Disease Spread Across Multiple Scales in a Spatial Hierarchy: Effect of Host Spatial Structure and of Inoculum Quantity and Distribution

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ABSTRACT

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Spatial patterns of both the host and the disease influence disease spread and crop losses. Therefore, the manipulation of these patterns might help improve control strategies. Considering disease spread across multiple scales in a spatial hierarchy allows one to capture important features of epidemics developing in space without using explicitly spatialized variables. Thus, if the system under study is composed of roots, plants, and planting hills, the effect of host spatial pattern can be studied by varying the number of plants per planting hill. A simulation model based on hierarchy theory was used to simulate the effects of large

versus small planting hills, low versus high level of initial infections, and aggregated versus uniform distribution of initial infections. The results showed that aggregating the initially infected plants always resulted in slower epidemics than spreading out the initial infections uniformly. Simulation results also showed that, in most cases, disease epidemics were slower in the case of large host aggregates (100 plants/hill) than with smaller aggregates (25 plants/hill), except when the initially infected plants were both numerous and spread out uniformly. The optimal strategy for disease control depends on several factors, including initial conditions. More importantly, the model offers a framework to account for the interplay between the spatial characteristics of the system, rates of infection, and aggregation of the disease.

The importance of spatial aspects of plant disease epidemics is well recognized (3,4,13) and efforts have been made in order to incorporate spatial heterogeneity into disease progress models (20,24). These models predict that aggregation of the disease reduces the speed of epidemics. Indeed, it has been shown that an aggregated spatial pattern of inoculum leads to a reduction of disease level compared with a more uniform pattern; for example, in the case of damping-off of cress, black rot in cabbage, or *Zucchini yellow mosaic virus* in zucchini squash (2,16,21). Therefore, cultural practices that would result in a more aggregated pattern of diseased plants may contribute to disease control.

Disease level is also influenced by plant density (6,7) as well as host spatial pattern (25). It has been shown theoretically that the spatial discontinuities introduced by host aggregation cause the infection rate to fluctuate as host aggregates are depleted of healthy plants and new aggregates are being colonized (1). Compared with a random pattern, an aggregated pattern of plants speeds up the epidemic development at the start of the epidemic and slows it down afterward. The best choice of host spatial pattern depends on the time and disease incidence at which the switch between acceleration and slowing down occurs. This depends, on the one hand, on the number of plants in each aggregate and on the infection rate between plants and, on the other hand, on the initial pattern of infected plants.

Therefore, the best strategies for reducing crop losses can only be reached by integrating the effects (on both mean disease level and disease aggregation) of the factors that are directly under the control of the farmer (such as plant density and spatial location)

and the factors that are related to the pathogen but might be indirectly influenced by cultural practices (such as inoculum quantity and spatial pattern or relative infection rate). A simulation model for disease spread across multiple scales in a spatial hierarchy has been developed (the Cascade model) (8). Although not explicitly spatial, this model takes into account some of the spatial characteristics of the system, such as host aggregation (e.g., aggregation of roots within plants and plants within planting hills) and initial spatial pattern of the pathogen (e.g., number of initially infected planting hills and initial number of infected plants per infected planting hill). The objective of the work presented here was to analyze the effect of host spatial structure and of inoculum quantity and distribution on disease temporal development and spatial pattern using the Cascade model.

THEORY AND APPROACHES

Outline of the model. *Definition of the system.* The model is described elsewhere (8); therefore, only the features relevant to this study will be presented here. It is an individual-based, spatially implicit, SI model which represents host population and its spatial pattern through a nested hierarchy of levels (e.g., field > planting hill > plant > root > site). Let us consider here a three-level system: planting hills, plants, and roots. Each level consists of units which infect each other within a unit of the higher level; for instance, roots infect each other within a plant. Levels are linked in two ways: high levels influence the level immediately below them by allowing the first infection to take place (e.g., the first infection of a root in a formerly healthy plant is allowed by plant-to-plant infection) and low levels influence the level immediately above them through the probability of infection at the higher level (e.g., the infectiousness of plants is dependent upon disease “severity” of diseased plants) (i.e., the conditional

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incidence at the root level [ratio of the number of diseased roots on the number of roots in diseased plants]).

Infection process. In the present study, the healthy and diseased plants (respectively roots) were supposed to mix perfectly within planting hills (respectively plants), leading to the following equation for infection (given here for the plant level but it is similar for the planting hill and root levels). In each planting hill i , with susceptible plants (S_i) and infected plants (I_i), the number of infections at each time step follows a binomial distribution (B):

$$(\Delta I_i / \Delta t) \rightarrow B[S_i, 1 - (1 - p_i)^{I_i}] \quad (1)$$

where p_i is the probability that one infected plant in planting hill i will infect one susceptible plant in the same planting hill:

$$p_i = \beta_{\text{plants}} \times C_i \quad (2)$$

where β_{plants} is the infectiousness of a totally infected plant, and C_i is the conditional incidence at the root level in planting hill i (i.e., number of infected roots in planting hill i divided by the number of roots in infected plants of planting hill i).

Initiation of the epidemics. For each level, the model allows specifying the number of units that are initially infected (per unit of the level above). Although this is not the same as the distribution of primary inoculum, the assumption made here is that the quantity and distribution of initially infected units is the same as those of primary inoculum.

Output. The model output is, for each level, the total number of units, number of diseased units, and variance of the number of diseased units per unit of the level above (V_{obs}). At each level, disease incidence is defined as the ratio of the number of diseased units on the total number of units.

Description of the simulations. The effects of the spatial structures of host and of primary inoculum on disease dynamics were analyzed by considering host populations of 25,000 plants containing 49 roots each. Two different distributions of the plants were simulated: 1,000 planting hills with 25 plants each (small planting hills) or 250 planting hills with 100 plants each (large planting hills). Initially, 25 roots were infected, belonging to 25 different plants. The spatial pattern of the inoculum was either regular (1 infected plant in each of 25 planting hills) or aggregated (25 infected plants in 1 planting hill).

The effect of inoculum quantity was analyzed by repeating these simulations with four times more inoculum: 100 initially infected roots. The spatial pattern of the inoculum was either uniform (1 infected plant in each of 100 planting hills) or aggregated (25 infected plants in each of 4 planting hills in the case of small planting hills, 100 infected plants in 1 planting hill in the case of large planting hills).

The maximum infectiousness (β) was $2E-4$, $2E-2$, and $2E-2$ at the planting hill, plant, and root levels, respectively. These values were chosen so that the transition time at the plant level (i.e., the time needed for a plant to go from having one infected root to all but one infected root) would be ≈ 10 days and the complete epidemics in small planting hills would develop in ≈ 150 days from a single infected root. The simulations were run for 300 time steps (i.e., days) and 100 repetitions were done for each set of parameters in order to obtain a distribution for the results with the level of precision required for our purposes.

Data analysis. The areas under disease progress curves (AUDPCs) at each level were computed between time steps 0 and 299 for each of the 100 repetitions. The normality of the distribution of AUDPC was tested with a Shapiro-Wilk test (the significance level was modified according to the Bonferroni correction to maintain experimentwise error rate below 5%) using R statistical software (<http://www.r-project.org>). The distributions of AUDPC obtained in the different treatments were compared with

a Kolmogorov-Smirnov test, with α corrected for multiple comparisons.

The relationship between incidences at two successive levels in the spatial hierarchy was plotted. If the infected units were randomly located among units of the level above, then the incidence at one level (Inc_{high}) would be equal to the first term of a binomial distribution $B(n, \text{Inc}_{\text{low}})$, where n is the number of units (level below) per unit and Inc_{low} is the incidence at the level below (20):

$$\text{Inc}_{\text{high}} = 1 - (1 - \text{Inc}_{\text{low}})^n \quad (3)$$

When the disease is aggregated, Inc_{high} is smaller than predicted by the binomial distribution; aggregation is maximal when the incidence-incidence curve follows the first bisector.

Under the null hypothesis of a random distribution of diseased units, the variance of the number of diseased units per unit of the level above is $V_{\text{th}} = n \times \text{Inc}_{\text{low}} \times (1 - \text{Inc}_{\text{low}})$. There is good empirical evidence for a power law relationship between the observed and theoretical (binomial) variance of the number of diseased units, which results in a linear relationship between the variances when plotted on a log scale (11,17):

$$\ln(V_{\text{obs}}) = a + b \ln[n \times \text{Inc}_{\text{low}} \times (1 - \text{Inc}_{\text{low}})] \quad (4)$$

where V_{obs} is the observed variance of the number of diseased units per unit of the level above; Inc_{low} and Inc_{high} are the proportions of diseased units at one level and at the level above, respectively; and a and b are the intercept and slope of a straight line, respectively. When $a = 0$ and $b = 1$, diseased units have a random spatial pattern which can be described by the binomial distribution. When $b = 1$ and $a > 0$, diseased units have an aggregated distribution but the degree of aggregation does not depend on disease incidence. When $a > 0$ and $b \neq 1$, the degree of aggregation (heterogeneity) changes systematically with disease incidence (11). The power law relationship can be decomposed into four phases over the course of an epidemic occurring within a spatial hierarchy (8): an initial increase in disease aggregation, followed by two phases of constant aggregation (before and after the incidence at the lower level has reached 0.5), and a final phase of decrease of aggregation. The intercept of the straight line when aggregation is constant (i.e., when the slope = 1) indicates the intensity of disease aggregation when the equilibrium between infection of new units and saturation of long-diseased units has been reached. The intercept was estimated by regression analysis for each of the 100 repetitions in each treatment. The normality of the distribution of the intercept values within each treatment was tested with a Shapiro-Wilk test (the significance level was modified according to the Bonferroni correction to maintain an experimentwise error rate $< 5\%$) using R statistical software. The distributions of intercepts obtained in the different treatments were compared with a Kolmogorov-Smirnov test, with α corrected for multiple comparisons.

RESULTS

The evolution of disease incidence at the planting hill, plant, and root levels is represented in Figure 1, and the AUDPCs at the plant level are represented in Figure 2. AUDPC at the planting hill and root levels followed similar patterns (Table 1). All AUDPC distributions followed a normal distribution as determined by the Shapiro-Wilk test, except in the case of large planting hills with aggregated inoculum, where the AUDPCs were skewed toward low values because the incidence in some of the 100 repetitions did not reach 1 in 300 time steps. The effect of inoculum aggregation was more important than the effect of host spatial pattern or inoculum quantity, the disease spreading more quickly when the initially infected plants were uniformly distributed. When the

pattern of inoculum was aggregated, the simulated AUDPC was always lower when the plants were grouped into large planting hills (100 plants) compared with small planting hills (25 plants). In the case of large planting hills, a fourfold increase in the number of initially infected plants did not result in a significant increase in AUDPC. When the initial pattern of diseased plants was uniform, the effect of the size of the planting hills depended on the initial number of diseased plants. If the level of inoculum was low (i.e., 25 initially infected plants), the AUDPC was significantly lower for large planting hills. On the contrary, if the initial level of inoculum was high (i.e., 100 initially infected plants) and the inoculum pattern was uniform, large planting hills resulted in a higher AUDPC than small planting hills.

Aggregation of diseased plants within planting hills and diseased roots within plants was indicated by the incidence-incidence relationship (Fig. 3) as well as the power law analysis (Fig. 4; Table 2). The intercept of the power law analysis followed a normal distribution as determined by the Shapiro-Wilk test,

except at the plant level in the case of the high inoculum quantity uniformly dispersed. Disease aggregation was influenced more by plant pattern than by either inoculum quantity or inoculum pattern. Inoculum quantity had an effect on disease aggregation only at the very beginning of the epidemic development (Figs. 3A and D and 4A and D): disease aggregation (both of diseased plants among planting hills and diseased roots among plants) was more pronounced at the beginning of the epidemic in the case where the inoculum quantity was low. Subsequently, the intensity of aggregation of diseased plants and diseased roots became similar between low and high inoculum quantities when inoculum was aggregated and almost similar when inoculum was uniform (Table 2).

Inoculum pattern also had an effect on disease aggregation but only at the very beginning of the epidemics and only at the plant level (Figs. 3B and 4B). Indeed, the epidemics that started from an initially uniform or initially aggregated pattern of diseased plants converged toward the same aggregation level as soon as the initially infected planting hills became saturated.

Plant pattern had a more important effect on disease aggregation, and this effect varied during the course of the epidemic (Figs. 3C and F and 4C and F): at the very beginning of the epidemic, large planting hills resulted in a less aggregated pattern of diseased plants (because initial hill disease incidence was higher in large planting hills [100/250] than in small planting hills [100/1,000] with the same plant disease incidence [100/25,000]), but rapidly diseased plants became more aggregated within large planting hills than within small planting hills (Table 2). This happened because of the effect of the number of plants and planting hills on the hill-to-hill and plant-to-plant infection rates. The effect of the number of plants per planting hill was also reflected down to the aggregation of diseased roots which were, at the beginning, less aggregated in plants that were grouped into large planting hills than in plants that were grouped into small

Fig. 1. Effect of the host's and inoculum's spatial structures on the dynamics of the proportion of diseased A and B, planting hills; C and D, plants; and E and F, roots in simulated epidemics. A, C, and E, Low inoculum quantity (25 initially infected plants); B, D, and F, high inoculum quantity (100 initially infected plants). Solid lines: small planting hills (1,000 planting hills made from 25 plants); dashed lines: large planting hills (250 planting hills made from 100 plants); thick lines: aggregated inoculum (25 infected plants in 1 hill for low inoculum; 25 plants in 4 hills for high inoculum and small planting hills; 100 plants in 1 hill for high inoculum and large hills); thin lines: uniform inoculum (1 infected plant in each of 25 hills for low inoculum and 1 infected plant in each of 100 hills for high inoculum).

Fig. 2. Effect of the host spatial structure (thin lines: small planting hills made from 25 plants, thick lines: large planting hills made of 100 plants) on the area under the disease progress curve of the incidence at the plant level in simulated epidemics as a function of the quantity (25 or 100 initially infected plants) and spatial pattern of inoculum (aggregated inoculum = 25 infected plants in 1 hill for low inoculum; 25 plants in 4 hills for high inoculum and small planting hills; 100 plants in 1 hill for high inoculum and large hills; uniform inoculum = 1 infected plant in each of 25 hills for low inoculum and 1 infected plant in each of 100 hills for high inoculum).

TABLE 1. Effect of plant pattern, inoculum quantity, and inoculum pattern on the mean area under disease progress curve (AUDPC) at each level in epidemics simulated on a three-level system (planting hill > plant > root)

Hill size, pattern ^x	Inoculum quantity ^y	AUDPC (standard deviation) ^z		
		Planting hills	Plants	Roots
Small	High	258.9 (0.9) B	238.2 (0.9) B	232.0 (0.9) B
		236.6 (1.7) C	215.9 (1.7) C	209.7 (1.7) C
	Low	224.6 (3.5) D	204.0 (3.5) D	197.8 (3.5) D
		200.3 (8.4) F	179.6 (8.4) E	173.4 (8.4) E
Large	High	268.4 (1.6) A	252.5 (1.6) A	246.3 (1.6) A
		222.1 (4.4) E	206.1 (4.4) D	199.9 (4.4) D
	Low	124.4 (27.6) G	108.8 (26.8) F	102.8 (26.5) F
		119.2 (27.4) G	103.7 (26.7) F	97.7 (26.3) F

^x Planting hill size: small planting hills = 1,000 planting hills made of 25 plants; large planting hills = 250 planting hills made of 100 plants. Initial inoculum pattern of diseased plants: uniform = 1 plant in each of 25 or 100 planting hills for low or high inoculum quantity, respectively; aggregated = 25 plants in 1 planting hill for low inoculum quantity, 25 plants in 4 planting hills for high inoculum quantity and small planting hills, and 100 plants in 1 planting hill for high inoculum quantity and large planting hills.

^y Number of initially infected plants: high = 100 and low = 25.

^z Distributions with the same letter (within one column) are not significantly different.

Fig. 3. Relationship between the disease incidences at two successive levels in the spatial hierarchy in epidemics simulated on a three-level system (planting hill > plant > root); data plotted every time step until $t = 50$, once every 10 time steps afterward. The dashed line represents the case of a random distribution (equation 1, $n = 25$ for plants in small planting hills [black lines in A and B, gray line in C], $n = 100$ for plants in large planting hills [black line in C], $n = 49$ for roots [D, E, and F]). In all graphs, the gray dots are the same reference: small planting hills (25 plants), high level of inoculum (100 initially infected plants), uniformly dispersed (i.e., 1 initially infected plant in each of 100 planting hills). A and D, Effect of inoculum quantity, black dots = low inoculum quantity (25 initially infected plants). B and E, Effect of inoculum pattern, black dots = maximal aggregation of inoculum (i.e., 25 plants initially infected in each of four planting hills). C and F, Effect of the host spatial structure, black dots = large planting hills (100 plants in each planting hill). A, B, and C, Relationship between planting hill disease incidence and plant disease incidence. D, E, and F, Relationship between plant disease incidence and root disease incidence.

planting hills and subsequently became more aggregated (Figs. 3F and 4F; Table 2).

DISCUSSION

Novelty and limits of the approach. The hierarchical model Cascade was used to test different scenarios under which a disease could spread across three scales in a spatial hierarchy. The example presented above was disease spread among planting hills, among plants, and among roots; however, the model would equally be suited to aerial diseases (with plants, leaves, and leaflets as levels, or even agricultural regions, farms, and fields), as long as strong discontinuities in spatial or temporal scales exist between the different hosts levels (8). Host spatial structure was represented by the degree of clustering of plants in planting hills, and the effect of the spatial structure of primary infection was represented by the distribution of initially diseased plants among planting hills. Thus, the model allowed the address of spatial questions although the equations used for infection were non-spatial at each level.

Considering several scales allowed the analysis of the effect of host spatial pattern and quantity and distribution of initially

infected plants not only on disease dynamic but also on disease aggregation, characterized as incidence–incidence relationships and power law relationships. This had been addressed using spatialized models, in particular concerning the effect of inoculum spatial pattern (28,29), but these models only considered two levels (plant > unit). Few approaches have considered disease epidemics at different scales in a hierarchy (20,23,26). Considering several hierarchical scales is important because processes acting at one scale have consequences at the other scales (e.g., plant clustering had an effect on both mean incidence and disease aggregation at the root level), and because the processes that are relevant for establishing crop losses (e.g., effect of disease aggregation on compensatory growth) might occur at a different scale than the scale that was chosen for host clustering (planting hill) (9,10).

The hierarchical model Cascade was not intended to simulate a specific epidemic. On the contrary, it is a strategic model (19): its aim is to reflect in a simple way the complexity of epidemics developing across several scales simultaneously. As with any simplification, it cannot be applied to any system without caution. In addition to the importance of identifying relevant hierarchical levels (8), two assumptions made in this study must be kept in

Fig. 4. Variance (simulated by the model) of the number of **A** to **C**, diseased plants per planting hill or **D** to **F**, diseased roots per plant against the variance under the hypothesis of a random distribution of diseased plants or roots, on a log scale. Data plotted every time step until $t = 50$, once every 10 time steps afterward. In all graphs, the gray dots are the same reference: small planting hills (25 plants), high level of inoculum (100 initially infected plants), uniformly dispersed (i.e., 1 initially infected plant in each of 100 planting hills). **A** and **D**, Effect of inoculum quantity, black dots = low inoculum quantity (25 initially infected plants). **B** and **E**, Effect of inoculum pattern, black dots = maximal aggregation of inoculum (i.e., 25 plants initially infected in each of four planting hills). **C** and **F**, Effect of the host spatial structure, black dots = large planting hills (100 plants in each planting hill). **A**, **B**, and **C**, Relationship between planting hill disease incidence and plant disease incidence. **D**, **E**, and **F**, Relationship between plant disease incidence and root disease incidence.

mind when extrapolating the results to real systems. First, the assumption of perfect mixing will probably not hold for any size of planting hill: as more plants are entered into planting hills, it will become necessary to consider the distance between plants. Similarly, the assumption of perfect mixing may or may not be appropriate for all levels, depending on the dispersal mechanism at each level; in particular, if hill-to-hill infection occurred over short distances, two-dimensional infection might be more appropriate. Second, the maximal infectiousness of plants and planting hill were kept constant whatever the size of the planting hill; however, it might be reasonable to think that small and large planting hills do not have the same infectiousness (large planting hills might have a larger infectiousness, because they can contain more infected plants). Furthermore, host clustering itself might have an effect on the pattern of primary infection: when the host is aggregated, some propagules of primary inoculum will be situated outside any planting hill, whereas the propagules in contact with a planting hill will be liable to infect more plants, thus leading to an aggregated pattern of primary infection. Despite these simplifications, the model was able to simulate important features of plant disease epidemics, such as aggregation or the effect of the host's and inoculum's spatial structure on disease dynamics.

Effect of host and inoculum spatial structures on disease dynamic. The simulations showed that aggregating the initially infected plants always resulted in slower epidemics than spreading out the initial infections uniformly (Fig. 1), which is consistent with previous results, both experimental (2,16,21) and theoretical (15,27). Thus, it would be advisable to reduce the spread of the inoculum propagules as much as possible; for example, by reducing soil displacement through cultivation in the case of soil-borne propagules, as suggested by spatially explicit models (22). However, this recommendation might depend on other factors that were not tested here, in particular the link function relating the probability of infection at the planting hill level with the conditional incidence at the plant level. If there were a strong link between the two levels, the dilution effect brought on by the spread of the primary inoculum might be sufficient so that the low conditional incidence at the plant level could noticeably reduce the probability of infection at the planting hill level (8). Further-

more, an aggregated pattern of disease can lead to a reduction in compensatory growth by healthy plants (because fewer plants are adjacent to diseased ones), thus increasing yield loss (14,18). Thus, both beneficial and detrimental effects of an aggregated pattern of the disease must be considered when selecting a management strategy.

Simulation results also showed that, in most cases, disease epidemics were slower with large host aggregates (100 plants/hill) than with smaller aggregates (25 plants/hill): the plants that were situated in initially infected planting hills became infected more rapidly but the ones that were in healthy hills were somewhat protected by the lower hill-to-hill infection rate. However, the simulations show that this strategy would fail if the initially infected plants were both numerous and spread out uniformly; in this case, it would be better not to clump the plants, because all plants in an initially infected hill can quickly become infected. More detailed analyses are required to determine up to what level of inoculum the aggregation of host is advisable, and whether the results hold if the plant level is bypassed (i.e., planting hills were made of roots), because it might be more appropriate if roots from neighboring plants largely overlap.

A reduction in the rate of disease spread by host clustering had already been demonstrated in the case of vector-borne diseases (5). Other authors have advised a random or even spacing of plants but the disease dynamics were similar and the recommendation was made on the ground that the slowing down of the epidemic by host clustering only occurs at a time that may be posterior to the date of harvest (1). Obviously, this will depend on the pathosystem under study and the choice of planting pattern should be made individually, based on the characteristics of the field (quantity and spatial pattern of the primary inoculum) and on the biological characteristics of the disease (strength of the link between levels, effect of disease aggregation on crop losses, and effect of the physical modifications of the environment caused by host clustering on infectiousness at each level) and the crop (time of harvest and effect of host clustering on yield).

In summary, the simulations showed that the best strategy concerning host distribution was dependent upon the quantity and spatial pattern of primary infections: when there were a few initially infected plants or when the initial pattern of the disease was aggregated, large planting hills resulted in slower epidemics; however, when the primary infections were both numerous and uniformly spread out, small planting hills resulted in less disease. Validation of strategic models is done by consent by the scientific community and consistency with accepted knowledge rather than comparison with actual data (12); therefore, the fact that the results are in agreement with empirical and theoretical studies supports the model.

Accounting for disease aggregation. Another argument in favor of the model is its ability to reproduce the patterns observed in many real epidemics: the shape of the relationship between incidences at two successive levels (Fig. 3) and the power law relationship between observed and binomial variances (Fig. 4). Furthermore, the model offers a conceptual framework in which to explain the mechanisms responsible for the interplay between host structure, disease spread, and disease aggregation. Indeed, aggregation results from the balance between spread at one level and spread at the levels above (8) and these, in turn, depend on host spatial structure and inoculum pattern. For example, it is possible to explain the unusual patterns of the incidence-incidence relationships obtained with large planting hills and a high level of uniformly distributed inoculum (Fig. 3C and F, black dots).

In this scenario, aggregation of diseased plants within planting hills initially increased, then there was a temporary decrease in aggregation, and then aggregation became constant (Fig. 3C, black dots). This is due to the fact that aggregation started artificially low, because 100 planting hills were initially infected on one plant only; therefore, the high plant-to-plant infection rate

TABLE 2. Mean intercept of the power law relationship in the segment where the slope = 1 (i.e., $\ln[V_{\text{obs}}] = a + \ln[V_{\text{th}}]$) at the plant and the root levels in epidemics simulated on a three-level system (planting hill>plant>root)

Hill size, pattern ^x	Inoculum ^y	Intercept (standard deviation) ^z	
		Plants	Roots
Small	Uniform	High 2.66 (0.04) E	3.74 (0.02) D
		Low 2.80 (0.06) D	3.78 (0.02) C
	Aggregated	High 2.80 (0.04) D	3.78 (0.01) C
		Low 2.79 (0.05) D	3.78 (0.02) C
Large	Uniform	High 4.44 (0.3) C	3.70 (0.05) E
		Low 4.47 (0.12) B	3.84 (0.01) B
	Aggregated	High 4.51 (0.04) A	3.84 (0.01) A
		Low 4.52 (0.05) A	3.85 (0.02) AB

^x Planting hill size: small = 1,000 planting hills made of 25 plants and large = 250 planting hills made of 100 plants. Initial inoculum pattern of diseased plants: uniform = 1 plant in each of 25 or 100 planting hills for low or high inoculum quantity, respectively; aggregated = 25 plants in 1 planting hill for low inoculum quantity, 25 plants in 4 planting hills for high inoculum quantity and small planting hills, and 100 plants in 1 planting hill for high inoculum quantity and large planting hills.

^y Number of initially infected plants: high = 100 and low = 25.

^z Plants = relationship between the observed and the binomial variances of the number of diseased plants in each planting hill; Roots = relationship between the observed and the binomial variances of the number of diseased roots in each plant. Distributions with the same letter (within one column) are not significantly different.

compared with hill-to-hill infection led to the intensification of the disease within infected hills and to an increase in aggregation. As a result, the conditional incidence at the plant level (i.e., plant disease incidence in infected hills) became high and the rate of hill-to-hill infection increased, leading to the temporary decrease in aggregation, until aggregation of diseased plants reached its equilibrium, when infection of new hills balanced saturation of long-infected hills. Note that the intercept of the power law is much higher for large planting hills than for small planting hills (Table 2) but aggregation indices for diseased plants cannot be compared directly between large and small planting hills, because the number of plants per planting hill is different.

At the root level, the observed pattern (i.e., initial apparent decrease in aggregation, followed by an increase almost up to the maximum aggregation when the incidences approached 0.4, and then a small decrease to reach a constant level) (Fig. 3F, black dots) can also be explained. Indeed, in the first part of the epidemic (when plant disease incidence was <0.4), the disease observed at the root level developed almost as if the system comprised only the plants in the initially infected planting hills, as can be verified by running the simulation again with a null probability of infection between planting hills (results not shown). This means that the observed decrease and then increase in aggregation in the first part of the epidemic is only due to the fact that the observed population of roots is not the population actually involved in the epidemic: aggregation of infected roots among the plants from the infected planting hills was actually constant. In the second part of the epidemic (after the incidences at the root and plant levels was >0.4), aggregation of diseased roots decreased because plant-to-plant infection suddenly became higher as new susceptible plants “entered” the system when new planting hills became infected, and aggregation reached its equilibrium (balance between saturation of plants and infection of new plants, supplied by the infection of new planting hills).

As can be seen above, the model accounts for the dynamic and relative nature of aggregation. Indeed, aggregation is relative to the limits of the system considered (e.g., the decrease and then increase in aggregation of diseased roots among plants, when all planting hills were considered, turned out to be constant aggregation when only initially infected planting hills were considered), and the limits of the system within which an epidemic develops at one level change with time, as elements of the levels above get infected.

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