Inheritance of resistance in cotton cultivars to the HV1 isolate and a mixture of two races of *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye in Nigeria

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ABSTRACT

Cotyledons and true leaves of cotton cultivars 'S295', TAMCOT CAMD-E, SAMCOT-6 and SAMCOT-8 and their F_1, F_2 and backcross progenies were inoculated with the HV1 isolate and a mixture of races 7 and 10 of Xanthomonas campestris *pv* malvacearum. *Race 10 is the most virulent and widely distributed in Nigeria. Disease reactions were rated on a scale of 1-10. Disease grade frequency distributions in the crosses involving S295 and the susceptible SAMCOT varieties indicated a single gene with complete dominance for resistance to the race mixture and HV1 isolate in S295. Frequency distribution also indicated a single gene difference between S295 and multi-adversity resistant cultivar, TAMCOT CAMD-E that confers resistance in S295 to the HV1 isolate. This resistance appears to be conferred by two closely linked genes that behave like a single with a large effect. The implications of these results in breeding immune or highly resistant cotton cultivars to Xcm in Nigeria are discussed.*

Introduction

Bacterial blight of cotton, induced by *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye occurs throughout the cotton-producing areas of the world. In Nigeria, it is the most economically important disease of cotton, causing an estimated annual yield loss of 10 – 20% (Dransfield, 1965). Losses to bacterial blight on an annual basis, depending on the disease manifestation and cotton species, have been shown to vary from 5 – 80% in some parts of Africa (Innes, 1983). Management involving exclusion, eradication and sanitation control the disease in areas such as California, in the United States of America, where it is practicable to enforce regulations governing these actions (Presley and Bird, 1969). In most cotton growing areas of the world, it is difficult to use management alone for control. In Nigeria for example, there is the "close season" regulation. To "close the season" for any cotton field, it is recommended that all plants be uprooted or cut at the soil-line and burnt. This annual operation is to commence as from mid-March and to last up to first week of June prior to the beginning of the new season. However, this legal and mandatory operation has not been followed strictly by farmers. None adherence to cotton "close season" has resulted in cotton ratoon. The ratoon or off-season cotton is a reservoir for insects and diseases that hibernate in alternative host plants. Although, chemical seed treatment in Nigeria and other African countries is a popular and effective method of minimizing disease loss; breeding for and use of resistant varieties has been shown in different parts of the world to be the most economic means of controlling the disease (Bird, 1986; Alabi *et al*., 2000). Breeding for resistance to bacterial blight has been an integral component of the overall cotton improvement program at the Institute for Agricultural Research, Samaru, Zaria. To date, several cotton varieties varying in levels of resistance to bacterial blight have been developed and released for commercial production (Alabi, 1995). Six cotton hybrid pools (ASA, AASA, ACSA, RSA, RASA and SMSA) derived from crosses between Samaru Allen (SA) and different exotic cultivars (Albar 51/474, Reba W296, Reba B50, BJA 592, Acala 1517c and their derivatives) have for a long time constituted the genetic pool for the cotton varietal improvement program at the IAR, Samaru, Zaria. Amongst the various hybrid pools or families, the RASA and RSA families have been found to be most resistant (Mustafa, 1981). SAMCOT-9 and SAMCOT-10, the current commercial cultivars derived from the RASA pool ($B_9LB_{10}L$) have been shown to be highly resistant to the disease and possess high yield attributes. However, of concern is the durability of the observed resistance in the presence of highly virulent isolates. Recent field surveys of cotton farms in the northern and eastern cotton growing zones have shown increased susceptibility and the development of severe symptoms on the commercial cultivars. Although, bacterial blight has been controlled in many countries through the use of resistant varieties, it still remains a potentially serious disease in Africa. The continued threat from the disease is due mainly to the variability of the pathogen. In 1981, a new virulent isolate, designated HV1, was identified in Africa (Follin, 1981 and 1983). In 1984, a cotton cultivar (S295) resistant to all known races and isolates of bacterial blight of cotton was identified in Chad (Girardot *et al*., 1986). Recent field surveys of cotton farms in the northern and eastern cotton growing zones of Nigeria have shown increased susceptibility and the development of severe symptoms on commercial cultivars.

Although the presence of the highly virulent isolates of bacterial blight pathogen identified in Chad and Burkina Faso have not been reported in Nigeria, it is feared that because of the heavy trans-border trade between Nigeria and Chad and other neighboring countries; and the north-easterly wind that blows across these regions, it is necessary to continuously monitor and prepare for the occurrence of the highly virulent isolates. Therefore, the objective of this study was to determine the nature and mode of inheritance of resistance in S295 to the HV1 isolate and a mixture of two predominant and widely distributed races of bacterial blight in Nigeria.

Experimental procedure

Four parent materials, namely: 'S295'; TAMCOT CAMD-E; SAMCOT-6 and SAMCOT-8 were selected for this study based on their differential response to infection by *Xanthomonas campestris* pv. *malvacearum*.

S295 is highly resistant to all known races and isolates of bacterial blight (Girardot *et al*., 1986). TAMCOT CAMD-E is resistant to all USA and Nigerian races but susceptible to HV1. SAMCOT-8 is resistant to races 2 and 6 but moderately susceptible to races 7 and 10. SAMCOT-6 is susceptible to all Nigerian races and contains no gene for resistance to bacterial blight.

The four parents were planted during the rainy season of 1998 at the Institute for Agricultural Research (IAR) Farm, Samaru. Self-pollinated seed was harvested from individual plants and bulked. In 1999, crosses were made among the parents and F_1 seed were produced. During the rainy season of 2000, the resulting F_1s from the series of crosses and their parents were grown in the field. Each F_1 was backcrossed to each of its parent to generate the backcrosses. At the same time, fresh F_1s were made and later advanced to the F_2 generation through controlled self-pollination. In 2001, the resulting populations including four (4) parents, six (6) F_1s, six (6) F_2s and twelve (12) backcrosses (6 BC_1s and 6 BC_2s) were evaluated for bacterial blight resistance. The twenty-eight populations were planted in (12 x 10 cm) plastic pots filled with heat-sterilized soil (two parts of soil to one part of sand). Six seeds were planted in each pot and entries arranged in a completely randomized design with four repetitions. Parental and F_1 entries consisted of two pots per repetition. Four pots represented a single plot for BC_1 and BC_2 entries and eight pots represented a single plot for F_2 entries. Plants were inoculated and evaluated at the cotyledon and true-leaf growth stages.

Races 7 and 10 and the HV1 isolate were cultured at 25 °C on potato carrot dextrose agar media (PCDA). Inoculum was prepared from 5 day-old cultures by suspending a small portion of bacterial colony in sterile water contained in a small glass vial. The bacterial suspension was adjusted to produce an inoculum density of approximately 1.0×10^6 bacteria/ml. Equal portion (1:1) of the inoculum of races 7 and 10 were combined for the race mixture. After inoculation and evaluation at the cotyledon growth stage, seedlings in each pot were randomly thinned to few plants. Two main stem leaves at nodes seven to nine were inoculated and evaluated for the true-leaf growth stage.

Plants were evaluated for disease reaction from 15–21 days after inoculation depending upon disease development. Disease expression was graded using the scale of 1 (immunity) to 10 (fully susceptible) described by Bird and Hadley (1958). 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 the 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_2s and backcross generations were examined to determine the type of distribution exhibited. Numbers occurring in segregating classes were tested using Chi-square for goodness of fit to classical phenotypic ratios.

Results

SAMCOT-6 x S295; SAMCOT-8 x S295

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 1:1 mixture of races 7 and 10 and the HV1 isolate. The cultivar S295 was resistant to both the mixture and the HV1 isolate while SAMCOT-6 was highly susceptible (Table 1). Slightly higher disease grades were recorded on cotyledons compared to true leaves in the presence of both the mixture of races and the HV1 isolate. Bimodal F_2 disease grade frequency distributions were obtained for the two growth stages when inoculated with the race mixture (Figure 1). Disease grades distributions indicated a resistant and susceptible class represented by Grade 1 – 3 and 4 – 10, respectively. Chi-square test for goodness of fit to a 3 resistant: 1 susceptible phenotypic ratio was not significant. A perfect fit to a 3:1 ratio for the cotyledons would have given 147 resistant: 49 susceptible seedlings and 96 resistant: 32 susceptible plants for true leaves. However, the results gave a close fit to a 3:1 segregating ratio, 150: 42 for cotyledon and 96: 32 for true leaves (Table 2). When cotyledons were inoculated with the HV1 isolate, a bimodal F_2 disease grade frequency distribution was observed (Figure 2). The F_2 population segregated 3 resistant: 1 susceptible (Table 3). For the true leaves, in the presence of the HV1 isolate, a bimodal disease grade frequency distribution was also observed in the F_2. However, there were more resistant plants than would have been expected. The expected segregation ratio was 96 resistant: 32 susceptible plants. The observed results were 116 resistant: 30 susceptible plants. A 3: 1 phenotypic ratio was the only acceptable fit. The backcross to the susceptible parent filled closely a 1:1 phenotypic ratio under monogenic control. Similar segregating patterns of the F_2 and BC_2 generations were also obtained in the cross SAMCOT-8 x S295 (Table 1).

SAMCOT-6 x TAMCOT CAMD-E; SAMCOT-8 x TAMCOT CAMD-E

Cotyledons and true leaf disease grades indicated resistance in TAMCOT CAMD-E in the presence of the mixture of races 7 and 10 and susceptibility to the HV1 isolate. TAMCOT CAMD-E, SAMCOT-6 and SAMCOT-8 were susceptible to the HV1 isolate. Bimodal F_2 disease grade frequency distributions were obtained for both cotyledons and true leaves (Figure 3). The 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. The grading distribution of the F_2 population fitted closely a

3 resistant: 1 susceptible segregating ratio. The F_1 and BC_1 populations were resistant. The backcross population to the susceptible parent segregated and fitted closely a 1:1 phenotypic ratio. Similar trends were observed in the cross between SAMCOT x TAMCOT CAMD-E (Table 2). In the presence of the HV1 isolate, parental, F_1, BC_1, BC_2 and F_2 populations were all susceptible (Table 3). Means and frequency distributions indicated little, if any, detectable genetic differences between TAMCOT CAMD-E and SAMCOT-6 for resistance to the HV1 isolate.

TAMCOT CAMD-E x S295

Both parent cultivars were resistant to the 1:1 race mixture of races 7 and 10 in reactions of both cotyledons and true leaves with no significant differences among parents or backcrosses. No segregation for resistance to the race mixture in the cross between TAMCOT CAMD-E x S295 was observed (Table 2). When inoculated with the HV1 isolate, TAMCOT CAMD-E was susceptible and S295 was highly 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 (Figure 4). The segregation in the F_2 population did not fit a known classical phenotypic ratio because of the excess number of plants in the intermediate and susceptible range. When the F_1 was crossed with the susceptible parent, the BC_2 population exhibited a bimodal distribution but did not fit a 1:1 phenotypic ratio. The same trend was observed with the true-leaves.

Discussion

The results of the present study show that the disease grades in the presence of the HV1 isolate were higher than those of the 1:1 mixture of races 7 and 10, irrespective of the growth stages. In addition higher disease grades were recorded with cotyledons compared to true leaves. Disease reactions to the HV1 isolate and the mixture of races indicated the variation in the level of virulence or aggressiveness of the pathogen. According to Innes (1967) and Keay *et al.* (1969), disease reactions vary with the age and kind of host tissue. Young succulent tissues tend to be more susceptible than older tissues (Innes, 1961b).

The F_2 disease grade frequency distributions for the crosses SAMCOT-6 x S295 and SAMCOT-8 x S295 indicated a single gene inheritance of resistance to the 1:1 mixture of races 7 and 10 and HV1. Dominance for resistance was complete. Both cotyledon and true leaf F_2 and BC disease grade distributions indicated monogenic inheritance. These results corroborate those of Follin *et al.* (1981), Wallace and El-Zik (1989) and Alabi *et al.* (2000).

In the presence of the 1:1 race mixture, the F_2 disease grade distribution of the crosses SAMCOT-6 x CAMD-E and SAMCOT-8 x CAMD-E indicated a single gene inheritance of resistance. It is interesting to note that TAMCOT-CAMD-E indicated a monogenic inheritance. TAMCOT-CAMD-E is known to contain the B_2B_3 gene combination with a modifier complex (B_{sm}). Therefore, one would have expected a multigenic inheritance. It is probable that the interaction of B_2B_3 gene combination with the minor gene complex created a large gene effect comparable to that of a major gene. When TAMCOT-CAMD-E was crossed with S295 in the presence of the race mixture, disease grade frequency distributions indicated the presence of a major gene effect with dominance in the direction of resistance in S295. However, in the crosses between SAMCOT-6 x CAMD-E and SAMCOT-8 x CAMD-E in the presence of HV1 the F_1s, F_2s and backcross progenies were all susceptible. These results tend to suggest a single gene difference between S295 and CAMD-E that confers resistance in S295.

The monogenic inheritance of disease resistant observed in this study when the variety S295 was crossed with SAMCOT-6 and SAMCOT-8, tends to suggest from a practical point of view that the backcross breeding method could be used to transfer the major gene resistance to susceptible types, if the cultivar S295 is used as a source of total resistance. However, a major drawback of this method has been that such a system does not exploit genetic variability for characters other than resistance; and is unlikely within a reasonable working period, to restore a critical balance between genotype and environment which has been built up by continued selection in well adapted varieties of rainfed cotton type.

As a result of the uncertainty of the durability of a major gene resistance, Follin *et al.* (1988) suggested that rather than breed for total resistance, it might just be encouraging for breeders to develop cultivars with as much horizontal resistance as possible through recurrent selection method, which encourages the accumulation of minor genes which can lead to the creation of new linkages which behave like major genes. From a series of genetic studies, El-Zik and Bird (1970), Innes (1974) and Bird (1982), indicated that the combinations of two or more genes and a modifier complex was responsible for the observed immunity in commercial cotton varieties grown in the USA for more than two decades. In order to derive long term immunity to bacterial blight of cotton, Brinkerhoff *et al.* (1984) suggested an approach which involves the combination of several gene resistance factors preferably two or more genes with large effect into a polygenic resistance background. The success of this approach predicates largely on the availability and the use of pathogen genotypes that permit consistent and rigorous screening of plant genotypes in segregating generations.

In conclusion, since genes for resistance to bacterial blight in cotton are known to be accumulative in their effect, it is necessary to find combinations of these genes that would give the greatest degree of resistance.

The potentials of the multi-adversity cultivars in the current germplasm collection should be fully evaluated with the view to identifying desirable combinations that could be used in hybridization program with Samaru cultivars in order to develop varieties that are highly resistant to bacterial blight and with good agronomic and acceptable fiber quality attributes.

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Table 1. *Cotyledon and true leaf disease grades[1] of parental cotton cultivars inoculated with a mixture of races 7 and 10 and the HV1 isolate of* Xanthomonas campestris *pv.* malvacearum.

	Mixture of races[2]		HV1[2]	
Parents	Cotyledon	True-leaf	Cotyledon	True-leaf
S295	1.4[a]	1.2[a]	2.8[a]	2.3[a]
Tamcot-CAMD-E	3.8[b]	3.2[b]	7.5[b]	6.2[b]
SAMCOT-6	6.4[d]	6.1[d]	8.6[d]	7.4[d]
SAMCOT-8	4.9[c]	4.5[c]	6.7[c]	6.2[c]

[1] Grades based on a scale of 1 (immunity) to 10 (fully susceptible).

[2] Any two mean grades within a column having a letter in common are not significantly different at P=0.05 according to Duncan's new multiple range test.

Table 2. *Segregation for cotyledon and true-leaf disease reactions to a mixture of Nigerian races 7 and 10 of* Xanthomonas campestris *pv.* malvacearum *among parental cotton cultivars and their F1, F2, BC1, BC2 generations.*

	Cotyledons					True leaves			
	Observed segregation					Observed segregation			
Parents and progeny	Expected segregation	Number R[1]	Number S[2]	X^2 value	P	Number R	Number S	X^2 value	P
S295	R	48	0	-	-	30	0	-	-
SAMCOT-6	S	0	48	-	-	0	30	-	-
SAMCOT-8	S	4	44	-	-	2	28	-	-
CAMD-E	R	46	2	-	-	30	1	-	-
SAMCOT-6 x S295									
F_1	R	48	0	-	-	32	0	-	-
BC_1 (F_1 x S295)	R	96	0	-	-	64	0	-	-
BC_2 (F1 x SAMCOT-6)	1:1	49	47	0.04	0.90 – 0.75	33	31	0.06	0.90 – 0.75
F_2	3:1	150	42	1.00	0.30 – 0.35	94	34	0.17	0.75 – 0.50
SAMCOT-8 X S295									
F_1	R	48	0			32	0		
BC_1 (F_1 x S295)	R	96	0			64	0		
BC_2 (F1 x SAMCOT-8)	1:1	54	42	1.50	0.25 – 0.10	30	34	0.25	0.75 – 0.50
F_2	3:1	147	45	0.25	0.75 – 0.50	92	36	0.67	0.50 – 0.25
CAMD-E x S295									
F_1	R	46	0	-	-	40	0	-	-
BC_1 (F_1 x CAMDE-)	R	94	0	-	-	80	0	-	-
BC_2 (F1 x SAMCOT-6)	R	96	0	-	-	80	0	-	-
F_2	R	190	0	-	-	172	0	-	-
SAMCOT-6 x CAMD-E									
F_1	R	42	0	-	-	39	0	-	-
BC_1 (F_1 x CAMDE-)	R	88	0	-	-	77	0	-	-
BC_2 (F1 x SAMCOT-6)	1:1	40	38	0.05	0.90 – 0.75	35	29	0.56	0.50 – 0.25
F_2	3:1	152	40	0.45	0.50 – 0.25	124	38	0.25	0.75 – 0.50
SAMCOT-8 x CAMD-E									
F_1	R	46	0	-	-	32	0	-	-
BC_1 (F_1 x CAMDE-)	R	84	0	-	-	76	0	-	-
BC_2 (F1 x SAMCOT-8)	1:1	44	36	0.60	0.50 – 0.25	36	29	0.56	0.50 – 0.25
F_2	3:1	137	52	0.35	0.50 – 0.25	128	34	0.38	0.75 – 0.50

[1] R = resistant

[2] S = susceptible

Table 3. *Segregation for cotyledon and true-leaf disease reactions to the HV1 isolate of* Xanthomonas campestris *pv.* malvacearum *among parental cotton cultivars and their F1, F2, F2, BC1 and BC2 generations.*

	Cotyledons					True leaves			
	Observed segregation					Observed segregation			
Parents and progeny	Expected segregation	Number R[1]	Number S[2]	X^2 value	P	Number R[1]	Number S[2]	X^2 value	P
S295	R	40	0	-	-	30	1	-	-
SAMCOT-6	S	0	40	-	-	0	30	-	-
SAMCOT-8	S	0	40	-	-	0	30	-	-
CAMD-E	R	0	38	-	-	0	29	-	-
SAMCOT-6 x S295									
F_1	R	48	0	-	-	30	1	-	-
BC_1 (F_1 x S295)	R	94	0	-	-	68	2	-	-
BC_2 (F1 x SAMCOT-6)	1:1	57	39	3.38	0.10 – 0.05	48	33	0.39	0.75 – 0.50
F_2	3:1	143	49	0.03	0.90 – 0.95	116	30	0.17	0.75 – 0.50
SAMCOT-8 X S295									
F_1	R	45	0			30	1	-	-
BC_1 (F_1 x S295)	R	96	0			70	2	-	-
BC_2 (F1 x SAMCOT-8)	1:1	55	41	2.40	0.25 – 0.10	37	27	1.6	0.25 – 0.10
F_2	3:1	147	45	0.25	0.75 – 0.50	101	27	1.04	0.50 – 0.25
SAMCOT-6 x CAMD-E									
F_1	S	1	44	-	-	0	30	-	-
BC_1 (F_1 x CAMDE-)	S	2	95	-	-	0	64	-	-
BC_2 (F1 x SAMCOT-6)	S	1	92	-	-	1	58	-	-
F_2	S	2	189	-	-	1	128	-	-
SAMCOT-8 x CAMD-E									
F_1	S	1	46	-	-	0	36	-	-
BC_1 (F_1 x CAMDE-)	S	1	92	-	-	0	60	-	-
BC_2 (F1 x SAMCOT-8)	S	0	94	-	-	1	67	-	-
F_2	S	2	190	-	-	1	144	-	-

[1] R = resistant

[2] S = susceptible