Reactivation of latent infections with migration shapes population-level disease dynamics

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Abstract

Annual migration is common across animal taxa and can dramatically shape the spatial and temporal patterns of infectious disease. Although migration can decrease infection prevalence in some contexts, these energetically costly long-distance movements can also have immunosuppressive effects that may interact with transmission processes in complex ways. Here we develop a mechanistic model for the reactivation of latent infections driven by physiological changes associated with migration (i.e., "migratory relapse") and its consequences for infectious disease dynamics. We determine conditions under which migratory relapse can amplify or reduce infection prevalence across host and pathogen traits and transmission phenologies. We show that migratory relapse can dramatically increase prevalence across the annual cycle and may be crucial for maintaining pathogens with low transmissibility and short infectious periods in migratory populations. Conversely, migratory relapse can reduce the prevalence of highly virulent pathogens by amplifying culling of infected hosts during costly migration, especially for highly transmissible pathogens and those transmitted during migration or the breeding season. Our study provides a mechanistic foundation for understanding the spatiotemporal patterns of relapsing infections in migratory hosts, with implications for zoonotic surveillance and understanding how infection patterns will responds to shifts in migratory propensity associated with environmental change. Furthermore, our work suggests that incorporating within-host processes into population-level models of pathogen transmission may be crucial for reconciling the range of migration–infection relationships observed across migratory host species.

Introduction

Long-distance migration is increasingly recognized to shape the spatial and temporal patterns of infectious disease [1,2]. As these seasonal movements between breeding and wintering grounds occur across animals [3], migration can facilitate the geographic spread of zoonotic pathogens such as filoviruses and West Nile virus [4,5]. Pathogens can also threaten migratory hosts, as observed with sockeye salmon and some waterfowl [6,7]. Accordingly, quantifying the conditions under which migration enhances or dampens pathogen transmission is important to identify when and where infection risks in such species are greatest and to predict the epidemiological consequences of shifting migrations with changing land-use and climate [8,9].

Empirical studies have suggested several ecological mechanisms by which animal migration could increase or decrease infection risks. Seasonal movement from breeding to wintering grounds could expose hosts to a greater number of infected individuals, arthropod vectors, or environmental infectious stages across habitats (i.e., migratory exposure [10–12]). However, because long-distance movement is energetically demanding, migration could also reduce infection prevalence by causing high mortality of infected hosts (i.e., migratory culling [13]). Migration could also allow hosts to escape habitats with high infection risk (i.e., migratory escape [14]) or temporally separate from groups of particularly infectious conspecifics (i.e., migratory allopatry [15]). Additionally, certain habitats experienced during migration could also directly cull pathogens sensitive to environmental conditions (i.e., migratory recovery [16]).

Despite theoretical support [17,18] and grounding of transmission-reducing mechanisms in some empirical systems [13,15,19], recent data syntheses suggest animal migration does not uniformly reduce wildlife disease risks. For example, a meta-analysis across host taxa found relatively weak negative effects of infection status and intensity on survival during migration

60 [20], and a comparative study across migrant, resident, and nomadic ungulates also demonstrated
61 little evidence for migratory escape or culling [21]. Additionally, similar analyses have suggested
62 mixed support for migratory exposure to explain greater infection risks with long-distance
63 movement. Although habitat diversity predicts parasite richness across ungulates, migratory or
64 nomadic species do not sample more diverse habitats than resident counterparts [21]. Further,
65 whereas bird species with greater habitat diversity likewise have higher helminth richness,
66 migratory and resident hosts do not systematically differ in habitat use [22]. Such work suggests
67 greater pathogen exposure of migrants may be insufficient to explain observed infection patterns
68 and that other factors related to host migration more likely drive increased infection risks.
69 One underexplored mechanism focuses on how energetic costs of migration could drive
70 within-host infection processes [23]. Long-distance movement requires substantial energy [24],
71 with some songbirds and temperate bats investing 25–50% of their body mass in fat reserves for
72 flight [25]. Such energy demands and physiological changes can negatively affect migrant
73 immunity [26,27]. Impaired immunity prior to and during migration could make hosts more
74 susceptible to new infections [28]. However, greater susceptibility would likely increase
75 pathogen transmission only when exposure occurs at these stages of the annual cycle (e.g.,
76 breeding grounds before fall migration, wintering grounds in early spring, and stopover sites
77 [11,29]). As an alternative mechanism, immunosuppression associated with migration could
78 cause latent infections (e.g., obtained in a previous season) to reactivate. In a rare experimental
79 test, modified photoperiod of redwings caused the relapse of latent *Borrelia burgdorferi*
80 infections upon initiation of migratory restlessness [30]. Pathogen reactivation caused by
81 physiological preparations for long-distance movement (i.e., “migratory relapse”) could thus

82 facilitate migratory hosts arriving at their breeding grounds and their wintering grounds
83 particularly primed to infect susceptible conspecifics, arthropod vectors, and spillover hosts.
84 Pathogens that exhibit cycles of latency and reactivation (i.e., susceptible–infected–
85 latent–infected dynamics) are relatively common, particularly for viruses, bacteria, and some
86 protozoa [31–33], and infect migratory hosts ranging from birds to ungulates to bats (Table 1).
87 Immunosuppression from stressors broadly drives the reactivation of pathogens such as
88 herpesviruses in ungulates [34,35], haemosporidians and flaviviruses in songbirds [36,37], and
89 henipaviruses in flying foxes [38,39]. Similarly, migration has been implicated in the relapse of
90 not only *Borrelia burgdorferi* in thrushes but also West Nile virus in white storks [40] and
91 haemosporidians in rusty blackbirds [41], among other examples (Table 1). Recent comparative
92 analyses across birds also support migration-induced relapse, as temperate bird species with a
93 broader range of migratory movements from their wintering grounds (where vector exposure is
94 unlikely) to their breeding grounds are more capable of infecting susceptible ticks [42]. Such
95 findings more generally underscore that animal migration could play an underrecognized role in
96 the spatial and temporal dynamics of relapsing pathogens across diverse mobile host species.
97 Mechanistic models of relapsing infections in closed populations broadly suggest that
98 reactivation is an important determinant of pathogen persistence [39], especially when
99 transmission is seasonal (e.g., many vector-borne diseases [43,44]). For migratory species,
100 although pathogen transmission often occurs at the breeding grounds [17], exposure
101 opportunities can take place at other or multiple stages of the annual cycle [11,29,45,46]. If
102 transmission occurs in only the breeding or wintering grounds, relapse during spring and fall
103 migration could facilitate pathogen persistence, akin to biannual birth pulses [47], and elevate
104 prevalence through increasing the force of infection. However, migratory relapse may have

strong impacts only when enough hosts are latently infected, suggesting that pathogen traits could moderate when and where relapse increases prevalence. Short infectious periods relative to migration duration could limit how strongly migrants contribute to pathogen transmission upon arrival at the breeding or wintering grounds. Similarly, relapse of particularly virulent pathogens could rapidly cull infected hosts and reduce prevalence [1]. Integrating relapse into general theory for infection dynamics in a migratory host population could accordingly help to disentangle when animal migration is most likely to magnify infectious disease risks.

We here develop a mechanistic model of relapsing infections in a migratory host to explore the conditions under which migration-induced pathogen reactivation amplifies or dampens infection risks. We expand prior modeling frameworks for a host undergoing a two-way annual migration between the breeding and wintering grounds [17,18]. By assuming the start of spring and fall migration causes some fraction of latently infected hosts to relapse, we explore how the frequency of reactivation affects infection dynamics across the annual cycle of a migratory species. Additionally, we identify pathogen traits for which migratory relapse most influences disease risk and assess how the phenology of pathogen transmission modifies infection outcomes. Using models to explore these scenarios could help establish the kinds of host–pathogen systems for which reactivation driven by animal migration can amplify or reduce infection prevalence and thus better guide future empirical tests of model predictions [48].

Methods

Model structure

We developed a differential equation model describing population and infection dynamics during breeding, migration, and overwintering periods. We coarsely parameterized our model around a

migratory songbird, but our model structure remains general enough to apply to a range of migratory host and pathogen systems in which latent infections reactivate.

Host demography and migration

In the absence of infection, our model tracks the host population size, $N(Y,\tau)$, in year Y and within-year time τ, where τ takes values between 0 and 1 corresponding to the start and end of the calendar year, respectively. The population dynamics of the migratory host are described by ordinary differential equations across each stage of the annual cycle [17]. To avoid offspring contributing to reproduction in their hatch year, fecundity is proportional to the number of migrants returning at the start of the breeding season ($N_{breed}(Y) = N(Y,\tau_0)$ [49]. Per-capita offspring production takes the density-dependent form $b_0 - b_1 N$, where b_0 and b_1 are constants. The per-capita mortality rate is assumed to be independent of host density but varies across the annual cycle (μ_j, where $j = b, m, w$ denotes breeding, migration, and wintering, respectively). The probability of survival at each stage is given by the following equation:

$$\sigma_j = e^{-\mu_j T_j}$$

Here, T_j ($j = b, m, w$) represents the proportion of the year occupied per stage of the annual cycle. I the breeding season (i.e., $\tau_0 \leq \tau < \tau_0 + T_b$), population dynamics are as follows:

$$\frac{dN}{d\tau} = (b_0 - b_1 N)N_{breed} - \mu_b N$$

150 During spring and fall migrations, hosts travel between breeding and wintering grounds each for
151 a duration T_m and experience a per-capita mortality rate μ_m. Hosts spend the remainder of the
152 annual cycle at the wintering grounds (i.e., $T_w = 1 - T_b - 2T_m$) with a per-capita mortality rate
153 μ_w. During migration and winter, population dynamics are given by the following equations:
154

155 $$\frac{dN}{d\tau} = -\mu_m N$$

156 and

157 $$\frac{dN}{d\tau} = -\mu_w N$$

158

159 <u>Seasonal infection dynamics</u>

160 To represent the dynamics of relapsing infections, we categorize hosts by their current infection
161 status (i.e., susceptible [S], infected [I], and latent [L]), following the SILI model framework
162 [33,39]. Transmission from infectious to susceptible hosts in each stage of the annual cycle
163 occurs at the density-dependent rate β_j (see scenarios below), with infected animals becoming
164 latent at rate ρ (i.e., $1/\rho$ is the duration of non-lethal acute infections). We model stage-specific
165 costs of infection as proportional reductions in host fecundity (c_f) and survival (c_j), with each
166 ranging from 0 (i.e., no change) to 1 (i.e., infected hosts have a 100% reduction in fecundity or
167 lifespan). During the breeding season, we model infection dynamics as follows:
168

169 $$\frac{dS}{d\tau} = \left(b_0 - b_1\left(S + I\left(1 - c_f\right) + L\right)\right) N_{breed} - \mu_b S - \beta_b SI$$

170 $$\frac{dI}{d\tau} = \beta_b SI - \frac{\mu_b}{1-c_b} I - \rho I$$

171 $$\frac{dL}{d\tau} = \rho I - \mu_b L$$

Migratory relapse is modeled by the parameter ε, representing the fraction of latent hosts that return to the infectious class. Because latent infections can reactivate shortly after the initiation of migratory restlessness [30], we assume relapse occurs instantaneously at the start of both spring and fall migrations. The starting values for latent and infected individuals at the initiation of each migration are reset accordingly:

$$L = L(1 - \varepsilon)$$

and

$$I = I + L\varepsilon$$

Owing to immunosuppression during migration, we assume acute infections do not subside during migration (i.e., $\rho = 0$). Infection dynamics are thus given by the following equations:

$$\frac{dS}{d\tau} = -\mu_m S - \beta_m SI$$

$$\frac{dI}{d\tau} = \beta_m SI - \frac{\mu_m}{1-c_m} I$$

$$\frac{dL}{d\tau} = -\mu_m L$$

Infection dynamics are modeled in a similar manner at the overwintering grounds, although infections can here become latent owing to relatively weaker physiological tradeoffs:

$$\frac{dS}{d\tau} = -\mu_w S - \beta_w SI$$

$$\frac{dI}{d\tau} = -\frac{\mu_w}{1 - c_w} I - \rho I$$

$$\frac{dL}{d\tau} = \rho I - \mu_w L$$

Parameterization

We parameterized our model using the dark-eyed junco (*Junco hyemalis*), a temperate songbird with diverse migratory strategies throughout North America [50]. Juncos breed across Alaska, Canada, and the northern United States, migrating south in autumn and wintering throughout the central and eastern United States. The timing of the junco annual cycle is well-characterized, with migrants beginning to depart their wintering grounds in early March, migrations spanning four to eight weeks, and reproduction starting at the breeding grounds in May [51,52]. Migrants depart their breeding grounds in early October and arrive at the wintering grounds in November. Juncos lay an average of four eggs per clutch across 1–2 broods, resulting in a maximum per-capita fecundity of eight juveniles reared over the five-month breeding season [50]. The survival probability at the breeding grounds is high, whereas that during spring and fall migration is lower. As the survival probability during winter is slightly higher than during migration [51], we assume $\sigma_m < \sigma_w < \sigma_b$ to characterize temperate migrants. However, we also assess sensitivity to this assumption by modeling equal winter and breeding survival probabilities ($\sigma_w = \sigma_b$), as likely occurs for many Neotropical migrants [53]. Although migratory relapse has not been observed in juncos, this species can be infected with relapsing pathogens (e.g., *B. burgdorferi* and haemosporidians) and displays physiological costs of long-distance migration [19,54,55].

Model analysis and infection scenarios

Because our main aim was to assess how migratory relapse affects long-term infection prevalence (I/N), we varied the proportion of hosts that relapse at the start of migration (ε) from 0 (i.e., no reactivation of infection) to 1 (all latent infections reactivate at the onset of migration). In our baseline scenario, we assumed pathogen transmission occurs only at the breeding grounds [17] (i.e., $\beta_b = \beta,\ \beta_m = \beta_w = 0$). This assumption holds for systems where only the breeding season produces enough susceptible hosts to enable transmission or where exposure is driven by breeding behavior [56]. Limiting transmission to the breeding grounds can also coarsely approximate vector-borne diseases given the phenology of many arthropod vectors [31].

To identify pathogen traits that might shape the degree to which migratory relapse affects infection prevalence, we next systematically varied the duration of acute infection ($1/\rho$) and transmission rate (β) alongside our relapse parameter (ε). Because past work on juncos supports migratory culling in this particular host system [19], and because particularly virulent pathogens could rapidly cull latent hosts that become infected with migratory relapse [1], we also varied the cost of infection for survival during migration (c_m). We assumed that the costs of infection to fecundity (c_f) and survival during both breeding (c_b) and wintering (c_w) stages were low.

Lastly, because host aggregations (e.g., at stopover sites [29]) or particular environmental conditions (e.g., tropical habitats supporting winter activity of vectors [11]) can facilitate some pathogens being transmitted outside of only the breeding season, we explored model behavior across three additional transmission phenologies: (*i*) winter only ($\beta_w = \beta,\ \beta_b = \beta_m = 0$), (*ii*) migration only ($\beta_m = \beta,\ \beta_b = \beta_w = 0$) or (*iii*) year-round ($\beta_b = \beta_m = \beta_w = \beta$).

Across all parameterizations, we ran our model for at least five years and allowed simulations to continue until the maximum mean difference in prevalence per timestep in the previous five years was below 0.0001, representing a steady state annual cycle. Most iterations

239 reached steady state in 40 years. Figure 1 shows our model structure and example population
240 sizes. All simulations were conducted in R using the *deSolve* package [57]. For each unique set
241 of model parameters, we recorded the maximum infection prevalence across the steady state
242 annual cycle. Maximum prevalence is notably important for understanding the epidemiological
243 consequences of migratory relapse for both pathogen spillover and wildlife conservation [49].
244

245 **Results**

246 *Migratory relapse and host–pathogen dynamics*

247 In the absence of pathogen relapse ($\varepsilon = 0$) and when transmission occurs only in the breeding
248 season, infection prevalence in the migratory population is relatively low for our baseline
249 parameterization. Prevalence increases early in the breeding season, attains its maximum prior to
250 fall migration, and decreases over the winter through lack of transmission (Fig. 2). In contrast,
251 migratory relapse generally amplifies prevalence across the annual cycle, especially for low-
252 virulence pathogens (i.e., causing a modest increase in migratory mortality); at its most extreme
253 ($\varepsilon = 1$), reactivation of all latent hosts upon initiation of migration triggers dramatic pulses of
254 infection back into the host population (Fig. 2A). Prevalence then declines across the breeding
255 season, as relapsed hosts transition to latency. As more hosts acquire infection, migratory relapse
256 reduces population size, with the largest reductions occurring for pathogens of intermediate
257 virulence (Fig. 2A). Intermediate infection costs decrease the mean and variance in prevalence
258 with migratory relapse across the annual cycle (Fig. 2B). However, when infection poses strong
259 costs for migrant survival and the fraction of reactivation is high, migratory relapse converts
260 hosts from latency to actively infected and in turn enhances the effect of migratory culling on
261 infected individuals (i.e., mortality), thereby reducing population-level prevalence (Fig. 2C).

262

263 *Sensitivity to pathogen traits and host overwinter survival*

264 To understand broader contexts where migratory relapse amplifies or minimizes infection
265 prevalence, we systematically covaried the cost of infection to migratory survival (c_m), the
266 fraction of relapsing hosts upon migration (ε), pathogen transmissibility (β), and the duration of
267 non-lethal acute infection ($1/\rho$). We explored patterns for two host migration scenarios: that of a
268 temperate migrant with winter survival lower than breeding survival ($\sigma_w < \sigma_b$) and that of a
269 Neotropical migrant where winter survival is equivalent to breeding survival ($\sigma_w = \sigma_b$).

270 For the baseline (i.e., temperate migrant), migratory relapse broadly increases the
271 maximum prevalence when costs of infection are low (Fig. 3). For pathogens with low
272 transmissibility ($\beta = 0.05$) and short acute infections (i.e., one month relative to the six-week
273 migration), migratory relapse allows pathogens to persist that otherwise would be unable to
274 invade the host population (Fig. 3). As expected, pathogens with higher transmissibility and
275 longer acute infections attain greater peak prevalence when costs of infection are low to
276 intermediate. However, migratory relapse can decrease prevalence relative to models assuming
277 no relapse ($\varepsilon = 0$) when costs of infection and transmission rates are both high (Fig. 3).

278 These patterns were generally consistent when winter survival reflected Neotropical
279 migrant systems ($\sigma_w = \sigma_b$). Maximum prevalence was overall greater, with the pathogen
280 typically able to persist without relapse (Fig. S1). With low costs of infection for migrant
281 survival, migratory relapse produced greater increases in prevalence, particularly with higher
282 transmission rates and longer infectious periods. At higher infection costs, the difference in
283 maximum prevalence between models without relapse ($\varepsilon = 0$) and with complete relapse ($\varepsilon =$
284 1) was minimized so that migratory relapse only dampened infection by virulent pathogens.

Sensitivity to transmission phenology

We lastly considered how the timing of pathogen transmission within the migratory host annual cycle affects model outcomes. The amplifying effects of migratory relapse on prevalence are most pronounced when transmission rates are low, irrespective of transmission phenology (Fig. 4). Yet when transmission rates are high, complete migratory relapse ($\varepsilon = 1$) can reduce prevalence relative to a model without relapse ($\varepsilon = 0$) especially when transmission occurs during migration and throughout the annual cycle. For the former, this infection-dampening effect of relapse is particularly pronounced for pathogens with short infectious periods (Fig. S2). These patterns were similar for the Neotropical migrant scenario (i.e. high winter survival), although fewer regions of parameter space reduced infection prevalence with relapse (Fig. S3).

Discussion

Determining the conditions under which migration amplifies or dampens pathogen transmission is important to identify when and where infection risks are greatest in highly mobile species. Migratory species host various pathogens with cycles of latency and reactivation, including several with zoonotic potential [30,39,40], but theory to date on migratory host–pathogen interactions does not account for migration-induced pathogen reactivation and its population-level consequences. Using a mathematical model, we found that migratory relapse can increase or decrease infection prevalence throughout the annual cycle, dependent upon pathogen traits and transmission phenology. For relatively low-virulence pathogens, gains to the infectious class through relapse outpace losses of infected individuals through migratory culling to increase prevalence. In contrast, migratory relapse can exacerbate migratory culling to reduce prevalence

308 for pathogens that are especially virulent, highly transmissible, and can be spread during the
309 breeding and migratory stages of the annual cycle. Past theory on host–pathogen dynamics
310 generally concludes that migration reduces infection prevalence by reducing transmission
311 opportunity (i.e., migratory escape) and infected host survival (i.e., migratory culling) [16–18].
312 Our work instead suggests that incorporating physiological processes into host–pathogen models
313 (i.e., energetic demands of migration that lead to immunosuppression and relapse [24,26,27])
314 could account for the wider range of migration–infection relationships observed in wildlife.

315 For an annual cycle typical of migratory songbirds, and a range of biologically plausible
316 pathogen traits, our model generated dramatic increases in infection prevalence with an increase
317 in the proportion of hosts experiencing migratory relapse. In some cases, relapse was necessary
318 to maintain persistence of less-transmissible pathogens with shorter acute infections across the
319 annual cycle. Although prior theory has not examined relapse induced by physiological changes
320 with migration, past models of relapsing pathogens have demonstrated that reactivation can
321 facilitate pathogen persistence. In the absence of immigration, recurrent cycles of acute infection
322 and latency were necessary to explain henipavirus dynamics in straw-colored fruit bats [39], and
323 more frequent relapse optimized pathogen invasion potential for human malaria (*Plasmodium*
324 *vivax*) [44]. Our model suggests migratory relapse can generate pronounced biannual peaks in
325 prevalence that maintain more infection throughout the annual cycle than by direct transmission
326 alone. This pattern differs in its seasonal timing and magnitude from those generated by seasonal
327 birth pulses or relapse induced by immune tradeoffs with reproduction [36,38,58,59], especially
328 in systems where offspring benefit from prolonged maternal immunity [60]. Because this pattern
329 was most evident when infection had low costs for migrant survival, our model predictions may
330 be especially applicable to migratory reservoir hosts that themselves do not experience high

331 morbidity or mortality (e.g., henipaviruses in flying foxes and *Borrelia burgdorferi* in songbirds
332 [30,39]). Given the high prevalence of actively infectious hosts during migration, migratory
333 stopover sites and arrival periods of migrants returning to breeding or wintering grounds could
334 be prioritized as important targets for surveillance and interventions to limit spillover risk [61].
335 Our model also identified contexts in which migratory relapse can decrease prevalence,
336 or even cause pathogen extinction, by removing more actively infectious hosts with migratory
337 culling. Reductions in prevalence were most likely to be seen in highly transmissible pathogens,
338 those with long infectious periods, and when transmission occurred at the breeding grounds or
339 during migration. Systems for which such conditions are met include rhabdoviruses in salmonid
340 fish as well as haemosporidian parasites and flaviviruses in some songbirds [7,19,62,63]. High
341 relapse frequency and high mortality of infected migrants might be more common for species
342 undertaking strenuous migrations, such as those involving prolonged periods of powered flight.
343 This supports prior predictions that migratory culling is more likely to operate in long-distance
344 migrations with fewer stopovers than shorter migrations or where migratory species move and
345 forage in a stable environmental window (e.g., ungulates "surfing the green wave") [17,64].
346 Given the sensitivity of our model to the degree of relapse and the costs of infection for
347 migrant survival, estimation of these parameters will be important for applying this framework to
348 empirical systems. Detecting pathogen relapse requires sampling hosts across their annual cycle
349 [33], which can be logistically challenging for highly mobile species [65]. However,
350 advancements in tracking technologies (e.g., lightweight geolocators, stable isotopes) are
351 improving inference into migratory networks, which could facilitate temporal sampling across
352 breeding, wintering, and stopover habitats [66]. Additionally, multiple diagnostic efforts are
353 necessary to differentiate compartments within the SILI framework. For example, serology,

354 PCR, and microscopy can distinguish uninfected (i.e., susceptible), acutely infected, and latent
355 hosts for haemosporidian parasites [67], and pathogen genotyping could disentangle whether
356 infections are more likely to indicate reactivation rather than novel transmission events [33].
357 Integrating longitudinal sampling and these diagnostics with mark–recapture could help to
358 estimate infection- and stage-specific survival probabilities and infer pathogen costs [68].
359 To examine conditions under which relapse during migration increases or decreases
360 infection risks, we focused our model on a simple system with a single interaction between a
361 migratory host and its pathogen. For tractability, we ignored alternative ecological interactions
362 such as multiple host species (and thus a more generalist pathogen) or explicit arthropod vectors.
363 However, high prevalence in relapsing migrants suggests migrants could introduce pulses of
364 infection into seasonally sympatric co-occurring species. This could be particularly important for
365 pathogen maintenance in partially migratory species such as juncos, where long-distance
366 migrants form mixed flocks with residents at shared wintering grounds [50]. For vector-borne
367 pathogens with cycles of latency and reactivation, such as *Borrelia burgdorferi* and some
368 flaviviruses [30,69], migrants undergoing relapse could also have disproportionate influence on
369 infecting vectors upon their arrival at the breeding grounds. Future extensions to our model could
370 assess how phenological overlap between migrants and vectors shapes disease outcomes [49,70].
371 Because many species are shifting the timing and extent of their migratory movements in
372 response to climate and anthropogenic change [9,71], associated changes to host physiology and
373 survival could alter how migratory relapse influences infection patterns. Deterioration of
374 resources at breeding or wintering sites could increase the proportion of relapsing hosts,
375 increasing prevalence of low-virulence pathogens or preventing persistence of high-virulence
376 pathogens if migratory mortality is also elevated. Additionally, many migratory species are

377 increasingly overwintering in anthropogenic habitats. For example, Anna's hummingbirds and
378 European blackcaps both spend more of their annual cycle in urbanized regions owing to
379 abundant and predictable anthropogenic food resources [72,73]. Such resources could dampen or
380 amplify the likelihood of pathogen relapse at spring departure depending on their nutritional
381 quality or effects on host density and crowding [74]. Other stressors associated with urban
382 habitats, such as artificial light at night, could further amplify the likelihood of relapse [75,76].
383 These consequences of urban habituation could be especially relevant for human health in the
384 context of wildlife reservoirs of relapsing zoonoses, such as flying foxes and henipaviruses [77].
385 Many pathogens of concern to human, domestic animal, and wildlife health exhibit cycles
386 of latency and reactivation. Our model provides a generalizable and mechanistic framework for
387 understanding the dynamics of such infections in migratory hosts. We demonstrate theoretical
388 support for migratory relapse increasing infectious disease risks but also emphasize the context
389 dependence of these patterns on host and pathogen traits. Empirical estimates of relapse and
390 survival across the annual cycle will be important for linking such frameworks with empirical
391 systems, which will be critical for predicting how anthropogenic changes in both climate and
392 land use alter animal migration, reactivation of latent infections, and pathogen spillover risks.
393

394 **Data accessibility**

395 R code for reproducing the main analyses is available in the Dryad Digital Repository:
396 https://datadryad.org/stash/share/eT4DN6oJ0BlGmv5fqjg16cm3HThgXmvgllWemPpInYY.
397

398 **Author contributions**

DJB and RJH designed the model, DJB and EDK parameterized the model, and DJB performed the analyses and wrote the first draft of the manuscript. All authors contributed to revisions.

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Competing interests

We declare no competing interests.

415 **Tables**

416 Table 1. Examples of pathogens that exhibit cycles of latency and reactivation, their detection in

417 migratory or nomadic host species, and likely drivers of observed relapse events.

Pathogen	Host taxon	Behavior	Likely driver of relapse	Reference
Borrelia burgdorferi	Redwing (*Turdus iliacus*)	Migrant	Migration	[30]
Eastern equine encephalitis virus	Passeriformes	Migrant	Migration, reproduction	[69]
Plasmodium spp.	Passeriformes	Migrant	Migration, reproduction	[31]
West Nile virus	White storks (*Ciconia ciconia*)	Migrant	Migration	[40]
Haemosporida	Rusty blackbird (*Euphagus carolin*)	Migrant	Migration	[41]
Infectious hematopoietic necrosis virus	Sockeye salmon (*Oncorhynchus nerka*)	Migrant	Migration	[63]
Brucella abortus	Elk (*Cervus canadensis*)	Migrant	Reproduction	[58]
Equine herpesviruses	Grévy's zebras (*Equus grevyi*)	Migrant	Translocation	[35]
Henipaviruses	Flying foxes (*Pteropus* spp.)	Nomad	Reproduction, nutritional stress	[38]

418

Table 2. Model parameters, their definitions, and default values. All rates are given in units of years^{-1}. Parameterization for host demography and migration is based on juncos [50–52].

Parameter	Definition	Value
Annual cycle		
T_b	Proportion of year spent at breeding grounds	0.42 (~5 months)
T_m	Proportion of year spent in each migration	0.12 (1.5 months)
T_w	Proportion of year spent at wintering grounds	0.34 (~4 months)
τ_0	Start of the breeding season per annual cycle	0.33 (~early May)
Host demography		
b_0	Density-independent reproduction rate	3.3
b_1	Density-dependent reproduction rate	0.003
σ_b	Breeding survival probability	0.95
σ_m	Migration survival probability (one-way)	0.79
σ_w	Winter survival probability	0.85 (temperate), 0.95 (Neotropical)
Pathogen traits		
β	Density-dependent transmission rate	0.05 (low), 0.5 (high)
ρ	Infected-to-latent rate	4 (1 month), 12 (3 months)
ε	Fraction of relapsing hosts at the start of migration	0–1
c_f	Cost of infection for fecundity	0.2
c_b	Cost of infection for breeding survival	0.2
c_m	Cost of infection for migratory survival	0.2–0.8
c_w	Cost of infection for winter survival	0.2

Figures and legends

Figure 1. (A) Model schematic of relapsing infections in a migratory host through the annual cycle, for a pathogen transmitted at the host breeding grounds. Filled circles depict the number susceptible (S), infectious (I) and latently infected (L) hosts, and arrows represent gain or losses to each class through demographic or infection processes. Arrows between panels illustrate that a fraction (ε) of latently infected hosts relapse at the start of migration. (B) Example dynamics over the steady state annual cycle. Solid lines depict numbers of susceptible (S, light gray), infectious (I, pink), and latent (L, dark gray) hosts, and vertical dashed lines delineate the spring and fall migratory periods. Infection parameters used are the transmission rate ($\beta_b = 0.05$), cost of infection for migrant survival ($c_m = 0.5$), rate of transition from infectious to latent ($\rho = 4$, corresponding to a three-month duration of acute infection), and the probability of relapse at the onset of migration ($\varepsilon = 0.75$); demographic parameters are the default values listed in Table 2.

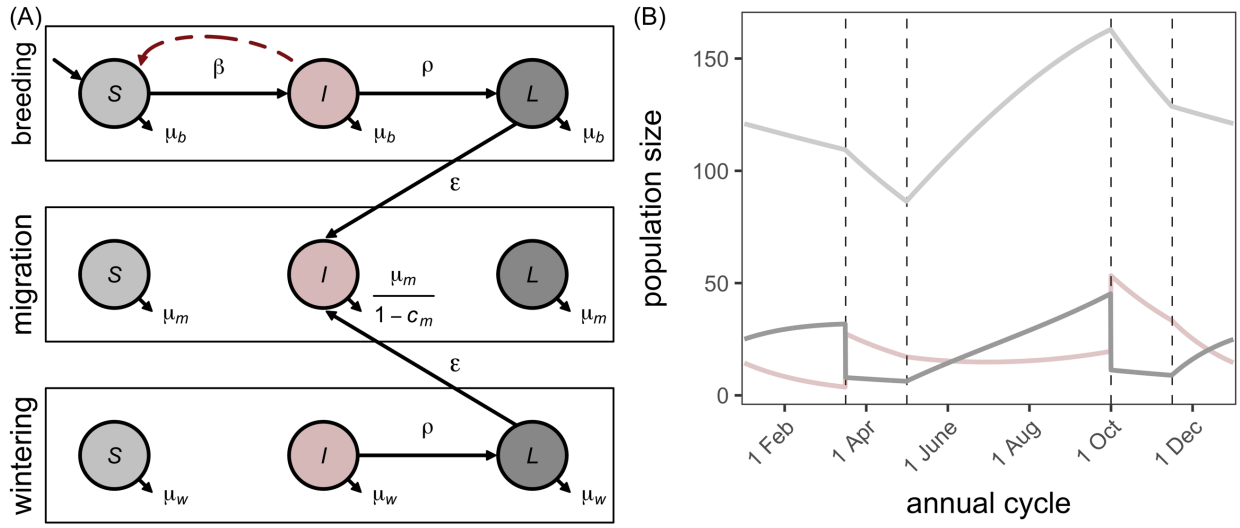


Figure 2. Steady state population size (top row) and infection prevalence (bottom row) across the annual cycle as a function of the fraction of relapse at migration onset (ε, colored lines) and low, medium, and high infection costs for migratory survival (c_m, columns). Transmission occurs at the breeding grounds ($\beta_b = 0.5$), infection is acute for three months ($\rho = 4$), and winter survival represents a temperate migrant ($\sigma_w = 0.85$). All other parameter values are listed in Table 2.

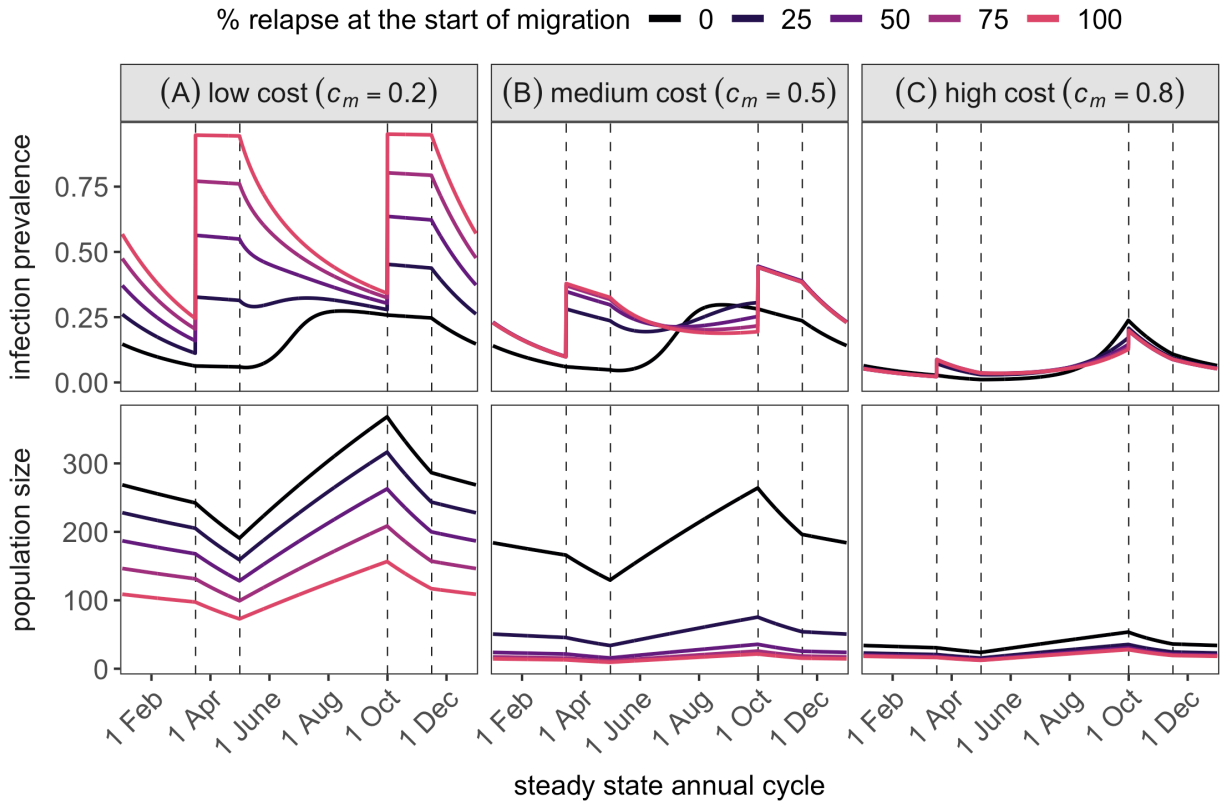


Figure 3. Sensitivity of the relationship between peak infection prevalence and virulence (i.e., cost of infection for migratory survival, c_m) to pathogen traits. Transmission occurs only at the breeding grounds for moderate ($\beta_b = 0.05$, top) and high ($\beta_b = 0.5$, bottom) transmissibility. Lines show the fraction of relapsing hosts at the start of migration (ε), and columns indicate an increasing duration of acute infection ($1/\rho$) from 1 to 3 months. Winter survival represents a temperate migrant ($\sigma_w = 0.85$), and all other parameter values are listed in Table 2.

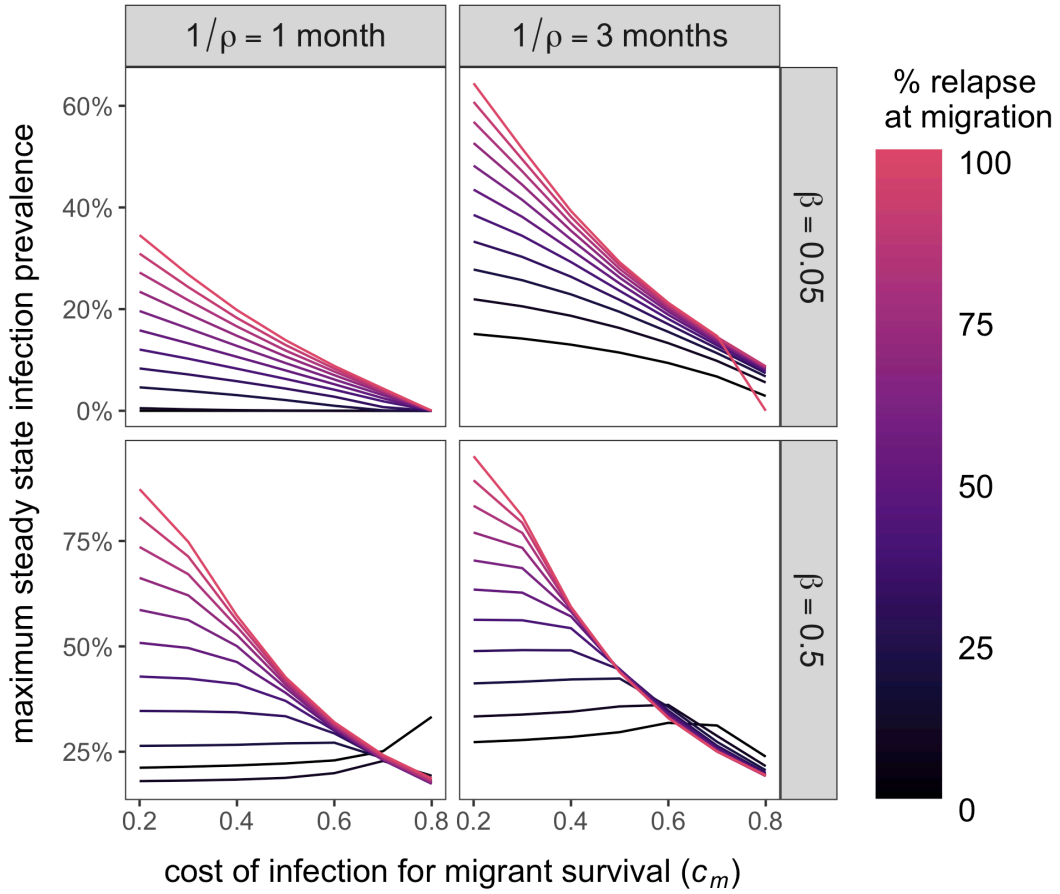
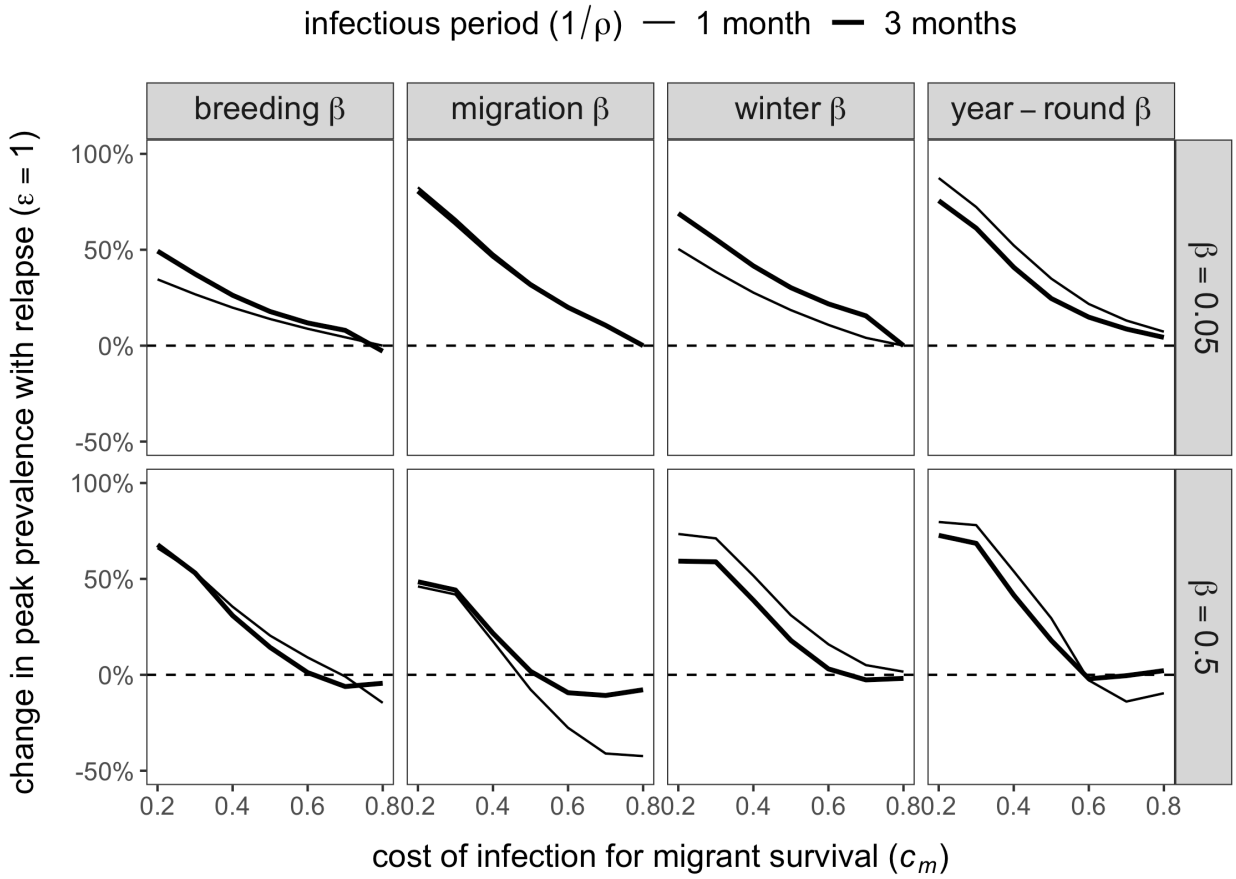


Figure 4. Effects of transmission phenology and pathogen traits on how infection prevalence responds to migratory relapse. We calculate the response of infection prevalence to relapse (vertical axis) as the difference in maximum prevalence between models where all individuals relapse ($\varepsilon = 1$) and no individuals relapse ($\varepsilon = 0$); values above or below the dashed line indicate that relapse increases or decreases peak prevalence, respectively. Columns represent different transmission phenologies (breeding only, migration only, winter only, or year-round), Rows represent moderate ($\beta = 0.05$, top) and high ($\beta = 0.5$, bottom) transmissibility, line width represent short versus long infectious periods ($1/\rho$), and the horizontal axis represents costs of infection for migrant survival (c_m). Winter survival is representative of temperate migrants ($\sigma_w = 0.85$), and all other parameter values are provided in Table 2.



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