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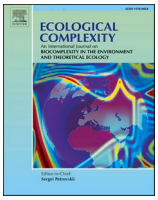

Emergent spatial structure and pathogen epidemics: the influence of management and stochasticity in agroecosystems

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A B S T R A C T

Organisms susceptible to disease, from humans to crops, inevitably have spatial geometry that influence disease dynamics. Understanding how spatial structure emerges through time in ecological systems and how that structure influences disease dynamics is of practical importance for natural and human management systems. Here we use the perennial crop, coffee, *Coffea arabica*, along with its pathogen, the coffee leaf rust, *Hemileia vastatrix*, as a model system to understand how spatial structure is created in agroecosystems and its subsequent influence on the dynamics of the system. Here, we create a simple null model of the socio-ecological process of death and stochastic replanting of coffee plants on a plot. We then use spatial networks to quantify the spatial structures and make comparisons of our stochastic null model to empirically observed spatial distributions of coffee. We then present a simple model of pathogen spread on spatial networks across a range of spatial geometries emerging from our null model and show how both local and regional management of agroecosystems interact with space and time to alter disease dynamics. Our results suggest that our null model of evolving spatial structure can capture many critical features of how the spatial arrangement of plants changes through time in coffee agroecosystems. Additionally, we find small changes in management factors that can influence the scale of pathogen transmission, such as shade tree removal, and result in a rapid transition to epidemics with lattice-like spatial arrangements but not with irregular planting geometries. The results presented here may have practical implications for farmers in Latin America who are in the process of replanting and overhauling management of their coffee farms in response to a coffee leaf rust epidemic in 2013. We suggest that shade reduction in conjunction with more lattice-like planting schemes may result in coffee being more prone to epidemic-like dynamics of the coffee leaf rust in the future.

1. Introduction

Organisms susceptible to disease, from humans to crops, inevitably have spatial geometry that influences disease dynamics. While it may be argued that spatial components of disease-host systems in mixed environments are less important (e.g. plankton), it is certainly true that most plants and animals have non-trivial spatial structure, whether exogenously imposed by abiotic environment (Gratzer et al., 2004) or emerging endogenously from ecological dynamics (Li et al., 2016). It has been a standard epidemiological question to ask how disease propagates through space (Keeling et al., 1999; Park et al., 2002; Balcan et al., 2009; Craft et al., 2010), but less obvious is how the space is constructed in the first place and how that space influences subsequent disease dynamics. At one extreme, a feedback likely exists between host and disease, where hosts may alter their spatial distribution in response to the presence of disease, such systems may include humans (Levine & Levine, 1994). On the other hand, there exist many hosts-pathogen systems where hosts do not alter their spatial distribution over the course of pathogen dynamics, such as plants.

The construction of spatial structure becomes complicated when considering human managed systems such as agroecosystems. The spatial arrangement of crops varies across agroecosystems due to a suite of interacting cultural, social, economic, and ecological factors. Here we focus our attention on perennial agroecosystems where plant mortality and replanting can occur iteratively, generating spatial distribution with a signature of the prior spatial arrangements, continuously inherited from one harvest to the next. This contrasts with annual systems which will be effectively fixed during the course of pathogen spread, due to the seasonal harvest/destruction and replanting of all plants. In perennial systems, the spatial arrangement is a consequence of farmer decisions about initial planting combined with continual replanting in spaces where individual plants had become damaged or die. The initial planting frequently begins with a lattice-like structure consisting of ordered rows and semi-constant interplant distances, but evolves over time with the dynamics of replanting. Replanting can be understood as a response to thinning, from a variety of causes, including the pathogens themselves. Consequently, the pattern of disease occurrence in agroecosystems is conditioned first by the structure of the plant distributions (effectively a socioecological process) and second by the dynamics of transmission (mainly an ecological process).

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The coffee agroecosystem and its most notorious pathogen, the coffee leaf rust, *Hemileia vastatrix*, provides a useful model system to interrogate the interaction of spatial pattern construction and its subsequent influence on pathogen dynamics. Coffee (both *Coffea arabica* and *Coffea robusta*) is a long-lived plant subject to a variety of management styles from intensive latticed monocultures to polycultures beneath the shade of forests (Moguel & Toledo, 1999). Transmission of the pathogen operates at two distinct spatial scales, locally, from coffee bush to coffee bush, and regionally, from farm to farm (Vandermeer et al., 2015; Vandermeer and Rohani, 2014). While regional pathogen dynamics is clearly important (Avelino et al., 2012), here we focus on the local dynamics in which the spatial distribution of coffee plants is evidently important to local transmission (Vandermeer et al., 2018). At this local scale, transmission likely results from a number of interacting factors, for example from plants being so densely planted that their leaves touch, or spore dispersal via air turbulence to neighboring plants, all of which are, in practice, influenced by management decisions such as how many and what kind of shade trees are incorporated in the system.

Prior work on coffee and the coffee leaf rust has employed a network approach to understanding spatial dynamics (Vandermeer et al., 2018), and here we build on that work. By focusing on local transmission dynamics, an intuitive approach for modeling pathogen dynamics is evident. We presume there exists some critical distance (D_{crit}) for which the pathogen is able to spread from plant to plant. We conceptualize the distribution of plants and the implied spread of the pathogen, as a network where the nodes consist of the plants and the edges are defined by the D_{crit}. Fig. 1 illustrates the approach on three 20 × 20m coffee plots from a coffee farm in southern Mexico and shows the clear interaction of the scale of transmission (D_{crit}) and the underlying spatial arrangement of coffee plants. The sub-networks within a given plot show us the extent to which the pathogen could theoretically spread if any member of the sub-network were infected. In one case (Vandermeer et al., 2018) the emergent spatial sub-networks predict observed pathogen dynamics, showing that plant to plant pathogen transmission is more likely within a sub-network than between subnetworks, suggesting a utility in employing the subnetwork framework to more generally study pathogen dynamics.

Our proposed time-dependent process of spatial reorganization of plants via death and replanting within a plot is in part inspired by observations from the field with areas under cultivation for different periods of time. The three plots in Fig. 1 correspond to a one-year-old plot (Sandino), a four-year-old plot (Che), and a fifteen-year-old plot (Leon). Note how the youngest plot has a lattice-like spatial pattern and the distributions become more disorganized as the plots age. Exploring the mortality/replanting mechanism, we propose a null-model to simulate the socio-ecological processes of plant death and replanting. Initiating a perfect lattice arrangement of plants, we simulate stochastic death and replanting within a fixed radius of the plant's prior position. The emergent spatial patterns are then compared to empirical spatial distributions (Fig. 1), and the range of spatial patterns from the null-model are used simulate pathogen spread to understand how the scale of pathogen transmission (D_{crit}) interacts with the underlying spatial pattern.

2. Methods

2.1. Model of Spread on Spatial Networks

Given that the intensity of pathogen infection is empirically correlated with the sub-network structure (Vandermeer et al., 2018), we stipulate D_{crit}, the maximum distance the pathogen can spread to neighboring plants (the spatial scale of transmission) and create a community of sub-networks for which dynamics are simulated (see Fig. 1, which illustrates how D_{crit} creates sub-networks). Given the coordinates of each plant, as they emerge from the simulations from the plot spatial evolution, a spatial scale of pathogen transmission is stipulated via particular values of D_{crit} which in turn creates a collection of spatial sub-networks (frequently referred to as "connected components"), called C,

$$C = \{C_1, C_2, C_3 \ldots C_m\}$$

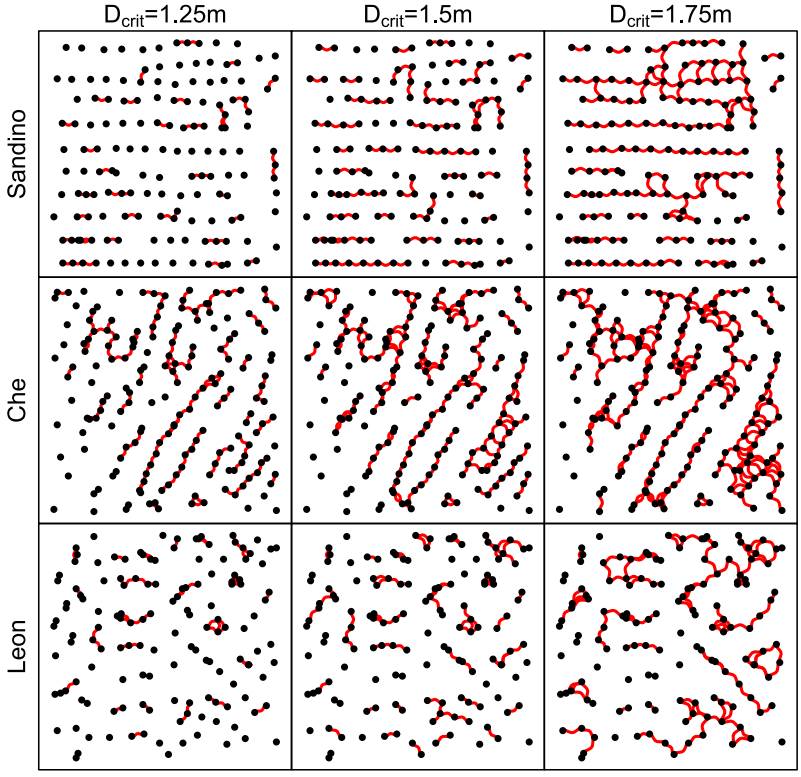

Fig. 1. The spatial distribution of coffee plants on three 20 × 20m plots in southern Mexico and the subsequent spatial networks that emerge from different spatial scales of pathogen transmission (D_{crit}), illustrating how the underlying spatial distribution of plots changes through time where Sandino is a one-year-old plot, Che is a four-year-old plot, and Leon is a 15 year-old plot. The spatial pattern moves from highly organzed lattice-like (1 year old plot - Sandino) to disorganized spatial structure (15 year old plot - Leon). We suggest that this gradient of organization emerges from the stochastic mortality/replanting phenomenon.

Where m is the number of sub-networks in the system. Note that each subnetwork in C contains a unique collection of plants corresponding to a given scale of pathogen transmission D_{crit}. For example, from a collection of n plants we might obtain,

$$C = \{\{p_1, p_2\}, \{p_3, p_4, p_5\}, \{p_6\}, \ldots \{p_a, p_b, p_c, \ldots p_n\}\}$$

for a particular D_{crit}. Note that the indices for each plant, p, are unique across all subsets within C, and come from the set P,

$$P = \{p_1, p_2, p_3 \ldots p_n\}$$

Where n represents the total number of plants in the system.

In the model, we keep track of all the infected plants with the set I, which is initialized as an empty set.

$$I = \{\}$$

For each time step in the model we iterate through all nodes (plants) in P, and there is a fixed probability, β, that a given plant becomes infected. If P_i (the i^{th}plant in P) becomes infected via

$$p_i \lambda(\beta)$$

Where

$$\lambda(\beta) = \begin{cases} 0, & \beta \geq U(0,1) \\ 1, & \beta < U(0,1) \end{cases}$$

then the whole cluster, C_j, which is a subset of C and contains p_i, is join in union with I. This is done for all p's where $\lambda(\beta) = 1$ (i.e. when there is a successful infection).

$$I(t+1) = I(t) \cup C_j$$

Conceptually, each sub-network represents the extent to which the pathogen instantaneously spreads from a single infected plant to all plants in that sub-network. We use the inevitability of the spread within a sub-network as a simplifying assumption and assume that all plants that fall within the sub-network denoted by the scale of the spread, D_{crit}, become infected instantaneously. This abstraction simplifies the system and allows for us to focus on the interplay of pathogen dynamics and the spatial geometry. With the assumption of instantaneous spread within a sub-network, our model only has one parameter associated with the epidemic process, the probability of a random plant in the plot being infected β, which can be thought of as being a measure of the regional pathogen propagule density.

When simulating pathogen spread, we allowed simulations to proceed until 90% of the plants became infected to quantify the time to epidemic of the pathogen on the spatial geometries arising from the null model. To account for the inherent stochasticity of the null model and the spreading process we replicated time step snapshots from the null model five times with subsequent ten replicate simulations of the spreading process for the D_{crit} ranging from 0 to 3. We then used the mean time until 90% infected hosts.

2.2. Null model of evolving plant spatial geometry

Despite the fact that coffee bushes are often planted with the intention of a strict lattice structure (planted in rows), the real distribution of coffee plants on a farm rarely reflects perfectly that initial intent. As time goes by, some coffee bushes die and usually are replanted, but rarely in precisely the same location, leading eventually to a loss of the initial planting pattern. To the farmer these small deviations may not seem consequential for the dynamics of pathogens and pests, yet they can accumulate significantly to change the basic spatial pattern (e.g., Fig. 1). Although a host of complicated local factors are involved in planting decisions, we initially approach the problem with a null model of planting spatial evolution.

We begin with plants arranged in a lattice bound within a x and y coordinate range and modify the structure over time. The simple model simulates stochastic death and replanting within an area of relatively proximity of the prior plant position. The coordinates change over time according to,

$$x_i(t+1) = x_i(t) + \xi(\phi)\mathcal{D}(\alpha)$$

$$y_i(t+1) = y_i(t) + \xi(\phi)\mathcal{D}(\alpha)$$

Where

$$\xi(\phi) = \begin{cases} 0, & \phi \geq U(0,1) \\ 1, & \phi < U(0,1) \end{cases}$$

and $x_i(t)$ and $y_i(t)$ represent the two coordinates corresponding the position of plant i at time t. $\xi(\phi)$ is the death/replanting rate and ϕ is the mortality probability for plant i. $\mathcal{U}(a, b)$ is a uniformly distributed random variable with range (0,1) and $\mathrm{D}(\alpha)$ is a random variable drawn from a uniform distribution with mean α, that stipulates the "replanting radius" of the new plant. For all simulations, $\phi = 0.05$ and $\alpha = 0.25$. The simulations were run iteratively for each plant in the plot 100 times.

To understand how our null model approximates the planting arrangements of real agroecosystems, we use an empirical data set of three 20×20 m plots on an organic coffee farm in the Soconusco region of Chiapas, Mexico. Each of the three plots have different ages (time since the area had all plants removed and replanted) corresponding to approximately one year, four years and fifteen years (Fig. 1). They represent what we propose to be the progression of spatial structure across the lifecycle of an area in cultivation. Given these three 20×20m plots with differing numbers of plants, for each comparison, the simulated evolution of spatial structure was done with the same planting density as the real plot it was intended to simulate. Our empirical plots have 177, 147 and 140 plants. To approximate the lattice-like initial conditions of each of these plots we used 12×15, 15×10, and 14×10 planting arrangements for the simulated plots. By controlling for the planting density, our null model of plot spatial evolution allows us to make comparisons with our empirical 20×20m plots and understand to what extent our null model approximates the empirical spatial geometry across the ontogeny of the plots through time.

2.3. Quantification of spatial structure

Similar to modeling the spread of the pathogen in space as described in the previous section, to quantify the spatial pattern of a plot we focus again on the sub-networks that emerge from imposing D_{crit}. By looking at a range of D_{crit} for a given spatial pattern we quantify how the number of sub-networks changes across spatial scales and can subsequently make comparisons to our empirical spatial patterns. Fig. 1 illustrates how different spatial patterns give rise to varying numbers of sub-networks for a single D_{crit}. The variability in the number of sub-networks reflects the clustering and over-dispersion and various spatial scales within a particular plot. The number of sub-networks not only uses the same tools for modeling pathogen spread, but also provides biologically relevant information for the dynamics of the pathogen that the use of a traditional dispersal kernel does not. For example, a given distribution of sub-networks for a D_{crit} gives us the minimum number of outside infections needed to infect a total area in cultivation.

To understand the extent to which the simulations approximate the empirical patterns, we use Δ_s, or the difference in the number of sub-networks in the empirical spatial patterns minus the number of sub-networks in the simulated spatial patterns. For a perfect spatial approximation in terms of number of subgraphs we expect a $\Delta_s = 0$. Importantly, we are interested in Δ_s across a relatively wide range of D_{crit} to quantify the spatial pattern, although we are constrained at the low end where no plants are connected and the high end where the whole plot is connected. For each simulation, we extracted the pattern at the first step and subsequently every 10 steps through 100 rounds of replanting. For our analyses, we used D_{crit} that ranges from 0 to 3m to

quantify the spatial structure.

3. Results

3.1. Plot evolution and approximations of empirical structure

Considering the pattern of sub-network emergence as a function of D_{crit}, we expect that as time advances (iterations in the model), early iterations will approximate the younger empirical plots and later interactions of the model will approximate the older empirical plots (as is evident in Fig. 2). Data for the three empirical plots are roughly approximated by the null model for various spatial scales (values of D_{crit}), and the range of colors in Fig. 3 show the variation in plot evolution, where light grey is the lattice and dark red is after 100 rounds of replanting. It is apparent that simulations start far from the empirical distributions and move towards them (i.e., Δ_s,=0) with continued plant death and replanting.

The largest deviations (Δ_s) are typically found at the distance that separates rows of the lattice, which ranges from 1.3-1.6m. This suggests that the empirical planting geometries are more clustered and over dispersed at scales that the model cannot approximate. For example, the empirical plot in Fig. 3a shows the model consistently unable to approximate at D_{crit} from 1.3-2m, and we see in the empirical data that this likely emerges from irregularities within row structure. It is evident from Fig. 3a that the deviation from the lattice emerges from missing plants and clustered plants but within the row structure itself. While simulations move plants away from the lattice structure randomly, the empirical data suggest that attempts to maintain semblance of row structure results in plants being replanting within the row but in an over dispersed or clustered fashion. Similar deviations are found between the empirical plots and simulations in Fig. 3b and c and are consistent prior to the scale that join rows of the lattice, as denoted by the lite grey line from the simulations. These deviations occur because the simulated plots are more clustered at these smaller distances as shown by the approximations being below the zero line.

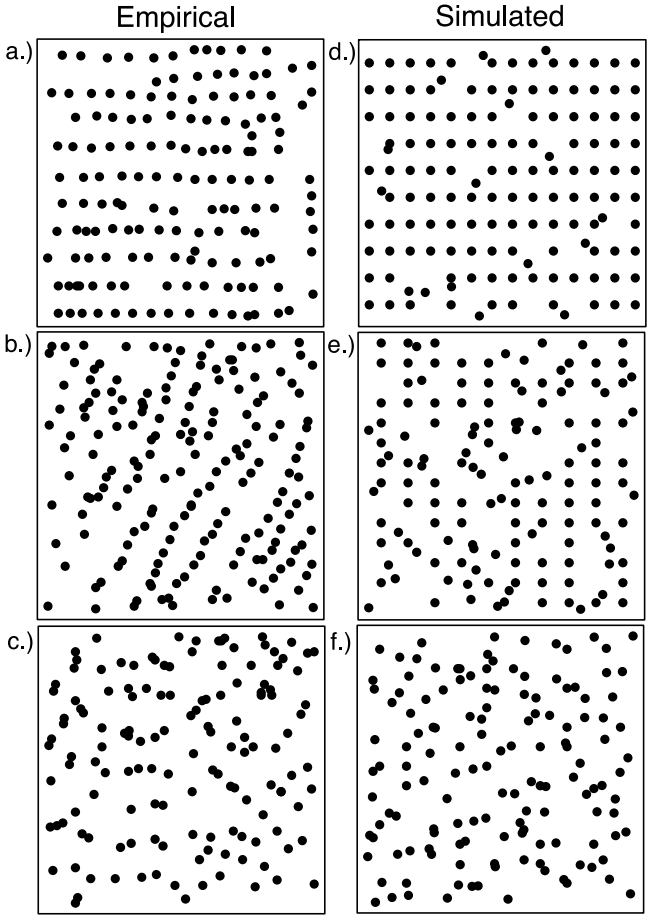

Fig. 2. Three 20 × 20M plots illustrating the position of all coffee plants. a. a one-year old plot, b. a 4 year old plot, c. approximately a 15 year old plot, d. simulated plot after 25 time units, e. simulated plot after 50 time units, f. simulated plot after 100 time units.

3.2. Modeling pathogen spread on spatial networks

Using time to reach 90% infection as a state variable, we illustrate its response to the two variables of interest, "plot evolution time" which is to say the time the null model is permitted to run, and the D_{crit} parameter which stipulates the threshold scale of transmission between plants. In Fig. 4, we summarize the general dynamics of the system from a two-dimensional parameter sweep of, 1) the scale of the pathogen transmission (D_{crit}), 2) the time steps involved in the plot evolution simulation, and 3) the state variable, time to epidemic (time to reach 90% of the trees infected). A pathogen spreading across the different plot geometries (represented by plot evolution time) reaches epidemic status regardless of spatial geometries due to the fixed probability of outside infection of plants β. At low transmission levels, it is apparent that plants become more clustered as the plot geometry evolves away from a lattice-like structure resulting in a small but detectable difference in the time to epidemic. At large scales there are few differences in the dynamics pathogen transmission due to almost the entire plot being connected resulting instantaneously infection once a colonizing infection reaches the plot.

It is intuitive that at very large transmission values, the pathogen will move quickly to infect the whole plot and at smaller scales it will move slowly, with little effect of the planting geometry. It is at intermediate transmission levels that we find non-obvious interactions with the geometry of plants. For these intermediate scales of transmission we find that lattice-like geometries are sensitive to small increases in transmission and generate a drastic jump in pathogen dynamics where the time to epidemic shows a pattern similar to that of a critical transition (Fig. 4). As the plot becomes less lattice-like through stochastic plant death and replanting this critical transition-like dynamic becomes less pronounced. At the two extremes of plant geometries, we see critical transition-like behavior for highly organized lattice-like arrangements and a gradual change in time to epidemic for more unorganized pseudorandom arrangements as the scale of pathogen transmission changes. These results suggest that a more random-like pattern of the plants buffers drastic changes in the overall dynamics of the pathogen.

Given the basic biology of most pathogens, it makes sense to think about not only the dynamics within a plot but also how the regional dynamics impact the system. Our model results suggest that the dynamics of the spatial host-pathogen system changes as the probability of plants being infected from outside of the plot increases (Fig. 5). As the regional infection probability increases, the interaction between the spatial geometries of the plants and scale of pathogen transmission becomes less pronounced. The critical transition-like behavior observed for relatively low regional infection probabilities is buffered as the regional infection probability increases, suggesting that under epidemic levels of a pathogen in the environment, the spatial arrangement of plants on a given farm becomes less important for the overall dynamics of the system.

4. Discussion

The management practices that create the spatial geometries of plants in agroecosystems emerge from socioecological processes structured by a number of influences, from cultural practices to the economic position of the farmer among other factors. Our approach here has been to try and recreate the range of observed spatial planting geometries by

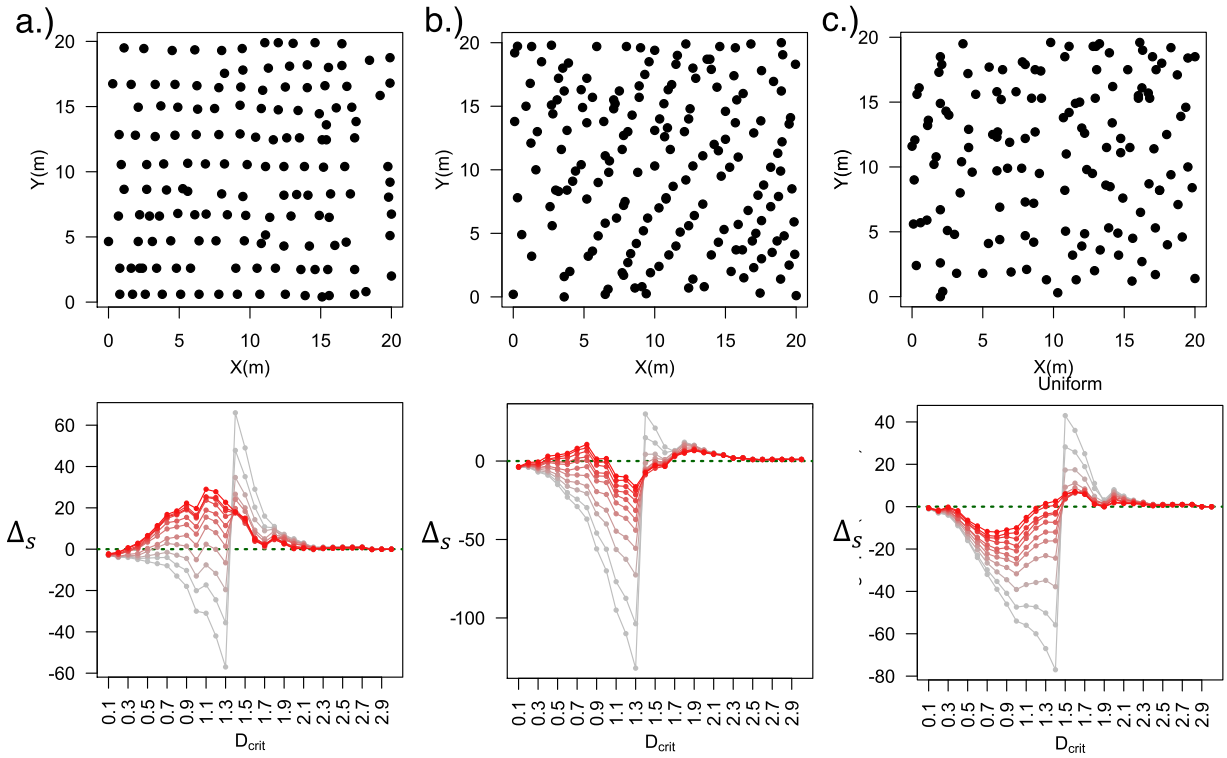

Fig. 3. (a-c) The bottom rows shows Δ_s as a function of the critical distance at various stages in the evolution of plot structure using the null model. Shading goes from light (the first stage in the simulation) to dark red (final step of simulation). Note that the dashed horizontal line corresponds to a 1:1 approximation of the model to the empirical plots. Note that the y-axes differ for each of the sub-network comparison plots. The top row shows the empirical spatial distribution of of plants that are compared to the simluations.

using a simple null model that strips away most of these real-world complexities. We show that a simple process of stochastic plant death and replanting within a small radius surrounding the dead plant can recreate many of the features observed in the real distribution of planting geometry. Furthermore, we suggest that the observed spatial geometries in agroecosystem can be the result of different snapshots in time of this dynamic process (Fig. 2). The comparisons between our model and empirical data (Fig. 3) provides support for the idea that at least for the sampled plots, the distributions of plants fall along different times of this stochastic death and replanting process. For the most lattice-like geometry of our empirical plot (Fig. 3a) the model passes through the 1:1 approximation of our empirical data for a wide range of spatial scales, suggesting that early stages in the simulations that move away from the lattice approximate it better than later more unorganized steps in the model. Furthermore, our simulations pass that same 1:1 approximation but at a much later time in the simulations for our plot of intermediate lattice structure (Fig. 3b), while our plot farthest from the lattice structure (Fig. 3c) is never well approximated by our model across the full range of D_{crit}.

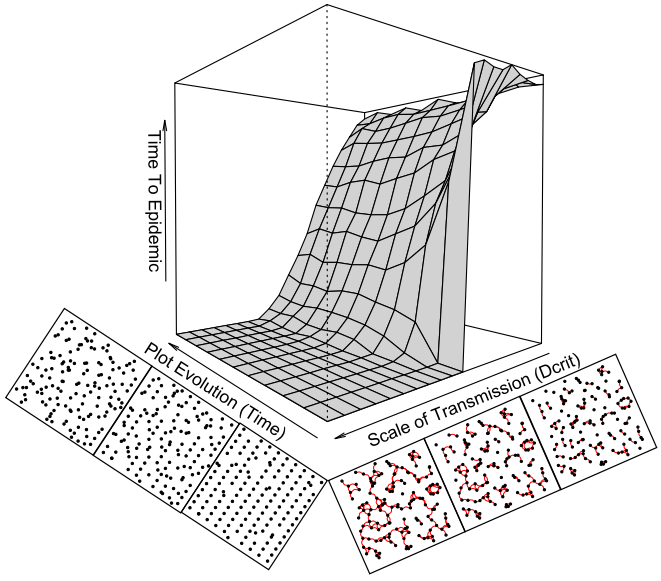

Fig. 4. Shows the time until 90% infection across a range of scales of pathogen transmission (D_{crit}) as well as planting geometries generated by the plot evolution model. Plots along the two axes are to illustrate the changing spatial network structure along scale of spread and the changing panting geometry in the plot evolution model. The regional infection probability is $\beta = 0.1$.

The interaction of spatial structure and spatial transmission of the pathogen suggests that they interact in a non-linear way. We observe that there is a critical transition-like behavior that emerges from the interaction of both the scale of pathogen transmission and the underlying spatial pattern of the hosts. Relatively small changes in pathogen transmission (D_{crit}) with lattice-like spatial geometry can lead to a dramatic jump in dynamics of the pathogen (Fig. 4). Thus, with lattice-like planting, the pathogen may be held at relatively low densities, but a small change in management that may influence of scale of pathogen spread (discussed below) can result in a devastating shift in dynamics. The uniform nature of the lattice creates conditions such that, once the threshold that connects rows is met, the whole plot becomes a connected network on which the pathogen can spread across. As the death and replanting process moves the spatial pattern away from a lattice planting geometry, it disrupts row structures and subsequently buffers the critical transition-like jump in pathogen dynamics. Thus it might be expected that uniform lattice-like planting geometries are far more sensitive to small changes in the scale of transmission of the

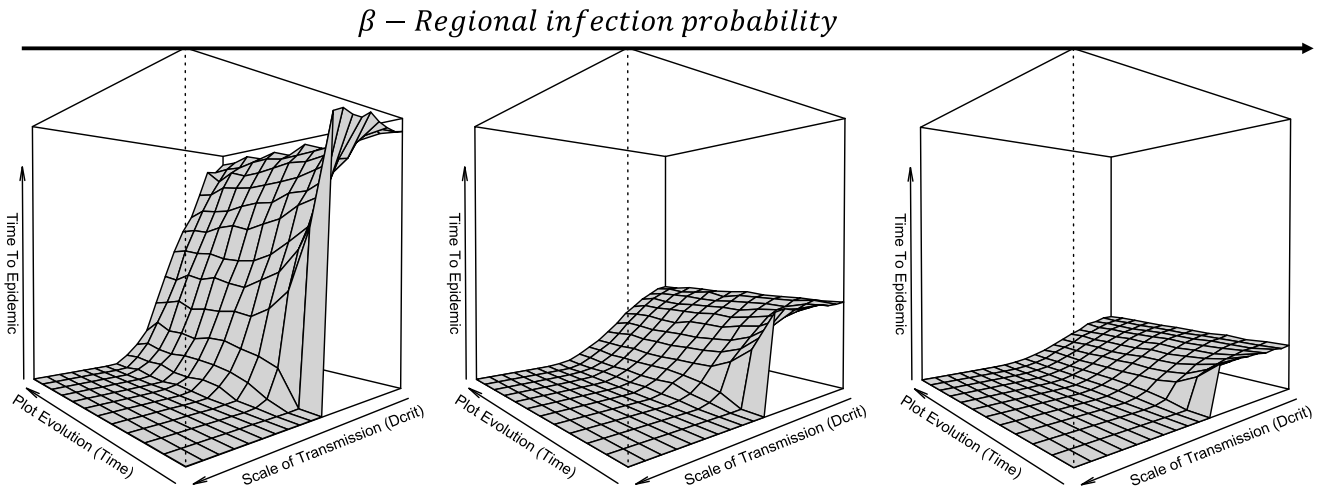

Fig. 5. Shows how the pathogen dynamics change as the regional infection probability increases. The first figure is $\beta = 0.1$, then $\beta = 0.2$ then $\beta = 0.5$.

pathogen and even a relatively small amount of disruption from the highly organized state can buffer against variability in pathogen spread. As we move away from the lattice-like geometry, there may be higher pathogen infection at low transmission due to some clusters of plants, but that same geometry ultimately prevents the pathogen from spreading through the whole area. Furthermore, as the regional infection probability increases the qualitative results of the model stay the same however they are damped, and the critical transition-like behavior is almost non-existent for high regional infection probabilities.

The results presented here have practical significance for the management of pathogens in agricultural systems, and in particular for the system which inspired the study, the coffee leaf rust (CLR). The CLR propagates both as a random propagule rain, spores arriving from the regional pool of spores in the environment, and from plant to plant on a local level through local wind current instabilities, branch-branch-contact, and splashing (Vandermeer et al., 2018; Avelino et al., 2004). Our model simulations mainly focused on the local level transmission and how that interacts with the planting geometry of the agroecosystem. There are a number of management factors within coffee agroecosystems that have the potential to influence parameters associated with the scale of the CLR transmission (Avelino et al., 2004). Shade is one of the most commonly managed aspects of coffee agroecosystems and its impact on the dynamics of the CLR has been contentious with some reporting beneficial impacts of shade reducing CLR (Soto-Pinto et al, 2002), and others reporting the opposite (Lopez-Bravo et al., 2012). The classic recommendation has been to reduce shade to manage the CLR, as the microclimatic implications of shade such as increasing humidity could potentially be beneficial to the germination of spores (Staver et al., 2001), but it is important to understand that the transmission of spores and the viability of spores are two different forces that need to be simultaneously managed. Bourot et al. (2016) provided evidence that shade within coffee agroecosystems reduces the spread of spores, thus providing support for the idea that shade trees within a coffee plantation act as a wind breaks and prevent local dispersal. In Avelino et al. (2012) the surrounding landscape of pasture land was correlated with the CLR on individual farms. Shade is a single management factor that has the potential to influence the local scale of pathogen transmission and probability of the pathogen establishing infection. In the context of the analysis presented here, we suggest that the amount of shade locally will modify D_{crit} by creating wind breaks which reduce the plant to plant (local) transmission.

While the question of what initially caused the outbreak of the CLR in 2013 in Latin America is still unclear, it set up the necessary conditions to overhaul many coffee agroecosystems throughout the region. Due to the prevalence of plant death from the epidemic itself, in conjunction with the promotion of resistant varieties, most farmers throughout Latin America are likely replanting whole farms now. This is particularly important moment in the dynamics of the CLR in Latin America, as following classical agronomical recommendations for combating the CLR would mean a reduction in shade, thus potenitally increasing the scale of CLR transmission locally, as well as replanting with resistant varieties, will likely lead to planting geometries that are more lattice-like when the whole system is replanted. Studies have found that this process is already underway in parts of Central America (Valencia et al., 2018), and as this study shows, the combination of shade reduction and moving towards a uniform planting structure, increases the likelihood of the critical transition-like epidemic dynamics observed in our model.

Declaration of Competing Interest

None.

Acknowledgments

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ecocom.2020.100872.

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