Cellular Landscapes and Infectious Disease

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1. Introduction

Recent patch models of epidemics (PME) have been based on models from metapopulation biology, which have been developed in conservation ecology (Lloyd and May 1996). PME primarily are used to examine stability conditions, i.e., when infection becomes absent, endemic, or at stable equilibria.

Some of the results of mathematical PME have counterintuitive results such as less disease prevalence with higher mobility of infectives (Allen et al. 2006). More spatially explicit representations of landscapes in conservation ecology have shown different results for species dynamics than those shown for many metapopulation models (e.g., Malanson 2002). Cellular simulations are used to determine how spatial heterogeneity of disease risk from an underlying environment and the spatial behaviour of individuals affect disease dynamics.

2. Methods

Here, “discretized continuous fields” (sensu Wilson and Burrough 1999) are used to represent heterogeneous landscapes of risk of infection and risk of recovery in a toy model of infectious disease for two states, infectives and susceptibles. Healthy and unhealthy cells in the landscape are derived from either a fractal surface generated by the random midpoint displacement method, through simple geometrical structures (fig. 1). Additional spatial variation is added in some cases by imposing barriers to capture quarantine and *cordon sanitaire* methods of limiting epidemics.

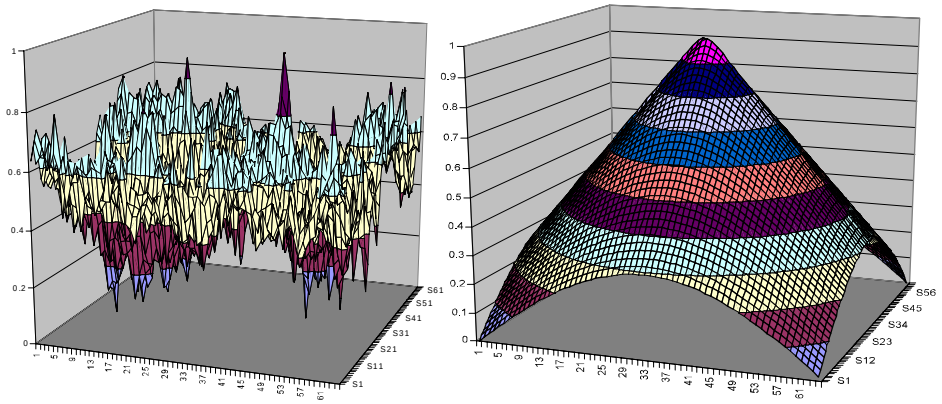

Figure 1. Fractal and structured representations of the risk quality of the landscape (actually wrapped as a torus in the simulation).

The spatial spread of individuals is simulated by random walks, biased walks, and varying speed – and with differences between infected and non-infected individuals. The population is held constant with neither births nor deaths. Initial conditions include both a homogenous widespread distribution and a few localized infectives.

3. Results

Differences in spatial structure of the environment and in the spatial behaviour of the individuals affect outcomes (e.g., fig. 2). When the initial conditions are a small group of invasives among many susceptibles, the equilibrium value of the invasives is in nonlinear proportion to the total population, not the proportion for the two found for the initial condition of widespread distribution of infectives and susceptibles. Equilibria depend on the relations between the spatial extent of the environment and the number of individuals because infection is due to co-occurrence as well as environmental quality.

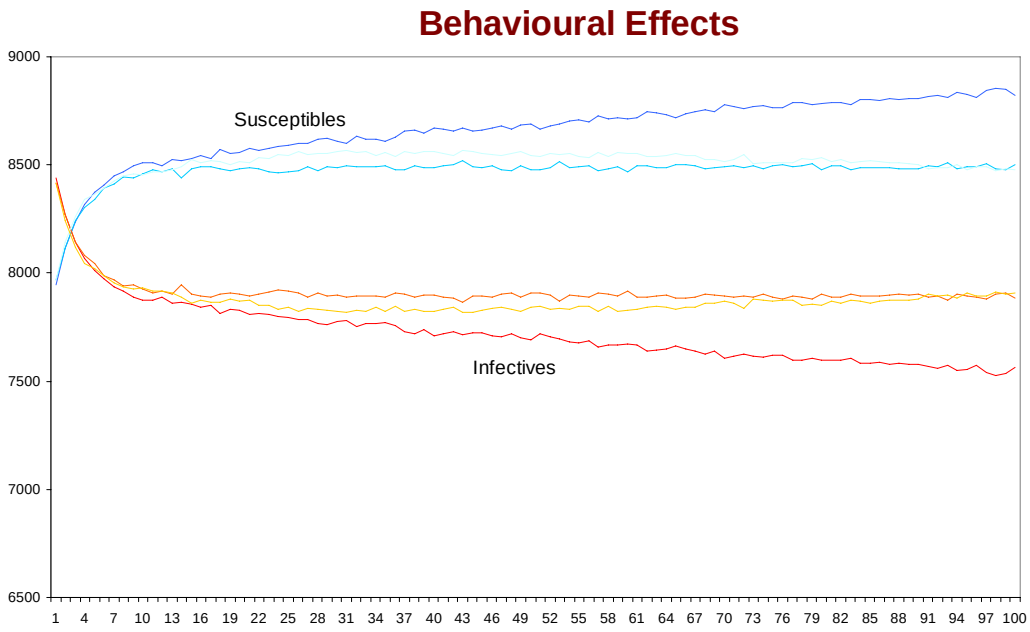

Figure 2. Biased and random walks in relation to environmental quality alter outcomes.

Infectives increase from random to fractal to simply structured risk landscapes, but different fractal dimensions of the landscape surface of risk are unimportant. Pronounced spatial structures such as cones or hills produce definite locations of infectives and susceptibles, but small differences in numbers; it appears that a spatially heterogeneous environment segregates the two and thus limits infection (fig 3). The increased interaction created by biased walking toward areas of lower infection risk is balanced by the strong spatial pattern of the lower risk on the conical surface.

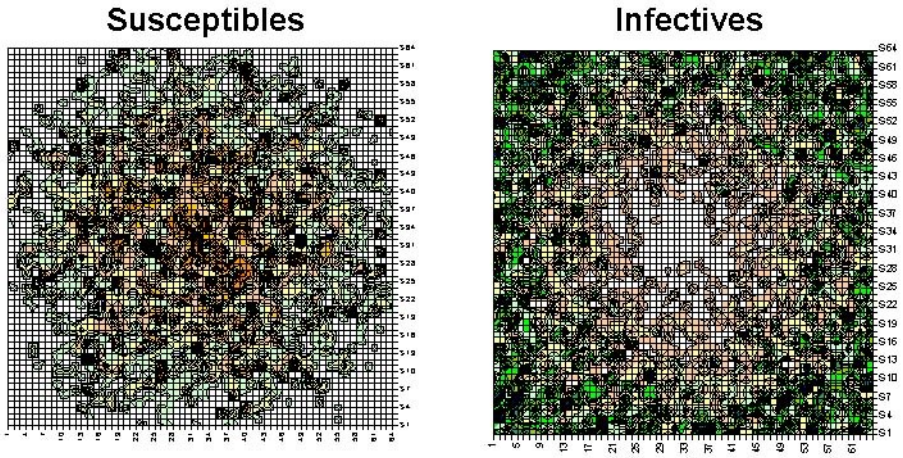

Figure 3. On a conical surface of infection risk, infective and susceptible individuals are segregated even though both walk toward the area of low risk.

Although the fractal landscapes might be expected to be a more realistic representation of the spatial variation in infection risk for an organism, more has been learned in this case from the simplest heterogeneous landscape: a cone of risk. Although some results merely confirm the mathematical approach of Allen et al. (2006), the additional spatial detail seems worth pursuing in order to fully explain the process. Additional research is called for on the relationships between the number of cells in categories of risk and the number of individuals, as well as more realistic representations of natality, mortality, latency, and immunity.

7. References

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