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Inheritance of Response to Cotton Leaf Curl Virus (CLCuV) Infection in Cotton

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Abstract: To study inheritance of CLCuV, nine selected varieties/lines (four resistant five susceptible) of up land cotton (*G. hirsutum* L.) were used for five F_1 combinations. The F_1 was sown for raising F_2 and back crossing purpose. Subsequently P_1, P_2 F_1, F_2, BC_1 and BC_2 population were established and to ensure the inoculation of CLCuV no pesticide was used to control insect population. The results indicated that duplicate dominant epistasis was involved in control of resistance of CLCuV. Virus resistance was controlled by two dominant duplicate genes as F_2 ratio was modified to 15:I from 9:4:4:1 and the test cross ratio was modified to 3:1 instead of 1:1.

Key words: Cotton, leaf curl virus, inheritance, genetic resistance

Introduction

During the year 1987-88, a virus like disease appeared in cotton which gradually increased and became epidemic from the year 1992-93 and caused heavy damage to the crop during the year 1994-95 when the cotton production declined from 12.8 to 7.9 million bales (Anonymous, 1997). This disease was then identified and named as cotton leaf curl virus (CLCuV). The yield loss has become a constant phenomenon every year due to CLCuV and about 7.1 million bales have been lost during last decade (Mahmood, 1999).

Similar type of damage to cotton (*G. hirsutum* L.) has been done by the CLCuV in Nigeria, Sudan and Tanzania. The disease caused a reduction in number of bolls by 87.4 and 38.8% in boll weight and 92.2% in seed cotton yield (Singh *et al*., 1999). The crop management strategies proved ineffective against CLCuV attack and the only option was to build genetic resistance in varieties to provide the solution of this disease (Khan and Khan, 1995). Much attention has been paid towards breeding for resistant varieties in Pakistan and as a result eleven CLCuV resistant varieties have been approved until now for general cultivation which provided genetic barrier against CLCuV along with other desirable traits. The country is still under constant threat as many of the farmers are still growing old varieties. However, CLCuV resistant varieties has become an essential integral component of the national cotton breeding program. Therefore, inheritance studies on CLCuV were also carried out in the present research.

Leaf curl disease of cotton (*G. hirsutum* L.) is characterized by vein thickening, enation formation and curling of leaves. Generally, the virus induces two types of leaf curling i.e., upward (Up) or downward (Dw). Both types of symptoms are observed in almost all commercially cultivated cotton varieties in naturally infected as well as artificially inoculated plants. In addition, mixed type (Dw and Up) can also be seen in same plants (Khalid and Shah, 1999; Khalid and Masood, 1999).

Wilson and Brown (1991) studied the inheritance of cotton leaf crumple virus (CLCV) infection in cotton. The results indicated that factor controlling symptoms/expression (Susceptibility) were inherited as duplicate factors and that the susceptible phenotype (genotype c_1c_1 c_2c_2) was recessive to the phenotype i.e., resistant phenotype (genotype c_1-, c_2-), which mean that at least one dominant gene must be present in a genotype for resistance to CLCV.

Ali (1997) studied that the inheritance of CLCuV in upland cotton and reported that resistance was monogenic and dominant in nature. Mansoor *et al.* (1999) reported that cotton genotypes, susceptible to CLCuV accumulated several fold higher level of viral DNA, as compared to the tolerant varieties.

Sanz *et al.* (1999) studied the genetic variability of natural population of cotton leaf curl "Gemini virus" and suggested that cotton leaf curl virus (CLCuV) of Pakistan belongs to "Gemini virus" group of genus Begomo virus. They further reported that whitefly (*Bemisia tabaci*) transmitted "Gemini virus" in cotton.

Materials and Methods

The experiment was conducted at Central Cotton

Research Ansated, Multan on clay loam soil. Four CLCuV resistant varieties viz. S-III, MNH554, CIM448 and CIM1100 and five CLCuV susceptible varieties viz. 124F, MNH516, S-14, MNH465 and NIAB Karishma were crossed as: S-111 X 124F, MNH564 x MNH516, CIM448 X S-14, CIM1100 X MNH465, NIAB KARISHMA X MNH465.

All these crosses were attempted during the year 1996. The F_1 generation along with their parents was raised during the 1997. Plants of the F_1 generation were self pollinated and subsequently back crossed to the two parents. Parents were also crossed during the subsequent years (1997, 1998) in above-mentioned fashion.

The parents P_1, P_2, F_1, F_2 and back cross (BC_1, BC_2) populations of each cross were planted on 15th June, 1998. One row of P_1, P_2 and F_1, six rows of F_2 and three rows of each back cross were planted keeping row to row distance 75 cm and plant to plant 25 cm, while the length of row was 6 m. The F_1 seed of year 1998 was again sown in next sowing season 1999 as F_2 generation, keeping 8 rows of each cross. Techniques regarding artificial infestation were not available and observations were taken in the environment of natural infestation (Ali, 1997). As the whitefly is vector for transmission of CLCuV (Sanz *et al*., 1999). Keeping in view this point, no pesticide was applied before 15th September, providing opportunity to whitefly and other sucking insects of cotton to breed and multiply frequently for transmission of CLCuV inoculum. All plants of all generations were observed periodically and rated during the growing season for CLCuV symptoms, following the method of Wilson and Brown (1991). Plants were rated as "A" for asymptomatic (resistant) and "S" for virus affected symptoms (CLCuV susceptible).

Symptom ratings obtained from the final data collection (Sept., 1998 to 1999) were used for further analysis. The data of six generations P_1, P_2, F_1, F_2, BC_1 and BC_2 of each cross for the year 1998 and F_2 generation during 1999 were used for the goodness of fit by the chi-square test as discussed by Gomez and Gomez (1983).

Results and Discussion

It is revealed from data presented in Table 1 that all the parent plants of 124F, MNH516, S-14, MNH465, NIAB-Karishma and F_1 of NIAB- Karishma X MNH465 showed CLCuV symptoms. While plants of varieties S-111, MNH564, CIM448, CIM1100 and F_1 plants of following crosses (1) S111 X 124F, (2) MNH564 x MNH516, (3) CIM448 X S-14, (4) CIM1100 X MNH465 were asymptomatic (A phenotype). The F_2 population segregated in 1:15, S:A (CLCuV diseased plants : healthy plants) of all crosses except the cross NIAB-Karishma X MNH465, as the whole F_2 population of this cross was disease affected (S phenotype). It was due to the reason that both parents used in crossing were CLCuV susceptible and as a result the F_1, BC_1, BC_2 and F_2 generations of this cross were also CLCuV susceptible.

The data regarding the back cross populations (BC_1 and BC_2) raised from the first four F_1 hybrids from serial number one to four presented in Table 1 revealed that the BC_1 population (F_1 x resistant parents) did not show any segregation as all plants were resistant. The back cross (BC_2) populations of respective susceptible parents segregated into 1:3 ratio, S:A (CLCuV diseased plants : Healthy plants). The 1:15 ratio in F_2 population of all the four crosses except NIAB- Karishma x MNH465 between S:A were also observed during the following year 1999-2000, which confirmed the previous year results. Chi Square test completely advocated the goodness of fit for F_2 and back cross ratios (Table 2).

These results indicated that inheritance of CLCuV susceptibility was conditioned by duplicate factors, with the S conferring alleles recessive and hypostatic to A (healthy plant from CLCuV). The data F_1, F_2, BC_1 and BC_2 populations of cross NIAB-Karishma x MNH465 showed those resistant plants (phenotype) could not be achieved in F_1 by crossing the susceptible parents with each other. It was suspected that at least one copy of each of the two factors (designated VR_1, VR_2) must be present for the A (resistant to CLCuV) response and plant bearing genotype VR_1-, VR_2-, VR_1- Vr_2Vr_2, Vr_1Vr_1 and VR_2- will also be resistant to CLCuV and only genotypes having $Vr_1Vr_1Vr_2Vr_2$ will be susceptible.

The present studies revealed that virus resistant (A) in S111, MNH564, CIM448, CIM1100 was dominant over susceptibility in 124F, MNH516, 3-14, MNH465 and that the character was controlled by two dominant genes. Segregation ratio of F_2 9:3:3:1 based on two genes was modified to 1:15 as a result of duplicate dominant epistasis in this experiment. The pattern of segregation of F_2 is as under:

$VR_1VR_1VR_2VR_2$ x $Vr_1Vr_1Vr_2Vr_2$

F_1	VR_1 Vr_1 VR_2 Vr_2	Resistant
F_2	9=VR_1 – VR_2-	Resistant
	3= VR_1- Vr_2 Vr_2	Resistant
	3= Vr_1 Vr_1 VR_2-	Resistant
	1= Vr_1 Vr_1 Vr_2 Vr_2	Susceptible

F2 ratio changes to 1:15 (Susceptible : resistant)

Table 1: Inheritance of CLCuV during 1998

		Observed		Expected		
Cross	Total plants	S	A	S	A	χ^2
124F	26	26	0	-	-	
S-111	27	0	27	-	-	
S-111 X 124F (F_1)	59	0	59	0	59	
F_1 X S-111(BC_1)	84	0	84	0	84	
F_1 X124F (BC_2)	88	21	67	22	66	0.06
S-111 X124F(F_2)	185	13	172	11.6	173.4	0.18
MNH564	27	0	27	-	-	
MNH516	26	26	0	-	-	
MNH564 X MNH516 (F_1)	56	0	56	0	56	
F_1 X MNH564 (BC_1)	83	0	83	0	83	
F_1 X MNH516 (BC_2)	85	24	61	21.25	63.75	0.47
MNH564 X MNH516 (F_2)	172	10	162	10.75	161.25	0.06
CIM448	26	0	26	-	-	
S-14	21	21	0	-	-	
CIM448 X S-14 (F_1)	55	0	55	0	55	
F_1 X CIM448 (BC_1)	79	0	79	0	79	
F_1 X S-14 (BC_2)	81	23	58	20.25	60.75	0.50
CIM448 X S-14 (F_2)	175	8	167	10.9	164.1	0.82
MNH465	29	29	0	-	-	
CIM1100	25	0	25	-	-	
CIM1100X MNH465(F_1)	56	0	56	0	56	
F_1 X CIM1100 (BC_1)	74	0	74	0	74	
F_1 X MNH516 (BC_2)82	82	19	63	20.5	61.5	0.15
CIM1100 X MNH465(F_2)	171	8	163	10.7	160.3	0.73
NIAB Krishma	26	26	0			
MNH465	23	23	0			
N. Krishma X MNH465(F_1)	52	52	0			
F_1X NIAB Krishma(BC_1)	81	80	1			
F_1X MNH465 (BC_2)	73	73	0			
N. Krishma X MNH465(F_2)	172	171	1			

Table 2: Inheritance of CLCuV in F_2 during 1999

		Observed		Expected		
Cross	Total plants	S	A	S	A	χ^2
S111 X124F	229	16	213	14.3	214.7	0.22
MNH564 X MNH516	207	14	193	12.9	194.1	0.10
CIM448 X S14	212	11	201	13.25	198.75	0.41
CIM1100 X MNH465	209	15	194	13.1	195.9	0.29
NIAB Krishma X MNH465	215	214	1	-	-	-

$P \leq 0.05$, S = CLCuV effected(Susceptible), A = Asymptomatic(Resistant)

Test cross ratio (F_1 x Susceptible parent)

$VR_1Vr_1VR_2Vr_2$(resistant F_1) x $Vr_1\ Vr_1Vr_2Vr_2$ Susceptible parent

1= $VR_1\ Vr_1VR_2Vr_2$	Resistant
1= $Vr_1\ Vr_1VR_2Vr_2$	Resistant
1= $VR_1Vr_1\ Vr_2Vr_2$	Resistant
1= $Vr_1Vr_1Vr_2Vr_2$	Susceptible

As a result the test cross ratio was modified to 3:1 from 1:1.

These results are in according to Wilson and Brown (1991) and are contrasting with Siddig (1968) and Ali (1997). As the *G. hirsutum* L. is an allotetraploid species, an example of duplicate factor inheritance in cotton has been documented by Wilson (1987). It is therefore, concluded that for future improvement it might be necessary to identify lines that breed true for the resistant A. Following evidences support the presence of two dominant genes hypothesis.

The test cross ratio 1:3 confirms digenic model as in monogenic, it must be 1:1 ratio. All the F_1, test cross ratio, back cross ratio and F_2 ratio confirmed the homozygosity of the parents.

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