New Physiologic Race of *Phytophthora megasperma* f. sp. *glycinea*

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ABSTRACT

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A new physiologic race of *Phytophthora megasperma* f. sp. *glycinea* has been identified after hypocotyl inoculation of the differential soybean (*Glycine max*) cultivars Harosoy, Harosoy 63, Sanga, Mack, Altona, PI 171442, and PI 103091. The new race was proposed as number 25. Race 25 is compatible with or virulent to those cultivars with the following genes at the rps_1 locus: Rps_1, Rps_1^b, Rps_1^c, Rps_1^k, and Rps_1^{Har} but incompatible with or avirulent to those cultivars with the genes Rps_3, Rps_4, Rps_5, and Rps_6.

Twenty-four physiologic races of *Phytophthora megasperma* Drechs. f. sp. *glycinea* Kuan & Erwin (13) (*P. m.* f. sp. *glycinea*) (syn. *P. megasperma* Drechs. var. *sojae* Hildeb.), the causal fungus of Phytophthora root rot of soybean (*Glycine max* (L.) Merr.), have been identified since the disease was first reported in 1955 (12). Race 2 was reported in 1965 (17), race 3 in 1972 (18), race 4 in 1974 (19), races 5 and 6 in 1976 (9), races 7–9 in 1977 (14), races 10–16 in 1980 (10), races 17–20 in 1982 (11), races 21 and 22 in 1983 (15), race 23 in 1983 (20), and race 24 in 1984 (12).

In 1984, we collected 62 new isolates of *P. m.* f. sp. *glycinea* to investigate the variation in pathogenicity among isolates within a given physiologic race. All isolates but one (84-1-2) were identified as known races of *P. m.* f. sp. *glycinea* on the differential soybean cultivars Harosoy, Harosoy 63, Sanga, Mack, Altona, PI 171442, and PI 103091. The reactions of the differentials to isolate 84-1-2 are reported, and the isolate is proposed as physiologic race 25.

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MATERIALS AND METHODS

Dying plants symptomatic of Phytophthora root rot caused by *P. m.* f. sp. *glycinea* were collected in seven commercial soybean fields in west central Indiana. A portion (10–12 cm) of the basal stem consisting of diseased and healthy tissue was placed in a plastic bag and kept on ice until sectioned. After removing the epidermis, four or five cross sections were removed from the transition area between diseased and healthy tissue and aseptically transferred to a selective medium in petri plates. The medium consisted of 0.6 g of Bacto yeast extract, 1 g of sucrose, 0.01 g of cholesterol, 0.001 g of benomyl, 0.027 g of PCNB, 0.2 g of vancomycin (hydrochloride), 20 g of agar, 40 ml of V-8 juice, and 1,000 ml of water. One isolate from each plate was transferred and maintained on potato-dextrose agar slants at 20–24 C in an unlighted cabinet.

Inoculum was prepared by growing the isolates on oatmeal agar in petri plates in an unlighted cabinet for 2–3 wk at 24 C. Inoculations were made by the hypocotyl method, which consists of inserting a piece of mycelium (2 × 2 mm) into a longitudinal slit in the hypocotyl and covering with petrolatum to prevent desiccation of the inoculum and host tissue. Ten 10-day-old seedlings of the seven differential cultivars were inoculated with each isolate and grown in the greenhouse at 24–27 C with supplemental fluorescent and incandescent light. Six days after inoculation, the seedlings were classified as susceptible (dead) or resistant (no external symptoms) to the isolates. Inoculation with isolate 84-1-2 was repeated at three different times.

RESULTS AND DISCUSSION

The reactions of the differential cultivars indicated that 21 isolates were race 1, 34 were race 3, five were race 4, one was race 7, and one (race 25) was different from the previously described races 1–24. Harosoy, Harosoy 63, Sanga, and Mack were susceptible and Altona, PI 171442, and PI 103O91 were resistant to race 25. Race 25 differed from race 4 only by its reaction on Sanga and from race 20 only by its reaction on PI 171442.

Each differential susceptible to race 25 has a gene at the rps_1 locus (16): Harosoy (rps_1 or Rps_1^{Har}), Harosoy 63 (Rps_1), Sanga (Rps_1^b), and Mack (Rps_1^c). The cultivar Williams 82, which has the gene

Rps_1^k (7), was also susceptible to race 25. However, PI 171442 (Rps_3) (3), PI 86050 (Rps_4) (2), L62-904 (Rps_5) (8), and Altona (Rps_6) (1) were resistant to race 25.

The recently released resistant cultivars Keller (4) and Miami (5) ($Rps_1^c Rps_3$) and Winchester (6) ($Rps_1^b Rps_3$) are resistant to race 25 because each has the gene Rps_3 in addition to Rps_1^b or Rps_1^c. Also, the germ plasm strains with the genotypes $Rps_1^b Rps_3 Rps_6$, $Rps_1^c Rps_3 Rps_4$, $Rps_1^c Rps_3 Rps_6$, $Rps_1^k Rps_4$, and $Rps_1^k Rps_6$, which we have developed for release to soybean breeders, are resistant to race 25 and to most or all other known races of the pathogen.

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