Inheritance of Resistance of Soybeans to Peronospora manshurica

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Downy mildew, Peronospora manshurica (Naoum.) Syd., is widely distributed in the United States. It was reported first on soybeans in North Carolina in 1924 by Lehman and Wolfe (6), in Illinois in 1929 by Boewe (1), and in Indiana in 1929 by Gardner (2). The infection first appears on the upper surface of the leaves as indefinite chlorotic areas which later change to well-defined grayish brown lesions with dark brown chlorotic borders. Grayish colored masses of conidiophores are usually present on the undersurface of the lesions. Some indication of the disease has been observed on the stem. Seed-borne oospores are the source of initial infection while conidia are responsible for the spread of the disease in the field.

Jones and Torrie (5) reported systemic infection of downy mildew in soybeans. They observed differences in varietal reactions in both the field and greenhouse. Geeseman (3) found a wide range in varietal reaction to downy mildew from complete resistance to complete susceptibility. He also observed that the fungus developed best under cool, humid conditions. Yarwood reported (7, 8) that the downy mildews of tobacco, onion, and cabbage required conditions for sporulation similar to those of downy mildew of soybeans. The present study is concerned with the mode of inheritance of resistance to each of the three physiologic races of downy mildew reported by Geeseman (4).

Materials and Methods

The study was made in the greenhouse during the winter of 1947-48 and the summer of 1948 at Madison, Wis. The crosses used to determine the mode of inheritance to the three races of downy mildew are listed in Table 1. In general, all the varieties in Table 1 are resistant to the three races except Illini and Richland which are susceptible. However, Richland showed partial resistance to race 3. The reaction of the parental varieties to the three races was presented by Geeseman (4). The reactions of the F1, F2, and F3 generations were determined for race 1, with the exception of crosses involving two resistant varieties where only the F2 was tested. Only the F2 generation was used for the crosses tested with races 2 and 3. Since these races were not discovered until late in the study, F1 and F2 seed was not available at that time. Thirty seeds from each of the 72 F2 lines were tested for each cross with the exception of Illini × Richland where 96 F2 lines were used. The same F2 lines were tested to each of the races if sufficient seed was available where more than one race was involved.

The seedlings which were grown in flats were inoculated by spraying with a spore suspension of conidia in water as soon as the primary leaves had expanded. The flats containing inoculated plants were placed in a moist chamber at a temperature of 18°C. Lesions developed on the leaves approximately 14 days during the winter and in 8 to 10 days during the summer. Illini which is susceptible to race 1, was used as a check for the severity of infection by the fungus.

The plants were classified for mildew reaction according to the reaction of the primary leaves as follows:

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Severity Class | Percentage of lesions
0             | 0
1             | 2
2             | 5
3             | 7
4             | 8
A             | Pin-point
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For the genetic interpretation of the data from severity classes 0 and A were considered resistant and 2 to 4 as susceptible. Since varieties which reacted O, 1 and 3 gave a 1 type of reaction to race 2, segregation, given susceptibility of resistance over segregating F1 lines of these crosses all gave good

Inheritance of Genes for Reaction to Race 1

All the F2 plants for the crosses involving resistant varieties were as resistant as the physiologic race 1. Since no segregation was observed, the F2, the F3 generation was not tested. The F3 of the F1, F2, and F3 generations of crosses resistant × susceptible and susceptible × susceptible varieties and their parents to physiologic race 1, is presented in Table 2. F1 plants of all crosses between resistant and susceptible varieties were given the distinct pin-point type of lesion. The F2 and the segregating F3 lines of these crosses were given the distinct pin-point type of lesion. The F2 and the segregating F3 lines of these crosses were given the distinct pin-point type of lesion.