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# Evaluation of Perennial *Glycine* Species for Resistance to Soybean Fungal Pathogens That Cause Sclerotinia Stem Rot and Sudden Death Syndrome

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## ABSTRACT

**The cultivated soybean [*Glycine max* (L.) Merr.] has a relatively narrow genetic base and most commercial cultivars are susceptible to *Sclerotinia sclerotiorum* (Lib.) de Bary and *Fusarium solani* (Mart.) Sacc. f. sp. *glycines*, which, respectively cause Sclerotinia stem rot (SSR) and sudden death syndrome (SDS). The objective of this study was to screen all the available accessions of the perennial *Glycine* species for resistance to the pathogens that cause SSR and SDS. For SSR evaluations, five seedlings of each of 787 accessions were screened once in a series of eight non-replicated runs. Seedlings were inoculated with an agar plug cut from the edge of a 1-d-old fungal culture by placing the plug next to the stem. Of the 787 accessions, 183 had partial resistance with 144 of these accessions being *G. tabacina* (Labill.) Benth. A selected set of 53 accessions was further screened in two replicated trials with five plants per each of four replications. *Glycine tabacina* had several accessions that were consistently rated as partially resistant. For SDS evaluations, five plants of each of 767 accessions were screened once in a series of eight runs. Plants were inoculated by a layered technique in which infested sorghum seed were placed below the transplanted seedlings. In the initial evaluation of 767 accessions, 134 had partial resistance with 65 of these accessions being *G. tomentella* Hayata. In a replicated set of selected accessions, *G. tomentella* had several accessions that were consistently rated as partially resistant. These perennial *Glycine* species represent potential untapped sources for improving disease resistance in soybean.**

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THE GENUS *Glycine* Willd. is composed of two subgenera, *Glycine* and *Soja* (Moench) F. J. Herm. The cultivated soybean and its wild annual progenitor *Glycine soja* Sieb. and Zucc. belong to the subgenus *Soja*. Both species are diploid ($2n = 40$) and are cross compatible. The subgenus *Glycine* contains 16 wild perennial species. They are indigenous to Australia and grow in diverse geographical areas under a wide range of climatic conditions. These species are diploid ($2n = 40$), with aneuploidy ($2n = 38$ and 78) and tetraploidy ($2n = 80$) occurring in *G. tomentella*, *G. tabacina*, and *G. hirticaulis* Tind. and Craven (Tindale and Craven, 1993; Kollipara et al., 1997; Singh et al., 1998). Genomic symbols have been assigned to each species on the basis of cytogenetic, biochemical, and molecular studies (Kollipara et al., 1997).

Useful traits have been identified from accessions of at least some of the perennial *Glycine* species. Some species carry resistance to soybean pathogens like *Heterodera glycines* Ichinohe (Riggs et al., 1998), *Microsphaera diffuse* Cke. & Pk. (Mignucci and Chamberlain, 1978), *Phakopsora pachyrhizi* H. Sydow & Sydow (Hartman et al., 1992; Schoen et al., 1992), *Phytophthora soja* Kaufmann & Gerdemann (Kenworthy, 1989), *Septoria glycines* Hemmi (Lim and Hymowitz, 1987), and yellow mosaic virus (Horlock et al., 1997).

*Sclerotinia sclerotiorum* on soybean is referred to as

**Abbreviations:** SDS, sudden death syndrome; SSR, Sclerotinia stem rot.

Sclerotinia stem rot (SSR) or as white mold on soybean and other crops. The disease is one of the most important on soybean grown in the north central states of the USA and in regions of southern Ontario, Argentina, and Brazil (Grau and Hartman, 1999; Hartman et al., 1998). In 1994, SSR was ranked second to soybean cyst nematode in yield-reducing diseases in the USA (Wrather et al., 1997). The importance of finding better sources of resistance in soybean was recently demonstrated when all *G. max* accessions in the USDA-ARS collection from maturity group III and earlier were screened for *S. sclerotiorum* resistance. Although none of these accessions are known to be immune, a few of them have good resistance to SSR and are being reevaluated in a number of field locations (B. Diers, 1999, personal communication).

Sudden death syndrome (SDS) of soybean, caused by the soilborne fungus *Fusarium solani* (Mart.) Sacc. f. sp. *glycines* (Roy et al., 1989; Rupe, 1989; Roy, 1997), has been reported from Argentina, Brazil, and the USA (Rupe and Hartman, 1999). Disease symptoms include mottling, interveinal chlorosis, and necrosis on the upper leaves at flowering and also root rot, crown rot, vascular discoloration of stems, defoliation, and pod abortion (Roy et al., 1989; Rupe, 1989). Disease is most severe in wet conditions and decreases as soil moisture decreases. Soybean cultivars and lines that exhibit various levels or resistance to *F. solani* f. sp. *glycines* have been reported on the basis of tests conducted in the growth chamber, greenhouse, microplots, and field. Resistance to *F. solani* f. sp. *glycines* was reported to be conditioned by a dominant gene (*Rfs*) in cultivar Ripley (Stevens et al., 1993) and quantitatively in cv. Forrest (Hnetkovsky et al., 1996). A number of accessions in the soybean germplasm have been screened for resistance with several new sources with at least partial resistance being reported (Hartman et al., 1997).

The cultivated soybean, having a relatively narrow genetic base, may have insufficient levels of disease resistance. Other germplasm, including the wild perennial *Glycine* species that has more genetic diversity than cultivated soybean, may contribute to increasing disease resistance levels through interspecific crosses. The objective of this study was to screen all the available accessions of the perennial *Glycine* species for resistance to the pathogens that cause SSR and SDS.

## MATERIALS AND METHODS

Seeds of the perennial *Glycine* species were scarified by scoring seed coats on the opposite side of the hilum with a razor blade. Seeds were then germinated on moist filter paper inside petri dishes under light at 25°C for 4 to 6 d. Germinated seedlings were planted in a 1:1 steam-sterilized soil:sand mix (1:1 v/v) at pH 7. The soil was a Drummer silt loam. All plants were grown in greenhouses under a photoperiod of 16 h of daylight at 24 ± 3°C/18 ± 3°C day/night temperature. For all evaluations, transplanted seedlings were grown for 2 to 3 wk before being inoculated. Seeds of the soybean checks were sown when the perennial accessions were transplanted. The seedlings were grown in a greenhouse and watered daily for 10 to 14 d before inoculating.

For SSR evaluations, five seedlings of each of the 787 accessions (Table 1) were screened once in a series of eight non-replicated runs that averaged 98 accessions per run. Cultivar Williams 82 was used as a susceptible check in each run. A selected set of 53 accessions (Screen 2), which were based on the results of the non-replicated runs with some accession representing most species, was further screened in two replicated trials with five plants in each of four replications. Soybean cultivars Asgrow A2242 (susceptible) and NK S19-90 (partially resistant) were used as checks (Hartman, 1997). A final selected set of 12 accessions (Screen 3A and 3B), which were based on those 53 accessions that preformed well within some of the species, was screened in two trials with three replications each.

An isolate of *S. sclerotiorum* originating from infected

**Table 1. List of the species in the genus *Glycine* Willd., ploidy level, genome symbol, and the number of accessions in each rating category based on percentage of plant survival for plants inoculated with *Sclerotinia sclerotiorum* (Sclerotinia stem rot, SSR) and disease severity rating for plants inoculated with *Fusarium solani* f. sp. *glycines* (sudden death syndrome, SDS).**

| Species | 2n | Genome† | No. of accessions | SSR survival (%) | | | | No. of accesssions | SDS severity | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | 75–100 | 51–75 | 26–50 | 0–25 | | 1–2 | 2–3 | 3–4 | 4–5 |
| *G. arenaria* Tind. | 40 | HH | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 |
| *G. argyrea* Tind. | 40 | $A_2A_2$ | 3 | 0 | 0 | 1 | 2 | 3 | 2 | 1 | 0 | 0 |
| *G. canescens* F.J. Herm. | 40 | AA | 75 | 5 | 4 | 9 | 57 | 75 | 18 | 28 | 21 | 8 |
| *G. clandestina* Wendl. | 40 | $A_1A_1$ | 107 | 6 | 12 | 30 | 60 | 102 | 25 | 33 | 23 | 21 |
| *G. curvata* Tind. | 40 | $C_1C_1$ | 4 | 0 | 0 | 0 | 4 | 3 | 2 | 1 | 0 | 0 |
| *G. cyrtoloba* Tind. | 40 | CC | 25 | 0 | 4 | 2 | 19 | 23 | 12 | 6 | 4 | 1 |
| *G. falcata* Benth. | 40 | FF | 13 | 3 | 1 | 1 | 8 | 11 | 1 | 3 | 2 | 5 |
| *G. latifolia* (Benth). Newell & Hymowitz | 40 | $B_1B_1$ | 42 | 16 | 10 | 11 | 5 | 37 | 3 | 6 | 14 | 14 |
| *G. latrobeana* (Meissn.) Benth. | 40 | $A_3A_3$ | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| *G. microphylla* (Benth.) Tind. | 40 | BB | 31 | 8 | 11 | 5 | 7 | 31 | 0 | 13 | 18 | 0 |
| *G. pindanica* Tind. & Craven | 40 | $H_2H_2$ | 2 | 0 | 0 | 2 | 0 | 2 | 0 | 1 | 1 | 0 |
| *G. tabacina* (Labill.) Benth. | 40 | $B_2B_2$ | 13 | 9 | 1 | 3 | 0 | 13 | 0 | 5 | 4 | 4 |
| *G. tabacina* | 80 | Complex‡ | 132 | 79 | 36 | 6 | 11 | 132 | 1 | 36 | 47 | 48 |
| *G. tabacina* | ? | | 87 | 56 | 17 | 6 | 8 | 87 | 5 | 15 | 25 | 42 |
| *G. tomentella* Hayata | 38 | EE | 22 | 0 | 1 | 0 | 21 | 22 | 3 | 4 | 2 | 13 |
| *G. tomentella* | 40 | DD | 47 | 0 | 1 | 0 | 46 | 44 | 9 | 12 | 12 | 11 |
| *G. tomentella* | 78 | Complex§ | 56 | 0 | 0 | 4 | 52 | 55 | 18 | 17 | 11 | 9 |
| *G. tomentella* | 80 | Complex§ | 53 | 1 | 4 | 5 | 43 | 52 | 17 | 14 | 13 | 8 |
| *G. tomentella* | ? | Complex§ | 73 | 0 | 0 | 6 | 67 | 73 | 18 | 29 | 11 | 15 |
| Total | | | 787 | 183 | 103 | 91 | 411 | 767 | 134 | 225 | 208 | 200 |

† Genomically similar species carry the same letter symbols (after Kollipara et al., 1997).
‡ Allopolyploids (A and B genome) and segmental allopolyploids (B genome).
§ Allopolyploids (A and D genomes, D and E, A and E, or any other unknown combination).

plants from a field near Dekalb, IL, in 1994 was maintained in the dark at 4°C on potato dextrose agar. Mycelial plugs were transferred to acidified potato dextrose agar (APDA) and after 1 d, this culture was used as starter inoculum by transferring 3-mm-diam plugs from the margin of the colony to new APDA plates. After 1 to 2 d, 3-mm-diam plugs from the colony edges were used to inoculate plants. To inoculate the perennial plants, a single plug, mycelial-side down, was placed on the newest leaf petiole touching the main stem or in the plant whorl. For soybean plants, a single mycelial plug was placed mycelial-side down on a cotyledon approximately 2 mm from the stem of each seedling. Following inoculation with the plug method, all seedlings were lightly misted with water from a hand atomizer to increase humidity and then covered with a plastic dome that fit over individual flats. The dome-covered flats were placed about 1 m under black mesh shade cloth (80% light reduction) to prevent heat build up inside the domes. After 2 d, the domes and shade cloth were removed. The number of total seedlings that survived was counted daily usually beginning with the day the dome was removed until plants stopped dying, which was 4 to 5 d after the first rating. For the last set, the area under the survival curve (AUSC) was calculated on the basis of four ratings recorded daily (Shaner and Finney, 1977).

For SDS evaluations, five plants of each of the 767 accessions (Table 1) were screened once in a series of eight runs. The cultivar Spencer was used as a susceptible check in each run. A selected set of 67 accessions (Screen 2), which were based on the results of the non-replicated runs with some accession representing most species, was further screened in two replicated trials with five plants per each of four replications. A final selected set of 15 accessions, which were based on those 67 accessions that preformed well within some of the species, was screened in two trials with three replications each. Seeds of the soybean checks [Spencer (susceptible), PI520 733 (partially resistant)] were sown when the perennial accessions were transplanted.

A *F. solani* f. sp. glycines isolate, Mont-1 (Hartman et al., 1997), was subcultured on 2% (w/v) water agar for 3 wk before infesting sorghum seed. Two hundred cubic centimeters of sorghum seed were soaked in distilled water in a 1-L Erlenmeyer flask overnight, drained, and autoclaved on two consecutive days for 20 min at 121°C. Ten mycelial plugs (4-mm diameter) were transferred from the water agar colonies. Cultures on sorghum grains were incubated under a 12 h-photoperiod with fluorescent light (60 $\mu$mol $m^{-2}$ $s^{-1}$) for 10 to 14 d at 25°C.

Nine entries of five seedlings or seeds were sown in 20- by 30-cm trays. Soil was infested by placing colonized sorghum seed below transplanted *Glycine* spp. and beneath soybean seeds at planting (Hartman et al., 1997). A template was used to make three furrows that were 30 cm long, 2 cm deep, and 3 cm apart. Five cubic centimeters of infested sorghum seed was evenly distributed in each furrow about 800 $cm^3$ of soil mix was added to cover the infested seed to a depth of 2 cm. Transplanted seedlings or seeds were added to each furrow after reapplying the template to make a 2-cm-deep furrow directly over the infested sorghum seeds. Trays were placed in a greenhouse at 20 to 30°C and watered daily. Disease severity was rated for each plant 21 d after planting based on a scale of 1 to 5: 1 = no symptoms, 2 = light symptom development with slight mottling and mosaic (1–20% foliage affected), 3 = moderate symptom development with interveinal chlorosis and necrosis (21–50% foliage affected), 4 = heavy symptom development with interveinal chlorosis and necrosis (51–80% foliage affected), and 5 = severe symptom development with interveinal chlorosis and necrosis and/or dead plants (81–100% foliage affected). For the last set, the area under the disease progress curve (AUDPC) was calculated on the basis of four ratings recorded 2 to 4 wk after inoculating (Shaner and Finney, 1977).

A correlation coefficient was caculated to compare ratings for the initial nonreplicated evaluations of SSR and SDS. The data for Screens 3A and 3B were analyzed by analysis of variance (SAS Institute, Cary, NC). Means were compared by least significant differences at $P = 0.05$.

## RESULTS AND DISCUSSION

The average survival of plants representing the 787 accessions screened for resistance to *S. sclerotiorum* was 39% whereas the survival of Williams was 34% (Table 1). Approximately 25% of the accessions were considered to have partial resistance (survival > 75%). Plants representing accessions of *G. tabacina* had the highest survival percentage (84%) followed by *G. latifolia* (Benth.) newell & Hymowitz (71%), and *G. microphylla*

**Table 2. Plant survival percentage and area under the survival curve (AUSC) of perennial *Glycine* species inoculated with *Sclerotinia sclerotiorum* (Sclerotinia stem rot).**

| | | | | Survival (%)§ | | AUSC¶ | |
|---|---|---|---|---|---|---|---|
| Species | Entry† | 2*n* | Genome‡ | Screen-1 | Screen-2 | Screen-3A | Screen-3A |
| *canescens* | 690 | 40 | AA | 100 | 63 | 105 | 94 |
| *clandestina* | 895 | ? | $A_1A_1$ | 100 | 82 | 170 | 111 |
| *latififolia* | 916 | 40 | $B_1B_1$ | 100 | 27 | 60 | 107 |
| *microphylla* | 307 | 40 | BB | 100 | 78 | 178 | 176 |
| *microphylla* | 1070 | 40 | BB | 100 | 75 | 302 | 182 |
| *tabacina* | 322 | 80 | BBBB# | 100 | 83 | 253 | 304 |
| *tabacina* | 456 | 40 | $B_2B_2$ | 100 | 92 | 360 | 338 |
| *tabacina* | 460 | 40 | $B_2B_2$ | 100 | 82 | 355 | 199 |
| *tabacina* | 509 | 80 | BBBB# | 100 | 95 | 338 | 201 |
| *tabacina* | 726 | 80 | $AAB_2B_2$ | 100 | 83 | 223 | 197 |
| *tabacina* | 1084 | 80 | $AAB_2B_2$ | 100 | 89 | 375 | 251 |
| *tomentella* | 782 | 40 | DD | 75 | 59 | 83 | 83 |
| *max* | A2242 | 40 | GG | – | 29 | 115 | 151 |
| *max* | S19-90 | 40 | GG | – | 100 | 377 | 324 |
| LSD | | | | | | 112 | 108 |

† Entry is either the University of Illinois accession number of the wild perennial *Glycine* spp. or is designation of soybean cultivar.
‡ Genomically similar species carry the same letter symbols.
§ Percentage survival based on five plants per accession in the initial screen of 787 accessions. For the second screen, mean percentage survival of those accessions is based on data of two trials with 52 accessions of four replications with five plants per replication.
¶ Data for the two trials are based on each of three and four replications for Trials 1 and 2 respectively, with five plants per replication.
# Segmental allopolyploids (B genome).

**Table 3. Sudden death syndrome (SDS) severity and area under disease progress curve (AUDPC) of perennial *Glycine* species inoculated with *Fusarium solani* f. sp. *glycines*.**

| Species | Entry† | 2*n* | Genome‡ | Severity§ | | AUDPC¶ | |
|---|---|---|---|---|---|---|---|
| | | | | Screen-1 | Screen-2 | Screen-3A | Screen-3B |
| *cyrtoloba* | 783 | 40 | CC | 3.3 | 2.7 | 21 | 17 |
| *microphylla* | 652 | 40 | BB | 1.8 | 2.9 | 19 | 19 |
| *microphylla* | 778 | 40 | BB | 2.3 | 1.5 | 18 | 17 |
| *tabacina* | 349 | 40 | $B_2B_2$ | 2.5 | 2.7 | 27 | 24 |
| *tabacina* | 905 | 80 | Complex | 2.0 | 3.0 | 25 | – |
| *tomentella* | 353 | 80 | AD | 1.3 | 2.7 | 17 | – |
| *tomentella* | 632 | 78 | DE | 1.0 | 1.5 | 16 | 21 |
| *tomentella* | 653 | 78 | DE | 1.0 | 1.8 | 22 | 20 |
| *tomentella* | 694 | 78 | DE | 1.0 | 1.4 | 21 | 17 |
| *tomentella* | 707 | 78 | DE | 1.0 | 1.8 | 18 | 18 |
| *tomentella* | 722 | 78 | DE | 1.0 | 1.6 | 18 | 19 |
| *tomentella* | 773 | 38 | EE | 1.0 | 1.2 | 18 | 14 |
| *tomentella* | 871 | 80 | AD | 1.0 | 1.3 | 17 | 14 |
| *tomentella* | 885 | 80 | AD | 1.3 | 1.5 | 16 | 16 |
| *tmentella* | 1232 | 38 | EE | 1.5 | 1.3 | 18 | 18 |
| *max* | Spencer | 40 | GG | 4.0 | 3.7 | 26 | 28 |
| *max* | PI523 733 | 40 | GG | – | 2.0 | 19 | 13 |
| LSD | | | | | | 5.9 | 6.9 |

† Entry is either the University of Illinois accession number of the wild perennial *Glycine* spp. or is cultivar or plant introduction of soybean.
‡ Genomically similar species carry the same letter symbols.
§ Percentage severity based on five plants per accession in the initial screen of 767 accessions. For screen-2, mean disease severity of 67 accessions that had lower disease severity ratings from screen-1 within a species. Data for those two trials were based on four replications for trials 1 and 2, respectively, with five plants per replication.
¶ Data for the two trials are based on each of three and four replications for trials 1 and 2, respectively, with five plants per replication.

(Benth.) Tind. (64%); *G. tomentella* had one of the lowest survival percentages (9%). Plant survival greater than 75% was not found in *G. arenaria* Tind., *G. argyrea* Tind., *G. curvata* Tind., *G. cyrtoloba* Tind., *G. latrobeana* (Meissn.) Benth., and *G. pindanica* Tind. & Craven (Table 1). In the second screen, all selected *G. tabacina* accessions for the third screen had greater than 82% survival (Table 2). In the third screen, four of the *G. tabacina* accessions had ratings that did not differ from the partially resistant soybean check (S19-90), while the rest of the accessions had ratings that did not differ from the susceptible check (A2242) (Table 2).

On the basis of the intitial SSR screen, accessions with the B genome (*G. latifolia*, *G. microphylla*, and *G. tabacina*) had better survival ratings along with some accessions with the A genome (*G. canescens* F.J. Herm. and *G. clandestine* Wendl.) and the F genome (*G. falcata* Benth.) (Table 1). Resistance within the *G. tabacina* accessions was not associated with distribution within a specific region or country of origin. In addition, within the polyploid *G. tabacina*, resistance to *S. sclerotiorum* was approximately equal in those accessions lacking adventitious roots (genome $AAB_2B_2$) and those accessions having adventitious roots (genome BBBB) (Costanza and Hymowitz, 1987).

The average disease severity ratings of plants representing the 767 accessions screened for resistance to *F. solani* f. sp. *glycines* was 3.1, whereas the rating of Spencer was 3.8 (Table 1). Plants representing accessions of *G. argyrea* had the lowest disease severity ratings (1.7) followed by *G. curvata* (1.8), and *G. cyrtoloba* (2.1); *G. tabacina* had the highest disease severity ratings (3.6). Disease severity ratings of less than 2 were not found in *G. arenaria*, *G. latrobeana*, *G. microphylla*, and *G. pindanica* (Table 1). In the second screen, all but five of the selected perennial accessions for the third screen had lower disease severity ratings than the partially resistant check PI 520 733 (Table 3). In the third screen, most of the *G. tomentella* accessions had disease ratings that did not differ from the partially resistant check (Table 3).

There was a significant ($P < 0.0001$) positive correlation ($r = 0.3$, $n = 765$) between SSR survival ratings and SDS disease ratings. In general, the species that had higher survival ratings after inoculating with *S. sclerotiorum* were more susceptible to *F. solani* f. sp. *glycines* and vice versa. It is not known why there are these differences in stem versus root resistance, but it is very likely that the mechanism of resistance between the two are different. In addition, it is not known how these wild perennial *Glycine* species would perform under epidemic conditions in the field. Although field evaluations are important, the lack of adaptation in these perennial *Glycine* species to field conditions would make it difficult to evaluate this unique collection in the field.

Novel sources of resistance were identified in accessions of the perennial *Glycine* species. Accessions belonging to *G. tabacina*, *G. tomentella*, and *G. clandestina* represented the majority of accessions screened. Of the 16 perennial *Glycine* species, *G. albicans* Tind. & Craven, *G. hirticaulis* Tind. & Craven, and *G. lactovirens* Tind. & Craven, were not evaluated because these species are recalcitrant regarding multiplication under greenhouse conditions at Urbana, IL.

A number of useful traits have been identified from accessions of at least some of the perennial *Glycine* species. Sources of resistance have been reported for some soybean fungal pathogens (Lim and Hymowitz, 1987; Kenworthy, 1989; Mignucci and Chamberlain, 1978; Hartman et al., 1992; Schoen et al., 1992), nematodes (Riggs et al., 1998), and viruses (Horlock et al., 1997). Because of the complexity of the different ecological niches that these perennials occupy in their natural habitat, it is likely that a multitude of useful traits includ-

ing disease resistance will be found in these perennial *Glycine* species. Singh et al. (1990, 1993, 1998) produced fertile backcrossed progeny from a soybean × *G. tomentella* hybrid. This success sets the stage whereby useful germplasm within the wild perennial *Glycine* species can be utilized by soybean breeders.

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## REFERENCES

Costanza, S.H., and T. Hymowitz. 1987. Adventitious roots in *Glycine* subgenus *Glycine*: Morphological and taxonomic indicators of the "B" genome. Plant Syst. and Evol. 158:37–46.

Grau, C.R., and G.L. Hartman. 1999. Sclerotinia stem rot. p. 46–48. *In* G.L. Hartman et al. (ed.) Compendium of Soybean Diseases. APS Press, St. Paul, MN.

Hartman, G.L. 1997. Screening soybeans for resistance to Sclerotinia stem rot. p. 1–12. *In* Proc. 27th Soybean Seed Research Conference 1997, Chicago, IL. American Seed Trade Association, Washington, DC.

Hartman, G.L., Y.H. Huang, R.L. Nelson, and G.R. Noel. 1997. Germplasm evaluation of *Glycine max* for resistance to *Fusarium solani*, the causal organism of sudden death syndrome. Plant Dis. 81: 515–518.

Hartman, G.L., L. Kull, and Y.H. Huang. 1998. Occurrence of *Sclerotinia sclerotiorum* in soybean fields in east-central Illinois and enumeration of inocula in soybean seed lots. Plant Dis. 82:560–564.

Hartman, G.L., T.C. Wang, and T. Hymowitz. 1992. Sources of resistance to soybean rust in perennial *Glycine* species. Plant Dis. 76: 396–399.

Hnetkovsky, N., S.J.C. Chang, T.W. Doubler, P.T. Gibson, and D.A. Lightfoot. 1996. Genetic mapping of loci underlying field resistance to soybean sudden death syndrome (SDS). Crop Sci. 36: 393–400.

Horlock, C.M., D.S. Teakle, and R.M. Jones. 1997. Natural infection of the native pasture legume, *Glycine latifolia*, by alfalfa mosaic virus in Queensland. Australasian Plant Path. 26:115–116.

Kenworthy, W.J. 1989. Potential genetic contributions of wild relatives to soybean improvement. p. 883–888. *In* A.J. Pascale (ed.) World Research Conference IV. Proc., Buenos Aires. 5–9 Mar. 1989. Argentina. Assoc. Soja, Buenos Aires.

Kollipara, K.P., R.J. Singh, and T. Hymowitz. 1997. Phylogenetic and genomic relationships in the genus *Glycine* Willd. based on sequences from the ITS region of nuclear DNA. Genome 40:57–68.

Lim, S.M., and T. Hymowitz. 1987. Reactions of perennial wild species of genus *Glycine* to *Septoria glycines*. Plant Dis. 71:891–893.

Mignucci, J.S., and D. W. Chamberlain. 1978. Interactions of *Microsphaera diffusa* with soybeans and other legumes. Phytopathology 68:169–173.

Riggs, R.D., S. Wang, R.J. Singh, and T. Hymowitz. 1998. Possible transfer of resistance to *Heterodera glycines* from *Glycine tomentella* to soybean. Supp. J. Nematology 30(4S):547–552.

Roy, K.W. 1997. *Fusarium solani* on soybean roots: Nomenclature of the causal agent of sudden death syndrome and identity and relevance of *F. solani* form B. Plant Dis 81:259–266.

Roy, K.W., G.W. Lawrence, H.H. Hodges, K.S. McLean, and J.F. Killebrew. 1989. Sudden death syndrome of soybean: *Fusarium solani* as incitant and relation of *Heterodera glycines* to disease severity. Phytopathology 79:191–197.

Rupe, J.C. 1989. Frequency and pathogenicity of *Fusarium solani* recovered from soybeans with sudden death syndrome. Plant Dis. 73:581–584.

Rupe, J.C., and G.L. Hartman. 1999. Sudden death syndrome. p. 37–39. *In* G.L. Hartman et al. (ed.) Compendium of Soybean Diseases. APS Press, St. Paul, MN.

SAS Institute. 1992. SAS/STAT Guide for Personal Computers. 6th ed. SAS Institute, Cary, NC.

Schoen, D.J., J.J. Burdon, and A.H.D. Brown. 1992. Resistance of *Glycine tomentella* to soybean leaf rust *Phakopsora pachyrhizi* in relation to ploidy level and geographical distribution. Theor. Appl. Genet. 83:827–832.

Shaner, G., and R.E. Finney. 1977. The effect of nitrogen fertilization on the expression of slow mildewing resistance in Knox wheat. Phytopathology 67:1051–1056.

Singh, R.J., K.P. Kollipara, and T. Hymowitz. 1990. Backcross-derived progeny from soybean and *Glycine tomentella* Hayata intersubgeneric hybrids. Crop Sci. 30:871–874.

Singh, R.J., K.P. Kollipara, and T. Hymowitz. 1993. Backcross (BC2-BC4)-derived fertile plants from *Glycine max* and *G. tomentella* intersubgeneric hybrids. Crop Sci. 33:1002–1007.

Singh, R.J., K.P. Kollipara, and T. Hymowitz. 1998. Monosomic alien addition lines derived from *Glycine max* (L.) Merr. and *G. tomentella* Hayata: Production, characterization, and breeding behavior. Crop Sci. 38:1483–1489.

Stephens, P.A., C.D. Nickell, and F.L. Kolb. 1993. Genetic analysis of resistance to *Fusarium solani* in soybean. Crop Sci. 33:929–930.

Tindale, M.D., and L.A. Craven. 1993. *Glycine pindanica* (Fabaceae: Phaseolae), a new species from West Kimberley, Western Australia. Aust. Syst. Bot. 6:371–376.

Wrather, J.A., T.R. Anderson, D.M. Arsyad, J. Gai, L.D. Ploper, A. Porta-Puglia, H.H. Ram, and J.T. Yorinori. 1997. Soybean disease loss estimates for the top 10 soybean producing countries in 1994. Plant Dis. 81:107–110.