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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 (but potentially with pathogen reservoir with infection rate of γ_i).

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, cite 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 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 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 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 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 non-symmetrically about 1). For these simulations, at each of the 10 longevities used above, we simulated 100 metapopulations for each of four

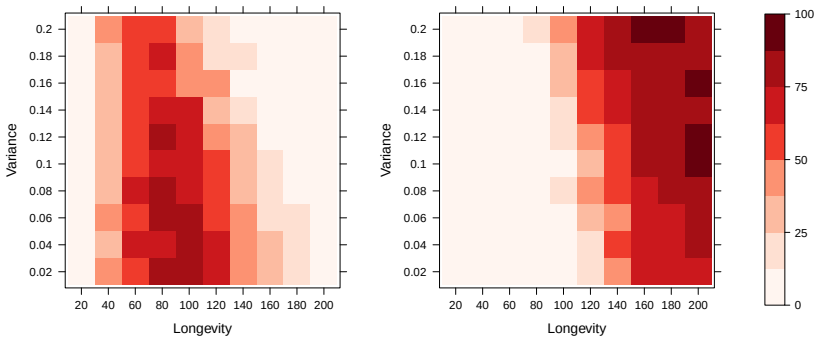

Fig. 1 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

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

Similar to above edits to methods, need to better distinguish the different analyses and what they're testing.

The pathogen persisted in 70% of the 10000 simulations, spreading through the entire susceptible host population (i.e. a pandemic) in 37% of the 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 (lattice structure, $\alpha = 0$, $\delta = 0.1$) produced qualitatively similar results, though pathogen spread in these models was generally more difficult (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 infectious 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 distribution toward low quality patches (distribution ranging from 0.25 to 1.25) increased mean susceptible occupancy, while shifting towards high quality patches

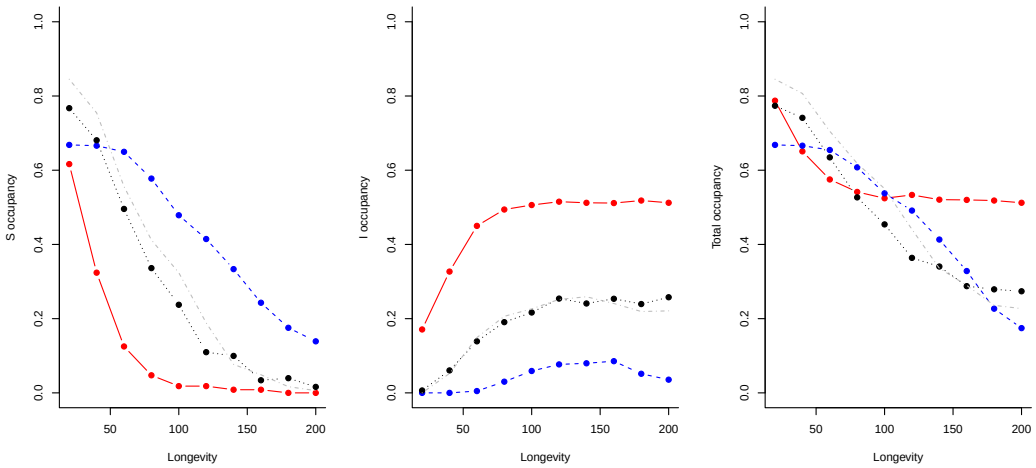

Fig. 2 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

(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 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, 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 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 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 progressed, high quality patches increasingly supported infectious occupants, while low quality patches supported infectiouss only briefly and infrequently (Fig 3). In

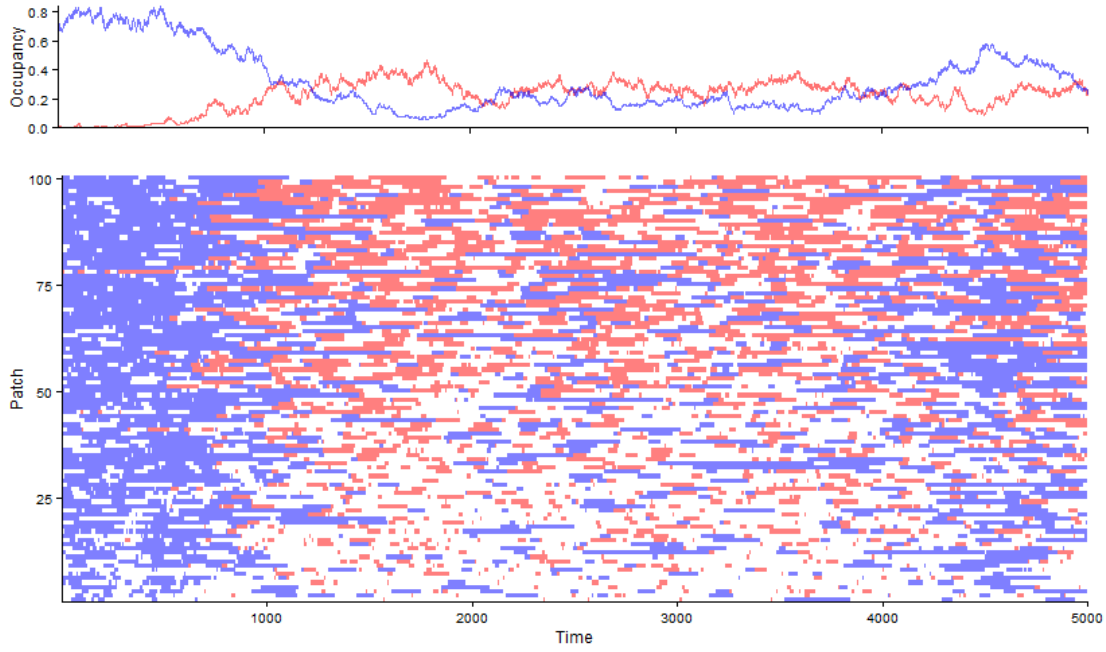

Fig. 3 Results from a single simulation with variance 0.2 and longevity 80. The top panel shows total occupancy of susceptible (blue) and infectious (red) patches over time. The bottom panel shows the state – susceptible (blue), infectious (red), or unoccupied (white) – of individual patches through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

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). 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 infectious. 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

Need to emphasize novelty of this work, especially compared to Hess, Gog, and Park. Discuss that movement in this model is not only heterogeneous, but also directional and what that means. Also consider reorganizing to put the more interesting elements at the front, and get rid of the less interesting parts (i.e. the first paragraph). As with notes for results and methods, need to be clear about which analyses support which claims, and specifically need to be careful about

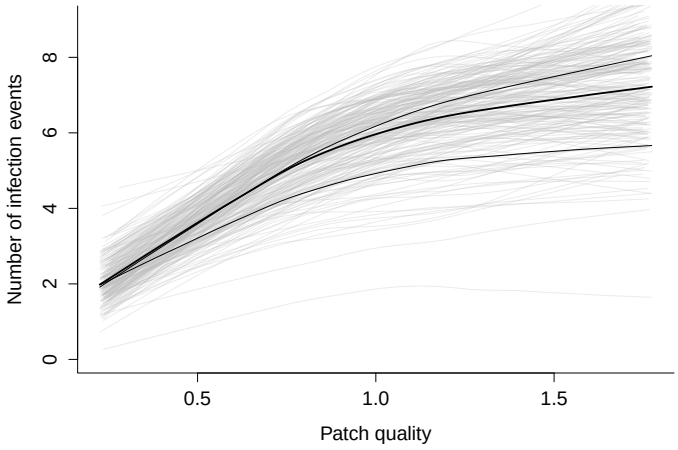

Fig. 4 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40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

discussing the different patch quality distributions and distinguishing between the symmetric and asymmetric distributions.

The positive relationship between pathogen spread and environmental longevity demonstrated in the theoretical model above is well documented in models of empirical systems. Using a model parameterized for chronic wasting disease in mule deer, Almberg et al (2011) find that increasing prion survival in the environment increases the resulting force of infection and peak prevalence, with the importance of reservoir 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. Look into George et al (2013).

By modeling the spatial spread of such an environmentally-transmitted pathogen, 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 as 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.

Emphasize own results relative to the contact network literature. Should try to avoid the "main model results suggest X, but sensitivity analysis models suggest Y" format and instead try to present a more complete picture/story. In the primary model, the increase in pathogen spread from high variance metapopulations likely results from the presence of high quality patches and their ability to overwhelm low quality patches at high longevity through a combination of their relative stability, the high connectivity due to the uniform spatial structure, and the relative strength of direct infection. The assumptions tested in the other models all weaken these effects – by constraining movement through the lattice structure; by limiting the influence of direct infection and thus the influence of colonists from infected high quality patches; and by reducing the stability of high quality patches relative to low – thus preventing low quality patches from being overwhelmed by the high quality patches.

The differences observed between the different parameterizations of the model highlight the fact that 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 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 the 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)).

High qualily patches also experience more infection events because they are able to support susceptibles long enough for them to get infected, whereas susceptibles on low quality patches are probably going to go extinct before they can become infected (though the effect persists when $\alpha = 0$ and is substantially diminished in the lattice model and when $\delta = 0.1$, so it seems that it is driven largely by connectivity and direct infection

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 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).

And Hess (1996) who finds that implementing a quarantine on centralized patches reduces pathogen spread.

In contrast, 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 represent a dead-end for the pathogen, reducing the number of patches from which it can spread, and thereby reducing infectious occupancy (Fig 2).

Though this model provides evidence that high quality patches can serve as traps and superspreaders, and low quality patches can serve as susceptible refuges, the effects of these processes on total metapopulation occupancy depend on the strength and structure of the two transmission pathways. Need more here? In particular, these findings suggest the presence of a trade-off between facilitating metapopulation stability and inhibiting disease spread, with a pathogen's environmental longevity and ease of spread controlling the roles of different quality patches with respect to this trade-off. When pathogen spread is limited or slow (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.

Maybe lay out the big picture in this paragraph: that there are essentially three different scenarios, very low pathogen spread, intermediate/endemic pathogen spread, and easy/pandemic pathogen spread and then go through each one and its consequences on the roles played by high and low quality habitat.

However, in cases where the pathogen spreads 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 reducing overall occupancy (Fig 2c). In these cases, where pathogen longevity is intermediate, or pathogen spread is limited by metapopulation structure, 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 stochas-

tic 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, we see that low quality patch distributions, by hindering the spread of the pathogen and providing refuges for susceptibles, produce higher occupancy than high quality distributions.

What does this model offer beyond the literature cited? WRT Hess 1996, the extension to explicit habitat patches offers insight into the roles of specific patches; even though connectivity is an important driving factor in this model, it also allows us to consider the role of other processes (e.g. extinction, explicity pathogen transmission in space).

Also discuss Sharp and Pastor (2011), who show that management actions that increase carrying capacity can increase disease spread and destabilize population.

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).

As with the notes above, emphasize not just how this work fits with other literature, but also what it adds. WRT these other metapop disease modeling papers, may not want to equate increased patch quality with increased movement so easily and instead delve more into what including patch quality offers us. "This refelcts the the results of blah in that ..., but offers the additional insight that ..."

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.

Again, use this last paragraph not just to summarize the results, but to emphasize the novelty of these insights.

5 Appendix

5.1 Sensitivity Analyses

5.1.1 Lattice structure

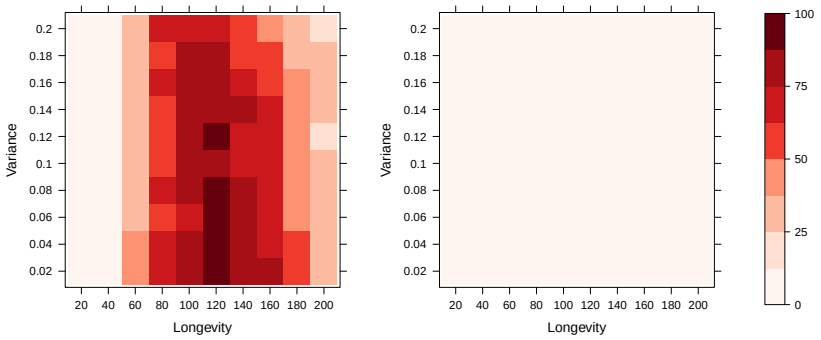

Fig. 5 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

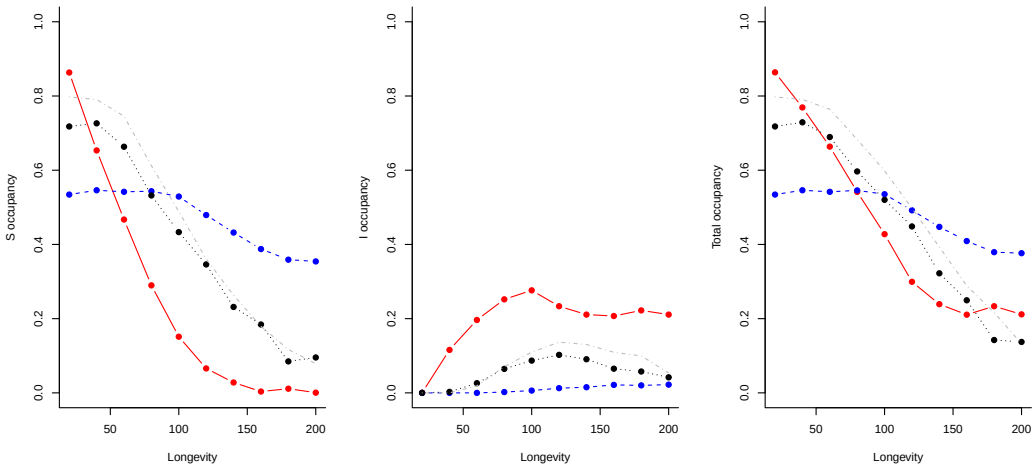

Fig. 6 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

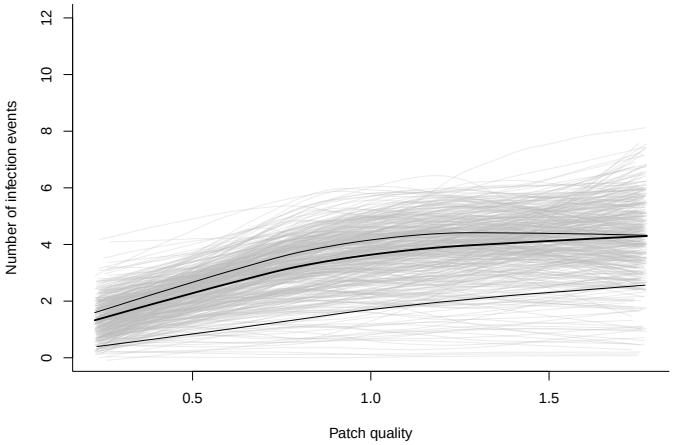

Fig. 7 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of 40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

373 *5.1.2 Extinction scaling,* $\alpha = 0$

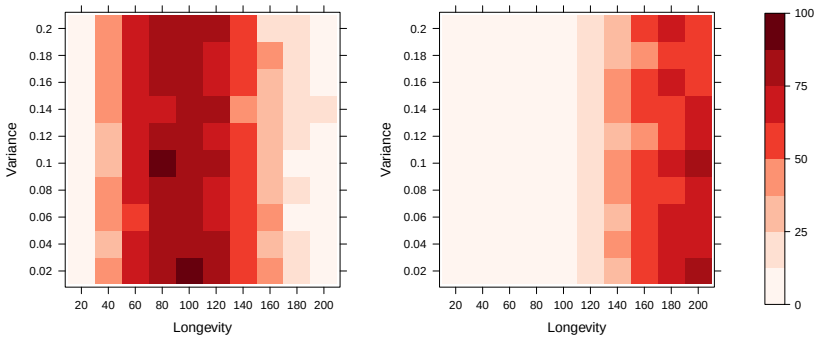

Fig. 8 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

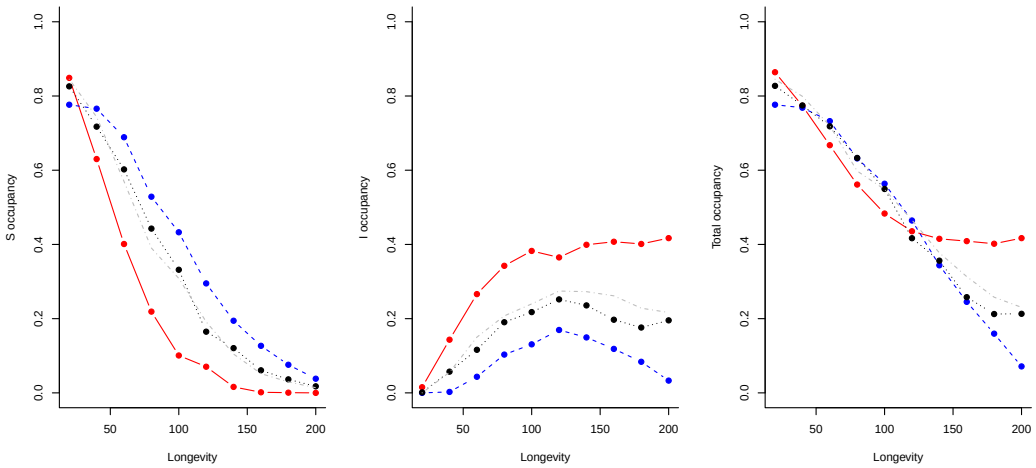

Fig. 9 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

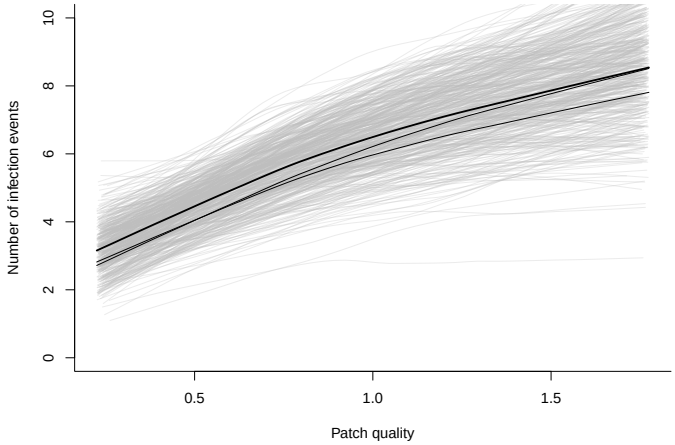

Fig. 10 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of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

374 *5.1.3 Weak direct transmission,* $\delta = 0.1$

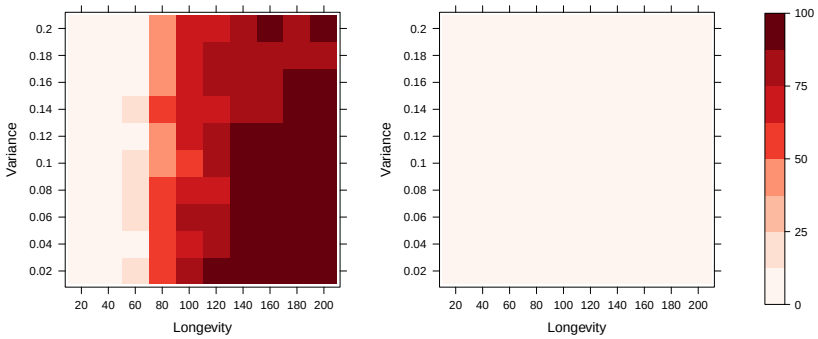

Fig. 11 Percent of simulations resulting in (a) endemic disease (both susceptibles and infectiouss persist) and (b) pandemic disease (no susceptibles persist), as a function of the pathogen's environmental longevity (half-life of infectivity decay) and the variance of the patch quality distribution.

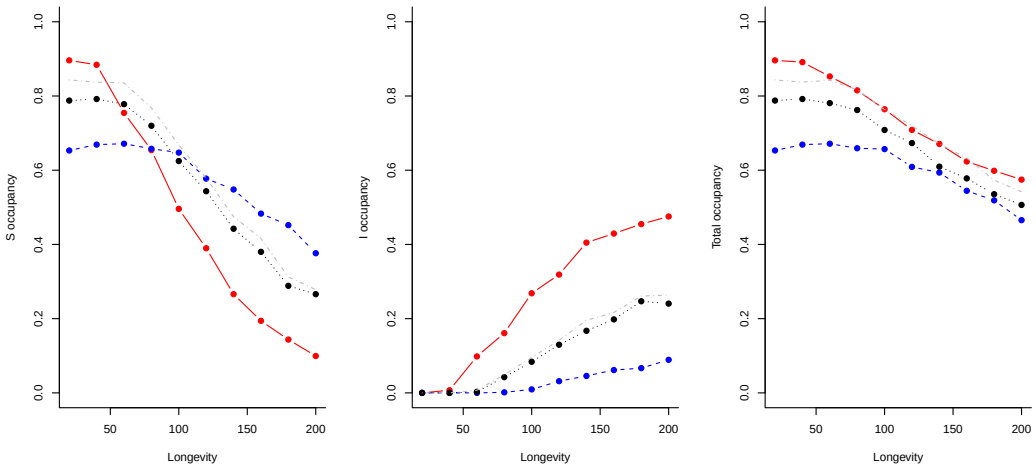

Fig. 12 The effect of the presence of high and low quality patches on (a) mean susceptible occupancy, (b) mean infectious occupancy, and (c) mean total occupancy, measured relative to a low variance metapopulation. Red solid lines show hiqh quality metapopulations, blue dashed show low quality, and black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

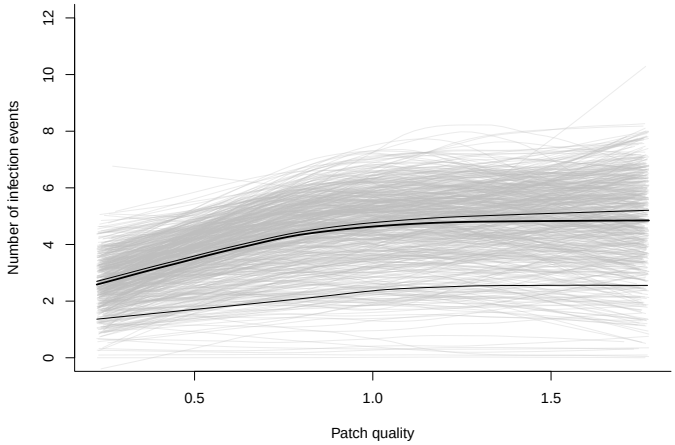

Fig. 13 The number of infection events (S to I transitions) for individual patches in endemic simulations. Light gray lines show smoothed fit for single simulations, while the thick black line shows the smoothed fit over all simulations. The thin black lines show the smoothed fits for longevities of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

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