Analysis of Stem Canker Epidemics in Irrigated and Nonirrigated Conditions on Differentially Susceptible Soybean Cultivars

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ABSTRACT

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Incidence and severity of stem canker caused by *Diaporthe phaseolorum* var. *caulivora* were quantified from an inoculum point source in irrigated and nonirrigated plots of resistant, moderately resistant, and susceptible soybean cultivars (Bay, Wilstar 550, and Bedford, respectively) during the 1988–90 seasons. Stem canker severity at each disease assessment was higher on all cultivars in irrigated plots. In general, disease progress was described better by the logistic model than by the monomolecular model for all cultivars in both irrigated and nonirrigated plots. The apparent infection rates of the logistic model were higher on all cultivars in irrigated plots compared with those in nonirrigated plots. Similarly, the gradient and velocity of spread were greater in the irrigated plots than in the nonirrigated plots. Greater stem canker severity and higher apparent infection rates occurred on the susceptible cultivar Bedford than in the moderately resistant and resistant cultivars. Pycnidia and perithecia occurred on the infected plants during each growing season, indicating that the fungus produces more than one cycle of inoculum within a season. This secondary inoculum was capable of infecting soybean plants in the greenhouse at reproductive growth stages. Better fit of the disease progress data to the logistic model also indicates that secondary infections do occur in the field. However, in the field symptoms from infections by the secondary inoculum are irregular during the crop season because of the prolonged incubation period. Therefore, stem canker should be classified as a both primary inoculum-dependent and infection rate-dependent disease for apparent lack of regular symptom expression from secondary infections and the confounding effects of symptomless, secondary infections.

Additional keywords: epidemiology, *Glycine max,* irrigation, monocyclic, polycyclic, resistance.

Stem canker, caused by *Diaporthe phaseolorum* (Cooke & Ellis) Sacc. var. *caulivora* K. L. Athow & R. M. Caldwell, is currently an important disease of soybeans (*Glycine max* (L.) Merr.) throughout the southeastern United States (3,9,11,12,18,24,25). The disease was prevalent and reduced yields of susceptible cultivars in the midwestern United States during the 1940s and 1950s (1). It has ceased to be a problem in that region because of the use of resistant cultivars (3). The pathogens causing stem canker in the Midwest and southern United States are considered distinct formae speciales on the basis of cultural, morphological, and physiological differences (2,3).

D. p. caulivora overwinters on diseased and apparently diseased, symptomless, soybean stem. The debris from the previously infected crop serves as the principal source of primary inoculum for successive soybean crops (3,18,21). Seed also has been suspected as a source of primary inoculum (13), but its role in the disease cycle is uncertain (3). Optimal (−0.04 MPa) and maximal (0 to −0.02 MPa) soil moisture initiate the production of perithecia on the debris, and continued availability of soil moisture favors ascospore production (27). Factors affecting the formation of pycnidia and production of conidia are not well understood (27). Both ascospores and conidia are capable of causing infection (6,18). Ascospores and conidia are dispersed by splashing raindrops, and the splash-borne spores fall on petioles, petiole bases, and stem tissue of soybean seedlings, causing infection. Pinpoint lesions on the stems and petioles appear early in the season, and canker symptoms typically appear at reproductive growth stages after 50–80 days of incubation (3,7,19).

Free moisture is critical for spore dispersal, infection, and disease development (6,19). Continuous or discontinuous free moisture after inoculation reduces incubation period while increasing the incidence and severity of the disease (6). Stem canker infections occur over a wide temperature range (19).

Soybean cultivars differ in their susceptibility to stem canker (3,7,10,11,29). Similarly, isolates of the pathogen also vary in their virulence to soybean cultivars (11). Soybean cultivars have been classified as susceptible, moderately susceptible/resistant (intermediate), and resistant on the basis of their reactions to the pathogen (9). An integrated approach of altered tillage (21), planting of resistant cultivars (3,7), and use of fungicides (3) is followed to prevent or manage the disease.

Increased stem canker incidence and severity and associated yield losses occur in years with abundant rainfall. However, experimental evidence is lacking to confirm this association. As part of a project to develop a forecasting system for stem canker, we evaluated stem canker development on three differentially susceptible soybean cultivars in irrigated and nonirrigated conditions. Preliminary results have been reported (26).

MATERIALS AND METHODS

Experiments were conducted during the 1988–90 soybean growing seasons at the Ben Hur Research Farm near Baton Rouge, LA. During 1989, the experiment was also conducted at the Citrus Research Station in Port Sulphur, LA.

Plot establishment. Three soybean cultivars (Bedford, susceptible; Wilstar 550, intermediate; and Bay, resistant) in maturity group V were chosen for the study because of their reaction to the stem canker pathogen (9) and because stem canker epidemics had been previously characterized on them (7). The experiments were planted in fields with no previous history of stem canker

on 20 May 1988, 26 May 1989, and 28 May 1990 at Ben Hur and on 24 May 1989 at Port Sulphur. The treatments were arranged in a split-plot design and replicated three times, wherein the replications and irrigation treatments were the main plot experimental units. The cultivars formed the subplots. Subplot dimensions were 9.75 × 9.75 m, consisting of 13 rows with a row spacing of 0.75 m. The subplots were separated by a 2-m fallow space, and main plots were separated by a 25-m fallow space to reduce plot interactions. Before sowing, the seeds of each cultivar were treated with soybean inoculant (Legume Aid, Kalo Inc., Overland Park, KS).

Inoculations. Each year the subplots were inoculated either on the day of planting or within 3 days after planting at the V_0 stage of soybean growth (8). The method of inoculation has been described previously (7) and is summarized here. An inoculum point source was established in the center of each subplot with 0.5 kg of stem canker-infested soybean debris and 2 L of *D. p. caulivora*-colonized oat kernels on the surface of the soil. The inoculum was contained within a 20-cm-wide wire mesh that extended 5 cm into the soil and was supported at the corners with garden stakes.

Irrigations. For subplots in the nonirrigated treatment, rainfall was the source of irrigation. To simulate additional rainfall in excess of the ambient, the subplots in the irrigated treatments were subjected to periodic sprinkler irrigation. Sprinklers were activated once each week for 1 h each in the mornings (8:30–9:30 a.m.) and evenings (6:00–7:00 p.m.) on rain-free days, beginning with the date of inoculation.

Maximum and minimum temperature and leaf wetness were continuously monitored in the canopies of irrigated and nonirrigated blocks using sensors on a CR21 micrologger (Campbell Scientific Inc., Logan, UT). Daily rainfall data were also obtained from the Bench Mark weather stations located at each experimental site for the period from inoculation to final disease assessment.

Disease assessment. Stem canker incidence (ratio of the number of plants infected to the total plants assessed) and stem canker severity (area of the main stem covered by cankers, scored on a 0–100% scale) were assessed on 10 randomly chosen plants at 2-wk intervals in 1988 and 1989 and at weekly intervals in 1990. Disease assessments began on the date of appearance of canker symptoms. In 1988 and 1989, disease assessments were made 0.75, 1.5, 2.25, 3.0, 3.75, and 4.5 m away from the focal center in eight different directions. In 1990, canker severity and incidence were assessed at only one sampling point that was 0.75 m away from the point source in each subplot. To confirm that stem canker was the disease assessed, at each disease assessment pathogen isolations were made from the cankered tissue on a semiselective medium for *D. p. caulivora* (17) in petri dishes, and the cultural characteristics were compared with a known culture of the fungus.

Secondary inoculum and infection of soybean plants. To determine whether the fungus produces secondary inoculum during the season, at each disease assessment 50 cankers from each cultivar were examined under a stereoscope for the presence of pycnidia and perithecia. To determine the effectiveness of the secondary inoculum in causing secondary infections, soybean plants of the susceptible cultivar Bedford at reproductive growth stages R_2–R_5 (8) grown in 30-cm-diameter pots were inoculated with ascospores (10^6 ascospores per milliliter obtained by incubating canker-infested soybean debris at the optimal soil moisture [27]) or conidia (10^6 conidia per milliliter obtained by incubating canker-infected stem pieces in moist chambers at 25 ± 1 C) and incubated on benches in a temperature-controlled greenhouse (30 ± 2 C) under 16 h of moisture per day. The inoculated plants were monitored for stem canker development. This experiment was repeated two times.

Statistical analyses. Areas under the disease progress curves (AUDPC) were calculated following the procedure outlined by Campbell and Madden (5) for each cultivar in both irrigated and nonirrigated plots. Analysis of variance was used to determine the effects of irrigation (main plot), cultivar (subplot), and interactions each year on AUDPC and final stem canker severity. In 1989, the effects of location were also determined. The error terms used to test location, irrigation treatment, and cultivar effects were variances of replication-location, replication-treatment, and replication-treatment-cultivar interactions, respectively.

Linearized forms of monomolecular (5,28) and logistic (5,28) models were evaluated for goodness of fit to the disease progress data (severity) for each cultivar in irrigated and nonirrigated conditions. Criteria for goodness of fit of linear models included examination of the plot of disease progress over time, coefficient of determination for regression for each model, and plots of standardized residuals (5).

The logistic model was the most appropriate model according to the goodness of fit criteria mentioned above, and apparent infection rates calculated from this model were used to compare cultivars, seasons, and effects of irrigation using a paired *t* test. Disease gradients were also calculated for each cultivar in irrigated and nonirrigated conditions for the data from the 1988 and 1989 seasons using the logit-linear model (5, p. 264), and the data were consistent temporally from plot to plot. The gradient parameters for the three cultivars between the irrigated and nonirrigated treatments were compared using a paired *t* test. The apparent infection rates for each cultivar in irrigated and nonirrigated conditions were divided by the corresponding gradient estimates to obtain the velocity of spread of the epidemics (14). The velocities of the epidemics were also used to compare cultivars, seasons, and effects of irrigation using a paired *t* test.

The occurrence of secondary infections during the season was determined indirectly by fitting the logistic and monomolecular models to disease progress data between 0 and 90–110 days after inoculation. In our disease progress data, this meant omitting the final disease assessment data in 1988 and 1989 and the final two disease assessment data in 1990 from the analysis. If the monomolecular model fitted the data better than the logistic model, then it is indirect evidence for secondary infections in the field. Analyses were conducted using SAS procedures (22).

RESULTS

Stem canker severities and AUDPC were significantly different in the two locations in 1989. Sprinkler irrigation significantly affected both stem canker severity and AUDPC each year. Both stem canker severity and AUDPC were significantly different in the three cultivars each year (Table 1). All two-way and three-way interactions were nonsignificant except for cultivar × location for canker severity in 1989 and cultivar × irrigation treatment for both canker severity and AUDPC in 1990 (Table 1).

Stem canker progress in irrigated and nonirrigated treatments. Each year, canker symptoms on all cultivars in the irrigated plot consistently appeared 50–65 days after inoculation. Symptom expression in the nonirrigated plots was delayed by 2 wk. Differences in canker severity on all cultivars between irrigated and nonirrigated plots were recorded throughout the disease progress, although these differences were less pronounced in the early stages of disease progress. Canker severity was invariably higher in irrigated plots than in nonirrigated plots of all cultivars (Fig. 1). Stem canker was most severe in 1988 (Fig. 1A and B), intermediate in 1989 (Fig. 1C–F), and least severe in 1990 (Fig. 1G and H). In 1989, higher stem canker severities were recorded on all cultivars at Baton Rouge (Fig. 1C and D) than at Port Sulphur (Fig. 1E and F).

Apparent infection rates calculated from the logistic model were significantly ($P \leq 0.05$) higher on all cultivars in irrigated plots than in nonirrigated plots. The differences in the rates of disease progress in irrigated and nonirrigated plots were better expressed in the intermediate and susceptible cultivars than in the resistant cultivar. The rate of disease progress was higher in all cultivars during the 1988 season. During the 1989 season, the disease progressed faster at Baton Rouge than at Port Sulphur (Table 2).

The extent of stem canker spread measured by the disease gradients from the focal center were significantly ($P \leq 0.05$) steeper

TABLE 1. Abbreviated analysis of variance F tests and significance ($P > F$) for the effects of irrigation treatment (main plot) and cultivar (subplot) on final stem canker severity and the areas under the disease progress curve (AUDPC) on soybean plants

Source of variation[a]	df	1988		1989		1990	
		F	$P > F$	F	$P > F$	F	$P > F$
Stem canker severity							
L	1	...	...	1,156.26	0.0009	...	...
T	1	20.21	0.0461	217.81	0.0046	204.93	0.0048
L × T	1	...	...	8.27	0.1026	...	...
C	2	23.56	0.0004	292.74	0.0001	1,860.35	0.0001
C × L	2	...	...	15.08	0.0019	...	...
C × T	2	0.40	0.6380	0.38	0.6956	65.60	0.0001
L × T × C	2	...	...	0.24	0.7921	...	...
AUDPC							
L	1	...	...	78.87	0.0124	...	...
T	1	23.16	0.0406	23.12	0.0400	26.08	0.0363
L × T	1	...	...	6.05	0.1331	...	...
C	2	31.38	0.0002	31.29	0.0002	47.65	0.0001
C × L	2	...	...	1.62	0.2566	...	...
C × T	2	1.72	0.2391	4.20	0.0566	8.01	0.0123
L × T × C	2	...	...	3.52	0.0801	...	...

[a] L, location; T, treatment; C, cultivar.

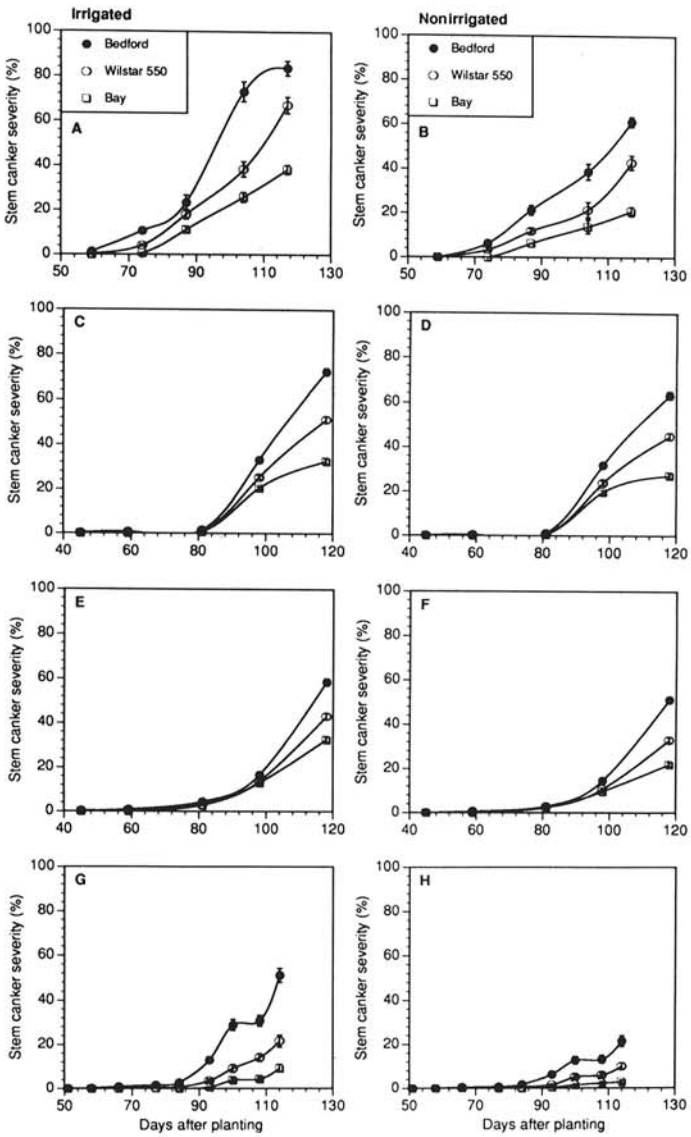

Fig. 1. Stem canker (severity) progress curves for three soybean cultivars. **A** and **B,** Irrigated and nonirrigated plots, respectively, at Baton Rouge in 1988; **C** and **D,** irrigated and nonirrigated plots, respectively, at Baton Rouge in 1989; **E** and **F,** irrigated and nonirrigated plots, respectively, at Port Sulphur in 1989; **G** and **H,** irrigated and nonirrigated plots, respectively, at Baton Rouge in 1990.

in most nonirrigated plots than in the irrigated plots (Table 3). Similarly, stem canker spread occurred with significantly greater velocity in all irrigated plots than in nonirrigated plots except in one case (Table 3).

Rainy days were equal in number in 1988 and 1989 at Baton Rouge and approximately the same at Port Sulphur in 1989 and at Baton Rouge in 1990. The irrigated plots received 11–14 irrigations in different years and locations (Table 4). In irrigated plots, the average leaf wetness per day was 13.23 h, compared with 8.67 h in the nonirrigated plots. The average maximum and minimum temperatures in irrigated plots were 31–37 and 15–19 C, respectively.

Stem canker progress on different cultivars. Although the shapes of the stem canker progress curves were similar for the three cultivars, the canker severities were significantly different (Fig. 1). Canker severities were greatest on Bedford, intermediate on Wilstar 550, and lowest on Bay (Fig. 1). In irrigated plots, stem canker severity was most severe on all cultivars in 1988, followed by 1989 and 1990 (Fig. 1). These differences were also evident with the apparent infection rates except in 1988 (Table 2). Disease progress was fastest on Bedford in both irrigated and nonirrigated plots, followed by Wilstar 550 and Bay (Table 2). The disease gradient was significantly steeper in nonirrigated plots of all cultivars, locations, and years than disease gradient in the irrigated plots except for Bay and Wilstar 550 at Baton Rouge and Wilstar 550 at Port Sulphur in 1989. Similarly, velocity of spread was significantly greater for all cultivars in the irrigated plots than in the nonirrigated plots except for Wilstar 550 in 1989 at Port Sulphur (Table 3).

Secondary inoculum and infection of soybean plants. A schematic representation of the events in the disease cycle is presented in Figure 2. About 80 days after inoculation, when the soybean plants were in R_2 and R_3 growth stages, blisterlike structures within the cankers were observed. Closer microscopic examination of these structures revealed that they were pycnidia. Whole mounts made from the pycnidia revealed abundant alphaconidia. Production of pycnidia was consistently observed in the cankers of all cultivars at each disease assessment after their initial occurrence. Perithecia also appeared approximately 40 days after the appearance of pycnidia when the plants were at R_5 stage. However, production of perithecia was less frequent and was observed only on susceptible Bedford. Inoculations of soybean plants at reproductive growth stages R_2–R_5 with ascospores and conidia produced typical canker symptoms in the greenhouse each time the experiment was conducted.

Modeling temporal stem canker progress. Compared with the monomolecular model, the logistic model provided a superior fit to the data in a majority of cases (80%) (Table 2). The rates of disease progress calculated from the logistic model discrimi-

nated the irrigated and nonirrigated treatments and the cultivars better than the rates obtained from the monomolecular model, as indicated by the significance of t tests for all treatment combinations (Table 2).

The model fits to detect possible secondary cycles within a season were better for the monomolecular model than for the logistic model in approximately 80% of the cases. The model fits were higher for 1988 and 1989 than for 1990. However, in 1990, the logistic model fitted the data better than the monomolecular model (Table 5).

DISCUSSION

The development of stem canker epidemics caused by *D. p. caulivora* was significantly increased in the susceptible, intermediate, and resistant cultivars by frequent sprinkler irrigations. The stem canker symptom appearance was also hastened by the sprinkler irrigations. The higher severity of stem canker epidemics was the result of increased rate of disease progress and extent and velocity of spread. This work provides the first experimental evidence for the common assertion that increased stem canker incidence and severity are associated with abundant rainfall.

Both ascospores and conidia are produced in a gelatinous matrix by *D. p. caulivora* (3,19,27), which depends on rain splash for dispersal and deposition on susceptible host tissue. During the soybean-growing season in Louisiana, the days are hot and humid and the nights are rich in dew, which may contribute to the production of inoculum. However, successful infection of plants depends on the efficient dispersal of this inoculum. Overhead sprinkler irrigation wets nearly the entire surface of the soil and the crop; more importantly, it increases splash dispersal of soil and inoculum (20). In our experiments, sprinkler irrigations may have contributed to increased production of inoculum and dispersal and subsequent infection of soybean plants in the irrigated plots. The sprinkler irrigations also lengthened the average wetness duration in the irrigated plots.

Previous studies have shown that stem canker development is moisture-dependent (6,19). Maximum levels of stem canker incidence and severity and minimum length of incubation period were observed with at least 8-h wetness duration (6). In the irrigated plots, the average wetness duration measured was 10.67 h, which satisfied the requirements to achieve maximum levels of

TABLE 2. Apparent infection rates (with corresponding standard errors of mean) calculated from logistic and monomolecular models fitted to stem canker severity on three soybean cultivars in irrigated and nonirrigated conditions in the 1989–90 seasons

Location and year	Cultivar	Irrigated	R^2	Nonirrigated	R^2	t Test[a]
Monomolecular model						
Baton Rouge, 1988	Bay	0.010 ± 0.0019	0.40	0.005 ± 0.0011	0.39	*
	Wilstar 550	0.020 ± 0.0025	0.61	0.010 ± 0.0020	0.57	*
	Bedford	0.036 ± 0.0055	0.55	0.029 ± 0.0070	0.29	NS
Baton Rouge, 1989	Bay	0.008 ± 0.0001	0.99	0.007 ± 0.0004	0.96	*
	Wilstar 550	0.014 ± 0.0009	0.96	0.011 ± 0.0009	0.94	**
	Bedford	0.025 ± 0.0032	0.89	0.020 ± 0.0020	0.92	NS
Port Sulphur, 1989	Bay	0.009 ± 0.0010	0.89	0.005 ± 0.0004	0.94	*
	Wilstar 550	0.012 ± 0.0017	0.86	0.009 ± 0.0011	0.88	NS
	Bedford	0.019 ± 0.0032	0.82	0.016 ± 0.0023	0.84	NS
Baton Rouge, 1990	Bay	0.001 ± 0.0003	0.59	0.004 ± 0.0001	0.70	*
	Wilstar 550	0.003 ± 0.0008	0.67	0.001 ± 0.0003	0.70	*
	Bedford	0.010 ± 0.0022	0.69	0.003 ± 0.0007	0.76	*
Logistic model						
Baton Rouge, 1988	Bay	0.199 ± 0.0024	0.66	0.171 ± 0.0027	0.50	**
	Wilstar 550	0.180 ± 0.0022	0.64	0.148 ± 0.0027	0.42	**
	Bedford	0.208 ± 0.0013	0.75	0.189 ± 0.0016	0.66	**
Baton Rouge, 1989	Bay	0.099 ± 0.0012	0.90	0.081 ± 0.0010	0.90	**
	Wilstar 550	0.114 ± 0.0011	0.93	0.091 ± 0.0009	0.93	**
	Bedford	0.115 ± 0.0006	0.98	0.101 ± 0.0007	0.96	**
Port Sulphur, 1989	Bay	0.066 ± 0.0003	0.98	0.057 ± 0.0004	0.96	**
	Wilstar 550	0.073 ± 0.0003	0.99	0.074 ± 0.0006	0.95	*
	Bedford	0.084 ± 0.0004	0.99	0.081 ± 0.0004	0.98	*
Baton Rouge, 1990	Bay	0.067 ± 0.0013	0.94	0.056 ± 0.0014	0.89	*
	Wilstar 550	0.131 ± 0.0008	0.97	0.118 ± 0.0009	0.96	**
	Bedford	0.139 ± 0.0006	0.98	0.129 ± 0.0008	0.96	**

[a] NS = Paired t tests not significant at $P = 0.05$. * = and ** = Paired t tests significant at $P = 0.05$ and 0.01, respectively.

TABLE 3. Gradients (g) and velocity (v) of spread calculated using stem canker severity on three soybean cultivars in irrigated and nonirrigated conditions in 1988 and 1989 seasons

		g^{-m}			v^{m-day}		
Location and year	Cultivar	Irrigated	Nonirrigated	t Test[a]	Irrigated	Nonirrigated	t Test
Baton Rouge, 1988	Bay	1.000	1.395	**	0.199	0.123	*
	Wilstar 550	0.878	0.968	*	0.205	0.153	*
	Bedford	0.422	0.561	*	0.493	0.337	**
Baton Rouge, 1989	Bay	0.538	0.611	NS	0.184	0.133	*
	Wilstar 550	0.594	0.641	NS	0.192	0.142	*
	Bedford	0.550	0.742	**	0.210	0.136	**
Port Sulphur, 1989	Bay	0.504	0.595	*	0.131	0.096	*
	Wilstar 550	0.507	0.532	NS	0.144	0.139	NS
	Bedford	0.516	0.593	*	0.163	0.137	*
LSD ($P \leq 0.05$)		0.077			0.026		

[a] NS = paired t tests not significant at $P = 0.05$. * = and ** = Paired t tests significant at $P = 0.05$ and 0.01, respectively.

infection. The sprinkler irrigations in the irrigated plots represent seasons with increased rain events. Canker severities were greater in the irrigated plots of 1988 and 1989 at Baton Rouge (Fig. 1A, C, and E). Significantly higher stem canker severity was observed on all cultivars in 1988 at Baton Rouge (Fig. 1A and B) than in 1989 at Port Sulphur (Fig. 1E and F), although the total rainfall was comparable between the locations in the 2 yr. However, more rainy days (including the irrigation events) occurred in 1988, suggesting the importance of the number of rainy days compared with the total rainfall. The number of rainy days was significantly correlated with stem canker severity (K. V. Subbarao, *unpublished data*). Recorded temperatures in irrigated and nonirrigated plots differed little. Stem canker infections occur over a wide temperature range (19); therefore, temperature is unlikely to be a major factor influencing stem canker severity during the soybean season in Louisiana.

Cultivar susceptibility affected disease severity, rate of disease progress, and extent and velocity of spread. Increasing levels of cultivar resistance contributed to decreasing levels of disease severity, rate of disease progress, and extent and velocity of spread. On the basis of the different parameters, Bedford was ranked as the most susceptible, Wilstar 550 as intermediate, and Bay as the most resistant. These rankings are consistent with the characterization of resistance in these cultivars by previous workers (7,9). The logistic rates for the three cultivars are also consistent with the rates previously reported on these cultivars (7).

Each year, *D. p. caulivora* pycnidia were consistently observed in the cankers of all three cultivars. Backman et al (3) initially reported that pycnidia are not observed in season; subsequently, Backman et al (2) reported the in-season production of pycnidia in the southern United States. Other workers (10,12) have reported the production of perithecia during the soybean season. In our experiments, production of perithecia was less common and was observed only on Bedford. These observations indicate the production of secondary inoculum by *D. p. caulivora*. The fungus can also survive and sporulate as a saprotroph on wheat (*Triticum aestivum* L.) and weed species such as *Euphorbia heterophylla* L., *Aeschynomene americana* L., *Xanthium strumarium* L., and *Sesbania macrocarpa* Muhl. (Y. H. Lee, *unpublished data*). It is generally assumed that *D. p. caulivora* ascospores and conidia are incapable of infecting soybean plants at reproductive growth stages because of the hardiness and woody nature of soybean plants. The role of secondary inoculum in the epidemiology of the disease has not been studied (3). Inoculations of susceptible Bedford plants at reproductive growth stages in the greenhouse with ascosporial and conidial inocula caused stem canker, indicating that the secondary inoculum produced in the field is capable of causing infections on plants at reproductive growth stages. In our study, plausible secondary cycles were indirectly detected in a majority of stem canker disease progress data (Table 5). In the field, symptoms on the plants infected by the secondary inoculum are not regularly observed within the same season because of the lengthy incubation period. It is likely that the plants infected by the secondary inoculum remain asymptomatic (2,23) and increase the inoculum potential for the following season (isolations of *D. p. caulivora* from numerous asymptomatic plants were successful, and postseason observations of the apparently healthy plants revealed the production of perithecia on them). Production of perithecia is greatly influenced by the moisture status of the internodes, whereas production of pycnidia is unrelated to the moisture (27) and is governed by temperature (15). These observations have important disease management implications. In years with high rainfall, spraying fungicide on the crop to prevent postseason sporulation on asymptomatic plants and planting a resistant cultivar the following season should help reduce the initial inoculum and, concomitantly, the stem canker.

Velocity of an epidemic is defined as the spatial advance of a pathogen generation within a crop cycle (14); therefore, the concept of velocity can only be used for polycyclic diseases. Secondary inoculum cycles resulting in successful stem canker infections were detected in our study, which in effect identify the disease as polycyclic. Hence, it is reasonable to use velocity to characterize stem canker epidemics.

The Weibull model differentiates between monocyclic and polycyclic diseases on the basis of the estimated shape parameter (4,7,16). Damicone et al (7) used stem canker incidence to characterize resistance and the Weibull model to understand the nature of stem canker epidemics. Damicone et al (7) did not detect secondary inoculum during the season and characterized the disease as monocyclic. The shape parameters estimated from the Weibull model fitted to both incidence and severity data in this study were also indicative of the compound interest nature of stem canker epidemics (K. V. Subbarao, *unpublished data*). The infection rates on the three cultivars are nearly identical between the two studies. It is unclear whether the differences in the con-

TABLE 4. Total number of rainy days, total rainfall (cm), and number of irrigations in the irrigated plots at each experimental site from inoculation to final disease assessment

Location	Season	Number of rainy days	Total rainfall	Number of irrigations
Baton Rouge	1988	61	51.03	12
	1989	61	93.23	14
	1990	43	39.30	11
Port Sulphur	1989	44	55.40	14

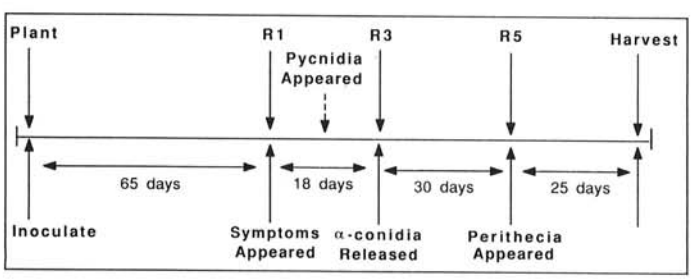

Fig. 2. Schematic representation of the events in a typical soybean stem canker disease cycle in Louisiana.

TABLE 5. Coefficients of determination for the monomolecular and logistic models fitted to detect secondary cycles in stem canker progress by omitting the final disease severity in 1988 and 1989 and the last two disease severities in 1990

			R^2 (adjusted)	
Location and year	Cultivar	Status	Monomolecular	Logistic
Baton Rouge, 1988	Bay	Irrigated	0.90	0.76
		Nonirrigated	0.91	0.73
	Wilstar 550	Irrigated	0.86	0.70
		Nonirrigated	0.88	0.64
	Bedford	Irrigated	0.80	0.70
		Nonirrigated	0.75	0.62
Baton Rouge, 1989	Bay	Irrigated	0.99	0.91
		Nonirrigated	0.99	0.96
	Wilstar 550	Irrigated	0.99	0.96
		Nonirrigated	0.94	0.92
	Bedford	Irrigated	0.98	0.98
		Nonirrigated	0.96	0.90
Port Sulphur, 1989	Bay	Irrigated	0.95	0.91
		Nonirrigated	0.91	0.88
	Wilstar 550	Irrigated	0.93	0.86
		Nonirrigated	0.95	0.85
	Bedford	Irrigated	0.97	0.94
		Nonirrigated	0.96	0.91
Baton Rouge, 1990	Bay	Irrigated	0.72	0.63
		Nonirrigated	0.47	0.74
	Wilstar 550	Irrigated	0.56	0.91
		Nonirrigated	0.50	0.92
	Bedford	Irrigated	0.59	0.92
		Nonirrigated	0.67	0.94

clusions between our study and those of Damicone et al (7) are the result of the choice of disease quantification variable. In this study, the choice of disease quantification variable did not affect the conclusions. Damicone et al (7) attributed the differences in the directional spread of the disease to water movement patterns in the plots. Canker severities in the eight directions of disease assessment in our study were also significantly different and followed the water movement in the plots (K. V. Subbarao, *unpublished data*). The significantly higher disease in the irrigated plots, steeper gradients, and higher velocity of spread observed in our plots also support this conclusion. Vanderplank (28) classified plant diseases into primary inoculum-dependent and infection rate-dependent categories. A monocyclic epidemic is defined as that which occurs when the inoculum comes from a reservoir and increases without multiplication during the vegetation season (28). A polycyclic epidemic is defined as that which occurs when the inoculum increases through multiplication during the vegetation season (28). Although soybean stem canker fits the classical definition of infection rate-dependent diseases, it should be classified as both primary inoculum-dependent and infection rate-dependent because of the irregular symptom expression from secondary inoculum and the confounding effect of symptomless, secondary infections.

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