



Reaction of some Segregating Populations and Newly Introduced Cotton (*Gossypium hirsutum*) Germplasm to *Xanthomonas campestris* pv. *malvacearum*

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ABSTRACT

Resistance of cotton cultivars in the Sudan to *Xanthomonas campestris* pv. *malvacearum* conferred by $B_2 B_6$ gene combination is no longer effective after the appearance of new races of the pathogen. Thus, new sources of resistance or new gene combinations are needed to protect the crop. This study was carried out to identify such a new source or gene combination to confer resistance to the prevailing races in the Sudan. Highly resistant plants from some F_2 plants were recovered. The S295 cotton cultivar (B_{12}) showed high resistance to post-Barakat race. Some MAR cotton germplasm (MAR 5 & 6) was also showed high resistance to this race. This high resistance was also observed in the F_4 segregating populations of the introduced crosses $E1043 \times B_2 B_6$ and $B_2 B_6 \times Pima 32$.

Introduction

Bacterial blight [*Xanthomonas campestris* pv. *malvacearum* (Smith) Dye] is a serious disease on cotton that occurs in most producing regions of the world. It attacks the leaves (angular leaf spot), stems and branches (blackarm) and bolls (bacterial boll rot). The pathogen can survive on undecomposed plant debris and in planting seed. In regions experiencing strong wind-driven rain that disseminates the pathogen, the disease becomes very destructive.

Sanitary practices using acid-delinted and fungicide treated seed and removal of plant debris of the previous crop are used in areas where the disease occurs to minimize loss in yield and quality but host plant resistance is the only practical control measure. Genetic analysis of resistance to bacterial blight has led to the identification of twenty major resistance genes. There have been different reports on the effectiveness of specific "B" genes and gene combinations in conferring resistance (Knight and Hutchinson, 1950; El Zik and Bird, 1970) The real challenge in developing immune cotton cultivars to bacterial blight is the utilization of wide genetic variability. Saunder & Innes (1963) stated that certain "B" gene combinations do give additive effects under Sudan conditions.

The resistant cotton cultivars Barakat, Barac 67B and Albar (57)12 are grown in the Sudan. The gene combination present in these cultivars is $B_2 B_6$ but the appearance of the new race (post-Barakat) has nullified the resistance conferred by this gene combination. There have been several reports on major genes in addition to the minor genes in developing cotton cultivars that are highly resistant to bacterial blight (Last, 1959; Brinkerhoff, 1959; Knight, 1964). This study was to identify new sources or new gene combinations to confer resistance to the new race in the Sudan.

Material and Methods

Reba w296 (B_9), Acala (93)H ($B_2 B_6$), Acala (93)M ($B_2 B_6$), cotton cultivars, Introduction 5 and Introduction 15 from the USA and Bar 58 (Wild $B_4 B_{11}$) were used as parents. Crosses were made in the field and the F_1 seedlings were grown in the greenhouse to produce the F_2 seeds. The F_2 , F_1 and the parents were grown in 450×85 cm soil beds in the greenhouse in a randomized block design with three replications. Fully expanded cotyledons of each plant were inoculated using the toothpick scratch inoculation method (Bird, 1986). Post-Barakat race was cultured in petri dishes on potato carrot dextrose agar (PCDA) at $25^\circ C$. Inoculum was prepared from 5-7 day-old cultures. The suspension of bacteria was approximately 1.0×10^6 cells/ml. Disease reactions were rated 15 days after inoculation on scale of 1 to 10, 1 representing immunity and 10 severe susceptibility (Bird and Hadley, 1958). The plants were then allowed to grow and inoculated again at the six leaf node using the same method of inoculation. Histograms and mean disease grades were only taken from true leaves data.

MAR (5&6) lines, cotton cultivars and the segregating populations of the crosses $E1014 \times B_2 B_6$ and $B_2 B_6 \times Pima 32$ materials (Table 1) were kindly provided by Dr. Kamal El Zik of the Texas A&M University. Seedlings of these lines and cultivars were grown in 25×32 cm clay pots in the greenhouse in a randomized complete block design. Three pots representing each line or a cultivar in each replication and each pot contained 5-6 plants. Only cotyledons were inoculated using the same inoculation technique and inoculum but only cotyledons were inoculated. The experiment was conducted in the greenhouse with a daily average temperature of $27^\circ C$.

Results

F₂ population of the cross Acala (93) H × Introduction 5 produced the lowest disease grade (4.3), followed by Reba w296 × Introduction 5 (6.2), Reba w296 × Bar 58 (5.6). The highest disease grade was produced by the F₂ of the cross Acala (93) M × Introduction 5 (6.5). The analysis of disease grade in all F₂ populations for all crosses revealed significant differences, indicating different genes were segregating for resistance. F₂ individuals that were more resistant than their parents were recovered in these crosses. This indicated transgressive segregation for resistance in these parents. The distributions of disease grade in the F₂ populations of these crosses inoculated with post-Barakat race are discrete (Figs 1-4). In F₂ population of the crosses Reba w296 × Introduction 5, Reba w296 × Bar 58 and Acala (93) M × Introduction 15, some plants showed immunity or high resistance (Figs 1-4). In these figures three peaks or more representing resistant and susceptible individuals can be seen. The F₂ population of the cross Acala (93) H × Introduction 5 showed two peaks (Fig.4). Average disease grade for introductions are shown in Table 1. The MAR-5 BLLCAGYBS-3-86 breeding line showed immune reaction. LBBCHU2GS-1-87 (MAR 5), Deltapine 5409 and LBBCDBOAKH-1-90 (MAR 6) showed high resistance. MAR lines; CABD3CABCH-1-89, CA3HCHULBH-1-88, CD3HCABCUH-1-89, LBBCABCHUS-1-87 and Tamcot HQ95 showed moderate resistance. The remaining lines and cultivars showed susceptible reaction (Table 1). The average disease grade of the two introduced crosses E1043 X B₂ B₆ and B₂ B₆ X Pima 32 were 1.8 and 1.5 respectively.

Discussions

The discrete distributions of the F₂ disease grade indicated that resistance was governed by major genes. Although the number of the F₂ populations in all crosses was very small, resistant plants were recovered. If this number was large, we could get different representative disease grades and hence more detailed distributions. Transgressive segregation for resistance observed in the F₂ of these crosses indicated that there were different genes in the two parents segregating for resistance.

In the cross Reba W296 × Introduction 5, B₉ resistance gene is present in Reba W296 and B₃, B₄ and B₇ that may present in Introduction 5 (Introduction from the USA), could be responsible for the observed resistance. The resistance observed in the F₂ population of the cross Reba W296 × Bar 58 is possibly due to B₄ B₉ and B₁₁ that may present in Bar 58. B₃ B₄ or B₇ in addition to B₂B₆ present in Acala (93) M could be responsible for the observed resistance in the F₂ of the cross Acala (93) M × Introduction 15.

Innes (1965) reported the effectiveness of B₂B₉, B₂B₄ gene combination which gave high resistance similar to B₂B₆ gene combination at that time.

The combination of two or more genes and a modifier complex was found very effective and gave high resistance (El Zik & Bird, 1970; Innes 1974; Bird 1982). The combinations of two or more major genes from B₂, B₃, B₄, B₆, B₇ and B₉ that were probably present in the parents of the above crosses might be responsible for the observed resistance. Resistance observed in the MAR 5 & 6 material could be attributable to the gene combination B₂ B₃ B₇ or B₂ B₃ B₆ B₇ present in this material. Differences in disease grade for the same gene combination in different genotypes may be due to the genetic background of these genotypes or a minor gene complex. LBBCABCHUS-3-86 (MAR5) breeding line showed immune reaction to post-Barakat race. This resistance could be due to the effect of B₂ B₃ B₇ gene combination, most probably enhanced by other minor genes; since other genotypes having the same gene combinations produced different reactions. The B₂B₃, B₂B₆, B₂B₃B₆ gene combinations were found to be susceptible to the new race in the Sudan. Therefore, either B₂B₇, B₃B₇ or B₂B₃B₆B₇ gene combinations were responsible for the resistance observed. Resistance to post-Barakat race showed by S295 cotton cultivar may be due to the major resistance gene B₁₂. These results agreed with Wallace and El Zik (1992) and Wallace (1987). Resistance showed by the introduced crosses E1014 B₂B₆ and B₂B₆ Pima 32 may be due to other major genes in E1014 or Pima 32 compatible with B₂B₆ or other minor genes present in these parents or both.

In conclusion, it is possible to develop resistant cotton cultivars to the prevailing bacterial race (s) in the Sudan from a compatible gene combination using B₂, B₃, B₄, B₆, B₇, B₉ and B₁₂. The pedigree and the backcross selection methods are adequate for such purpose.

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Table 1. Average disease grade₁ of introduced germplasm including MAR (5&6), cotton cultivars and two segregating generations.

Genotype	Grade	Genotype	Grade	Genotype	Grade
Tamcot CAB-C8	5.4	B ₂ B ₆ Pima 32	1.5	LBBCC4HUGS-1-89	3.7
Tamcot HQ95	3.6	B51	8.2	CABD3SHP35-1-90	4.4
Tamcot SPHINX	4.0	K210	8.5	BLCABPD86S-1-90	4.2
Deltapine 50	4.7	S295	3.3	MAR5PD2085-4-90	3.9
Deltapine 5409	1.8	CABCHUS-2-86	5.8	CAHUGARPIH-1-88	6.5
Paymaster HS26	4.7	BLLCABS-3-86	1.0	CD3HHARCIH-1-88	6.2
Paymaster 280	5.0	LBBCABCHUS-1-87	2.5	CD3HCAHUGH-2-88	3.8
Paymaster 330	4.9	LBBCHUSGS-187	1.5	CD3HCHULBH-1-88	2.9
Stoneville 474	6.6	CDP37HPIH-1-86	4.1	CABD3CABCH-1-89	2.6
Stoneville 887	5.7	LBBCD3H-1-87	4.0	CD3HCABCUH-1-89	2.6
E1043 B ₂ B ₆	1.8	CAHUGLBBCS-1-88	4.2	LBBCDBOAKH-1-90	1.4

₁ denotes immunity and grade 10 denotes full susceptibility.