Widely Virulent Isolates of *Phytophthora megasperma* f. sp. *glycinea* Causing Root and Stem Rot of Soybeans in New York

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ABSTRACT

Tooley, P. W., Bergstrom, G. C., and Wright, M. J. 1984. Widely virulent isolates of *Phytophthora megasperma* f. sp. *glycinea* causing root and stem rot of soybeans in New York. Plant Disease 68:726-727.

Phytophthora megasperma f. sp. *glycinea* was confirmed for the first time in New York State on the soybean cultivar Amsoy 71. Five isolates obtained from a single field showed wide virulence on single gene differential soybean cultivars; only one isolate (race 8) could be classified using the existing numerical race designation scheme. Virulence formulae (effective/ineffective host genes) were used to describe the isolates. A multirace-resistant cultivar containing gene Rps_1^c had been grown the year before these widely virulent isolates were detected, revealing that rapid selection for virulence may occur.

Phytophthora root and stem rot of soybean caused by *Phytophthora megasperma* Drechs. f. sp. *glycinea* Kuan & Erwin (7) has long been a major disease throughout the predominant soybean-growing regions of the United States and Canada, (4,6,12). As soybean acreages in the eastern United States increased, outbreaks of root and stem rot seemed likely in this region as well. Indeed, the disease was reported in Maryland in 1982 (2) and may exist undetected in other states.

In 1983, a very wet spring, late planting, and an unusually dry summer resulted in poor soybean stands throughout the soybean-growing regions of New York State. Wilting and dying plants were noted at several locations, and collections were made to determine the cause. Taproots of wilted plants from several locations showed internal and external discoloration. However, only in one field were the classic symptoms of Phytophthora root and stem rot observed, ie, wilted, yellowed plants with flagging leaves and a distinct brown stem lesion progressing up the lower stem (4,6). This paper reports the presence of highly virulent isolates of *P. megasperma* f. sp. *glycinea* (Pmg) in New York State.

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MATERIALS AND METHODS

Isolations were made from wilted soybeans from six different fields in Tompkins and Seneca counties as well as from samples submitted to the Plant Disease Diagnostic Laboratory at Cornell University for analysis. Soybean growth at the six locations was being monitored by agronomists at Cornell University, as growers had experienced problems in previous years. Small tissue pieces from roots and hypocotyls were surface-disinfested in 2% sodium hypochlorite, blotted on a paper towel, and plated on a modified V-8 juice medium (9).

Colonies morphologically similar to Pmg were transferred to V-8 juice agar for further use. Such isolates were examined using 10× and 40× magnification phase contrast microscopy. To test for production of sporangia and zoospores, agar disks were cut from colony margins with a No. 2 cork borer and placed in 5 ml of sterile distilled water in 35-mm-diameter petri dishes at room temperature.

Seeds of seven differential soybean cultivars (8) were planted in vermiculite in 15-cm-diameter plastic pots in a greenhouse maintained at 24–27 C. The soybean differentials and their major genes for resistance to Pmg (1) were: Harosoy 63 (Rps_1^a), Sanga (Rps_1^b), Wells II (Rps_1^c), PI 86972-1 (Rps_3), Altona (Rps_6), and Harosoy and PI 103091 (genes unidentified).

Ten-day-old seedlings of each genotype were hypocotyl-inoculated with the above isolates as described previously (11). Five days after inoculation, disease reactions were recorded. Differentials for which any inoculated plants sustained complete stem girdling and hypocotyl collapse were rated as susceptible; others were rated as resistant.

RESULTS

Pmg was isolated only from plants of cultivar Amsoy 71 from a field near Ovid, NY, where classic root and stem rot symptoms had been observed. The previous year (1982), the grower had planted a soybean cultivar with multirace resistance to Pmg (Williams 79, Rps_1^c, resistant to races 1–3, 6–11, 13, 15, 17, and 21) in this field. Soybeans were not grown in this field in 1980 or 1981; the field history prior to 1980 was not available. The soil in this field was predominantly a Darien silt loam with 0–3% slope.

Five isolates (each originating from a different plant) were identified as Pmg, based initially on colony morphology. Abundant oospores with amphigynous antheridia were observed in all cultures, and sporangia and zoospores were observed after 48 hr near margins of the agar disks placed in distilled water.

Virulence formulae (3,11) for the five isolates are presented in Table 1. An unexpected array of virulence was found; only one of the five isolates fit the existing numerical race designation scheme (8).

DISCUSSION

The finding of highly virulent races of Pmg in New York State is important for several reasons. Although soybean production may date back about 20 yr in certain areas of New York, soybeans have not been grown intensively over wide

acreages as in other states. However, the widely virulent phenotypes found in a single field rival those found in other regions only after intense selection pressure exerted by the continued planting of resistant cultivars.

Second, disease outbreaks caused by new Pmg races in other states after the wide planting of cultivars containing gene Rps_1[a] have been characterized by high frequencies of race 3 (virulence formula 1b, 1c, 3, 6, 103/Har., 1a), which contains no "unnecessary" virulence when attacking such cultivars. Thus, we were initially surprised to find races in New York with much wider virulence than necessary to attack their host cultivar (Amsoy 71, Rps_1[a]). We were less surprised by this finding when we learned that a cultivar containing the gene Rps_1[c] had been grown the previous year. A similar array of widely virulent Pmg isolates was found in Wisconsin immediately after the planting of multirace-resistant cultivars containing the Rps_1[c] allele (11). Thus, it is apparent that the intense selection pressure exerted on Pmg populations by the growing of multirace-resistant cultivars can cause rapid selection of widely virulent races.

Furthermore, our results show that even after 2 yr without soybeans, selection for new Pmg races may occur in a single season when a multirace-resistant cultivar is planted. Hobe and Schmitthenner (5) found many new races of Pmg in soil that were not isolated from diseased plants grown in that soil. As soil populations of Pmg thus comprise a reservoir of pathogenic variation, the array of races we detected in a single field could have existed prior to 1982. Selection imposed by the planting of Williams 79 in 1982 probably caused an increase in the frequency of races capable of overcoming the Rps_1[c] gene. Rapid response to such selection might similarly occur when additional new genes for resistance to Pmg are incorporated into commercial soybean cultivars. Our results support the judicious deployment of such cultivars in reducing the buildup of widely virulent Pmg races.

Table 1. Virulence formulae for New York isolates of *Phytophthora megasperma* f. sp. *glycinea*

Isolate no.[a]	ATCC no.[b]	Virulence formulae[c]	Race[d]
805	52697	1b, 1c, 3/1a, 6, Har., 103	8
804	52696	1c/1a, 1b, 3, 6, Har., 103	Unknown
801	52693	6/1a, 1b, 1c, 3, Har., 103	Unknown
802	52694	/1a, 1b, 1c, 3, 6, Har., 103	Unknown
803	52695	/1a, 1b, 1c, 3, 6, Har., 103	Unknown

[a] Isolates, all obtained from plants of Amsoy 71 soybeans grown in a single field near Ovid, NY, are ranked in order of increasing virulence.

[b] American Type Culture Collection accession number.

[c] Host genes for resistance that were effective (resistant reaction type) are separated by a slash from those that were ineffective (susceptible reaction type). Alleles Rps_1[a], Rps_1[b], Rps_1[c], Rps_3, and Rps_6 are designated as 1a, 1b, 1c, 3, and 6, respectively. Harosoy and PI 103091, which do not contain named resistance genes, are abbreviated Har. and 103, respectively.

[d] Standard designations used by other workers (8). Unknown = isolates with a range of virulence not conforming to standard race designations.

Had our sample size been larger, we might have detected Pmg in additional fields. Thus, future surveys should be performed to determine whether the single field containing Pmg represents a "point source" or whether the pathogen is present more widely in New York State. If it was a point source, Pmg may have been introduced on machinery or in debris or infested soil. Measures could be taken to prevent spread to other areas. A more likely prospect is that Pmg already exists undetected in other fields. If this is true, effective long-term control via the planting of cultivars with race-specific resistance seems unlikely, given the rapid evolution of pathogen genotypes that can overcome such resistance.

Cultivars containing rate-reducing resistance to Pmg (10), possibly combined with use of fungicides, may offer the best prospect for long-term control of Phytophthora root and stem rot of soybean.

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LITERATURE CITED

1. Athow, K. L., and Laviolette, F. A. 1982. Rps_6, a major gene for resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathology 72:1564-1567.
2. Beagle, J. E., Rissler, J. F., and Kantzes, J. G. 1982. Phytophthora root rot of soybeans in Maryland. Plant Dis. 66:371-372.
3. Green, G. J. 1965. Stem rust of wheat, rye, and barley in Canada in 1964. Can. Plant Dis. Surv. 45:23-29.
4. Hildebrand, A. A. 1959. A root and stalk rot of soybeans caused by *Phytophthora megasperma* Drechsler var. *sojae* var. nov. Can. J. Bot. 27:927-957.
5. Hobe, M. A., and Schmitthenner, A. F. 1981. Direct isolation of new races of *Phytophthora megasperma* var. *sojae* from NW Ohio soils. (Abstr.) Phytopathology 71:226.
6. Kaufman, M. J., and Gerdemann, J. W. 1958. Root and stem rot of soybean caused by *Phytophthora sojae* n. sp. Phytopathology 48:201-208.
7. Kuan, T. L., and Erwin, D. C. 1980. Formae speciales differentiation of *Phytophthora megasperma* isolates from soybean and alfalfa. Phytopathology 70:333-338.
8. Laviolette, F. A., and Athow, K. L. 1983. Two new physiologic races of *Phytophthora megasperma* f. sp. *glycinea*. Plant Dis. 67:497-498.
9. Schmitthenner, A. F. 1973. Isolation and identification methods for *Phytophthora* and *Pythium*. Proc. Woody Ornament. Dis. Workshop, 1st. University of Missouri, Columbia.
10. Tooley, P. W., and Grau, C. R. 1982. Identification and quantitative characterization of rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean seedlings. Phytopathology 72:727-733.
11. Tooley, P. W., Grau, C. R., and Stough, M. C. 1982. Races of *Phytophthora megasperma* f. sp. *glycinea* in Wisconsin. Plant Dis. 66:472-475.
12. Walters, H. J. 1961. Phytophthora rot of soybeans. Arkansas Farm Res. 10:2.