Inheritance of cotton leaf curl virus disease (CLCuD) in upland cotton

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Abstract: Inheritance of cotton leaf curl virus disease (CLCuD) was studied in four crosses parents involving resistant and susceptible parents to this disease. Six generations *i.e.* P_1 , P_2 , F_1 , F_2 , BC_1 and BC_2 were generated. Resistant reaction is dominant over susceptibility as all the plants in F_1 generation were resistant in both R x S crosses and R x R cross. All the plants in F_1 generation of S x S cross were susceptible. In F_2 generation of both R x S crosses showed the duplicate dominant (15 resistant: 1 susceptible) effect for inheritance of cotton leaf curl virus disease in upland cotton. These results were further confirmed by observed ratio in backcross generations.

Key words: Dominant, generation, inheritance, resistant, susceptible, virus disease

Cotton as a crop as well as commodity plays an important role in the agrarian and industrial activity of the nation and has a unique place in the economy of our country. It is contributing about 65 per cent of the raw material for the textile industry. Our economy is consistently influenced by cotton through its production, processing and by generating direct and indirect employment to more than eight million people. Cotton is grown in tropical and sub tropical regions of more than 80 countries world over viz., China, the former Soviet Union (primarily Uzbekistan), Brazil, Australia and the United States, Bulgaria, Russia, China and Korea, India and Pakistan. The leading cotton producing countries are China, USA, India and Pakistan (Meyer et al., 2013).

India produces around 37.50 million bales of cotton ranging from short staple to extra long staple from an area of 11.53 m ha with productivity of 552 kg / ha, while the world productivity is 622 kg / ha. In Haryana, cotton area under cotton is about 5.57 lakh hectare with a production of 23 lakh bales and a productivity of 702 kg/ha lint. More than 90 per cent area of cotton is under *G. hirsutum*. Low productivity of cotton is mainly due to high incidence of insect pests and diseases caused by fungal, bacterial and viral pathogens. Of these viral diseases alone or in combination with other factors are quite destructive and are limiting factor for the cotton production resulting significant loss in seed cotton yield.

The cotton leaf curl virus disease is caused by Gemini virus are of considerable concern. The climate of India is favorable for the growth and spread of its vector *i.e.* whitefly (Bemisia tabaci Genn). It transmits Gemini virus which pose serious threat to cotton production. In India, cotton leaf curl virus disease was first reported in upland cotton in Sriganganagar area of Rajasthan state during 1993 (Ajmera, 1994) and during 1994 it appeared in Haryana and Punjab (Rishi and Chauhan, 1994; Singh et al., 1994) states and posed a major threat to its cultivation in northern India (Varma et al., 1995). The disease has appeared in an epidemic form during 1997 in the Rajasthan affecting an area of 0.1 million ha (Anonymous, 1998).

Cotton leaf curl virus disease is initially characterized by small vein thickening (SVT) type symptoms on young upper leaves of plants. The disease is further characterized by upward or downward curling of leaves and affected leaves become thick, leathery, brittle and greener than healthy leaves. Later formation of cup shaped or leaf laminar outgrowth called "enation" appear on the underside of the leaf.

Use of chemicals in controlling this disease is costly and not so effective. Moreover, it may be hazardous to men and environment. Extensive use of pesticides has also cause damage to soil quality and fertility. Therefore, development of a resistant variety to this disease is the most effective, long term, less expensive and safe method to fight against this disease and to enhance and stabilize the productivity of cotton. Research efforts to develop resistant varieties/hybrids through conventional/ biotechnological approaches along with cultural and management practices are in progress for effective control of this disease.

The successful exploitation of the source of resistance also requires information on the genetic control for development of resistant variety. The information on these aspects may helpful to meet out the objectives. Keeping these in mind the present study was conducted.

MATERIALS AND METHODS

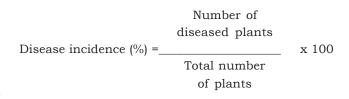
The present investigation was conducted at Cotton Research Area, Department of Genetics and Plant Breeding, CCS Haryana Agricultural University, Hisar during *kharif*, 2013. Four parents which includes two resistant (H 1098-i and H 1117) and two susceptible (B 59-1678 and HS 6) to cotton leaf curl virus disease were chosen to generate the experimental material for the present study. These four parents were used to develop four crosses, H 1098-i x B 59-1678 (R x S), H 1117 x HS 6 (R x S), H 1098-i x H1117(R x R) and B 59-1678 x HS 6 (S x S). These crosses were designated as cross I, cross II, cross III and cross IV, respectively.

During *kharif*, 2011, the parents were identified to fulfill the objectives and F_1 crosses between these parents, namely H1098-I, H 1117, B 59-1678 and HS 6 were made. The F_1 hybrids and parents were raised during *kharif*, 2012. Each

 F_1 was selfed to obtain F_2 generation and simultaneously backcrossed to both of its parents to produce backcross generations (BC₁and BC₂). Fresh crosses were also made to obtain the F_1 seed and all the parents were selfed to get their seeds for the next year. Thus, the experimental material finally comprised of six generations, namely P_1 , P_2 , F_1 , F_2 , BC₁ (backcross to first parent P_1) and BC₂ (backcross to second parent P_2).

The experimental material comprised of six generations *i.e.* parents (P_1 and P_2), F_1 , F_2 and back crosses (B_1 and B_2) of four crosses was grown in a compact family block design with three replications during kharif, 2013 at Cotton Research Area of CCS Haryana Agricultural University, Hisar. There was a single row of non segregating generations (P_1 , P_2 and F_1), 20 rows of F₂ and 8 rows of each back cross 1 and back cross 2 generations. The length of each row was 6 m with a spacing 67.5 x 30 cm. In order to build up heavy inoculum pressure one row of highly susceptible line (HS 6) to cotton leaf curl virus disease was planted at the periphery of the experimental area. Normal cultural practices were followed except no insecticidal spray for control of white fly population in the field which is vector of cotton leaf curl virus disease. Reaction of cotton leaf curl virus disease was recorded on all the plants in all replications.

Observation on cotton leaf curl virus disease (CLCuD) : Observation on cotton leaf curl virus disease was recorded under field condition in each replication on all the plants of each of the non segregating generations (P_1 , P_2 and F_1), backcross generations and the F_2 generation. Disease was scored on 0 -5 grade depending upon the response to the cotton leaf curl virus disease.



- 0= Immune; complete absence of symptoms, per cent disease incidence =0 per cent
- 1= Highly resistant; very minute thickening of veins, per cent disease incidence = 0 - 10 per cent
- 2= Resistant; thickening of small group of veins, per cent disease incidence = 10 - 20 per cent
- 3= Susceptible; severe vein thickening and leaf curling developed at the top of the plant, per cent disease incidence = 20 - 40 per cent
- 4= Moderately susceptible; severe vein thickening and leaf curling developed on the half of the plant canopy, per cent disease incidence = 40 - 70 per cent
- 5= Highly susceptible; severe vein thickening, leaf curling, enation and full stunting of plant, per cent disease incidence = 70 - 100 per cent

Plants were selected on the basis of above grading scale. There were very few plants that belonged to grade 0 (immune). Most of the resistant plant were of grade 1 and 2. Their average per cent disease incidence ranged from 0-20 per cent.

Statistical analysis

The Chi – square test of goodness of fit : For testing the agreement of observed frequencies with those expected upon a given hypothesis, the chi- square (a^{"2}) test of goodness of fit. For carrying out this test the following formulae was used:

$$a^{n^2} = i = \Sigma$$
 (Oi – Ei)2 / Ei
1

whereas;

 O_i = observed frequency of ith class

 E_i = expected frequency of ith class

For testing the significance, the a"² tabulated value was seen at n-1 d.f.

RESULTS AND DISCUSSION

The incidence of cotton leaf curl virus disease during the experimental year *i.e.* 2013-2014 was very severe under field condition particularly nearby Hisar areas including CCS HAU, cotton research area. During this year no variety/ strain was observed completely immune to this disease. Even in highly resistant strains only few plants showed immunity. The F₁s viz., H 1098-I x B 59-1678 and Cross H 1117 x HS 6 had resistance to CLCuD indicated that resistance is a dominant trait. The expression of resistance in both (R x S) crosses revealed that there was no cytoplasmic inheritance for the expression of susceptibility to CLCuD. The dominance nature of resistance over susceptibility was further confirmed by backcrosses and F₂s. there were two distinct classes, *i.e.*, resistant and susceptible in F₂ and backcross population. The pattern of segregation in F2 gave a good fit to 15 resistant: 1 susceptible (Table 1) indicating the duplicate type of gene action. Disease was expressed in those plants which had recessive genes at both the loci. Duplicate type of gene interaction for CLCuD was further confirmed by a good fit of 3 resistant: 1 susceptible ratio of backcross with susceptible parents.

The breeding for cotton leaf curl disease resistance has been achieved through the assemblage of minor genes by recurrent selection and according to Azhar *et al.*, (2010) resistance depends on major genes (dominant genes) which may lose quickly because of the evolution of pathogen for these genes. The F_1 of crosses between highly susceptible S 12, highly resistant LRA 5166 varieties were found all virus free plants and their F_2 was close to 3:1 ratios which exhibit the presence of a single gene for the inheritance of resistance against CLCuD reported by Rehman *et al.*, (2005). Ahuja *et al.*, 2006 reported 4 types

| Parent/ generation | Plants | | | Expected | a"2 | Р |
|-------------------------------------|------------------|-----------|-------------|----------|------------|-------|
| | Screened | Resistant | Susceptible | ratio | calculated | value |
| Cross I (R x S): | H 1098-I x B 5 | 9-1678 | | | | |
| P ₁ | 54 | 54 | 0 | | | |
| P ₂ | 56 | 0 | 56 | | | |
| F_1 | 61 | 61 | 0 | | | |
| F_2 | 364 | 348 | 16 | 15:1 | 2.27 | 0.131 |
| BC ₁ | 218 | 218 | 0 | | | |
| BC_2 | 243 | 173 | 70 | 3:1 | 1.77 | 0.183 |
| Cross II(R x S):Cross H 1117 x HS 6 | | | | | | |
| P ₁ | 61 | 61 | 0 | | | |
| P ₂ | 57 | 0 | 57 | | | |
| F_1 | 59 | 59 | 0 | | | |
| F_2 | 358 | 331 | 27 | 15:1 | 0.74 | 0.389 |
| BC_1 | 221 | 221 | 0 | | | |
| BC_2 | 231 | 168 | 63 | 3:1 | 0.575 | 0.448 |
| Cross III (R x R |): H 1098-I x H1 | 117 | | | | |
| P ₁ | 54 | 54 | 0 | | | |
| P_2 | 59 | 59 | 0 | | | |
| F_1 | 58 | 58 | 0 | | | |
| F_2 | 347 | 347 | 0 | | | |
| BC_1 | 236 | 236 | 0 | | | |
| BC_2 | 221 | 221 | 0 | | | |
| Cross IV(S x S): | 59-1678 x HS 6 | 5 | | | | |
| P ₁ | 59 | 0 | 59 | | | |
| P ₂ | 59 | 0 | 59 | | | |
| F_1 | 56 | 0 | 56 | | | |
| F_2 | 358 | 0 | 358 | | | |
| BC ₁ | 230 | 0 | 230 | | | |
| BC_2 | 235 | 0 | 235 | | | |

Table 1. Inheritance of CLCuD in upland cotton

of segregation patterns in the F_2 generations. A good fit for 15 (resistant):1 (susceptible), 13 (resistant):3 (susceptible), 9 (resistant):7 (susceptible) ratios indicated digenic control of the trait with duplicate dominant, dominant inhibitory, and duplicate recessive epistasis, respectively. Three gene controls with triplicate dominant epistasis was obtained in one of the crosses.

In F2 generation of cross H 1098-I x H1117 (R x R), all the plant were resistant resistant to CLCuD indicated that genes involved in the resistance of CLCuD were present at the same locus in both the parents. Hence no segregation pattern was observed in F2 and backcrosses. In cross $59-1678 \times HS 6$ (S x S), all the plants in F1, F2 and backcross generations

were susceptible to CLCuD. The disease reaction in all generations was similar to the reaction of parents suggesting that there was no complimentary interaction between the genes for susceptibility in both the susceptible parents.

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