Inheritance of resistance to bacterial blight in some crosses of cotton

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ABSTRACT

*Five generations, P_1, P_2, F_1, F_2, and F_3 of each of four cotton cultivars (*Gossypium hirsutum*) crosses were utilized in the genetic analysis of bacterial blight resistance in cotton by using generation means analysis. Analysis of generations derived from these cultivars showed that the genetic variation in bacterial blight resistance was controlled by additive gene action. Non-allelic interaction and dominance genetic effects were detected in some crosses. The additive × additive gene effects were predominant. These results indicated that breeding for resistance improvement to bacterial blight is practicable through selection.*

Introduction

Bacterial blight of cotton affects all plant parts during all growth stages. The disease can be extremely damaging under the Sudan conditions and particularly to long staple cotton *Gossypium barbadense*. Therefore, if improvement of resistance is to be made an understanding of its genetical basis is essential. Quantitative analysis to bacterial blight indicated additive, dominance, and epistatic gene action (Bird, 1960; El-Zik and Bird, 1967; Innes *et al*., 1974; Wallace and El-Zik, 1990). A substantial number of diallel studies indicated the importance of both additive and dominance effects on bacterial blight resistance (Innes and Brown, 1969, 1974; El-Zik and Bird, 1967; Luckett, 1989; Wallace and El-Zik, 1990).

Estimates of gene effects involved in the inheritance of bacterial blight resistance from four cotton cultivars crosses are reported in this paper.

Experimental procedure

A half diallel mating system was initiated by crossing four *Gossypium hirsutum* cotton cultivars: (Acala (93) H, S295, LEBO-1-78, and Tamcot HQ 95. These parents were selected because of their moderate resistance to bacterial blight and their different genetic background. Acala (93) H is a newly released cotton cultivar in the Sudan, S295 is a cotton cultivar from Chad, LEBO-1-78, and Tamcot HQ 95 are cotton cultivars from the USA. Crossing and selfing of the four parents produced six F_1, six F_2, and six F_3 for a total of 24 populations for each set of parents. The parents, F_1, F_2, and F_3 generations were grown in the field using a randomized complete block design with three replications. Plots were 12.5 m in length and 0.8 m wide, with 50-cm hill spacing, two plants per hill. Parental and F_1 generations were grown in one-row plots. The F_2 and F_3 progenies were grown in three-rows plots.

Field inoculation and scoring of disease severity described by Knight (1946) were used. They involved spraying the undersurface of the leaves of six-week old plants with a suspension of bacteria (Post Barakat race) prepared by soaking 5 lb air-dried infected leaves in 40 gallons of water for 2 hours, after which the suspension is strained through sacking. "Knapsacks sprayers" were used for spraying twice a day for two successive days. Disease severity was recorded 21 days after inoculation using scale of (0-10). Zero represents immunity and 10 represents full susceptibility.

Adequacy of the additive-dominance model was tested using Hayman and Mather scales (1955). Gene effects for bacterial blight were estimated using the five-parameter model following Hayman (1958). The significance of gene effects was tested by calculating variances, standard error, and 't' values separately for each effect (Singh & Chaudhary, 1977).

Results and Discussion

Mean disease grades ranged from 4.4 to 5.6 for parental cultivars and from 4.4 to 5.3 for F_1 populations grown in the field (Table 1). Analysis of variance indicated significant differences for leaf disease grade among different populations. To ascertain the type of gene action involved in determining the variation between generation means, the results obtained by scaling tests and model-fitting procedures to the generation means are shown in Tables 1 and 2. A satisfactory fit of expected and observed generation means was obtained with the three-parameter model for all crosses (Table 3). Both simple scaling test C and D were not significant indicating the absence of non-allelic interaction. Therefore, the additive dominance model is adequate to explain genetic variability for blight resistance in these crosses. The joint scaling test indicated the adequacy of the additive dominance model for crosses (3, 4, 5, and 6). Therefore, only additive dominance effects were involved to determine variation between generation means in these crosses. Conversely, the joint scaling test indicated the inadequacy of the additive dominance model in crosses, (1 and 2), and hence indicated the presence of epistasis (Table 2). The additive × additive type of epistatic interaction (fixable component) was significant for crosses (1, 2 and 6). While, dominance × dominance type of epistatic interaction was not significant for all crosses (Table 3). This implies the importance of additive × additive type of epistatic interaction in these crosses. Significant additive genetic effects (d) were detected for all crosses (Table 3). Whereas, significant dominance (h) genetic effects were detected for crosses (2 and 6). This indicated the predominance of additive genetic effects in the inheritance of bacterial blight resistance. These results agreed with (Green and Brinkerhoff, 1956; Bird and Handley, 1958; Bird, 1960; Arnold, 1963; El-Zik, 1967; Innes and Brown, 1969; Innes *et al*., 1974; Wallace and El-Zik 1990) who showed that most of the genetic variance was accounted for by additive, dominance, and

epistatic gene action. The predominance of additive × additive type of interaction along with prominent additive effects would imply some possibility for selection in the segregating generations. The additive and non-additive components can be utilized by inter-mating of desirable segregants followed by selecting superior genotypes. This study suggests the possibility of breeding for improvement in resistance to bacterial blight disease.

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Table 1. *Mean disease grade for parental and F_1 populations inoculated in the field with Post Barakat race.*

	P1	P2	P3	P4
P1	4.4	4.8	4.7	4.4
P2		4.6	4.6	5.3
P3			5.6	5.0
P4				5.4

Grades based on a scale 1 (higly resistant) to 10 fully susceptible

Table 2. *Scaling tests and their standard errors.*

	Cross [b]					
Scaling test [a]	1	2	3	4	5	6
C	- 0.6 ± 0.8	- 0.2 ± 0.8	0.1 ± 1.1	- 0.3 ± 1.2	0.4 ± 1.2	- 0.3 ± 0.8
D	- 0.4 ± 0.7	- 1.4 ± 0.7	- 0.9 ± 1.2	0.1 ± 1.2	- 1.6 ± 0.8	- 1.5 ± 0.7
χ^2 for joint scaling test						
Additive dominance[1]	19.2*(2)	14.3*(2)	4.9(2)	4.8(2)	4.8(2)	5.93(2)

*P < 0.05

[1] Figure between parentheses indicates degrees of freedom.

[a] Scaling tests are: *C - dominance and D - additive.*
[b] Crosses are: **1** - Acala (93) H×S295, **2** – Acala (93) H×*LEBO2-1-78,* **3** - Acala (93) H× Tamcot HQ 95, **4** - S295× *LEBO2-1-78,* **5** - S295× Tamcot HQ 95 and **6** - *LEBO2-1-78*× Tamcot HQ 95 respectively.

Table 3. *Weighted least square estimates and their standard errors.*

Genetic	Crosses [b]					
Parameters [a]	1	2	3	4	5	6
m	4.8 ± 0.20	4.7 ± 0.21	4.4 ± 0.28	4.6 ± 0.28	5.3 ± 0.20	5.0 ± 0.21
d	0.50* ± 0.09	0.30* ± 0.07	0.15* ± 0.07	0.15* ± 0.08	0.2* ± 0.09	0.35* ± 0.09
h	0.47 ± 0.49	1.2* ± 0.57	0.47 ± 0.92	- 0.27 ± 0.92	1.3 ± 0.68	1.2* ± 0.60
l	0.27 ± 1.70	- 1.60 ± 1.90	- 1.3 ± 2.70	0.53 ± 2.7	- 2.7 ± 2.40	- 1.6 ± 01.9
i	1.2* ± 0.56	1.5* ± .60	0.85 ± 0.88	0.45 ± 0.88	1.5 ± 0.77	1.7* ± 00.62

*P < 0.05

[a] Genetic Parameters are: *m - mean, d - additive, h - dominance, I- d×d interaction, l - h×h interaction components*
[b] Crosses are: **1** - Acala (93) H×S295, **2** – Acala (93) H×*LEBO2-1-78,* **3** - Acala (93) H× Tamcot HQ 95, **4** - S295× *LEBO2-1-78,* **5** - S295× Tamcot HQ 95 and **6** - *LEBO2-1-78*× Tamcot HQ 95 respectively.