Effects of Quadrat Size and Shape, Initial Epidemic Conditions, and Spore Dispersal Gradient on Spatial Statistics of Plant Disease Epidemics

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ABSTRACT

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The spatiotemporal spread of plant diseases was simulated using a stochastic model to study the effects of initial conditions (number of plants initially infected and their spatial pattern), spore dispersal gradient, and size and shape of sampling quadrats on statistics describing the spatiotemporal dynamics of epidemics. The spatial spread of disease was simulated using a half-Cauchy distribution with median dispersal distance μ (units of distance). A total of 54 different quadrat types, including 23 distinct sizes ranging from 4 to 144 plants, were used to sample the simulated epidemics. A symmetric form of the binary power law with two parameters (α , β) was fitted to the sampled epidemic data using each of the 54 quadrats for each replicate simulation run. The α and β estimates were highly correlated positively with each other, and their estimates were comparable to those estimated from observed epidemics. Intraclass correlation (κ) was calculated for each quadrat

type; κ decreased exponentially with increasing quadrat size. An asymmetric form of the binary power law with three parameters (α_1 , β_1 , β_2) was used to relate κ to the disease incidence (p); β_1 was highly correlated to β : $\beta_1 \approx \beta - 1$. In general, initial conditions and quadrat size affected α , β , α_1 , β_1 , and β_2 greatly. The parameter estimates increased as quadrat size increased, and the relationships were described well by a linear regression model on the logarithm of quadrat size with the slope or intercept parameters dependent on initial conditions and μ . Compared with initial conditions and quadrat size, the overall effects of μ and quadrat shape were generally small, although within each quadrat size and initial condition they could be substantial. Quadrat shape had the greatest effect when the quadrat was long and thin. The relationship of the index of dispersion (D) to p and quadrat size was determined from the α and β estimates. D was greatest when p was 0.5 and decreased when p approached 0 or 1. It increased with quadrat size and the rate of the increase was maximum when p was 0.5 and decreased when p approached 0 or 1.

Characterization of the spatiotemporal dynamics of plant disease incidence and severity over time and space is of fundamental importance in epidemiology for describing and understanding the development of diseases, for evaluating treatments (12,16,37), and for developing efficient sampling schemes (2,10,18). Many statistical methods have been developed and used to analyze the spatiotemporal dynamics of plant disease epidemics and have been reviewed recently (11,16,25,37).

The beta-binomial distribution is more appropriate than the negative binomial distribution for examining spatial patterns of disease incidence for the presence of aggregation (9,16). An alternative measure of variability is the intraclass correlation (4), which measures the correlation between the binary responses (diseased or not diseased) of individual plants within a sampling unit. The dispersion or aggregation parameter of the beta-binomial distribution is related mathematically to the intraclass correlation coefficient. Spatial autocorrelation is used to determine the spatial dependence of disease incidence over various spatial lags (1,5,17). The binary form of the power law relationship, modified from Taylor's power law (29), has been used to investigate the temporal dynamics of aggregation of diseased plants (8,16).

These spatiotemporal statistics and models are very useful in describing and summarizing observed epidemics. They can also

be used in comparative epidemiology to characterize individual epidemics, to identify similarities and differences between epidemics, and, thus, to identify the key determinants of epidemic development (13,14). Their usefulness in comparative epidemiology, however, depends critically on our understanding of their relationships with the underlying biological and physical processes and the relative effects of biological and physical processes on these statistics. Using an individual-based and spatially explicit stochastic simulation model, Xu and Ridout (33) investigated the effects of initial epidemic conditions (initial number of infected plants and their spatial pattern), sporulation rate, and spore dispersal gradient on the spatiotemporal dynamics of plant disease epidemics. They demonstrated clearly the importance of initial epidemic conditions and spore dispersal gradient in influencing intraclass correlation (based on a 5×5 quadrat) and spatial autocorrelation, agreeing with Yang (35).

The objective of this paper was to study the effects of sampling unit size and shape on intraclass correlation and the binary form of the power law relationship under various initial epidemic conditions using the simulation model described previously (33). The simulation model is not intended to be an exact model of any specific epidemic, but it reflects the main processes involved in the development of an epidemic. Intraclass correlation was calculated from the simulated epidemic data; the binary form of the power law was fitted to the simulated data. The relationships of the calculated intraclass correlation and the estimated binary power law parameters to the underlying biological and physical processes and to quadrat size and shape were investigated.

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THEORY AND APPROACHES

The binary form of the power law relationship. Taylor's power law (29) has been extended to relate the observed variance (V_0) of disease incidence between quadrats to the variance expected (V_R) if a binomial distribution is assumed for the number of diseased plants per quadrat (8,16). This relationship is called the binary form of the power law and can be used to investigate the temporal dynamics of aggregation of diseased plants. There are two forms of this relationship. One is a symmetric form:

$$V_0 = AV_R^\beta = A[p(1-p)/n]^\beta \quad (1)$$

in which p is the disease incidence and n is the total number of plants per quadrat. The other is a more general (potentially) asymmetric form:

$$V_0 = Ap^{\beta_1}(1-p)^{\beta_2} \quad (2)$$

in which quadrat size (n) is not included explicitly but is incorporated into A .

The following discussion uses equation 1 only. When V_0 is greater than V_R , aggregation is indicated. Thus the condition for aggregation is

$$V_0/V_R = A[p(1-p)/n]^{\beta-1} > 1 \quad (3)$$

This variance/variance ratio is called the index of dispersion (D) (3,16) and provides a measure of the degree of aggregation of diseased plants. The value of D is determined jointly by A , n and β ; it is not possible to infer D from either A or β alone.

If D is plotted against p , parameter A affects only the amplitude of the curve, whereas β determines its shape. The greater the value of A , the greater the value of D . The greater the value of β , the lower the value of D , since $p(1-p)/n < 1$. If $A < 1$, β must be less than 1 to satisfy equation 3. When $\beta = 1$, D is constant and equal to A ; when A and $\beta = 1$, there is no aggregation or over-dispersion ($D = 1$). When $\beta > 1$, D increases with p , reaches a maximum, $A[1/(4n)]^{\beta-1}$, at $p = 0.5$, and then decreases toward 0 as p approaches 1. However, for $\beta < 1$, D increases as p approaches 0 or 1. For all the plant disease incidence data that have been fitted to the binary form of the power law so far, estimates of β have been greater than or equal to 1 (16,17,19).

Another related statistic based on quadrat sampling is intraclass correlation (κ), which measures the tendency of the plants within a sampling quadrat to have a similar disease status. Positive values of κ indicate aggregation of disease; κ is related to the binary form of the power law and index of dispersion (16):

$$\kappa = (D - 1)/(n - 1) \quad (4)$$

where D is the index of dispersion and n is the quadrat size.

The simulation model and details of simulation runs. Details of the model have been described previously (33). The model is a two-dimensional stochastic spatial contact model (21) in which the distance that spores are dispersed follows a specific long-tailed probability distribution, the half-Cauchy distribution (26).

The spread of disease is modeled on a rectangular lattice of plants consisting of $n_r = 150$ rows with $n_c = 150$ plants in each row. The distance between adjacent plants within a row is $d_c = 1$, and the distance between rows is $d_r = 1$. Each plant is assumed to consist of $n_s = 30$ units that are susceptible to the pathogen. 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 lattice.

Having completed a latent period of $t_L = 5$ days, an infected unit becomes infectious for $t_I = 10$ days, producing viable spores according to a Poisson process at a rate of λ (per day, sporulation rate). Spores are assumed to be dispersed independently of one another and to travel in straight lines of random orientation, with the dispersal distance varying randomly according to a half-Cauchy distribution (26), with median dispersal distance parameter μ . If the spore falls beyond the end of a row by a

distance of more than $d_r/2$, 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 and this unit becomes infected if it has not been infected previously; otherwise the spore has no effect. The model allows for the spread of disease within as well as between plants. Random numbers were generated using the uniform random number generator of Wichmann and Hill (32).

The simulation variables included the initial number of infected plants (Y_0) and their spatial pattern (Y_S) and the median spore dispersal parameter (μ). In the present study, the sporulation rate was constant ($\lambda = 0.7 \text{ day}^{-1}$), as previous studies (33) showed that λ did not affect spatial statistics describing the dynamics of epidemics directly, only indirectly via disease incidence. The initial number of infected plants (Y_0) was 1, 4, or 9, and each had a single infected unit when it entered its infectious period. The initial infected plants were located with one of three spatial patterns (Y_S): clumped, regular, and random. With $Y_0 = 1$, the regular and clumped patterns were the same, i.e., the infected plant was placed in the (approximate) center of the grid. 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 4 or 9 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 generator (32) for each simulation run. Thus, in total, there were eight combinations of Y_0 and Y_S , i.e., eight initial epidemic conditions (Y). Five values of μ were used: 0.25, 0.50, 1.0, 2.0, and 4.0, in the same units as d_r . Thus, there were in total 40 (8×5) combinations of the three simulation variables (Y_0 , Y_S , and μ). For each combination of Y_0 , Y_S , and μ , the model was simulated 50 times, with each run terminating at various times, ranging from 48 to 145 days, to ensure that the final disease incidence was greater than 80%. This ensured a wide range of disease incidence values for estimating the intraclass correlation and variance-mean relationship.

Statistical analysis. The effects of the initial number of infected plants (Y_0) and their spatial configuration (Y_S), the dispersal parameter (μ), and the sampling quadrat size (Q_S) and shape (Q_P) on intraclass correlation and on the binary power law relationship describing the spatiotemporal dynamics of the simulated epidemics were studied.

Temporal rates of disease progress were not calculated because previous studies (33) showed that the rate of increase in disease incidence (p) over time was well described by the logistic model, with the rate determined mainly by λ and μ . The binary form of the power law was fitted to the data sampled from each simulated epidemic using quadrats of various sizes and shapes. Plants with at least one unit infected were classified as diseased. Although the epidemic was simulated on a 150×150 grid, disease incidence data were sampled only from the central 120×120 grid at an interval of 3 days, using a contiguous array of 1 of 54 quadrat types (Q). The 54 quadrat types were chosen according to the following criteria: (i) the length and width of the quadrat were exact divisors of 120, so that an array of contiguous quadrats covered the whole central grid; (ii) the maximum quadrat area was 144 plants; (iii) all possible square quadrats were included; and (iv) rectangular quadrats were included if there were at least two quadrat shapes of the same area.

At each sampling time, the variance (V_0) of disease incidence among quadrats and intraclass correlation, calculated as $\kappa = (D - 1)/(n - 1) = (V_0/V_R - 1)/(n - 1)$, were estimated for each of the 54 quadrat types; these calculations were implemented directly in the simulation program. Equations 1 and 2 were fitted to the data obtained from each simulated epidemic. However, there was little difference in the goodness-of-fit of the two models. Therefore, only the results from fitting equation 1 are presented in this paper.

For parameter estimation, a linearized form of equation 1 was used:

$$\ln(V_0) = \ln(A) + \beta \ln[p(1-p)/n] = \alpha + \beta \ln[p(1-p)/n] \quad (5)$$

where $\alpha = \ln(A)$.

As in the previous paper (33), the asymmetric form of the binary power law (equation 2) was used to relate κ to disease incidence (p):

$$\kappa = A_1 p^{\beta_1} (1-p)^{\beta_2} \quad (6)$$

where A_1 , β_1 , and β_2 are parameters to be estimated. A linearized form of equation 6 was used for parameter estimation:

$$\ln(\kappa) = \alpha_1 + \beta_1 \ln(p) + \beta_2 \ln(1-p) \quad (7)$$

where $\alpha_1 = \ln(A_1)$. Equations 5 and 7 were fitted to the data for each simulated epidemic by least squares regression, assuming a random error with constant variance. In fitting the two models, data points with $p < 0.01$ were excluded from analysis to remove small negative κ values.

The relationship between α and β was analyzed using regression analysis; in the analysis, β and α were arbitrarily treated as independent and dependent variables respectively. Average values of α and β over each combination of Q , Y , and μ were used in the regression analysis. The effects of Q , Y , and μ on the α - β relation were also investigated using regression analysis. Both μ and Y were regarded as factors (categorical variables), while Q (as Q_S) was treated as a continuous variable.

A factorial analysis of variance (ANOVA) was used to assess the overall effects of Q_S , Q_P , Y_0 , Y_S , and μ on the five parameters (α , β , α_1 , β_1 , β_2). In ANOVA, Y_0 and Y_S were combined into a single factor, termed initial epidemic condition (Y), because the combinations of Y_0 and Y_S do not form a complete factorial set, and the emphasis of this study is on quadrat size and shape. Furthermore, in ANOVA, Q_S was nested within each Y and μ combination, and Q_P was nested within Q_S . Therefore, the percentage of the total variance in the parameters that was accounted for by Y and μ was calculated in relation to the variation between simulation runs. In contrast, the percent variance accounted for by Q_S and Q_P was calculated in relation to the variation within simulation runs and, therefore, cannot be

compared directly with those by Y and μ . To assess the effect of Q_S on the parameters without the confounding effects of Q_P , ANOVA was also applied to square quadrats only. To assess how the effects of μ , Q_S , and Q_P on the parameters varied with Y , a separate ANOVA was performed for each of the eight initial conditions.

Regression analysis was also used to determine the relationship of the parameters with the simulation variables. In the regression analysis, both μ and Y were regarded as factors (categorical variables), while Q_S was treated as a continuous variable. All the statistical analyses were done using Genstat (Numerical Algorithms Group Ltd., Oxford).

RESULTS

Binary form of the power law. General results. The higher the value of μ , the greater the rate of increase in disease incidence, as shown in the previous study (33). None of the 2,000 simulated epidemics died out (i.e., failed to increase), and equation 5, thus, was fitted to data from all 2,000 simulation runs, with a separate regression for each of the 54 quadrat types. In total, there were 108,000 regression analyses. The percent variance accounted for by equation 5 ranged from 84 to 100%, with an average of 98.5%. The estimated values of α ranged from 0.6 to 10.1 with a mean of 3.7; most values were in the range of 1 to 6; the β estimates ranged from 0.93 to 1.94, with a mean of 1.22. The distribution of a third binary power law parameter, $\gamma = \alpha - \beta \ln(n)$, ranged from -1.88 to 0.59, with a mean of -0.41.

Relationship between α and β . There was a highly significant positive correlation between α and β ($r = 0.79$) (Fig. 1). Regression analysis showed that $\approx 63\%$ of the total variation in β was accounted for by α . Including Y as a factor in the model increased the variation accounted for to 81%, including μ as a factor increased this further to 91% and including the logarithm of quadrat size as a covariate increased the percent variance accounted for to 96%. Neither Y nor μ had any significant effect on the regression coefficient of either α or $\ln(Q_S)$; furthermore, there were no significant interactions between Y and μ . Increasing μ and Y_0 both led to greater intercepts. The regular pattern of initial infected plants led to greater intercepts than the random pattern; in turn, the random pattern had greater intercepts than the clumped pattern (Table 1).

Fig. 1. Relationship of α , averaged for each combination of Q (quadrat size and shape), Y (initial epidemic condition), and μ (median spore dispersal distance), with the corresponding average β ; α and β are the two parameters of the binary power law model fitted to the simulated epidemics.

TABLE 1. Parameter estimates of the regression model of β on $\alpha^{a,b}$

Variable ^c	Estimate
Constant (i.e., Y_1 : Y_S = regular or clumped; $Y_0 = 1$)	1.086 $\pm$ 0.0037
α	0.142 $\pm$ 0.0015
$\ln(n)$	-0.130 $\pm$ 0.0023
Y_2 , Y_S = random, $Y_0 = 1$	-0.015 $\pm$ 0.0019
Y_3 , Y_S = clumped, $Y_0 = 4$	As Y_2
Y_4 , Y_S = regular, $Y_0 = 4$	0.038 $\pm$ 0.0023
Y_5 , Y_S = random, $Y_0 = 4$	0.023 $\pm$ 0.0026
Y_6 , Y_S = clumped, $Y_0 = 9$	As Y_2
Y_7 , Y_S = regular, $Y_0 = 9$	0.089 $\pm$ 0.0023
Y_8 , Y_S = random, $Y_0 = 9$	0.054 $\pm$ 0.0028
$\mu = 0.50^d$	0.004 $\pm$ 0.0018
$\mu = 1.0$	0.014 $\pm$ 0.0018
$\mu = 2.0$	0.035 $\pm$ 0.0019
$\mu = 4.0$	0.078 $\pm$ 0.0020

^a α and β are the two parameters of the binary power law model fitted to the simulated epidemics. Model parameters are described in text (equations 1 and 5)

^b Initial epidemic condition (Y) and median spore dispersal distance (μ) were treated as factors and only affected the intercept (constant). Y has eight levels and μ has five levels. Y = initial conditions specified by the initial number of infected plants ($Y_0 = 1, 4$, or 9) and their spatial pattern (Y_S = regular, random, or clumped); μ = median spore dispersal distance.

^c n = sampling unit (quadrat) size

^d For $\mu = 0.25$, it is zero.

Parameter α . Figure 2 shows the effects of Y , μ , and Q_5 on the parameter α , averaged over Q_p . ANOVA (Table 2) showed that Y and μ accounted jointly for 75% of the total variation in α between simulation runs, of which 10% was due to μ alone and 4% to their interaction. The mean values of α with $Y_0 = 1, 4$, and 9 plants were, respectively, 3.65, 3.70, and 3.82, and 3.26, 3.60, and

4.21 for the clumped, regular, and random patterns, respectively. The mean values of α increased from 3.57 to 4.06 as μ increased from 0.25 to 4.0; effects of μ decreased as the number of initial infected plants increased (Fig. 2). Q_5 accounted for $\approx 94\%$ of the total variation in α within simulation runs. Increasing quadrat size led to higher α (Fig. 2); the rate of increase was greatest for the

Fig. 2. Relationship of α (averaged over quadrat shapes) to the logarithm of quadrat size for each of the eight initial conditions. $\circ$, $\square$, $\triangle$, ∇ , and $\diamond$ represent median spore dispersal distance $\mu = 0.25, 0.50, 1.0, 2.0$, and 4.0 , respectively. Y_0 = initial number of infected plants.

random pattern when $Y_0 > 1$. The average values of α ranged from 1.06 for $Q_S = 4$ to 6.02 for $Q_S = 144$. The overall effects of Q_P were very small compared with Q_S (Fig. 3A). To analyze the effects of Q_S on α without the confounding effect of Q_P , a separate ANOVA was applied to the square quadrats only; the results were similar to those shown in Table 2 except that the effect of μ and Q_S increased slightly.

To focus on how the effects of Q_S and Q_P on α varied with the initial conditions, a separate ANOVA was done for each set of initial conditions (Table 3). The contribution of μ to variation in α between simulation runs ranged from 11 to 49% and was greater when $Y_0 = 1$ or 4, due mainly to the difference between $\mu = 4.0$ and the others (Fig. 2). The percent variance within simulation runs due to Q_S exceeded 95% for all eight initial conditions. The contributions of Q_P and interactions between Q_S and μ were small. The results of ANOVA applied only to the square quadrats were similar to those shown in Table 3.

A separate ANOVA was done for each combination of Y and Q_S to determine how the effects of μ and Q_P on α varied with quadrat size within each initial condition. The percent variance accounted for by μ and Q_P varied greatly with quadrat size. For μ , it ranged from 2 to 84%; for Q_P , it varied from 7 to 96%. The contribution of μ to the variation in α generally decreased with increasing Q_S , and was least for the clumped pattern. Greatest variation in α was attributed to Q_P for the clumped pattern.

A regression model was used to describe the effects of quadrat size on average α for each combination of simulation variables (Table 4); this model was fitted to the full data set and to a restricted data set comprising only the square quadrats. For both data sets, the models accounted for more than 94% of the total variation in α . Slope parameter b_1 depended on initial epidemic conditions, but intercept parameter a_1 did not. The value of b_1 was greatest for the random pattern. Including μ as an additional explanatory variable improved the fit of the models significantly, but the increase in the percent variance accounted for was less than 2%.

Parameter β . The overall effects of Y , μ , and Q_S on parameter β , averaged over Q_P , were very similar to those on parameter α , except that the effect of μ was considerably greater. ANOVA (Table 2) showed that Y and μ accounted jointly for 82% of the total variation in β between simulation runs; 56 and 23% were due to the main effects of Y and μ respectively. The mean values of β with $Y_0 = 1, 4$, and 9 plants were, respectively, 1.17, 1.21, and 1.25 and 1.12, 1.22, and 1.28 for the clumped, regular, and random patterns, respectively. As μ increased from 0.25 to 4.0, the mean value of β increased from 1.17 to 1.31, and the increase was mainly restricted to $\mu = 0.5$ to 4.0; the effect of μ decreased with increasing Y_0 . Q_S accounted for $\approx 63\%$ of total variation in β within simulation treatments. Increasing quadrat size led to higher β ; the average values of β ranged from 1.07 for $Q_S = 4$ to 1.34 for $Q_S = 100$. Approximately 8% of the total variation within each simulation run was due to Q_P . Generally, the effects of Q_P were mainly restricted to those quadrat sizes containing quadrat types

with a long shape, such as $M \times 20$ or 24 (Fig. 3B). There were also large interactions between Y and Q_S , accounting for approximately 12% of the total variation within simulations. The rate of the increase in β with increasing Q_S was least for the clumped pattern and greatest for the random pattern. ANOVA, performed only for the square quadrats, gave results very similar to those shown in Table 2.

To focus on how the effects of μ , Q_S , and Q_P on β varied with initial conditions, ANOVA was applied to each Y (Table 3). The contribution of μ to variation in β between simulation runs ranged from 29 to 73% and was least for the clumped pattern. The percent variance due to Q_S , ranging from 54 to 87%, increased with increasing Y_0 , was least for the clumped pattern when $Y_0 > 1$, and was similar for the random and regular patterns. The contribution of Q_P ranged from 7 to 39% and was greatest for the clumped pattern. Results of ANOVA applied to the square quadrats were similar to those shown in Table 3, except that the contribution of Q_S increased considerably.

The contribution of μ and Q_P to variation in β within each combination of Y and Q_S varied greatly with Q_S . For μ , the percent variance accounted for ranged from 5 to 59%; for Q_P , it ranged from 0 to 95%. The interaction between μ and Q_P was generally negligible. The contribution of μ decreased with increasing Y_0 and was less for the clumped and regular patterns. Q_P accounted for less variation in the regular and random patterns.

A regression model was used to describe the effects of quadrat size on the average β for each combination of simulation variables (Table 4). This model was fitted to the full data set and to a restricted data set comprising only the square quadrats. For both data sets, the model accounted for more than 86% of the total variation in β . Slope parameter b_2 depended on initial conditions, and intercept parameter a_2 depended on μ . Slope b_2 was greatest for the random pattern and smallest for the clumped pattern and increased with increasing Y_0 . For the intercept, there was no significant difference between $\mu = 0.25$ and 0.5, but a_2 increased with increasing μ from 0.5 to 4.0.

Intraclass correlation. *General results.* Intraclass correlation (κ) initially increased and then decreased with increasing disease incidence (p); the relationship between κ and p became gradually more symmetrical from the clumped, to the regular, and then to the random pattern. The κ values decreased with increasing quadrat size, and the maximum value of κ decreased as Y_0 increased. Figure 4 shows the average κ for each quadrat type over all levels of disease incidence and over all combinations of Y and μ . Clearly, average κ decreased with increasing quadrat sizes. There were large differences between different quadrat types within each quadrat size (i.e., different quadrat shape). Most of the differences between quadrat shapes were due to the small κ values for those quadrats with one extremely long side (20 or 24). Indeed, for these quadrats, the average κ varied little as quadrat size increased (Fig. 4). Square quadrats had the greatest κ values for a given quadrat size. Furthermore, for square quadrats average

TABLE 2. Percentage of variance accounted for by simulation variables (μ , Y , Q_S , Q_P)^a in five model parameters (α , β , α_1 , β_1 , β_2)^b describing the binary power law relationship and the relationship of intraclass correlation to disease incidence

Parameter	Between simulation runs			Within simulation runs				
	μ	Y	$\mu \times Y$	Q_S	Q_P	$\mu \times Q_S$	$Y \times Q_S$	$\mu \times Y \times Q_S$
Binary power law								
α	10.3	60.5	4.1	94.2	0.5	0.3	2.3	0.1
β	22.9	56.4	2.8	63.4	8.4	0.8	11.8	0.5
Intraclass correlation								
α_1	3.9	49.6	11.4	6.2	9.7	0.4	17.2	1.4
β_1	26.8	51.2	2.6	52.1	17.6	0.9	8.4	0.8
β_2	3.8	6.8	16.2	45.5	13.1	3.4	3.4	1.3

^a Y = initial conditions specified by initial number of infected plants ($Y_0 = 1, 4$, or 9) and their spatial pattern (Y_S = regular, random, or clumped); μ = median spore dispersal distance; Q_S = sampling unit (quadrat) size; Q_P = sampling unit (quadrat) shape.

^b Model parameters are described in the text (equations 1,5,6, and 7).

Fig. 3. The averages of parameters α , β , α_1 , β_1 , and β_2 for each of the 54 different quadrat sizes and shapes over all other simulation variables (initial conditions and spore dispersal gradient) and over all levels of disease incidence. ● represents square quadrats; ▽ and □ represent quadrats with one side of 20 and 24, respectively; ○ represents all other quadrats. α_1 , β_1 , and β_2 are the three parameters of the asymmetric form of the binary power law model describing the relationship of intraclass correlation to disease incidence for each simulated epidemic.

κ values decreased approximately exponentially with increasing quadrat size.

Equation 7 was fitted successfully to most of the 108,000 simulation runs. The percentage of variance accounted for ranged from 1 to 100%, with an average of 93%. There were 203 runs where 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 mainly restricted to the clumped pattern (163 cases) or to $\mu = 0.25$ (109 cases) or 0.5 (94 cases) but was not related to Q_S . The α_1 estimated values ranged from -1.86 to 0.63, with a mean of -0.40. The β_1 estimates ranged from -0.01 to 0.89 with a mean of 0.28. The β_2 estimates were more symmetrically distributed than β_1 and ranged from -0.14 to 1.36, with a mean of 0.41. All the pairwise correlations between the three parameters were small (<0.44).

Parameter α_1 . Figure 5 shows the effects of Y , μ , and Q_S on parameter α_1 , averaged over Q_P . ANOVA (Table 2) showed that Y and μ accounted jointly for 65% of the total variation in α_1 between simulation runs, of which 4% was due to μ alone and 11% to their interaction. The mean values of α_1 with $Y_0 = 1, 4$, and 9 plants were, respectively, -0.29, -0.39, and -0.49, and were -0.40, -0.56, and -0.24 for the clumped, regular, and random patterns, respectively. The mean values of α_1 were similar, approximately -0.38, as μ increased from 0.25 to 2, but the mean value was -0.49 for $\mu = 4$; the difference was mainly in simulations with initial conditions of the regular pattern at $Y_0 = 4$ or 9 and the random pattern at $Y_0 = 9$. Q_S only accounted for approximately 6% of the total variation in α_1 within simulation runs. However, there were considerable interactions between Q_S and Y , accounting for approximately 17% of total variation; depending on initial conditions, increasing quadrat size could lead to lower (e.g., Fig. 5B and E) or higher α (Fig. 5G). Q_P accounted

for approximately 10% of the total variation in α within simulation runs; much of this was due to the extremely small α values for quadrats with one side of 24 (Fig. 3C).

A separate ANOVA was done for each set of initial conditions to determine how the effects of Q_S and Q_P on α_1 varied with initial conditions (Table 3). The contribution of μ to variation in α_1 between simulation runs ranged from 4 to 78% and was much greater for the regular pattern when $Y_0 = 4$ or 9. The percent variance within simulation runs due to Q_S was generally small, ranging from 18 to 33%. The contributions of Q_P varied greatly from 6 to 66%; it was greater for the clumped and regular patterns than for the random pattern. The interactions between Q_S and μ were very small.

A separate ANOVA was done for each combination of Y and Q_S to determine how the effects of μ and Q_P on α_1 varied with quadrat size within each initial condition. The percent variance accounted for by μ and Q_P varied greatly with quadrat size. For μ , variance ranged from 1 to 84%; for Q_P , it ranged from 0 to 93%. The contribution of μ to the variation in α_1 was greatest for the regular pattern when $Y_0 > 1$ (average 64%, whereas for other initial conditions it was 9%); the contribution of μ was not related to Q_S . The greatest variation in α_1 was attributed to Q_P for the clumped and regular patterns. In general, interactions between μ and Q_P were small, ranging from 0 to 27%, and were highest for the regular pattern.

Parameters β_1 and β_2 . There was very high correlation between β and β_1 ($r = 0.97$): $\beta_1 \approx \beta - 1$. Thus, the overall effects of simulation variables on β_1 were very similar to those on β (Tables 2 and 3; Fig. 3B and D), and therefore are not presented. Figure 6 shows the effects of Y , μ , and Q_S on the parameter β_2 , averaged over Q_P . ANOVA (Table 2) showed that Y and μ accounted jointly for only 27% of the total variation in β_2 between simulation runs.

TABLE 3. Percentage of variance accounted for by the simulation variables (μ , Q_S , Q_P)^a in a separate analysis of the five model parameters (α , β , α_1 , β_1 , β_2)^b describing the binary power law relationship and intraclass correlation for each set of initial conditions^c

Model parameter	Variable	Condition 1		Condition 4			Condition 9		
		Regular	Random	Clumped	Regular	Random	Clumped	Regular	Random
Binary power law									
α	μ	45.4	47.8	35.1	48.8	43.0	10.6	10.9	15.9
	Q_S	96.2	95.6	95.3	95.4	97.1	95.4	96.7	98.2
	Q_P	1.6	0.8	3.0	2.5	0.8	3.5	1.5	0.4
	$\mu \times Q_S$	0.8	0.7	0.5	0.7	0.4	0.2	0.4	0.1
β	μ	60.2	59.8	54.0	72.6	66.5	28.5	54.1	50.2
	Q_S	63.6	56.5	55.6	68.6	78.0	53.5	85.7	86.9
	Q_P	23.1	15.3	34.1	21.0	11.5	39.3	7.3	6.7
	$\mu \times Q_S$	2.6	2.1	1.4	2.3	1.0	0.6	1.8	0.4
Intraclass correlation									
α_1	μ	3.9	5.6	3.0	56.7	4.7	15.4	77.7	18.4
	Q_S	22.1	17.6	29.6	32.5	30.0	29.3	19.6	18.2
	Q_P	38.8	10.4	46.9	39.0	6.4	51.8	66.4	18.5
	$\mu \times Q_S$	2.3	0.4	1.2	5.2	1.2	1.8	0.8	4.6
β_1	μ	62.9	52.5	48.7	63.6	71.5	40.4	60.2	61.7
	Q_S	49.1	42.2	49.1	57.7	62.1	48.8	72.5	70.5
	Q_P	37.2	24.2	41.6	30.2	22.4	44.7	17.1	17.2
	$\mu \times Q_S$	0.7	0.5	0.5	2.8	0.9	0.2	2.6	2.5
β_2	μ	27.6	17.0	18.4	19.0	24.7	22.9	38.8	2.6
	Q_S	48.9	40.0	50.5	56.5	53.8	52.7	33.0	53.3
	Q_P	26.8	19.6	31.0	17.4	14.2	35.1	27.5	8.3
	$\mu \times Q_S$	4.3	3.4	3.7	8.7	1.4	1.4	7.3	7.0

^a μ = median spore dispersal distance; Q_S = sampling unit (quadrat) size; Q_P = sampling unit (quadrat) shape.

^b Model parameters are described in the text (equations 1, 5, 6, and 7).

^c Initial conditions are specified by initial number of infected plants ($Y_0 = 1, 4$, or 9) and their spatial pattern (Y_S = regular, random, or clumped).

The random pattern had the smallest β_2 values for $Y_0 > 1$ (Fig. 6). Q_S accounted for approximately 46% of the total variation in β_2 within simulation treatments. Increasing quadrat size generally led to higher β_2 (Fig. 6); the average values of β_2 ranged from 0.31 for $Q_S = 4$ to 0.49 for $Q_S = 120$. Approximately 13% of the total variation within simulation runs was due to Q_P . A large proportion of the Q_P contribution was due to the large β_2 values for quadrats with one side of 20 or 24 (Fig. 3E). All the interaction terms were small.

To focus on how the effects of μ , Q_S , and Q_P on β_2 varied with initial conditions, ANOVA was applied separately for each value of Y (Table 3). The contribution of μ to variation in β_2 between simulation runs was generally low, ranging from 3 to 39%. The contribution of Q_S ranged from 33 to 57%. The contribution of Q_P ranged from 8 to 35% and was lowest for the random pattern. Results of ANOVA applied to the square quadrats were similar to those shown in Table 3.

The contribution of μ and Q_P to variation in β_2 varied greatly between combinations of Y and Q_S . For μ , its contribution ranged from 2 to 97% (average 22%); for Q_P , it ranged from 0 to 92% (average 49%). The interaction between μ and Q_P was generally small (average 6%). The contribution of μ was largest for the regular pattern at $Y_0 = 9$, least for the random pattern at $Y_0 = 9$, and similar for other initial conditions. Q_P accounted for the least and largest variation for the random and clumped patterns, respectively.

A linear regression model was used to describe the effect of the logarithm of quadrat size on average β_2 for each combination of simulation variables. This model was fitted to the full data set and to a restricted data set comprising only the square quadrats; for both data sets, the regression model accounted for more than 67% of the total variation in β_2 . The slope parameter did not depend on initial conditions or μ , but the intercept parameter depended on the interaction between Y and μ , which is why the values of these parameter estimates are not presented here. The slope estimate was greatest for the random pattern and smallest for the clumped pattern, and increased with increasing Y_0 . For the intercept, there was no significant difference between $\mu = 0.25$ and 0.5 but it increased with increasing μ from 0.5 to 4.0 .

Index of dispersion (D). Substituting for A ($= \exp(\alpha)$) and β in equation 3 with the equations relating α and β to quadrat size and simulation variables (Table 4) gave the relationship of D to disease incidence and sampling quadrat size as:

$$D = \exp(\alpha)[(1-p)p/n]^{\beta-1} = Cn^{b_1}[(1-p)p/n]^{a_2+b_2 \ln(n)-1} \quad (8)$$

where $C = \exp(a_1)$. Since b_1 and b_2 were dependent on initial conditions, it follows that D was also influenced by initial conditions. Figure 7 shows D for quadrat size up to 150 over the incidence range of 0.01 to 0.99 for three sets of initial conditions (Y_6 , Y_7 , and Y_8), with parameters based on the square quadrats (Table 4). As quadrat size increased, D increased with the rate of increase approximately linearly, especially for $n > 30$. For the regular pattern, D was much less than the random or the clumped pattern; D decreased more steeply with decreasing ($p < 0.5$) or increasing ($p > 0.5$) p for the random pattern than for the clumped pattern.

DISCUSSION

The symmetrical form of the binary power law (8) successfully described all the simulated epidemics for the 54 quadrat types with size ranging from 4 to 144 plants. Estimates of the two parameters (α , β) were comparable to those estimated from real data (16,17,19). As in the previous study (33), the asymmetrical

Fig. 4. Average intraclass correlation (κ) for each of the 54 quadrat types over all other simulation variables (initial conditions and spore dispersal gradient) and over all levels of disease incidence. ● represents square quadrats; ▽ and □ represent quadrats with one side of 20 and 24, respectively; ○ represents all other quadrats.

TABLE 4. Parameter estimates of the models^a describing the relationships of α and β ^b to quadrat size and simulation variables^c

Y	Y_S	Y_0	α				β			
			All quadrats		Square quadrats only		All quadrats		Square quadrats only	
			a_1	b_{1i}	a_1	b_{1i}	a_2	b_{2i}	a_2	b_{2i}
1	Regular	1		1.383±0.0088		1.405±0.0214		0.063±0.0013		0.055±0.0027
2	Random	1		1.440±0.0088		As Y_1		As Y_1		As Y_1
3	Clumped	4		1.303±0.0088		1.281±0.0233		0.047±0.0013		As Y_1
4	Regular	4	-1.254±0.0283	As Y_1	-1.228±0.0724	As Y_1	0.908±0.0043 ^d	0.074±0.0014	0.885±0.0099 ^d	0.073±0.0034
5	Random	4		1.613±0.0097		1.579±0.0268		0.097±0.0013		0.098±0.0030
6	Clumped	9		As Y_3		As Y_3		As Y_3		As Y_1
7	Regular	9		As Y_2		As Y_1		As Y_5		As Y_5
8	Random	9		1.711±0.0097		1.693±0.0268		0.122±0.0014		0.121±0.0034
	$\mu = 1.0$						0.018±0.0030		0.017±0.0081	
	$\mu = 2.0$						0.066±0.0030		0.067±0.0081	
	$\mu = 4.0$						0.144±0.0030		0.147±0.0081	
% Variance accounted for			94.5		94.4		86.5		86.6%	

^a The following equations were used to model the effect of quadrat size (n) on average α and β for each combination of initial epidemic conditions and median spore dispersal distance: $\alpha = a_1 + b_1 \ln(n)$ and $\beta = a_2 + b_2 \ln(n)$, where a_1 , b_1 , a_2 , and b_2 are parameters to be estimated.

^b α = The amplitude term in the binary power law model (16) for the variance of disease incidence data between quadrats; β = the power term in the binary power law model.

^c Simulation variables are initial conditions (Y) and median spore dispersal distance (μ). Y is specified by the initial number of infected plants ($Y_0 = 1, 4$, or 9) and their spatial pattern (Y_S = regular, random, or clumped).

^d For $\mu = 0.25$ or $\mu = 0.5$, it is zero.

form of the binary power law satisfactorily summarized the relationship between intraclass correlation and disease incidence. Analyses showed clearly that quadrat size has a large effect on the parameters of the binary power law models describing the variance-mean and intraclass correlation-incidence relationships. Furthermore, the effect of quadrat size depended on initial epidemic conditions. Compared with quadrat size, quadrat shape had small effects on the parameters.

Parameters α and β were significantly positively correlated; this significant correlation could also be observed from those α and β estimates obtained in experimental studies for tobacco virus diseases (8), strawberry leaf blight (30), and grape downy mildew (17). The initial epidemic conditions (number of initially infected plants and their spatial pattern) and the spore dispersal gradient (μ) had significant effects on the α - β linear relationship. For a given α value, increasing μ led to a greater β value, which indicates less

Fig. 5. Relationship of α_1 (averaged over quadrat shapes) to the logarithm of quadrat size for each of the eight initial conditions. ○, □, Δ, ▽ and ◇ represent median spore dispersal distance $\mu = 0.25, 0.50, 1.0, 2.0$, and 4.0 , respectively. Y_0 = initial number of infected plants.

aggregation of diseased plants. Increasing μ results in more long-distance dispersal and hence is expected to result in less aggregation near the source plants. The regular and clumped patterns had the highest and lowest β values, respectively. Increasing the number of initially infected plants led to greater β values for the random and regular patterns. For the regular pattern, initially infected plants were least aggregated, the aggregation of initially infected plants was greatest for the clumped pattern,

while aggregation of initially infected plants for the random pattern was between the two extremes. The greater the number of initially infected plants, the more likely the larger area will contain an initially infected plant for the random and regular patterns, thus leading to less aggregation over the simulation grid for these two patterns.

Initial epidemic conditions had considerable effects on the five parameters (α , β , α_1 , β_1 , β_2). Furthermore, initial conditions

Fig. 6. Relationship of β_2 (averaged over quadrat shapes) to the logarithm of quadrat size for each of the eight initial conditions. $\circ$, $\square$, $\triangle$, ∇ and $\diamond$ represent median spore dispersal distance $\mu = 0.25, 0.50, 1.0, 2.0$, and 4.0 , respectively. Y_0 = initial number of infected plants.

A Clumped; $Y_0 = 9$

B Regular; $Y_0 = 9$

C Random; $Y_0 = 9$

Fig. 7. The index of dispersion in relation to disease incidence and quadrat size calculated using equation 8. The index is based on the models derived from the square quadrats only for three of the eight initial conditions (Table 4). Y_0 = initial number of infected plants.

affected the relative contributions of quadrat size and shape and the spore dispersal gradient (μ) to variation in these parameters. The importance of initial epidemic conditions on the spatio-temporal dynamics of plant diseases has been demonstrated previously, theoretically and experimentally. In an experiment with southern blight of processing carrot, the rate of disease increase generally increased as the number of initial disease foci increased (27). An aggregated pattern of initial disease resulted in lower disease incidence than a uniform pattern for *Zucchini yellow mosaic virus* (22) and rice sheath blight (24). Analytical and simulation modeling studies have shown that aggregation slows the rate of disease development (20,31,33,36) and that spatial statistics are affected significantly by the initial conditions (33,35).

As expected, increasing μ led to less aggregation, i.e. smaller D or κ values, as it led to more long-distance dispersal. However, its overall effect on α , β , α_1 , β_1 , and β_2 was generally small compared with that of initial conditions, but it varied greatly with initial conditions, confirming previous simulation studies (33). In general, for each set of initial conditions, μ explained a greater proportion of the variation in α , β and β_1 than in α_1 or β_2 . Furthermore, the effects of μ on the parameters were much greater for the regular pattern than for the other patterns. This is likely to be due to the confounding effects of aggregation of initially infected plants with subsequent aggregation due to μ during the epidemic development period. For the regular pattern, initially infected plants were least aggregated when the epidemic started, thus aggregation of subsequent diseased plants due to μ was least confounded with the aggregation of initially infected plants.

Compared with other factors, quadrat size was the single most important factor affecting spatial statistics such as intraclass correlation, index of dispersion, and binary power law. An effect of quadrat size on spatiotemporal statistics was expected theoretically (15,16,34) and has been demonstrated experimentally (6,19). For example, heterogeneity for strawberry Phomopsis leaf blight, measured as the beta-binomial parameter θ , index of dispersion, and the binary power law parameters, was affected by the number of sampling units (15 leaflets or 5 leaves) per quadrat (30). Increasing quadrat size resulted in greater values of α , β , α_1 , β_1 , and β_2 . Indeed, more than 90% of the total variation in α was accounted for by a simple linear regression on the logarithm of quadrat size, with the slope parameter depending on initial conditions. Theoretically, the constant terms in these empirical models relating α and β to the logarithm of quadrat size (equations 8 and 9) should be 0 and 1 respectively because, for a quadrat of size 1 α and β are expected to be 0 ($A = 1$) and 1, respectively. However, setting the constants to zero or one as appropriate resulted in models that significantly overestimated and underestimated α and β at small and large quadrat sizes, respectively, indicating some nonlinear feature in the relationship. Nonetheless, the linear relationships provide a reasonable empirical fit to the data over the range of quadrat sizes considered. The relationship between quadrat sizes and the parameters was of a log-linear type. However, it is not immediately clear why the relationship should be of this form. It might be due to the Cauchy spore dispersal distribution used in the simulation model or to the nature of quadrat sampling, or to other causes. Further studies are needed to understand this.

Intraclass correlation (κ) increased initially and then declined with increasing disease incidence (p). This relationship has been observed in several experiments (17,19,30) (the beta-binomial parameter θ instead of κ was used [17,30]: κ is a monotonic function of θ). Increasing quadrat size led to decreasing κ values; again this relationship is of log-linear form. Declines in intraclass correlation with quadrat size have been observed by Madden et al. (19) and Tanne et al. (28); these authors showed that the beta-binomial parameter θ decreased with increasing quadrat size. It is expected that κ will decrease as quadrat size increases. Intraclass

correlation κ is defined as the mean of the complete set of pairwise correlations between plants within a quadrat. Spatial autocorrelation of individual plants declined with distance for this simulation model (33) and in experimental studies for citrus canker (7) and strawberry leather rot (23). As the quadrat size increases, the average distance between a pair of plants within quadrats will increase and, thus, lead to smaller κ . Furthermore, the previous study (33) showed that spatial autocorrelation declines approximately exponentially with distance for this simulation model, which may explain the observed log-linear relationship between κ and quadrat size. Increasing quadrat size led to decreasing κ values, implying less aggregation. However, the index of dispersion (D) increased with quadrat size, approximately linearly for quadrat size >30 ; this relationship is critically dependent on initial conditions. Since $D = 1 + \kappa(n - 1)$, it implies that the rate of decline in κ with increasing quadrat size must be less than $1/(n - 1)$. Thus, increasing quadrat sizes led to apparently contradictory results: smaller κ indicated less aggregation, larger D indicated greater aggregation. This stresses the importance of interpreting spatial statistics correctly.

Compared with the effect of quadrat size, the effect of quadrat shape was generally small, especially for α and β . However, there were large effects for certain sets of initial conditions and quadrat sizes. The greatest differences in spatial statistics were observed between square quadrats and quadrats of the same size but with a very long side (i.e., 20 or 24). Indeed, average intraclass correlation for these long and thin quadrats was very small and did not vary with increasing quadrat size. This may be due to the fact that, for the extremely long-shaped quadrats, κ was predominantly determined by the pairwise correlations of plants over a larger distance, which is very small. Generally the effect of quadrat shape was smaller for the random initial pattern than for the clumped or regular patterns. Some caution may be needed in interpreting the effects of quadrat shape because of the limited range of quadrat shapes that are available within each quadrat size.

This study shows clearly that spatiotemporal statistics derived from quadrat data are influenced greatly by quadrat size and initial epidemic conditions, even though within a fixed quadrat size under a particular set of initial conditions the effects of spore-dispersal gradient and quadrat shape can be large and significant. Thus, it is very important to select appropriate initial conditions in experiments for obtaining maximum information and to avoid using extreme long-shaped quadrats for sampling. Furthermore, the differences among the values of these spatial statistics reported from different studies may reflect, at least in part, differences in quadrat sizes and initial conditions and, to a lesser extent, differences in the spore-dispersal gradient.

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