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# Empirical evidence of spatial thresholds to control invasion of fungal parasites and saprotrophs

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## Summary

• The ability to forecast invasion of harmful and beneficial organisms is becoming increasingly important in agricultural and horticultural production systems as well as in natural plant communities.
• In this paper we examine the spread of a fungus through a population of discrete sites on a lattice, using replicable, yet stochastically variable experimental microcosms.
• We combine epidemiological concepts to summarise fungal growth dynamics with percolation theory to derive and test the following hypotheses: first fungal invasion into a population of susceptible sites on a lattice can be stopped by a threshold proportion of randomly removed sites; second random removal of susceptible sites from a population introduces a shield which can prevent invasion of unprotected sites; and third the rate at which a susceptible population is invaded reduces with increasing number of randomly protected sites.
• The broader consequences of thresholds for fungal invasion in natural and agricultural systems are discussed briefly.

**Key words:** invasion thresholds, biological control, percolation, *Rhizoctonia solani*, epidemiology.



## Introduction

Fungi play an essential role in ecosystem functioning via processes ranging from nutrient cycling and maintenance of soil structure (Copley, 2000; Yaalon, 2000) to causing diseases in agricultural systems (Gilligan, 2002a), as well as determining plant abundance, species diversity and succession in natural environments (Packer & Clay, 2000; Van der Putten, 2000). Underlying these processes is the ability of fungi to spread by mycelial growth or spore movement and to invade and persist in structurally and nutritionally heterogeneous environments. For many microorganisms spread occurs in environments with food supply in discontinuous, discrete patches, and with a spatially heterogeneous biota, which can have a considerable impact on the ability of a fungus to colonise soil aggregates (Toyota *et al.*, 1996). Typical examples include the colonisation of roots by pathogenic and mycorrhizal fungi, the colonisation of particulate organic matter in soil, or networks formed by wood-decaying basidiomycetes. Despite considerable empirical knowledge on the foraging behaviour of fungi (Cooke & Rayner, 1984; Rayner, 1991; Ritz, 1995; Boddy, 1999), and recent developments of mathematical models for fungal growth (Davidson *et al.*, 1996; Stacey *et al.*, 2001), simple criteria for invasion of fungi into heterogeneous environments are still lacking. Underlying the invasion is the complex interplay between the nonlinear dynamics of fungal growth and the spatial heterogeneity of the soil environments. Here we evaluate empirical evidence and theoretical arguments for the existence of spatial thresholds to control invasion of soil-borne fungi in heterogeneous populations of substrates.

In this paper we deal with thresholds for invasion of fungi by mycelial spread. Our approach is centred on the hypothesis that the invasion of soil-borne fungi by mycelial spread through populations of organic substrates, such as fragments of plant tissues (roots, leaves), or populations of plants and living roots, is analogous to the spread of infection through discrete host populations. The essential features of fungal spread involve the primary colonisation of available (cf. susceptible, $S$) fragments from resting spores or introduced propagules in soil, followed by secondary colonisation as mycelium spreads from

a colonised (cf. infected, *I*) towards another susceptible fragment, till the colonised fragment is depleted of nutrients (cf. removed, *R*). The dynamics of fungal spread can then be described by compartmental *S-I-R* models, which have been widely studied in epidemiological contexts (Van der Plank, 1968; Burdon *et al.*, 1989; Anderson & May, 1991; Jeger, 2000; Shea *et al.*, 2000; Gilligan, 2002b). Crucially, these studies have led to criteria for invasion and persistence of disease in animal, human or plant populations. There is a significant body of literature on spatial invasion for epidemics on lattices, which can be used to formulate criteria for fungal invasion (Grassberger, 1983; Bunde & Havlin, 1991; Stauffer & Aharony, 1991; Cardy, 1996; Newman, 2002). In particular, these studies show that there is a threshold probability of spread between individual sites (cf. fungal spread from a colonised to an uncolonised substrate) above which invasion of populations of substrates occurs. In addition, percolation theory predicts that if a certain fraction of sites is unavailable for colonisation, invasion stops. The ecological importance is that competitors and antagonists can prevent invasive spread by colonising not all, but a threshold proportion of sites.

In this paper we quantify thresholds to block invasion of fungal parasites and saprotrophs by mycelial spread. First, we rehearse the invasion theory for fungal spread previously described and tested by Bailey *et al.* (2000), and explain how the theory also predicts that a threshold density of randomly protected sites can prevent invasion. Secondly, we quantify the invasion of the saprotrophic and parasitic fungus *Rhizoctonia solani* Kühn through replicated populations of discrete nutrient sites. An increasing number of removed sites selected at random from a population is used as an analogue of locally successful but spatially incomplete exclusion from a site by a range of processes including competition, antagonism (biocontrol) and chemical control. In particular we demonstrate the following:

- biological invasion into a population of susceptible sites on a lattice can be stopped by a threshold proportion of randomly removed sites;
- random removal of susceptible sites from a population introduces a shield which can prevent invasion of unprotected sites;
- the rate at which a susceptible population is invaded reduces with increasing number of randomly protected sites.

The consequences of thresholds for fungal invasion in natural and agricultural systems are discussed. More generally, we discuss how the analysis can be extended to a broad class of microorganisms invading heterogeneous environments (similar to the lattices presented here) with plant communities and agricultural crops occurring in mosaics of many species in patterns ranging from fields (e.g. the distribution of fields of susceptible crops or resistant varieties), to within-crop genetic diversity (Burdon *et al.*, 1989; Zhu *et al.*, 2000).

## Materials and Methods

### Percolation theory and invasion of disease

Grassberger (1983) showed that a general *SIR* epidemic model, in which susceptible hosts (*S*) become temporarily infected (*I*) after which they become permanently immune (*R*), can be treated as a percolating system. Percolation represents the simplest model of a spatial system. Consider a lattice where each site is randomly occupied with probability $1 - p$ or empty with probability $p$ (Fig. 1a). For simplicity, we assume that fungal spread can only occur between neighbouring susceptible sites, and that this occurs with a probability of 1. For botanical epidemics, especially soil-borne disease, transmission is often limited to a local neighbourhood, although spores do offer opportunities for larger scale interactions. In statistical physics, such a network model with local interactions is referred to as site percolation (Bunde & Havlin, 1991).

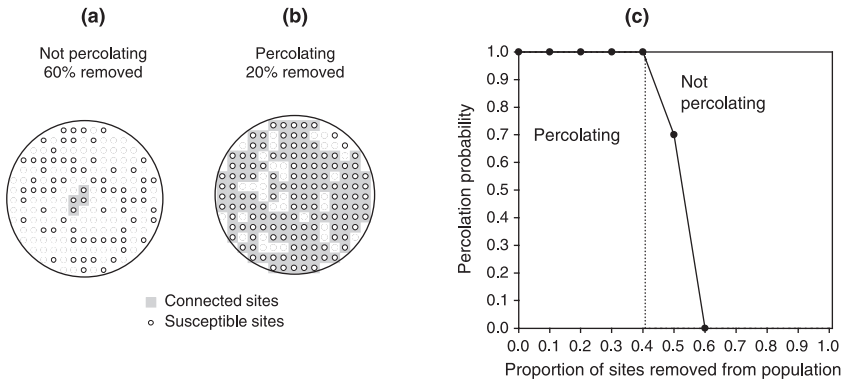


**Fig. 1** Examples of microcosms comprising nutrient sites on a square lattice from which the proportion of sites that is removed equals (a) 0.6, which results in only a few sites forming a finite connected network of neighbouring sites, or (b) 0.2, with nearly all sites part of an infinite connected network. (c) at a critical fraction of randomly removed sites percolation theory predicts that the connected patch switches from infinite to finite (dotted line); in smaller, experimental microcosms this transition is less steep but still highly nonlinear (continuous line; each point is the proportion of 10 simulated microcosms in which spread initiated from the central site can reach the edge of the system via connected neighbouring sites). Removed sites are shown by gaps in the lattice.

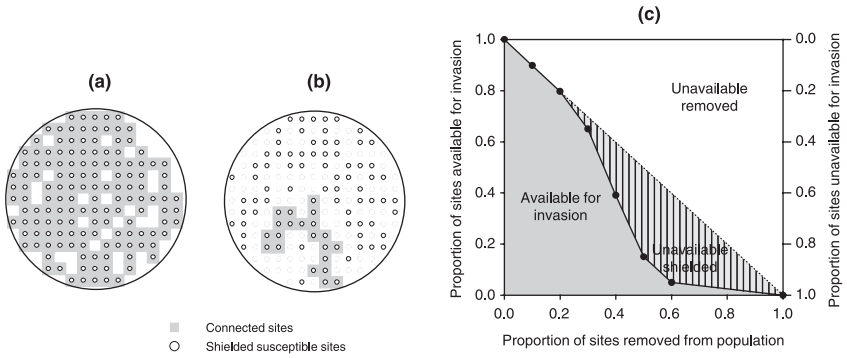


**Fig. 2** Shielding of unprotected sites from invasion. (a) example of a population in which 20% of the sites are removed, with all remaining sites staying connected with each other, providing many pathways connecting the central site with the edge of the system; (b) example of a population in which 50% of the sites are removed. From the central site only a few tortuous pathways of neighbouring sites are available for spread, most of which are dead-ended. (c) with increasing proportion of sites randomly removed from a population, the fraction of remaining sites that are shielded from invasion increases.

Percolation theory relates the fraction of sites that are removed from a susceptible population to the probability of invasion. Fungal spread is assumed to be constrained to sites that belong to the same cluster. Two sites belong to the same cluster if they are connected by a path of directly neighbouring sites that are available for colonisation. The validity of the assumption that fungal spread is constrained will be shown below. If the proportion of removed or protected sites in the population is large, the sites available for colonisation form small clusters or occur as isolated sites (Fig. 1a). A central cluster spanning the system size does not exist, and the fungus cannot invade. At low proportions of removed sites, on the other hand, many pathways between opposite edges of the system exist, and in principle the fungus can invade (Fig. 1b). At some intermediate fraction of removed sites a threshold proportion ($p_c$) must therefore exist above which for the first time a fungus can percolate from one end of the system to the other. Below the threshold we have noninvasive spread (i.e. successful control), whereas above the threshold the fungus can spread invasively.

The precise value of the threshold depends on the lattice. Lattices can range from random lattices to lattices with some element of spatial correlation. To test if the theory will hold for fungal spread, we concentrate here on spread through a simple square lattice. For a two-dimensional, infinitely large system with a square lattice, the percolation threshold of randomly removed sites is *c.* 0.407 (Bunde & Havlin, 1991). For smaller systems, the spread is always finite, and it is more appropriate to quantify the probability that a cluster of neighbouring sites spans the length of the population. For simulated populations of 160 sites on a square lattice (identical to the experimental system described below) the proportion out of 10 replicates in which the cluster of neighbouring sites initiated from the centre reaches the edge of the system dropped dramatically with increasing proportion of removed sites (Fig. 1c). Although the transition is less abrupt than for an infinite system, the relationship between the fraction of removed sites and the ability to invade remains highly nonlinear (Fig. 1c).

In addition to the invasion threshold for randomly removed sites as introduced above, two other features are important for spread through a population. Firstly, the fraction of available sites that are shielded from invasion increases as the proportion of sites removed from the population increases (Fig. 2). This is distinctly different from a nonspatial system (or an epidemic dominated by primary infection), where the fraction of sites unavailable for colonisation is directly proportional to the fraction of removed sites (dotted line in Fig. 2c). In a spatial system, removal of sites introduces an additional benefit by shielding available sites from invasion (Fig. 2c). Secondly, the sites belonging to the same cluster are less connected. Typically, small pockets of well connected sites are connected with each other by bottle necks of single occupiable sites (Fig. 2a,b). The pathway for spread becomes more tortuous with fewer neighbouring sites, which ought to reduce the rate of fungal spread.

## Empirical quantification of fungal invasion

To test if the features of percolation introduced above are observed in experimental systems, we quantified the patch dynamics of the fungal plant pathogen and saprotroph *Rhizoctonia solani* Kühn (R5, AG2-1, IMI385769) following introduction into replicate populations of agar sites (Bailey *et al.*, 2000). Each site comprised a 3-mm diameter agar dot of 10% (w/v) PDA. We summarised the fungal spread between two individual neighbouring sites in probability profiles (Gilligan & Bailey, 1997). Probability profiles were determined by placing pairs of agar dots comprising a colonised donor and an uncolonised recipient site at 8, 10, 13, 16, 19, 22, and 25 mm apart ($n$ = 20). Recipient dots were assessed daily for colonisation. At each distance, the probability of colonisation increases with time to an asymptotic level as the donor site becomes depleted of nutrients. Fig. 3 shows the probability of colonisation on day 12, after which no further changes in the colonisation were observed. To ensure a high probability of colonising a neighbouring site while at the same time minimising the probability of colonising the site beyond nearest neighbour we selected an intersite distance of 8 mm for our population studies.

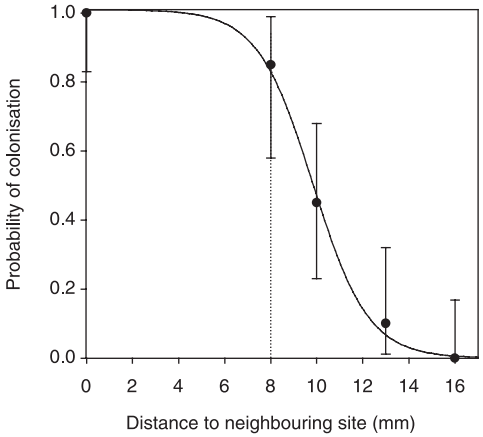


**Fig. 3** Spread of *R. solani* from a donor colonised agar site to a recipient uncolonised site, characterised by a probability profile that describes the change in colonisation with distance. Each point is the number of successful colonisations out of 20 replicates; the colonisation efficiency is summarised by a sigmoidal curve. The vertical line at 8 mm represents the distance between neighbouring sites that was selected for the population experiments. The bars show the 95% lower and upper binomial confidence limits.

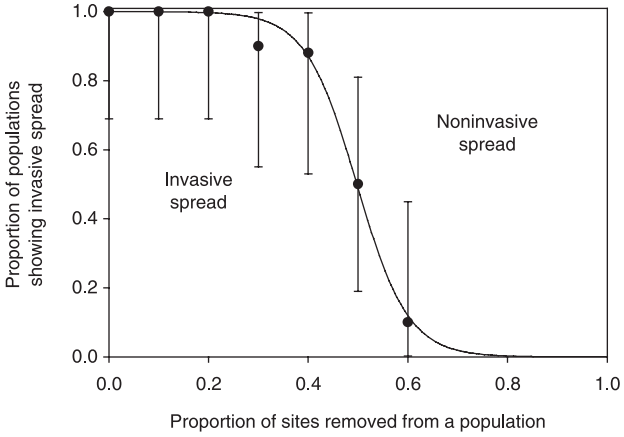


**Fig. 4** The fraction out of 10 populations in which the fungus *R. solani* spreads invasively as affected by the proportion of sites that were removed from the population. The continuous line represents a sigmoidal curve fitted to the data. The bars show the 95% lower and upper binomial confidence limits.

For the population studies, sites of agar dots were spotted onto a square lattice in large Petri plates (140-mm diameter) at 8-mm apart. A fraction of 0, 10, 20, 30, 40, 50, or 60% of the lattice points was randomly selected to remain empty (Fig. 1a). Each population was replicated 10 times using independent randomisation schemes, leading to a total of 70 populations. The central agar site of each population was inoculated with a single hyphal strand (1 mm length) removed from the edge of a 4-d-old colony of *R. solani* growing on water agar. To avoid desiccation of the agar, moist filter paper was placed into the lid of each plate. The plates were sealed and incubated in the dark at 23°C, and assessed daily for 28 d using a dissecting microscope (magnification × 40) and the number and locations of colonised sites recorded.

Spatial maps of the dynamics of fungal colonisation are used: first to count the changes in cumulative number and the daily increment in newly colonised sites, hereafter referred to as the rate of invasion, and second to identify those replicates that spread invasively. We quantify how the rate of invasion changes with time as the fungus spreads through the population of susceptible sites, and analyse how these dynamics are affected by the removal of sites in particular as small clusters of well-connected susceptible sites linked through bottle necks are being formed. We relate invasiveness to the final pattern of colonised sites and define noninvasive spread as those replicates in which fungal spread initiated from a single (central) site fails to reach the edge of the population. By default the remaining replicates that did reach the edge of the system are classified as invasive (cf. Bailey *et al.* (2000)). We subsequently quantify the ability to invade as the proportion of replicate populations with invasive spread.

## Results

### Threshold for invasion

The central question in this paper is how many sites need to be removed to prevent invasion of the fungus. There is a transition from invasive to noninvasive spread with increasing number of removed sites, even though these are randomly dispersed throughout the population (Fig. 4). Removal of up to 30% of the sites had a minimal effect on the ability of *R. solani* to invade. Removal of more than 60% of the sites was sufficient to stop invasion in almost all replicates (Fig. 4). The relationship between the proportion of patches showing invasive spread and the fraction of removed sites is not as sharp as would be expected for an infinite system, but closely followed the simulated value for our replicated smaller microcosms (Fig. 1c).

### Shielding of susceptible sites

The spread of *R. solani* through populations of discrete nutrient sites follows closely the pattern of neighbouring susceptible sites, with only occasional spread to sites beyond nearest neighbour (Fig. 5a). For the populations in which every lattice point contains a susceptible site, rapid wave-like spread occurs in all directions. Nevertheless, the spread is not homogeneous and small gaps of one or two sites appear even in these populations. For populations in which sites are removed, spread appears more directional, reminiscent of a line of colonised sites weaving through the population, occasionally encountering a small patch of interconnected sites (Fig. 5a). Spread through these populations appears more irregular with large gaps.

The fungus was unable to colonise all available sites in any of the treatments (Fig. 5b). In a population without removed sites, however, nearly all sites became colonised (90%). With

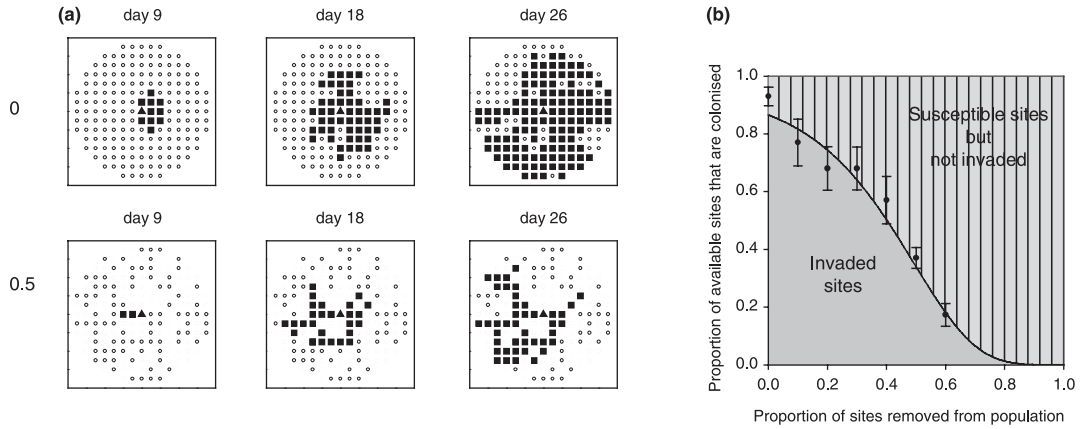


**Fig. 5** Spatial dynamics of fungal spread through populations with randomly removed sites. (a) examples of maps showing the spread of *R. solani* on 9, 18, and 26 d after inoculation of the central site in a population of sites distributed on a square lattice with all sites occupied or 50% of the sites removed. (b) the mean ($n$ = 10) relationship between the proportion of sites removed from the population and the proportion of sites that ultimately are colonised. Legend: o susceptible sites; solid square, sites that have been colonised by *R. solani*; solid triangle, site of inoculation. Gaps are shown by empty sites; SE shown by bars.

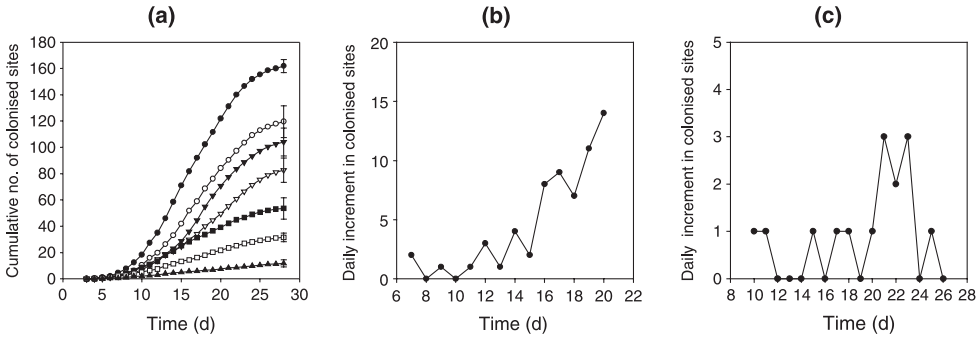


**Fig. 6** Temporal dynamics of fungal spread through populations with randomly removed sites. (a) mean colonisation ($n$ = 10) for populations with a fraction of removed sites of 0 (solid circle), 0.1 (open circle), 0.2 (solid inverted triangle), 0.3 (open inverted triangle), 0.4 (solid square), 0.5 (open square), or 0.6 (solid triangle). SE on last sampling day shown by bars. (b,c) two typical examples of daily increments showing (b) accelerating rate of invasion for populations in which all sites are occupied and (c) a reduced and variable rate of invasion with occasional outbursts for epidemics in which 40% of the sites are removed. Data are only shown until the colony reached the edge of the population.

increasing number of removed sites, the ability of the fungus to access the remaining sites dropped rapidly. This confirms that removal or protection of sites in a population has the additional benefit of shielding remaining sites from colonisation. This experimental result is in agreement with the simulations shown in Fig. 2(c).

### Rate of fungal invasion

The rate of fungal invasion is summarised by colonisation progress curves, analogous to disease progress curves ordinarily used to describe epidemics. The mean dynamics of invasion typically describe a sigmoidal shape (Fig. 6a). All replicates tend towards an asymptote even though not all available sites have become colonised. There is a significant reduction in the cumulative number of sites that have become colonised at each time with an increasing fraction of removed sites.

The sigmoidally shaped mean dynamics (Fig. 6a) summarise average behaviour of the populations, but also obscure some interesting aspects of the dynamics that are observed in each replicate separately. If all sites in the population are available, the daily increment in newly colonised sites typically increases with time as long as the edge of the system is not reached (Fig. 6b). Some replicates display more variability than others, but the increase associated with an accelerating process is typical for a wave-like colony expansion. This contrasts with populations in which a substantial proportion of the sites is removed. Typically in these populations, the colonisation does not change smoothly, but proceeds with short bursts of activity (Fig. 6c). Such dynamics may result from bottle necks which are apparent in the spatial patterns (Figs 2b and 5a) as pockets of four or more well connected sites are linked via single strings of one or two.

## Discussion

Invasion of saprotrophic and pathogenic fungi into crop and plant communities is fundamental to many critical processes

in soil, including nutrient cycling, crop productivity and the spread of diseases. In this paper we quantified the mycelial spread of the fungal saprotroph and plant pathogen, *R. solani*, through a heterogeneous lattice of susceptible sites. Using epidemiological models to summarise the growth dynamics of fungi combined with simple concepts from percolation theory we designed experiments to show that if a critical proportion of susceptible sites is removed from a population, invasive spread of a fungus can be prevented. The significance of this work is that it demonstrates that spatially heterogeneous coverage of a control strategy can prevent biological invasion by rendering not all but only a critical proportion of susceptible sites unavailable.

The experimental design focussed on a dynamical approach involving phase transitions for saprotrophic growth of soil-borne fungi. Analysis of the fungal growth through a population of discrete nutrient sites revealed three epidemiological and ecologically important behaviours: first removal of a critical fraction of susceptible sites from a population is sufficient to prevent invasion into a population that otherwise would have been invaded; second in a spatial system with short-range transmission, removal of susceptible sites introduces a shield preventing a large proportion of the remaining sites from being invaded; and third with an increasing number of removed or protected sites, the remaining susceptible sites are less well connected, forming a tortuous network with bottle necks resulting in a reduced rate of invasion, but with occasional out-bursts of fungal spread.

We tested the ability of fungi to invade for mycelial spread only. Many fungi have more than one method of dispersal spreading via mycelial growth or by spores that can be dispersed over longer distances. Clearly dispersal mechanisms affect the ability to invade. However, even for spread via spores, dispersal kernels can be constructed similar to the colonisation profiles shown in Fig. 3. For spore movement a threshold for invasion can therefore still be predicted.

Although the current experimental system is rather simple with nutrient sources placed in a square lattice, it has the strong advantage of repeatability, which often cannot be achieved in other systems. Moreover, although the hypotheses could in principle be tested by simulation alone, the empirical testing goes beyond this in testing the hypotheses in systems subject to variability that is endemic in experimental systems (Gilligan, 2002a). Sources of such variability include random differences in size and hence nutrient strength of susceptible sites in the population, small differences of intersite distance resulting from inaccurate placing of the susceptible sites, as well as randomness in fungal colony growth. These sources of variability are subsumed in the probability profile (Fig. 3), from which it can be seen that there is a small but nonzero chance of spread beyond nearest neighbour, and a high but less than a 100% chance of contact between nearest neighbours (Fig. 3). Above the threshold for invasion (e.g. when no sites are removed), the variability resulted in fewer sites becoming colonised than expected, since the probability of colonising a neighbouring site is less than 1, which is assumed in the derivation of our hypotheses from percolation theory. Below the threshold for invasion (e.g. when 60% of the sites are removed) more sites become colonised than expected as an occasional spread beyond nearest neighbour can connect two clusters of susceptible sites that otherwise would not have been connected. Accordingly one of the 70 populations showed invasive spread when noninvasive spread was expected. However, despite the inevitable variability, there was still a remarkable correspondence between the observed fungal invasion and the predicted behaviour from percolation theory. Under more natural conditions, greater variability may be observed due to soil physical, chemical and biological properties (Toyota *et al.*, 1996; Otten *et al.*, 2001; Otten *et al.*, 2004), resulting in probability profiles that are less steep with longer tails. Further work on the effect of longer tails as well as transmission beyond the nearest neighbour on the threshold for invasion is required as the number of sites that need to be removed may differ in these situations.

Thresholds for invasion have direct consequences for the application of control strategies. For example, in order to prevent invasion into a susceptible population, control measures need be applied only to a critical fraction of sites. Reducing the area at which control is applied whilst still preventing invasion can be beneficial for example with increased risk of failure through resistance build-up in the pathogen population, or if full spatial coverage is difficult to achieve (e.g. biological control agents on a root system (Weller, 1988)). Even above the threshold a significant reduction in the rate of invasion is achieved, which for instance can increase the longevity of a fungicide. In addition, phase transitions contribute to variability in the efficiency of biocontrol as a small difference in the number of sites controlled in a population can account for the difference between successful and unsuccessful control of an epidemic.

Percolation theory can be applied at a range of scales and has been used to analyse bacterial movement through soil (Li *et al.*, 1996), or saprotrophic spread of fungi (Bailey *et al.*, 2000) as well as larger scale spread of epidemics (Tan *et al.*, 2000; Newman, 2002; Sander *et al.*, 2002; Meyers *et al.*, 2003). Each case considers the spread through a population of suitable (susceptible) sites. Concepts derived from percolation theory can therefore play a prominent role in the design of management strategies for disease control at a range of scales. At the farm and regional scale, transmission between sites in a community can be broken by application of biological control agents or pesticides, as well as the introduction of resistant varieties and cultural practices, including crop rotation or organic farming. There are concerns about the spatial and temporal scales at which to apply such practices, with the increasing risk of herbicide and pesticide resistance and the need to anticipate the occurrence of novel or re-emerging disease problems (Stephens *et al.*, 1998; Shea *et al.*, 2000).

Underlying many of the problems lies the concept of invasion: Will a disease invade a population?; What is the minimum spatial coverage required for control to reduce the risk of invasion? Will the introduction of more susceptible crops (e.g. via an increased area of organic farming) enhance the risk of disease at a regional scale? These sorts of questions typically relate to percolation models, where there is a critical threshold for transmission of disease between adjacent sites above which disease is likely to invade (Grassberger, 1983; Newman, 2002).

Strictly we tested aspects of percolation that refer to static systems in which the transmission properties and status of each host are fixed on a lattice at the beginning. This is still somewhat distant from most agricultural systems and natural plant communities. In natural systems, transmission can occur over a range of scales with more than a single mode of transmission typified by mycelial spread, spore movement and vector dispersal. Moreover this occurs through a nonrandom spatially heterogeneous and temporally fluctuating environment. Hence spread will often occur beyond nearest neighbour, or be more variable than in our controlled environment, each of which may considerably affect the threshold for invasion. However, percolation models are still an area of active research in statistical physics (Strogatz, 2001), with further developments into percolation through sites off-lattice (Callaway *et al.*, 2000), and in the analysis of dynamical landscapes typically encountered in agricultural environments (Keymer *et al.*, 2000; Gilligan, 2002). Examples of dynamical landscapes extend through a range of scales, encompassing root systems, where root growth and death create a three-dimensional population of appearing and disappearing susceptible sites, as well as regional spread through fields with crop rotation over several seasons. We suggest that the approaches followed here provide a fertile basis for the analysis of dynamical systems leading to relatively simple estimates for the ability of microbes to invade heterogeneous environments.

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