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Environmental pathogen reservoirs and habitat heterogeneity in a metapopulation

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1 Introduction

Many wildlife diseases are caused by pathogens that can persist, and remain infectious, for long periods of time in the environment. Examples include chronic wasting disease (Miller et al (2006)), anthrax (Dragon and Rennie (1995)), plague (Eisen et al (2008)), and white nose syndrome (Lindner et al (2011)), among others. This environmental persistence creates environmental pathogen reservoirs from which hosts can become infected without direct contact with an infected individual. This additional transmission pathway can have important consequences for disease dynamics, with models showing that increased environmental persistence generally facilitates increased pathogen persistence and spread relative to direct transmission alone (Almberg et al (2011), Sharp and Pastor (2011), Breban et al (2009).

Since environmental transmission is spatially explicit (i.e. environmental reservoirs can only infect local residents), its role in disease dynamics may further depend on the spatial structure of the host population. In particular, we expect that host population structure and movement will be influenced by heterogeneity in quality among the habitat patches in a metapopulation. Indeed, the quality of a habitat patch can affect its extinction and colonization rates, as well as its contribution to the colonization of other empty patches (Moilanen and Hanski (1998)). These processes in turn may then influence where environmental pathogen reservoirs get established and how they affect disease dynamics and host occupancy throughout the metapopulation.

Specifically, we expect that high quality habitat patches, which support greater host density and traffic than lower quality habitat, might be more likely to form pathogen reservoirs. As these reservoirs are undetectable to the host, we predict that high quality patches will continue to attract – and infect – susceptible immigrants, effectively creating an ecological trap (Almberg et al (2011)). In addition, the greater traffic through high quality patches may further facilitate pathogen spread by positioning high quality patches as metapopulation-scale superspreaders (citation). Similarly, we expect that low quality patches, which see relatively

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less host traffic, will be less likely to develop pathogen reservoirs and thus may serve as refuges on which susceptible hosts can escape infection.

Many, if not all, of the pathogens listed above affect spatially structured host populations (e.g. plague in prairie dog colonies), and thus understanding how environmental transmission interacts with patterns of habitat quality is critical to managing disease in these systems. In this study then, we seek to explore how a pathogen's environmental longevity (how long it can persist and remain infections in the environment), and habitat heterogeneity (the variance in the distribution of patch quality in a metapopulation) interact to influence pathogen spread and host occupancy, with a specific interest in the roles played by high and low quality habitat.

2 Methods

2.1 Model Structure

To address the above questions, we developed a theoretical stochastic patch occupancy model (SPOM) in which each patch can be in one of three possible states: occupied by susceptible hosts (S), occupied by infected hosts (I), and unoccupied by the host. State transitions are governed by host colonization, extinction, and infection rates, where a susceptible population can become infected either through direct contact with infected immigrants, or through a local pathogen reservoir.

Building on the framework developed by Hanski (1994), the rate at which patch i, with quality A_i, is colonized by individuals in state $X \in (S, I)$ is given by its connectivity:

$$C_{Xi} = A_i^{\xi_{im}} \sum_{i \neq j} \phi_j A_j^{\xi_{em}} e^{-D d_{ij}}, \tag{1}$$

where ϕ_j is an indicator function that is 1 if patch j is in state X and is 0 otherwise; ξ_{im} and ξ_{em} control how rates of immigration and emigration, respectively, scale with patch quality; D is the inverse of mean dispersal distance; and d_{ij} is the distance between patches i and j. Essentially, C_{Xi} sums the colonization effort from all patches in state X to patch i.

The extinction rate of patch i in state X is given by:

$$E_{Xi} = \frac{e_X}{A_i^{\alpha}}, \tag{2}$$

where e_X is the extinction rate of a patch of unit quality in state X (here we assume that $e_I > e_S$), and α controls how extinction rate scales with patch quality. When an infected population goes extinct, we assume that the infected hosts leave behind an infectious pathogen reservoir on the patch.

Susceptible populations are infected via direct contact with infected colonists at rate δC_{Ii}, where δ is the transmission probability. In addition, environmental transmission can take place when a susceptible population occupies a patch with an environmental reservoir, and occurs at rate:

$$\gamma_i = \gamma_0 exp(-r t_{Ii}), \tag{3}$$

where γ_0 is the initial infection rate of the pathogen reservoir, t_{Ii} is the time since last infected occupancy, and r is the pathogen's decay rate in the environment.

Table 1 Parameters of SPOM model and their interpretations.

Parameter	Interpretation	Default Value
ξ_{im}	Scaling parameter for effect of target patch quality on immigration	0.5
ξ_{em}	Scaling parameter for effect of target patch quality in emigration	0.5
D	Inverse of mean dispersal distance	5
d_{ij}	Distance between patch i and j	1 for all i, j
e_S	Extinction rate of unit quality susceptible patch	0.1
e_I	Extinction rate of unity quality infected patch	0.5
α	Strength of environmental stochasticity	1
δ	Probability of direct infection	0.5
γ_0	Initial rate of infection from reservoir patch	0.5

2.2 Model Implementation and Analysis

The model outlined above was parameterized (Table 1) according to the following assumptions. (1) High quality patches maintain higher population sizes and therefore produce more colonists than low quality patches and have lower extinction rates than low quality patches ($\xi_{im} = 0.5$, $\alpha = 1$, Hanski and Ovaskainen (2003)). (2) High quality patches attract more colonists than low quality patches ($\xi_{em} = 0.5$, Hanski and Ovaskainen (2003)). (3) Connectivity among patches is determined entirely by quality ($d_{ij} = 1$ for all $i \neq j$). In addition to these assumptions, parameters were chosen so that without infection, approximately 0.75 of the metapopulation was occupied ($e_S = 0.1$, $D = 5$), and a range of epidemiological behaviors were feasible ($e_I = 0.5$, $\delta = 0.5$, $\gamma_0 = 0.5$).

With the above parameters fixed, we explored the effects of pathogen environmental persistence and habitat heterogeneity by varying pathogen longevity (10 values ranging from 20 to 200 time steps, with values representing the half-life of the pathogen's infectivity), and the variance of the patch quality distribution (10 values ranging from 0.02 to 0.2). For each simulation (100 replicates for each of the 100 longevity-variance combinations), qualities for 100 patches were drawn from a uniform distribution with mean 1 and given variance. In each case, an entirely susceptible population was simulated until it reached an approximate steady state, at which point a randomly chosen occupied patch was infected. The state of the metapopulation was then tracked for 5000 time steps. To address the metapopulation-level questions above, we recorded the proportion of patches in each state at the end of each simulation and classified the simulation as host extinction, disease-free (no infected populations after 5000 time steps), endemic (both susceptible and infected populations present), or pandemic (only infected populations). In addition, to address patch-level questions, for every simulation, we recorded the quality of each patch, the proportion of time spent in each state, and the number of transitions between each state.

In the above simulations, the mean of the patch quality distribution was held constant, and thus the range was expanded symmetrically about one as the variance was increased. To further parse out the relative roles of high and low quality patches, we performed a series of simulations where we expanded the range of the quality distribution non-symmetrically to include high and low quality patches separately. For these simulations, at each value for longevity, we simulated 100 metapopulations for each of four patch quality distributions: low varaince, (range

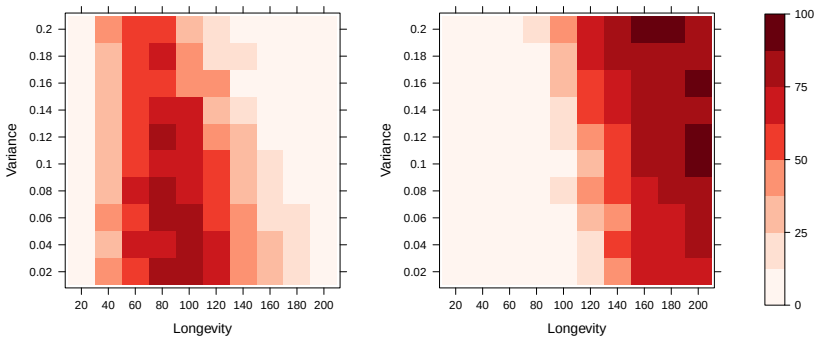

Fig. 1 Percent of simulations resulting in (a) endemic disease (both susceptibles and infecteds persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

0.75 to 1.25); intermediate variance, high quality (range 0.75 to 1.75); intermediate variance, low quality (range 0.25 to 1.25); and high variance (range 0.25 to 1.75).

In addition, to explore the effects of the model assumptions and parameterization, we repeated the above analyses for three additional model parameterizations: with patches arranged in a lattice structure; with $\alpha = 0$ to remove the effect of patch quality on extinction rate; and with $\delta = 0.1$ to reduce the relative importance of direct transmission.

Continuous time stochastic simulations of the above model were implemented in the R language (citation) using the Gillespie algorithm (Gillespie (1977)). Code is available at `https://github.com/clint-leach/Metapop-Disease`.

3 Results

The pathogen persisted in 7087 of the 10000 simulations, spreading through the entire susceptible host population (i.e. a pandemic) in 3721 simulations. Pathogen persistence increased substantially with longevity, with intermediate longevities favoring endemic disease, and high longevities facilitating pandemic dynamics (Fig 1). The effect of longevity, however, depended on the variance of the patch quality distribution, with low variance metapopulations favoring endemic dynamics for a wider range of longevities (Fig 1a), and high variance metapopulations lowering the longevity required for pandemic dynamics (Fig 1b). The three alternative parameterizations produced qualitatively similar results, though pathogen spread was generally more difficult under these assumptions (i.e. fewer pandemics), and the habitat quality variance exerted a relatively weaker influence on the epidemiological outcome (Figs 5, 8, 11).

To separate the influence of high and low quality patches, we explored how susceptible and infected patch occupancy changes when high and low quality patches are included separately in the patch quality distribution. Relative to a low-variance metapopulation (patch qualities roughly between 0.75 and 1.25), shifting the dis-

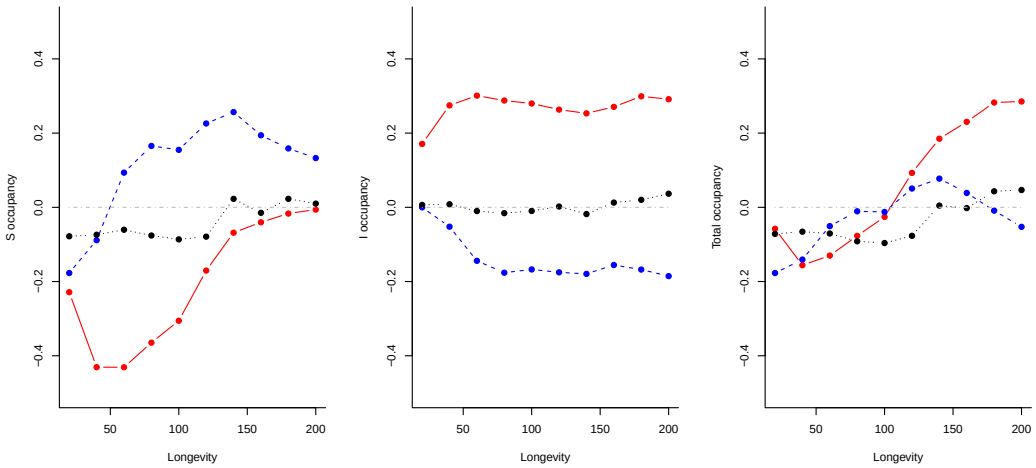

Fig. 2 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infected occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black shows high variance.

tribution toward low quality patches (distribution ranging from 0.25 to 1.25) increased mean susceptible occupancy, while shifting towards high quality patches (range from 0.75 to 1.75) decreased mean susceptible occupancy, with the largest effects observed at intermediate longevities (Fig 2(a)). Conversely, a shift towards low quality patches decreased infected occupancy, while a shift towards high quality patches increased infected occupancy (Fig 2(b)). These general trends were consistent across the three other parameterizations (Figs 6, 9, 12).

However, at high pathogen longevities, high quality patch distributions had a net positive effect on total occupancy relative to the other patch quality distributions (Fig 2(c)). Due to their lower extinction rates and high connectivity, high quality patches were able to maintain stable occupancy despite the widespread infection facilitated by high longevity. On the other hand, when high pathogen longevities lead to pandemic dynamics, low quality distributions were unable to support infected occupancy, resulting in an increase in host extinction events and a decrease in mean occupancy. The model in which the extinction rate does not scale with quality (i.e. $\alpha = 0$) produced very similar results (Fig 9(c)). However, in simulations with weak direct infection ($\delta = 0.1$), the high quality patch distributions always produced higher occupancy than low quality patch distributions (Fig 12(c)), while in the model with the lattice structure, low quality patch distributions actually produced higher occupancies than high quality distributions at high longevity (Fig 6(c)).

To understand the mechanisms behind these effects, we further examined within patch dynamics in high variance metapopulations (with qualities ranging from 0.25 to 1.75) that produced endemic dynamics. Because of their lower extinction rate and higher recolonization rate, high quality patches supported more consistent occupancy than low quality patches (Fig 3). However, as the epidemic

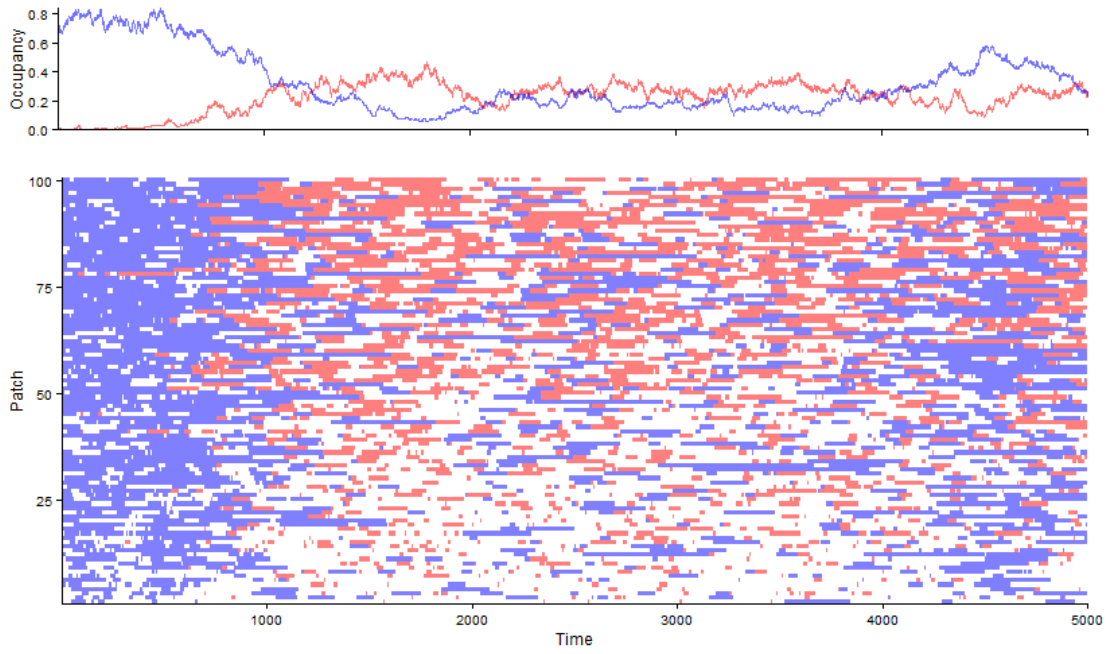

Fig. 3 Results from a single simulation with variance 0.2 and longevity 80. The top panel shows total occupancy of susceptible (blue) and infected (red) patches over time. The bottom panel shows the state – susceptible (blue), infected (red), or unoccupied (white) – of individual patches through time. Patches are stacked vertically with the lowest quality (~ 0.25) at the bottom and the highest quality (~ 1.75) at the top.

progressed, high quality patches increasingly supported infected occupants, while low quality patches supported infecteds only briefly and infrequently (Fig 3). In addition, while high quality patches did support susceptible occupancy, they experienced a greater number of infection events (susceptible to infected transitions) than low quality patches (Fig 4). This relationship became weaker as longevity increased, due to the fact that as longevity increases and the pathogen spreads more easily, there are fewer susceptibles remaining in the metapopulation to become infected. A similar trend was observed across the other parameterizations, though the observed relationship between patch quality and number of infection events was shallower for the lattice and weak direct infection models (Figs 7, 10, 13).

4 Discussion

The positive relationship between pathogen spread and environmental longevity demonstrated in the theoretical model above is well documented in models of empirical systems. Almberg et al (2011) find that, in plausible simulations of chronic wasting disease in deer, increasing prion survival in the environment increases the resulting force of infection and peak prevalence, with the importance of reservoir

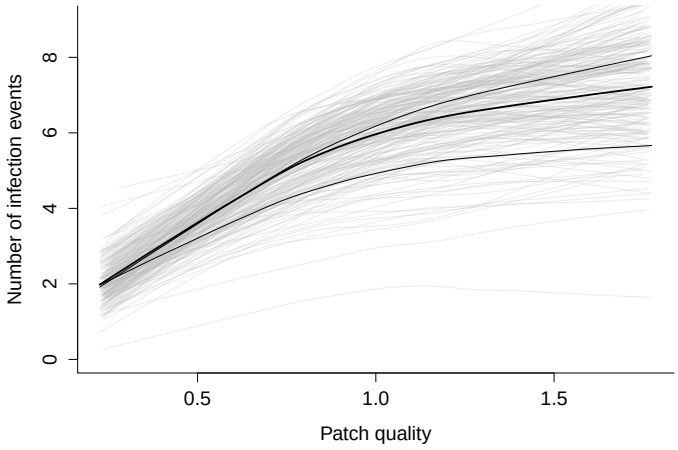

Fig. 4 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40 (top) and 140 (bottom).

transmission increasing through time. Similarly, Breban et al (2009) show that environmental transmission of avian influenza virus allows it to persist in situations where direct transmission alone is insufficient. *Other examples.*

Moreover, these simulations suggest that high variance metapopulations are more prone to widespread pathogen spread than low variance metapopulations (Fig 1). In particular, metapopulations with high patch quality variance require lower environmental longevities to experience pandemic dynamics (Fig 1).The mechanisms underlying this behavior can be understood by drawing parallels with models of pathogen spread through a contact network. In this context, our metapopulation can be viewed a network with patches as nodes connected through immigration (with the strength of the connection measured by connectivity). Many studies have shown that networks with high degree variance (where a node's degree is roughly equivalent to its total connectivity in our metapopulation model) are easily invaded by pathogens, which are then able to spread much more rapidly than in less heterogeneous networks (Pastor-Satorras and Vespignani (2001)). However, as the alternative parameterizations show, the overall effect of patch quality variance depends on the structure of the metapopulation, the resulting colonization and extinction rates, and the relative balance of the effects of low and high quality patches. In the focal model, the dynamics of high variance metapopulations were driven largely by the presence of high quality patches, whereas in the models with the lattice structure and with equal extinction rates across all patches, high and low quality patches were more balanced in their effects, thus canceling each other out as variance increased.

The effects of patch quality variance, and its interaction with longevity, result from the combined influence of both low and high quality patches in pathogen spread and metapopulation stability. Since a patch's connectivity is determined,

at least in part, by its quality, high quality patches function as highly connected hubs in the metapopulation. Specifically, high quality patches both attract more colonists from other patches and produce more colonists that spread to other patches. These two properties have important implications for pathogen spread in the metapopulation. Attracting colonists helps position high quality patches as the ecological "traps" hypothesized above, wherein susceptible hosts repeatedly colonize high quality patches and subsequently become infected (Fig 4). This infection pressure results from both transmission from the environmental reservoir and from direct transmission from infected immigrants attracted to the high quality patch, reflecting results from the contact network literature that show that highly connected nodes have higher infection risk (Christley et al (2005), Keeling and Eames (2005)).

This trap effect, coupled with the relatively low extinction rate on high quality patches helps to create a stable platform from which the pathogen can spread through the rest of the metapopulation. Indeed, high quality patches are effectively metapopulation scale superspreaders (Lloyd-Smith et al (2005), Paull et al (2012)). Once the pathogen infects high quality patches, the larger number of host colonists produced by high quality patches allow it to spread to the rest of the metapopulation relatively easily. This process helps to maintain infected occupancy throughout the metapopulation, which in turn feeds back on high quality patches to ensure a steady stream of infected colonists that help maintain the trap effect. Thus, through these two interacting mechanisms – the trap and superspreader effects – the presence of high quality patches serves to significantly increase pathogen spread and infected occupancy in the metapopulation (Fig 2).

In contrast, low quality patches help to limit pathogen spread and increase susceptible occupancy (Fig 2). Even though individual low quality patches are unable to support consistent occupancy (Fig 3), at the metapopulation level, the presence of low quality patches increases overall susceptible occupancy relative to a lower variance metapopulation (Fig 2). This phenomenon represents the other side of the hub role played by high quality patches in that low quality patches are relatively weakly connected to the metapopulation and are therefore infrequently colonized by infected individuals. Moreover, the instability of low quality patches (i.e. the high extinction rate), together with the increased mortality of infected populations, means that infected populations do not persist long on low quality patches. Because low quality patches are so infrequently colonized, at low to intermediate longevities, the environmental reservoir left behind by these infected populations generally decays before it has the opportunity to infect new susceptible colonists. As a result, low quality patches represent a dead-end for the pathogen, reducing the number of patches from which it can spread, and thereby reducing infected occupancy (Fig 2).

However, the ability of low quality patches to influence disease dynamics depends on the relative strength of the different transmission pathways – direct and environmental – and on the structure of the metapopulation. At intermediate longevities, the timescale of pathogen decay roughly matches the timescale of the colonization and extinction processes. In contrast, for low longevities, when the pathogen reservoir decays much faster than colonizations and extinctions occur, environmental transmission has little effect on disease dynamics and the effect of patch quality is relatively weak. Conversely, when the pathogen decays much slower than colonizations and extinctions occur, i.e. high longevity, the pathogen

reservoir persists on nearly all patches and the effects of patch quality are washed out. In these cases, the role of patch quality lies primarily in its effect on extinction rates. Due to their lower extinction rates, high quality patches are able to maintain stable occupancy despite widespread (pandemic) infection, while low quality patches are unable to support infected occupancy, resulting in an increase in host extinction events at the highest longevities.

These findings suggest the presence of a trade-off between facilitating metapopulation stability and inhibiting disease spread, with a pathogen's environmental longevity controlling the roles of different quality patches with respect to this trade-off. At low to intermediate longevities, the addition of high quality patches functions similarly to increasing movement in the model developed by Hess (1996), increasing pathogen spread and reducing overall occupancy (Fig 2c). In these cases, rather than serving as sources in the metapopulation, high quality patches become sinks, and more importantly, they become ecological traps, which can be even more detrimental to the overall population (Kristan (2003)). From a management perspective, it is critical to understand this shift, as previous theoretical work suggests that management should focus on patches where conditions are most favorable (Strasser et al (2010)). Generally, this means that maintaining high quality habitat should form the focus of managers, but Strasser et al. (2010) show that stochastic disturbance (e.g. disease) can lead to cases where focusing on low quality patches is more effective in increasing population growth rate.

Also discuss Sharp and Pastor (2011), who show that management actions that increase carrying capacity can increase disease spread and destabilize population. And Hess (1996) who finds that implementing a quarantine on centralized patches reduces pathogen spread.

However, as pathogen longevity increases, the trade-off shifts and the addition of high quality patches is a net benefit to overall occupancy, reflecting the findings of Gog et al (2002), who find that under background infection from an alternative host, increasing movement rarely reduces occupancy, despite facilitating pathogen spread. Park (2012) further develops this work by noting that the magnitude of the detrimental effects of increased moevement on occupancy depends on the relative strength of different transmission pathways. Specifically, he finds that as environmental transmission becomes stronger (e.g. higher longevity) relative to direct transmission, then total occupancy increases monotonically with movement, i.e. movement is a net benefit to the metapopulation. These studies, coupled with the results presented here, suggest that when there is a persistent source of infection throughout the metapopulation (e.g. when the pathogen is long-lived in the environment), factors that facilitate metapopulation stability (i.e. increased movement, high quality patches) are a greater benefit than factors that inhibit pathogen spread (i.e. decreased movement, low quality patches).

Spatial structure and disease spread represent two major factors influencing the dynamics of wildlife populations. As we have seen, in cases where the pathogen is able to persist and remain infectious in the environment, these two factors can interact in complex and interesting ways. Failing to consider these interactions and their effect on the roles played by high and low quality habitat risks overlooking the importance of low-quality habitat for the persistence of wildlife populations and the importance of high-quality habitat for the persistence of wildlife disease.

5 Appendix

5.1 Sensitivity Analyses

5.1.1 Lattice structure

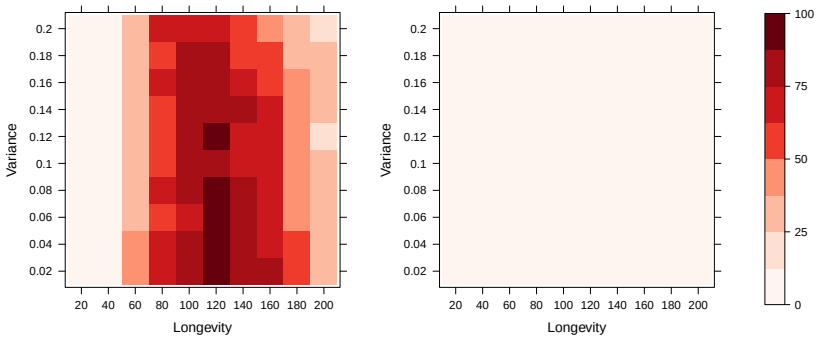

Fig. 5 Percent of simulations resulting in (a) endemic disease (both susceptibles and infecteds persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

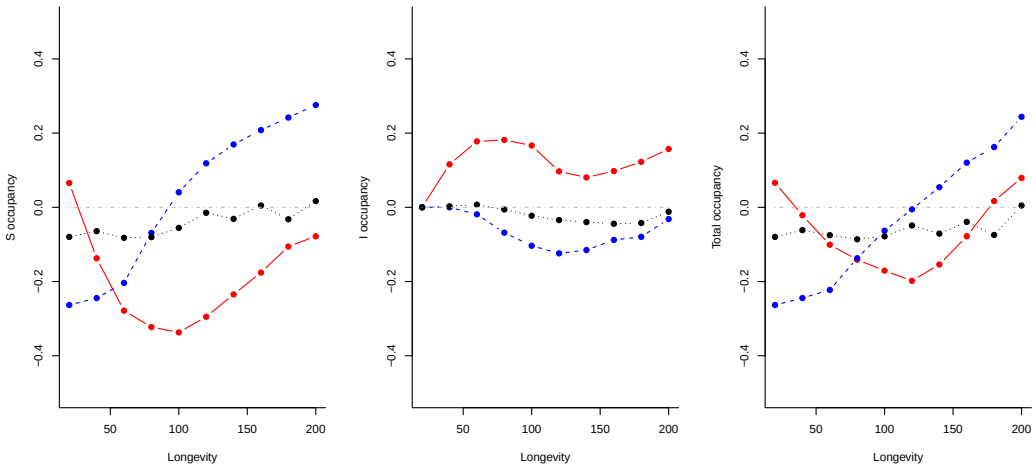

Fig. 6 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infected occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black shows high variance.

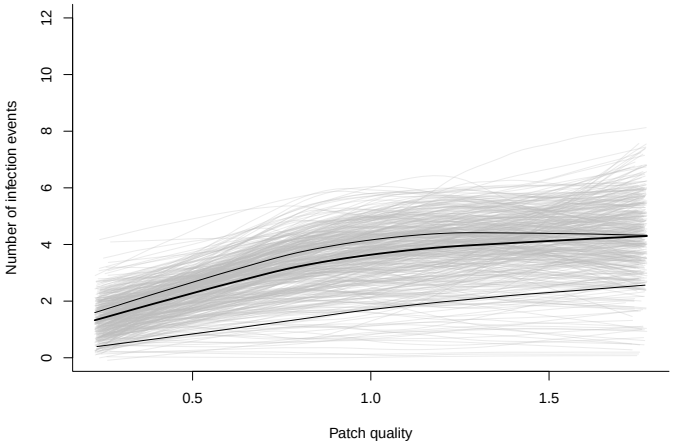

Fig. 7 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40 (bottom) and 140 (top).

305 *5.1.2 Extinction scaling,* $\alpha = 0$

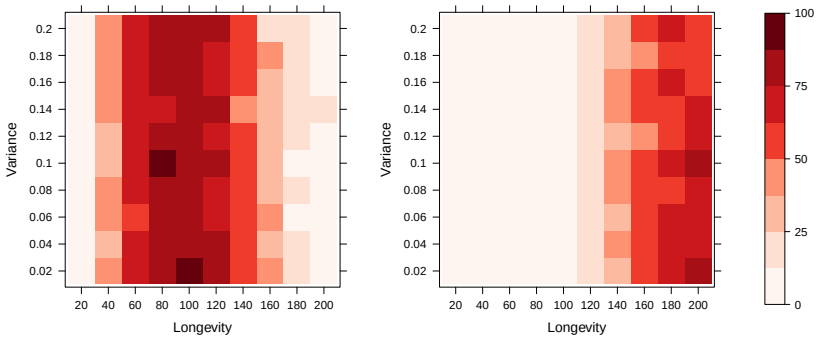

Fig. 8 Percent of simulations resulting in (a) endemic disease (both susceptibles and infecteds persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

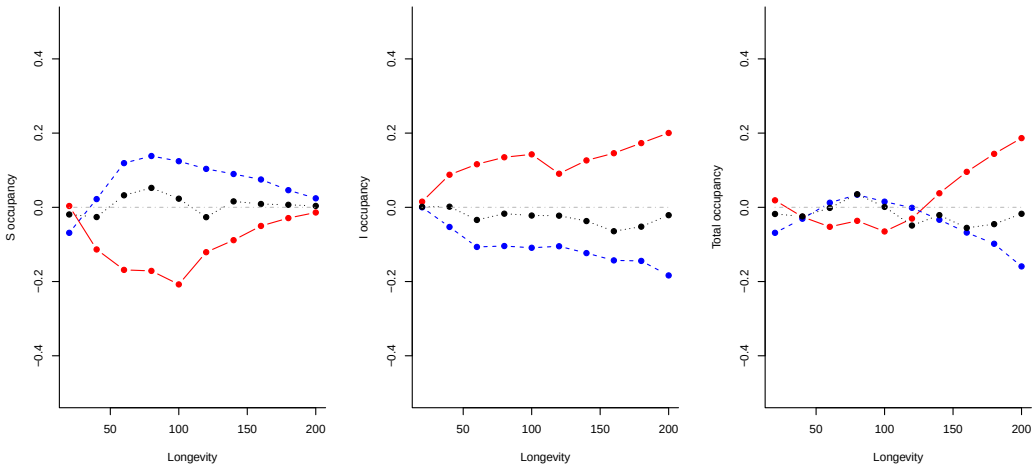

Fig. 9 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infected occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black shows high variance.

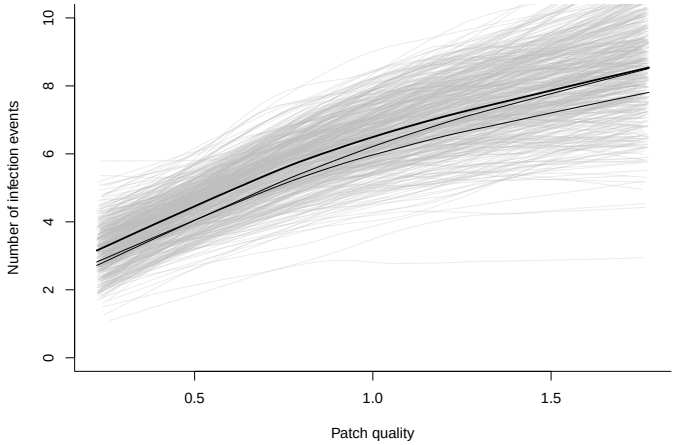

Fig. 10 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40 (top) and 140 (bottom).

306 *5.1.3 Weak direct transmission,* $\delta = 0.1$

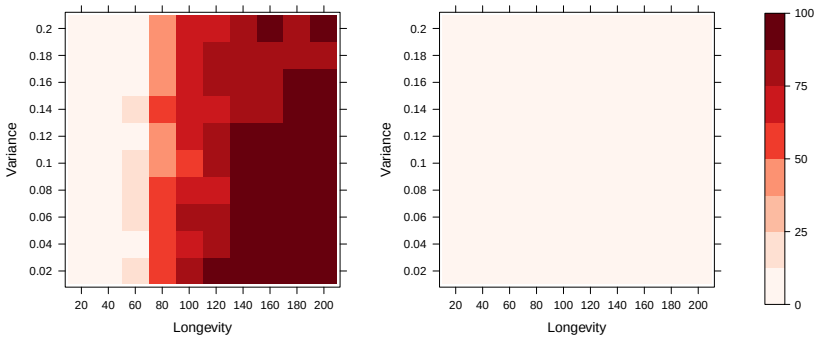

Fig. 11 Percent of simulations resulting in (a) endemic disease (both susceptibles and infecteds persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

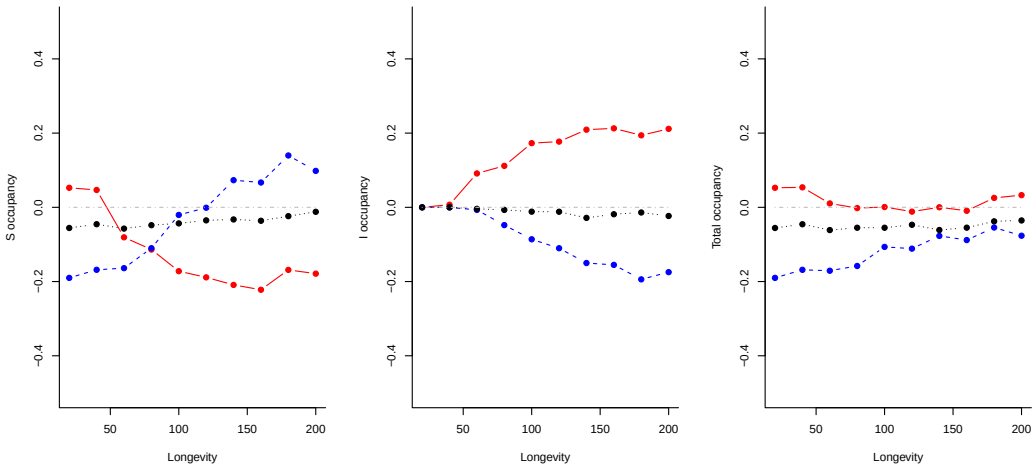

Fig. 12 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infected occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black shows high variance.

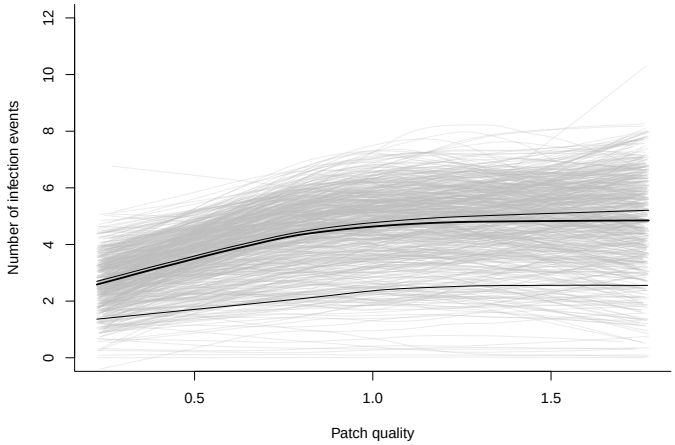

Fig. 13 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 60 (bottom) and 120 (top).

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