Published September, 1989

Inheritance of Resistance in Three Cotton Cultivars to the HV1 Isolate of Bacterial Blight

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ABSTRACT

Immunity to the bacterial blight pathogen, *Xanthomonas campestris* pv *malvacearum* (Smith) Dye, of cotton, *Gossypium hirsutum* L., has been stable for over 20 yr. A new virulent isolate of the pathogen (HV1) was identified in Africa and a cultivar (S295) resistant to all known races and isolates was subsequently developed. The objectives of this research were to determine the inheritance of resistance in 'S295', 'Tamcot CAMD-E', and 'Stoneville 825' to a mixture of USA races and the HV1 isolate, and to examine the relationship between resistance at the cotyledon stage with that at the true-leaf stage. Progenies from three crosses were artificially inoculated and disease reaction evaluated on a scale of 1 to 10. Disease grade frequency distributions indicated a single gene with complete dominance for resistance to the USA race mixture in Tamcot CAMD-E and S295. Frequency distributions also indicated a single-gene difference between S295 and Stoneville 825 for resistance to the HV1 isolate. Resistance in S295 to HV1 and the USA race mixture appears to be controlled by the same gene or two closely linked genes. Resistance to HV1 in S295 was inherited as a single gene, designated B_{12}, with complete dominance for resistance. Correlations between cotyledon and true-leaf disease grades and phenotypic ratios indicated that resistance was controlled by the same genetic mechanism at both plant stages.

BACTERIAL BLIGHT of cotton occurs in most cotton-producing regions of the world. The disease potentially is very destructive where wind-driven rain or sprinkler irrigation disseminates the pathogen (7,16). Infection occurs primarily through open stomata of leaves, but the pathogen may also infect the stem, branches, and bolls. Bacterial blight caused an estimated yield loss of 1.0% annually in the USA from 1952 to 1984, with a range of 0.1 to 2.3% (10). Resistant cultivars offer the most economical means of controlling bacterial blight. Resistance to the bacterial blight pathogen in upland cotton breeding lines and cultivars has been stable for more than 20 yr (8). This resistance has been attributed to the combination of two or more major bacterial blight resistance genes and a modifier complex (4,11,14,15). Nineteen races of the pathogen currently are recognized in the USA (5). In 1981, a new virulent isolate, designated HV1, was identified in Africa (9,12,13). In 1984, a cotton cultivar (S295) resistant to all known races and isolates of bacterial blight of cotton was identified in Chad (14). Understanding the inheritance of resistance to the HV1 isolate will provide valuable basic information needed to facilitate development of adapted, blight-resistant cultivars.

The objectives of this research were to: (i) determine the inheritance of resistance to the HV1 isolate in S295, (ii) determine the inheritance of resistance in S295 to the mixture of USA races that is used in screening germplasm for blight resistance in the multi-adversity resistance (MAR) cotton improvement program at Texas A&M University, and (iii) compare disease reactions of cotyledons and true leaves.

MATERIALS AND METHODS

'Tamcot CAMD-E', 'Stoneville 825', and 'S295' were selected as parents for this inheritance study. S295 is highly resistant to all known races and isolates of bacterial blight of cotton (14). Tamcot CAMD-E is highly resistant to all USA races but susceptible to the HV1 isolate. Stoneville 825 is susceptible to all USA races and to the HV1 isolate. Self-pollinated seed was harvested from individual field-grown plants and bulked.

Crosses were made among the three parents and F_1 seed were produced in the greenhouse in 1985. Backcrosses to both parents and additional F_1 seed were obtained in the field in 1986. Seed harvested from each population was bulked, saw ginned, and acid delinted. Fifteen populations: three parents, three F_1s, three F_2s, and six BCs were planted in soil in 300-mm pots. Six seeds were planted in each pot and entries arranged in a randomized complete block design in the greenhouse. Parental entries consisted of one plot/replication. Four pots represented a single plot for BC_1 and BC_2 entries and nine pots represented a single plot for F_2 entries. A single greenhouse bench represented one of seven replications. After emergence, seedlings were thinned only as required to facilitate a uniform seedling establishment. Plants were inoculated and evaluated at the cotyledon and true-leaf growth stage. Initial thinning resulted in a total of

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Published in Crop Sci. 29:1114–1119 (1989).

30 seedlings for each parental and F_1 population, 268 seedlings for each F_2, and 118 seedlings for each BC_1 and BC_2 population. Fully expanded cotyledons of seedlings were inoculated with each inoculum source using the toothpick scratch method (5). After inoculation and evaluation at the cotyledon growth stage, seedlings in each pot were randomly thinned to three plants. Two main-stem leaves at Nodes 7 to 9 were inoculated and evaluated for the true-leaf growth stage.

The USA races 1, 2, 7, and 18 and the HV1 isolate were cultured at 22 °C on potato carrot dextrose agar media (PCDA). Inoculum was prepared from 5 to 7-d-old cultures by placing a small portion of the bacterial growth inside a small glass vial containing sterile water. The bacterial suspension was adjusted to produce an inoculum density of approximately 1.0×10^6 bacteria/mL. Equal portions of the inoculum of USA races 1, 2, 7, and 18 were combined for the USA race mixture.

Plants were evaluated for disease reaction from 15 to 21 d after inoculation depending upon disease development. Day and night temperatures in the greenhouse were controlled to provide a diurnal temperature variation of approximately 8 °C to favor disease development. Daily average temperatures for the cotyledon and true-leaf stages were 27 and 24 °C, respectively. Disease expression was graded using the scale of 1 (immunity) to 10 (fully susceptible) described by Bird and Hadley (6).

Disease reactions of the two cotyledons and two true leaves, within a single plant, were examined and the highest disease grade was recorded to represent the disease reaction for that plant tissue. Statistical analyses were used to detect differences among parents and progenies and heterogeneity for number of resistant phenotypes among replications. Frequency histograms of F_2, BC_1, and BC_2 generations were evaluated to determine if they exhibited bimodal distributions. Numbers occurring in segregating classes were tested using chi-square for goodness of fit to classical phenotypic ratios. Pearson product-moment correlation coefficients were calculated using replication means to compare disease reactions of cotyledons and true leaves.

RESULTS

Tamcot CAMD-E × Stoneville 825 Cross

Analysis of variance of the data indicated significant differences ($P < 0.01$) among parental, BC_1 and BC_2 populations for both cotyledons and true leaves inoculated with the USA race mixture. Cotyledon and true leaf disease grades indicated resistance in Tamcot CAMD-E and susceptibility in Stoneville 825 to the USA race mixture (Table 1). Bimodal F_2 disease grade frequency distributions were observed in both cotyledons and true leaves (Fig. 1). Parental disease grades and F_2 distributions indicated two classes of disease reactions, with Grade 1 to 3 being resistant and Grade 4 to 10 being susceptible. A chi-square test for homogeneity indicated that replications were homogenous; subsequently, they were pooled. The F_1 plants were resistant but the F_2 population segregated into a 3 resistant:1 susceptible phenotypic ratio for both cotyledons and true leaves (Table 2). When backcrossed to the susceptible parent Stoneville 825, the population segregated into a 1 resistant:1 susceptible phenotypic ratio for both cotyledons and true leaves (Table 2).

The analysis of variance for the Tamcot CAMD-E × Stoneville 825 cross indicated a significant differ-

Table 1. Cotyledon and true-leaf disease grades of cotton parental cultivars inoculated with the USA race mixture and HV1 isolate of *Xanthomonas campestris* pv *malvacearum*.

Inoculum‡	Tissue inoculated	Disease grade†		
		Tamcot CAMD-E	Stoneville 825	S295
USA race mixture	Cotyledon	1.9	8.5	2.3
	True leaf	2.2	4.9	2.1
HV1 isolate	Cotyledon	8.8	8.2	2.2
	True leaf	7.5	7.1	2.8

† Grades based on a scale of 1 (immunity) to 10 (fully susceptible).
‡ Main stem leaves inoculated at 7, 8, or 9 node position.

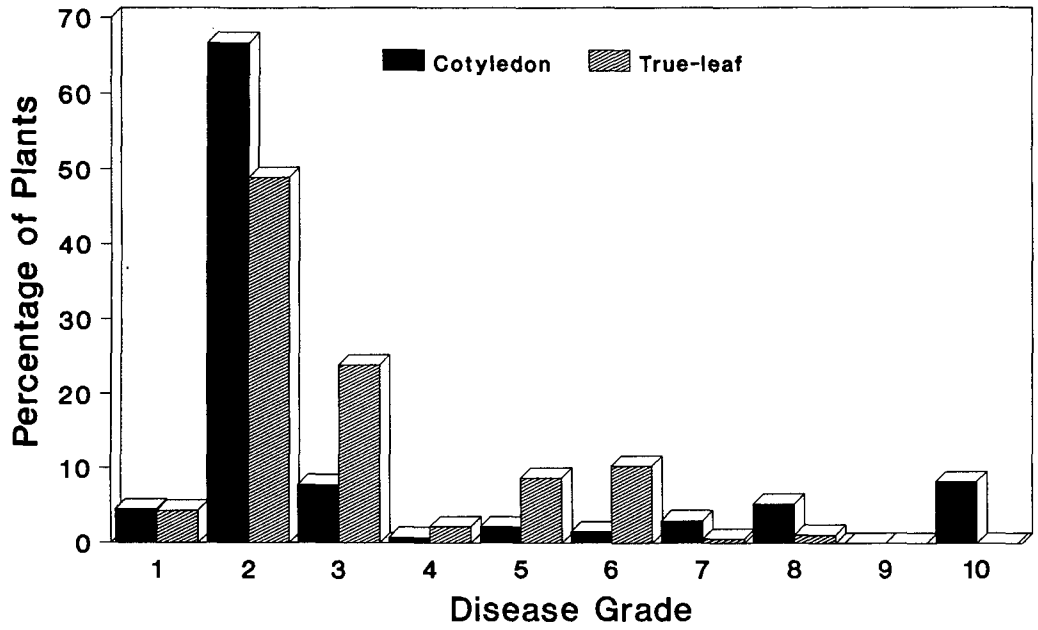

Fig. 1. The F_2 frequency distributions of cotyledon and true-leaf disease grades for the cotton cross Tamcot CAMD-E × Stoneville 825 (St. 825) inoculated with the USA race mixture of *Xanthomonas campestris* pv *malvacearum*.

Table 2. Segregation for cotyledon and true-leaf disease reactions to a mixture of USA races 1, 2, 7, and 18 of *Xanthomonas campestris* pv *malvacearum* among parental cotton cultivars† and their F_1, F_2, BC_1, and BC_2 generations.

Parents and progeny	Expected segregation	Cotyledons				True leaves			
		Observed segregation				Observed segregation			
		R‡	S‡	X^2 value	P	R‡	S‡	X^2 value	P
		no.				no.			
CAMD-E†	R	30	0	–	–	20	1	–	–
St. 825†	S	2	28	–	–	2	18	–	–
S295†	R	29	0	–	–	20	0	–	–
CAMD-E × St. 825									
F_1	R	29	0	–	–	20	0	–	–
BC_1 (F_1 × CAMD-E)	R	118	0	–	–	79	0	–	–
BC_2 (F_1 × St. 825)	1:1	61	57	0.14	0.75–0.50	44	37	0.60	0.50–0.25
F_2	3:1	212	56	2.41	0.25–0.10	142	42	0.46	0.50–0.25
CAMD-E × S295									
F_1	R	27	0	–	–	21	0	–	–
BC_1 (F_1 × CAMD-E)	R	118	0	–	–	84	0	–	–
BC_2 (F_1 × S295)	R	82	0	–	–	82	0	–	–
F_2	R	191	0	–	–	184	1	–	–
St. 825 × S295									
F_1	R	27	0	–	–	21	0	–	–
BC_1 (F_1 × S295)	R	115	2	–	–	80	0	–	–
BC_2 (F_1 × St. 825)	1:1	59	48	1.19	0.50–0.25	45	37	0.78	0.50–0.25
F_2	3:1	114	45	0.92	0.50–0.25	141	47	0	1

† Tamcot CAMD-E, Stoneville 825 (St. 825), and S295.
‡ R = resistant, S = susceptible.

ence ($P < 0.05$) between the parental cultivars only for cotyledon reaction when inoculated with the HV1 isolate. Tamcot CAMD-E and Stoneville 825 cultivars were susceptible to the HV1 isolate regardless of the plant tissue considered (Table 1). No differences were detected among the backcross populations for either cotyledons or true leaves. Means and frequency distributions indicated little, if any, detectable genetic differences between Tamcot CAMD-E and Stoneville 825 for resistance to the HV1 isolate.

Stoneville 825 × S295 Cross

Stoneville 825 was susceptible to the USA race mixture and HV1, and S295 was resistant to both (Table 1). Bimodal disease grade frequency distributions were obtained when cotyledons and true leaves of the F_2 progenies were inoculated with the USA race mixture (Fig. 2). Disease grade frequency distributions indicated a resistant and susceptible class represented by Grade 1 to 3 and 4 to 10, respectively. A chi-square test for homogeneity of cotyledon disease grade distributions among replications was significant and indicated that one replication had an excess of susceptible plants, while another replication had an excess of resistant plants. A chi-square test for goodness of fit to a 3 resistant:1 susceptible phenotypic ratio performed before removing these blocks had a probability value between 0.5 and 0.95. The chi-square values after removal of these two replications were lower and nonsignificant (Table 2). True leaf disease grades for

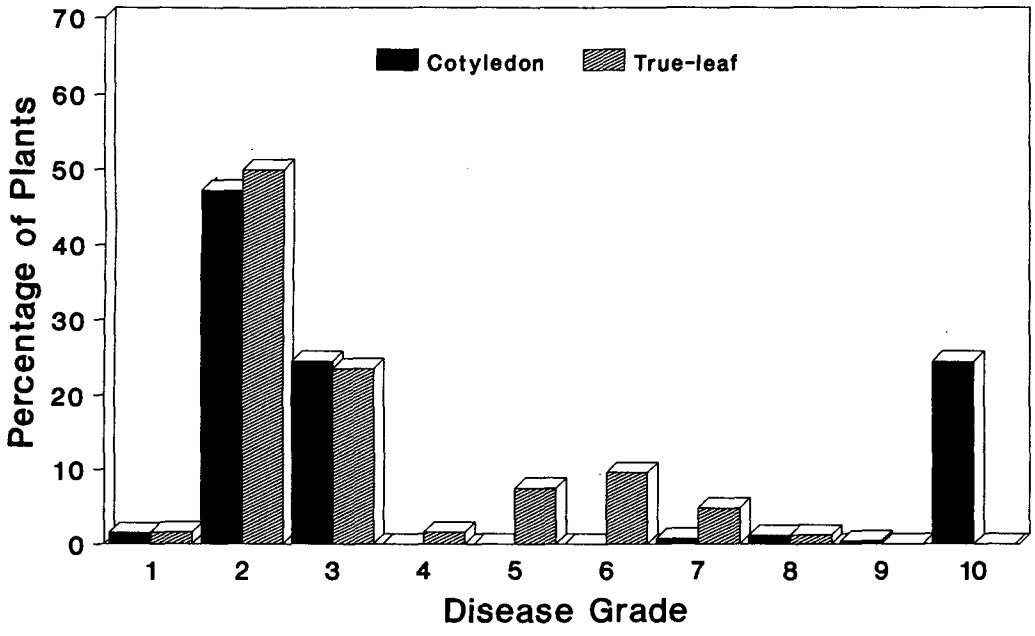

Fig. 2. The F_2 frequency distributions of cotyledon and true-leaf disease grades for the cotton cross Stoneville 825 (St. 825) × S295 inoculated with the USA race mixture of *Xanthomonas campestris* pv *malvacearum*.

Table 3. Segregation for cotyledon and true-leaf disease reactions to the HV1 isolate of *Xanthomonas campestris* pv *malvacearum* for two cotton crosses† and their parental, F_1, F_2, BC_1, and BC_2 generations.

Parents and progeny	Cotyledons					True leaves				
	Expected segregation	Observed segregation		X^2 value	P	Observed segregation		X^2 value	P
		R‡	S‡			R‡	S‡		
		no.				no.			
CAMD-E†	S	0	30	–	–	0	21	–	–
St. 825†	S	0	30	–	–	2	18	–	–
S295†	R	28	1	–	–	20	0	–	–
CAMD-E × St. 825									
F_1	S	1	28	–	–	0	20	–	–
BC_1 (F_1 × CAMD-E)	S	2	114	–	–	0	20	–	–
BC_2 (F_1 × St. 825)	S	1	117	–	–	1	80	–	–
F_2	S	3	265	–	–	1	183	–	–
St. 825 × S295									
F_1	R	27	0	–	–	20	1	–	–
BC_1 (F_1 × S295)	R	114	3	–	–	72	8	–	–
BC_2 (F_1 × St. 825)	1:1	60	47	1.58	0.25–0.10	44	38	0.44	0.75–0.50
F_2	3:1	186	76	2.24	0.25–0.10	139	49	0.11	0.75–0.50

† Stoneville 825 (St. 825) × S295 and Tamcot CAMD-E × St. 825.
‡ R = resistant, S = susceptible.

the USA race mixture also showed a good fit to a 3:1 ratio. The BC_2 population segregated into a 1 resistant:1 susceptible phenotypic ratio (Table 2).

When cotyledons were inoculated with the HV1 isolate, a bimodal F_2 disease grade frequency distribution was observed (Fig. 3). The F_2 population segregated 3 resistant:1 susceptible (Table 3). Inoculation of true leaves with the HV1 isolate also yielded a bimodal disease grade frequency distribution in the F_2 (Fig. 3). Reactions of true leaves differed from reactions of cotyledons, such that the test for a 3:1 ratio gave a nonsignificant chi-square only when Grade 1 through 4 were included in the resistant class (Table 3). Other dihybrid F_2 phenotypic ratios were tested; however, a 3:1 phenotypic ratio was the only acceptable fit.

Tamcot CAMD-E × S295 Cross

Parental cultivars Tamcot CAMD-E and S295 were highly resistant to the USA race mixture in reactions of both cotyledons and true leaves with no significant differences among parents or backcrosses. Disease grades were slightly higher for S295 compared to Tamcot CAMD-E for cotyledon reaction (Table 1). No segregation for resistance to the USA race mixture in the Tamcot CAMD-E × S295 cross was observed

When inoculated with the HV1 isolate, Tamcot CAMD-E was susceptible and S295 was resistant for both cotyledon and true-leaf disease reactions (Table 1). Cotyledon disease grade frequency distributions of the F_2 generation for this cross indicated a bimodal distribution (Fig. 4). However, the F_2 population had an excess number of plants that graded in the intermediate (Grade 4–6) and susceptible (Grade 7–10) range of the grading scale and the data failed to fit any classical phenotypic F_2 ratio. When the F_1 was crossed with the susceptible parent, the BC_2 population exhibited a bimodal distribution. However, a 1:1 phenotypic ratio was not observed. The backcross (BC_1) to

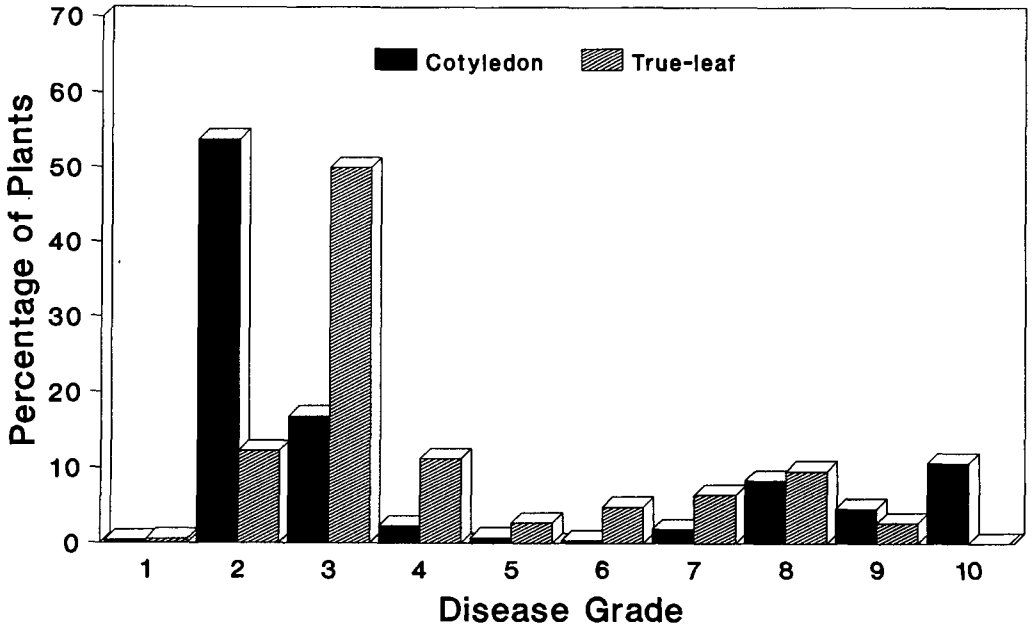

Fig. 3. The F_2 frequency distributions of cotyledon and true-leaf disease grades for the cotton cross Stoneville 825 (St. 825) × S295 inoculated with the HV1 isolate of *Xanthomonas campestris* pv *malvacearum*.

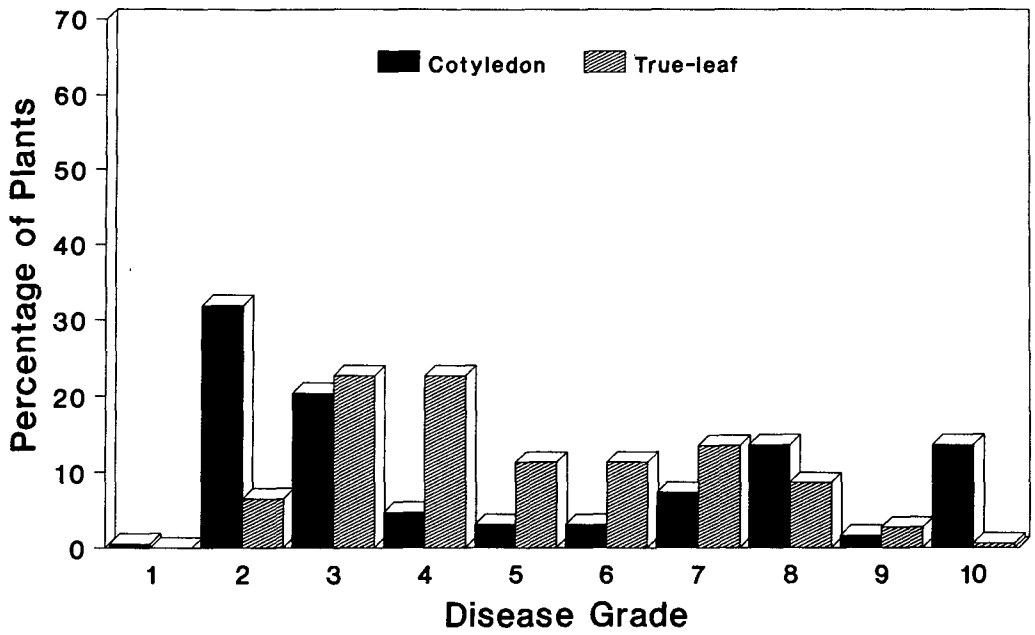

Fig. 4. The F_2 frequency distributions of cotyledon and true-leaf disease grades for the cotton cross Tamcot CAMD-E × S295 inoculated with the HV1 isolate of *Xanthomonas campestris* pv *malvacearum*.

the resistant parent yielded a phenotypic ratio of 58 resistant:23 susceptible progenies. When true leaves were considered, bimodal F_2 and BC distributions were less distinct compared to cotyledon disease reactions. In each case, an excess number of progenies were observed in the susceptible range (Grade 4–10) of the disease grading scale and resulted in a lack of fit to classical phenotypic ratios. The distributions, however, suggested major gene effects with dominance in the direction of resistance, as opposed to continuous variation.

Association of Resistance

The F_2 and backcross progenies from the Stoneville 825 × S295 cross were examined for independent assortment of resistance to the USA race mixture and HV1 isolate. Cotyledon and true-leaf disease reactions to the race mixture and the isolate were compared on an individual plant basis. For the cotyledon disease grades, only a single plant from a total of 383 plants was observed that had a high grade (Grade 10) for the USA race mixture and a low grade (Grade 3) for the HV1 isolate. When true leaves in the same plot were examined, however, disease grades in the resistant range (Grade 1–3) were observed. A poor inoculation may have been the reason for a single F_2 plant exhibiting a high and low grade. However, the preponderance of evidence suggests that resistance to the USA race mixture and HV1 isolate was conditioned by the same gene or two closely linked genes.

Cotyledon and True Leaf Correlation

Correlation coefficients using replication means of disease grades revealed a significant and positive correlation between cotyledon and true-leaf disease grades for both the USA race mixture ($r = 0.88$) and HV1 isolate ($r = 0.94$). The age of the cotyledon or true leaf appeared to influence the degree of disease expression. In general, younger cotyledons and true leaves of susceptible plants scored higher on the disease grading scale than did older tissues.

DISCUSSION

The F_2 disease grade frequency distributions for the cross Tamcot CAMD-E × Stoneville 825 indicated a single gene difference with complete dominance for resistance to the mixture of USA races 1, 2, 7, and 18 in the resistant parent Tamcot CAMD-E. The apparent single gene inheritance, however, conflicts with the reported multigenic resistance in Tamcot CAMD-E (3). Innes et al. (17) analyzed a diallel cross involving the line 101–102B, which was the source of resistance utilized in developing parental germplasm of Tamcot CAMD-E, and suggested that a stable "super gene" with dominant gene action had been synthesized. Brinkerhoff et al. (8) suggested that a chromosome inversion including the genes for resistance would greatly reduce recombination between the loci. The monogenic inheritance of resistance indicates that pedigree breeding would be adequate for transferring this resistance into susceptible genotypes. When inoculated with the HV1 isolate, parental, F_1, F_2, and backcross progenies for the Tamcot CAMD-E × Stoneville 825 cross gave susceptible disease reactions.

Single gene inheritance of resistance to the USA race mixture and HV1 isolate was indicated in S295 when crossed with Stoneville 825. Dominance for resistance was complete. The pedigree of S295 has been reported as a three-way cross of a line derived through panmixy of 34 parents, a line derived from a recurrent selection procedure, and another line related to the recurrent selected line (14). The three parents were reported to be resistant to USA races but susceptible to the HV1 isolate. The pedigree of S295 suggests that

resistance to the HV1 isolate would be multigenic in nature. However, both cotyledon and true-leaf F_2 and BC disease grade distributions clearly indicated monogenic inheritance of resistance. It is possible that a single-point mutation occurred in S295. Examination of individual F_2 and BC plants from the Stoneville 825 × S295 cross suggested that resistance to the HV1 isolate and the USA race mixture in S295 is conferred by the same gene or by two closely linked genes.

When the HV1-susceptible parent Tamcot CAMD-E was crossed with S295, disease grade frequency distributions for HV1 indicated the presence of a major gene effect with dominance in the direction of resistance in S295. Simple Medelian inheritance, however, was not verifiable. When either Tamcot CAMD-E or S295 was crossed with Stoneville 825, a single gene difference was observed when plants were inoculated with the USA race mixture. The single gene difference between S295 and Stoneville 825, however, also conferred resistance in S295 to the HV1 isolate. If the gene for resistance to the USA race mixture in Tamcot CAMD-E was the same gene conferring resistance in S295, then the progenies from crossing these parents would have indicated a single gene difference when inoculated with the HV1 isolate. The genetic background of Tamcot CAMD-E, however, influenced the expression of resistance to the HV1 isolate. An excess number of susceptible phenotypes were observed in the progenies of the cross Tamcot CAMD-E × S295, suggesting a negative type of interaction between the gene for resistance to the USA race mixture in Tamcot CAMD-E with the gene for resistance to the USA race mixture and HV1 isolate in S295. These findings are in agreement with previous reports on the influence of the genetic background on the expression of genes for resistance to bacterial blight (4,11,14,15). To facilitate future studies concerning this new source of resistance to the HV1 isolate, it is proposed that this major gene for resistance in S295 be designated B_{12}. This follows the numerical sequence of previously identified *B* genes as recommended by the committee on rules for symbolizing genes and chromosome aberrations in cotton (18).

Conflicting evidence for a basic mechanism controlling resistance to bacterial blight in different plant parts and at different stages of plant growth have been reported (1,2). Arnold and Brown (2) suggested that screening for resistance should be conducted at several different stages of plant growth. Cotyledon and true-leaf disease grades in the present study were positively correlated, and when monogenic inheritance was observed in the cotyledon stage of growth, the same was observed in the true-leaf stage of growth. These results support the concept of a common basic mechanism controlling resistance in the two different stages of plant growth and suggest that selection for resistance can be accomplished in either growth stage. Evaluation for resistance at both the cotyledon and true-leaf stages would give an added measure of assurance in correctly identifying resistant genotypes.

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