Letter to the Editor

A Simple Analysis of Disease Foci

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This research was supported in part by the U.S. Department of Agriculture under CSREES, special grant agreement 93-34135-8696, managed by the Pacific Basin Administrative Group (PBAG).
This is journal series 4144 of the Hawaii Institute of Tropical Agriculture and Human Resources.
I thank R. Berger, University of Florida Department of Plant Pathology, and P. Rodriguez de Gonzalez, University of Hawaii Department of Plant Pathology, for the use of their data on citrus variegated chlorosis and zucchini yellow mosaic, respectively. I also thank Y. Xia and V. Sanaka, University of Hawaii Department of Information and Computer Science, for their assistance with computer programming.
Accepted for publication 20 December 1995.

This letter was written to articulate a problem for nonepidemiologists, to propose a possible solution, and to discuss applications of the solution. The function of this letter is to advocate for simplicity in spatial data analysis, to describe a simple and effective alternative to more complex forms of analysis, and to encourage the study of epidemics as populations of disease foci, rather than as populations of diseased individuals.

The problem. Much of quantitative epidemiology is not very straightforward and appears to consist in obscuring the results of simple investigations in sophisticated analyses. This type of criticism certainly can be directed at the area of spatial pattern analysis. Some forms of analysis are too complex for most to understand. They fail to simplify data. The problem lies with the idea that if something is relatively new and complex, then it must be better.

At a basic level, the objectives of spatial analysis of presence/absence data for epidemics are straightforward. Investigators first try to detect departure from spatial randomness, and then questions concerning aspects of pattern are asked (e.g., cluster or focus size, shape, number, and orientation of axes within the lattice). Some methods of pattern analysis lack the simplicity and forthrightness of these objectives. The complexity of some techniques (i.e., statistical theory, mathematics, and interpretation), the obliqueness of the results, and the largely undeveloped links to disease management applications are factors that render these techniques difficult to use, understand, and apply to disease management. Inevitable consequences are the confusion of the casual reader or nonepidemiologists and the relative inaccessibility of techniques to those outside a small circle of epidemiologists.

A trend in botanical epidemiology is the increasing sophistication and complexity of spatial data analysis technologies. During the past decade, a group of relatively sophisticated spatial/spatiotemporal analysis methods has emerged. Among the methods are spatiotemporal autocorrelation (14) and geostatistics (3,15). These methods are based upon relatively advanced statistical theory as compared with simpler forms of analysis (mapping, quadrat variance methods, discrete probability distributions, and indices of dispersion) (2). This inherent complexity and sophistication renders these methods intimidating and/or inaccessible to many researchers who wish to conduct epidemiological analysis of their data. In addition to an increase in theoretical complexity, technical jargon also has proliferated, as with two-dimensional and spatiotemporal distance class analyses (5,10,13). Understanding and interpreting these analyses and keeping up-to-date with their glossaries have become more challenging.

One objective of data analysis is to reduce the complexity of data and organize data into a clear and meaningful system. Some forms of spatial pattern analysis fail to accomplish this, and may even amplify the complexity of data. Two-dimensional distance class analysis (2DCLASS) is a case in point. For example, a distance class matrix is often as spatially complex as its mapped data. And, with its relative lack of statistical rigor, a proliferating jargon, and several arbitrary decision guidelines, it is not an extremely precise method for hypothesis testing. Aspects of distance class matrices (e.g., core and reflected clusters) are used as indirect evidence for quantitative aspects of actual disease foci (e.g., cluster number, size, shape, and orientation) (11). This evidence is indirect and oblique because the matrix is only an image of the mapped data superimposed upon random scenarios, and not the actual mapped data. Although the method has merit, the interpretive nature of the decision criteria and the roundabout way of arriving at cluster attributes invite misuse and tenuous and potentially inaccurate conclusions.

In this communication, I offer a simple alternative to more complex methods of spatial pattern analysis. The method concerns the direct description and summary of disease foci, which, thereby, reduces the complexity of mapped data. The method is an extension of mapping. Its simplicity and utility are demonstrated in applications to disease data, and computer software for the analysis is made available. Before the method is described, a few related concepts are reviewed and clarified.

Definitions and assumptions. A disease "focus" is "a site of localized concentration of diseased plants or discrete lesions, either about a primary source of infection or coinciding with an area originally favorable to establishment, and tending to influence the pattern of further transmission of the disease" (1). Similarly, a disease "cluster" may be defined as a number of diseased plants grouped closely together. Thus, the two terms are synonymous when used in relation to plant disease epidemics. However, an important distinction is made herein between these two terms: a disease focus may consist of one diseased plant or unit, whereas a disease cluster contains more than one diseased plant or unit.

A concept of spatial proximity is borrowed here from the game of chess and from spatial lag correlation analysis of continuous data (4,9). The concept may be applied to defining a disease focus for binary data. The concept establishes a spatial proximity-distance criterion for diseased plants within a disease focus or cluster (an immediate proximity criterion). A diseased plant that is adjacent to another diseased plant (spatial lag = 1) and shares an edge (rook's case) or a corner (bishop's case) with that diseased plant in a lattice are considered to be part of the same disease focus (Fig. 1A).

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Publication no. P-1996-0202-02R

Within this concept, related variables may be defined and their values calculated to enhance the understanding of spatial dynamics. Focus "number" (N) is defined as the total number of disease foci in the matrix. Focus "size" (s) is defined as the number of diseased plants in a disease focus (each plant meeting the immediate proximity criterion). A spatially isolated, diseased plant has $s = 1$. Focus "dimensions" are defined as the maximum "row" (r) and "column" (c) distances spanned by a focus. A "proximity index" (PI) may be used as an indication of compactness of focus organization. The index, calculated as (s/rc), is borrowed here from spatiotemporal distance class analysis, in which values for PI were used to characterize the compactness of the core cluster (10). A high frequency of rook's case spatial connections results in a relatively high PI in relation to a high frequency of bishop's connections among plants. Thus, PI varies inversely with compactness. Examples are provided to illustrate the calculation of N, s, r, c, and PI (Fig. 1B).

Two assumptions underlie this simple model. First, it is assumed that binary data (presence/absence) are relevant simplifications of the system. Distances between rows and between plants within rows are assumed to be constant, although not necessarily equal.

A computer program with a recursive subroutine was written to find and describe disease foci in two-dimensional space. The program (FOCI) prints a map of the observed data, identifies the number of disease foci, and calculates s, r, c, and PI for each focus. Programming language is Microsoft Visual Basic for Windows. Input data sets are as described previously (13). Data from two pathosystems are presented to illustrate the use and application of the method.

Example 1: Citrus variegated chlorosis (CVC). Data were furnished by R. D. Berger, University of Florida, Department of Plant Pathology. The data were from an epidemic of CVC from 1989 to 1992 in a Brazilian citrus (*Citrus sinensis* (L.) Osbeck) orchard. Trees were cultivar Natal sweet orange on Cleopatra mandarin (*Citrus reshni* Hort. ex Tan.) rootstocks. Between-row spacing was 8 m, and within-row spacing of trees was 6 m. A sub set of the orchard (71 rows, 21 trees per row) was selected for analysis.

Yearly maps of CVC incidence show the accumulation of disease in the orchard from 1989 to 1992 (Fig. 2). The number of CVC disease foci increased linearly from 1989 to 1991 to approximately 80 foci, and then decreased from 1991 to 1992 by 66% as coalescence among CVC disease foci occurred (Fig. 3A). The reduction in focus number (coalescence) occurred when disease incidence increased from 22 to 46% (Fig. 3A). Values for mean PI also increased linearly (1989 to 1991) and then declined (0.75 to 0.65, 1991 to 1992, respectively) (Fig. 3B). Thus, as the number of disease foci increased (1989 to 1991), so did the relative compactness of focus organization. Conversely, as distinct foci coalesced into a larger disease focus, the organization of the resulting focus was less compact than that of the original, coalescing foci. Values for s remained relatively constant from 1989 to 1990, but increased significantly thereafter (1991 to 1992) during the period of presumed coalescence (Fig. 3B). A typical CVC disease focus was isodiametric from 1989 to 1992, as evident in the nearly identical values representing row and column dimensions for foci (Fig. 3C). A frequency distribution for s indicated that from 1989 to 1991 the most commonly observed disease focus size was 1 (Fig. 3D). No focus with $s > 4$ was observed for 1989 or 1990. In 1991 and 1992, the frequency of smaller foci decreased as larger foci formed because of coalescence of proximal disease foci.

Example 2: Zucchini yellow mosaic potyvirus (ZYMV). Data were available from experimental epidemics of ZYMV on zucchini squash (*Cucurbita pepo* L.) in 20-row by 20-column lattices on the island of Maui, Hawaii, in 1994 . Plant spacing was 1 m and row spacing was 1.3 m. These data were analyzed by spatiotemporal distance class analysis elsewhere (12).

Researchers wished to compare ZYMV epidemics with different spatial starting conditions. They started different epidemics (via inoculation) with the same number of inoculations (12 plants). Varying the configurations of inoculated plants resulted in the establishment of different numbers of disease foci between treatments. After inoculation, ZYMV was allowed to spread naturally. Data from two treatments were selected here, a uniform pattern (UP) and an aggregated pattern (AP) of initial disease (Fig. 4A and B, with 12 and 2 foci, respectively). Maps of disease incidence from seven disease assessment dates were prepared to illustrate the general spatiotemporal progress of ZYMV infection in each plot (Fig. 4A and B).

The progress curves for ZYMV-incidence were 'S'-shaped for the UP and the AP data (Fig. 5A). Higher levels for disease incidence were observed for UP at most dates than for AP (Fig. 5A). Values for final disease incidence for AP and UP were 0.73 and 0.91, respectively (Fig. 5A). Rate of disease progress was reduced for AP as compared with UP, indicating the effect that aggregation can have upon temporal aspects of epidemics.

The total number of disease foci increased at approximately the same rate for both AP and UP for the first several assessment dates, but peaked at different times and at different levels (Fig. 5B). In general, a peak in a curve of N versus time indicates the point in time at which coalescence of foci (extinction of old foci) occurs more frequently than either the creation of new foci or the simple, radial expansion of existing foci. The peak for AP was delayed in relation to UP (Fig. 5B).

During the last 7 to 9 days of the ZYMV experiment, large differences in average focus size were observed between AP and UP

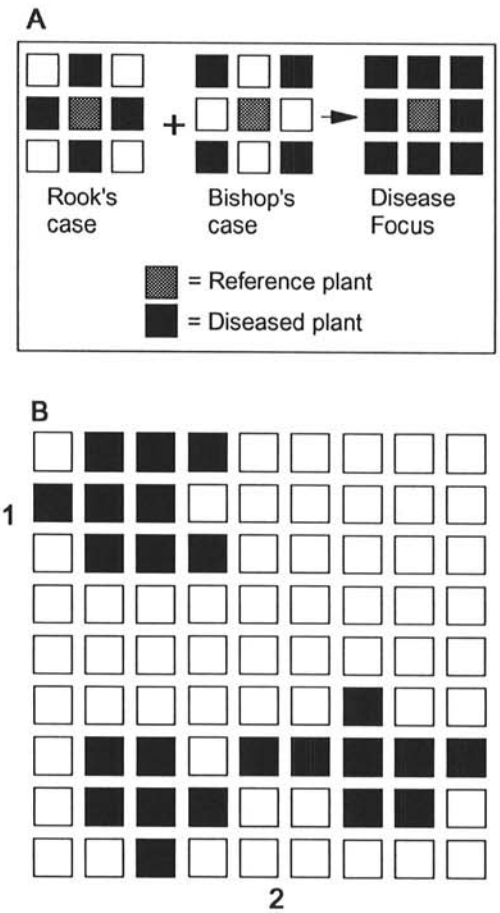

Fig. 1. A, The immediate proximity criterion, defined in relation to the reference plant (diseased) for the rook's and bishop's case (spatial lag = 1) among diseased plants in a plant distribution lattice. This criterion is the basis for defining disease foci: foci consist of diseased plants that meet this criterion for two-dimensional adjacency. **B,** Map of diseased (■) and nondiseased (□) plants in a nine-row by nine-column lattice. The two disease foci (1 and 2, respectively) differ in focus size and proximity index. For focus 1, focus size (s) = 9; maximum row length (r) = 3, maximum column width (c) = 4, and proximity index (PI) = (s/rc) = 0.75. For focus 2, $s = 14$, $r = 4$, $c = 8$, and $PI = 0.44$.

treatments (Fig. 5C). Prior to that, average size of disease foci remained low and constant for the first four assessment dates (Fig. 5C). However, the rapid increase of ZYMV in the UP plot resulted in a single, large disease focus by 39 days after inoculation. The focus spanned the entire row- and column-length of the plot (Figs. 4A and 5D). In general, disease foci were more numerous and larger for the UP data than for the AP treatment.

Cluster shape for the UP plot was, on average, isodiametric during the epidemic. For AP, cluster shape was roughly rectangular between 28 and 39 days after inoculation, on average, with long axis in directions of rows (Fig. 5D).

In light of these data, we know that epidemics with different starting conditions can have quite different spatial and temporal attributes. But we can't say why, exactly. To what extent was the initial number of disease foci responsible for subsequent differences (AP versus UP) for focus attributes? How is disease incidence related to disease focus attributes? The ZYMV experiment was not designed to answer these questions. Only a carefully designed experiment could, and would, reveal that aspects of disease increase in space and time are interrelated.

Discussion. Data generated by this method may be analyzed by well-known statistical methods (e.g., analysis of variance and means separation). Confidence limits on mean values of *s*, *r*, *c*,

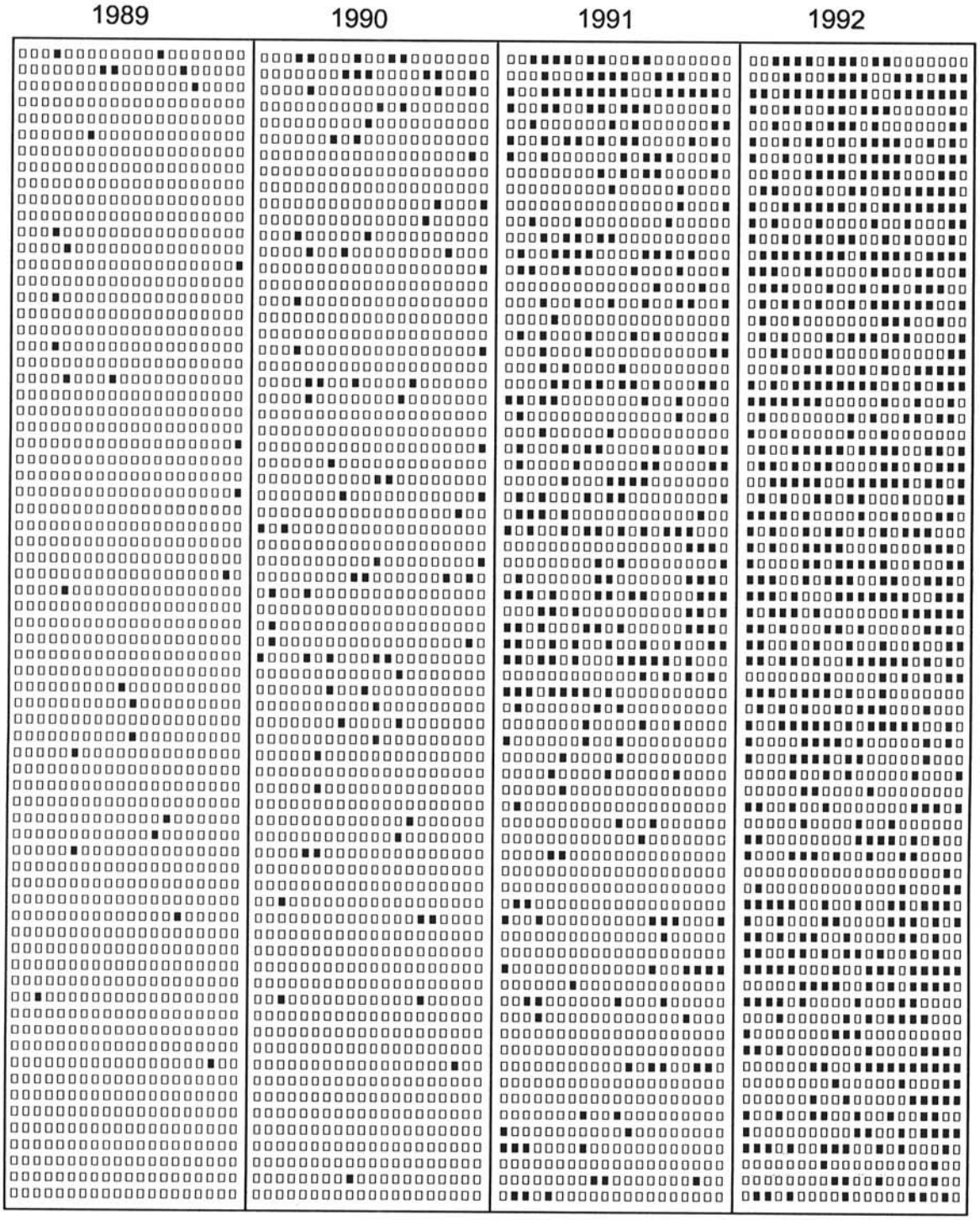

Fig. 2. Yearly maps (1989 to 1992, respectively) of diseased (■) and nondiseased (□) citrus trees during an epidemic of citrus variegated chlorosis in a 71-row by 20-column section of a Brazilian citrus orchard. Maps show the accumulation of disease from year to year in the same orchard.

and *PI* could be calculated. Measures of variance and central tendency have the potential to provide important insights into epidemic dynamics. For example, a measure of variation in *s*, *r*, *c*, and *PI* might be helpful in describing the spatial pattern of a disease. Under some conditions, very different epidemics might have similar measures of central tendency (i.e., mean values of *s* and *PI*). Thus, some measure of variance about these means might be helpful in distinguishing such epidemics. The potential importance of variation in mean *s* is illustrated by the histogram in Figure 3D. The method also generates temporal data that can be fit to nonlinear models (e.g., logistic) that describe system behavior over time. Parameters from these nonlinear models may even prove useful in predicting the spatiotemporal dynamics of disease spread.

There are very few techniques currently available that provide useful information about disease foci for intensively mapped, binary data (focus size, shape, number, etc.). The simple method described here fills a need in this area and provides an improved alternative for describing clusters. The method may be used as a direct supplement to 2DCLASS. 2DCLASS and spatial autocorrelation analysis both provide limited information about focus size, shape, number, etc. The output matrices and proximity patterns produced by these analyses are reflections of the actual data and may even be more complex than the original data. Although distance class analysis is useful in detecting departures from spatial randomness and edge effects, its estimates of cluster number and size are imprecise and of unproven reliability, since informa-

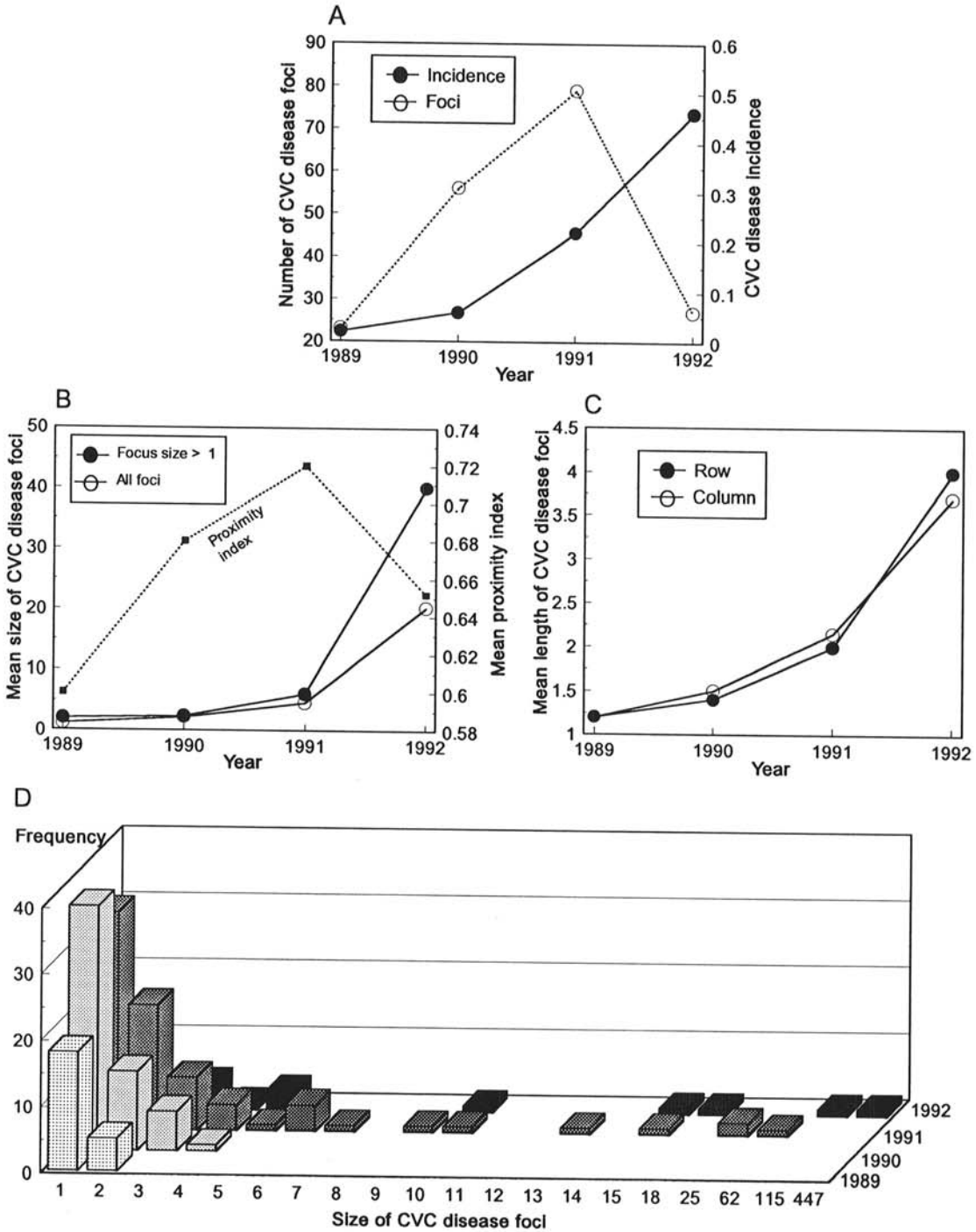

Fig. 3. Disease foci data from an epidemic of citrus variegated chlorosis (CVC) in a 71-row by 20-column Brazilian citrus orchard from 1989 to 1992. **A,** Total number of disease foci and incidence of symptomatic trees. **B,** Mean size of disease foci and mean proximity index for disease foci. **C,** Mean maximum row and column lengths of disease foci. **D,** Histogram of frequency distribution for size (i.e., number of diseased plants) of disease foci.

tion is gathered from the distance class matrix and not the mapped data directly.

By using the simple approach described here in conjunction with distance class analysis, improved estimates (accuracy and precision) of disease focus attributes may be obtained. For example, the CVC data and ZYMV data were analyzed by 2DCLASS and the simple method, and quite different estimates were obtained (Table 1). Of interest is the underestimation of s by 2DCLASS for the UP treatment in the ZYMV data in relation to direct analysis of disease foci (Table 1). The misestimation of s occurred because the core cluster area of the distance class matrix was only a reflection of average spatial relationships among diseased plants in comparison with a random scenario. Conversely, direct analysis of foci is done from mapped data and no comparison with random scenarios is done. Misestimation of N occurred because of a basic difference in the way N is calculated by the two analyses. In distance class analysis, N is estimated by counting the number of discrete groups of distance classes within the

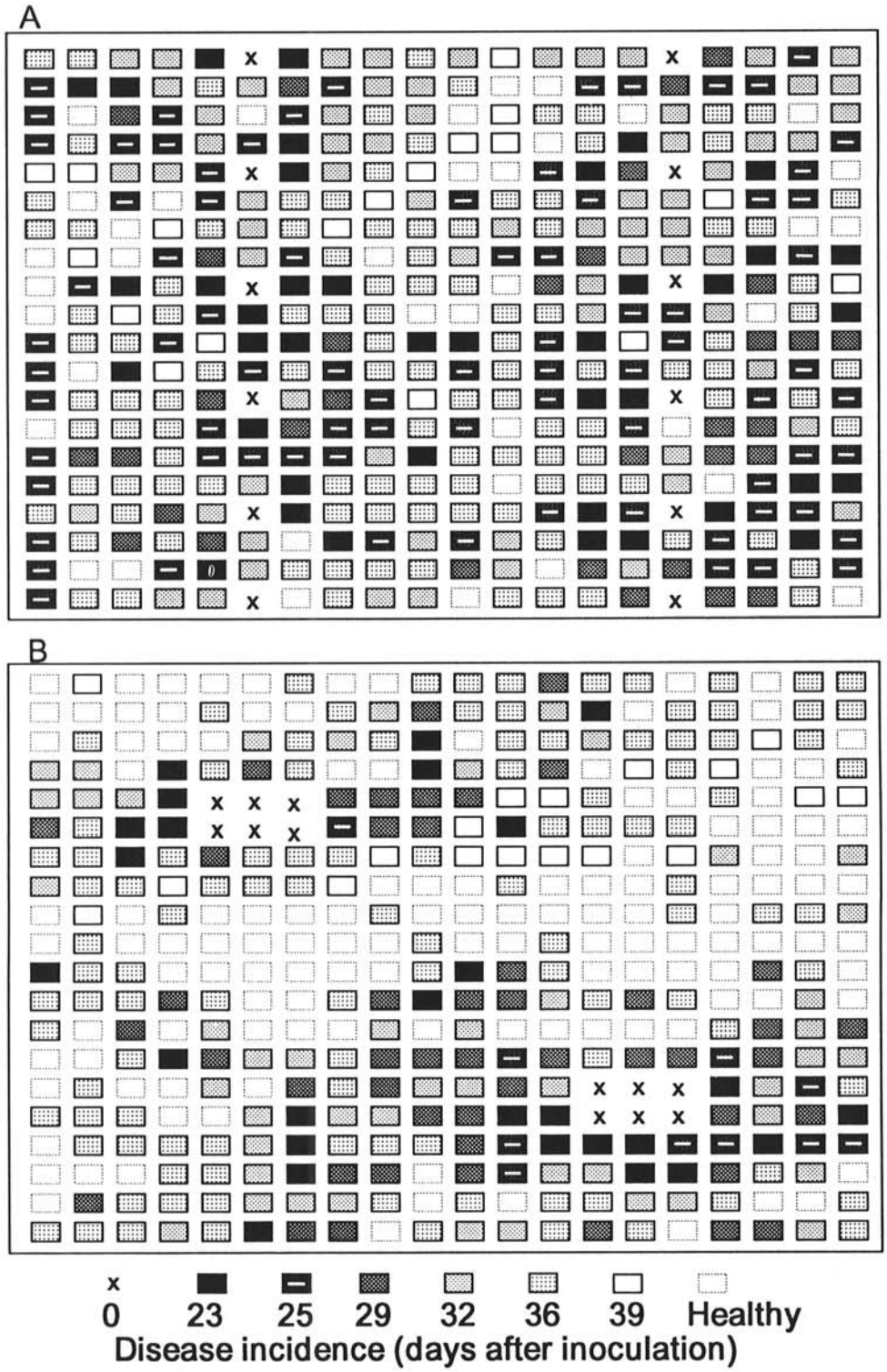

Fig. 4. Two spatiotemporal maps of diseased and nondiseased zucchini plants during epidemics of zucchini yellow mosaic virus (ZYMV) during 1994 in Maui, Hawaii. At day 0, 12 seedlings were inoculated (×) in each of two lattices (each 20-rows by 20-columns). **A,** Uniform patterns of inoculated plants. **B,** Aggregated patterns of inoculated plants. Disease spread occurred naturally by aphids after inoculation.

distance class matrix. These groups of distance classes represent spatial relationships among diseased plants within the lattice (in comparison with random scenarios) and expose patterns of aggregation. The same rules for proximity used to identify foci in this paper are used in distance class analysis to identify discrete groups of distance classes in the distance class matrix (the reflected clusters). However, single, significant distance classes that are spatially isolated are not counted in the estimation of N. In the simple model described here, single and spatially isolated diseased plants are considered disease foci and are counted in the estimate of N.

Links with other forms and systems of analysis are possible. Data on focus attributes or additional parameters generated by using the data in other models have potential to be utilized by crop loss models (7) or disease forecasting systems. The method described here may be used in any physical or biological system for which binary data (presence/absence) data are relevant. The method is distinguished from forms of analysis of sparsely sampled or continuous data. The strength of the method is in the comparison of epidemics.

There are potential limitations to this method of analysis. It is well established from experimental data that aggregation varies with disease mean and, therefore, usually with time (8,16). Thus, parameters of spatial and temporal models that are used to understand a pathosystem also vary with disease mean and are density dependent.

There exists a degree of density dependence for the elements of the model described here (e.g., N, s, r, c, and PI). In other words, a variable (N) can assume a range of values depending on the level of disease incidence in the population. An example of the mathematical basis for dependency in this system is as follows.

Within a given rectangular lattice, there is space for a finite number of disease foci (N_{max}). This maximum number of foci is realized when foci are minimum size ($s = 1$) and distributed uniformly within the lattice (and have no rook's- or bishop's-case position occupied by another diseased plant). N_{max} may be expressed as a function of focus dimensions (row and column length): $N_{max} = (rc/4)$; in which $r = r + 1$ or $c = c + 1$, when r or c are odd numbers, respectively. Thus, for the CVC data (71 rows, 20 columns), $N_{max} = [(72)(20)/4] = 360$, the maximum number of disease foci possible for this lattice. When $s = 1$, N is a measure of disease incidence (DI). DI at N_{max} can then be expressed as follows: $DI = (N_{max}/rc)$. Using this equation, CVC DI at theoretical N_{max} (i.e., 360 foci) was 0.253. Actually, CVC DI at observed N_{max} (i.e., 79 foci) was 0.22 (Fig. 3A). For lattices with an even number of rows and columns, DI at $N_{max} = 0.25$. This is the level of disease incidence at theoretical N_{max} in the lattice. It follows that as DI approaches 0.25 it becomes possible to observe the maximum number of foci during an epidemic. Thus, values for N are density dependent. When DI is very low or high, values for N are expected to be low. This means that comparisons of N among lattices of different sizes or levels of disease incidence should proceed with caution and may be relatively meaningless.

Focus size also is density dependent. Because plants remain diseased in this model, a disease focus cannot shrink, it can only get larger or remain the same size. Thus, as disease spreads (incidence increases), foci tend to enlarge. Also, space within a matrix is limited. As DI approaches its maximum, coalescence among foci occurs (larger s values).

Values for PI vary with s. (Remember that s is the number of diseased plants, not a measure of focus dimension. Focus dimensions, r and c values, assume a range that is dependent upon s.) For example, as s increases, the minimum value possible for PI (i.e., PI_{min}) decreases: $PI_{min} = (s/s^2)$. As the minimum value for PI decreases, the range and number of values possible for PI increases. As s increases, the range in possible focus shapes and

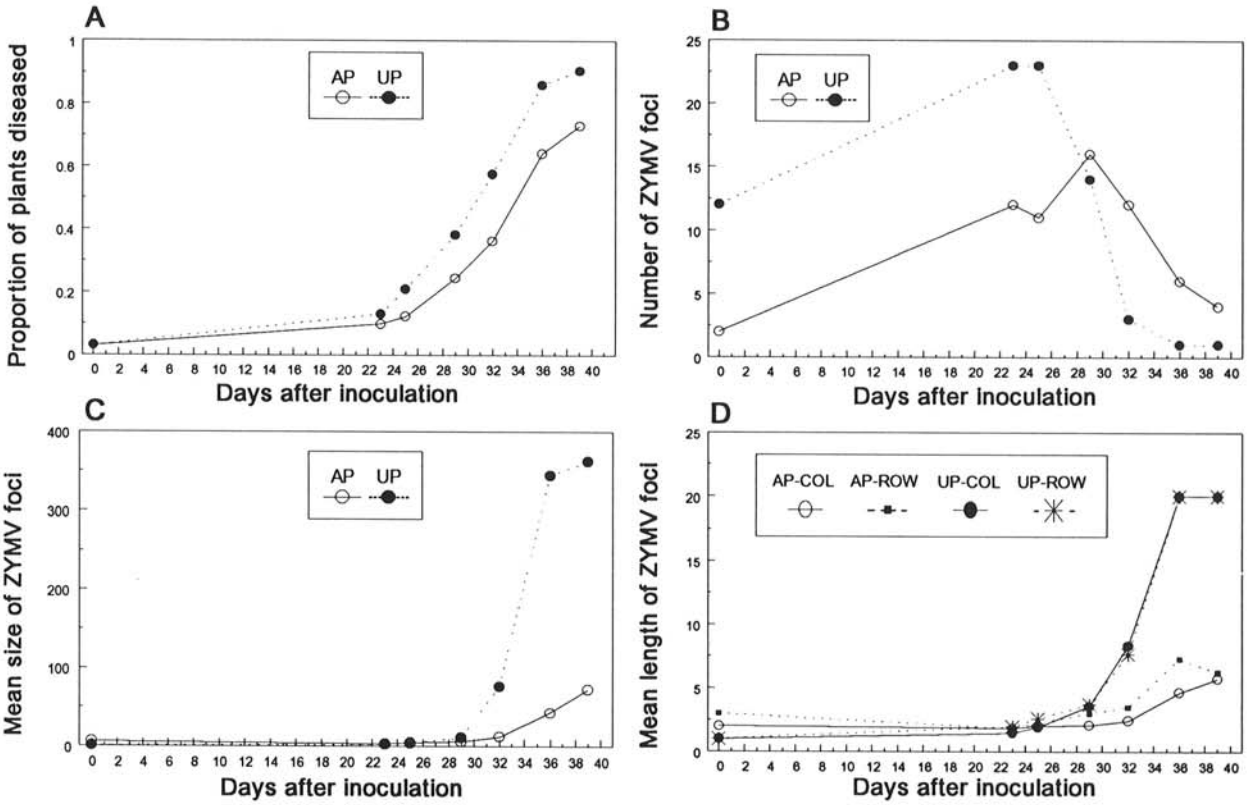

Fig. 5. Comparative disease foci data versus time from seven disease assessment dates during two epidemics of zucchini yellow mosaic virus (ZYMV) on zucchini squash in 1994 in Maui, Hawaii, in 20-row by 20-column lattices. Data are from epidemics with two different spatial starting conditions, a uniform pattern (UP) of 12 ZYMV-infected plants and an aggregated pattern (AP) of 12 ZYMV-infected plants. **A,** Proportion of ZYMV-infected plants. **B,** Total number of ZYMV disease foci. **C,** Mean size of ZYMV disease foci. **D,** Maximum row and column length of ZYMV disease foci.

dimensions increases. These concepts are illustrated as follows. First, with $s = 1$, $PI = 1$. There is only one possible PI value when $s = 1$. With $s = 2$, PI can assume two values (1.0 and 0.5), depending on whether the connection between the two diseased plants is rook's or bishop's case, respectively. With $s = 3$, PI can assume the following values: 1.00, 0.75, 0.50, and 0.33. Thus, mean PI values, which are based upon data from many small foci ($s = 1$), are relatively inflated. Larger foci will tend to have smaller values for PI, on average. If PI is dependent upon s, and s is dependent upon density (DI), then PI is density dependent also.

TABLE 1. Comparison of disease foci attributes from two forms of spatial analysis (two-dimensional distance class analysis [2DCLASS] and an analysis of disease foci [FOCI]) for epidemics of citrus variegated chlorosis (CVC) in Brazil during 1989 to 1992 and zucchini yellow mosaic virus (ZYMV) on zucchini squash in Hawaii in 1994

Disease focus attribute	2DCLASS	FOCI
CVC (year):		
Cluster size	Core cluster size[a]	Mean focus size[b]
1989	2	1.1
1990	3	2.2
1991	47	4.6
1992	5	20.3
Cluster number	Reflected clusters[c]	Number of foci[d]
1989	24	23
1990	17	56
1991	14	79
1992	13	27
Cluster compactness	Core cluster proximity index[e]	Mean proximity index[f]
1989	0.5	0.6
1990	0.75	0.68
1991	0.52	0.72
1992	0.63	0.65
ZYMV (days after inoculation):		
	Core cluster size	Mean focus size
Cluster size	AP[g] / UP[h]	AP / UP
23	11 / 3	3.3 / 2.2
25	19 / 4	4.5 / 3.7
29	30 / 5	6.1 / 11
32	38 / 5	12.0 / 76.7
36	29 / 3	42.7 / 344.0
39	25 / 2	73.0 / 362
	Reflected clusters	Number of foci
Cluster number	AP / UP	AP / UP
23	3 / 8	12 / 23
25	2 / 4	11 / 23
29	1 / 6	16 / 14
32	1 / 3	12 / 3
36	1 / 5	6 / 1
39	1 / 5	4 / 1

[a] Core cluster size defined as the number of significant ($P \leq 0.05$) and adjacent distance classes in the [0,0] region of the 2DCLASS matrix.
[b] Mean focus size is defined as the average number of diseased plants in a disease focus.
[c] A reflected cluster is defined as any group (>1) of significant ($P \leq 0.05$) and adjacent distance classes in the 2DCLASS matrix, excluding the [0,0] region of the matrix.
[d] A disease focus is a discrete group of spatially isolated diseased plants (≥1 diseased plant).
[e] Proximity index is a measure of the compactness of cluster organization. For the core cluster, the index is calculated as the core cluster size/[X,Y] dimensions of the core cluster.
[f] Proximity index is a measure of the compactness of cluster organization. In FOCI, the index is calculated as focus size/(row · column dimensions of the disease focus).
[g] AP = aggregated pattern of 12 initially diseased plants.
[h] UP = uniform pattern of 12 initially diseased plants.

Density dependence of variables within this model does not preclude its use, just as other density-dependent forms of spatial analysis are not precluded from use. Unfortunately, it may not be possible to assess the significance of some values because it may not be possible to determine whether the temporal changes in attributes of foci were because of i) changes in the spatial pattern of the disease or ii) changes in the incidence of disease. However, useful comparisons of epidemics are made possible by comparing levels of one variable at a constant level of another variable (e.g., s at 25% DI).

For illustration, let us compare values for mean s for the ZYMV data at a fixed DI level (0.70). From Figure 5A, it is evident that 0.70 DI occurred at 34 days after inoculation for the UP treatment and at 39 days after inoculation for the AP treatment. From Figure 5C, a comparison of s at 0.70 DI is possible by comparing the values for s at 34 days after inoculation (s = approximately 200, UP treatment) and at 39 days after inoculation (s = approximately 55, AP treatment). This approximately fourfold difference in values for s between treatments indicates that s, and each other variable from this analysis, is not strictly density-dependent and can assume a relatively wide range of values.

In addition, with careful attention to experimental design, it may be possible to distinguish between effects due to pattern and effects due to disease incidence, or density.

Finally, variables and parameters derived from this method are interrelated and should be interpreted as such. The mapped data should always be revisited after performing an analysis to ensure that strange or bizarre foci do not allow statistics to obscure the truth.

Among its positive aspects, the method described here is quite simple, conceptually and in application and interpretation, and helps to alleviate the lack of simple techniques available to the nonepidemiologist. A computer is not needed. Calculations are made directly from mapped data. The method reduces complexity of mapped data by transforming many maps into single, curvilinear relationships between variables (e.g., s versus time). The method allows relatively precise quantification of important attributes of clustering: focus size, shape, orientation, compactness, and the variances associated with these attributes. In addition, the method permits us to look at epidemics from the standpoint of a population of disease foci rather than a population of individually diseased plants, a concept that finds support within the ecological literature (6).

Computer software capable of performing the analysis is available from the author upon request.

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