Effects of Tillage, Cultivar, and Planting Date on Percentage of Soybean Leaves with Symptoms of Sudden Death Syndrome

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ABSTRACT

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A 4-year experiment, 1991 to 1994, was conducted in a field where sudden death syndrome (SDS) had previously been observed. The objective was to determine the effects of tillage, planting date, and soybean cultivar on the percentage of leaves with symptoms of SDS at R6 growth stage. The soybean cultivars Essex, Forrest, Hartwig, and Rhodes were each planted in 75-cm-wide rows in disk-till, ridge-till, and no-till plots. The planting dates were mid-May, mid-June, and late June to early July each year. Symptoms of SDS developed in 1991, 1992, and 1994, but in not 1993. There were significant year × cultivar (P = 0.0001) and tillage × planting date × cultivar (P = 0.05) interactions for the percentage of leaves with symptoms of SDS. Essex, Forrest, and Rhodes had a greater percentage of leaves with symptoms of SDS than did Hartwig in 1991 and 1994; differences among cultivars did not occur in 1992. The percentage of Essex, Forrest, and Rhodes leaves with symptoms of SDS was greater for no-till than for either disk-till or ridge-till in mid-May plantings. There were no significant correlations between the percentage of leaves with SDS and yield.

Sudden death syndrome (SDS), caused by Fusarium solani (Mart.) Sacc. (17), has been a problem of soybean, Glycine max (L.) Merr., in the central United States, including Arkansas, Illinois, Indiana, Iowa, Kentucky, Mississippi, Missouri, and Tennessee, since the mid-1980s. Leaf symptoms of SDS first appear as interveinal chlorotic blotches that become necrotic while the midvein and major lateral veins remain green (17). Severely affected plants may defoliate prematurely, and pod abortion may also occur. The vascular tissue in the upper taproot and lower stem turns gray-brown, but the pith remains white. This disease has caused extensive yield losses in some fields (5).

There are reports that SDS is affected by tillage systems. Ploper (11) observed that SDS incidence was higher in the nontilled than in the tilled areas of a field in Argentina. Von Qualen et al. (22) determined that premature dying of soybean in Indiana was higher in nontilled than in tilled plots; the foliar symptoms they observed in 1985 were similar to SDS; and the symptoms of both SDS and brown stem rot occurred in 1986.

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The effect of tillage systems on soybean diseases is important because there is a trend toward reduced tillage in soybean in the United States (7). Tillage before planting a full-season soybean crop can affect the incidence of brown spot (21) and stem canker (13,21). Tillage before planting soybean in a double-crop rotation can affect the incidence of brown spot and bacterial blight (23). Tillage before planting can also affect the population dynamics of soilborne pests such as soybean cyst nematode (SCN) (3,20) and *Rhizoctonia* spp. (10).

We initiated an experiment in 1991 to determine the effects of three tillage treatments, three planting dates, and four soybean cultivars on the occurrence of soybean diseases.

Symptoms of SDS were observed in plots. Treatment effects on SDS symptom expression are reported here. The site was infested with *Heterodera glycines*.

MATERIALS AND METHODS

In March 1991, tillage treatments were established in two separate areas of a field where soybean was planted in 1989 and corn was planted in 1990. The areas were separated by a 15-m alley. In 1991, corn, Zea mays L., was planted in one area and soybean in the other. Soybean and corn were rotated each year thereafter. The tillage treatments were maintained in the same plots each year. Corn was always planted in mid-April. Corn and soybean were rotated to make the data more acceptable and realistic to Missouri soybean farmers, because most of them follow a

similar rotation program.

Plots were established in 1991 near Portageville, Missouri, on a Tiptonville sandy loam soil (Typic Argiudolls) consisting of 70% sand, 25% silt, and 5% clay, where sudden death syndrome had previously been observed. The soil was infested with H. glycines race 4 (12). Prior to soybean planting in 1991 and each subsequent year, the plots were disk-tilled (disked twice), ridge-tilled (soil mounded over the previous year's row with a diskbedding implement), or not tilled (left undisturbed). The top 10-cm of the ridgetill beds was pushed off just prior to planting to form a flattop ridge. Each 8row (75-cm spacing) soybean plot was 6 m wide and 6 m long and was treated with a preemergence application of imazaquin at 0.14 kg a.i./ha and alachlor at 0.4 kg a.i./ha. All plots were treated with a postemergence application of bentazone at 0.181 kg a.i./ha. Disk-till and ridge-till plots were cultivated once, and all plots were hand-weeded if necessary.

A split-split plot design with four replicates comprised three tillage treatments (disk-till, ridge-till, no-till) as main plots and three soybean planting dates (mid-May, mid-June, and late June to early July) as subplots. The soybean cultivars Essex, Forrest, Hartwig, and Rhodes, all maturity group V, were randomized within subplots. Hartwig is resistant to all races of SCN; Essex is susceptible to all races; and Forrest and Rhodes are resistant to races 1 and 3 but susceptible to race 4. Soybean planting dates were 22 May, 18 June, and 9 July 1991; 14 May, 15 June, and 7 July 1992; and 12 May, 2 June, and 21 June 1993. Soybeans were harvested at maturity and yield was adjusted to 13% moisture.

During early May of 1991 through 1994, each soybean plot was sampled for *H. glycines*. Each sample was a composite of 10 soil cores (2.5-cm-diameter × 20-cm-deep) collected from the two center rows of each plot. The sample was mixed and a 100-cm³ subsample of soil was processed with a semiautomatic elutriator (1) for extraction of cysts. Eggs were freed from cysts using the Ten-Broek tissue homogenizer and counted.

The center two rows of each soybean plot were examined for SDS symptoms at the R6 (4) stage of plant development, when pods contain full-size green beans at one of the four uppermost nodes; and the percentage of leaves exhibiting SDS

symptoms was estimated. In 1991, 1992, and 1994, 20 arbitrarily selected plants with symptoms typical of SDS were collected from the entire test area, but not from each plot. Plants were not collected in 1993 because no SDS symptoms developed. The plants were examined for symptoms of diseases such as stem canker, SDS, and brown stem rot. Isolations were made from the upper taproot tissue of the plants within 1 day of collection using the techniques described by Rupe (15).

The percentage of leaves with SDS symptoms was subjected to analysis of variance (6). Fisher's least significant difference test was used for mean comparison, and correlation analysis was used to investigate relationships between numbers of H. glycines eggs, percentage of leaves with SDS symptoms, and soybean yield. Analyses were conducted with SAS (SAS Institute, Cary, NC).

RESULTS AND DISCUSSION

Symptoms of SDS were observed among plots in 1991, 1992, and 1994, but not in 1993. F. solani was isolated from the upper taproot tissue from all sampled plants collected that exhibited foliar symptoms of SDS during those 3 years. The isolates were similar to those described by Roy et al. (14) and Rupe (15). Symptoms of stem canker and brown stem rot were not observed on any of these plants.

Analysis of variance for data collected in 1991, 1992, and 1994 indicated that all main effects were significant (Table 1).

Table 1. Analysis of variance for percentage of soybean leaves with sudden death syndrome symptoms by tillage, soybean cultivar, planting date, and yeara

Source of variation	df	Mean square ^b	
Year (Y)	2	2,971.88	**
Rep	3	683.76	
Error A	6	271.93	
Tillage (T)	2	2,493.62	***
$Y \times T$	4	469.59	**
Error B	18	204.89	
Plant date (PD)	2	1,540.84	****
Y×PD	4	77.85	
$T \times PD$	4	369.91	***
$Y \times T \times PD$	8	49.17	
Error C	54	65.47	
Cultivar (C)	3	1,736.61	****
Y×C	6	330.89	****
$T \times C$	6	340.32	****
$PD \times C$	6	161.24	*
$Y \times T \times C$	12	81.34	
$Y \times PD \times C$	12	74.83	
$T \times PD \times C$	12	129.15	*
$Y \times T \times PD \times C$	24	53.85	
Error D	243	57.88	

^a Tillages were no-till, disk-till, and ridge-till. Planting dates were mid-May, mid-June, and late June to early July. Cultivars were Essex, Forrest, Hartwig, and Rhodes.

Likewise, several two-way interactions were significant, but only one three-way interaction was significant. The percentage of leaves with symptoms of SDS was greater in Essex, Forrest, and Rhodes than in Hartwig in 1991 and 1994; differences among cultivars did not occur in 1992 (Table 2). The percentage of leaves with symptoms varied among years within a cultivar. The percentage of Essex, Forrest, and Rhodes leaves with symptoms was greater in no-till than in either disk-till or ridge-till for mid-May plantings (Table 3). In mid-June plantings, the percentage of Forrest and Rhodes leaves with symptoms was greater in no-till than in either disk-till or ridge-till. Differences in symptoms among cultivars and tillage treatments was not observed for late-June to early-July plantings.

Symptoms of SDS were mild in 1991, 1992, and 1994, and there was no significant correlation between percentage of leaves with SDS symptoms and soybean yield. Thus, yield data are not included here. Hershman et al. (5) demonstrated that soybean yields were generally not correlated with area under the SDS disease progress curve when symptoms were mild and developed relatively late (pod-fill or later). Stephens et al. (19) also demonstrated that soybean yields were not correlated with SDS severity.

Our results confirm those of Hershman et al. (5) that SDS symptoms are greater in early- than late-planted soybean. However, we only observed this in no-till plantings. Symptoms of SDS were mild in our test, and that may be the reason no differences in SDS symptoms were observed among planting dates in tilled plots as Hershman et al. reported. They stated that the soil moisture at the V3 growth stage, when three nodes are present on the main stem beginning with the unifoliate node, was greater in early than in late plantings; and that may have enhanced infection of plants by F. solani. So SDS symptoms may have been more severe in early than in late plantings because of differences in infection. F. solani was found in soybean roots as soon as 3 weeks after emergence (2), but the effect of soil moisture on earlyseason infection is poorly defined. The effect of early-season infection on SDS symptom development is also poorly defined.

Our results also confirm the observations of Ploper (11) and Von Qualen et al. (22) that symptoms of SDS are greater in nontilled than in tilled plantings. The nontilled plots may have been more an-

Table 2. The effect of year and cultivar on percentage of soybean leaves^a with symptoms of sudden death syndrome averaged over tillage treatments^b and planting dates^c

Year	Cultivar				
	Essex	Forrest	Hartwig	Rhodes	
1991	13.61	11.39	0.28	15.83	
1992	0.00	1.39	0.00	3.44	
1994	7.64	7.08	0.42	9.72	

 $LSD_{(0.05)}$ cultivar means for same year = 3.52 $LSD_{(0.05)}$ year means for same or different cultivar = 1.63

Table 3. The effects of tillage, planting date, and cultivar on percentage of soybean leaves^a with symptoms of sudden death syndrome averaged over 3 years^b

Tillage treatment	Cultivar	Planting date		
		Mid-May	Mid-June	Late June
No-till	Essex	25.42	8.75	7.50
	Forrest	23.00	10.83	6.67
	Hartwig	0.00	0.00	0.00
	Rhodes	22.00	17.92	5.83
Disk-till	Essex	7.50	4.17	3.33
	Forrest	2.50	3.33	1.25
	Hartwig	0.00	0.00	0.00
	Rhodes	9.92	4.58	5.42
Ridge-till	Essex	1.67	3.33	2.08
	Forrest	9.08	0.83	2.08
	Hartwig	0.00	0.00	0.00
	Rhodes	10.42	7.08	3.33

 $LSD_{(0.05)}$ same tillage and planting date = 7.70

^b Significance levels: * P = 0.05; ** P = 0.01; *** P = 0.001; **** P = 0.0001.

^a Percentage of leaves with symptoms evaluated at the R6 stage of soybean growth.

^b Tillages were no-till, disk-till, and ridge-till.

c Planting dates were mid-May, mid-June, and late June to early July.

 $LSD_{(0.05)}$ same tillage but across planting dates = 7.82

 $LSD_{(0.05)}$ across tillage same or different planting date = 7.29

^a Percentage of leaves with symptoms evaluated at the R6 stage of soybean development.

^b Years were 1991, 1992, and 1994; no symptoms developed in 1993.

aerobic, cool, and moist than tilled plots. Any of these conditions may have affected the soybean–*F. solani* interaction and thus SDS symptom expression.

The differences in the percentage of leaves with SDS between no-till and disk-till or ridge-till plots were observed in our plots even though SDS was mild. We speculate that these differences in symptom development between tillage treatments may also occur when symptoms are severe.

This is not the first report of variation in SDS symptoms among cultivars and between years. Hershman et al. (5), Melgar and Roy (9), and Rupe et al. (16) found SDS varied among cultivars. Hershman et al. (5) and Rupe et al. (16) also found that SDS severity on a cultivar varied between years. The variation in symptom expression between cultivars can be genetically controlled (18). The variation in SDS symptoms between years is probably due to yearly environmental variation. McLean and Lawrence (8) speculated that a growing season that begins with cool temperatures followed by relatively high temperatures and adequate season-long moisture creates optimal conditions for SDS symptom development. The mechanism by which the environment affects SDS symptom development is unknown.

There was no significant correlation between preplant numbers of H. glycines eggs and percentage of leaves with SDS symptoms even in the SCN-susceptible cultivars. Thus egg data are not included here. Others have reported that H. glycines may enhance SDS symptoms. In greenhouse (14) and field microplot studies (8), foliar symptoms of SDS were more severe when F. solani and H. glycines were both present than with F. solani alone. Hershman et al. (5) observed that in a field infested with both pathogens, cultivars resistant to H. glycines were less affected by SDS than were susceptible cultivars. However, correlations between area under the disease progress curve for SDS and H. glycines cyst density at harvest were not significant for most cultivar and planting date combinations.

Our data demonstrate that SDS symptoms were higher in nontilled than in tilled systems. Soybean losses to SDS may therefore increase as the acreage of nontilled soybean production in the United States increases. Expression of SDS symptoms may be less in nontilled soybean when planting late rather than early, but the yield of soybean declines as planting is delayed. Efforts to develop cultivars resistant to SDS and alternative management systems must be enhanced to protect the U.S. soybean crop from this disease.

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