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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 infectious 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 (Paull et al (2012)). Similarly, we expect that low quality patches, which see

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Table 1 State transitions and their rates for patch i in a metapopulation simulation. S denotes occupied by susceptible hosts, I denotes occupied infectious hosts, and $\emptyset$ denotes unoccupied by the host. All patches are characterized by an environmental infection rate, γ_i, which is non-zero only if the patch has been previously occupied by infectious hosts.

State Transition	Rate
$S \to I$	$\delta C_{Ii} + \gamma_i$
$S \to \emptyset$	e_{Si}
$I \to \emptyset$	e_{Ii}
$\emptyset \to S$	C_{Si}
$\emptyset \to I$	C_{Ii}

relatively 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, George et al (2013)), 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 the distribution of habitat 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 infectious hosts (I), and unoccupied by the host ($\emptyset$). State transitions are governed by host colonization, extinction, and infection rates, where a susceptible population can become infectious either through direct contact with infectious immigrants, or through a local pathogen reservoir (Table 1).

Building on the framework developed by Hanski and Ovaskainen (2003), 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 $e_I > e_S$ to account for disease-induced mortality), and α controls how extinction rate scales with patch quality. When an infectious population goes extinct, we assume that the hosts leave behind an infectious pathogen reservoir on the patch.

Susceptible populations are infected via direct contact with infectious colonists at rate δC_{Ii}, where δ is the direct transmission probability. In addition, environmental transmission can take place when a susceptible population occupies a previously infected patch, 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 infectious occupancy ($t_{Ii} = \infty$, and thus $\gamma_i = 0$, if the patch has never been occupied by infectious hosts), and r is the pathogen's decay rate in the environment.

Table 2 Parameters of SPOM model, their meaning, and the values assigned under different parameterizations. Empty cells indicate same value as the default parameterization.

Parameter	Interpretation	Default	Lattice	Equal Ext.	Weak Direct Inf.
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	2	–	–
d_{ij}	Distance between patch i and j	1 $\forall i \neq j$	1 $\forall i, j$ neighbors	–	–
e_S	Extinction rate of unit quality susceptible patch	0.1	–	–	–
e_I	Extinction rate of unity quality infectious patch	0.5	–	–	–
α	Strength of environmental stochasticity	1	–	0	–
δ	Probability of direct infection	0.5	–	–	0.1
γ_0	Initial rate of infection from reservoir patch	0.5	–	–	–

2.2 Model Parameterization

The model outlined above was parameterized in four different ways to explore the consequences of different assumptions (Table 2). The baseline model was parameterized 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 – disease-free, endemic disease, and pandemic disease (i.e. all host populations infectious) – was feasible, depending on the pathogen's environmental longevity ($e_I = 0.5$, $\delta = 0.5$, $\gamma_0 = 0.5$).

The baseline model above assumes that all patches are equally accessible from all other patches (i.e. are separated by distance 1), so to examine the effects of a more rigid spatial structure, we also implemented a model with patches arranged in a square lattice such that each patch is only accessible from its four neighboring patches (Lattice, Table 2). In addition, we explored a model with $\alpha = 0$ so that the extinction rate was constant with patch quality, simulating the effects of high environmental stochasticity, i.e. environmental stochasticity is strong enough that the population size of a patch does not affects its extinction rate (Equal extinction, Table 2). Lastly, we parameterized a model with $\delta = 0.1$ to reduce the relative importance of direct transmission and explore the disease dynamics when transmission is driven largely by the environmental reservoir (Weak direct infection, Table 2).

2.3 Simulation studies

For each of the above parameterizations, we performed two different simulation experiments to explore the combined effects of pathogen environmental longevity and habitat quality distribution on disease dynamics and metapopulation occupancy. In the first of these, we investigated the effect of increasing habitat heterogeneity on the dynamics of pathogens with a range of environmental longevities. For these simulations, we explored 10 values of pathogen longevity ranging from 20 to 200 time steps, with values representing the half-life of the pathogen's infectivity, and 10 values for the variance of the patch quality distribution, 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 fixed 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. 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 infectious populations after 5000 time steps), endemic (both susceptible and infectious populations present), or pandemic (only infectious populations). In addition, to identify the roles played by individual patches of varying quality, for every simulation, we recorded the quality of each patch, the proportion of time spent in each state (i.e. occupied by susceptible hosts, occupied by infectious hosts, and unoccupied), and the number of transitions between each state.

The above simulations held the mean of patch quality distribution constant and explored the effect of increasing habitat heterogeneity around that mean (i.e. by expanding the quality distribution symmetrically about 1). To further parse out the relative influence of high and low quality patches separately, we performed a second series of simulations where we expanded the range of the uniform quality distribution to skew towards either high or low quality patches (i.e. by expanding the quality distribution asymmetrically about 1). For these simulations, at each of the 10 longevities used above, we simulated 100 metapopulations for each of four

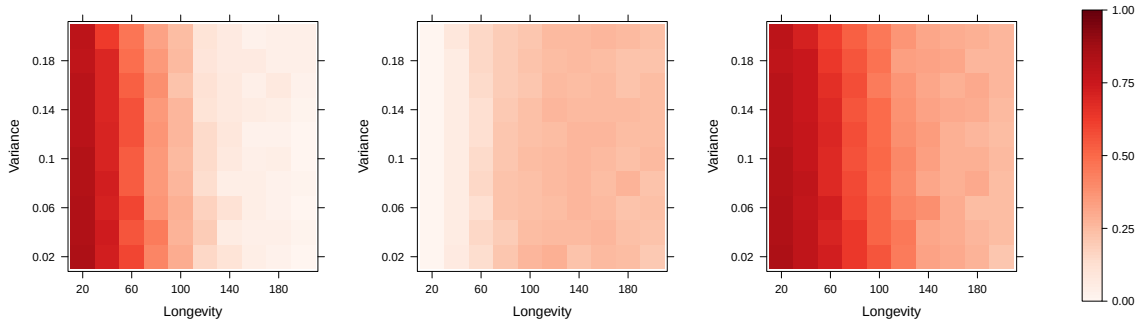

Fig. 1 Mean occupancy of (a) susceptible patches, (b) infectious patches, and (c) both, as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

patch quality distributions: low variance, (patch qualities ranging 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).

Continuous time stochastic simulations of the above model were implemented in the R language (R Core Team (2014)) using the Gillespie algorithm (Gillespie (1977)). Code is available at `https://github.com/clint-leach/Metapop-Disease`.

3 Results

In the baseline parameterization, infectious occupancy increased substantially with longevity, while susceptible and total occupancy decreased (Fig 1). Increasing the variance of the patch quality distribution around a mean of 1 had relatively little effect on average occupancy (Fig 1). However, though the mean showed little change, the variance in both susceptible and infected occupancy increased with the variance of the patch quality distribution, especially at intermediate longevities. This increase in variance was due to the greater variability in epidemiological outcome in high variance metapopulations, which generated both more pandemics and more disease die-outs than lower variance metapopulations. The three alternative parameterizations (models with lattice structure, equal extinction rates, and weak direct infection) produced qualitatively similar results, though pathogen spread in these models was generally more limited (Figs 5, 8, 11).

When we explored the influence of high and low quality patches separately (i.e. in simulations that expanded the patch quality distribution asymmetrically), we found that, relative to a low-variance metapopulation (patch qualities roughly between 0.75 and 1.25), shifting the distribution to include 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 differences observed at intermediate longevities (Fig 2(a)). Conversely, a shift towards low quality patches decreased

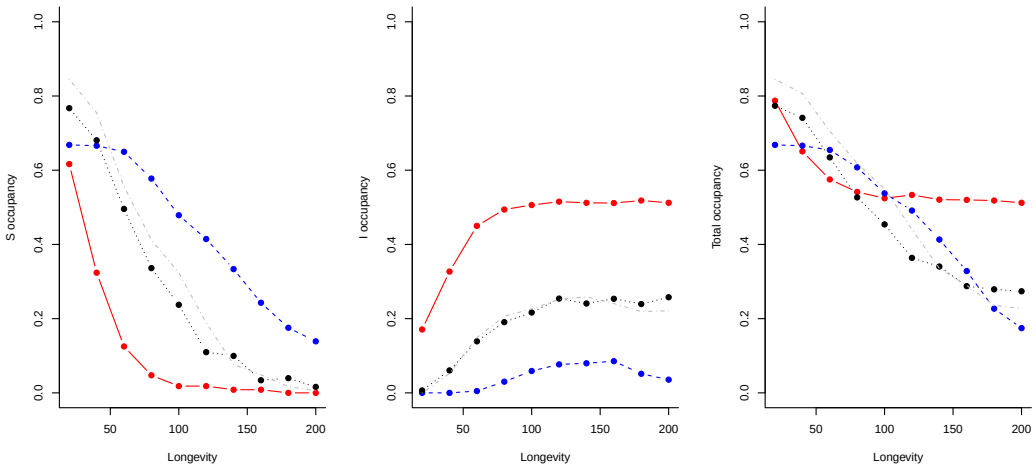

Fig. 2 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

infectious occupancy, while a shift towards high quality patches increased infectious occupancy (Fig 2(b)). These general trends were consistent across the three other parameterizations (Figs 6, 9, 12).

However, despite facilitating pathogen spread, 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 pathogen longevity. On the other hand, though low quality patch distributions inhibited pathogen spread at intermediate longevities, when high pathogen longevities led to pandemic dynamics, low quality distributions were unable to support infectious occupancy, resulting in an increase in host extinction events and a decrease in mean total occupancy. The model in which the extinction rate did not scale with quality (i.e. $\alpha = 0$) produced very similar results, though with less pronounced differences between the different quality distributions (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 longevities (Fig 6(c)).

To understand the mechanisms behind these effects, we further examined within patch dynamics in the baseline model. To compare across the full range of patch qualities, and to ensure the opportunity for both suscepible and infectious occupancy, we limit this analysis to 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

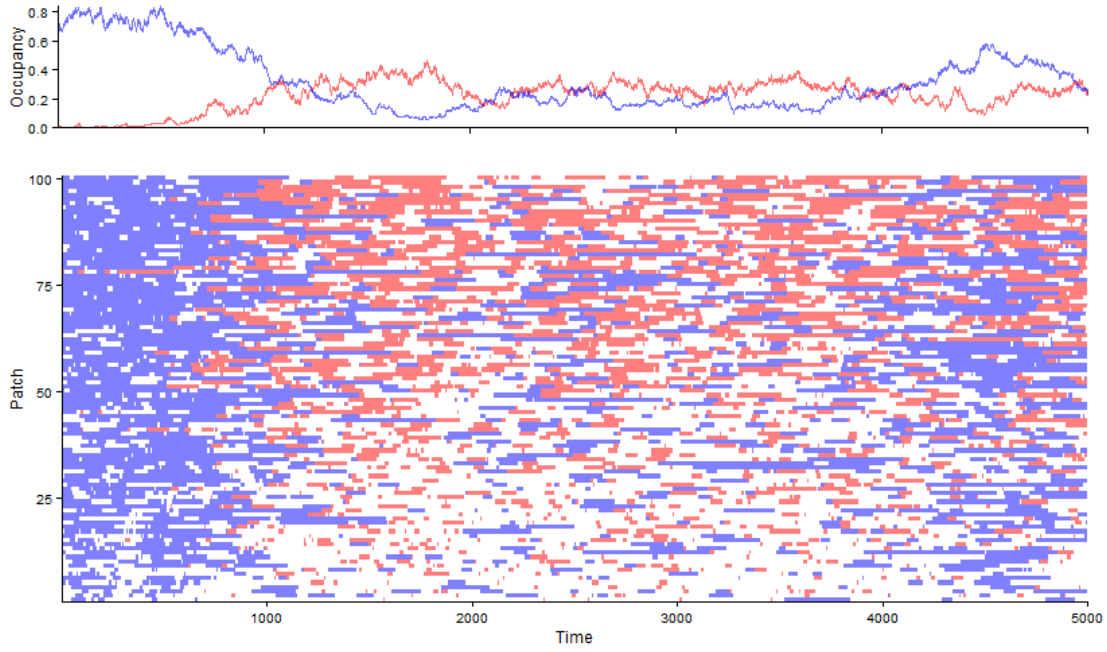

Fig. 3 Results from a single representative simulation with variance 0.2 and longevity 80. The top panel shows total occupancy of susceptible (blue) and infectious (red) patches over time. The bottom panel shows the state – susceptible (blue), infectious (red), or unoccupied (white) – of individual patches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

supported more consistent occupancy than low quality patches (Fig 3). However, as the epidemic progressed, high quality patches increasingly supported infectious occupants, while low quality patches supported infectious hosts 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 infectious transitions) than low quality patches (Fig 4). 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

This work demonstrates that the distribution of habitat quality in a metapopulation can have substantial impacts on the dynamics of environmentally transmitted pathogens and the resulting patterns of host occupancy. These impacts are driven largely by the contrasting effects of low and high quality habitat, and their relative balance in the metapopulation. These contrasting effects derive from differences in colonization and extinction rates, which in turn drive how low and high quality patches interact with and modulate pathogen transmission, either augmenting it

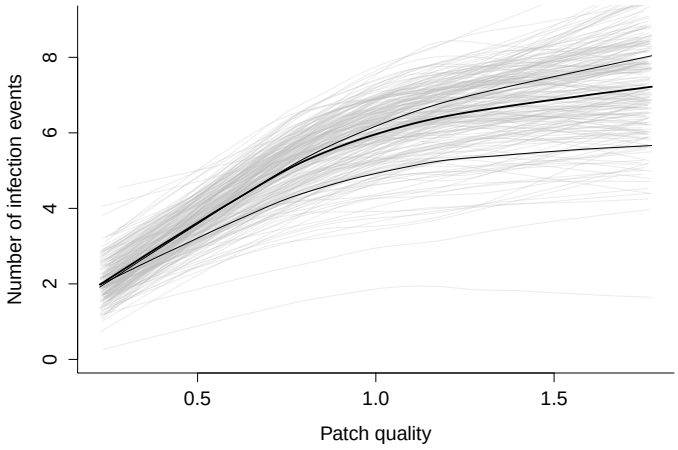

Fig. 4 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40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

in the case of high quality patches, or dampening it in the case of low quality habitat.

Since a patch's connectivity is determined by its quality (Eqn 1), 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 on high quality patches results from both transmission from a persistent environmental reservoir and from direct transmission from infectious 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 these patches allow it to spread to the rest of the metapopulation relatively easily. This process helps to maintain infectious occupancy throughout the metapopulation, which in turn feeds back on high quality patches to ensure a steady stream of infectious colonists that help maintain the environmental reservoir and 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 infectious occupancy in the metapopulation (Fig 2).

In contrast to high quality habitat, low quality patches help to limit pathogen spread and increase susceptible occupancy (Fig 2). Even though individual low quality patches, due to their high extinction rates and low colonization rates, are unable to support consistent occupancy (Fig 3), 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 infectious individuals. Moreover, the instability of low quality patches (i.e. the high extinction rate), together with the increased mortality of infectious populations, means that infectious 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 infectious populations generally decays before it has the opportunity to infect new susceptible colonists. As a result, low quality patches effectively represent a dead-end for the pathogen, reducing the number of patches from which it can spread, and thereby reducing infectious occupancy (Fig 2).

These findings, that low quality habitat inhibits disease spread while high quality habitat enhances it, suggest the presence of a trade-off between between processes that facilitate metapopulation stability at the expense of disease spread, and processes that hinder disease spread at the expense of metapopulation stability. This work thus expands on previous studies of disease spread in a metapopulation that focused on movement rate as the driver of this trade-off, with increased movement both improving metapopulation stability and increasing disease spread (Hess (1996), Gog et al (2002), Park (2012)). Shifting the patch quality distribution towards high quality patches in our model is in many ways analogous to increasing movement in the models of Hess, Gog, and Park, but by including habitat heterogeneity and metapopulation structure, we are able to investigate the role of individual patches and their contribution to total occupancy.

These contributions, and the relative strength of the above trade-offs, depend largely on the strength and structure of the different avenues of pathogen transmission. Broadly, there are three different scenarios that emerge from this and the above studies: conditions that support very weak pathogen spread, conditions that facilitate intermediate/endemic pathogen spread, and conditions that produce easy/pandemic pathogen spread. When pathogen spread is weak (e.g. due to low longevity, or weak direct transmission), high quality patches are a net benefit to the metapopulation, as their connectivity and stability facilitate greater occupancy with little additional pathogen spread.

However, in cases where the pathogen is able to spread more easily (i.e. at an endemic level), the addition of high quality patches functions similarly to increasing movement in the model developed by Hess (1996): increasing pathogen spread and as a consequence reducing overall occupancy (Fig 2c). In these cases, where pathogen longevity is intermediate, or dispersal is limited by metapopulation structure (e.g. a lattice), rather than serving as sources in the metapopulation, high quality patches become sinks. 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 maintaining patches where conditions are most favorable (Strasser et al (2010)). Generally, this means

that managers should focus on maintaining high quality habitat, but Strasser et al. (2010) show that stochastic disturbance (e.g. disease-induced mortality) can lead to cases where focusing on low quality patches is more effective in increasing population growth rate. Indeed, in these situations, our model suggests that low quality patch distributions, by hindering the spread of the pathogen and providing refuges for susceptible hosts, can produce higher occupancy than high quality distributions.

As pathogen longevity increases and environmental reservoirs become more widespread, the trade-off shifts and the addition of high quality patches is again a net benefit to overall occupancy. In these situations, environmental reservoirs overwhelm the susceptible refuges on low quality patches, such that they remain contaminated even across infrequent colonization events. Under these conditions of widespread infection, high quality patches help to maintain metapopulation stability in the face of disease-induced mortality, thereby increasing total occupancy relative to more balanced or low quality patch distributions.

These dynamics reflect the findings of Gog et al (2002), who found that under strong background infection from an alternative host, increasing movement increased total occupancy, despite facilitating pathogen spread. Park (2012), by adding environmental transmission to the mix, found more generally that in cases where background and environmental transmission were strong relative to direct transmission, increasing movement increased total occupancy monotonically, i.e. movement was 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 and direct infection is relatively less important), 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 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

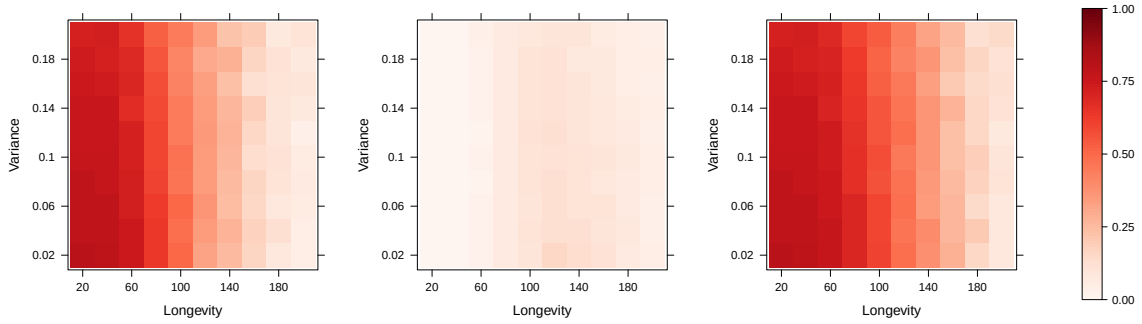

Fig. 5 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

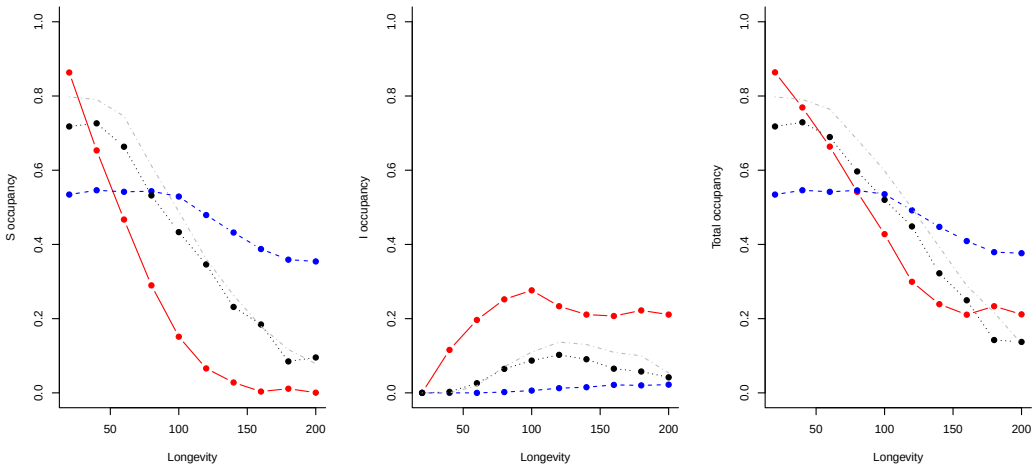

Fig. 6 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

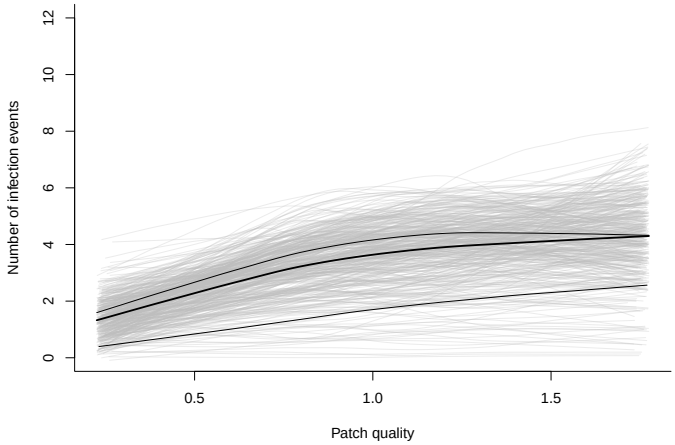

Fig. 7 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40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

318 *5.1.2 Extinction scaling,* $\alpha = 0$

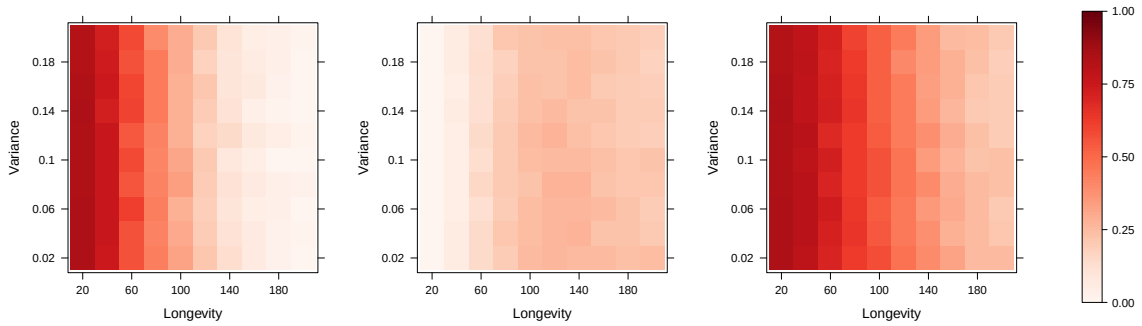

Fig. 8 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

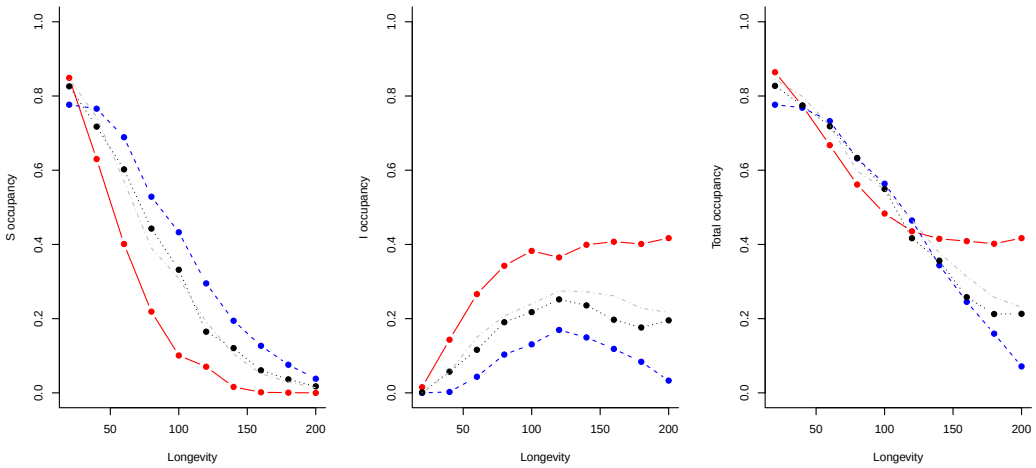

Fig. 9 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

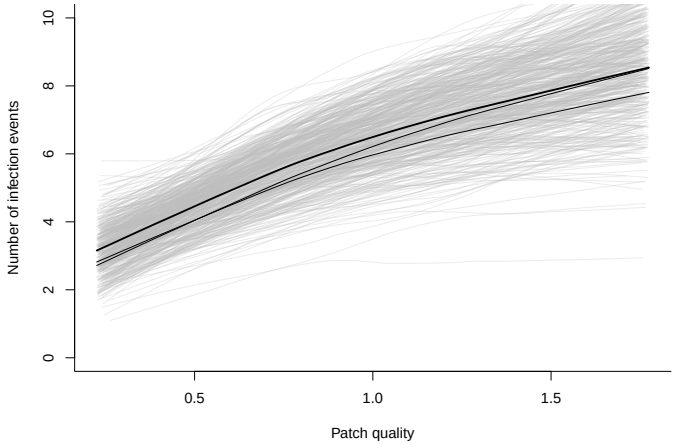

Fig. 10 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of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

319 *5.1.3 Weak direct transmission,* $\delta = 0.1$

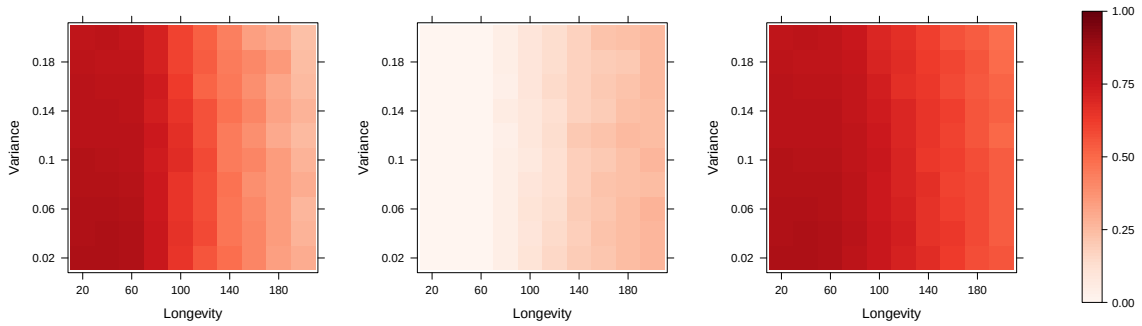

Fig. 11 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

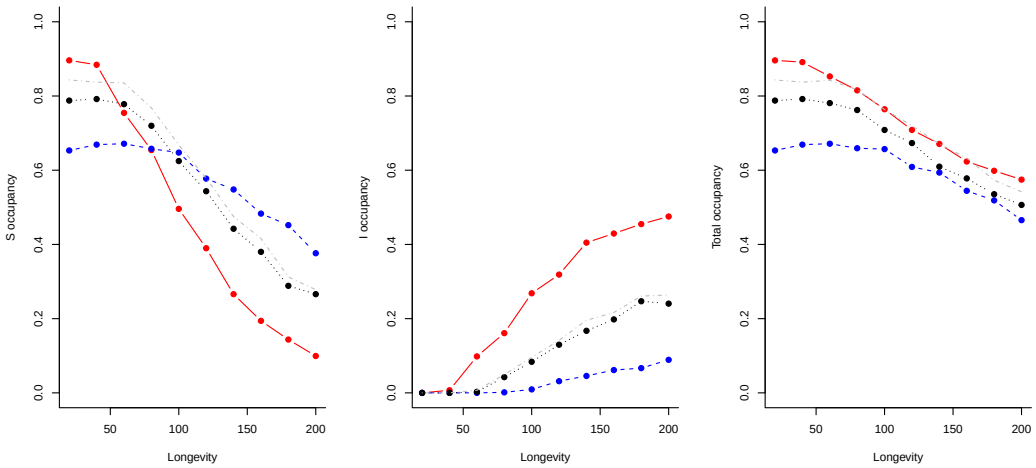

Fig. 12 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

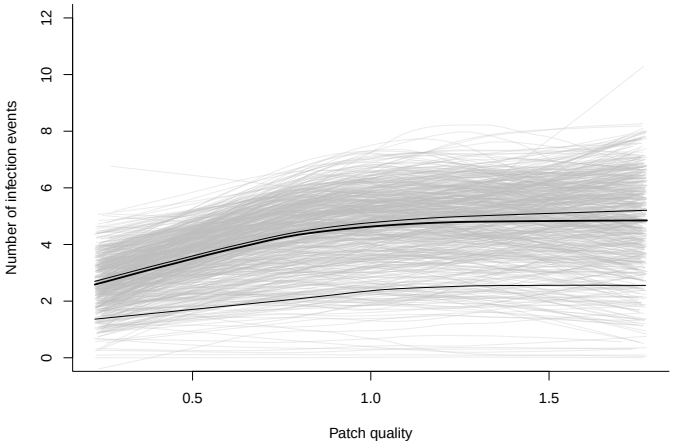

Fig. 13 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of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

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