Effect of Plant Age, Maturity Group, and the Environment on Disease Progress of Sudden Death Syndrome of Soybean

J. C. RUPE, Associate Professor, Department of Plant Pathology, and E. E. GBUR, JR., Associate Professor, Agricultural Statistics Laboratory, University of Arkansas, Fayetteville 72701

ABSTRACT

Rupe, J. C., and Gbur, E. E., Jr. 1995. Effect of plant age, maturity group, and the environment on disease progress of sudden death syndrome of soybean. Plant Dis. 79:139-143.

Disease progress of sudden death syndrome (SDS) of soybean, caused by *Fusarium solani,* was determined in a standard susceptible cultivar, Lee 74, planted uniformly throughout the test site and in 42 soybean cultivars representing maturity groups (MG) IV–VIII, 1986–1989. Disease severity was assessed weekly from the middle of July through the first of October using a visual rating scale of percent leaf area exhibiting foliar symptoms of SDS. The disease ratings were used to calculate the absolute infection rate (dy/dt) for each cultivar at each rating time and the area under the disease progress curve (AUDPC). In Lee 74, SDS progressed in two phases: an initial rapid increase in disease to about 35% leaf area affected and a slower increase after that point. This two-phase disease progress curve was fit by a segmented model using two exponential equations. The fits of these equations were highly significant ($P < 0.0001$) in all 3 yr. The equations were joined on 30 August 1986, 13 August 1987, and 17 August 1988, which corresponded to 74, 69, and 83 days after planting (DAP) in 1986, 1987, and 1988, respectively. These join points occurred at accumulated temperatures from planting of 925, 852, and 963 degree-days in 1986, 1987, and 1988, respectively, using 15 C as the base temperature. Disease progress in the 42 test cultivars was compared by determining the DAP of the first large increase in disease (dy/dt > 1%/day or, if lower than 1%/day, the highest dy/dt attained). DAP and dy/dt were significantly ($P < 0.0001$) related to AUDPC ($R = -0.58$ and $R = 0.38$, respectively). Cultivars susceptible to race 6 of the soybean cyst nematode (SCN), *Heterodera glycines,* the predominate race in the field, developed SDS 10 days earlier and had significantly higher levels of disease than cultivars resistant to race 6 of SCN. The earliest increases in SDS occurred between 71 and 77 DAP in 1986, 69 and 74 DAP in 1987, and 75 and 83 DAP in 1988 for most MGs. Accumulated heat for these periods was 910–942, 852–916, and 857–963 degree-days in 1986, 1987, and 1988, respectively. In 1987, three cultivars in MG VII and VIII had large increases 52 DAP after 608 degree-days. In general, however, disease increased at the same time across MGs, but at no specific reproductive stage.

Sudden death syndrome (SDS) is a mid-season disease of soybean (*Glycine max* (L.) Merr.) favored by high-yield environments (14). Aboveground symptoms of SDS usually begin at or after flowering as interveinal chlorotic spots. These spots develop into interveinal necrotic streaks, finally resulting in defoliation of the leaflets with the petioles remaining attached to the stem. While most SDS symptoms are aboveground, the pathogen *Fusarium solani* (Mart.) Sacc. (10,11), is confined to the roots. In addition, SDS is often associated with the soybean cyst nematode (SCN) *Heterodera glycines* Ichinohe, although the nematode is not necessary for disease development (4,6,8,10,14,17). SDS appears to be strongly influenced by the environment, growth stage of the plant, and cultivar susceptibility. Cooler than normal temperatures during reproductive development, abundant rainfall or irrigation (14), and increased soil fertility (15) have been associated with SDS development. SDS usually occurs during reproductive development, but early symptoms have been observed several weeks before flowering (14). Cultivar susceptibility has a strong effect on disease development and is currently the only reliable control measure available to growers (13). Susceptibility is usually measured by the severity of foliar symptoms at full seed (R6) (6,15,17); however, there have been no reports on how disease progresses to this point or how cultivar susceptibility affects disease progress.

Published with the approval of the Director, Arkansas Experiment Station, manuscript #94086.

Accepted for publication 14 October 1994.

The objectives of this paper were to determine the importance of certain environmental factors and plant development stage to disease development and describe disease progress in several soybean cultivars. An initial report has been published (13).

MATERIALS AND METHODS

The experiments were conducted at the Pine Tree Experiment Station, Colt, AR, in a field with a history of SDS and SCN during 1986–1988. The soil in the field was a Crowley silt loam and was infested with race 6 of SCN (13). Overhead irrigation was provided by fixed sprinklers placed in lines separated by 18 rows of soybeans. Management of the field employed standard farming practices for optimum soybean growth.

The tests were planted on 17 June 1986, 5 June 1987, and 26 May 1988. A previous report (13) incorrectly stated that the 1986 planting date was 3 June. Plots were planted at the rate of 10 seeds per 30 cm of row and consisted of one row, 6 m long with 0.9 m between rows and 1.5 m alleys separating each tier of plots. The experimental design was a randomized complete block with four replications. Each replication consisted of six rows and 15 tiers. To compensate for within-field variability of SDS, a standard susceptible cultivar, Lee 74, was planted in nine continuous rows so that each test cultivar was adjacent to a row of Lee 74. This resulted in 135 plots of Lee 74. Forty-two test cultivars from maturity groups (MG) IV-VIII were selected from cultivars grown in Arkansas. Within each MG, cultivars ranged from susceptible to resistant based on results of earlier cultivar tests and ob-

Table 1. Dates of planting and the number of days after planting (DAP) of the disease ratings for sudden death syndrome of soybean

		Sample										
Year	P[y]	1	2	3	4	5	6	7	8	9	10	11	12
1986													
Date	6/17	7/22	7/30	8/5	8/20	8/27	9/2	9/9	9/17	9/24	9/30	10/8	...[z]
DAP	0	35	43	49	64	71	77	84	92	99	105	113	...
1987													
Date	6/5	7/1	7/17	7/27	8/13	8/18	8/26	9/3	9/10	9/16	9/24	10/1	10/7
DAP	0	26	42	52	69	74	82	90	97	103	111	118	124
1988													
Date	5/26	7/14	7/22	7/27	8/2	8/9	8/17	8/23	8/31	9/6	9/14	9/20	9/28
DAP	0	49	57	62	68	75	83	89	97	103	111	117	125

[y] Planting date.
[z] No rating taken.

servations in grower fields (13). Lee 74 was included as a test cultivar as well as serving as the standard susceptible cultivar.

Severity of SDS and growth stage of the soybean (3) were assessed each year at approximately weekly intervals from the middle of July until the first of October (Table 1) on a 0–5 scale with 0 = no disease, 1 = 1–10%, 2 = 11–30%, 3 = 31–70%, 4 = 71–90%, and 5 > 90% of the leaf area affected by SDS. Percent leaf area affected was determined by converting each rating to the midpoint of its range, i.e., 0 = 0%, 1 = 5%, 2 = 20%, 3 = 50%, 4 = 80%, and 5 = 95%. These converted ratings were used to determine the average disease severity and the absolute infection rate (dy/dt [2]) at each time and the area under the disease progress curve (AUDPC [18]). Growth stages were those of Fher et al (3) and represented full bloom (R2), beginning pod (R3), full pod (R4), beginning seed (R5), and full seed (R6).

Temperature data were collected at two weather stations, one at Wynne, AR, 17 km northeast from the test site and the other at Brinkley, AR, 36 km southwest from the test site. Daily high and low temperatures were averaged between the stations and used as an estimate of the temperature at the test site. Daily rainfall data were collected at the Pine Tree Experiment Station approximately 800 m east of the test site. Irrigation was by overhead sprinklers to maintain optimum soil moisture for soybean growth.

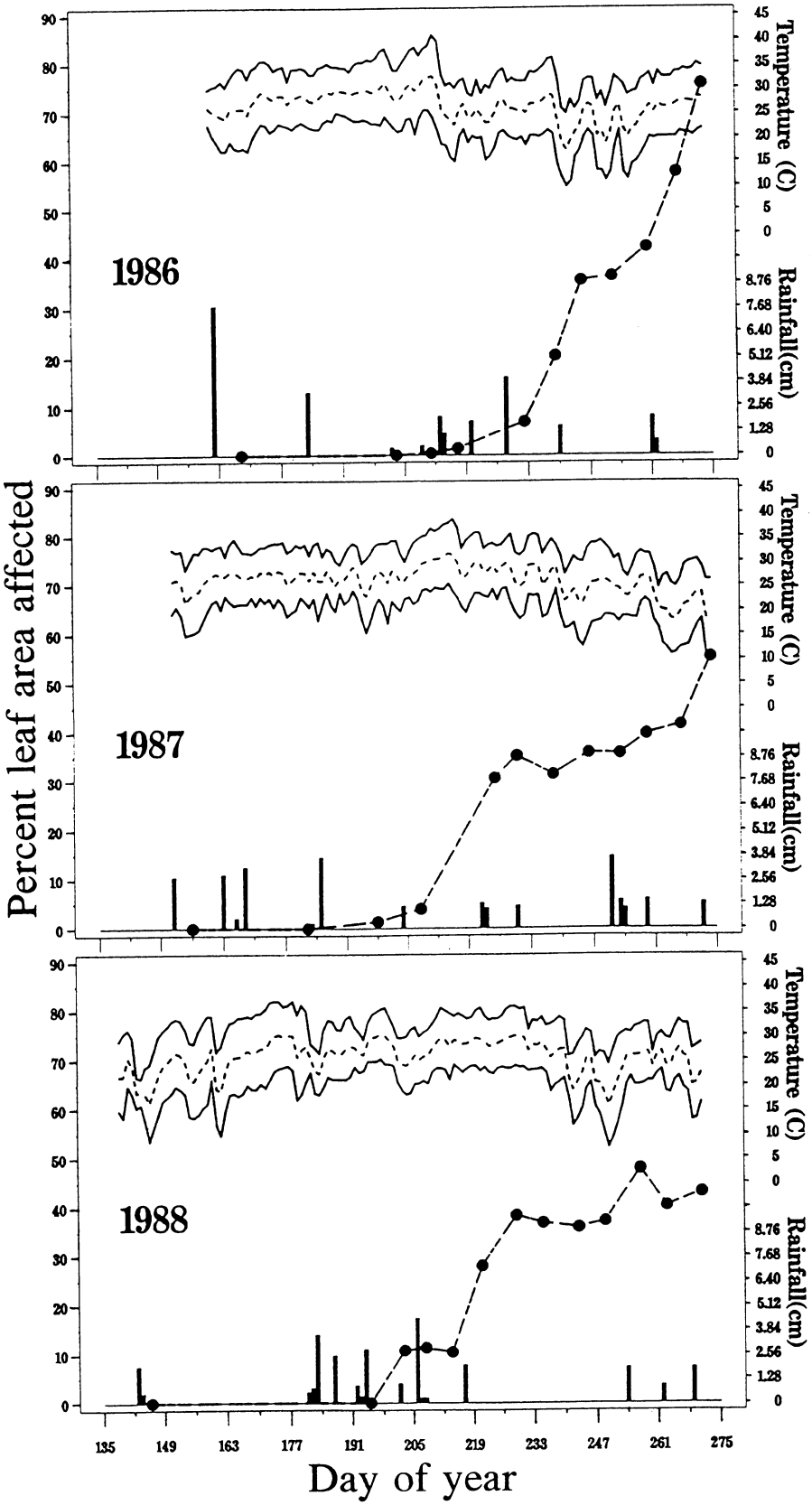

Fig. 1. Disease progress of sudden death syndrome in the soybean cultivar Lee 74 (•-----•), daily high (upper solid line), low (lower solid line), and average (middle dashed line) temperatures, and daily rainfall (vertical bars) in 1986, 1987, and 1988 at the Pine Tree Station, Colt, AR.

RESULTS

Disease progress, Lee 74. Disease progress of SDS consisted of two phases in the standard susceptible cultivar Lee 74: a rapid increase (13–15 days) to about 35% leaf area affected followed by a phase with a slower rate of disease increase (27–44 days) (Fig. 1). Each phase of the epidemic was best fit by an exponential model. For each year, the natural logarithm of percent disease was modeled as a function of day of the year. The day of the year associated with the point of intersection (X_j) was assumed to be unknown. Least squares estimates of the slopes and intercepts of the lines and the coordinates of the point of intersection were obtained using the algorithm described in Hudson (7) and Lerman (9). Slope estimates were compared across years using the two-sample Student's t test allowing for unequal variances. Calculations were carried out using SAS. The two phase models fit the data well (F test significant at $P < 0.0001$). The resulting equations had points of intersection on 30 August 1986, 13 August 1987, and 17 August 1988 (day of the year 242, 225, and 229, respectively), which occurred at 74, 69, and 83 DAP, respectively. Within each year the rate of disease increase was significantly slower in the second phase compared with the first phase of the epidemic in all 3 yr. Between years, the rate of disease increase in the first phase of the epidemic was significantly different among all years with the fastest rate occurring in 1986, followed by 1987 and 1988 (Table 2). In the second phase, 1987 and 1988 were significantly different from 1986, but not from each other. The rate in the

second phase of the epidemic in 1988 was not significantly different from 0.

The first phase of the epidemic was not associated with any obvious changes in temperature or rainfall (Fig. 1), but was associated with accumulated degree-days. Using a base temperature of 15 C selected from growth chamber studies because no disease developed at 15 C, but did at 20 C (J. C. Rupe, *unpublished data*), the accumulated degree-days (2) from planting to X_j were calculated, resulting in 925, 852, and 963 degree-days for 1986, 1987, and 1988, respectively. In the second phase of the epidemic, temperatures were initially cooler in 1986 and then increased compared with the other years when the temperatures were warmer and then decreased (Fig. 1).

Rainfall was associated with the initiation of the first phase of the epidemic in 1986, but not in 1987 or 1988 (Fig. 1). However, heavy rainfall in early July 1988 preceded an initial increase in SDS to the 10% level 3 wk before the rapid rise in disease to 35% leaf area affected.

While early symptoms of SDS occurred on Lee 74 during vegetative growth stages in all years, rapid increases in disease did not occur until reproductive development (Fig. 1). The reproductive stages R2, R3, R4, R5, and R6 were reached approximately on 5, 20, 27 August, 2 and 17 September 1986, 13, 18, 27 August, 3 and 16 September 1987, and 2, 9, 17, 23 August, and 14 September 1988, respectively, in each year.

Disease progress, cultivars. A single model did not adequately describe disease progress of each cultivar due to variability associated with differences in cultivar susceptibilities and in the number of replications. To compare all cultivars in the test, the absolute infection rates (dy/dt) were calculated for each cultivar in each year at each rating time. A dy/dt > 1%/day or the highest dy/dt attained was used to indicate the first rapid increase in disease. Absolute infection rates ranged from 0.2 to 4.8% per day and occurred 52–125 DAP. SDS did not develop in a few cultivars. There was a high correlation between dy/dt and AUDPC ($R = 0.38$, $P < 0.0001$) and disease at growth stage R6 ($R = 0.45$, $P < 0.0001$), but not DAP. DAP was highly correlated to AUDPC ($R = -0.58$, $P < 0.0001$) and disease at R6 ($R = -0.60$, $P < 0.0001$).

The relationship of cultivar susceptibility to SCN to development of SDS was compared in 33 cultivars with known reactions to race 6 of SCN (22 susceptible and 11 resistant) using the two-sample Student's *t* test allowing for unequal variances. Susceptibility to race 6 of SCN was associated with earlier appearance of SDS (10 days) and with more severe SDS (AUDPC and R6) than cultivars resistant to race 6 (Table 3).

In each year, the earliest increases occurred at about the same number of DAP for all MGs (Table 4). In general, the earliest increase in disease occurred 71–77 DAP in 1986, 69–74 DAP in 1987, and 75–83 DAP in 1988. These DAPs corresponded to accumulated degree-days of 910–942 in 1986, 852–916 in 1987,

Table 2. Estimated intercepts, slopes, and join points (X_j) of segmented exponential model[v] of development of sudden death syndrome of soybean in cv. Lee 74 at the Pine Tree Station, Colt, AR, in 1986, 1987, and 1988

Segment	Year	Intercept (%)	Slope (%/day)	Estimated standard error of slope	X_j[w]
1	1986	−29.047	0.134 a[x]	0.006	242
1	1987	−22.698	0.116 b	0.004	225
1	1988	−8.991	0.055 c	0.008	229
2	1986	−3.395	0.028 a	0.007	...[y]
2	1987	1.377	0.009 b	0.002	...
2	1988	3.146	0.002[z] b	0.005	...

[v] Model consists of two exponential equations; the first segment includes all dates up to and including X_j, the second segment includes all dates at or above X_j.
[w] Join point dates were 30 August 1986, 13 August 1987, and 13 August 1988.
[x] Estimated slopes within a segment followed by the same letter are not significantly different at $P = 0.05$ using a two-sample Student's *t* test with unequal variances.
[y] Not applicable.
[z] Not significantly different from 0 at $P = 0.05$.

Table 3. Relationship of susceptibility to race 6 of the soybean cyst nematode (SCN), *Heterodera glycines*, and various parameters of disease progress of sudden death syndrome (SDS) among 33 soybean cultivars at the Pine Tree Station, Colt, AR, from 1986 to 1989

SCN Race 6	SDS			
	dy/dt[v]	DAP[w]	AUDPC[x]	R6[y]
Susceptible	1.42	86.27	766.03	19.27
Resistant	1.80	96.87	388.97	8.18
P[z] values	0.125	0.025	0.013	0.007

[v] Average size of the first absolute infection rate (dy/dt) of at least 1%/day or, if less, the highest dy/dt attained.
[w] Number of days after planting of the first rapid increase in dy/dt.
[x] Area under the disease progress curve (18).
[y] Severity of SDS (percent leaf area affected) at the R6 growth stage (3).
[z] Indicate at what level of significance the values within a column differ using a two-sample Student's *t* test allowing for unequal variances.

Table 4. Number of cultivars that had their first rapid increase in sudden death syndrome (SDS)[w] in each rating period

Year	Maturity group	Days after planting[x] (number of cultivars[y])										
		49–52	57–64	68–71	74–77	82–84	89–92	97–99	103–105	111–113	117–118	124–125	No SDS
1986	IV	0	0	2	1	0	0	0	0	...[z]	...	...	1
	V	0	0	2	4	0	3	3	0	...	...	...	2
	VI	0	0	2	3	2	2	5	0	...	...	...	0
	VII-VIII	0	0	0	4	0	1	3	2	...	...	...	0
1987	IV	0	...	2	0	2	0	0	0	0	0	0	0
	V	0	...	3	1	4	0	0	1	0	3	0	2
	VI	0	...	2	1	4	1	1	2	1	2	0	0
	VII-VIII	3	...	1	0	2	0	0	0	2	2	0	0
1988	IV	0	0	0	0	2	1	0	1	0	0	0	0
	V	0	0	0	3	2	2	2	3	0	1	0	1
	VI	0	0	0	5	2	0	0	1	0	2	4	0
	VII-VIII	0	0	0	4	4	0	0	1	1	0	0	0

[w] First rapid increase in SDS was defined as the first absolute infection rate (dy/dt) of at least 1%/day or, if less, the highest dy/dt attained.
[x] The number of days after planting (DAP) of the first rapid increase in SDS. Intervals reflect rating dates with similar DAPs (Table 1). Planting dates were 17 June 1986, 5 June 1987, and 26 May 1988.
[y] The same 4 maturity group (MG) IV, 14 MG V, 14 MG VI, and 10 MG VII-VIII cultivars were tested in each year.
[z] Data not taken.

and 857–963 in 1988. The exceptions were three MG VII-VIII cultivars in 1987 that had increases 52 DAP after 608 degree-days.

The growth stage associated with the dy/dt differed with year and MG (Table 5). In 1986, the earliest increases occurred during R5 for MG IV and V, R3 and R4 for MG VI and R3 for MG VII-VIII. In 1987, the earliest increases occurred during R5 for MG IV, R3 for MG V, R2 for MG VI, and during late vegetative growth for MG VII-VIII. In 1988, the earliest increases occurred during R5 for MG IV, R3 and R4 for MG V and VI, and R2 for MG VII-VIII.

DISCUSSION

SDS disease progress in the standard susceptible cultivar, Lee 74, was best described by a model using two exponential equations that described a two-phase epidemic (Fig. 2). In each year, there was a rapid increase in disease to about 35% leaf area affected followed by a slower increase in disease thereafter. Comparing among years of slopes in the first phase is probably not as useful (due to a missed observation in 1987 and the early increase in disease due to heavy rain in 1988 [Fig. 1]) as comparing when the two equations were joined in each year, X_j. These X_js were similar (74, 69, and 83 DAP in 1986, 1987, and 1988, respectively) although planting dates were as much as 22 days apart (1986 versus 1988). The first phase of the epidemic was not clearly associated with a specific rain event or a rapid change in temperature (Fig. 1) but did appear to be associated with accumulated heat units. The largest difference in accumulated heat units (1987 versus 1988) was only 11.5%. This effect of accumulated heat units was also observed with the test cultivars. There was overlap among all 3 yr in accumulated heat units for the first cultivars in each MG to exhibit the initial rapid increase in disease (910–942, 852–916, and 857–963 degree-days for 1986, 1987, and 1988, respectively).

Changes in plant physiology associated with reproductive development may be another factor influencing SDS development. Typically, SDS develops during soybean reproductive growth (14). In the standard susceptible and the test cultivars, rapid increases in SDS occurred during reproductive development but were not tied to any particular stage, ranging from R2 to R5 (Table 5). The stage during which rapid disease development occurred depended on the MG and the planting date. However, the change from vegetative growth to reproductive growth may also have some effect on SDS, because this change is associated with a change in host physiology. As the plant enters reproductive development, net root growth is dramatically reduced (1; C. A. Berouty, *personal communication*), and photosynthates are transported to the developing pods and seeds instead of to the vegetative parts of the plants, which may reduce the plant defenses. However, severe SDS can be induced in greenhouse inoculations on 2- to 3-wk-old plants that are not in reproductive growth (10,11). Furthermore, in 1987 three MG VII-VIII cultivars (Coker 237, Braxton, and FFR 771) had large increases in SDS during vegetative growth (Table 4).

SCN appeared to affect SDS development. On average, cultivars susceptible to race 6 of SCN (the predominate race in the field [13]) developed SDS 10 days sooner and had higher levels of disease than SCN resistant cultivars (Table 3). This agrees with greenhouse studies reporting that SDS appeared sooner and was more severe in the presence of SCN than in treatments without the nematode (8,10). Field studies also report that SCN resistance was associated with lower levels of SDS in some cultivars (4,13). In the southern U.S., SCN populations usually have gone through two generations by the time of the first rapid increase in SDS (5,16). The stress caused by the increased populations may contribute to the earlier appearance and greater severity of SDS in the SCN-susceptible cultivars than in SCN-resistant cultivars.

Another important factor associated with SDS is optimum soil moisture from either rainfall or irrigation (14). Although soil moisture was not measured in this test, the plots were irrigated as needed for optimum growth. Later studies have revealed that the irrigation system used in this test was able to maintain optimum soil moisture from planting until flowering, a period during which stored soil moisture was higher and plant demands for water were lower than after flowering (J. C. Rupe, *unpublished data*). From these later studies, it seems likely that soil moisture was not a limiting factor during the first phase of the epidemic but was probably the limiting factor in the second phase of the epidemic. This is indicated by increases in SDS during the second phase of the epidemic associated with rainfall in the middle of September in 1986 and 1987. In 1988, SDS did not increase in the second phase of the epidemic, and there was also very little rainfall. Rapid development of SDS in grower fields has been observed within 1 wk of flood or furrow irrigation (J. C. Rupe, *personal observation*).

Too much rainfall during vegetative growth may reduce or delay SDS. In 1988, heavy rainfall during the first 2 wk of July resulted in standing water in low parts of the field. This heavy rainfall was followed by an early increase in SDS to the 10% leaf area affected level; however, changes in disease distribution were noted (12). Specifically, little SDS developed in the low areas of the field until 4 or 5 wk after the rest of the field (J. C. Rupe, *unpublished data*). Disease development in plots in these low areas in 1986 and 1987 (years without flooding) was identical to that in the other plots in the test suggesting that stress during vegetative growth from flooding could delay or reduce SDS development. It is not clear if this early flooding stress affected root colonization by *F. solani* or affected plant susceptibility.

Cultivar susceptibility also affected disease development. As indicated by the high correlations between dy/dt and DAP with AUDPC and disease levels at R6, resistance was expressed in most cultivars as a delay in the onset of disease (increased DAP) and a reduction in the speed of the increase (lower dy/dt). Although highly significant, these correlations accounted for only 38–60% of the variability in the data, indicating that SDS may progress in a number of different ways. These differences in disease

Table 5. Plant growth stage associated with the first rapid increase in sudden death syndrome (SDS)[x] at Colt, AR

Year	Maturity group	Growth stage[y] (number of cultivars)[z]						
		Veg.	R2	R3	R4	R5	R6	No SDS
1986	IV	0	0	0	0	2	1	1
	V	0	0	0	0	6	6	2
	VI	0	0	1	3	3	7	0
	VII-VIII	0	0	3	1	0	6	0
1987	IV	0	0	1	0	3	0	0
	V	0	0	3	1	4	4	2
	VI	0	3	4	0	3	4	0
	VII-VIII	3	2	1	0	0	4	0
1988	IV	0	0	0	0	3	1	0
	V	0	0	1	3	5	4	1
	VI	0	0	6	1	1	6	0
	VII-VIII	0	4	4	0	1	1	0

[x] First rapid increase in SDS was defined as the first absolute infection rate (dy/dt) of at least 1%/day or, if less, the highest dy/dt attained.

[y] Growth stages are those of Fehr et al (3); R2 = full bloom; R3 = beginning pod; R4 = full pod; R5 = beginning seed; R6 = full seed.

[z] The same 4 maturity group (MG) IV, 14 MG V, 14 MG VI, and 10 MG VII-VIII cultivars were tested in each year.

development may be due to the environment, uneven disease distribution, resistance to SCN, or different types of plant resistance to SDS.

These results suggest that yield loss to SDS may be controlled by delaying planting (4) or by selecting early-maturing cultivars. Both of these strategies would result in SDS developing at a later reproductive stage, which may result in lower yield losses.

ACKNOWLEDGMENTS

This research was funded in part by a grant from the Arkansas Soybean Promotion Board. We would like to thank Robert Turner, Roger Eason, and William Johnson for management of the plots and Jeffery Velie for computer assistance.

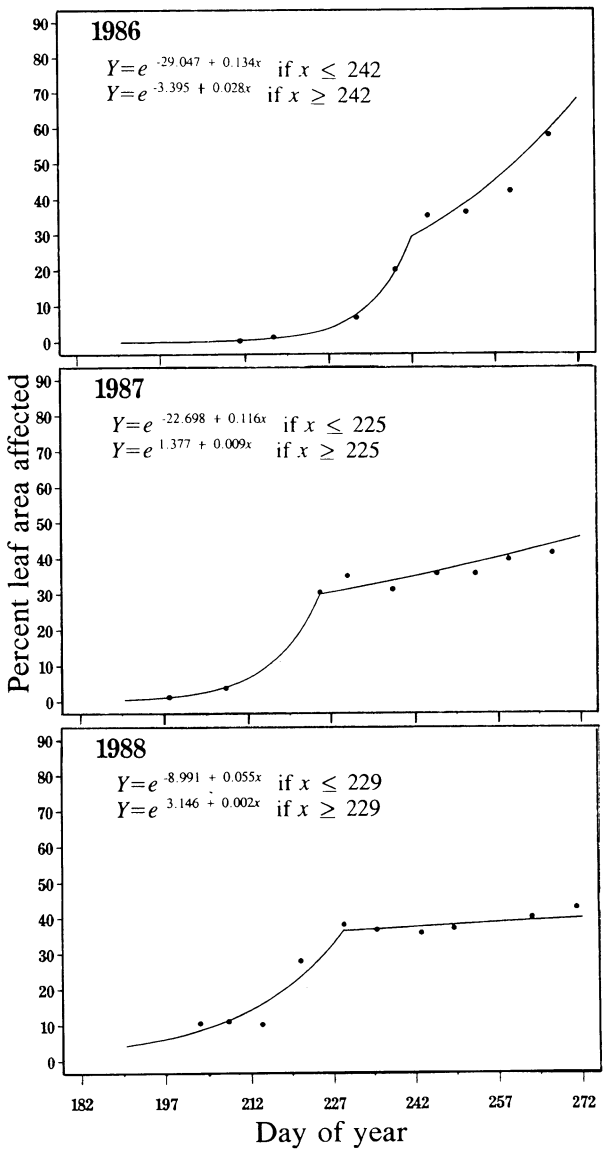

Fig. 2. Observed (•) and predicted disease progress values for sudden death syndrome of soybean using segmented exponential equations for epidemics occurring at the Pine Tree Station, Colt, AR, in 1986, 1987, and 1988. Fitted equations for each year appear in the upper left-hand corner of each figure with Y = percent leaf area affected and x =day of the year.

LITERATURE CITED

1. Brown, D. A. 1984. Characterizing soybean root growth and distribution. Arkansas Farm Res. 33:3.
2. Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, Inc. New York.
3. Fehr, W. R., Caviness, C. E., Burmood, D. T., and Pennington, J. S. 1971. Stage development descriptions for soybean, *Glycine max* (L.) Merrill. Crop Sci. 11:929-931.
4. Hershman, D. E., Hendrix, J. W., Stuckey, R. E., Bachi, P. R., and Henson, G. 1990. Influence of planting date and cultivar on sudden death syndrome in Kentucky. Plant Dis. 74:761-766.
5. Hill, N. S., and Schmitt, D. P. 1989. Influence of temperature and soybean phenology on dormancy induction of *Heterodera glycines*. J. of Nematol. 21:361-369.
6. Hirrel, M. C. 1986. Disease severity and yield loss comparisons of soybean maturity groups affected in sudden death syndrome. Page 61 in: Proc. Annu. Meet. South. Soybean Dis. Workers, 13th.
7. Hudson, D. J. 1966. Fitting segmented curves whose join points have to be estimated. J. of Amer. Stat. Assoc. 61:1097-1129.
8. Lawrence, G. W., Roy, K. W., and McLean, K. S. 1988. Soybean cyst nematode associations with sudden death syndrome of soybeans. (Abstr.) Phytopathology 78:1514.
9. Lerman, P. M. 1980. Fitting segmented regression models by grid search. Appl. Statist. 29:77-94.
10. Roy, K. W., Lawrence, G. W., Hodges, H. H., McLean, K. S., and Killebrew, J. F. 1989. Sudden death syndrome of soybean: *Fusarium solani* as incitant and relation of *Heterodera glycines* to disease severity. Phytopathology 79:191-197.
11. Rupe, J. C. 1989. Frequency and pathogenicity of *Fusarium solani* recovered from soybeans with sudden death syndrome. Plant Dis. 73:581-584.
12. Rupe, J. C., Gbur, E. E., and Marx, D. B. 1989. Spatial and temporal distribution of sudden death syndrome of soybeans. (Abstr.) Phytopathology 79:1219.
13. Rupe, J. C., Gbur, E. E., and Marx, D. M. 1991. Cultivar responses to sudden death syndrome of soybean. Plant Dis. 75:47-50.
14. Rupe, J. C., Hirrel, M. C., and Hershman, D. E. 1989. Sudden death syndrome. Pages 84-85 in: Compendium of Soybean Diseases. J. B. Sinclair and P. A. Backman, eds. American Phytopathological Society, St. Paul, MN.
15. Rupe, J. C., Sabbe, W. E., Robbins, R. T., and Gbur, Jr., E. E. 1993. Soil and plant factors associated with sudden death syndrome of soybean. J. Prod. Agric. 6:218-221.
16. Shilling, K. 1984. Investigations into the spatial distribution and the population dynamics of *Heterodera glycines* Ichinohe. Ph.D. thesis, University of Arkansas, Fayetteville.
17. Sciumbato, G. L., and Keeling, B. L. 1985. Sudden death syndrome (SDS) of soybeans in Mississippi in 1984. Page 64 in: Proc. Annu. Meet. South. Soybean Dis. Workers, 12th.
18. Shaner, G., and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing in Knox wheat. Phytopathology 67:1051-1056.