Effects of Rotation, Tillage, and Fumigation on Premature Dying of Soybeans

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ABSTRACT

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Premature dying of Miami soybeans was reduced by crop rotation and fumigation and increased with reduced tillage in the USDA ARS-Purdue Integrated Pest Management systems in 1985 and 1986. In 1985, symptoms of premature dying of soybean plants in central Indiana were similar to those reported for sudden death syndrome (SDS). *Diaporthe* spp. were consistently associated with plants dying at the R6 growth stage. These plants were shorter, had fewer nodes, and yielded less than plants that did not die prematurely. Premature dying was more extensive in a continuous soybean (S-S) cropping than in a corn-soybean (C-S) rotation and was least extensive in a wheat-corn-soybean (W-C-S) rotation (11, 6, and 2%, respectively). Yields of the S-S, C-S, and W-C-S rotations were 2,650, 3,047, and 3,262 kg/ha, respectively. The dying pattern paralleled yield reductions. Soil fumigation with metham (sodium methyldithiocarbamate) reduced the incidence of SDS and increased yield both years. In 1986, symptoms of SDS and brown stem rot occurred. *Phialophora* and *Diaporthe* were isolated from prematurely dying plants. Classical pith discoloration symptoms caused by *P. gregata* were predominant. More plants died prematurely in the S-S rotation (11%) than in the W-C-S rotation (2%), and fumigation of soil with metham reduced premature dying from an average of 5 to 3%. Premature dying in the S-S rotation was consistently higher in no-tillage plots than in either conventionally tilled or chisel-plowed plots both years.

Sudden death syndrome (SDS) of soybeans (*Glycine max* (L.) Merr.) has been observed in the Midwest in recent years. Symptoms of the disease were first reported in the early 1970s in Arkansas (12). Hirrel described SDS as a premature dying of soybeans after flowering and before maturity; he described the symptoms as interveinal chlorosis developing into interveinal necrosis within 5–10 days until only tissue near the major leaf veins remains green (9,12). These symptoms appear throughout the plant but are most severe on the top leaves. Leaves abscise at the apex of the petiole, leaving leafless stems; later, the petioles may also abscise. When symptoms occur before the R6 growth stage, pods also abscise. The abscission of pods is most pronounced at the top of the plant. Plants that develop symptoms later produce smaller, lighter seed. The vascular tissues of affected plants become reddish brown, but the pith retains its normal white appearance. The discoloration progresses outward from vascular tissue. Roots of affected plants appear normal, but plants with advanced symptoms have decayed lateral roots. Hirrel reported yield losses of 20–70% in affected fields (10). The role or roles of dominant soybean pathogens in SDS or in association with recently described strains of *Fusarium solani* Snyd. & Hans. (K. W. Roy, *personal communications*) have not been verified, although numerous pathogens and environmental conditions have been associated with SDS.

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Table 1. Agronomic characteristics of soybean plants in 1985

	Healthy plants	Prematurely dying plants[z]
Plant weight (g/20 plants)	527	254**
Seed yield (g/20 plants)	123	50**
Height (cm)	86	73**
Number of nodes	14	12**
Root length (cm)	8	7 ns

[z]** = Highly significant difference ($p = 0.01$) between healthy and prematurely dying plants, according to the F value from the analysis of variance; ns = no significant difference at $p = 0.05$, according to the F value from the analysis of variance.

Table 2. Fungal colonization of soybean stems in 1985

Pathogen	Healthy plants[y]	Prematurely dying plants[z]
Septoria	1.8	1.4 ns
Macrophomina	1.5	1.8 ns
Cercospora	3.5	1.9**
Colletotrichum	1.2	1.8*
Glomerella	1.9	2.6**
Diaporthe	1.9	3.6**

[y]Fungal colonization was rated on a scale of 1–5 (1 = no colonization detected; 5 = severe colonization, with 71–100% of the stem sample colonized).

[z]* = Significant difference between healthy and prematurely dying plants at $p = 0.05$; ** = significant difference at $p = 0.01$; ns = no significant difference at $p = 0.05$.

The recent appearance of SDS in Indiana poses a serious threat to the valuable soybean crop in that state. In 1985, premature dying similar to that reported by Hirrel occurred in Indiana, primarily in the southern part of the state but also in the central part, and in soybeans in the USDA-Purdue Integrated Pest Management study initiated in 1980 (18).

This study was established to determine the effects of rotation, tillage, fumigation with Vapam (sodium methyldithiocarbamate), and foliar fungicide on premature dying of soybeans.

MATERIALS AND METHODS

The Integrated Pest Management plots were established in 1980 and are part of a USDA-Purdue University project located at the Purdue Agronomy Farm, where the soil is primarily Treaty, a Chalmers-like silty clay loam. The soybean cultivar Miami (maturity group II) was planted (20 May 1985 and 4 June 1986) in rows 76 cm apart, at 333,580 plants per hectare. The incidence and severity of premature dying in three rotations, including continuous soybean (S-S) cropping and corn-soybean (C-S) and wheat-corn-soybean (W-C-S) rotations, were studied in soybean plots with optimum or moderate weed control (18). Three tillage systems, including conventional moldboard plowing, chisel plowing, and no-tillage, were evaluated with each rotation.

A split-split-plot design with three replications was utilized in this study. The whole plots were factorial combinations of the three crop rotation systems and the three tillage treatments, randomized in each replication. The whole plots were repeated so that all crops with a given rotation-tillage combination could be studied each year. The section of each whole plot used for this study was 25.8 × 3 m. The whole plots were split in half, and a randomly chosen half of each plot was fumigated with Vapam (32.7% sodium methyldithiocarbamate, also referred to as metham) at the rate of 1,124 L/ha. The fumigant was applied as a 1:1 solution of Vapam and water, by a modified anhydrous ammonia applicator equipped with a pump, and was delivered at a depth of 15 cm from 25-cm flat sweeps spaced 76 cm apart. Care was taken to leave crop debris intact in the chisel-plowed and no-tillage plots. The injector, with its valves off, was pulled through the unfumigated subplots to disturb the soil as in the fumigated subplots. Immediately before planting, the surface debris in the fumigated subplots was sprayed with captan (*N*-[(trichloromethyl)thio]-4-cyclohexene-1,2-dicarboximide, Captan 50WP) at 1.13 kg a.i./ha. Miami soybeans were planted 2½ wk after fumigation. The fumigated and unfumigated subplots were split further into split-split plots for foliar fungicide treatments and controls. In plots receiving foliar fungicide treatment, the soybeans were sprayed to a drip stage with a mixture of benomyl (methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate, Benlate 50WP) plus captan (Captan 50WP), each at 0.56 kg a.i./ha, every 20 days, beginning at the R2 growth stage (5). A backpack sprayer, powered by carbon dioxide, was used to apply the fungicides on the canopy of the plants at a constant pressure of 2.1 kg/cm^2.

The plants were monitored visually for disease development at growth stages R2, R4, R6, R7, and R8. Plants that died prematurely, at the R6 growth stage (2 September 1985 and 4 September 1986), were counted in each plot. Prematurely dying plants were counted and examined macroscopically for symptoms or signs of pathogens. In each main plot (rotation-tillage combination), 10 plants that had died prematurely and 10 apparently healthy plants were tagged on 5 September 1985, when they were in the late R6 growth stage, and were collected at maturity. The stems were visually rated on a 1–5 scale developed to describe the incidence and severity of fungal colonization: 1 = no sign of colonization on stems (none); 2 = one or two stems colonized (mild); 3 = 20–40% of the combined surface area of all stems colonized (moderate); 4 = 41–70% of the combined surface area of all stems colonized (high); 5 = 71–100% of stems colonized and total surface area of stems affected (severe). Isolations were made on potato-dextrose agar (PDA) from stem sections that had been placed in moist chambers for 48 hr.

After the stems were rated for colonization, plants that had died pre-

Table 3. Seed characteristics of soybeans in 1985

	Healthy plants	Prematurely dying plants[z]
Seed weight (g/100 seed)	23	17**
Germination (%)	83	93 ns
Infection by *Diaporthe* (%)	1	4 ns
Infection by *Cercospora* (%)	2	1 ns

[z]** = Highly significant difference ($p = 0.01$) between healthy and prematurely dying plants, according to the F value from the analysis of variance; ns = no significant difference at $p = 0.05$, according to the F value from the analysis of variance.

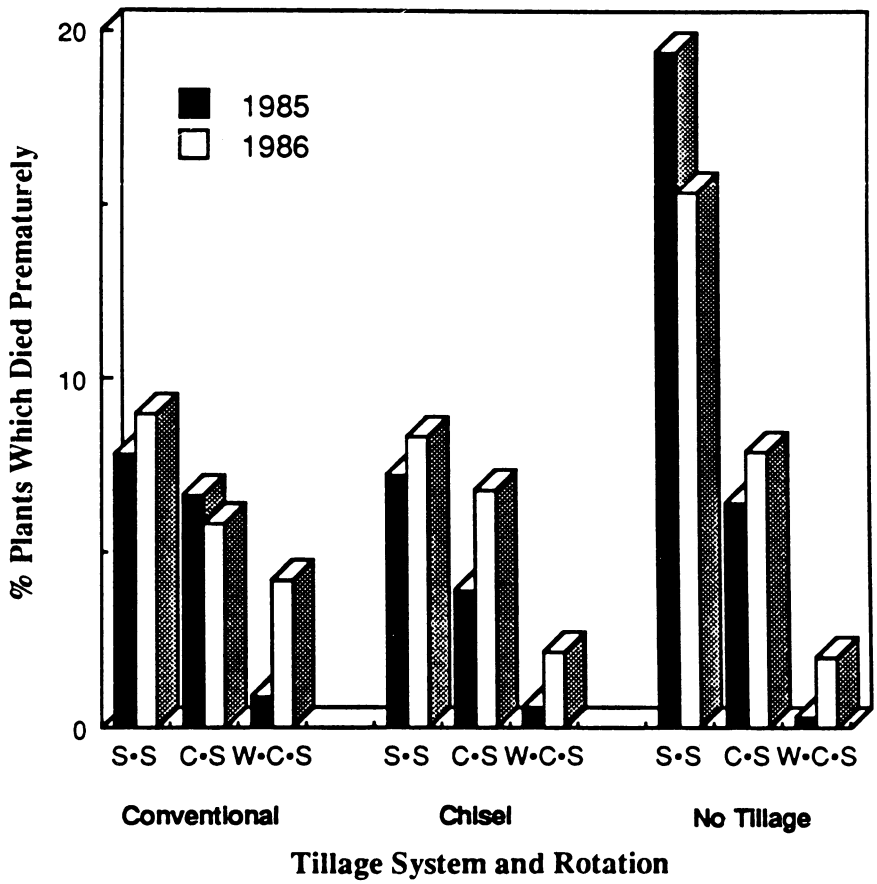

Fig. 1. Effects of rotation (S-S = continuous soybean cropping; C-S = corn-soybean rotation; W-C-S = wheat-corn-soybean rotation) and tillage (conventional tillage with a moldboard plow, chisel tillage, and no-tillage) on premature dying of soybeans. Premature dying was determined in early September of both 1985 and 1986, when healthy plants were in the R6 growth stage.

maturely and those that had senesced naturally were put into separate groups. Three bundles of 20 plants each were randomly chosen from these two groups. These plants were evaluated for total weight, height, number of nodes, root length, and seed yield. From each treatment, 100 seeds were sampled, weighed, surface-sterilized by soaking in 95% ethanol for 20 sec and then in a 0.1% hypochlorite solution for 1 min, and plated on PDA. The PDA plates were incubated for 7 days, and then microbial growth was identified.

In the field at maturity, 2.4 m of row was harvested from each subplot. Seeds were dried to 13% moisture and weighed, and a 100-seed sample was used to determine seed infection, by plating as described above. Also in 1985, five randomly chosen plants from each subplot were collected at maturity, and their stems rated for fungal colonization as described above. On 10 September 1986, 10 prematurely dying plants from each main plot (rotation-tillage combination) were examined macroscopically for the presence of pathogens. Stems of these plants were surface-sterilized with 0.1% sodium hypochlorite and placed on PDA for 6 days to isolate potential pathogens.

Data from 1985 and 1986 were analyzed separately. The Waller-Duncan Bayesian LSD was used for mean separations. Means that separated at $K = 100$ were considered significant (22). Where only two means were compared, the difference between them was considered significant if the calculated value of F exceeded the tabular value of F at the 0.05 probability level, and highly significant if it exceeded the tabular F at the 0.01 probability level.

Table 4. Effect of rotation on late-season soybean pathogens in 1985

	Disease assessment[w,x]		
	S-S	C-S	W-C-S
Seed infection (%)[y]			
Diaporthe	4.2 a	3.6 a	1.6 b
Cercospora	4.6 a	3.1 a	4.0 a
Stem colonization[z]			
Cercospora	2.6 a	2.6 a	2.7 a
Septoria	1.7 a	1.6 a	1.5 a
Diaporthe	2.5 a	2.1 b	1.7 c
Macrophomina	2.0 a	1.6 b	1.6 b
Glomerella	1.8 a	1.7 a	1.4 b
Colletotrichum	1.5 a	1.4 a	1.1 b
Dead plants (%)	12.7 a	6.2 ab	0.7 b

[w] S-S = continuous soybean cropping; C-S = corn-soybean rotation; W-C-S = wheat-corn-soybean rotation.
[x] Values in the same row followed by the same letter are not significantly different according to the Waller-Duncan Bayesian LSD (K = 100).
[y] Assessed by isolation.
[z] Rated on a scale of 1–5 (1 = no colonization; 5 = severe colonization, with 71–100% of the stem sample colonized).

RESULTS

In both 1985 and 1986, soybeans died prematurely at the R6 growth stage. In both years, dying was most severe in the S-S cropping in no-tillage plots. Soil fumigation reduced premature death but was least effective in the no-tillage plots.

In 1985, the symptoms of affected plants included dead stems, dried brown pods, and complete defoliation with petioles remaining intact. Vascular tissues were discolored brown, especially at the soil line. Some dying plants had extensive interveinal chlorosis and necrosis, and only tissue adjacent to major veins remained green. Symptoms developed rapidly on 20% of the soybeans in the no-tillage S-S plots; no symptoms were noticeable 10 days prior to the R6 growth stage. These symptoms were similar to those reported for SDS (9–12).

Plants that died prematurely in 1985 were significantly shorter, weighed less, and had fewer nodes and lower yields than healthy or naturally senesced plants (Table 1). A number of fungi pathogenic to soybeans were consistently found on mature stems: *Septoria glycines* Hemmi; *Cercospora kikuchii* (T. Matsu. & Tomoyasu) Gardner; *Diaporthe* spp.—*D. phaseolorum* var. *caulivora* Ath. & Caldw. and *D. p.* var. *sojae* (Lehman) Wehm. (grouped with its imperfect stage, *Phomopsis sojae* Lehman); *Glomerella glycines* (Hori) Lehman & Wolf; *Colletotrichum dematium* (Pers. ex Fr.) Grove var. *truncatum* (Schw.) Arx; and *Macrophomina phaseolina* (Tassi) Goid. These are all hereafter referred to by their genus names. Stems of plants that died prematurely were colonized significantly more by *Diaporthe, Glomerella*, and *Colletotrichum* ($p = 0.05$) and significantly less by *Cercospora* ($p = 0.01$) than stems of healthy plants (Table 2). The greatest differences between the stem colonization ratings of healthy plants and those of prematurely dying plants were in colonization by *Diaporthe* and *Cercospora*. Seed harvested from plants that died at the R6 growth stage was significantly lighter than seed from healthy plants (Table 3).

Similar levels of dying occurred at the R6–R7 stages of seed development each year, but the predominant symptoms of plants that died prematurely in 1986 were somewhat different from those observed in 1985. In 1986, the leaves of most prematurely dying plants remained attached to the plants, and the pith of the these plants was discolored a dark reddish brown. Also in 1986, less than 1% of the plants exhibited symptoms of defoliation, petioles attached to stems, normally colored pith, and discolored vascular tissue. Isolations made from stems in 1986 yielded *Phialophora gregata* (Allington & Chamberl.) W. Gams, *Diaporthe*, and *Macrophomina*. Isolations from stems with symptoms similar to those of the previous year also yielded *Diaporthe* and *Macrophomina*. *Fusarium* spp. occurred infrequently. Since the incidence of premature dying in 1986 was similar to that in 1985 and was extensive in both years, pathological and agronomic data are presented for 1985 plant samples tagged at growth stage R6 (healthy vs. prematurely dying) and randomly selected at R8.

Table 5. Effect of crop rotation, tillage, and soil fumigation on soybean yield in 1985

	Seed yield (kg/ha)[x,y]			
Tillage	S-S	C-S	W-C-S	Average
Conventional plowing	2,452 c	2,948 b	3,172 a	2,857
Increase due to fumigation[z]	134*	104*	222*	153*
Chisel plowing	2,717 b	2,869 ab	2,962 a	2,849
Increase due to fumigation	231*	12	214*	152*
No-tillage	2,552 b	2,749 b	3,038 a	2,780
Increase due to fumigation	203*	0	95	99*
Average	2,574 b	2,855 a	3,057 a	
Increase due to fumigation	189*	39	177*	

[x] S-S = continuous soybean cropping; C-S = corn-soybean rotation; W-C-S = wheat-corn-soybean rotation.
[y] Values for unfumigated plots in the same row followed by the same letter are not significantly different; * = significant increase in fumigated plots, according to the Waller-Duncan Bayesian LSD (K = 100).
[z] Fumigated plots were injected with Vapam (32.7% sodium methyldithiocarbamate) at 1,124 L/ha.

Effect of crop rotation. During both years, more plants died prematurely in the S-S rotation (11.2%) than in the C-S (6.3%) or the W-C-S rotation (1.7%) (Fig. 1). This pattern occurred in all tillage systems each year and was particularly obvious in no-tillage soybeans. In both years, significantly more plants died prematurely in the S-S rotation than in the W-C-S rotation. In all three rotations, *Diaporthe* and *Cercospora* colonized stems and infected seed more than the other fungal pathogens in 1985 (Table 4). Of these

two dominant organisms, only *Diaporthe* increased in the S-S and C-S rotations, compared to the W-C-S rotation. *Macrophomina* increased only in the S-S rotation. *Colletotrichum* and *Glomerella* increased in the S-S and C-S rotations, compared to the W-C-S rotation (Table 4). Crop rotation had a significant influence on yield in both years. Yield levels were similar in 1985 and 1986 and were consistently highest in the W-C-S plots. Yields were significantly lower in the S-S rotation than in the C-S or the W-C-S rotation (2,574, 2,855, and 3,057 kg/ha, respectively, in 1985).

Effect of tillage. More plants died prematurely in no-tillage plots than in either conventionally tilled or chisel-plowed plots, but these differences were not significant across crop rotation in either year (Fig. 1). Significantly more plants died prematurely in the no-tillage S-S plots than in plots with other rotation-tillage combinations (Fig. 1), but there was no significant interaction between rotation and tillage.

Evaluations of seed from the bulk yield and plants randomly chosen at maturity indicate tillage did not significantly ($p = 0.05$) affect the occurrence of the dominant pathogens, *Diaporthe* and *Cercospora*, in 1985. Also, the other pathogens colonizing soybean stems were not significantly influenced ($p = 0.05$) by tillage. In both 1985 and 1986, yields were consistently higher with conventional or chisel tillage than with no-tillage. These differences were significant in 1986, but not in 1985. Tillage affected yields most within the S-S rotation (Table 5), but rotation × tillage effects were not significant. Yields in the S-S rotation were higher with chisel plowing than with conventional tillage and were intermediate with no-tillage.

Effect of fumigation. Soil fumigation and the fungicide drench significantly reduced premature dying of soybeans (Fig. 2). The effect of fumigation was greatest in the conventionally tilled plots, intermediate in the chisel-plowed plots, and least in the no-tillage plots. With conventional tillage (in which fumigation was most effective) and within the S-S rotation (in which the incidence of pathogens was greatest), fumigation reduced premature dying. Also, the rating for stem colonization by *Diaporthe* in the randomly chosen mature plants was reduced from 3.7 to 3.0, and infection of mature seed by *Diaporthe* in the bulk yield samples was significantly reduced. Seed infection by *Cercospora* increased with fumigation (from 4 to 5%), and the rating for stem colonization by *Cercospora* (2.0) was unchanged. The less prevalent pathogens were not significantly influenced by fumigation.

The reduction in premature dying with fumigation was paralleled by yield increases with fumigation in both years. Yield differences for fumigation × tillage were also observed. Yields of conventionally tilled and chisel-plowed plots increased significantly with fumigation (with increases of 153 and 152 kg/ha, respectively, in 1985), but fumigation did not give a consistent yield increase in no-tillage plots (Table 5).

Effect of foliar fungicide. Foliar fungicides were not effective in significantly reducing the percentage of plants that died prematurely in either 1985 or 1986. In 1985, similar levels of premature dying occurred in soybean subplots with and without the foliar fungicide treatment. In 1986, the only significant difference was within the conventional tillage system, in which 6.4% of the plants sprayed with the foliar fungicide died prematurely, in contrast to 5.0% of the untreated plants. However, the foliar fungicide treatment reduced the incidence and severity of the late-season phases of seed infection and stem colonization by pathogens (Table 6). Seed yield of soybeans treated with the foliar fungicide was significantly higher than that of untreated soybeans (3,069 vs. 2,919 kg/ha). Yield increases due to the foliar fungicide were significant in the S-S and C-S rotations but not in the W-C-S rotation.

DISCUSSION

In 1985 and 1986, similar patterns of premature dying were observed in soybeans. However, two different groups of pathogens were associated with this premature dying. In 1985, *Diaporthe* was consistently associated with affected plants; in 1986, *Phialophora* and *Diaporthe* were involved.

Even though plants appeared to die suddenly in 1985, the agronomic characteristics of healthy and prematurely dying plants (Table 1) suggest premature dying was the end result of a sustained process. Physiologic maturity occurs at the R7 growth stage, and plants would not have grown as much between the R6 and R8 growth stages as suggested by the highly significant differences in plant height, weight, and number of nodes. These differences in plant size are likely due to latent infection, which did not manifest disease symptoms until the late R6 growth stage. The possibility that SDS symptoms can be modified by major soybean pathogens, particularly pathogens in the *Diaporthe-Phomopsis* complex, and occur after the development of latent infections, needs further study.

Since premature dying occurred during the R6 growth stage prior to physiologic maturity, seed size and yield were reduced, because of the shortened pod-filling period. The fact that the plants that died prematurely remained in the field until the other plants were mature may account for the increased infection of seed by *Diaporthe*. However, the fact that less *Cercospora* was recovered from the seed of prematurely dying plants than from healthy plants suggests that *Diaporthe* had colonized the prematurely dying plants and thus prevented

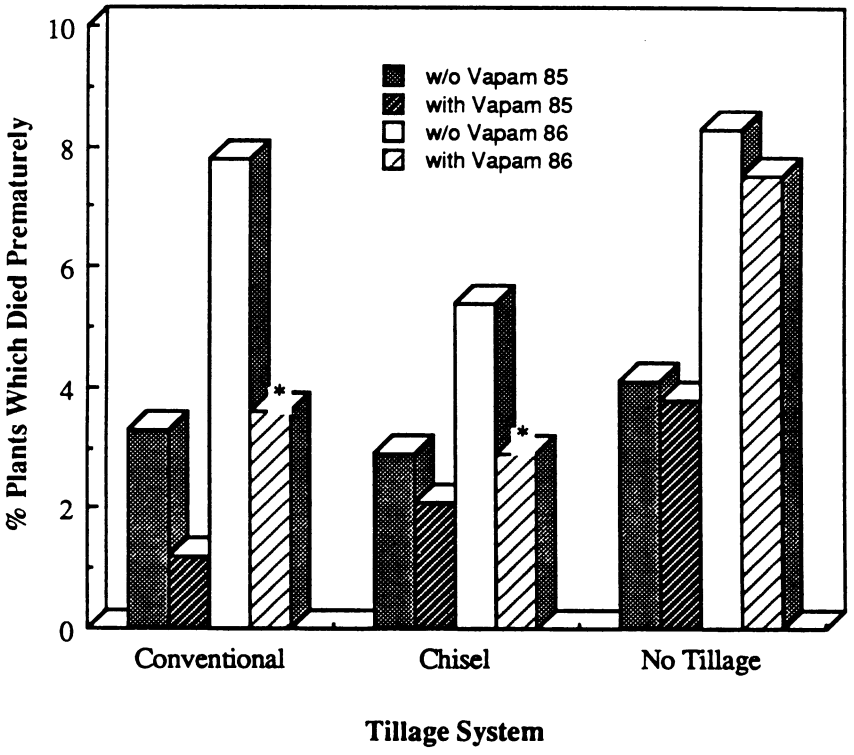

Fig. 2. Effects of preplant fumigation with Vapam on premature dying of soybeans in various tillage systems in 1985 and 1986. * = significantly fewer prematurely dead plants in the fumigated plots ($p = 0.05$), according to the F value from the analysis of variance.

the development of *Cercospora*, by the antagonistic relationship between these pathogens (17). The increased germination of seed from affected plants is difficult to explain. Improved germination has been reported with smaller seeds, but these comparisons were among different genotypes (4,16).

The percentage of plants that died prematurely, the percentage of seed infected by *Diaporthe*, and yield were influenced by rotation. Premature dying and seed infection by *Diaporthe* were most common in the S-S rotation, less in the C-S rotation, and least in the W-C-S rotation. Although it tended to be more common in S-S no-tillage plots, premature dying was not significantly reduced by conventional or chisel tillage. A similar pattern was observed in 1986.

In both years, the pathogens causing premature death did not increase after corn or wheat rotation, and control was better after a 2-yr rotation than after a 1-yr rotation. Hirrel (12) also observed that a 1-yr rotation was not effective in controlling SDS. Others have reported increased brown stem rot with S-S cropping (2,15). Dunleavy (1) reported good control of brown stem rot with a 5-yr corn rotation.

Both premature dying and the percentage of seed infected with *Diaporthe* were reduced with fumigation. This reduction was greatest with conventional tillage. The increased effectiveness of fumigation with conventional or chisel tillage is probably due to the greater dissipation of the fumigant and thus better control of pathogens. These results suggest the causal agent of the SDS-like symptoms is biological. Fumigation with Vapam has been shown to reduce the incidence of *Macrophomina* and *Phialophora* in soybeans and to increase soybean yields (8,13). Widin and Kennedy (24) associated yield increases in Vapam-fumigated plots with reduced fungal colonization of nodules and increased nitrogenase activity. Nodule colonization by fungi may reduce nitrogen fixation and induce stress on the plant, which could render the plant more susceptible to other pathogens. Because more than one pathogen is associated with SDS symptoms (12), and because we did not evaluate nodules closely in this study, the role of nodule invasion by pathogens as related to SDS needs further study.

Table 6. Effect of foliar fungicide on late-season soybean pathogens in 1985

	Disease assessment[w]	
	No fungicide	Fungicide[x]
Seed infection (%)[y]		
Diaporthe	3.1 a	0.2 b
Cercospora	3.9 a	0.1 b
Stem colonization[z]		
Cercospora	2.4 a	1.0 b
Septoria	2.1 a	1.0 b
Diaporthe	2.4 a	1.3 b
Macrophomina	1.9 a	1.3 b
Glomerella	1.7 a	1.2 b
Colletotrichum	1.3 a	1.2 a
Dead plants (%)	3.4 a	3.0 a

[w] Values in the same row followed by the same letter are not significantly different according to the Waller-Duncan Bayesian LSD (K = 100).

[x] Benomyl plus captan foliar fungicide.

[y] Assessed by isolation.

[z] Rated on a scale of 1–5 (1 = no colonization; 5 = severe colonization, with 71–100% of the stem sample colonized).

The soybean cyst nematode (SCN), *Heterodera glycines* Ich., did not appear to be involved in the premature death observed in 1985, since it was not present in the roots of soybeans involved in this study. Trace levels of SCN were identified in S-S plots in 1986. However, soybean plants with trace levels of SCN and depressed growth occurred in only a few isolated spots, outside the area sampled in these studies.

Environmental factors during August and September may account for the different roles of fungal pathogens associated with premature dying in these two years. Climatological data for Lafayette, Indiana, recorded at the Purdue Agronomy Farm (State Climatologist, Agronomy Department, Purdue University), show that August 1985 was unusually cool and wet, with temperatures as low as 9 C and rainfall amounting to 18 cm (double the average for West Lafayette in August). In contrast, rainfall in August 1986 was exceptionally low, amounting to only 5 cm. Soil temperatures at the 5- and 10-cm depths were below average in 1985 and above average in 1986. The two growing seasons were thus quite different and favored different pathogens. Hirrel observed that SDS is favored by cool, wet conditions and is most frequent in irrigated soybeans (10–12). The excessive moisture available in 1985 would also favor colonization by *Diaporthe* (which was most closely associated with premature dying), even at low temperatures (7,19,21,23). Brown stem rot, the predominant disease in 1986, is not associated with excessive moisture, and charcoal rot, caused by *Macrophomina*, is associated with dry conditions (3,6,14, 20,25). In this study, edaphic factors apparently were important in determining which pathogen dominated in the premature death of soybeans, and *Diaporthe* infections are identified as having a high potential to modify the severity of SDS.

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