A Major Gene for Resistance to Anthracnose Stalk Rot in Maize

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ABSTRACT

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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 Wf9. 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

dominance for resistance or susceptibility at some loci. Carson and Hooker (4) studied five sets of crosses involving four resistant and two susceptible inbreds. Results indicated that additive genetic effects accounted for more than 90% of the total variation among generation means, with important dominance genetic effects in some populations. As a follow-up of this study, Carson and Hooker (5) used reciprocal translocation testcross analysis to locate genes for ASR resistance in the inbred line A556. The results revealed that the long arms of chromosomes 1, 4, and 8 and both arms of chromosome 6 carry resistance genes in A556. Carson (3) investigated the mode of inheritance of resistance to ASR in the maize inbred MP305. Results indicated that MP305 has two dominant genes for resistance, one with a major and the other with a minor effect. Linkage studies revealed that the long arms of chromosomes 1, 4, 6, and 8 and the short arm of chromosome 6 carry genes for resistance (3).

The inbred LB31, developed at Cornell University, has been found to carry a high level of ASR resistance that appeared to be simply inherited. The purpose of this study was to determine the mode of inheritance of resistance to ASR of LB31.

MATERIALS AND METHODS

The resistant maize inbred LB31 was derived by six generations of selfing of an international synthetic (seed obtained in 1973 from Dr. M. Johnson, Pennsylvania State University, University Park, PA 16802), and the susceptible inbred B37 was derived from Iowa Stiff Stalk Synthetic. The exact origin of the international synthetic is unknown, but it is believed to contain a mixture of tropical and temperate germ plasm. LB31 was crossed with B37, and the several F_1 hybrid plants were selfed to produce the F_2 generation. Backcrosses of F_1 plants to each parent were also made to produce the BC_1 and BC_2 generations. The resistant inbred LB31 was also crossed to several other elite inbreds.

The two parental inbreds F_1 and F_2 and the backcross generations were tested at the Agronomy Research Farm at Aurora, NY, and at the Agricultural Engineering field at Newark, DE, for ASR resistance in 1985 in a randomized complete block design with four replications. Single-row plots, each consisting of 24 plants, were used, with two plants per hill. Plants were spaced 30 cm within rows and rows were 90 cm apart, giving a final population of approximately 59,280 plants per hectare. Each replication contained one plot each for the P_1, P_2, and F_1 generations, two plots for the BC_1 and BC_2, and five plots for the F_2.

Plants in each plot were inoculated about 2 wk after mid-silking by injecting 1 ml of conidial suspension of *C. graminicola* into the first elongated internode above the brace roots. The stalk injections were made with a 50-cm^3 Vaco pistol-grip rubber plunger syringe (Ideal Instruments, Inc., Chicago, IL 60612) fitted with a stainless steel needle as described by White and Humy (19).

An isolate of *C. graminicola*, Cg 151 NY-82, obtained from naturally infected corn leaves in Tioga County, New York, in 1982, was used for the studies. The conidial suspensions for the stalk inoculations were prepared by scraping spores from the surface of 2- to 3-wk-old oatmeal agar cultures and filtering through four layers of cheesecloth to remove mycelial debris. The spore suspension was then adjusted with distilled water to 5×10^5 conidia per milliliter.

Rating for ASR was done 4 wk after inoculation. Plants were cut off two internodes above the ear and then split longitudinally to ground level. The total numbers of rotted internodes and internodes more than 75% rotten were recorded. ASR scores for individual plants were obtained by summing the number of internodes showing any rot plus the number of internodes with more than 75% rot—i.e., if three internodes exhibited some rot and one was rated 75%, a total score of 3+1 or 4 was obtained. The highest score of the resistant parent was 4. Any plants with scores of 1–3 were considered resistant. The susceptible inbred showed an average rating of 10 with a range of 8 to 14. Plants in segregating generations rated 8 or higher were considered susceptible.

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 discolored 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 F_1, F_2, 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 rot and those showing rot on three or more internodes with two to five internodes showing 75% or more rot. The F_1 progenies were all rated as resistant (1–4) at Newark, and 94 of 102 were resistant at Aurora. Several F_1 plants at Aurora were rated between 5 and 8, showing 75% rot in one or more internodes. However, most F_1 plants were rated 1–4. Of 458 F_2 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 F_2 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 F_1 generation was resistant and the F_2 and 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 borers (*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 F_1 and BC_1 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 Wf9 types

TABLE 1. Stalk rot reactions of generations derived from a cross between LB31 and B37 when inoculated with *Colletotrichum graminicola*

Generation	Observed R	Observed S	Expected R	Expected S	χ_2	*P* value
Aurora						
LB31	78	0	78	0		
B37	0	87	0	87		
(LB 31 × B37) LB31	167	11	178	0		
(LB 31 × B37) B37	93	92	92.5	92.5	0.005	$.9 < P < 1$
(LB31 × B37) F_1	94	8	102	0		
(LB31 × B37) F_2	307	119	319.5	106.5	1.960	$.1 < P < .5$
Newark						
LB31	76	0	76	0		
B37	0	90	0	90		
(LB31 × B37) LB31	133	0	133	0		
(LB31 × B37) B37	96	85	90.5	90.5	0.669	$.1 < P < .5$
(LB31 × B37) F_1	72	0	72	0		
(LB31 × B37) F_2	327	131	343.5	114.5	3.170	$.05 < P < .1$

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).

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