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RESEARCH ARTICLE

Pathogen evolution following spillover from a resident to a migrant host population depends on interactions between host pace of life and tolerance to infection

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Abstract

1. Changes to migration routes and phenology create novel contact patterns among hosts and pathogens. These novel contact patterns can lead to pathogens spilling over between resident and migrant populations. Predicting the consequences of such pathogen spillover events requires understanding how pathogen evolution depends on host movement behaviour. Following spillover, pathogens may evolve changes in their transmission rate and virulence phenotypes because different strategies are favoured by resident and migrant host populations. There is conflict in current theoretical predictions about what those differences might be. Some theory predicts lower pathogen virulence and transmission rates in migrant populations because migrants have lower tolerance to infection. Other theoretical work predicts higher pathogen virulence and transmission rates in migrants because migrants have more contacts with susceptible hosts.
2. We aim to understand how differences in tolerance to infection and host pace of life act together to determine the direction of pathogen evolution following pathogen spillover from a resident to a migrant population.
3. We constructed a spatially implicit model in which we investigate how pathogen strategy changes following the addition of a migrant population. We investigate how differences in tolerance to infection and pace of life between residents and migrants determine the effect of spillover on pathogen evolution and host population size.
4. When the paces of life of the migrant and resident hosts are equal, larger costs of infection in the migrants lead to lower pathogen transmission rate and virulence following spillover. When the tolerance to infection in migrant and resident populations is equal, faster migrant paces of life lead to increased transmission rate and virulence following spillover. However, the opposite can also occur: when the migrant population has lower tolerance to infection, faster migrant paces of life can lead to decreases in transmission rate and virulence.

5. Predicting the outcomes of pathogen spillover requires accounting for both differences in tolerance to infection and pace of life between populations. It is also important to consider how movement patterns of populations affect host contact opportunities for pathogens. These results have implications for wildlife conservation, agriculture and human health.

KEYWORDS
disease ecology, emerging infectious disease, evolutionarily stable strategies, migratory culling, movement ecology, pace of life, tolerance, virulence

1 | INTRODUCTION

Changes to animals' patterns of habitat use and movement induced by climate and land use change create novel contact patterns among hosts and pathogens (Altizer et al., 2013). Such novel contact patterns can lead to pathogen spillover events, with implications for the health of wildlife, livestock and humans (Altizer et al., 2011). Changes to patterns of seasonal migration, a type of movement in which animals make an annual round trip between habitats (Dingle & Drake, 2007), have affected disease dynamics by altering contact patterns between resident and migrant populations (e.g. in monarchs, Satterfield et al., 2018; elk, Rayl et al., 2021; salmon, Ashander et al., 2012). Predicting the effect of pathogen spillover events between resident and migrant hosts requires an understanding of how these spillover events affect the evolution of pathogen transmission and virulence.

Before considering virulence in the context of migration and residency, we consider the selective pressures that act on pathogens generally. It is generally thought that pathogens experience a tradeoff between transmission rate (β) and virulence (μ) (Anderson & May, 1982; Cressler et al., 2016; Lipsitch & Moxon, 1997). These parameters are used in SIS models which are often used to study directly transmitted infectious diseases for which infection does not confer immunity (Hethcote, 2000) and can be used to describe environmentally transmitted diseases under some conditions (Benson et al., 2021). We define virulence as pathogen-induced host mortality and note that although virulence is often taken to be a pathogen property, it is an emergent property of a host-pathogen-environment system (Turner et al., 2021). We refer to the component of virulence attributed to the pathogen as the pathogen's virulence phenotype (Read, 1994). High transmission rates can be achieved by reproducing rapidly within a host but this may come with high host mortality costs (Acevedo et al., 2019). Long-term transmission opportunities from a host can be maintained by reproducing slower at a lower cost to the host, but this may come with lower transmission rates (Acevedo et al., 2019). The total number of new infections from a single host (R_0) depends on infection duration and the per capita rate at which infected individuals produce new infections (βS; the transmission rate multiplied by the number of susceptible individuals, which we call the 'infection rate') (Nelson & May, 2017). The tradeoff between transmission rate and virulence means that infection duration and infection rate are negatively correlated.

Properties of host populations affect which pathogen strategies lead to the highest pathogen fitness (Ewald, 1983; Restif et al., 2001). Tolerance to infection is one host property that determines the host response to a given pathogen burden (McCarville & Ayres, 2018). If a host has a lower tolerance to infection (a higher mortality rate for a given pathogen burden), this reduces the infection duration associated with a given transmission rate, which may select for a lower virulence and transmission rate pathogen phenotype (Altizer, 2001; Altizer et al., 2011, 2018). Host and population properties that increase the number of opportunities for contact with susceptible individuals may select for higher virulence pathogen phenotypes by increasing the infection rate associated with a given virulence phenotype (e.g. high levels of salmon density in aquaculture settings select for more virulent salmon lice phenotypes Ugelvik et al., 2017). Migrant and resident populations may differ in properties that select on pathogen virulence phenotypes.

Migrants and residents may differ in their tolerance to infection. The energetic costs of migration may decrease tolerance to infection, leading to culling of hosts infected with highly virulent pathogens (Altizer et al., 2011; Table 2). Thus, for a given pathogen phenotype, virulence may be higher in migrants, which may select for lower virulence and transmission rate pathogen phenotypes. It may also be the case that migration selects for more infection-tolerant hosts, which could select for more virulent pathogen phenotypes (Altizer et al., 2011; Table 2). Therefore, differences in tolerance between migrants and residents may lead to higher or lower virulence pathogen phenotypes evolving in migrant host populations.

Migrants and residents may also differ in their rates of contact with susceptible hosts and pathogen-independent mortality rates. Much of the empirical work on pathogen virulence in resident and migrant hosts (Table 2) has shown that pathogens infecting migrants have less virulent phenotypes. In many of these examples, residents have more contact opportunities with susceptible hosts since they are in agricultural settings with high host population densities (Krauss et al., 2010; Morgan et al., 2007). More frequent contact opportunities with susceptible hosts likely select for more virulent phenotypes. It may not be generally true that migrants have fewer contact opportunities than residents. Migrants often aggregate at high densities (Rubenstein & Hack, 2013), which could

lead to high contract rates with susceptible individuals (Krauss et al., 2010). Migrants may also have faster paces of life (higher fecundity and mortality rates) than residents (Soriano-Redondo et al., 2020). Faster pace of life may select for more virulent pathogen phenotypes because higher fecundity rates (Ewald, 1983) and higher pathogen-independent mortality rates (e.g. due to faster pace of life) (Restif et al., 2001) can both select for more virulent pathogen phenotypes. Higher host fecundity rates select for more virulent pathogen phenotypes by increasing the rate at which susceptible hosts are added to the population, thus increasing the rate of contact with susceptible hosts (Ewald, 1983). Higher mortality from factors other than infection by the focal pathogen reduces infection duration, selecting for higher transmission rate pathogen phenotypes (Restif et al., 2001).

We investigate how pathogen virulence phenotype changes following spillover from residential hosts to migratory hosts using a proof-of-concept model (Servedio et al., 2014). We have compiled some examples of host-pathogen systems for which our model is relevant in Table 2, which include host populations of the same or different species. In particular, we consider how differences in tolerance to infection and pace of life between migrants and residents interact to determine whether spillover leads to an increase or decrease in pathogen virulence and transmission phenotype. Both pace of life and tolerance to infection can vary between species or between populations of the same species (de Roode et al., 2008; Mathot & Frankenhuis, 2018; Power & Mitchell, 2004). Past work on how the evolution of virulence depends on host migration has either considered differences in contact opportunities (Poulin & de Angeli Dutra, 2021) or differences in tolerance (Altizer, 2001; Osnas et al., 2015; Ugelvik et al., 2017) between migrants and residents. These mechanisms operate in opposite directions and we consider both. We explore a range of parameter values that allows us to discover the range of outcomes possible when these mechanisms operate together, improving our understanding of how spillover from a resident to a migrant population affects the evolution of pathogen transmission rate and virulence phenotypes.

2 | METHODS

The goal of our model is to investigate how host population properties related to migration strategy affect which pathogen strategies dominate at equilibrium. The foundation of our model is a spatially implicit SIS (susceptible-infected-susceptible) model in which there are multiple pathogen strategies that vary in transmission and virulence phenotypes and host populations vary in their pace of life and tolerance to infection (see Table 1 for model parameters and variables). Our choices to model pathogen dynamics as density-dependent and explore differences in tolerance between migratory and non-migratory hosts are related to relevant empirical case studies described in Table 2. We use a numerically simulated adaptive dynamics approach (Best et al., 2017; Shaw et al., 2019) in order to identify how the dominant pathogen strategy depends on host traits. We begin by asking how the pathogen strategy that dominates at equilibrium depends on the host

TABLE 1 Model symbols, definitions and values (where applicable).

Symbol	Definition [units]	Value(s)
α	Pathogen strategy [unitless]	Integers between 0 and 10
β_α	Transmission rate for pathogen strategy α [individual^{-1} time^{-1}]	$0.0005 \times \alpha$
γ	Recovery rate [time^{-1}]	0.1
m_X	Steepness of association between transmission rate and host mortality rate in population X (host tolerance to infection) [time$^{-1/2}$]	Varied (0–2)
f_X	Density-independent component of the fecundity rate for individuals in population X [time^{-1}]	Varied (0.1–2)
N_X	Number of individuals in population X [individuals]	Varies
N^*	Number of individuals when the population is at equilibrium without infection [individuals]	Varied (100–1000)
$\mu_{X,S}$	Mortality rate of susceptible individuals in population X [time^{-1}]	Varied based on f_X and N^* values
δ	Strength of density dependence [individuals^{-1}]	0.001
$\mu_{X,\alpha}$	Mortality rate for individuals in population X infected with pathogen strategy α [time^{-1}]	$\left(\sqrt{\mu_{X,S}}+m\left(\frac{\beta_\alpha}{0.005}\right)\right)^2$
p	Probability of mutation [unitless]	0.001
ε	Stability cutoff [individuals]	0.001
S_X	Number of susceptible individuals in population X [individuals]	Varies
$I_{X,\alpha}$	Number of individuals in population X infected with pathogen transmission strategy α [individuals]	Varies
$f(S_X, I_X)$	Density-dependent fecundity rate [individuals time^{-1}]	$f_X(1-\delta(S_X+I_X))$
$V(X)$	Mean pathogen strategy value in population X [unitless]	Evolves

TABLE 2 Examples of systems in which migratory and non-migratory hosts share a pathogen/parasite and transmission is density dependent.

Host species	Pathogen/parasite species	Transmission and recovery dynamics	Migration and resident dynamics	Virulence details-differences in pathogen phenotype (if known)	Virulence details-differences in host tolerance/resistance (if known)	Spillover details (if known)	References
House finches	Bacteria (*Mycoplasma gallisepticum*)	Direct transmission and short-term environmental transmission Transmission has been modelled using susceptible-infected-susceptible (SIS) and susceptible-infected-recovered models	In the eastern U.S. population, migrants and residents share breeding grounds, migrants travel south-west for the winter The western U.S. population is not migratory	Initial virulence of strains in the western population is lower than in the eastern population. Virulence in both populations increased following spillover. The virulence in the two populations is now similar	The costs of long-distance migration may decrease resistance Non-migrants may experience decreased tolerance in the winter due to cold	Initial spillover into the eastern population presumably from poultry Spillover from eastern into western population in the 2000s	Altizer et al. (2004), Hawley et al. (2013), Hurtado (2008)
Migratory waterfowl and poultry	Avian influenza viruses	Transmission details are uncertain. Has been modelled as SIS	Domestic poultry are sedentary. Poultry on flyways can overlap with migrating waterfowl	Lower virulence pathogen phenotypes in wild migratory birds than in poultry	Ducks have high levels of tolerance Vaccination can increase tolerance/resistance in poultry	Initial transmission to poultry seems to be from wild waterfowl. Spillback from poultry to wild migratory birds has occurred	Alexander (2007), Endo and Nishiura (2018), Smith et al. (2015)
Saiga and domestic sheep	Foot and mouth disease virus	Direct Transmission. Has been modelled using susceptible-latent-infected-recovered	Saiga migrate and come into contact with sedentary sheep during part of their migration	Unknown	Saiga have higher infection-induced mortality than domestic ruminants	Transmission from domestic ruminants to saiga is widely assumed. Anecdotal evidence of spillback from saiga to livestock	Morgan et al. (2006)
Atlantic salmon and pink salmon	Salmon lice	Direct transmission. Free-swimming stages are transmitted to fish without an intermediate host	Farmed salmon are sedentary; wild salmon migrate between rivers for breeding and ocean for maturation and pass by farms en route	Higher virulence pathogen phenotypes in farmed fish than in wild ones	Unknown	Both spillover (wild to farmed) and spillback (farmed to wild) occur	Ashander et al. (2012), Krkošek et al. (2005), Ugelvik et al. (2017)

TABLE 2 (Continued)

Host species	Pathogen/parasite species	Transmission and recovery dynamics	Migration and resident dynamics	Virulence details-differences in pathogen phenotype (if known)	Virulence details-differences in host tolerance/resistance (if known)	Spillover details (if known)	References
Monarch butterflies	Protozoan (*Ophryocystis elektroscirrha*)	Transmission is typically maternal but can be environmental Infected individuals cannot recover, so best modelled as SI	Eastern population migrates farther than the western population. Resident population in Florida. Some overlap between migratory and non-migratory individuals	Parasites from resident populations are more virulent than those from migratory ones	Migrant hosts more resistant to infection. Migration may lead to culling of hosts infected with more virulent pathogen phenotypes	Spillover from residents to migrants occurs when they share habitat	Altizer (2001), Altizer et al. (2011), Satterfield et al. (2018)
Zebra	Helminths	Environmental transmission	Separate populations of resident, migrant and sedentary zebra	Unknown	Possible that migrant and sedentary zebra populations have lower resistance than the resident population	Unknown	Maina et al. (2022)
Sparrows	Nematodes	Environmental transmission	Migrant and resident species that overlap during migration	Unknown	Residents had lower resistance than migrants	Unknown	Carbó-Ramírez and Zuria (2015)

pace of life and tolerance to infection in a single population in order to propose a source for the differences in pathogen strategy between migratory and non-migratory populations that are sometimes seen in empirical systems (Table 2). We then ask how pathogen strategy changes following spillover from a resident into a migrant population where the migrant and resident populations can differ in their pace of life, tolerance to infection, both or neither. We focus on how the predictions about pathogen strategy evolution following spillover differ from what could be predicted in the single population case.

2.1 | Pathogen strategy

Pathogen strategy α (where α is an integer between 0 and 10) gives a pathogen transmission and virulence phenotype. Each pathogen strategy is defined by a transmission rate (β_α) that is a linear function

$$\beta_\alpha = 0.0005 \times \alpha, \tag{1}$$

of α. This transmission rate is related to the virulence of the pathogen, taken to be the pathogen-induced rate of host mortality in some host population X. This is the difference between the mortality rates of hosts in population X infected with a pathogen of strategy α ($\mu_{X,\alpha}$) and susceptible individuals in population X ($\mu_{X,S}$). The mortality rate of infected hosts is taken to be a quadratic function of transmission rate (Alizon & van Baalen, 2005),

$$\mu_{X,\alpha} = \left(\sqrt{\mu_{X,S}} + m\left(\frac{\beta_\alpha}{\beta_{10}}\right)\right)^2, \tag{2}$$

where m ($0 \le m \le 2$) and the mortality rate of susceptible individuals ($\mu_{X,S}$) are host properties. The parameter m (hereafter referred to as the host's tolerance to infection) governs how steep the relationship between virulence and transmission rate is (Figure A1) (McCarville & Ayres, 2018). High values of m mean low tolerance to infection and low values of m mean high tolerance to infection.

2.2 | Single population model

2.2.1 | Infection

In an SIS model of infection dynamics in some population X ($X = R$ or M where R stands for resident and M stands for migrant), susceptible hosts become infected by pathogen strategy α at a rate proportional to the number of susceptible hosts in the population (S_X) and the number of hosts in the population infected with pathogen strategy α ($I_{X,\alpha}$), that is, transmission is direct and density-dependent as seen in many pathogens in Table 2. Infected hosts recover at a rate γ ($\gamma = 0.1$) and immediately become susceptible again. Hosts can become infected with any of the 11 pathogen strategies in our model. We ignore coinfection with multiple pathogen strategies for simplicity despite its potential importance (Rigaud et al., 2010).

2.2.2 | Fecundity

Per-capita host fecundity rate is independent of infection status and all individuals are born into the susceptible class. The density-independent component of the per-capita fecundity rate is given by f_X $(0 \le f_X \le 3)$ and the density-dependent per-capita fecundity rate in population X, $f(S_X, I_X)$, is given by

$$f(S_X, I_X) = f_X(1 - \delta(S_X + I_X)), \tag{3}$$

where δ ($\delta = 0.001$) is the strength of density dependence. Fecundity occurs continuously during the breeding season, which lasts for half of each simulation year.

2.2.3 | Mutation

The role of mutation in this model is to explore the strategy space and identify the strategies that dominate at equilibrium after competition with other strategies. As the pathogen is transmitted to a new host, it can mutate to each adjacent strategy with some small probability p ($p = 0.001$). That is, if a host is infected with strategy $\alpha = 4$, it will typically transmit the same strategy ($\alpha = 4$) with probability $1 - 2p$, but will sometimes transmit to a lower strategy ($\alpha = 3$) with probability p, or a higher strategy ($\alpha = 5$) with probability p. When $\alpha = 0$ or $\alpha = 10$, there is only one adjacent strategy. In these edge cases, the rate of mutation into the adjacent class remains p, but the transmission will result in infection with the original pathogen strategy with probability $1 - p$ instead of $1 - 2p$. We show the differential equations for all infection classes in the single population model, but for simplicity, omit the edge cases in presenting the full two population model.

2.2.4 | Model

Bringing together the infection dynamics with fecundity and pathogen strategy-specific mortality, the rate of change of the susceptible individuals in population X in the breeding season is given by

$$\frac{dS_X}{dt} = -\sum_{\alpha=0}^{10}\beta_\alpha S_X I_{X,\alpha} + \sum_{\alpha=0}^{10}\gamma I_{X,\alpha} + f_X\left(1 - \delta\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right)\right)\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right) - \mu_{X,S}S_X. \tag{4a}$$

The rate of change of the individuals infected by strategy α in population X is given by

$$\frac{dI_{X,\alpha}}{dt} = (1-2p)\beta_\alpha S_X I_{X,\alpha} + p\beta_{\alpha-1}S_X I_{X,\alpha-1} + p\beta_{\alpha+1}S_X I_{X,\alpha+1} - \gamma I_{X,\alpha} - \mu_{X,\alpha}I_{X,\alpha}, \tag{4b}$$

for $1 \le \alpha \le 9$ and by

$$\frac{dI_{X,0}}{dt} = (1-p)\beta_0 S_X I_{X,0} + p\beta_1 S_X I_{X,1} - \gamma I_{X,0} - \mu_{X,0}I_{X,0}, \tag{4c}$$

when $\alpha = 0$ and

$$\frac{dI_{X,10}}{dt} = (1-p)\beta_{10} S_X I_{X,10} + p\beta_9 S_X I_{X,9} - \gamma I_{X,10} - \mu_{X,10}I_{X,10}, \tag{4d}$$

when $\alpha = 10$. During the non-breeding season, the equations for the infected classes remain the same, but the equation for the susceptible class

$$\frac{dS_X}{dt} = -\sum_{\alpha=0}^{10}\beta_\alpha S_X I_{X,\alpha} + \sum_{\alpha=0}^{10}\gamma I_{X,\alpha} - \mu_{X,S}S_X, \tag{4e}$$

does not include the fecundity term.

2.3 | Two population model

In the two population model, a resident population R and a migrant population M share an environment and can infect each other during the breeding season. During the non-breeding season, since the two populations do not share an environment, they cannot infect each other. This modelling choice is related to the fact that migrant and resident populations that share a pathogen often overlap for only part of the year (Table 2). Individuals are born into the same population as their parents and remain in the same population following infection and recovery. During the breeding season, the rates of change for the susceptible resident ($X = R$) and migrant populations ($X = M$) are given by

$$\frac{dS_X}{dt} = -\sum_{\alpha=0}^{10}\beta_\alpha S_X(I_{R,\alpha} + I_{M,\alpha}) + \sum_{\alpha=0}^{10}\gamma I_{X,\alpha} + f_X\left(1 - \delta\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right)\right)\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right) - \mu_{X,S}S_X. \tag{5a}$$

The rate of change of the infected populations of the resident ($X = R$) and migrant ($X = M$) populations are given by

$$\begin{aligned}\frac{dI_{X,\alpha}}{dt} = {} & (1-2p)\beta_\alpha S_X(I_{R,\alpha} + I_{M,\alpha}) + p\beta_{\alpha-1}S_X(I_{R,\alpha-1} + I_{M,\alpha-1}) \\ & + p\beta_{\alpha+1}S_X(I_{R,\alpha+1} + I_{M,\alpha+1}) - \gamma I_{X,\alpha} - \mu_{X,\alpha}I_{X,\alpha},\end{aligned} \tag{5b}$$

during the breeding season. During the non-breeding season, the rates of change for the susceptible resident and migrant populations are given by Equation (4e) and the rate of change for the infected resident and migrant populations are given by Equations (4b–4d) where $X = R$ or M.

2.4 | Pace of life

To isolate the effect of host pace of life on pathogen evolution independent of the effects on population size, we find pairs of mortality and fecundity rates that yield the same equilibrium population sizes N^* ($100 \le N^* \le 1000$) in the absence of infection. To do this, we consider the host population dynamics in the absence of infection, given by

$$\frac{dN}{dt} = f(1 - \delta N)N - \mu N, \tag{6a}$$

in the breeding season and

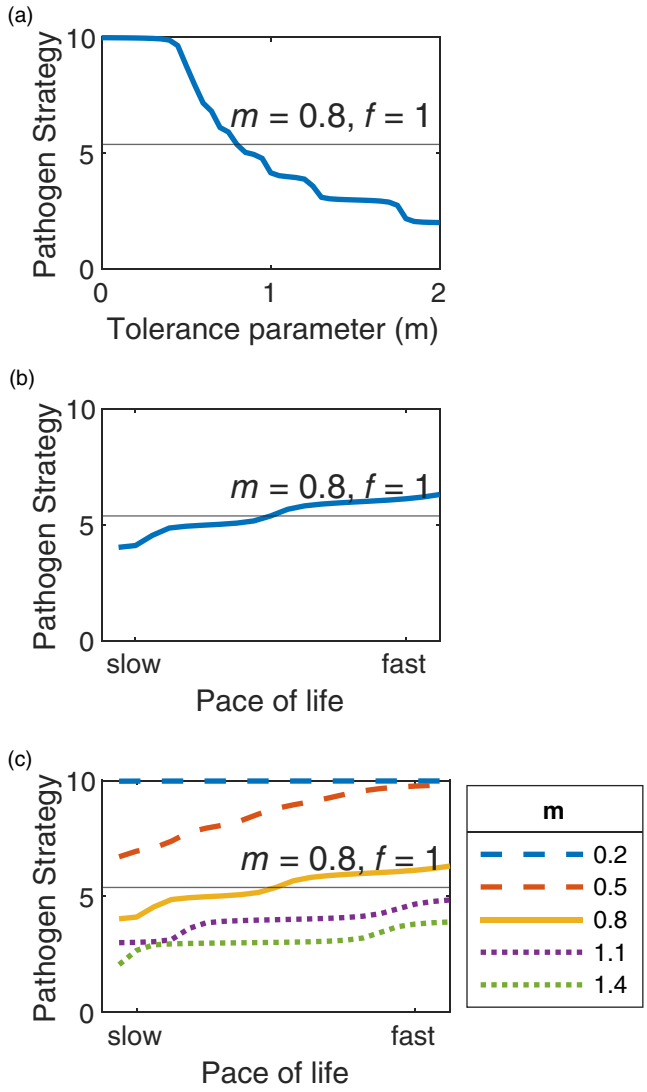

FIGURE 1 Pathogen strategies in one population. In a single population in which $N^*=800$ (a) the pathogen strategy that evolves decreases monotonically as tolerance to infection decreases (m increases) ($f=1$), (b) the pathogen strategy that evolves increases monotonically as fecundity (f) increases ($m=0.8$) and (c) the pathogen strategy that evolves increases monotonically as fecundity (f) increases and decreases monotonically as tolerance to infection decreases (m increases) when both tolerance to infection and pace of life vary. The pathogen strategy that evolves in the resident population in the two population simulation ($m=0.8$ and $f=1$) is shown as a horizontal line in all panels for easy comparison with the pathogen strategy that evolves in the migrant population in the two population simulations.

$$\frac{dN}{dt} = -\mu N, \tag{6b}$$

in the non-breeding season, where each season lasts for half of a year. Next, we look for the equilibrium population size (N^*), that is, the population size at the beginning of the breeding season such that the growth during the breeding season is equal to the

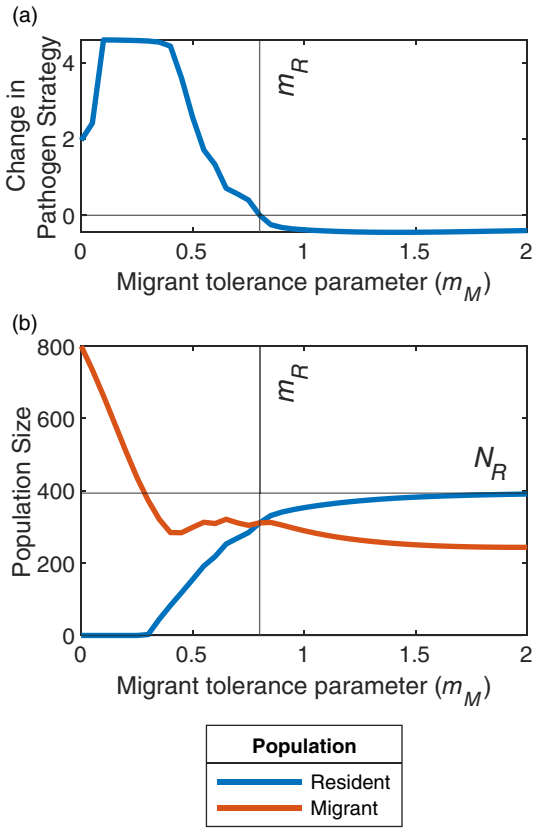

FIGURE 2 Migrant and resident populations differ in tolerance to infection. Following spillover from a resident into a migrant population in which $N^*=800$ for both populations, $f_R=f_M=1$ and $m_R=0.8$ (a) the change in pathogen strategy generally decreases monotonically as migrant tolerance to infection decreases (m_M increases). When $m_M=m_R$ (m_R is shown as a vertical line for reference), there is almost no change in pathogen strategy. (b) The size of the resident population increases as migrant tolerance to infection decreases (m_M increases) and size of the migrant population generally decreases as migrant tolerance to infection decreases (m_M increases). The number of resident individuals before spillover is shown as the horizontal line labelled N_R for reference.

population decline during the non-breeding season. Solving (6a and 6b), we find that

$$N^* = \frac{(f-\mu)\left(1-e^{\mu-\left(\frac{f}{2}\right)}\right)}{f\delta\left(e^{\frac{\mu}{2}}-e^{\mu-\left(\frac{f}{2}\right)}\right)}. \tag{6c}$$

To find different fecundity (f) and mortality (μ) rate pairs that yield a particular population size, we fix values of N^* and f and solve numerically for μ using vpasolve in MATLAB. We also run analyses in which we vary fecundity (f) and mortality (μ) rate separately in order to determine the effect of each rate separately and identify the effect of varying them together. While the pace of life syndrome idea can encompass many physiological and behavioural traits (Mathot & Frankenhuis, 2018), we restrict our attention here to the effects of fecundity and mortality rate.

2.5 | Simulations

We begin by initializing a one population model of the resident population with 100 individuals in the susceptible class, 10 individuals in the middle infection class ($\alpha = 5$) and no individuals in any other class. We run simulations until there is no class whose populations at the end of the non-breeding season change in number by more than ε between 2 years ($\varepsilon = 0.001$). When this stability condition has been met, we calculate which pathogen strategy dominates in the resident population alone. We also initialize a migrant population with N^* susceptible individuals and no infection. We use these stable populations as the starting condition for a two population model. When stability is reached in the two population simulation, we calculate what pathogen strategy dominates and how many individuals are in the migrant and resident populations. As a representative example, we explore simulations in which $N^* = 800$. When not taken as variables, we set the fecundity rate of the host population to $f_x = 1$ and the tolerance value to $m = 0.8$. These parameter choices were made because they led to intermediate levels of virulence at equilibrium in a single population, allowing virulence to increase or decrease following the addition of a second population. We also performed a sensitivity analysis to assess the generality of our findings (Supporting Information).

2.6 | Calculating pathogen strategy

We chose average pathogen strategy at equilibrium as our metric because when multiple pathogen strategies are present at equilibrium they are adjacent to one another. To calculate the average pathogen strategy in a single population X ($V(X)$), we take the sum of α multiplied by the proportion of infected individuals infected with pathogen strategy α overall values of α

$$V(X) = \frac{\sum_{\alpha=0}^{10} I_\alpha \alpha}{\sum_{\alpha=0}^{10} I_\alpha}. \quad (7)$$

To find the change in mean pathogen strategy resulting from adding in the migrant population, we calculate the difference in average pathogen strategy between the resident population at equilibrium before and after the addition of the migrant population.

3 | RESULTS

The addition of a migrant population can lead to an increase, decrease or no change in the pathogen strategy (i.e. the pathogen transmission rate and virulence phenotype) depending on the tolerance to infection in the migrant population and the pace of life of the migrants. We first explain how tolerance to infection and pace of life operate separately and together in a single host population to determine pathogen strategy. Next, we consider these mechanisms in the context of spillover in a two population model with residents and migrants and show how changes in relative population size can generate results that cannot be predicted solely from the single population model.

3.1 | Pathogen strategy evolution in a single host population

The pathogen strategy ($\alpha = 0, \ldots, 10$) that evolves decreases monotonically as the host's tolerance to infection decreases (as m increases) (i.e. the transmission and virulence phenotypes of pathogens are lower for lower host tolerance to infection) (Figure 1a). The pathogen strategy that evolves increases monotonically with the pace of life of the host population (i.e. the transmission and virulence phenotypes of pathogens are higher for faster host paces of life) (Figure 1b). When fecundity and mortality rates are considered separately, the pathogen strategy that evolves increases monotonically with the mortality rate (Figure A2) and generally does not change as a function of fecundity rate (Figure A4). When taken together, the effects of the host's tolerance to infection and pace of life are additive (Figure 1c).

3.2 | Change in pathogen strategy following spillover from a resident into a migrant population

We consider what happens when migrants and residents (1) differ only in their tolerance to infection, (2) differ only in their paces of life and (3) differ in both tolerance to infection and pace of life.

We begin by considering the case where migrants differ from residents in their tolerance to infection but have the same pace of life ($f_M = 1$ and m_M varies) (Figure 2). Following spillover into a migrant population, one might expect that the pathogen strategy would shift toward whatever strategy is favoured in that migrant population. Indeed, the qualitative pattern of the change in virulence phenotype follows the pattern of the single population simulations (Figure 1a). There is a monotonic decrease in pathogen strategy as migrant tolerance to infection decreases. The pathogen strategy increases when the migrant's tolerance to infection is higher than the residents and decreases when the migrant's tolerance to infection is lower (Figure 2a). Pathogen strategy changes more when the migrant has a higher tolerance to infection than residents than when the migrant has a lower tolerance to infection because of the effect of spillover on population sizes.

As the migrant's tolerance to infection decreases, the size of the migrant population decreases and the size of the resident population increases (Figure 2b). When the migrant's tolerance to infection is extremely high, the resident population is extirpated due to the increase in pathogen virulence and transmission phenotypes. In these cases, the pathogen strategy shifts all the way to what is optimal in the migrant population alone. When the migrant's tolerance to infection is lower than the residents, the migrants make up a smaller proportion of the total number of hosts than the residents and the pathogen strategy does not move as far toward what is optimal in the migrant population. When $m_M = m_R$, the migrants and residents

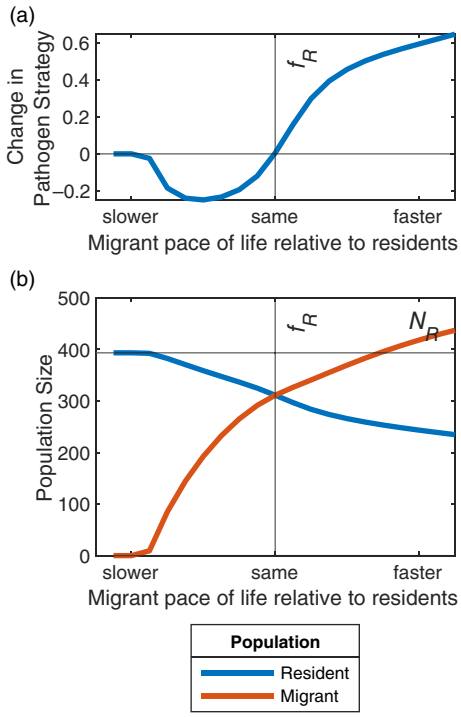

FIGURE 3 *Migrant and resident populations differ in pace of life.* Following spillover from a resident into a migrant population in which $N^*=800$ for both populations, $m_R = m_M = 0.8$ and $f_R=1$ (a) the change in pathogen strategy following spillover decreases and then increases as migrant fecundity rate (f_M) increases. When $f_M = f_R$ (f_R is shown as a vertical line for reference), there is almost no change in pathogen strategy. (b) The size of the resident population following spillover decreases as migrant fecundity rate (f_M) increases and size of the migrant population generally increases as migrant fecundity rate (f_M) increases. The number of resident individuals before spillover is shown as the horizontal line labelled N_R for reference.

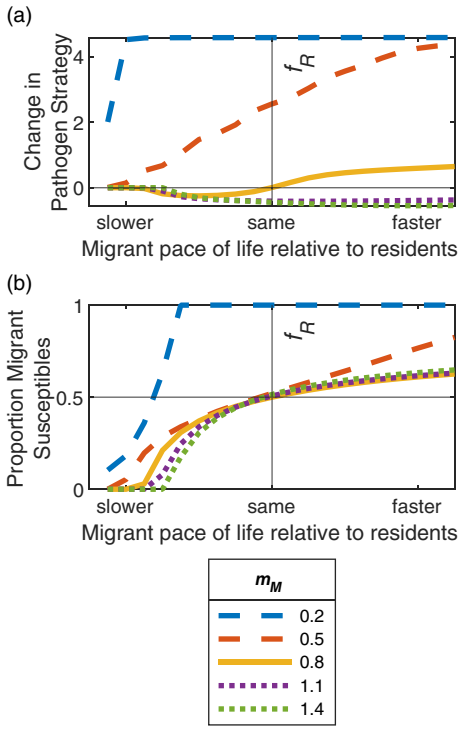

FIGURE 4 Migrant and resident populations differ in tolerance to infection and pace of life. Following spillover from a resident into a migrant population in which $N^*=800$ for both populations, $m_R=0.8$ and $f_R=1$ (with f_R shown as a vertical line for reference) (a) the change in pathogen strategy following spillover decreases as migrant tolerance to infection decreases (m_M increases) and can either increase or decrease as migrant fecundity rate (f_M) increases. (b) The proportion of susceptible individuals at equilibrium that are migrants typically increases as migrant fecundity rate (f_M) increases and as migrant tolerance to infection increases. A horizontal line is shown where migrants and residents make up an equal proportion of the susceptible population for reference.

have the same population size because they are both equally affected by the pathogens. Although in this case there is almost no change in pathogen strategy (Figure 2a), we see a decrease in resident population size because the infection is density-dependent and the addition of the migrant population increases host density for half of the year.

We now consider the case in which migrants and residents differ in their pace of life, but have the same tolerance to infection ($m_M=0.8$ and f_M varies) (Figure 3). As would be predicted from the single population case (Figure 1b), we see that spillover into a migrant with a slower pace of life leads to a decrease in pathogen strategy and spillover into a migrant population with a faster pace of life leads to an increase in pathogen strategy. When we consider mortality rate and fecundity rate separately, as would be expected from the single population case (Figures A2 and A4), spillover into a migrant population with a lower mortality rate leads to a decrease in pathogen strategy (Figure A3a), spillover into a migrant population with a higher mortality rate leads to an increase in pathogen strategy (Figure A3a) and the fecundity rate of the migrant population has next to no effect on pathogen strategy (Figure A5a). However, unlike in the single population case (Figure 1b) and unlike the case in which migrant mortality rate but not migrant fecundity rate is varied (Figure A3a), the relationship we see between migrant pace of life and change in pathogen strategy is not monotonic (Figure 4a). When migrants have a very slow pace of life, there is no change in pathogen strategy. Then, as migrant pace of life gets faster, there is a larger decrease in pathogen strategy until a certain point ($f_M=0.6$) past which we see a monotonic increase of pathogen strategy as migrant pace of life gets faster.

This non-monotonicity can be explained by considering how migrant and resident population sizes vary with migrant pace of life (Figure 3b) and especially how population sizes vary with migrant fecundity rate (Figure A5b). When migrants have very slow paces of life (low fecundity rates), their population is extirpated and they exert no selection pressure on pathogen strategy. This contrasts with the large migrant population sizes when migrant mortality rates are lower than residents and migrant and resident fecundity rates are equal (Figure A3b). Then, as migrant fecundity rates get faster, migrants make up a larger portion of the population, leading to a larger influence on pathogen strategy. Since initially migrants have a lower average pathogen strategy than the residents, this leads to a larger decrease in pathogen strategy. However, as the

migrant pace of life continues to increase, the difference between what pathogen strategies are optimal in each population separately also gets smaller (Figure 1b), leading to a smaller decrease in pathogen strategy. The combination of these effects leads to a peak in the size of the decrease in pathogen strategy for an intermediate pace of life. When $f_R < f_M$, increases in f_M lead to both larger differences in optimal pathogen strategy between migrants and residents (Figure 1b) and increases in the size of the migrant population (Figure 3b). This differs from the case in which migrants have higher mortality rates than residents but equal fecundity rates, in which higher migrant mortality rates lead to smaller migrant population sizes (Figure A3b). Thus, we see a monotonic increase in change in pathogen strategy as the pace of life gets faster when $f_R < f_M$ with increases in pathogen strategy that are larger in magnitude than if only migrant mortality rate was varied. Thus, although migrant mortality rates are largely responsible for the direction of the change in pathogen strategy following spillover, migrant fecundity rates qualitatively affect the magnitude of the change through the effect of fecundity on population size.

Finally, we consider the case in which migrants and residents differ both in pace of life and tolerance to infection (Figure 4). We find that the effects of these two factors on pathogen strategy are not simply additive as they were in the single population case (Figure 1c). Although a larger tolerance to infection always leads to a larger increase in pathogen strategy (lines with small m_M are typically above those with larger m_M in Figure 4a), a faster pace of life sometimes leads to an increase in pathogen strategy and sometimes leads to a decrease (some lines in Figure 4a increase, others decrease). The cases where a faster migrant pace of life leads to a larger decrease in pathogen strategy, even when $f_R < f_M$ are the cases where the migrant's tolerance to infection is lower.

As with the non-monotonicity in Figure 3a, this can be understood by considering how the migrant pace of life influences the proportion of the total number of hosts that are migrants at equilibrium along with the difference in optimal pathogen strategy between residents and migrants (Figure 1c). When $m_R < m_M$, there are cases when the pathogen strategy that would evolve in the migrant is lower than that in residents even when the migrant's pace of life is faster (Figure 1c). In these cases, adding in the migrant exerts a downward pull on pathogen strategy. Meanwhile, the proportion of migrants at equilibrium increases monotonically with migrant fecundity rate meaning that the migrant population exerts a larger pull on the pathogen strategy (Figure 4b). Thus, when the migrant population has a lower tolerance to infection, faster paces of life mean that more of the difference between the pathogen strategy that is optimal in the migrant and resident populations is realized as a change in pathogen virulence phenotype. This explains why the change in pathogen strategy continues to decrease even when $f_R < f_M$ in cases where $m_R < m_M$ (Figure 4a). When the tolerance to infection and pace of life mechanisms act together, a faster pace of life does not always lead to higher pathogen strategy values, but instead can serve to amplify the direction of the effect caused by differences in tolerance to infection.

4 | DISCUSSION

Predicting the outcome of disease spillover events between migrant and resident populations is increasingly important as altered movement patterns lead to novel contact opportunities (Altizer et al., 2013). This requires understanding how pathogen virulence and transmission rate phenotypes evolve following spillover. There are many host-pathogen systems in which some hosts migrate and others do not (Table 2). However, there are very few systems for which we understand how pathogen virulence phenotypes differ between migratory and non-migratory host populations (Alexander, 2007; Altizer, 2001; Altizer et al., 2004; Ugelvik et al., 2017). There are even fewer systems for which we have information about the trajectory of pathogen evolution following a spillover event (Hawley et al., 2013). Finally, we found no empirical systems for which we know how pathogen strategy differs in the case where migratory and non-migratory host populations come into repeated contact versus the case where migratory and non-migratory host populations are separate. The lack of empirical data on a phenomenon of relevance in a broad range of host-pathogen systems necessitates the development of general theory.

Despite limited theoretical work about the evolution of virulence following spillover between resident and migrant host populations, we can form intuition from studies on pathogen evolution in other contexts. Some theory predicts that pathogens should evolve lower rates of transmission and lower virulence phenotypes in migrant populations if migratory hosts have lower tolerance to infection or fewer susceptible contacts (Osnas et al., 2015). We might also expect that pathogens should evolve phenotypes with higher rates of transmission and virulence in migratory hosts if migratory hosts have more contacts with susceptible hosts or higher tolerance (Ewald, 1983; Poulin & de Angeli Dutra, 2021). Differences in tolerance to infection and contact rate with susceptible individuals have not been considered simultaneously.

We began by considering how tolerance to infection and pace of life affected pathogen strategy evolution in a single population. In agreement with previous work, we found that a faster pace of life increased the virulence and transmission rates of the pathogen phenotypes that evolved, lower tolerance to infection decreased the virulence and transmission rates of the pathogen phenotypes that evolved and the effects of these two factors were additive when combined. When we investigated spillover, pathogen effects on host population size led to results that could not be directly predicted from the single population case. When migrants have a faster pace of life, spillover into a migrant population leads to the evolution of pathogen phenotypes with higher rates of transmission and virulence. This leads to a decrease in resident population size. This is an example of pathogen-mediated apparent competition, in which spillover reduces the abundance of one of the host populations (Power & Mitchell, 2004). When migrants have lower tolerance to infection, spillover into a migrant population leads to the evolution of pathogen phenotypes with lower rates of transmission and virulence. When these differences are combined, with migrants having

lower tolerance to infection and faster paces of life, the size of the decrease in pathogen transmission and virulence phenotypes can increase with faster paces of life, reversing the direction of the effect of migrant pace of life when values of tolerance to infection are equal. Thus, the direction of the effect of pace of life on the evolution of virulence following a spillover event depends on differences in tolerance to infection.

Although our study was primarily concerned with pathogen evolution, feedback with the host population response to infection was important. Other ways in which coevolution between hosts and pathogens following spillover might affect pathogen evolution would require consideration of the phylogenetic distance between host populations. The only host traits we vary are pace of life and tolerance to infection and the only pathogen traits we vary are transmission rate and virulence phenotype. Neither the evolution of host resistance to infection nor pathogen-host breadth is included. How these factors operate during spillover may depend on phylogenetic relatedness between host populations. The relationship between phylogenetic distance between host populations and resistance to infection is complicated because as phylogenetic distance increases, nonhost resistance increases and evolved resistance decreases (Antonovics et al., 2013). Adding considerations of resistance and host breadth as a function of phylogenetic distance between hosts to our model would add valuable nuance.

Our model is based on the commonly held and broadly theoretically and empirically supported assumption that there is a tradeoff between transmission rate and virulence (Acevedo et al., 2019; Alizon & van Baalen, 2005; Bonneaud et al., 2020; Cressler et al., 2016; de Roode et al., 2008; Lipsitch & Moxon, 1997; Turner et al., 2021). There are cases in which this tradeoff might not apply. For example, when pathogens infect a host that is a 'dead-end' in terms of transmission or when transmission occurs primarily through vectors, transmission might be disconnected from virulence (Farrell & Davies, 2019). Predicting the outcome of spillover in these cases would require a different model.

Future theoretical work should consider whether different ways to implement virulence and contact rates with susceptible individuals yield qualitatively different predictions. We considered virulence only as increased host mortality and considered differences in pace of life as drivers of differences in contact rates with susceptible hosts. Future spatially explicit models could consider mechanisms explicitly related to movement. For example, sub-lethal costs to movement ability might drive pathogen evolution differently than mortality costs. Manipulating contact rate through host movement or aggregation may yield different predictions than manipulating pace of life. Modelling movement mechanisms could increase our understanding of the effects of host migration on pathogen evolution separate from the associations between migration and pace of life and tolerance to infection.

As migration patterns change, it is increasingly important to understand and predict the trajectory of spillover events between migrant and resident populations, including the evolutionary trajectory of pathogen strategy. Predicting the trajectories of spillover events involving migratory species is important to maintaining wildlife, livestock and human health in a changing world (Altizer et al., 2011).

AUTHOR CONTRIBUTIONS

Allison K. Shaw conceived the idea and secured funding; Martha Torstenson and Allison K. Shaw formulated the research goals; Martha Torstenson developed, coded and analysed the model; Martha Torstenson wrote the original draft; and Martha Torstenson and Allison K. Shaw edited the final draft.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest.

DATA AVAILABILITY STATEMENT

Model code is available from Zenodo at https://doi.org/10.5281/zenodo.10723759 (Torstenson & Shaw, 2024).

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REFERENCES

Acevedo, M. A., Dillemuth, F. P., Flick, A. J., Faldyn, M. J., & Elderd, B. D. (2019). Virulence-driven trade-offs in disease transmission: A meta-analysis. *Evolution*, *73*(4), 636–647. https://doi.org/10.1111/evo.13692

Alexander, D. J. (2007). An overview of the epidemiology of avian influenza. *Vaccine*, *25*(30), 5637–5644. https://doi.org/10.1016/j.vaccine.2006.10.051

Alizon, S., & van Baalen, M. (2005). Emergence of a convex trade-off between transmission and virulence. *The American Naturalist*, *165*(6), E155–E167. https://doi.org/10.1086/430053

Altizer, S. (2001). Migratory behaviour and host-parasite co-evolution in natural populations of monarch butterflies infected with a protozoan parasite. *Evolutionary Ecology Research*, *3*, 611–632.

Altizer, S., Bartel, R., & Han, B. A. (2011). Animal migration and infectious disease risk. *Science*, *331*, 296–302. https://doi.org/10.1126/science.1194694

Altizer, S., Becker, D. J., Epstein, J. H., Forbes, K. M., Gillespie, T. R., Hall, R. J., Hawley, D. M., Hernandez, S. M., Martin, L. B., Plowright, R. K., Satterfield, D. A., & Streicker, D. G. (2018). Food for contagion: Synthesis and future directions for studying host–parasite responses to resource shifts in anthropogenic environments. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *373*(1745), 20170102. https://doi.org/10.1098/rstb.2017.0102

Altizer, S., Hochachka, W. M., & Dhondt, A. A. (2004). Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American house finches. *Journal of Animal Ecology*, *73*(2), 309–322. https://doi.org/10.1111/j.0021-8790.2004.00807.x

Altizer, S., Ostfeld, R. S., Johnson, P. T. J., Kutz, S., & Harvell, C. D. (2013). Climate change and infectious diseases: From evidence to a predictive framework. *Science*, *341*, 514–519. https://doi.org/10.1126/science.1239401

Anderson, R. M., & May, R. M. (1982). Coevolution of hosts and parasites. *Parasitology*, *85*(2), 411–426. https://doi.org/10.1017/S0031182000055360

Antonovics, J., Boots, M., Ebert, D., Koskella, B., Poss, M., & Sadd, B. M. (2013). The origin of specificity by means of natural selection: Evolved and nonhost resistance in host–pathogen interactions. *Evolution*, *67*(1), 1–9. https://doi.org/10.1111/j.1558-5646.2012.01793.x

Ashander, J., Krkošek, M., & Lewis, M. A. (2012). Aquaculture-induced changes to dynamics of a migratory host and specialist parasite: A case study of pink salmon and sea lice. *Theoretical Ecology*, *5*(2), 231–252. https://doi.org/10.1007/s12080-011-0122-4

Benson, L., Davidson, R. S., Green, D. M., Hoyle, A., Hutchings, M. R., & Marion, G. (2021). When and why direct transmission models can be used for environmentally persistent pathogens. *PLoS Computational Biology*, *17*(12), e1009652. https://doi.org/10.1371/journal.pcbi.1009652

Best, A., Ashby, B., White, A., Bowers, R., Buckling, A., Koskella, B., & Boots, M. (2017). Host-parasite fluctuating selection in the absence of specificity. *Proceedings. Biological sciences*, *284*(1866), 20171615. https://doi.org/10.1098/rspb.2017.1615

Bonneaud, C., Tardy, L., Hill, G. E., McGraw, K. J., Wilson, A. J., & Giraudeau, M. (2020). Experimental evidence for stabilizing selection on virulence in a bacterial pathogen. *Evolution Letters*, *4*(6), 491–501. https://doi.org/10.1002/evl3.203

Carbó-Ramírez, P., & Zuria, I. (2015). Immune condition and blood parasites in three sparrow species with different migratory status in Central Mexico. *Avian Biology Research*, *8*(3), 167–174. https://doi.org/10.3184/175815515X14371521830098

Cressler, C. E., McLeod, D. V., Rozins, C., Van Den Hoogen, J., & Day, T. (2016). The adaptive evolution of virulence: A review of theoretical predictions and empirical tests. *Parasitology*, *143*(7), 915–930. https://doi.org/10.1017/S003118201500092X

de Roode, J. C., Yates, A. J., & Altizer, S. (2008). Virulence-transmission trade-offs and population divergence in virulence in a naturally occurring butterfly parasite. *Proceedings of the National Academy of Sciences of the United States of America*, *105*(21), 7489–7494. https://doi.org/10.1073/pnas.0710909105

Dingle, H., & Drake, V. A. (2007). What is migration? *Bioscience*, *57*(2), 113–121. https://doi.org/10.1641/B570206

Endo, A., & Nishiura, H. (2018). The role of migration in maintaining the transmission of avian influenza in waterfowl: A multisite multispecies transmission model along east Asian-Australian flyway. *Canadian Journal of Infectious Diseases and Medical Microbiology*, *2018*, 1–7. https://doi.org/10.1155/2018/3420535

Ewald, P. W. (1983). Host- parasite relations, vectors, and the evolution of disease severity. *Annual Review of Ecology and Systematics*, *14*, 465–485. https://doi.org/10.1146/annurev.es.14.110183.002341

Farrell, M. J., & Davies, T. J. (2019). Disease mortality in domesticated animals is predicted by host evolutionary relationships. *Proceedings of the National Academy of Sciences of the United States of America*, *116*(16), 7911–7915. https://doi.org/10.1073/pnas.1817323116

Hawley, D. M., Osnas, E. E., Dobson, A. P., Hochachka, W. M., Ley, D. H., & Dhondt, A. A. (2013). Parallel patterns of increased virulence in a recently emerged wildlife pathogen. *PLoS Biology*, *11*(5), e1001570. https://doi.org/10.1371/journal.pbio.1001570

Hethcote, H. W. (2000). The mathematics of infectious diseases. *SIAM Review*, *42*(4), 599–653. https://doi.org/10.1137/S0036144500371907

Hurtado, P. (2008). The potential impact of disease on the migratory structure of a partially migratory passerine population. *Bulletin of Mathematical Biology*, *70*(8), 2264–2282. https://doi.org/10.1007/s11538-008-9345-y

Krauss, S., Stallknecht, D. E., Negovetich, N. J., Niles, L. J., Webby, R. J., & Webster, R. G. (2010). Coincident ruddy turnstone migration and horseshoe crab spawning creates an ecological 'hot spot' for influenza viruses. *Proceedings of the Royal Society B: Biological Sciences*, *277*(1699), 3373–3379. https://doi.org/10.1098/rspb.2010.1090

Krkošek, M., Lewis, M. A., & Volpe, J. P. (2005). Transmission dynamics of parasitic sea lice from farm to wild salmon. *Proceedings of the Royal Society B: Biological Sciences*, *272*(1564), 689–696. https://doi.org/10.1098/rspb.2004.3027

Lipsitch, M., & Moxon, E. R. (1997). Virulence and transmissibility of pathogens: What is the relationship? *Trends in Microbiology*, *5*(1), 31–37. https://doi.org/10.1016/S0966-842X(97)81772-6

Maina, L. G. M., Maingi, N., Ng'ang'a, C. J., Waruiru, R. M., & Gakuya, F. (2022). Diversity, prevalence, and intensity of gastrointestinal helminth infections in migratory, resident, and sedentary plains zebras (*Equus quagga*) in Masai Mara National Reserve and Lake Nakuru National Park, Kenya. *Veterinary Parasitology: Regional Studies and Reports*, *33*, 100750. https://doi.org/10.1016/j.vprsr.2022.100750

Mathot, K. J., & Frankenhuis, W. E. (2018). Models of pace-of-life syndromes (POLS): A systematic review. *Behavioral Ecology and Sociobiology*, *72*(3), 41. https://doi.org/10.1007/s00265-018-2459-9

McCarville, J., & Ayres, J. (2018). Disease tolerance: Concept and mechanisms. *Current Opinion in Immunology*, *50*, 88–93. https://doi.org/10.1016/j.coi.2017.12.003

Morgan, E. R., Lundervold, M., Medley, G. F., Shaikenov, B. S., Torgerson, P. R., & Milner-Gulland, E. J. (2006). Assessing risks of disease transmission between wildlife and livestock: The Saiga antelope as a case study. *Biological Conservation*, *131*(2), 244–254. https://doi.org/10.1016/j.biocon.2006.04.012

Morgan, E. R., Medley, G. F., Torgerson, P. R., Shaikenov, B. S., & Milner-Gulland, E. J. (2007). Parasite transmission in a migratory multiple host system. *Ecological Modelling*, *200*(3), 511–520. https://doi.org/10.1016/j.ecolmodel.2006.09.002

Nelson, P. G., & May, G. (2017). Coevolution between mutualists and parasites in symbiotic communities may lead to the evolution of lower virulence. *American Naturalist*, *190*(6), 803–817. https://doi.org/10.1086/694334

Osnas, E. E., Hurtado, P. J., & Osnas, E. E. (2015). Evolution of pathogen virulence across space during an epidemic. *American Naturalist*, *185*(3), 332–342. https://doi.org/10.1086/679734

Poulin, R., & de Angeli Dutra, D. (2021). Animal migrations and parasitism: Reciprocal effects within a unified framework. *Biological Reviews*, *96*, 1331–1348. https://doi.org/10.1111/brv.12704

Power, A. G., & Mitchell, C. E. (2004). Pathogen spillover in disease epidemics. *The American Naturalist*, *164*(S5), S79–S89. https://doi.org/10.1086/424610

Rayl, N. D., Merkle, J. A., Proffitt, K. M., Almberg, E. S., Jones, J. D., Gude, J. A., & Cross, P. C. (2021). Elk migration influences the risk of disease spillover in the Greater Yellowstone Ecosystem. *Journal of Animal Ecology*, *90*(5), 1264–1275. https://doi.org/10.1111/1365-2656.13452

Read, A. F. (1994). The evolution of virulence. *Trends in Microbiology*, *2*(3), 73–76. https://doi.org/10.1016/0966-842X(94)90537-1

Restif, O., Hochberg, M. E., & Koella, J. C. (2001). Virulence and age at reproduction: New insights into host–parasite coevolution. *Journal of Evolutionary Biology*, *14*(6), 967–979. https://doi.org/10.1046/j.1420-9101.2001.00355.x

Rigaud, T., Perrot-Minnot, M.-J., & Brown, M. J. F. (2010). Parasite and host assemblages: Embracing the reality will improve our knowledge of parasite transmission and virulence. *Proceedings of the Royal Society B: Biological Sciences*, *277*(1701), 3693–3702. https://doi.org/10.1098/rspb.2010.1163

Rubenstein, D. I., & Hack, M. A. (2013). Migration. In *Encyclopedia of biodiversity* (pp. 309–320). Elsevier. https://doi.org/10.1016/B978-0-12-384719-5.00095-2

Satterfield, D. A., Maerz, J. C., Hunter, M. D., Flockhart, D. T. T., Hobson, K. A., Norris, D. R., Streit, H., de Roode, J. C., & Altizer, S. (2018). Migratory monarchs that encounter resident monarchs show life-history differences and higher rates of parasite infection. *Ecology Letters*, *21*(11), 1670–1680. https://doi.org/10.1111/ele.13144

Servedio, M. R., Brandvain, Y., Dhole, S., Fitzpatrick, C. L., Goldberg, E. E., Stern, C. A., Cleve, J. V., & Yeh, D. J. (2014). Not just a theory—The utility of mathematical models in evolutionary biology. *PLoS Biology*, *12*(12), e1002017. https://doi.org/10.1371/journal.pbio.1002017

Shaw, A. K., Craft, M. E., Zuk, M., & Binning, S. A. (2019). Host migration strategy is shaped by forms of parasite transmission and infection cost. *Journal of Animal Ecology*, *88*(10), 1601–1612. https://doi.org/10.1111/1365-2656.13050

Smith, J., Smith, N., Yu, L., Paton, I. R., Gutowska, M. W., Forrest, H. L., Danner, A. F., Seiler, J. P., Digard, P., Webster, R. G., & Burt, D. W. (2015). A comparative analysis of host responses to avian influenza infection in ducks and chickens highlights a role for the interferon-induced transmembrane proteins in viral resistance. *BMC Genomics*, *16*(1), 574. https://doi.org/10.1186/s12864-015-1778-8

Soriano-Redondo, A., Gutiérrez, J. S., Hodgson, D., & Bearhop, S. (2020). Migrant birds and mammals live faster than residents. *Nature Communications*, *11*(1), 1–8. https://doi.org/10.1038/s41467-020-19256-0

Torstenson, M., & Shaw, A. K. (2024). Model code associated with: Pathogen evolution following spillover from a resident to a migrant host population depends on interactions between host life history speed and cost of infection. *Journal of Animal Ecology*. *Zenodo*. https://doi.org/10.5281/zenodo.10723759

Turner, W. C., Kamath, P. L., Van Heerden, H., Huang, Y. H., Barandongo, Z. R., Bruce, S. A., & Kausrud, K. (2021). The roles of environmental variation and parasite survival in virulence-transmission relationships. *Royal Society Open Science*, *8*(6), 1–21. https://doi.org/10.1098/rsos.210088

Ugelvik, M. S., Skorping, A., Moberg, O., & Mennerat, A. (2017). Evolution of virulence under intensive farming: Salmon lice increase skin lesions and reduce host growth in salmon farms. *Journal of Evolutionary Biology*, *30*(6), 1136–1142. https://doi.org/10.1111/jeb.13082

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

Appendix S1. Appendix S1 includes supplementary figures showing the meaning of the host tolerance parameter and results of simulations examining the effects of host mortality and fecundity rates separately. Appendix S1 also contains methods, results, and figures for the sensitivity analysis.

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