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Sudden Death Syndrome of Soybean

Sudden death syndrome (SDS) is a relatively new disease of soybean (*Glycine max* (L.) Merr.) caused by blue-pigmented strains of *Fusarium solani* (Mart.) Sacc. (39,47,50,51,53,58) that have recently been designated *F. solani* f. sp. *glycines* (47).

SDS was first observed by H. J. Walters in Arkansas in 1971. The disease remained an unnamed curiosity until 1982, when M. C. Hirrel determined that it had severely reduced yields in that state. Hirrel coined the name of the disease in response to the perceived rapidity with which the above-ground symptoms developed (20,21,22). The name is not always descriptive, however, since disease progress is slowed and little premature plant death occurs when the cultivar involved has some degree of resistance and/or environmental conditions do not favor disease development. Nevertheless, rapid death of foliar tissue and whole plants can occur under favorable conditions.

SDS appeared to be confined to Arkansas until it was identified in several counties in Mississippi, Missouri, Kentucky, and Tennessee in 1984. By 1986, the disease had been found in Illinois and Indiana, and its current distribution in the United States now also includes Alabama, Georgia, Iowa, Kansas, Ohio, and Wisconsin (22,25,51,54,58,62,74; D. V. Phillips and A. F. Schmitthenner, *personal communications*). SDS also has been reported from southern and northern Argentina (45) and Brazil (40). Although the range of SDS has expanded greatly in the United States during the last decade, disease occurrence and annual yield losses remain greatest in counties along the lower Ohio River Valley and upper Mississippi River Delta (18,39).

Yield losses to SDS are especially dependent upon growth stage at the time of infection, cultivar reaction, and environment. In the United States, losses of up to

80% in individual fields have been attributed to SDS, but losses in the range of 5 to 15% are more common (18,21,22,62). Throughout the southern region of the United States, SDS caused an estimated average annual yield loss of 0.1% from 1988 to 1994, representing a loss of 280,000 t annually (72). In the north central United States, estimated average annual yield losses of 175,619 t occurred during 1989 to 1991 (9). In Argentina in 1994, SDS caused estimated yield losses of up to 90% in individual fields and an overall yield loss of 134,000 t. Losses to SDS have greatly increased in Brazil during 1991 to 1994. In that country, yield losses in individual fields have been as high as 30 to 40%, and in 1994 the overall yield loss was 15,000 t (71). These estimates illustrate the potential importance of SDS as a factor limiting soybean production in the United States and foreign countries.

Although many questions about SDS remain unanswered, much has been learned about this disease during the past decade. The main objectives of this report are to summarize our current knowledge of SDS, indicate how this knowledge can be used to manage the disease, and identify future research needs.

Symptoms and Signs

Symptoms usually develop at or shortly after flowering. The first leaf symptoms are circular to irregularly shaped, scattered, interveinal, pale green to chlorotic spots a few millimeters or more in diameter that produce a mottled appearance. Rugosity and a slight marginal cupping of young leaves is sometimes associated with mottling (Fig. 1A to C). The spots may enlarge and become necrotic or they may coalesce and form elongated regions of interveinal chlorosis. Eventually, part or all of the chlorotic tissue becomes necrotic and green tissue remains only near the major leaf veins (Fig. 1D to F). Some severely diseased leaflets may drop off, leaving the still-attached petioles bare (Fig. 1G). Since interveinal necrosis progresses faster on the upper than the lower leaves, the upper leaves are first to defoliate. Complete defoliation may occur when disease is severe (Fig. 1H). Severe infection during flowering and pod formation often results in the

abortion of flowers and pods (Fig. 1I). The uppermost flowers and pods tend to abort first (18–20,22,39,54,58,62). Remission of leaf symptoms is associated with hot, dry conditions (18,22,54). In plants with asymptomatic leaves or with only mild leaf symptoms, there may be no external root symptoms; root symptoms become prominent with increasing severity of the leaf symptoms. Internally, a grayish to reddish brown discoloration beginning near the pith and radiating outward through the vascular tissue may extend from the taproot up the stem for several nodes, with the pith remaining white (Fig. 1J). In plants with severe leaf symptoms, the internal root and stem discoloration is more pronounced, and there is considerable necrosis of the taproots and lateral roots as well as a reduction in root volume (Fig. 1J to L). A certain percentage of such plants may die prematurely. Severely infected plants are easily pulled from the soil. A bluish sporulation (macroconidia) may be produced by *F. solani* f. sp. *glycines* on the taproot and lower stem of some severely diseased plants (48) (Fig. 1M).

Plant height appears to be unaffected by SDS in the field (19,39). However, height reductions have occurred on artificially inoculated plants in the greenhouse (51,58).

The disease pattern in the field is at first typically scattered spots or streaks of affected plants. Diseased plants may be unevenly distributed in fields or extend throughout the fields (18,64).

Foliar symptoms of brown stem rot (*Phialophora gregata* (Allington & D.W. Chamberlain) W. Gams), red crown rot (*Cylindrocladium crotalariae* (C.A. Loos) D.K. Bell & Sobers), and stem canker (*Diaporthe phaseolorum* (Cooke & Ellis) Sacc. var. *caulivora* K.L. Athow & R.M. Caldwell) are similar to and may be confused with those of SDS (20,22). These diseases are distinguishable from SDS by symptoms: A distinct discoloration of the pith occurs in brown stem rot-infected plants; red perithecia are produced on stems of plants having red crown rot; and cankers occur on lower stems in stem canker-infected plants (4,20,22). In addition, the bluish sporulation on the root and lower stem helps distinguish SDS from these other diseases (48).

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Fig. 1. Symptoms of sudden death syndrome: (A) chlorotic spotting of leaves; (B and C) rugosity and chlorotic mottling of leaves; (D) severe foliar symptoms; (E and F) interveinal chlorosis and necrosis of leaves; (G) defoliation producing bare petioles; (H) nearly complete defoliation; (I) pod abortion; (J) stem and root vascular discoloration and root rot; (K) asymptomatic roots (left) compared with root rot and reduced root size in moderately (middle) and severely (right) diseased plants; (L) aerial parts of plants corresponding to roots in previous figure, showing defoliation and fewer pods in diseased compared with asymptomatic plants; and (M) blue sporulation (macroconidia) of *Fusarium solani* f. sp. *glycines* on taproot.

The Causal Agent

More than 9 years ago, Koch's postulates were completed for SDS by Roy et al. (50,51) and Rupe (58). They concluded that the causal agent was a blue-pigmented morphological form of *Fusarium solani*, which was designated form A (FSA) (50,51). Recently, FSA was designated *F. solani* f. sp. *glycines* form. nov. (47) on the basis of host (physiological) specialization (13,38), its distinction from *Fusarium solani* form B (cause of seedling disease and

root rot of soybean; 29,30) and other strains and formae speciales of *F. solani*, including *F. solani* f. sp. *phaseoli* (Burkholder) W.C. Snyder & H.N. Hans. (47), a closely related fungus (2,44), and further morphological and cultural characterization (47). Some researchers are of the opinion that SDS-inciting isolates of *F. solani* are *F. solani* f. sp. *phaseoli* (44) or that they represent a subgroup within *F. solani* f. sp. *phaseoli* or possibly a separate form species (2). The names *F. solani* f. sp.

phaseoli type A (6,23,42) and *F. solani* type A (41) have also been used in reference to the SDS causal fungus, but use of these names should be discontinued. Historical aspects of SDS etiology are found in other reports (47,50,51,53,58).

The persistent notion that *H. glycines* and other organisms may have direct causal involvement in SDS is perplexing. When Koch's postulates were first completed for SDS more than 9 years ago, *F. solani* f. sp. *glycines*, *H. glycines*, and *F. solani* form B (FSB) all were included in the pathogenicity tests (50,51,53). Only *F. solani* f. sp. *glycines* incited the symptoms of SDS. Moreover, Koch's postulates have been completed with *F. solani* f. sp. *glycines* several times, in both the greenhouse and the field, by at least four independent research groups (39,47,51,58,74).

F. solani f. sp. *glycines* grows slowly on potato dextrose agar (PDA), PDA amended with streptomycin and aureomycin (PDASA), modified Bilay's medium (47,48,50,51,52), potato sucrose agar, and V8 juice agar (K. W. Roy, unpublished). For example, colony diameter on PDA averaged only 24 mm after 7 days at 25°C, the optimum temperature for growth of *F. solani* f. sp. *glycines* on that medium (47). The fungus sporulates well on all four media (47,51,52; K. W. Roy, unpublished). Macroconidia are more uniform in morphology and are produced in greater numbers on modified Bilay's medium in continuous fluorescent light (52).

Colony morphology and pigmentation of *F. solani* f. sp. *glycines* are the same on PDASA and PDA (47). The blue pigmentation that has become associated with the SDS causal agent (47,48,51–53,58) is invariably produced by fresh cultures of *F. solani* f. sp. *glycines*. However, colonies exhibit considerable variation in surface pigmentation due largely to the amount of sporulation. Slimy masses of blue to blue-green macroconidia (Fig. 2A and B) are produced in distinct, effuse sporodochia. When viewed microscopically (×100), conidia appear reddish blue to dark blue. Isolates with varying degrees of sporulation produced colonies with varying amounts of blue pigmentation. In some profusely sporulating isolates, the blue to blue-green conidial masses occupy nearly the entire colony surface except for an outer fringe of grayish white, nonsporulating hyphae. The conidial mass radiating outward from the colony center usually is continuous, but small scattered groups of sporodochia sometimes appear (Fig. 2B). Scattered, small, cream-colored conidial masses sometimes occur in the older, central part of the colony (Fig. 2A and D). Dark blue (Fig. 2A and B), purple (Fig. 2C and D), and violet (Fig. 2E) spore masses tend to form and become admixed to varying degrees with the blue to blue-green spore masses as colonies age. The purple or violet spore masses sometimes occur as

Fig. 2. Variability in the surface morphology and pigmentation of approximately 14-day-old colonies of *Fusarium solani* f. sp. *glycines* isolates on potato dextrose agar (PDA). Isolates (A) A-15, (B) A-11, (C) A-14, (D) A-16, and (E) A-13 are first- or second-generation subcultures recovered from roots. Note typical blue to blue-green dominant pigmentation (A and B) with admixture of usually later-produced dark blue (A and B), purple (C and D), and violet (E) pigmentation and cream-colored spore masses (arrows; A and D). Isolate (F) A-15 was subcultured on PDA several times and stored in a PDA slant for 8 months prior to being transferred to plates of PDA.

sectors radiating from the colony center (Fig. 2C and E), particularly with frequent subculturing. Transfers from purple or violet spore masses usually produce, at least initially, colonies with blue to blue-green spore masses. Prolonged storage of the fungus on PDA invariably reduces sporulation and typical surface pigmentation with a concomitant increase in nonsporulating aerial mycelium (Fig. 2F). Transfers from sporulating regions of colonies that are almost entirely mycelial usually give rise to sporulating cultures (47,51). With aging of colonies, the agar darkens, the degree of darkening depending upon the isolate.

F. solani f. sp. *glycines* rarely, if ever, produces microconidia (51–53,58). It forms macroconidia on simple or branched conidiophores from monophialides (47,51–53) (Fig. 3A). Macroconidia are predominately three-septate, but four-septate conidia are numerous, and spores with five or more septa occur (51,52,58). On modified Bilay's medium, macroconidia range from approximately 34 to 66 μm long (50.5 μm average) by 3.9 to 5.0 μm wide (4.6 μm average) (47). On a rich medium such as PDA, macroconidia are larger, ranging from 30 to 65 μm long by 6 to 8 μm wide (58). Dimensions of macroconidia produced on soybean roots in the field range from 38 to 48 μm long (42.5 μm average) by 3.8 to 5.0 μm wide (4.5 μm average) (47). One or more chlamydospores may be formed in individual macroconidia (Fig. 3B), or they may be formed singly or in pairs, either terminally or in an intercalary fashion in hyphae (39,52).

F. solani f. sp. *glycines* has no known sexual stage. In India, a valid connection

was made between *F. solani* and *Nectria haematococca* Berk. & Broome from soybean (3), but this *F. solani* produced microconidia, and its colonies are unlike those of *F. solani* f. sp. *glycines* (47,51–53). A report connecting SDS-inciting isolates of *F. solani* with *N. haematococca* in the United States (1) was incorrect (44).

In addition to soybean, mung bean, green bean, lima bean, and cowpea are hosts of *F. solani* f. sp. *glycines* (13,38).

Differences in virulence occur among isolates of *F. solani* f. sp. *glycines* inoculated on soybean (15,38,58). Lim and Jin (33) presented preliminary data suggesting the fungus may be comprised of races, but this has not been confirmed.

In addition to *F. solani* f. sp. *glycines*, other *F. solani* strains occur on soybean roots: FSB (47,51), *F. solani* involved in the etiology of Essex syndrome (15), *F. solani* f. sp. *pisi*, and possibly other strains (15). Clearly much work remains in characterizing and distinguishing members of this complex. The use of molecular techniques (2) will speed the process. Recently, Achenbach et al. (2) used random amplified polymorphic DNA analysis to distinguish the SDS *F. solani* from *F. solani* f. sp. *phaseoli* and other strains of *F. solani* that do not incite SDS.

Disease Development and Epidemiology

Pathogenesis. Since macroconidia of *F. solani* f. sp. *glycines* convert to chlamydospores when incubated in a soil extract solution, and these chlamydospores occur in sloughed cortical tissue of soybean roots in the field (39), the fungus is assumed to reside in soil and in root debris mainly as

chlamydospores, which constitute the primary inoculum. The mode by which *F. solani* f. sp. *glycines* penetrates roots is unknown, but some evidence suggests it is direct (36). The fungus can colonize and infect roots of 2- to 3-week-old seedlings (42,51,57), and colonization appears to be limited primarily to cortical tissue of the root and lower stem (39,51–53,58). Hyphae grow intercellularly and intracellularly (Fig. 4), but mainly intracellularly. Hyphae are rarely observed in tissue above the crown region and in the stele only after considerable root degradation has occurred during the late stages of plant growth. At that time, chlamydospores may be observed in degraded, sloughed cortical tissue (39), and blue to blue-green masses of *F. solani* f. sp. *glycines* macroconidia may be visible on infected roots near the soil line (Fig. 1M). Sporulation on root surfaces is always associated with root rot and is more frequent during or immediately following periods of high soil moisture (39,48). In bean root rot, conidia produced on plants by *F. solani* f. sp. *phaseoli* are washed into soil by rain or irrigation and convert to chlamydospores, thereby increasing inoculum potential (31). The presumption that this occurs in the SDS disease cycle is compatible with recent data of Rupe and Becton (59; J. C. Rupe, unpublished). They found that inoculum density of *F. solani* (*F. solani* f. sp. *glycines*) increased near the time of soybean harvest. The inoculum densities ranged from 1,141 to 4,399 CFU/g of soil, depending upon the time of sampling soil in relation to the growth stage of the host (Table 1). Population densities of *F. solani* f. sp. *glycines* in soil were positively correlated with SDS severity (66). Much more information is needed on the relationship between inoculum density and SDS development.

Fig. 3. Reproductive structures of *Fusarium solani* f. sp. *glycines*: (A) light micrograph of macroconidiophores, monophialides, and macroconidia ($\times 825$); and (B) light micrograph of chlamydospores produced within macroconidia (scale bar = 20 μm).

Fig. 4. Scanning electron micrograph of intercellular and intracellular hyphae of *Fusarium solani* f. sp. *glycines* in soybean taproot cortical tissue. (scale bar = 25 μm)

Rupe and Gbur (60) reported that SDS disease progression was best described by a model consisting of two exponential equations that described a two-phase epidemic. In each of 3 years of their field study, there was a rapid increase in disease to about 35% leaf area affected followed by a slower increase in disease thereafter (Fig. 5). Rapid increases in SDS occurred during reproductive development, generally at the same time across cultivar maturity groups but at no specific reproductive stage; i.e., the rapid increases occurred from growth stages R2 to R5 (10). Symptom development was associated with accumulated temperature (degree-days).

The production of SDS leaf symptoms with concurrent localization of *F. solani* f. sp. *glycines* in the root and crown of soybean plants from the field (39,51–53,58) and inoculation experiments demonstrating that SDS leaf symptoms can be incited independently of root symptoms (58) suggest the involvement of a phytotoxin in leaf symptom expression, and this appears to be substantiated by more direct evidence. A phytotoxic polypeptide from culture filtrates of SDS-inciting isolates of *F. solani* caused browning of soybean calli, necrosis on detached cotyledons and leaves, and yellowing, curling, and drying of attached cotyledons and leaves. Isolates of *F. solani* that did not incite leaf symptoms on soybean also failed to produce the phytotoxic polypeptide (27,28). Monordin, another phytotoxin isolated from SDS-inciting isolates of *F. solani*, caused interveinal necrosis, curling, and drying of leaves when applied to soybean cuttings (5). These data strongly suggest that the unique ability of *F. solani* f. sp. *glycines* to incite SDS leaf symptoms (47) is conferred, at least in part, by one or more phytotoxic compounds.

Role of biotic and abiotic factors in SDS development. Few subjects related to SDS development have received more attention than the correlation of *H. glycines* and soil moisture and temperature to SDS symptom development. Field observations

by Hirrel in the early 1980s first suggested that the presence of *H. glycines*, and high soil moisture and cool temperatures during early soybean reproduction, favored disease development (22). He found from a survey of 30 SDS fields in four states that the nematode was associated with 70 to 80% of SDS-symptomatic plants. *H. glycines* is not necessary for infection of soybeans by *F. solani* f. sp. *glycines* but hastens the onset and increases the severity of SDS foliar symptoms (39,51). Rupe and Gbur (60) found that, on average, cultivars susceptible to race 6 of *H. glycines* developed SDS sooner and were more severely affected by SDS than were cultivars resistant to race 6. In microplot studies (39), tip dieback of pods occurred more frequently on SDS-symptomatic plants inoculated with *F. solani* f. sp. *glycines* and *H. glycines* than with *F. solani* f. sp. *glycines* alone. Despite these associations between *H. glycines* and SDS, positive correlation of *H. glycines* populations in soil at planting and harvest with severity of SDS leaf symptoms during soybean reproductive development has been absent or inconsistent (16,19,34,49,64,66). It may be that the reduced root systems of plants with severe SDS deprive the nematode of feeding sites or interfere with its reproduction (34,35). This response, combined with parasitism of different reproductive stages of *H. glycines* by *F. solani* f. sp. *glycines* (32,34–36,39), may actually result in reduced *H. glycines* populations in soil sampled during soybean reproduction and at harvest. This would account for inconsistent correlations between *H. glycines* populations at harvest and SDS foliar symptoms (64).

Enhancement of SDS by *H. glycines* (39,51), coupled with the typically irregular distribution of the nematode in fields (66), may help explain the occurrence of isolated areas of SDS-symptomatic plants, which are sometimes referred to as “hot spots.” Foci with a substantial nematode population density are likely to show SDS foliar symptoms before those with a low *H. glycines* population density (39,51,53).

Depending upon the local conditions, SDS may be confined to the hot spots, or if conditions favor SDS development, the remainder of the field may develop SDS. The size and shape of hot spots associated with high *H. glycines* populations may change over time as *H. glycines* is redistributed in fields by tillage, surface water movement, or other agents of dispersal (46).

F. solani f. sp. *glycines* was first isolated from *H. glycines* cysts in 1986 (K. W. Roy, SDS Workshop, St. Louis, MO, 1987, unpublished). Subsequent evidence for correlation between these two pathogens includes a report of *F. solani* f. sp. *glycines* hyphae found in association with *H. glycines* cysts rupturing the root epidermis (Fig. 6), chlamydospores of the fungus observed within cysts (39), and association of the fungus with various *H. glycines* reproductive structures (8,32,34–36,39). Roy et al. isolated *F. solani* f. sp. *glycines* from cysts from 46 of 56 fields affected by both SDS and *H. glycines* in the Midwest and South; maximum and average isolation frequencies were 28 and 7%, respectively (49,55). The fungus was found to be capa-

Table 1. Inoculum densities of *Fusarium solani* f. sp. *glycines* (CFU/g)^x at different soybean growth stages over 4 years in a field with sudden death syndrome at Pine Tree Station, Colt, Arkansas^y

Growth stage ^z	Year			
	1989	1990	1991	1992
Planting	2,117	2,531	1,425	1,541
Flowering (R2)	1,917	2,684	1,191	1,243
Full seed (R6)	...	2,328	1,378	1,221
Harvest (R8)	4,399	3,518	1,962	1,608
Average	2,811	2,765	1,487	1,403
LSD(0.05) growth stage × year = 623				
LSD(0.05) year = 326				

^x Colony-forming units per gram of soil.

^y J. C. Rupe, unpublished.

^z Growth stages of Fehr et al. (10).

Fig. 5. 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, Arkansas, 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. (From Rupe and Gbur [60]).

ble of surviving in cysts in vitro at approximately 10°C for a period sufficient to overwinter in cysts in the field (55), and isolates from cysts were pathogenic on soybean (55).

The ability of *F. solani* f. sp. *glycines* to overwinter in cysts may influence its inoculum potential and dispersal. Typically, the fungus is found growing alone within individual cysts (55), indicating little competition with other microorganisms for nutrients and space. In such an environment, the probability of its survival in overwintering cysts until spring may be greater than its survival free in soil or in soybean debris where competition with other microorganisms could be a factor. It also is possible that cysts provide a food base for germination of chlamydospores and growth of *F. solani* f. sp. *glycines* at the beginning of and during the soybean growing season (35,55). Recent reports of SDS occurrence in Georgia, Iowa, Kansas,

Ohio, and Wisconsin in the United States, and in Argentina and Brazil, indicate that *F. solani* f. sp. *glycines* may be slowly expanding its range. This expanded range may be, in part, the result of movement of *F. solani* f. sp. *glycines*-colonized cysts into these areas. However, although SDS occurs in Argentina (45), occurrence of *H. glycines* has not been documented there. Local dispersal of cysts colonized with the fungus could be achieved by any factor that moves soil, including man, farm machinery, surface and flood water, and animals. Distant dispersal of cysts could be mediated by migrating birds or by infested soil peds contaminating seed lots (46,67).

The association of high soil moisture with SDS occurrence and severity is a common field observation. Figure 7 shows a greater concentration of SDS-symptomatic plants near a ditch where soil moisture would be higher relative to other portions of the field. In greenhouse (51) and field (39) experiments, the incidence and severity of foliar symptoms were greater and occurred earlier in irrigated than in nonirrigated plants. Scherm and Yang (65) found that SDS foliar symptoms were less severe with decreasing soil moisture (Fig. 8A and B). High soil moisture during the vegetative period appears to have a greater predisposing effect than during postflowering (51,65). Rupe and Gbur (60) observed, however, that too much soil moisture during vegetative growth can reduce or delay SDS symptom development. The association of SDS symptom expression with unseasonably cool temperatures prior to or during flowering and pod set is also a common field observation (21,22; K. W. Roy, J. C. Rupe, D. E. Hershman, and T. S. Abney, *unpublished*). The optimum temperature for growth of *F. solani* f. sp. *glycines* in vitro, 25°C (47), parallels the temperature at which the internal colonization of hypocotyl tissue by

F. solani f. sp. *glycines* is greatest, as determined by lesion size (56). Symptom development is greater when soybean plants inoculated with *F. solani* f. sp. *glycines* are incubated at 25°C rather than at a temperature exceeding 35°C (Fig. 9A and B). Scherm and Yang (65) found that soil temperature differentially influenced the development of foliar symptoms and root symptoms, with foliar symptoms most severe at 22 to 24°C and root symptoms most severe at 15°C (Fig. 10A and B).

The results of Scherm and Yang (65) indicate that high soil moisture and low temperatures during the early part of the growing season, but warmer temperatures during soybean reproduction, are optimal for SDS symptom expression. Rupe and Gbur (60) found that early SDS development was correlated to accumulated temperatures (degree-days) from planting. During this period, rainfall maintained adequate soil moisture for vigorous growth. However, after the initial onset of SDS just after flowering, disease development continued only in years with frequent rainfall.

Yield and Seed Quality Losses

SDS reduces yield, sometimes dramatically, when symptoms are severe early during soybean reproductive development (11,12,16,20–22,37,43,51,62,64,65). The effect of SDS on yield depends on the growth stage of the host at the onset of symptom development and whether the disease progresses rapidly and becomes

Fig. 6. Scanning electron micrograph of *Fusarium solani* f. sp. *glycines* hyphae (arrows) colonizing soybean lateral root surface and associated with *Heterodera glycines* cyst (scale bar = 25 μ m).

Fig. 7. Soybean field in Ridgeway, Illinois illustrating effect of high soil moisture on sudden death syndrome. Diseased plants are concentrated near ditch and in lowest area of field.

Fig. 8. Relationship between soil matrix potential (ψ_m) and foliar disease severity (A) and between the number of days on which average ψ_m was drier than -0.01 MPa and foliar disease severity (B) of sudden death syndrome on potted soybean plants after inoculation with *Fusarium solani*. Red and blue symbols represent results from repeats 1 and 2 of the study, respectively. Each point is the mean of three replicates. (From Scherm and Yang [65])

severe. The likelihood that flower and pod abortion will be severe, with a resultant decrease in seed numbers, is greater if severe symptom development occurs during early reproductive stages prior to pod fill. Severe symptom development when seeds are expanding in the pod usually results in less pod abortion, but seed size may be reduced (19,20,22,62; K. W. Roy, J. C. Rupe, D. E. Hershman, and T. S. Abney, unpublished observations).

Greenhouse (51) and field studies (16,19,43,62,64) and observations (20,22) on the effect of SDS on yield components lead to the conclusion that both reduced seed numbers and size largely account for yield loss to SDS. Reduced seed numbers result from flower, seed, and pod abortion (20,22,62). Reduced seed size is probably due to arrested plant development, premature plant death, impaired capacity of the diseased plant to produce seed dry matter, or combinations of these (26).

Yield loss to SDS often is difficult to demonstrate experimentally, however. In fact, predicted loss based on the perception of severe SDS, particularly of severe aboveground symptoms, is often overestimated, if a statistically significant reduction occurs at all (34,69; K. W. Roy, J. C. Rupe, D. E. Hershman, and T. S. Abney, unpublished). This problem is probably largely due to experimental variability stemming from the interaction of SDS with one or more of the abiotic or biotic variables mentioned previously. Although numerous studies report that SDS reduces yield, few recorded the losses under experimental conditions in which at least some of the interacting factors were controlled. In these studies, experimental con-

ditions ranged from artificial inoculation of potted soybean plants in the greenhouse to natural infection in field plots (11,12,16,20–22,37,43,51,62,64,65). In controlled greenhouse experiments, *F. solani* f. sp. *glycines* reduced seed yield by 77%, and the number of seeds per plant and number of pods per plants were reduced by 75 and 65%, respectively. The reductions in yield and number of seeds occurred in continuously irrigated plants but not in periodically irrigated plants (58). Some of these early findings were later corroborated under controlled experimental field conditions by Scherm and Yang (65). Different levels of soil moisture produced varying degrees of SDS severity, which were reflected in yield losses; at the highest moisture level, yield was reduced by 37%, whereas no significant yield reductions occurred at intermediate or low levels of soil moisture (65). Clearly, soil moisture plays a critical role in the interrelationship between SDS severity and yield loss.

Few studies have actually shown experimentally that *F. solani* f. sp. *glycines* and *H. glycines* in combination exacerbate yield reduction (34,35,37). This and the inconsistent correlations between the nematode and SDS (16,19,34,49,64) indicate that much more research is needed to clarify the role *H. glycines* plays in yield losses to SDS.

Regression analysis has further characterized yield losses and added the element of predictability. Hirrel reported yield loss regression equations for the association of SDS leaf symptom severity (x) and yield (y , in bu/A) for fields in Arkansas and southern Illinois (22). In Arkansas, for maturity group V cultivars, the regression

was $y = 53.2 - 7.97x$, and for group VI cultivars, $y = 64.3 - 19.4x$, representing reductions of about 8 and 19 bu/A, respectively, for each unit increase in disease. In southern Illinois, for group III cultivars rated for SDS severity at growth stage R4 to R5, the equation was $y = 63.4 - 11.1x$, representing a reduction of about 11 bu/A for each unit increase in disease; for group IV cultivars rated at R3 to R4, it was $y = 67.1 - 12.8x$, representing a reduction of about 13 bu/A for each unit increase in disease; and for group V cultivars rated at R2 to R3, it was $y = 9.6 + 20.1x$. A comparison of the latter three equations indicated that disease assessment at R4 to R5 best described yield loss. In preliminary reports, Njiti et al. (43) reported that, for each unit increase in foliar disease severity, yield was reduced by 12 to 22%; and Gibson et al. (12) reported that an increase in 1% of disease incidence resulted in a 0.0 to 0.1 bu/A yield loss in one aspect of the study, and a 0.1 to 0.5 bu/A reduction in another.

It is somewhat surprising that severity of SDS leaf symptoms is incompletely correlated with yield. Some reports indicate no significant correlation (19,68,71), while others report statistically significant correlations ranging from $r = -0.40$ to -0.74 (11,64). More surprising is the lack of any report on the correlation between SDS root rot severity and yield and the inadequacy of our current knowledge of the interrelationship between root rot, leaf symptoms, and yield. Njiti et al. (42) reported significant but low ($r = 0.38$) to modest ($r = 0.61$) correlations, for two field environments, between the percentage of soybean taproots infected with *F. solani* and the severity of SDS foliar symptoms at growth stage

Fig. 9. Soybean plants inoculated with *Fusarium solani* f. sp. *glycines* and incubated at 25°C (A) versus >35°C (B).

Fig. 10. Relationship between soil temperature and foliar disease severity (A) and root disease severity (B) of sudden death syndrome on potted soybean plants after inoculation with *Fusarium solani*. Red and blue symbols represent results from repeat 1 and 2 of the study, respectively. Each point is the mean of three replicates. (From Scherm and Yang [65])

R6. They found even poorer or nonsignificant correlations between the severity of taproot infection, i.e., the percentage of taproot tissue sections yielding *F. solani*, and the severity of leaf symptoms. Rupe (58) found a correlation of $r = -0.90$ for the association of root mass with leaf symptom severity, yet Rupe (57) and Scherm and Yang (65) found no close correlation between root disease severity and foliar disease severity. Thus, questions of how taproot infection is related to severity of root symptoms, how severity of root rot is related to severity of leaf symptoms, and how these relationships influence the yield of SDS-symptomatic plants still remain unanswered. This information is crucial not only in defining and predicting yield loss to SDS but also in establishing criteria for selecting resistant cultivars. Hnetkovsky et al. (23) pointed out that cultivar resistance based on severity of SDS leaf symptoms does not necessarily guarantee protection from yield loss. Gray and Achenbach (14) found that the cultivar Ripley, reportedly resistant to SDS based on leaf symptoms (68), was susceptible to root rot. Njiti et al. (42) alluded to the inadequacy of using leaf symptoms alone in determining SDS severity and showed taproot infection to be an important aspect of SDS that should be considered in assessing disease severity. Their sound recommendation that both root infection and leaf symptoms be considered in assessing SDS severity should be modified to include and emphasize root rot severity.

F. solani f. sp. *glycines* has not been isolated from surface-disinfested seeds of either artificially (39) or naturally (52) infected SDS-symptomatic plants and therefore is presumed to have only an indirect effect on soybean seed quality. Rupe et al. (64) found that SDS decreased seed size and increased the number of dead and hard

seeds in harvested soybeans. They also observed an increased number of abnormal seedlings as SDS severity increased. These effects on seed quality and vigor associated with SDS may be attributable to increases in other diseases, such as pod and stem blight and *Phomopsis* seed decay, caused by the *Diaporthe/Phomopsis* complex (4). Plants killed prematurely by SDS but persisting in the field until normal harvest are, in effect, subjected to delayed harvest. Von Qualen et al. (70) suggested that this may explain why SDS-symptomatic plants have a high incidence of infection by *Diaporthe/Phomopsis*, fungi known to increase in seeds with delayed harvest (4). In other studies, frequency of occurrence of *Phomopsis* seed decay and SDS severity were positively correlated (D. Hershman and K. W. Roy, unpublished), and frequency of *Phomopsis* spp. in seeds was greater for seeds from plants with a high incidence of SDS than from plants with a low incidence (16).

Management of SDS

No single tactic will control SDS; however, the use of multiple management tactics will help minimize losses.

Disease resistance. Proper selection of soybean cultivars resistant to SDS is the cornerstone of an effective management program. Cumulative data from field studies documented that cultivars differ in their reactions to SDS (19,21,22,60,61,68,69), and cultivars having field resistance, e.g., Forrest, Jack, and Ripley, have been identified (42). Field resistance to SDS (which has been characterized as horizontal, rate-reducing [partial] resistance) is inherited multigenically (6,23,41,42) and is highly heritable (41). This in part contrasts with a report by Stephens et al. (68), who found in a greenhouse study that resistance to SDS leaf symptoms in Ripley was com-

plete and conferred by a single gene. However, in the field, this gene (*rps*) confers only partial resistance to SDS (11). Rate-reducing resistance to SDS was found by Njiti et al. (42) to be expressed during the growing season. The taproots of both resistant and susceptible cultivars were infected by the SDS causal fungus early in the growing season, but resistance was manifested later by reductions in the incidence and severity of taproot infection and by a reduction in the disease index (based on leaf symptoms standardized to the R6 growth stage) in resistant compared with susceptible cultivars. They suggested that resistance was due to an extended latent period. Similarly, Rupe and Gbur (60) found that resistance appeared to be expressed in most cultivars as a delay in the onset of disease, in addition to a reduction in the rate of disease progression. Some cultivars appear to have tolerance to SDS, as was implied by the occurrence of high levels of taproot infection without leaf symptom development in Essex and Asgrow 5403 (42). Cultivar reaction to SDS appears to be conditioned to some degree by *H. glycines*. Soybean cultivars with resistance to *H. glycines* are generally less susceptible to SDS than are those lacking resistance (19,22,41,60,61). This is not uniformly true, however (41,61). For instance, Njiti et al. (41) found that a group of soybean lines resistant to races 3 and 14 of *H. glycines* was significantly less susceptible to SDS than an *H. glycines*-susceptible group in three of six field environments. In another field environment, however, lines with resistance to races 3 and 14 of *H. glycines* were significantly more susceptible to SDS, on average, than the lines susceptible to those races and than lines with resistance to race 3 only. It may be possible to achieve concurrent management of SDS and *H. glycines*, since a

Table 2. Effect of planting date and cultivar on soybean sudden death syndrome in McLean County, Kentucky, 1986 to 1988

Cultivar	Maturity group	Area under the disease progress curve ^y						
		1986			1987		1988	
		21 May	16 June	30 June	15 May	22 June	17 May	11 July
Harper	III	42.9 aA ^z	48.0 bA	62.0 bcB	181.7 bB	59.1 bA	557.3 cB	0.0 NS A
Fayette	III	55.3 ab	41.0 bB	0.0 aA	135.9 bB	49.5 abA	395.5 bB	0.0 A
Williams	III	109.9 a-cAB	142.6 bcB	79.6 cA	250.5 cB	88.8 cA	194.4 aB	0.0 A
Pershing	IV	132.9 b-dA	214.9 cdAB	264.9 deB	302.0 cB	53.1 abA	666.5 dB	0.0 A
Mitchell	IV	176.6 cdA	238.1 dA	181.5 dA	465.2 dB	187.3 dA	597.9 cdB	0.0 A
Franklin	IV	77.0 abB	0.0 aA	38.4 aAB	65.2 aA	96.6 cA	130.1 aB	0.0 A
Essex	V	204.5 dA	263.1 dB	291.0 eB	477.5 dB	75.0 bcA	470.7 bcB	0.0 A
Coker 355	V	128.8 b-dA	93.3 bcA	92.3 cA	0.0 aA	0.0 aA	252.7 aB	0.0 A
Cultivar mean		116.0 A	130.6 A	126.2 A	234.8 B	76.2 A	445.6 B	0.0 A

^y R3 to R6 (10) based on a disease index calculated as the product of the scores from disease incidence and disease severity. Disease incidence: 1 = no symptoms, 2 = 1 to 10% plants with symptoms, 3 = 11 to 30%, 4 = 31 to 70%, 5 = >70%; ratings are for cultivars in the full pod (R6) stage. Disease severity: 1 = no symptoms; 2 = <10% chlorotic spotting, no necrosis; 3 = 11 to 30% chlorosis, ≤10% necrosis; 4 = 11 to 30% necrosis with no to light defoliation; 5 = 31 to 70% necrosis with light to moderate defoliation; 6 = >70% necrosis with severe defoliation; ratings are for cultivars in the full pod (R6) stage.

^z Values are averages of four replications (1986) or five replications (1987, 1988). Means in a column followed by a common lowercase letter are not significantly different, Duncan's multiple range test, $P = 0.05$; uppercase letters compare means among planting dates within each dependent variable and year.

genotype resistant to both has been registered (17).

In recent years, much progress has been made in elucidating the nature of inheritance of resistance to SDS (6,23,41,42). This progress was due in part to the use of DNA molecular markers to identify and locate alleles associated with field resistance to SDS, and four quantitative trait loci have been identified (6,23). Owing to the significant cultivar-by-environment interaction (11,19,41,60), the use of multiple field environments for selection is a necessity in conventional breeding. However, because marker-assisted selection is based on genotype rather than on phenotype, it reduces the need for testing in multiple environments and therefore is more efficient and less time-consuming than conventional methods.

Disease escape. Delayed planting generally reduces SDS incidence and severity (19,45,60,70,73). In Kentucky, Hershman et al. (19) found that disease progress was reduced in 2 of 3 years with planting in mid-June through early July, compared with planting in May (Table 2). This was consistent with the rarity of SDS in double-crop soybeans, which are typically planted in late June to early July in Kentucky. Wrather et al. (73) confirmed the finding of Hershman et al. (19), but in their study, reductions in SDS due to delayed planting occurred only in no-till plantings (Table 3). In addition, Wrather et al. (73) found that SDS severity was less in disk-tilled and ridge-tilled plantings of three cultivars planted in mid-May and of two of these cultivars planted in mid-June (Table 3), confirming previous reports (45,70) that tilled soybeans have less SDS than do nontilled soybeans. Results of these studies suggest that the reduction in SDS is probably related to soil temperature and

moisture. The soil is warmer and drier in later plantings than it typically is in earlier plantings (7), and the net effect delays the onset of SDS symptoms (18,19,60). Ridge tilling, which promotes warmer soil and reduces moisture on the soil surface (7), may be helpful (65,73). Also, planting early-maturing cultivars frequently results in SDS development at a later plant reproductive stage, which lowers the risk of serious yield losses (18,19,60). Planting multiple cultivars on a farm-wide basis can increase the probability of some acreage escaping serious SDS, since cultivar reactions vary among locations and years (19,22,61).

Managing *H. glycines*. Because of the earlier appearance and increased severity of SDS foliar symptoms associated with parasitism of plants by *H. glycines* (39,51), reducing *H. glycines* populations by planting nonhost crops or *H. glycines*-resistant cultivars (46), for example, may afford some control of SDS. Rupe et al. (63) reported that rotations with sorghum, fescue, wheat, or an *H. glycines*-resistant cultivar reduced *H. glycines* populations in fields affected by SDS. Presumably, *H. glycines* populations would have to be reduced well below 1,000 eggs and juveniles/100 cm³ of soil to be of any value in SDS management, since this *H. glycines* inoculum level enhanced SDS under greenhouse (51) and field microplot (39) conditions. Information on such *H. glycines* threshold populations is needed.

Other management practices. Although SDS is favored in highly fertile fields with a high yield potential (18,21,22,62), maintenance of the plant nutrient status may delay premature plant death caused by SDS and limit yield losses. This effect was evident during preliminary studies in Indiana, where disease

severity was reduced when stabilized foliar nitrogen was applied to soybeans during a late stage of vegetative growth (T. S. Abney, *unpublished*). In Tennessee (24), the incidence and severity of SDS foliar symptoms and the severity of root rot were reduced with increasing application rates of potassium chloride (KCl) fertilizer at or near planting, although yield enhancements were inconsistent. The use of both foliar nitrogen and KCl in SDS management requires additional research, but results of these initial studies are encouraging.

Von Qualen et al. (70) found that SDS was less severe in a corn-soybean-wheat rotation than in continuous soybeans. Rupe et al. (63) found that rotation of soybean with sorghum or wheat decreased inoculum densities of *F. solani* compared with continuous planting of a susceptible soybean cultivar. Fescue had no effect on inoculum densities (Table 4). Other studies (21,22; D. E. Hershman, *unpublished*) found crop rotation to have little or no effect on SDS.

Because SDS is generally favored by excess soil moisture (22,39,51,65), managing water in the field should help counteract this effect. Diverting surface water to encourage drainage, tilling poorly drained fields to break up compaction and hard pans, and altering crop irrigation practices to provide optimal but not excessive soil moisture are suggested. In addition, because soil moisture is generally greater in no-till than in conventionally tilled fields and perhaps accounts for increased SDS severity and premature plant death (70), an alternative to no-till might be considered for fields having a history of SDS.

In view of the predispositional effect premature plant death or delayed harvesting has on infection of soybean seeds by fungi (4,64,70), timely harvest of fields affected by SDS and the use of foliar fungicides in seed production fields may limit seed quality losses by reducing *Diaporthe/Phomopsis* infection of seeds on plants killed by SDS.

Summary

The potential now exists for substantial yield losses from SDS in all regions of production. Hirrel (22) described severe SDS in 1982, 1984, and 1985 in Arkansas, and Hartman et al. (16) described SDS as occurring in epidemic proportions in east-central Illinois in 1993. There is a pressing need, therefore, to put into place effective management procedures. These practices currently center around the use of resistant cultivars.

Our poor understanding of SDS epidemiology and host-pathogen interactions has hampered efforts to develop cultivars that consistently perform well across multiple environments. Therefore, many new areas of potential research must be addressed before significant gains are likely

Table 3. The effect of tillage, planting date, and cultivar on percentage of soybean leaves^y with symptoms of sudden death syndrome at Portageville, Missouri, averaged over 3 years^z

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

LSD(0.05) same tillage but across planting dates = 7.82

LSD(0.05) across tillage, same or different planting date = 7.29

^y Percentage of leaves with symptoms evaluated at the R6 stage of soybean development (10).

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

Table 4. Inoculum densities of *Fusarium solani* f. sp. *glycines* (CFU/g)^y with different crop rotations^z averaged over 4 years in a field at Pine Tree Station, Colt, Arkansas, with sudden death syndrome and soybean cyst nematode

Rotation crop: cultivar	Inoculum density
Susceptible soybean: Lee 74	2,471 a
Resistant soybean: Hartz 6130	2,108 ab
Sorghum: DMK 54	1,666 c
Fescue: Kentucky 31	2,125 ab
Wheat: Caldwell	2,007 bc

^y Soil densities (CFU/g = colony-forming units/gram of soil) averaged over four samplings (planting, R2, R6, and harvest [R8]) except in 1989, when the R6 (10) samples were not taken.

^z Plots were planted to the susceptible cultivar, Lee 74, in the first and fourth year and to the crops and cultivars listed in the second and third years.

to be made. Perhaps most importantly, we must further explore the nature of SDS resistance in soybean, identify additional sources of genetic resistance, and establish new and better ways to evaluate and incorporate resistance to SDS in high-yielding soybean cultivars. The use of marker-assisted selection and improved greenhouse screening procedures have great potential in the development and release of new cultivars with reliable SDS resistance. Inherent in this effort is the need for better understanding of the relationship between *H. glycines* and *F. solani* f. sp. *glycines*, a clear view of the biology, variability, and pathogenesis of *F. solani* f. sp. *glycines*, and more precise knowledge of SDS epidemiology. One of the main limitations in SDS research is the inconsistency of disease development in both the field and greenhouse. Researchers are faced with high experimental variability, which is a severe restraint on SDS research. Reducing experimental variability will require a better understanding of the infection process and how factors such as soil moisture, temperature, *H. glycines*, and host physiology affect disease development.

Determining the relationship of SDS foliar and root symptoms to yield loss is essential to assessing the true extent of the SDS problem in the United States and elsewhere. This effort will be assisted by the development of SDS yield loss equations and knowledge of pathogen inoculum densities on SDS-induced yield losses. Finally, evaluating the influence of various cultural practices and cropping systems on SDS may provide additional means of effectively managing the disease.

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