Analysis of Epidemics Using Spatio-Temporal Autocorrelation

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ABSTRACT

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The theory of spatio-temporal (ST) autocorrelation analysis is summarized. Methods of epidemic analysis that are specific to agricultural systems in which temporal observations are made on disease values in a regular spatial lattice are presented. Techniques for calculating ST autocorrelations and partial autocorrelations are given; these statistics are then used in identifying the order of autoregressive and moving average terms that should be included in an ST transfer function that describes epidemic development in time and space. Spread of leather rot of strawberries is used as an example.

Recent reviews of the analysis of spatial pattern of plant disease reflect a growing sophistication in the approaches employed by epidemiologists for understanding disease development as a spatial process (5,12,18). The latter reviews provide a thorough treatment of the analysis of spatial point patterns, and it is not our intent here to recapitulate those observations, other than to note, by way of introduction, that such analyses provide only limited, and often ambiguous, insights into the processes that have generated an observed spatial pattern. At least in part, this is because point pattern analyses generally do not conserve any information on location. This is not true of distance methods, but these have not found much practical use in plant pathology (19). The Moran I statistic (17) and other measures of spatial autocorrelation conserve the spatial information content of a field sampling scheme by making use of information on the location of each sample point (6,18). Nicot et al (18) have discussed the advantages of using the Moran I statistic to characterize the spatial pattern of disease. Noe and Campbell (19) used spatial autocorrelation to characterize the spatial pattern of several nematode species. Madden et al (13) have demonstrated how the use of spatial autocorrelation coefficients (16) calculated for a series of sample times can be used in conjunction with spatial point pattern statistics such as Lloyd's mean crowding and patchiness indices (22) to characterize changes in spatial aggregation over time.

Ultimately, the goal of spatial analysis in epidemiology should be not to simply analyze and describe spatial patterns of disease, but to identify and specify the physical and biological mechanisms that account for the evolution of spatial patterns of disease over time. The analytical methods described here could be viewed as intermediate between the purely descriptive and purely mechanistic approaches in that spatio-temporal (ST) autocorrelation analysis is intended to identify models that can account for the evolution of observed spatial patterns as opposed to simply describing a pattern at a fixed point in time. The general class of models that are used to describe pattern evolution in time and space are known as spatio-temporal transfer functions (STFs). In general, an STF may be composed of temporally and spatially distributed inputs, system variables, and errors (definitions of these variables are presented subsequently). The goal of the statistical procedures of process identification is to determine the limits of significant temporal and spatial dependence between events in a plane, while the goal of process specification is the actual estimation of parameters of the STF (3). The present paper is limited to a discussion of the theory and techniques of identification. It is important to note that spatial autocorrelation analysis is not sufficient for process identification, since this technique is limited to the description of an observed pattern at a single point in time. For instance, one could look at how spatial autocorrelation changes over time (13), but nothing in the latter approach would suggest the manner in which one pattern might arise from another (3).

STF identification involves an analytical technique known as spatio-temporal autocorrelation (2,3,14). ST autocorrelation can best be thought of as an extension of spatial autocorrelation analysis (6) that incorporates elements of classical time series analysis (4). Although the analytical theory is well developed, programs needed to perform such analyses are highly complex and not generally available, so we have developed a PASCAL language program (STAUTO) for this purpose. We first describe some of the necessary theoretical background, and then present an example analysis.

PHYSICAL BASIS FOR ANALYSIS

Representation of sample areas. A field can be viewed as a regular lattice on the Cartesian plane, each of whose N elements represents a sampling area. Commonly, these sampling areas are contiguous quadrats. For notational convenience, the N elements are designated with a single subscript, i ($i = 1 \ldots N$), rather than with the double Cartesian coordinate subscript. Suppose that we have T observations on the lattice over time. Then the quantity of interest (say, number of diseased individuals) at time t in element i will be designated $y_{i,t}$.

Spatial and temporal proximity and the concept of neighborhood. Ultimately, we will define an ST autocorrelation statistic, but first it is necessary to explain the meanings of spatial and temporal proximity, and the sense in which a set of lattice elements j may be said to be neighbors of an element i. First, consider the purely spatial case of proximity, which is defined in terms of a spatial pattern selected by the investigator and a spatial lag order. Cliff and Ord (6) give a number of examples of spatial proximity patterns, the most common of which are analogues from the game of chess and are designated "rook," "bishop," and "queen" (Fig. 1). Many other patterns are possible, and additional types that are included in STAUTO are also illustrated (Fig. 1). First- and second-order spatial lags are illustrated for each of the above patterns (Fig. 1). Extension of the definition of spatial proximity for each pattern to higher-order spatial lags is straightforward.

In strict analogy with the conventional correlation statistic, we are interested in the degree to which the value of some variable

(say, disease incidence) in element i covaries with values observed in elements at varying distances from i. To demonstrate, let j be the set of elements that satisfies the spatial requirements of the particular proximity pattern being considered for a given element i (Fig. 1). Then, for a given spatial lag s, we can define the spatial lag operator $L^s y_{i,t}$:

$$L^s y_{i,t} = \sum_{h \epsilon j} (y_{h,t}) / n_j^s \quad (1)$$

in which j denotes the set of elements that lie at lag distance s from element i, n_j^s denotes the number of elements in j at lag s, and the notation $h \epsilon j$ indicates that the summation is over all elements h that belong to the set j. For example, in the case of the rook's definition and given $s = 1$, the lattice elements included in the summation (Eq. 1) would be those immediately adjacent to element i in the same row or column (Fig. 1). These same elements are also said to be first-order spatial lag neighbors of i. Thus, neighborhoods of element i are defined with respect to a specific spatial order of lag distance, and we will refer to lattice elements satisfying the distance and pattern criteria as s-order spatial neighbors of element i.

Clearly, there must be some rational basis for selection of a particular pattern. Ideally, the choice of pattern should be based on the investigator's prior knowledge of how spatial orientation of lattice elements influences the ability of element pairs to interact. If there was no physical basis for assuming the presence of a directional bias, then a symmetrical pattern would be a logical choice (e.g., rook, bishop, queen, or square). Alternatively, if, say, wind direction exerted a strong influence on lattice element interactions, then one-sided patterns such as the within- or across-row patterns or analgous variants of the bishop pattern might be appropriate. It may not be possible to select a particular pattern *a priori* from within a subset of patterns that represents a logical choice. However, in such a case, one may use the objective criterion of selecting that pattern which maximizes the overall level of autocorrelation.

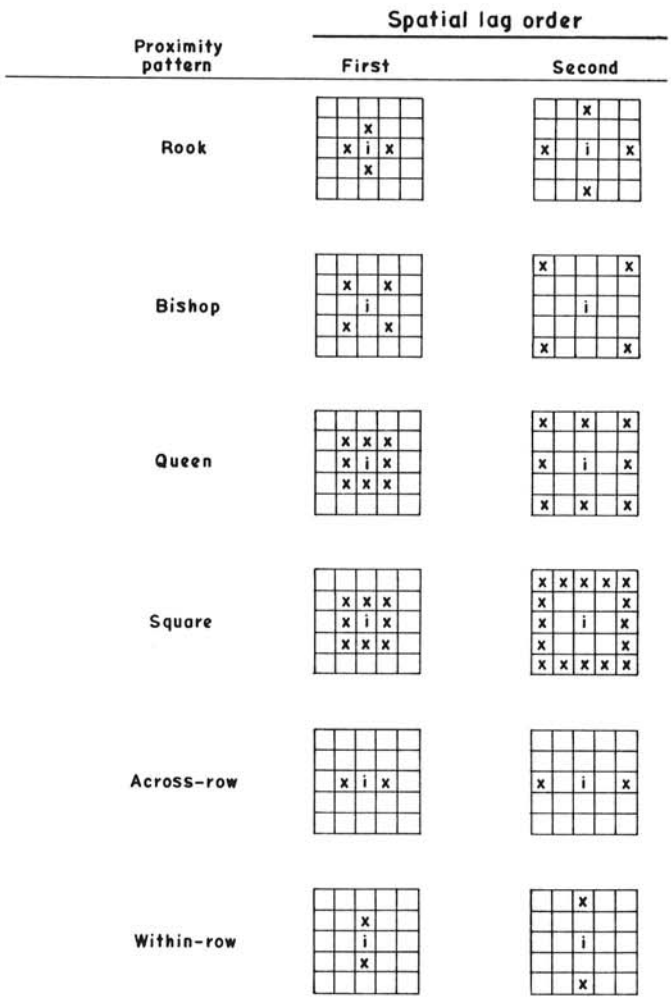

Fig. 1. Examples of spatial proximity patterns for first- and second-order spatial lags. For each pattern, examples of first- and second-order lags are illustrated. *i* indicates an arbitrary element of the lattice, and the *x*'s indicate neighbors for the pattern at the indicated lag. Lattice columns in the figure correspond to crop rows.

In addition to the problem of specifying which elements are neighbors of element i at any given spatial lag s (Eq. 1), there is also the matter of how the effects of neighbors at a particular distance s should be weighted. The choice of weight that is assigned to the set of neighbors j of lattice element i should reflect the investigator's knowledge of how disease is influenced by underlying physical processes such as propagule dispersal. For instance, knowledge of the dispersal process involved in a particluar disease might suggest the use of inverse lattice distance as the weighting measure employed in the case of a large-spored, aerially dispersed pathogen, whereas the square of the inverse distance might be appropriate in the case of a pathogen that is spread by rain splash. In the geography literature, binary neighbor weights have commonly been used (6), in which case no *a priori* weighting based on distance is assumed. By binary weight, we mean that the weighting function assumes a value of 1 for lattice elements that are s-order lag neighbors of element i, and a value of 0 otherwise. Binary weights are the best choice when no logical basis for assigning weights as a function of lag distance exists. However, in the context of pathogen dispersal within a field that is governed by physical processes, weighting by inverse distance or inverse of squared distance may often be a sensible alternative to binary weights. The generalized spatial lag operator, which includes a weighting effect is then given by:

$$L^s y_{i,t} = w_s \sum_{h \epsilon j} (y_{h,t}) / n_j^s \quad (2)$$

in which w_s is the weight applied to the neighbors of element i at spatial lag s.

The spatial lag operator (Eq. 2) can be further generalized to include simultaneous spatial and temporal lags:

$$L^s y_{i,t-k} = w_s \sum_{h \epsilon j} (y_{h,t-k}) / n_j^s \quad (3)$$

in which $y_{i,t}$ and $y_{h,t}$ have been replaced by $y_{i,t-k}$ and $y_{h,t-k}$, respectively, and k is the temporal lag order ($L^s y_{i,t-k}$). Hereafter, all references to the lag operator are with respect to equation 3 unless noted otherwise.

THE GENERAL CLASS OF SPATIO-TEMPORAL TRANSFER FUNCTIONS

The general form of the STF expresses the system variable $y_{i,t}$ (e.g., disease incidence in lattice element i at time t) as a function of three basic variable types, each of which may be spatially and temporally lagged with respect to $y_{i,t}$. System inputs are variables that are external to the system and which are not influenced by the system (e.g., weather variables). Inputs will be spatially distributed if the geographic scale of the model is suitably large. For example, weather inputs to an STF for development of stripe rust in the major wheat-producing states of the central United States would certainly be spatially distributed, since each lattice element would be on the order of 10^6 hectares. However, if the STF is intended to describe epidemic development of stripe rust in a single field, then weather inputs to the lattice elements can be considered to be fixed at a given time t.

In addition to system inputs, the STF may also include temporally and spatially lagged values of the system variable ($L^s y_{i,t-k}$), and these represent the autoregressive component of the STF. An STF may also include temporally and spatially lagged error terms ($L^s \xi_{i,t-k}$), which represent the moving average component of the model. The errors are often thought of as unknown external inputs and are frequently referred to as

disturbance terms, since the error terms account for the combined effects of all external inputs that are not explicitly accounted for in the vector of known external inputs.

Autoregressive models. A pure ST autoregressive process of order l in space and m in time, or STAR(l, m) process, is given by:

$$y_{i,t} = \sum_{s=0}^{l} \sum_{k=1}^{m} \beta_{s,k} L^s y_{i,t-k} + \xi_{i,t} + \mu \quad (4)$$

in which $L^s y_{i,t-k}$ is defined as in equation 3; the $\beta_{s,k}$ are parameters that define $y_{i,t}$ as a linear function of the terms $L^s y_{i,t-k}$; and μ is a constant that often is considered equal to zero. The first summation is for spatial lags from 0 to l. The variable $\xi_{i,t}$ is the error term for the i-th element and t-th time and represents the difference between the observed and expected $y_{i,t}$. For example, a STAR(1, 1) model would be:

$$y_{i,t} = \beta_{0,1} y_{i,t-1} + \beta_{1,1} L^1 y_{i,t-1} + \xi_{i,t} \quad (5)$$

since, by convention, $w_0 = 1$ is usually assumed, and $L^0 y_{i,t-1}$ is simply the variable value in i. To make the meaning of equation 5 clear, suppose that $y_{i,t}$ represents disease incidence in quadrat i at time t. Then, equation 5 states that disease incidence in element i at time t depends on the disease incidence observed in that same element at time $t-1$, and also depends on disease incidence in its first nearest neighbors (Fig. 1) at time $t-1$.

Moving average models. A pure ST moving average process of order p in space and q in time, or STMA(p, q) process is given by:

$$y_{i,t} = \sum_{s=0}^{p} \sum_{k=1}^{q} \gamma_{s,k} L^s \xi_{i,t-k} + \xi_{i,t} + \mu \quad (6)$$

in which $L^s \xi_{i,t-k}$ represents the temporally and spatially lagged error terms (analogous to Eq. 3), and μ is a constant equal to the mean of the $y_{i,t}$. The error terms are each assumed to be normally distributed and to have an expectation = 0, constant variance, and to be mutually uncorrelated; additionally, $\xi_{i,t}$ is uncorrelated with $y_{i,t}$. As an example, an STMA(1, 1) model would have the form:

$$y_{i,t} = \gamma_{0,1} \xi_{i,t-1} + \gamma_{1,1} L^1 \xi_{i,t-1} + \xi_{i,t} \quad (7)$$

in which we have again assumed that $w_0 = 1$, and that $L^0 \xi_{i,t-1}$ is simply the value of ξ in element i. The interpretation of equation 7 is strictly analogous to that for equation 5 except that predicted disease incidence in element i at time t is now based on the observed errors in the neighbors of element i, rather than on the disease incidence in these neighbors. The inclusion of moving average terms in a model accounts for the composite effect of any external factors that influence system behavior, but that are not explicitly included in the model. Notice that the sources of error are partitioned into a mean effect at time t (μ), as well as effects due to each combination of a spatial lag s and temporal lag k. It has been shown that low-order STMA models are equivalent to STAR models of infinite order, so that STMA models may often provide a more parsimonious model representation (3). Note also that the terms of equation 6 are random variables, so that STMA models are stochastic, whereas STAR models are deterministic, except for the error term.

Mixed models. Assuming that there is only one system input, the general STF model may be written as:

$$y_{i,t} = \sum_{s=0}^{u} \sum_{k=1}^{v} \alpha_{s,k} L^s x_{i,t-k} + \sum_{s=0}^{l} \sum_{k=1}^{m} \beta_{s,k} L^s y_{i,t-k} + \sum_{s=0}^{p} \sum_{k=1}^{q} \gamma_{s,k} L^s \xi_{i,t-k} + \xi_{i,t} + \mu \quad (8)$$

in which $L^s x_{i,t-k}$ represents the effect of temporally and spatially distributed inputs (analogous to Eq. 3), and $L^s y_{i,t-k}$ and $L^s \xi_{i,t-k}$ are as previously defined (Eqs. 4 and 6, respectively). When the $\alpha_{s,k} = 0$, but both the $\beta_{s,k}$ and $\gamma_{s,k}$ have nonzero terms for some s and k, the STF is known as an ST autoregressive moving average (STARMA) model. If the $\alpha_{s,k}$ are also nonzero for some s and k, considerable problems occur in model identification, since the $x_{i,t}$ will generally be correlated with each other as well as with the $y_{i,t}$. Fortunately, as discussed above, for many epidemiological applications, it will often be the case that the $x_{i,t}$ = constant for all i at a given time t, so that we can consider the somewhat simpler modified STARMA model:

$$y_{i,t} = \sum_{k=1}^{v} \alpha_k x_{t-k} + \sum_{s=0}^{l} \sum_{k=1}^{m} \beta_{s,k} L^s y_{i,t-k} + \sum_{s=0}^{p} \sum_{k=1}^{q} \gamma_{s,k} L^s \xi_{i,t-k} + \xi_{i,t} + \mu \quad (9)$$

As a further simplification, x_t can be reduced to a constant and not separable from μ. The analytical problem of model identification that we are concerned with in this paper involves determination of the order of both the autoregressive and moving average components for any process that can be represented by equation 9 using the methods of spatio-temporal autocorrelation analysis.

THE SPATIO-TEMPORAL AUTOCORRELATION COEFFICIENT

Calculation. A simple form of the estimate of the ST autocorrelation at spatial lag s and temporal lag k is given by Bennett (3) and Martin and Oeppen (14), and which will be designated $r_{s,k}$. However, the definition of $r_{s,k}$ as an ST autocorrelation statistic leads to some major theoretical problems (3,11). Martin and Oeppen (14) proposed a solution to these problems. Although the required computational algorithm is considerably more complex than that for $r_{s,k}$, the proposed statistic provides the most general solution for estimating the partial autocorrelations (see below). The elements of the autocorrelation matrix **R** are calculated as:

$$r_{h,j,s,k} = \frac{\sum_{t=v+1}^{T} \sum_{i=1}^{N} (L^h y_{i,t-j} - \bar{y})(L^s y_{i,t-k} - \bar{y})}{\left[\sum_{t=v+1}^{T} \sum_{i=1}^{N} (L^h y_{i,t} - \bar{y})^2\right]^{1/2} \left[\sum_{t=v+1}^{T} \sum_{i=1}^{N} (L^s y_{i,t} - \bar{y})^2\right]^{1/2}} \quad (10)$$

in which $r_{h,j,s,k}$ is the autocorrelation between spatial lag h at time j and spatial lag s at time k, L^s is the spatial lag operator defined in equation 3, $v = \max(j, k)$, and $\bar{y}$ is the grand spatio-temporal mean which is calculated as: $\bar{y} = \Sigma\Sigma y_{i,t} / NT$. The first row and column of **R** contain the ST autocorrelation function $r_{s,k}$. In addition, **R** is symmetric, and contains 1's on the diagonal.

The number of significant temporal and spatial lag orders of the autoregressive and moving average components of an STF can be judged in part from statistical tests of the ST autocorrelations and partial autocorrelations. Bennett (2,3) has warned that patterns in the autocorrelogram are affected by residual autocorrelations and multicollinearity, and that interpretation should be based on a careful examination of both the autocorrelogram and partial autocorrelogram. Detailed discussion on the interpretation of these patterns can be found in Bennett (3). For a correlation matrix of the form of **R**, Kendall and Stuart (11) define the partial autocorrelation between $y_{i,t}$ and $L^s y_{i,t-k}$ as:

$$\psi_{0,0,s,k} = -C_{0,0,s,k} / \left[(C_{0,0,0,0})(C_{s,k,s,k})\right] \quad (11)$$

in which $C_{s,k,s,k}$ is the cofactor of $r_{s,k,s,k}$ in the determinant of **R**.

Interpretation of the autocorrelogram. The pattern of autocorrelations (autocorrelogram) and partial autocorrelations (partial autocorrelogram) assume characteristic forms, depending on the type and order of STARMA process involved (2,3,14). For a purely autoregressive process of order l in space and m in time (STAR[l, m]), the autocorrelations will decay approximately exponentially (or in a damped exponential-sinusoidal pattern) over spatial and temporal lags, whereas the partials will be cut off (as opposed to trailing off gradually) after l lags in space and m lags in time. In contrast, for a pure moving average process of order p in space and order q in time (STMA[p, q]), the autocorrelations will be cut off after p lags in space and q lags in time, while the partials will decay exponentially. In the case of a mixed model (STARMA [l, m, p, q]), the autocorrelations begin to decay exponentially after the first $p-l$ lags in space and $q-m$ lags in time, while the partials

begin to decay after the first $l - p$ lags in space and $m - q$ lags in time.

However, it should be noted that the patterns we have described are, in effect, idealizations, and that, in general, the pattern may not always be so easily interpreted (3). The mixed STARMA model can be particularly difficult to identify when the maximum number of temporal and spatial lags is limited. Nevertheless statistical tests derived from time series analysis have been suggested as an aid to the interpretation of spatio-temporal autocorrelograms (3).

A test of the significance of the autocorrelations was derived by Box and Jenkins (4) from a simplified form of Bartlett's (1) formulae for the variance and covariance of the estimated autocorrelations. Jenkins (10) suggested using Quenouille's (20) formula for the variance of the estimate of the partial autocorrelations. Because the distributions of the estimates of the ST autocorrelations and partial autocorrelations are asymptotically normal, the significance of the estimates are usually tested with the familiar standard normal (z) statistic (10). However, it is important to note that tests conducted on the autocorrelations are tests of the significance of the moving average terms, while tests on the partials are tests of the significance of the autoregressive terms.

STATIONARITY REQUIREMENTS

An additional consideration that is related to the interpretation of pattern in autocorrelograms is the matter of system stationarity. A statistic (or function) is said to be stationary if the values (or functional form) are time invariant. Strict application of the methods of spatio-temporal autocorrelation requires that the system under study be stationary in order for model parameter estimates to have a meaningful physical interpretation (3). As will be seen below, nonstationarity will generally be the rule in plant pathosystems, so it is necessary to be able to recognize the symptoms of nonstationarity and take corrective steps where needed. Three sources of nonstationarity need to be considered. These are discussed in great detail by Bennett (3), but we summarize them briefly here.

Nonstationarity of level. Epidemiological systems are typically characterized by strong temporal trends in mean level of disease within a field as evidenced by the common use of the logistic or monomolecular models to represent disease progress. Furthermore, diseases with a strong focal character will exhibit strong spatial trends across a field at any given time t. Nonstationarity of level is indicated by a slow decline in autocorrelations with increasing lags. A basic assumption of the analytical methods we are describing is that the expected value of $y_{i,t}$ is the same for all i and t, or, equivalently, that the of y process is homogeneous and isotropic. Fortunately, temporal and spatial trends are generally easy to recognize and can be removed by temporal, spatial, or simultaneous temporal and spatial differencing. First-order temporal, spatial, and simultaneous temporal-spatial differencing are defined, respectively, by:

$$\nabla_T y_{i,t} = y_{i,t} - y_{i,t-1} \tag{12a}$$

$$\nabla_S y_{i,t} = y_{i,t} - L^1 y_{i,t} \tag{12b}$$

$$\nabla_{ST} y_{i,t} = y_{i,t} - y_{i,t-1} - L^1 y_{i,t} + L^1 y_{i,t-1} \tag{12c}$$

in which ∇_T, ∇_s, and ∇_{ST} are the respective difference operators. Any of the three types of differencing can be invoked by STAUTO, so that the required transformations can be easily implemented when needed.

Nonstationarity of variance. Problems similar to those noted above may also arise with respect to $Var[y_{i,t}]$. In these circumstances, a suitable transformation of $y_{i,t}$ is usually all that is required. Our program includes seven transformation options, including those for the logistic and monomolecular among others.

Nonstationarity of process. This form of nonstationarity occurs when the model parameters are themselves functions of time or some other ignored system quantity. Our experience with analysis of epidemiological systems has led us to conclude that most instances of nonstationarity in such systems can be handled by the methods already described, and that any persistent nonstationarity is most likely attributable to nonstationarity of process, in which case it may be possible to partition the data set into two or more stationary ones.

AN EXAMPLE OF SPATIO-TEMPORAL AUTOCORRELATION ANALYSIS

Description of the system. Epidemic development of leather rot of strawberry (*Fragaria* × *ananassa* Duch. 'Midway'), caused by *Phytophthora cactorum* (Leb. & Cohn) Schroet., was monitored in six field plots near Wooster, OH, in spring 1986. Each plot was 2 m long and three rows wide. Both sides of each row in a plot were divided into ten 20-cm-long quadrats, and the five flower cymes closest to the row edge were tagged in each quadrat. Thus, one plot constituted a 6 × 10 lattice. As an example of the analytical methods, preliminary results are presented for one of the plots.

Monitoring of epidemic development. Because leather rot of strawberry is spread by rain splash (21), assessments of epidemic development were made 5 days (approximate length of latent period) after a rain event, rather than at regular chronological intervals. At each assessment date, the number of tagged cymes bearing one or more berries infected with *P. cactorum* was recorded. Assessments were made for a series of 10 rain events, by which time incidence of cyme infections approached 100%.

Results of spatio-temporal autocorrelation analysis. Epidemic development in the example plot was characterized by a sigmoid curve, so the logistic transformation was applied to the data using Haldane's (9) correction for the proportion of disease when incidence equaled 0 or 1, and an analysis was performed on the nondifferenced data using binary weights and the rook's definition of spatial proximity. Aspatial summary statistics output by the program showed a strong temporal trend in mean disease level for both raw and transformed disease incidence as would be expected (Table 1). Notice that a strong trend in the variance also is apparent for both raw and transformed data (Table 1).

The autocorrelogram and partial autocorrelogram for nondifferenced data are presented for a maximum spatial lag of $s = 3$ and a maximum temporal lag of $k = 3$ (Table 2). The autocorrelogram indicates that disease incidence among neighbors is highly correlated with disease incidence in lattice element i at time t for all spatial lags and for temporal lags $\leqslant 2$ ($P \leqslant 0.001$, Table 2). The slow decline in autocorrelations over spatial lag is a strong indicator of a nonstationary spatial process and is consistent with the focal nature of epidemic development in this system. Although the temporal autocorrelations decay much more rapidly than the spatial autocorrelations, the general pattern here

TABLE 1. Aspatial statistics for leather rot development in strawberry for raw data and for analysis using the logit transformation, binary weights, the rook's definition of spatial proximity, and no differencing

Sample sequence	Raw data		Tranformed data[b]	
	Mean[a]	Variance	Mean	Variance
1	0.33	0.29	−1.98	0.42
2	0.40	0.41	−1.92	0.52
3	0.43	0.41	−1.88	0.54
4	0.57	0.48	−1.72	0.59
5	0.77	0.71	−1.51	0.74
6	0.93	0.96	−1.34	0.94
7	1.52	1.55	−0.84	1.23
8	2.47	3.02	−0.08	2.40
9	2.93	3.83	0.34	3.19
10	3.13	4.12	0.57	3.50

[a] Means for raw data are for the mean number of strawberry cymes bearing at least one infected fruit out of a possible maximum of five cymes per quadrat. Each plot consisted of a 6 by 10 lattice of quadrats.

[b] Raw data transformed to logits based a possible maximum of five cymes per quadrat.

also is suggestive of a nonstationary process, a fact readily confirmed by the aspatial statistics (Table 1). In contrast to the high autocorrelations seen in the correlogram, the partial autocorrelations for nondifferenced data indicate that only first-order autoregressive temporal effects at 0 and 1 spatial lags are significant, suggesting a STAR(1, 1) model. However, as we have indicated previously, the coefficients of a model based on the analysis of nonstationary data are not readily interpretable in terms of real physical processes.

We also present results for the same data set, but using temporal differencing and inverse distance weights (Tables 3 and 4). The logistic transformation and the rook's definition of spatial proximity have been maintained in this analysis. Temporal differencing of the data removed most of the temporal trend in mean disease incidence as well as the nonstationarity of variance (Tables 3 and 4). Use of inverse distance weighting probably represents a conservative estimate of the effect of distance on the epidemic process in our example. Nonetheless, the combination of temporal differencing and inverse distance weighting appear to remove most evidence of nonstationarity (Table 4), so that there appears to be no need to employ simultaneous spatial and temporal differencing in this instance. The patterns seen in the autocorrelogram and partial autocorrelogram indicate that the generating process for this leather rot epidemic might be modeled as a STARIMA(1, 1, 1, 1) process (when differencing has been employed the model is known as an ST autoregressive integrated moving average, or STARIMA model). Note, however, that at one temporal lag, the zero-order spatial lag is nonsignificant for both the moving average process (autocorrelogram) and the autoregressive process (partial autocorrelogram). Consequently, for the temporally differenced data, the parameters $\beta_{0,1}$ and $\gamma_{0,1}$ are both equal to 0, so that the model can be written as:

$$\nabla_T y_{i,t} = \beta_{1,1} L^1 (\nabla_T y_{i,t-1}) + \gamma_{1,1} L^1 (\nabla_T \xi_{i,t-1}) + \xi_{i,t} \qquad (13)$$

in which ∇_T is the temporal difference operator (Eq. 12a).

TABLE 2. Spatio-temporal correlogram and partial correlogram for data transformed by logits, and using binary weights, the rook's definition of spatial proximity, and no differencing

Temporal lag order	Autocorrelations[a] Spatial lag order 0	1	2	3
1	0.88***	0.75***	0.65***	0.59***
2	0.68***	0.55***	0.45***	0.39***
3	0.40***	0.25*	0.20*	0.16
Temporal lag order	**Partial autocorrelations Spatial lag order 0**	**1**	**2**	**3**
1	0.64***	0.27***	0.07	0.11*
2	−0.00	−0.10*	−0.06	−0.09
3	−0.07	−0.03	−0.00	0.03

[a] *, **, and *** indicate that the estimate is significantly different from 0 at $P = 0.05$, $P = 0.01$, and $P = 0.001$, respectively.

TABLE 3. Aspatial statistics for leather rot development in strawberry using the logit tranformation, inverse distance weights, the rook's definition of spatial proximity, and temporal differencing[a]

Sample sequence	Mean	Variance
2	0.06	0.05
3	0.04	0.05
4	0.16	0.18
5	0.20	0.23
6	0.17	0.17
7	0.50	0.52
8	0.76	0.92
9	0.42	0.84
10	0.22	0.32

[a] Counts of cymes per quadrat bearing at least one infected fruit per cyme were transformed to logits based on a possible maximum of five cymes per quadrat. Temporal differencing was performed on the logit-transformed data (Eq. 12a in text). Means are for the resulting mean differences in the logit of disease incidence.

DISCUSSION AND CONCLUSIONS

Equation 13 indicates that the change in disease incidence, as represented by change in logits, in lattice element i at time $t(\nabla_T y_{i,t})$ depends on several factors. First, as might well be anticipated, change in disease incidence in element i at time t is dependent on the previously observed value of disease incidence in i. Also, the spatial dependence of disease incidence in element i on change in disease incidence in surrounding elements does not extend beyond the first nearest neighbors of element i, and this spatial dependence only extends back to time $t-1$ (Eq. 13). The low spatial lag order of the model (Eq. 13) is consistent with the observation that infection gradients of splash-dispersed organisms are characteristically very steep (15). Furthermore, a maximum temporal lag order of 1 was expected, since spread of leather rot occurs in discrete, easily recognizable events, and the timing of disease assessments could be determined with a high degree of accuracy.

In the case of pathogens whose propagules are liberated by wind, the problem of selecting an appropriate sampling interval will be more complex. Geographers, who have analyzed the spread of various diseases in human populations, have used the disease incubation period as the sampling interval (6–8). In the case of plant disease epidemics, the latent period of a disease might serve as an appropriate sampling interval. The selection of an appropriate sampling interval is important, since an interval that is too small relative to the effective rate of the epidemic process will cause the values of temporal autocorrelations to remain artificially high (14). On the other hand, if a sampling interval is selected that is too large, temporal dependencies that actually exist may not be reflected in the resulting autocorrelogram.

The moving average component of equation 13 also is of order 1 in space and time. The occurrence of a significant moving average term in equation 13 may have been due to the fact that weather factors that might have affected epidemic development were not included in this example analysis. For example, the inclusion of moving average terms in equation 13 may indicate that the observed pattern of leather rot development was partly the result of local processes such as inoculum dispersal that depend on the degree of flooding in individual quadrats. Variation in such factors as the degree of local flooding would be difficult to quantity, and are probably best accounted for as moving average (e.g., error) terms. The full STARIMA(1, 1, 1, 1) model would then indicate that local dispersal (moving average process) was superimposed on a planar process (autoregressive process).

TABLE 4. Spatio-temporal correlogram and partial correlogram for data transformed by logits, and using inverse distance weights, the rook's definition of spatial proximity, and temporal differencing

Temporal lag order	Autocorrelations[a] Spatial lag order 0	1	2	3
1	0.05	0.26**	0.04	0.00
2	0.03	0.07	−0.06	−0.12
3	−0.05	−0.1	−0.10	−0.11
Temporal lag order	**Partial autocorrelations Spatial lag order 0**	**1**	**2**	**3**
1	−0.04	0.22***	0.04	0.07
2	−0.03	0.04	−0.04	−0.08
3	−0.04	0.05	−0.00	−0.01

[a] **, and *** indicate that the estimate is significantly different from 0 at $P = 0.01$, and $P = 0.001$, respectively.

ST autocorrelation analysis is a potentially powerful tool, but there are difficulties associated with its use because the selection of an appropriate proximity pattern and distance weighting criterion is generally not a trivial matter. The rook proximity pattern and binary weights are perhaps the most commonly used assumptions in the absence of definitive data, and their widespread use and acceptance within geography have made them standards (6). The attractiveness of the rook pattern lies in its simplicity and symmetry. As suggested earlier, the presence of prevailing winds may indicate the need for an across- or within-row, or even a diagonal pattern.

Our program, STAUTO, also includes provision for differential weighting of neighbors included in a lag operator summation (Eq. 3). Differential weighting is achieved by specifying a "barrier effect," which operates across lattice columns (which are assumed to represent crop rows). Crop row barrier effects can be specified in STAUTO by defining a barrier effect for each pair of adjacent crop rows. The specification of barrier effects in an analysis involves an extension of the equation for lagged neighbor summation (Eq. 3) so that it is possible to account for differences in pathogen dispersal within as opposed to across rows. With respect to equation 3, barrier effects are specified as:

$$L^s y_{i,t-k} = w_s \sum_{h \epsilon j} (b_{r,s} y_{h,t-k}) / n_j^s \qquad (14)$$

in which $b_{r,s}$ is the barrier effect for rows r and $r+1$ at spatial lag s, and other terms are as defined in equation 3. Barrier effects are specified for each adjacent pair of rows r and $r+1$, and these effects are assumed to be multiplicative across rows. For example, if the barrier effect across rows 1 and 2 is 0.5, and the effect across rows 2 and 3 is 0.4, then at spatial lag $s = 2$ the total effect across rows 1 and 3 would be 0.2. Note that in STAUTO, $b_{r,s}$ is always defined to be 1 for an element j that occupies the same data column (crop row) as element i. Use of a barrier effect might be considered in the analysis of an epidemic involving a row crop in which the rows present significant barriers to propagule dispersal. When the barrier effect option is combined with the distance weighting option in the program, relatively complex patterns of weights can be specified.

When two or more alternative patterns appear to be equally feasible on *a priori* grounds, the best choice would appear to be to select that pattern that produces the highest levels of ST autocorrelation in the lower-order spatial and temporal lags. Such an approach amounts to using an objective criterion as the basis for pattern selection. In our experience, the interpretation of ST autocorrelograms is not drastically different when results based on similar proximity patterns are compared. For instance, the square pattern is only a minor modification of the queen pattern, and results with these two patterns are usually quite similar. In contrast, the results obtained when using the rook pattern will often be quite different from the two latter patterns. The rook pattern does not include those elements j that fall on a 45 degree diagonal from element i. If the values of diagonal elements are poorly correlated with those in element i, then the rook pattern will yield generally higher ST autocorrelations. Similarly, the bishop pattern usually yields very different ST autocorrelograms from any of the above three patterns.

Binary distance weighting should be preferred in the absence of independent evidence to the contrary, but it should be noted that this weighting criterion will almost always produce higher ST autocorrelations than inverse distance weighting or similar schemes. However, the selection of binary weights, when, in fact, inverse distance weighting ought to have been used, does not pose significant problems for parameter estimation in the subsequent model specification stage, since, in the course of model fitting, parameter estimates will automatically be adjusted accordingly. At the worst, one might tend to overestimate the significance of higher-order spatial lags when using binary weighting. Consequently, we would suggest that higher-order spatial lag terms that are only marginally significant be ignored when binary weights are used. In connection with weight selection, it should also be noted that weights can only be selected to a level of scale. That is, the weight vector (1.0, 0.5, 0.25), corresponding to weights at spatial lags $s = 0$, $s = 1$, and $s = 2$, respectively, is identical in effect to the weight vector (0.5, 0.25, 0.125). Thus, the arbitrariness of defining $w_0 = 1.0$ is more apparent than real.

Throughout this paper we have suggested the use of a number of analytical operations, some of which reduce the level of ST autocorrelation apparent in the data, whereas others have the opposite effect. For instance, temporal and/or spatial differencing typically remove much of the autocorrelation, whereas the criterion for pattern selection is improvement in autocorrelation. Also, binary distance weighting will typically yield higher autocorrelations than other weighting schemes. The rationale for each effect can be summarized as follows. If there is strong theoretical or empirical evidence for a particular form of distance weighting, then the weighting should be applied *a priori* without regard to its effect on the level of spatial autocorrelations. The next major consideration is whether or not the data show evidence of nonstationarity as indicated by the failure of autocorrelations to decline rapidly over space and time, since a basic assumption of the analysis is that the spatio-temporal series is stationary. Epidemiological data will frequently require at least temporal differencing, since most studies deal with epidemics that are nondecreasing. If a weighting scheme other than binary weights is used, temporal differencing alone may be sufficient to achieve stationarity in the data. Ideally, the specification of barrier effects and proximity pattern also would be based on *a priori* considerations. However, practically, an investigator will frequently not have the necessary data to support strongly one particular alternative over a number of other closely related ones. In such circumstances, the objective criterion of maximizing autocorrelations with the emphisis on lower-order ones, seems logical.

Spatio-temporal autocorrelation analysis represents an important new approach to the study of spatial pattern in plant disease epidemiology. It is distinct from previous analytical methods in that the logical endpoint of analysis is not simply a statistical description of the observations, but rather a model that summarizes the spatial and temporal dependencies in the data, and therefore a model that can account for the evolution of observed patterns. Although such models are not mechanistic in the strict sense, they contain clear implications for spread mechanisms.

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