

Screening of Germplasms against Cotton Leaf Curl Disease under Natural Epiphytotic Environment

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Cotton is the most important *kharif* cash crop of north India. Several factors responsible for low production and productivity of cotton during last decade among them cotton leaf curl disease has been found one of the major limiting factor. High susceptibility of the presently grown cultivars to *Cotton leaf curl disease* (CLCuD) in north western states of India is one of the key factors for low yield of cotton in these states. To identify the source of resistant against CLCuD, present study was carried out at two locations i.e. Cotton Research Station, Sirsa, and Cotton Section, CCS HAU, Hisar during *kharif* 2014. Sixty-four genotypes of *Gossypium hirsutum* were evaluated against CLCuD at both the location under natural epiphytotic condition. It has been found that seven genotypes namely AUBURN, BLIGHT MASTER, B59-1678, PIL 8, PIL 8-5, PIL 104 and PKV 0804 showed resistant reaction against CLCuD while nine genotypes i.e. 101-102-B2, 1695-175 J, 7203-14-104, DELCOT 377, H 1098i, PIL 8-7, PIL-9,PUSA 31, RS 810 observed moderately resistant reaction against CLCuD. These genotypes may be utilized as sources of resistance in different breeding programme for evolving CLCuD tolerant/resistance varieties of cotton.

Keywords: *Gossypium hirsutum*, CLCuD, Resistant source, Genotypes.

Cotton is one of the most important fibre crops in India. It plays a very significant role in Indian economy. Cotton leaf curl disease (CLCuD) earlier known as African leaf curl of cotton is very crucial factor responsible for decreased productivity of cotton. First time CLCuD was reported from Nigeria on *Gossypium peruvianum* and *G. vitifolia* in 1912 by Faquharson, who reported that CLCuD is a viral disease and caused by *Gossypium virus-1*. In 1924, the disease was reported in Sudan and Tanzania^{6,7,8} and thereafter it spread to all the African Countries situated north of equator except Egypt, Maghreb, Benin, Chad,

Togo and Barkina Faso¹⁴. It severely started affecting cotton (*G. hirsutum*) in Pakistan since 1967⁴ bringing down the cotton production. In the year 1989, it was observed in the kitchen gardens in Bangalore, Karnataka¹⁰. Later it observed on *G. hirsutum* Sriganganagar, Rajasthan in 1993¹, and 1994 in Haryana¹³. High susceptibility of presently grown cultivars to CLCuD responsible for severe appearance of CLCuD. The only way to overcome this problem will be to 'stack' multiple resistances, based upon distinct mechanisms of action⁵. Resistant source may obtained by evaluating germplasms against CLCuD. Commonly used methods for screening of resistant germplasm include, the exploitation of virus spreader line (S-12) and white fly as a source of transmission vector¹¹. Another method that was used for

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screening is the sowing time difference i.e. normal and late sowing along with disease nursery¹².

MATERIALS AND METHODS

Sixty four genotypes of *G. hirsutum* were sown during *khari*, 2014 under unprotected natural epiphytotic field conditions at two locations i.e. CCS HAU Cotton Research Station Sirsa and Cotton Section CCS HAU Hisar. Each genotype was sown in two replications with spacing of 67.5x30 cm. in single row of 6.0 meter length.

Susceptible check variety HS-6 was sown after every fourth row and also as border around the experiment to ensure enough inoculum. All conventional agronomic practices were followed to keep the crop in good condition. However, no pesticides were sprayed to allow maximum whitefly population i.e. vector of *Cotton leaf curl virus* (CLCuV).

Observations recorded

PDI of CLCuD was recorded in the month of August and September according to scale described below

Symptoms	Disease Severity (grade)	Per cent Disease intensity	Disease reaction
Complete absence of symptoms	0	0	Immune /disease free
Thickening of few small scattered veins on one or few leaves of a plant observed after careful observation	1	0.1-10	Highly Resistant
Thickening of small group of veins, no leaf curling, no reduction in leaf size and boll setting	2	10.1-20	Resistant
Thickening of all veins, minor leaf curling, leaf enations, deformity of internodes with minor reduction in leaf size but no reduction in boll setting.	3	20.1-30	Moderately Resistant
Severe vein thickening, moderate leaf curling, leafy enations, minor deformity of internodes and minor reduction in leaf size and boll setting.	4	30.1-40	Moderately Susceptible
Severe vein thickening, moderate leaf curling, leaf enations and deformity of internodes with moderate reduction in leaf size and boll setting followed by moderate stunting.	5	40.1-50	Susceptible
Severe vein thickening, leaf curling, reduction in leaf size, leafy enations, deformed internodes and severe stunting of plant with no or few boll setting	6	> 50	Highly Susceptible

Calculation of PDI

Per cent disease Intensity (PDI) was calculated for each entry by using the following formula given below:

$$PDI = \frac{\text{Sum of all the numerical ratings of plants observed}}{\text{Total no. of plants observed} \times \text{Maximum grade}} \times 100$$

RESULTS AND DISCUSSION

Per cent disease intensity (PDI) of each genotype at every location and their mean is described in Table 1. The data of Table 2 revealed that among sixty four genotypes, none was found immune or disease free and highly resistant against CLCuD. Seven genotypes namely AUBURN, BLIGHT MASTER, B59-1678, PIL 8, PIL 8-5, PIL

104, PKV 0804 showed resistant reaction against CLCuD; nine genotypes i.e. 101-102-B2, 1695-175 J, 7203-14-104, DELCOT 377, H 1098i, PIL 8-7, PIL-9, PUSA 31, RS 810 observed moderately resistant reaction; twenty two genotypes namely 101-102B, B 56-181, BADNAWARI, C 100A, COKER 413-68, DELFOS, DELTAPINE SL, DUNN, G 67, GTSV 337, H 14, H 1117, H 1226, H 1300, H 655 C, IAN 1327 F, LUXMI, PAYMASTER, REBA B 50, RS 875, SHARDA, TAMCOT CAMPE expressed moderately susceptible reaction while, eighteen genotypes i.e. B 57-876, BC 68-2, F 1378, F 1794, G COT 100, GS 10, H 1236, HS 182, L 147, NECTARILESS, PKV 081, R 40 (Frego upland), RS 2097, RS 2098, STONEVILLE 62, S 344, TCH 1599, TX ORSZ 78 showed susceptible

Table 1. Evaluation of different genotypes against CLCuD under natural epiphytotic condition

S. No	Name of Genetic Collection	PDI		Mean
		CRS Sirsa	Cotton Section Hisar	
1.	101-102B	14.44	56.67	35.56
2.	101-102-B2	26.66	33.33	30.00
3.	1695-175 J	20.20	33.33	26.77
4.	105 F	52.60	56.67	54.64
5.	320 F	51.50	50.00	50.75
6.	7203-14-104	11.11	43.33	27.22
7.	AUBURN	12.24	16.67	14.46
8.	B 56-181	28.88	50.00	39.44
9.	B 57-876	33.33	56.67	45.00
10.	B59-1678	10.66	10.00	10.33
11.	BADNAWARI	11.11	50.00	30.56
12.	BLIGHT MASTER	15.50	20.66	18.08
13.	BC 68-2	28.88	56.67	42.78
14.	C 100A	20.60	56.67	38.64
15.	COKER 413-68	35.50	28.30	31.90
16.	DELCOT 377	21.88	32.66	27.27
17.	DELCOT 277	46.46	56.67	51.57
18.	DELTAPINE SL	33.30	43.33	38.32
19.	DELFOFOS	21.10	50.00	35.55
20.	DUNN	28.88	38.33	33.61
21.	F 1378	46.66	52.40	49.53
22.	F 846	54.40	56.67	55.54
23.	F 1794	32.40	50.00	41.20
24.	G COT 8 F	53.40	56.67	55.04
25.	G COT 100	40.00	50.66	45.33
26.	G 67	20.30	55.00	37.65
27.	GS 10	30.10	54.60	42.35
28.	GTSV 337	33.33	40.00	36.67
29.	H 655 C	20.00	50.00	35.00
30.	H 1098i	28.22	30.00	29.11
31.	H 1117	11.11	56.67	33.89
32.	H 1226	20.30	50.00	35.15
33.	H 1236	33.33	50.00	41.67
34.	H 1300	32.00	46.54	39.27
35.	H 14	18.33	56.67	37.50
36.	HS 182	33.33	56.67	45.00
37.	IAN 1327 F	29.10	33.33	31.22
38.	L 147	33.33	53.46	43.40
39.	LOCKET 4785 CREAM	56.40	56.67	56.54
40.	LUXMI	24.44	50.00	37.22
41.	NECTARILESS	28.88	56.67	42.78
42.	PAYMASTER	11.11	66.67	38.89
43.	PIL 104	11.11	16.67	13.89
44.	PIL 8	10.00	16.67	13.34
45.	PIL 8-5	12.40	20.44	16.42
46.	PIL 8-7	15.50	24.56	20.03
47.	PIL-9	21.11	32.46	26.79
48.	PKV 0804	22.20	10.00	16.10
49.	PKV 081	26.66	56.67	41.67

50.	PUSA 31	20.20	28.33	24.27
51.	PUSA 317	51.50	56.67	54.09
52.	R 40 (Frego upland)	41.11	46.66	43.89
53.	REBA B 50	52.80	16.67	34.74
54.	RS 2097	30.60	50.00	40.30
55.	RS 2098	40.00	50.00	45.00
56.	RS 810	21.11	36.44	28.78
57.	RS 875	21.10	53.33	37.22
58.	RST 9	44.44	50.00	47.22
59.	S 344	28.80	56.67	42.74
60.	SHARDA	16.66	56.67	36.67
61.	STONEVILLE 62	31.80	50.00	40.90
62.	TAMCOT CAMPE	18.88	56.67	37.78
63.	TCH 1599	25.50	56.67	41.09
64.	TX ORSZ 78	33.33	50.00	41.67

Table 2. Disease reaction of different genotypes against CLCuD

Reactions	PDI	No. ofgermplasmin each category	Genotypes
Disease Free/Immune	0	0	-
Highly resistant	0.1-10	0	-
Resistant	10.1-20	7	AUBURN, BLIGHT MASTER, B59-1678, PIL 8, PIL 8-5, PIL 104, PKV 0804.
Moderately Resistant	20.1-30	9	101-102-B2, 1695-175 J, 7203-14-104, DELCOT 377, H 1098i, PIL 8-7, PIL-9, PUSA 31, RS 810.
Moderately susceptible	30.1-40	22	101-102B, B 56-181,BADNAWARI , C 100A, COKER 413-68 , DELFOS, DELTAPINE SL, DUNN, G 67,GTSV 337, H 14,H 1117, H 1226, H 1300, H 655 C , IAN 1327 F, LUXMI, PAYMASTER ,REBA B 50, RS 875, SHARDA, TAMCOT CAMPE.
Susceptible	40.1-50	18	B 57-876,BC 68-2, F 1378,F 1794, G COT 100,GS 10,H 1236, HS 182, L 147,NECTARILESS, PKV 081, R 40 (Frego upland),RS 2097, ,RS 2098, STONEVILLE 62, S 344,TCH 1599, TX ORSZ 78.
Highly susceptible	>50.0	8	105 F, 320 F, DELCOT 277, F 846, G COT 8 F, LOCKET 4785 CREAM, RST 9, PUSA 317.

reaction and eight genotypes viz. 105 F, 320 F, DELCOT 277, F 846, G COT 8 F, LOCKET 4785 CREAM, RST 9, PUSA 317 were observed highly susceptible reaction against CLCuD.

Similarly, Monga *et al.*, (2008) screened a total of 1799 cotton germplasm lines during 1997-2006 under natural conditions. Field resistant lines were confirmed through grafting and whitefly inoculation. Twelve germplasm lines were found field resistant over the years and were subjected to graft inoculation and artificial transmission with

whitefly. Only seven lines, namely, BP-52-16, MB-LYHH, JBWR-21, CNH-2773, AKH-9620, B 59-1679-2, Super okra virescent and 59-CCD were recorded resistant to cotton leaf curl virus disease. CRSM-38 is a new variety exhibited an average of 77.9% higher degree of tolerance against cotton leaf curl virus (CLCuV) over respective checks, and attained an average of 46.52 % higher seed cotton yield than that of CIM-496 under various climatic conditions revealing its wider adaptability².

CONCLUSION

At present, no single variety of *G. hirsutum* is resistant to CLCuD. Resistant source may become a very effective tool to overcome the impact of CLCuD. Screening of germplasms will provide a resistance source toward cotton leaf curl since it contains valuable natural resource of plant diversity. Seven genotypes of *G. hirsutum* namely AUBURN, BLIGHT MASTER, B59-1678, PIL 8, PIL 8-5, PIL 104, PKV 0804 showed resistant reaction against CLCuD and nine genotypes i.e. 101-102-B2, 1695-175 J, 7203-14-104, DELCOT 377, H 1098i, PIL 8-7, PIL-9, PUSA 31, RS 810 observed moderately resistant reaction against CLCuD. These genotypes may leads to development of resistant varieties that effectively avoid the CLCuD infection.

REFERENCES

1. Ajmera, B.D., Occurrence of leaf curl virus on American Cotton (*G. hirsutum*) in north Rajasthan. Paper presentation, National Seminar on Cotton Production Challenges in 21st Century, 1994; 18-20 Hisar. India.
2. Ahmad, S., Hussain, A., Hanif, M., Mahmood, K., Nazeer, N. W., Mahmood A., Noor-ul-Islam, Malik, W., Qayyum, A. and Hanif K., CRSM-38, a new high yielding coupled with CLCuV tolerance cotton (*Gossypium hirsutum* L.) variety. *African J. of Biotech.*, 2012; **11**(19): 4368-4677
3. Farquharson, C.O., A report of the mycologist. A report Agric. Deptt. Nigeria. In Siddique MA and Hungus LC (Eds) Cotton growth in Gezira environment. W Haffer and Sons Ltd. Cambridge England. 1912; 106.
4. Hussain, T. And Ali, M., A review of cotton diseases in Pakistan. *Pak Cottons*, 1975; **19**: 71-86.
5. Ilyas, M., Amin, I., Mansoor, S., Briddon, R. W. and Saeed, M., Challenges for transgenic resistance against geminiviruses. In *Emerging Geminiviral Diseases and their Management*, 2011; 1-35.
6. Jones, G. H. and Mason, T.G., Studies on two obscure diseases of cotton. *Ann. Bot.*, 1926; **1**(16): 759-772.
7. Kirkpatrick, T.W., Leaf curl in cotton. *Nature*, 1930; **125**: 672.
8. Kirkpatrick, T.W., Further studies of leaf-curl of cotton in the Sudan. *Bull Ent Res.*, 1931; **12**: 323-363.
9. Monga, D., Kumar, M., Chander, S., Singh, N.P., Meena, R.A., Identification of cotton leaf curl virus disease (CLCuD) resistant lines *J. Cotton. Res. Dev.*, 2008; **22** (2): 234-237.
10. Nateshan, H.M. and Muniyappa, V., Leaf curl disease of cotton, a whitefly transmitted geminivirus from Southern India. *Indian Phytopathology.*, 1992; **45**: 164.
11. Perveen, R., and Sultan, K.M., Screening of cotton germplasm against cotton leaf curl begomovirus (CLCuV). *Pak. J. Phyto.*, 2005; **17**(1): 56-60.
12. Perveen, R., Fan, I., Islam, N.U., Haider, S., Chohan, S. and Rehman, A.U., Correlation of biweekly environmental conditions on CLCuV disease growth in Pakistan. *Eur J Sci.* 2010; **4**: 224-227.
13. Rishi, N. and Chauhan, M.S., Appearance of leaf curl disease of cotton in Northern India. *J. Cotton Res. Develop.*, 1994; **8**: 179-180.
14. Tarr, Saj. Leaf curl disease of cotton. *Common W Mycol Internat*, Kew, Surrey. 1951; 20-28.