Influence of Host Diversity on Development of Epidemics: An Evaluation and Elaboration of Mixture Theory

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ABSTRACT

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A spatiotemporal/integro-difference equation model was developed and utilized to study the progress of epidemics in spatially heterogeneous mixtures of susceptible and resistant host plants. The effects of different scales and patterns of host genotypes on the development of focal and general epidemics were investigated using potato late blight as a case study. Two different radial Laplace kernels and a two-dimensional Gaussian kernel were used for modeling the dispersal of spores. An analytical expression for the apparent infection rate, r , in general epidemics was tested by comparison with dynamic simulations. A genotype connectivity

parameter, q , was introduced into the formula for r . This parameter quantifies the probability of pathogen inoculum produced on a certain host genotype unit reaching the same or another unit of the same genotype. The analytical expression for the apparent infection rate provided accurate predictions of realized r in the simulations of general epidemics. The relationship between r and the radial velocity of focus expansion, c , in focal epidemics, was linear in accordance with theory for homogeneous genotype mixtures. The findings suggest that genotype mixtures that are effective in reducing general epidemics of *Phytophthora infestans* will likewise curtail focal epidemics and vice versa.

Additional keywords: crop arrangement, dispersal kernels, genotype mixtures, *Solanum tuberosum*.

Modern agricultural practices have resulted in a decline in crop diversity and an increase in genetically and spatially uniform plant populations. This lack of host diversity can lead to an escalation in the frequency and severity of epidemics. Conversely, many natural ecosystems are less prone to severe epidemics due to the diversity of host genotypes and their patchy distribution (1). Spatial diversification of host resistance therefore appears as an important approach in the effort to achieve successful and sustainable management of crop pathogens. Applications of this approach have included inter- and intra-specific diversity (6,14,17,44-46) both within fields (e.g., alternate strips or rows) and between fields (e.g., cultivar rotation and mosaic cropping).

The potential of genotype mixtures to slow down or prevent epidemic development has been established through the use of computer simulation (18,22,26,28) and by experimentation (1,7,11,12,16,23,25,30,33,44,48). Three factors account for disease reduction in genotype mixtures: (i) the dilution of inoculum due to the presence of resistant plants (lack or reduction of inoculum production on resistant plants, and loss of inoculum infectious to susceptible hosts as a result of spore deposition on resistant plants); (ii) the physical barrier to successful deposition created by the resistant plants; and (iii) the potential for induced host resistance when propagules are deposited on a neighboring plant for which they are avirulent (10). With regard to the spatial arrangement of genotypes within a mixture, the ground area of individual genotype units (genotype unit area) is an important

configuration variable influencing epidemic development (3,24,25,27,28). A genotype unit is defined as a spatially delineated unit of space planted with a single host genotype. A genotype unit can therefore refer to a single plant, a row or patch of plants of the same genotype, or an entire field as in a monoculture.

The rate of progress of epidemics that develop from initial blanket infections originating from large external sources of inoculum (general epidemics) can be characterized by the apparent infection rate, r (43). The apparent infection rate is therefore a good candidate parameter for describing the impact of spatial configuration in a genotype mixture upon the development of the disease in the field. The parameter r can be approximated by utilizing the theory of age-structured populations (19), whereby the apparent infection rate is related to the net lifetime reproduction (average number of daughter lesions produced per mother lesion) and the generation time (average time between birth of an individual and birth of its offspring); see also Segarra et al. (36).

For epidemics that develop from an initial point of infection (focal epidemics), the radial velocity of focus expansion, c , can be used to describe the impact of spatial variation upon disease progress. According to theory (39,41,42), focal expansion reaches a constant velocity after an initial phase of focus build-up. This rate has been shown (in theory) to increase linearly with the logarithm of the fraction, f , of susceptible plants in ideal mixtures of susceptible and resistant cultivars. This relationship has been confirmed experimentally in the field for stripe rust (*Puccinia striiformis*) on wheat (*Triticum aestivum*) (40) and for bean rust (*Uromyces appendiculatus*) on bean (*Phaseolus vulgaris*) (2). An ideal mixture is infinitesimally fine-grained. In practice, ideal mixing is approximated when the spore cloud around a lesion on a plant encompasses a large number of other susceptible plants. An ideal mixture can therefore often be approximated in the field

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through random seed mixing prior to planting, provided that the pathogen spreads its propagules over distances well exceeding the plant and row distances of the crop.

For the potato late blight pathosystem (*Phytophthora infestans*–*Solanum tuberosum*), the relatively large size of potato plants, as the smallest genotype unit possible, can result in high levels of autoinfection (34) defined as infection caused by a propagule from within the same genotype unit. The radial expansion of lesions further increases the extent of autoinfection, suggesting a somewhat negative outlook on the potential of genotype mixtures to control *P. infestans*. However, recent experimental studies (1,11,12) indicate that increased host diversity can contribute to a reduction of disease progress. There is evidence for a potentially beneficial effect of host diversity in reducing potato late blight in temperate regions such as France (1) and the United States (11), where late blight epidemics tend to develop from obvious foci. Recent experiments conducted by Andrivon et al. (1) suggest that part of the mixture effect might be due to differences in the spatiotemporal pattern of disease spread, with disease progressing mainly along the rows in mixed-cultivar plots (lines of least resistance) as opposed to spatial spread in direct correspondence with the mean wind direction. This indicates the need for further research into the effects of patch size, shape, and distribution (crop arrangement) on the development of epidemics in genotype mixtures of potato. However, recent field studies by Garrett et al. (12) suggest that the results of studies into host-diversity effects on potato late blight from temperate regions cannot be extrapolated to humid, tropical regions where general epidemics are commonly observed and success in the use of genotype mixtures has been variable. A hypothesis has been proposed that the level of outside inoculum is an important factor driving the magnitude of host-diversity effects and that lower amounts of outside inoculum generally improve the efficacy of mixtures against potato late blight (12).

The primary objective of this paper is to determine whether predictions on the effect of genotype mixing in homogeneous mixtures also hold in nonideal mixtures. A nonideal mixture is defined here as having a coarser grain than an ideal mixture, i.e., there is a level of aggregation of matching genotypes, e.g., into rows or blocks. A spatiotemporal modeling framework was developed to run disease scenarios for general and focal epidemics in nonideal genotype mixtures at different scales and patterns and deduct from these simulations predictive relationships regarding the use of genotype mixtures in nonideal configurations. The model is parameterized for potato late blight. Because disease epidemics occur in both large- and small-scale production systems around the world, we developed a theoretical framework for evaluating effects of host diversity that encompasses both general and focal epidemics. This will provide theoretical support for future experimental research efforts aimed at developing globally applicable management strategies based on diversified landscape/mosaics. The secondary objectives of this paper are as follows: (i) investigate theories relating to the importance of crop arrangement in the use of genotype mixtures for disease suppression; (ii) assess the effects of crop arrangement and different descriptions of dispersal on the growth rate parameters, r and c ; (iii) develop a method for predicting trends in r and c in nonideal mixtures; (iv) investigate the potential use of r as a predictor of c ; and (v) develop an approximation formula for r .

THEORY AND APPROACHES

Theoretical framework. Epidemics may be classified on a scale ranging from focal to general, depending on the extent of primary infections. Following the groundbreaking work on ideal genotype mixtures by Van den Bosch et al. (39,41,42), temporal progress in focal epidemics may be characterized by the radial velocity of focus expansion, c . According to the theory, c ap-

proaches a constant value after an initial phase of focus build-up. Van den Bosch et al. (39) showed that the rate of c in monocultures can be approximated as

$$c = a \times \ln(R_0) + b \quad (1a)$$

where R_0 is the net lifetime reproduction, defined as the average number of offspring per mother, and a and b are parameters. Table 1 provides a summary of symbol definitions and dimensions. For ideal cultivar mixtures, equation 1a is changed to include f , the fraction of susceptible host plants in the mixture (2):

$$c = a \times \ln(fR_0) + b \quad (1b)$$

Because the mixing of susceptible and resistant plants is assumed to occur at a very fine grain ("ideal mixture"), net lifetime reproduction within a mixture can be equated to the product of net lifetime reproduction of a population of only susceptible individuals, R_0 , and the fraction of susceptible plants, f .

The apparent infection rate, r , is a frequently used measure of temporal progress of general epidemics. This apparent infection rate is a parameter in the logistic growth equation (47) and may be estimated from a time series of disease progress after logit transformation of severity (5):

$$\text{logit}(x) = rt + c \quad (2)$$

where x is disease severity on a scale from 0 to 1, t is time, and c is $\text{logit}(x_0)$, with $\text{logit}(x) = \ln(x/(1-x))$. Another way of conceptualizing r is offered by the life history theory whereby r is related to net lifetime reproduction, R_0 , and to generation time T , the average age of the mother at birth (13,19,36):

$$r \approx \frac{\ln(R_0)}{T} \quad (3)$$

For ideal mixtures, R_0 is replaced by fR_0 , similar to equation 1b. It follows from equation 1a and equation 3 that there must be a linear relationship between c and r .

In this paper, we move from ideal mixtures of susceptible and resistant plants to mixtures in arbitrary spatial configurations. The primary mechanism driving epidemic dynamics in time and space is successful propagation, which in turn depends on the spatial arrangement of the host population. We hypothesize that for such cases net lifetime reproduction within the mixture depends on both the fraction of susceptibles and their spatial configuration and we summarize these two effects in a new parameter q , the genotype connectivity. In landscape ecology, connectivity generally refers to the spatial connectedness of like habitat units in a landscape, as it affects the influence of landscape structure on the ability of an organism to move through the landscape (20). An increasing interest in connectivity research has led to a proliferation of connectivity measures (Tischendorf and Fahrig, 38). However, what constitutes a spatial connection between genotype units clearly depends on the application or process of interest; genotype units that are connected for insect dispersal are not necessarily connected for pathogen dispersal. It is therefore apparent that spatial connectivity must be assessed with consideration for the nature and scale of the interaction between the organism of interest and the landscape. The parameter q expresses the overall probability that a propagule produced in a susceptible genotype unit in the field (or landscape), when dispersed according to the dispersal kernel, will be deposited within (upon) a susceptible genotype unit (whether the same unit or another unit). In an ideal mixture, $q = f$. For any spatially heterogeneous mixture, q can be calculated by taking into account the dispersal kernel of propagules and the spatial patterns and scale of genotypes. Let $s(x,y)$ express the density of spores at location (x,y) ; $s(x,y)$ is 1 m^{-2} if the genotype is susceptible and 0 m^{-2} otherwise. Inoculum dispersal from a source at location $(0,0)$ is described by a kernel (normalized to a volume or probability mass under the kernel of

1), denoted by $K(x,y)$. Let (ξ,ψ) denote source locations and (x,y) target locations. Then we can write

$$q = \frac{\iint_{\xi\psi} s(\xi,\psi) \iint_{xy} H(s(x,y)) K(x-\xi, y-\psi) dy dx d\psi d\xi}{\iint_{\xi\psi} s(\xi,\psi) d\psi d\xi} \quad (4)$$

where H is the Heaviside function, which is 1 when its argument is greater than 0, and 0 otherwise. The interpretation is that the top integral measures the total number of spores produced by susceptibles, which when dispersed fall on a susceptible plant, and the bottom integral measures the total number of spores produced by susceptibles. q is therefore the probability that the spores produced will land on a susceptible genotype; q therefore expresses connectivity between genotypes. In ideal mixtures, q equals f , but in nonideal mixtures with large genotype units and a high degree of autoinfection within genotype units, q will tend to unity. In general, $f \leq q \leq 1$. As the area of all the susceptible genotype units in the landscape is fA , where A is the total area of the landscape, the above equation can be simplified to

$$q = \frac{1}{fA} \iint_{\xi\psi} s(\xi,\psi) \iint_{xy} H(s(x,y)) K(x-\xi, y-\psi) dy dx d\psi d\xi \quad (5)$$

To investigate these hypothesized relations, a spatiotemporal model of the potato late blight pathosystem was constructed.

Development of an integro-difference equation model for spatial epidemics of *P. infestans*. A spatially explicit age-structured integro-difference equation model was developed to simulate general and focal epidemics of *P. infestans* and to explore the effect of heterogeneous genotype mixtures on the two parameters characterizing the rate of spread in both situations, r and c . Space

is described as a two-dimensional grid in which each cell represents a potato plant that is either susceptible or resistant to *P. infestans*. In addition to its coordinates, each cell is characterized by state variables describing the number of lesions and their associated radius in age classes of 1 day. All other variables are derived from these basic state variables. The equations describing the number and area dynamics of lesions for each plant are based on the following principles. Every day there is a new generation of lesions and these belong to the same age class. Lesions are latent during the first λ days of their existence, the latency period, which was set at 5 days based on the popular Dutch cv. Bintje (9). Concomitant to development, the lesion grows radially. It is assumed that the center part of a lesion becomes infectious at lesion age $(\lambda + 1)$ days. Infectiousness of a segment of leaf area is taken to last 1 day. During each successive day, as another annulus of daily radial growth reaches age $(\lambda + 1)$ days, this annulus will sporulate. Spore production intensity per unit of infectious leaf area is accounted for by the parameter λ (set at 10^8 sporangia m^{-2}). A recent paper by Berger et al. (4) indicates the importance of lesion expansion as an epidemic component in polycyclic epidemics. In this model, the lesion growth rate on a particular plant, λ , is area-limited, i.e., there is a limit to lesion expansion due to competition:

$$\rho = \rho_m(1 - x) \quad (6)$$

where ρ_m is a cultivar-specific potential lesion growth rate and x is disease severity, the fraction of the host plant that is either latently infected, infectious, or removed. Thus, lesion size is restricted by the leaf area per plant as opposed to the leaf area per leaflet. A limit to lesion expansion due to the size of an “average” leaflet was removed from the model because model outputs were found to be insensitive to this parameter (*unpublished data*). This is in

TABLE 1. Symbols used in this study

Symbol	Unit	Description
A	m^2	Total area of the landscape
A_L	m^2	Area of a lesion
A_p	m^2	Area of a plant
c	$m \text{ day}^{-1}$	Radial velocity of focus expansion
f	— ^a	Fraction of susceptible host plants in the mixture
i	—	Infection efficiency
IP	day	Infectious period
K	—	Dispersal kernel: probability distribution of dispersal distances
LAI	—	Leaf area index
N_i	—	Number of infected plants
q	—	Genotype connectivity: the overall probability that a propagule produced in a susceptible genotype unit, when dispersed according to the dispersal kernel, will be deposited upon a susceptible genotype unit (whether the same unit or another unit)
Q_i	m^2	Area of infected plant material
r	day^{-1}	Apparent infection rate
r_i	m	Focal radius
r_1	day^{-1}	Approximation for the apparent infection rate
r_2	day^{-1}	Approximation for the apparent infection rate
R	m	Radius of a leaflet
R_L	m	Radius of a lesion
R_0	—	Net lifetime reproduction
s	—	Susceptibility
S	—	Total spore production on a leaflet
t	day	Time
T	day	Mean generation time: average time between birth of an individual and birth of its offspring
x	—	Disease severity: overall percentage of diseased tissue on the susceptible genotype
α	m^{-1}	Steepness of disease gradient
δ	—	Dispersal efficiency
θ	m	Standard deviation in the two-dimensional Gaussian kernel
κ	—	Disease severity threshold
λ	day	Latent period
μ	day	Mean of the entire sporulation curve
μ_d	m	Mean dispersal distance
ρ	$m \text{ day}^{-1}$	Lesion growth rate
ρ_m	$m \text{ day}^{-1}$	Cultivar-specific potential lesion growth rate
σ	m^{-2}	Sporulation intensity (per unit of infectious leaf area)
σ_c	day	Standard deviation of the entire sporulation curve

^a — Signifies that the defined quantity is dimensionless.

agreement with analysis of a Leslie matrix model by Powell et al. (32), which shows that a leaflet restriction is not important in the calculation of the apparent infection rate.

For each individual potato plant, the model thus keeps track of the number and radius of lesions of all age classes and calculates the number of new lesions per plant at each time step. The areas of latent infectious and dead lesion area on each host plant are calculated by multiplying the annuli of lesions in the appropriate age category with their respective areas and summing over all lesions. The age-structured population model for lesions on each plant is updated daily by “aging” the existing lesions by 1 day and starting new lesions at a rate calculated as the product of the number of landed spores per square meter of leaf area in a day, the infection efficiency, and fraction of area not yet occupied by *P. infestans*, $(1 - x)$. No calculations were made involving individual lesions.

Dispersal. Host plants are linked through dispersal of spores. Ecological data on short-range dispersal of *P. infestans* are available (31). We describe where spores land using dispersal kernels, which describe the probability distribution of landing locations of spores in the two-dimensional plane. Spores were dispersed either according to a radial Laplace kernel:

$$K(x, y) = \frac{\alpha^2}{2\pi} e^{-\alpha\sqrt{x^2+y^2}} \quad (7)$$

or a radial Gaussian kernel:

$$K(x, y) = \frac{1}{2\pi\theta^2} e^{-\frac{x^2+y^2}{2\theta^2}} \quad (8)$$

In the Laplace kernel, α (m^{-1}) is a factor describing disease gradient steepness, while θ (m) represents the standard deviation in the two-dimensional Gaussian kernel. The positions x and y are distances from the source plant, and $K(x, y)$ is the probability density at location (x, y) . Both dispersal kernels describe the net results of aerial dispersal of spores in time coupled with deposition to the ground.

The radial Laplace and the radial Gaussian kernels can be compared in terms of mean dispersal distance, μ_d , in the plane, i.e., the mean distance traveled by spores. For a radial Laplace kernel, $\mu_d = 2/\alpha$, whereas for a radial Gaussian kernel, $\mu_d = \theta\sqrt{\pi/2}$. We used three dispersal kernels in simulation experiments (Fig. 1). The first two are radial Laplace kernels, parameterized with data from Paysour and Fry (31) for dispersal of spores of *P. infestans*, yielding $\alpha = 0.82 \text{ m}^{-1}$ in nonirrigated potatoes and $\alpha = 2.1 \text{ m}^{-1}$ in irrigated potatoes. The associated mean dispersal distances are $\mu_d = 2.44 \text{ m}$ in nonirrigated potatoes and $\mu_d = 0.95 \text{ m}$ in irrigated potatoes, providing a “wide Laplace kernel” and a “narrow Laplace kernel,” respectively. A radial Gaussian kernel was included as the third dispersal kernel in order to investigate the sensitivity of model outcomes to a differently shaped kernel (thinner tails) for the same μ_d . The value of θ for the Gaussian kernel was 1.95 m, yielding the same value for μ_d as the radial Laplace kernel for nonirrigated potatoes, i.e., 2.44 m.

Solution and implementation. The integro-difference equation (IDE) model was implemented in MATLAB version 5.3.0.10183 (The MathWorks, Natick, MA). IDEs enable continuous integration over space of spatial phenomena with a discrete temporal behavior. The IDE approach permits great flexibility in incorporating dispersal distributions and demographic processes and can be linked closely to empirical data, as in this study. Spatial phenomena are solved by multiplying fast Fourier transforms of spatial distributions and dispersal kernels and back-transforming the result. This solution method requires periodic boundary conditions; therefore in the Fourier or frequency domain, the defined field is represented as a torus. As a result, any spore dispersing outside of the field’s borders will instantaneously re-appear on the opposite edge of the field. As a result of this assumption, the

simulated domain should be interpreted as a “window” on an infinite repetition of identical windows contiguously over space. Back-transformation of the result to the spatial domain gives the distribution of dispersed spores over the simulated domain. Solution of spatial phenomena by fast Fourier transforms enhances the speed and accuracy of the numerical solution of the model.

Simulation. As a result of the solution methodology, each field we define in our simulation experiments is not an isolated island but is part of, and is influenced by, a larger virtual environment. Thus, any spore that leaves the edge of the field and then reappears on the opposite edge is assumed to be from a distant source of inoculum, i.e., a neighboring or distant potato field. This gives a realistic representation of general epidemics of potato late blight as they occur in large potato growing regions. For the simulation of general epidemics in isolated fields, genotype mixtures are “padded” with a large border of resistant space (padded mixtures); the simulated domain can therefore be thought of as a window to an infinite repetition of identical, isolated genotype mixtures. For the simulation of focal epidemics, we assume that the initial inoculum is from a smaller, local source, e.g., a volunteer plant, and that the influence of the larger virtual environment can be ignored. All focal simulation experiments were stopped long before the focus, or any spores, could reach the field edges; therefore, the toroidal symmetry did not interfere with focal expansion.

In the simulation of general epidemics, a simulation time of 15 days was sufficient to reach a disease severity level of approximately 40% and make robust estimates of r using equation 2. The length of each simulation for the focal simulation experiments was determined by a need for sufficient focus build-up with which to gain a reliable calculation of c , but preventing significant travel of spores over the field boundaries (and reappearing at the opposite boundary due to the toroidal symmetry). Simulation times were chosen appropriately for the dispersal kernel used and were derived by measuring the time taken to reach a final disease severity of 20% in a pure-line susceptible population, under each dispersal kernel. This ensured that focus expansion was stopped

Fig. 1. Dispersal kernels describing the probability distributions of landing locations of spores around the source in the two-dimensional plane, as used in model simulations of potato late blight. L1 = wide Laplace kernel, L2 = narrow Laplace kernel, and G = Gaussian kernel, with mean dispersal distances of 2.44, 0.95, and 2.44 m, respectively.

sufficiently early in each field. The resultant simulation times were 34 days for the wide Laplace kernel, 54 days for the narrow Laplace kernel, and 37 days for the Gaussian kernel.

Based on planting patterns in the Netherlands, the length of the side of a compartment cell was set at 0.75 m (distance between rows) and the width of each compartment cell at 0.3 m (distance between plants). The growth of the host plant was not considered in this model; therefore, LAI was held constant at a value of 5. This is approximately the maximum LAI that a potato crop can reach and was used to represent a well-developed potato crop in prime condition. We assumed perfect weather conditions for late blight development; therefore, other variables were held constant: $\lambda = 5$ days, $IP = 1$ day, $\rho = 0.004 \text{ m day}^{-1}$, $i = 0.01$, and $\sigma = 10^8 \text{ m}^{-2}$. These are estimates (averages) based on conditions found in the Netherlands (9). In the absence of any information in the literature, an estimate of 0.1 was used for the dispersal efficiency, δ .

Data analysis. Disease severity, x , was derived by calculating the overall proportion of diseased tissue on the susceptible genotype. In all simulation experiments for general epidemics, progress of disease was expressed as r , the apparent infection rate that was estimated by linear regression of $\logit(x)$ versus time (equation 2) as simulated with the IDE model.

Radial velocity of focus expansion, c. The radial velocity of focus expansion, c , was calculated as the slope of the linear regression of the calculated focus radius on time. Focal radius, r_t , was derived by first calculating the crop area infected. This was determined by counting the number of plants with a disease severity greater than a threshold of 1% (κ), multiplying this number by the area of a plant, and dividing the result by the fraction of susceptible host plants in the mixture:

$$Q_t = \frac{N_t A_p}{f} = \text{Area covered by focus} = \pi r_t^2 \quad (9)$$

where Q_t is the area infected, N_t is the number of infected plants above the severity threshold κ , A_p is the area of a plant, and f is the fraction of susceptibles in the mixture. Rewriting equation 9 to make r_t the subject gives

$$r_t = \sqrt{\frac{Q_t}{\pi}} \quad (10)$$

Radius calculations prior to day 25 were not used in order to avoid distortion of the final statistic created by the initial phase of focus build-up.

Fig. 2. Genotype mixtures used to study the effect of number of genotype units on the behavior of simulated potato late blight epidemics. White blocks represent plants susceptible to *Phytophthora infestans* and black areas represent resistant plants. Field dimensions are **A**, 1.5 × 2.4 m; **B**, 3 × 4.8 m; **C**, 6 × 9.6 m; **D**, 12 × 19.2 m; **E**, 24 × 38.4 m; **F**, 48 × 76.8 m; **G**, 24 × 38.4 m; and **H**, 48 × 76.8 m. Patterns **A** to **F** are “padded” with a border of nonhost plants approximately 50 times as wide as the mean dispersal distance of the dispersal kernel, isolating them from the influence of a larger virtual environment. Fields **G** to **H** have no border of resistant space and are influenced by a larger virtual environment. Genotype unit area is held constant at 0.3 × 0.75 m, providing **A** to **H**, 4, 16, 64, 256, 1,024, 4,096, 1,024, and 4,096 susceptible genotype units, respectively. The fraction, f , of susceptible host plants in the population is held constant at 0.25. Genotype units for patterns **A** to **H** are rectangular because the distance between rows is 0.75 m while the distance between plants is 0.3 m. The number of genotype units shown in each pattern has been decreased to enhance clarity.

For focal epidemics in a nonideal mixture, the spread of the focus can be restricted by the spatial configuration of the host plants, and thus, noncircular foci can occur, e.g., spread of disease along the length of an isolated row. In such cases there would be benefit to measuring spread velocity in several different compass directions; however, the area metric described above is sufficient to describe the impact of crop arrangement on the expansion of disease foci because it represents an average radial expansion over time.

There is evidence for an increasing frontal velocity when larger spatial scales are considered; the theory of turbulent diffusion suggests that spread could become more efficient as the focus expands (8,21,35). However, a constant c is an emergent property of this model, and as this model operates at the field scale, it gives a realistic description of the life cycle of the pathogen and a realistic description of short-scale dispersal.

Importance of scale in the use of genotype mixtures for disease suppression. Pioneering simulation and experimental work (3,24–28) revealed the importance of crop arrangement, in particular the genotype unit area, in the effectiveness of genotype mixtures for disease control. However, the results of further simulation studies by Mundt and Brophy (22) suggested that the number of host genotype units in a population is a more important determinant of the effectiveness of mixtures for disease control than genotype unit area. In order to investigate this, we tested the following null hypothesis: the size of the field (window) will not affect model results. The null hypothesis was tested by exploring the effects of increasing the number of genotype units in a field, while maintaining the proportion of susceptible crop and its spatial configuration. Mundt and Brophy (22) simulated potato late blight epidemics in mixtures surrounded by resistant space and isolated from external sources of inoculum, but in this study we will test this null hypothesis by simulating epidemics in mixtures that are isolated from outside inoculum (padded mixtures) and in mixtures that are part of an interconnected network of like fields (unpadded mixtures). Individual host plants of a genotype unit area of 0.225 m^2 ($0.3 \times 0.75 \text{ m}$) were first arranged in a 1:4 mixture (25% susceptible) with an area of 3.6 m^2 ($1.5 \times 2.4 \text{ m}$) (Fig. 2A). This area was successively quadrupled to give identical mixtures in terms of the configuration and proportion of susceptible host plants, but with areas of 14.4 m^2 ($3 \times 4.8 \text{ m}$), 57.6 m^2 ($6 \times 9.6 \text{ m}$), 230.4 m^2 ($12 \times 19.2 \text{ m}$), 921.6 m^2 ($24 \times 38.4 \text{ m}$), and $3,686.4 \text{ m}^2$ ($48 \times 76.8 \text{ m}$), respectively (Fig. 2B to F). In order to simulate epidemic development in fields that are isolated, each of the defined mixtures was padded with a border of nonhost plants approximately 50 times as wide as the mean dispersal distance of the dispersal kernel. In order to simulate the influence of a larger interconnected network of host fields on epidemic development, two further simulation experiments were devised, whereby the border of resistant space around the two largest padded mixtures previously defined was removed to give an infinite repetition of identical genotype mixtures contiguously over space (Fig. 2G to H). Only the two largest mixtures were used because smaller landscapes result in a small number of coefficients in the Fourier transform, which is not enough to represent the functions adequately. General inoculation in each experiment proceeded with the allocation of 1 cm^2 of infectious tissue to each host plant. This gives an initial disease severity of approximately 0.01%. Spore dispersal was provided by the wide Laplace kernel. The disease severities reached in each genotype mixture were then compared with those of pure-line susceptible populations of the same size to provide relative disease severity ratings.

Effect of crop arrangement and dispersal kernel on r and c . In order to investigate the effects of altering the configuration and density of susceptible genotype units on r and c in nonideal mixtures, two further sets of simulation experiments were devised. General epidemics were simulated in the first set of experiments and focal epidemics were simulated in the second set. In both sets

of simulation experiments, we assume that the genotype mixtures are part of a larger interconnected network of like fields; therefore, unpadded mixtures are defined. In each set, block and row patterns with genotype unit areas of $0.3 \times 0.75 \text{ m}$ and $76.8 \times 0.75 \text{ m}$, respectively, were generated for four different fractions, f , of susceptibles in the genotype mixtures: 0.125, 0.25, 0.5, and 1 (Fig. 3). Thus, identical genotype mixtures were used in each set of simulation experiments. The different f values were created by doubling the number of susceptible units in a mixture with each successive f category and locating the new susceptible units equidistantly between the old units. The genotype unit areas were held constant throughout at the aforementioned values. The size of the field remained fixed throughout at $76.8 \times 48 \text{ m}$. General inoculation proceeded as above with the allocation of 1 cm^2 of infectious tissue to each host plant, while focal inoculation proceeded with the allocation of 1 cm^2 of infectious tissue to each of the central nine host plants.

In order to gain insight into the effects of different dispersal kernels on our results, each individual simulation experiment outlined in this section was repeated for each of the three dispersal kernels described in the dispersal section.

Fig. 3. Genotype mixtures used to study the effect of spatial configuration and fraction, f , of susceptible host plants on the behavior of simulated potato late blight epidemics. White blocks represent plants susceptible to *Phytophthora infestans* and black areas represent resistant plants. f values are **A and D**, 0.125; **B and E**, 0.25; **C and F**, 0.5; and **G**, 1. The size of the field is held constant at $76.8 \times 48 \text{ m}$. Genotype unit areas are **A to C**, $0.3 \times 0.75 \text{ m}$; **D to F**, $76.8 \times 0.75 \text{ m}$; and **G**, $76.8 \times 48 \text{ m}$ —providing 2,048, 4,096, 8,192, 8, 16, 32, and 1 susceptible genotype units, respectively. Genotype units for patterns **A to C** are rectangular because the distance between rows is 0.75 m while the distance between plants is 0.3 m .

Parameter q as predictor of r and c . For the set of general simulation experiments outlined in the previous section, r was plotted against $\ln(q)$ to investigate if q could provide reliable predictions of crop arrangement on the progress of disease, in a similar manner as the rate of focus expansion, c , is linearly related to $\ln(f)$ (40). The same procedure was then repeated for the set of focal simulation experiments; c was plotted against $\ln(q)$ to investigate if q could predict or characterize the impact of crop arrangement on the progress of disease in focal epidemics.

Parameter r as predictor of c . In order to develop a theoretical framework for host diversity that incorporates both general and focal epidemics, it is necessary to determine whether the results of studies in host-diversity effects on potato late blight from temperate regions can be extrapolated to humid, tropical regions. The hypothesized relationship between the two emergent measures of epidemic development, r and c , was investigated by regressing radial velocity calculations against the apparent infection rates in corresponding genotype mixtures.

Approximation formulae for r . In addition to calculation of r based on simulation results, life table analysis for lesions was used to predict the value of r . This analysis is based on an original idea of Spijkerboer (37). A predictive estimate of r has been derived from q and equation 3 based on the following components: the genotype connectivity, q ; the sporulation intensity, σ ; the dispersal efficiency, δ ; the infection efficiency, i ; the radius of a leaflet, R ; the latency period, λ ; the potential radial growth rate of a lesion, ρ_m ; and time, t . The radius of a lesion is calculated as follows:

$$R_L = \rho_m t \quad (11)$$

in which it is assumed that $R_L = 0$ at $t = 0$ and only one infection occurs on the leaflet at $t = 0$. So, restriction of lesion expansion due to competition for space (equation 6) does not need to be taken into account. It therefore follows that the area of a lesion is given by

$$A_L = \pi R_L^2 = \pi (\rho_m t)^2 = \pi \rho_m^2 t^2 \quad (12)$$

The rate of change of lesion area over time is therefore given by the equation

$$\frac{d(A_L)}{dt} = 2\pi \rho_m^2 t \quad (13)$$

Total spore production on a leaflet, S , is now written as

$$S = \int_{\lambda}^{\lambda + \frac{R}{\rho_m}} 2\pi \sigma \rho_m^2 (t - \lambda) dt = \sigma \pi R^2 \quad (14)$$

where R is derived from an average leaflet area of 11 cm², approximating the size of small leaflets. The generation time T can now be written as

$$T = \frac{1}{S} \int_{\lambda}^{\lambda + \frac{R}{\rho_m}} t 2\pi \sigma \rho_m^2 (t - \lambda) dt = \lambda + \frac{2}{3} \frac{R}{\rho_m} \quad (15)$$

Multiplication of equation 14 by the dispersal efficiency, δ , and the infection efficiency, i , gives the final equation for the calculation of R_0 :

$$R_0 = \sigma \delta i \pi R^2 \quad (16)$$

where R_0 represents the net lifetime replacement of infected leaflets, assuming only one infection per leaflet. Substitution of equations 15 and 16 into equation 3 gives an approximation of r in homogenous space:

$$r_1 \approx \frac{\ln(\sigma \delta i \pi R^2)}{\lambda + \frac{2}{3} \frac{R}{\rho_m}} \quad (17)$$

Incorporation of q into the numerator of equation 17 gives a predictive measure for r in heterogeneous space:

$$r_1 \approx \frac{\ln(q \sigma \delta i \pi R^2)}{\lambda + \frac{2}{3} \frac{R}{\rho_m}} \quad (18)$$

Recent theory provides a second approximation formula for r (36):

$$r_2 \approx \frac{\ln R_0}{\mu} \left[1 + \frac{1}{2} \left(\frac{\sigma_c}{\mu} \right) \ln R_0 \right] \quad (19)$$

where μ and σ_c values can be calculated from measurements of the sporulation curve, and R_0 from equation 16 multiplied by q . In order to investigate the accuracy of these approximation formulae, r_1 and r_2 will be used to predict r in the set of simulation experiments for general epidemics.

RESULTS

Importance of scale in the use of genotype mixtures for disease suppression. For simulation experiments A to F (Fig. 2), an increase in the number of genotype units via an increase in the size of the field always resulted in an increase in q and in the final disease severity (Table 2). Relative disease severities decreased from patterns A to C (Fig. 2) and stabilized thereafter. For simulation experiments G and H (Fig. 2), an increase in the number of

TABLE 2. Simulated effect of the number and aggregation of susceptible host genotype units on disease severity of potato late blight

Pattern ^a	Overall area (m ²)	Number of units	q^b	% Disease severity ^c		
				Pure-line ^d	Mixture ^e	Relative ^f
A	3.6	4	0.05	5.41	3.01	0.56
B	14.4	16	0.10	15.23	6.73	0.44
C	57.6	64	0.16	29.43	12.02	0.41
D	230.4	256	0.20	40.04	16.27	0.41
E	921.6	1,024	0.23	45.89	18.74	0.41
F	3,686.4	4,096	0.24	48.94	20.04	0.41
G	921.6	1,024	0.25	52.08	21.40	0.41
H	3,686.4	4,096	0.25	52.08	21.40	0.41

^a Spatial configuration of host plants as shown in Figure 2.

^b Genotype connectivity: the overall probability that a propagule produced in a susceptible genotype unit, when dispersed according to the dispersal kernel, will be deposited upon a susceptible genotype unit (whether the same unit or another unit) (equation 5). Spore dispersal was provided by a wide radial Laplace kernel in order to simulate dispersal in nonirrigated potato plots.

^c Disease severity calculated as the percentage of diseased foliar surface on the susceptible genotype.

^d Final disease severity for pure-line susceptible population.

^e Final disease severity for mixtures consisting of 25% susceptible and 75% immune plants.

^f Relative disease severity, i.e., disease severity for the mixture divided by disease severity of the pure-line susceptible population of the same size.

genotype units via an increase in the size of the field had no effect on q or on the final or relative severity values (Table 2).

Effect of crop arrangement and dispersal kernel on r and c . In all simulation experiments, q was found to increase with increasing f values, with consistently higher values for the row patterns and the narrow Laplace dispersal kernel followed by the wide Laplace and Gaussian kernels (Table 3).

In the simulated general epidemics, r increased with increasing f values in an analogous fashion to q , and also demonstrated consistently higher values for the row patterns and the narrow Laplace dispersal kernel followed by the wide Laplace and Gaussian kernels (Table 3).

In the simulated focal epidemics, c reached a constant value and increased linearly with the logarithm of the fraction, f , of susceptible plants (Table 3; Fig. 4). Unlike the results for q and r , higher velocities were reached with the wide Laplace kernel followed by the Gaussian and narrow Laplace kernels (Table 3; Fig. 4).

Parameter q as predictor of r and c . Plotting r as a function of $\ln(q)$ gave an effective indication of trends in r for all three dispersal kernels for the block patterns ($R^2 > 0.99$, $P \leq 0.005$) (Fig. 5). For the row patterns, plotting r as a function of $\ln(q)$ gave an effective indication of trends in r for the wide Laplace and Gaussian kernels ($R^2 > 0.99$, $P \leq 0.005$); however, this relationship was weaker for the narrow Laplace kernel ($R^2 = 0.90$, $P = 0.05$) (Fig. 5). Plotting c as a function of $\ln(q)$ also gave an effective indication of trends in c for all three dispersal kernels for the block patterns ($R^2 > 0.99$, $P \leq 0.005$) and for row patterns for the wide Laplace and Gaussian kernels ($R^2 > 0.95$, $P \leq 0.05$), whereas this relationship was again weaker for the row patterns under the narrow Laplace kernel ($R^2 = 0.88$, $P = 0.06$) (Fig. 6). This slight disparity resulted from similarly high values of q for f values of 0.125 and 0.25. In these simulation experiments, the combination of a steep dispersal gradient, with $\mu_d = 0.95$ m, and relatively large

gaps between susceptible genotype units resulted in spore deposition only within rows. As a result, similar q values were observed in these two cases.

Parameter r as predictor of c . Figure 7 shows that in all simulation experiments, r and c were highly correlated ($R^2 > 0.95$), with little difference in the strength of the relationship for the three dispersal kernels. For the block patterns, these relationships were highly significant ($P \leq 0.001$), and also for the row patterns ($P \leq 0.05$). The spatial configuration of genotype units had little

Fig. 4. Relationship between the radial velocity of focus expansion, c , and the logarithm of the fraction of susceptible host plants, f , in simulated potato late blight epidemics in genotype mixtures of susceptible and fully resistant potato cultivars. Individual genotype units were aggregated on the basis of their resistance level into block patterns and row patterns. These configurations are given in Figure 3. Spores were disseminated using three dispersal functions: $\diamond$ = wide Laplace kernel, $\triangle$ = narrow Laplace kernel, and $\blacksquare$ = Gaussian kernel, with mean dispersal distances of 2.44, 0.95, and 2.44 m, respectively. The four points in each data set relate to calculations of c at four fractions, f , of susceptible plants in the host population: 0.125, 0.25, 0.5, and 1. Genotype unit area was held constant at 0.3×0.75 m for block patterns, 76.8×0.75 m for row patterns, and 76.8×48 m for pure-line susceptible host populations.

TABLE 3. Simulated effect of dispersal kernel and configuration, density, and aggregation of susceptible genotype units on development of general and focal potato late blight epidemics

Dispersal kernel	Pattern ^a	f^b	Genotype			General				Focal
			Unit area (m ²)	No. of units	q^c	r^d day ⁻¹	r_1^e day ⁻¹	r_2^f day ⁻¹	sev ^g (%)	c^h (m day ⁻¹)
Wide Laplace	A	0.125	0.225	2,048	0.130	0.400	0.328	0.336	14.851	0.680
	B	0.250	0.225	4,096	0.253	0.467	0.410	0.422	21.399	0.784
	C	0.500	0.225	8,192	0.501	0.542	0.494	0.512	33.217	0.893
	D	0.125	57.600	8	0.205	0.416	0.384	0.395	18.888	0.680
	E	0.250	57.600	16	0.280	0.472	0.422	0.436	22.765	0.794
	F	0.500	57.600	32	0.508	0.543	0.496	0.514	33.503	0.895
	G	1.000	3,686.400	1	1.000	0.627	0.579	0.604	52.078	1.027
Narrow Laplace	A	0.125	0.225	2,048	0.202	0.416	0.382	0.392	18.719	0.276
	B	0.250	0.225	4,096	0.301	0.475	0.431	0.445	23.803	0.311
	C	0.500	0.225	8,192	0.523	0.544	0.499	0.518	34.154	0.353
	D	0.125	57.600	8	0.492	0.461	0.492	0.510	32.873	0.294
	E	0.250	57.600	16	0.498	0.503	0.493	0.511	33.095	0.318
	F	0.500	57.600	32	0.599	0.553	0.516	0.536	37.408	0.354
	G	1.000	3,686.400	1	1.000	0.627	0.579	0.604	52.078	0.395
Gaussian	A	0.125	0.225	2,048	0.125	0.399	0.323	0.331	14.549	0.518
	B	0.250	0.225	4,096	0.250	0.466	0.408	0.421	21.224	0.578
	C	0.500	0.225	8,192	0.500	0.542	0.494	0.512	33.153	0.633
	D	0.125	57.600	8	0.156	0.406	0.350	0.359	16.270	0.479
	E	0.250	57.600	16	0.250	0.466	0.408	0.421	21.230	0.575
	F	0.500	57.600	32	0.500	0.542	0.494	0.512	33.153	0.634
	G	1.000	3,686.400	1	1.000	0.627	0.579	0.604	52.078	0.689

^a Spatial configuration of host plants as shown in Figure 3.

^b Fraction of susceptible host plants in the mixture.

^c Genotype connectivity: the overall probability that a propagule produced in a susceptible genotype unit, when dispersed according to the dispersal kernel, will be deposited upon a susceptible genotype unit (whether the same unit or another unit) (equation 5).

^d Apparent infection rate (equation 2).

^e Approximation for the apparent infection rate (equation 18).

^f Approximation for the apparent infection rate (equation 19).

^g Disease severity calculated as the percentage of diseased foliar surface on the susceptible genotype.

^h Radial velocity of focus expansion.

effect on these relationships, with the data for row patterns under the narrow Laplace dispersal kernel again exhibiting a slightly poorer fit.

Approximation formulae for r . Values of r_1 closely resembled those of r (Table 3) with the relative bias, $(r - r_1/r) \times 100$, ranging from 1.97 to 19.05%. The mean bias for the entire set of simulation experiments was 9.8%. A slightly better approximation to r was achieved with r_2 (Table 3), with the relative bias ranging between 1.65 and 17.10% and a mean bias of 7.2%.

DISCUSSION

The results from the first set of simulation experiments lead to the rejection of the null hypothesis; an increase in the number of genotype units via an increase in the size of the field did have an effect on the results, but only in the simulations of isolated fields where padded mixtures were used (Table 2, A to F). For simulation experiments G and H, an increase in the size of the field clearly had no effect on model results. These differences in model results were due to differences in assumptions regarding dispersal; in patterns A to F, the genotype mixtures are surrounded by a resistant space that facilitates spore loss from outside the mixture boundaries, whereas in patterns G and H, we assume that inoculum from the surrounding (alike) environment compensates for this loss. It is clear from Table 2 that the final disease severities for both the pure-line populations and the mixtures increase from patterns A to F. This is an expected result and can be interpreted with the use of q . As we increase the size of the field, a smaller proportion of inoculum is lost outside the field edges and, by cor-

ollary, the connectivity between the host plants increases; thus, q increases until its value approaches that of f . When we examine the relative severity values, we see that the effectiveness of a mixture, in terms of disease control, increases with an increase in the number of genotype units within a field and then finally stabilizes. Thus, if a genotype mixture is surrounded by a resistant space and isolated from external sources of inoculum, then the number of genotype units will exert an influence on model results and the null hypothesis is rejected. These results are in agreement with those of an earlier simulation study by Mundt and Brophy (22) where potato late blight epidemics were also simulated in isolated mixtures. As a result, it was concluded that the epidemiological impact of the number of genotype units is of more importance than the genotype unit area (22). However, in simulation experiments G and H, we make a different assumption regarding spore dispersal; the simulated domain is a window onto an infinite lattice of identical, contiguous windows, i.e., there is no resistant space between the mixtures in the lattice. As a result, increasing

Fig. 5. Relationship between the apparent infection rate, r , and the logarithm of the genotype connectivity parameter, q , in simulated potato late blight epidemics in genotype mixtures of susceptible and fully resistant potato cultivars. Individual genotype units were aggregated on the basis of their resistance level into block patterns and row patterns. These configurations are given in Figure 3. Spores were disseminated using three dispersal functions: $\blacklozenge$ = wide Laplace kernel, $\blacktriangle$ = narrow Laplace kernel, and $\blacksquare$ = Gaussian kernel, with mean dispersal distances of 2.44, 0.95, and 2.44 m, respectively. The four points in each data set relate to calculations of r and q at four fractions, f , of susceptible plants in the host population: 0.125, 0.25, 0.5, and 1. Genotype unit area was held constant at 0.3×0.75 m for block patterns, 76.8×0.75 m for row patterns, and 76.8×48 m for pure-line susceptible host populations.

Fig. 6. Relationship between the radial velocity of focus expansion, c , and the logarithm of the genotype connectivity parameter, q , in simulated potato late blight epidemics in genotype mixtures of susceptible and fully resistant potato cultivars. Individual genotype units were aggregated on the basis of their resistance level into block patterns and row patterns. These configurations are given in Figure 3. Spores were disseminated using three dispersal functions: $\blacklozenge$ = wide Laplace kernel, $\blacktriangle$ = narrow Laplace kernel, and $\blacksquare$ = Gaussian kernel, with mean dispersal distances of 2.44, 0.95, and 2.44 m, respectively. The four points in each data set relate to calculations of velocity at four fractions, f , of susceptible plants in the host population: 0.125, 0.25, 0.5, and 1. Genotype unit area was held constant at 0.3×0.75 m for block patterns, 76.8×0.75 m for row patterns, and 76.8×48 m for pure-line susceptible host populations.

Fig. 7. Relationship between apparent infection rate, r , and radial velocity of focus expansion, c , in simulated potato late blight epidemics in genotype mixtures of susceptible and fully resistant potato cultivars. Individual genotype units were aggregated on the basis of their resistance level into block patterns and row patterns. These configurations are given in Figure 3. Spores were disseminated using three dispersal functions: $\blacklozenge$ = wide Laplace kernel, $\blacktriangle$ = narrow Laplace kernel, and $\blacksquare$ = Gaussian kernel, with mean dispersal distances of 2.44, 0.95, and 2.44 m, respectively. The four points in each data set relate to calculations of c and r at four fractions, f , of susceptible plants in the host population: 0.125, 0.25, 0.5, and 1. Genotype unit area was held constant at 0.3×0.75 m for block patterns, 76.8×0.75 m for row patterns, and 76.8×48 m for pure-line susceptible host populations. The dashed line represents a 1:1 relationship.

the size of the field but holding all other aspects of crop arrangement constant has no effect on q (or other model results) because spore loss does not occur in these two simulation experiments. This demonstrates that spore loss from outside the field boundaries is the mechanism responsible for the difference in model results in patterns A to F. Because spore loss from any field is the net result of the influx of external inoculum and the dispersal of inoculum outside the field boundaries, it is clear that the number of genotype units within a field should not be considered independently from the resistant space that surrounds the field. Because *P. infestans* sporangia have been observed to disperse and infect potato fields up to 900 m from the source (49), it may be of more relevance, particularly in major potato growing regions, to assume that individual potato fields are part of an interconnected network of fields rather than isolated patches of host plants. This is particularly true in highland, tropical regions where year-round potato production and the continuous presence of late blight have been described (15,29). Our results suggest that in such regions where there may be a continual inflow of outside inoculum, the pattern and scale of the genotype units could be of more importance to the individual farmer than the size of his or her field.

In any spatial model of an aerially disseminated pathogen, the function used to disperse the propagules is of prime importance. The radial Laplace and Gaussian dispersal kernels represent two such commonly used functions, and in this study, the mean dispersal distances for these kernels were taken from the results of experiments on potato late blight contained in a seminal paper on interplot interference by Paysour and Fry (31). In the simulations of general epidemics, q and r values were slightly higher under the narrow Laplace kernel followed by the wide Laplace and Gaussian kernels. This is an expected result because steeper pathogen dispersal gradients often result in smaller host-diversity effects on disease due to a reduction in mixing effect (10). For focal epidemics, the simulation experiments showed that c can be calculated in nonideal mixtures; constant velocities were reached in all simulation experiments and c was found to increase linearly with the logarithm of f in all cases. Despite the occurrence of non-circular foci, the simulation results show that c can still be used as a measure of spatial spread over time. This is by virtue of the area method adopted to derive c , whereby the final velocity obtained represents an average radial expansion over time expressed in meters per day. For the focal simulation experiments, c values were highest under the wide Laplace kernel followed by the Gaussian and the narrow Laplace kernels. Here, there was a marked difference between the three sets of results. Unlike general epidemics where each plant is a source, in nonideal mixtures, focus expansion is governed by the spatial arrangement of susceptible genotypes in the host population. Table 3 shows that q values are higher for both block and row patterns under the narrow Laplace kernel; here, dampening of the spore cloud results in an increase in the number of autoinfections because a larger proportion of spores land back on the source plant. However, despite a loss in mixing effect, c values are considerably lower under this dispersal kernel. It is therefore apparent that simulated c values are greater under dispersal functions that afford the opportunity to breach areas of host resistance. These results reinforce the need to study dispersal when epidemics have a focal aspect; they further illustrate the importance of future experimental research aimed at gathering ecological data on the short-range dispersal of *P. infestans*.

Our results show that q can be used to predict the impact of crop arrangement on the development of both general and focal epidemics. The linear relationships between $\ln(q)$ and r and $\ln(q)$ and c were highly significant, indicating that q can be used effectively to predict changes in both r and c across heterogeneous host populations. However, simulation results for dispersal according to the narrow Laplace kernel in row patterns at f values

of 0.125 and 0.25 did not reveal a linear relationship between $\ln(q)$ and r or $\ln(q)$ and c . Here, the combination of a steep dispersal gradient, with $\mu_d = 0.95$ m, and multiple resistant rows of 0.75 m wide results in the pathogen being unable to breach the gap between neighboring susceptible rows. As a consequence, spores are deposited only within rows, leading to similarity in q values and in disease for these two cases (Table 3). In all other cases, the linear relationships between $\ln(q)$ and r and $\ln(q)$ and c approached direct proportionality. It is therefore apparent that parameter q is a sound measure for predicting the disease suppressiveness of landscapes. However, caution must be used in interpreting these results. In general epidemics, variation in the landscape is being "averaged out" because each plant is a source. This may be inferred from Figure 5 where the lines (excluding the anomaly caused by very wide inter-rows) for the various kernels and landscapes seem to almost coincide when superimposed. For focal epidemics, however, variation in the landscape at the scale of the dispersal kernel has a strong effect on the development rate of the epidemic. This can be inferred from Figure 6, where the lines, if combined in one graph, do not overlap. This demonstrates that for focal epidemics, information on q proves information on c only if information on the dispersal function is also available.

The results of this modeling study indicate that the spatial arrangement of the host population impacts the development of general epidemics and the spread of focal epidemics by a common mechanism. This explains the high degree of correlation between r and c across different spatial configurations of host plants and for different dispersal kernels. These findings provide evidence against the idea that results from studies of host-diversity effects on potato late blight in temperate regions cannot be extrapolated to more tropical regions (12). It was suggested that the likely difference between tropical and temperate systems is the level of outside inoculum arriving at newly planted fields; in tropical regions, there is often a continual presence of potato late blight (15,29), whereas in Europe and North America, blight epidemics must begin again with each growing season. However, this study suggests that the empirical results cannot be attributed to fundamentally different responses to changes in spatial configuration of host plants between focal and general epidemics.

The predictive measures of apparent infection rate, r_1 and r_2 , showed that information relating to emergent growth in time and space can be inferred from life table characteristics. The theory of age-structured populations (19) has been used to approximate temporal growth without any explicit reference to spatial constraints (equation 3). The inclusion of q in equation 16 provides a link between the spaceless world of temporal growth and the spatial variation that shapes the growth of the pathogen population in its habitat. The proposed approximation to r_1 was therefore able to produce accurate predictions of r , on average within 9.8% of r . The high degree of correlation between r and c suggests that r_1 would also be capable of predicting epidemic spread in focal epidemics.

The measures of epidemic progress developed in this study could be of benefit in the design of new experiments. Using the genotype connectivity parameter, q , it is possible to quickly gauge the efficacy of landscape designs for disease control without the need for any dynamic epidemic simulation. In addition, q could be used to predict trends in r and c for new pathogens or pathogen races where little is known about epidemic development in the field. If sufficient ecological information regarding life table processes is available, then q can be used as a spatial component as part of other indices, such as r_1 , to produce direct predictions of the effects of host diversity on epidemic development. Similarly, such predictive measures could be used to predict epidemic development in new cultivars via incorporation of the infection efficiency of the pathogen, for a particular cultivar, into the calculations of q . In this way, intermediate levels of host resistance could be incorporated into q values.

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