Patterns of Rhizoctonia Foliar Blight on Soybean and Effect of Aggregation on Disease Development

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This research was supported in part by the Louisiana Soybean and Grain Research and Promotion Board. Approved for publication by the Director of the Louisiana Agricultural Experiment Station as manuscript 89-38-3349.
Thanks are given to J. P. Damicone, J. W. Hoy, and K. V. Subba Rao for comments and suggestions.
Accepted for publication 30 August 1990 (submitted for electronic processing).

ABSTRACT

Yang, X. B., Snow, J. P., and Berggren, G. T. 1991. Patterns of Rhizoctonia foliar blight on soybean and effect of aggregation on disease development. Phytopathology 81:287-293.

Each of six soybean fields with a history of Rhizoctonia foliar blight was divided into 0.75- × 0.75-m quadrats. Diseased leaves in each quadrat were initially counted at soybean growth stage V8-V11. At soybean growth stage R4, disease incidence in each quadrat was assessed. Lloyd's index of patchiness (LIP) of diseased leaves ranged from 1.56 to 4.54 at the first rating. At the second disease rating, LIP of disease incidence for each plot ranged from 1.32 to 2.15, indicating a decrease in disease aggregation (except for 1987 Lake Arthur). Cluster size was examined using paired-quadrat variance analysis. Different cluster sizes were detected in different fields, indicating an environmental effect on pathogen dispersal. For each field, paired-quadrat variance curves from two rating dates had similar trends. The apparent infection rates of the disease were negatively correlated with the LIP at first rating ($P < 0.05$), indicating that spatial pattern influenced disease development. Disease incidence late in the season was predicted with a logistic model, using early infection as a predictor ($P < 0.01$). Incorporation of LIP into the logistic growth model to correct aggregation effect improved the prediction.

Additional keywords: epidemiology, *Glycine max, Rhizoctonia solani.*

Foliar blight caused by *Rhizoctonia solani* Kühn on soybean (*Glycine max* (L.) Merrill) (23) is a disease of having both soilborne and leafborne nature (29). Rain-splashed soils containing sclerotia and mycelium fragments are sources of primary inoculum (10). In the canopy, secondary spread occurs by mycelial growth from leaf-to-leaf and plant-to-plant (12,17,29), and disease foci expand horizontally only after the canopy reaches a certain density (29). The disease begins to spread both vertically and horizontally after canopy closure (29).

Density and distribution of the soilborne inoculum influence the incidence of many diseases (5). Information on influence of pattern and density of soil inoculum on the development of soybean foliar blight is needed. However, spatial pattern of foliar blight inoculum is difficult to determine because no sampling procedure can truly reflect the distribution pattern of a pathogen in soil (16). Alternatively, the number of primary lesions induced by *R. solani* on varied hosts has been considered a reflection of the amount of inoculum present (1–4,6). Research to evaluate spatial patterns of inoculum by assessing diseased tissues has been reported for hypocotyl rot of snapbean caused by *R. solani* (6) and for sorghum downy mildew (21). Primary lesions caused by *R. solani* on individual leaves or other discrete plant tissues should therefore help us demonstrate the effects of soil inoculum on the development of Rhizoctonia foliar blight on soybean.

Because of the leafborne nature of soybean foliar blight, the pattern of the disease may be highly aggregated, and the aggregation may therefore affect the epidemic process. Rouse et al (19) found that counts of colonies of *Erysiphe graminis tritici* Marchal on wheat were described by a negative binomial distribution, which indicated aggregation distribution of colonies. They suggested incorporation of the negative binomial distribution into a disease model. Waggoner and Rich (27) examined 112 examples of disease frequency counts and found that lesion distribution influenced the logistic increase of plant disease (27). Recently, Reynolds et al (18) found that within neighboring quadrats the incidence of strawberry leather rot (causal agent *Phytophthora cactorum*) at a given time was becoming progressively similar as the epidemic developed. Besides the pattern, the amount of early infection may also affect disease development. Smith et al (24) found that disease severity of southern blight of processing carrot caused by *Sclerotium rolfsii* was influenced

by the number of disease foci early in the season. Like southern blight of carrot, Rhizoctonia foliar blight also forms distinct disease foci in the fields (l2,17,29). Therefore, both number and distribution of primary infections may affect ultimate foliar blight development in the field.

The objectives of this work were to study spatial pattern of Rhizoctonia foliar blight both early and late in the growing season and to assess the influence of spatial pattern and quantity of early infections on disease development.

MATERIALS AND METHODS

Plot establishment and data collection. Experiments were conducted in soybean fields (Table 1) with a history of Rhizoctonia foliar blight. In 1987, the test sites were two fields at the Burden Plantation, Baton Rouge, and one field at Lake Arthur. Fields at the Burden Plantation were planted with cv. Davis at a row spacing of 75 cm. The field at Lake Arthur was planted with cv. Hartz 7126 at a row spacing of 25 cm. In 1988, three locations at the Burden Plantation, Ben Hur Farm (Baton Rouge), and Lake Arthur were selected. Fields in the first two locations were planted with cv. Davis at a 75-cm row spacing. Cultivar Asgrow 6785 was planted at Lake Arthur at a 25-cm row spacing. Planting dates ranged from 23 May to 3 June in both years. All cultivars were susceptible to the disease.

During soybean growth stages V3-V5 (8), a point was randomly chosen as an arbitrary origin for each field. At this point, a plot was established. Within the plot, a single row for 75-cm row spacings or three rows for the 25-cm row spacings were defined as a sampling row. One side of the plot was marked to provide a base line. From the base line, each sampling row was subdivided into contiguous 0.75- × 0.75-m quadrats. The number of quadrats in each plot varied with plot size (Table 1).

At soybean growth stages V8-V11 (8) (immediately before canopy closure), diseased leaves in every quadrat were counted for each plot. At the same time, 20 quadrats were randomly selected from each plot to determine number of leaves per quadrat. At soybean growth stage R4, disease incidence (percentage of infected leaves) of every quadrat was determined. Assessment of each site was completed in 2 days for the first rating and in 3 days for the second rating.

Spatial pattern analysis. To visually examine the spatial pattern, diseased leaves per quadrat and disease incidence were each divided into five classes and then mapped using SAS.

To determine the spatial pattern statistically, diseased leaves and disease incidence were each combined into 11 frequency classes. The mean and Lloyd's index of patchiness (LIP) (13) of diseased leaves per quadrat and of disease incidence were calculated for each plot. LIP can be used as a measure of aggregation for a population (13).

Paired-quadrat variance analysis (14) was used in this study to determine cluster size of the disease. In our experiments, data for ten sampling rows were randomly selected from the data set of a plot. Pairing quadrat spacings varied from 1 to 20 quadrats. Each sampling row had 20 pairings for each distance. Hence, there were 10 × 20 pairings for calculating a variance at pairing distance m (V_m) and the equation was

$$V_m = \frac{1}{2 \times 10 \times 20} \sum_{i=1}^{10} \sum_{j=1}^{20} [X_{ij} - X_{i(j+m)}]^2 \quad (1)$$

where X_{ij} and $X_{i(j+m)}$ were disease rating at the j-th quadrat and $(j + m)$-th quadrat in i-th sampled row, respectively. The variances were plotted against the quadrat spacing to examine the cluster size of disease in each field. This analysis was done with a computer program written in BASIC (28).

Influence of spatial pattern of early infection on disease development. It was assumed that the logistic model was appropriate for describing disease increase (26). An assumption for logistic increase is that inoculum is distributed according to a Poisson distribution. However, inoculum is usually not randomly distributed (6,7,9,15,22), and the distribution pattern of the pathogen may influence disease development (5,9,25,27). Therefore, the aggregation factor LIP should have a negative effect on increase of disease; i.e., the apparent infection rate is lowered when LIP is high. The logistic model can be written as:

$$dX/dt = \mathrm{LIP}rX(1 - X) \quad (2)$$

in which r is the apparent infection rate, X is disease proportion, dk/dt is the increase of disease at time t, and LIP is Lloyd's index of patchiness at first rating; LIPr is, therefore, the apparent infection rate corrected for aggregation. Integrating equation 2, assuming that LIP is constant, results in:

$$\ln(X_2/(1 - X_2)) = \mathrm{LIP}rt + \ln(X_1/(1 - X_1)) \quad (3)$$

in which $X_1$and X_2 are disease proportion at time 1 and 2 respectively. To demonstrate the relationship expressed by equation 3, two methods were used. First, the values of r were expected to decline as the LIP increased if r was plotted against the LIP. Secondly, a multiple regression using LIP and $\ln(X_1/(1 - X_1))$ as independent variables was compared with a simple linear regression using $\ln(X_1/(1 - X_1)$ as an independent variable. For the multiple regression, the partial regression coefficient for LIP will be negatively significant if LIP affects disease development, and the coefficient of determination will be improved compared with that of simple regression. Mathematical relation between 'rt' and aggregation factor has been given by Yang (28).

The above relationship can be tested when sets of X_1, X_2, and LIP are available. To obtain those data sets, each plot was divided into a series of nonoverlapped subplots, each of 10 × 10 quadrats. The number of subplots per plot ranged from 12 to 20, depending on plot size. Disease progress in each subplot was considered as one independent epidemic. From each subplot, X_1, X_2, and LIP were obtained. The X_1, X_2, and LIP within a plot were then considered a set of samples to examine the effect of the spatial pattern and level of early infection on disease development using equation 3.

The disease proportion at the first rating (X_1) was calculated by dividing the number of diseased leaves per subplot by the estimated total number of leaves per subplot. X_2 was calculated by taking the mean disease proportion of 100 quadrats per subplot. A computer program was written in BASIC (28) to split the plot into subplots and calculate X_1, X_2, and LIP at first rating. Regressions are done using SAS (20). Aptness of regression models was determined by R^2, F-tests, and residual plots.

TABLE 1. Cultivar, planting date, row spacing, plot size, and disease rating date for six soybean fields infested with *Rhizoctonia solani* in Louisiana

Year and location	Quadrats[a]	Row spacing (cm)	Cultivar	Planting date	Growth stage at rating[b] First	Second
1987						
Lake Arthur	40 × 43	25	Hartz 7126	25 May	V9	R4
Burden A	42 × 44	75	Davis	30 May	V9	R4
Burden B	35 × 49	75	Davis	3 June	V10	R4
1988						
Lake Arthur	40 × 50	25	Asgrow 6785	2 June	V8	R4
Ben Hur	40 × 50	75	Davis	23 May	V11	R4
Burden	40 × 50	75	Davis	1 June	V11	R4

[a]Number of quadrats per plot. Quadrat size = 0.75 = 0.75 m.
[b]Soybean growth stage on Fehr's scale (8).

RESULTS

In 1987, for the plots at Lake Arthur, Burden field A, and

Burden field B, mean diseased leaves per quadrat at the first rating were 1.7, 1.1, and 1.2. Average disease incidence at the second rating was 18, 18, and 25, respectively. In 1988, for the plots at Lake Arthur, Ben Hur, and Burden, mean diseased leaves per quadrat at the first rating were 0.50, 1.10, and 0.36. Disease incidence at second rating for each location was 27, 39, and 27, respectively.

Spatial pattern of disease. Only maps of Lake Arthur in 1987 and Ben Hur in 1988 are presented in Fig. 1. Other maps are published elsewhere (28). The disease maps had similarities in spatial patterns between the first and second rating at a given field (Fig. 1). At Lake Arthur, for the first rating (Fig. 1,A1) there were two areas of high density in the upper part of the plot, indicated in the figure by dark shading. These spots developed further into two clumps of high disease incidence at the second rating (Fig. 1,A2). However, disease in the two spots did not develop equally by the second rating, as indicated by different densities between the two areas. At Ben Hur, both ratings showed less disease on one side of the field (Fig. 1,B1 and B2). At the second rating, quadrats with greatest density of disease followed the row direction, indicating a prevailing within-row dispersal of disease.

The Lloyd's index of patchiness (LIP) of diseased leaves per quadrat and of disease proportion per quadrat was greater than one for each field (Table 2), indicating that distribution of the disease was aggregated. Values of LIP at the second rating were less than those at first rating except for 1987 at Lake Arthur.

For the Lake Arthur plots in 1987, a peak of the variance curves occurred at quadrat spacing eight (Fig. 2). According to Ludwig and Goodall (14), cluster sizes of an organism are indicated by peaks of paired-quadrat variance plotted against quadrat spacing. For Burden field A in 1987, the curves increased dramati-

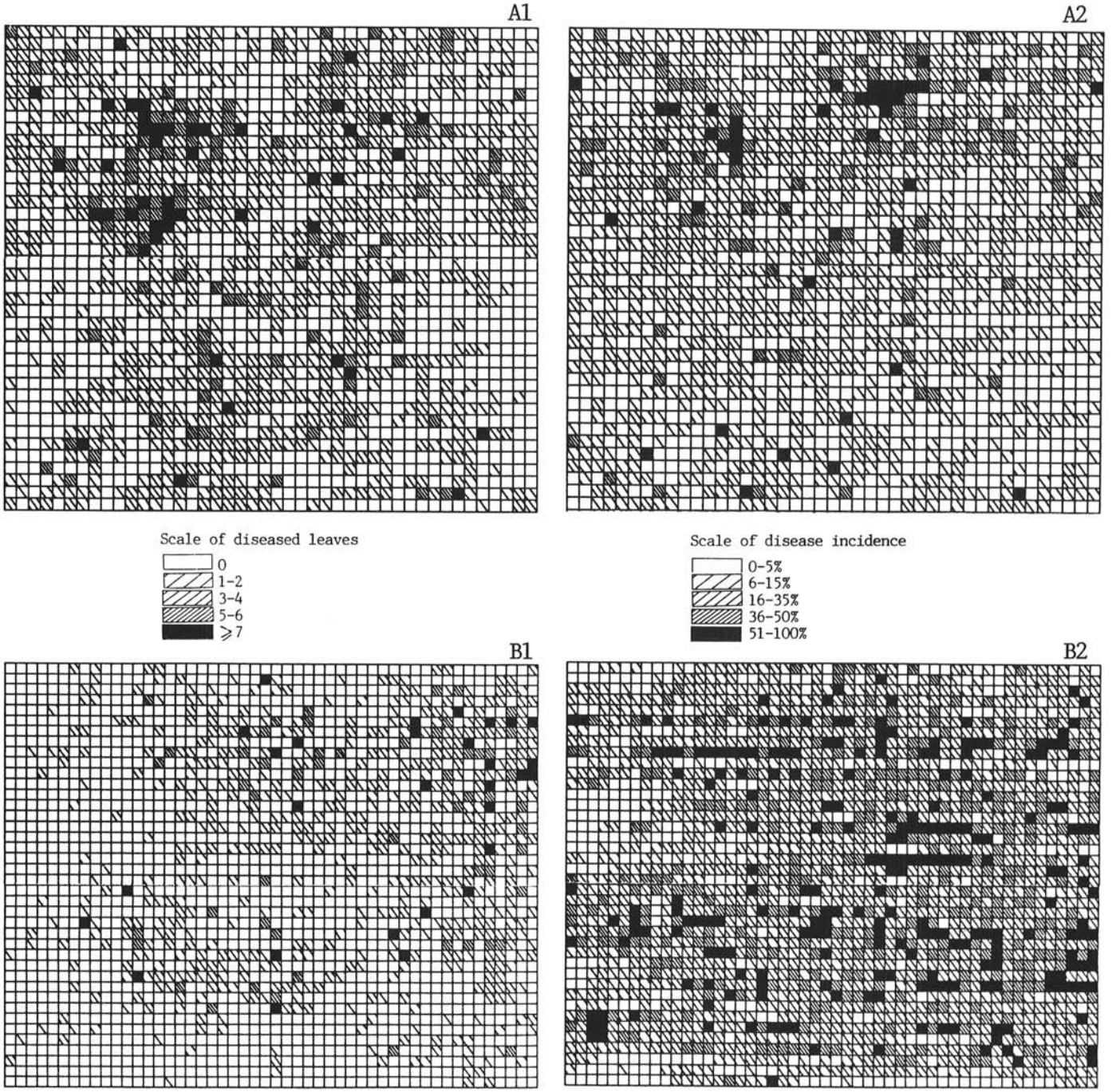

Fig. 1. Maps of number of diseased leaves per quadrat at soybean stage V9 or V11 (A1 = Lake Arthur, 1987; B1 = Ben Hur, 1988) and of disease incidence at R4 (A2 = Lake Arthur, 1987; B2 = Ben Hur, 1988). Fields were infested with *Rhizoctonia solani* and the row direction was horizontal.

TABLE 2. Lloyd's index of patchiness (LIP) for number of diseased leaves per quadrat at the first rating and disease incidence at the second rating in six soybean fields infested with *Rhizoctonia solani* in Louisiana

Year and location	LIP	
	First rating	Second rating
1987 Lake Arthur	1.56	1.68
1987 Burden A	3.31	2.15
1987 Burden B	2.03	1.47
1988 Lake Arthur	4.54	1.33
1988 Ben Hur	2.97	1.32
1988 Burden	4.28	1.46

cally from quadrat spacing one to three (Fig. 2), indicating that the basic clumping size was three quadrats. A second cluster size was indicated by a peak of the variance curves at quadrat spacing eight to nine. In Burden field B, the variance curves increased greatly from one to three, indicating that the basic cluster was three quadrats, and then fluctuated. In 1988 at Lake Arthur, the basic clumping size was one quadrat spacing for both sampling dates (Fig. 3). For the curve of the first rating at Ben Hur, peaks appeared at quadrat spacing 4, 8, 13, and 17. Curves from the first and second sampling at Burden deviated at quadrat spacings 1–10. Curves of paired-quadrat variance for the two ratings generally were similar, particularly for Lake Arthur in 1987 (Figs. 2 and 3).

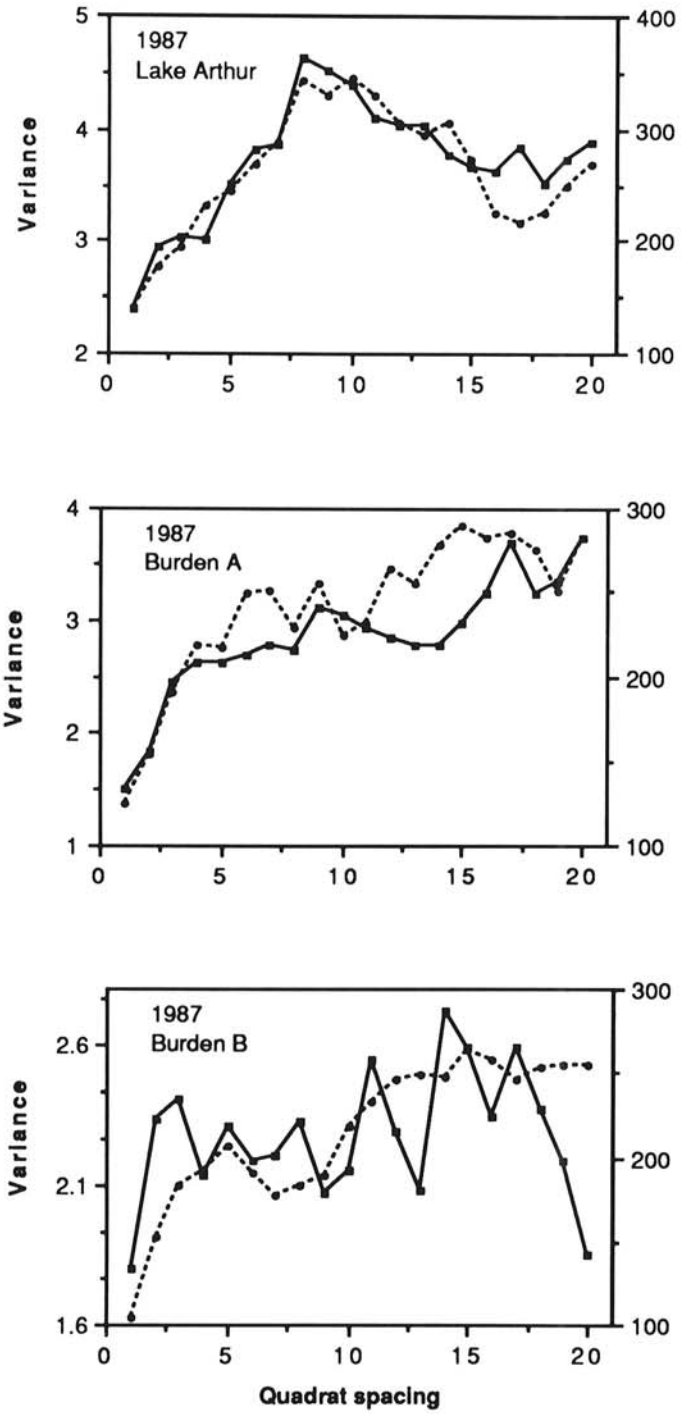

Fig. 2. Variance patterns for data of Rhizoctonia foliar blight of soybean from fields at Lake Arthur, Burden A, and Burden B in 1987. Solid line and left Y axis are for the first rating. Dashed lines and right Y axis are for the second rating. Cluster sizes are indicated by the peaks of the variance curves.

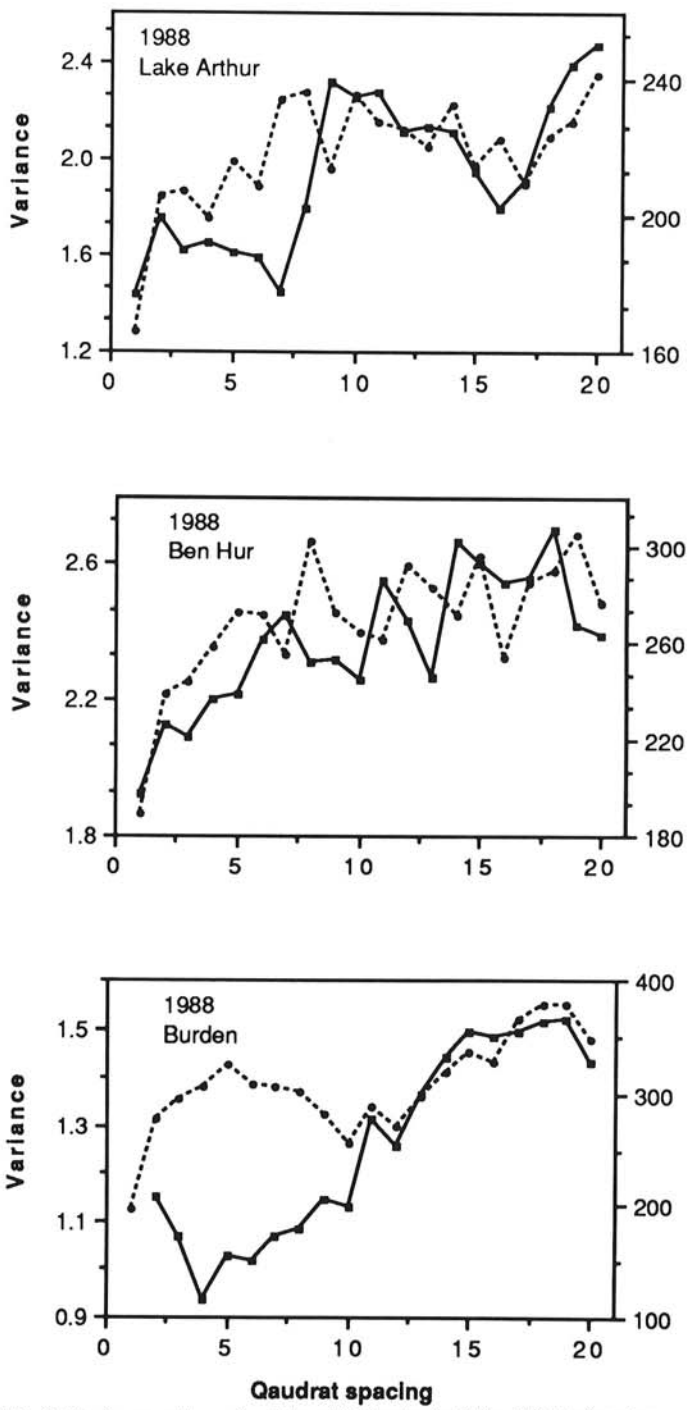

Fig. 3. Variance patterns for data of Rhizoctonia foliar blight of soybean from fields at Lake Arthur, Ben Hur, and Burden in 1988. Solid line and left Y axis are for the first rating. Dashed lines and right Y axis are for the second rating. Cluster sizes are indicated by the peaks of the variance curves.

Influence of number and spatial pattern of early infections on disease development. Values of LIP ranged from about 1 to 8 for different subplots. Value of apparent infection rate (r) declined as the value of LIP increased (Fig. 4). This relationship was negatively significant for every plot, particularly for the plots at Lake Arthur and Ben Hur in 1988 (Fig. 4). Disease incidence late in the season was predicted with a logistic equation using disease early in the season as an independent variable. Values of the coefficients of determination (R^2) ranged from 0.31 to 0.70 ($P \leq .011$) (Table 3). Regression intercepts (B_0) ranged from 0.62 to 4.47. The modified logistic equation (equation 3) fitted the data better than the unmodified version for some fields (R^2 ranged from 0.42 to 0.80). The partial regression coefficient (B_1) of equation 3 was significant for every plot.

DISCUSSION

Rhizoctonia foliar blight in Louisiana is caused by *R. solani* anastomosis group one (17,30). Other strains of *R. solani* are present in soil and cannot be separated in routine assays for foliar blight strains. Strandberg (25), as well as Campbell and Pennypacker (6), pointed out that it is reasonable to assess spatial pattern of inoculum using infected plants or tissues rather than the pathogen propagule itself. In our study, the first ratings of diseased leaves were made before the start of focus expansion (29). Therefore, an aggregation of diseased leaves early in the season may indicate an aggregated distribution of Rhizoctonia foliar blight inoculum in the soil.

Change of disease LIP during the season was reported by

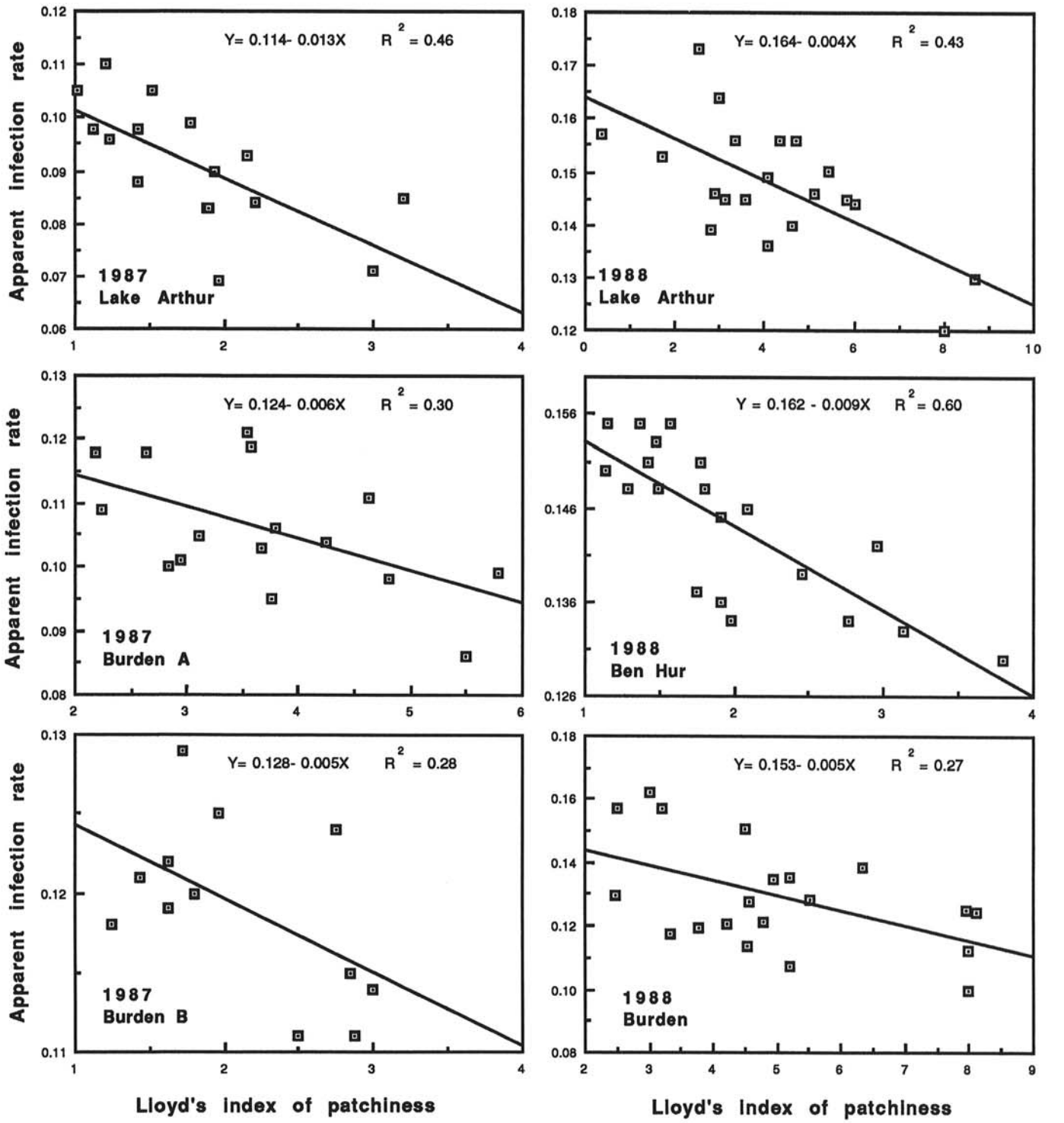

Fig. 4. Relationship between apparent infection rate and Lloyd's index of patchiness of Rhizoctonia foliar blight on six soybean fields in Louisiana.

Reynolds et al (18) in strawberry leather rot. The decreased values of LIP at the second rating compared with those at the first rating in foliar blight indicate that spread of disease resulted in a decrease of aggregation of disease late in the season. Spatial patterns of disease assessed at the second rating, therefore, may not reflect the pattern of soil inoculum as well as at the first rating. However, information from the second rating could be practically useful for planning disease sampling, resistance screening, and fungicide trials.

Paired-quadrat variance analysis for assessing disease aggregation has several theoretical advantages over other hierarchical variance analyses and allows detection of small scale clumping (11,14). In this study, a basic cluster size of one to three quadrats (0.75 to 2.25 m) was detected, which coincides with the observation that foci of Rhizoctonia foliar blight often have diameters of 1–2 m (29). Sampling for paired-quadrat analysis is less labor-intensive because distribution patterns can be assessed readily with one to several transects instead of entire fields (14).

Similarity of spatial patterns assessed from the two sampling times was consistently illustrated by different methods. For example, the early and late disease maps (Fig. 1) showed strong similarity in disease clump patch. The paired-quadrat variance analysis quantitatively illustrated the similarity of cluster size between the two ratings for most fields (Figs. 2 and 3). The significant correlation between early and late disease incidence (Table 3) further illustrates this similarity. Similarity between spatial patterns at the two sampling times suggests that secondary dispersal of the pathogen is limited. Therefore, a survey early in a season may outline the approximate disease boundary in a field late in the season.

Waggoner and Rich (27) incorporated an aggregation factor (k from the negative binomial distribution), both in a multiple infection transformation and in the logistic growth model. Ferrin and Mitchell applied the modified multiple infection transformation for the tobacco black shank disease (9), which is caused by a soilborne pathogen. No application of Waggoner and Rich's modified logistic growth model has been published, however, perhaps because the model's differential equation is complicated. Our modified equation (equation 2) tried to correct the logistic growth for the nonrandom patterns by adjusting the apparent infection rate. Equation 3 explained more variation in regression than an unmodified logistic model, indicating that it may be able to integrate the effects of spatial pattern on disease development.

TABLE 3. Summary of regression results for logistic model of disease increase $[\ln(X_2/(1-X_2)) = rt + \ln(X_1/(1-X_1))]$ and modified logistic models of disease increase $[\ln(X_2/(1-X_2)) = \mathrm{LIP}rt + \ln(X_1/(1-X_1))]$ for prediction of Rhizoctonia foliar blight in six soybean fields in 1987 and 1988[a]

Year and location	B_0	B_1 $\ln(X_1/(1-X_1))$	r^2	$P < F$
1987 Lake Arthur	0.931*	0.601**	0.46	0.004
1987 Burden A	3.926**	1.149***	0.70	0.001
1987 Burden B	4.475**	1.251*	0.56	0.005
1988 Lake Arthur	1.118**	0.404**	0.33	0.008
1988 Ben Hur	1.282**	0.396***	0.55	0.001
1988 Burden	0.618**	0.377*	0.31	0.011

	B_0	B_1 LIP	B_2 $\ln(X_1/(1-X_1))$	R^2	$P < F$
1987 Lake Arthur	2.87**	−0.460**	0.853**	0.64	0.001
1987 Burden A	3.61**	−0.146*	1.037***	0.80	0.001
1987 Burden B	4.02**	−0.217*	1.048*	0.62	0.022
1988 Lake Arthur	3.60**	−0.360**	0.820**	0.45	0.008
1988 Ben Hur	3.66**	−0.386***	0.716***	0.79	0.001
1988 Burden	3.20**	−0.112*	0.821**	0.43	0.002

[a] LIP is Lloyd's index of patchiness (13); B_0, B_1, and B_2 were regression coefficients; *, **, and *** indicate that the coefficient is significantly different from 0 at $P < 0.05$, 0.01, and 0.001, respectively. In the logistic model, B_0 equals 'rt'. In modified logistic model, 'rt' is the sum of B_0 and B_1 LIP.

However, these effects were statistically detected, which may depend on the nature of high aggregation (LIP up to 8) of Rhizoctonia foliar blight.

Subsampling plots is a useful method for studying effects of aggregation on the development of a soilborne disease, such as Rhizoctonia foliar blight of soybean. Among subplots of each field, differences in primary infections and aggregation would be expected if the pathogen distribution was not uniform, but the ambient effects, such as rainfall, temperature, and canopy density, would be homogenous as planting was uniform. Among subplots, therefore, differences in disease late in the season would be determined by the differences in early infection and in disease aggregation. However, the interference of inoculum between subplots might be a source of variation although the disease spread is limited.

Variation that our model could not explain might also come from three other sources. Primary infection by soil inoculum might occur after the first rating (29), which resulted in an underestimation of X_1. Low autocorrelation of disease progress has been reported for this disease (29) and may have occurred in this study. Furthermore, LIP is also quadrat size dependent, which may affect the efficiency to correct aggregation effects.

Rhizoctonia foliar blight is a highly clustered disease because it has both soilborne and leafborne nature. Difficulty in quantification and prediction of this type of disease has been noted (24,29). Our results suggest the possibility of using infection early in the season to predict foliar blight disease.

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