plants. It is unknown whether resistance is widespread among *L. esculentum* var. *cerasiforme* or if the biological basis for resistance is similar in these accessions. In a preliminary study, both 27-1A and LA1312 had fewer propagules of *P. parasitica* per gram of root tissue than other lines (unpublished data), which suggests that at least two of these accessions may have similar types of resistance.

Literature Cited

1. Bernhardt, E.A. and R.G. Grogan. 1982. Effect of soil matric potential on the formation and indirect germination of sporangia of *Phytophthora parasitica*, *P. capsici*, and *P. cryptogea*. Phytopathology 72:507–511.
2. Blaker, N.S. 1984. The effect of soil salinity on asexual reproduction by *Phythophthora parasitica* and severity of Phytophthora root rot of citrus. PhD Diss., Univ. of California, Davis.
3. Bolkan, H.A. 1985. A technique to evaluate tomatoes for resistance to Phytophthora root rot in the greenhouse. Plant Dis. 69:708–709.
4. Boukema, I. 1983. Inheritance of resistance to foot and root rot caused by *Phytophthora nicotianae* V. Breda de Haan var. *nicotianae* in tomato (*Lycopersicon* Mill.) Euphytica 32:103–109.
5. Heritage, A.D. and E.K.S. Harrigan. 1984. Environmental factors influencing safflower screening for resistance to *P. cryptogeae*. Plant Dis. 68:767–769.
6. Hoy, M.W., J.M. Ogawa, and J.M. Duniway. 1984. Effects of irrigation on buckeye rot of tomato fruit caused by *Phytophthora parasitica*. Phytopathology 74:474–478.
7. Ioannou, N. and R.G. Grogan. 1984. Water requirements for sporangia formation by *Phytophthora parasitica* in relation to bioassay in soil. Plant Dis. 68:1043–1048.
8. Kelman, M.K. and M.D. Coffey. 1985. Quantitative comparison of the resistance to Phytophthora root rot in three avocado rootstocks. Phytopathology 75:230–234.
9. Matkin, O.A. and P.A. Chandler. 1957. The UC type soil mixes. In: K.F. Baker (ed.). The U.C. system for producing healthy container-grown plants. Calif. Agr. Expt. Sta. Man. 23.
10. Mircetich, S.M. and M.E. Matheron. 1976. Phytophthora root and crown rot of cherry trees. Phytopathology 66:549–558.
11. Richardson, L.T. 1941. A *Phytophthora* tomato disease new to Ontario. Can. J. Res. 19:446–483.
12. Satour, M.M. and E.E. Butler. 1967. A root and crown rot of tomato caused by *Phytophthora capsici* and *P. parasitica*. Phytopathology 57:510–515.
13. Skadow, K. 1985. Eine Methode der Resistenzprufung von Tomaten gegen *Phytophthora nicotianae* v. Breda de Haan var *nicotianae* Arch. Phytopathol. Pflanzenschutz, Berlin 21:143–150.
14. Umaerus, V., M. Umaerus, L. Erjefalt, and B.A. Nilsson. 1983. Control of *Phytophthora* by host resistance: problems and progress, p. 315–326. In: D.C. Erwin, S. Bartnicki-Garcia, and P.H. Tsao (eds.). *Phytophthora*: its biology, taxonomy, ecology and pathology. Amer. Phytopathol. Soc., St. Paul, Minn.
15. USDA Soil Conservation Service. 1972. Soil Survey of Yolo County, Calif.

HortScience 22(1):105–108. 1987.

Resistance of Cucumber Lines to *Rhizoctonia solani* Damping-off: Not Related to Fruit Rot Resistance

Gerdien Booy[1], Todd C. Wehner[2], and Samuel F. Jenkins, Jr.[3]
North Carolina State University, Raleigh, NC 27695

Additional index words. seedling disease, *Cucumis sativus*, vegetable breeding, belly rot

Abstract. **Seedlings of 35 cucumber (*Cucumis sativus* L.) lines were evaluated for resistance to damping-off caused by *Rhizoctonia solani* Kuhn. Five variables were measured, with the corrected disease rating (a rating for disease severity including a correction for seed vigor) being the most useful. Differences in resistance occurred among the lines, with ratings varying from 1.5 to 5.9 on a 0 (no disease) to 9 (plant dead) scale. The ratings for damping-off resistance were compared with ratings collected previously for rhizoctonia fruit rot resistance. The correlations were low and nonsignificant ($r = -0.19$ to -0.10). Thus, the damping-off test would not be a good substitute for the fruit rot test.**

Rhizoctonia solani Kuhn. [telemorph: *Thanatephorus cucumeris* (Frank) Donk], a soilborne pathogen and facultative parasite, causes damping-off disease of cucumber seedlings (11). On cucumber fruits, *R. solani* causes a rot on the area of the fruit in contact with the soil and is also called belly rot (3). In the southern United States, losses averaged 7% to 9% of the fruits harvested and have reached 40% under conditions optimal for the pathogen (13). In North Carolina, the average annual loss is about 3.5% of the fruits harvested (4).

Growth of *R. solani* is favored by high moisture (11), with an optimum growth temperature of 25° to 30°C. Infections, however, are most severe at temperatures not necessarily optimal for the pathogen but unfavorable for the host (1).

R. solani has been divided into seven anastomosis groups (AG) in North America (2) and 15 AGs in Japan (5–7). Isolates of AG 4 exist at or near the soil surface and generally are responsible for damping-off as well as fruit rot (8, 10).

The purpose of this study was to develop a screening test for resistance to damping-off in cucumber seedlings and to determine whether the results were comparable to those of rhizoctonia fruit rot resistance of plants at the fruiting stage.

Inoculum preparation. Isolates of *R. solani* AG 4 (R-8C, R-8D), originally collected from cucumber fields in Arkansas, were maintained on potato dextrose agar (PDA). Inoculum was produced on sterilized oat grains. Five hundred cubic centimeters of oat grains and 250 ml water were placed in autoclavable bags and were autoclaved for 1 hr on each of two consecutive days. Plugs (1 cm^2) of colonized PDA were transferred into the bags of sterile oat grains. After 7 days at 20° to 25°C with periodic shaking, the oats were colonized by the fungus. They were then dried in a greenhouse for 4 days and stored in plastic bags at 4°.

Reaction of cucumber plants to *R. solani* was tested by planting seeds in flats of inoculated soil. Steamed soil was inoculated with infested oat grains 7 days before planting the seeds to enable the fungus to colonize the soil.

Damping-off screening tests. The first test was conducted in a germination chamber (18°C) and in a greenhouse (25° to 30°). Inoculum concentrations of isolate R-8C were 0, 40, 80, 160, 320, 640, and 1280 oat grains per 1000 cm^3 of soil. Sixteen seeds of two cultivars (Marketmore 76 and Sumter) were planted in flats on 27 Feb. 1985 and checked for emergence each day for 18 days. This experiment, which was not replicated, was conducted to determine the optimum temperature and inoculum concentration for evaluation of damping-off resistance.

A 2nd test was run using seven cultivars (Addis, Clinton, Earlipik 14, Marketmore 76, Pacer, Sumter, and Supergreen), which differed in resistance for fruit rot, and six concentrations (0, 10, 20, 40, 80, and 160 oat grains per 1000 cm^3) of two isolates (R-8C, R-8D). After seeding, the flats were placed in a chamber at 20°C and 14-hr daylength using cool-white fluorescent lights 0.2 m above soil level. Seeds were planted 18 Apr., and the flats were watered regularly. Twenty seeds of each line per isolate per concentration were planted. The experimental design was a randomized complete block with three replications.

Received for publication 20 Mar. 1986. Paper no. 10393 of the Journal Series of the N.C. Agricultural Research Service, Raleigh NC 27695-7601. Funding was provided by a grant from Vlasic Foods, Inc. The cost of publishing this paper was defrayed in part by the payment of page charges. Under postal regulations, this paper therefore must be hereby marked *advertisement* solely to indicate this fact.

[1]Research assistant.
[2]Associate Professor, Dept. of Horticultural Science.
[3]Professor (deceased), Dept. of Plant Pathology.

Table 1. Corrected ratings for seven cucumber cultivars at six concentrations of two isolates of *Rhizoctonia solani* in the 2nd damping-off test.

Isolate	Concn (grains/1000 cm³)	Corrected rating[z] Cultivar: Earlipik 14	Pacer	Sumter	Addis	Clinton	Marketmore 76	Supergreen	Range[y]
Check	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
R-8C	10	2.1	2.5	1.7	2.4	1.3	2.4	3.5	2.2
	20	2.2	1.9	2.2	1.4	3.5	5.1	3.7	3.7
	40	2.9	3.1	3.2	4.6	4.2	4.4	6.0	3.1
	80	4.3	4.5	4.2	4.8	5.5	4.9	6.4	2.2
	160	3.8	4.2	4.2	6.3	3.5	5.3	5.3	2.8
R-8D	10	1.7	1.3	1.9	2.3	3.3	3.5	3.0	2.2
	20	1.0	1.8	1.9	2.1	3.1	4.8	4.7	3.8
	40	3.3	3.4	4.1	2.6	4.2	4.7	4.8	2.2
	80	3.3	3.3	4.0	4.4	3.6	5.2	5.4	2.1
	160	3.4	4.7	3.8	4.5	3.5	4.3	5.3	1.9
x̄		2.8	3.1	3.1	3.5	3.6	4.5	4.8	2.0
LSD (5%) for cultivar × isolate × concentration means						3.0			

[z]Ratings ranged from 0–9 (0 = no disease, 1–3 = slight damage, 4–6 = moderate damage, and 7–9 = severe damage) and corrected for the percentage of emergence measured in the control treatment.
[y]Range = (mean of most-susceptible cultivar) − (mean of most-resistant cultivar).

In the 3rd test, seedlings of 35 lines were evaluated for resistance to damping-off using one concentration (40 oat grains per 1000 cm^3) of isolate R-8C. Treatments were arranged in a randomized complete block design with four replications. Twenty seeds of each line were planted for each treatment. Seedlings also were grown in uninfested soil as a check on germination and emergence. The flats were placed in a chamber at 20°C and 14-hr daylength using cool-white fluorescent lights 0.2 m above soil level. The seedlings were planted on 26 May 1985.

Data analysis. Mean days to emergence and percentage of emergence of the seedlings were calculated by counting the number emerged each day until the seedlings were scored for final disease severity. The emerged seedlings were rated for damping-off after 18 days for the first test and after 20 days for the 2nd and 3rd tests. A disease rating was given to the seedlings, using a scale ranging from 0 to 9, in which 0 = no disease, 1–3 = slight damage, 4–6 = moderate damage, and 7–9 = severe damage (9 = plant dead). A corrected disease rating was calculated by adjusting the disease rating by the percentage of stand in the control treatments: corrected rating = $\{[\Sigma(\text{rating} \times \text{no.})] + [9 \times (\text{total-emerged})]\}/\text{total}$, where: rating = disease rating for each group of seedlings with the same rating, no. = number of seedlings in the group being rated, total = total number of emerged seedlings in the uninoculated flats, and emerged = number of emerged seedlings in the inoculated flats.

The number of unemerged seedlings (total − emerged) was multiplied by 9 because it was assumed that those seedlings were dead. Correction for seed and seedling viability was made using total seedlings emerged from uninoculated flats as the 100% level (rather than the number of seeds planted in the inoculated flats).

Rhizoctonia fruit rot tests. The results were compared to data for fruit rot resistance of these lines (unpublished data). Correlation analysis was run with the damping-off scores and the percentage of each fruit covered with lesions in the fruit rot tests, using means over replications and harvest dates for each line.

Damping-off screening test results. As a result of the first test, a concentration range from 0 to 160 oat grains per 1000 cm^3 was selected for further testing to find an optimum concentration. An incubation temperature of 20°C was selected, because, at 18°, the percentage of germination of some cucumber lines was low, and infections seemed to be more severe at temperatures relatively less favorable for the pathogen than for the host (1).

An inoculum concentration of 20 oat grains per 1000 cm^3 using isolate R-8C was optimum in the 2nd test. A wide range in percentage of diseased seedlings and in corrected disease rating among the lines was chosen as the criterion for determining the optimum concentration of inoculum (Table 1). We chose to use 40 oat grains per 1000 cm^3 (even though 20 produced a wider range among cultivars) to help identify cultivars with a high level of resistance. At that concentration, damage ratings were in the proper range ('Supergreen' at 4.8–6.0 and 'Earlipik 14' at 2.9–3.3), leaving room to identify by comparison more-resistant (ratings of 0–2) or more-susceptible (ratings of 7–9) lines in a screening test.

In the 3rd test, number of days to first emergence and mean number of days to emergence were not effective variables for measurement of damping-off, as shown by the nonsignificant F value for treated vs. control seedlings (Table 2). In addition, those variables were not correlated with the disease ratings (Table 3). There was a large treatment effect and also a large line effect for percentage of emergence (Table 2). The correlation with the disease ratings was not high. Percentage of emergence was correlated only moderately with the corrected disease rating (Table 3). There were treatment effects and line effects for mean disease rating and for corrected disease rating, and the two ratings were highly correlated with each other (Tables 2 and 3). The corrected disease rating is a better variable to use because it includes a correction for seed vigor.

The mean of 3.6 for the corrected disease rating was low, indicating some damping-off resistance in all the lines tested. Also, the range among lines for corrected disease rating was not large, indicating their similarity in resistance to this disease. Others have been more successful in identifying resistance to damping-off. For peas (*Pisum sativum* L.), the difference between lines susceptible and moderately resistant to *R. solani* was most obvious 5 days after inoculation at high temperature or 15 days after inoculation at low temperature (9). In snapbeans (*Phaseolus vulgaris* L.), more differences among the lines occurred at higher inoculum densities than at lower densities (12).

Damping-off vs. fruit rot correlations. Correlations between damping-off rating and percentage of the fruit damaged in the fruit rot tests were low and nonsignificant (Table 3). Although the damping-off test was faster and easier to run than the fruit rot test, it has no value as a substitute due to the low correlation. All tests were run using the same isolates and similar soil inoculation techniques, so the lack of correlation indicates that different mechanisms of resistance are acting or that the isolates of *R. solani* used differ in virulence as fruit rot or seedling pathogens. Further studies are needed to evaluate a more extensive collection of cucumber germplasm for resistance to damping-off and to determine the effect of isolates from different AGs on damping-off resistance.

Table 2. Percentage of emergence (PE), number of days to first emergence (DFE), mean days to emergence (DME), mean disease score (MS), and corrected disease rating (CR) for 34 cucumber lines uninoculated and inoculated with infested oats (lines are ranked by CR).

	Inoculation level							
	Uninoculated			40 oat grains/ 1000 cm³				
Line	PE	DFE	DME	PE	DFE	DME	MS[z]	CR
PI 163216	---[y]	---	---	38	7	7.5	0.9	---
National Pickling	97	7	7.8	96	7	8.1	1.3	1.5
Earlipik 14	55	9	11.7	44	8	11.4	1.1	2.7
DEXP 130	90	7	8.5	83	6	9.2	2.0	2.8
Raider	98	7	7.4	90	6	7.6	2.3	2.8
Gy 14A	98	7	8.1	91	7	7.4	2.5	2.8
Sprint 440	70	7	8.6	75	7	8.5	2.8	3.0
Straight 8	93	7	8.1	93	7	8.0	2.7	3.0
Ashley	83	7	9.0	71	7	9.8	2.3	3.0
Sumter	98	7	7.7	85	7	8.2	2.4	3.1
Supergreen	72	7	8.3	60	7	8.4	2.7	3.2
Little Leaf	93	7	7.2	93	6	7.5	3.0	3.2
M 16	90	6	7.7	93	7	7.6	3.2	3.2
Carolina	80	7	9.1	88	7	9.0	3.1	3.3
Commander	48	8	9.4	49	8	10.5	2.3	3.3
Castlepik	82	7	7.7	93	7	7.6	3.0	3.3
Score	93	7	7.7	83	6	7.5	3.1	3.5
Pikmaster	100	6	6.7	96	7	7.1	3.3	3.5
Dasher II	90	6	7.3	83	6	7.5	2.9	3.5
M 23	95	7	7.1	89	7	8.6	3.0	3.5
Verino	92	7	10.1	76	7	8.9	2.5	3.6
Pioneer	90	7	6.7	81	6	7.5	2.8	3.6
M 15	87	7	8.5	84	7	8.1	3.4	3.6
Guardian	100	8	8.3	93	6	7.1	3.5	3.6
Pacer	98	7	8.7	93	6	7.5	3.5	3.7
Poinsett 76	100	7	9.3	80	7	8.5	2.8	3.7
Clinton	72	7	8.1	63	7	8.6	3.1	3.9
Calypso	85	7	10.6	74	7	8.8	3.2	3.9
SMR 18	88	7	7.9	83	7	7.7	3.7	4.0
Tamor	75	7	8.4	60	7	8.5	2.9	4.2
Castlemaster	88	7	9.3	68	9	10.3	3.1	4.3
M 21	75	7	9.3	56	8	9.9	3.2	4.4
Marketmore 76	73	7	8.5	50	8	9.9	2.8	5.4
Gy 3	37	12	14.0	19	14	16.0	2.5	5.7
Addis	30	9	10.8	24	8	10.7	4.6	5.9
x̄	83	7	8.6	75	7	8.8	2.9	3.6
LSD (5%)	22	2	2.0	17	2	1.5	1.4	1.8
CV (%)	16	14	15	16	16	12	34	37
F (0 vs. 1)				18	1	0	777	728

[z]The MS and CR for the control were 0 for all lines, so they were not listed in the table.
[y]Insufficient seeds available for uninoculated control treatment.

Table 3. Correlation among five variables for damping-off resistance to *Rhizoctonia solani* in cucumber and with percentage of fruit damage in field and laboratory tests[z].

	Correlation					
	Days to first emergence	Mean days to emergence	Mean rating	Corrected rating	Percentage of fruit damage	
Variable					Field	Lab
Percentage of emergence	−0.58**	−0.63**	−0.16	−0.37	−0.09	−0.25
Days to first emergence		0.83**	−0.11	0.11	0.16	−0.06
Mean days to emergence			−0.33	0.16	0.12	−0.10
Mean rating				0.89**	0.00	−0.24
Corrected rating					−0.10	−0.19
Percentage of damage (field)						0.58**

**Correlation significant at the 1% level.

Literature Cited

1. Baker, K.F. and R.J. Cook. 1974. Biological control of plant pathogens. Freeman, San Francisco.
2. Burpee, L.L., P.L. Sanders, H. Cole, Jr., and R.T. Sherwood. 1980. Anastomosis groups among isolates of *Ceratobasidium cornigerum* and related fungi. Mycologia 2:689–701.
3. MacNab, A.A., A.F. Sherf, and J.K. Springer. 1983. Identifying diseases of vegetables. College of Agr., Pennsylvania State Univ., University Park.
4. Main, C.E. and S.M. Nusser. 1985. 1984 estimates of crop losses in North Carolina due to plant diseases and nematodes. North Carolina State University, Raleigh, Dept. of Plant Pathology Special Publ.
5. Ogoshi, A. 1975. Studies on the anastomosis groups of *Rhizoctonia solani*. Jpn. Agr. Res. Quart. 9:198–203.
6. Ogoshi, A., M. Oniki, T. Araki, and T. Ui. 1983. Studies on the anastomosis groups of binucleate *Rhizoctonia* and their perfect states. J. Faculty Agr., Hokkaido Univ. 61(2):244–260.
7. Ogoshi, A. and T. Ui. 1985. Anastomosis groups of *Rhizoctonia solani* and binucleate Rhizoctonia, p. 57–58. In: C.A. Parker, A.D. Rovira, K.J. Morre, and P.T.W. Wong (eds.). Ecology and management of soilborne plant pathogens. Amer. Phytopathol. Soc., St. Paul, Minn.
8. Parmeter, J.R., Jr., R.T. Sherwood, and W.D. Platt. 1969. Anastomosis grouping among isolates of *Thanatephorus cucumeris*. Phytopathology 59:1270–1278.
9. Shehata, M.A., D.W. Davis, and N.A. Anderson. 1984. Resistance to rhizoctonia stem rot in peas as influenced by temperature, watering method, and period of disease development. Plant Dis. 68:22–24.
10. Sherwood, R.T. 1969. Morphology and physiology in 4 anastomosis groups of *Thanatephorus cucumeris*. Phytopathology 59:1924–1929.
11. Stevens, F.L. and J.G. Hull. 1915. Diseases of economic plants. MacMillan, New York.
12. Sumner, D.R. 1983. Resistance in snapbean breeding lines to *Rhizoctonia solani* and rhizoctonia-like fungi. Phytopathology 74:506. (Abstr.)
13. Sumner, D.R. and D.A. Smittle. 1976. Etiology and control of fruit rot of cucumber in single harvesting for pickles. Plant Dis. Rptr. 60:304–307.

HortScience 22(1):108–109. 1987.

Inheritance of Resistance in Cabbage Seedlings to Black Rot

M.D. Dickson[1] and J.E. Hunter[2]

New York State Agricultural Experiment Station, Cornell University, Geneva, NY 14456

Additional index words. breeding, genetics, disease, *Xanthomonas campestris* pv. *campestris*. *Brassica oleracea* Group *capitata*

Abstract. **Resistance to black rot of cabbage (*Brassica oleracea* L. Group *capitata*), caused by *Xanthomonas campestris* pv. *campestris* in PI 436606, was conditioned by one recessive gene. The data suggested the occurrence of one or two modifying genes for tolerance to black rot in an unrelated line.**

Black rot (BR) caused by *Xanthomonas campestris* pv. *campestris* is an important disease of cabbage. In Florida, Georgia, and elsewhere, large blocks of cabbage seedlings are grown for shipment to the north for early planting. Periodically, although the seeds are determined to be free of the pathogen according to the best current test methods, severe outbreaks of BR occur. Bain (1) in 1952 identified a high level of resistance in 'Early Fuji' and 'Hugenot' and reported that resistance was due to one or more dominant genes (2). Williams (6) reported that resistance to BR in 'Early Fuji' was due to a recessive gene, *f*. In the heterozygous condition, the expression of this gene was influenced by one recessive and one dominant modifier gene, resulting in varying segregation ratios following crosses of 'Early Fuji' to different lines. Most American and European cultivars do not have resistance to BR, whereas some Japanese hybrids are resistant.

Received for publication 7 Apr. 1986. The cost of publishing this paper was defrayed in part by the payment of page charges. Under postal regulations, this paper therefore must be hereby marked *advertisement* solely to indicate this fact.

[1]Dept. of Horticultural Sciences.

[2]Dept. of Plant Pathology.

Young seedlings (14– to 35–days–old) of inbred lines with resistance derived from 'Early Fuji' were not resistant when inoculum was introduced into the plants via hydathodes using a procedure reported previously (3). In contrast, mature plants were resistant. Therefore, we initiated a search for BR resistance that would be expressed in both seedlings and mature plants. Following screening of most of the cabbage accessions in the PI collection of the USDA at Pullman, Wash., we identified PI 436606 from China as having the highest level of seedling resistance (3). PI 436606 also exhibited excellent mature plant resistance (3).

To study the inheritance of resistance to BR in seedlings of PI 436606, we selected for seedling resistance to BR for two generations. The PI selection was crossed to BI-16 and LAWI-3 and then the F_2 and reciprocal backcrosses were produced in the greenhouse. 'Roundup' was grown as a standard susceptible check.

Seeds were planted in early May in individual sections of Speedling styrofoam trays (5 × 5-cm cell size). Fourteen days later, when the first true leaf was 1 to 2 cm in length, the seedlings were placed overnight in a mist chamber to induce formation of guttation droplets. Plants were inoculated on three consecutive days with 1.25×10^8 colony-forming units of *Xanthomonas campestris* pv. *campestris* (Wisconsin strain PHW 117) grown on YDC agar as reported previously (3, 5). The plants were grown in a greenhouse at 25°–30°C day, 20° night temperatures, under good light (late spring), to obtain maximum disease and resistance expression.

The disease severity was recorded on a scale of 0 to 4 (0 = no disease; 1 = trace, and 2, 3, and 4 = 1, 1.5, and >2.0 cm^2 of diseased leaf tissue, respectively).

The disease reaction was recorded 14 days after inoculation, at which time all plants with scores of 2, 3, or 4 were discarded as susceptible. The survivors were reevaluated 7 days later and reclassified as susceptible in the case of a few plants. The survivors were transplanted to the field and allowed to grow to maturity. In August they were sprayed with inoculum early in the morning on three successive days when they were covered with dew and guttation droplets to assess their mature plant resistance.

The seedling test was very severe and all the susceptible check plants became severely infected, with little difference expressed between LAWI-3 and 'Round-up'. In our trials to identify seedling resistance, LAWI-3 always has shown more resistance than any other line, except PI-436606, including several lines that have repeatedly shown good mature plant resistance (3). Under these conditions, the subselection of PI 436606 was free of disease in most, but not all, plants. The severity of the test was indicated by the fact that even the LAWI-3 plants died.

The results obtained from the cross of PI 436606 with BI-16 (Table 1) suggest that resistance in the seedling stage is conferred by a single recessive gene. In the cross of PI 436606 x LAWI-3, there were more resistant plants than expected if there were only a single recessive gene involved in resistance ($P = 0.10$). The χ^2 probability was improved if resistance was due to one recessive gene plus one recessive modifier ($P = 0.20$). Further probability improvement occurred if resistance was due to one recessive gene plus one recessive and one dominant modifier ($P = 0.5$).