Spatial and Temporal Patterns of Spotted Wilt Epidemics in Peanut

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ABSTRACT

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The spatio-temporal patterns of spotted wilt disease in peanut, caused by thrips-vectored tomato spotted wilt tospovirus (TSWV), were analyzed by several methods. The spatial distributions of symptomatic plants were mapped at 2-wk intervals during two consecutive years in fields of the susceptible peanut cultivar Florunner and in the resistant cultivar Southern Runner. The disease frequency data were subjected to quadrat analyses, including indices of dispersion (variance/mean ratio and the negative binomial k parameter), comparison of frequency distributions of symptomatic plants to expected distributions derived from several models of spatial dispersion (binomial, Poisson, and negative binomial), and two-dimensional distance class analysis at each sampling interval. Although significant spatial aggregation of diseased plants was detected in most samples, random or nearly random clusters of infected plants that apparently arose from a continuous immigration of viruliferous vectors dominated spatial aspects of epidemic progress. Aside from smaller incidence of disease in the resistant compared to the susceptible cultivar, no effect of cultivar was noted in relation to spatial or temporal patterns of disease development in this pathosystem. Spotted wilt temporal progress was well described by monomolecular models of disease progress in each cultivar and year. The data and analyses were consistent with the hypothesis that most infections arise as a result of primary transmission and that there is limited secondary spread of TSWV after it becomes established in the field.

Additional keywords: *Arachis hypogaea*, spatial analysis, Thysanoptera.

Spotted wilt, caused by tomato spotted wilt tospovirus (TSWV), is now recognized as a serious threat to peanut (*Arachis hypogaea* L.) production in the southeastern United States. Severe damage occurred to peanut in Texas during 1986, 1990, and again during 1991, with yield reductions of up to 95% (1,4,5,6). Incidence of spotted wilt in Georgia peanut has increased dramatically since 1986 (14,15,16). By 1990, field surveys in Georgia reported infections in 100% of sampled fields in 10 counties. Although the economic impact of spotted wilt on peanut in Georgia has been limited, the severe losses in Texas peanut and similarly drastic reductions in both pepper and fall tomato in Georgia during 1990 emphasize the potential for significant damage.

TSWV is vectored by thrips (Thysanoptera). Two of the seven known vector species, the tobacco thrips, *Frankliniella fusca* (Hinds), and the western flower thrips, *F. occidentalis* (Pergande), occur in most peanut-producing areas of the southeastern United States (3, 35,36,41). TSWV is acquired by immature thrips feeding on infected host plants. Larvae maintain the virus but usually do not transmit it. Virus transmission occurs primarily through the feeding activities of adult thrips, who have a midgut barrier that prevents virus acquisition (42). Ullman et al. (43) have recently reported that TSWV replicates within vectors, rendering them capable of transmitting the virus for long periods of time; viruliferous adult thrips, therefore, potentially are able to infect many plants. We have observed that spotted wilt symptoms can appear as early as 10 days after infection but may take much longer, possibly making assessments of disease incidence based solely on visual surveys of symptomatic plants prone to underestimation. Some infected plants, while yielding a positive serological assay for TSWV, never exhibit symptoms (17).

The transmission dynamics of TWSV in peanut are poorly understood. Reddy et al. (34) reported that primary infection played a larger role in the development of spotted wilt epidemics than did secondary spread from plant to plant, but their conclusions were inferred from thrips population data rather than from analyses of disease progress or spatial pattern. Additionally, the Indian tospovirus, causing bud necrosis disease in peanut, is known to differ significantly from the U.S. isolate (37) and is now reclassified as bud necrosis virus. These factors warrant a separate examination of spotted wilt transmission and disease progress in the southeastern peanut-producing region of the United States, where the disease is known to be caused by TSWV.

An analysis of the spatial attributes of spotted wilt disease is important in understanding and managing epidemics (9,10,12,24, 27,28,29,40). Spatial patterns of disease are influenced by a variety of biotic and abiotic factors operating at scales ranging from the pathogen's interaction with individual host plants to community- and landscape-level ecological processes that influence the dispersal and host-selection behavior of insect vectors. As the disease progresses, patterns of infected plants may undergo temporal change relative to one another and the architecture of the host crop (21). For example, disease transmission through primary infection is often associated with an initially random spatial distribution of infected plants (13,24,25,27,40), although truly random patterns of infection are infrequent in plant pathosystems (26). Secondary spread from one infected host plant to adjacent plants frequently leads to clusters of infected plants. Cluster size, location, and density of symptomatic plants can provide information about the rate of disease spread, the underlying dynamics

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of virus transmission, vector dispersal, and the effects of cultural practices (22,23,24,27,29,30,33,40). Cultivar resistance can affect the spatial pattern of diseased plants by lengthening the latent period and lowering virus acquisition efficiency of the vector, although extreme resistance is probably necessary to completely disrupt the characteristic patterns of particular epidemics (29).

This study analyzes the spatial and temporal development of spotted wilt epidemics in peanut fields under cropping systems typical in Georgia. We examined disease progress data from two growing seasons that had different levels of disease incidence. In addition, we looked for a cultivar effect by simultaneously analyzing the development of epidemics in the susceptible peanut cultivar Florunner and in the moderately resistant cultivar Southern Runner. Our objectives were to gain insight into the development of spatial pattern in spotted wilt epidemics over time and the relative importance of primary and secondary virus transmission.

MATERIALS AND METHODS

Experimental plots and data collection. Two 22.8 × 152-m plots of peanut were planted in 1989 and 1990 on the University of Georgia Attapulgus Research Farm in Attapulgus, on Dothan loamy sand. One was planted with the TSWV-susceptible cultivar Florunner and the other with the moderately resistant cultivar Southern Runner (7,18). There were 24 rows of plants parallel to the long dimension in each field, separated by 0.9 m in accordance with commercial practices typical of peanut cropping systems in the southeastern United States. Plots were planted on 13 April in 1989 and 1990. No insecticides were applied for thrips control. Complete details of field preparation and the subsequent cultural practices were described previously by Culbreath et al. (18).

Beginning on 12 June 1989 and 30 May 1990, each field was examined five times at 14-day intervals for plants with symptoms of spotted wilt. Peanut germination and stand establishment requires approximately 2 weeks for early-season plantings when the soil temperature is still relatively cool, and although virus transmission can begin as soon as viruliferous thrips begin to enter the field and feed on the developing plants, it is not unusual for the widespread appearance of symptoms to require several more weeks. The initial survey dates were chosen because they closely followed the general appearance of spotted wilt symptoms. Symptomatic plants were marked with colored surveyor's flags, using a different color on each survey date, preserving both the location and detection date of each symptomatic plant. Tissue samples were taken from symptomatic plants for serological testing (enzyme-linked immunosorbent assay [ELISA]) as previously described (18). In 1989, every symptomatic plant was assayed; in 1990, every tenth symptomatic plant in each row was tested. The location of each symptomatic plant was recorded prior to harvest. In addition, the mean within-row plant spacing and number of plants per field were estimated by sampling 2 rows × 3.0 m × 10 replications in 1989 and 2 rows × 7.6 m × 25 replications in 1990.

Data analysis. Maps of each field were digitized and divided into contiguous 0.9 × 0.9-m quadrats arrayed in 24 rows × 166 columns (3,984 total), with each row of quadrats centered over a row of peanut. Quadrat dimensions reflected the spacing between rows, produced square quadrats without overlap, and assured that each quadrat contained plants (23). The analysis routines allowed pooling of adjacent quadrats to increase quadrat size (9). Whenever this was necessary, quadrats were first combined along rows (parallel to the long edge of the fields), followed by pooling across rows (Table 1, footnote).

Symptomatic plants were mapped into the matrices to within 0.03 m of their position in each field by translating the [row, distance] coordinates of each symptomatic plant to the nearest corresponding matrix coordinates, using:

$$i_n = 14 + r(d_r/0.03)$$
$$j_n = 1 + \text{integer}\ (d_c/0.03)$$

where i_n and j_n are the matrix coordinates of the n^{th} symptomatic

TABLE 1. Tomato spotted wilt tospovirus (TSWV) incidence, indices of dispersion and $\chi 2$ goodness-of-fit to Poisson and negative binomial frequency distributions for TSWV-infected peanut at Attapulgus, GA

Year	Cultivar	DPP[a]	y[b] ± SE	Quadrat size (m)	VM[c]	k[d]	Poisson		Negative binomial	
							χ^2	P[e]	χ^2	P
1989	Southern Runner	61	0.001 ± 2.3e-5	7.2 × 3.6	1.37	0.22	‡[f]	‡[f]	3.40	0.07
	Southern Runner	75	0.002 ± 6.0e-5	3.6 × 3.6	1.39	0.06	9.25[b]	**	0.99	0.61
	Southern Runner	89	0.003 ± 1.2e-4	3.6 × 1.8	1.35	0.11	33.07	**	2.94	0.23
	Southern Runner	103	0.006 ± 2.2e-4	1.8 × 1.8	1.42	0.17	87.02[d]	**	3.80	0.28
	Southern Runner	118	0.007 ± 2.5e-4	1.8 × 0.9	1.45*[e]	0.18	184.23	**	6.39	0.09
1989	Florunner	61	0.002 ± 5.4e-5	7.2 × 3.6	1.48	0.03	6.18	0.05	0.04	0.98
	Florunner	75	0.005 ± 1.6e-4	1.8 × 1.8	1.50	0.11	200.84[d]	**	1.81	0.61
	Florunner	89	0.010 ± 3.1e-4	1.8 × 0.9	1.64**	0.17	361.53	**	1.13	0.77
	Florunner	103	0.015 ± 4.8e-4	0.9 × 0.9	1.58**	0.28	511.28	**	1.09	0.78
	Florunner	118	0.017 ± 5.4e-4	0.9 × 0.9	1.58**	0.31	498.53	**	3.90	0.27
1990	Southern Runner	48	0.011 ± 3.2e-4	1.8 × 1.8	0.97	†[g]	1.35[d]	0.51	1.32	0.25
	Southern Runner	62	0.022 ± 6.2e-4	1.8 × 0.9	1.09	1.02	4.47	0.11	0.09	0.77
	Southern Runner	76	0.046 ± 1.3e-3	0.9 × 0.9	1.32**	0.58	149.49	**	5.22	0.16
	Southern Runner	90	0.064 ± 1.8e-3	0.9 × 0.9	1.41**	0.63	245.94	**	4.06	0.25
	Southern Runner	104	0.075 ± 2.1e-3	0.9 × 0.9	1.40**	0.75	323.17	**	4.47	0.22
1990	Florunner	48	0.025 ± 3.3e-4	0.9 × 0.9	1.10	1.46	18.47	**	0.62	0.43
	Florunner	62	0.080 ± 1.1e-3	0.9 × 0.9	1.46**	1.09	398.68	**	3.68	0.45
	Florunner	76	0.178 ± 2.4e-3	0.9 × 0.9	1.65**	1.59	910.81	**	5.68	0.46
	Florunner	90	0.235 ± 3.1e-3	0.9 × 0.9	1.58**	2.27	692.78	**	10.96	0.14
	Florunner	104	0.265 ± 3.5e-3	0.9 × 0.9	1.58**	2.47	701.44	**	15.75	0.03

[a] DPP = Days postplanting.

[b] y = Disease incidence.

[c] VM = Variance/mean ratio.

[d] k = negative binomial k parameter.

[e] * = $P < 0.05$; ** = $P < 0.01$.

[f] There was insufficient data to fit this model.

[g] Estimated value for k was less than zero.

plant (14 matrix rows represent the unplanted field margin at the top and bottom of the fields), r is the field row containing the n^{th} symptomatic plant, d_r is the distance (in meters) between planted field rows (the unplanted margin on the left side of the fields begins at j_1), and d_c is the distance from the left field edge to the n^{th} symptomatic plant.

Frequency distributions of symptomatic plants per quadrat were compared to expected distributions under binomial, Poisson, and negative binomial models by a χ^2 goodness-of-fit test to assess their spatial dispersion as regular, random, or aggregated, respectively (19,20). Field patterns of disease were considered aggregated if the observed frequency distribution of numbers of diseased plants per quadrat was indistinguishable from expected values under the negative binomial model at $P < 0.05$. In several instances, there was insufficient data at the smallest quadrat sizes to calculate a χ^2 statistic with degrees of freedom greater than 0 after expected frequencies less than 1 were combined in the tails of the distributions. Whenever this occurred, adjacent quadrats were combined until a χ^2 value could be calculated; the smallest quadrat size for which a χ^2 statistic could be tested then was substituted for the usual 0.9 × 0.9-m quadrats. When necessary, quadrats were first combined parallel to the long edge of the field, then parallel to the short edge. Additional combinations alternated in similar fashion. For illustration purposes, the expected (discrete) frequencies under binomial, Poisson, and negative binomial models were smoothed using cubic spline approximations to make the figures easier to interpret (2,38). Aggregation of symptomatic plants also was assessed using the variance/mean ratio (39,40) and the negative binomial k parameter (20,40). The significance of values obtained for variance/mean deviations from unity were determined with a t test (20).

Maps of spotted wilt incidence from 1990 were subjected to two-dimensional (2D) distance class analysis (24,29,30,31, and 33 for additional background material). The counts of symptomatic plants in the 24 × 166-quadrat sampling lattice were converted to binary (presence/absence) data. Two-dimensional distance class analysis compared the standardized count frequencies (SCF) of diseased plants in each [X,Y] distance class to lattices of expected random SCF generated by Monte Carlo simulations. All data sets subjected to 2D-distance class analysis had mean quadrat incidences of disease between 0.20 and 0.85 and were analyzed using 400 sets of simulated data to model the expected SCF. Random numbers for the Monte Carlo simulations were obtained with a long period ($> 2 \times 10^{18}$) pseudorandom number generator (32). Significance levels for the observed SCF and their 95% confidence limits were calculated as described by Gray (24). [X,Y] distance classes were accepted as having significantly higher numbers of infected quadrats than expected when $P < 0.05$ and fewer infected quadrats than expected when $P > 0.95$. Data were summarized with regard to average cluster size, relative strength of aggregation, cluster shape, and the strength of edge effects.

The proportions of symptomatic plants in each field were fitted to the monomolecular model of disease progress, $y = 1 - \beta e^{rt}$, where y is disease incidence, β and r are fitted parameters (r is a rate coefficient), and t is the time since planting (9). Nonlinear regressions were performed with the S-plus data analysis package (38). Model performance was evaluated using the residual standard error (RSE) and R^2 (the coefficient of determination).

RESULTS

Disease incidence and indices of dispersion. In 1989, estimated numbers of plants (±SE) in each field were 49,860 ± 1,868 plants for Southern Runner and 44,520 ± 1,462 plants for Florunner. Plant numbers were lower for both cultivars in 1990, with approximately 16,502 ± 354 plants in the Southern Runner field and 25,110 ± 453 plants in the Florunner field. Over 95% of the plants identified as symptomatic by visual inspection were confirmed by a positive ELISA test for TSWV infection in both years (18).

In 1989, the proportion of symptomatic plants was low in both Florunner and Southern Runner (Table 1). By the final field-assessment date at 118 days after planting, fewer than 1% of Southern Runner plants exhibited symptoms of TSWV infections, and approximately 1.7% of Florunner was symptomatic. The variance/mean ratio of symptomatic plants per quadrat in 1989 exceeded unity significantly ($P < 0.05$, df = 3,981; Table 1) on the last field assessment, 118 days postplanting (DPP) in Southern Runner and on the final three assessments in Florunner, beginning on 89 DPP. Negative binomial k parameters were less than one for all field

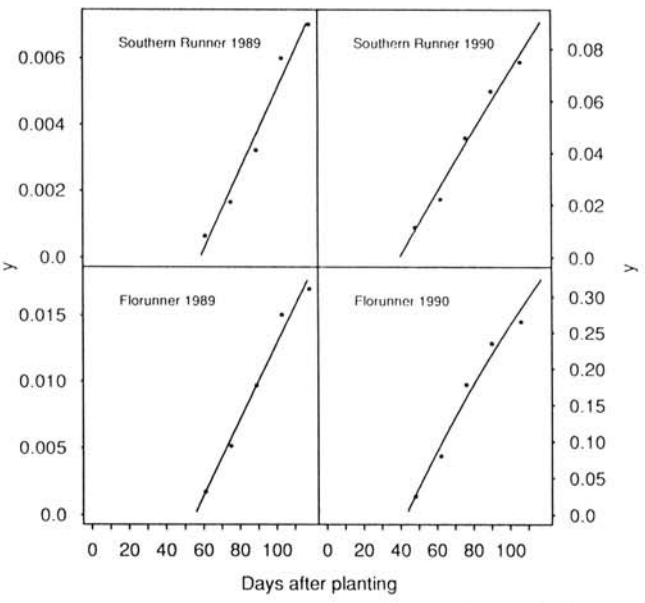

Fig. 1. Temporal progress of tomato spotted wilt tospovirus epidemics on peanut at Attapulgus, GA, fitted to the monomolecular model of disease progress. Solid line is the predicted increase in disease incidence, y, and dots indicate observed incidence of spotted wilt on the field-assessment dates.

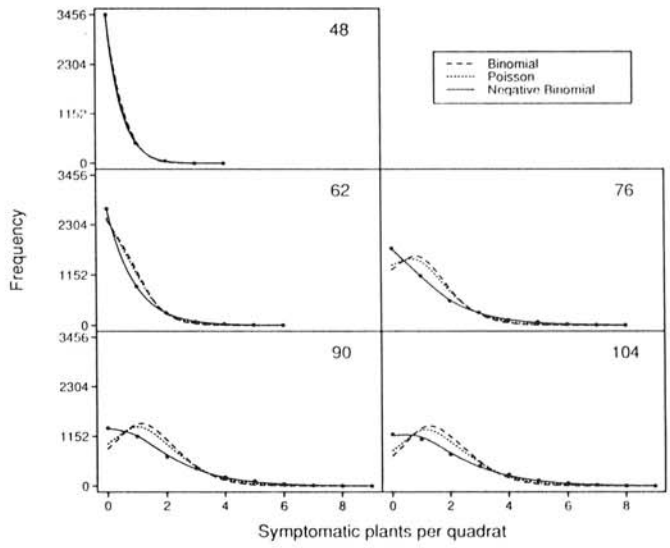

Fig. 2. Comparison of observed frequency distributions of the number of peanut plants with tomato spotted wilt tospovirus symptoms per quadrat (solid circles) with expected distributions (lines) under binomial, Poisson, and negative binomial processes for susceptible cultivar Florunner in 1990 at Attapulgus, GA. For clarity, expected distributions were smoothed with the cubic spline approximation, and all axes were made equivalent. The number of days postplanting for each field assessment appears in the upper right corner of each plot.

surveys in both cultivars as well, suggesting weak aggregations of plants with spotted wilt disease.

In 1990, the disease incidence in both cultivars was greater at all assessment dates, ranging from approximately 1.1 to 7.8% in Southern Runner and from 2.4 to 25.3% in Florunner over the sampling interval (Table 1). In Southern Runner, the variance/mean ratio among quadrats exceeded unity on the third through fifth assessment dates ($P < 0.01$; Table 1). The estimated negative binomial k parameter ranged from 1.02 on day 62 to 0.75 on day 104. The variance/mean ratio obtained for Florunner on the first assessment date also was lower than on subsequent assessments and was indistinguishable from one (1.10 on day 48; Table 1). By day 62, this ratio had risen to 1.46 ($P < 0.01$, df = 3,981; Table 1) and continued to suggest weak but statistically significant aggregation of symptomatic plants during the remainder of the season. The negative binomial k parameter ranged from a low of 1.09 to a high of 2.47.

Temporal disease progress. Spotted wilt disease progress was slow in both cultivars in 1989 (Fig. 1; Table 1). The monomolecular β and rate parameters for Southern Runner plants were 1.01 ± 0.001 and 0.0001 ± 0.00001, respectively (±SE, RSE = 0.0005, df = 3, $R^2 = 0.97$). The rate of disease progress in Florunner was more rapid, with $\beta = 1.02 \pm 0.002$ and $r = 0.0003 \pm 0.00002$ (RSE = 0.001, df = 3, $R^2 = 0.98$). In 1990, there was an increase in both the rate of disease progress and the final disease incidence in both cultivars, although spotted wilt incidence remained consistently lower in Southern Runner than in Florunner. The monomolecular parameters for Southern Runner in 1990 were $\beta = 1.05 \pm 0.008$ and $r = 0.001 \pm 0.0001$ (RSE = 0.004, df = 3, $R^2 = 0.98$), suggesting a 10-fold increase in the rate of epidemic progress in 1990. The increase in disease progress rate was similar in Florunner, with $\beta = 1.24 \pm 0.05$ and $r = 0.005 \pm 0.0005$ (RSE = 0.02, df = 3, $R^2 = 0.97$).

Models of spatial dispersion processes. None of the observed frequency distributions of symptomatic plants per quadrat could be fit to a binomial model using 0.9 × 0.9-m quadrats (Fig. 2; Table 1) ($P < 0.01$, χ^2 test, df = 3,981). In only one instance was a fit to a binomial model obtained for larger quadrats with 95% certainty (3.6 × 1.8-m quadrats of symptomatic Southern Runner plants on field-assessment day 48). This data set, however, also was described by Poisson and negative binomial models ($P > 0.05$; Table 1).

In 1989, the distributions of symptomatic Southern Runner plants were described by a negative binomial model on all five assessment dates. The Poisson model of a random spatial process was rejected at all but the earliest field survey, when disease incidence was too low to calculate a χ^2 statistic for any of the quadrat sizes. However, all of the results for Southern Runner in 1989 required combining adjacent quadrats to some degree (Table 1), reflecting the difficulty in obtaining sufficient degrees of freedom for a goodness-of-fit test at low disease-frequency levels. Observed distributions of symptomatic Florunner plants in 1989 also were indistinguishable from the expected negative binomial distributions on all five assessment dates (Table 1). The Poisson model was rejected on all but the first date. Although the incidence of spotted wilt disease was higher in Florunner than in Southern Runner, the three earliest assessments nonetheless required some combination of adjacent quadrats to obtain sufficient expectations.

The 1990 data followed similar patterns. In Southern Runner, all field survey data were adequately described by a negative binomial model, although the first two, on assessment days 48 and 62, also were indistinguishable from a Poisson distribution. After day 62, however, the Poisson model was rejected. In Florunner, the Poisson model was rejected on all five field observation dates; the negative binomial model was accepted on only the first four dates. On the last date (104 DPP), the negative binomial model was not accepted for 0.9 × 0.9-m quadrats but was acceptable for 1.8 × 1.8-m quadrats ($P = 0.32$; Fig. 2).

2D-distance class analysis. For Southern Runner in 1990, the proportions of quadrats containing at least one infected plant were 0.20 and 0.23 on field-assessment days 90 and 104, respectively, permitting 2D-distance class analyses on these final two assessment dates (31). On day 90, there were 257 SCF that were higher than expected under a random spatial distribution and 218 SCF that were lower than expected. The average cluster size was four to seven quadrats, indicated by higher than expected SCF in the [1,0], [2,0], and [1,1] distance classes (31). Distance classes with X coordinates > 85 contained both random and lower than expected SCF, whereas distance classes with X coordinates < 85 contained a mixture of random, lower, and higher than expected SCF, suggesting a weakly aggregated spatial distribution of small clusters of symptomatic quadrats within a generally random matrix (Fig. 3A and B). On day 104, there were 281 SCF that were higher than expected under a random spatial distribution and 253 SCF that were lower than expected. The average cluster size had increased to 5 to 11 quadrats, indicated by higher than expected SCF in the [0,1], [1,0], [1,1], [2,0], and [3,1] distance classes (Fig. 3C and D). Furthermore, quadrat pairs with significantly higher SCF than would be expected under a random distribution began to segregate in the upper left quadrant of the distance class diagram ([X < 85, Y < 12], $P \leq 0.05$), with most of the low SCF

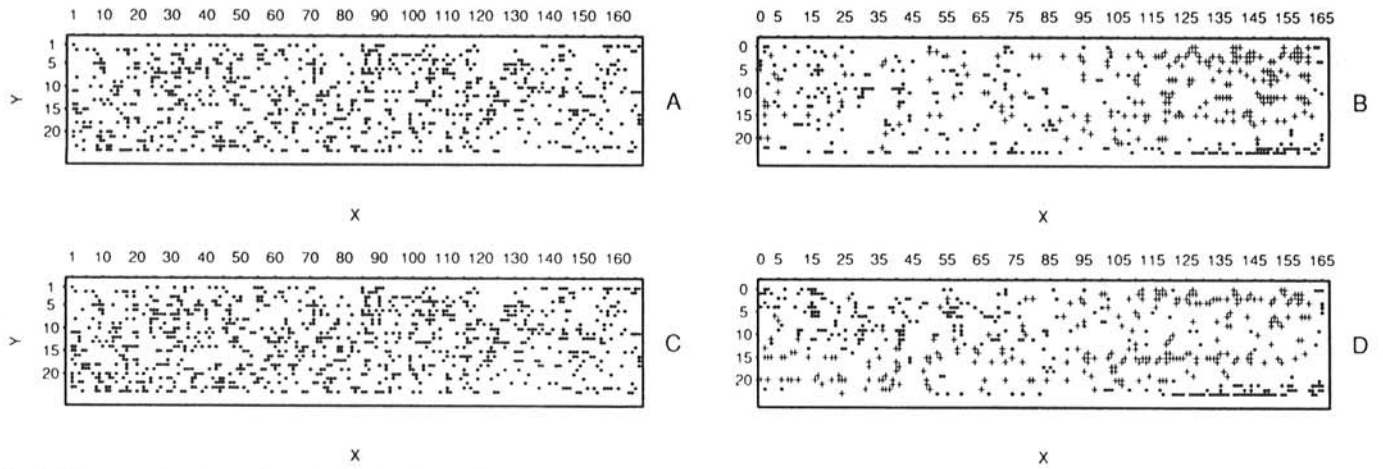

Fig. 3. Binary quadrat data and two-dimensional (2D) distance class analyses of tomato spotted wilt tospovirus incidence in Southern Runner peanut in 1990 at Attapulgus, GA, **A and B,** at 90 days postplanting (DPP) and **C and D,** at 104 DPP. The left column in **A and C** illustrates presence (solid circles) or absence (empty cell) of spotted wilt in each 0.9 × 0.9-m quadrat. The right column in **B and D** shows the results of 2D-distance class analyses of the corresponding 24 × 166-quadrat sampling lattice at each field-assessment date. Solid circles represent standardized count frequencies (SCF) significantly higher than expected under a random spatial dispersion ($P < 0.05$) and crosses represent SCF lower than expected ($P > 0.95$).

($P \geq 0.95$) occurring in the outer three quadrants. This suggested both an increase in average cluster size as the epidemic progressed and some degree of continuing organization toward weak aggregation within a random background matrix. The higher than expected SCF in the outermost rows and columns, [163 to 165, 22 to 24] on days 90 and 104 were fewer than the 15 to 20% of the total number of SCF described by Nelson et al. (31) as indicative of a discernible edge effect and, therefore, are likely to be artifacts of the analysis.

In Florunner, the proportions of quadrats with symptomatic plants were 0.13, 0.33, 0.56, 0.66, and 0.70 on the 1990 field-assessment dates. On day 62, there were 209 SCF with values higher than expected, mostly in the upper left quadrant of the distance class diagram, and 424 with values lower than expected, the majority occurring in the outer quadrants (Fig. 4A and B). Random SCF ($0.05 < P < 0.95$) were distributed throughout the distance class diagram but were sparse in the [X > 110] distance classes, indicating that distances between symptomatic quadrat pairs were frequently less than would be expected under a random model. The average cluster size was three to five quadrats, indicated by higher than expected SCF in the [1,0], [2,0], and [0,1] distance classes. On day 76 there were 505 SCF that were greater than expected at $P \leq 0.05$ and 1,019 that were less than expected at $P \geq 0.95$. It was not possible to determine an average cluster size because many of the greater than expected SCF were clustered in the lower [X,Y] distance classes, and the corresponding clusters of quadrats containing symptomatic plants were contiguous in much of the lattice (Fig. 4C and D). Nonetheless, a nonrandom component was apparent; the null hypothesis of random spatial distribution was excluded for SCF in 62% of the [X,Y] distance classes. This pattern continued on days 90 and 104, with 643 and 771 greater than expected SCF, respectively, and with 1,216 and 1,321 lower than expected SCF (Figs. 4E–H). These nonrandom pair distances comprised a decreasing proportion of the total number of available SCF, however, accounting for 53% on day 90 and 47% on day 104, suggesting that the influence of processes producing random distributions of symptomatic plants increased in dominance as the epidemic progressed in this cultivar.

DISCUSSION

The data indicated weakly aggregated TSWV infections that were described by negative binomial frequency distributions of infected plants per quadrat. They were consistent with a number of spatial processes influencing disease-transmission dynamics; Campbell and Noe (10) cited various authors who established at least 15 distinct processes yielding a negative binomial model and others giving the related Neyman Type A model. We focus our discussion on three such processes.

First, the observed slightly aggregated patterns of disease may have resulted from transmission of the virus by the progeny of the adult thrips vectors that initially introduced the pathogen. This hypothesis proposes that arriving adult thrips transmitted the virus to a number of plants, oviposited on those newly infected hosts, and then continued to feed on them or to move to other plants randomly. Neonates and early instar thrips, which have limited mobility and a high likelihood of feeding on the oviposition plants, acquired the virus from oviposition plants before moving to closely adjacent host plants, either as late instar larvae or as young adults. These adults transmitted the virus to the new hosts as they fed. Black et al. (5) reported higher incidence of spotted wilt in peanut plots surrounded by the susceptible cultivar Tamrun 88 than in those surrounded by more resistant cultivars, suggesting that some degree of secondary transmission occurred in that system. Nonetheless, we have observed in other field tests that massive and numerous applications of insecticide provided good control of larval thrips but that control of adult thrips was erratic, implying that vector immigration was continuous. In these trials, spotted wilt incidence was usually unaffected by larval thrips control, and the use of systemic insecticides even resulted in significant increases in spotted wilt incidence in spite of highly efficient control of larval thrips (J. W. Todd, *unpublished data*).

Alternatively, clusters of infected plants may have resulted from primary infection if viruliferous vectors were capable of redispersing short distances after arriving in a field. The twining habit and dense plantings of peanut plants, with their overlapping runners, made it easy for thrips to move to adjacent plants. In many cases, thrips, simply walking from one shoot to another, might have moved to another host. This redispersal, followed by vector feeding and consequent virus transmission, may have produced some aggregation of infected plants without transmission of the virus by progeny of viruliferous vectors.

Our data were consistent with this interpretation. Infection appeared to occur mainly through primary inoculation by viruliferous vectors entering the field from outside reservoirs, either as fully winged immigrants from the surrounding landscape or as brachypterous thrips that overwintered within the field (11). The weak aggregation of plants infected with TSWV resulted from the vectors (or their progeny) that moved to and fed on closely adjacent plants.

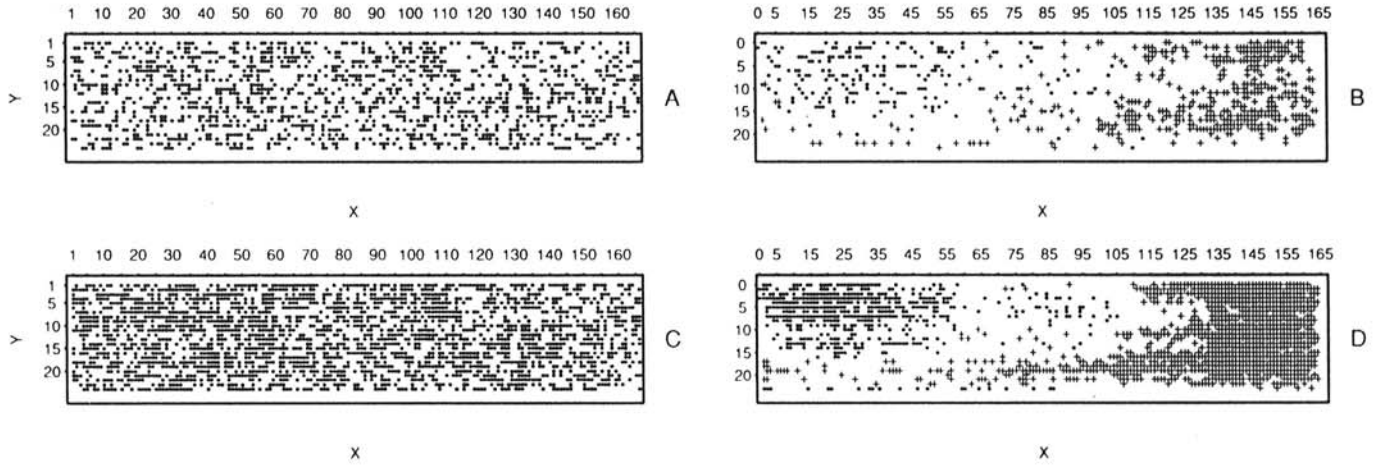

Fig. 4. Binary quadrat data and two-dimensional (2D) distance class analyses of tomato spotted wilt tospovirus incidence in Florunner peanut in 1990 at Attapulgus, GA, **A and B,** at 62 days postplanting (DPP) and **C and D,** at 76 DPP. The left column in **A and C** illustrates presence (solid circles) or absence (empty cell) of spotted wilt in each 0.9 × 0.9-m quadrat. The right column in **B and D** shows 2D-distance class analyses for the corresponding 24 × 166-quadrat sampling lattice at each field-assessment date. Solid circles represent standardized count frequencies (SCF) significantly higher than expected under a random spatial dispersion ($P < 0.05$) and crosses represent SCF lower than expected ($P > 0.95$).

The lack of disease suppression in insecticide-treated plots, the inability of adult thrips to acquire TSWV (42), the likelihood that adults who acquired the virus during larval feeding remained capable of transmitting TSWV for long periods of time (43), and the apterous condition of larval thrips (8) all support the hypothesis that primary transmission during adult thrips dispersal and host seeking accounted for most observed incidence of spotted wilt in peanut. Analysis of temporal disease progress can help distinguish between weak aggregations produced by secondary within-field transmission, and similar patterns caused by exogenous forces during primary infection. In the former case, disease progress should accelerate as secondary inoculum accumulates within the field, whereas in the latter instance disease progress will be independent of disease incidence (9). The adequacy of the monomolecular model for describing disease progress curves in this pathosystem is consistent with an interpretation favoring primary transmission of inoculum. We must emphasize, however, that statistical information regarding model fits must be interpreted with extreme caution before drawing conclusions about the underlying biological mechanisms driving disease progress in any pathosystem (9). Despite the excellent performance of the monomolecular model, the possibility of multiple within-field disease cycles remains, particularly since the opportunity for some degree of secondary infection via larval thrips cannot be completely dismissed without additional data. Nonetheless, we believe that primary infection played a more important role in this pathosystem. A finer temporal scale of disease progress measurements would help resolve this, as would data on larval thrips movement.

Finally, a third process capable of yielding the observed spatial distribution of disease suggests that plants previously infected with TSWV might be slightly more attractive to immigrating vectors than healthy plants. If this was the case, immigrating viruliferous thrips would be more likely to begin feeding on a previously infected plant or on a closely adjacent healthy one. Such behavior could lead to aggregation surrounding early-season inoculations. The present data provide some weak support for this interpretation, particularly in Southern Runner (Fig. 3). Furthermore, lettuce plants with spotted wilt attracted higher numbers of thrips than healthy plants did (R. Mau, *personal communication*). Investigations to determine the relative likelihood of thrips bias toward infected and healthy peanut plants are needed to evaluate whether thrips are attracted differentially to plants with spotted wilt.

This investigation corroborated earlier evidence for resistance to TSWV in the peanut cultivar Southern Runner (7,18). In both 1989 and 1990, the final cumulative incidence of spotted wilt was lower in Southern Runner than in Florunner. Previous studies showed that this was not attributable to a preference by thrips for one host cultivar over the other (18).

Although 2D-distance class analysis was developed to analyze lattices of individual plants, we know of no technical difficulty preventing its use on lattices of binary quadrat data; indeed, Nelson et al. (31) also have suggested this use. Quadrat size can be selected such that all quadrats contain plants, making it unnecessary to deal with vacancies. Use of 2D-distance class analysis on binary quadrat data renders analysis of plant-to-plant interactions impossible, however. This sacrifice of spatial resolution was necessary in the present case because of the large size that matrices of individual plants would have attained and because the locations of healthy plants and vacancies were not mapped (the data were originally collected for a remote-sensing study requiring only the locations of symptomatic plants). Most of the existing literature on 2D-distance class analysis used smaller data matrices than those presented here (24,25,29,30,31). Such matrices were well suited for studying disease increase among individual plants. Unfortunately, the use of this technique to detect spatial patterns in large matrices is limited by the size of the arrays generated during the Monte Carlo simulations (24). For example, in 1989 we estimated that there were approximately 45,000 plants in the Florunner field. An array containing 400 Monte Carlo simulations on a 45,000-plant matrix would require storage for 1.8×10^7 floating point numbers or nearly 141 megabytes of memory for 8-byte floating point numbers.

The 2D-distance class analysis of the 1990 Southern Runner data, although limited to the last two field-assessment dates, suggested that the distribution of quadrats containing diseased plants was weakly aggregated into clusters that were distributed within a generally random background matrix (Fig. 3). The analyses supported the hypothesis that the overall spatial distribution of epidemic progress was dominated by vector immigration and, to a lesser extent, by within-field vector movement. Nonrandomness was indicated when the SCF in greater than 5 to 10% of the [X,Y] distance classes were distinct from the mean SCF under a random model (31). By the final two field-assessment dates in Southern Runner in 1990, SCF in 12 and 13% of the [X,Y] distance classes were significantly nonrandom ($0.95 < P < 0.05$).

The 2D-distance class analyses for 1990 Florunner were more difficult to interpret, and they raised a disturbing question about the rigor of the technique (Fig. 4). Beginning on the third field-assessment date (76 DPP), when 56% of the quadrats contained at least one infected plant, extreme segregation of higher than expected SCF, random SCF, and lower than expected SCF was observed (Fig. 4). The entire field was interpreted as a single cluster, in spite of the proportions of infected quadrats on each of these field-assessment dates being well within the published guidelines for 2D-distance class analyses (31). These analyses provided no useful information and were excluded from additional interpretation. Further work is needed to resolve these difficulties, perhaps using simulation studies designed to test the range of admissible large data sets, i.e., those containing many potential [X,Y] distances, and the behavior of 2D-distance class analyses on large data sets containing a variety of known spatial patterns of disease distribution.

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