Evaluating Soybean Cultivars for Resistance to *Sclerotinia sclerotiorum* Under Field Conditions

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ABSTRACT

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Forty-two soybean (*Glycine max*) cultivars were evaluated for resistance to *Sclerotinia sclerotiorum* in field conditions. The most resistant cultivars in four field trials were Maple Arrow and Ace, but Maple Presto and McCall appear promising. The wide range in cultivar reactions indicated that breeding for resistance to this pathogen is possible. Disease incidence at harvest was correlated with cultivar height, lodging severity, and maturity as well as number of apothecia under the canopy. These results suggest that disease escape may be an important consideration in Sclerotinia stem rot of soybean.

Reports of Sclerotinia stem rot of soybean (*Glycine max* (L.) Merr.) caused by *Sclerotinia sclerotiorum* (Lib.) de Bary indicate that this disease is increasing in both incidence and severity of outbreaks in Illinois (2), Wisconsin (13), and Ontario (1). This increase has been associated with the cultivation of soybean after other crops infected by *S. sclerotiorum* (13,16), expansion of soybean acreage into areas with favorable climate for disease, and the introduction of cultural practices such as irrigation and decreased row spacing (12). Sclerotinia stem rot was first reported in Ontario in 1946 (14,15) but since 1978 has increased with the cultivation of shorter season cultivars in areas with a history of white mold in dry edible white bean (*Phaseolus vulgaris* L.).

Previous reports indicated that differences in resistance were present within soybean cultivars. Limited-term-inoculation (LTI) was the best of three methods for detecting partial resistance among cultivars in controlled-environment studies (2), but the ability of this technique to predict effective resistance in field conditions has not been reported. Resistance was also investigated in other studies, and a number of cultivars were identified as more resistant than others (1,11,13). Resistance in these studies did not appear related to disease escape mechanisms such as cultivar maturity or plant architecture. The ranking of cultivars in field studies was similar at row spacings of 25–38 and 76 cm, but disease severity was higher at narrower row widths and under biweekly irrigation schemes (12). Current knowledge of sources and stability of resistance in soybean to *S. sclerotiorum* is limited because few cultivars have been evaluated. The purpose of this study was to compare the disease reactions of soybean cultivars to *S. sclerotiorum* among different locations and years and to evaluate the utility of greenhouse screening methods to predicting field performance of these cultivars.

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MATERIALS AND METHODS

Four separate experiments were conducted during these investigations. In 1981 and 1982, 23 and 18 soybean cultivars, respectively, were rated for resistance to *S. sclerotiorum* in a field with a known history of disease at Woodstock, Ontario. These tests were planted as randomized, complete block designs with four replicates each year. Each four-row plot was planted with 50 seeds per square meter in rows spaced 36 cm apart. Each row was 7 m long at planting and later trimmed to 6 m. The herbicides metolachlor (Dual) and metribuzin (Lexone) were applied as a tank-mixed, preplant-incorporated treatment at rates of 1.75 and 0.42 L/ha, respectively. Tests were planted on 20 May 1981 and 16 May 1982.

On 31 August 1981 (growth stages R6–R7 [6]), the two middle rows of each plot were evaluated for the number of plants with and without symptoms of Sclerotinia stem rot (disease incidence). On 2 September 1982 (growth stages R6–R7), only one of the two middle rows was evaluated for disease incidence. Cultivar height (cm), lodging severity (1 = plot erect to 5 = plot completely lodged), and maturity (days after planting) were recorded at harvest.

In 1982, an additional experimental trial was planted in the same field at Woodstock. This experiment consisted of seven soybean cultivars selected from the 1981 trial to represent a range of cultivar disease reactions from susceptible to resistant. The experimental design was a randomized complete block planted as described previously. During the growing season, counts were made of apothecia between the two middle rows of each plot (1.4 m^2) and fresh and senescent blossoms on four plants of each cultivar; disease incidence was evaluated in one of the two middle rows of each plot. Cultivar height, lodging severity, and maturity were recorded at harvest.

On 1 June 1984, a field trial of 24 soybean cultivars was planted at Arkell, Ontario, in a field with a known history of white mold in dry edible white bean. The field plot was planted as described previously. Metolachlor and sethoxydim (Poast) herbicides were applied preplant-incorporated (1.75 L/ha) and post-emergence (1.3 L/ha), respectively. Sclerotinia stem rot incidence was determined in one of the two middle rows for each treatment on 4 October 1984 (growth stages R7–R8). Cultivar height and lodging severity were also recorded as described previously.

Differences in disease incidence between cultivars were evaluated with a cluster analysis method for mean separation (10,19). All disease incidence data employed in statistical analyses were transformed with the arc sine transformation (20). All reported values are the means of untransformed data.

RESULTS

Symptoms of Sclerotinia stem rot were observed in all cultivars; however, significant differences among cultivars were apparent in each field trial. The cultivar Evans was chosen as a susceptible check because of its known susceptibility under Ontario conditions.

Disease incidence ranged from 4 to 82% in 1981, and 15 cultivars had significantly ($P = 0.05$) less disease than Evans (Table 1). The most resistant cultivars were Maple Arrow, OAC 1981-3, OAC Pisces, OAC 1981-4, and Bicentennial. In 1982, disease incidence ranged from 2 to 57%, and Maple Arrow, McCall, and Ace were significantly less diseased than Evans (Table 1). The second field trial at Woodstock in 1982 showed Ace, Maple Arrow, and Maple Presto with the lowest disease incidences (Table 2). The field trial at Arkell in 1984 ranged from 0.3 to 22% diseased plants, and Maple Presto, Maple Arrow,

Table 1. Incidence of Sclerotinia stem rot among soybean cultivars in field evaluations at Woodstock in 1981 and 1982 and Arkell, Ontario, in 1984

Cultivar	Diseased plants (%)
1981	
Maple Arrow	4.4 a[z]
OAC 1981-3	4.8 a
OAC Pisces	7.2 a
OAC 1981-4	8.3 a
Bicentennial	12.2 a
Ozzie	23.2 b
Ace	23.2 b
OAC 1981-5	26.1 b
M71-52	31.7 b
Pioneer 0877	33.5 b
Apache	33.6 c
M71-25	47.9 c
Grande	48.4 c
M72-107	51.4 c
Hodgson	55.4 c
Evans	62.4 d
S1346	67.0 d
1282	69.4 d
A1564	70.4 d
Gesto	71.3 d
1981	79.5 d
B152	81.0 d
Prestige	82.0 d
1982	
Maple Arrow	2.4 a
McCall	8.4 a
Ace	9.8 a
A1564	20.0 b
Ozzie	20.9 b
1282	21.8 b
Evans	23.1 b
OAC 1982-8	23.8 b
OAC 1982-6	24.1 b
0877	25.0 b
Bicentennial	26.0 b
OAC Libra	28.9 b
Hodgson	31.4 b
Grande	32.5 b
Apache	32.8 b
OAC Pisces	36.9 b
Gesto	51.6 c
J82	56.8 c
1984	
Maple Presto	0.3 a
Maple Arrow	1.6 a
McCall	1.8 a
Ace	2.0 a
Bicentennial	5.6 b
A2481	6.0 b
B203	6.3 b
1981	7.2 b
Evans	7.5 b
A1937	7.6 b
Maple Amber	8.5 b
Coles	9.2 b
Crusader	9.6 b
Corsoy 79	10.6 b
S1346	10.8 b
Eagle	11.6 b
AP10	11.7 b
A1564	11.7 b
Harcor	12.5 b
1677	14.3 b
B152	14.7 b
DSR171	16.5 b
0877	17.2 b
Hawk	22.4 b

[z]Numbers within columns followed by the same letter are not significantly different ($P = 0.05$) according to Scott-Knott cluster analysis (19).

McCall, and Ace were the most resistant cultivars evaluated (Table 1).

Correlations of disease incidence among cultivars common to the four field trials determined that cultivar reactions varied among individual field trials. In general, the field responses of cultivars were significantly correlated between the 1982 and 1984 trials ($r = 0.80$–0.92, $P = 0.05$) but not between these two trials and the 1981 trial ($r = 0.43$–0.65).

Table 3 presents correlation coefficients relating disease incidence of each cultivar to a number of variables that were hypothesized to affect cultivar response. In 1981, cultivar height, maturity, and lodging severity were each positively correlated with disease incidence of the 23 cultivars evaluated. In 1982, disease incidence within the large research plot (1982a) was not correlated with any variables, whereas disease incidence in the adjoining plot (1982b) was correlated with cultivar height and apothecial number between the two center rows on 6 August 1982. Disease incidence in the 1984 field trial was positively correlated with cultivar height and lodging severity.

DISCUSSION

Some cultivars showed a stable and high level of resistance under field conditions. Maple Arrow and Ace were the only cultivars that had less disease than the susceptible check, Evans, in all field trials. Of the cultivars tested more than once, Maple Arrow, Ace, Maple Presto, and McCall were most resistant to *S. sclerotiorum* under field conditions. The high levels of resistance evident in Maple Arrow and Ace represent new sources of disease resistance and indicate that breeding for Sclerotinia stem rot resistance shows promise as a control strategy. This has been noted also in studies of other cultivars (2,12). Maple Arrow was also resistant to Sclerotinia stem rot in growth room trials (1).

A number of cultivars showed considerable variation in disease incidence among the four trials. For instance, the cultivar Bicentennial had significantly less disease than Evans in 1981 but was comparable to Evans in 1982 and 1984. The cultivar OAC Pisces also ranged from relatively resistant in 1981 to relatively susceptible in 1982. This is the first report of such variation in soybean cultivar responses to *S. sclerotiorum*, and these location or year effects indicate the presence of genotype-environment interactions that can cause difficulty in identifying the most stable, resistant cultivars (5,22).

Variation in cultivar response among trials was associated with a number of crop and pathogen variables. Disease incidence increased with an increase of cultivar height, lodging severity, and maturity, indicating that disease escape is an important mechanism affecting disease incidence in some cultivars. The effects of these crop variables differed among the four trials. Cultivar height in 1981, 1982b, and 1984, lodging severity in 1981 and 1984, and cultivar maturity in 1981 were all significantly correlated with disease incidence. Apothecial number was also correlated with disease on one of the five dates it was monitored. This variable was correlated with cultivar height ($r = 0.80$, $P = 0.03$) but not with maturity ($r = 0.17$, $P = 0.72$) or lodging severity ($r = 0.54$, $P = 0.21$). These results suggest that cultivar height may affect disease incidence by influencing production of apothecia within each

Table 2. Number of apothecia and incidence of Sclerotinia stem rot in seven soybean cultivars in field evaluations at Woodstock, Ontario, in 1982

	Apothecia[y] (no.)	Diseased plants (%)
Ace	7.5 a[z]	3.6 a
Maple Arrow	21.2 b	3.8 a
Maple Presto	11.2 a	4.6 a
OAC 1981-5	24.8 b	23.9 b
Bicentennial	26.2 b	25.4 b
Evans	27.5 b	25.7 b
Prestige	27.8 b	31.4 b

[y]Number of apothecia between two middle rows of plot (1.4 m^2) on 6 August 1982.
[z]Numbers within columns followed by the same letter are not significantly different ($P = 0.05$) according to Scott-Knott cluster analysis (19).

Table 3. Correlation coefficients relating incidence of Sclerotinia stem rot of soybean to selected cultivar and pathogen variables

Variable	Field trials			
	1981	1982a	1982b	1984
Cultivar height	0.63**[z]	0.44	0.82*	0.45*
Cultivar maturity	0.58**	0.33	0.69	0.27
Lodging severity	0.73**	0.23	0.35	0.69**
Number of apothecia on				
22 July 1982	...	...	0.49	...
29 July 1982	...	...	0.72	...
6 August 1982	...	...	0.86*	...
12 August 1982	...	...	0.57	...
26 August 1982	...	...	0.13	...
Total number of apothecia	...	...	0.68	...

[z]Correlation coefficients between disease incidence and selected variables. Statistical significance at * = $P = 0.05$ and ** = $P = 0.01$.

cultivar. In addition, the duration of extended periods of plant surface wetness associated with increased disease incidence may explain differences among cultivars.

In addition to the correlations between disease and crop and pathogen variables, there were intercorrelations between some of the crop variables evaluated. Cultivar height was correlated with lodging ($r = 0.59$–0.74, $P = 0.05$–0.001) and maturity ($r = 0.55$–0.75, $P = 0.006$–0.001) in all field trials, whereas lodging was correlated with maturity only in the 1982 trial ($r = 0.60$, $P = 0.008$). Cultivars that were tall tended to lodge more and result in increased disease incidence.

Field trials and greenhouse evaluations (1) were not correlated for cultivars common to both studies. Correlation coefficients ranged from 0.03–0.41 and were not significant. Cline and Jacobsen (2) reported that ratings obtained by the LTI method paralleled observations of field-infected plants but did not report a statistical analysis of the association. A lack of correlation between field and growthroom studies is not uncommon (17), and Sclerotinia stem rot of soybean appears to be associated with the influence of additional host and pathogen variables on the disease cycle in the field. Small differences in resistance to infection can be hidden by variation in environmental or biological factors (17) that can be induced by differences in cultivar architecture.

The effect of plant architecture on disease avoidance mechanisms in white mold of *P. vulgaris* has been demonstrated (3,4,7–9,18,21). Grau et al (13) did not consider disease escape to be a significant factor in explaining resistance of soybean cultivars to Sclerotinia stem rot in Wisconsin because plant architecture or cultivar maturity group were not associated with disease reaction. Disease escape may be a significant factor in other locations, however, as cultivar height, lodging severity, maturity, and the number of apothecia significantly affected disease reactions in Ontario.

The identification of cultivar characteristics that affect the responses of soybean cultivars to disease under field conditions increases the difficulty of identifying sources of field resistance to this pathogen in the growth room. Cultivars with low disease incidence or severity in field evaluations should be tested in the growth room also to determine if disease escape or physiological resistance is present. Further studies are required on the nature of inheritance of resistance and the relative importance of physiological resistance and disease escape mechanisms under field conditions. Comparative evaluations of all reported sources of resistance would be valuable in identifying the most resistant cultivars for use in breeding programs. Development of soybean cultivars with resistance to this pathogen appears feasible and may represent an effective and economic stategy for disease control in areas where this disease is prevalent.

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