A Major Gene for Resistance to Anthracnose Stalk Rot in Maize

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ABSTRACT

Maize inbred LB31, derived from an international synthetic composed of temperate and tropical germ plasm, is highly resistant to anthracnose stalk rot (ASR) caused by the fungus Colletotrichum graminicola. Resistance to ASR appeared to be controlled by a single, dominant gene as determined by a generation means analysis of the cross LB31 × B37. ASR resistance of LB31 was also exhibited in hybrids involving inbreds related to Mo17, B14A, A632, and W9. The results of this study suggest that a single, dominant gene conditions ASR resistance in the inbred LB31.

Additional key words: breeding for disease resistance.

Anthracnose, caused by the fungus Colletotrichum graminicola (Ces.) Wils., is one of the major diseases that attack maize (Zea mays L.). The fungus may infect maize roots, leaves, stalks, ears, kernels, tassels, or even silks, and significant yield losses have been reported in the United States (1,6,8,9,12) and other parts of the world (14,16). The most common symptoms are the anthracnose leaf blight (ALB) and anthracnose stalk rot (ASR). ASR is considered a more significant factor in crop loss.

Anthracnose stalk rot is now considered a major problem for maize production in the United States. Complete losses of crop in sweet corn fields in Benton County, IN, because of damage by C. graminicola were reported in 1972 (18). Up to 17.2% reduction in grain yield from natural ASR infection caused primarily by premature plant death during grain filling has been documented (15). Severe outbreaks of ASR have been reported on maize in North Carolina in 1972 and 1973 (12); losses were attributed to increased lodging and premature death. Incidence and severity of ASR have increased in New York maize fields in recent times (1).

Reports on the mode of inheritance of resistance to ASR are limited. A study conducted by Lim and White (13) indicated that resistance to ASR is conditioned by genes at several loci with additive effects more important than dominance effects. Significant heterotic effects were detected, indicating partial

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RESULTS AND DISCUSSION

Resistant inbred LB31 showed a high level of resistance to ASR. Stalk rot was always restricted to the inoculated internode, and even when corn borer activity was evident, there was no more than one discoloration internode. Also, plants of LB31 rarely had any internodes more than 75% rotten. No plant of LB31 was rated higher than a 4 on the 1-14 scale. Susceptible inbred B37, on the other hand, had a significant amount of disease spread within the stalk, and in most cases the disease spread up to more than two internodes above the ear. Also, a high percentage of internodes had more than 75% rot, resulting in ratings of 8-14 for the B37 inbred.

The anthracnose stalk rot reactions of F1, F2, and backcross generations from the crosses of LB31 with B37 are presented in Table 1. It was evident from the reactions of segregating generations that two classes of progeny occurred, those that showed rot on one to four internodes with no internodes showing 75% or more and those showing rot on three or more internodes with two to five internodes showing 75% or more. The F1 progeny were all rated as resistant (1-4) at Newark, and 94 of 102 were resistant at Aurora. Several F2 plants at Aurora were rated between 5 and 8, showing 75% rot in one or more internodes. However, most F1 plants were rated 1-4. Of 455 F2 plants at Newark, 327 were resistant (1-4) to ASR whereas 131 showed susceptibility (8-14), thus providing a good fit to a 3:1 ratio expected for a single-gene model of inheritance. Similarly, the F2 population at Aurora segregated in a ratio of 3 resistant (1-4) to 1 susceptible (8-14) (307:119).

All progeny of the backcross to the resistant parent were resistant (1-4) at Newark; a few susceptible (8-14) progeny were found in the backcross to the resistant parent at Aurora. The progeny of the backcross to the susceptible parent segregated in a resistant:susceptible ratio of 1:1 at both locations (Table 1), thus supporting the hypothesis that a single, dominant gene controls ASR resistance.

The results of the generations mean analysis suggest that LB31 carries a single, dominant gene for resistance to anthracnose stalk rot. The F1 generation was resistant and the F2 backcross to the susceptible parent followed the ratios expected for a single, dominant gene model of inheritance.

The relatively higher proportion of susceptible plants in all generations at Aurora, compared with Newark, may be attributed to a severe infestation of European corn borer (Ostrinia nubilalis Hübner) in the plots at Aurora. European corn borer damage has been shown to increase the incidence and severity of ASR (2,11). The presence of a few intermediate plants in the F1 and BC1 generations at Aurora may have resulted from misclassification of other stalk rots as ASR or from actual increases in the amount of ASR damage because of corn borer damage. At any rate, the overall data strongly support a single, dominant gene model of inheritance of ASR resistance. Crosses of LB31 onto a series of elite inbreds including Mo17, B37, B14A, A632, and W94 types.

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<th>TABLE 1. Stalk rot reactions of generations derived from a cross between LB31 and B37 when inoculated with Colletotrichum graminicola</th>
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indicate that the ASR resistance of LB37 holds in each of these diverse hybrids.

This is the first report indicating that stalk rot resistance in maize is controlled by a single, dominant gene. All previous genetic studies have shown that stalk rot resistance in maize is inherited in a quantitative manner with several genes involved (7,8,10,12,17). The most simple pattern of inheritance reported previously is two dominant genes with modifiers (3). Observations of the ASR development and spread in the inoculated internodes revealed that there was very little spread of the stalk rot in resistant plants and that in most cases the rot was restricted to only the inoculated internode, even in the presence of severe corn borer damage. It is difficult to visualize a mechanism of resistance that so effectively blocks pathogen spread and development. This is especially significant since C. graminicola is a more aggressive stalk rot pathogen than Diplodia maydis and Gibberella zeae (20).

LITERATURE CITED