Resistance of 17 Soybean Cultivars to Foliar, Latent, and Seed Infection by *Cercospora kikuchii*

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ABSTRACT

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Seventeen soybean (*Glycine max*) cultivars, representing maturity groups I to IV, were screened under greenhouse and field conditions for resistance to foliar and seed infection by *Cercospora kikuchii*. Although significant differences among cultivars in purple seed stain incidence, caused by *C. kikuchii*, were documented in greenhouse and field trials, predicting the ranking in cultivar resistance to purple seed stain under field conditions based on greenhouse results was largely unsuccessful. Four cultivars (BSR 101, Amsoy 71, Hack, and Miami) were consistently susceptible, and one cultivar (Resnick) was resistant to foliar infection in both greenhouse and field trials. However, significant differences in foliar disease severity were detected only in one of two field seasons. No significant differences in cultivar susceptibility to latent infections by *C. kikuchii* were detected in either the greenhouse or the field. The incidence of purple seed stain among 17 soybean cultivars was unrelated to disease development on the foliage.

Purple seed stain and Cercospora leaf blight of soybeans (*Glycine max* (L.) Merr.) are caused by *Cercospora kikuchii* (Matsumoto & Tomoyasu) M.W. Gardner (8,14). Purple seed stain is ubiquitous in soybean cultivation areas of the world (12). Cercospora lesions on leaves are rarely observed prior to growth stage R4, and blighting has been reported in late-season epidemics in the southern United States (17). The disease is of economic significance primarily because it reduces the quality of harvested seed (15).

Infected seeds, when used as planting material, can result in reduced germination and weakened seedlings; and they constitute one source of primary inoculum (19). Previous research on cultivar susceptibility to foliar infection and seed infection has revealed no strong relationship, with some cultivars showing resistance to seed stain but susceptibility to leaf blight (18). Spore trapping in Pennsylvania soybean fields has detected abundant inoculum in the absence of visible symptoms or crop residues from the previous season, indicating an alternative inoculum source (C. E. Orth and W. Schuh, *unpublished data*). Latent infections of soybean foliage can be caused by at least 14 fungal pathogens, including *C. kikuchii* (1,2,10,15). *C. kikuchii* latent infections result from active penetration of the host epidermal cell wall by the fungus subsequent to limited colonization of one to a few cells (10). Latent infecting hyphae resume growth and sporulate following the death of the host tissue (10). Infections developing into typical purple lesions result from stomatal penetration and colonization of the intracellular spaces (5,7,10). The role of latent infections in providing the secondary inoculum for sustaining the epidemic before seed infection is currently being investigated.

Screening for resistance under field conditions is influenced by variations in disease pressure and environment, as well as being labor intensive. Under controlled conditions, these variations can be minimized. The purposes of this study were to compare results from field and greenhouse evaluations, and determine if resistance to one infection type (visible lesion, latent infection, and seed infection) is a reliable predictor of resistance to the other infection types. The results should be useful in evaluating methods of screening new cultivars for resistance to purple seed stain, and in determining strategies for resistance breeding.

MATERIALS AND METHODS

Greenhouse plant production. Seventeen soybean cultivars representing maturity groups (MG) I–IV (4) were used in the experiments (Table 1). Seeds of each cultivar were inoculated with *Bradyrhizobium japonicum* (Kirchner) Jordan inoculum (Agway Inc., Syracuse, NY), and two seeds were planted per pot in 473-ml plastic containers in a mixture of steam-disinfected sand:peat:loam (1:2:2, v/v/v). Pots were placed on greenhouse benches where temperature and relative humidity, as monitored by hygrothermograph, ranged from 20 to 25 C and 20 to 60%, respectively. Plants were watered daily with deionized water, and at growth stage (GS) V1 they were thinned to one per pot and fertilized with Osmocote 20:20:20 (N-P-K) fertilizer. Sunlight was supplemented with 1,000-W high-pressure sodium lamps for 14 hr per day. Plants were maintained in the greenhouse until the beginning of the respective treatments. Plants used to screen for resistance to purple seed stain were produced as above except that they were grown in 2-L plastic pots.

Inoculum production and maintenance. An isolate of *C. kikuchii* obtained from infected seed collected at the Russell E. Larson Agricultural Research Center of Pennsylvania State University was used for all experiments. The isolate was stored in liquid nitrogen as a mycelial plug in 10% glycerol solution. The isolate was cultured on petri plates containing V8 agar. Ten-day-old fungal colonies were induced to sporulate by alternating 12-hr light and dark cycles at 25 C (3, 13,20). After three consecutive fungal transfers, new fungal inoculum was obtained from stocks stored in liquid nitrogen.

Inoculation at growth stage V3. Mature conidia were harvested as described (13). The inoculum concentration was adjusted to 1.25×10^5 conidia per milliliter of distilled water in 0.02% Tween 20 using a hemacytometer. A spore settling tower of dimensions $1.5 \times 1 \times 1$ m (ht $\times$ l $\times$ w) was used for inoculation. Five plants at GS V3 were placed on a rotating disk at the base of the tower. The conidial suspension was sprayed by airbrush into the top of the settling tower to deliver a volume of approximately 21 ml for each inoculation event. The suspension was allowed to settle for 2 min, a time previously determined to provide uniform spore deposition. After inoculation, plants were moved to dew chambers and incubated for 24 hr under continuous darkness at 25 C. The plants were then returned to the greenhouse.

Inoculation of soybean seed pods. From 10 to 15 pods at GS R4 (0.5–2 cm in length) per plant were labeled and sprayed with a spore suspension of 1.25

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$\times 10^5$ conidia per milliliter until runoff. The pods were air-dried for 15 min and then incubated in dew chambers for 24 hr at 25 C under continuous darkness. They were then returned to the greenhouse.

Disease severity assessment. For the first replicate only, plants were observed daily for 14 days following inoculation to determine differences between cultivars in the incubation period: the time in days until 50% of the plants developed lesions. Since there were no differences among cultivars, disease severity (percent leaf area covered) was assessed at days 7 and 21 postinoculation in subsequent replications. Lesion numbers were counted and lesion area was estimated by overlaying a transparent grid delineated into 2 × 2 mm squares onto the leaf. The number of squares overlaying lesions was counted, and the leaf area was measured with a Li-Cor Leaf Area Meter, Model LI-3000 (Lambda International Corp., Lincoln, NE). Disease severity was recorded as percent leaf area covered, i.e., as (0.04 cm^2 × number of grid squares counted)/leaf area.

Latent infection assessment. The numbers of latent infections per inoculated leaf was determined through destructive sampling. All inoculated leaves were removed 21 days after inoculation and surface disinfected for 15 sec in 70% ethanol and 40 sec in 0.5% sodium hypochlorite. Leaves were then rinsed in sterile distilled water for 2 min and air-dried for 24 hr in petri plates placed in incubators at 25 C with 12 hr light (2.5 W m^{-2} of cool-white, fluorescent light) and 12 hr dark. The dry leaflets were moistened with sterile distilled water and maintained for 4 days in the incubators, after which latent infections became visible as sporulating lesions and were counted for each leaf separately. Four days was enough time for symptom expression.

Seed infection assessments. Inoculated pods were harvested at GS R8. Seeds were removed from the pods and surface disinfected in 70% ethanol for 15 sec and in 0.5% sodium hypochlorite for 90 sec. These seeds were then plated on V8 agar to detect symptomless infections, i.e., seeds showing no purple discolorations. After 6 days of incubation at 25 C with 12 hr light and 12 hr dark, the percentage of seeds with purple discolorations and/or producing *C. kikuchii* colonies was determined.

Experimental design. The experiments were a randomized complete-block design. There were four blocks, replicated over time, with four units per block per cultivar. The order of inoculation of cultivars and the assignment of dew chambers were random. Data on seed infection and latent infections were collected on only two of the four blocks.

Data analysis. Differences in disease severity and number of latent infections among cultivars and cultivars within maturity group were analyzed by the general linear models procedure (9) using Minitab (Minitab, Inc., State College, PA).

Field experiments. Field trials were conducted over two growing seasons, 1991 and 1992, at the Russell E. Larson Research Center. All seeds were inoculated with *B. japonicum* and planted in five rows, 0.98 × 0.45 m, on 8.8-cm centers using an experimental plot planter. Plot dimensions were 3.7 × 1.4 m (l × w). Blocks were separated by 0.5-m-wide alleyways. Latent infection incidence was determined on five fully developed leaves per plot at GS R4 by removing leaves from approximately the same node in the upper canopy of each plant and assaying as described above. Visible symptoms were rated when they became apparent at GS R6. The three center rows of each plot were rated as a unit using the Horsfall-Barratt scale (6) and transformed to percentages. In 1991, seeds were hand harvested at physiological maturity (GS R7) and mechanically harvested at harvest maturity (GS R8) using a small plot combine. In 1992, only one seed sample was harvested by combine at harvest maturity (GS R8). Approximately 300 randomly selected seeds were used per block and cultivar. Purple seed stain was determined by visual examination. Data presented were the percentage of the total number of seeds stained.

Experimental design and analysis. The experimental design was a randomized complete-block design with cultivars randomized within blocks. There were four blocks representing the replicates. Infection on leaves and seeds was the result of natural inoculum. Data were analyzed by the GLM procedure using Minitab.

Table 1. Seed and foliar disease responses of 17 soybean cultivars to *Cercospora kikuchii*

Cultivar, maturity group	Foliar disease severity			Latent infections			Percent seed stained		
		Field[u]			Field[w]			Field[y]	
	GH[t]	1991	1992	GH[v]	1991	1992	GH[x]	1991	1992
BSR 101, I	2.3	29.7 ab	6.9	4.2	10.2	7.2	25	25 ab	0.6 b
Weber 84, I	2.2	21.1 ab	18.2	7.1	7.6	7.0	15	4 c	0.1 b
Amsoy 71, II	2.4	18.8 ab	33.5	8.1	7.3	2.3	13	23 ab	2.6 a
Hack, II	9.1	4.7 b	38.3	10.7	10.8	4.7	28	30 a	0.5 b
Preston, II	3.1	14.1 ab	14.6	7.5	8.8	4.2	18	10 bc	0.8 b
Century 84, II	5.1	39.1 a	10.3	7.1	9.7	2.2	7	18 abc	0.3 b
Elgin 87, II	2.1	41.6 a	30.4	4.4	10.5	6.7	20	12 bc	0.6 b
Miami, II	1.9	37.5 a	37.1	5.2	8.4	10.5	32	24 ab	1.0 b
Pella 86, III	4.2	10.5 ab	4.9	6.7	15.9	4.5	14	14 bc	0.1 b
Chamberlain, III	2.1	4.1 b	6.4	6.1	6.4	3.7	13	10 bc	0.8 b
Harper 87, III	3.8	16.4 ab	41.4	6.0	12.7	6.2	16	9 bc	0.6 b
Resnick, III	2.4	16.4 ab	3.8	3.6	9.8	2.5	1	8 bc	0.1 b
Hobbit 87, III	1.6	18.8 ab	29.7	3.4	11.1	7.2	2	12 bc	0.2 b
Winchester, III	4.2	15.2 ab	43.0	5.1	13.2	4.2	23	11 bc	0.2 b
Fremont, III	2.1	39.6 a	68.0	9.3	9.4	5.8	12	16 bc	0.2 b
Sparks, IV	2.6	4.1 b	24.6	5.0	8.9	3.0	18	13 bc	0.3 b
Flyer, IV	3.2	5.8 b	25.0	4.4	9.8	3.7	18	5 c	0.0 b
HSD[z] 0.05	NS	NS	32.88	NS	NS	4.72	NS	8.4	0.9

[t] Disease severity (% leaf coverage) in greenhouse experiments based on four replicates with four leaves per replication.
[u] Disease severity (% leaf coverage) in field experiments at growth stage (GS) R6. Severity estimated by Horsfall-Barratt scale (based on four replicates) randomized complete-block design (RCBD). Means followed by the same letter are not significantly different.
[v] Incidence of latent infections in greenhouse experiments based on two replicates with four subsamples per replication and one leaf per subsample.
[w] Incidence of latent infections in field experiments at GS R4 based on five leaves per plot in four replications, RCBD.
[x] Incidence of purple stained seed in greenhouse trials. Means based on two replicates with two plants per replication and two to 12 pods harvested per plant.
[y] Incidence of purple stained seed from the field trail harvested at GS R8. Means based on four replications (approximately 1,200 randomly selected seeds), RCBD. Means followed by the same letter are not significantly different.
[z] Honest significant difference, based on Tukey's pairwise comparison procedure, with the family error rate protected at $P = 0.05$.

RESULTS

Disease severity and latent infections in the greenhouse. Disease severity on the foliage of plants at GS V3 ranged from 1.6 to 9.1% among cultivars, but significant differences were not detected ($P = 0.598$). Disease severity (%) among the maturity groups, averaged over cultivars, was in the range of 2.9–3.2% ($P = 0.668$). Neither cultivar nor maturity group responses to latent infection incidence were significant ($P = 0.619$ and $P = 0.341$, respectively) (Table 1). Mean number of latent infections per leaf was lowest for Hobbit 87 and highest for Hack, 3.4 and 10.7, respectively. There was a significant blocking effect ($P < 0.001$) for both disease severity and latent infections, with less disease developing in replications conducted during winter months (blocks 1 and 4), when relative humidity and light quality were lowest. Analysis of individual blocks did not reveal any significant differences, and since the variances were homogeneous, blocks were treated as replications.

Disease severity and latent infections in the field. Typical disease symptoms of Cercospora infection on leaves were not discernible prior to GS R5 on any cultivars. All cultivars were rated at GS R6 for severity (percent leaf coverage) by evaluating discrete lesions and bronzing symptoms (17). Significant differences were detected in 1991 for both MG and cultivars, $P = 0.002$ and 0.001, respectively. Cultivars in MG IV had significantly less severe symptoms, with 4.9% of the leaf area affected, compared to 24.3% recorded for those belonging to MG II (Table 1). Among cultivars, Century 84, Elgin 87, Miami, and Fremont had significantly higher disease severity than Hack, Chamberlain, Sparks, and Flyer. No significant differences were detected for latent infections in the 1991 field trials for either cultivar or maturity group.

In the 1992 trial, no significant differences in percent leaf area affected were detected for either maturity group (range of 3.7–5.9%) or cultivar (range of 2.2–10.5%). However, latent infection incidence for 1992 was significant for cultivar ($P = 0.055$) at the 10% significance level. This is probably accounted for by the large number of latent infections detected on cultivar Miami relative to the other cultivars.

Incidence of purple seed stain disease in greenhouse and field trials. Significant differences were not detected in the greenhouse trial for maturity group, cultivar, or replicate. There was a wide range in percent seed infection, with Miami displaying the greatest susceptibility (mean of 32%) and Resnick the least (mean of 1%) (Table 1). The MGs ranked I, II, IV, and III from most to least susceptible and ranged in means from 20% for MG I to 12% for MG III.

In 1991 field trials, both harvest dates, physiological (GS R7) and harvest maturity (GS R8) of seeds, revealed significant differences ($P < 0.05$) for both maturity group and cultivar in purple seed stain incidence. The MGs ranked II, I, III, and IV from most to least susceptible. The GS R8 harvest revealed a significant difference among MG II, III, and IV, with means of 19.6, 11.2, and 8.7%, respectively (Table 1). Cultivars Century 84, Miami, and Hobbit 87 developed greater than 5% additional purple seed stain with harvest at GS R8 relative to GS R7. There was little difference in seed stain incidence between pods harvested at GS R7 and GS R8 among the other cultivars. Cultivars Hack, BSR 101, Miami, and Amsoy 71 had greater than 20% purple stained seed at GS R8. Cultivars Flyer and Weber 84 were most resistant, with 5 and 4% purple stained seed, respectively (Table 1).

In the field trials of 1992, the proportion of infected seed was greatly reduced relative to 1991. Although initial inoculum levels were similar, disease pressure, as determined through spore trapping (C. E. Orth and W. Schuh, *unpublished data*), was low during the 1992 season. Nevertheless, significant differences among cultivars could be detected. Amsoy 71, an MG II cultivar, displayed the greatest percentage of infected seed, and Flyer, MG IV, displayed the least, 2.6 and 0.0% seed infected, respectively. There was also a significant effect of maturity group, with MG II having significantly greater percent purple stained seed (1.0%) than any other maturity group.

Relationship between foliar disease development and purple seed stain incidence. There were no significant differences detected in greenhouse trials relative to latent infection or foliar symptom development. Ranking cultivars and maturity groups from most to least susceptible did not reveal any consistent pattern of susceptibility to latent infection and lesion development. Among maturity groups, MG II had the greatest disease severity as well as the highest level of latent infections. The cultivar Hack had both the greatest incidence of latent infections and the most severe symptom development under greenhouse conditions, and was responsible for MG II cultivars displaying the greatest susceptibility. There was no other cultivar that could be ranked consistently by these two criteria in the greenhouse trials.

In the 1991 and 1992 field trials, no relationship was detected between foliar disease symptom severity and incidence of latent infection among cultivars or maturity groups. Greenhouse results for latent infection and symptom development were not correlated with field results for these variables relative to either cultivar or maturity group rankings.

The incidence of purple seed stain detected in greenhouse trials revealed Hack to be one of the most susceptible cultivars. This was also the case for latent infections and foliar lesion development. Hobbit 87 was one of the most resistant to seed infection and the foliar infections. No other cultivar showed consistent rankings among these three disease expressions. The correlation between maturity group and seed infection in the greenhouse trials was −0.183.

In the 1991 and 1992 field trials, little relationship was found between foliar disease expression, whether latent or visible lesions, and the proportion of seed stain among the tested cultivars. The maturity groups had correlation coefficients of −0.310 and −0.337 for the GS R7 and GS R8 harvests of the 1991 trial, respectively. Only Amsoy 71 was found to be significantly more susceptible than the other cultivars. The correlation between seed infection in the greenhouse and the 1991 GS R8 harvest was approximately 0.28. Cultivars BSR 101, Hack, and Miami were all susceptible. In general, the greenhouse trials identified cultivars that were most susceptible to seed infection in the 1991 field trial.

DISCUSSION

The incidence of purple seed stain observed in the greenhouse and in the field in 1991 significantly exceeded the maximum allowable incidence of 5% for no. 1 grade yellow soybeans, as defined by the U.S. Department of Agriculture (16). Host plant resistance is the preferred strategy for avoidance of such losses. Greenhouse trials were not successful in identifying cultivars with resistance to purple seed stain in the field, since no significant differences among cultivars could be detected. Cultivars such as Weber 84, Harper 87, and Flyer, which performed well in the field, ranked intermediate in greenhouse experiments. However, cultivars that were the most susceptible in greenhouse experiments (BSR 101, Hack, Amsoy 71, and Miami) were also the most susceptible in the 1991 field experiment. The inability to detect differences in cultivar resistance in the greenhouse experiments may be due to inadequate disease pressure exerted on the test material. Pods were susceptible to infection starting early in their development (13). In the field, they are therefore exposed to multiple infection events, as contrasted with the single point inoculation used in the greenhouse trials. Increasing the inoculum dosage, multiple inoculations, or extending the pod wetness period (13) might improve the cultivar separation by increasing disease pressure.

Even though foliar infection, resulting in latent or visible lesions, may be of little economic importance, its role in sustaining the epidemic through production of inoculum for pod infections makes incorporation of this type of resistance into

cultivars desirable. Latent infections form sporulating lesions after leaf drop and senescence. A significant amount of leaf drop, under Pennsylvania conditions, is observed around canopy closing (GS R1). The potential effect of inoculum reduction through host resistance to foliar infection can be seen in the 1992 data. Inoculum densities, as measured through spore trapping, were low in 1992 compared to 1991. This may have contributed to the drastically reduced levels of purple seed stain. Even though the reduced levels of inoculum in 1992 were probably the result of environmental factors, host plant resistance to foliar infection could have a similar effect; i.e., it could reduce the level of seed infection through reduction of inoculum levels during the season.

As has been previously reported, resistance to foliar infection resulting in visible lesions was not a reliable predictor of subsequent incidence of purple seed stain (18). Furthermore, the late-season blighting that has been associated with Cercospora infection (17) also was not strongly correlated with the final incidence of infected seeds.

The absence of visible symptoms of leaf infection until GS R6 points to the potential role of latent infections for inoculum production up to that developmental stage. No significant differences among cultivars were observed either in greenhouse or field experiments. Haustoria resulting from direct penetration of the leaf surface are restricted in their subsequent growth due to the collapse of neighboring cells (10). This type of resistant reaction, along with uniform levels of infection among cultivars within each experimental approach, suggests that resistance was widespread and general. Further investigations based on a variety of fungal isolates are needed to conclusively prove this statement.

Ploper et al (11) reported that the duration of GS R7–R8 is critical to seed infection. They determined that cultivars with a short duration of GS R7–R8 had reduced levels of seed infection. In this study, seven of 13 cultivars had increased levels of purple seed stain when assessed at the R7 vs. R8 growth stage. The increases were, however, small. No attempt was made to determine the duration of the R7–R8 period.

With the methodologies used in this study, results obtained in greenhouse studies were not a reliable indicator of cultivar performance in the field. Resistance to foliar infection (visible lesions and latent infections) was not related to, nor was it a reliable indicator of, resistance to seed infection. Resistance to latent infection shows potential for decreasing the inoculum produced during the season and should be investigated using a larger number of cultivars and fungal isolates.

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