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► **To cite this version:**

Marieke Jesse, Hans Heesterbeek. Divide and conquer? persistence of infectious agents in spatial metapopulations of hosts. *Journal of Theoretical Biology*, 2011, 275 (1), pp.12. 10.1016/j.jtbi.2011.01.032 . hal-00676094

**HAL Id: hal-00676094**

**<https://hal.science/hal-00676094>**

Submitted on 3 Mar 2012

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## Author's Accepted Manuscript

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PII: S0022-5193(11)00044-0  
DOI: doi:10.1016/j.jtbi.2011.01.032  
Reference: YJTBI6344

To appear in: *Journal of Theoretical Biology*

Received date: 12 July 2010  
Revised date: 17 January 2011  
Accepted date: 20 January 2011

Cite this article as: Marieke Jesse and Hans Heesterbeek, Divide and conquer? persistence of infectious agents in spatial metapopulations of hosts, *Journal of Theoretical Biology*, doi:[10.1016/j.jtbi.2011.01.032](https://doi.org/10.1016/j.jtbi.2011.01.032)

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Accepted manuscript

18 Divide and conquer? Persistence of infectious agents in  
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23 **Abstract**

Persistence of an infectious agent in a population is an important issue in epidemiology. It is assumed that spatially fragmenting a population of hosts increases the probability of persistence of an infectious agent and that movement of hosts between the patches is vital for that. The influence of migration on persistence is however often studied in mean-field models, whereas in reality the actual distance travelled can be limited and influence the movement dynamics. We use a stochastic model, where within- and between-patch dynamics are coupled and movement is modelled explicitly, to show that explicit consideration of movement distance makes the relation between persistence of infectious agents and the metapopulation structure of its hosts less straightforward than previously thought. We show that the probability of persistence is largest at an intermediate movement distance of the host and that spatially fragmenting a population of hosts is not necessarily beneficial for persistence.

24 *Key words:* stochastic model, migration, movement distance, patch  
25 neighbourhood, lattice

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26 **Introduction**

27 Once an infectious disease invades a population, it can go extinct immediately,  
28 fade out after an epidemic or persist in the population. In homogeneously mixing  
29 populations, infectious agents tend to die out when the host population size is

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30 below a critical community size (Bartlett, 1957; Grenfell & Harwood, 1997),  
31 related to the demographic processes that regulate the inflow of new susceptible  
32 hosts in the population (Anderson & May, 1992). However, populations are  
33 often structured in space and then the situation becomes much more subtle.  
34 Examples are humans living in cities and towns (Hall et al., 2007), cattle on  
35 farms (Le Menach et al., 2005; Kao et al., 2007) or wildlife populations, for  
36 example the water voles in the U.K. (Telfer et al., 2001), the badgers in the  
37 U.K. (Rogers et al., 1998) or the great gerbils in Kazakhstan (Davis et al.,  
38 2007a). These populations live in a set of spatially separated patches of suitable  
39 habitat, connected to each other by migration of the individuals (e.g. travel of  
40 humans between cities, purchasing and selling of cattle and juvenile migration of  
41 wildlife). Each patch moreover, has its own dynamics influenced by the incoming  
42 and outgoing migrants and by the local conditions. Such a population is called  
43 a (spatial) metapopulation (Hanski, 1999; Leibold et al., 2004). The patch size  
44 measured is the size of the inhabiting population of interest and ranges from  
45 very large (e.g. cities, farms), to very small (for example wildlife often lives in  
46 small (family)groups).

47 The structure of such metapopulations for a given species is distinct from sin-  
48 gle homogeneously mixing populations, resulting in differences in dynamics and  
49 persistence of the species itself, and other species it interacts with. For exam-  
50 ple, infectious disease agents both influence and are influenced by the structure  
51 and migration of their host (Grenfell & Harwood, 1997). Hanski (1999) showed  
52 that, if the total population size is below the critical community size, then spa-  
53 tially fragmenting this population has a positive influence on the ability of an  
54 infectious agent to persist and that notably movement of hosts between patches  
55 is crucial. The latter aspect has received substantial attention in the literature  
56 (e.g. Swinton et al., 1998; Keeling, 2000; Park et al., 2002; Hagenaars et al.,  
57 2004; Lindholm & Britton, 2007).

58 However, there is a much broader range of aspects that influence and con-  
59 trol persistence in a metapopulation (Swinton et al., 1998): (i) demographic  
60 aspects: birth and death; (ii) epidemic aspects: length of infectious period and

61 the specifics of the transmission process; (iii) spatial aspects: movement distance  
62 of the host and frequency of migration or length of stay in patches. Notably the  
63 interaction between these aspects appears to be important, and is not well un-  
64 derstood. Indeed, seemingly new phenomena arise from analysis of real systems  
65 (Davis et al., 2007b, 2008). In this theoretical paper we aim to contribute to  
66 this debate and explore the question: how does the interaction between all the  
67 aspects mentioned shape persistence of an infectious disease agent in a spatially  
68 structured host population with movement modelled explicitly?

69 For studying persistence in a metapopulation often a mean-field approxima-  
70 tion is used (Gog et al., 2002; McCallum & Dobson, 2002; Jesse et al., 2008),  
71 but here we introduce a (preferred) movement distance of the host: hosts are  
72 only allowed to migrate to patches within this movement distance. The set of  
73 patches that can be reached by individual hosts migrating out of a given patch  
74 within one time step is seen as the neighbourhood of that patch. The effect of  
75 the neighbourhood of a patch on persistence has been studied both in an eco-  
76 logical setting (Durrett & Levin, 1994; Hanski & Ovaskainen, 2000; Vuilleumier  
77 et al., 2007) and in an epidemiological context (Levin & Durrett, 1996; Rhodes  
78 & Anderson, 1996).

79 Keeling (2000) pointed out that there are two ways of coupling between  
80 subpopulations. The first is based on movement rate of individuals between  
81 subpopulations, which is the coupling we use in the present paper. The second  
82 is by using a parameter that describes how much the global average affects  
83 the dynamics of a subpopulation. Keeling used the latter way of coupling and  
84 concluded that persistence is maximized at intermediate levels of coupling (in  
85 the second interpretation), but expects the qualitative behaviour of both ways  
86 of coupling to be similar. In this paper we show that using a movement rate and  
87 in addition a movement distance indeed results in persistence being maximized  
88 at intermediate levels of coupling.

89 Here, we take a much broader view by regarding an epidemiological model  
90 with demography, explicit modelling of movement (distance and frequency of  
91 movement) and coupling of between- and within-patch dynamics. Among other

92 results, we show that there can be an optimum for persistence at intermediate  
 93 neighbourhood size. We explore the reasons behind this and how persistence  
 94 of an infectious agent is influenced by the demographic, epidemic and spatial  
 95 parameters. Moreover, we show that, in contrast to what was generally believed,  
 96 a metapopulation is not necessarily more beneficial for the persistence of an  
 97 infectious agent compared to a single homogeneously mixing population.

## 98 **Methods**

### 99 *Model*

100 The model describes a host population divided into subpopulations, each inhab-  
 101 iting a patch with suitable habitat (details in Jesse et al., 2008). In contrast to  
 102 Jesse et al. (2008) we now consider a spatially explicit metapopulation, where  
 103 patches are structured in space, in our case on a regular square lattice. At least  
 104 initially there are no unoccupied patches and the patches are identical in the  
 105 sense that all parameter values are the same for all subpopulations. An overview  
 106 of the parameters and their default values used in the numerically studied cases  
 107 is given in Table 1.

108 In each patch there is homogeneously mixing and *SIR*-type dynamics oc-  
 109 curs, with hosts being in one of the three following states: susceptible (*S*),  
 110 infectious (*I*) or recovered/immune to the infection (*R*). The infectious disease  
 111 is transmitted directly and hosts do not lose immunity, but eventually die and  
 112 are replaced by susceptibles. Births are locally density dependent, meaning that  
 113 they depend on the number of hosts present in that patch.

Parameter	Value	Definition
$P$	100	number of patches
$K$	10	carrying capacity
$b$	0.2	birth rate per week
$\mu$	0.01	natural mortality rate per week
$m$	[0, 1]	movement rate per week
$\beta$	10	transmission rate per week
$\gamma$	0.5	recovery rate per week

Table 1: Definition and default values of the model parameters for numerically studied cases.

Random variable	Distribution	Parameter	Definition
$\mathbf{B}(N_x, t)$	Poisson	$\max[0; bN_x(t)(1 - \frac{N_x(t)}{K})]$	# births
$\mathbf{D}(Z_x, t)$	Binomial	$\mu$	# deaths
$\mathbf{Inf}(S_x, t)$	Binomial	$1 - \exp(-\frac{\beta I_x(t)}{N_x(t)})$	# infecteds
$\mathbf{Rec}(I_x, t)$	Binomial	$\gamma$	# recovereds
$\mathbf{M}_{\text{out}}(Z_x, t)$	Binomial	$m$	# emigrants
$\mathbf{M}_{\text{in},Z}(x, t)$	Multinomial	$1/P$	# immigrants

Table 2: The random variables with their distribution and parameters. The state is given explicitly or by  $Z$  with  $Z \in \{S, I, R\}$ .

114 The model is stochastic and discrete in time, one time step denotes one  
 115 week, where  $S_x(t)$ ,  $I_x(t)$  and  $R_x(t)$  denote the number of hosts per infectious  
 116 state in patch  $x$  at time  $t$ . The total number of hosts in patch  $x$  at time  $t$  is  
 117 given by  $N_x(t)$ . Events occur successively in the interval  $[t, t+1)$  in the order of  
 118 birth, death, infection, recovery and migration. Of course, ordering the events  
 119 is artificial, but it is necessary in a discrete model so that in the bookkeeping  
 120 individual-level events, such as dying or recovering, are taken into account in a  
 121 consistent way. The complete set of stochastic difference equations is given by:

$$\begin{aligned}
 S_x(t+1) &= S_x(t) - \mathbf{D}(S_x, t) - \mathbf{Inf}(S_x, t) + \mathbf{B}(N_x, t) - \mathbf{M}_{\text{out}}(S_x, t) + \mathbf{M}_{\text{in},S}(x, t) \\
 I_x(t+1) &= I_x(t) - \mathbf{D}(I_x, t) + \mathbf{Inf}(S_x, t) - \mathbf{Rec}(I_x, t) - \mathbf{M}_{\text{out}}(I_x, t) + \mathbf{M}_{\text{in},I}(x, t) \\
 R_x(t+1) &= R_x(t) - \mathbf{D}(R_x, t) + \mathbf{Rec}(I_x, t) - \mathbf{M}_{\text{out}}(R_x, t) + \mathbf{M}_{\text{in},R}(x, t)
 \end{aligned}$$

122 The random variables and their distributions (Table 2) are calculated with  
 123 the number of hosts at that point in the time interval, using the order of the  
 124 events; for example, the number of newly infected hosts at time interval  $[t, t+1)$   
 125 depends on the number of infectious hosts at time  $t-1$  minus the number of  
 126 infectious hosts that died at time  $t$ .

### 127 *Structure*

128 Above we described the dynamics within each patch, but these patches are con-  
 129 nected to each other via migration of the hosts, which we also model explicitly.  
 130 Where in Jesse et al. (2008) a mean-field approximation was used, here a finite  
 131 spatially explicit structure is looked at. The patches to which a host can mi-  
 132 grate, are given by the adjacency matrix  $A$ . This adjacency matrix describes



133 the connections between the patches of the metapopulation: if  $A_{ij} = 1$  it is  
 134 possible to move from patch  $i$  to patch  $j$ , otherwise  $A_{ij} = 0$ . We view the  
 135 metapopulation of patches as being arranged spatially in a finite regular lattice  
 136 where a node is a patch and where an edge indicates a connection in the above  
 137 sense. The network is non-directed, which means that if movement is possible  
 138 from patch  $i$  to patch  $j$ , then it is also possible from patch  $j$  to patch  $i$ , i.e.  
 139  $A_{ij} = A_{ji}$ .

Of particular interest is the movement distance of the hosts, measured as path length in the lattice. For movement distance  $d$ , a host can migrate along at maximum  $d$  edges in the regular lattice of patches. The host can, with equal probability, migrate to all patches that can be reached within this number of edges. This movement distance can be seen as the capacity of the host to migrate to patches at that distance. When the movement distance equals 1, hosts can only migrate to their nearest neighbours and when the movement distance is large enough, hosts can move to every patch. The matrix  $M$  gives the patches to which a host can move within a movement distance  $d$  and has entries such that, for  $i \neq j$ :

$$M_{ij} = \begin{cases} 1 & \text{if patch } i \text{ and } j \text{ are connected, i.e. } (\sum_d A^d)_{ij} > 0 \\ 0 & \text{otherwise} \end{cases},$$

140 and  $M_{ii} = 0$ , preventing hosts from migrating back immediately to the patch  
 141 they just left (i.e. no loops of length 1 are allowed). The matrix  $M$  is calculated  
 142 by means of  $\sum_d A^d$ , where  $A_{ij}^d$  gives the number of paths of length  $d$  from patch  
 143  $i$  to patch  $j$ .

#### 144 *Simulations*

145 The model is stochastic and hence there can be large variation between runs of  
 146 the model, even though the set of parameter values is identical. The stochastic  
 147 difference equations were simulated for a range of parameter values. The pro-  
 148 gram is written in R (package 2.10.1, [www.cran.r-project.org](http://www.cran.r-project.org)). At time  $t = 1$  one  
 149 infectious host is introduced into one patch in an otherwise completely suscepti-  
 150 ble metapopulation. For the results the location of this (index) patch, where the

151 first infectious host is introduced, does not matter. This coincides with findings  
152 of Pautasso & Jeger (2008), when they studied a model with *SI*-type dynamics  
153 on a directed network, where each node represents an individual.

154 After introduction of this one infectious host in the metapopulation, there  
155 are four possible situations: the infectious agent may (i) not spread at all in  
156 the initial patch, (ii) spread in the initial patch, but not between patches (iii)  
157 spread within and between patches and then fade out or (iv) spread within and  
158 between patches and (quasi)persist in the metapopulation (Jesse et al., 2008).  
159 Here, we are interested in the fourth case, persistence in the metapopulation.

160 We defined the infectious agent to be persistent in a simulation if it is still  
161 present in the population after twice the expected life span of the host. The  
162 expected life span of a host is  $1/\mu$ , therefore the simulations were run for  $2/\mu$   
163 time steps. The choice of twice the expected life span is arbitrary, but within  
164 this number of time steps the infectious agent has survived two generations of  
165 hosts, and has spread between the patches.

166 The results obtained are from 1000 simulations. This number of simulations  
167 per datapoint was chosen such that variation between several runs of 1000 sim-  
168 ulations for the same set of parameter values is small. Therefore, increasing the  
169 number of simulations smoothens the graphs depicting our results, but does not  
170 alter or add to conclusions we draw.

171 The default parameter values are shown in Table 1. The default death rate  
172 equals 0.01, meaning that the life span of a host is on average 100 weeks, i.e. a  
173 host lives around two years. And the default recovery rate equals 0.5, so that  
174 a host is infectious for on average two weeks. The metapopulation consists of  
175 100 patches, all with carrying capacity 10. This carrying capacity is chosen  
176 such that persistence of the infectious agent in a single patch is not possible.  
177 In a single homogeneously mixing population of size 1500 around 90% of the  
178 simulations will result in a persisting infection for the default parameter values.  
179 The metapopulation is arranged on a square  $10 \times 10$  lattice, with reflective  
180 boundaries. These boundaries can be considered as natural boundaries such as

181 water or mountains, which cannot be crossed by the host.

## 182 **Results and Conclusions**

183 In this section we will first present the influence of the spatial aspects on persis-  
184 tence of infectious agents in the metapopulation. Then the demographical and  
185 epidemiological aspects will be presented.

### 186 *Spatial aspect*

187 In Figure 1a, the influence of the spatial aspects on persistence of the infectious  
188 agent are explored. In this figure, contour lines show the fractions of simulations  
189 with persisting infection as functions of the migration rate and the movement  
190 distance of the host. Fixing the movement distance and then increasing the  
191 migration rate resulted in a higher fraction of simulations with persisting in-  
192 fection. A higher migration rate means that hosts spend a shorter time in one  
193 patch and thus change patches more frequently. By changing patches more fre-  
194 quently, hosts increase the number of contacts with other hosts and therefore  
195 have a higher probability of finding sufficient susceptible hosts to infect while  
196 they are still infectious.

197 Fixing the migration rate and increasing the movement distance shows a  
198 maximum in the fraction of simulations with persisting infection at intermediate  
199 movement distance. This result is also shown in Figure 1b for four different  
200 migration rates. Clearly there is a peak at intermediate movement distance,  
201 except for relatively low migration rates (a migration rate of 0.2 means that a  
202 host moves once every 5 weeks). The default infectious period is two weeks, so  
203 at low migration rates the host spends, on average, most of its entire infectious  
204 period within one patch. Since the patch size is too low for persistence, there is  
205 only a very small fraction of simulations showing persistence.

### 206 *Effect of movement distance on patch level*

207 As already shown, varying only the movement distance already results in dis-  
208 similarities between the fraction of simulations with persisting infection. In

209 order to understand where these dissimilarities come from, we focus on the dif-  
210 ferences that occur on patch level. Because the infectious hosts are the key for  
211 persistence of the infectious agent in the metapopulation, we focused on some  
212 properties of patches with respect to infectious hosts that might be influenced  
213 by the movement distance:

- 214 i) the total number of time steps (not necessarily consecutive) that a patch  
215 contains at least one infectious host;
- 216 ii) the number of times that a patch gets infected, i.e. how often are there  
217 transitions from no infectious host in a patch to at least one infectious host  
218 in that patch;
- 219 iii) the total number of infectious hosts per patch, i.e. the sum of the number  
220 of infectious hosts at each time step in a patch;
- 221 iv) the number of consecutive time steps a patch contains an infectious host.

222 Only simulations with persisting infection were taken into account when calcu-  
223 lating the above measures. The results are presented as box-and-whisker-plots  
224 in Figure 2.

225 It is immediately clear that the medians for movement distance 6 and 14 are  
226 approximately the same for all four measures, but that the range differs: the  
227 variation between patches for intermediate movement distance is larger than  
228 for large movement distance. Movement distance 14 corresponds to a situation  
229 where the patches are almost fully connected and the system starts behaving  
230 like one single homogeneously mixing population.

231 On average a patch contains 35 out of the 200 time steps (17.5% of the time)  
232 at least one infectious host for low movement distance (Fig. 2a). The average  
233 number for the other movement distances is slightly higher. The number of  
234 transitions from no infectious hosts in a patch to at least one is the lowest  
235 for the low movement distance (median 15 times, Fig. 2b). This might be  
236 due to (infectious) hosts returning more often back to a patch they visited  
237 just a few time steps ago (fewer patches to choose from). On average the

238 total number of infectious hosts per patch is about 1.5 times larger for low  
239 movement distance (median of 120) than for the other two movement distances  
240 (Fig. 2c). Because the number of infected patches at each time step is equal  
241 for all movement distances, the number of infectious hosts in each patch must  
242 be higher for low movement distance than for the other movement distances.  
243 Again, this is probably caused by hosts returning more often to a patch they just  
244 visited at low maximum movement distances. Finally in Figure 2d the median  
245 number of consecutive time steps a patch is inhabited by infectious hosts is  
246 shown. Basically, this is just the total number of time steps a patch is infected  
247 divided by the number of transitions from having no infectious host in a patch  
248 to having at least one. Here, the low movement distance shows that a patch  
249 contains at least one infectious host for a longer uninterrupted number of time  
250 steps.

251 With the four measures we looked at, we can conclude that at low movement  
252 distance, patches are on average longer infected and contain over the whole time  
253 period more infectious hosts than at large movement distance. This implies that  
254 the infectious agent remains within the same area at low movement distance  
255 until that area runs too low on available susceptible hosts. At large movement  
256 distance on the other hand, the infectious agent is more scattered throughout the  
257 whole metapopulation, patches get (re)infected more often, but it is for a short  
258 period of time and with only a few infectious hosts. Hence, at low movement  
259 distance the spreading of the infectious agent occurs more locally and gradually  
260 becomes more global when the movement distance increases.

#### 261 *Demographical and epidemiological aspects*

262 As mentioned earlier, persistence of an infectious agent is also influenced by  
263 demographical and epidemiological aspects. The fraction of simulations with  
264 persisting infection is therefore studied as function of each of the four parameters  
265 controlling these aspects: birth rate, death rate, transmission rate and recovery  
266 rate. The fraction is shown in Figure 3 for three different movement distances,  
267 namely 2, 6 and 14, and for a single homogeneously mixing population.

268 This single homogeneously mixing population consists of one patch with a  
269 carrying capacity  $K$  of 1000 hosts; the total size of the metapopulation and the  
270 single population are therefore equal. Moreover, this figure shows how a change  
271 in a relevant parameter influences the fraction of simulations with persisting  
272 infection. For all parameters, the fraction of simulations with persisting infection  
273 is the highest for intermediate movement distance, corresponding to the previous  
274 result that there is a maximum in the fraction of simulations with persisting  
275 infection at this movement distance.

276 The natural death rate (i.e. not related to the infection) determines the  
277 life span of a host and influences also, because of the density-dependent birth  
278 rate, the population turnover: a lower death rate implies a longer life span and  
279 therefore a slower population turnover; i.e. a slower inflow of new susceptible  
280 hosts. The life span is used to define when an infectious agent is persistent  
281 in our set up, so varying the death rate means that the number of time steps  
282 before an infectious agent is called persistent varies as well. Increasing the death  
283 rate, i.e. increasing population turnover, has a positive effect on the fraction  
284 of simulations with persisting infection (Fig. 3a). At a low death rate, while  
285 the speed by which the infectious agent spreads through the whole population  
286 remains equal, it takes longer for new susceptible hosts to arise. This time gap is  
287 more difficult to bridge in a single homogeneously mixing population, where all  
288 susceptible hosts are immediately available for contact to the infectious agent.  
289 Therefore, a low death rate is more beneficial for persistence in a metapopulation  
290 than in a single homogeneously mixing population.

291 The birth rate in this model is density dependent; a high birth rate implies  
292 that each subpopulation is around carrying capacity and dead hosts are replaced  
293 by susceptible new-borns quickly. Hence, increasing the birth rate also increases  
294 the fraction of simulations with persisting infection (Fig. 3b). At low birth rate,  
295 a single homogeneously mixing population is more beneficial for the infectious  
296 agent, because the few susceptible hosts that are born per unit of time are found  
297 quickly, while in the metapopulation an infectious host needs time to find the  
298 susceptible hosts left. When the birth rate is large, susceptible hosts arise faster

299 and are immediately available for contact with infectious hosts in case of a single  
300 homogeneously mixing population. This could mean that the infectious agent  
301 ‘burns through’ the available susceptibles too fast to allow persistence. It is  
302 then advantageous for the infectious agent to be in the metapopulation: not all  
303 susceptible hosts are immediately available for contact, allowing the infectious  
304 agent to persist in the population for a longer time (i.e. the risk of fade out is  
305 spread).

306 In Figure 3c the fraction of simulations with persisting infection is shown for  
307 various transmission rates. For low transmission rates the fraction of simulations  
308 with persisting infection is higher in a metapopulation than in a single homoge-  
309 neously mixing population, but the opposite holds at high transmission rates.  
310 In a single homogeneously mixing population an infectious host has immediate  
311 access to all susceptible hosts. At high transmission rate, this availability of all  
312 susceptibles in a single homogeneously mixing population is beneficial for the  
313 infectious agent, because even when there are only a few susceptibles left, there  
314 is a high probability that they will be contacted. In a metapopulation on the  
315 other hand, an infectious host needs time to travel to patches where the few  
316 susceptibles remain, and before successful contact has been made this infectious  
317 host may have died or recovered. At low transmission rate, the above situations  
318 are reversed.

319 The recovery rate determines the infectious period of a host and Figure 3d  
320 shows that the fraction of simulations with persisting infection as a function of  
321 recovery rate is hardly influenced by the movement distance of the host. When  
322 the recovery rate is large, the infectious period is short and, on average, fewer  
323 infectious hosts leave the patch during the infectious period, making it more  
324 difficult for the infectious agent to spread to other patches. The movement  
325 distance does not have a large influence on this, because the ability to spread is  
326 here, in the first instance, mostly determined by the chance to leave the patch  
327 at all.

328 Furthermore, a short infectious period results in a population of many re-  
329 covered hosts. In Figure 3d, the fraction of simulations with persisting infection

330 coincides for both the single homogeneously mixing population and a metapop-  
331 ulation where hosts have an intermediate movement distance. However, this  
332 fraction is very much influenced by the migration rate of the host (not shown).  
333 Persistence of the infectious agent is more difficult at low migration rate, because  
334 the infectious host does not change patches frequently enough to sustain the in-  
335 fectious agent. A high migration rate in a metapopulation performs slightly  
336 better than a single homogeneously mixing population, because running out of  
337 susceptible hosts happens faster in a single population than in a metapopulation.

338 Finally, both the shape of the lattice and the number of patches do not  
339 qualitatively change the results (not shown). Reshaping the lattice, for example  
340 into  $4 \times 25$  with the same total population size as the  $10 \times 10$  case, still gives  
341 a peak at the same intermediate movement distance of 6. Interestingly, the  
342 mean distance of one patch to any other patch is for the  $4 \times 25$  lattice 9.2  
343 steps and for the  $10 \times 10$  lattice 6.6. Increasing the number of patches to, for  
344 example, a  $12 \times 12$  lattice also gives a peak at intermediate movement distance.  
345 The latter peak occurs at a slightly higher movement distance compared to the  
346 above cases, however, the  $12 \times 12$  case is not directly comparable, because both  
347 the total population size and the mean distance from one patch to any other  
348 is larger. Thus the size of the lattice does not appear to essentially change the  
349 results.

#### 350 *Balance*

351 Persistence of an infectious agent in a metapopulation results from a balance  
352 between the spatial, demographical and epidemiological components. We stud-  
353 ied the components in isolation above, but now look at their combined effect  
354 (Fig. 4).

355 Each panel in this figure shows curves leading to the same fraction of simu-  
356 lations with persisting infection ('isopersistence level') as function of the death  
357 and the transmission rate. The isopersistence levels are shown for low, interme-  
358 diate and high movement distance in a metapopulation and for a single homo-  
359 geneously mixing population and for the four parameter combinations low/high



360 migration rate and low/high birth rate. For all four subfigures the default re-  
361covery rate is used, because Figure 3d showed that for the various movement  
362distances differences in the fraction of simulations with persisting infection are  
363not strongly affected by the recovery rate. In order to compare situations we  
364used in Figure 4a,c an isopersistence level of 0.3 and in Figure 4b,d a level of  
3650.6. These levels are chosen with use of Figure 3d, where for migration rate 0.4  
366the fraction of simulations with persisting infection is around 0.3 at the peak  
367and for migration rate 0.8 around 0.6.

368 At low birth rate the total population size is smaller than at high birth rate,  
369because in the latter situation the population is close to carrying capacity. For  
370low birth rates both the panels with low and high migration rates (Fig. 4a-b)  
371show that in a single homogeneously mixing population the isopersistence level  
372is achieved at much lower transmission and death rates than in a metapopula-  
373tion. In that situation, a single homogeneously mixing population is thus more  
374beneficial for the infectious agent than a metapopulation, because at a low birth  
375rate susceptible hosts are not replenished at the same rate as at a high birth  
376rate; it takes more time before new susceptible hosts arise. When they do arise  
377it is more difficult for an infectious host in a metapopulation to successfully meet  
378this susceptible host, because the infectious host first has to move to a patch  
379with sufficient susceptible individuals within its infectious period. In a single  
380homogeneously mixing population the new susceptible hosts are immediately  
381available for contact.

382 At high birth and low migration rate (Fig. 4c), the isopersistence level for  
383the single homogeneously mixing population and the intermediate movement  
384distance almost coincide. At high transmission rate the isopersistence levels of  
385all situations coincide, because infectious hosts then have successful contacts  
386with more susceptible hosts per unit of time.

387 At both high birth and high migration rate (Fig. 4d), the differences between  
388the four cases are very small, especially at a large transmission rate all movement  
389distances coincide more or less with the single homogeneous mixing population.

390 All panels in this figure show the same pattern as described in previous

391 sections: persistence of an infectious agent in a metapopulation is easier accom-  
392 plished at intermediate movement distance of the host.

### 393 **Discussion**

394 In this paper we showed that persistence of an infectious agent in a metapopu-  
395 lation results from of a balance between spatial, demographical and epidemio-  
396 logical parameters. We showed on lattices a robust non-linear relation between  
397 the fraction of simulations with persisting infection and movement distance of  
398 hosts, with this fraction being largest at an intermediate movement distance of  
399 the host. Furthermore we showed that dividing a single homogeneously mixing  
400 population into several smaller populations that are connected to each other, is  
401 not necessarily beneficial for the persistence of the infectious agent.

402 The insight we obtain is based on simulations only. While an important  
403 part of relevant parameter space has been explored, there is considerable addi-  
404 tional value in providing analytical proof of the observed qualitative phenom-  
405 ena, to obtain robustness of results and deeper understanding. There have been  
406 many approaches in the literature where authors have focussed on analytical  
407 results concerning infectious disease in spatial metapopulations, and even more  
408 concerning single-species populations, predator-prey systems, and notably host-  
409 parasitoid systems. Mostly these studies concentrate on stability analysis of the  
410 trivial equilibrium (i.e. invasion problems, see Jansen & Lloyd, 2000, and refer-  
411 ences given there) or on providing analytical expressions for the expected time  
412 to extinction from a quasi-steady state (see N asell, 1999; Hagensars et al., 2004;  
413 Lindholm & Britton, 2007, and the references given there), as persistence in  
414 stochastic models is always temporary (Hanski, 1998; Lloyd-Smith et al., 2005).

415 In discrete-time models it is not possible to determine the quasi-stationary  
416 distribution and other measures have to be used; for example, persistence time  
417 (Rhodes & Anderson, 1996) or a certain number of time steps the infectious dis-  
418 ease is present in the metapopulation (Swinton et al., 1998; Vuilleumier et al.,  
419 2007; Jesse et al., 2008; Courcoul & Ezanno, 2010). We focus on persistence  
420 (long-term quasi-stability), i.e. we look at the fraction of realisations of the

421 stochastic process persisting for a time beyond a certain minimum (set arbitrar-  
422 ily by twice the average life span of the host species). We noticed that increasing  
423 this minimum did not qualitatively change the results. However, taking twice  
424 the life span of the host as measure for persistence for hosts with very short life  
425 spans, may lead to a time scale that is too short to speak of persistence.

426 In our system we merged a stochastic description with discrete spatial struc-  
427 ture, with on top of that non-uniform dispersal (due to a maximum movement  
428 distance for hosts). This combination did not allow us to obtain analytical re-  
429 sults, nor does the set-up allow for easy simplified models with only two patches,  
430 say, that might be amenable to direct analysis. We can, however, compare to  
431 studies where discrete space was studied analytically with different choices for  
432 dispersal. Rohani et al. (1996) conclude that, in predator-prey type interac-  
433 tion with uniform dispersal, the equilibrium becomes unstable, compared to a  
434 single homogeneous population, when there is a big difference in the dispersal  
435 between the two species. We have identical dispersal for our two ‘predator-prey  
436 species’ (infected and susceptible individuals, respectively). In our set-up we  
437 show, by simulation, differences in persistence, if dispersal is not uniform, but  
438 constrained. In the discussion of Rohani et al. (1996), it is argued that the  
439 equilibrium may destabilise when dispersal is not uniform. Similar equilibrium  
440 (in)stability results are discussed analytically by White & White (2005) studying  
441 coupled map lattices with integro-differential equations. They show in addition  
442 that destabilising influences of dispersal can only occur in “exploiter-victim”  
443 type of relationships.

444 In a much simpler model, Funk et al. (2005) study the dynamics of virus par-  
445 ticles in a spatial metapopulation of host cells. This is basically a one-species  
446 system because only the virus particles disperse. The dispersal mechanism,  
447 however, is closer to ours, as dispersal is constrained to be to (eight) nearest  
448 neighbours only. The analytical results allow a large class of dispersal mech-  
449 anisms to be studied. One of their results is that if dispersal is too fast the  
450 infection cannot be maintained. Webb et al. (2007) show, for a host-parasite  
451 system, that highly local systems, i.e. where dispersal is limited, have reduced

452 persistence of the infectious agent. Both these observations (Funk et al., 2005;  
453 Webb et al., 2007) are, although from very different models, in broad agreement  
454 with our finding of an optimal persistence for intermediate dispersal frequency  
455 and distance. This is in contrast to results by Hagenaars et al. (2004) who study  
456 persistence (measured as time to extinction from a quasi steady state and as  
457 mean outbreak duration) in a spatial and stochastic *SIR* set-up similar to ours.  
458 Their analytical and simulation results show a rather more monotone rise in  
459 persistence as a function of increasing between-patch contacts. However, there  
460 is an important difference with our model: their model has no actual dispersal  
461 within the metapopulation, the only migration comes from outside the system,  
462 and is essentially a model with two levels of contacts (within-patch and with  
463 individuals in other patches) for individuals that are otherwise fixed in their  
464 patch. Together, the above comparisons to related studies lead us to expect  
465 that our observation of non-monotone persistence as a function of coupling cru-  
466 cially depends on the physical migration between patches of individuals of at  
467 least two types in an “exploiter-victim” relationship, with constraints on the  
468 distance over which individuals can migrate within a time step.

469 An important characteristic of a metapopulation is that the infectious hosts  
470 have to move to find susceptible hosts. At low movement distance infectious  
471 hosts encounter a lower number of susceptible hosts than the average host,  
472 because the number of susceptible hosts is depleted by the presence of other  
473 infectious hosts. These infectious hosts are competing with each other over the  
474 available susceptibles and thereby reduce the potential for each one of them to  
475 transmit the infectious disease to a susceptible (Rhodes & Anderson, 2008). At  
476 a relatively high migration rate, i.e. with a short sojourn time per patch, a  
477 host can meet more others than at a low migration rate. An infectious host will  
478 therefore have a larger number of contacts with susceptible hosts, because they  
479 visit more patches during their infectious period. Compared to a single homo-  
480 geneously mixing population, hosts in metapopulations can have the advantage  
481 that they do not reach all susceptible hosts at the same time, which can improve  
482 the probability of survival of the infectious agent.

483 As is also shown by Cross et al. (2005), the relation between migration  
484 and recovery is very important for the ability of an infectious agent to persist,  
485 because the infectious period determines the time an infectious host has to infect  
486 susceptible hosts. And the migration rate is an important determinant of the  
487 level of mixing.

488 Regarding movement we studied a wide range of migration rates: from mi-  
489 grating almost never, to migrating every week. We also assumed that all indi-  
490 viduals experience the same migration rate. In reality there is a lot of difference  
491 in movement behaviour among species (Hawkes, 2009). Movement may, for ex-  
492 ample, be restricted to mainly juveniles leaving their nest to settle elsewhere or  
493 to foraging outside the own habitat patch. The first mechanism can be thought  
494 of at low migration rates, although there are no age-classes defined in the model.  
495 The latter one at high migration rates, where individuals leave their own patch,  
496 visit another one and then return to their home patch again.

497 This situation could be even more resembled when the probability to move to  
498 a certain patch depends on the distance of that patch with respect to the resident  
499 patch. We assumed random dispersal, within the movement distance, but it  
500 can also be argued that hosts may have a preference for nearby patches. The  
501 influence of a dispersal function, favouring nearby patches, on the persistence  
502 of the infectious agent should therefore be studied more carefully.

503 In this paper we defined the movement distance by the number of edges  
504 crossed. Another option would have been using a radial movement distance,  
505 where individuals can move to all patches within a certain radius around their  
506 resident patch. However, using a radial movement distance instead, does not  
507 affect the results. Durrett & Levin (1994) also concluded that the qualitative  
508 behaviour is independent of the exact definition of movement distance when  
509 they studied interacting particle systems for various definitions of movement  
510 distance.

511 There have been studies of the effect of the neighbourhood of a patch on the  
512 persistence of the infectious agent in a spatially structured population. In an  
513 ecological context Vuilleumier et al. (2007) and in an epidemiological context

514 Rhodes & Anderson (1996) concluded that increasing the movement distance  
515 maximises the probability of persistence. They considered patches as the unit  
516 of study and had many unoccupied patches in the population. Another study  
517 (Courcoul & Ezanno, 2010) did take within-patch dynamics into account, but  
518 they also considered indirect transmission. The probability of infection of a sus-  
519 ceptible host in a patch depended then not only on the available infectious hosts  
520 in the same patch, but also on the prevalence of the infection in neighbouring  
521 patches. Courcoul & Ezanno (2010) varied the number of neighbouring patches  
522 that influence the infection dynamics in a patch, but did not see a peak in the  
523 fraction of simulations with persisting infection at an intermediate number of  
524 neighbours. However, due to the different ways persistence can be defined, one  
525 should be cautious in comparing the various studies.

526 The model used in this paper is as simple as possible. For example, it is not  
527 taken into account that infectious hosts might experience reduced mobility or  
528 death as a result of the infectious disease. Another assumption is continuous  
529 birth, as opposed to seasonal birth, where births occurs in a short period of  
530 a year, which is often the case in animal populations. Seasonal births reduces  
531 the probability of persistence of the infectious agent, because there are fewer  
532 infectious hosts between epidemics, which increases the probability of fade-out  
533 due to stochasticity (Grassly & Fraser, 2006). But, each year there is also a burst  
534 of new susceptible hosts into the population (Altizer et al., 2006), creating the  
535 possibility of a new epidemic. One of the next steps in exploring the balance  
536 of the various aspects (spatial, demographical and epidemiological) involved in  
537 the persistence of an infectious agent would be to include one or more of the  
538 above mentioned options.

### 539 **Acknowledgements**

540 This research is supported by The Netherlands Organisation for Scientific Re-  
541 search (NWO grant 918.56.620).

542 **References**

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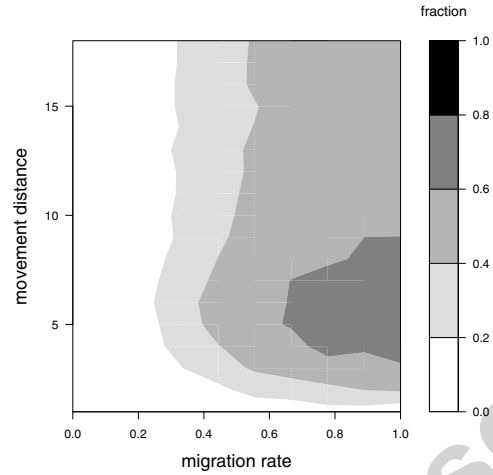
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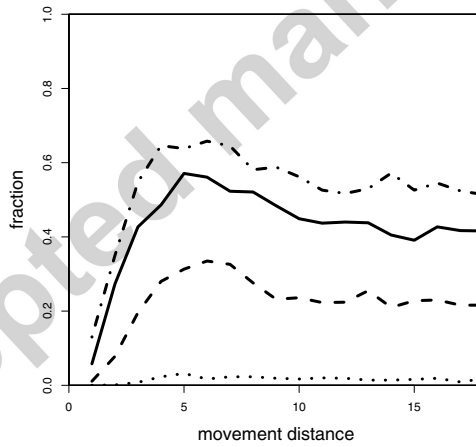


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(a)



(b)

Figure 1: In *a*) the contour lines mark the area where the combination of the migration rate and the movement distance yield the same minimum fraction of simulations with persisting infection. In *b*) the fraction of simulations with persisting infection is shown with migration rate 0.2 (dotted line), 0.4 (dashed line), 0.6 (solid line) and 0.8 (dot-dashed line). The results are taken from 1000 simulations and the default parameter values are  $P = 100$ ,  $K = 10$ ,  $\beta = 10$ ,  $\gamma = 0.5$ ,  $b = 0.2$  and  $\mu = 0.01$ .

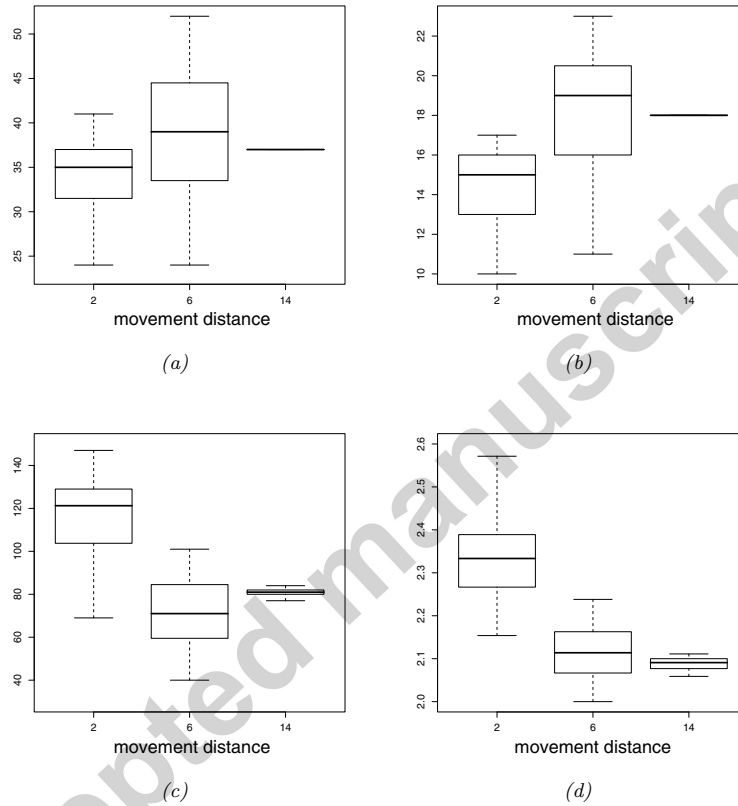


Figure 2: A box-and-whisker-plot, where the thick line denotes the median, the box encloses 50% of the observations and the whiskers show the 2.5 and 97.5 percentile. Panel *a*) shows the total number of time steps that a patch contains at least one infectious host is shown; panel *b*) the number of times that a patch gets infected, i.e. the number of transitions from no infectious host in a patch to at least one infectious host; panel *c*) the total number of infectious hosts per patch and panel *d*) the number of consecutive time steps a patch contains an infectious host. The results are from 1000 simulations with persisting infection, run till  $t = 200$ . The default parameter values are  $P = 100$ ,  $K = 10$ ,  $\beta = 10$ ,  $\gamma = 0.5$ ,  $b = 0.2$  and  $\mu = 0.01$ .

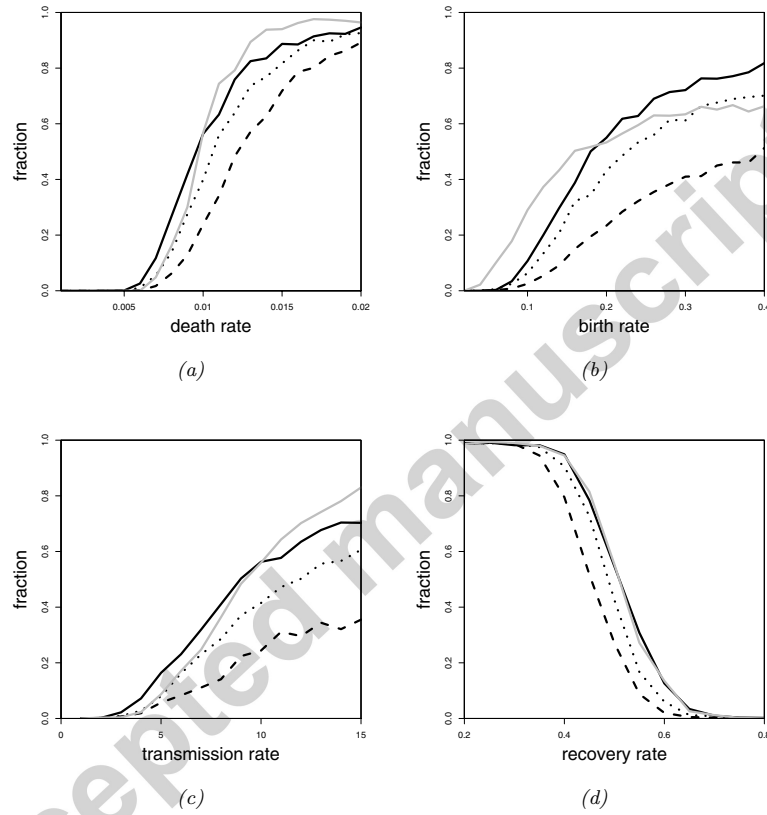


Figure 3: The fraction of simulations with persisting infection for a range of values of four demographical and epidemiological parameters. In each panel, the results (from 1000 simulations) are shown for the movement distance 2 (dashed line), 6 (solid line) and 14 (dotted line) and for a single homogeneously mixing population (grey line). A migration rate of 0.6 is used, and the default parameter values are  $P = 100$ ,  $K = 10$ ,  $\beta = 10$ ,  $\gamma = 0.5$ ,  $b = 0.2$  and  $\mu = 0.01$ .

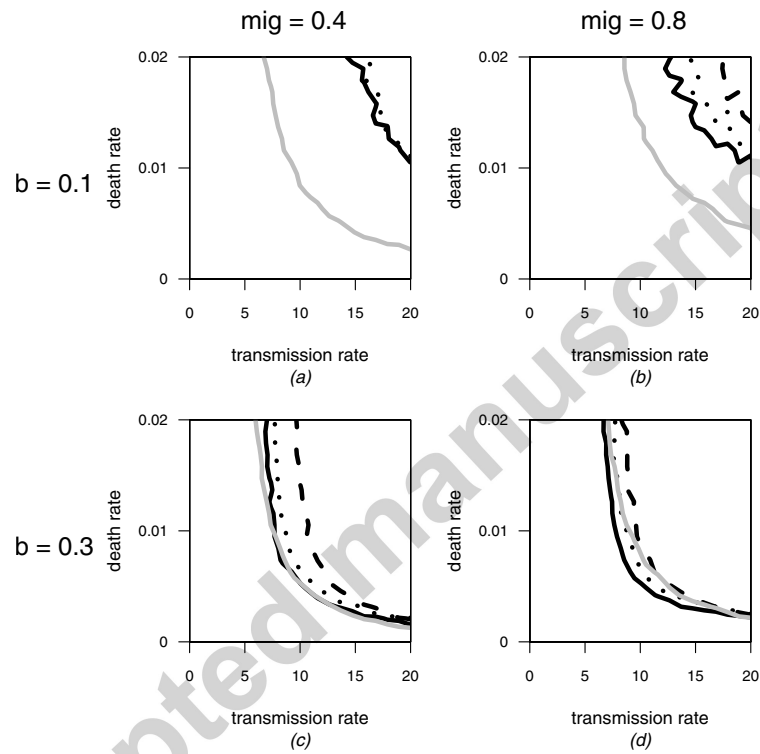


Figure 4: Curves for parameter combinations leading to the same fraction of simulations with persisting infection, where for panel *a*) and *c*) this fraction is 0.3 and for panels *b*) and *d*) the fraction is 0.6. The results are for movement distance 2 (dashed line), 6 (solid line) and 14 (dotted line) and for a single homogeneously mixing population (grey line). As migration rates 0.4 (panel *a*) and *c*) and 0.8 (panel *b*) and *d*) are used and birth rate 0.1 (panel *a*) and *b*) and 0.3 (panel *c*) and *d*). The other parameters are set to the default values:  $P = 100$ ,  $K = 10$  and  $\gamma = 0.5$ .