Field Characterization of Rate-Reducing Resistance to *Phytophthora megasperma* f. sp. *glycinea* in Soybean

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ABSTRACT

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Field studies were performed in 1980 and 1981 to compare methods of quantifying rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycinea* (Pmg) and to determine how such resistance is expressed under field conditions. Eight variables that provided estimates of resistance were examined in 1980: metalaxyl was applied at four rates so that resistance could be expressed in terms of fungicide equivalency. Quadratic (rather than linear) equations best fit the disease intensity:metalaxyl rate response function. Moreover, cultivars showed significantly different quadratic responses to increasing metalaxyl application rates. Of 21 variables examined in 1981, 14 allowed the detection of significant cultivar differences in rate-reducing resistance. The variables most useful in differentiating cultivars were disease incidence at growth stages R3, R5, and R7; area under the disease progress curve; and disease severity rating at the R5 growth stage. These variables were highly correlated with one another, indicating that the incidence of dead or dying plants may be used to predict the internal disease severity in surviving plants. Most plants of cultivars showing the lowest disease incidence showed no internal discoloration of taproots and lower stems. Thus, resistance was characterized by the apparent ability of some cultivars to restrict the activity of Pmg in the taproot and lower stem tissue. As this phenomenon closely parallels that observed with the same cultivars in response to cotyledon inoculation, we believe that a primary component of rate-reducing resistance to Pmg is the ability to restrict fungal colonization of the plant tissue.

Many soybean cultivars without race-specific resistance to *Phytophthora megasperma* Drechs. f. sp. *glycinea* (Pmg) (15) (syn. *P. megasperma* Drechs. var. *sojae*) yield well and have few plants killed when grown in the presence of races to which they are susceptible via standard hypocotyl inoculation (26,27,34,38). Over the years, this phenomenon has variously been referred to as moderate resistance (38), field resistance (27), field tolerance (1,11), or simply tolerance (6,26) to distinguish it from race-specific resistance identified through hypocotyl inoculation (14–16).

Cultivars that exhibit this field phenomenon restrict tissue colonization by the pathogen in response to cotyledon inoculation (34). This, and the fact that reduced disease severity is associated with the yield increase reported for such cultivars, indicate that a form of resistance is being expressed which we have termed rate-reducing resistance (34).

Rate-reducing resistance can be evaluated in seedling assays (6,11,34). We found previously that estimates of such resistance obtained via cotyledon inoculation correlated well with yield, percent dead plants, and simple interest infection rate (34).

Further studies were required to elucidate how resistance is expressed in the field and to determine whether the correlation between cotyledon and field reaction resulted from the presence of similar or related resistance mechanisms. Also, we wished to determine which of the measures of rate-reducing resistance obtained from field studies could best be used to compare cultivar levels of such resistance. This information would be useful to plant breeders and others interested in quantitatively estimating rate-reducing resistance.

Our goals in the present study, therefore, were to determine which epidemiological parameters are best for differentiating cultivar levels of rate-reducing resistance and to investigate more extensively how such resistance is expressed under field conditions.

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In 1980, we evaluated disease incidence, area under the disease progress curve (AUDPC), simple interest infection rate (r_s), and the rate of metalaxyl fungicide application necessary to achieve a final disease incidence of 15% (F_{15}) as measures for comparing cultivars in rate-reducing resistance. In 1981, we increased the number of cultivars studied, assessed disease more frequently throughout the season while monitoring plant growth, and based disease assessments on internal as well as external symptoms to better characterize the expression of rate-reducing resistance.

MATERIALS AND METHODS

Cultivar selection. Cultivars selected for field studies represented several maturity groups and did or did not contain race-specific resistance to races 1 and 2 of Pmg conferred by the Rps_1 allele (2). These cultivars had been tested in previous years at various Pmg-infested locations and were known to differ in yield potential and susceptibility as determined by the percentage of plants killed by Pmg. The cultivars, their maturity groups, and their race-specific genes for resistance to Pmg are as follows: Steele, group I, Rps_1; Beeson, Harosoy 63, Marshall, Amsoy 71, Wells, Asgrow A2656, Asgrow A2575, and Century, group II, Rps_1; Corsoy and Northrup King S1492, group II, no race-specific resistance; Wayne, group III, no race-specific resistance. In 1980, only cultivars Steele, Amsoy 71, Asgrow A2656, and Wayne were grown; in 1981, all 12 cultivars were grown.

Field plot location. The field plot was located in Racine County, WI, on an Elliot silty clay loam soil type with a high water-holding capacity and poor internal drainage. The plot had been used for soybean cultivar evaluation in previous years and was known to be infested with numerous races of Pmg (35).

Experimental design. In 1980, four-row plots of each cultivar were hand planted on 22 May in 3.4-m rows 76 cm apart at a seeding rate of 24 seeds per meter of row. The four cultivars were arranged in a randomized complete block design with three replications per treatment. The systemic fungicide metalaxyl (Ridomil; Ciba-Geigy Corp., Greensboro, NC 27409) formulated 15% granular was applied in an 18-cm-wide band in strips across one row of each cultivar in each replication at rates of 0, 0.56, 1.12,

and 2.24 kg of active ingredient (a.i.) per hectare (ha). This procedure generated a split-block experimental design (17, page 115) with cultivars as the main plot and metalaxyl treatment as the subplot.

In 1981, eight-row plots of each cultivar were planted on 19 May with a four-row planter mounted on an International Cub tractor. The seeding rate was 24 seeds per meter of row. Rows were 4.6 m long and 76 cm apart. The 12 cultivars were arranged in a randomized complete block design with five replications per treatment. Metalaxyl formulated 15% granular was applied in an 18-cm-wide band (1.12 kg a.i./ha) over each of four rows in each eight-row plot. This procedure generated a split-plot experimental design (17, page 87) with cultivars as the main plot and metalaxyl treatment as the subplot.

Field plot observation. Disease incidence was determined by counting the number of plants dead or dying, and expressed as a percentage of the total number of plants emerged at 26 days (in 1980) or at 15 days (in 1981). In 1980, disease incidence was recorded in each row 26, 61, and 90 days after planting. In 1981, disease incidence was recorded for plants in the center two rows of each four-row subplot 35, 42, 51, 57, 70, 86, and 112 days after planting. Rounded to the nearest whole growth stage, these dates represented the V1, V7, and R5 stages and the V1, V3, V5, V7, R3, R5, and R7 stages of soybean development (5) in 1980 and 1981, respectively. These stages represent an average over all cultivars; for the earlier-maturing Steele and the later-maturing Wayne, approximately one growth stage should be added to or subtracted from these values, respectively.

In 1981, plants showing symptoms of Phytophthora root and stem rot (9,14) were marked with a 15-cm-long wooden pot label indicating the date and whether they were infected or dead. Plants marked infected at one date were also marked with another stake when dead and the number of days were recorded. In this way, the progress of disease on each plant could be followed throughout the season. In one row (designated row three), plants classified as having been killed by Pmg (defined in terms of permanent wilt of the plant's growing point) were removed from the row, while in the other row (designated row two) they were left in place. In addition, the ratio of dead plants to total diseased plants for each cultivar at each date was calculated as: (number of dead plants in the row divided by the total number of symptomatic and dead plants in the row) × 100.

The AUDPC was calculated to express the amount of disease for the entire season as follows:

$$\text{AUDPC} = \sum_{i=1}^{n-1} [(X_{i+1} + X_i)/2]\,[t_{i+1} - t_i]$$

in which X_i = the cumulative disease incidence, expressed as a proportion at the ith observation, t_i = time (days after planting) at the ith observation, and n = total number of observations. The infection rate (r_s) of simple interest disease was estimated by regressing the transformed value of $\ln [1/(1-X)]$ of cumulative disease incidence on time (37).

To determine whether plants of the 12 cultivars grown in 1981, once visibly infected, were dying at the same rate, the time from first symptom expression to plant death was calculated as the number of days (determined by sequential disease observation dates) from the time plants were marked as visibly infected (symptomatic) to the time they were marked as dead.

In 1981, ten consecutive plants were randomly sampled from the outer two rows of each four-row treatment combination at the same times that observations were made on the inner two rows. These samples were used to evaluate disease severity based on internal symptoms, as well as to investigate the effects of Pmg on plant growth parameters. Plant heights were measured in centimeters between the taproot-hypocotyl interface (~1 cm below the uppermost root) and the plant's growing point. At the first and second sampling dates, vegetative growth stage (5) was estimated by counting the numbers of nodes on the main stem with a fully-developed leaf for at least 10 sampled plants of all cultivars. Starting at the third sampling date, however, every sampled plant was examined and its vegetative stage rating was recorded. Plant height was divided by the number of nodes on the main stem for each plant, to yield internode lengths. At 70 and 86 days after planting, the reproductive stage (5) of each sampled plant was recorded along with the vegetative stage.

In 1981, tissue pieces from the hypocotyl-lower stem area as well as from roots of the 10 sampled plants, were surface disinfested in 1% sodium hypochlorite for 1–2 min, blotted on paper towels, and plated on a modified V8-juice agar medium (35). This was done at each sampling date for plants of all 12 untreated cultivars and for metalaxyl-treated plants of cultivars Steele and Asgrow A2656. Colonies that were morphologically similar to Pmg were transferred to V8-juice agar and cornmeal agar slants and later tested for pathogenicity via hypocotyl inoculation (35).

In 1981, each of the 10 sampled plants was rated for disease severity based in the first three samplings upon visible lesion development and root necrosis. As the season progressed, however, it became apparent that visual estimation based on external symptoms alone tended to underestimate disease severity. Therefore, for the third through sixth samplings, hypocotyls and taproots of all plants were split with a scalpel and the plants were rated on a 0–6 scale based on internal necrosis and external symptoms as follows: 0 = no internal or external discoloration of taproots or stems; 1 = slight discoloration in taproot; 2 = extensive taproot discoloration, but no external brown stem lesion evident; 3 = extensive taproot discoloration, external brown stem lesion present below cotyledonary node; 4 = external brown stem lesion present between cotyledonary node and unifoliate node; 5 = external brown stem lesion reaching above unifoliate node, plant alive; 6 = external brown stem lesion reaching above unifoliate node, plant dead.

For purposes of comparison with disease incidence, disease severity is usually expressed as the amount of diseased plant tissue as a percentage of the total (10,25,28). In 1981, we were able to express disease severity in this manner by calculating it for each sampled plant by the following formula: disease severity (%) = (maximum internode to which internal and/or external symptoms had advanced divided by the total number of internodes on the plant) × 100, with the taproot-lower stem area below the cotyledonary node counted as the first internode.

Data were analyzed via analysis of variance. Mean comparisons were made according to the Bayes least significant difference (BLSD) procedure (31). In analyzing cultivar response to metalaxyl treatment, multiple comparison procedures such as the BLSD were deemed inappropriate because of the quantitative nature of the treatment factor (4,12). Instead, the functional relationship between metalaxyl level and cultivar response was explored through the use of mutually orthogonal single-degree-of-freedom contrasts (orthogonal polynomials). For each of the five variables, treatment sums of squares for metalaxyl rate and the cultivar-rate interaction were partitioned into linear, quadratic, and lack-of-fit components which were tested against the appropriate error mean square in the analysis of variance. As metalaxyl treatment levels were not equally spaced, the coefficients of orthogonal polynomials were derived manually rather than obtained from available tables (8, page 111).

RESULTS

The 1980 experiment. High initial disease incidence resulted from heavy rainfall and thus standing water in the plot following seedling emergence. Table 1 presents five measures of rate-reducing resistance for the four soybean cultivars grown in 1980 and treated with metalaxyl at different rates. Significant differences ($P = 0.05$) among cultivars and rates of metalaxyl were found for disease incidence at the V1 growth stage. Significant cultivar and rate differences as well as a significant cultivar-rate interaction ($P = 0.05$) were found for disease incidence at the V7 and R5 growth stages, for r_s, and for the AUDPC.

Within the untreated plots, cultivar means differed significantly for all five variables (Table 1). For disease incidence at growth stages V1, V7, and R5, for r_s, and for AUDPC, the numbers of pairwise mean comparisons declared significant via the BLSD

procedure in the untreated plots were one, four, four, and five, respectively, of a total possible of six. In addition, cultivar rankings remained constant for three of the five variables. At the metalaxyl rate of 0.56 kg a.i./ha, cultivars differed significantly in disease incidence at growth stages V7 and R5, in r_s and AUDPC, but not in disease incidence at growth stage V1. At the metalaxyl rate of 1.12 kg a.i./ha, cultivars differed significantly in disease incidence at growth stages V7 and R5 and in AUDPC, but not in disease incidence at growth stage V1 or in r_s. At the metalaxyl rate of 2.24 kg a.i./ha, no significant differences among cultivars were found for any of the five variables (Table 1). Over all levels of metalaxyl treatment, the numbers of pairwise cultivar mean comparisons declared significant via the BLSD procedure (of 120 possible comparisons) were 18, 59, 67, 37, and 57 for disease incidence at the V1, V7, and R5 growth stages, r_s, and AUDPC, respectively.

For all five variables assessed in 1980, the average cultivar response to increasing rates of metalaxyl showed significant linear and quadratic trends. The partitioned cultivar-rate interaction for disease incidence at growth stage V1 showed no significant interaction effects, while that for disease incidence at growth stages V7 and R5 and for r_s showed significant cultivar-rate (linear) and cultivar-rate (quadratic) effects. The partitioned interaction for AUDPC showed only significant cultivar-rate (linear) effects. Significant lack-of-fit components were found for the main effect of rate for disease incidence at growth stage V7 and for r_s, and for the cultivar-rate interaction effect for only disease incidence at the V7 growth stage. A representative analysis of variance table (that for disease incidence at growth stage R5) is presented in Table 2.

Because significant quadratic as well as linear trends were found for cultivar response to metalaxyl rate, it was apparent that the relationship between final disease incidence and rate of metalaxyl could best be described by a quadratic equation. Since cultivars differed significantly in response to increasing rate of metalaxyl (indicated by the significant cultivar-rate [linear] and cultivar-rate [quadratic] effects), such relationships needed to be derived for individual cultivars. Analysis of variance was thus performed on individual cultivars for disease incidence at the R5 growth stage (final disease incidence), and the treatment sum of squares for metalaxyl rate was partitioned into linear, quadratic, and lack-of-fit components by using orthogonal polynomials. All four cultivars showed significant ($P = 0.05$) or highly significant ($P = 0.01$) rate (linear) and rate (quadratic) effects. Only cultivar Steele showed a significant rate (lack-of-fit) effect.

For each replication and cultivar, therefore, a quadratic equation of the form $Y = aX^2 + bX + c$ describing the relationship between disease incidence at growth stage R5 (Y) and rate of metalaxyl (X) was fit to the data. The equations were then solved for the rate of metalaxyl necessary to achieve a disease incidence at growth stage R5 (final disease incidence) of 15%. We termed this variable F_{15}. In increments of 5%, the value 15% was the lowest percentage that did not yield at least one complex solution to the quadratic equations. Higher percentage values yielded some negative solutions to the equations, which were difficult to interpret biologically.

A measure of rate-reducing resistance used by Fry (7) is the equivalence of resistance to the effect of fungicide applied to a susceptible cultivar. By using cultivar Steele as the susceptible standard (having sustained the highest disease incidence throughout the season), we calculated the magnitude of resistance for the other three cultivars by determining the differences between Steele and the other cultivars in rates of metalaxyl necessary to achieve a final disease incidence of 15% (DF_{15}).

Finally, since measuring resistance in terms of its equivalence to the effect of fungicide applied to a susceptible cultivar does not

TABLE 1. Measures of rate-reducing resistance for four soybean cultivars grown in 1980 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

Rate of metalaxyl (kg a.i./ha)	Cultivar	Measure of resistance: Disease incidence at growth stage[w] V1	V7	R5	r_s (per day)[x]	AUDPC[y]
0.0	Steele	39 a[z]	65 a	71 a	0.0112 a	37.9 a
	Amsoy 71	18 abc	60 a	64 a	0.0139 a	31.8 a
	Wayne	25 ab	29 b	33 b	0.0020 bc	18.5 b
	Asgrow A2656	7 bc	16 bcd	21 bc	0.0025 bc	9.4 cde
0.56	Steele	8 bc	19 bc	24 b	0.0032 b	11.0 bcd
	Amsoy 71	11 bc	20 bc	21 bc	0.0020 bc	11.5 bcd
	Wayne	0 c	0 e	0 d	0.0001 c	0.1 f
	Asgrow A2656	5 bc	6 cde	6 d	0.0000 c	3.7 def
1.12	Steele	16 bc	26 b	26 b	0.0021 bc	14.8 bc
	Amsoy 71	0 c	0 e	0 d	0.0000 c	0.0 f
	Wayne	2 c	2 de	2 d	0.0000 c	1.5 ef
	Asgrow A2656	1 c	1 e	2 d	0.0002 c	0.9 ef
2.24	Steele	8 bc	8 cde	8 cd	0.0000 c	5.3 def
	Amsoy 71	9 bc	9 cde	9 cd	0.0000 c	6.0 cdef
	Wayne	3 c	3 de	3 d	0.0000 c	1.9 ef
	Asgrow A2656	10 bc	10 cde	10 cd	0.0000 c	6.3 cdef

[w] Cumulative disease incidence at the V1, V7, and R5 growth stage (26, 61, and 90 days after planting), expressed as a percentage.
[x] The simple interest infection rate, r_s, obtained by regressing the transformed value of $\ln[1/(1-x)]$ of cumulative disease incidence on time.
[y] AUDPC represents the area under the disease progress curve.
[z] Means within a column followed by the same letter do not differ significantly according to the Bayes least significant difference (BLSD) procedure, $k = 100$. See text for metalaxyl rate trend comparisons. Data are means of three replications.

TABLE 2. Analysis of variance for data on disease incidence at growth stage R5 obtained in 1980 for four soybean cultivars treated with four rates of metalaxyl and grown in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

Source	df	Mean square	F
Block	2	5.17	4.0 ns[a]
Cultivar	3	1,509.83	17.39 **
Error *a*	6	86.84	
Rate of metalaxyl	3	4,346.62	33.41 **
linear	1	7,347.23	56.47 **
quadratic	1	5,039.07	38.73 **
lack-of-fit	1	653.57	5.02 ns
Error *b*	6	130.11	
Cultivar × Rate	9	387.86	6.96 **
C × R (linear)	3	766.11	13.76 **
C × R (quadratic)	3	239.20	4.30 *
C × R (lack-of-fit)	3	158.27	2.84 ns
Error *c*	18	55.68	2.84

[a] ns = not significant; * = significant, $P = 0.05$; and ** = significant, $P = 0.01$.

TABLE 3. Measures of rate-reducing resistance in terms of fungicide equivalency for four soybean cultivars grown in 1980 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

Cultivar	F_{15}[v] (kg a.i./ha)	DF_{15}[w] (kg a.i./ha)	RF_{15}[x] (%)
Steele	1.305[y] a[z]	···	0.0 a
Amsoy 71	0.700 b	0.605 a	39.0 b
Wayne	0.288 bc	1.017 b	68.7 c
Asgrow A2656	0.195 c	1.110 b	79.6 c

[v] F_{15} represents resistance in terms of the rate at which metalaxyl would need to be applied to the cultivar to result in a final disease incidence of 15%.
[w] DF_{15} represents a measure of resistance in terms of the reduction (savings) in the rate of metalaxyl application to a given cultivar, relative to the susceptible cultivar Steele, to result in a final disease incidence of 15%.
[x] RF_{15} represents resistance in terms of the percentage reduction in F_{15} relative to cultivar Steele.
[y] Data are means of three replications.
[z] Means within a column followed by the same letter do not differ significantly according to the Bayes least significant difference procedure ($k = 100$).

allow a measure of resistance for the susceptible cultivar itself, we calculated as an additional measure of resistance the percentage reduction in F_{15} relative to Steele for each replication and cultivar. We termed this variable RF_{15}.

Mean values of the three variables representing measures of resistance in terms of fungicide equivalency are presented in Table 3. Cultivars differed significantly ($P = 0.05$) and rankings were consistent for all three variables. For the variables F_{15}, DF_{15}, and RF_{15}, four, two, and five pairwise mean comparisons were declared significant via the BLSD procedure, of a total of six possible (Table 3).

The 1981 experiment. Symptoms of Phytophthora root and stem rot were first observed 15 days after planting. Disease incidences did not differ significantly for rows two and three at any observation date, nor was there a significant cultivar-row interaction. Henceforth, rows were analyzed as subsamples within each treatment (33, page 153).

Significant differences among untreated cultivars in disease incidence were apparent at the first observation date and remained so throughout the season (Table 4, Fig. 1). The greatest increase in disease came between the V7 and R3 growth stages. The number of pairwise mean comparisons between untreated cultivars declared significant via the BLSD procedure for disease incidence at growth stages V1, V3, V5, V7, R3, R5, and R7 were 17, 17, 15, 17, 20, 24, and 25, respectively, of a total of 66 possible comparisons.

No significant differences in disease incidence were found among metalaxyl-treated cultivars at any of the seven observation dates. Differences in disease incidence between metalaxyl-treated and untreated plants were significant for the two and three most susceptible cultivars at growth stages V1 and V3, respectively, and for the six most susceptible cultivars at growth stages V5, V7, R3, R5, and R7 (Table 4).

Significant differences were observed for AUDPC and r_s values among cultivars within the untreated class but not the treated class (Table 5). In addition, treated rows had significantly lower AUDPC and r_s values than did untreated rows for five and one of the twelve cultivars, respectively (Table 5). Cultivars also differed significantly in the time from first symptom expression to plant death (Table 5). The number of pairwise cultivar comparisons declared significant for AUDPC, r_s, and time from first symptom expression to plant death were 22, 11, and 16, respectively, of a total of 66 possible comparisons.

Cultivars (untreated) differed significantly in ratios of dead plants to total diseased plants at the V5 and R7 growth stages. Cultivar means for this variable ranged from 0 to 1.00, from 0.33 to 1.00, from 0.11 to 0.85, from 0.19 to 0.85, from 0.45 to 0.83, from 0.45 to 0.87, and from 0.54 to 0.92 at the V1, V3, V5, V7,R3, R5, and R7 growth stages, respectively. At the V5 growth stage, cultivar means ranged from 0.11 to 0.85, and 11 of 66 possible pairwise mean comparisons were declared significant via the BLSD procedure. At the R7 growth stage, cultivar means ranged from 0.54 to 0.92 with only seven significant pairwise mean comparisons. Significant correlation coefficients ($P = 0.01$) between these ratios and disease incidence for the cultivars were obtained at the R5 and R7 growth stages ($r = 0.82$ and 0.92, respectively). In addition, a significant negative correlation between such ratios and time from first symptom expression to plant death ($P = 0.05$) was obtained at the R7 growth stage ($r = -0.58$).

Disease ratings based on internal as well as external symptoms represented disease severity more accurately than those based on external symptoms alone since internal colonization was often not apparent by viewing the root exterior. Table 6 represents mean disease ratings at the third through the sixth sampling dates (V5, V7, R3, and R5 growth stages), in which plants were rated based on internal as well as external symptoms. Significant differences among cultivars were found in disease rating at the R3 and R5 growth stages; 11 and 22 pairwise cultivar comparisons (untreated) were declared significant, respectively. In addition, metalaxyl-treated plants showed significantly lower mean disease ratings than untreated plants for four and seven of the 12 cultivars at the R3 and R5 growth stages, respectively.

Table 7 presents the distribution of plants of each cultivar among the disease rating classes at the R5 growth stage. Cultivars with low disease severity, Wayne, NK S1492, and Asgrow A2575, all had a majority of plants in the zero rating class.

Correlation coefficients were calculated among the various measures of rate-reducing resistance obtained for the 12 cultivars grown in 1981 (Table 8) and the four cultivars grown in 1980. In 1981, all correlations were highly significant ($P = 0.01$), while in 1980, the only significant correlation was between AUDPC and disease incidence at the R5 growth stage ($r = 0.99$).

The relationship between disease severity and disease incidence was further explored in 1981 because of interest in the possibility of predicting disease severity from disease incidence (10,25,28). Percent disease severity was regressed against percent disease incidence at the V5, V7, R3, and R5 growth stages. A significant linear relationship ($P = 0.05$) was obtained at the V5 growth stage, while highly significant linear relationships ($P = 0.01$) were

TABLE 4. Disease incidences expressed as percentages of symptomatic and dead plants for 12 soybean cultivars grown in 1981 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

	Disease incidence (%)[w] at: Days after planting (and at growth stage)[x]						
Cultivar	35(V1)	42(V3)	51(V5)	57(V7)	70(R3)	86(R5)	112(R7)
Steele	10 a[y]*[z]	14 a*	20 a*	23 ab*	41 a*	45 a*	48 a*
Harosoy 63	5 b*	10 ab*	20 a*	25 a*	34 ab*	36 ab*	38 ab*
Marshall	3 bc	6 bc*	17 ab*	19 abc*	28 abc*	31 abc*	36 ab*
Amsoy 71	1 c	2 c	12 abc*	19 abcd*	24 abcd*	26 bcd*	29 abc*
Wells	1 bc	2 c	12 abc*	17 abcde*	21 bcde*	23 bcd*	25 bcd*
Corsoy	2 bc	4 bc	11 abc*	15 abcdef*	19 bcdef*	21 bcde*	22 bcd*
Asgrow A2656	0 c	1 c	5 bc	9 bcdef	13 cdef	14 cde	16 cde
Beeson	2 bc	4 bc	6 bc	10 bcdef	11 cdef	12 de	12 cde
Asgrow A2575	1 c	1 c	5 bc	6 cdef	10 cdef	11 de	12 cde
Century	0 c	1 c	4 c	4 def	7 def	8 de	10 de
Wayne	0 c	0 c	1 c	2 ef	3 ef	3 e	4 de
NK S1492	0 c	0 c	1 c	1 f	2 f	2 e	2 e
Mean	2	4	10	12	18	19	21

[w](Cumulative number of plants symptomatic or dead divided by total number of plants) × 100. Data are means of 10 observations on plots not treated with metalaxyl.

[x] Growth stages as described by Fehr et al (5). Values presented represent, to the nearest whole growth stage, the mean growth stage over all cultivars.

[y] Means within a column followed by the same letter do not differ significantly ($k = 100$) according to the Bayes least significant difference procedure (BLSD).

[z] An asterisk indicates a significant difference (BLSD, $k = 100$) between the means of the metalaxyl-treated and untreated plots for a given cultivar; mean values of treated plots ranged from 0 to 1, from 0 to 1, from 0 to 2, from 0 to 2, from 0 to 3, from 0 to 3, and from 0 to 6% at growth stages V1, V3, V5, V7, R3, R5, and R7, respectively.

obtained at the V7, R3, and R5 growth stages ($r^2 = 0.09, 0.18, 0.76$, and 0.29, respectively, 58 df). Fig. 2 presents disease severity plotted against disease incidence at the R3 growth stage. The square root of disease severity was also regressed on disease incidence; that resulted in slightly higher values of r^2 (the coefficient of determination) at three of the four observation dates ($r^2 = 0.09$, 0.24, 0.68, and 0.34 at the V5, V7, R3, and R5 growth stages, respectively).

Pmg was first isolated 42 days after planting (V3 growth stage) from hypocotyls of untreated plants. A uniform set of seven differential soybean cultivars (16) plus cultivar Tracy were used to determine the race identity of 11 isolates from the plot; races 3, 8, 9, and previously undescribed races were detected.

DISCUSSION

The results of this study show that soybean cultivars differ in levels of rate-reducing resistance to Pmg and may be differentiated in terms of several epidemiological parameters. In 1980, the AUDPC was the best variable for detecting cultivar differences in untreated plots, followed by the r_s and disease incidence at growth stages V7 and R5.

In 1981, we detected significant differences among cultivars for 14 of the 21 variables examined. The greatest numbers of cultivar differences were detected with disease incidence at growth stages R3, R5, and R7; with AUDPC; and with disease severity at the R5 growth stage.

For plant breeders and others interested in identifying rate-reducing resistance to Pmg in soybean, two important considerations are ease of data collection and ability to detect

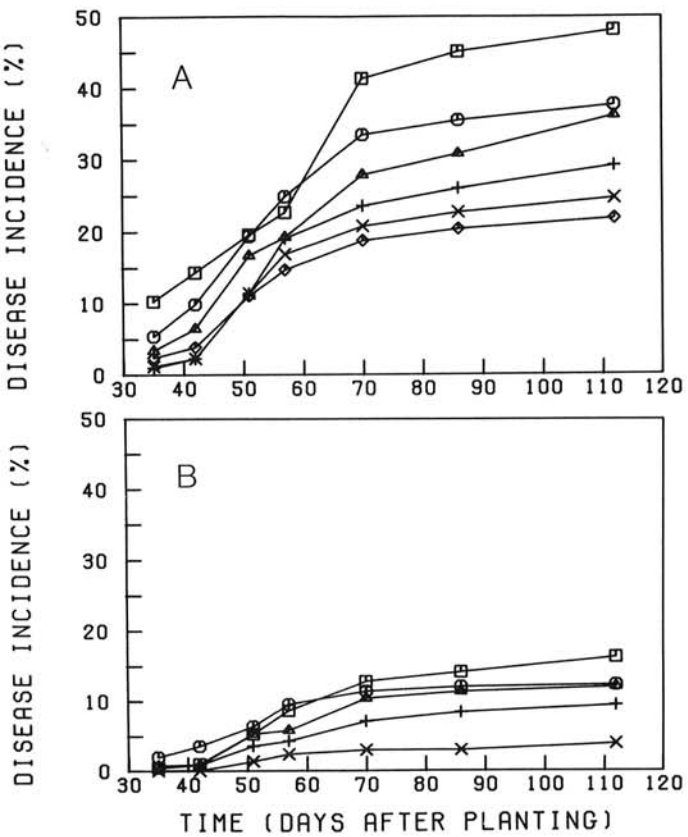

Fig. 1. Disease progress curves for 12 soybean cultivars grown in 1981 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*. Data are means of 10 observations on plants not treated with metalaxyl. **A,** Squares represent the cultivar Steele, circles represent Harosoy 63, triangles represent Marshall, plus signs represent Amsoy 71, multiplication signs represent Wells, and diamonds represent Corsoy. **B,** Squares represent cultivar Asgrow A2656, circles represent Beeson, triangles represent Asgrow A2575, plus signs represent Century, multiplication signs represent Wayne, and diamonds represent Northrup King S1492.

TABLE 5. Additional measures of rate-reducing resistance for 12 soybean cultivars grown in 1981 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

Cultivar	AUDPC[v]	r_s[w] (per day)	Time (days) between initial symptom expression and plant death
Steele	26.9[x] a[y]*[z]	0.0196[x] a[y]*[z]	14.7 a[y]
Harosoy 63	22.1 ab*	0.0147 ab	13.9 a
Marshall	19.0 abc*	0.0100 abc	16.2 ab
Amsoy 71	15.6 abcd*	0.0054 bc	15.8 a
Wells	14.4 bcd*	0.0043 bc	19.9 ab
Corsoy	12.5 bcde	0.0034 bc	16.5 ab
Asgrow A2656	8.2 cde	0.0025 bc	18.7 ab
Beeson	7.5 cde	0.0019 bc	16.8 ab
Asgrow A2575	6.4 de	0.0022 bc	13.3 a
Century	4.8 de	0.0016 bc	15.6 a
Wayne	1.9 e	0.0005 c	22.9 b
NK S1492	1.3 e	0.0003 c	36.3 c
Mean	11.7	0.0055	16.1

[v] AUDPC represents the area under the disease progress curve.
[w] The simple interest infection rate, r_s, obtained by regressing the transformed value of $\ln[1/(1-x)]$ of cumulative disease incidence on time.
[x] Data are means of ten observations representing plots not treated with metalaxyl.
[y] Means within a column followed by the same letter do not differ significantly ($k = 100$) according to the Bayes least significant difference (BLSD) procedure.
[z] An asterisk indicates a significant difference (BLSD, $k = 100$) between the means of the metalaxyl-treated and untreated plots for a given cultivar; mean values for treated plots ranged from 0.1 to 1.7 for AUDPC and from 0 to 0.0008 for r_s.

TABLE 6. Disease ratings obtained from 12 soybean cultivars grown in 1981 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

Cultivar	Disease rating[u] at: Days after planting (and at growth stage)[v] 51(V5)	57(V7)	70(R3)	86(R5)
Steele	0.36[w,x]	1.06[w,x]	2.35[w] a[y]*[z]	3.20[w] a[y]*[z]
Harosoy 63	0.34	0.64	1.46 abc*	1.90 ab*
Marshall	0.26	0.96	1.48 abc	2.06 ab*
Amsoy 71	0.26	0.64	1.92 ab*	1.98 bc*
Wells	0.00	0.00	1.53 abc*	2.02 abc*
Corsoy	0.16	0.32	1.25 abc	1.50 bc*
Asgrow A2656	0.10	0.32	0.88 bc	1.70 bc*
Beeson	0.08	0.04	0.42 c	1.14 bcd
Asgrow A2575	0.12	0.00	0.40 c	0.84 cd
Century	0.00	0.14	0.51 c	1.00 bcd
Wayne	0.00	0.38	0.27 c	0.27 d
NK S1492	0.00	0.48	0.27 c	0.28 d
Mean	0.14	0.41	1.05	1.49

[u] Based on disease rating classes 0 to 6 as follows: 0 = no internal or external discoloration of taproots or stems; 1 = slight discoloration in taproot; 2 = extensive taproot discoloration but no external brown stem lesion evident; 3 = extensive taproot discoloration, external brown stem lesion present below cotyledonary node; 4 = external brown stem lesion present between cotyledonary node and unifoliate node; 5 = external brown stem lesion reaching above unifoliate node but plant alive; 6 = external brown stem lesion reaching above unifoliate node, plant dead.
[v] Growth stages as described by Fehr et al (5). Values presented represent, to the nearest whole growth stage, the mean growth stage over all cultivars.
[w] Data are means of 50 observations (sampled plants) from plots not treated with metalaxyl.
[x] At 51 and 57 days after planting there were no significant differences ($P = 0.05$) among cultivars or between metalaxyl-treated and untreated plots.
[y] Means within a column followed by the same letter do not differ significantly ($k = 100$) according to the Bayes least significant difference procedure (BLSD).
[z] An asterisk indicates a significant difference in the BLSD procedure ($k = 100$) between the means of the metalaxyl-treated and untreated plots for a given cultivar; mean values of treated plots ranged from 0 to 0.60, from 0 to 0.12, from 0 to 0.50 and from 0.02 to 0.34 at 51, 57, 70, and 86 days after planting, respectively.

significant cultivar differences via statistical procedures. Our data show that the AUDPC as well as disease incidence assessed between the V7 and R7 growth stages are the variables which best meet these criteria.

Metalaxyl proved highly effective in reducing disease incidence and severity and was a useful tool for studying rate-reducing resistance to Pmg. As metalaxyl rate increased (1980 experiment), fewer cultivar differences were found; no differences were detected at the rate of 2.24 kg a.i./ha.

We analyzed the disease intensity:metalaxyl rate response function and found that quadratic equations best represented this relationship. All four cultivars tested in 1980 showed significant quadratic responses to metalaxyl rate; however, the magnitude of such responses varied dramatically with the cultivar, making a single equation inappropriate for representing all cultivars.

Anderson and Buzzell (1) also found that the efficacy of metalaxyl in controlling Pmg varied with the amount of fungicide and the relative "tolerance" of the soybean cultivar. Based on a multiple comparison test used to compare metalaxyl rates, they concluded that the disease incidence of Amsoy 71 and Harcor was not significantly reduced by metalaxyl rates up to 0.75 kg a.i./ha.

TABLE 7. Frequency distribution of sampled plants within disease rating classes 0 to 6[a] at 86 days after planting (R5 growth stage) for 12 soybean cultivars grown in 1981 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*

	Number of plants per class[b] in disease rating class[a]:						
Cultivar	0	1	2	3	4	5	6
Steele	13	10	2	0	1	2	22
Harosoy 63	15	11	12	0	3	6	3
Marshall	4	21	13	3	1	5	3
Amsoy 71	15	15	7	1	1	3	8
Wells	15	8	11	2	5	9	0
Corsoy	15	23	3	0	3	2	4
Asgrow A2656	9	25	6	1	3	3	3
Beeson	19	21	4	0	3	2	1
Asgrow A2575	26	17	3	1	0	2	1
Century	18	22	8	0	0	0	2
Wayne	37	13	0	0	0	0	0
NK S1492	37	12	1	0	0	0	0
Total	223	198	70	8	20	34	47

[a] Disease rating classes are as follows: 0 = no internal or external discoloration of taproots or stems; 1 = slight discoloration in taproot; 2 = extensive taproot discoloration but no external brown stem lesion evident; 3 = extensive taproot discoloration, external brown stem lesion present below cotyledonary node; 4 = external brown stem lesion present between cotyledonary node and unifoliate node; 5 = external brown stem lesion reaching above unifoliate node, plant alive; 6 = external brown stem lesion reaching above unifoliate node, plant dead.

[b] Fifty plants per cultivar (ten plants times five replications) were sampled from plots not treated with metalaxyl. For metalaxyl-treated plots, the number of plants of each cultivar in the zero rating class ranged from 37 to 49 of 50.

TABLE 8. Correlation coefficients among four measures of rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycinea* in 12 soybean cultivars grown in 1981 in a naturally infested field plot

Measure of resistance	Measure of resistance				
	r_s[a]	AUDPC[b]	DI[c]	DSR[d]	% DS[e]
Value of r_s[a]	1.000[f]				
AUDPC[b]	0.937	1.000			
DI[c]	0.932	0.997	1.000		
DSR[d]	0.833	0.934	0.941	1.000	
% DS[e]	0.795	0.761	0.776	0.867	1.000

[a] Mean value of r_s, the simple interest infection rate.

[b] Mean area under the disease progress curve.

[c] Mean final disease incidence.

[d] Mean disease severity rating at growth stage R5.

[e] Mean disease severity at growth R5, expressed as a percentage.

[f] All correlations were highly significant ($P = 0.01$, 10 df).

Similarly, Vaartaja et al (36) did not find a difference in disease incidence between metalaxyl rates of 0.5 and 1.0 kg a.i./ha for cultivar Steele. We found by analyzing the metalaxyl response function that, conversely, Amsoy 71 and Steele did show significant responses apparent from 0 to 2.24 kg a.i./ha.

The three variables we calculated from metalaxyl response functions to represent cultivar resistance in terms of fungicide equivalency were nearly equal in their resolution of cultivar differences. Since response to increasing metalaxyl rate differed among cultivars, relative measures of resistance in terms of fungicide equivalency would vary depending on the value of the disease intensity (independent) variable chosen for which to solve the response equations. A value of 15% final disease incidence allowed us to differentiate cultivars with the variable F_{15} while ranking them in a manner consistent with other estimates of resistance. In years characterized by less severe epidemics than in 1980, the response functions may vary such that equal or superior cultivar differentiation could be achieved using lower values of the independent variable.

The variable DF_{15} allows a measure of resistance in terms of fungicide rate applied to a susceptible standard cultivar. By this measure (Table 3), the resistance present in Amsoy 71, Wayne, and Asgrow A2656 was equivalent to metalaxyl applied to Steele at 0.605, 1.017, and 1.110 kg a.i./ha, respectively.

Expressing resistance in terms of the percentage reduction in the rate of metalaxyl necessary to reduce final disease incidence to 15% (RF_{15}) allowed us to obtain a value (zero) for the susceptible standard cultivar itself. This is useful in determining which of the other cultivars show significantly greater levels of resistance compared to the susceptible standard. The variable RF_{15} also showed greater precision in cultivar differentiation than did F_{15}; Amsoy 71 and Wayne differed significantly in terms of RF_{15} but not in terms of DF_{15}.

Fry (7), who also expressed resistance in terms of fungicide equivalence, plotted the log of the apparent infection rate against fungicide dosage and fitted a straight line to nonlinear data to allow calculation of fungicide equivalency for cultivar comparison. Because of the nonlinear nature of the relationship, quadratic rather than linear equations may better describe the response function resulting in more precise estimates of fungicide equivalence.

Additional variables examined in 1981 aided in further characterization of rate-reducing resistance. First, cultivars differed significantly in rate of death expressed as the mean number of days between appearance of the first aboveground symptom expression and plant death. These results support the hypothesis that cultivar resistance may act by restricting pathogen

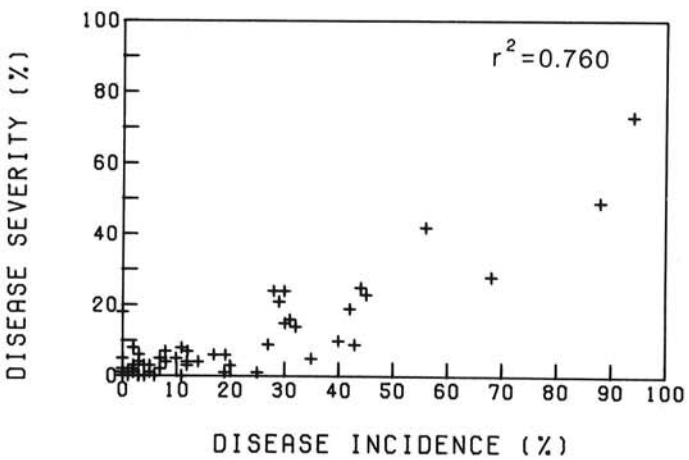

Fig. 2. The relationship between disease severity and disease incidence at the R3 growth stage (70 days after planting) for 12 soybean cultivars grown in 1981 in a field plot naturally infested with *Phytophthora megasperma* f. sp. *glycinea*. Percent disease severity is plotted against mean values of disease incidence over rows two and three, yielding five replications times 12 cultivars = 60 observations.

colonization in the plant tissue. The significant negative correlation between time between first symptom expression and death and ratio of dead plants to total diseased plants at 112 days after planting indicates that the cultivars that took longer to die also tended to be those that had greater proportions of infected versus dead plants. Early in the season this trend seemed to be reversed as shown by a positive correlation obtained between these two variables. Since time between first symptom expression and death was not significantly correlated with final disease incidence, final disease rating, AUDPC, or value of r_s, it is apparent that some of the more resistant cultivars, even though capable of restricting pathogen movement in the taproot-lower stem tissue, may die just as quickly as susceptible cultivars once infection has progressed into aboveground plant parts. It is possible that different physiological mechanisms could be involved in resistance expressed belowground versus aboveground, or that time between first symptom expression and plant death more accurately represents a measure of disease tolerance (21, page 21) rather than resistance.

Secondly, it is apparent from ratios of dead plants to total diseased plants that either cultivars differ in their propensity to die once infected or else cultivars differ in their propensity to become newly infected. Correlations between such ratios and disease incidence showed sequential increases throughout the season and were highly significant at the V5 and R7 growth stages. This seems to indicate that cultivars that show low disease incidence (indicating higher levels of resistance) tended to have greater proportions of dead plants early in the season (perhaps indicating a critical susceptible stage) but lower proportions of dead plants later in the season (perhaps indicating a form of adult-plant resistance). Conversely, susceptible cultivars with high disease incidences tended to have high proportions of infected plants early in the season, but also progressively higher proportions of dead plants throughout the season. Thus, these cultivars appear to remain susceptible throughout their development.

Variables estimating rate-reducing resistance were highly correlated with one another, indicating consistency in cultivar ranking among the variables. In both years, the highest correlation was between final disease incidence and AUDPC. The failure to detect more significant correlations among variables in 1980 could be due in part to the low number of cultivars tested.

For the four cultivars common to both 1980 and 1981, high correlations over years were not found for variables estimating rate-reducing resistance. This appeared to be largely due to the change in rank of Wayne and Asgrow A2656 over years, which could have been caused by different environmental conditions. Previous studies (34) have shown that the expression of rate-reducing resistance to Pmg may be affected by inoculum density, temperature, photoperiod, and plant age, and that the magnitude of these effects differ with the cultivar. Because large differences in seedling-stage inoculum densities existed between 1980 and 1981 (due to early flooded conditions in 1980), we were not surprised that Wayne and Asgrow A2656 changed in rank because our previous studies with these cultivars revealed that they differed substantially in response to increased inoculum density via cotyledon inoculation (34, Table 2).

Significant correlations among variables within years indicate that, in the future, workers may be able to assess certain more easily measurable variables and use these values to estimate values of highly correlated variables. This would be of use in estimating disease severity from disease incidence data, the latter of which are much easier to collect for root and stem rot of soybean. Our studies indicate that such assessments should be performed at the R3 growth stage at which the strongest linear relationship between these variables was found (Fig. 2).

In addition, the relationship between disease incidence and severity may require a different interpretation for soilborne than for foliar pathogens. In our assessments, plants were not included in disease incidence counts until they showed aboveground symptoms. By this time, however, these plants had reached an advanced stage of disease severity. This is in contrast to measurements performed on foliar plant diseases (10,25,28) in which infected plant units generally are identified very early, and then progressive disease severity is measured on those same units. Thus, we were not surprised to find a significant linear relationship between incidence and severity. Campbell et al (3) also found such a correlation for bean root rot. However, the large differences between coefficients of determination (r^2) we obtained at the V5, V7, R3, and R5 growth stages indicate that other factors such as inoculum distribution could be affecting the relationship. Fig. 2 suggests that, in spite of a significant linear relationship between incidence and severity, this relationship might be more accurately described by a nonlinear function.

Although we have defined resistance in terms of its effect on the rate of epidemic development, the simple interest infection rate (r_s) itself was not as good a variable for differentiating cultivars in 1981 as in 1980. As the 1980 epidemic was more severe than that of 1981, it is possible that the usefulness of r_s for cultivar differentiation may vary with the severity of the epidemic. In addition, several other sources of error inherent in the calculation of r_s (29) may lead to large error terms associated with means of r_s values, resulting in fewer significant differences among cultivars. Thus, our results agree with those of others who have noted drawbacks in the use of infection rates for characterizing epidemics (29,39).

Infection rates have not been widely used to describe epidemics caused by soilborne plant pathogens possibly due to confusion or uncertainty regarding the simple versus compound interest (sensu Vanderplank [37]) nature of such diseases and thus the appropriate transformation to use in linearizing disease progress data (20,23,32). Kannwischer and Mitchell (13) considered black shank of tobacco caused by *Phytophthora parasitica* var. *nicotianae* to be a multiple cycle (compound interest) disease because secondary infection could have resulted from the spread of inoculum in the field. The etiology of Phytophthora root and stem rot of soybean is similar to that of black shank of tobacco; however, we have chosen to represent disease increase in terms of the simple interest model for the following reasons:

First, critical experiments have not been performed to show that secondary infection cycles occur. Our own observations indicate that significant plant-to-plant spread probably did not occur because we did not observe more disease in the rows in which dead plants were not removed (due to contact with healthy roots of adjacent plants or movement of inoculum from root systems of dead plants to healthy plants) compared to those in which dead plants were removed.

Secondly, we do not feel that proof of existence of secondary infection precludes use of the simple interest model. Vanderplank (37, page 30) states that as long as the inoculum present in the soil at the beginning of the season remains the *main* source of inoculum during a single season, the simple interest model is appropriate. Pfender and Hagedorn (24) recently pointed out for Aphanomyces root rot of peas that even though the pathogen was capable of spreading from plant-to-plant, the extent to which secondary relative to primary infection may affect epidemic progress in any given season will vary with the density and distribution of initial inoculum. In light of the variable contribution that secondary infection may make to overall disease progress, it is apparent that epidemic progress of a single disease may be better characterized by the simple interest model under some conditions and the compound interest model under other conditions. Because in our plot, inoculum density was very high due to an intensive soybean cropping history, it seems reasonable to assume that secondary infection, if it occurred, would contribute little to epidemic development compared with infection resulting from the initial soil inoculum. Thus, the simple interest model would be appropriate.

The 1981 experiment allowed us to determine more extensively how rate-reducing resistance is expressed under field conditions. We observed large cultivar differences in disease severity ratings based on internal necrosis. Highly significant correlations between such ratings and other variables used for estimating resistance revealed that cultivars showing the greatest ability to suppress epidemic development were also the most capable of restricting the pathogen's activity in the taproot and lower stem tissue of the plant.

This tissue localization phenomenon closely parallels that observed with the same cultivars in response to cotyledon inoculation (34). We believe therefore, that a primary component of rate-reducing resistance to Pmg is the ability to restrict fungal colonization of the plant tissue (34). Although the biochemical and physiological factors responsible for such resistance remain unknown, phytoalexins could play a role, as they have been detected in soybean roots (18).

The large number of zero disease ratings (indicating no internal or external discoloration) shown by cultivars with the greatest ability to reduce epidemic development led us to speculate that an additional component of rate-reducing resistance (22) may be the ability to restrict pathogen penetration into lateral or taproot tissue. In a recent study with alfalfa, host as well as nonhost resistance to *P. megasperma* was found not to be expressed prior to root penetration but during colonization of seedling roots (19). In addition, cytological studies (30) have indicated that Pmg can complete its life cycle within roots of the 'field tolerant' cultivar Wayne as well as the susceptible cultivar Amsoy; furthermore, Wayne roots showed no discoloration. These observations support the hypothesis that rate-reducing resistance to Pmg is expressed during root colonization.

Finally, rate-reducing resistance appears to be active against numerous races of the pathogen; it was expressed in the presence of a wide array of pathotypes. This phenomenon was also observed in response to cotyledon inoculation of cultivars Steele and Asgrow A2656 (34). If it is truly race nonspecific in nature, this form of resistance should provide a means of long-lasting disease control.

In summary, we have characterized rate-reducing resistance to Pmg in terms of diverse epidemiological parameters and investigated how such resistance is expressed under field conditions. Future work should be carried out in several areas. For further characterization of resistance in terms of fungicide equivalency, disease intensity:fungicide rate relationships must be established for additional cultivars. The disease incidence:disease severity relationship should be further explored as it has been for foliar pathogens (10,25,28) as an aid in disease assessment. The effects of environmental variables on the expression of rate-reducing resistance for specific cultivars should be explored in field studies in other locations and years. In addition, work is needed to elucidate the physiological basis of the tissue localization phenomenon involved in rate-reducing resistance to Pmg in soybean. Finally, future studies should be designed to determine whether secondary infection occurs and its role, relative to primary infection, in contributing to the progress of root and stem rot epidemics.

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