

**Infections on the move: How transient phases of host
movement influence disease spread**

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1 **Infections on the move: How transient phases of host**
2 **movement influence disease spread**

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19 Running Title: Transient phases affect disease spread

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25 **Abstract**

26 Animal movement impacts the spread of human and wildlife diseases, and there
27 is significant interest in understanding the role of migrations, biological
28 invasions, and other wildlife movements in spatial infection dynamics. However,
29 the influence of processes during the transient phases of host movement on
30 infection is poorly understood. We propose a conceptual framework that
31 explicitly considers infection dynamics during transient phases of host
32 movement to better predict infection spread through spatial host networks.
33 Accounting for host transient movement captures key processes that occur while
34 hosts move between locations, which together determine the rate at which hosts
35 spread infections through networks. We review theoretical and empirical studies
36 of host movement and infection spread, highlighting the multiple factors that
37 impact the infection status of hosts. We then outline characteristics of hosts,
38 parasites and the environment that influence these dynamics. Recent
39 technological advances provide disease ecologists unprecedented ability to track
40 the fine-scale movement of organisms. These, in conjunction with experimental
41 testing of the factors driving infection dynamics during host movement, can
42 inform models of infection spread based on constituent biological processes.

43

44 **Key Words:** Epidemiology, Disease ecology, Movement ecology, Spatial
45 modelling, Metapopulations, Networks, Host-parasite interactions

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48 **1. Introduction**

49 Understanding how infectious diseases spread through spatial networks
50 of hosts has been called a “holy grail” of epidemiology [1]. Spatial host networks
51 portray host populations as a set of nodes in which hosts reside, and host
52 movement among those locations serves as the links (i.e. edges) connecting the
53 network [2,3]. Since most disease-causing parasites cannot actively disperse,
54 host movement also provides critical links for parasite infections to spread [2].
55 Characterizing these links is not straightforward, however. Multiple processes
56 act on hosts during movement across the landscape that potentially influence
57 infections. Dispersal ecologists refer to this period of movement after organisms
58 depart a discrete location (e.g. household, habitat patch), but before arriving to a
59 different location, as the transient phase [4]. Explicitly considering transient
60 movement phases has provided a deeper understanding of the causes and
61 consequences of wildlife movement [4], but this phase has largely been ignored
62 in studies of disease spread.

63 Moving hosts are subject to changes in biotic and abiotic conditions that
64 alter existing infections [5], cause mortality [6,7] or facilitate acquisition of new
65 infections [8,9]. The infection status of individuals arriving into new locations
66 may therefore be indirectly or unrelated to their infection status when
67 movement is initiated. Here, we review the limitations of current approaches to
68 studying infection spread and emphasise the benefits of explicitly considering
69 the processes that occur during transient phases of host movement (hereafter
70 referred to as “host transience”). We first overview the existing methods
71 examining the link between host movement and infection spread. Second, we
72 propose a modelling framework that explicitly considers host movement and

73 infection dynamics during transient phases, before developing testable
74 hypotheses about the importance of factors influencing infection dynamics
75 during host transience. We conclude by discussing how our framework can guide
76 future research testing the role of host transience in the spatiotemporal
77 dynamics of wildlife and human disease.

78

79 **2. Current approaches for investigating the link between host** 80 **movement and infection spread**

81 Most research has focused on seasonal host migrations [5,7], but we
82 broaden this perspective to consider any movement that connects spatially
83 discrete resident locations of hosts. This includes large-scale seasonal migrations
84 between breeding and non-breeding habitats, but also routine, local movements
85 within populations (e.g., foraging between resource patches, mate searching
86 among subgroups) or more regionally between different populations (e.g.,
87 dispersal). This definition of movement aligns well with existing spatial network
88 frameworks and permits comparisons of infection dynamics during host
89 transience at various scales.

90

91 **a) Theoretical Studies**

92 Spatial network models specify the geographic locations of hosts and
93 their infections over time [3,10]. We define four broad categories of models
94 describing the spatial dynamics of infection spread (Fig. 1), with some examples
95 of each type provided in Table S1 (Supplementary Material). Many existing
96 spatial network models use metapopulation approaches [10], where the unit of

97 measurement is the resident location rather than the individual, each with
 98 standard epidemiological states (e.g. susceptible, exposed, infected, and
 99 recovered). The simplest versions are *phenomenological metapopulation models*
 100 (Fig. 1a) [11], which do not explicitly parameterise host movement, but instead
 101 model connectivity of groups, with rates of spread determined by physical
 102 processes, such as gravitation [12], percolation [13] and radiation [14]. Despite
 103 their simplicity, phenomenological models have accurately reproduced patterns
 104 of disease spread in human and wildlife populations. For example, the spread of
 105 plague in populations of great gerbils (*Rhombomys opimus*) occurs between
 106 resident locations (burrows) that are in closest proximity to one another [13],
 107 while the spread of influenza in humans is explained by the proximity and size of
 108 resident locations, with larger locations experiencing increased host movement
 109 and higher rates of infection [15]. *Kernel-based metapopulation models* (Fig. 1b)
 110 extend these models by including an explicit parameter for host movement (the
 111 mobility kernel, m , [16]) that specifies a proportion of hosts that change
 112 locations between time steps. The rate at which infections spread to susceptible
 113 nodes (S) is a function of the mobility kernel, the number of infected nodes (I)
 114 and the probability that each movement successfully spreads the infection (β^*):

$$\frac{dS}{dt} = -m\beta^*IS \quad \text{Eq. 1}$$

$$\frac{dI}{dt} = +m\beta^*IS \quad \text{Eq. 2}$$

115 Kernel-based metapopulation models have seen widespread application
 116 in disease ecology and have been extended to consider effects of habitat quality
 117 in resident locations [17,18], host phenotypic variation [19], and the presence of
 118 alternative hosts [20]. Simpler models assume a fixed rate of movement between

119 locations [11], or in proportion to the density of hosts in source locations [21].
120 However, Levy or random walks that characterize heterogeneities in movement
121 trajectories of individuals are increasingly applied [22]. *Coupled metapopulation*
122 *models* (Fig. 1c) incorporate within-location infection dynamics (e.g.,
123 transmission, recovery, births and deaths), and link these to the between-
124 location dynamics of host movement (m) and infection spread (β^*IS) [23].
125 Finally, while kernel-based and coupled metapopulation models track cohorts of
126 hosts that move over time, *individual-based (or agent-based) metapopulation*
127 *models* (Fig. 1d) have nodes that represent individuals, permitting tracking of the
128 movement and transmission of each individual host [24]. Individual-based
129 metapopulation models may uphold assumptions of homogenous mixing within
130 locations [25], though some agent-based models explicitly account for
131 heterogeneous contact rates within locations [26].

132 While many models do explicitly account for host movement, infection
133 spread *per se* is generally described in much simpler terms, typically as a
134 constant probability of infected hosts spreading infection (β^*). This
135 simplification overlooks the potential for infections to be acquired [1,12] or lost
136 [11,21], or hosts to die [27] while moving. Although models may accurately
137 reproduce spatial patterns of infection, ignoring the underlying mechanisms
138 driving those patterns do not allow extrapolation to predict disease spread
139 under alternative environmental scenarios. In subsequent sections, we consider
140 the consequences of relaxing these constraints.

141

142 **b) Empirical Studies**

143 Owing to the difficulty in determining the location and infection status of
144 moving hosts, many empirical approaches, such as mark-recapture (MR) surveys
145 and genetic analyses (Table S2), infer movement and infection spread from data
146 collected at resident locations. Ultimately, the lack of information on host
147 transience poses limitations that cannot be overcome without additional
148 approaches. For example, MR surveys of cliff swallows (*Petrochelidon*
149 *pyrrhonota*) showed that prevalence of parasites in swallow colonies rose with
150 increased arrivals by non-residents. However, colonies with the highest
151 prevalence were also those with the most nests [28], highlighting how the
152 contribution of movement to infection spread is difficult to disentangle from
153 within-location factors solely through MR. Correlations between host arrival
154 rates and prevalence may also reflect increases in susceptible hosts if many
155 arriving are uninfected [29]. Studies have also found weak [9] and even negative
156 associations between host arrival and infection prevalence, for example after fish
157 migrations [30].

158 Population genetics has revealed congruent patterns of gene flow
159 between hosts and parasites. These overlaps, which have been found for
160 parasites of both humans [31,32] and wildlife (reviewed by [33]), are considered
161 as evidence of the link between infection spread and host movement. Sampling
162 of rapidly evolving RNA viruses, which have generation times short relative to
163 the rate of host movement [34,35] have improved the temporal scale at which
164 genetic analyses can focus. Streicker et al. [35] used this approach to reconstruct
165 the recent spread of rabies in populations of vampire bats (*Desmodus rotundus*),
166 and higher rates of viral gene flow than maternally inherited bat genes suggested
167 male-biases in spread. Whereas the above techniques cannot distinguish

168 individual movements, Bayesian assignment tests, which use host and parasite
169 genotypes, allow for individual-based assessments of host movement between
170 resident locations [36]. Assignment tests have also proved useful for determining
171 how landscape features affect infection spread by impeding host movement [36],
172 but this technique is error prone [37]. Any genetic approach cannot reconstruct
173 the path travelled by, and infection status of, hosts during transience.

174 Biologging techniques, such as radio telemetry and GPS tags, can
175 overcome these issues by providing a more complete picture of host movement
176 [38]. Craft et al. [19] used GPS devices on nomadic and terrestrial lions (*Panthera*
177 *leo*) in a spatial network of prides in the Serengeti, which provided data for
178 disease simulations that explicitly included host transience. Other biologging
179 studies linked GPS locations to environmental data to assess effects of elevation
180 [39] and landscape structure [26] on infection spread. A key challenge of
181 biologging is acquiring infection data from hosts in transience. Capturing hosts to
182 obtain samples may be dangerous and disrupt natural movement behaviours. As
183 a result, remote tracking has provided detailed empirical data for modelling host
184 movement in host networks, but infection spread must be inferred [19]. In
185 addition, remote tracking is feasible for relatively few wildlife host-parasite
186 systems, and remains costly.

187 The long distances travelled by many migratory hosts allow researchers
188 to survey infections in hosts along different points in the migratory route, which
189 perhaps has provided the most insight into infection dynamics during host
190 transience (Table S2). Positive associations between host migration and spatial
191 expansion of infections have been reported [40]. However, reduced infection
192 prevalence among migrating animals have also been widely observed [7,30]

193 (Table S2), possibly due to increased mortality of infected hosts [7], avoidance of
194 infection through “migratory escape” [7], or recovery from infection while
195 moving [5]) (see Section 4 for further discussion). Direct quantification of any of
196 these processes in the wild is currently lacking.

197

198 **3. Framework for integrating host transience into spatial** 199 **network models of infection spread**

200 To better understand how transient phases of host movement factor into
201 spatial infection dynamics, we propose a framework that integrates concepts
202 from dispersal ecology and spatial disease modelling (Fig. 2a). We conceptualize
203 our framework as an individual-based metapopulation, but it could be applied to
204 any of the spatial network models shown in Fig. 1. Briefly, host movement
205 between spatially discrete locations is broken into three phases: departure,
206 transience, and arrival. While in transience, hosts can acquire infections
207 (transmission) or recover from infections (recovery), and all hosts are subject to
208 mortality, potentially at different rates for infected and uninfected hosts.

209 To illustrate mathematically the effect of these processes on host and
210 infection dynamics, and the factors affecting them, we describe the dynamics of a
211 cohort of moving hosts of size M , comprising I infected hosts and S uninfected
212 hosts ($M=S+I$). Here we used a simple host-microparasite framework [41], which
213 ignores the infection load of hosts, for ease of illustration. More complex, tailored
214 models could be developed as required. Host and infection dynamics during the
215 transient phase can be described by:

$$\frac{dM}{dt} = -M(d + I/M \alpha + v) \quad \text{Eq. 3}$$

$$\frac{dI}{dt} = \Lambda(M - I) - I(d + \alpha + v + \sigma) \quad \text{Eq. 4}$$

216 where d is the background host mortality rate, α is the parasite-induced host
 217 mortality rate, v is the host arrival rate at the recipient location (i.e., 1/duration
 218 spent moving) and σ is the host recovery rate from infection (for simplicity here,
 219 we assumed infected hosts recover to be susceptible to reinfection, but this could
 220 be relaxed). Finally, Λ represents the force of infection on susceptible individuals
 221 during the transient phase, and can take different forms depending on the
 222 transmission mode of the parasite. For example, for a parasite that undergoes
 223 direct transmission within the cohort of hosts, $\Lambda = \beta I$ (where β is the standard *per*
 224 *capita* transmission rate). However for a parasite that infects from a pre-existing
 225 environmental reservoir Λ will simply be a constant, reflecting the number of
 226 infectious stages in the environment encountered per unit time. Given this
 227 framework, the dynamics of hosts that successfully arrive at the recipient
 228 location (total: A ; infected: A_I) is given by:

$$\frac{dA}{dt} = vM \text{ and } \frac{dA_I}{dt} = vI \quad \text{Eq. 5}$$

229 such that the total number of individuals arriving ($A_{(\infty)}$) and number of infected
 230 individuals arriving ($A_{I(\infty)}$) is:

$$\frac{dA_{(\infty)}}{dt} = v \int_0^{\infty} M_{(t)} dt \text{ and } \frac{dA_{I(\infty)}}{dt} = v \int_0^{\infty} I_{(t)} dt. \quad \text{Eq. 6}$$

231 Example dynamics for this model are shown in Fig. 3. Using this general
 232 framework, models can be developed that are tailored to the dynamics of specific
 233 host-parasite systems while meeting logistical constraints or data limitations.

234 We emphasise that we do not aim here to provide a comprehensive analysis of
235 the dynamical properties of this model, which is beyond the scope of this review.
236 Instead, we present this framework to clarify the occurrence and connection of
237 the various processes that affect infection spread during host transience.

238 Importantly, the parameters in this framework are likely to be influenced
239 in different ways by host (H), parasite (P) and environmental (E) factors, and any
240 interactions between them. As such, these parameters should be considered as
241 functions, dependent on H , P and E ; for example:

$$d = f_d(H, E), \alpha = f_\alpha(H, P), v = f_v(H, E), \sigma = f_\sigma(H, P, E), \Lambda = f_\Lambda(H, P, E) \quad \text{Eq. 7}$$

242 We argue that closer attention to each of these functions and, ideally,
243 parameterising (at least some of) the host, parasite and environmental
244 dependencies within them, will lead to a clearer and more mechanistic
245 understanding of spatial host and infection dynamics than currently exists. In the
246 following sections we consider existing empirical evidence for these
247 dependencies, and highlight gaps where further information is required.

248

249 **4. Factors influencing transient phase infection dynamics**

250 **a) Recovery (σ) and relation to host arrival rate (v)**

251 Recovery from infections during host transience acts to decouple
252 infection spread from host movement. As a consequence, so called “structural
253 delay effects” [42], whereby parasite circulation predominantly occurs within
254 resident locations, may occur even in host networks highly connected by
255 movement. Since a given time period (on average $1/\sigma$ time units in our
256 framework) is required before recovery occurs [11], rates of recovery during

257 transience depend fundamentally on the amount of time the hosts spend in
258 transience (on average, $1/\nu$ time units). The duration of transience is at least in
259 part related to the linear distance travelled, and so simpler models may account
260 for variation in recovery rates by considering differences in movement distances.
261 Growing empirical evidence of infection recovery during long-distance seasonal
262 migrations (Table S2) [7] suggests that decoupling effects of host recovery are
263 particularly pronounced with longer linear distances. Substantial variation in the
264 direction and velocity of intergroup movements can also occur within
265 populations [49], so in many cases the time that hosts spend in transience may
266 not correspond to the linear distance travelled. Characterizing variation in
267 movement trajectories may therefore be important for parameterizing recovery
268 rates. Even if the time that hosts spend in transience is, on average, longer than
269 the infectious period, outlying cases of rapid movement or longer persistence of
270 infection may sustain infection spread between resident locations. Thus, the
271 degree of overlap in the variation in transient phase duration and infectious
272 period should more accurately estimate rates of spread throughout spatial host
273 networks.

274 Factors related to hosts and the environment that affect the time that
275 hosts spend in transience may influence rates of spread. For example,
276 behavioural responses to mitigate risks and costs of infection are well-
277 documented in wildlife and can be manifested through changes in host
278 movement patterns [44]. Landscape structure can also influence the duration of
279 host transience with implications for infection spread [45]. Behavioural and
280 landscape effects on host movement can be captured in our framework by

281 allowing arrival rates (ν) to vary with infection loads and/or the presence of
282 habitat features in the movement path.

283 Since most local movements between nearby resident locations are likely
284 too brief for infection recovery to occur, infection spread may be better
285 predicted by transmission during host transience or by characteristics of
286 resident locations (e.g. infection status [21], population size [1], spatial
287 arrangement [13]). Recovery should not be completely disregarded for local
288 dynamics, however. Abrupt changes in abiotic conditions that often occur when
289 entering transience could result in rapid recovery events, for example, when fish
290 move through saline waters [30,46]. Livestock lose ectoparasites during daily
291 ranging movements between woodlands (favourable for ticks) and pasture
292 (unfavourable for ticks), which modelling suggests can modulate infection
293 prevalence in the broader population (Fig. 2c) [47].

294

295 **b) Host mortality (background, d , or parasite-induced, α)**

296 Mortality of hosts during transience clearly will affect the number of hosts
297 that arrive (A). However, if infected hosts are differentially affected [via, for
298 example, increased pathogenic effects (α) during movement] host mortality
299 during transience will also affect the proportion of immigrants that carry
300 infections to the destination (A_i). This process may therefore inhibit parasite
301 persistence both through reductions in infection spread and reductions in
302 susceptible hosts available for infection in recipient locations. Experimental
303 work supports the hypothesis that infection-induced mortality is a mechanism
304 underlying observed decreases in protozoal infections with distance migrated by
305 monarch butterflies (*Danaus plexippus*, Fig. 2b) [48]. Immunological factors

306 should play a role in this process. Some species balance the energetic costs of
307 prolonged movement with immunosuppression [49], which clearly increases
308 infection risk, and likely mortality, during host transience. Alternatively,
309 adaptations that enhance immune function during periods of travel, particularly
310 tolerance responses that aid host survival without resulting in parasite clearance
311 [50] could facilitate infection spread. Such adaptations are evidenced by
312 migratory birds that experience immune activation when preparing to migrate
313 (Fig. 2d) [51] and by larger immune defences organs of migratory versus non-
314 migratory bird species [52].

315 In addition to host-related factors, both parasite-related factors (rate of
316 host exploitation) and environmental conditions, may also affect infection-
317 induced (α) and background (d) mortality rates of moving hosts at both local and
318 regional scales. Traversing habitats with unfavourable conditions (e.g. extreme
319 temperatures) or high densities of predators could drive host deaths during
320 transience, irrespective of the distance travelled. Similarly, infections from highly
321 virulent parasites acquired within source locations could conceivably
322 compromise host health to an extent that even modest energy expenditures
323 during local movement could cause death in transit.

324

325 **c) Force of Infection (Λ)**

326 In contrast to recovery and mortality, transmission during host transience
327 (either among moving hosts, at per capita rate β , or from the environment, at
328 rate Λ) generally facilitates infection spread among host networks. This process
329 therefore strengthens the link between infection spread and host movement but
330 weakens the link between spread and prevalence in source resident locations.

331 Since gains in infection are contingent on susceptible hosts encountering
332 infective stages, either from other infected hosts or in the environment, we
333 expect that the rate of acquisition of new infections during host transience is
334 most dependent on parasite transmission mode, the habitats traversed in the
335 transient phase, and the grouping patterns of moving hosts. For
336 environmentally-transmitted parasites, acquisition of infection during host
337 transience results when moving hosts traverse habitats supporting infective
338 stages. Primates typically acquire helminth infections during daily ranging [53],
339 and modelling suggests that transmission during local ranging of primate
340 individuals can allow parasites to invade and expand in their populations [54].
341 Acquisition of infection during host transience may also explain the apparent
342 importance of inter-burrow movement of pygmy blue-tongued lizards (*Tiliqua*
343 *adelaidensis*) for local infection spread (Fig. 2g) [9].

344 At broader scales, the epidemiological relevance of transmission during
345 host transience is well-illustrated by seasonal migrations of Saiga (*Saiga tatarica*,
346 [8]. Saiga acquire infections while moving through pastures with sheep faecal
347 matter that harbour infective nematode stages (Fig. 2f). For nematodes
348 therefore, spatial spread is contingent on transmission in Saiga during the
349 transient phase rather than transmission within resident locations [8],
350 emphasizing again how habitats traversed during host transience can factor into
351 spatial infection dynamics. Energy expenditure and immunosuppression during
352 regional movements may amplify transmission by activating infections from
353 dormant parasite stages. Outbreaks of latent bacterial (*Borrelia garinii*)
354 infections occurred in redwing thrushes (*Turdus iliacus*) when migratory
355 restlessness was induced (Fig. 2h) [55]. Activation of latent fungal infections

356 have also been reported in natterjack toads (*Epidalea calamita*) when moving
357 from terrestrial to aquatic habitats [56].

358 For vector-borne infections, transmission during host transience depends
359 on moving hosts encountering habitats favourable for vectors as well as the
360 parasites they harbour. Daily movements of humans can increase time in
361 habitats harbouring mosquito-borne dengue virus [57] and result in spatial
362 patterns of infection risk that diverge from those predicted by abundance of
363 mosquitoes in households [57]. These findings support the hypothesis that
364 exposure during host transience (captured by the force of infection parameter, Λ ,
365 in our framework) may decrease the influence of resident locations on patterns
366 of infection spread.

367 Grouped travel likely enhances transmission of directly-transmitted
368 parasites among moving hosts. Studies of shoaling movements in fish
369 demonstrate that parasitic infections can be transmitted in traveling groups [58].
370 Documentation of avian influenza virus transmission during stopovers along
371 bird migration routes lend further support for the potential of grouped travel to
372 promote transmission during host transience (Fig. 2i) [59]. Alternatively,
373 assortative grouping patterns could inhibit transmission among transient hosts
374 (i.e. migratory allopatry). Migration by juvenile pink salmon (*Oncorhynchus*
375 *gorbuscha*) prevents acquisition of infection through separation from infective
376 adults (Fig. 2e) [60]. This case is represented in our framework through a β
377 parameter equal to zero and would result in structural trapping of infection to
378 locations occupied by adult hosts.

379

380 **5. Future Direction**

381 This review highlights that obtaining field data on infection dynamics
382 during the transient phase of movement presents a key challenge to
383 understanding the mechanistic links of host movement and infection spread.
384 Owing to recent innovations of tracking and computational technology that
385 permit detailed individual-based tracking of wildlife systems [38], we argue that
386 collection of such data is now feasible for some wildlife systems. Utilization of
387 automated image-based tracking methods [69] allows ecologists to characterize
388 at high resolutions the behavioural patterns of infected and uninfected hosts in
389 controlled environments that mimic transient phases. These approaches also
390 provide the opportunity to quantify effects of host grouping on transmission
391 during transient phases. A key advantage of these experimental approaches is
392 the feasibility of monitoring changes in infections in individual hosts at fine
393 temporal scales, which can be directly linked to environmental conditions and
394 host behaviours. Nevertheless, owing to costs and logistical constraints, image-
395 based tracking is typically performed in small experimental units. Distinguishing
396 departure, transience and arrival in small units can be problematic. Future effort
397 can be made to develop larger experimental tracking systems, such as
398 mesocosms, capable of capturing all phases of hosts movement and infection
399 spread.

400 The radio-tracking and GPS studies highlighted above [19,39,61] are
401 strong initial attempts at directly quantifying transient phase host movements in
402 the wild. Future work can improve on these approaches by combining movement
403 paths with individual infection data at multiple points during transience. Doing
404 so can better identify factors that decouple rates of infection spread from linear
405 host movement assumed in conventional models, which might resolve

406 unexpected and inconsistent findings of prior work [9,19]. For organisms that
407 cannot be feasibly surveyed for infection during transient phases, biologging
408 devices may be developed that remotely assay infection status of moving hosts in
409 the wild. This could also be done indirectly. Since immune function in
410 ectothermic animals is strongly linked to body temperature, fitting migratory
411 ectotherms such as amphibians and snakes with temperature sensors may
412 provide insights into how host susceptibility varies during periods of movement.
413 For larger-bodied mammals, GPS devices combined with accelerometers can
414 identify critical periods of movement during which increased energy
415 expenditure poses heightened infection risk [38].

416 Considering the importance of the structure and abiotic conditions of the
417 habitat matrix surrounding resident locations for transient phase infection
418 dynamics, approaches used by landscape epidemiologists can benefit spatial
419 network models of infection spread. Landscape epidemiologists apply
420 environmental data from satellite imagery to identify the habitats in which
421 diseases proliferate. Integrating habitat data into metapopulation models has
422 been carried out extensively [45,62,63], but models have typically only
423 considered effects of habitat on host movement. Future work can advance by
424 considering realistic effects that differential quality of habitats in the matrix have
425 on transmission and host recovery during periods of movement [17,18].
426 Additionally, the coarse resolution of much environmental data used in
427 landscape epidemiological studies limits the utility of these data to regional
428 movements such as migrations and dispersal. Local scale heterogeneities in
429 external conditions (e.g. moisture levels [64], vegetation cover [65], temperature
430 [64,66], predation risk [67]) are known to affect infection risk and prevalence

431 and may also affect host infections during local movements. Experiments that
432 manipulate habitat can complement landscape ecological approaches by testing
433 how movement through the habitat matrix alters courses of infection within
434 hosts. In addition, field and experimental data on the abundance and persistence
435 of parasite infective stages and/or infection vectors in the habitat matrix can
436 inform parameterization of rates of environmental transmission in transient
437 hosts. Theoretical work has begun to use these types of data to explore infection
438 dynamics in single locations [68], and our framework can guide spatially explicit
439 extensions of these models that distinguish environmental transmission rates at
440 each phase of host movement. Finally, human alteration of habitats comprising
441 host networks, while posing various potentially detrimental consequences for
442 population viability, may afford natural experiments for testing the abiotic
443 factors involved in transience phase infection dynamics. Satterfield et al. [70]
444 were able to use human-mediated amplification of exotic milkweed (*Asclepias*
445 *curassavica*) in the United States, a preferred breeding and nutrient resource of
446 monarch butterflies, to model how loss of migratory behaviour in monarch
447 populations caused by year-round resource availability altered population-level
448 infection dynamics. Human activities that alter the habitats spanning spatial host
449 networks may allow ecologists to measure the effects of habitat structure,
450 temperature, moisture and other abiotic variables on infection in transient hosts.
451 Such data would enhance the ability to predict patterns of disease spread amid
452 environmental change.

453

454 **6. Conclusion**

455 Identification of relevant biological processes is the first step in building
456 mechanistic models of ecological dynamics. With an explicit transient phase, our
457 conceptual framework unpacks infection spread into its constituent biological
458 processes: transmission, infection recovery, and infection-induced mortality. In
459 so doing, our framework links patterns of infection spread described by existing
460 spatial models to specific mechanisms that otherwise are hidden in their
461 assumptions. While our framework can be simplified as needed, evidence of
462 these processes from the empirical studies reviewed here provides a strong
463 rationale for building this added complexity into disease models. Owing to
464 technological developments, movement ecology is experiencing an exciting
465 renaissance of big data that is affording new insights in the mechanisms driving
466 animal movements as well as their ecological consequences. These developments
467 provide equally exciting opportunities for disease ecologists to advance our
468 understanding of the consequences of host movement for infection spread, the
469 factors that determine those consequences, and how to model spatial infection
470 dynamics.

471

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482 DD and AM conceived of review. All authors assisted in formulating the
483 conceptual approach. AF developed the mathematical framework. DD wrote the
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485

486 **Competing interests**

487 The authors have no competing interests

488

489 **References**

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- 703

704 **Tables and Figures**

705

706 Fig. 1. Metapopulation-based spatial disease models track locations of hosts
707 and either simulate infection spread based on connectivity measures
708 without explicitly considering host movement (a) or define proportion of
709 hosts change locations between time steps (white arrow) with infection
710 spread occurring from a proportion of hosts that change from infected
711 locations to susceptible locations (b, red arrow). *Coupled metapopulation*
712 *models* link local processes such as transmission (thin red arrow) to the
713 between-location processes of host movement and infection spread (c).
714 *Individual-based network models* track movements of each host (denoted by
715 subscripts i,j) (d).

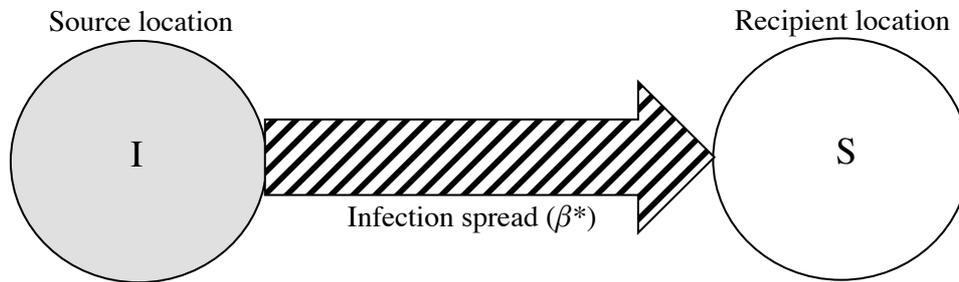
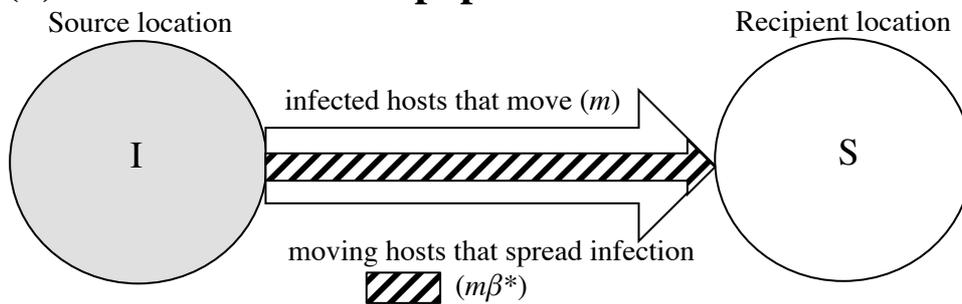
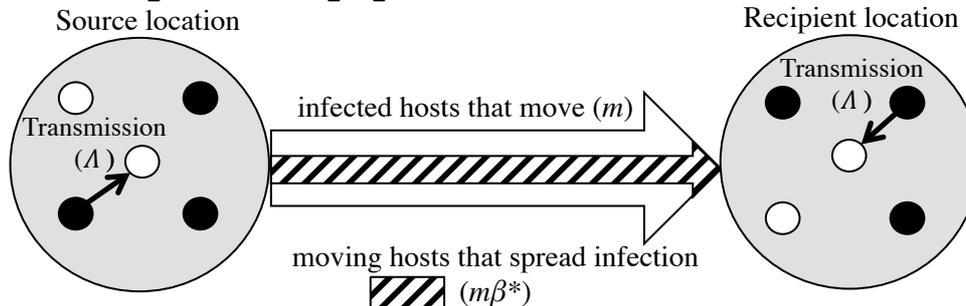
716

717 Fig. 2a.) Framework for capturing transient phase infection dynamics. The
718 movement path of hosts and their infections (intensity/probability
719 represented by shading of arrow with darker red being higher
720 intensity/probability) are categorized into three phases: departure,
721 transience and arrival. During transience, infections are lost/reduced
722 through background or disease-induced mortality of infected hosts, or as
723 conditions during transience decrease exposure and/or cause deterioration
724 of infections (i.e. recovery). Mechanisms that drive recovery include: (b-c)
725 movement through habitats unsuitable for infections, which may occur with
726 protozoal infections during monarch butterfly migrations [6] and with tick
727 infections during ranging movements of livestock [47]; (d) enhancement of
728 immune function during periods of movement, which may occur in

729 migratory red knots [51]; (e) dispersion of hosts that reduces contact, as
730 evidenced by sea lice infections in migratory pink salmon [60]. Mechanisms
731 that increase the force of infection during transience include: (g-f)
732 movement through habitats with viable infective stages, which occurs with
733 parasitic nematodes in migratory saiga [8] and dispersing pygmy blue
734 tongue lizards [9]; (h) immunosuppression, such as the proliferation of
735 latent bacterial infections in migratory redwing thrushes [55]; and (i) host
736 aggregation, which occurs with Avian Influenza Virus (AIV) infections
737 during stopovers by migrating sandpipers [59].

738

739 Figure 3. Dynamics of the total number of hosts and the number of infecteds
740 during the transient moving phase as predicted from a mathematical model,
741 assuming parasite transmission from the environment. (a) total number of
742 individuals (M) and number of infected individuals (I) undergoing transient
743 movement through time. (b) cumulative total number of individuals (A) and
744 number of infected individuals arriving at the destination location through
745 time (A_I). We emphasise this figure is for illustrative purposes only, created
746 using arbitrary parameter values that do not relate to values from any
747 particular empirical system ($d=1, \alpha=0.1, \Lambda=1, \sigma=0.1, \nu=0.2$).

(a) Phenomenological metapopulation**(b) Kernel-based metapopulation****(c) Coupled metapopulation****(d) Individual-based metapopulation**