Effects of Initial Epidemic Conditions, Sporulation Rate, and Spore Dispersal Gradient on the Spatio-Temporal Dynamics of Plant Disease Epidemics

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ABSTRACT

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A stochastic model that simulates the spread of disease over space and time was developed to study the effects of initial epidemic conditions (number of initial inocula and their spatial pattern), sporulation rate, and spore dispersal gradient on the spatio-temporal dynamics of plant disease epidemics. The spatial spread of disease was simulated using a half-Cauchy distribution with median dispersal distance μ (units of distance). The rate of temporal increase in disease incidence (β_t , per day) was influenced jointly by μ and by the sporulation rate λ (spores per lesion per day). The relationship between β_t and μ was nonlinear: the increase in β_t with increasing μ was greatest when μ was small (i.e., when the dispersal gradient was steep). The rate of temporal increase in disease severity of diseased plants (β_s) was affected mainly by λ : β_s increased directly with increasing λ . Intraclass correlation (κ_t), the correlation of disease status of plants within quadrats, increased initially with disease incidence, reached a peak, and

then declined as disease incidence approached 1.0. This relationship was well described by a power-law model that is consistent with the binary form of the variance power law. The amplitude of the model relating κ_t to disease incidence was affected mainly by μ : κ_t decreased with increasing μ . The shape of the curve was affected mainly by initial conditions, especially the spatial pattern of the initial inocula. Generally, the relationship of spatial autocorrelation ($\rho_{t,k}$), the correlation of disease status of plants at various distances apart, to disease incidence and distance was well described by a four-parameter power-law model. $\rho_{t,k}$ increased with disease incidence to a maximum and then declined at higher values of disease incidence, in agreement with a power-law relationship. The amplitude of $\rho_{t,k}$ was determined mainly by initial conditions and by μ : $\rho_{t,k}$ decreased with increasing μ and was lower for regular patterns of initial inocula. The shape of the $\rho_{t,k}$ curve was affected mainly by initial conditions, especially the spatial pattern of the initial inocula. At any level of disease incidence, autocorrelation declined exponentially with spatial lag; the degree of this decline was determined mainly by μ : it was steeper with decreasing μ .

The characterization of disease incidence and severity over time is of fundamental importance in epidemiology for describing and understanding the dynamics of diseases as well as for evaluating treatments. Typically, a measure of the extent of disease such as the number of lesions, the amount of diseased tissue, or the number of diseased plants is plotted against time to give a disease progress curve. The mathematical properties of disease progress curves and aspects of their statistical analysis have been reviewed extensively (2,21,23,41).

Several important parameters defining the temporal dynamics of an epidemic such as the rate of increase of the disease and the asymptotic disease level can be estimated from the disease progress curve. These parameters have been used to compare outbreaks of the same disease at different times, at different locations, or under different management practices (8,30,35). They have also been used to characterize individual epidemics with the aim of identifying similarities and differences between epidemics and, thus, identifying the key elements that determine the course of epidemics (19,20). This is often termed comparative epidemiology. The most important parameter (2) is the rate of increase of the disease, describing the temporal progress of the epidemic. The rate has been often interpreted in relation to biological processes such as sporulation and infection and the host and environmental factors that influence these processes, even though it is recognized that spatial aspects of an epidemic may affect the rate parameter (2). These spatial effects can compli-

cate the interpretation of a temporal analysis, particularly when one wishes to compare epidemics that differ in their spatial aspects.

Recently, there has been increased interest in the statistical description and theoretical modeling of the spatio-temporal dynamics of epidemics (17,23,45). Several statistical methods exist for describing and analyzing spatial patterns of epidemics. The beta-binomial distribution has been shown to be more appropriate than the negative-binomial distribution for examining spatial patterns of disease incidence for the presence of aggregation (14,23). The beta-binomial distribution has two parameters: one is the probability of disease (estimated by disease incidence) and the other represents variability in disease incidence among quadrats; both are usually estimated by the maximum-likelihood method, which requires iterative calculations. An alternative measure of variability that is simpler to calculate is the intraclass correlation (7). This measures the correlation between the binary responses (diseased or not diseased) of individual plants within a quadrat. The dispersion or aggregation parameter of the beta-binomial distribution is directly related mathematically to the intraclass correlation. In fact, the beta-binomial distribution can be written in terms of the intraclass correlation. An alternative approach is spatial autocorrelation, which determines the spatial dependence of disease incidence over various spatial lags (2,11,23). Geostatistical methods have also been used to analyze spatial disease patterns, and the results are often presented as a semivariogram representing the average squared difference between pairs of samples separated by a given distance to detect the spatial dependence (3,12,34). This semivariogram is closely related to the spatial autocorrelation (1). Recently, spatio-temporal stochastic models have been fitted to experimental data using stochastic integration methods (10); this approach has been devel-

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oped further using Markov chain Monte Carlo methods (9). These spatio-temporal statistics are very useful in describing and summarizing observed epidemics. However, their usefulness in inferring the underlying biological and physical processes that lead to the observed epidemic is not clear, because the relationships of these statistics to the underlying processes have not been studied adequately.

The focus of this paper is to study the relationships of spatio-temporal statistics to the underlying processes governing epidemics using a stochastic simulation model. Although the simulation model is not intended as an exact model of any specific epidemic, it is intended to reflect, in a simplified way, the main processes involved in the development of an epidemic. Several spatio-temporal statistics were derived from the epidemic data generated by the simulation model, and their relationships with parameters of the underlying processes were investigated.

DESCRIPTION OF THE SIMULATION MODEL

The model is a two-dimensional stochastic spatial-contact model (28) in which the distance that spores are dispersed follows a long-tailed probability distribution. There are several similarities with the one-dimensional model proposed by Minogue and Fry (27).

The spread of disease is modeled on a rectangular grid of plants consisting of n_r rows with n_c plants in each row. The distance between adjacent plants within a row is d_c , while the distance between rows is d_r . Each plant is assumed to consist of n_s units that are susceptible to the disease. The epidemic is initiated by a single infected unit on one or more plants. These initially infected plants may be located systematically or randomly within the grid.

When a unit becomes infected, it enters a latent period of length, t_L time steps. It then becomes infectious for t_I time steps, producing spores that can initiate new infections, after which it no longer produces spores. During the infectious period, the infectious unit is assumed to produce viable spores according to a Poisson process with constant rate parameter λ , called the sporulation rate. Although the spread of disease is a continuous process in time, it is more convenient computationally to work with a discrete-time model. At each time step, here taken as 1 day, the number of spores produced by an infectious unit is, therefore, a Poisson random variable, with parameter λ , the mean number of spores produced per lesion per day.

To simulate the spread of disease, spores are assumed to be dispersed independently of one another and to travel in straight lines of random orientation, determined by a uniform random number on the interval $(0^\circ, 360^\circ)$. The dispersal distance varies randomly according to a half-Cauchy distribution (31) with median dispersal distance parameter μ . With this distribution, the dispersal distance (d) of a particular spore is simulated by taking $d = \mu \tan(\pi x/2)$, in which x is a uniform random number on the interval $(0,1)$. If the spore falls beyond the end of a row by a distance of more than $d_c/2$ or beyond the extreme rows by a distance of more than $d_r/2$, it is ignored. Otherwise it lands on a randomly selected unit on the nearest plant, again determined by a uniform random number. This unit becomes infected if it has not been infected previously; otherwise the spore has no effect. The plant that is nearest to the spore when it lands may be the plant from which it originated, and the model, therefore, allows for the spread of disease within plants as well as between plants. Random numbers were generated using the uniform random number generator of Wichmann and Hill (43).

DESCRIPTION OF SIMULATION RUNS

The effects of the initial number of infected plants (Y_0) and their spatial configuration (Y_S), the sporulation rate (λ), and the dispersal parameter (μ) on the spatio-temporal dynamics of epidemics were studied. Several summary statistics (temporal rates, intraclass correlation, and spatial autocorrelation) were calculated to describe the spatio-temporal dynamics of each simulated epidemic. In all the simulations, there were $n_r = 150$ rows and $n_c = 150$ plants

within a row, with $d_r = d_c = 1$; each plant had $n_s = 30$ susceptible units; and the latent and infectious periods lasted for $t_L = 5$ days and $t_I = 10$ days, respectively.

The initial number of infected plants (Y_0) was one, four, or nine, and each had a single infected unit on the point of entering its infectious period. The initial infected plants were located with one of three spatial patterns (Y_S): clumped, regular, or random. With $Y_0 = 1$, the single infected plant was at the (approximate) center of the grid for the regular pattern. With $Y_0 = 4$ or 9, the infected plants were placed in the (approximate) central 2×2 or 3×3 grid for the clumped pattern. For the regular pattern, the central 120×120 grid was partitioned into four or nine equal-sized square blocks, and one infected plant was placed at the (approximate) center of each block. For the random pattern, locations for the infected plants were chosen within the central 120×120 grid of plants using a uniform random number. Thus, in total, there were eight combinations of Y_0 and Y_S . Five values of λ were used: 0.3 to 0.7 day⁻¹ with an increment of 0.1 day⁻¹, covering the range of most experimental rate estimates (18). Ten values of μ were used: 0.25, 0.50, 0.75, 1.0, 1.5, 2.0, 3.0, 4.0, 5.0, and 6.0, in the same units as d_r . Thus, there were, in total, 400 ($= 8 \times 5 \times 10$) combinations of the four simulation variables (Y_0 , Y_S , λ , and μ); for each combination, the model was simulated 50 times, with each run terminating after 60 days. Data were sampled at an interval of 3 days for subsequent statistical analysis; thus, there were 20 temporal sampling points for each simulation, 21 if the initial point was included.

Additional simulations were run for eight combinations of μ and λ , for each set of initial conditions ($Y_0 \times Y_S$). These were four levels of μ (0.25, 0.75, 2.0, and 6.0) and two levels of λ (0.3 and 0.7 day⁻¹), giving a total of 64 combinations, each simulated 50 times. These simulations were terminated at various times, ranging from 54 to 210 days, to generate epidemics with a final disease incidence level exceeding 95%. These simulations were used to model intraclass correlation and spatial autocorrelation over a wider range of disease incidence than was possible with the main set of simulations.

DATA ANALYSIS

Although the epidemic was simulated on a 150×150 grid, all statistical analyses were based on data from the central 120×120 grid, to reduce edge effects. Plants with at least one unit infected were classified as diseased. The effects of simulation variables (Y_0 , Y_S , λ , and μ) on plant disease incidence, p_t , and disease severity, S_t , were studied. p_t was defined as the overall proportion of plants that had become infected by time t . In the literature, S_t is often defined as the percentage of tissue area diseased per plant. However, in this study, S_t was defined as the mean number of infected units per infected plant, because S_t defined in this way was less highly correlated with p_t than 'conventional' S_t . The definition of S_t used in this study could be termed 'conditional disease severity,' since it measures the severity of disease, conditional on the plants being infected. The definitions of both disease incidence and severity include all plants and units that had become infected by time t , irrespective of whether they were infectious at time t .

The increase in p_t over time was generally well described by the logistic model

$$\log\left(\frac{p_t}{1-p_t}\right) = \alpha_I + \beta_I t, \quad (1)$$

in which t is time in days. To estimate the parameters α_I and β_I , we calculated the empirical logistic transformation of p_t (4), namely $\log[(y_t + 0.5)/(n_t - y_t + 0.5)]$, in which y_t and n_t are the number of infected plants and the total number of plants ($= 120 \times 120$), respectively, at time t , and regressed this on t (4). The focus of our attention is the parameter β_I , which characterizes the temporal rate of increase of the epidemic, but the percentage of variance accounted for by the regression was also recorded.

A: $\mu = 0.25, \lambda = 0.70, Y_0 = 1$

B: $\mu = 1.0, \lambda = 0.70, Y_0 = 1$

C: $\mu = 6.0, \lambda = 0.70, Y_0 = 1$

Fig. 1. Three examples of simulated epidemics on the central 120×120 grid after 45 days. There was one initial infected plant at the center of the grid. μ = median dispersal distance. λ = sporulation rate.

The increase in S_t was also well described by the logistic model

$$\log \left(\frac{S_t + 0.5}{n_s - S_t + 0.5} \right) = \alpha_s + \beta_s t, \quad (2)$$

in which n_s is the number of susceptible units per plant, which is 30 in this study.

Intraclass correlation at time t (κ_t) measures the tendency of the plants within a sampling unit (quadrat) to have a similar disease status. Positive values of κ_t indicate aggregation of disease. κ_t was calculated as (7)

$$\kappa_t = 1 - \frac{1}{p_t(1-p_t)NT(T-1)} \sum_{i=1}^N Y_i(T-Y_i), \quad (3)$$

in which Y_i is the number of infected plants in the i th quadrat, N is the total number of quadrats, and T is the number of plants in a quadrat. We calculated intraclass correlation using a contiguous array of 5×5 quadrats on the central 120×120 grid of plants. Thus, $N = 576$ and $T = 25$.

Spatial autocorrelation at time t and spatial lag k ($\rho_{t,k}$) was calculated for disease incidence of individual plants up to a lag of 25, in the same units as d_t . Spatial autocorrelation was only calculated along the rows, since spatial autocorrelation along the columns was expected to be the same because, in the simulation model, the direction of spore dispersal is chosen at random. Algorithms for calculating both intraclass correlation and spatial autocorrelation were implemented directly in the simulation program.

The binary form of Taylor's (36) power law (23) was used as the basis for relating κ_t to p_t :

$$\kappa_t = A p_t^{\beta_1} (1 - p_t)^{\beta_2}, \quad (4)$$

in which A , β_1 , and β_2 are parameters to be estimated. This is a close approximation to equation 1 in Hughes et al. (15), if the more general equation 6b in Madden and Hughes (23) for the binary power law is used. Parameter A affects only the amplitude of the curve, and β_1 (power of disease incidence) and β_2 (power of proportion of healthy plants) determine the shape of the curve. In particular, when $\beta_1 > 0$ and $\beta_2 > 0$, the curve has a maximum at $p_t = \beta_1 / (\beta_1 + \beta_2)$. For parameter estimation, a linearized form of equation 4 was used, i.e.,

$$\ln(\kappa_t) = \alpha + \beta_1 \ln(p_t) + \beta_2 \ln(1 - p_t), \quad (5)$$

in which $\alpha = \ln(A)$.

The following model was used to relate spatial autocorrelation to p_t and spatial lag, k :

$$\rho_{t,k} = A p_t^{\beta_1} (1 - p_t)^{\beta_2} \exp[\beta_3 k (1 - p_t)], \quad (6)$$

in which A , β_1 , β_2 , and β_3 are parameters to be estimated. Parameter A affects the amplitude of $\rho_{t,k}$ at any spatial lag, β_1 and β_2 determine the shape of the curve in relation to p_t , and β_3 (degree of exponential decline over spatial lags) determines the rate of decline of $\rho_{t,k}$ over spatial lags at any particular level of p_t . For parameter estimation, a linearized form of equation 6 was used, i.e.,

$$\ln(\rho_{t,k} + c) = \alpha + \beta_1 \ln(p_t) + \beta_2 \ln(1 - p_t) + \beta_3 k (1 - p_t), \quad (7)$$

in which $\alpha = \ln(A)$ and c is a constant ($= 0.125$) added to $\rho_{t,k}$ to avoid zero or negative values. The maximum value of $(\rho_{t,k} + c)$ was still less than 1.0.

Equations 5 and 7 were fitted by least squares to data from individual simulations. The overall effects of Y_0 , Y_s , λ , and μ on β_t ,

β_s , and the seven parameters in equations 5 and 7 were assessed by analysis of variance (ANOVA). The data are unbalanced, partly because one factor combination ($Y_0 = 1$, $Y_s = \text{clumped}$) does not arise, and partly because a few points were excluded from the analysis (described below). Because of this, the results of ANOVA are dependent on the order in which factors are included in the analysis. Analysis showed, however, that the effects of including factors in different orders were very small (data not shown). To assess how the contribution of λ and μ to the variation in β_l , β_s , and the seven parameters of equations 5 and 7 varied with the initial epidemic conditions, separate ANOVAs were performed for each of the eight initial conditions. All the statistical analyses were done using Genstat (Numerical Algorithms Group Ltd., Oxford, England).

RESULTS

General results. Figure 1 shows three examples of simulated patterns of disease incidence and illustrates that distinct clusters of infected plants may form some distance from the original source of infection. This is a consequence of using a long-tailed distribution for dispersal distance (26,31). It can also be seen clearly that disease incidence increased as the dispersal parameter μ increased. There was considerable variation between runs with the same set of parameter values. The standard deviation of disease incidence between simulation runs increased approximately in proportion to the mean incidence.

The epidemic died out before any new plants were infected in 84 of the 20,000 simulation runs. These simulation runs were ex-

Fig. 2. Relationship of final level of disease incidence (expressed here as a percentage rather than a proportion) at 60 days to the sporulation rate (λ) and the median dispersal distance (μ) for each of the eight initial conditions. The symbols ●, ○, ▼, ▽, and ■ represent $\lambda = 0.3, 0.4, 0.5, 0.6$, and 0.7 , respectively. Y_0 = initial number of infected plants.

cluded from the subsequent analysis. A further six simulation runs were excluded because the epidemic died out after only a few new infections when, as a result of stochastic variation, no further infections were produced. The logistic models were thus fitted to 19,910 simulation runs. As Y_0 increased, the likelihood of the epidemic dying out was reduced. All of the 90 simulation runs omitted had only a single initial infection, with equal numbers arising from the regular and random patterns. The number of epidemics that died out decreased with increasing λ ; there were 56, 22, 7, 2, and 3 simulation runs omitted with $\lambda = 0.3, 0.4, 0.5, 0.6$, and 0.7 , respectively. There was no dependence on μ .

Rate of increase in disease incidence (β_I). Figure 2 shows the effect of Y_0 , Y_S , λ , and μ on the incidence of disease at 60 days. The regular pattern of initial infections resulted in the highest incidence, and the clumped pattern resulted in the lowest incidence. As

expected, increases in either Y_0 or λ resulted in higher disease incidence. The greater the value of μ , the higher the final disease incidence, but the relationship was nonlinear. The increase in the final disease incidence in relation to μ was greatest for small values of μ .

The increase in disease incidence over time was well described by the logistic model (equation 1). The percentage of variance accounted for exceeded 90% in all but 6 out of 19,910 simulations analyzed and was never less than 70%. Figure 3 shows the mean estimate of β_I for each of the 400 combinations of Y_0 , Y_S , λ , and μ . As would be expected, β_I increased as λ increased. There was also a sharp increase in β_I as μ increased when μ was small, but, as μ became larger, further increases had little effect on β_I . ANOVA (Table 1) showed that λ and μ jointly accounted for about 85% of the total variation in β_I , of which only 6% was due to their interaction.

Fig. 3. Relationship of the logistic rate of temporal increase in disease incidence (β_I) to the sporulation rate (λ) and the median dispersal distance (μ) for each of the eight initial conditions. The symbols $\bullet$, $\circ$, $\blacktriangle$, ∇ , and $\blacksquare$ represent $\lambda = 0.3, 0.4, 0.5, 0.6$, and 0.7 , respectively. Y_0 = initial number of infected plants.

About 2.5% of the variation in β_I was due to Y_0 . The mean values of β_I with one, four, and nine plants infected initially were 0.130, 0.138, and 0.148 per day, respectively. The effect of Y_S (pattern) on β_I accounted for 2.8% of the variation. The average values of

β_I were 0.134, 0.147, and 0.137 per day for the clumped, regular, and random patterns, respectively. With $Y_0 = 1$, β_I was 0.132 and 0.128 per day for the regular and random patterns, respectively. This difference is because the probability that subsequent spores

TABLE 1. Percentage (%) of variance accounted for by the four simulation variables^a in the analysis of variance of model parameters^b describing temporal disease increase, intraclass correlation, and spatial autocorrelation

Term	df	Temporal disease increase		Intraclass correlation			Spatial autocorrelation			
		β_I	β_S	α	β_1	β_2	α	β_1	β_2	β_3
Y_0	2	2.5	8.4	5.3	4.3	1.3	11.6	9.3	4.7	8.8
Y_S	2	2.8	0.4	5.6	44.4	2.7	37.6	44.0	24.6	4.3
μ	9	30.0	0.3	54.8	4.6	7.4	28.5	0.2	19.7	57.6
λ	4	49.3	62.3	0.0	0.1	0.2	0.2	0.2	0.5	2.8
$Y_0 \times Y_S$	3	0.5	0.2	0.5	9.9	1.1	3.8	5.7	1.8	1.0
$Y_0 \times \mu$	18	1.3	2.8	3.0	2.1	2.7	0.2	0.3	0.7	1.1
$Y_0 \times \lambda$	8	2.0	4.1	0.0	0.0	0.0	0.6	1.5	1.1	0.1
$Y_S \times \mu$	18	0.3	0.2	4.1	0.9	2.2	0.4	1.4	1.4	2.5
$Y_S \times \lambda$	8	1.1	1.5	<0.1	<0.1	0.5	1.4	4.2	1.1	0.8
$\mu \times \lambda$	36	6.0	13.3	3.8	1.3	3.1	0.3	0.6	0.3	1.0

^a The four simulation variables are the number of initial infections (Y_0), spatial pattern of the initial infections (Y_S), the spore dispersal parameter (μ), and the sporulation rate (λ).

^b α = the amplitude of spatial autocorrelation (or intraclass correlation) in relation to disease incidence. β_1 = power of disease incidence. β_2 = power of proportion of healthy plants. β_3 = degree of exponential decline over spatial lags. β_I = rate of temporal increase in disease incidence. β_S = rate of temporal increase in disease severity of diseased plants. Equations 1, 2, 5, and 7 further define the model parameters.

TABLE 2. Percentage (%) of variance accounted for by the sporulation rate (λ) and the spore dispersal parameter (μ) in a separate analysis of variance of model parameters describing temporal disease increase, intraclass correlation, and spatial autocorrelation for each set of initial conditions^a

		Initial conditions							
		1		4			9		
Model parameters ^b	Variables	Regular	Random	Clumped	Regular	Random	Clumped	Regular	Random
Temporal disease increase									
β_I	μ	33.7	32.4	40.8	31.2	33.3	41.6	28.7	32.1
	λ	57.1	57.4	50.9	57.4	57.6	48.1	60.9	55.6
	$\mu \times \lambda$	2.9	2.2	6.4	8.7	5.4	9.3	8.9	9.3
	Total SS ^c	2.15	1.78	3.33	5.18	2.95	4.99	9.20	5.50
β_S	μ	17.9	17.2	0.2	0.5	1.5	4.5	2.3	1.6
	λ	55.7	56.8	70.8	77.6	79.9	65.9	79.9	82.2
	$\mu \times \lambda$	9.3	7.9	22.7	17.6	13.4	26.8	16.0	14.3
	Total SS	0.16	0.18	0.26	0.38	0.36	0.38	0.81	0.68
Intraclass correlation									
α	μ	51.7	27.0	70.0	81.6	63.2	73.3	85.9	72.6
	λ	0.8	0.3	0.4	0.0	0.1	0.5	0.5	0.0
	$\mu \times \lambda$	8.4	5.5	5.7	2.7	6.5	8.9	1.9	4.2
	Total SS	46.8	47.9	41.7	95.0	73.4	27.5	140.0	100
β_1	μ	28.3	34.1	14.5	4.4	17.8	22.6	3.3	8.9
	λ	0.0	0.2	0.7	0.1	0.4	0.9	1.4	0.2
	$\mu \times \lambda$	2.7	0.8	1.9	5.1	5.0	6.1	3.6	8.8
	Total SS	2.8	2.7	1.8	1.8	2.5	0.9	2.0	2.6
β_2	μ	2.8	6.1	12.0	31.8	24.5	3.8	39.3	41.3
	λ	0.3	0.7	0.1	0.0	2.1	0.2	0.5	2.8
	$\mu \times \lambda$	3.3	4.0	7.5	9.2	8.2	10.0	5.2	6.0
	Total SS	62.7	45.6	5.0	9.2	16.8	2.4	9.3	17.9
Spatial autocorrelation									
α	μ	69.9	47.4	82.2	65.2	58.1	87.0	44.3	55.7
	λ	3.9	10.1	1.4	3.1	5.4	0.7	10.8	5.8
	$\mu \times \lambda$	6.2	5.2	1.1	1.0	1.3	1.7	4.6	0.5
	Total SS	28.8	40.9	31.7	48.1	48.9	35.6	39.2	43.4
β_1	μ	2.3	3.0	10.5	2.3	7.4	26.7	5.1	2.8
	λ	5.2	16.9	0.0	22.2	9.4	15.9	30.9	6.1
	$\mu \times \lambda$	16.0	8.3	1.3	2.0	2.3	8.0	8.0	1.9
	Total SS	0.28	0.70	0.28	1.16	1.36	0.60	2.22	1.63
β_2	μ	22.9	18.7	60.3	58.3	34.9	71.2	22.4	16.6
	λ	1.7	6.6	0.8	0.5	5.8	3.2	1.9	4.4
	$\mu \times \lambda$	7.4	2.3	5.0	1.1	0.4	2.4	8.0	0.4
	Total SS	11.1	22.8	5.1	5.1	6.75	6.6	3.99	5.50
β_3	μ	72.5	69.3	75.8	79.4	54.9	82.3	78.6	60.0
	λ	2.7	2.5	1.7	5.3	3.8	0.0	6.9	5.1
	$\mu \times \lambda$	1.4	0.4	0.6	0.0	0.0	0.0	0.0	0.9
	Total SS	0.05	0.05	0.05	0.16	0.10	0.06	0.18	0.12

^a Initial conditions are specified by the number of initial infections ($Y_0 = 1, 4, \text{ or } 9$) and their spatial pattern ($Y_S = \text{regular, random, or clumped}$).

^b α = the amplitude of spatial autocorrelation (or intraclass correlation) in relation to disease incidence. β_1 = power of disease incidence. β_2 = power of proportion of healthy plants. β_3 = degree of exponential decline over spatial lags. β_I = rate of temporal increase in disease incidence. β_S = rate of temporal increase in disease severity of diseased plants. Equations 1, 2, 5, and 7 further define the model parameters.

^c Total sum of squares from analysis of variance.

will fall outside of the grid, and therefore fail to contribute to the development of the epidemic, is minimized when the infected plant is at the center of the grid. With $Y_0 = 4$ or 9 , an additional factor contributes to the higher average rate of disease increase when the infected plants are arranged regularly. A regular pattern minimizes the average distance from a healthy plant to the nearest infectious plant, and, therefore, maximizes the probability that a healthy plant will become infected; a clumped pattern minimizes this probability, whereas the random pattern gives an intermediate value.

To investigate how the contribution of λ and μ to variation in β_I varied with the initial conditions, a separate ANOVA was performed for each combination of Y_0 and Y_S (Table 2). The contribution of λ to variation in β_I ranged from 48 to 61% and that of μ ranged from 29 to 42%. The contribution of λ was least for the clumped initial pattern when $Y_0 = 4$ or 9 , and it was similar for all the other com-

binations of Y_0 and Y_S . The contribution of μ was greatest for the clumped pattern when $Y_0 = 4$ or 9 , it was similar for all the other combinations of Y_0 and Y_S . The percentage of variance due to the interaction between λ and μ increased slightly as Y_0 increased.

Rate of increase in disease severity (β_S). The percentage of variance accounted for by the logistic model (equation 2) of disease severity increase over time was less than that for disease incidence, but still averaged 84%. In addition to those 90 simulation runs excluded because the epidemic did not develop, a further 441 simulation runs were excluded from the following analysis because the percentage of variance accounted for was less than 50%. As Y_0 increased, the number of simulation runs excluded decreased: there were 359, 72, and 10 runs omitted for $Y_0 = 1, 4$, and 9 , respectively. There was no dependence on Y_S . The number of runs excluded was less for intermediate values of μ than for large or small

Fig. 4. Relationship of the logistic rate of temporal increase in disease severity (β_S) to the sporulation rate (λ) and the median dispersal distance (μ) for each of the eight initial conditions. The symbols $\bullet$, $\circ$, ∇ , ∇ , and $\blacksquare$ represent $\lambda = 0.3, 0.4, 0.5, 0.6$, and 0.7 , respectively. Y_0 = initial number of infected plants.

values. The number of epidemics excluded decreased with increasing λ ; there were 269, 108, 43, 13, and 8 runs omitted from simulations with $\lambda = 0.3, 0.4, 0.5, 0.6$, and 0.7 , respectively.

Figure 4 shows the mean estimate of β_S for each of the 400 combinations of Y_0 , Y_S , λ , and μ . ANOVA (Table 1) showed that λ accounted for 62% of the variation in β_S . As would be expected, β_S increased as λ increased. Less than 1% of the variation was due to the main effect of μ , but its interaction with λ accounted for 13% of the total variation. This interaction can be clearly seen from Figure 4; β_S decreased as μ increased for small values of λ and increased with increasing μ for higher values of λ . About 8% of the variation in β_S was due to Y_0 . The mean value of β_S was 0.019, 0.024, and 0.029 per day for $Y_0 = 1, 4$, and 9, respectively.

Table 2 shows the contribution of λ and μ to the variation in β_S within each combination of Y_0 and Y_S . Percentage of variance ac-

counted for by λ ranged from 56 to 82% and that of μ ranged from 0.2 to 18%. The contribution of λ and its interaction with μ generally increased as Y_0 increased from 1 to 4, whereas for μ , the highest percentage of contribution occurred when Y_0 was 1. As with β_I , the contribution of λ was least for the clumped pattern with $Y_0 > 1$.

Intraclass correlation (κ_i). Figure 5 shows the average value of κ_i at each temporal sampling point plotted against the average disease incidence for each of the 400 combinations of Y_0 , Y_S , λ , and μ . Theoretically, intraclass correlation is zero when there are no diseased plants (at the start of an epidemic), and then increases and finally decreases to zero again when all the plants are infected at the end of an epidemic (22); this pattern can be seen clearly in Figure 5. Because a value of $\kappa_i > 0$ indicates clumping (aggregation) at some spatial scale, it can be seen that clumping occurred for all simulations, even when initial conditions were regular or

Fig. 5. Relationship of the average intraclass correlation (κ_i) at each temporal sampling point to average disease incidence (expressed here as a percentage rather than a proportion) for each of the eight initial conditions. Y_0 = initial number of infected plants.

random. The greater number of points at the lower end of the disease incidence axis reflects the fact that the levels of final disease incidence at 60 days were low for some combinations of simulation variables (Fig. 2). For the regular and random patterns, the maximum value of κ_i decreased as Y_0 increased (Fig. 5). For the clumped pattern, the relationship between κ_i and p_i was largely independent of Y_0 (Fig. 5C and F), with the maximum value of κ_i occurring at an intermediate level of p_i . The relationship between κ_i and the p_i peak was also dependent on Y_S , with the lowest values of κ_i for the regular pattern.

In the additional 3,200 (64×50) simulation runs, the epidemic failed to develop in only 28 runs, all of which had $Y_0 = 1$ and $\lambda = 0.3$. Equation 5 was fitted successfully to the remaining simulation runs. The percentage of variance accounted for ranged from

56 to 99% with an average of 89% and was less than 70% in only 31 cases. The effects of Y_0 , Y_S , λ , and μ on the three estimated parameters (α , β_1 , and β_2) were assessed by ANOVA (Table 1). Figure 6 illustrates the effects of μ , Y_0 , Y_S , and p_i on κ_i by plotting κ_i against p_i for various combinations of μ , Y_0 , and Y_S using the average values of α , β_1 , and β_2 over λ (Table 3). Averaging over λ was justified, because λ accounted for very little of the variability (Table 1) in α , β_1 , and β_2 .

The parameter α (equation 5) affects the amplitude of κ_i . About 5% of the variation in α was accounted for by Y_0 ; the average values of α for $Y_0 = 1, 4$, and 9 were $0.079, -0.039$, and -0.186 , respectively, showing the increasing amplitude with decreasing initial disease. Y_S accounted for about 6% of the variation; the mean values of α were $-0.151, -0.159$, and 0.083 for the clumped,

Fig. 6. Estimated relationship of intraclass correlation (κ_i), given by equation 5, to disease incidence (expressed here as a percentage rather than a proportion) for μ (median dispersal distance) = 0.25, 0.75, 2.0, and 6.0 for each of the eight initial conditions. Parameter values (Table 3) were averaged over λ (sporulation rate) = 0.3 and 0.7, because the effect of λ on the model parameters was very small. The solid, dotted, dashed, and dashed-dotted lines represent $\mu = 0.25, 0.75, 2.0$, and 6.0 , respectively. Y_0 = initial number of infected plants.

regular, and random patterns, respectively, showing the lowest amplitude for a regular initial pattern and highest amplitude for a random initial pattern. These differences can be seen in differences in the maximum values of κ_t shown in Figure 6. About 55% of the total variation in α was due to μ . The mean values of α were 0.246, 0.119, -0.005, and -0.623 for μ of 0.25, 0.75, 2.0, and 6.0, respectively, implying that the maximum values of κ_t decreased as μ increased (Fig. 6). Although little variation (<1%) was attributable to the main effect of λ , its interaction with μ accounted for 4% of the total variation: the decrease in α with increasing μ was less for λ of 0.7 than for 0.3.

Jointly, Y_0 and Y_S accounted for 59% of the total variation in parameter β_1 (Table 1). The mean values of β_1 were 0.309, 0.464, and 0.461 for the clumped, regular, and random patterns, respectively. For both the random and regular patterns, β_1 increased as Y_0 increased; in contrast, for the clumped pattern, β_1 decreased as Y_0 increased (Table 3). About 5% of the variation in β_1 was due to μ . However, there was no clear pattern associated with μ (Table 3). For parameter β_2 , simulation factors only accounted for 22% of the total variation, of which 7% was due to μ . The mean values were 0.594, 0.527, 0.529, and 0.415 when μ was 0.25, 0.75, 2.0, and 6.0, respectively.

Table 2 shows the ANOVA results for each combination of Y_0 and Y_S . For parameters α and β_2 , the contribution of μ increased as Y_0 increased, but the opposite was true for β_1 . Of the three parameters, α was most affected by μ , especially with the regular pattern.

Spatial autocorrelation ($\rho_{t,k}$). Equation 7 was fitted successfully to most of the 3,200 simulation runs. The percentage of variance accounted for ranged from 4 to 99% with an average of 82%. There were 149 runs in which the percentage of variance accounted for was less than 50%, and these were excluded from the subsequent analysis. The number of epidemics with poor fit was not related to

Y_0 or Y_S , but was related to λ and μ : 56 of the 149 runs excluded occurred with $\lambda = 6.0$ and $\mu = 0.7$. Table 1 shows ANOVA results on the effects of Y_0 , Y_S , λ , and μ on the four parameters (α , β_1 , β_2 , and β_3). Figure 7 illustrates the effects of μ , Y_0 , Y_S , and p_t on $\rho_{t,k}$ by plotting $\rho_{t,k}$ against p_t and k for various combinations of μ , Y_0 , and Y_S using the average values of α , β_1 , β_2 , and β_3 over λ (Table 3).

The parameter α in equation 7 affects the amplitude of $\rho_{t,k}$ at any spatial lag in relation to p_t . Jointly, Y_0 and Y_S accounted for 53% of the total variation in α . The mean values of α were -0.090, -0.646, and -0.144 for the clumped, regular, and random patterns, respectively, reflecting the difference shown in Figure 7. For Y_0 of 1, 4, and 9, the mean values of α were -0.060, -0.309, and -0.479, respectively. The interaction between Y_S and Y_0 was due mainly to the greater reduction in α with increasing Y_0 for the regular pattern than for the other two patterns. About 29% of the variation in α was due to μ ; the mean values of α were -0.114, -0.156, -0.266, and -0.746 when μ was 0.25, 0.75, 2.0, and 6.0, respectively.

About 59% of the total variation in β_1 was due to Y_S and Y_0 (Table 1). The average values of β_1 were 0.185, 0.108, and 0.228 for the clumped, regular, and random patterns, respectively, and 0.212, 0.173, and 0.147 for Y_0 of 1, 4, and 9, respectively.

For parameter β_2 , the four simulation variables accounted for 59% of the total variation, of which 25% was due to Y_S and 20% to μ . The mean values of β_2 were 0.417, 0.226, and 0.327 for the clumped, regular, and random patterns, respectively, and 0.388, 0.358, 0.318, and 0.180 when μ was 0.25, 0.75, 2.0, and 6.0, respectively. Y_0 accounted for about 5% of the total variation in β_2 : β_2 decreased as Y_0 increased.

Among the four simulation variables, μ accounted for the most variation in β_3 (58%). The mean values of β_3 were -0.058, -0.056, -0.047, and -0.024 per unit distance when μ was 0.25, 0.75, 2.0, and 6.0, respectively. About 9% of the total variation was due to

TABLE 3. Average values of model parameters describing intraclass correlation and spatial autocorrelation for each combination of the three simulation variables^a

		Initial conditions							
		1		4			9		
Model parameters ^b	μ	Regular	Random	Clumped	Regular	Random	Clumped	Regular	Random
Intraclass correlation									
α	0.25	0.178	0.288	0.257	0.216	0.386	−0.015	0.217	0.438
	0.75	0.110	0.260	0.104	0.103	0.272	−0.162	0.047	0.225
	2.0	0.077	0.298	0.009	−0.055	0.131	−0.290	−0.218	0.042
	6.0	−0.457	−0.134	−0.467	−0.904	−0.496	−0.622	−1.199	−0.696
β ₁	0.25	0.340	0.327	0.365	0.460	0.440	0.255	0.560	0.528
	0.75	0.335	0.337	0.340	0.458	0.434	0.231	0.533	0.497
	2.0	0.383	0.402	0.358	0.488	0.475	0.232	0.540	0.535
	6.0	0.449	0.475	0.409	0.487	0.517	0.286	0.524	0.565
β ₂	0.25	0.505	0.592	0.573	0.577	0.686	0.460	0.623	0.732
	0.75	0.498	0.604	0.505	0.519	0.583	0.420	0.523	0.562
	2.0	0.570	0.776	0.516	0.478	0.508	0.434	0.483	0.482
	6.0	0.380	0.558	0.465	0.345	0.409	0.448	0.337	0.538
Spatial autocorrelation									
α	0.25	−0.005	0.259	0.131	−0.442	0.008	0.239	−0.847	−0.185
	0.75	−0.077	0.257	0.032	−0.531	0.052	0.064	−0.834	−0.201
	2.0	−0.148	0.178	−0.108	−0.600	−0.055	−0.080	−1.041	−0.285
	6.0	−0.606	−0.291	−0.588	−1.185	−0.625	−0.518	−1.365	−0.808
β ₁	0.25	0.183	0.234	0.201	0.096	0.218	0.213	0.042	0.205
	0.75	0.179	0.242	0.190	0.086	0.241	0.186	0.042	0.206
	2.0	0.190	0.254	0.184	0.109	0.253	0.168	0.036	0.230
	6.0	0.181	0.236	0.176	0.090	0.215	0.161	0.080	0.202
β ₂	0.25	0.419	0.493	0.504	0.303	0.362	0.576	0.189	0.279
	0.75	0.375	0.487	0.436	0.244	0.391	0.463	0.176	0.293
	2.0	0.374	0.450	0.383	0.202	0.346	0.417	0.111	0.258
	6.0	0.202	0.232	0.248	0.061	0.188	0.273	0.070	0.166
β ₃	0.25	−0.049	−0.046	−0.054	−0.071	−0.054	−0.058	−0.070	−0.058
	0.75	−0.045	−0.044	−0.051	−0.069	−0.053	−0.054	−0.070	−0.059
	2.0	−0.039	−0.037	−0.044	−0.056	−0.045	−0.049	−0.055	−0.049
	6.0	−0.022	−0.020	−0.026	−0.025	−0.024	−0.030	−0.023	−0.025

^a The three simulation variables are spore dispersal parameter (μ), and the number of initial infections ($Y_0 = 1, 4$, or 9) and their spatial pattern ($Y_S =$ regular, random, or clumped).

^b α = the amplitude of spatial autocorrelation (or intraclass correlation) in relation to disease incidence. β_1 = power of disease incidence. β_2 = power of proportion of healthy plants. β_3 = degree of exponential decline over spatial lags. Equations 5 and 7 further define the model parameters.

Y_0 , and the mean values of β_3 with Y_0 of 1, 4, and 9 were -0.037 , -0.048 , and -0.050 per unit distance, respectively. About 4% of the variation was due to Y_5 ; the regular pattern had a lowest value of β_3 (-0.050) and the random pattern had the highest (-0.043).

Table 2 shows parts of the ANOVA results for each combination of Y_0 and Y_5 . The contribution of μ to the variation in α varied considerably with Y_0 and Y_5 , ranging from 44 to 87%. The highest percentage of variation accounted for by μ was with the clumped pattern, and this increased as Y_0 increased. For β_1 , little variation was explained by μ or λ . For β_2 , the percentage of variation attributable to μ varied greatly with Y_0 and Y_5 , ranging from 17 to 71%. With $Y_0 > 1$, a higher percentage of variation was accounted for by μ with the clumped pattern than with the other two patterns. For

β_3 , the percentage of contribution of μ did not vary greatly with Y_0 , but it was much less with the random pattern than with the other two patterns when $Y_0 > 1$.

DISCUSSION

The simulation model shows clearly that the rate parameters of nonlinear models describing disease progress, especially disease incidence, are determined jointly by temporal processes such as sporulation and by the spatial dispersal of the pathogen propagules. Temporal curves are usually derived on the assumption that all the susceptible tissues/plants are equally likely to be infected. In practice, however, the further away the susceptible plants are

Fig. 7. Estimated relationship of spatial autocorrelation ($\rho_{i,k}$), given by equation 7, to disease incidence (expressed here as a percentage rather than a proportion) and spatial lag for μ (median dispersal distance) = 0.25 and 2.0 and for three spatial patterns. The number of initial infections (Y_0) is 9. Parameter values (Table 3) were averaged over λ (sporulation rate) = 0.3 and 0.7, because the effect of λ on the model parameters was very small.

from the infectious plants, the less likely they are to be infected. The extent to which the assumption of homogeneous mixing between infectious and healthy plants affects the rate parameter depends on the spore dispersal gradient. Gubbins and Gilligan (13) demonstrated that a model that allows heterogeneous mixing between a pathogen and its biocontrol agent fitted the observed data better than a model that assumes homogeneous mixing. A steeper spore dispersal gradient leads to a more aggregated pattern of disease (as defined by κ_i and $p_{i,k}$) and to a lower rate of increase in disease incidence (β_i). The effect of spore dispersal gradient is, however, nonlinear, i.e., the effect is more pronounced for small μ . Assuming a constant degree of aggregation over time, analytical modeling has also shown that the effect of aggregation on the rate of increase is less with low aggregation (44). For airborne or rain-splashed pathogens, wind speed, turbulence, and properties of rainfall events, together or separately, affect the spore dispersal gradient. These factors may, therefore, have important effects on the development of an epidemic when μ is small.

Initial epidemic conditions (number of initial infected plants and their spatial pattern) had considerable effects on β_i and β_s . Increasing the number of initial infected plants led to increasing β_i and β_s . A priori, one might expect the initial number of infected plants not to have an effect, since an epidemic that begins with a single infected plant will soon have four or nine infected plants and should then increase at the same rate as an epidemic that begins with four or nine infected plants. The effects of the number of initial inocula may be due to the fact that all initial infections were synchronized. In the simulations that were started with four or five infected plants, the infected unit on each plant was set to be at the start of its infectious period. This synchronization, which will not occur generally in developing epidemics, resulted in the increased values of β_i . However, this situation can arise in artificial-inoculation experiments. In an experiment with Southern blight of processing carrot, the rate of disease increase generally increased as the number of initial disease foci increased (33). A more aggregated pattern of initial infections led to lower rate of disease increase, especially disease incidence. Analytical modeling has shown that aggregation will slow the rate of disease development (25,42,44). In experimental studies with zucchini yellow mosaic potyvirus, an aggregated pattern of initial disease resulted in lower disease incidence than a uniform pattern (29).

The relatively high contribution of both λ and μ to variation in β_i was generally consistent over the different initial conditions. In contrast, the contribution of λ to β_s varied considerably with initial conditions: it was greater with more than one initial infection that were not clumped together. β_s was influenced mainly by sporulation rate (λ). Interestingly, about 20% of the variation in the rate of increase of the conventional measure of disease severity (based on all plants, not just on the infected plants) was due to the initial conditions (data not shown), but only 9% for β_s . Thus, β_s is a useful statistic to supplement the information obtained from β_i for understanding temporal dynamics of disease, though more detailed data are needed to estimate β_s .

Intraclass correlation (κ_i) increased initially with disease incidence, reached a maximum, and finally declined as disease incidence approached 1.0. This relationship of aggregation with disease incidence has been observed for grape downy mildew (24) and is consistent with the binary form of Taylor's (36) power law (23) for variances. The power-law model for κ_i (equation 4) satisfactorily summarized the relationship between intraclass correlation and disease incidence. The precise form of this relationship between κ_i and p_i depended primarily on μ as well as on initial epidemic conditions. Importantly, λ had little direct effect on any of the three model parameters. The λ parameter affected intraclass correlation only indirectly through its effect on disease incidence. The amplitude parameter α was influenced mainly by μ : in particular, the flatter the dispersal gradient the lower the maximum value of intraclass correlation. The influence of μ on α also depended on initial epidemic conditions; the percentage of variance

accounted for by μ ranged from 27 to 86% for the eight initial conditions. Thus, for comparative experiments made with controlled initial conditions, the choice of initial conditions will affect the extent to which differences in α will reflect differences in μ , rather than random variation. The parameter β_1 (power of disease incidence) was determined mainly by initial conditions, especially the spatial pattern of initial infections, whereas only a small proportion of variation in the parameter β_2 (power of proportion of healthy plants) was due to any of the four simulation variables. Most of the variation in β_2 was simply stochastic variation. These observations may explain why this binary power law successfully fitted such a wide range of parameter combinations. In the model, the effect of λ was essentially incorporated into disease incidence, and the effect of μ was absorbed into both disease incidence and the parameter α , the effect of initial epidemic conditions (Y_0 and Y_S) in β_1 and other (random) variations in β_2 .

A four-parameter model generally well described the relationship of spatial autocorrelation ($p_{i,k}$) to disease incidence and spatial lag. In this model, the relationship between spatial autocorrelation and disease incidence at any spatial lag was of a binary power-law form: at any level of disease incidence, spatial autocorrelation declined exponentially over distance. As with the intraclass correlation, λ had little direct effect on any of the four model parameters. The parameter α (amplitude of spatial autocorrelation in relation to disease incidence) was influenced by both initial conditions and μ . The flatter the dispersal gradient, the lower the amplitude. Under some initial conditions, the contribution of μ to the variation in α could be as high as 87%. Interestingly, as with κ_i , parameter β_1 was determined mainly by initial conditions, especially the spatial pattern of initial infections. The parameter β_2 was influenced by both initial conditions and μ , but to a lesser extent than parameter α . The parameter β_3 (degree of exponential decline over spatial lags) was determined mainly by μ , especially with the clumped and regular patterns of initial infections. The decline over spatial lag was steeper with decreasing μ .

The relationships of intraclass correlation and spatial autocorrelation with disease incidence indicate the importance of frequent sampling in time to estimate these statistics, as recognized a decade ago (22). In the current study, μ was in the same units as the distance between plants. More information might be obtained by analyzing data generated by varying the size of the sampling unit. Hughes et al. (16) recently exploited the relationships between disease incidence at two levels in a spatial hierarchy to design an effective sampling method and to show how disease aggregation at the lower scale (e.g., leaves) can reduce the rate of disease increase at the upper scale (e.g., plants).

The simulation model uses a half-Cauchy distribution, a particular form of inverse power-law distribution, to model spore dispersal. Another distribution that is used commonly to model spore dispersal is the exponential. Some observed disease gradient and spore dispersal distributions are fitted better by one model and some by the other, and sometimes both models provide an adequate fit (5,6). The spatial spread of disease with the exponential distribution has been studied extensively (37–40,45), and simulation studies have shown that the exponential distributions produce a more regular radial-spatial pattern of disease spread, while long-tailed distributions such as the Cauchy produce more long-distance dispersal and the resulting spatial pattern appears to be clumped or fractal (26,31,32). Further work is needed to study the extent to which the conclusions drawn in this study concerning the effects of initial epidemic conditions, spore dispersal gradient, and sporulation rate on spatio-temporal statistics apply to epidemics in which spore dispersal follows exponential distribution.

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LITERATURE CITED

- Burrough, P. A. 1987. Spatial aspects of ecological data. Pages 213-251 in: Data Analysis in Community and Landscape Ecology. R. H. G. Jongman, C. F. G. Braak, and O. F. R. van Tongeren, eds. PUDOC, Wageningen, the Netherlands.
- Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York.
- Chellemi, D. O., Rohrbach, K. G., Yost, R. S., and Sonoda, R. M. 1988. Analysis of the spatial pattern of plant pathogens and diseased plants using geostatistics. *Phytopathology* 78:221-226.
- Cox, D. R., and Snell, E. J. 1989. Analysis of Binary Data, 2nd ed. Chapman and Hall, London.
- Ferrandino, F. J. 1996. Length scale of disease spread: Fact or artifact of experimental geometry. *Phytopathology* 86:806-811.
- Fitt, B. D. L., Gregory, P. H., Todd, A. D., McCartney, H. A., and MacDonald, O. C. 1987. Spore dispersal and plant disease gradients: A comparison between two empirical models. *J. Phytopathol.* 118:227-242.
- Fleiss, J. L. 1981. Statistical Methods for Rates and Proportions, 2nd ed. John Wiley & Sons, New York.
- Fry, W. E. 1982. Principles of Plant Disease Management. Academic Press, New York.
- Gibson, G. J. 1997. Markov chain Monte Carlo methods for fitting spatio-temporal stochastic models in plant epidemiology. *Appl. Stat.* 46:215-233.
- Gibson, G. J., and Austin, E. J. 1996. Fitting and testing spatio-temporal stochastic models with application in plant epidemiology. *Plant Pathol.* 45:172-184.
- Gottwald, T. R. 1995. Spatio-temporal analyses and isopath dynamics of citrus scab in nursery plots. *Phytopathology* 85:1082-1092.
- Gottwald, T. R., Cambra, M., Moreno, P., Camarasa, E., and Piquer, J. 1996. Spatial and temporal analysis of citrus tristeza virus in eastern Spain. *Phytopathology* 86:45-55.
- Gubbins, S., and Gilligan, C. 1997. A test of heterogeneous mixing as a mechanism for ecological persistence in a disturbed environment. *Proc. R. Soc. Lond. B* 264:759-763.
- Hughes, G., and Madden, L. V. 1993. Using the beta-binomial distribution to describe aggregated patterns of disease incidence. *Phytopathology* 83:759-763.
- Hughes, G., Madden, L. V., and Munkvold, G. P. 1996. Cluster sampling for disease incidence data. *Phytopathology* 86:132-137.
- Hughes, G., McRoberts, N., Madden, L. V., and Gottwald, T. R. 1997. Relationships between disease incidence at two levels in a spatial hierarchy. *Phytopathology* 87:542-550.
- Jeger, M. J. 1989. Spatial Components of Plant Disease Epidemics. Prentice Hall, Englewood Cliffs, NJ.
- Kranz, J. 1968. Eine Analyse von annuellen Epidemien pilzlicher Parasiten. II. Qualitative und quantitative Merkmale von Befallskurven. *Phytopathol. Z.* 61:170-190.
- Kranz, J. 1974. Comparison of epidemics. *Annu. Rev. Phytopathol.* 12: 355-374.
- Kranz, J. 1978. Comparative anatomy of epidemics. Pages 33-62 in: Plant Disease. Vol. 2, How Disease Develops in Populations. J. G. Horsfall and E. B. Crowling, eds. Academic Press, New York.
- Madden, L. V. 1986. Statistical analysis and comparison of disease progress curves. Pages 55-84 in: Plant Disease Epidemiology. Vol. 1, Population Dynamics and Management. K. J. Leonard and W. E. Fry, eds. MacMillan Publishing Company, New York.
- Madden, L. V. 1989. Dynamic nature of within-field disease and pathogen distributions. Pages 96-126 in: Spatial Components of Plant Disease Epidemics. M. J. Jeger, ed. Prentice-Hall, Englewood Cliffs, NJ.
- Madden, L. V., and Hughes, G. 1995. Plant disease incidence: Distributions, heterogeneity, and temporal analysis. *Annu. Rev. Phytopathol.* 33: 529-564.
- Madden, L. V., Hughes, G., and Ellis, M. A. 1995. Spatial heterogeneity of the incidence of grape downy mildew. *Phytopathology* 85:269-275.
- McRoberts, N., Hughes, G., and Madden, L. V. 1996. Incorporating spatial variability into simple disease progress models for crop pathogens. Pages 75-82 in: Aspects of Applied Biology, Vol. 46, Modelling in Applied Biology: Spatial Aspects. The Association of Applied Biologists, Wellesbourne, Warwick, England.
- Minogue, K. P. 1989. Diffusion and spatial probability models for disease spread. Pages 127-144 in: Spatial Components of Plant Disease Epidemics. M. J. Jeger, ed. Prentice-Hall, Englewood Cliffs, NJ.
- Minogue, K. P., and Fry, W. E. 1983. Models for the spread of disease: Model description. *Phytopathology* 73:1168-1173.
- Mollison, D. 1977. Spatial contact models for ecological and epidemic spread. *J. R. Stat. Soc. Ser. B* 39:283-326.
- Nelson, S. C. 1996. A simple analysis of disease foci. *Phytopathology* 86:332-339.
- Shaner, G., and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. *Phytopathology* 67:1051-1056.
- Shaw, M. W. 1995. Simulation of population expansion and spatial pattern when individual dispersal distributions do not decline exponentially with distance. *Proc. R. Soc. Lond. B* 259:243-248.
- Shaw, M. W. 1996. Simulating dispersal of fungal spores by wind, and the resulting patterns. Pages 165-172 in: Aspects of Applied Biology, Vol. 46, Modelling in Applied Biology: Spatial Aspects. The Association of Applied Biologists, Wellesbourne, Warwick, England.
- Smith, V. L., Campbell, C. L., Jenkins, S. F., and Benson, D. M. 1988. Effects of host density and number of disease foci on epidemics of Southern blight of processing carrot. *Phytopathology* 78:595-600.
- Stein, A., Kocks, C. G., Zadoks, J. C., Frinking, H. D., Ruissen, M. A., and Myers, D. E. 1994. A geostatistical analysis of the spatio-temporal development of downy mildew epidemics in cabbage. *Phytopathology* 84:1227-1239.
- Sweetmore, A., Simons, S. A., and Kenward, M. 1994. Comparison of disease progress curves for yam anthracnose (*Colletotrichum gloeosporioides*). *Plant Pathol.* 43:206-215.
- Taylor, L. R. 1961. Aggregation, variance and the mean. *Nature (Lond.)* 189:732-735.
- van den Bosch, F., Frinking, H. D., Metz, J. A. J., and Zadoks, J. C. 1988. Focus expansion in plant disease. III: Two experimental examples. *Phytopathology* 78:919-925.
- van den Bosch, F., and Metz, J. A. J. 1996. The velocity of spatial population expansion: An overview of the individual based approach. Pages 231-238 in: Aspects of Applied Biology, Vol. 46, Modelling in Applied Biology: Spatial Aspects. The Association of Applied Biologists, Wellesbourne, Warwick, England.
- van den Bosch, F., Zadoks, J. C., and Metz, J. A. J. 1988. Focus expansion in plant disease. I: The constant rate of focus expansion. *Phytopathology* 78:54-58.
- van den Bosch, F., Zadoks, J. C., and Metz, J. A. J. 1988. Focus expansion in plant disease. II: Realistic parameter-sparse models. *Phytopathology* 78:59-64.
- Waggoner, P. E. 1986. Progress curves of foliar diseases: Their interpretation and use. Pages 3-37 in: Plant Disease Epidemiology. Vol. 1, Population Dynamics and Management. K. J. Leonard and W. E. Fry, eds. MacMillan Publishing Company, New York.
- Waggoner, P. E., and Rich, S. 1981. Lesion distribution, multiple infection, and the logistic increase of plant disease. *Proc. Natl. Acad. Sci. U.S.A.* 78:3292-3295.
- Wichmann, B. A., and Hill, I. D. 1982. An efficient and portable pseudo-random number generator. *Appl. Stat.* 31:188-190.
- Yang, X. B., and TeBeest, D. O. 1992. Dynamic pathogen distribution and logistic increase of plant disease. *Phytopathology* 82:380-383.
- Zadoks, J. C., and van den Bosch, F. 1994. Expansion and spatial spread of disease. *Annu. Rev. Phytopathol.* 32:503-521.