Cultivar Responses to Sudden Death Syndrome of Soybean

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ABSTRACT

Rupe, J. C., Gbur, E. E., and Marx, D. M. 1991. Cultivar responses to sudden death syndrome of soybean. Plant Dis. 75:47-50.

Forty-two soybean (*Glycine max*) cultivars representing maturity groups IV–VIII were evaluated for their response to sudden death syndrome (SDS), caused by *Fusarium solani*, over 3 yr in a field with a history of the disease. Disease severity was assessed weekly from the middle of July through September using a rating scale of 0–5 where 0 = 0%, 1 = 1–10%, 2 = 11–30%, 3 = 31–70%, 4 = 71–90%, and 5 = >90% of the leaf area exhibiting foliar symptoms of SDS. Area under the disease progress curve was calculated for each cultivar. The susceptible cultivar Lee 74 was planted adjacent to each cultivar tested and served as a standard for comparison of disease levels. Disease progress on plants of Lee 74 was used to estimate disease for each of the plots with plants of the test cultivars using the geostatistical technique, "kriging." Estimates of relative amounts of disease were used as covariates in the cultivar analysis. Foliar symptoms of SDS were noted at or shortly after flowering with cultivars of maturity group IV but before flowering with many of the cultivars in maturity groups V–VIII, depending on the year. Significant differences in disease development between cultivars occurred at the R_3 growth stage and were more distinct at the R_6 growth stage. Cultivar rankings at R_3 were generally highly correlated to their rankings at R_6. Most cultivars had similar levels of disease between years and with previous observations. However, some cultivars had significant differences in disease development between years. Disease development in a few cultivars differed markedly with previous observations. These differences appeared to be associated with the response of the cultivar to race 6 of the soybean cyst nematode (SCN), *Heterodera glycines*, the predominate race in the test field. Cultivars susceptible to race 6 were susceptible to SDS and those that were moderately resistant or resistant to race 6 had low levels of SDS. With other cultivars, susceptibility to SCN was not related to susceptibility to SDS.

Sudden death syndrome of soybean (SDS) is a recently described root disease of high-yield-potential soybeans (*Glycine max* (L.) Merrill [5,14]). First observed in 1971, the disease was confined to Arkansas until 1984 and 1985 when it was reported in Illinois, Indiana, Kentucky, Mississippi, Missouri, and Tennessee (14). Foliar symptoms of SDS begin before flowering for soybeans in late maturity groups or after flowering for cultivars in early maturity groups. These symptoms first appear as chlorotic interveinal spots that enlarge and become necrotic, eventually leaving only the veins green. Such severely affected leaflets may dehisce and leave the petioles attached. Yield reductions when disease is severe are caused by seed and pod abortion and can lead to almost total crop loss (14). The causal agent of SDS is *Fusarium solani* (Mart.) Sacc. (9,13). The soybean cyst nematode (SCN), *Heterodera glycines* Ichinohe, is often but not always associated with SDS (5–7,9,11,14,16).

One possible control measure for SDS is the use of cultivars with low susceptibility to the disease. Marked differences in cultivar responses to SDS have been reported (3,4,6,10,11,16). Sciumbato and Keeling (16) reported that SDS was more severe in cultivars in maturity groups VI and VII than in maturity group V. However, Hershman et al (4) found no correlation between maturity group and response to SDS but did find a weak positive correlation between severity to SDS and populations of SCN with SCN-susceptible cultivars.

Development of symptoms of SDS by a cultivar may not be related to the level of infection of the roots by *F. solani*. The percentage of root segments infected with *F. solani* was roughly the same in Lee 74, a susceptible cultivar, as in the asymptomatic cv., Hartz 6130 (11). At least with cv. Hartz 6130, this apparent resistance may actually be tolerance.

The objectives of this study were to confirm cultivar differences in response to SDS, determine if cultivar responses are consistent between years, and compare disease progress among cultivars from different maturity groups. Preliminary reports from this study have been published (10,12).

MATERIALS AND METHODS

The experiment was conducted during 1986–1988 in a field with a history of both SDS and SCN at the Pine Tree Experiment Station at Colt, Arkansas. Overhead irrigation was provided from fixed sprinklers placed in lines separated by 18 rows of soybeans. The soil in the field was a Crowley silt loam and was infested with race 6 of SCN (R. T. Robbins, *unpublished data*). Standard farming practices for optimum soybean growth were used.

Test cultivars were planted on 3 June 1986, 5 June 1987, and 26 May 1988 at

Published with approval of the director of the Arkansas Agricultural Experiment Station, Fayetteville. Supported in part by the Arkansas Soybean Promotion Board.

Accepted for publication 24 June 1990 (submitted for electronic processing).

10 seeds per 30 cm of row in single, 6-m rows with 0.9 m between rows in a randomized complete block design with four replications. Each block was six rows wide. To compensate for possible within-field variability of SDS, a standard susceptible cultivar, Lee 74, was planted in rows three and six of blocks one and four; rows one, three, and six of block two; and rows one and four of block three. Row one in blocks one and four and row six in block three were used as border rows and not assessed in the experiment. With this arrangement, each test cultivar was next to a standard susceptible cultivar. Forty-two test cultivars were selected from maturity groups grown in Arkansas (groups IV–VIII) that had either high, moderate, or low reactions to SDS in previous tests or from observations in commercial fields (J. C. Rupe and M. C. Hirrel, *unpublished data*).

Severity of SDS was assessed each year at approximately weekly intervals from the middle of July until the first of October on a scale of 0–5 where 0 = no disease, 1 = 1–10%, 2 = 11–30%, 3 = 31–70%, 4 = 71–90%, and 5 = >90% of the leaf area affected by chlorotic or necrotic symptoms of SDS. Disease scores were used to determine area under the disease progress curve (AUDPC [17]) up to soybean growth stages R_3 (pod development) and R_6 (full pod [2]) for each cultivar and all plots of the standard susceptible cultivar. The AUDPCs for the standard susceptible cultivar were kriged and used to obtain estimates of the relative amounts of disease for each plot containing a test cultivar; that is an estimate of the amount of disease in terms of AUDPC that would have occurred in a test plot if Lee 74 had been planted (1). Kriging is a statistical technique for predicting a response, taking into account spatial correlation among the observations. The AUDPCs for the test cultivars were analyzed by maturity group and growth stage using analysis of covariance. The covariate was the estimated level of relative amount of disease (15). The whole plot factor in the analysis was year and the split plot factor was cultivar.

RESULTS

SDS was unevenly distributed within the field in all 3 yr of the study. AUDPCs for the standard susceptible cultivar (Lee 74) ranged from 8 to 180, 45.5 to 202.5, and 0 to 225 rating days on 24 September 1986, 24 September 1987, and 20 September 1988, respectively. R. T. Robbins used the Baermann funnel technique to determine the levels of J2 larvae of the SCN at planting and at flowering in 1988 for all plots of the standard susceptible cultivar. At planting, SCN ranged from 8 to 1,104 J2 larvae per 500 cc of soil and averaged 215.4 J2 per 500 cc of soil. At flowering, SCN ranged from 72 to 3,480 J2 per 500 cc of soil and averaged 755.2 J2 per 500 cc of soil. The onset of foliar symptoms of SDS occurred at flowering (R_2) or later for cultivars in maturity group IV in all 3 yr of the study (Table 1). However, some cultivars in each of the other maturity groups developed foliar symptoms before flowering in 1987 and 1988.

Analysis of covariance revealed that replication and year did not significantly affect disease development at R_3 or R_6 in any maturity group. However, the covariate (Lee 74 AUDPC) and cultivar both were highly significant ($P < 0.0001$) in all maturity groups at both R_3 and R_6. The cultivar × year interaction was also highly significant except in maturity group IV.

Significant differences in development of SDS occurred between cultivars in all maturity groups and in most years at growth stage R_3 and in all years at growth stage R_6 (Table 2). Some cultivars (such as Mack and Deltapine 506) had similar levels of disease development between years, while others had significant differences in disease development between years (such as Coker 425 and Asgrow 6520). Cultivar rankings were similar to previous observations with most cultivars. However, Essex, Wilstar 550, and Tracy M had high levels of disease development in this test but ranked low in previous observations. The cvs. Asgrow 5474 and Leflore had high levels of disease development at other locations but very little SDS in this test. Essex, Wilstar 550, and Tracy M were all susceptible to race 6 of SCN, and Asgrow 5474 and Leflore were moderately resistant and resistant, respectively, to this race (8). There was no consistent pattern between susceptibility to race 6 of SCN and SDS development with the other cultivars.

The disease rankings of the cultivars at R_6 were highly correlated by Spearman's rank correlation test (15) to the ranking at R_3, except for maturity group IV in 1987 ($P = 0.0513$) and 1988 ($P = 0.6$) and for maturity groups VII and VIII in 1986 ($P = 0.1173$ [Table 3]).

DISCUSSION

The onset of foliar symptoms of SDS appeared to be independent of the reproductive age of the plant and more dependent on the chronological age of the plant. Many cultivars developed foliar symptoms before reproductive development, especially in maturity groups VI–VIII. Even the cultivars in maturity group IV developed symptoms just as the plants were flowering, well before the pod-fill stage as reported in the Midwest (14). Differences in the onset of foliar symptoms of SDS between the South and Midwest may be attributable to the growth type of soybeans grown in these regions. In the South, soybeans with determinate growth (with little vegetative growth occurring once the plant flowers) are grown, while indeterminate soybeans (those with vegetative growth continuing after flowering) are grown in the Midwest. It is not clear, however, whether symptom expression of SDS is dependent on growth type or some other trait associated with cultivars of indeterminate growth type.

Once disease onset began, significant differences in disease development could be detected (Table 2). With most of the cultivars in maturity groups IV and VI–VIII, differences in disease development were probably caused by differences in the rate of disease development because symptom onset occurred at about the same time for most cultivars in most years (Table 1). However, disease development with the cultivars in maturity group V was also influenced by symptom onset, which occurred from vegetative growth through R_6.

Differences in disease development were consistent between years and with previous observations of most cultivars, but with others, significant differences occurred between years or with previous

Table 1. Number of soybean cultivars[a] that first developed foliar symptoms of sudden death syndrome (SDS) at each growth stage over 3 yr at the Pine Tree Experiment Station at Colt, Arkansas

Year	Maturity group	Growth stage[b]						No SDS
		Veg	R_2	R_3	R_4	R_5	R_6	
1986	IV	0	0	3	0	1	0	0
	V	0	5	5	1	1	1	1
	VI	1	3	4	4	2	0	0
	VII & VIII	0	7	2	1	0	0	0
1987	IV	0	3	0	0	1	0	0
	V	3	1	4	0	4	2	0
	VI	7	5	0	0	0	2	0
	VII & VIII	7	3	0	0	0	0	0
1988	IV	0	2	1	0	1	0	0
	V	5	3	2	3	0	0	1
	VI	11	1	1	0	0	1	0
	VII & VIII	10	0	0	0	0	0	0

[a]There were four, 14, 14, and 10 cultivars in maturity groups IV, V, VI, and VII and VIII, respectively.

[b]Growth stages were: Veg = vegetative, R_2 = full bloom, R_3 = beginning pod, R_4 = full pod, R_5 = beginning seed, and R_6 = full seed (2).

observations (Table 2). Differences from year to year generally involved changing rank between the top and middle (as with cv. Jeff) or between the middle and bottom (as with cv. Ringaround 606). These year-to-year differences may be attributable to differing responses of cultivars to predisposition factors. These factors have not yet been determined. A few cultivars had very different responses in this test compared to previous observations. These differences were associ-

Table 2. Response of soybean cultivars in maturity groups IV–VIII grown at the Pine Tree Experiment Station at Colt, Arkansas, in 3 yr to sudden death syndrome (SDS), their prior response to SDS[a], and their reaction to race 6 of the soybean cyst nematode *Heterodera glycines* Ichinohe

Cultivar	SCN[c] race 6 reaction	Growth stage (AUDPC)[b]						Prior SDS response
		R_3			R_6			
		1986	1987	1988	1986	1987	1988	
Maturity group IV								
Mitchell	···	12.7	18.6	4.7	69.8	74.3	49.6	H
Ringaround 480	S	11.4	12.2	6.4	66.1	69.6	43.1	H
Pershing	S	8.1	0.0	5.7	37.2	20.0	25.3	L
Nathan	S	2.5	0.0	2.6	8.2	0.0	10.9	M
LSD within year[d]				9.6				25.3
LSD across years[e]				13.1				32.5
Maturity group V								
Deltapine 105	S	6.6	28.2	11.0	73.0	106.2	55.6	H
Mack	MR	3.5	14.5	5.4	56.1	60.6	37.8	H
Essex	S	11.2	4.4	4.4	72.5	33.6	40.2	L
Wilstar 550	S	5.1	5.4	0.8	61.5	44.2	32.5	L
Coker 425	S	4.7	4.7	7.1	20.9	48.7	50.1	M
Narow	S	2.9	9.8	1.3	24.4	42.0	27.9	H
Pioneer 5482	···	7.6	0.4	0.7	55.4	16.4	14.3	M
Forrest	S	5.1	2.2	0.2	30.1	23.5	14.9	M
Asgrow 5980	R	3.3	0.0	1.5	21.9	9.6	22.0	M
Pioneer 9571	MR	1.4	0.0	2.6	2.3	0.9	21.4	M
Coker 355	MR	1.9	0.0	0.9	8.1	0.0	14.6	L
Asgrow 5474	MR	3.8	0.0	0.0	5.8	4.3	0.5	H
Bedford	S	0.9	0.0	1.2	0.0	0.0	5.2	M
Epps	MR	0.5	0.0	0.0	0.0	0.0	0.0	L
LSD within year				7.6				27.2
LSD across years				8.2				27.6
Maturity group VI								
Lee 74	S	13.2	43.1	34.2	103.6	145.1	109.6	H
Funks Experimental 1492	···	15.1	11.4	40.9	103.0	77.3	126.2	H
Deltapine 506	S	13.1	12.2	9.6	76.2	77.6	71.6	H
Tracy M	S	15.8	11.0	8.6	73.9	61.8	56.5	L
Jeff	R	18.0	3.8	11.5	105.7	29.9	47.7	H
Hartz 6383R	MR	13.9	15.5	1.5	52.9	68.8	58.0	M
Centennial	MR	13.6	9.0	6.6	59.0	55.0	59.8	M
Ringaround 604	···	8.9	9.7	6.6	41.6	60.3	58.4	M
Davis	S	8.2	4.1	5.0	51.8	33.8	52.6	L
Sumter	···	7.2	1.8	0.0	27.1	22.7	20.2	L
Asgrow 6520	S	8.1	3.8	3.5	26.2	2.4	40.3	L
Ringaround 606	S	10.1	1.2	0.0	37.3	20.5	0.0	L
Hartz 6130	MR	7.9	2.5	1.3	16.2	5.1	24.9	L
Leflore	R	7.4	0.0	0.0	28.9	0.0	2.1	H
LSD within year				10.1				33.3
LSD across years				12.3				36.3
Maturity groups VII & VIII								
Coker 237	S	17.4	45.8	12.0	66.4	151.6	103.3	H
Braxton	S	18.6	35.9	20.1	80.4	114.9	106.4	H
Asgrow 7372	···	18.7	29.0	29.4	45.3	124.3	125.1	H
FFR 771	···	23.0	37.8	9.6	108.9	111.7	67.2	M
Starr	S	20.4	16.9	20.6	92.4	73.0	102.0	M
Bragg	S	17.3	7.2	9.3	64.8	59.1	74.2	M
Gordon	MR	20.3	11.8	0.0	65.0	52.8	65.0	M
Coker 6727	···	18.7	12.2	0.0	50.5	60.2	36.6	M
Deltapine 497	S	19.1	6.7	0.2	34.6	31.8	45.3	L
Hartz 8112	S	15.8	0.0	0.0	19.8	0.0	5.7	L
LSD within year				16.6				36.9
LSD across years				20.7				39.7

[a] Susceptibility of cultivars to SDS based on other tests or observations in grower's fields in Arkansas before 1986: H = high, M = moderate, and L = low susceptibility (J. C. Rupe and M. C. Hirrel, *unpublished*).

[b] Area under the disease progress curve in rating days at soybean growth stages R_3 and R_6 based on a 0–5 rating scale of foliar symptoms where 0 = no symptoms, 1 = 1–10%, 2 = 11–30%, 3 = 1–70%, 4 = 71–90%, and 5 >90% of the leaf area with symptoms.

[c] Reaction of cultivars to race 6 of the soybean cyst nematode, *Heterodera glycines* Ichinohe: S = susceptible, MR = moderately resistant, R = resistant, and ··· = not tested (8).

[d] Least significant difference calculated at the $P = 0.05$ level for AUDPCs within a column (year).

[e] Least significant difference calculated at the $P = 0.05$ level for AUDPCs across columns (years).

Table 3. Correlation coefficients (R) of cultivar ranking[a] for sudden death syndrome (SDS) between growth stages R_3 and R_6 over 3 yr at the Pine Tree Experiment Station at Colt, Arkansas

Maturity group[b]	Year		
	1986	1987	1988
IV	1.00 (0.0001)[c]	0.95 (0.0513)	0.40 (0.6000)
V	0.84 (0.0002)	0.95 (0.0001)	0.86 (0.0001)
VI	0.85 (0.0001)	0.95 (0.0001)	0.85 (0.0001)
VII & VIII	0.53 (0.1173)	0.94 (0.0001)	0.91 (0.0002)

[a]Correlation coefficients derived by Spearman's rank correlation procedure (15).
[b]There were four, 14, 14 and 10 cultivars in maturity groups IV, V, VI, and VII and VIII, respectively.
[c]Probability levels for significant correlations.

ated with the response of the cultivar to race 6 of SCN, the predominate race in this field (8). The cultivars in this group that were susceptible to race 6 (Essex, Wilstar 550, and Tracy M) had high levels of SDS. Those that were moderately resistant or resistant (Asgrow 5474 and Leflore) had very little SDS but in previous observations had exhibited low and high levels of SDS, respectively. It is not known if SCN was present in the fields where the previous observations were made or, if present, what the predominate race was. With the other cultivars, there was no consistent association between response to race 6 of SCN and SDS development. SCN has been associated with SDS (4,14), and the presence of SCN in greenhouse tests has hastened and intensified the development of foliar symptoms of SDS (9). It may be that SCN is an important predisposition factor with some, but not all, cultivars.

The later disease was assessed the greater the differences in disease development but, generally, the cultivar ranking within a maturity group did not change (Tables 2 and 3). Thus, the ranking of a cultivar at R_3 is a strong indication of what its ranking will be at R_6 even if there are no significant differences in disease at R_3. Because it may not always be possible to make disease assessments at the optimum time for cultivars in each maturity group, disease rankings can be used to indicate how cultivars differ in their response to SDS.

From the results presented in this paper, we have shown that there are significant differences in cultivar response to SDS. While these differences are consistent from year to year and location to location for most cultivars, the disease response of some cultivars is quite variable. Because of this variability, cultivars should be evaluated over a number of years and locations. Results from different states should also be compared. Comparisons between states would be greatly aided by uniform testing procedures, particularly a uniform rating scale, use of standard susceptible and resistant check cultivars, and assessment of the presence and the predominate race of SCN. Further research is needed to determine the factors that influence SDS and cause variability in results between years and locations, the role of SCN in the development of SDS, and the genetic nature of resistance to SDS.

ACKNOWLEDGMENTS

We thank Robert W. Turner, William F. Johnson, and Roger L. Eason for maintenance of the field plot; Michael L. Courtney for technical assistance; Kevin C. Thompson and Lori S. Spurgeon for statistical programming assistance; and the Arkansas Soybean Promotion Board for partial funding of this project.

LITERATURE CITED

1. Clark, I. 1979. Practical Geostatistics. Elsevier Applied Science Publishers, Essex, England. 129 pp.
2. Fehr, W. R., Caviness, C. E., Burmood, D. T., and Pennington, J. S. 1971. Stage development descriptions for soybeans, *Glycine max* (L.) Merrill. Crop Sci. 11:929-931.
3. Gibson, P. T., Oliver, J. W., and Anders, M. L. 1988. Soybean sudden death syndrome: Variety testing in Illinois. (Abstr.) Proc. Am. Soc. Agron. 80:81.
4. Hershman, D. E., Hendrix, J. W., Stuckey, R. E., Bachi, P. R., and Grove, J. H. 1987. Effect of soybean cultivars and planting date on development of soybean sudden death syndrome. (Abstr.) Phytopathology 77:1689.
5. Hirrel, M. C. 1983. Sudden death syndrome of soybean-A disease of unknown etiology. (Abstr.) Phytopathology 73:501-502.
6. Hirrel, M. C. 1986. Disease severity and yield loss comparisons of soybean maturity groups affected in sudden death syndrome. (Abstr.) Page 61 in: Proc. Annu. Meet. South. Soybean Dis. Workers, 13th.
7. 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.
8. Riggs, R. D., Hamblen, M. L., and Rakes, L. L. 1988. Resistance in commercial soybean cultures to six races of *Heterodera glycines* and to *Meloidogyne incognita*. Ann. Appl. Nematol. 2:70-76.
9. 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.
10. Rupe, J. C. 1987. Soybean cultivar response to sudden death syndrome. Arkansas Farm Res. 36:7.
11. Rupe, J. C. 1988. Relationship of cultivar susceptibility, *Fusarium solani*, and soybean cyst nematode to soybean sudden death syndrome (SDS). (Abstr.) Phytopathology 78:1545.
12. Rupe, J.C. 1988. Soybean sudden death syndrome cultivar testing procedures. (Abstr.) Page 70 in: Proc. Annu. Meet. South. Soybean Dis. Workers, 15th.
13. Rupe, J. C. 1989. Frequency and pathogenicity of *Fusarium solani* recovered from soybeans with sudden death syndrome. Plant Dis. 73:581-584.
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. 106 pp.
15. SAS Institute, Inc. 1988. SAS User's Guide: Statistics. Version 6.03 ed. Cary, NC. 1028 pp.
16. Sciumbato, G. L., and Keeling, B. L. 1985. Sudden death syndrome (SDS) of soybeans in Mississippi in 1984. (Abstr.) Page 64 in: Proc. Annu. Meet. South. Soybean Dis. Workers, 12th.
17. 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.