Spatial and Temporal Spread of Soybean Stem Canker from an Inoculum Point Source

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ABSTRACT

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Incidence and severity of stem canker arising from inoculum point sources of *Diaporthe phaseolorum* var. *caulivora* was quantified on resistant (Bay), intermediate (DPL105, W550), and susceptible (Bedford) soybean cultivars in the field during 1986 and 1987. Secondary production of spores arising from perithecia or pycnidia of *D. p. caulivora* was not detected during either growing season. Disease progress was described by the Weibull model. The shape parameter (c) of the model was equal to the idealized c value of a monocyclic disease for Bedford. Infection rates (k) declined as resistance levels of cultivars increased (Bedford, $k = 0.12$; W550, $k = 0.09$; Bay, $k = 0.02$ per week). An exponential model fit the disease gradients. An increase in stem canker spread in 1987 compared with 1986 was accompanied by a reduced gradient steepness. Increasing levels of cultivar resistance generally increased gradient steepness, particularly when severity was used to measure disease. Yield losses in intermediate and susceptible cultivars were linearly related to stem canker incidence with regression lines having similar slopes. No yield loss occurred in Bay as lesions failed to elongate or cause severe necrosis. The greater spread of stem canker in 1987 compared with 1986 was correlated with cumulative rainfall. Differences in directional spread were attributed to water movement patterns in plots.

Stem canker of soybean ((*Glycine max* (L.) Merr.) caused by *Diaporthe phaseolorum* (Cke. & Ell.) var. *caulivora* Athow & Caldwell, is currently a serious and widespread disease over much of the southern United States. The disease reduced yields of susceptible cultivars in the midwestern United States during the late 1940s and early 1950s (1). Stem canker became severe in the South by the early 1980s, causing yield losses in susceptible cultivars of up to 80% (17). The strains of the pathogen in the South and the Midwest differ in cultural morphology (20) and physiologic specialization (11–14,20).

Conidia and ascospores of *D. p. caulivora*, produced in culture and on stem debris in the field (2,5,26), are both capable of inciting stem canker (26). Presumably, spores are dispersed and deposited on plant surfaces by water (2). Final severity of disease is closely related to incidence of infection during early stages of vegetative growth (2,30,33,34). The elongated cankers that produce foliar and stem necrosis and eventually kill plants develop following incubation periods of 50 or more days (5,26). Alternatively, cankers may not develop on infected plants or their development may be restricted on stems and petiole bases (5,13,28).

Soybean cultivars differ in susceptibility to stem canker (2,9, 12,13,35). Reactions have been classified as susceptible, intermediate (moderately susceptible, moderately resistant), and resistant (2,34). Resistant cultivars such as Tracy M and Bay possess genetic resistance controlled by two genes and one gene, respectively (15,35). Yield losses of intermediate cultivars are typically less than those of susceptible cultivars in the presence of stem canker. However, symptom development and yield losses in intermediate cultivars can approach those of a susceptible cultivar when conditions are favorable for severe disease development (2). The genetic basis of their resistance is not known.

In fields infested with the pathogen, occurrence of stem canker is often sporadic from year to year (2,28,32,33). This has been attributed in part to variability in environmental conditions (2,29, 30,32,33) and/or reduced planting of susceptible cultivars (28). Research to date has focused on identifying factors that affect infection and disease development under controlled conditions. Free moisture is critical for both infection and disease development (5,26). Postinoculation application of continuous or discontinuous free moisture increased disease incidence and severity while reducing the incubation period (5). Infections by ascospores and conidia occur over a wide temperature range (27).

The epidemiology of stem canker in the field has received less attention. Reduction in primary inoculum with tillage systems that bury infested crop residue is known to reduce stem canker compared with no-till systems (29,30). An increased understanding of the severe and unpredictable epidemics caused by this disease depends on knowledge of the factors that affect spread of *D. p. caulivora* and development of stem canker. The objective of this research was to quantify the pattern and extent of stem canker spread from a point source of inoculum. We also report the effects of levels of cultivar resistance and environmental factors on field stem canker development and soybean yield.

MATERIALS AND METHODS

Collection and preparation of inoculum. Stem pieces with fruiting structures of *D. p. caulivora* were collected after the 1985 season from a commercial production field, placed in polyethylene bags, and stored under shelter in the field before use in 1986. Infested stem pieces for use in 1987 were collected from experimental plots after the 1986 season.

Artificial inoculum was also produced to supplement natural inoculum. Single conidia were recovered from isolates of *D. p. caulivora* that originated from cankered soybean plants collected in different areas of southern Louisiana. Five single-conidial isolates, determined to be pathogenic to the susceptible breeding line J77-339 with inoculations in the greenhouse using the toothpick technique (12), were stored at 4 C on silica gel (31). Whole oat kernels were saturated in distilled water and autoclaved for 1 hr on two consecutive days. Aliquots (500 ml) of oats were inoculated with mycelium from 7-day-old cultures of each isolate. Inoculated oats were incubated for 3 wk on a lab bench before use.

Field plot descriptions and design. Field studies were done in a Commerce silt loam soil at the LSU Ben Hur Research Farm

in Baton Rouge in 1986. The experimental field was fallowed in 1985 and in pasture before 1985. The field had a uniform 1% slope to the east. An adjacent field was used in 1987 with the same soil type, slope, orientation, and cropping history, except that it was cropped to soybean in 1986. A second field of Sharkey clay soil at the LSU Iberia Research Station in Jeanerette was also used in 1987. The field had zero slope and had been previously cropped to soybean. Neither field that had been previously cropped to soybean had a history of stem canker nor could signs of *D. p. caulivora* be detected on stem debris before planting.

Plot dimensions in all fields were 9.75 × 9.75 m consisting of 13 rows spaced 0.75 m apart. Buffers between plots were three-row planted strips (2.25 m wide) consisting of either one or two rows of the cultivar in the plot and one or two rows of the adjacent cultivar. Rows ran in an east-west direction and were seeded at a rate of 23–26 seed per meter with double-disc opening planters. Soybean cultivars used in all field studies were from maturity group V. Cultivars Delta and Pine Land 105 (DPL105) and Wilstar 550 (W550), reported to be susceptible and intermediate, respectively, in response to stem canker (9) were planted on 15 May 1986. Sixteen plots of each cultivar were completely randomized within the field. In 1987, cultivars planted were Bedford (susceptible), W550 (intermediate), and Bay (resistant) (2). Ten plots of each cultivar were planted in a completely randomized design at Baton Rouge on 24 May. Four plots of W550 were planted at Jeanerette on 22 May. Weeds and insects were controlled using standard local practices.

Forty-eight subplots were defined in each plot that were 0.75 × 0.75 m and extended in eight directions from the center subplot (Fig. 1). Each subplot contained a single row. The perimeter of the center subplot of each plot was surrounded by a 20-cm-wide vertical strip of aluminum window screening that extended 5 cm into the soil and was supported by 25-cm-long wood garden stakes. Plots were infested at the V_0 stage of soybean development (7) by placing 0.25 kg of infested stem debris and 2 L of oat inoculum on the soil surface in the center subplot of all but one plot per cultivar (control).

Rainfall and temperature were continuously monitored in fields at Baton Rouge and 0.25 km from the field in Jeanerette with CR21 Microloggers (Campbell Scientific, Logan, UT). Rainfall was measured with tipping bucket rain gauges and temperature with Campbell model 101 temperature probes.

Disease and yield evaluation. Disease was quantified over time, beginning 30 days after planting at Baton Rouge in 1986 and 1987. Four ratings were made on 3-wk intervals in 1986 and six were made on 2-wk intervals in 1987. Final ratings were made at the mid-R_5 (pod fill) (7) stage. A plot was visited only once per season to avoid possible mechanical spread by movement through a plot. In 1986 at Baton Rouge, three infested plots per cultivar were rated each date. In 1987 at Baton Rouge, one infested plot per cultivar was rated each date except the last, when three were rated. On a given date, plots to be rated were selected at random from remaining plots not yet visited. Only mid-R_5 ratings were made at Jeanerette. Control plots were also rated at mid-R_5 at all locations.

Ratings were made by recording the number of cankers per plant on 10 randomly selected plants in each subplot per plot. Only cankers on the main stem were considered. Early canker development caused by *D. p. caulivora* was differentiated from anthracnose caused by *Colletotrichum* spp. by gently scraping

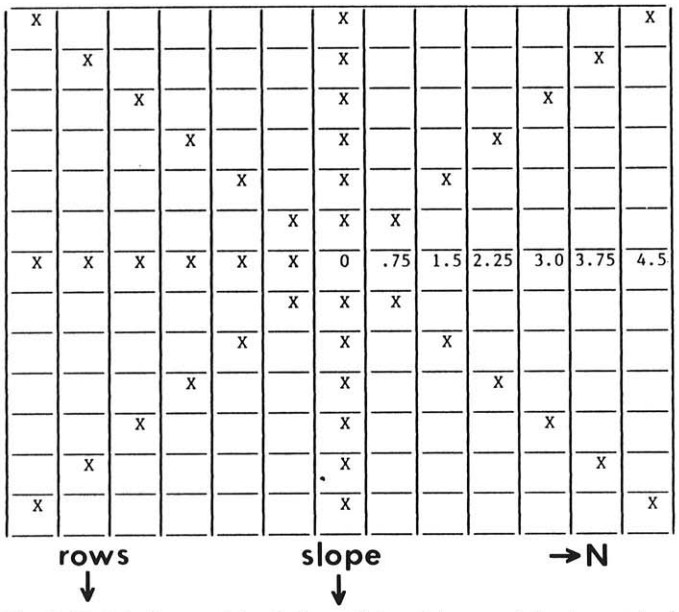

Fig. 1. Plot design used to study spatial and temporal development of soybean stem canker. Each subplot is 0.75 × 0.75 m. X = subplot for disease evaluations; numbers are distances in meters from the inoculum source, 0.

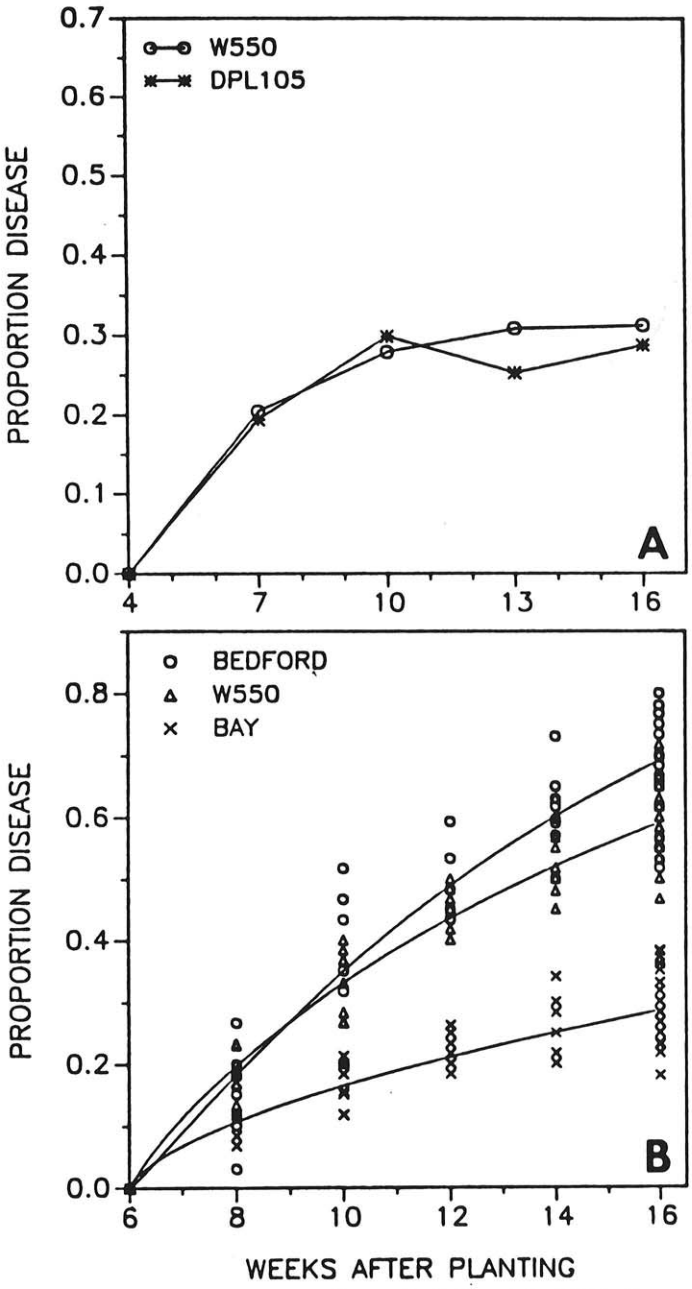

Fig. 2. Stem canker progress over time **A,** in 1986 and, **B,** 1987 in Baton Rouge. Cultivars DPL105 and W550 are intermediate in response to stem canker, Bedford is susceptible, and Bay is resistant. Data points in A represent the mean value of three plots across 48 subplots. In B the Weibull model is fit to means values across distances ($N = 6$) for one plot per cultivar in weeks 6–14 and three plots per cultivar on week 16 ($N = 18$).

the lesion surface. Reddish-brown discoloration associated with stem canker extended into the vascular system while anthracnose caused only superficial discoloration. Canker identification was corroborated with isolations made on a semiselective medium (25). The number of plants killed by stem canker per 10 plants in a subplot was also recorded. Incidence was expressed as the proportion of plants affected with stem canker (maximum = 1.0). Severity was expressed as number of cankers per plant and the proportion of plants killed by stem canker.

Substrates that might support secondary (in-season) sporulation by *D. p. caulivora* were sampled. Necrotic leaves, petioles, and stems were collected in plots on all sampling dates. These were incubated for 72 hr in moist chambers on a lab bench. Spores arising from fruiting structures were then streaked onto the semiselective medium.

Yield was measured in the same three plots rated for disease at mid-R_5 at Baton Rouge in 1986 and 1987. All plants in a subplot were hand-harvested and mechanically threshed with a rasping-bar plot thresher. Yield was expressed as seed weight (g) per subplot adjusted to 13% moisture.

Data analysis. Nonlinear regression using the derivative-free option was used to quantify spatial and temporal disease development. The Weibull model:

$$y = 1 - \exp(-(kt)^c)$$

where y = proportion of plants diseased, k = infection rate (wk^{-1}), t = time ($t = 0$ at 6 wk after planting), and c = shape parameter, was fitted to disease incidence values over time. The Weibull model has been used to theoretically differentiate between polycyclic and monocyclic diseases based on variation in shape (3,24). A value of $c = 1$ is expected for a monocyclic disease and $c > 2$ for many polycyclic diseases.

TABLE 1. Analysis of temporal progress of stem canker caused by *Diaporthe phaseolorum* var. *caulivora* on three soybean cultivars in Baton Rouge, 1987[a]

Cultivar	Parameter[b] k (rate/wk)	Parameter[b] c (shape)	R^{2c}	$P > F^d$ (lack of fit)
Bedford	0.116 ± 0.007	1.097 ± 0.156	0.92	0.471
W550	0.088 ± 0.007	0.872 ± 0.120	0.92	0.693
Bay	0.020 ± 0.008	0.683 ± 0.141	0.85	0.407

[a] Weibull model fitted to mean incidence over six distances from one plot per assessment at 6–14 wk and three plots per assessment at 16 wk after planting ($N = 64$).
[b] k = rate (per week) parameter, and c = shape parameter. 95% confidence interval follows each estimate.
[c] Coefficient of determination.
[d] Significance of F test for lack of fit, large P indicates good fit.

An exponential model for spatial analysis (8,16) was fitted to final disease incidence and severity values over distance. The model is:

$$y = a\exp(-bx),$$

where y is the same as described above, a = y-intercept, and b = spread parameter (m^{-1}, a measure of gradient steepness). The half-distance (= $0.693/b$), i.e., the distance at which disease level was reduced by 50% (8), was also determined. Estimates of disease incidence and plants killed per plot were made by determining the area under the gradient equation (by integration from 0.75 to 4.5 m) and dividing by the total area (21,23).

Analysis of variance and means separation were employed to assess the influence of direction from the inoculum source on incidence of stem canker at mid-R_5. Analysis was performed on mean values over distance from the source.

Linear regression was employed to quantify yield loss. Regression of yield (g/subplot) on stem canker incidence provided an estimate of maximum yield in the absence of disease (the y-intercept). Yield loss models were then constructed by regression of percent of maximum yield on disease incidence.

Aptness of a regression model was assessed by an F test for significance of a relationship between y and x, F test for lack of fit, and the coefficient of determination (R^2) (22). Comparisons of parameter estimates between different regression models were made by using 95% confidence intervals (22).

RESULTS

Temporal progress of stem canker. The increase in incidence of stem canker was similar for cultivars DPL105 (susceptible) and W550 (intermediate) in 1986 (Fig. 2A). Symptoms consisting of small (3–4 mm), sunken, and reddish-brown lesions with discoloration extending into the vascular tissue were first observed 7 wk after planting at R_1 stage. *D. p. caulivora* was isolated from such lesions. Maximum levels of disease occurred from 10 to 16 wk after planting. The sampling interval of 3 wk did not result in sufficient observations for model fitting.

Differences in progress of stem canker were evident in 1987 between cultivars Bedford (susceptible), W550 (intermediate), and Bay (resistant). Initial symptoms (described above) were first observed 8 wk after planting at the V_{10} stage. Maximum levels occurred 16 wk after planting. The fit of the Weibull model to the data (Fig. 2B) was good for all cultivars with R^2 values ranging from 0.85 to 0.92 (Table 1). The infection rate (k) was highest for Bedford, intermediate for W550, and lowest for Bay (Table 1). The shape parameters (c) of the model were equal to 1.0 for Bedford and less than 1.0 for W550 and Bay.

The causal fungus was not recovered during the 1986 or 1987 growing seasons from substrates assayed. Fungi that sporulated readily on necrotic stems and petioles were the Phomopsis form of *D. p. sojae* (Lehman) or *Colletotrichum* spp.

TABLE 2. Relationship between stem canker incidence at mid-R_5 (y) and distance from the inoculum source (x) for four soybean cultivars[a]

Cultivar	Year-location[c]	Parameter[b] Intercept (a)	Parameter[b] Spread (-m) (b)	R^{2d}	$P > F^e$ (lack of fit)	Half distance (m)	Incidence[f] (%) Est.	Incidence[f] (%) Mean
W550	1986–B.R.	1.85 ± 0.32	0.89 ± 0.15	0.95	0.85	0.78	27.2	31.2
DPL105	1986–B.R.	1.82 ± 0.32	0.95 ± 0.16	0.96	0.93	0.73	24.2	28.7
Bedford	1987–B.R.	1.12 ± 0.06	0.20 ± 0.02	0.95	0.78	3.50	68.3	71.3
W550	1987–B.R.	1.04 ± 0.09	0.24 ± 0.04	0.91	0.80	2.84	56.8	59.2
W550	1987–Jean.	0.99 ± 0.11	0.32 ± 0.05	0.91	0.18	2.17	45.9	47.2
Bay	1987–B.R.	0.78 ± 0.13	0.58 ± 0.11	0.92	0.99	1.19	20.6	22.1

[a] Exponential model was fitted to mean incidence over eight directions from three plots ($N = 18$).
[b] Parameters represent the predicted incidence (y) at $x = 0$ (a) and rate of decline in y over x (b) (= spread parameter). 95% confidence interval follows each estimate.
[c] B.R. = Baton Rouge, Jen. = Jeanerette.
[d] Coefficient of determination.
[e] Significance of F test for lack of fit, large P indicates good fit.
[f] Est. = stem canker incidence estimated from integrating exponential model (see text). Mean = mean incidence from three plots per cultivar.

Spatial development of stem canker. Plot-to-plot movement of *D. p. caulivora* did not appear to greatly influence levels of stem canker observed. Stem canker did not develop in uninoculated (control) plots in 1986 and incidence was less than 3% in 1987.

Mean incidence of stem canker per plot at mid-R_5 was similar for DPL105 and W550 in 1986 (Table 2). Incidence declined with distance from the inoculum source for both cultivars, from about 90% at 0.75 m to near 0% at 4.5 m (Fig. 3). Gradients were described by the exponential model with R^2 values greater than 0.90 and with no significant lack of fit (Table 2; Fig. 3). Estimates of b were not significantly different for the two cultivars. The half-distance for both cultivars was approximately 1 subplot increment.

Mean incidence of stem canker per plot at mid-R_5 in 1987 was greatest for Bedford (susceptible), intermediate for W550 (intermediate) at two locations, and lowest for Bay (resistant) (Table 2). Incidence for W550 was 90 and 51% greater at the Baton Rouge and Jeanerette locations, respectively, compared with 1986. Incidence in 1987 also declined with distance from the source, but differences between cultivars were apparent (Fig. 3). Declines in incidence for Bedford (susceptible) and for W550 (intermediate) did not approach 0% even by 4.5 m from the source (Fig. 3D–F). Incidence for Bay (resistant) was near 0% by 4.5 m from the source, and incidence close to the source was lower compared with the other cultivars (Fig. 3C). Estimates of b for W550 were significantly lower and resulting half-distances higher in 1987 in comparison to 1986 (Table 2). The intercept

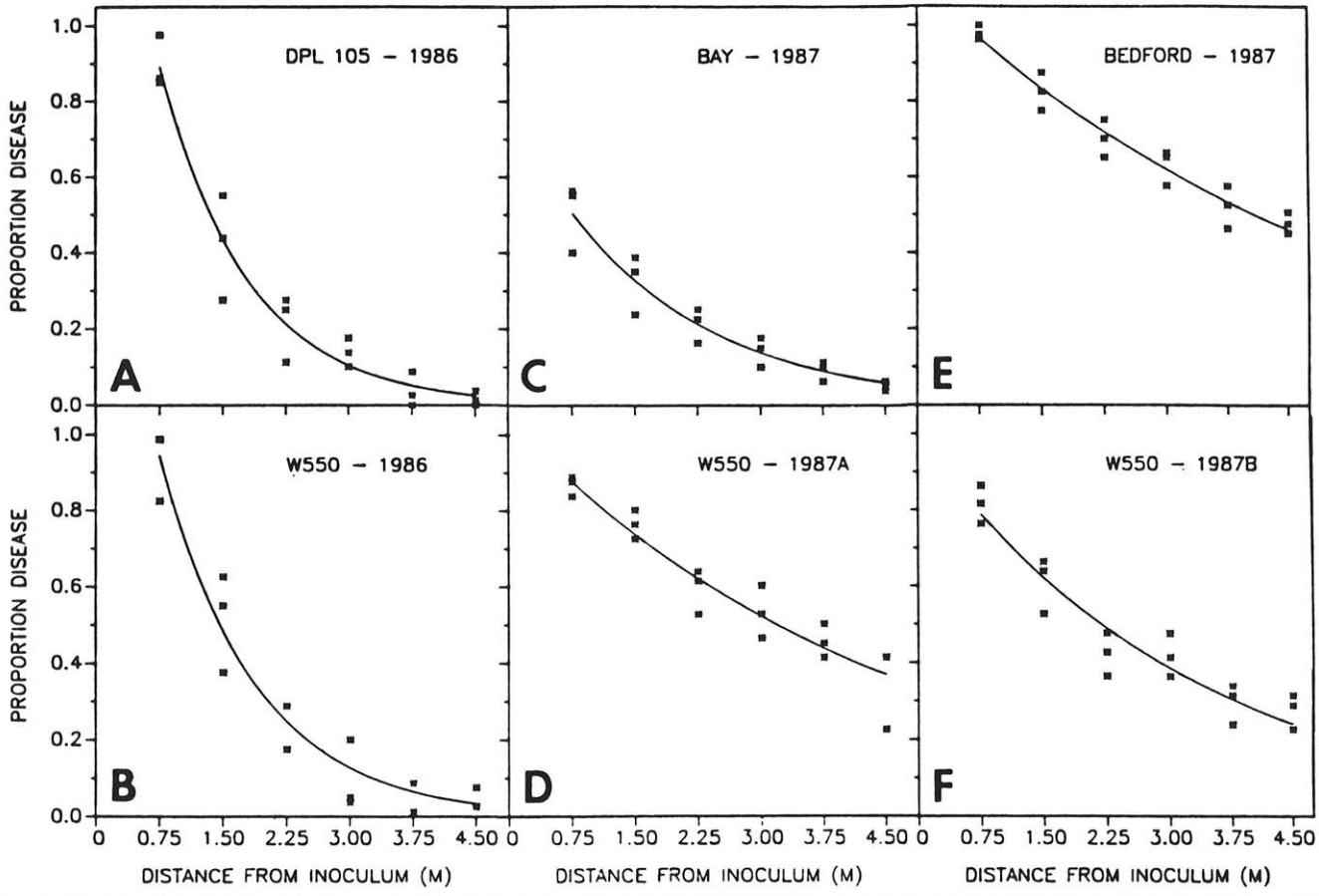

Fig. 3. Gradients of stem canker incidence at mid-R_5 fit to the exponential model from, **A and B,** Baton Rouge in 1986, **C–E,** Baton Rouge in 1987, and **F,** Jeanerette in 1987. DPL105 and W550 are intermediate in response to stem canker, Bedford is susceptible, Bay is resistant. Data points represent mean values of three plots per cultivar across eight directions ($N = 18$).

TABLE 3. Relationship between proportion of plants killed by stem canker at mid-R_5 (y) and distance from the inoculum source (x) for four soybean cultivars[a]

Cultivar	Year-location[c]	Parameter[b] Intercept (a)	Parameter[b] Spread (-m) (b)	R^{2d}	$P > F^e$ (lack of fit)	Half distance (m)	Incidence[f] (%) Est.	Incidence[f] (%) Mean
W550	1986–B.R.	1.07 ± 0.53	1.64 ± 0.58	0.89	0.87	0.42	5.1	7.7
DPL105	1986–B.R.	2.56 ± 1.22	2.20 ± 0.60	0.96	0.96	0.31	6.0	10.6
Bedford	1987–B.R.	0.87 ± 0.11	0.26 ± 0.06	0.84	0.79	2.66	45.7	46.2
W550	1987–B.R.	1.08 ± 0.29	0.63 ± 0.19	0.82	0.91	1.10	25.8	28.5
W550	1987–Jean.	0.82 ± 0.33	0.49 ± 0.24	0.60	0.94	1.41	26.0	27.5
Bay	1987–B.R.	...	...	...	...	...	...	0

[a] Exponential model was fitted to mean proportion of plants killed over eight directions from three plots per cultivar ($N = 18$).
[b] Parameters represent the predicted proportion of plants killed (y) at $x = 0$ (a) and rate of decline in y over x (b) (= spread parameter). 95% confidence interval follows each estimate.
[c] B.R. = Baton Rouge, Jen. = Jeanerette.
[d] Coefficient of determination.
[e] Significance of F test for lack of fit, large P indicates good fit.
[f] Est. = proportion of plants killed estimated from integrating exponential model (see text). Mean = mean proportion from three plots per cultivar.

TABLE 4. Relationship between number of cankers per plant at mid-R_5 (y) and distance from the inoculum source (x) for four soybean cultivars[a]

Cultivar	Year-location[c]	Parameter[b] Intercept (a)	Parameter[b] Spread (-m) (b)	R^{2d}	$P > F^e$ (lack of fit)	Half distance (m)	Mean[f]
W550	1986–B.R.	13.42 ± 2.59	1.25 ± 0.20	0.97	0.95	0.55	1.45
DPL105	1986–B.R.	18.63 ± 4.75	1.54 ± 0.29	0.97	0.94	0.45	1.48
Bedford	1987–B.R.	3.18 ± 0.50	0.25 ± 0.07	0.76	0.91	2.82	1.76
W550	1987–B.R.	3.31 ± 0.37	0.43 ± 0.06	0.94	0.45	1.39	1.24
W550	1987–Jean.	2.12 ± 0.29	0.30 ± 0.07	0.85	0.25	2.34	1.05
Bay	1987–B.R.	1.14 ± 0.23	0.71 ± 0.15	0.91	0.99	0.98	0.26

[a] Exponential model was fitted to mean number of cankers per plant over eight directions from three plots ($N = 18$).
[b] Parameters represent the predicted number of cankers per plant (y) at $x = 0$ (a) and rate of decline in y over x (b) (= spread parameter). 95% confidence interval follows each estimate.
[c] B.R. = Baton Rouge, Jen. = Jeanerette.
[d] Coefficient of determination.
[e] Significance of F test for lack of fit, large P indicates good fit.
[f] Mean number of cankers per plant of three plots per cultivar.

was lower and b was greater for Bay in comparison to Bedford and W550 in 1987. Half-distances for Bedford and W550 in 1987 were approximately 3–4 subplot increments.

Estimates of stem canker incidence per plot, made by calculating the area under the exponential equations, provided a reasonable estimate when compared to the actual mean (Table 2). The estimated densities were, however, always lower than the actual plot mean.

Mean incidence of plants killed per plot at mid-R_5 was similar for DPL105 and W550 in 1986 (Table 3). Incidence of dead plants in 1987 was greatest for Bedford, intermediate for W550, and zero for Bay. Incidence of dead plants for W550 was 2.7 and 2.6 times greater for the two locations in 1987 compared with 1986. Estimates of b did not differ between the two cultivars in 1986 and were greater than those for disease incidence. Estimates of b for W550 at Baton Rouge in 1987 were less than in 1986. In 1987, b was less for Bedford than for W550. Half-distances approximated 0.5 subplot increments in 1986, and ranged from approximately 1.5 (W550) to 3 (Bedford) subplot increments in 1987. Estimated incidence of dead plants per plot (by integration) was always less than but near the mean incidence.

Mean number of cankers per plant at mid-R_5 was the same for DPL105 and W550 in 1986 (Table 4). Estimates of b for canker number were similar for the two cultivars. Mean cankers per plant in 1987 were greatest for Bedford, intermediate for W550, and least for Bay. Canker number for W550 in 1986 was 14 and 27% lower than in 1987 at Baton Rouge and Jeanerette, respectively. The estimate of b for W550 in 1986 was less than in 1987. Estimates of b at Baton Rouge in 1987 were lowest for Bedford, intermediate for W550, and greatest for Bay. Half-distances for intermediate cultivars were less than 1 subplot increment in 1986 and greater than 1 subplot increment in 1987.

Direction from the source had a significant effect on disease incidence at each location ($P = 0.01$). The effect of cultivar was not significant in 1986, but was in 1987 at Baton Rouge ($P = 0.01$). The cultivar × direction interaction was not significant at Baton Rouge in 1986 or 1987, therefore, data were pooled across cultivars (Table 5). In 1986, incidence was greatest to the E, down the row and parallel to the field slope. Incidence was also high to the N, across rows and near parallel to prevailing winds during the first 60 days after planting (from SSE in both years). Directional differences were less apparent at Baton Rouge in 1987 where disease incidence was high (Table 5). Incidence was again high to the E and N, however, not significantly higher than several other directions. In 1987 at Jeanerette (Table 5), incidence was high to the E and W, parallel to the row, and to the N near parallel to prevailing winds (from SSE).

Effect of stem canker on yield. Yield was negatively correlated with stem canker incidence, number of cankers per plant, and incidence of dead plants. Linear regression of stem canker incidence on yield (g/subplot) generally resulted in the highest R^2 values. The linear relationship was significant for all cultivars in 1986 and in 1987 except for Bay (Table 6). Lesions on Bay did not elongate. Predicted yield losses for susceptible and intermediate cultivars at 100% disease incidence (= regression slope) ranged from 50 to 60% and were not significantly different. Variation was higher for DPL105 in 1986 and for Bedford in 1987 than for W550 in 1986 and 1987 (Fig. 4A–B, D–F). Stem canker incidence had no significant effect on yield of Bay (Fig. 4C).

TABLE 5. Relationship between stem canker incidence at mid-R_5 and direction from the inoculum source at three locations

Direction	Proportion disease[a] Baton Rouge-1986[b]	Baton Rouge-1987[c]	Jeanerette-1987[d]
E	0.59 a	0.59 a	0.53 b
SE	0.21 cde	0.51 bc	0.33 cd
S	0.25 cde	0.52 abc	0.53 b
SW	0.16 e	0.43 c	0.22 d
W	0.28 cd	0.49 bc	0.58 ab
NW	0.19 de	0.47 bc	0.40 bcd
N	0.42 b	0.55 ab	0.72 a
NE	0.29 c	0.51 bc	0.46 bc

[a] Values in a column followed by the same letter are not significantly different at $P = 0.05$ according to Duncan's multiple range test. Range test performed on mean values over six distances from the inoculum source.
[b] Mean values of three plots of each W550 and DPL105 ($n = 6$).
[c] Mean values of three plots of each Bay, Bedford, and W550 ($n = 9$).
[d] Mean values of three plots of W550 ($n = 3$).

Environmental influences. Temperature and rainfall data were summarized only by month (30-day interval) after planting because of the slow development of stem canker in the field. (Table 7). Although only three locations were used, correlations were made between mean disease incidence and weather data to obtain preliminary information. Correlations were made for intermediate and susceptible cultivars ($N = 5$) and the corresponding weather variables at each location (Table 7). Average of daily minimum temperature was positively correlated with incidence in month 3 after planting ($P = 0.01$). Average of daily maximum temperature was positively correlated with incidence in month 1 ($P = 0.1$) and negatively correlated with incidence in months 3 and 4 ($P = 0.05$). Cumulative rainfall was positively correlated with incidence in months 1 ($P = 0.05$) and 3 ($P = 0.01$). No significant ($P < 0.1$) correlations were obtained for number of rainy days with incidence.

DISCUSSION

The pattern and extent of stem canker spread differed depending on the level of cultivar resistance and year. Stem canker incidence and severity was reduced with increasing cultivar resistance. While conditions were conducive for stem canker development in both years and locations, spread was more extensive in 1987. The increase in disease incidence in 1987 over 1986 was believed to

be related to differences in cumulative rainfall. These factors help explain the sporadic nature of severe stem canker outbreaks that have been observed in the southern region in recent years.

The selection of cultivars used in this study was based on reported field reactions to stem canker and unpublished observations made in Louisiana. The responses of the selected cultivars paralleled reported responses with the exception of DPL105. DPL105 gave an intermediate response, in contrast to unpublished reports in Louisiana but in agreement with published results of others (2). Variation in response of cultivars to the strain(s) of *D. p. caulivora* that cause stem canker in the southern region have been reported. This has been attributed to variation in virulence among isolates from different geographical areas (12) and to environment (2). For this reason, several isolates of *D. p. caulivora* collected in different areas of the state were used for inoculum in this study.

Temporal progress of stem canker was typical of a monocyclic disease. The progress curves in 1986 exhibited inverted j-shapes characteristic of monocyclic disease increase (3). The shape parameters obtained from fitting the Weibull model to disease progress curves in 1987 were not significantly different from the idealized value of a monocyclic epidemic (3,24). The lack of in-season production of either pycnidia or perithecia during the 1986 and 1987 seasons suggests that secondary spread of this disease probably does not occur. The long incubation period required for symptom development also negates secondary spread as a plausible mechanism of disease increase.

The effect of resistance on disease progress was expressed

TABLE 6. Relationship of stem canker incidence at mid-R_5 (x) to percent of maximum yield (y) for four soybean cultivars at Baton Rouge in 1986 and 1987[a]

Cultivar	Year	df	Parameter[b] Intercept (a)	Parameter[b] Slope (b)	R^{2c}	$P > F^d$ (regression)	$P > F^e$ (lack of fit)
W550	1986	18	99.99 ± 8.62	−49.10 ± 18.98	0.65	0.001	0.339
DPL105	1986	18	108.21 ± 24.60	−59.78 ± 58.30	0.23	0.045	0.585
Bedford	1987	18	99.99 ± 25.52	−51.97 ± 34.97	0.38	0.006	0.788
W550	1987	18	100.19 ± 10.99	−58.63 ± 17.72	0.75	<0.001	0.188
Bay	1987	18	99.99 ± 9.55	4.64 ± 34.96	0.005	0.782	0.910
W550	2 yr	36	99.97 ± 6.11	−55.08 ± 11.23	0.74	<0.001	0.268

[a] Linear model was fitted to mean values over eight directions from three plots per cultivar.
[b] Parameters represent the predicted percent of maximum yield (y) at $x = 0$ (a) and rate of change in y over x (b). 95% confidence interval follows each estimate.
[c] Coefficient of determination.
[d] Significance of F test for regression.
[e] Significance of F test for lack of fit, large P indicates good fit.

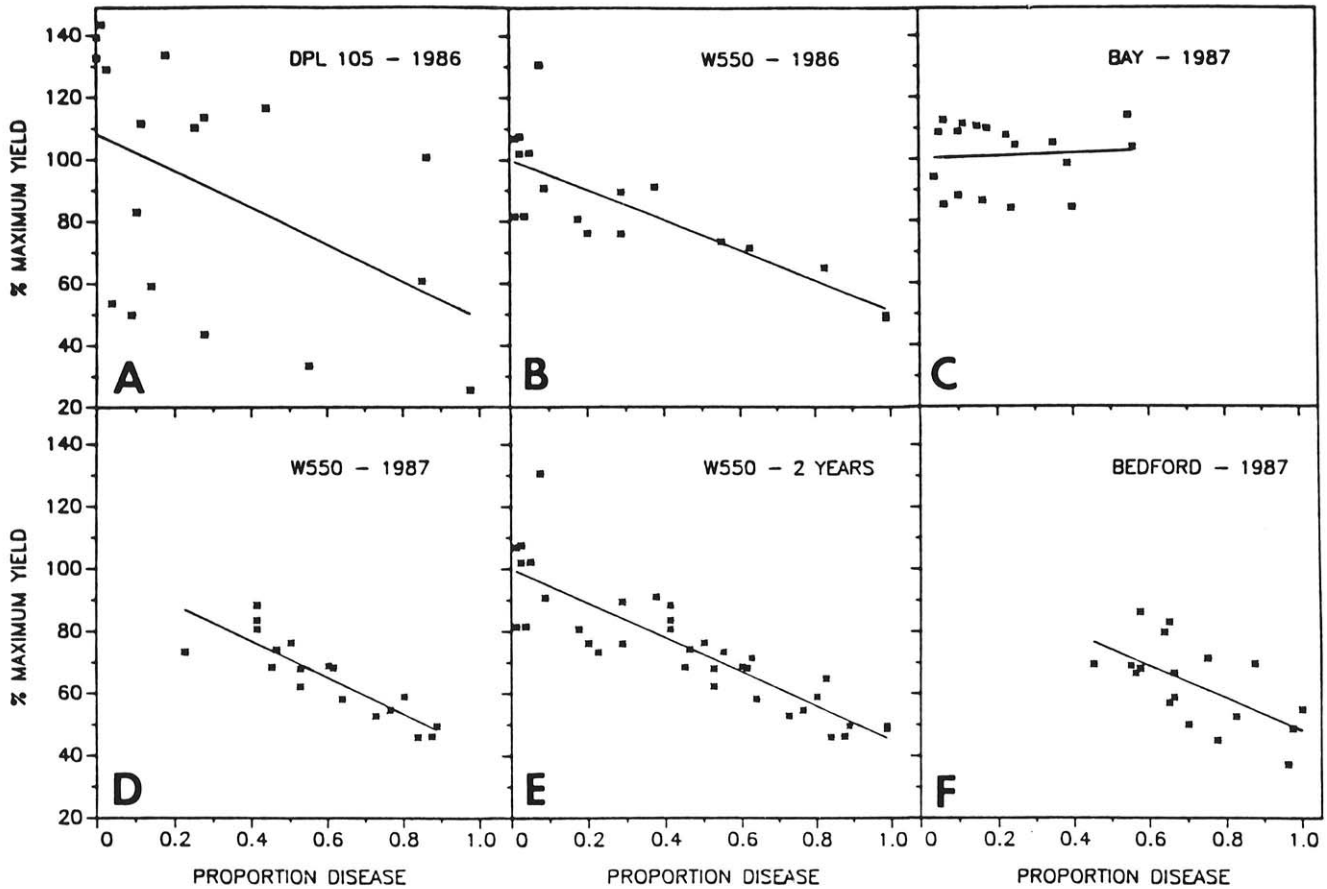

Fig. 4. Linear relationships between yield loss and stem canker incidence at, **A and B,** Baton Rouge in 1986, **C–E,** Baton Rouge in 1987, and **F,** combined 1986 and 1987. DPL105 and W550 are intermediate in response to stem canker, Bedford is susceptible, and Bay is resistant. Data points represent mean values of three plots per cultivar across eight directions ($N = 18$).

through a reduction in both infection rate and maximum level of disease. A third parameter for generalized progress of monocyclic diseases as described by Berger and Bartz (3) is the incubation period. Resistance and year had no effect on incubation period in this study as initial symptoms consisting of restricted lesions appeared on all cultivars 7–8 wk after planting (Figs. 1 and 2). Damicone et al (5) reported that the duration of free moisture after spore deposition influenced the length of the incubation period of stem canker with a susceptible breeding line. Although complete vegetative wetness data were not obtained in this study, data in Baton Rouge during 1986 and 1987 (Damicone, *unpublished*) indicate vegetative wetness routinely occurs 8–10 hr per night in the summer regardless of rainfall. Eight hours of daily vegetative wetness after spore deposition was shown to be sufficient for stem canker development (5).

Disease gradients were affected by both environment and cultivar resistance. The steeper gradients and restricted spread observed for W550 in 1986 compared with 1987 were believed to be a result of differences in cumulative rainfall. Increases in rain splash and runoff with with high levels of rainfall possibly results in spore deposition over a larger area from the source, thus reducing gradient steepness. Effects of levels of resistance on gradient steepness were more pronounced for disease severity (cankers/plant, proportion dead plants) than for incidence. At Baton Rouge in 1987, gradients of stem canker incidence differed only between resistant and susceptible cultivars while gradients of severity were different for all three levels of resistance. These results are in contrast to reports that gradients are not reliable indicators of cultivar resistance (4,10,18,19). Diseases studied in these reports were polycyclic rusts. Slow-rusting resistance is characterized in part by reduced lesion size and/or spores produced per lesion. Slow-rusting results in reduced secondary inoculum and slower epidemic development. In contrast, stem canker disease develops slowly from only primary inoculum. Intermediate resistance in W550 would have been overlooked by comparing gradients of incidence with susceptible Bedford at Baton Rouge in 1987 (Table 2).

Ploetz and Shokes (27) reported a linear effect of inoculum concentration on stem canker infection. A spore concentration of 10^5 spores per milliliter was required for infection of 50% of the stem nodes that were inoculated. Damicone et al (5) used 10^6 spores per milliliter sprayed on plants to runoff to achieve one or two cankers per plant under high moisture conditions. *D. p. caulivora* apparently has a low infection efficiency. The differing gradients observed between cultivars in 1987 apparently were a result of differential response of cultivars to inoculum concentration.

Relationships between yield loss and stem canker incidence were constant for intermediate and susceptible cultivars; however, variation was high for DPL105 and Bedford. DPL105 was adversely affected by a pod-blanking syndrome of unknown etiology. Plant stand irregularities also contributed to yield variation with Bedford. The slopes of yield loss models for susceptible and intermediate types were the same as those obtained in a plot test using fungicide treatments to produce variation in stem canker incidence (6). Backman et al (2) reported a strong linear relationship between yield and a pretransformed rating scale based on the percent of plants dead or dying in a plot at late R_6. Disease evaluations in this study and in the fungicide test (6) were made by determining the condition of individual plants at mid-R_5. No transformation was required to linearize the data. No special consideration was given to plants dying during the evaluation. These differences may have contributed to the apparent discrepancies in model predictions. The yield loss model of Backman et al (2) predicted 88% yield loss at the highest disease rating. Predicted yield losses at 100% incidence for susceptible and intermediate cultivars in this study ranged from 49 to 60%. Predicted yield loss at 100% incidence for the maturity group VII cultivar Bragg (susceptible) (2) in the fungicide test was 50% (6).

Preliminary correlations revealed that temperature and cumulative rainfall but not rain days were related to disease incidence. Ploetz and Shokes (27) reported infection by *D. p. caulivora* at temperatures ranging from 10 to 34 C. At 10–22 C and above 34 C, however, infection was markedly lower than at 28–34 C (27). Low minimum temperatures in month 3 and high maximum temperatures in months 3 and 4 may have limited infection or disease development in this study. Early-season rainfall during the vegetative stages of plant growth is probably critical for stem canker development in a field infested with *D. p. caulivora*. Infections during the vegetative stages precede a long incubation period (>50 days) (5,26) and are most closely related to final disease severity during the reproductive stages (2,33,34). Moisture from rainfall during this period should affect spore development and release from stem debris on the soil surface. Spread and deposition of spores released in a gelatinous matrix onto plant surfaces would also require rainfall. Preliminary evidence in support of the importance of rainfall was the positive correlation between cumulative rainfall and disease incidence in this study. The lack of correlation between the number of rainy days and disease incidence differed from another report (32). Perhaps the intensity and longevity of rain events differed between studies.

Differences in disease development by direction from the source appeared to be influenced by field slope, row direction, and prevailing wind. Disease development across rows was greatest downwind (Table 5). Splashing rain driven by prevailing winds may have deposited spores downwind. Surface water movement patterns in plots could also account for directional differences. Planters used in this study formed elevated ridges parallel to each row. These ridges may have confined water movement to rows. During periods of heavy rain it is plausible that spores moved with surface runoff down rows parallel to the slope (Baton Rouge), or moved in both directions in the source row where there was no slope (Jeanerette).

These results reveal several strategies for management of stem canker. Since secondary cycles apparently do not occur, destruction of primary inoculum by burial, burning, and/or crop rotation

TABLE 7. Monthly (30-day) summaries of temperature and rainfall data and correlation of weather variables with disease incidence from field plots in Baton Rouge in 1986 and 1987, and Jeanerette in 1987

	Average daily minimum temperature (C)				Average daily maximum temperature (C)				Rain days (no.)				Cumulative rainfall (cm)			
Month[a]	1[b]	2[c]	3[d]	r[e]	1	2	3	r	1	2	3	r	1	2	3	r
1	20.4	21.1	21.6	0.65 ns	29.5	30.1	30.2	0.82*	13	15	18	0.46 ns	11.5	33.9	30.4	0.92**
2	21.9	21.9	22.7	−0.01 ns	31.2	31.7	32.0	0.68 ns	16	16	13	0.01 ns	16.4	13.6	17.3	−0.79 ns
3	22.7	23.2	23.0	0.96***	33.1	32.2	32.3	−0.91**	11	12	16	0.22 ns	15.0	28.2	22.4	0.97***
4	21.0	20.1	21.3	−0.78 ns	32.4	30.1	32.3	−0.90**	9	8	10	−0.59 ns	3.9	5.7	16.5	0.15 ns

[a] Month after planting.
[b] Baton Rouge, 1986.
[c] Baton Rouge, 1987.
[d] Jeanerette, 1987.
[e] Correlation coefficient (r) for relationship between monthly weather variable and disease incidence (see Table 2) for susceptible and intermediate cultivars (W550, DPL105, and Bedford) ($N = 5$); *** = $P = 0.01$, ** = $P = 0.05$, * = $P = 0.1$, ns = not significant.

should reduce stem canker. This is in agreement with reported reductions in disease incidence using certain tillage systems (29,30). Destruction of residue could be integrated with planting a cultivar with some level of resistance to limit disease incidence and development. Unfortunately, there are relatively few resistant cultivars, and those with resistance often lack traits such as cyst nematode resistance or are not adapted to particular locations. For example, the maximum yield obtained for the resistant cultivar Bay in this study was 31% less than the maximum for for the intermediate cultivar W550 in the presence of stem canker. Bay is the only known resistant cultivar in maturity group V (2). Producers thus often select intermediate cultivars and continue to risk losses to stem canker. Fungicides can reduce losses from stem canker (2,6); however, they must be applied before symptom development without knowledge of eventual disease outcome. Researchers are continuing to use the methods outlined herein to study stem canker spread over several years and locations to better understand the effects of environment on it.

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