Microplot Comparison of Rate-Reducing and Race-Specific Resistance to *Phytophthora megasperma* f. sp. *glycinea* in Soybean

P. W. Tooley and C. R. Grau

Former graduate research assistant and associate professor, respectively, Department of Plant Pathology, University of Wisconsin, Madison 53706. Present address of first author: U.S. Department of Agriculture, Agricultural Research Service, Foreign Disease-Weed Science Research Unit, Fort Detrick Building 1301, Frederick, MD 21701.
Research supported by the College of Agricultural and Life Sciences, University of Wisconsin-Madison and by Hatch Project 2665.
Accepted for publication 9 December 1985.

ABSTRACT

Tooley, P. W., and Grau, C. R. 1986. Microplot comparison of rate-reducing and race-specific resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathology 76:554-557.

Microplots were artificially infested with races 1, 3, and 4 of *Phytophthora megasperma* f. sp. *glycinea* alone and in all possible combinations. Five soybean (*Glycine max*) cultivars containing different levels of rate-reducing resistance alone or in combination with race-specific resistance to *P. m.* f. sp. *glycinea* were grown in microplots for 3 yr in succession. Cultivars with high levels of rate-reducing resistance sustained low disease levels regardless of the race or race combination of *P. m.* f. sp. *glycinea*. In microplots infested with incompatible races of the fungus, specific resistance was not significantly more effective than a high level of rate-reducing resistance in all 3 yr. Also, race-specific resistance was ineffective in the presence of race combinations that contained a proportion of compatible races of *P. m.* f. sp. *glycinea*. Since diverse races of the pathogen now exist over wide areas, our results indicate that a high level of rate-reducing resistance offers the best means of stable, long-term control of soybean root and stem rot.

We have reported (23–25) the performance of soybean cultivars with rate-reducing resistance to *Phytophthora megasperma* Drechs. f. sp. *glycinea* Kuan and Erwin. We observed that rate-reducing resistance tends to remain effective in diverse field environments. In contrast, race-specific resistance to *P. m.* f. sp. *glycinea*, although effective initially, can be overcome by new races of the pathogen (7–10,12,15,16,18).

Use of race-specific resistance is considered by some soybean breeders to be effective for the control of root and stem rot caused by *P. m.* f. sp. *glycinea* (1–3). In particular, the incorporation of multiple alleles for resistance to the pathogen into a single soybean genotype offers promise for control of root and stem rot caused by diverse races of *P. m.* f. sp. *glycinea* (2).

Other workers (5,16,27,28) have evaluated rate-reducing (nonrace-specific) resistance to *P. m.* f. sp. *glycinea* and have attempted to incorporate both types of resistance into commercial soybean cultivars. However, quantitative information on the stability and relative efficacy of race-specific and rate-reducing resistance is lacking. Such information is needed by soybean breeders and plant pathologists to provide the basis for development of improved disease management strategies for Phytophthora root and stem rot.

Studies of the effectiveness of the two types of resistance in controlling the disease have been reported (5,14,17,21) but these include limited information on the race composition of the pathogen. Specific resistance is active against only certain races of the pathogen. Conversely, rate-reducing resistance is assumed to act equally against all pathogen races (13), but this assumption has not been tested in field studies with *P. m.* f. sp. *glycinea*. Detailed knowledge of race composition is necessary for evaluating the stability of rate-reducing resistance and for quantitative comparison of the two types of resistance.

Our objective was to use microplots artificially infested with known races and race combinations of *P. m.* f. sp. *glycinea* to evaluate the stability of rate-reducing resistance and to accurately compare the efficacies of race-specific and rate-reducing resistance to *P. m.* f. sp. *glycinea*.

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MATERIALS AND METHODS

Cultivar selection. Cultivars represented several maturity groups with or without race-specific resistance to races 1 and 2 conferred by the Rps_1 allele or to races 1–3, 6–11, 13, 15, 17, and 21 conferred by the Rps_1^c allele (12). The cultivars, their maturity groups (MG), race-specific genes for resistance to *P. m.* f. sp. *glycinea*, and levels of rate-reducing resistance (24) are as follows: Steele, MGI having Rps_1 and low rate-reducing resistance; Asgrow A2656, MGII having Rps_1 and high rate-reducing resistance; Wells II, MGII having Rps_1^c and intermediate rate-reducing resistance; Corsoy, MGII having no race-specific resistance and low rate-reducing resistance; Wayne, MGIII having no race-specific resistance and high rate-reducing resistance.

Preparation of inoculum. Isolates of *P. m.* f. sp. *glycinea* were obtained from diseased soybeans collected in southeastern Wisconsin and races were identified as described previously (26). Four isolates each of races 1, 3, and 4 were grown on V8-juice agar in 9-cm-diameter petri dishes for 7 days at room temperature. Twenty-one petri dishes of each isolate were comminuted in a blender at high speed for 10 sec and hand-mixed with 3,000 cc of sterile vermiculite. Inocula of isolates of the same race were combined and mixed thoroughly with about 8,000 cc of a sterilized mixture of loam soil, sand, and peat (2:1:1, v/v). Inoculum combinations were then produced to yield all possible single-race and multiple-race treatment combinations.

Microplot design and infestation. Microplots were located at Arlington, WI, in a field with no previous history of soybean cultivation and 15 yr of continuous corn before this study. Microplots were circular, 1 m in diameter, and bordered by a 25-cm-wide fiberglass strip buried edgewise in the ground to a depth of 13 cm. Adjacent microplots were separated by 3 m of fallow ground.

Inoculum (750 cm^3) of each race or race combination was used to infest each of two 0.6-m rows 33 cm apart in each microplot. The inoculum was placed 2.5 cm deep and covered with 1.3 cm of field soil; 25 soybean seeds were planted in each row (50 seeds per microplot) on 14 June 1979, 29 May 1980, and 21 May 1981 and covered with 1.3 cm of field soil. In 1980 and 1981, microplots were

not reinfested with inoculum. Instead, soybean roots remaining from the previous season were left in the microplots and seeds were planted in rows just adjacent to the original seedbed rows.

The five soybean cultivars, seven races or race combinations of *P. m.* f. sp. *glycinea*, and a control treatment which consisted of 750 cm^3 of uninfested soil placed in the microplot rows yielded 40 treatments in a factorial experiment arranged in a randomized complete block design with three replications.

Disease assessment and yield determination. The incidence of plants killed by *P. m.* f. sp. *glycinea* in each microplot was assessed periodically throughout the growing season. Disease incidence was assessed six times in 1979 (at 9, 23, 38, 50, 68, and 83 days after planting) and three times in 1980 and 1981 (at 26, 47, and 81 days after planting in 1980 and at 18, 48, and 86 days after planting in 1981). Disease incidence data were used to calculate areas under the disease progress curve (AUDPC) and simple-interest infection rates (r_s) as described previously (24).

Microplots were hand-harvested at the end of the season with a small-plot thresher and yield was assessed as grams of seed per microplot. Yield was not assessed in 1981 because plants of some cultivars were killed by frost before maturity.

For each cultivar within each replication, percent yield reductions were calculated as follows: Yield reduction (%) = [(yield from control microplot − yield from infested microplot)/(yield from control microplot)] × 100.

To determine the relationship between disease measures and yield loss in 1979 and 1980, percent yield reduction was regressed on final disease incidence, AUDPC, and r_s.

Stability of rate-reducing resistance was evaluated and resistances were compared by formulating a nonorthogonal set of 18 single-degree-of-freedom contrasts among means (20, page 177). Contrasts were calculated separately for each year.

RESULTS

Disease incidences, AUDPCs, and infection rates of root and stem rot were similar in 1979 and 1980, but disease levels were higher in 1981 (Fig. 1, Table 1). The higher disease levels in 1981 were probably caused by the buildup of inoculum in the microplots during the previous 2 yr.

The highest levels of disease in all 3 yr developed on cultivars Steele and Corsoy, which have low levels of rate-reducing resistance (Fig. 1, Table 1). Cultivars Asgrow A2656 and Wayne, which have high levels of rate-reducing resistance, showed low disease levels relative to other cultivars (Fig. 1, Table 1). In all 3 yr, analysis of variance revealed highly significant ($P < 0.01$) effects due to cultivar, race treatment, and cultivar-race interaction for final disease incidence, AUDPC, and r_s.

In all 3 yr, Asgrow A2656 and Wayne (high rate-reducing resistance) showed significantly lower ($P < 0.01$) final disease incidence than Steele and Corsoy (low rate-reducing resistance) in the presence of race 3 alone, race 4 alone, and races 3 and 4 combined. Similarly, Asgrow A2656 and Wayne showed significantly lower disease incidence than did Steele, Corsoy, and Wells II in the presence of race 4 alone.

Disease incidence of cultivars in the presence of single races and two-race and three-race combinations were compared. Increasing the number of races did not increase the disease incidence for Wayne or Asgrow A2656.

In all 3 yr, Wayne did not differ significantly from Steele and Wells II in the presence of race 1. Similarly, Wayne and Asgrow A2656 did not differ significantly from Wells II in the presence of race 3. There were no significant differences in disease incidence between Wayne and Asgrow A2656 or between Wayne and Wells II in the presence of race 1. Similarly, Wayne did not differ significantly from Wells II in the presence of races 1 and 3 combined.

In all 3 yr, Wayne showed significantly lower disease incidence than Steele in the presence of races 1 and 3 combined, races 1 and 4 combined, and races 1, 3, and 4 combined. Wayne also showed significantly lower ($P < 0.05$) disease incidence than Wells II (intermediate rate-reducing resistance) in the presence of races 1, 3, and 4 combined but not in the presence of races 1 and 4 combined or races 3 and 4 combined.

Analyses identical to those above were performed for AUDPC and r_s and results were nearly identical to those obtained for final disease incidence.

In both 1979 and 1980, highly significant ($P < 0.01$) linear relationships were observed between yield reduction and final disease incidence, AUDPC, and r_s. Regression coefficients (slopes)

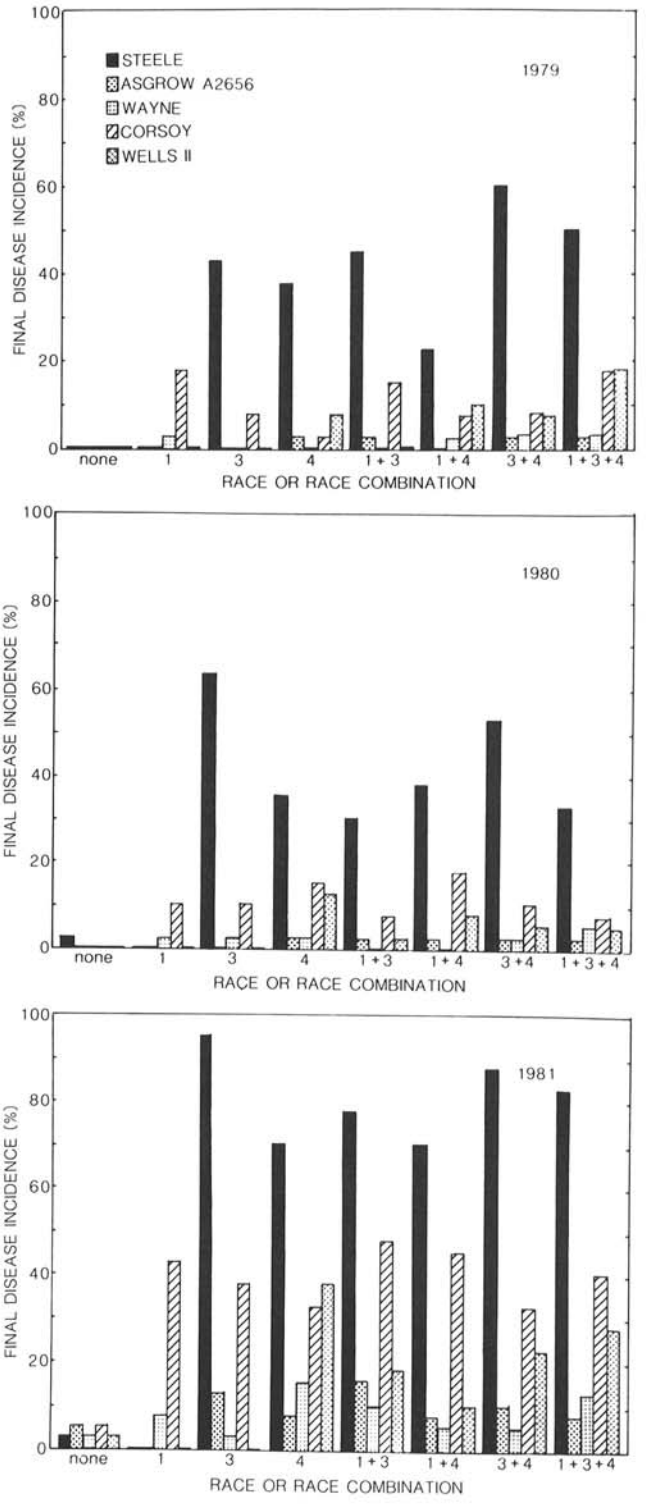

Fig. 1. Final disease incidence for five soybean cultivars grown in microplots artificially infested with seven races or race combinations of *Phytophthora megasperma* f. sp. *glycinea* in **A**, 1979; **B**, 1980; and **C**, 1981. Values of Bayes' least significant difference (BLSD) (19) for final disease incidence were 14.6, 9.8, and 18.7% in 1979, 1980, and 1981, respectively.

TABLE 1. Area under the disease progress curve (AUDPC) and simple interest infection rate (r_s) for five soybean cultivars grown in microplots artificially infested with seven races or race combinations of *Phytophthora megasperma* f. sp. *glycinea*

Year and cultivars	AUDPC[a] Race or race combination								r_s[a] (per day × 10^{-4}) Race or race combination							
	None	1	3	4	1 + 3	1 + 4	3 + 4	1 + 3 + 4	None	1	3	4	1 + 3	1 + 4	3 + 4	1 + 3 + 4
1979																
Steele	0[b]	0	16.9	16.6	18.4	11.2	24.5	21.2	0	0	84	78	93	39	138	123
Asgrow A2656	0	0	0.5	1.0	1.8	0.3	1.2	0.6	0	0	1	2	4	1	2	3
Wayne	0	2.1	0	0	0.5	1.4	1.9	2.0	0	3	0	0	1	2	3	4
Corsoy	0	9.8	3.8	0.9	8.9	4.2	4.8	9.8	0	26	8	2	20	8	12	24
Wells II	0	0	0	4.0	0.3	5.7	4.9	9.0	0	0	0	12	1	13	12	30
				BLSD[c] = 7.5								BLSD = 40				
1980																
Steele	0.4	0.4	23.5	9.3	10.0	11.2	17.6	11.6	3	1	158	81	58	82	121	65
Asgrow A2656	0.1	0.1	0.4	0.8	0.5	0.7	1.1	1.4	2	1	0	6	3	1	2	3
Wayne	0	0.7	0.7	1.3	0.1	0.4	0.9	1.9	0	6	1	1	1	2	1	6
Corsoy	0.1	4.5	3.7	6.4	2.5	8.3	3.3	3.6	1	10	14	20	9	19	12	11
Wells II	0	0	0	3.1	0.7	3.4	2.4	1.0	0	0	0	24	2	6	3	8
				BLSD = 3.2								BLSD = 27				
1981																
Steele	0.2	0	49.5	33.7	34.1	29.5	42.0	35.5	2	0	527	187	214	167	507	239
Asgrow A2656	1.6	0	4.9	2.1	6.9	2.4	3.6	3.4	5	0	18	12	23	12	17	11
Wayne	0.3	3.0	0.3	6.5	4.2	1.5	1.9	4.8	2	13	2	21	11	6	7	17
Corsoy	1.5	23.3	19.9	16.7	26.4	24.1	16.4	20.9	5	65	73	49	74	70	52	67
Wells II	0.8	0	0	16.8	6.2	4.8	7.9	12.3	2	0	0	64	26	12	35	40
				BLSD = 9.2								BLSD = 192				

[a] AUDPC and r_s (10^{-4}) values were calculated as described previously (24).
[b] Data are means of three replications.
[c] Bayes' least significant difference (k = 100) for comparing cultivar means within each race treatment and for comparing race treatment means within cultivars (19).

obtained by regressing percent yield reduction on final disease incidence were close to 1 in both years (b = 0.90 and 1.11 in 1979 and 1980, respectively), indicating that a 1% increase in final disease incidence resulted in approximately a 1% decrease in yield.

DISCUSSION

The results confirm that rate-reducing resistance offers a more stable means of control of soybean root and stem rot than does race-specific resistance. Cultivars containing high levels of rate-reducing resistance to *P. m.* f. sp. *glycinea* sustained low disease incidence for 3 yr in the presence of several different races and race combinations. Two-race and three-race combinations of the pathogen caused no more disease on these cultivars than did single races. These results are consistent with field observations (*unpublished*) that cultivars containing high levels of rate-reducing resistance suffer less disease and lower yield losses when grown in regions characterized by varied pathogen race composition. Our results also agree with previous results from growth chamber studies of cotyledon inoculation (23) which showed rate-reducing resistance to be expressed against four races of *P. m.* f. sp. *glycinea*.

Race-specific resistance was very unstable in the presence of mixed pathogen populations that contained a proportion of compatible races of *P. m.* f. sp. *glycinea*. Race-specific resistance was ineffective when about one-third of the initial pathogen population consisted of races capable of overcoming the resistance.

Cultivars with low rate-reducing resistance sustained severe plant mortality if they did not also contain specific resistance active against all races present in the microplot. Race-specific resistance was very effective in microplots infested only with incompatible races of *P. m.* f. sp. *glycinea*. However, cultivars that contained only high levels of rate-reducing resistance and no race-specific resistance suffered slightly, but not significantly, more disease than those that contained effective race-specific resistance.

Diverse races of *P. m.* f. sp. *glycinea* are now present throughout the major soybean-growing regions of the United States and Canada (4,6,11,26,29), and widely virulent races have been found in some regions new to soybean cultivation (22). Thus, the potential control offered by race-specific resistance may not be realizable in these areas. Also, the cost of breeding for race-specific resistance in addition to rate-reducing resistance may not be justified, given the unstable nature of race-specific resistance. If race-specific resistance is used, it should be combined with a high level of rate-reducing resistance to prevent extreme losses caused by new races of *P. m.* f. sp. *glycinea*.

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