Laboratory and Field Assessment of Resistance in Soybean to Stem Rot Caused by *Sclerotinia sclerotiorum*

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ABSTRACT

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To evaluate resistance to stem rot, commercial soybean cultivars were grown in a greenhouse without fertilizer for 5 wk, then cut apices of excised plants with leaves and stem apices removed were inoculated with mycelial disks from cultures of *Sclerotinia sclerotiorum* grown for 5 days on millet seed agar. The inoculated stems were incubated on moist vermiculite in trays at 20–22 C. The trays were covered with aluminum foil for 3 days, then exposed to ambient light on a laboratory bench. Lesion lengths were measured after 7 days. Commercial soybean cultivars of maturity groups 0, I, II, and III differed in their responses to stem rot with the laboratory method, but results of different experiments often were not consistent. The same cultivars also were tested in an infested field. Disease incidence ranged from 2.2 to 40% in 1984 and from 0 to 52% in 1985. The disease reactions of 13 cultivars tested in both years were significantly correlated ($r = 0.72$) with each other. Seed yields of 16 cultivars in 1985 were inversely correlated with stem rot incidence ($r = -0.94$). For every 10% increase in disease from 0 to 52%, yield was reduced by 7.8% of the maximum yield of 3,024 kg/ha (45 bu/acre). Lesion lengths in commercial cultivars assessed by the laboratory method showed varying correlations with disease incidence in the field experiments ($r = -0.17$–0.86). Ten cultivars of field-grown plants subjected to the laboratory assay gave reactions significantly correlated ($r = 0.68$) with field reactions of those cultivars.

Sclerotinia stem rot or white mold of soybean caused by *Sclerotinia sclerotiorum* (Lib.) de Bary is increasing in importance in Michigan because of the expansion of soybean acreage to infested land formerly cropped to a susceptible host, navy beans. In addition, the practice of growing soybeans with narrow row spacings favors disease development (5).

Differences in susceptibility of soybeans to stem rot have been demonstrated in the field (4,7) and in the greenhouse (1–3). Evaluation of varietal resistance in the field is extremely laborious and permits only one cycle of evaluations during a growing season. Moreover, disease may be variable (6), such that selection of single resistant plants in a breeding program would be impossible. Greenhouse evaluations of disease reactions also are laborious. We first attempted to evaluate resistance in the greenhouse but later decided that a rapid, reliable laboratory method would have advantages of economy in time and labor over greenhouse testing. In this report, we describe 1) an attempt to develop a laboratory method of assessing resistance to stem rot based on the rate of lesion development on excised soybean stems, 2) the reactions of several soybean cultivars to stem rot in field experiments, and 3) attempts to validate the laboratory assay using disease reactions in the field as a standard. Brief reports of some of this work have been published (2,7).

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MATERIALS AND METHODS

The pathogen. *S. sclerotiorum* isolate A from navy bean and isolate G from soybean were obtained from W. L. Casale of our department. These were maintained on millet seed agar. Millet seed was ground in a Wiley mill to pass through a sieve with 0.85-mm openings (20-mesh). Twenty grams of ground seed was mixed per liter of 2% agar and autoclaved, then 20 ml was poured into 9-cm-diameter petri dishes. The agar was inoculated in the center of the plate, and cultures were grown at 23 ± 1 C for 3–14 (usually 5) days. The inoculum consisted of 5-mm-diameter disks cut with a cork borer from two concentric circles 1–1.5 cm from the edge of the culture. In cultures older than 5 days, sclerotial initials had sometimes begun to form. In preliminary experiments, isolate G was more virulent than isolate A on excised soybean stems. Isolate G was used throughout this work

Soybean plants. Seeds were germinated in moist vermiculite for 2 days. Uniform seedlings were transplanted into 11-cm-diameter × 14-cm plastic pots of 946-cm^3 (32 oz) capacity containing a potting mix, with three to seven (usually three) plants per pot. Plants were grown for 3–7 wk in a greenhouse at various times of the year, without fertilizer unless stated otherwise. The temperatures in the greenhouse ranged from 20 to 35 C, and day lengths were extended to 12 hr in the winter by fluorescent lamps. Plants were cut off at ground level, put in polyethylene bags, and brought into the laboratory, where leaves and growing tips were excised before inoculation. The cultivars Corsoy and Evans were used routinely in the development of the method. Corsoy was known to be partially resistant to stem rot in the field (5,6), whereas Evans was regarded as susceptible (6). Later, Weber 84, which was highly susceptible in our field experiments, was substituted for Evans.

Inoculation procedure. Excised stems were placed on 500 cm^3 of moist silica sand (1 cm deep) or 1,000 cm^3 of vermiculite (2 cm deep) in plastic trays 26 × 18 × 6 cm. The trays were first lined with a single layer of plastic film (Borden Sealwrap, Borden Chemical Co, North Andover, MA), two or three layers of which also were used to cover the trays to retain moisture. The sand was moistened with 200 ml of distilled water and the vermiculite with 500 ml distilled water; 12–16 stems were arranged parallel to the short axis of each tray. Stems were inoculated by applying disks of inoculum at various sites on the stems. In most experiments, inoculum was applied either to the axil of the first trifoliolate leaf or on the cut apex of the stem. To aid in adhesion, each disk was dipped in 0.3% water agar before being applied to the stem. Trays with inoculated stems were incubated on a laboratory bench for 5–7 days at about 25 ± 3 C or in later experiments at 21 ± 1 C.

Lesion lengths were measured as the distance from the site of inoculation to the farthest macroscopically visible extent of the lesion. Mean lesion lengths in each replicate were calculated as the sum of individual lesion lengths divided by the total number of inoculated plants, whether infected or not.

Experimental design. Various designs were employed. In general, there were eight to 35 stems per treatment, arranged in three to seven replicates, with one or two trays serving as a replicate. Significant differences between means were determined by Student's *t* test or by Fisher's protected least significant difference (LSD) or Tukey's honestly significant difference (both following analysis of variance). In some experiments, stems were not apportioned into

replicates and standard errors of the mean were calculated.

Field experiments. In 1984, 20 soybean cultivars from maturity groups 0–III were evaluated for reactions to Sclerotinia stem rot in a field on the Michigan State University farm. The field had been artificially infested in 1982 by broadcasting screenings containing sclerotia of *S. sclerotiorum* obtained from elevators used to store navy beans. The field was planted to soybeans in 1983. On 18 June, four 5-m (17-ft) rows of each cultivar were planted 50 cm (20 in.) apart in each of three replicates in a 5 × 4 triple rectangular lattice design. Beginning at flowering and until the end of August, about 5 cm of water was applied to the field weekly by overhead irrigation. On 29 and 30 August, the center two rows of each plot were evaluated for disease severity by rating each plant in the middle two rows on a scale of 0–3, where 0 = no disease, 1 = localized lesions, 2 = expanded lesions but not affecting pod fill, and 3 = expanding lesions resulting in poor pod fill or plant death.

In 1985, 16 cultivars were assessed in the same field. On 20 May, four 4-m (14-ft.) rows of each cultivar were planted 50 cm (20 in.) apart in each of five replicates in a balanced 4 × 4 lattice design. Beginning at flowering, about 4 cm of water was applied weekly by overhead irrigation. On 9 and 10 September, disease was determined in the center two rows of each plot as follows: 0 = no disease; 1 = infected plant, remaining alive; and 2 = infected plant, dead. In late October, soybeans were harvested from the center two rows of each plot and yields were determined.

Data were analyzed by analysis of variance, and differences between means were distinguished by an LSD test.

RESULTS

Greenhouse experiments. Initially, attempts were made to inoculate different soybean cultivars grown in pots in a greenhouse. After inoculation, plants and pots were covered with polylethylene bags to maintain a high humidity. Lesions lengths were measured after 9 days. Several factors were tested for their effects on lesion development. Placement of inoculum on different internodes or at the axil of the first trifoliolate leaf of intact plants had no discernible effect on disease development. Longer lesions were obtained using disks (5 mm diameter) of pathogen-colonized millet seed agar than of wheat seed agar, potato-dextrose agar, oatmeal agar, or colonized soybean stem segments (5 mm long × 2 mm thick). There was no advantage to removing inoculum after 2 days, contrary to results of others (1,3). Infection was similar when inoculum disks of millet seed agar were dipped into either 0.3% water agar, 2% soluble starch, or 2% carboxymethylcellulose to aid adhesion to the plants. Longer lesions developed on plants 3 wk old than on plants 4 wk old.

Because of difficulty in obtaining uniform disease and the labor involved in the greenhouse tests, an attempt was made to develop a laboratory assay for the disease using excised soybean stems.

Handling of plants. Intervals of 1 or 4 hr between excision and inoculation of stems or keeping excised stems on ice or at ambient temperature did not influence subsequent disease development. Therefore, cut stems were kept at ambient temperature and were inoculated within 2 hr of cutting.

Stems with apices excised near the first trifoliolate leaf and intact stems of cultivars Evans and Corsoy were compared to determine whether excision would affect lesion development. Stems were inoculated at the axils of the first trifoliolate leaves and incubated at 21 ± 1 C. Because there was no effect on lesion development, apices were routinely excised for uniformity. Stems usually were incubated under the ambient light of the laboratory.

Age of inoculum. Inoculum disks from cultures 5, 8, and 11 days old were used to inoculate excised stems of 23-day-old cultivar Evans and 16-day-old cultivar Corsoy at leaf axils. Inoculated plants were incubated at 25 ± 3 C. Disks from cultures 5 and 8 days old were more virulent than disks from cultures 11 days old (Table 1). In another experiment, disease produced by 5-day-old cultures was more severe than that produced by 8-or 11-day-old cultures. In general, inoculum disks from cultures 5 days old were used.

Age of plants. Preliminary experiments indicated that longer lesions developed on 3- than on 4-wk-old stems of Corsoy and Evans when both cultivars were inoculated at the leaf axils and incubation was at 25 ± 3 C. Mean lesion lengths were 4.4 and 0.4 cm, respectively, on 3- vs. 4-wk-old stems of Corsoy and 8.2 and 2.9 cm, respectively, on Evans ($P = 0.05$). More 3- than 4-wk-old plants also were infected, i.e., 83 vs. 22% for Corsoy and 100 vs. 61% for Evans.

Lesion development also was compared on stems of Corsoy and Weber 84 from plants 3, 4, 5, 6, and 7 wk old. These ages corresponded, respectively, to growth stages V2, V3, V4, V5–V6, and V7–R1. Plants were fertilized by applying 0.6 g of 20-20-20 (NPK) to the soil in each pot once a week after the plants reached 2 wk old. Stems were inoculated at the cut apices and were incubated at 21 ± 1 C. In Corsoy, lesion lengths tended to decrease in older plants, whereas in Weber 84, only the lesions in 7-wk-old plants were shorter than those in younger plants (Table 2). Differences between the two cultivars were greatest in plants 5, 6, or 7 wk old ($P = 0.05$).

Supplementary fertilizer. During some of the assays, plants grown longer than 4 wk showed symptoms of nitrogen deficiency. Several experiments indicated that the use of fertilizer to correct the deficiency resulted in reduced mean lesion lengths. For example, soil in the pots in which four cultivars were growing was supplemented with 0.6 g of fertilizer (20-20-20, NPK) weekly after the second week. Stems from 5-wk-old plants were inoculated at cut tips and incubated at 21 ± 1 C. Mean lesion length for stems of fertilized plants was 3.8 cm, and that for unfertilized plants was 7.6 cm. These differences were statistically significant ($P = 0.05$).

Site of inoculation. The axil of the first trifoliolate leaf and the internode between the first and second trifoliolate leaves were compared as inoculation sites on stems from 4-wk-old plants of cultivar Evans. Incubation was at 25 ± 3 C. Lesions tended to be longer in stems inoculated at the axil of the first trifoliolate leaf than at the internode in two tests (3.5 ± 1.0 vs. 2.1 ± 0.8 cm and 2.7 ± 0.8 vs. 0.6 ± 0.4 cm).

The axil of the first trifoliolate leaf and the cut stem apex were compared as

Table 1. Effect of inoculum age on development of Sclerotinia stem rot on excised stems of soybean cultivars Corsoy and Evans in a laboratory assay[a]

Culture age (days)	Lesion length (cm)[b]		Infected plants (%)	
	Corsoy	Evans	Corsoy	Evans
5	4.0 ± 0.6	8.0 ± 0.8	83	88
8	4.0 ± 0.6	8.6 ± 0.6	75	96
11	1.4 ± 0.5	4.4 ± 0.7	29	70

[a] Stems (24 per treatment per cultivar) were inoculated in the axil of the first trifoliolate leaf with 5-mm diameter disks cut from the advancing edges of colonies grown on millet seed agar.
[b] Plus-minus standard error of the mean.

Table 2. Effect of age of soybean cultivars Corsoy and Weber 84 on the development of Sclerotinia stem rot on excised stems in the laboratory[y]

Plant age (wk)	Lesion length (cm)[z]	
	Corsoy	Weber 84
3	9.3 ab	10.3 a
4	6.7 bcd	8.3 abc
5	5.8 cd	10.3 a
6	4.7 de	9.9 a
7	2.4 e	6.2 bcd

[y] Stems (two per treatment per cultivar in each of seven replicates) were inoculated at excised apices with mycelial inoculum disks.
[z] Means followed by the same letter did not differ by Tukey's honestly significant difference ($P = 0.05$).

inoculation sites, using stems from 4- and 5-wk-old plants of Corsoy and Evans and an inoculation temperature of 21 ± 1 C. Inoculation of cut terminals gave longer lesions and more infected stems than did inoculation at the leaf axils of plants at either age ($P = 0.05$). For example, lesion lengths on 4-wk-old stems of Corsoy were 9.6 cm for cut terminal inoculation and 3.8 cm for leaf axil inoculation; corresponding values for Evans were 12.6 and 3.7 cm. Respective percentages of infected plants were 100 vs. 56 for Corsoy and 100 vs. 67 for Evans.

Incubation in light vs. darkness. Stems of 4-wk-old plants of cultivars Evans and Corsoy were incubated in trays kept on a bench under the ambient light of the laboratory or were covered with aluminum foil and placed on the same bench at 25 ± 3 C. Inoculum was placed at the axil of the first trifoliolate leaf. In four such experiments, lesion lengths were not affected by light or darkness ($P = 0.05$). Later, trays were covered with aluminum foil for 3 days to prevent upturning of the stem tips, which sometimes dislodged inoculum disks, then were incubated in the ambient light of the laboratory.

Temperature of incubation. To assess the effect of incubation temperature on disease development, stems of cultivars Corsoy and Evans were incubated at 15, 20, 25, and 30 C in the dark and on the laboratory bench at 25 ± 3 C in ambient laboratory light. Plants were 4 wk old and were inoculated at cut ends. Stems incubated at 15 and 20 C had longer lesions and a greater proportion of infected stems than those incubated at 25 and 30 C (Table 3). Stems in trays incubated on the laboratory bench had the least disease development of all. Once infection was established on a stem, lesion development progressed at a similar rate for the cultivar regardless of temperature. Lesions appeared earliest at 20 C, accounting for the longer lesions at that temperature. In a second experiment, inoculated stems were incubated at temperatures of 20, 22, 24, and 27 C. Percentages of plants infected and lesion lengths again were greatest at 20 and 22 C. Henceforth, stems for all experiments were incubated on a laboratory bench at 21 ± 1 C.

Commercial cultivars. At various stages in the development of the laboratory method, commercial cultivars of soybean were assayed for their reactions to stem rot. Statistically significant differences among cultivars usually occurred, but cultivar rankings were not always consistent from one experiment to another (Table 4). Despite variability between experiments, Corsoy frequently was the most resistant of the various cultivars in the laboratory assay.

Commercial cultivars were evaluated for stem rot reaction in an infested field in 1984 and 1985. In 1984, the use of a disease index resulted in the same ranking of the 20 cultivars as did expressing the data as disease incidences. Therefore, only the latter values are given in Table 4. There were essentially three groups. Gnome and Weber 84 had the highest disease incidences, 39.5 and 31.1%, respectively. Century and Elgin were intermediate with infection incidences of 19.1 and 15.5%, respectively. The 16 other cultivars had values ranging from 13.8 to 2.2%, which did not differ significantly ($P = 0.05$).

Data for the 1985 field experiment also are expressed as disease incidence (Table 4). The most susceptible cultivars were Gnome, Weber 84, and Hobbit, with 40.4–52.4% of plants infected. Nebsoy, Sprite, Century, Wells II, Elgin, and Beeson 80 were intermediate with 18.0–29.3% of plants diseased. Evans, Corsoy, Corsoy 79, Hardin, Pella, Hodgson 78, and Ozzie had the least disease, with 0–12% disease incidence. Yields of soybeans across all cultivars were inversely correlated with the disease incidence ($r = -0.94$) (Fig. 1). For every 10% increase in disease from 0 to 52%, yield was reduced by 235 kg/ha (3.5 bu/acre), or 7.8%, from a maximum of 3,024 kg/ha (45 bu/acre). Thus, 52% disease, the maximum for any cultivar, reduced yield by about 40%. The results indicate the very great loss in soybean yield that stem rot can cause and also demonstrate that even low amounts of disease can result in losses. Yields from a disease-free test at East Lansing were available for 13 of the 16 cultivars used in

Table 3. Effect of temperature on development of Sclerotinia stem rot in excised stems of soybean cultivars Corsoy and Evans[x]

Temperature (C)	Lesion length (cm)[y]		Infected plants (%)[y]	
	Corsoy	Evans	Corsoy	Evans
15	6.3 ab	7.6 ab	100 a	100 a
20	9.0 a	10.8 a	100 a	100 a
25	4.2 bc	6.7 bc	57.3 b	81.0 a
30	4.8 bc	3.0 cd	57.0 b	38.0 b
Room[z]	1.1 c	1.8 d	14.3 c	23.7 b

[x] Stems (seven per treatment in each of three replicates) were inoculated with mycelial inoculum disks.
[y] Means in a column followed by the same letter did not differ by Tukey's honestly significant difference ($P = 0.05$).
[z] Temperature of 25 ± 3 C.

Table 4. Reactions of soybean cultivars to Sclerotinia stem rot in laboratory assays and in field tests in 1984 and 1985

Cultivar	Maturity group	Lesion lengths in laboratory assays (cm)		Disease incidence (%) in field experiments[a]	
		Test[b,c]	Test[c,d]	1984[c]	1985[c]
Gnome	II	7.6	9.4	39.5	52.4
Weber 84	I	8.4	7.5	31.1	48.5
Sprite	III	1.8	8.4	–	28.8
Century	II	7.5	7.3	19.1	28.1
Elgin	II	1.5	8.5	15.5	21.2
Vickery	II	–	–	13.8	–
Amsoy 71	II	–	–	13.7	–
Amcor	II	–	–	13.4	–
Corsoy	II	6.8	3.3	–	7.9
Corsoy 79	II	5.8	3.0	13.3	5.3
Lakota	I	–	–	13.3	–
Beeson 80	II	10.9	8.7	12.6	18.0
Harcor	II	–	–	12.4	–
Wells	II	7.9	7.0	11.9	23.1
Nebsoy	II	9.5	10.8	10.0	29.3
Hodgson 78	I	1.9	7.9	6.8	2.8
Pella	III	4.8	6.7	6.7	3.8
Simpson	0	–	–	6.1	–
Hardin	I	5.9	2.7	5.7	7.1
Hobbit	III	5.9	9.5	4.1	40.4
Dawson	0	–	–	2.8	–
Evans	0	7.0	5.2	2.2	12.0
Ozzie	0	5.3	6.7	–	0.0
LSD		3.0	2.1	12.4	13.6

[a] Percentage of diseased plants in two 5-m rows in each of three replicates (1984) or two 4-m rows in each of five replicates (1985). LSD values are Fisher's protected LSD ($P = 0.05$).
[b] Data are based on 3.5-wk-old excised stems (nine per cultivar) inoculated at leaf axils and incubated at 25 C.
[c] Correlation coefficients: test 1 × test 2 = 0.08, 1984 × 1985 = 0.72, test 1 × 1984 = 0.25, test 1 × 1985 = 0.31, test 2 × 1984 = 0.28, test 2 × 1985 = 0.61.
[d] Data are based on 5.5-wk-old excised stems (nine per cultivar) inoculated at cut terminals and incubated at 20 C.

the disease trial. The reduction of yield in the diseased plots relative to the disease-free plots was 257 kg/ha (3.8 bu/acre) for each 10% increase in disease incidence, which is close to that for the diseased plot.

There was no apparent relationship of maturity groups I, II, or III with disease reaction in either year. For example, in 1985, these maturity groups were represented among the cultivars with greatest and least disease. However, the group 0 cultivars Simpson, Dawson, and Evans in 1984 and Ozzie and Evans in 1985, were among the least diseased cultivars.

Thirteen of the same cultivars were tested in both years, and their disease reactions in the two tests were significantly correlated ($r = 0.72$, $P = 0.05$). The greatest discrepancy occurred with Hobbit, which had 4.1% infected plants in 1984 and 40.4% in 1985. When Hobbit was excluded, the correlation coefficient for the remaining 12 cultivars was 0.89. The reason for the large discrepancy with this cultivar is unknown.

Attempts were made to verify the results obtained by the laboratory assay by comparing these data with disease incidence obtained with commercial cultivars in the field. During 1984, half of the cultivars, i.e., 10, were tested in the laboratory at one time. Correlation coefficients for each such experiment with the 1984 and 1985 field results were determined where common cultivars existed. These ranged from $r = -0.53$ to $r = 0.86$. The latter value was the only one of eight such correlations to be statistically significant ($P = 0.01$); it involved seven cultivars compared with the 1985 field results.

Ten assays performed in the laboratory using the 16 field-tested cultivars in 1985 also had varying correlations with the 1984 and 1985 field results, ranging from $r = -0.17$ to $r = 0.63$. Two examples are given in Table 4.

On 28 September 1985, healthy green stems of 10 cultivars that had not yet senesced were brought from the field and assayed by stem inoculation in the laboratory. The results showed a significant ($P = 0.05$) correlation with the field data ($r = 0.68$), indicating that the laboratory assay could, to some extent, predict the field reaction to the disease.

DISCUSSION

The method described may prove useful in assessing resistance to Sclerotinia stem rot in soybean cultivars and introductions and in screening populations segregating for resistance to the disease in a breeding program. Advantages include its speed and economy; an experiment requires only 6–7 wk, 5–6 wk to grow the plants and 1 wk for disease development, and it requires a minimum of laboratory space.

In early experiments, many stems escaped infection, or the initiation of lesion development was asynchronous, contributing to variability, which limited the ability to distinguish differences among treatments or cultivars. This problem was overcome to some extent by standardizing such factors as age of inoculum and of plants, but variability was reduced most by placing the inoculum at cut stem apices rather than at the leaf axils and by reducing the incubation temperature after inoculation from 25 ± 3 C to 21 ± 1 C. With these changes, infection is usually close to 100% and variability in lesion lengths also has been reduced. Results using plants of different ages suggest that differences in cultivar response may be greatest in plants at least 5 wk old.

A remaining problem is that of reproducibility of results. This may be due to varying environmental conditions in the greenhouse, but it is not yet known what growing conditions will produce plants whose reaction to stem rot most closely mimics that in the field. In comparing the responses of a number of cultivars in the field with stem lesions of plants grown in the greenhouse under varying conditions throughout the year, correlation coefficients ranged from $r = -0.53$ to $r = 0.86$. This suggests that growing conditions exist that will produce plants capable of responding similarly to those in the field and lends some validity to the laboratory assay. Such a suggestion also is supported by the similar response ($r = 0.68$) of 10 cultivars in the field and by stem inoculation of the same field-grown cultivars in the laboratory. Work is in progress to define the appropriate growing conditions for plants to be used in the laboratory assay. It is perhaps noteworthy that Corsoy frequently has been the most resistant of the cultivars tested in the laboratory, and it also had relatively little disease in our field tests and those of others (5,6). Corsoy (3) and the closely related cultivar, Corsoy 79 (1), also were relatively resistant in growth chamber tests. At the other extreme, the cultivar Ozzie had no disease in the field in 1985, yet its stems were fully susceptible in the laboratory assay. Whether a consistently high correlation between field and laboratory reactions of plants to stem rot can be achieved is not known. Even if not, a cultivar or introduction in which a lesion cannot develop by stem inoculation would seem likely to be highly resistant to the disease.

It is not to be expected that the rate at which lesions extend in the excised stems should be correlated perfectly with the responses of plants to stem rot under field conditions. Inoculum in the field is presumed to be ascospores that infect the flowers; from this initial infection, the pathogen invades the soybean stem (3,6). Therefore, components of resistance may also reside in the flowers and in the cuticle or wax layers on the stem, both of which are bypassed by inoculating cut ends of plants.

The two field experiments revealed great differences in the responses of soybean cultivars to Sclerotinia stem rot, and there was a reasonable correlation between the reactions of those cultivars tested in both years ($r = 0.72$). Possibly, these results could provide a basis for selecting a soybean cultivar for use in an area where stem rot is a problem. Our field results agree with those of Grau et al (5,6) in that Corsoy and Hodgson 78 were

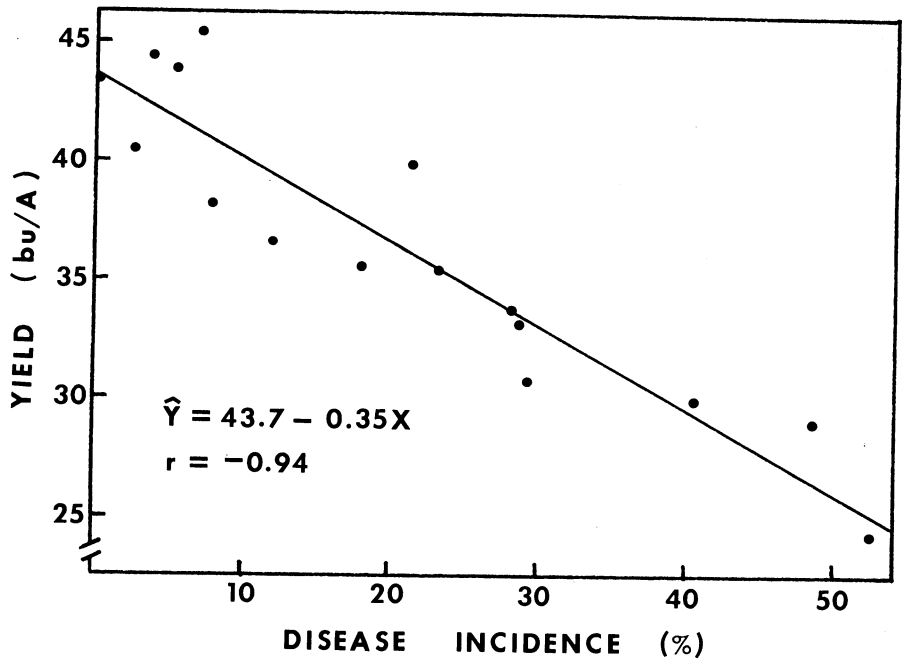

Fig. 1. Relation of incidence of Sclerotinia stem rot to seed yields in 16 soybean cultivars in a field experiment in 1985.

among the more resistant commercial cultivars, whereas Gnome was highly susceptible. In our tests, however, Evans and Hardin also had little disease, whereas Grau et al (6) considered them susceptible. These differences could be due to the great variability inherent in field testing or to differences in varietal specificity among different populations of the pathogen. Differences in field reactions could not be correlated with maturity group I, II, or III, although in both years, group 0 cultivars were among those with least disease. Grau et al (6) found no relation of maturity group 0, I, or II to resistance.

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