lower prevalence than residents for a narrower region of parameter space (electronic supplementary material, figure S2). Mortality during migration reduces survival of migrants (many of whom are infected), and thus leads to susceptible individuals making up a larger portion of the populations and lower population prevalence. This mechanism, described as migratory culling [53], has been explored in previous models and shown to reduce infection prevalence [37]. Our results also provide a helpful comparison with the recent paper by Teitelbaum *et al*. [30] which hypothesizes that environmental sampling should increase parasite richness of migrants while migratory culling should decrease both parasite richness and infection prevalence of migrants (compared to residents). Our model, which includes both mechanisms, suggests that migratory culling may act primarily on infection prevalence with less impact on parasite richness.

Empirically, this study adds to the growing body of evidence (e.g. table 1) that migratory species harbour a higher diversity of parasite species than non-migratory species. This was true for nearly every migratory category, but especially so for catadromous fishes, which move between fresh and saltwater environments. Anadromous fishes do the same but live as adults in saltwater as opposed to fresh, suggesting that freshwater environments may be more conducive to a broader array of parasite life histories. That this pattern of increased parasitism is consistent with the model explored here is reassuring, but unsurprising given that this model was constructed with such results in mind. We found no evidence that migratory fish species had either higher prevalence or higher intensity of infection, contrasting with results from other organisms (table 1). As discussed above, our model predicts a somewhat lower prevalence in migratory species across a broad array of conditions, consistent with results from monarch butterflies [31] but not supported in the comparative analyses reported here. However, we note that the modelled differences in prevalence between migrants and non-migrants were relatively small (on the order of 10–15%) and may be difficult to detect in cross-species comparative analyses. Although our model did not quantify intensity *per se*, empirical results from previous studies (both fishes and other organisms) suggest that migrants have lower intensity: this result was not corroborated here. It is important to note that our analyses of prevalence and intensity were by no means comprehensive: we systematically sampled the available literature to capture data from a reasonable sample of host types in each migratory class. Consequently, our sample sizes for these analyses were small (prevalence: 168 parasite species from 94 host species; intensity: 143 parasite species from 67 host species) compared with those for parasite richness (643 host species). Capture of additional prevalence and intensity data from the published literature may prove fruitful. In addition, we agree with [46] that better quantification of sampling effort in studies of parasite richness, prevalence and intensity are necessary, both in papers reporting such work and databases summarizing that work.

The finding that different infection metrics can provide very different understandings of the impact of infection on host migration is particularly important given that empirical studies linking migration and parasites often use only a single metric when quantifying infection (table 1). It seems reasonable to expect that a combination of parasite diversity (quantified by 'richness', or other variant), prevalence and intensity would drive selection on migration differently in different systems. For example, infection intensity might be more important for ectoparasites under some circumstances but not others; species that fly or swim experience more drag than those that walk [54], with drag amplified by the presence of ectoparasites [15]. Other parasites might have a threshold effect, where infection beyond a certain intensity is lethal [55]. Which metrics are important probably depends on the nature of the parasite and whether it causes chronic illness or is a kill-or-recover pathogen. With the former, as in helminth infections, intensity or richness may be crucial in determining host fitness, whereas in viral infections, viral load may be less important than infection presence in the first place [56]. Of course, it may also be the case that parasites have no selective effect on migration but are rather just a side effect of migration that evolved for other reasons [13].

As is often the case with ecological processes [57], the impacts of parasites on their hosts may operate differently at different scales. Although our focus here is on seasonal migration, parasite infection can influence host movement at other scales, including dispersal and daily movements. As is the case with migration, infection risk can be expected to either increase or decrease host movement at these scales [15]. Hosts may disperse to escape infested habitats [58] or stay to pay the cost of 'known' parasites rather than risk exposure to unknown ones [59]. Hosts may be manipulated by their parasites to either move more and increase transmission [60], or move less and increase predation and thus trophic transmission

[61]. Finally, host movement can be important for parasites as well, enabling the spread of parasites to new areas [62], driving the dynamics of disease outbreaks [63] and influencing the structure of parasite communities [64].

Our findings have implications for how we think about parasites and migration at a range of scales from the macroevolutionary to the ontogenetic. At the macroevolutionary scale, if parasitism influences the evolution of migration, and migratory behaviour influences the ability of lineages to colonize new regions, then parasites may have a significant role in shaping host biogeographical patterns and diversification rates. For instance, during the Great American Biotic Interchange [65], many more North American lineages of birds and mammals appear to have dispersed into and subsequently diversified in South America than the converse [66–70], a result potentially attributable to changes in life history and dispersive abilities associated with host–parasite coevolution [71,72]. Avoidance of parasite infection may also help explain the maintenance of long-distance migration in some lineages across massive changes in climate across millions of years of evolution [73].

At the population (ontogenetic) scale, parasites have been shown to influence life history in a variety of contexts, including fecundity, longevity and susceptibility to predation [74–76], each of which can in turn affect the likelihood of migration. For example, individuals that shed their parasites annually will have different effects on host–parasite interactions than individuals with persistent infections. By modelling the abundance of parasites within hosts, one could compare the effect of infection intensity with both parasite richness and prevalence. Other future work could include examining investment in tolerance versus resistance [77] and exploring the effect of host migratory strategy on parasite virulence (which probably depends on the amount of time hosts and parasites are in contact).

Data accessibility. All model code, data and analysis code are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.47t0b41 [38].

Authors' contributions. A.K.S. and M.Z. conceived of the study, J.S. did the literature search and data collection, A.K.S. and J.S. developed and analysed the model, J.S. and F.K.B. analysed data, and all authors contributed to writing and editing the final manuscript.

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References

1. Dingle H. 2014 *Migration: the biology of life on the move*, 2nd edn. Oxford, UK: Oxford University Press.
2. Russell AP, Bauer AM, Johnson MK, Elewa MAT. 2005 Migration in amphibians and reptiles: an overview of patterns and orientation mechanisms in relation to life history strategies. In *Migration of organisms* (ed. AMT Elewa), pp. 151–203. Berlin, Germany: Springer.
3. Wolcott TG, Wolcott DL. 1985 Factors influencing the limits of migratory movements in terrestrial crustaceans. *Contrib. Mar. Sci.* **68**, 257–273.
4. Common IFB. 1954 A study of the ecology of the adult bogong moth, *Agrotis infusa* (Boisd) (Lepidoptera: Noctuidae), with special reference to its behaviour during migration and aestivation. *Aust. J. Zool.* **2**, 223–263. (doi:10.1071/ZO9540223)
5. Lampert W. 1989 The adaptive significance of diel vertical migration of zooplankton. *Funct. Ecol.* **3**, 21–27. (doi:10.2307/2389671)
6. Gales NJ, Cheal AJ, Pobar G, Williamson P. 1992 Breeding biology and movements of Australian sea-lions, *Neophoca cinerea*, off the west coast of Western Australia. *Wildl. Res.* **19**, 405–415. (doi:10.1071/WR9920405)
7. Bonte D *et al.* 2012 Costs of dispersal. *Biol. Rev. Camb. Philos. Soc.* **87**, 290–312. (doi:10.1111/j.1469-185X.2011.00201.x)
8. Shaw AK. 2016 Drivers of animal migration and implications in changing environments. *Evol. Ecol.* **30**, 991–1007. (doi:10.1007/s10682-016-9860-5)
9. Heape W. 1931 *Emigration, migration and nomadism*. Cambridge, UK: Heffer and Sons.
10. Brodersen J, Nilsson PA, Hansson L-A, Skov C, Brönmark C. 2008 Condition-dependent individual decision-making determines cyprinid partial migration. *Ecology* **89**, 1195–1200. (doi:10.1890/07-1318.1)
11. Hebblewhite M, Merrill EH. 2009 Trade-offs between predation risk and forage differ between migrant strategies in a migratory ungulate. *Ecology* **90**, 3445–3454. (doi:10.1890/08-2090.1)
12. Stefanescu C, Askew RR, Corbera J, Shaw MR. 2012 Parasitism and migration in southern Palaearctic populations of the painted lady butterfly, *Vanessa cardui* (Lepidoptera: Nymphalidae). *Eur. J. Entomol.* **109**, 85–94. (doi:10.14411/eje.2012.011)
13. Poulin R, Closs GP, Lill AWT, Hicks AS, Herrmann KK, Kelly DW. 2012 Migration as an escape from parasitism in New Zealand galaxiid fishes. *Oecologia* **169**, 955–963. (doi:10.1007/s00442-012-2251-x)
14. Wood CL, Johnson PT. 2015 A world without parasites: exploring the hidden ecology of infection. *Front. Ecol. Environ.* **13**, 425–434. (doi:10.1890/140368)
15. Binning SA, Shaw AK, Roche DG. 2017 Parasites and host performance: Incorporating infection into our understanding of animal movement. *Integr. Comp. Biol.* **57**, 267–280. (doi:10.1093/icb/icx024)
16. Pedersen AB, Jones KE, Nunn CL, Altizer S. 2007 Infectious diseases and extinction risk in wild mammals. *Conserv. Biol.* **21**, 1269–1279. (doi:10.1111/j.1523-1739.2007.00776.x)
17. Folstad I, Nilssen AC, Halvorsen O, Andersen J. 1991 Parasite avoidance: the cause of post-calving migrations in *Rangifer*? *Can. J. Zool.* **69**, 2423–2429. (doi:10.1139/z91-340)
18. Loehle C. 1995 Social barriers to pathogen transmission in wild animal populations. *Ecology* **76**, 326–335. (doi:10.2307/1941192)
19. Shaw AK, Binning SA. 2016 Migratory recovery from infection as a selective pressure for the evolution of migration. *Am. Nat.* **187**, 491–501. (doi:10.1086/685386)
20. Daversa DR, Fenton A, Dell AI, Garner TWJ, Manica A. 2017 Infections on the move: how transient phases of host movement influence disease spread. *Proc. R. Soc. B* **284**, 20171807. (doi:10.1098/rspb.2017.1807)
21. Figuerola J, Green AJ. 2000 Haematozoan parasites and migratory behaviour in waterfowl. *Evol. Ecol.* **14**, 143–153. (doi:10.1023/A:1011009419264)
22. Morgan ER, Medley GF, Torgerson PR, Shaikenov BS, Milner-Gulland EJ. 2007 Parasite transmission in a migratory multiple host system. *Ecol. Model.* **200**, 511–520. (doi:10.1016/j.ecolmodel.2006.09.002)
23. MacColl ADC, Chapman SM. 2010 Parasites can cause selection against migrants following dispersal between environments. *Funct. Ecol.* **24**, 847–856. (doi:10.1111/j.1365-2435.2010.01691.x)
24. Bush AO, Lafferty KD, Lotz JM, Shostak AW. 1997 Parasitology meets ecology on its own terms: Margolis *et al*. revisited. *J. Parasitol.* **83**, 575. (doi:10.2307/3284227)
25. Ricklefs RE *et al*. 2017 Avian migration and the distribution of malaria parasites in New World passerine birds. *J. Biogeogr.* **44**, 1113–1123. (doi:10.1111/jbi.12928)

26. Slowinski SP, Fudickar AM, Hughes AM, Mettler RD, Gorbatenko OV, Spellman GM, Ketterson ED, Atwell JW. 2018 Sedentary songbirds maintain higher prevalence of haemosporidian parasite infections than migratory conspecifics during seasonal sympatry. *PLoS ONE* **13**, e0201563. (doi:10.1371/journal.pone.0201563)
27. Jenkins T, Thomas GH, Hellgren O, Owens IP. 2012 Migratory behavior of birds affects their coevolutionary relationship with blood parasites. *Evolution* **66**, 740–751. (doi:10.5061/dryad.qr8v5f4v)
28. Koprivnikar J, Leung TLF. 2015 Flying with diverse passengers: greater richness of parasitic nematodes in migratory birds. *Oikos* **124**, 399–405. (doi:10.1111/oik.01799)
29. Leung TLF, Koprivnikar J. 2016 Nematode parasite diversity in birds: the role of host ecology, life history and migration. *J. Anim. Ecol.* **85**, 1471–1480. (doi:10.1111/1365-2656.12581)
30. Teitelbaum CS, Huang S, Hall RJ, Altizer S. 2018 Migratory behaviour predicts greater parasite diversity in ungulates. *Proc. R. Soc. B* **285**, 20180089. (doi:10.1098/rspb.2018.0089)
31. Satterfield DA, Maerz JC, Altizer S. 2015 Loss of migratory behaviour increases infection risk for a butterfly host. *Proc. R. Soc. B* **282**, 20141734 (doi:10.1098/rspb.2014.1734)
32. Altizer SM, Oberhauser KS, Brower LP. 2000 Associations between host migration and the prevalence of a protozoan parasite in natural populations of adult monarch butterflies. *Ecol. Entomol.* **25**, 125–139. (doi:10.1046/j.1365-2311.2000.00246.x)
33. Carbó-Ramírez P, Zuria I. 2015 Immune condition and blood parasites in three sparrow species with different migratory status in central Mexico. *Avian Biol. Res.* **8**, 167–174. (doi:10.3184/175815515X14371521830098)
34. Bandelj P, Blagus R, Trilar T, Vengust M, Rataj AV. 2015 Influence of phylogeny, migration and type of diet on the presence of intestinal parasites in the faeces of European passerine birds (*Passeriformes*). *Wildl. Biol.* **21**, 227–233. (doi:10.2981/wlb.00044)
35. Mysterud A, Qviller L, Meisingset EL, Viljugrein H. 2016 Parasite load and seasonal migration in red deer. *Oecologia* **180**, 401–407. (doi:10.1007/s00442-015-3465-5)
36. Sikkel PC, Welicky RL, Artim JM, McCammon AM, Sellers JC, Coile AM, Jenkins WG. 2016 Nocturnal migration reduces exposure to micropredation in a coral reef fish. *Bull. Mar. Sci.* **93**, 475–488. (doi:10.5343/bms.2016.1021)
37. Johns S, Shaw AK. 2016 Theoretical insight into three disease-related benefits of migration. *Popul. Ecol.* **58**, 213–221. (doi:10.1007/s10144-015-0518-x)
38. Shaw AK, Sherman J, Barker FK, Zuk M. 2018 Data from: Metrics matter: the effect of parasite richness, intensity and prevalence on the evolution of host migration. Dryad Digital Repository. (doi:10.5061/dryad.47t0b41)
39. Geritz SAH, Kisdi E, Meszena G, Metz JA. 1998 Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.* **12**, 35–57. (doi:10.1023/A:1006554906681)
40. Maynard Smith J, Price G. 1973 The logic of animal conflict. *Nature* **246**, 15–18. (doi:10.1038/246015a0)
41. Strona G, Lafferty KD. 2012 FishPEST: an innovative software suite for fish parasitologists. *Trends Parasitol.* **28**, 123. (doi:10.1016/j.pt.2012.02.001)
42. Froese R, Pauly D. 2016 FishBase. See www.fishbase.org.
43. Gibson DI, Bray RA, Harris EA. 2005 Host–parasite database of the Natural History Museum. See http://www.nhm.ac.uk/research-curation/scientific-resources/taxonomy-systematics/host-parasites/index.html
44. Hoffman G. 1999 *Parasites of North American freshwater fishes*. Ithaca, NY: Comstock Publishing Associates.
45. Hipp R. 2015 SQLite v3.9.2. See https://www.sqlite.org.
46. Walther BA, Cotgreave P, Price RD, Gregory RD, Clayton DH. 1995 Sampling effort and parasite species richness. *Parasitol. Today* **11**, 306–310. (doi:10.1016/0169-4758(95)80047-6)
47. Bloom D. 2016 VertNet_Fishes_Sept2016. See http://datacommons.cyverse.org/browse/iplant/home/shared/commons_repo/curated/Vertnet_Fishes_Sep2016.
48. R Development Core Team. 2012 *R: a language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.
49. Hadfield JD. 2010 MCMC methods for multi-response generalized linear mixed models: the MCMCglmm R package. *J. Stat. Softw.* **33**, 1–22. (doi:10.18637/jss.v033.i02)
50. Hadfield JD, Nakagawa S. 2010 General quantitative genetic methods for comparative biology: phylogenies, taxonomies and multi-trait models for continuous and categorical characters. *J. Evol. Biol.* **23**, 494–508. (doi:10.1111/j.1420-9101.2009.01915.x)
51. Rabosky DL, Santini F, Eastman J, Smith SA, Sidlauskas B, Chang J, Alfaro ME. 2013 Rates of speciation and morphological evolution are correlated across the largest vertebrate radiation. *Nat. Commun.* **4**, 1958. (doi:10.1038/ncomms2958)
52. Spiegelhalter DJ, Best NG, Carlin BP, Van Der Linde A. 2002 Bayesian measures of model complexity and fit. *J. R. Stat. Soc. Ser. B Stat. Methodol.* **64**, 583–639.
53. Bradley CA, Altizer S. 2005 Parasites hinder monarch butterfly flight: implications for disease spread in migratory hosts. *Ecol. Lett.* **8**, 290–300. (doi:10.1111/j.1461-0248.2005.00722.x)
54. Schmidt-Nielsen K. 1972 Locomotion: energy cost of swimming, flying, and running. *Science* **177**, 222–228. (doi:10.1126/science.177.4045.222)
55. Jones S, Hargreaves N. 2009 Infection threshold to estimate *Lepeophtheirus salmonis*-associated mortality among juvenile pink salmon. *Dis. Aquat. Organ.* **84**, 131–137. (doi:10.3354/dao02043)
56. Lello J, Boag B, Hudson P. 2005 The effect of single and concomitant pathogen infections on condition and fecundity of the wild rabbit. *Int. J. Parasitol.* **35**, 1509–1515. (doi:10.1016/j.ijpara.2005.06.002)
57. Levin SA. 1992 The problem of pattern and scale in ecology: the Robert H. MacArthur award lecture. *Ecology* **73**, 1943–1967. (doi:10.2307/1941447)
58. Brown CR, Brown MB. 1992 Ectoparasitism as a cause of natal dispersal in cliff swallows. *Ecology* **73**, 1718–1723. (doi:10.2307/1940023)
59. Heeb P, Werner I, Mateman A, Kolliker M, Brinkhof MWG, Lessells C, Richner H. 1999 Ectoparasite infestation and sex-biased local recruitment of hosts. *Nature* **400**, 63–65. (doi:10.1038/21881)
60. Stafford CA, Walker GP, Ullman DE. 2011 Infection with a plant virus modifies vector feeding behavior. *Proc. Natl Acad. Sci. USA* **108**, 9350–9355. (doi:10.1073/pnas.1100773108)
61. Lafferty KD, Shaw JC. 2013 Comparing mechanisms of host manipulation across host and parasite taxa. *J. Exp. Biol.* **216**, 56–66.
62. Fèvre EM, Bronsvoort BM de C, Hamilton KA, Cleaveland S. 2006 Animal movements and the spread of infectious diseases. *Trends Microbiol.* **14**, 125–131. (doi:10.1016/j.tim.2006.01.004)
63. Ferrari MJ, Grais RF, Bharti N, Conlan AJK, Bjørnstad ON, Wolfson LJ, Guerin PJ, Djibo A, Grenfell BT. 2008 The dynamics of measles in sub-Saharan Africa. *Nature* **451**, 679–684. (doi:10.1038/nature06509)
64. Blouin MS, Yowell CA, Courtney CH, Dame JB. 1995 Host movement and the genetic structure of populations of parasitic nematodes. *Genetics* **141**, 1007–1014.
65. Stehli FG, Webb SD. 1985 *The Great American Biotic Interchange*. New York, NY: Plenum Press.
66. Marshall LG, Webb SD, Sepkoski Jr JJ, Raup DM. 1982 Mammalian evolution and the Great American Interchange. *Science* **215**, 1351–1357. (doi:10.1126/science.215.4538.1351)
67. Webb SD. 2006 The Great American Biotic Interchange: patterns and processes. *Ann. Mo. Bot. Gard.* **93**, 245–257. (doi:10.3417/0026-6493(2006)93[245:TGABIP]2.0.CO;2)
68. Weir JT, Bermingham E, Schluter D. 2009 The Great American Biotic Interchange in birds. *Proc. Natl Acad. Sci. USA* **106**, 21737–21742. (doi:10.1073/pnas.0903811106)
69. Smith BT, Klicka J. 2010 The profound influence of the Late Pliocene Panamanian uplift on the exchange, diversification, and distribution of New World birds. *Ecography* **33**, 333–342. (doi:10.1111/j.1600-0587.2009.06335.x)
70. Bacon CD, Silvestro D, Jaramillo C, Smith BT, Chakrabarty P, Antonelli A. 2015 Biological evidence supports an early and complex emergence of the Isthmus of Panama. *Proc. Natl Acad. Sci. USA* **112**, 6110–6115. (doi:10.1073/pnas.1423853112)
71. Ricklefs RE. 1992 Embryonic development period and the prevalence of avian blood parasites. *Proc. Natl Acad. Sci. USA* **89**, 4722–4725. (doi:10.1073/pnas.89.10.4722)

72. Ricklefs RE. 2002 Splendid isolation: historical ecology of the South American passerine fauna. *J. Avian Biol.* **33**, 207–211. (doi:10.1034/j.1600-048X.2002.330301.x)
73. Winger BM, Barker FK, Ree RH. 2014 Temperate origins of long-distance seasonal migration in New World songbirds. *Proc. Natl Acad. Sci. USA* **111**, 12115–12120. (doi:10.1073/pnas.1405000111)
74. Zuk M, Stoehr AM. 2002 Immune defense and host life history. *Am. Nat.* **160**, S9–S22. (doi:10.1086/342131)
75. Lee KA, Wikelski M, Robinson WD, Robinson TR, Klasing KC. 2008 Constitutive immune defences correlate with life-history variables in tropical birds. *J. Anim. Ecol.* **77**, 356–363. (doi:10.1111/j.1365-2656.2007.01347.x)
76. Johnson PTJ, Rohr JR, Hoverman JT, Kellermanns E, Bowerman J, Lunde KB. 2012 Living fast and dying of infection: host life history drives interspecific variation in infection and disease risk. *Ecol. Lett.* **15**, 235–242. (doi:10.1111/j.1461-0248.2011.01730.x)
77. Boulinier T, McCoy K, Sorci G. 2001 Dispersal and parasitism. In *Dispersal* (eds J Clobert, E Danchin, AA Dhondt, JD Nichols), pp. 169–179. Oxford, UK: Oxford University Press.