Geostatistical Analysis of *Phytophthora* Epidemic Development in Commercial Bell Pepper Fields

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This research was funded in part by grant number 92-37303-7715 from the National Research Initiatives Competitive Grants Program of the USDA. The technical assistance of Greg Parra is greatly appreciated. We also thank Sarah Potter, Candace Coats, and Melissa Brake for their assistance in data collection and processing, and C. Lee Campbell and Barbara Shew for their critical review of this manuscript.

Accepted for publication 14 November 1994.

ABSTRACT

Larkin, R. P., Gumpertz, M. L., and Ristaino, J. B. 1995. Geostatistical analysis of *Phytophthora* epidemic development in commercial bell pepper fields. Phytopathology 85:191-203.

Spatial patterns of disease, soil population levels of *Phytophthora capsici,* and soil water content were characterized during epidemics of Phytophthora root and crown rot using geostatistical techniques in three commercial bell pepper fields in two consecutive years. Disease incidence and severity increased with time in all fields, ranging from a maximum linear rate of 1.01% per day and 0.060 severity units per day (0–6 scale) in the lower portion of field three in 1992 to a minimum rate of 0.33% per day and 0.011 severity units per day in the upper portion of field three in 1993. Semivariograms constructed over four directions of orientation (0°, 45°, 90°, and 135°) were used to determine the spatial dependence of mean disease severity by quadrat (1 m^2) in each field over time. In most fields, disease severity displayed strong spatial dependence and a high degree of anisotropy over time, indicating strongly aggregated patterns of disease with distinct directional orientation. The within-row (0°) orientation generally was the direction of maximum spatial correlation and the predominant direction of disease spread. Diagonal directions also demonstrated considerable spatial dependence in most fields, whereas spatial dependence in the across-row direction varied with soil water conditions and was lower than other directions in three of four fields. The range of spatial dependence for final disease severity averaged 15 m or greater within rows and 3–15 m across rows for fields one and two in 1992 and the lower portion of field three in 1992 and 1993. Disease severity displayed little spatial dependence in field one in 1993 and the upper portion of field three in both years. Two-dimensional distance class analysis indicated nonrandom spatial patterns of disease incidence in all fields, as well as an increasing level of aggregation of disease over time and greater spread of disease within rows than across rows in most fields. Patterns of soil water content also were spatially dependent and correlated with patterns of disease severity in fields that had distinct moisture gradients. Early season population levels of *P. capsici,* as estimated by either a leaf disk bioassay or soil dilution plating, did not consistently demonstrate strong spatial dependence and were not closely associated with the occurrence or severity of disease throughout the season. These results emphasized the importance of soil water in the development and spread of disease caused by *P. capsici* in bell pepper.

Additional keywords: *Capsicum annum,* spatial pattern analysis

Spatial patterns of diseased plants over time reflect patterns of initial inoculum, dispersal processes, infection, and production of secondary inoculum, and result from the interaction of physical, biological, and environmental factors (2,21,37). Statistical methods that quantitatively define spatial patterns of disease development can be used to identify specific physical and biological mechanisms responsible for these patterns. Spatial pattern analyses and their applications in plant disease epidemiology have been reviewed by Campbell and Madden (2). Methods such as variance-to-mean ratios (37), indices of dispersion (2,20,37), and frequency distributions (10,28), do not take into account the actual location of sample sites or their relationship to neighboring sites, and result in a loss of valuable spatial information in the analysis (2,27). Recently, more sophisticated quantitative methodologies that incorporate these spatial relationships in the analysis have been used, including two-dimensional distance class analysis (8,9, 25,26,33,34), spatial and spatio-temporal autocorrelation analysis (7,22,29,30), and spatio-temporal autoregressive integrative moving average (STARIMA) techniques (11).

Geostatistical analysis has also been used in plant pathology to quantitatively characterize changes in spatial patterns of disease or pathogen populations over time (3,15,17,24,35,38). Geostatistics are a group of statistical techniques based on the theory of regionalized variables, i.e., variables that have a spatial distribution. These techniques take into account both the random and structured characteristics of spatially distributed variables and provide quantitative tools for their description and optimal, unbiased estimation (4,12,13,39). The spatial correlation structure can be described with the semivariogram and correlogram, which quantify spatial dependence by measuring the variation among samples separated by the same distance. These functions provide a quantitative representation of the spatial variation within a field. From this, the degree and strength of correlations, area or range over which samples are spatially dependent, and directional effects can be determined.

Geostatistical analysis has several advantages over other methods of spatial pattern analysis (3,4,39). Geostatistical techniques can be used on all types of continuous variables from complex data sets and require less rigid assumptions of stationarity compared with spatial autocorrelation techniques (6,13,39). Spatial dependence can be analyzed in specific directions or omnidirectionally. Geostatistics can be used to predict and interpolate spatial patterns and sample values in areas not sampled (kriging), and multiple interrelated factors can be analyzed to determine how they co-vary with distance and direction (4,12,19,39,41).

We are studying *Phytophthora capsici* Leonian as a model system to understand the modes of dispersal of inoculum in the field. The pathogen causes a root and crown rot, as well as an aerial blight of leaves, fruit, and stems on bell pepper (*Capsicum*

annuum L.) (1,18,31). Diseases caused by many *Phytophthora* species are polycyclic, i.e., multiple cycles of inoculum production and infection occur within a growing season, and dispersal of inoculum from previously infected plants plays a significant role in the development of epidemics (1,31,36). In addition, changes in soil water matric potential can significantly affect disease development, pathogen dispersal, pathogen population densities in soil, and the relationship between inoculum density and the development of disease (1,31,32). Analysis of spatial patterns of inoculum in soil and soil water status may be important in understanding the evolution of spatial patterns of Phytophthora diseases and dispersal mechanisms. However, little quantitative information is available that relates the spatial pattern of *P. capsici* populations in soil to soil water status and disease development in naturally infested fields.

Previously, we used two-dimensional distance class analysis to characterize the spatial and temporal development of disease and symptom expression in three commercial bell pepper fields naturally infested with *P. capsici* (33,34). Two-dimensional distance class analysis was based on binary data, the presence or absence of disease within a quadrat, and was used to quantify the size, location, and changes over time in clusters of quadrats containing diseased plants (25,26). Plants with wilt, crown lesions, stem lesions, or dead plants were found to be aggregated in each field, and cluster sizes increased over time. Disease spread was greater within rows than across rows (33,34). Wilting preceded the observation of crown symptoms, suggesting that root infection and subsequent colonization to crowns of plants occurred frequently. Inoculum movement to roots and movement of surface water within rows were probable mechanisms of inoculum dispersal (33,34).

Geostatistical analysis was used in the present study to quantify the spatial pattern of disease over time in the fields described previously and in two additional commercial bell pepper fields. Our objectives were to determine the degree and range of spatial correlation of disease severity in four directions over time and to determine potential mechanisms of disease spread and inoculum dispersal operating in this pathosystem. In addition, spatial patterns of soil water content and *P. capsici* populations in soil were analyzed and compared with spatial patterns of disease over time to determine how these factors were interrelated. We also evaluated the information derived from geostatistical analysis by comparing it with the information derived from two-dimensional distance class analysis for these fields. Preliminary results have been published previously (16).

MATERIALS AND METHODS

Some of the data reported were collected as part of a series of 1992 field experiments for which many of the methods have been described (33). Experiments were conducted in three commercial pepper fields in 1992 and in two of the same three fields in 1993. Fields were located in the Coastal Plain region of North Carolina and were naturally infested with *P. capsici*. Locations of the sampling areas within fields were similar but not identical in both years. Soil characteristics were described previously (33). In each field, *P. capsici* was isolated from infected plants on a *Phytophthora*-selective medium (PARPH) (14) and the presence of the pathogen in soil was confirmed by soil dilution plating.

Fields were cultivated and prepared by growers using standard cultural practices as described previously (33). Fields one and two in 1992 and all fields in 1993 consisted of 20 rows, each of which was divided into 20 contiguous quadrats of approximately 1 m^2 (400 quadrats total). In 1992, field three was divided into two adjacent 40 row × 35 quadrat sections corresponding with the upper and lower portions of the field to account for differences in slope, elevation, and drainage patterns in this field. Individual quadrats contained two to three transplants of the susceptible pepper cultivar Jupiter and within-row plant spacing was approximately 30 cm in all fields.

Incidence of disease on plants within quadrats was assessed nine times in field one, 10 times in field two, and three times in the upper and lower portions of field three in 1992. In 1993, disease incidence was assessed 14 times in field one, 13 times in the lower portion of field three, and seven times in the upper portion of field three. Fewer assessments were made in the upper portion of field three due to the absence of disease through the first half of the season in this field. Each plant within a quadrat was examined for specific disease symptoms, including wilting without visible lesions, lesions on crowns, stems, fruit, or leaves, or plant death due to disease.

Disease severity was evaluated based on categorical symptom classes observed on each plant using a 0–6 scale: 0 = healthy plant; 1 = plant with fruit or leaf lesion; 2 = wilted plant without visible lesions; 3 = plant with stem lesion above soil; 4 = plant with crown lesion at soil line; 5 = plant with both stem lesion and crown lesion; and, 6 = dead plant. The severity scale was based on the typical progression of disease symptoms following initial infection (33,34). Mean disease severity was determined for each quadrat by calculating the arithmetic mean for all plants in a quadrat. Mean disease severity should not be interpreted to correspond with actual disease symptom classes but represents an average value on a scale of increasing degrees of disease severity. Regression analysis was used to determine and compare the average linear rates of increase of disease incidence and disease severity for each field. Mean disease severity by quadrat was mapped for all fields at each assessment date.

Populations of *P. capsici* in soil and soil water contents were estimated from soil samples taken from each quadrat in fields one and two in 1992 and fields one and three in 1993. Soil samples were collected early and late in epidemic development in each

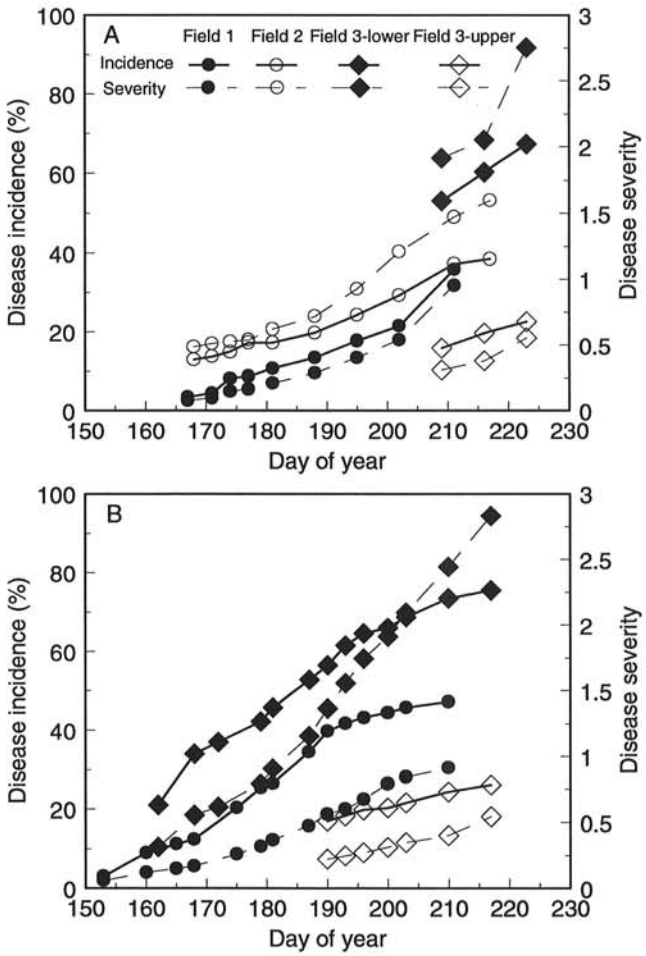

Fig. 1. Mean disease incidence and severity caused by *Phytophthora capsici* in three commercial bell pepper fields in the **A,** 1992 and **B,** 1993 growing seasons. Disease severity on individual plants ranged from 0 = healthy plant to 6 = dead plant.

field, with the exception of field three in 1993, when soil was sampled only in the early stages of the epidemic. Six soil cores (1.9 × 15 cm deep) were removed from opposite sides of each pepper plant in each quadrat. Cores were combined into a composite sample for each quadrat, stored in plastic bags at room temperature, and assayed within 3 days of sampling. Pathogen population levels were estimated from soil samples using a pepper leaf disk bioassay in 1992. However, in order to improve quantification of pathogen levels, populations were estimated by soil dilution plating on Masago's medium (23) amended with 20 mg/L hymexazol (99.5% a.i. Tachiagaren) in 1993 assays.

In the 1992 assays, 55 ml of soil from each quadrat was placed in a plastic cup (250 ml) with drainage holes, saturated with water, and allowed to drain for 5 days at 24 C. Drainage holes then were plugged, soil was flooded with water, and five 6-mm-diameter pepper leaf disks were floated on the water. After 24 h, leaf disks were removed, surface disinfested in 0.5% sodium hypochlorite for 1 min, rinsed in sterile distilled water, and blotted dry on sterile paper towels. The disks were then placed on amended Masago's medium (23) and disks colonized by *P. capsici* were counted 72 h later. Identification of *P. capsici* was verified by culturing on V8 agar and observing sporangial characteristics.

Relative soil population levels of *P. capsici* were represented by the percentage of leaf disks colonized, and calculated as the number of colonized disks compared with the total number of leaf disks. In 1993 assays, 20 g of each composite soil sample was added to 80 ml sterile 0.25% water agar, stirred, and 1 ml of the resulting suspension was spread on each of five replicate plates of amended Masago's medium. After incubation in the dark at 20–24 C for 72 h, plates were rinsed with water to remove soil residue and colonies of *P. capsici* were counted. Population levels were estimated as the number of cfu per gram of soil. Gravimetric soil water content was determined on a dry weight basis for composite soil samples from each quadrat at the same time as the pathogen assay in both years.

The spatial arrangement of diseased plants and plants with different symptom types were characterized by two-dimensional distance class analysis (8) using 2DCLASS software (26) as described previously (33). Spatial statistics obtained from two-dimensional distance class analysis included minimum core and reflected core cluster size, total number of clusters, and within and across-row effects (33). Results from 2DCLASS analysis were compared with results from geostatistical analysis to provide additional spatial characterization of disease development.

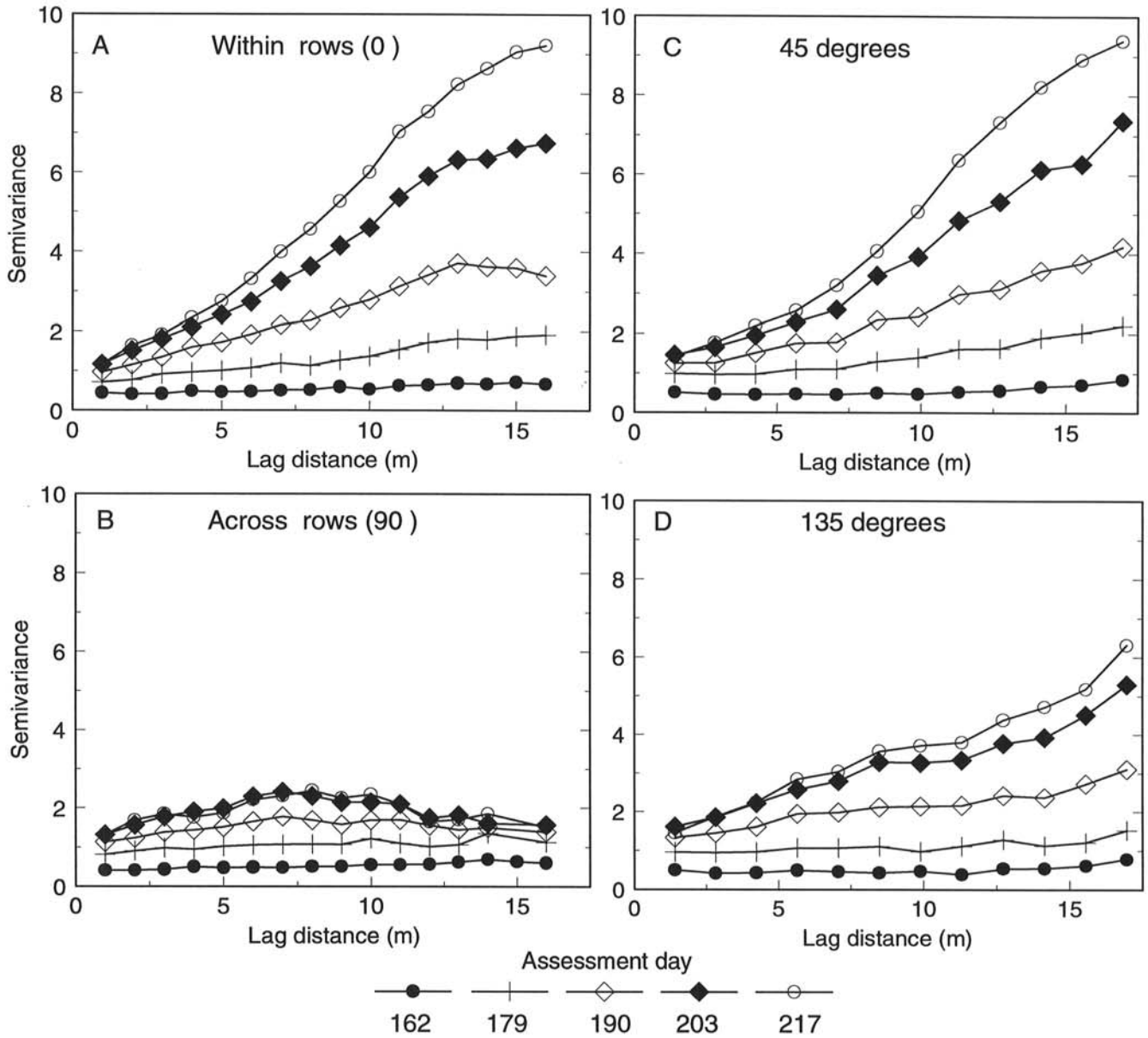

Fig. 2. Oriented semivariograms for disease severity caused by *Phytophthora capsici* on five assessment dates in the lower portion of field three in 1993 for the **A,** within-row (0°), **B,** across-row (90°), **C,** 45°, and **D,** 135° directions.

Geostatistical analysis was conducted on mean disease severity data for each quadrat using GSLIB version 1.2 software (5). Semivariance, covariance, and correlation functions were calculated for lag distances up to 15 m for the 20 row × 20 quadrat fields and up to 20 m for the 40 row × 35 quadrat fields. At these distances there were at least 100 quadrat pairs for computing each semivariogram value. The sample semivariogram is defined as one-half the average squared difference between pairs of sample data values separated by a given lag distance (12,13). Analysis was conducted in four directions, 0, 45, 90 and 135° azimuth, with 0° representing the within-row direction. Semivariograms were prepared for each field at each assessment date by plotting semivariance versus lag distance for all four directions. Semivariance values that start out small and increase with increasing lag distance indicate spatially dependent samples (12,13,39). The lag distance at which the semivariance approaches a constant value is called the range of spatial dependence, and indicates the distance beyond which the sample values are no longer correlated with each other (12,13,39). Samples separated by distances closer than the range are spatially correlated, whereas those separated by distances greater than the range are not spatially correlated. The constant value at which the semivariogram levels off is called the sill and generally approximates the sample variance. The point at which the semivariogram crosses the Y axis is called the nugget variance. This value reflects a discontinuity in the semivariogram at lag zero and is composed primarily of measurement error and microdistributional error, or the spatial variation occurring below the minimum scale of the study (12,13,39).

Linear and nonlinear models were fitted to semivariograms by weighted least squares regression using Statistical Analysis Systems ver. 6.04 (SAS Institute, Cary, NC). Models used to describe semivariograms included the spherical, Gaussian, exponential, and linear forms (4,5,12,40), and the model resulting in the lowest mean square error was considered the best fit. Weights were calculated by dividing the number of sample pairs by the model semivariance squared at a given lag distance (4). Semivariograms that did not fit any model (e.g., periodic curves) were described only by visual inspection of the graph. Characteristics of semivariograms determined from model parameters included nugget variance, range of spatial dependence, and sill for nonlinear models or slope for linear models. Anisotropy, which represents the occurrence and degree of directional differences in spatial dependence, was determined by comparison of semivariogram characteristics. Oriented semivariograms that displayed differences among semivariogram characteristics for different directional orientations indicated anisotropy or directionality in the degree of spatial dependence. Covariograms and correlograms also were constructed by plotting covariance or correlation functions versus lag distance. For clarity of presentation, only the semivariograms and disease severity maps for three assessment dates in four fields, representing early, middle, and late stages of epidemic development, are presented.

RESULTS

Disease incidence and severity. Disease symptoms observed on plants in each field in 1992 and 1993 included wilting without lesions, crown lesions, stem lesions, and dead plants. No plants were observed with leaf or fruit lesions in any field in 1992 and only a small percentage of plants (<2%) were observed with leaf or fruit lesions in 1993.

Disease incidence in 1992 increased at average rates of 0.65% per day in field one, 0.54% per day in field two, and 1.01 and 0.48% per day in the lower and upper portions of field three in 1992 (Fig. 1A). In 1993, disease incidence increased at rates of 0.89% per day in field one, and 1.0 and 0.33% per day in the lower and upper portions of field three (Fig. 1B). Mean final

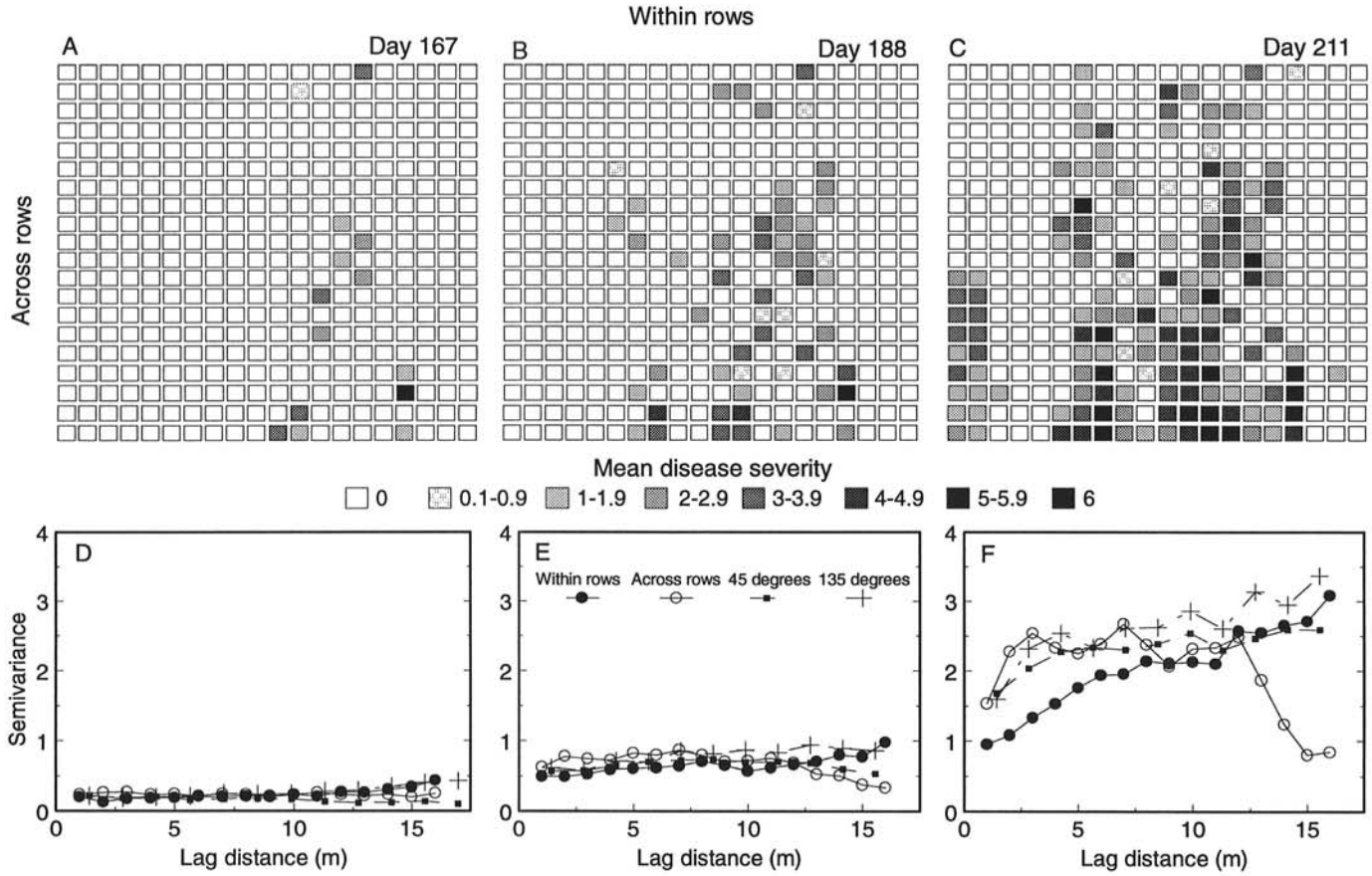

Fig. 3. Spatial pattern maps and semivariograms of disease severity caused by *Phytophthora capsici* on bell pepper in field one in 1992 on **A, D,** day 167, **B, E,** day 188, and **C, F,** day 211.

disease incidence at the end of the season was 35.8, 38.5, 67.3, and 22.6%, for fields one, two, and the lower and upper portions of field three in 1992, and 47.3, 75.5, and 26%, for field one and the lower and upper portion of field three in 1993, respectively.

Wilting without lesions preceded the occurrence of crown lesions by 1–2 wk on plants in most fields. Crown lesions were the predominant symptom class observed in field one through most of the season in 1992. Crown lesions also predominated in field two, although by the end of the season dead plants were the most frequent class observed. Wilting without lesions was the predominant symptom observed in field one in 1993 and in the upper portion of field three in 1992 and 1993. Crown lesions and dead plants were the predominant symptom classes observed in the lower portion of field three in 1992 and 1993.

Disease severity was greatest in the lower portion of field three in both years (Fig. 1). Average rate of linear increase in disease severity was 0.060 severity units per day in 1992 and 0.047 severity units per day in 1993. Disease severity was lowest in the upper portion of field three in both years and the average rate of increase was 0.018 in 1992 and 0.011 in 1993. Disease severity in fields one and two also increased over time and was greater in field two than in field one in 1992 (Fig. 1).

Geostatistical analysis. Characteristic changes were observed in semivariograms over time and are shown in detail for the lower portion of field three in 1993 (Fig. 2). Semivariograms for all directions were essentially flat at the early stages of the epidemic, indicating that disease in quadrats was not spatially related with disease in neighboring quadrats and variation between quadrats was random. Disease severity tended to become more spatially dependent over time, and the degree of spatial dependence differed with the direction of orientation (strong anisotropy). The slope of the semivariogram increased over time in the within-row (0°) direction (Fig. 2A), as did the sill and range of spatial dependence, whereas smaller changes in semivariogram characteristics with time generally were observed across rows (90°) (Fig. 2B). Semivariogram slopes that increase over time indicate an increase in the disease gradient in that direction. Changes in semivariogram characteristics over time also were observed for the diagonal (45 and 135°) directions for most fields (e.g., Fig. 2C and D), but the degree of change varied among fields. In general, spatial variation was maximum within rows and minimum across rows. Nugget variance (Y intercept) increased as disease severity, spatial dependence, and the season progressed, but in most fields it remained small relative to total variance. This indicated a strongly spatially dependent structure with a large proportion of the variation explained by spatial distribution. Variogram shapes were described by a variety of models within and among fields, including spherical, Gaussian, exponential, and linear forms.

Several small clusters of quadrats containing diseased plants coalesced into a large cluster of varying disease severity by day 211 in field one in 1992 (Fig. 3A–C). Little spatial dependence of disease was observed in any direction on days 167 or 188

TABLE 1. Semivariogram characteristics and model parameters of final disease severity for epidemics caused by *Phytophthora capsici* in commercial bell pepper fields in 1992 and 1993

Year/field	Day of year	Analysis direction[a] (degrees)	Model or shape[b]	Mean square error	Nugget variance[c] (C_0 or a)	C_1 or b[d]	Sill[e]	Range of spatial dependence[f]	Correlation at lag 1[g]
1992									
Field 1	211	0	Linear	1.49	0.88	0.14	...[h]	>15	0.60
		90	Periodic	...	...	...	(2.5)	(3)	0.37
		45	Exponential	0.33	1.12	1.38	2.50	8.05	0.30
		135	Exponential	1.65	1.26	1.71	2.97	12.2	0.33
Field 2	217	0	Linear	1.94	0.76	0.41	...	>15	0.76
		90	Periodic	...	...	...	(7)	(5)	0.64
		45	Spherical	1.46	0.66	3.51	4.17	6.6	0.65
		135	Gaussian	2.57	1.88	5.10	6.98	8.1	0.55
Field 3-lower	223	0	Gaussian	1.00	1.59	5.64	7.23	15.1	0.74
		90	Spherical	1.08	1.78	2.77	4.55	14.5	0.67
		45	Gaussian	0.73	2.03	5.81	7.84	18.9	0.65
		135	Gaussian	0.50	2.01	6.38	8.39	15.1	0.64
Field 3-upper	223	0	Linear-(Flat)	3.97	1.09	0.007	...	0	0.38
		90	Linear-(Flat)	2.73	1.30	0.014	...	0	0.15
		45	Linear-(Flat)	1.95	1.33	0.000	...	0	0.17
		135	Linear-(Flat)	2.02	1.31	0.007	...	0	0.18
1993									
Field 1	211	0	Linear-(Flat)	1.62	1.19	0.004	...	0	0.17
		90	Linear-(Flat)	5.64	1.39	0.007	...	0	0.16
		45	Linear-(Flat)	1.18	1.38	0.017	...	0	0.11
		135	Linear-(Flat)	1.28	1.40	0.010	...	0	0.10
Field 3-lower	217	0	Gaussian	0.74	1.24	10.08	11.32	>15	0.74
		90	Spherical	3.33	1.20	0.90	2.10	7.3	0.70
		45	Gaussian	0.35	1.36	15.88	17.24	>15	0.68
		135	Linear	0.47	1.08	0.273	...	>15	0.67
Field 3-upper	217	0	Linear-(Flat)	1.68	0.88	0.017	...	0	0.24
		90	Linear-(Flat)	1.16	0.88	0.023	...	0	0.10
		45	Linear-(Flat)	0.54	0.88	0.017	...	0	0.13
		135	Linear-(Flat)	21.8	0.96	0.001	...	0	0.07

[a] Direction of analysis refers to orientation of lag neighbors used in analysis in degrees azimuth (0° = within rows).
[b] Model refers to regression model that best fit the semivariogram according to weighted least squares analysis. Model resulting in lowest error mean square indicated best fit. Semivariograms that did not fit any model were described only by their shape (periodic curves).
[c] Nugget variance is Y intercept and represents microdistributional and measurement error. Values were determined from model parameters C_0 for nonlinear models or a for linear models.
[d] Values represent model parameters for structural variance C_1 ($C_0 + C_1$ = sill) for nonlinear models or slope b for linear models.
[e] Sill defined as semivariance value beyond range of spatial dependence. Parameter estimates of sill and range for periodic curves estimated visually from semivariograms and shown in parentheses.
[f] Range of spatial dependence defined as the lag distance at which the semivariance approaches a constant value and the distance at which samples are no longer spatially related. Values indicated as >15 represent ranges greater than largest lag distance measured.
[g] Correlation at lag 1 refers to correlation between adjacent quadrats (lag = 1 m) in each direction.
[h] Parameter unestimable by model used.

(Fig. 3D and E), but strong spatial dependence was evident within rows (0°) by day 211, as indicated by the strong positive linear slope of the semivariogram (Fig. 3F). The semivariogram for the across-rows (90°) direction was flat early in the epidemic, and then developed multiple peaks and valleys by the end of the season (Fig. 3D–F). This periodicity reflects fluctuations in disease correlations among quadrats and was probably due to the presence of multiple foci of disease in the across-row direction. Semivariogram characteristics varied considerably in the four directions of orientation, indicating strong anisotropy. The range of spatial dependence was greatest in the within-row direction, at >15 m, compared with 3–12 m for all other directions at the end of the season (Table 1). Spatial dependence also was observed in the diagonal directions, characterized by the exponential model at the end of the season (Table 1). Correlations among adjacent quadrats (lag distance = 1 m) were greater within rows than for all other directions (Table 1).

In field two in 1992, a large cluster of quadrats with diseased plants occurred early in the epidemic in the lower right-hand corner of the field (Fig. 4A). Disease spread from this cluster both within and across rows and an additional elongated cluster developed down rows near the center of the field by day 217 (Fig. 4B and C). Semivariograms representing the within-row (0°) and across-row (90°) directions reflected similar characteristics of range of spatial dependence, sill, and slope at days 168 and 188 (Fig. 4D and E), but at day 217 the semivariogram for the across-row direction revealed a periodicity caused by a second cluster of disease in the center of the field (Fig. 4F). Spherical and Gaussian semivariograms were observed for the 45 and 135° directions at day 217 (Table 1). The range of spatial dependence was greatest and correlations among adjacent quadrats were highest in the within-row direction at day 217 compared with all other directions (Table 1).

The lower portion of field three was dominated by a large cluster of quadrats with diseased plants in 1992, and disease was most severe in the center of the cluster and less severe around the periphery (Fig. 5A–C). Spatial dependence was evident and comparable in all four directions on all three assessment dates late in the season (Fig. 5D–F), and the range of spatial dependence was 14–19 m in all directions at day 223 (Table 1). Semivariograms for the within-row, 45, and 90° directions fit a Gaussian model, whereas the across-row direction was closest to a spherical model and had a lower sill than the other directions (Table 1). Range of spatial dependence was greatest in the 45° diagonal direction and corresponded with a low drainage area across the field in that direction. Correlations among adjacent quadrats were high in all directions (Table 1).

The upper portion of field three (Fig. 6A) had lower disease incidence and severity than the lower portion of the field in 1992 (Fig. 5C). Disease severity was variable in the scattered clusters of quadrats with diseased plants and disease was most severe in a diffuse, elongated cluster near the left side of the field at day 223. Disease severity for the upper portion of field three in 1993 was characterized by a diffuse elongate cluster on the right side of the field and scattered quadrats of low disease severity throughout the field (Fig. 6B). Two-dimensional distance class analysis indicated two clusters that did not change in size or shape over time, and aggregations of diseased plants were greater within rows than across rows. Disease severity also was lower in field one in 1993 than in 1992. Disease occurred in widely scattered clusters of low severity in field one in 1993 (Fig. 6C). Two-dimensional distance class analysis indicated that aggregations of diseased plants were small scattered clusters that did not consistently increase in size or number as the season progressed. In field one in 1993 and the upper portion of field three in 1992 and 1993, semivariograms were essentially flat and correlations among adjacent quadrats were low in all directions throughout the season, indicating that variation between quadrats was random and not spatially dependent (Table 1).

Two large clusters of quadrats with diseased plants occurred

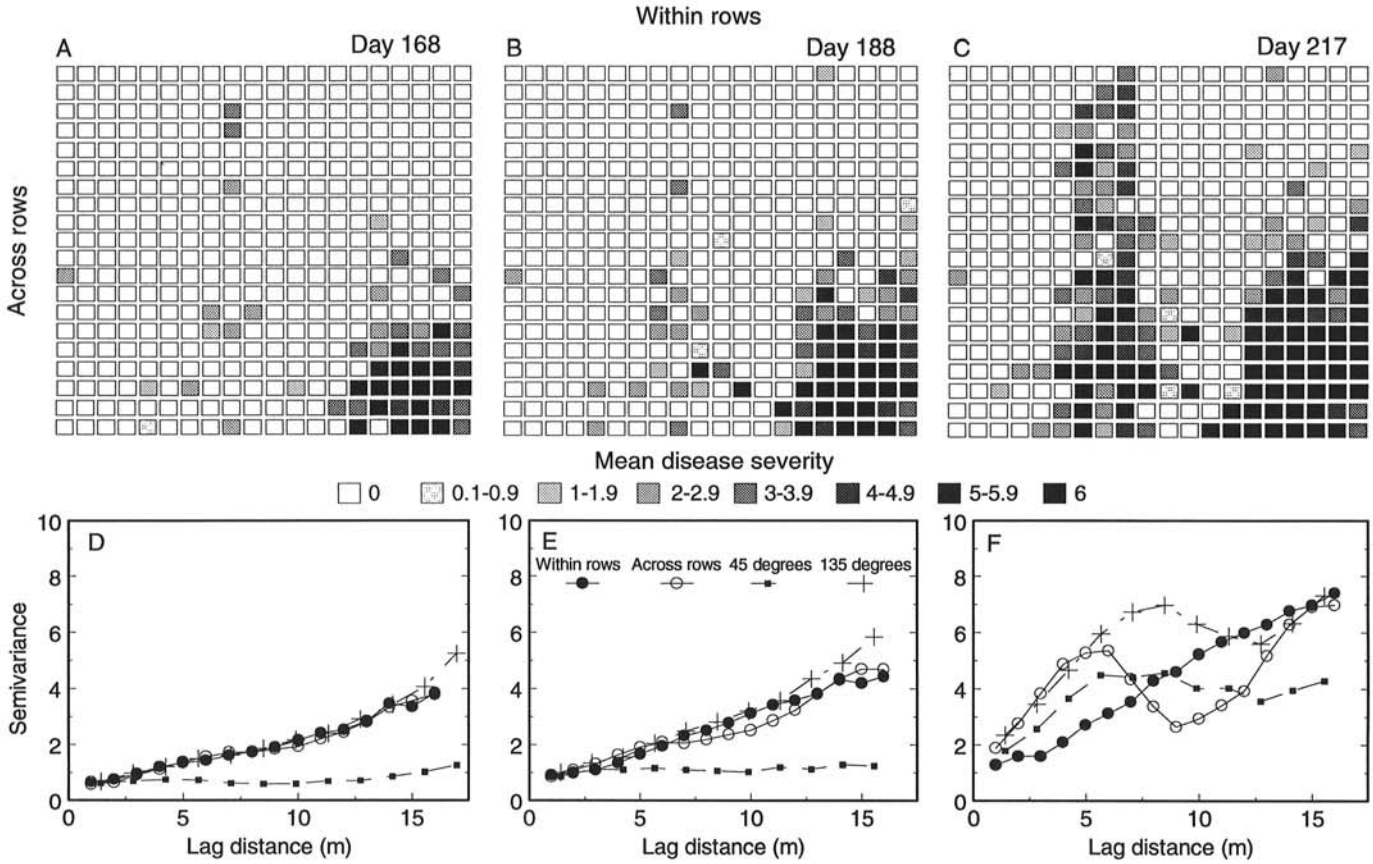

Fig. 4. Spatial pattern maps and semivariograms of disease severity caused by *Phytophthora capsici* on bell pepper in field two in 1992 on **A, D,** day 168, **B, E,** day 188, and **C, F,** day 217.

at opposite sides of the lower portion of field three on day 162 in 1993 (Fig. 7A). The clusters coalesced into one large cluster of severe disease encompassing two-thirds of the field by day 217 (Fig. 7B and C). Two-dimensional distance class analysis indicated a core cluster of 16 quadrats and a large reflected cluster of 54 quadrats on day 162, and one large cluster of 152 quadrats by day 217. Semivariograms were flat for all directions at the early stages of the epidemic (Fig. 7D), but distinct differences in spatial dependence due to directional effects were apparent by day 190 (Fig. 7E). A strong disease gradient was apparent in the within-row (0°) direction at day 217, whereas the semivariogram for the across-row (90°) direction was nearly flat, indicating very little disease gradient across rows (Fig. 7F). Correlations among adjacent quadrats were high in all directions by the end of the season (Table 1).

Inoculum of *P. capsici* in soil and soil water content. The spatial pattern maps and semivariograms for soil water content measured on day 181 in field one in 1992 indicated spatial dependence in all directions (Fig. 8A and C) and correlations among adjacent quadrats ranging from 0.61 to 0.65 for the four directions. The pathogen occurred mainly along the right side of the field, as indicated by the spatial pattern of quadrats containing *P. capsici* (Fig. 8B). Semivariograms of leaf disk colonization data indicated only a small degree of spatial dependence and correlations among adjacent quadrats were less than 0.3 for all but the within-row direction (data not shown). Spatial patterns of soil water content and leaf disk colonization were not highly correlated with each other ($r = 0.27$), or with the occurrence of disease at any time throughout the season (Table 2).

Semivariograms of soil water content for field two at day 171 indicated stronger spatial dependence than observed in field one in 1992. Soil water content correlations among adjacent quadrats for the four directions ranged from 0.80 to 0.85. A large area of high soil water content occurred in the lower-right corner of field two (Fig. 8D). There was a steep gradient from the lower-right corner to the upper-left corner of the field (135° direction), whereas there was little change in water content in the opposite direction (45°) (Fig. 8D and F). The spatial pattern for leaf disk colonization at day 171 indicated occurrence of the pathogen was scattered (Fig. 8E). Semivariograms of the percentage of leaf disks colonized indicated no spatial dependence in any direction (data not shown). The spatial pattern of soil water content was highly correlated with disease severity throughout the season (Fig. 4D–F, Table 2). Maximum correlation between soil water content and disease severity occurred on day 188 ($r = 0.67$) (Fig. 4E). Cross-correlograms indicated a strong spatial cross-dependence of soil water content with disease severity at day 188 (Fig. 9A). Cross-correlations between soil water content and disease severity in adjacent quadrats were high in all directions ($r = 0.67$–0.69), but correlations in the within-row direction remained high (>0.6) up to lag distances of 9 m.

Soil water content and pathogen population levels indicated strong spatial dependence and anisotropy in the lower portion of field three on day 172 in 1993 (Fig. 10A–D). Correlations among adjacent quadrats in the within-row direction for soil water content and pathogen populations were 0.58 and 0.66, whereas correlations among adjacent quadrats in all other directions were smaller, averaging 0.45 for soil water content and 0.27 for pathogen populations. Spatial patterns of soil water content (Fig. 10A) were highly correlated with the occurrence and severity of disease throughout the season, with the highest correlation occurring at the end of the season ($r = 0.65$) (Fig. 7C, Table 2). Spatial patterns of pathogen populations (Fig. 10B) were not highly correlated with disease severity at the end of the season ($r = 0.20$). Cross-correlations for soil water content and final disease severity among adjacent quadrats were high in all directions, but correlations remained high (>0.5) in the across-row direction at distances up to 15 m away (Fig. 9B). Soil water content was not correlated with pathogen population levels, however, and cross-correlations among adjacent quadrats were low (<0.2) in all directions.

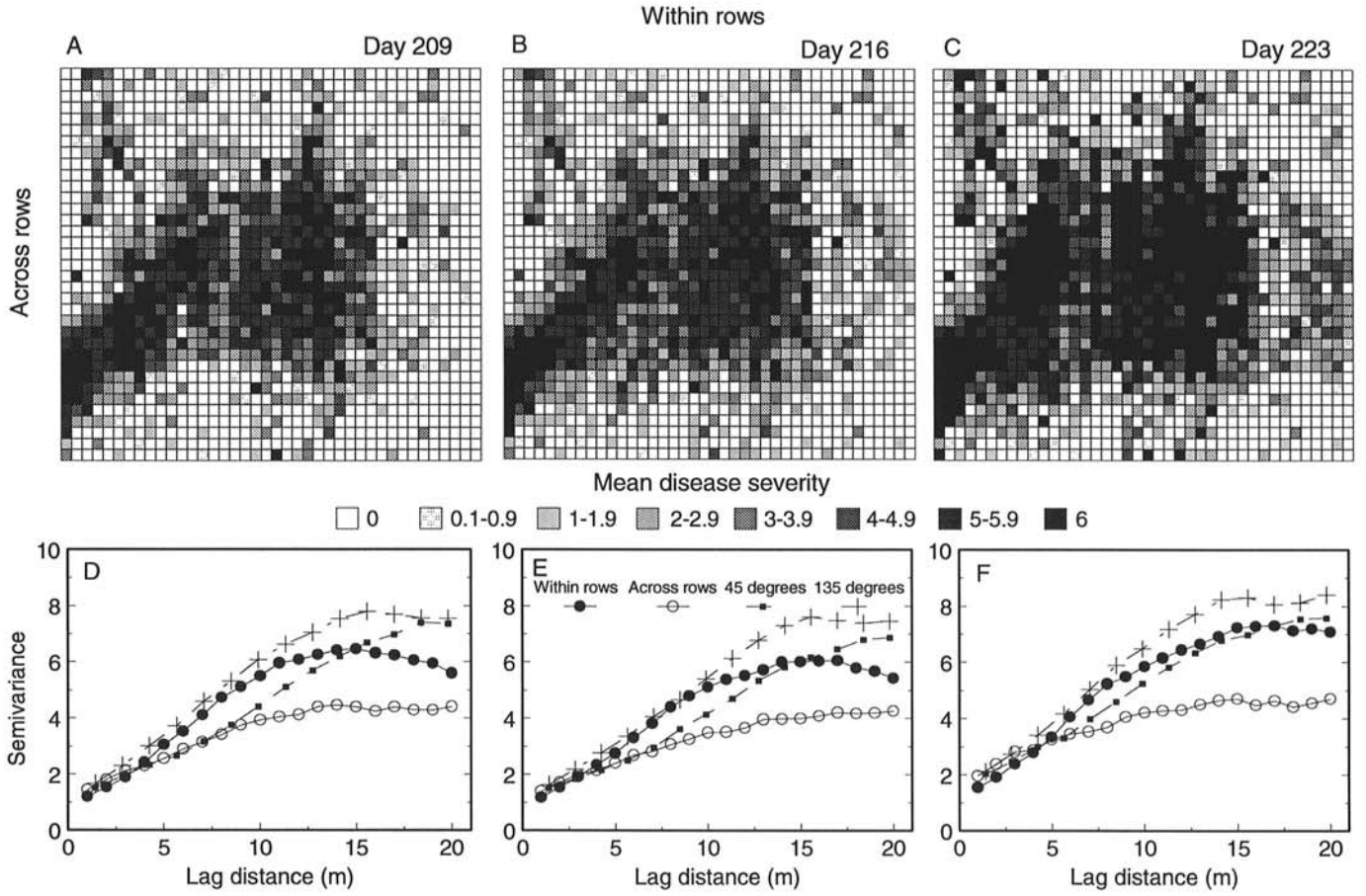

Fig. 5. Spatial pattern maps and semivariograms of disease severity caused by *Phytophthora capsici* on bell pepper in the lower portion of field three in 1992 on **A, D,** day 209, **B, E,** day 216, and **C, F,** day 223.

Semivariograms for soil water content measured on day 153 in field one in 1993 indicated spatial dependence in all directions (data not shown), and correlations among adjacent quadrats ranged from 0.53 to 0.59 for the four directions. No spatial dependence was observed for population levels of *P. capsici* in this field, however. Soil water content and propagule counts also were not correlated with the occurrence of disease in this field at any time (Table 2).

DISCUSSION

Spatial patterns of disease caused by natural inoculum of *Phytophthora capsici* in four of seven fields examined were not random, but were distinctly aggregated. In many of the fields, the degree of aggregation increased over time, as clusters of diseased plants expanded in several directions. Spatial patterns of soil water content early in the season also were aggregated and in three of four fields examined were correlated with disease severity. Population levels of *P. capsici* in the soil were not consistently spatially dependent and distribution of the pathogen early in epidemic development was not closely associated with the development of disease in these fields.

Disease incidence and severity observed in the 1993 field season followed trends similar to those in the 1992 season (33,34); however, some differences were observed. For example, in field one, disease incidence was slightly greater in 1993, but average disease severity was slightly lower than in 1992. Spatial patterns of disease also differed somewhat in these fields from 1992 to 1993. In field one, distinct clusters of diseased plants were evident in 1992 and grew in size throughout the season, whereas in 1993, disease in field one was scattered throughout the field and distinct clusters did not develop. Differences from year to year most likely resulted from differences in environmental factors and in distribution and level of initial inoculum. Rainfall was low during both summers, but lower rainfall during early July in 1993 may have restricted pathogen movement by water in these fields. In addition, assessment fields were located in the same approximate location, but were not in the same exact position from one year to the next.

Wilting in the absence of lesions was the earliest symptom observed on most plants and generally preceded the observation of crown lesions. Thus, infections probably began in the roots and subsequently spread to crowns. This suggests that root contact with inoculum, either by inoculum movement to roots, root growth to inoculum, root-to-root contact, or any combination of these, was important in epidemic development. In fields that showed spatial correlation, disease spread tended to be greater within rows than across rows, and disease spread quickly and unilaterally down rows. This suggests that movement of inoculum in surface water was an important dispersal mechanism. However, we did not sample for the presence of the pathogen in surface water to confirm this hypothesis. Movement of *P. capsici* in surface water has been shown to be an important dispersal mechanism in previous studies, both under natural conditions in the field and from inoculated point sources (1,31,33,34,36). Splash dispersal of inoculum was very limited in these fields, as evidenced by the absence or low occurrence of fruit, leaf, and stem lesions in 1992 and 1993 (33,34).

Soil water matric potential is known to have important effects on the development of *Phytophthora* diseases and on pathogen populations and dispersal (1,31,32,33). Soil water content was more highly correlated with disease development and spread than pathogen levels early in epidemic development in three of the four fields examined. Soil water content was most highly correlated with disease development in fields where there was a large moisture gradient, that is, where there were distinct low-lying areas of high soil water content as well as more well-drained areas of lower soil water content. Clusters of disease often corresponded with these low-lying areas where soil water content generally exceeded 10%. A gravimetric soil water content of 10% was equivalent to a soil water matric potential of about −10 J/kg (−100 mb) for all fields. In these cases, soil water content could be used to identify and differentiate the more disease-conducive

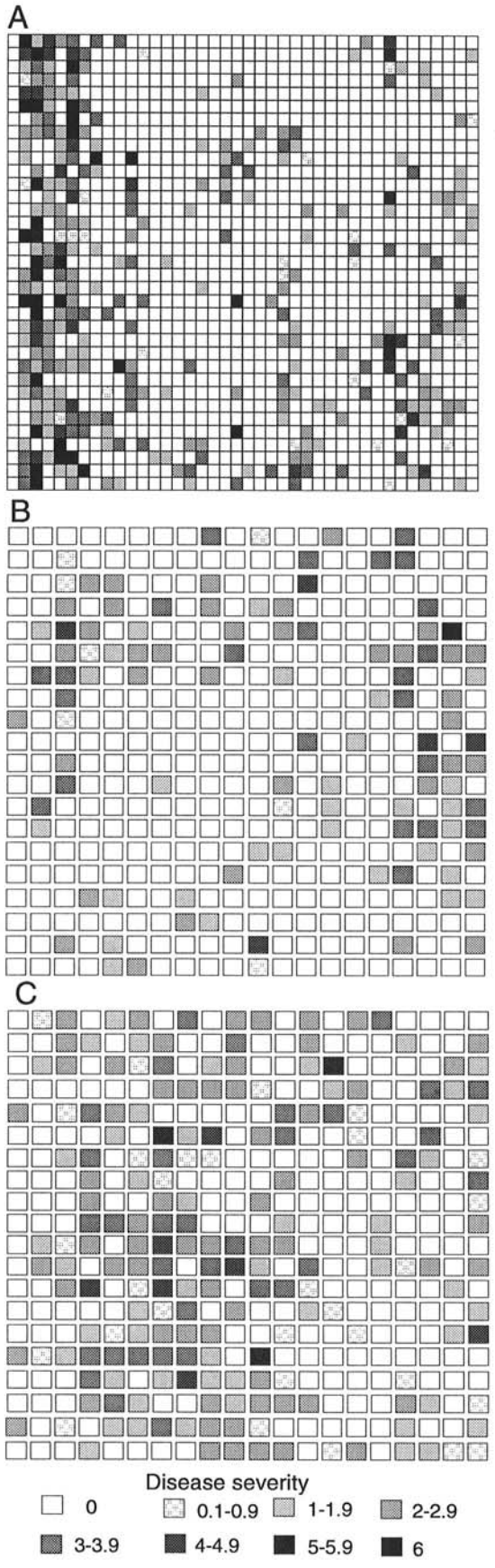

Fig. 6. Spatial pattern maps of disease severity caused by *Phytophthora capsici* on bell pepper in **A,** the upper portion of field three on day 223 in 1992, **B,** the upper portion of field three on day 217 in 1993, and **C,** field one on day 211 in 1993.

areas of the field. Within clusters of high soil water content, disease spread was comparable in both within- and across-row directions due to inoculum movement in soil and surface water. In the lower portion of field three in 1993, which had a large low-lying wet area, disease covered the entire width of the field in 1993 and then spread outward from this area primarily within rows (Fig. 7).

Our data demonstrate that even when adequate inoculum is present, soil water has a major effect on epidemic development. Generally, when soil water content was favorable for disease development in these soils, disease occurred. A soil water content of 10% was comparable with a soil matric potential of approximately −10 J/kg (−100 nb) for all fields. Although the pathogen was widely distributed in most of these fields, disease developed primarily in the areas of higher soil water content. Since soil water content was evaluated only once or twice during the season in each field, the values at the time of the assay may not represent average conditions. However, the patterns of moisture gradients, which permit us to distinguish areas of the field that tend to be wetter from those that tend to be drier, are assumed to be representative of conditions over time. These moisture gradients provided high correlations between soil water content and disease even in fields in which soil water content was fairly low at the time of the assay, such as the lower portion of field three in 1993.

The spatial pattern of *P. capsici* inoculum, as determined in this study, was generally not aggregated and was not highly correlated with disease development. In contrast, many investigators have found this pathogen and similar soilborne pathogens often are highly aggregated in the soil (1,3,32). All of the fields in our study had a history of *Phytophthora* epidemics and the pathogen may have been widely distributed throughout the fields prior to our study. Furthermore, neither soil assay method used in these tests was entirely satisfactory. Oospores generally do not germinate on agar media and are not detected by soil dilution plating, but are thought to be the overwintering propagule for this pathogen. Colony morphology on soil dilution plates also is not definitive and may lead to inaccurate estimates. The leaf disk assay generally provides better detection of oospores, but does not accurately quantify pathogen density. In short, current methods of estimating inoculum in the field may not accurately represent the true pathogen density and distribution patterns. We are continuing experiments to improve our abilities to quantify *P. capsici* populations in soil. The lack of correlation of pathogen levels to disease may also be related to the polycyclic nature of epidemics of *P. capsici*. Initial inoculum may only be important for establishing initial foci of disease, with subsequent spread associated mainly with secondary inoculum.

Disease severity in four of the seven fields examined showed strong spatial dependence and anisotropy. The direction of maximum spatial variation was within rows for three of the four fields, indicating a distinct disease gradient increasing over time. In each of these fields, disease severity in the within-row direction demonstrated the strongest spatial dependence, as evidenced by higher correlations, larger ranges, and increasing slope. The range of spatial dependence was often >15 m (15 quadrats) in the within-row direction at advanced stages of the epidemic. Large ranges observed for the diagonal directions in some fields were the combined result of within- and across-row disease spread and may also have been due to drainage of surface water in these directions. Overall, disease severity among quadrats became more spatially dependent over time, and the range, sill, correlation, and slope parameters all increased over time.

Semivariograms were best described using a variety of linear and nonlinear models in this study. Although many of the curves could be approximated by a linear function with a sill, the best fits were generally provided by the spherical or Gaussian models. These models provided improved estimation of semivariogram parameters. A few semivariograms could not be fit to any existing model. These were periodic in shape due to fluctuating disease gradients and may have been caused by the occurrence of addi-

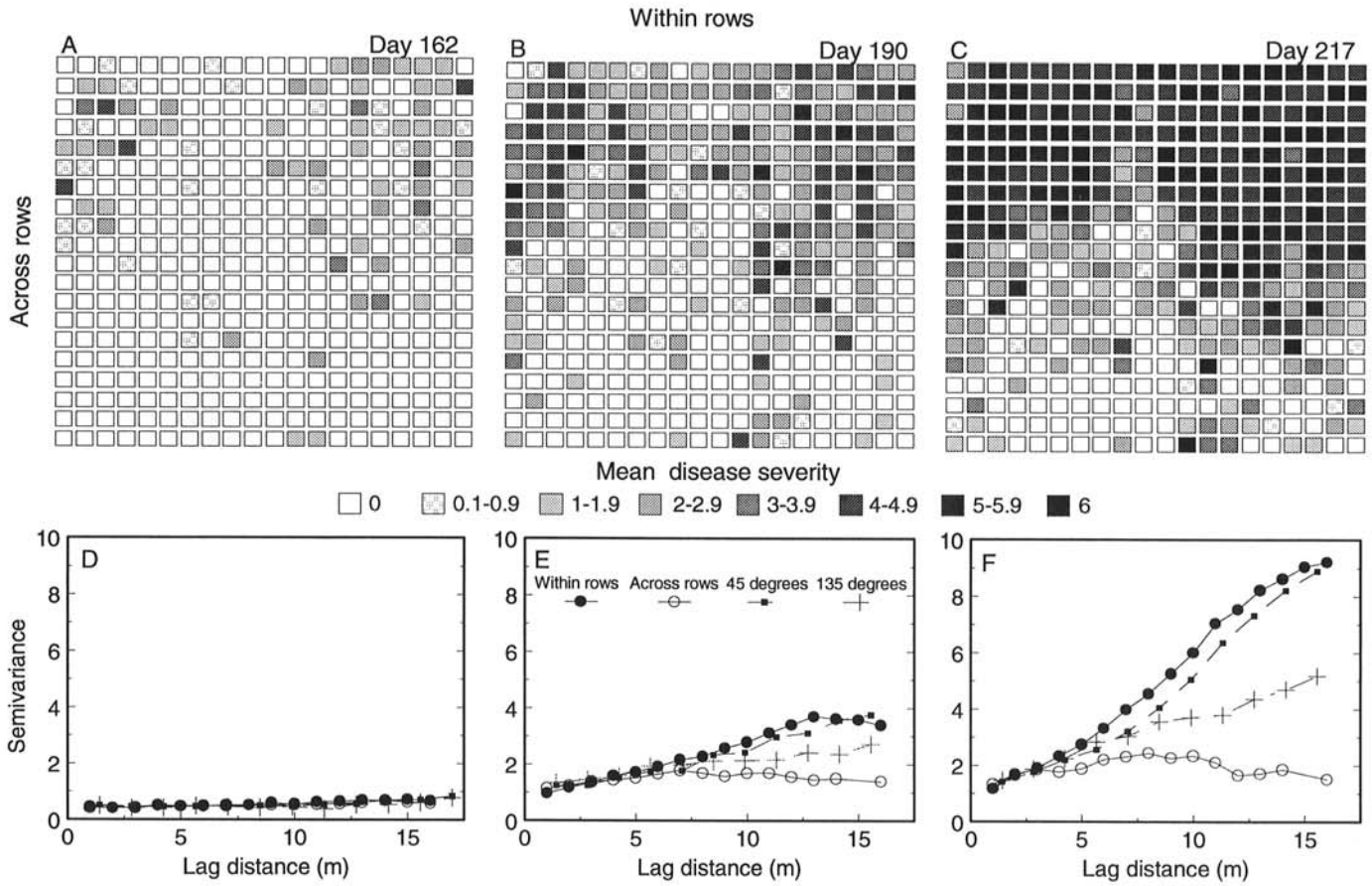

Fig. 7. Spatial pattern maps and semivariograms of disease severity caused by *Phytophthora capsici* on bell pepper in the lower portion of field three in 1993 on days **A, D,** 162, **B, E,** 190, and **C, F,** 217.

tional disease foci. Periodic semivariograms were observed primarily in the across-row direction and indicated less pathogen movement in that direction than any other. Because of the limited size of the sampling area (generally 20 × 20 grids of 1-m^2 quadrats), semivariance often did not level off within the limits of the lag distances tested. This indicates that disease in quadrats was spatially related beyond the distances measured.

Semivariograms for some fields indicated the presence of a steep gradient in one direction and virtually no gradient in the opposite direction, as for example, soil water content in field 2 in 1992 (Fig. 8D and F). This gradient could also be viewed as a nonstationary spatial trend in mean water content and modeled as a function of row and quadrat position. If the mean were modeled this way, the residual spatial correlation would

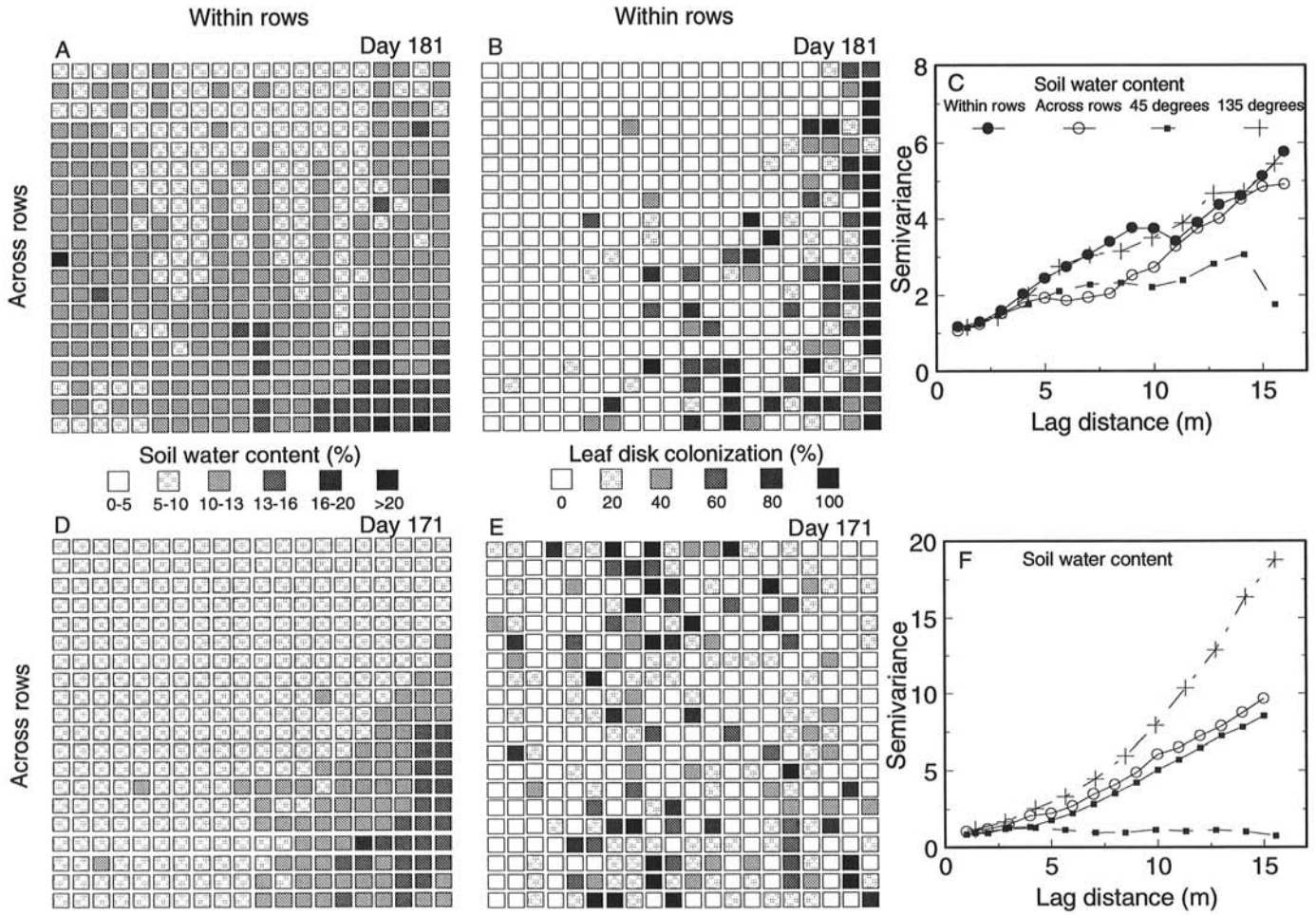

Fig. 8. Spatial pattern maps and semivariograms of gravimetric soil water content and soil population levels of *Phytophthora capsici* in **A–C,** field one on day 181 in 1992, and **D–F,** field two on day 171 in 1992. Pathogen population levels were estimated by a leaf disk bioassay (% leaf disks colonized).

TABLE 2. Gravimetric soil water content (%) and soil population level estimates of *Phytophthora capsici* and their correlation with disease severity in four commercial bell pepper fields

	Soil water content[a]					Pathogen population level[b]					
			Correlation coefficients (r)[c]					Correlation coefficients (r)[d]			
Year/field	Range of values (%)	Average ± (SD) (%)	Early disease	Mid-disease	Final disease	Range of values	Average ± (SD)	Early disease	Mid-disease	Final disease	Soil water
1992						% Leaf disks colonized					
Field 1	6.2–25.8	10.8 (1.8)	0.13	0.10	0.16*[e]	0–100	14.7 (29.2)	0.07	0.03	0.08	0.27*
Field 2	5.2–26.0	8.8 (2.4)	0.60*	0.67*	0.53*	0–100	18.3 (27.8)	0.08	0.11	0.23*	0.01
1993						Cfu/g soil					
Field 1	7.2–23.8	10.4 (2.1)	−0.02	−0.05	−0.11	0–47.3	2.6 (3.2)	0.10	0.12	0.06	−0.02
Field 3-lower	2.4–13.9	5.2 (1.7)	0.54*	0.61*	0.65*	0–68.0	10.3 (10.4)	0.07	0.13*	0.20*	0.15*

[a]Gravimetric soil water content measured on composite soil samples from each quadrat collected early in epidemic development. A soil water content of 10% was comparable to a soil water matric potential of about −10 J/kg (−100 mb) for all fields.
[b]Population levels of *Phytophthora capsici* in soil were determined from composite soil samples for each quadrat collected early in epidemic development by a leaf disk bioassay in 1992 (% leaf disks colonized) and by soil dilution plating in 1993 (cfu/g soil).
[c]Correlation coefficients refer to Pearson's product-moment correlations of soil water content with disease severity ratings in each quadrat at early, middle, and final stages of epidemic development.
[d]Correlation coefficients refer to Pearson's product-moment correlations relating pathogen population levels with disease severity ratings at early, middle, and final stages of epidemic development, and for inoculum levels with soil water content.
[e]Asterisks represent correlation coefficients that were significant at $P < 0.01$.

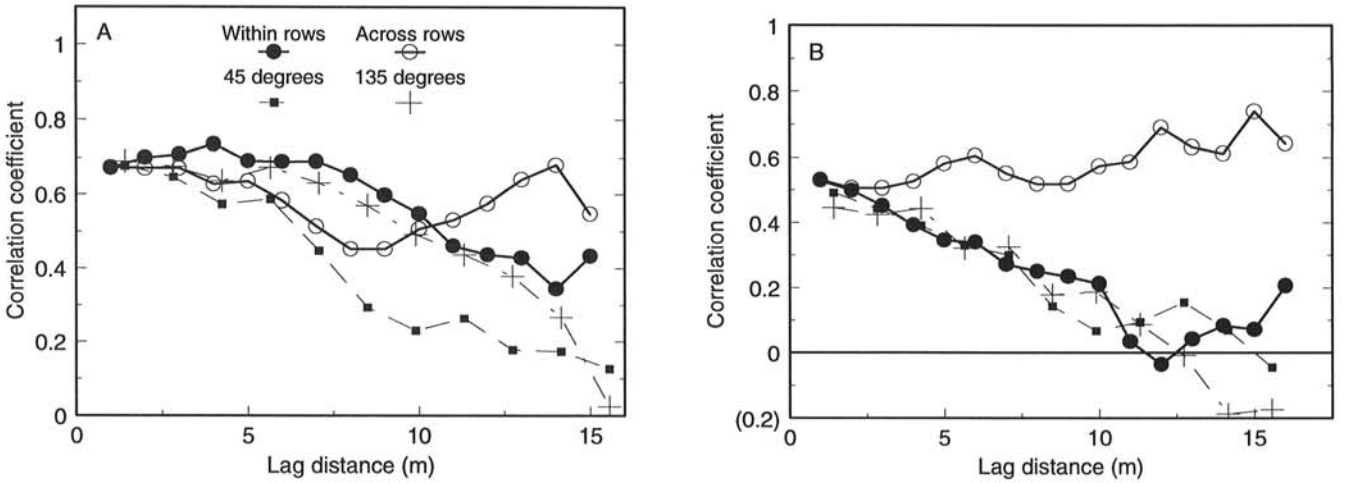

Fig. 9. Cross-correlograms for soil water content and disease severity in **A,** field two on day 188 in 1992, and **B,** the lower portion of field three on day 217 in 1993.

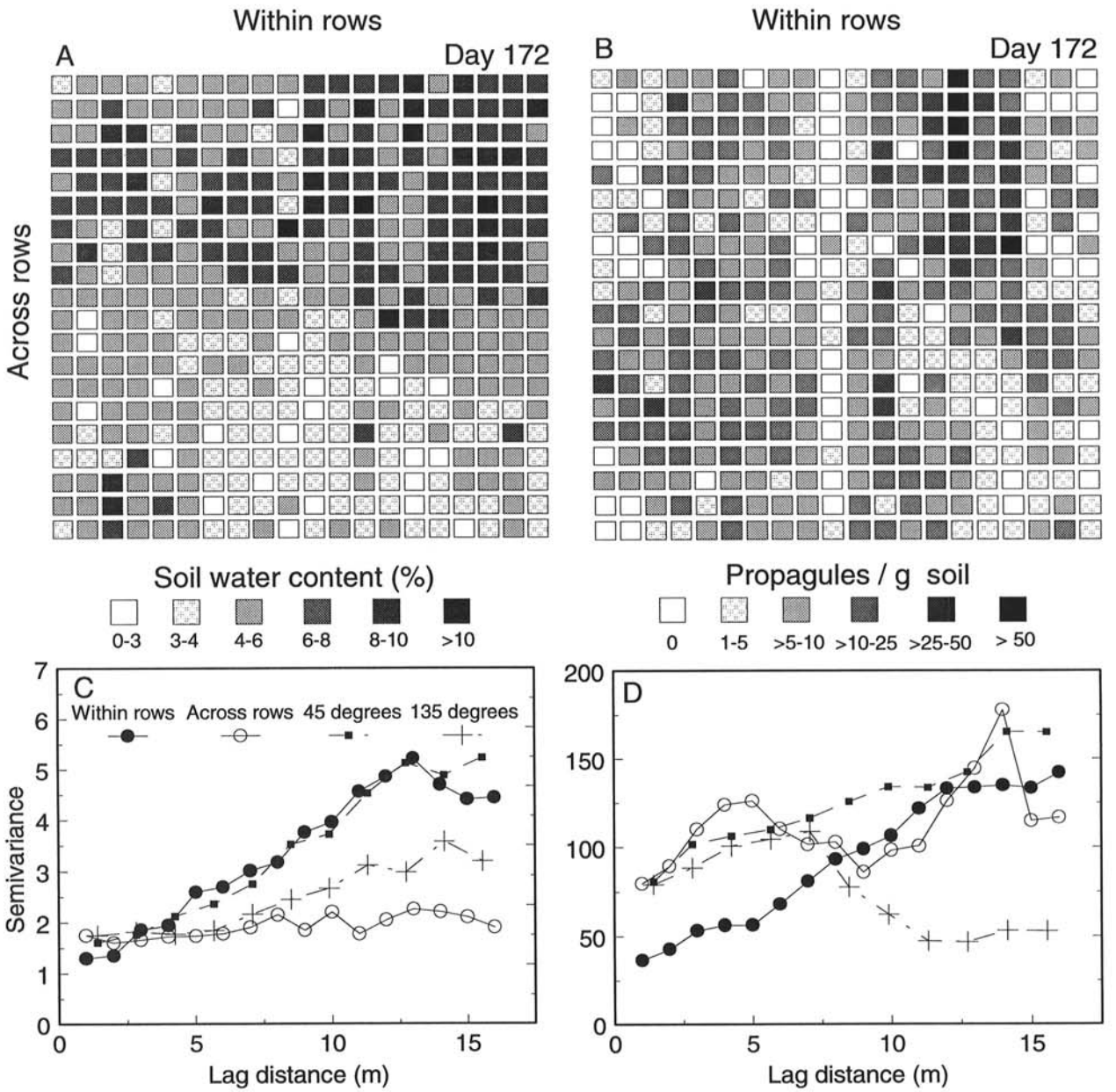

Fig. 10. Spatial pattern maps and semivariograms of **A, C,** gravimetric soil water content and **B, D,** soil population levels of *Phytophthora capsici* in the lower portion of field three on day 172 in 1993. Soil population levels were estimated as cfu/g soil by soil dilution plating.

be low in all directions and no anisotropy would be evident in the residuals. The anisotropy would show up as a directionality in the trend function for the mean. However, rather than remove this trend through detrending procedures, we chose to incorporate such spatial trends into our analysis and use the semivariogram to describe the relationships among various distances and directions of quadrats across the field. In this way, spatial variation could be compared among quadrats in all directions.

The range of spatial dependence for disease severity in fields in our study averaged 15–20 m. The ranges of spatial dependence reported for other pathosystems have depended largely on the scale of the experiment and the type of organism involved. Chellemi et al (3), using small study plots (3.6 × 3.6 m grid), observed ranges of >2 m for *P. nicotianae* var. *parasitica* on pineapple. Munkvold et al (24) reported ranges of 15–25 m for Eutypa dieback disease of grapes. Lecoustre et al (17) found ranges of >120 m for the whitefly-vectored African cassava mosaic virus. Todd and Tisserat (38) observed ranges of spatial dependence of 10–16 m (24 × 21 m grid) for the distribution of two nematode species on creeping bentgrass.

Although two-dimensional distance class analysis and geostatistics use very different approaches to analyze spatial variation and work with different types of data, the results from each analysis indicated similar overall conclusions for these fields (33,34). Both analyses indicated predominant disease spread down rows and quantified various aspects of spatial variation. Two-dimensional distance class analysis provided assessments of cluster size and shape and geostatistics added the range of spatial dependence and the degree of influence in multiple directions. Geostatistical techniques were better able to quantify subtle aspects of disease spread using disease severity measurements and gradients. Variation of disease severity within clusters was apparent and geostatistics allowed the differentiation of multiple foci within large clusters of disease, as evidenced by periodic semivariograms. Two-dimensional distance class analysis is designed to detect aggregation and was more sensitive than geostatistics in detecting overall aggregation. Spatial patterns of disease for all fields were found to be aggregated using two-dimensional distance class analysis, although field one and the upper portion of field three in 1993 had relatively weak aggregation (<15% SCFs for individual symptom types). Geostatistical analysis, on the other hand, does not specifically test for aggregation and did not detect systematic spatial correlation in field one in 1993 and the upper portion of field three in 1992 and 1993. When disease incidence and severity are low, spatial correlations also tend to be low. At low levels of disease, disease gradients may not be evident or are erratic, and spatial patterns of disease severity may not be distinguished from random patterns.

Geostatistics provided additional tools for the quantitative analysis of spatial distribution of disease data and enabled us to characterize attributes of epidemic development, such as the range and degree of spatial dependence in multiple directions over time. Geostatistical analysis also allowed the evaluation of spatial patterns of pathogen populations and soil water content as well as their relationship to disease development. The result is a more comprehensive understanding of the processes that influence the spatial pattern of disease and plant pathogen populations. These spatial correlation patterns may help us predict the probability of disease in naturally infested fields and also improve sampling strategies at different scales in the future.

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