Resistance of Soybean Cultivars to Sclerotinia sclerotiorum

C. R. GRAU and V. L. RADKE, Department of Plant Pathology, University of Wisconsin, Madison 53706, and F. L. GILLESPIE, Agricultural Agent, University of Wisconsin Extension, Sturtevant 53177

ABSTRACT

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Twenty-three soybean (Glycine max) cultivars were evaluated for their resistance to Sclerotinia sclerotiorum in naturally infested field plots. The cultivars Corsoy, Hodgson, and Hodgson 78 were less susceptible to S. sclerotiorum than other cultivars. Resistance or susceptibility to S. sclerotiorum was not related to plant architecture or soybean maturity group.

Additional key words: Sclerotinia stem rot

Sclerotinia stem rot of soybean (Glycine max (L.) Merr.), caused by Sclerotinia sclerotiorum (Lib.) de Bary (= Whetzelinia sclerotiorum (Lib.) Korf & Dumont), is generally considered to be a minor disease of soybean in Wisconsin. Localized outbreaks, however, have been observed in central and southeastern Wisconsin in fields previously cropped to susceptible crops such as green beans (Phaseolus vulgaris L.), cabbage (Brassica oleracea L.), or sunflowers (Helianthus annuus L.). Hine and Wheeler (11) also observed a high incidence of Sclerotinia stem rot when soybeans followed a crop of lettuce (Lactuca sativa L.) that was previously destroyed by S. sclerotiorum. Sclerotinia stem rot has also been reported in Illinois (5), Ontario (10), and South Africa (15), but prior cropping history was not associated with disease prevalence and severity.

S. sclerotiorum has long been a difficult pathogen to control. Most control strategies have involved crop rotation or the use of fungicides, but not the planting of resistant cultivars. Resistance to S. sclerotiorum has been reported in P. coccineus L. (3) and P. vulgaris (4,7), and an earlier report indicates that soybean cultivars differ in susceptibility to S. sclerotiorum (9).

This report provides more evidence that resistance to S. sclerotiorum exists in soybean and that breeding for resistance to this pathogen should be possible. Symptoms of Sclerotinia stem rot are also described and associated with growth stages of soybean (8).

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MATERIALS AND METHODS

Soybean cultivars were evaluated for their resistance to S. sclerotiorum in naturally infested field plots located in Racine County, WI, in 1978–1980. All plots were plowed in the fall and disked in the spring for seedbed preparation. Soybeans were hand-planted with a V-belt planter and seeded at a rate of 20 seeds per meter of row. Trifluralin $(\alpha, \alpha, \alpha$ -trifluoro-2, 6-dinitro-N, N-dipropyl-ptoluidine) was incorporated into the soil before planting at the rate of 0.84 kg a.i./ha in 1978 and 1980, and plots were hand-weeded each year.

In 1978, eight cultivars were planted on 16 May in plots consisting of four rows spaced 38 cm apart and 6.1 m in length. Each cultivar was replicated five times in a randomized complete block design. In 1979, 18 cultivars were planted on 17 May in individual plots consisting of five rows spaced 25 cm apart and 6.1 m long. Each cultivar was replicated four times in a randomized complete block design. In 1980, nine soybean cultivars were planted on 22 May in plots consisting of four rows spaced 38 cm apart and 6.1 m long. Each cultivar was replicated three times in a randomized complete block design.

A disease severity index based on symptoms observed and a disease severity formula as described by Sherwood and Hagedorn (13) was used to rate soybean cultivars for their resistance to S. sclerotiorum. The following disease classes were established: 0 = nosymptoms; 1 = only lateral branches showing lesions; 2 = lesions on the main stem, but little or no effect on pod-fill; and 3 = lesions on main stem resulting in plant death and poor pod-fill. The disease severity index (DSI) ranged from 0 (no disease) to 100 (all plants killed and with poor pod-fill) and was calculated for each cultivar by using the following formula:

DSI = $\Sigma \frac{\text{class} \times \text{no. of plants in class} \times 100}{\text{total no. of plants} \times 3}$

Disease severity readings over the three

crop seasons were taken between 1 and 9 September, when plants had pods that contained full-sized, green seeds in the uppermost nodes (R-6 growth stage). Disease severity indexes for each cultivar were determined by rating 50 consecutive plants in two rows of each replicate.

RESULTS

Disease development. Symptoms and signs of Sclerotinia stem rot were not observed until the late flowering stage (R-2), and disease development was generally minimal until the beginning of pod development (R-3 to R-5). Symptoms were most frequently observed on the main stem 15-40 cm above the soil surface. Lateral branches were less frequently diseased, and symptoms were rarely observed on the main stem at the soil line. Stem lesions originated at leaf axils and advanced up and down the stem. Lesions were initially gray but later appeared tan and water-soaked (Figs. 1A) and B).

Symptoms on lateral branches also originated at nodes. White, fluffy mycelium developed on infected tissue, and black sclerotia developed externally among the white mycelium (Figs. 1C and D) and internally in the stem pith. Pod infection without infection of the main stem was occasionally observed. Infection of the main stem resulted in wilt and eventual necrosis of stem and foliar tissue above the infection site, and pod development was generally poor.

Disease severity indexes. The disease severity indexes ranged from 10 to 85 (Table 1). The relative ranking of all cultivars was similar for each year of evaluation, although the actual indexes differed for each year. The indexes for Corsoy, Hodgson, and Hodgson 78 differed significantly from those of all other cultivars evaluated in 1978. Corsoy, Hodgson, and Hodgson 78 also had low disease severity indexes in 1979, but they were not significantly different from the cultivars Vickery and Amsoy 71, as they were in 1978.

Several additional cultivar comparisons are of interest. Although Corsoy is a recurrent parent of Vickery and Corsoy 79, the latter two cultivars had higher disease severity indexes in 1978 and 1979, respectively. Gnome, a determinate dwarf cultivar, had the highest DSI for each year of evaluation. Disease severity indexes were not associated with plant architecture or relative maturity of the cultivars.

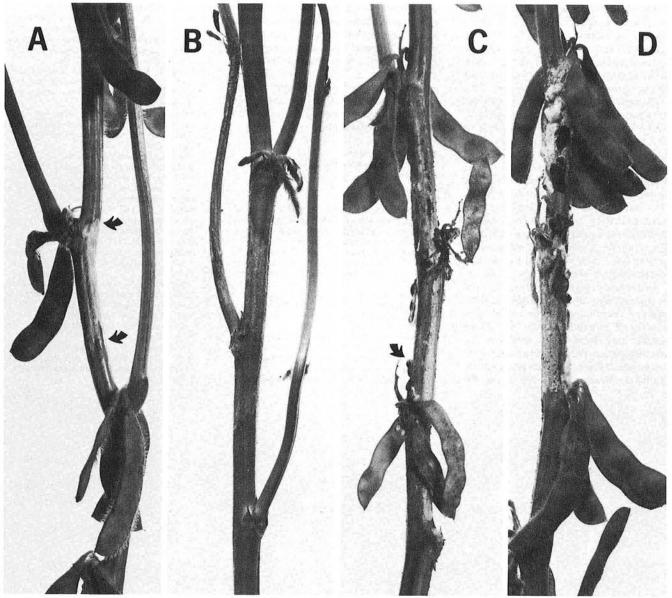


Fig. 1. Symptoms of Sclerotinia stem rot on soybean: (A) Gray, water-soaked lesion on stem (arrows). Lesions in soybeans originate at leaf axils and progress up and down the stem. (B) Tan, water-soaked lesions that eventually encircle the stem. Lesions later appear white to light tan. (C) Fluffy mycelium may be present, and sclerotia begin to form (arrow). (D) Stem lesions completely encompass the stem, and black sclerotia are well developed.

DISCUSSION

The mechanism of resistance in soybean to *S. sclerotiorum* is not known. Stem symptoms originated at leaf axials and rarely at the soil line; thus, ascospores are believed to have been the primary inoculum and not mycelium from myceligenic germination of sclerotia. Field observations and comparison with other susceptible crop species indicate that *S. sclerotiorum* may first colonize senescent flower parts and then progress into stem tissues (1,2,12).

All cultivars with white flowers were relatively susceptible to *S. sclerotiorum*, but some cultivars with purple flowers were less susceptible. We doubt if resistance to the pathogen is expressed when floral parts are infected. However, resistance to stem invasion appears to be related to a factor associated with purple flowers.

Table 1. Mean disease severity indexes (DSI) for Sclerotinia stem rot on soybean cultivars grown in fields naturally infested with Sclerotinia sclerotiorum

1978		1979				1980	
Cultivar	DSI ^a	Cultivar	DSI	Cultivar	DSI	Cultivar	DSI
Corsoy	10	Corsoy	19	Wayne	49	M70-153	14
Hodgson	21	Hodgson	22	Wells	53	A-3	20
Hodgson 78	23	Hodgson 78	31	Wells II	57	Century	33
Vickery	42	Vickery	31	Century	58	Sprite	35
Evans	44	Hark	33	Coles	68	Weber	38
Amsoy 71	51	Amsoy 71	35	Beeson 80	68	Amcor	42
Wells	54	Corsoy 79	40	Beeson	74	Hardin	45
Wayne	74	Amcor	43	Weber	76	Nebsoy	47
		Harcor	44	Gnome	86	Gnome	65
Tukey's testb							
P = 0.05	16		30		30		24
P = 0.10	14		27		27		21

 $^{^{}a}DSI = \Sigma \frac{class \times no. \text{ of plants in class} \times 100}{total \text{ no. of plants} \times 3}$, where the classes are established as follows:

0 = no symptoms; 1 = only lateral branches showing lesions; 2 = lesions on the main stem, but little or no effect on pod-fill; and 3 = lesions on main stem resulting in plant death and poor pod-fill. ^bTukey's test for comparison of treatment means (14).

Klendusity or disease escape should be considered as a factor that could affect severity of Sclerotinia stem rot. Corsov (group II) and Hodgson (group I) were less susceptible to the pathogen than Wells (group II), Weber (group I), and Evans (group 0). Thus, cultivars with high and low disease severity indexes were present within the maturity groups. In our opinion, klendusity is less likely to be a factor than physiologic resistance in explaining the observed differences in susceptibility to S. sclerotiorum. Plant architecture has been shown to affect susceptibility of Great Northern beans (6), but it was not associated with susceptibility or resistance to S. sclerotiorum in our cultivar evaluations. For example, Corsoy has a more bushy growth habit than Wells, but Corsoy had lower disease severity indexes.

In our study, soybean cultivars differed in susceptibility to S. sclerotiorum; thus, cultivar selection could be useful as a control of Sclerotinia stem rot. These results may serve as a guideline for selecting parents for crosses to incorporate resistance to S. sclerotiorum into soybean cultivars. Research is needed on the

epidemiology of Sclerotinia stem rot and subsequent relationships between disease severity ratings and yield loss. The disease severity indexes we report are general guidelines to rate soybean cultivars for resistance to *S. sclerotiorum*. Disease severity was recorded only once during the growing season, and the relationship between disease severity indexes and yield was not determined.

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