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Evaluation of Soybean Cultivars with the *Rps1k* Gene for Partial Resistance or Field Tolerance to *Phytophthora sojae*

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ABSTRACT

Phytophthora root rot, caused by *Phytophthora sojae* Kaufmann and Gerdeman, primarily attacks the roots of soybean [*Glycine max* (L.) Merr.] plants. Partial resistance and field tolerance in 14 commercial glyphosate [*N*-(phosphonomethyl)glycine] tolerant soybean cultivars with the *Rps1k* resistance gene were studied. Partial resistance to compatible *P. sojae* races 28 and 30 was evaluated by the agar layer technique. Relative to the percentage of the control, all of the commercial cultivars with the *Rps1k* had reductions in top mass and plant height that were not significantly different from the partial resistant check 'Conrad' that had 83% top mass and 77% plant height reduction; two of the 14 commercial cultivars had significantly lower root mass (28 and 31% lower) than Conrad (84%). In addition, there was no significant difference in disease ratings (root or whole plant) of the 14 commercial cultivars with the *Rps1k* compared with Conrad. Field tolerance, studied in six field experiments at Urbana, IL, during 2002–2004, was identified when there were no significant differences between the yield of inoculated treatments with or without mefenoxam [methyl *N*-(methoxyacetyl)-*N*-(2,6-xylyl)-D-alaninate] fungicide seed treatment or between inoculated and noninoculated treatments. There were no significant cultivar × inoculation × fungicide treatment interactions found in any of the field experiments, and a significant cultivar × inoculation treatment interaction was found in only one field experiment. Therefore, most of the cultivars appeared to be tolerant to *P. sojae*. It should be noted that field tolerance was not distinguished from partial resistance in the field component of this study.

PHYTOPHTHORA root and stem rot (PRR) is a soil borne disease that causes pre- and post-emergence damping-off, root and stem rot, and yellowing and wilting of lower leaves of soybean plants. The importance of PRR and complexities of genetic interaction of *P. sojae* races to specific resistance has been summarized (Schmitthenner, 1999).

Two types of resistance to *P. sojae*, complete and partial resistance, have been identified in soybean. Complete resistance is race-specific, monogenic dominant in inheritance, and conditions whole-plant immunity to infection, with one exception, *Rps2*, that only consistently conditions root resistance (McBlain et al., 1991). Eight loci conditioning race-specific, complete resistance, designated *Rps* genes, have been identified in soybean (Dorrance et al., 2003). Multiple alleles have been found at two of the loci, *Rps1* and *Rps3*, and are designated with a letter following the locus number, e.g., *Rps1k*. There is a gene-for-gene interaction between avirulence genes in *P. sojae* isolates and soybean *Rps* genes (Dorrance and St. Martin, 2000). At least 55 physiological races of *P. sojae* have been identified on the basis of compatible (susceptible) or incompatible (resistant) reactions after inoculation on a set of differential soybean lines possessing eight different *Rps* genes (Dorrance and St. Martin, 2000; Dorrance et al., 2003).

Deployment of race-specific resistance genes in soybean cultivars has been the primary method used to control PRR (Schmitthenner, 1999). Frequencies of new virulent *P. sojae* pathotypes that can overcome race-specific resistance genes increase in *P. sojae* soil populations as the pathogen adapts to the continued use of cultivars possessing *Rps* genes (Abney et al., 1997; Schmitthenner, 1999, 1994). This results in a breakdown of effective resistance, necessitates a changeover to cultivars with new PRR resistance and is an example of the "boom and bust cycle" phenomenon in plant resistance gene deployment (Priestley, 1978). However, the widespread deployment of *Rps1k* has remained effective in most soybean production areas beyond the 8- to 15-yr period of effectiveness of other *Rps* genes (Schmitthenner et al., 1994), although new *Rps1k*-virulent *P. sojae* populations have recently been reported to be increasing in some areas (Dorrance et al., 2003).

An alternative to race-specific resistance to P. *sojae* is partial resistance, also called field resistance, general resistance, and rate-reducing resistance (Buzzell and Anderson, 1982; Schmitthenner and Walker, 1979; St. Martin et al., 1994; Thomison et al., 1988; Tooley and Grau, 1981; Walker and Schmitthenner, 1984). Expression of partial resistance is incomplete because pathogen colonization occurs on inoculated plants, but the extent of colonization is limited compared with colonization on fully susceptible soybean genotypes. Partial resistance has been identified by challenging soybean lines with a compatible *P. sojae* isolate and determining the extent of colonization indirectly by measuring the amount of plant damage (Dorrance et al., 2003; McBlain et al., 1991; McBlain and Schmitthenner, 1991; Olah and Schmitthenner, 1985; Schmitthenner, 1985; Walker and Schmitthenner, 1984). The inoculum layer test has become the established method to evaluate partial resistance to *P. sojae* in soybean genotypes (McBlain et al., 1991; Schmitthenner et al., 1994). Partial resistance is quantitative in expression (Buzzell and Anderson, 1982; Olah and Schmitthenner, 1985), polygenic (Glover and

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Abbreviations: MG, maturity group; PRR, Phytophthora stem and root rot.

Scott, 1998; Walker and Schmitthenner, 1984), and is thought to be nonrace-specific (Schmitthenner and Walker, 1979). Two putative quantitative trait loci conditioning partial resistance were identified in the public soybean cultivar Conrad (Burnham et al., 2003). Soybean cultivars with high levels of partial resistance have been developed (Beuerlein et al., 2000).

Tolerance to *P. sojae*, an alternative to resistance, is often defined in plant pathology literature as better productivity in one plant line relative to another despite similar levels of pathogen colonization. For soybean and *P. sojae*, tolerance has alternatively been defined as the ability of a soybean genotype to remain productive, even under severe disease pressure (St. Martin et al., 1994). It offers yield stability when conditions favor disease development. Soybean cultivars that are tolerant to PRR have been identified (Olah and Schmitthenner, 1985). The objectives of this study were to identify partial resistance and field tolerance to two races of *P. sojae* compatible with the *Rps1k* gene in private commercial glyphosate tolerant cultivars.

MATERIALS AND METHODS

Confirmation of Race-Specific Resistance

The experimental design was a randomized complete block design with three treatments blocked four times. The experimental unit was a pot of five seedlings of each of 16 commercial cultivars (Table 1), 'Williams 82', and 'Williams.' Plants were grown in 10-cm diameter plastic pots (Hummert International, Earth City, MO) in a soil-less medium (Sunshine mix, LC1, Sun Gro Horticulture Inc., Bellevue, WA) and were fertilized with 8.2 g of slow release pellets (Nutricote, 18–6-6) spread over the surface of the soil. Treatments were *P. sojae* races 1, 28, and 30. The pathogen was transferred to V8–10 agar 1 to 2 wk before inoculating plants. Plants were inoculated at the unifoliolate stage (V1) using the hypocotyl injection method (Keeling, 1976) 7 d after planting. After inoculation, the plants were incubated in a dew chamber (100% relative humidity) at 22°C without light for 3 d, and then placed on a greenhouse bench at 28°C in ambient light supplemented by illumination from 1000 w metal halide and 1000 w high-pressure sodium lamps. Light intensity averaged 2.1 PAR/s/m^2, measured with a light meter (LI-COR, Inc., Lincoln, NE 68504). Five days after inoculation, plant survival was evaluated. Each experiment was repeated twice.

Table 1. Characteristics of 16 soybean cultivars provided by commercial seed companies.

Cultivar	Company†	*Rps* gene	Maturity	Height (cm)	PRR field tolerance‡
DSR199	Dairyland	*1k*	1.9	82.5	H
DSR241	Dairyland	*1k*	2.4	72.5	H
DSR297	Dairyland	*1k*	2.9	87.5	H
FS2105	FS HiSOY	unknown	2.1	medium	H
GR3101	Midwest	*1k*	3.1	67.5	H
P92B84	Pioneer	*1k*	2.8	-§	M
P93B01	Pioneer	*1k*	3	–	M
GR3331	Midwest	*1k*	3.3	72.5	H
AG3201	Asgrow	*1k*	3.2	–	M
AG3302	Asgrow	*1c*	3.3	–	L
DKB3151	Dekalb	*1k*	3.1	–	M
DSR321	Dairyland	*1k*	3.2	90	H
DSR322	Dairyland	*1k*	3.1	85	H
GH3135	Golden Harvest	*1k*	3.1	–	–
P93B09	Pioneer	*1k*	3	–	M
P93B36	Pioneer	*1k*	3.3	–	M

† Asgrow, Dairyland Seed Co., Inc., Dekalb, FS HiSOY, Golden Harvest, Midwest Seed Genetics, Inc., Pioneer Hi-Bred International, Inc.
‡ PRR = Phytophthora root and stem rot. Advertised tolerance level: H = high field tolerance, M = medium field tolerance, and L = low field tolerance.
§ Information not provided.

Greenhouse Evaluation of Commercial Cultivars for Partial Resistance

Sixteen commercial cultivars (Table 1), 'Conrad', Williams 82, Williams and 'Essex' were inoculated with *P. sojae* race 28 and race 30 in a single experiment. Inoculum consisted of 12-d-old *P. sojae* cultures grown on V8–10 agar. V8–10 agar without *P. sojae* was used in mock inoculation treatments. Five plants of each cultivar were grown in 10-cm diameter polystyrene cups with holes punched in the bottom for drainage. The cups were filled with 300 mL of sand followed by V8–10 agar from plates (100 × 15 mm) with and without *P. sojae*, and then covered with an additional 300 mL of sand. Five seeds of each cultivar were placed on top of the sand and covered with an additional 100 mL of sand. Plants were watered to flooding twice per day. Ambient light was supplemented by illumination from 1000 w metal halide and 1000 w high-pressure sodium vapor lamps (13 h photoperiod). Light intensity averaged 2.1 PAR/s/m^2, measured using a light meter (LI-COR, Inc., Lincoln, NE). Plants were grown at 24/18°C day/night temperatures.

The experimental design was a factorial in randomized complete blocks with six blocks. Preliminary experiments repeated twice using the same treatments and experimental units indicated that six blocks were optimum to detect differences within 10% of the mean at the 5% level of significance. Treatments were the 20 cultivars and the two inoculation treatments, V8–10 agar with either race 28 or race 30, and the mock-inoculated control treatment.

Three weeks after planting, plant heights were measured from the base of the plants at the soil line to the top leaf node. Mean height of plants within each cup was calculated. Plants were removed from cups and roots were washed to remove sand. Two disease-rating scales, a root rot rating scale and a whole plant rating scale, were used to assess disease severity, (Schmitthenner and Bhat, 1994; Schmitthenner et al., 1994). The root rot rating scale was 0 = no root mass lost, 1 = 1 to 10% root mass lost, 2 = 11 to 35% root mass lost, 3 = 36 to 65% root mass lost, 4 = 66 to 90% root mass lost, and 5 = 91 to 100% root mass lost. The whole plant rating scale was 1 = no root rot, 2 = trace of root rot, 3 = bottom third of root mass rotted, 4 = bottom two-thirds of mass rooted, 5 = all roots rotted, 10% seedling kill, slight stunting of tops of plants, 6 = 50% seedling kill, moderate stunting of tops, 7 = 75% seedling kill, severe stunting of tops, 8 = 90% seedling kill, 9 = all seedlings dead, and 10 = no seedling emergence. After rating, plants were wrapped in paper towels and placed in an oven at 33 to 35°C for 3 d. Dry root mass (g) and top (above the soil line) mass (g) were measured from each group of five plants in a pot.

In our study, cultivars were considered to have partial resistance if they had limited reductions in plant height and root and top mass in inoculated pots relative to the mock-inoculated control, and/or if they had lower root and whole plant disease ratings. The soybean cultivar, AG3302 with the *Rps1c* gene and incompatibility with *P. sojae* races 28 and 30, was used as a resistant check. The cultivars without an *Rps* gene or with the defeated *Rps1k* gene were expected to be compatible with the races tested and express susceptibility or partial resistance to infection relative to the noninoculated control. In addition to recording the root and whole plant disease ratings, cultivar response to infection was determined

by calculating the percentage of plant height, root mass, and top mass relative to the mock-inoculated control in each block within a cultivar, e.g., for cultivar A, (plant height of inoculated plants/plant height of noninoculated plants) × 100%. The relative responses of the commercial soybean cultivars were compared with susceptible Essex and Williams, Williams 82 (with *Rps1k*), and Conrad, with high partial resistance.

An analysis of variance (SAS Institute Inc., Cary, NC) was performed using JMP Vers. 5.1 (SAS Institute Inc. 2004) to determine the significance of the main effects blocks, soybean cultivar, inoculation, and the interaction between cultivar and inoculation for each measured trait. The mean square error of the cultivar × inoculation interaction effect was partitioned into single degree of freedom comparisons of the means between inoculated and mock-inoculated control for plant height and root and top masses for each cultivar using contrasts. Disease ratings were transformed by $\log_{10}(X + 1)$ before analysis of variance to stabilize the sample variance. Least significant differences (LSD at $P = 0.05$) were calculated to separate treatment means. Correlation analysis using the multivariate pairwise correlation procedure in JMP vers. 5.1 was performed on the percent plant height, root mass, and top mass relative to the mock-inoculated control, as well as root disease rating and whole plant rating, to determine the significance of the relationships between these measurements.

Field Experiments

Sixteen glyphosate tolerant soybean cultivars, 14 possessing the *Rps1k* gene, one with the *Rps1c* gene, and, one with no known race-specific genes for resistance (Table 1) were used for field tolerance evaluation. Some of these cultivars had advertised tolerance levels (Table 1), but there were no known PRR tolerant or PRR nontolerant glyphosate tolerant soybean cultivars to use as checks based on published research. The 16 cultivars were placed into two separate groups of eight cultivars that were close in maturity date to aid in harvesting the plots. Each group of eight cultivars was planted into two separate experiments at Urbana, IL, in 2002 and 2003. Experiment 1 had the earlier maturing cultivars while the cultivars in Exp. 2 were later maturing. The set of two experiments were identical in 2002 and 2003. On the basis of yield performance in 2002 and 2003, the two experiments were pared down in 2004 to include 'DSR199', 'DSR241', 'DSR297', and 'P93B01' in Exp. 1 and 'AG3201', 'AG3302', 'DKB3151', and 'GH3331' in Exp. 2.

The experimental units were two-row plots spaced 76 cm apart. Approximately 200 seeds were sown into a 5.2-m row. Planting dates were 6 July in 2002, 23 May in 2003, and 15 May in 2004. The soil types present at the Urbana, IL, location where all experiments were conducted were an Elburn silt loam (fine-silty, mixed, mesic Aquic Argiudolls) and a Thorp silt loam (fine-silty, mixed, mesic Agiaquic Argialbolls).

Experimental units (plots) were arranged in a split-split-plot design in four complete blocks in 2002 and 2003 and in six complete blocks in 2004. Cultivars were the main plots. The subplots were inoculation treatments consisting of a noninoculated control, *P. sojae* race 28 inoculation, and *P. sojae* race 30 inoculation. Sub-subplots were Apron fungicide treated seed and nontreated seed.

To increase inoculum, white millet (23 kg) (Siemer Enterprises, Inc. Teupolis, IL) was added to 20 L of boiling water and 500 g of L-asparagine H_2O (Fisher Scientific Company L.L.C., Hanover Park, IL). The mixture was stirred for 15 min, strained, and collected in 57-L buckets. The millet was thoroughly washed with hot tap water. Two liters of millet were packed into plastic autoclavable bags (Fisher Scientific Company), sealed with foam plugs to close 3-cm diameter openings cut with a punch (Greenlee Tool Company, Rockford, IL), and tied with plastic tiers. The bags were placed on aluminum trays and autoclaved for 2 h at 115°C. After cooling, each bag was inoculated with a different isolate of *P. sojae* which was maintained on V8–10 agar. Each Petri dish culture was removed and combined with 250 mL dH_2O in a blender and macerated at medium speed for 2 min. After blending, 10 mL of the homogenate was aseptically added to each bag with a hypodermic needle through the bag opening after removing the foam plugs and the plugs were immediately replaced following infestation. The bags were then kept in the dark at 20°C for 4 wk.

Inoculation was applied by spreading 250 mL of air-dried, infested millet inoculum on the surface of each row and manually raking it in 2 d after planting (8 and 9 July in 2002, 27 May in 2003, and 17 May in 2004). The next day, the plots were irrigated until saturated with overhead sprinklers. Additional irrigation was applied as needed throughout the season.

For the treatments receiving the fungicide seed treatment, mefenoxam (Apron XL, Syngenta Crop Protection, Guelph, ON) was applied to seed at the rate of 1 mL of Apron XL (0.024 a.i.) per kg of seed. Seed and fungicide were gently agitated together to ensure full coverage on the seed. The treated seed was air-dried before planting.

Plants were observed weekly for symptoms. Rows were trimmed to a uniform 5.1-m length with a rototiller. Plant populations in each row were determined 2 wk after planting by counting the number of plants in 90 cm of a randomly selected row. Plant heights were measured in the middle of the plot with a graduated stick as the average distance in cm from the ground to the apex of plants between the R5 to R6 growth stage (Fehr and Caviness, 1977). Two rows of each plot were harvested. Seed was cleaned of debris before recording total seed weight. Seed weights were standardized to 135 g kg^{-1} moisture.

Analyses of variance were calculated for stand counts, plant height, and seed yield for each experiment to determine the significance of the effects of block, the main plot treatment (cultivar), the subplot treatment (inoculation), the sub-subplot treatment (seed fungicide treatment), and the interactions cultivar × inoculation, inoculation × seed fungicide treatment, cultivar × seed fungicide treatment, and cultivar × inoculation × seed fungicide treatment. In addition, when the effect of the inoculation treatments was significant, single degree of freedom comparisons of the noninoculated control vs. inoculated treatments and *P. sojae* race 28 vs. race 30 inoculation treatments were made using orthogonal contrasts to partition the mean square error for inoculation treatments.

In our study, tolerance in a susceptible cultivar, lacking complete resistance *Rps*1k genes incompatible with *P. sojae* races 28 and 30, was identified when there were no significant differences between its yield in inoculated treatments with or without Apron seed treatment or between inoculated and noninoculated treatments. Conversely, a cultivar was considered to be nontolerant to *P. sojae* infection if the differences between the means of its yield in the inoculated treatments with or without Apron seed treatment or between inoculated and noninoculated treatments were significantly different.

Statistical analyses were performed using JMP Vers. 5.1 (SAS Institute Inc. 2004). The Restricted Maximum Likelihood Method (REML) was used to calculate the analysis of variance for the split-split plot experimental design. The effects tested were block, cultivar, inoculation treatments, fungicide treatments, and the interactions of inoculation × fungicide, cultivar × fungicide, and cultivar × inoculation × fungicide. Block × cultivar and block × inoculation [cultivar] (block × inoculation nested within cultivar) were random effects and error terms. *F* tests were done using the block ×

cultivar variance component to test the effects of block and the main plot cultivar, the block × inoculation [cultivar] variance component was used to test the effect of subplot inoculation treatment and the cultivar × inoculation interaction, and the residual was used to test the sub-subplot fungicide treatment effect and remaining interaction effects. The significance of differences between the treatments in orthogonal comparisons using contrasts were determined with Student's *t* tests. Correlation analysis was performed using the multivariate pairwise correlation procedure in JMP vers. 5.1 to determine the relationships between plant stand, plant height, and yield.

RESULTS

Confirmation of Race-Specific Resistance

Using the hypocotyl injection inoculation technique (Keeling, 1976), all cultivars were resistant to *P. sojae* race 1 except FS2105 and Williams. All cultivars were susceptible to *P. sojae* Races 28 and 30, except AG3302. These results confirmed the presence of the *Rps1k* gene in the test set of cultivars that were reported to have the gene (Table 1) and that AG3302 possessed the *Rps1c* gene.

Greenhouse Evaluation of Commercial Cultivars for Partial Resistance

Analysis of the greenhouse measurements indicated that the effects of *P. sojae* race and *P. sojae* race × cultivar interaction were not significant for any of the measurements; therefore, the data were summarized across races of *P. sojae* (Table 2). Single degree of freedom comparisons of control vs. inoculated treatments using contrast statements revealed nonsignificant differences for plant height, root mass, and top mass for AG3302, which has the *Rps1c* gene. Among the cultivars with *Rps1k*, differences between control and inoculated treatments for top and root mass of DKB3151 and for root mass of GR3101 and GR3331 were not significant (Table 2).

The mean percentage of control of inoculated plants was 76% for top mass, 67% for root mass, and 75% for plant height (Table 2). There was considerable variability in root mass, top mass, and plant height measurements. The percentage of control exceeded 100% in some cases. However, despite the high variability, there were significant differences among the cultivars for percentage of control for each of the greenhouse measurements.

All of the *Rps1k* commercial cultivars had reductions in top mass and plant height, relative to the control (percentage), and disease ratings that were not significantly different from the partial resistant check Conrad (Table 2). Two of the 14 susceptible commercial *Rps1k* cultivars, P92B84 and P93B09, had significantly lower root mass relative to the control than Conrad (Table 2). The susceptible check Essex had significantly greater reduction in top mass, root mass, and plant height than Conrad (Table 2).

Table 2. Reactions of 16 private and four public soybean cultivars, after inoculation with *Phytophthora sojae* races 28 and 30, for plant height, root and top mass, root disease rating, and plant disease rating, compared with noninoculated controls in a greenhouse experiment using six replications of five plants (experimental unit) per cultivar. Data are summarized across both *Phytophthora sojae* races 28 and 30.

	Top mass‡ (mg)			Root mass§ (mg)			Plant height¶ (cm)				
Cultivar†	Control	Inoculated	% Control	Control	Inoculated	% Control	Control	Inoculated	% Control	Root disease rating# (0–5)	Plant disease rating†† (1–10)
AG3201	266	222*	84	131	98**	77	13	11*	84	1.8	3.8
AG3302	220	199	91	119	127	107	12	11	92	0.4	1.5
Conrad	184	125**	83	91	61*	84	12	8**	77	2.8	5.2
DKB3151	209	184	90	100	86	92	12	9**	76	2.6	5.4
DSR199	211	153**	73	117	65**	58	12	9**	75	2.9	5.3
DSR241	210	153**	73	119	72**	61	11	8**	75	3.1	5.4
DSR297	234	188*	81	103	70**	69	13	10**	78	2.3	4.8
DSR321	239	146**	67	108	58**	60	13	9**	77	3.5	6.3
DSR322	262	176**	68	109	64**	60	14	10**	71	3.3	6.0
Essex	257	167**	66	133	70**	54	13	8**	62	3.3	5.8
FS2105	239	191**	80	146	91**	63	12	9**	77	2.5	5.0
GH3135	215	175*	82	130	82**	63	11	9*	81	2.3	4.6
GR3101	238	176**	75	118	95	81	13	10**	72	2.7	5.3
GR3331	240	188*	79	103	80	80	13	11**	81	2.0	4.2
P92B84	223	139**	68	121	59**	53	12	8**	71	3.9	6.6
P93B01	176	132*	76	93	62*	67	10	7**	73	3.3	5.8
P93B09	229	160**	71	126	69**	56	12	8**	72	3.2	5.6
P93B36	263	184**	71	131	74**	58	13	9**	70	3.1	6.0
Williams	299	196**	81	166	76**	56	13	8**	69	2.8	5.3
Williams 82	275	178**	65	139	68**	49	13	9**	69	3.1	5.9
Mean	234.4	171.4	76.1	120.2	76.2	67.5	12.4	9.0	75.1	2.7	5.2
LSD (0.05)	46.6	46.6	16.3	27.8	27.8	26.8	2.08	2.08	14.4	1.3	1.7

* Indicates significance of differences of single degree of freedom comparisons at $P \leq 0.05$ between inoculated and noninoculated pots within each cultivar.
** Indicates significance of differences of single degree of freedom comparisons at $P \leq 0.01$ between inoculated and noninoculated pots within each cultivar.
† All private cultivars had the *Rps1k* gene except AG3302 (*Rps1c* gene) and FS2105 (no known *Rps* genes), verified by the hypocotyl inoculation method. Controls were Conrad (high partial resistance), Williams 82 (*Rps1k*), Williams (susceptible), and Essex (susceptible).
‡ Dry top mass was the mean weight of plants above the soil line.
§ Dry root mass was the mean weight of plants below the soil line.
¶ Plant heights were the mean height from the soil line to the apex.
# Root disease rating: 0 = no visible root mass loss compared with the noninoculated control, 1 = 1–10% root mass loss, 2 = 11–35% root mass loss, 3 = 36–65% root mass loss, 4 = 66–90% root mass loss, 5 = 90–100% root mass loss.
†† Plant disease rating (Schmitthenner et al., 1994): 1 = no root rot, 2 = trace of root rot, 3 = bottom third of root mass rotted, 4 = bottom 2/3 of mass rotted, 5 = all roots rotted, 10% seedling kill, slight stunting of tops of plants, 6 = 50% seedling kill, moderate stunting of tops, 7 = 75% seedling kill, severe stunting of tops, 8 = 90% seedling kill, 9 = all seedlings dead, 10 = all seedlings killed before emergence. Complete race-specific resistance interactions typically receive a rating of 1 or 2.

Reduction of the top mass of seven of the *Rps1k* cultivars was not significantly different from the resistant check AG3302, and two from that group of seven, DKB3151 and GR3101, had root mass reduction not significantly different from AG3302 (Table 2). All of the *Rps1k* cultivars had significantly greater reductions in plant height and significantly higher root and whole plant disease ratings than AG3302 (Table 2).

All greenhouse measurements were significantly correlated with each other, with correlation coefficients (r) ranging from 0.27 for the correlation between plant height and root mass to 0.86 for the correlation between the root and the plant disease ratings. Root mass, top mass, and plant heights were positively correlated with each other and were negatively correlated with root and plant disease ratings.

Field Experiments

There were no significant fungicide × inoculation × cultivar interactions for yield in any of the field experiments (Tables 3, 4, and 5). This indicated that the cultivars did not interact differentially between inoculation and between fungicide treatments. Therefore, differences between the sub-subplot treatments, Apron-treated, and untreated seed within inoculation treatments for each cultivar were not found. There was a significant fungicide × inoculation × cultivar interaction for plant stand in Exp. 1 in 2002 (Table 3), indicating that the plant stand of cultivars differed depending on the inoculation and fungicide treatment applied.

A significant fungicide × inoculation interaction for yield was found in Exp. 1 in 2003 (Table 4) and in Exp. 1 in 2004 (Table 5). Orthogonal comparisons indicated that Apron seed treatment significantly increased yield in inoculated treatments, while it had no effect on yield in noninoculated treatments. Apron seed treatment boosted yield in Race 28 inoculations by 232 kg/ha or 12% and by 137 kg/ha or 7% in Race 30 inoculations in Exp. 1 in 2003 and by 137 kg/ha or 4% and 202 kg/ha or 6% in *P. sojae* Race 28 and Race 30 inoculations, respectively, in Exp. 1 in 2004 (data not shown). There was a significant fungicide × inoculation interaction for plant height in Exp. 2 in 2003 (Table 4). Inoculation with *P. sojae* Race 28 and Race 30 significantly reduced plant height by 9 and 8%, respectively, without Apron seed treatment, while Apron had no effect on plant height without inoculation (data not shown).

No significant interaction between fungicide treatments and cultivars for yield was found in any of the field experiments (Tables 3–5). Apron seed treatment did not differentially affect the yield performance of the cultivars. However, there was a significant fungicide × cultivar interaction for plant height in Exp. 2 in 2002 (Table 3), Exp. 2 in 2003 (Table 4), and Exp. 2 in 2004 (Table 5). The growth response of later maturing cultivars was more variable to the Apron seed treatment than the growth response of the earlier maturing cultivars in this study.

Effects of sub-subplot fungicide treatments were significant in five of the six field experiments. Differences among the Apron seed fungicide and the untreated sub-subplot treatments for yield were significant without interactions with other treatments in Exp. 2 in 2002 (Table 3), Exp. 2 in 2003 (Table 4), and Exp. 2 in 2004 (Table 5). Apron seed treatment boosted yields by 6% or 80 kg/ha Exp. 2 in 2002, 7% or 153 kg/ha in Exp. 2 in 2003, and 2% or 80 kg/ha in Exp. 2 in 2004 (data not shown).

Table 3. Analysis of variance of the effects of cultivar, *Phytophthora sojae* inoculation treatments, seed fungicide treatment, and their interactions on plant stand, plant height, and yield in two field experiments with eight different commercial glyphosate tolerant soybean cultivars in each experiment conducted in Urbana, IL, in 2002.

Experiment	Source of variation†	df	Plant stand	Plant height	Yield
1	Block	3	2.44	11.64***	30.82***
	Cultivar	7	0.99	9.46***	3.99**
	Block × cultivar&random	21			
	Inoculation	2	2.13	3.61*	1.48
	Noninoculated vs. inoculated	1		0.30	
	Race 28 vs. Race 30	1		2.67*	
	Cultivar × inoculation	14	0.52	0.92	1.27
	Block × inoculation[cultivar]&random	48			
	Fungicide	1	0.00	0.02	0.07
	Fungicide × cultivar	7	0.72	1.16	0.55
	Fungicide × inoculation	2	1.30	2.54	0.20
	Fungicide × inoculation × cultivar	14	1.94*	1.05	1.31
2	Block	3	4.25*	17.47***	14.48***
	Cultivar	7	1.85	7.73***	5.21**
	Block × cultivar&random	21			
	Inoculation	2	1.41	1.32	5.06*
	Noninoculated vs. inoculated	1			2.92**
	Race 28 vs. Race 30	1			1.26
	Cultivar × inoculation	14	0.72	0.78	1.01
	Block × inoculation[cultivar]&random	48			
	Fungicide	1	2.53	8.05**	18.72***
	Fungicide × cultivar	7	1.74	2.71*	1.89
	Fungicide × inoculation	2	0.41	1.08	0.10
	Fungicide × inoculation × cultivar	14	0.60	0.69	0.89

* Significant at $P \leq 0.05$.
** Significant at $P \leq 0.01$.
*** Significant at $P \leq 0.001$.
† The Restricted Maximum Likelihood (REML) method was used to calculate the analysis of variance for the split-split plot experimental design with four randomized complete blocks in JMP vers. 5.1.

Table 4. Analysis of variance of the effects of cultivar, *Phytophthora sojae* inoculation treatments, seed fungicide treatment, and their interactions on plant stand, plant height, and yield in two field experiments with eight different commercial glyphosate tolerant soybean cultivars in each experiment conducted in Urbana, IL, in 2003.

Experiment	Source of variation†	df	Plant stand	Plant height	Yield
1	Block	3	9.62***	0.53	1.75
	Cultivar	7	4.10**	1.80	3.53*
	Block × cultivar&random	21			
	Inoculation	2	28.2***	21.06***	7.28**
	Noninoculated vs. inoculated	1	7.41***	6.38***	3.76***
	Race 28 vs. Race 30	1	0.22	1.17	0.62
	Cultivar × inoculation	14	0.96	1.50	2.13*
	Block × inoculation[cultivar]&random	48	1.46		
	Fungicide	1	7.38**	26.97***	6.47*
	Fungicide × cultivar	7	0.57	0.73	0.56
	Fungicide × inoculation	2	0.78	1.28	4.83*
	Noninoculated with Apron vs. noninoculated without Apron	1			0.94
	Inoculated with Apron vs. inoculated without Apron	1			3.78***
	Fungicide × inoculation × cultivar	14	0.80	0.58	0.70
2	Block	3	5.77**	0.92	6.55**
	Cultivar	7	3.40*	2.90*	0.79
	Block × cultivar	21			
	Inoculation	2	36.76***	31.01***	16.45***
	Noninoculated vs. inoculated	1	8.57***	7.76***	5.60***
	Race 28 vs. Race 30	1	0.05	1.31	1.25
	Cultivar × inoculation	14	1.19	0.96	0.79
	Block × inoculation[cultivar]	48			
	Fungicide	1	38.63***	63.86***	11.62**
	Fungicide × cultivar	7	1.08	2.25*	0.26
	Fungicide × inoculation	2	0.92	8.37***	2.91
	Noninoculated with Apron vs. noninoculated without Apron	1		1.31	
	Inoculated with Apron vs. inoculated without Apron	1		8.86***	
	Fungicide × inoculation × cultivar	14	1.28	0.90	0.16

* Significant at $P \leq 0.05$.
** Significant at $P \leq 0.01$.
*** Significant at $P \leq 0.001$.
† The Restricted Maximum Likelihood (REML) method was used to calculate the analysis of variance for the split-split plot experimental design with four randomized complete blocks in JMP vers. 5.1.

Significant differences in plant height between the Apron and untreated seed treatments were found in Exp. 1 in 2003 (Table 4) and Exp. 1 in 2004 (Table 5) and for plant stand in Exp. 1 and 2 in 2003 (Table 4) and in Exp. 1 in 2004 (Table 5) in the absence of significant interactions with other treatments. The Apron seed treatment significantly increased plant height by 5% in Exp. 1 in 2003 and 2% in Exp. 1 in 2004 and plant stand by 7, 19, and 2% in Exp. 1, 2003, Exp. 2, 2003, and Exp. 1, 2004, respectively (data not shown).

There was a significant cultivar × inoculation treatment interaction ($P < 0.05$) for yield in Exp. 1 in 2003 (Table 4). Race 30 reduced yields of FS2105 and DSR241 more than Race 28, while Race 28 reduced yields of the six other cultivars more than Race 30. Orthogonal comparisons of inoculation treatments within cultivars made using contrasts indicated that there were significant differences in yield between the noninoculated control and inoculated treatments for four of the eight cultivars (Table 6), indicating that they were nontolerant to *P. sojae* infection, while the other four cultivars suffered no significant yield reduction due to inoculation and appeared to be PRR tolerant using this analysis. There were no significant differences between Race 28 and Race 30 inoculated treatments for any of the cultivars.

The effect of the subplot inoculation treatments was significant in three of the six field experiments (Tables 3–5). Where there were no significant interactions between inoculation treatments and the other main effects, significant differences between the inoculation treatments were found in Exp. 2 in 2002 (Table 3) and Exp. 2 in 2003 (Table 4). Differences between noninoculated and inoculated treatments were significant in both experiments, while the differences between Race 28 and Race 30 inoculations were not significant. *Phytophthora sojae* inoculations reduced yield by 93 kg/ha or 6% and 56 kg/ha or 4%, with Race 28 and Race 30, respectively, in Exp. 2 in 2002. In Exp. 2 in 2003, inoculations reduced yield by 421 kg/ha or 17% and 325 kg/ha or 13%, with Race 28 and Race 30, respectively.

There were significant differences among cultivars in Exp. 1 and 2 in 2002 (Table 3) and Exp. 1 and 2 in 2004 (Table 5) without significant interactions with other treatments. Mean yields were 1225 and 1515 kg/ha in Exp. 1 and 2 in 2002, respectively (data not shown). In 2004, mean yields were 3309 and 3405 kg/ha in Exp. 1 and 2, respectively (data not shown).

Yield was significantly correlated with plant height in 2002 through 2004 (Table 7), and was significantly correlated with plant stand in 2002 and 2003 but not in 2004. Plant height was correlated with plant stand in all three years.

DISCUSSION

Soybean plants that have complete resistance provided by the race-specific *Rps* genes were identified by the hypocotyl injection inoculation procedure. The hypocotyl inoculations in our study confirmed the presence of *Rps1k* in 14 private cultivars and Williams 82, *Rps1c* in AG3302, and no known *Rps* genes in FS2105. Plants that limit the colonization or damage caused by a compatible

Table 5. Analysis of variance of the effects of cultivar, *Phytophthora sojae* inoculation treatments, seed fungicide treatment, and their interactions on plant stand, plant height, and yield in two field experiments with four different commercial glyphosate tolerant soybean cultivars in each experiment conducted in Urbana, IL, in 2004.

Experiment	Source of variation†	df	Plant stand	Plant height	Yield
1	Block	5	0.94	0.68	2.66
	Cultivar	3	2.49	33.23***	58.40***
	Block × cultivar	15			
	Inoculation	2	8.47***	14.60***	1.51
	Noninoculated vs. inoculated	1	4.12***	5.07**	
	Race 28 vs. Race 30	1	0.07	0.07	
	Cultivar × inoculation	6	0.55	0.93	1.96
	Block × inoculation[cultivar]&random	40			
	Fungicide	1	0.84	9.81**	31.65***
	Fungicide × cultivar	3	0.94	2.31	1.74
	Fungicide × inoculation	2	2.25	1.16	3.29*
	Noninoculated with Apron vs. noninoculated without Apron	1			1.37
	Inoculated with Apron vs. inoculated without Apron	1			5.91***
	Fungicide × inoculation × cultivar	6	1.05	1.76	2.21
2	Block	5	2.57	2.25	3.18*
	Cultivar	3	2.25	8.7**	8.40**
	Block × cultivar&random	15			
	Inoculation	2	25.2***	9.28***	0.21
	Noninoculated vs. inoculated	1	6.75***	4.01***	
	Race 28 vs. Race 30	1	2.20*	1.56	
	Cultivar × inoculation	6	1.23	0.19	0.84
	Block × inoculation[cultivar]&random	40			
	Fungicide	1	4.01*	4.90*	7.12**
	Fungicide × cultivar	3	1.46	3.75*	1.60
	Fungicide × inoculation	2	0.28	1.09	0.75
	Fungicide × inoculation × cultivar	6	0.55	1.94	0.99

* Significant at $P \leq 0.05$.
** Significant at $P \leq 0.01$.
*** Significant at $P \leq 0.001$.
† The Restricted Maximum Likelihood (REML) method was used to calculate the analysis of variance for the split-split plot experimental design with six randomized complete blocks in JMP vers. 5.1.

P. sojae isolate have partial resistance. All cultivars with *Rps1k* were compatible with Races 28 and 30, enabling the measurement of partial resistance in those cultivars. AG3302, that had *Rps1c*, was incompatible with Races 28 and 30 and served as a resistant check in the tests.

The approach to evaluate partial resistance in this study was similar to the approach used in other research (Dorrance et al., 2003). This study focused on measuring partial resistance in compatible private commercial cultivars possessing the same defeated *P. sojae* race-specific complete resistance gene, *Rps1k*. Differences between inoculations with Races 28 and 30 and noninoculated controls for plant height, root and top mass, and ratings of root and plant symptoms identified both the race-specific resistance conferred by *Rps1c* in AG3302 and differences in compatible cultivars (Table 2). Partial resistance in the compatible cultivars was estimated by comparing the percentage of control among the cultivars for each of the greenhouse measurements. Significant differences in percentage of control indicated that there were differences among the compatible cultivars in the ability to limit PRR symptom development and the damage caused by *P. sojae* colonization in the absence of the protection conferred by *Rps1c* (Table 2). Cultivars DKB3151, GR3101, and GR3333 with *Rps1k* appeared to have a relatively high level of partial resistance. This result suggests that differences in partial resistance may underlie complete resistance. Partial resistance may contribute, along with complete resistance genes, to the overall resistance of a genotype.

Buzzell and Anderson (1982) proposed combining partial resistance and *Rps* genes to provide long term management of *P. sojae* and avoid breakdown of race-specific resistance. Our results demonstrated that partial resistance occurred or coexisted with race-specific resistance after challenging plants with compatible isolates that defeat race-specific resistance genes known to exist in the cultivars. Measurements of reductions in top mass and plant height, relative to controls, and disease ratings (Table 2) indicated that all of the commercial cultivars with *Rps1k* had levels of partial resistance comparable with Conrad, a public cultivar previously reported to have a high level of partial resistance in tests using the inoculum layer technique (Burnham et al., 2003; Dorrance et al., 2003). The private seed companies that developed the cultivars tested in our study appeared to have been successful in developing soybean cultivars with both race-specific, complete resistance, and nonspecific partial resistance. The combination of partial resistance with *Rps1k* and other *Rps* genes may have helped increase the durability of resistance in current cultivars apparent in most soybean production areas.

Individual defeated race-specific, complete resistance genes can have residual activity that contributes to partial resistance (Brodney et al., 1986). Some of the cultivars in this study possessing defeated *Rps1k* had a higher level of resistance than FS2105, a private cultivar that had no known *Rps* genes. However, Williams 82, which possesses *Rps1k*, was less resistant than isoline Williams, which has no known *Rps* genes. In addition, Conrad, with no known *Rps* genes, had a relatively high level of partial resistance. Therefore, there was no strong evidence of residual activity on infection expressed by *Rps1k* in this study. This is in agreement with the results

Table 6. Effect† of the *Phytophthora sojae* inoculation treatments, noninoculated control, race 28, and race 30, on the yields of eight commercial glyphosate soybean cultivars in Experiment 1 in 2003 in Urbana, IL.

Cultivar	Inoculation treatment	Yield (kg/ha)	t ratio‡
DSR199	Noninoculated control	2635	
	Race28	2113	
	Race30	2317	
	Noninoculated vs. inoculated		2.45*
	Race 28 vs. Race 30		1.03
DSR241	Noninoculated control	1957	
	Race28	1488	
	Race30	1206	
	Noninoculated vs. inoculated		3.57***
	Race 28 vs. Race 30		1.43
DSR297	Noninoculated control	1907	
	Race28	2210	
	Race30	2324	
	Noninoculated vs. inoculated		2.10*
	Race 28 vs. Race 30		0.56
FS2105	Noninoculated control	2311	
	Race28	2335	
	Race30	2067	
	Noninoculated vs. inoculated		0.64
	Race 28 vs. Race 30		1.36
GR3101	Noninoculated control	2445	
	Race28	1850	
	Race30	2248	
	Noninoculated vs. inoculated		2.31*
	Race 28 vs. Race 30		2.01
GR3331	Noninoculated control	2530	
	Race28	2240	
	Race30	2224	
	Noninoculated vs. inoculated		1.74
	Race 28 vs. Race 30		0.08
P92B84	Noninoculated control	2548	
	Race28	2369	
	Race30	2533	
	Noninoculated vs. inoculated		0.57
	Race 28 vs. Race 30		0.83
P93B01	Noninoculated control	2501	
	Race28	2232	
	Race30	2265	
	Noninoculated vs. inoculated		1.47
	Race 28 vs. Race 30		0.17

* Significant at $P \leq 0.05$.
** Significant at $P \leq 0.01$.
*** Significant at $P \leq 0.001$.
† The Restricted Maximum Likelihood (REML) method was used to calculate the analysis of variance for the split-split plot experimental design with four randomized complete blocks in JMP vers. 5.1.
‡ The significance of differences between the treatments in the single degree of freedom comparisons using contrasts were determined with Student's *t* tests.

where no expression of residual resistance was found from defeated *Rps1, Rps3, Rps4, Rps5*, and *Rps6* genes in near-isogenic soybean lines after challenging them with compatible *P. sojae* Race 7 (Young et al., 1994).

Lack of significant cultivar × race interaction for partial resistance indicated that there was no specificity toward *P. sojae* Races 28 and 30. Previous reports are in agreement with this lack of specificity (Schmitthenner and Walker, 1979). In contrast, stability of root resistance and race-specific slow rotting mechanisms were found to be dependent on the level of pathogenic variability in the *P. sojae* soil population and the rates of recombination on the soybean roots (McBlain et al., 1991).

Table 7. Pairwise correlations (*r*) of yield, plant height, and plant stand in field tests inoculated with *Phytophthora sojae* in Urbana, IL, in 2002–2004. Number of observations in 2002 and 2003 was 384, and number of observations in 2004 was 288.

	2002	2003	2004
Comparison	*r*	*r*	*r*
Yield and plant height	0.73***	0.65***	0.75***
Yield and plant stand	0.32***	0.18***	−0.03
Plant stand and plant height	0.17***	0.29***	0.12*

* Significant at $P \leq 0.05$.
** Significant at $P \leq 0.01$.
*** Significant at $P \leq 0.001$.

Level of *P. sojae* colonization in the greenhouse was measured indirectly in this study. Direct measurement of infection could be accomplished by enumeration of the pathogen (McBlain et al., 1991), recording the length of rotted tissue, or determining the total oospore production in infected tissue, after inoculation with compatible isolates (Slusher and Sinclair, 1973) and may predict field performance better than the indirect measurements used in this study. Lack of correlations between inoculum layer and slant board tests using *P. sojae* Race 4 in the greenhouse with tolerance scores in a field naturally infested with *P. sojae* were attributed to the ability of greenhouse tests to measure seedling tolerance but not field tolerance (McBlain et al., 1991). The value of greenhouse tests may only be in identifying soybean genotypes that can withstand early season *P. sojae* infection. Root disease ratings were the simplest measurement to collect in this study and appeared to be as useful as any of the other measurements taken (Table 2).

Field tolerance to *P. sojae* in a susceptible cultivar lacking complete resistance *Rps* genes was determined by comparing its yield in inoculated treatments with or without Apron seed treatment or between inoculated and noninoculated treatments. A cultivar was considered to have tolerance if the yield differences were not significant. Fungicide treatments have previously been used to aid in the identification of field tolerance (Anderson and Buzzell, 1982; Dorrance et al., 2003; Schmitthenner, 1985).

All of the commercial glyphosate cultivars used in this study were advertised by the seed companies to be moderate to highly tolerant to *P. sojae* infection, except for AG3302, advertised to have low tolerance but possesses *Rps1c,* and GH3135, that had no information (Table 1). Because no significant cultivar × inoculation × fungicide interaction for yield was found in any of the field experiments, despite the significance of differences between inoculation treatments in three of the six field experiments and between fungicide treatments in five of the six field experiments (Tables 3–5), all of the cultivars were apparently tolerant using the comparison between the inoculated Apron seed treatment and the inoculated treatment without Apron seed treatment sub-subplot treatments as the basis for determining tolerance. Significant differences between Apron treatments in inoculated plots would have identified nontolerant cultivars and nonsignificant differences for tolerant cultivars. Perhaps Apron seed treatment did not have a strong enough effect on yields in inoculated plots to produce significant differences, although, overall, including noninoculated plots, there was a significant effect, possibly because the Apron was controlling other pathogens, such as *Pythium* spp., that might have affected yields.

In Exp. 1 in 2003 (Table 4), there was a significant cultivar × inoculation interaction for yield, indicating that

there were differences in the responses of the cultivars with the inoculation treatments for yield. Although there was no nontolerant check included in this experiment for comparison, single degree of freedom comparisons of the yields of cultivars in inoculated vs. noninoculated treatments (Table 6) indicated that cultivars FS2105, GR3331, P93B09, and P93B36 possess PRR tolerance as advertised, while other cultivars failed to demonstrate the advertised tolerance. This approach to evaluating tolerance may be a better indicator of tolerant cultivars than a comparison with a suitable nontolerant check.

Classical definitions in plant pathology literature generally distinguish tolerant from nontolerant plant genotypes by their relative productivity when equally diseased. Tolerance to *P. sojae* had been defined as the ability of a soybean genotype to remain productive even under severe disease pressure (St. Martin et al., 1994). The amount of disease in the field experiments was not directly measured in this study. Therefore, the appearance of tolerance in this study may actually have been due to unequal levels of *P. sojae* colonization on the cultivars. Level of *P. sojae* infection or plant damage caused by the pathogen may have been limited by the expression of partial resistance to *P. sojae* infection in the field. Indeed, according to the definition of partial resistance used in this study and four of the five greenhouse measurements taken, all of the *Rps1k* commercial cultivars tested had partial resistance that appeared to be equal to Conrad, the partial resistant check (Table 2). However, a comparison of partial resistance determinations in the greenhouse and field was not possible without measurement of infection or damage caused by infection in the field experiments. Commercial soybean breeders may be directly or indirectly selecting for increased tolerance or partial resistance in breeding nurseries, especially if there is strong pressure of *P. sojae* to lower yields.

A possible mechanism of soybean PRR tolerance may be the ability of soybean plants to compensate and recover from early season root damage if environmental conditions nonconducive to PRR development occur later in the growing season. Through aggressive branching, soybean plants can compensate for a loss of 50% of the stand and produce 90% of the yields in full stands (Berglund, 2003). Plants resistant to *P. sojae* infection were shown to compensate for the loss of stand in blends with susceptible lines (Wilcox and St. Martin, 1998). In this study, yield was highly significantly correlated with plant stand in 2002 and 2003 (Table 7), suggesting that stand loss compensation may not have been a major factor involved in the response to *P. sojae* infection in the field.

The significant correlation between yield and plant height in this study (Table 7) suggests plant height measurements taken at growth stage R6 could be used in addition to yield data to determine the response to *P. sojae* infection in the field. Soybean breeders could exploit this relationship to efficiently screen large numbers of breeding lines for partial resistance or tolerance to *P. sojae* infection using suitable infected and noninfected controls.

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