Effectiveness of Specific Genes and Gene Combinations in Conferring Resistance to Races of Xanthomonas malvacearum in Upland Cotton

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ABSTRACT

Eight strains of *Gossypium hirsutum* were intercrossed to produce a complete diallel set. Plants in the field were inoculated with separate suspensions of *Xanthomonas malvacearum,* races 1 and 12, and a 1:1 mixture of the two. Each plant was assigned a disease grade (1-10) representing the extent of leaf infection. Race 12 was more virulent than race 1. Virulence of the mixture was intermediate between races 1 and 12. The parental lines having the major gene B_4 and the gene combination B_2B_3 were the most resistant to both races, followed by the B_2B_6 and $B_2B_3B_7$ combinations. Austin (B_7) was highly resistant to race 1 and less resistant to the mixture and race 12. Empire WR (B_{Sm}), Deltapine TPSA (B_{Dm}), and Texacala (B_{Tm}) were susceptible to both races. The B_7 gene negatively influenced the gene combination B_2B_3 in the presence of race 12. The major genes showed isodirectional dominance towards resistance. Transgressive segregation for the minor genes was indicated. Symptom expression with race 12 was influenced by environment more than with race 1. The B_4 and B_2B_3 genes were less influenced by environmental fluctuations than others. The genetic background was important in the expression of the major genes. Phytopathology 60:441-447.

RÉSUMÉ

Huit lignées de *Gossypium hirsutum* étaient entrocroisés pour produire une diallel complète. Les plantes aux champs étaient inoculées avec suspension séparé de *Xanthomonas malvacearum,* races 1 et 12, et 1:1 mélange des deux. Une maladie graduée (1-10) était assignée à chaque plante, représentant l'étendue de l'infection des feuilles. Race 12 était plus virulente que race 1. La virulence du mélange était intermédiaire entre races 1 et 12. Les lignées ayant les gènes majeurs B_4 et l'association des gènes B_2B_3 étaient les plus résistants aux deux races, suivis par B_2B_6 et $B_2B_3B_7$. Austin (B_7) était hautement résistant à race 1 et moins résistant au mélange et race 12. Empire WR (B_{Sm}), Deltapine TPSA (B_{Dm}), et Texacala (B_{Tm}) étaient susceptibles aux deux races. Le gène B_7 à influencé négativement l'association des gènes B_2B_3 en la présence de race 12. Les gènes majeurs ont manifestés dominance isodirectionel vers la résistance. La ségrégation transgressive des gènes mineurs étaient indiquée. L'expression des symptômes avec race 12 était influencée plus par le climat qu'avec race 1. Les gènes B_4 et B_2B_3 étaient moins influencés par le climat que d'autres. L'arrière-plan génétique était important dans l'expression des gènes majeurs.

Bacterial blight of cotton, incited by *Xanthomonas malvacearum* (E.F.Sm.) Dows., occurs throughout the cotton-producing areas of the world. Sixteen major genes have been reported that condition bacterial blight resistance in cotton (16, 21, 25, 27).

Knight (24, 25) summarized his investigations on the genetics of blight (blackarm) resistance. He identified and described ten major genes (B_1-B_{10}) from five species of *Gossypium*; all except b_8 were reported to be either fully or partially dominant for varying degrees of resistance. Knight (25) transferred nine of these genes to commercial Sakel (*G. barbadense* L.) and Upland (*G. hirsutum* L.) varieties grown in the Sudan.

Green & Brinkerhoff (16) described three major dominant genes which control resistance to blight in Upland strains. Lagière (27) identified two major dominant genes, and gave them the symbols B_9 and B_{10}. Lagière's B_9 was nonhomologous with Knight's B_1 to B_7 and B_9 (20). Innes (21) described the gene B_{11}. The influence of minor genes and the genetic background on the expression of the major genes has been emphasized by several workers (2, 8, 19, 25).

Bird (4) reported that in *G. hirsutum* the B_4 gene conditioned significantly higher resistance to five races of *X. malvacearum* than did B_2, B_3, or B_7. The B_4 gene was as effective as $B_2B_3B_6$ and significantly stronger than B_2B_3, B_2B_6, and $B_2B_3B_7$ gene combinations. In the presence of six races of the pathogen, B_2, B_3, and B_7 in an Empire background behaved as minor genes, whereas B_4 behaved as a strong major gene (5).

The effect of a number of the major genes, individually and combined, in conferring resistance has been reported from several countries in Africa (19, 24, 27, 28).

Reports on the variability of the pathogen (1, 13, 14, 15, 17, 31) emphasize that new and more virulent races do evolve. The existence of several physiologic races of the organism has been adequately demonstrated in the United States (6, 9, 12, 17). Fifteen races of *X. malvacearum* were recently described by differential reactions of eight *G. hirsutum* strains (18).

The objectives of this study were to determine the effectiveness of five major genes in conferring resistance to two races of *X. malvacearum,* their differential response in three genetic backgrounds, and their potentials for breeding immune strains of cotton.

MATERIALS AND METHODS.—Eight strains of Upland cotton (*G. hirsutum*) were chosen for the study. Five carried known specific genes or gene combinations for resistance to bacterial blight in an Empire WR background (Table 1). Thus, the B_{Sm} minor gene complex was common to them. Three were commercial varieties representing different minor gene backgrounds. The B_2B_3, B_2B_6, and B_4 genes had been transferred by backcrossing from Knight's Sakel to Empire WR (2).

The eight strains were intercrossed to produce a complete diallel set. Parental, F_1, and F_2 generations were produced in the field by making controlled cross- or self-pollinations. A factorial experiment with three replications was conducted over a 2-year period in the field. The two factors utilized in the 1964 experiment were entries (64 combinations: 8 parents, 28 F_1's and their 28 reciprocals) and races (races 1 and 12, and a 1:1 mixture of the two). Race designation as described by Hunter et al. (18) was used. In the 1965 experiment, one factor was the entries which consisted of 120 combinations: 8 parents, 56 F_1's and 56 F_2's. The second factor was races, with two levels; race 1 and race 12. In both experiments, seed of each entry were planted directly in the field in single row plots, 9.75 m long with rows 1 m apart. Seedlings were thinned to a maximum of 22 plants/plot.

Inocula of *X. malvacearum* races 1 and 12 were prepared from 5-day-old bacterial colonies increased by streaking on potato-carrot-dextrose agar (PCDA) in petri dishes. Bacteria from each dish were diluted with 7.60 liters of water. Each race was handled separately, and the inoculum was made up in quantities of 189 liters. Inoculum concentration was approximately 10^6 bacteria/ml. For the 1:1 mixture of races 1 and 12, bacterial growth from one plate of each race was diluted with 7.60 liters of water to give approximately 2×10^6 bacteria/ml.

Plants were inoculated 6 weeks after planting (plants with six to eight true leaves) by spraying a bacterial suspension, at 300 psi, onto the lower surface of the leaves. With this method, water-soaking is accomplished by forcing the inoculum through the open stomata into the substomatal cavity (7).

Plants were assessed for disease reaction 3-4 weeks after inoculation. Each plant was assigned a disease grade representing the extent of *X. malvacearum* leaf infection. The grading system consisted of 10 grades (8). Grade 1 represented no infection or immunity, grade 10, maximum infection and susceptibility. The grades were based on lesion size, shape, coalescence of lesions, and presence or absence of exudate (8).

RESULTS.—During the period from inoculation to grading in 1964 and 1965, the daily average relative humidity was 69.6% and 85.8%, the average temperature 27.1 C and 28.4 C, respectively. Total rainfall was 0.20 cm for 1964, 3.28 cm for 1965. Based on the known influence of these environmental variables, the disease grades should have been higher in 1965 than in 1964.

The over-all average disease grades for the parents were similar for the 2 years, but the F_1 population had higher average disease grades in 1965 than in 1964 (Tables 2, 3). The average disease grades of the parents and F_1's with race 1 were higher in 1964 than in 1965; the reverse was true for race 12. The average grades for the races and the mixture of the parental genotypes were similar in 1964, but in 1965 race 12 was more virulent than race 1. In both years, race 12 was more virulent than race 1 on the F_1 and F_2 populations, and virulence of the mixture was intermediate between both races (Tables 2, 3).

The combined analysis of variance of plot means for races indicated significant differences between races, parental genotypes, and race × genotype interaction.

Both races were effective in differentiating resistant parents from susceptible ones (Tables 2, 3). Among resistant parents, race 12 was more effective both years than race 1 in measuring the effects of the single major genes and gene combinations. Race 1 was more consistent than race 12 in differentiating effects of minor gene backgrounds among the susceptible parents. This resulted in a significant years × parents interaction for race 12 (Table 4).

In 1964, the parental lines with the genes B_4, B_2B_3, B_2B_6, and $B_2B_3B_7$ were highly resistant to both races

TABLE 1. Eight parental cotton lines used in studying the inheritance of resistance to races of *Xanthomonas malvacearum,* their designations and genotypes

Parents	Designations	Genotypes	Abbreviations[a]
146-25, UF 62	P_1	$b_2 b_2 b_3 b_3 B_4 B_4 b_6 b_6 b_7 b_7 B_{Sm} B_{Sm} b_{Dm} b_{Dm} b_{Tm} b_{Tm}$	$B_4 B_{Sm}$
Austin, UF 62	P_2	$b_2 b_2 b_3 b_3 b_4 b_4 b_6 b_6 B_7 B_7 B_{Sm} B_{Sm} b_{Dm} b_{Dm} b_{Tm} b_{Tm}$	$B_7 B_{Sm}$
14G, B.V. 61	P_3	$B_2 B_2 b_3 b_3 b_4 b_4 B_6 B_6 b_7 b_7 B_{Sm} B_{Sm} b_{Dm} b_{Dm} b_{Tm} b_{Tm}$	$B_2 B_6 B_{Sm}$
34G, B.V. 61	P_4	$B_2 B_2 B_3 B_3 b_4 b_4 b_6 b_6 b_7 b_7 B_{Sm} B_{Sm} b_{Dm} b_{Dm} b_{Tm} b_{Tm}$	$B_2 B_3 B_{Sm}$
91-92A, B.V. 61	P_5	$B_2 B_2 B_3 B_3 b_4 b_4 b_6 b_6 B_7 B_7 B_{Sm} B_{Sm} b_{Dm} b_{Dm} b_{Tm} b_{Tm}$	$B_2 B_3 B_7 B_{Sm}$
Deltapine TPSA, UF 62	P_6	$b_2 b_2 b_3 b_3 b_4 b_4 b_6 b_6 b_7 b_7 b_{Sm} b_{Sm} B_{Dm} B_{Dm} b_{Tm} b_{Tm}$	B_{Dm}
Empire WR, UF 62	P_7	$b_2 b_2 b_3 b_3 b_4 b_4 b_6 b_6 b_7 b_7 B_{Sm} B_{Sm} b_{Dm} b_{Dm} b_{Tm} b_{Tm}$	B_{Sm}
Texacala, UF 62	P_8	$b_2 b_2 b_3 b_3 b_4 b_4 b_6 b_6 b_7 b_7 b_{Sm} b_{Sm} b_{Dm} b_{Dm} B_{Tm} B_{Tm}$	B_{Tm}

[a] These abbreviations will be used in referring to the genotypes.

TABLE 2. Average disease grades of the parental cotton lines for each race of *Xanthomonas malvacearum* for the 1964 experiment

Parents	Genotypes	Race 1[a]	Race 12	R-1:R-12 mixture	Avg of races
1	$B_4 B_{Sm}$	1.64 a	1.60 a	1.55 a	1.60 a
4	$B_2 B_3 B_{Sm}$	1.47 a	1.68 a	1.71 a	1.62 a
5	$B_2 B_3 B_7 B_{Sm}$	1.72 a	2.07 b	1.87 ab	1.89 b
3	$B_2 B_6 B_{Sm}$	1.91 a	2.09 b	1.97 ab	1.99 bc
2	$B_7 B_{Sm}$	1.69 a	2.41 c	2.43 b	2.18 c
7	B_{Sm}	5.49 b	5.50 d	5.64 c	5.54 d
6	B_{Dm}	6.63 c	5.80 d	5.60 c	6.01 e
8	B_{Tm}	6.64 c	6.40 e	6.53 d	6.52 f
Avg of parents		3.40 a	3.44 a	3.41 a	3.42
Avg of F_1's		2.34 a	2.58 c	2.47 b	2.46

[a] Duncan's multiple range test for the 5% level.

TABLE 3. Average disease grades of the parental cotton lines for each race of *Xanthomonas malvacearum* for the 1965 experiment

Parents	Genotypes	Race 1[a]	Race 12	Avg of races
1	$B_4 B_{Sm}$	1.16 a	1.57 a	1.37 a
4	$B_2 B_3 B_{Sm}$	1.37 a	2.02 ab	1.70 ab
5	$B_2 B_3 B_7 B_{Sm}$	1.23 a	2.58 b	1.91 b
3	$B_2 B_6 B_{Sm}$	1.64 a	2.30 ab	1.97 b
2	$B_7 B_{Sm}$	1.24 a	4.36 c	2.80 c
7	B_{Sm}	5.78 b	5.81 d	5.80 d
6	B_{Dm}	6.00 bc	5.76 d	5.88 de
8	B_{Tm}	6.39 c	6.18 d	6.29 e
Avg of parents		3.10 a	3.82 b	3.46
Avg of F_1's		2.04 a	3.19 b	2.62
Avg of F_2's		2.59 a	3.73 b	3.16

[a] Duncan's multiple range test for the 5% level.

(Table 2). Austin (B_7) was highly resistant to race 1 and less resistant to the mixture and race 12. Empire WR, Deltapine TPSA, and Texacala were susceptible to both races.

The same trend occurred in 1965. The first four genotypes were highly resistant to both races (Table 3). The B_7B_7 genotype was highly resistant to race 1 (grade 1.24), but approached susceptibility in the presence of race 12 (grade 4.36). Parents 6, 7, and 8 were susceptible to both races (grades 5.49-6.64).

The B_4 gene and B_2B_3 gene combination were more consistent in conditioning higher levels of resistance (grades 1.16-2.02) to both races across years, as compared with the other genes. Although the $B_2B_3B_7$, B_2B_6, and B_7 genes conferred high resistance, significant differences existed among them, especially in the presence of race 12 (Tables 2, 3). Averaged over races and years, the order of resistance and significant grouping of genes and gene combinations was as follows: B_4 and B_2B_3 conditioned the highest level of resistance, followed by B_2B_6 and $B_2B_3B_7$, which were equal, and B_7.

The combination $B_2B_3B_7$ tended to be more susceptible than B_2B_3, especially in the presence of race 12 (Tables 2, 3). This indicated that under some conditions the B_7 gene exerted a negative effect on the B_2B_3 gene combination.

In 1964, the B_{Sm} minor gene complex was more effective in the presence of race 1 than either B_{Dm} or B_{Tm} (Table 2). B_{Sm} and B_{Dm} were more effective in the presence of race 12 than the B_{Tm} minor gene complex. In 1965, the effect of minor genes on plant reaction to race 1 was similar to the 1964 test, but to race 12 they were all equal in effect (Table 3).

Average disease grades for the F_1's followed two patterns when race 1 was used in 1965 (Table 5). First, in crosses between parents with the major genes (parents 1-5), the F_1's were highly resistant. This was also true in crosses between resistant and susceptible parents in which the F_1 values were close to the resistant parents. Second, F_1's derived from crosses between the susceptible parents (parents 6-8) were all susceptible. Average disease grades for the F_2's gave

TABLE 4. Analysis of variance of average disease grades of the parental cotton lines for *Xanthomonas malvacearum* races 1 and 12

Source of variation	d.f.	Race 1		Race 12	
		Mean squares	F	Mean squares	F
Years	1	1.0681	16.83[a]	1.7139	21.83[a]
Parents	7	35.2027	554.68[a]	23.1176	294.43[a]
Blocks within years	4	0.0243	0.38	0.1019	1.30
Years × Parents	7	0.1258	1.98	0.6913	8.80[a]
Error	28	0.0635		0.0785	
Total	47				

[a] Significant at the 1% level.

TABLE 5. Frequency distributions of disease grades for the F_2 generation, and average parental, F_1, and F_2 disease grades in the presence of *Xanthomonas malvacearum* race 1

Crosses	% Plants within each disease grade[b]									Avg disease grades[c]			
$P_A \times P_B$[a]	1	2	3	4	5	6	7	8	9	P_A	P_B	F_1	F_2
1 × 2	58.1	25.6	7.0	6.2	3.1	0	0	0	0	1.16	1.24	1.34	1.70
1 × 3	64.5	26.0	7.1	1.6	0.8	0	0	0	0	1.16	1.64	1.24	1.48
1 × 4	70.4	23.0	2.5	3.3	0.8	0	0	0	0	1.16	1.37	1.28	1.40
1 × 5	69.3	26.9	3.8	0	0	0	0	0	0	1.16	1.23	1.19	1.35
1 × 6	42.0	18.3	9.2	4.6	8.4	9.9	3.8	3.8	0	1.16	6.00	1.24	2.83
1 × 7	50.0	25.4	7.1	5.6	3.2	6.3	2.4	0	0	1.16	5.78	1.40	2.16
1 × 8	42.2	22.7	9.4	3.9	4.7	8.6	2.3	3.9	2.3	1.16	6.39	1.45	2.77
2 × 3	57.2	36.6	3.1	2.3	0.8	0	0	0	0	1.24	1.64	1.43	1.53
2 × 4	47.2	36.6	8.9	4.9	2.4	0	0	0	0	1.24	1.37	1.44	1.79
2 × 5	70.2	25.2	4.6	0	0	0	0	0	0	1.24	1.23	1.24	1.34
2 × 6	29.1	29.1	15.0	7.9	6.3	7.9	2.3	1.6	0.8	1.24	6.00	1.82	2.78
2 × 7	40.2	27.5	11.8	3.9	7.1	7.9	1.6	0	0	1.24	5.78	1.82	2.39
2 × 8	32.3	26.8	6.3	4.7	6.3	9.4	9.4	3.2	1.6	1.24	6.39	1.87	3.17
3 × 4	50.4	45.7	3.9	0	0	0	0	0	0	1.64	1.37	1.41	1.53
3 × 5	52.7	43.4	3.9	0	0	0	0	0	0	1.64	1.23	1.41	1.51
3 × 6	18.7	41.5	17.1	8.1	5.7	6.5	2.4	0	0	1.64	6.00	2.15	2.70
3 × 7	26.0	37.8	17.3	7.1	5.5	4.7	1.6	0	0	1.64	5.78	1.78	2.48
3 × 8	14.6	40.8	19.2	6.1	5.4	4.6	5.4	3.1	0.8	1.64	6.39	1.97	3.02
4 × 5	71.2	27.3	1.5	0	0	0	0	0	0	1.37	1.23	1.36	1.30
4 × 6	40.5	31.8	6.3	4.0	7.9	6.3	1.6	1.6	0	1.37	6.00	1.51	2.40
4 × 7	36.9	28.5	13.8	5.4	5.4	5.4	2.3	2.3	0	1.37	5.78	1.47	2.51
4 × 8	31.3	28.3	9.9	7.6	3.8	6.1	6.9	4.6	1.5	1.37	6.39	1.91	3.02
5 × 6	36.8	40.8	12.0	2.4	4.8	1.6	1.6	0	0	1.23	6.00	1.60	2.09
5 × 7	48.9	28.2	9.2	6.1	2.3	3.8	1.5	0	0	1.23	5.78	1.57	2.03
5 × 8	26.3	36.4	18.6	6.2	3.1	2.3	4.7	0.8	1.6	1.23	6.39	1.71	2.63
6 × 7	0	0	4.6	19.1	25.2	32.1	11.4	6.1	1.5	6.00	5.78	5.68	5.51
6 × 8	0	0	0.8	4.0	15.1	34.1	22.2	14.3	9.5	6.00	6.39	6.49	6.54
7 × 8	0	0	0.8	7.1	15.1	23.0	34.1	13.5	6.4	5.78	6.39	6.46	6.46

[a] P_A and P_B refer to the two parents of each cross.

[b] Grade 1 = immune; grade 9 = fully susceptible.

[c] Plant populations for each parent and per cross ranged from 59-66 for the parental lines, 79-120 for F_1's, and 122-132 for F_2's.

the same pattern as the F_1's, but the values obtained were slightly higher.

When race 12 was used in 1965, the F_1 averages indicated three patterns (Table 6). First, crosses between parents 1, 3, 4, and 5 and all other genotypes resulted in the most resistant F_1's. Second, in crosses between parent 2 (B_7) and parents with the major genes, the F_1 averages were close to the mid-parent. The F_1 averages were close to the susceptible parents in crosses between parent 2 and the three susceptible parents. Third, F_1 averages from crosses between the three susceptible parents were all in the susceptible range. The F_2 averages followed the same pattern as the F_1's.

All gradations in disease grades between the parental values were observed in the F_2 populations. The ranges for the F_2 populations were smaller with race 1 than with race 12 (Tables 5, 6). It seems evident from the data that isodirectionality for the major genes conditioning resistance was complete. The skewness due to directional dominance towards resistance was obtained for all major genes. The B_7 gene was an exception when inoculated with race 12. In crosses between parent 2 and the susceptible parents, F_2 frequency distributions approached normality, and they were shifted towards the susceptible side (Table 6). In crosses between the three susceptible parents, F_2 frequency distributions approached normality.

The recovery of resistant plants in the F_2 progenies from crosses between the susceptible parents indicated transgressive segregation (Tables 5, 6). It was expressed most in the F_2 progeny derived from the $P_6 \times P_7$ cross. The range of disease grades for the two parents was 4 to 8, with more than 90% of the population over grade 5. In the presence of both races, 4.6% of the F_2 population was resistant (grade 3).

The data presented in Tables 5 and 6 further show gene effects when transferring major genes to different genetic backgrounds. The three backgrounds are attributed to different minor gene complexes in the susceptible parents. Average disease grades for the F_1's and F_2's were generally lower and the ranges smaller in crosses between the five parents having the major genes and parents 6 and 7 than in crosses with parent 8. Also, more susceptible plants were obtained in crosses with parent 8 than in those with parents 6 and 7. It is evident from these results that the B_4 gene was very effective in the three backgrounds, but most effective in Empire WR. The B_7 gene combined more favorably with the B_{Sm} minor genes of Empire, and B_2B_3 genes with the B_{Dm} minor genes of Deltapine. The B_2B_6 and $B_2B_3B_7$ gene combinations were effective in both the Deltapine and Empire backgrounds. The B_4 gene and B_2B_3 gene combination were the most effective in Texacala (B_{Tm}).

DISCUSSION.—Normally, lesions of bacterial blight

TABLE 6. Frequency distributions of disease grades for the F_2 generation, and average parental, F_1, and F_2 disease grades in the presence of *Xanthomonas malvacearum* race 12

Crosses	% Plants within each disease grade[b]									Avg disease grades[c]			
$P_A \times P_B$[a]	1	2	3	4	5	6	7	8	9	P_A	P_B	F_1	F_2
1 × 2	25.8	46.9	7.1	0	2.3	7.8	7.8	2.3	0	1.57	4.36	1.67	2.72
1 × 3	32.6	45.7	11.6	4.7	5.4	0	0	0	0	1.57	2.30	1.62	2.05
1 × 4	29.4	46.0	12.7	8.7	3.2	0	0	0	0	1.57	2.02	1.52	2.11
1 × 5	28.2	51.2	10.7	6.1	3.8	0	0	0	0	1.57	2.58	1.87	2.06
1 × 6	28.1	34.4	6.2	4.7	8.6	7.8	7.8	1.6	0.8	1.57	5.76	1.67	2.99
1 × 7	29.8	38.9	10.7	2.3	6.1	8.4	3.8	0	0	1.57	5.81	1.62	2.56
1 × 8	22.0	35.4	8.7	6.3	8.7	5.5	8.7	3.9	0.8	1.57	6.18	1.72	3.20
2 × 3	5.6	36.0	26.4	12.0	9.6	8.0	2.4	0	0	4.36	2.30	2.99	3.19
2 × 4	6.9	38.9	25.2	16.8	5.3	3.8	2.3	0.8	0	4.36	2.02	2.71	2.99
2 × 5	0.8	17.7	21.5	26.9	18.5	9.2	4.6	0.8	0	4.36	2.58	3.16	3.94
2 × 6	0	0.8	6.1	18.5	26.9	28.5	14.6	3.8	0.8	4.36	5.76	5.65	5.38
2 × 7	0	3.9	7.1	20.5	26.0	28.3	12.6	1.6	0	4.36	5.81	5.48	5.10
2 × 8	0	0	6.9	11.4	15.3	29.0	25.2	8.4	3.8	4.36	6.18	6.08	5.94
3 × 4	16.3	48.0	28.7	6.2	0.8	0	0	0	0	2.30	2.02	2.47	2.28
3 × 5	8.4	41.2	36.6	11.5	2.3	0	0	0	0	2.30	2.58	2.75	2.58
3 × 6	6.2	30.0	19.2	20.0	10.8	6.9	5.4	1.5	0	2.30	5.76	2.51	3.49
3 × 7	0	23.2	32.0	22.4	8.8	11.2	1.6	0.8	0	2.30	5.81	2.79	3.60
3 × 8	0	20.5	20.5	18.9	15.0	9.4	10.2	4.7	0.8	2.30	6.18	2.67	4.28
4 × 5	4.7	52.0	29.1	9.5	4.7	0	0	0	0	2.02	2.58	2.43	2.59
4 × 6	11.1	28.6	20.7	12.7	9.5	9.5	7.9	0	0	2.02	5.76	2.26	3.41
4 × 7	8.1	35.5	21.0	12.9	7.2	12.9	1.6	0.8	0	2.02	5.81	2.55	3.26
4 × 8	5.5	40.6	22.7	10.9	3.9	3.9	7.8	4.7	0	2.02	6.18	2.59	3.34
5 × 6	1.5	16.2	23.9	22.3	13.8	12.3	8.5	1.5	0	2.58	5.76	2.98	4.11
5 × 7	3.8	18.5	24.6	15.4	16.1	15.4	5.4	0.8	0	2.58	5.81	3.39	3.93
5 × 8	1.6	8.3	24.8	20.7	13.2	14.0	8.3	5.0	4.1	2.58	6.18	3.06	4.61
6 × 7	0	0.8	4.6	5.4	22.5	25.6	30.2	7.0	3.9	5.76	5.81	5.77	6.05
6 × 8	0	0	0.8	11.2	17.6	27.2	24.0	14.4	4.8	5.76	6.18	6.71	6.24
7 × 8	0	0	0	4.7	15.0	28.3	27.6	16.5	7.9	5.81	6.18	6.71	6.58

[a] P_A and P_B refer to the two parents of each cross.
[b] Grade 1 = immune; grade 9 = fully susceptible.
[c] Plant populations for each parent and per cross ranged from 58-66 for the parental lines, 82-115 for F_1's, and 121-131 for F_2's.

are expected to be larger in a wet environment than a dry one. This was true with race 12, and the opposite with race 1. This suggests that the components of a given environment may be less important than the race of the pathogen in determining lesion size.

The influence of environment on host resistance and susceptibility has been emphasized by several workers, and was recently reviewed (18). It was noted in our results that some of the genotypes, especially B_4B_4 and $B_2B_2B_3B_3$, were more stable against environmental fluctuations than others. The B_7B_7 genotype was influenced the most by environment.

Differences in virulence between the two races were more evident among F_1 and F_2 populations than among the homozygous parents. Race 12 was more virulent than race 1. Virulence of the mixture of races 1 and 12 was generally intermediate between both races, and suggests that the effectiveness of race 12 was partly reduced by the presence of race 1.

Both races were more effective in measuring the effects of the major and minor genes in the drier 1964 environment than they were in 1965. This was especially true for race 12, which indicates that in a dry environment races such as race 12 would be best for a program of developing blight-resistant cottons. However, this would probably lead to selection for specific resistance to race 12. The best alternative would be to use a mixture of races to select for general resistance. Bird (3) has shown that a mixture of four to six races provides a broader base of pathogen variability and intense selection pressure for identifying favorable levels of genetic resistance.

In the B_{Sm} minor gene background, the B_4 gene was more effective than B_7, and as effective or better than the gene combinations B_2B_3, B_2B_6, or $B_2B_3B_7$. Among the minor gene backgrounds, the B_{Sm} gene complex was the most effective. Consistently high resistance, over both races and years, was obtained with the more effective major genes. This points out that resistance or near immunity is dependent not only on the number of genes in a strain but also on the effectiveness of combined major and minor genes. The results of this study agree with Knight & Hutchinson (26) that effective resistance could only be established by utilizing the major genes. Materials with two or more minor gene complexes, such as B_{Dm} and B_{Sm}, would provide an excellent genetic background for the major genes to obtain immunity. This would provide a broader base for stable resistance to the variability of the pathogen.

Differences in the F_2 distribution patterns in crosses with the three susceptible parents are due to the effect of minor genes on the expression of the major genes. Also, the intensifying effect of minor genes resulted in a slight deficit of susceptible plants in each of the F_2 progenies. Transgressive segregation for the minor genes indicates that they are different gene complexes.

The minor gene complex of Texacala is designated B_{Tm}.

The data confirm the importance of using different genetic backgrounds in studying disease resistance in cotton (8). Similarly, genes conditioning resistance should be compared in related genetic backgrounds.

Results from the F_1 and F_2 generations indicate that dominance effects of the major genes were in the direction of resistance to blight, except B_7 in the presence of race 12.

Simpson & Weindling (32) selected Stoneville 20, a blight-resistant strain of Upland cotton from Stoneville 2A. Blank (11) described a single gene conditioning Stoneville 20 resistance; it was designated B_7 by Knight (23). The present data indicated two patterns of gene action for the B_7 gene when inoculated with the two races. Resistance was due to dominant gene action in the presence of race 1 (Table 5). When inoculated with race 12, additive gene action was indicated (Table 6). Also, different levels of resistance were obtained when crossing the B_7B_7 genotype with the three minor gene backgrounds. These results confirm that the expression of the B_7 gene was influenced by minor genes (7, 8, 16, 23).

The method of classification has a direct bearing on the genetic interpretation of results, especially when plants are classified into resistant and susceptible classes only. To elucidate this point, if we consider that grade 1 represents the resistant class and plants within grades 2-10 the susceptible class, then resistance would be interpreted as a recessive character with susceptibility being dominant (Table 6). Alternatively, when grades 1-3 represent resistance and 4-10 susceptibility, the conclusion would be that resistance is inherited as a dominant character and susceptibility is recessive. Therefore, it is important that the grading system should adequately describe the range of phenotypic variability of disease symptoms.

The physiologic race of pathogen, genetic background of host, method of assessing resistance, and environment could explain the different reports on the inheritance of the B_7 gene as to whether resistance to blight was dominant (23) or recessive (7, 11, 16, 27). Further explanation for these differences may be drawn from recent studies (10, 22, 29, 30) suggesting that there is more than one physiological and/or biochemical pathway that controls and influences resistance to races of *X. malvacearum* in cotton.

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