INT. J. BIOL. BIOTECH., 18 (3): 517-524, 2021.

GENETIC ANALYSIS FOR INHERITANCE OF RESISTANCE TO COTTON LEAF CURL VIRUS (CLCV) DISEASE

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ABSTRACT

The present research was conducted to estimate the gene action for inheritance of resistant to CLCV disease. Adequacy of additive and dominance model was conducted, both scaling test designated that hypothesis was fit for genetic analysis. Significant of both additive (D) and dominance parameters (H_1 and H_2) endorsed the presence of additive and dominance genes for inheritance of resistant to CLCV. Additive (D) effect was more prominent than dominance (H_1 and H_2) suggested additive genes in legacy of resistant in both generations. Uneven values between H_1 and H_2 is sign of unequal dispersal of recessive and dominance genes. Values of $H_2/4H_1$, was less than (0.25), recommended allelic frequency of positive and negative effects gene was asymmetrical. F parameter, $\sqrt{4DH_1 + F}/\sqrt{4DH_1 - F}$ and $\frac{1}{4}\sqrt{4DH_1 + \frac{1}{2}F}$ / $\frac{1}{4}\sqrt{4DH_1 - \frac{1}{2}F}$ values were also proved equal proportion with relative frequency of dominant and recessive alleles in parents, h^2 value directed the existence of dominance effects due to heterozygous loci. Genetic ratio h^2/H_2 signalized that at least one genetic group is involved in controlling to resistance of CLCV disease in F_1 and F_2 generation. High narrow sense heritability indicated, selection in early segregating generations, because of traits of genotypes heritage to its offspring. Resistant offspring in F_1 and due to segregation F_2 population ratio was observed as 3:1 (resistant plants: diseased plants) among Mac-7 population, which legitimated the single dominant gene proved monogenic dominance nature of CLCV disease resistant in cotton. The Wr-Vr graph also shown Mac-7 carried maximum dominant genes and USD16-3058 contained maximum recessive genes. While, rest of the parents had different constitution of dominant and recessive genes. The high values of PCV and GCV proved that resistant of CLCV did not affected due to environment. High board sense heritability coupled with high genetic advance, suggested role of additive genes. Therefore, single plant selection could be fruitful in early segregating generation.

Keywords: Gene action, genetic analysis, cotton leaf curl virus, additive, dominance

INTRODUCTION

Pakistan is 4th largest cotton producing country in the world, whereas 3rd in consumption globally during last five years from 2014-15 to 2018-19. Counties economic development depends upon the production of cotton, because the nation mainly dependent on industry of cotton and related with textile sector. That's why the principle status has been given to the cotton crop. In Pakistan over 2.895 million hectares area is covered for cotton cultivation, however 12.72 million bales were achieved as production. As regards the provincial status of this crop, Punjab sown on 2.145 million hectares and achieved with the production of 7.90 million bales. However, Sindh Province produced 4.60 million bales with the cultivated area was 0.640 million hectares (Cotton Review, 2020).

Cotton leaf curl virus (CLCV) is most severe and destructive disease of cotton. The CLCV appeared in epidemic form during 1991 and subsequent years which causing drastic reduction in cotton production 12.82 million bales and 8.04 million bales in 1994. The CLCV disease has caused huge losses 7.1 million bales amounting of 1.2 billion dollars to the national economy of Pakistan during last decade (Mahmood, 1999). It given extent of losses to cotton production and is future disaster for cotton crop in Pakistan, after the presence of new burewala CLCuD stains which caused a collapse of resistance in most of the cultivable varieties of cotton (Mahmood *et al*., 2003 and Mansoor *et al*., 2003). CLCV again emerged in Pakistan during couple of years and every year thousands of acres destroyed due to this disease. Khan *et al*. (2007) reported that additive gene effect specified more too CLCV resistant in deviation with dominance gene effect. The polygenic heritage perception of CLCV disease was altered into single dominant nature of gene (with minor modifier genes) in cotton (Aslam and Gilani, 2000 and Ahuja *et al.*, 2006). The F_1 hybrids which were crossed between highly resistant and susceptible cotton cultivars that were found to be resistant/virus free in F_1 generation and during segregating generation in F_2, the ratio was nearby to 3:1, it indicated the proportion of CLCV resistant and CLCV diseased plants. It suggested the appearance of single dominant nature of gene action for the legacy of resistant for CLCV (Mahmood, 20004 and Rehman *et al.* 2005). However, uncertainty triumphs whether the CLCV control managed by dominant or recessive genes and it might be due to monogenic or polygenic.

MATERIALS AND METHODS

The present research was conducted at Central Cotton Research Institute Sakrand during the year 2017-2019. Ten parents (CRIS-129, FH-142, MNH-886, NIA-Noori, Baghdadi, CIM-602, NIAB-824, CEMB-33, USD16-3058 and Mac-7) were selected. The Griffings (1956) half diallel matting design were applied, 10 parents were hybridize according to suggested methods and 45 F_1 hybrids/F_2 population were produced. The F_1 and F_2 seeds were planted in the field conditions with randomized complete block design (RCBD) with three replications. The plant to plant space was kept 30cm and row to row 75 cm distance were retained. The nutrients/inputs and plant protection measures were applied as per need whenever required. The cotton leaf curl virus (CLCV) disease data was noted and analyzed with given disease rating scale by Akhtar *et al.* (2010) and Farooq *et al.* (2011). The research was conducted to assess the additive and dominance gene effect for legacy of resistance to CLCV according to Jinks and Hayman (1953). The gene action was elucidated by following the genetic components formulated by Hayman's (1954). The genetic components of variance were studied which suggested by Morley-Jones (1965) with modification of Hayman's approach. The data was analyzed to calculate the genetic components according to the methods of Hayman (1954) and Jinks (1954).

D = components of variance due to additive effect of genes
F = relative frequency of dominant and recessive alleles in the parents
H_1 = components of variance due to dominance effect of genes
H_2 = Dominance component indicating asymmetry of position and negative effects of genes
h^2 = Dominance effects over all loci
E = environmental variation

$F_1 = \sqrt{(H_1/D)}$ and $F_2 = \sqrt{\frac{1}{4}(H_1/D)}$ = Average degree of dominance

$H_2/4H_1$ = Proportion of dominant genes with positive and negative effect

$F_2 = \sqrt{4DH_1 + F}/\sqrt{4DH_1 - F}$ and $F_2 = \frac{1}{4}\sqrt{4DH_1 + \frac{1}{2}F} \ / \ \frac{1}{4}\sqrt{4DH_1 - \frac{1}{2}F}$ = Proportion between dominant and recessive alleles

h^2/H^2 = Number of gene groups/genes which control the trait and exhibit dominance

However, the per se performance/mean values and variance were statistically analyzed by following the method of Singh and Choudhry (1977). Whereas, further genetic components were adopted as genotypic and phenotypic and coefficient of variation (Burton and Devane 1953). Heritability (Hanson *et al.,* 1956) and genetic advance (Johanson *et al.,* 1955).

RESULTS AND DISCUSSION

The analysis of variance mean square values of F_1 and F_2 were significant for cotton leaf curl virus (CLCV) disease which indicated presence of genetic diversity in material (Table 1). The statistical results of F_1 and F_2 generation were also found significant for "a" and "b" components suggested that inheritance was controlled due to additive and dominance genetic effect for expression of resistance to cotton leaf curl virus. Greater value of "a" additive variance than the "b" dominance advocated role of additive gene action for the legacy of resistance to cotton leaf curl virus in F_1 and F_2 generation. The b_1 component was non-significant in F_1 generation and significance in F_2 generation indicated the presence of directional dominance effect. The significant of b_2 item in both the generations (F_1 and F_2) revealed asymmetrical distribution of genes among the parents. However, significance of b_3 items expressed presence of specific gene action other than attributable to b_1 and b_2 for resistance to cotton leaf curl virus in F_1 and F_2 generation. Mather and Jinks (1982) reported that b_3 significance indicate the dominance effect for particular cross.

Adequacy of Additive-Dominance Model

Before interpreting data for genetic analysis, it is necessary to test the validly for assumption (Hayman, 1954). The supposition of Haman's additive and dominance models are; (1) diploid segregation; (2) parents should be homozygous, due to continuous inbreeding, it assumed that parents will be homozygous; (3) no reciprocal differences. While, rest of suppositions of additive and dominance genetic model (Mather and Jinks, 1982) are (4) no multiple allelism; (5) absence of non-allelic interaction (epistasis); (6) independent gene distribution between the parents. The additive and dominance model's accuracy were tested with study of scaling test by conducting t^2 test

and regression analysis. Conferring with Mather and Jinks (1982) statement, the data of genetic interpretation will only be validated, if regression coefficient value "b" must be deviated from zero (0) but not from unity (1). Nevertheless, the "b" value was exceeded from 0.5 in F_1 and F_2 generation for cotton leaf curl virus indicated the absence of epistasis. The second test was the t^2 test. The results of additive-dominance model are presented in Table 2, indicated that t^2 test was non-significant in both generations, while "b" was also deviated from zero but not from unity. Both scaling test designated that the hypothesis of the genetic analysis is fit for the trait cotton leaf curl virus in both the generations F_1 and F_2.

Table 1. Analysis of mean of squares for cotton leaf curl virus in F_1 generation and F_2 population.

Source	**D.F**	**Cotton Leaf Curl Virus (CLCuV)**	
		F_1	**F_2**
Replication	2	73.72	125.86
Genotypes	54	1050.35**	1544.77**
a	9	5195.70**	8311.85**
b	45	221.36**	191.35**
b_1	1	77.91^{ns}	1117.93*
b_2	9	509.20**	463.43**
b_3	35	151.44*	94.91**
Error	108	83.91	37.81

Table 2. Additive-Dominance model for cotton leaf curl virus in F_1 and F_2 generation.

Generation	**t^2 test**	**Regression Analysis (t value of b)**		**Remarks**
		b/SE	**b0, b1**	
F_1	0.79^{NS}	0.79±0.12	b0 =6.46** b1 = 1.67**	Data is fit for genetic analysis
F_2	0.19^{NS}	0.92±0.09	b0 =10.70** b1 = 0.82**	Data is fit for genetic analysis

Table 3. Components of variance and genetic parameters for cotton leaf curl virus in $F_1$and F_2generation.

Components/Parameters	**Cotton Leaf Curl Virus (CLCuV)**	
	F_1Generation	**F_2 Generation**
D	369.47**±31.91	467.53**±21.55
H_1	167.77**±67.93	233.24**±45.88
H_2	71.96±57.73	115.19**±38.99
h^2	93.22**±18.14	133.96**±26.10
E	83.91**±9.62	37.81**±6.50
F	0.0	0.0
$F_1 = \sqrt{(H_1/D)}$ $F_2 = \sqrt{\frac{1}{4}(H_1/D)}$	0.67	0.35
$H_2/4H_1$	0.11	0.12
$F_1 = \sqrt{4DH_1 + F}/\sqrt{4DH_1 - F}$ $F_2 = \frac{1}{4}\sqrt{4DH_1 + \frac{1}{2}F}\ /\ \frac{1}{4}\sqrt{4DH_1 - \frac{1}{2}F}$	1.0	1.0
h^2/H_2	1.29	1.16
Heritability % (h^{ns})	70	81

Table 4. Cotton Leaf Curl Virus (CLCV) disease incidence % of cross combinations in F_1 and F_2 generation.

Cross Combinations	**F_1 Generation**		**F_2 Generation**	
	Disease %	**Disease reaction on rating scale**	**Disease %**	**Disease reaction on rating scale**
CRIS-129 x MNH-886	40.7	Tolerant	47.9	Tolerant
CRIS-129 x FH-142	31.7	Tolerant	43.8	Tolerant
CRIS-129 x NIA-Noori	37.7	Tolerant	58.6	Susceptible
CRIS-129 x Baghdadi	23.9	Tolerant	41.4	Tolerant
CRIS-129 x CIM-602	61.8	Tolerant	54.6	Tolerant
CRIS-129 x NIAB-824	38.8	Tolerant	52.4	Tolerant
CRIS-129 x CEMB-33	28.5	Tolerant	52.7	Tolerant
CRIS-129 x MAC-7	0	Resistant	4.1	Highly Tolerant
CRIS-129 x USD16-3058	49.6	Tolerant	75.4	Susceptible
MNH-886 x FH-142	38.2	Tolerant	37.7	Tolerant
MNH-886 x NIA- Noori	25.0	Tolerant	50.1	Tolerant
MNH-886 x Baghdadi	20.3	Highly Tolerant	46.0	Tolerant
MNH-886 x CIM-602	34.1	Tolerant	63.8	Susceptible
MNH-886 x NIAB-824	31.5	Tolerant	65.6	Susceptible
MNH-886 x CEMB-33	23.2	Tolerant	50.9	Tolerant
MNH-886 x MAC-7	0	Resistant	3.8	Highly Tolerant
MNH-886 x USD16-3058	43.7	Tolerant	66.3	Susceptible
FH-142 x NIA- Noori	31.1	Tolerant	64.6	Susceptible
FH-142 x Baghdadi	24.3	Highly Tolerant	44.1	Tolerant
FH-142 x CIM-602	49.4	Tolerant	51.8	Tolerant
FH-142 x NIAB-824	23.3	Tolerant	47.8	Tolerant
FH-142 x CEMB-33	16.9	Highly Tolerant	43.5	Tolerant
FH-142 x MAC-7	0	Resistant	2.4	Highly Tolerant
FH-142 x USD16-3058	40.6	Tolerant	66.5	Susceptible
NIA- Noori x Baghdadi	33.9	Tolerant	48.2	Tolerant
NIA- Noori x CIM-602	50.3	Tolerant	63.5	Susceptible
NIA- Noori x NIAB-824	33.2	Tolerant	48.1	Tolerant
NIA- Noori x CEMB-33	36.4	Tolerant	64.5	Susceptible
NIA- Noori x MAC-7	0	Resistant	3.9	Highly Tolerant
NIA- Noori x USD16-3058	61.6	Tolerant	69.3	Susceptible
Baghdadi x CIM-602	29.6	Tolerant	59.1	Tolerant
Baghdadi x NIAB-824	19.7	Highly Tolerant	41.1	Tolerant

Baghdadi x CEMB-33	20.4	Tolerant	48.3	Tolerant
Baghdadi x MAC-7	0	Resistant	2.9	Highly Tolerant
Baghdadi x USD16-3058	47.0	Tolerant	62.4	Susceptible
CIM-602 x NIAB-824	40.3	Tolerant	55.1	Tolerant
CIM-602 x CEMB-33	44.7	Tolerant	63.4	Susceptible
CIM-602 x MAC-7	0	Resistant	5.4	Highly Tolerant
CIM-602 x USD16-3058	56.1	Tolerant	73.8	Susceptible
NIAB-824 x CEMB-33	45.0	Tolerant	48.7	Tolerant
NIAB-824 x MAC-7	0	Resistant	3.3	Highly Tolerant
NIAB-824 x USD16-3058	52.6	Tolerant	66.0	Susceptible
CEBM-33 x MAC-7	0	Resistant	3.2	Highly Tolerant
CEBM-33 x USD16-3058	45.9	Tolerant	72.8	Susceptible
MAC-7 x USD16-3058	0	Highly Tolerant	9.4	Highly Tolerant

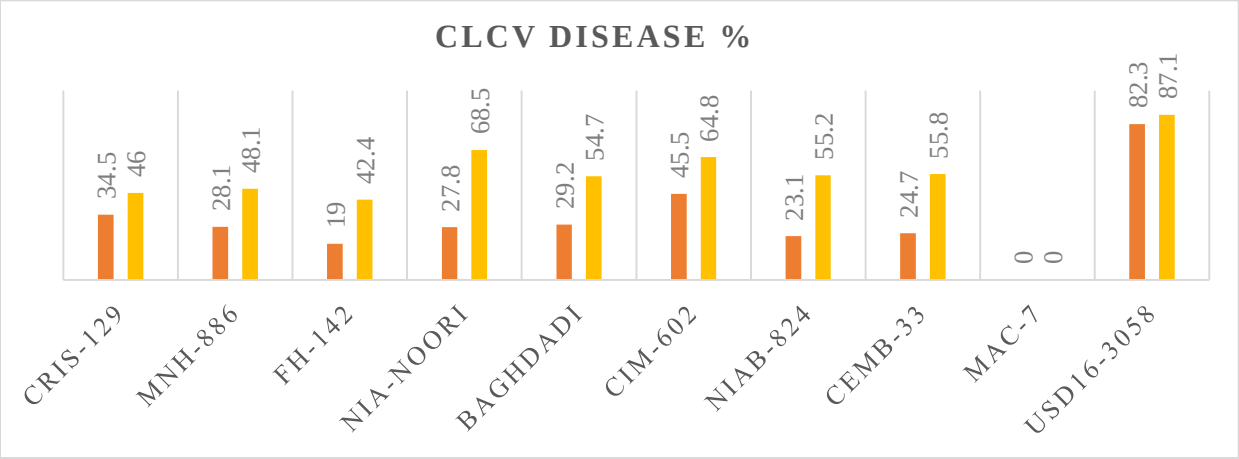

Fig. 1. CLCV Disease % of the varieties during 2 years screening at field conditions.

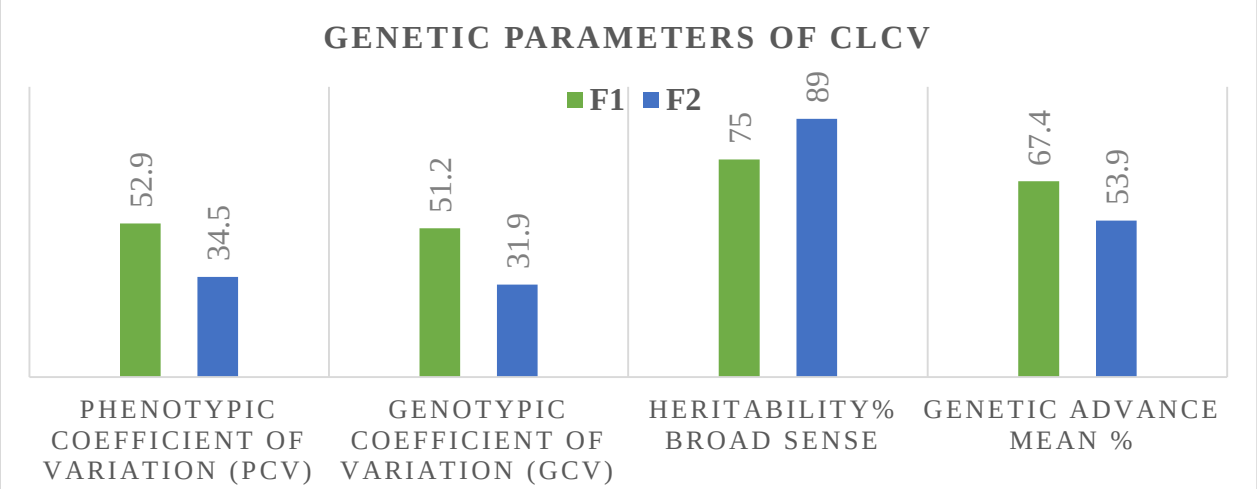

Fig. 2. Genetic parameter of CLCV.

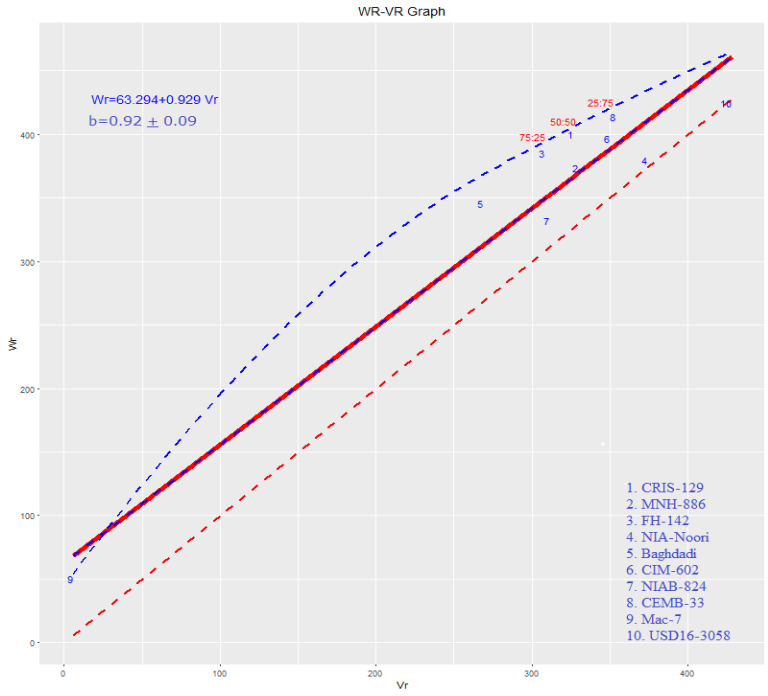

Fig. 3. Vr Wr graph of CLCV in F_1 generation.

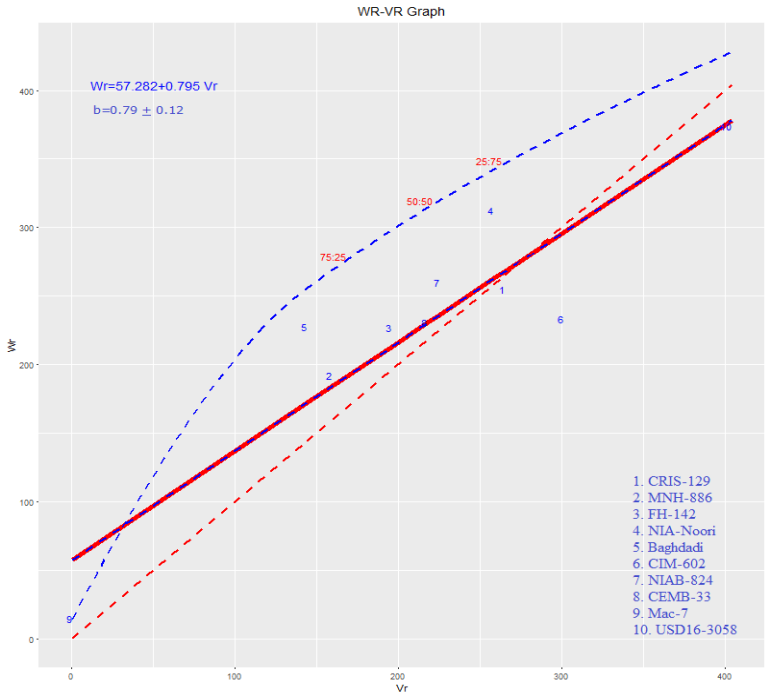

Fig. 4. Vr Wr graph of CLCV in F_2 generation.

Estimation Parameters of genetic components of variance

The genetic component of variance in F_1 and F_2 generation for cotton leaf curl virus depicted in Table 3. The additive (D) and dominance (H_1) were significant, but non-significant of dominance (H_2) was found in F_1

generation. While in F_2 generation, the additive (D) and dominance parameters (H_1 and H_2) were also significant for cotton leaf curl virus disease (Table 3), which endorse the presence of additive and dominance genes for inheritance of resistant to cotton leaf curl virus. The additive (D) effect was higher than the dominance (H_1 and H_2) for CLCV, hence it is suggested that additive genes effect are involved for inheritance of resistant to cotton leaf curl virus in in both F_1 and F_2 generations. The ratio of average degree of dominance in F_1 generation $\sqrt{(H_1/D)}$ and F_2 generation $\sqrt{\frac{1}{4}(H_1/D)}$ is less than unity, also validated nature of genes was partial dominance. The uneven values between H_1 and H_2 in both generations is sign of unequal dispersal of recessive and dominance alleles in the parents. It was also confirmed through values of $H_2/4H_1$, which was less than the maximum value (0.25), recommended that the allelic frequency of positive and negative effects gene was asymmetrical. The F parameter was non-significant and obtained zero value that is the indication of equivalent proportion of dominance and recessive alleles in the parents with relative frequency. The values of Proportion between dominant and recessive alleles in F_1 generation $\sqrt{4DH_1 + F}/\sqrt{4DH_1 - F}$ and F_2 generation $\frac{1}{4}\sqrt{4DH_1 + \frac{1}{2}F}$ / $\frac{1}{4}\sqrt{4DH_1 - \frac{1}{2}F}$ was obtained one (1.0), that specified recessive and dominance genes in the parent are in equal proportion. Significant of h^2 value directed the existence of dominance effects owing to heterozygous loci. The genetic component h^2/H_2 processed to know the number of group of genes that control trait and exhibit dominance. The study suggested that the value of genetic ratio h^2/H_2 signalized that at least one genetic group is involved in the controlling to resistance to cotton leaf curl virus (CLCV) disease in both F_1 and F_2 generations. The narrow sense heritability (h^{ns}) table 3 shown that in both the generations high narrow sense heritability (h^{ns}) was recorded for CLCV. It is direction that good potential for selection of single plants. The high heritability is the indication that the selection is done in early segregating generation because of traits of genotypes heritage to its offspring. The field screening of varieties shown only Mac-7 as resistant parent during 2 years, while remaining parents were affected with CLCV disease (Fig 1). The findings reflected that the Mac-7 resistant parent shown entirely resistant offspring in F_1 generation for CLCV disease, which legitimated the single dominant gene was responsible for inheritance of resistant to CLCV. While, in F_2 generation due to segregation the F_2 population ratio was observed as 3:1 (resistant plants: diseased plants) among Mac-7 population. Hence, it is proved monogenic dominance nature of CLCV disease resistant in cotton (Table 4). Siddig (1968), Aslam *et al.* (2000), Mahmood (2004), Rehman *et al.* (2005) and Khan (2013) reported single dominant gene or closely linked genes for CLCV resistance in cotton. Aslam and Gilani (200) and Hussain *et al.* (2012) suggested that generally dominant genes are responsible for resistant to CLCV as alleles responsible for susceptible to CLCV.

Estimation of Genetic Variability Parameters

The genetic parameters of F_1 and F_2generation are presented in Fig 2, which exhibited that phenotypic coefficient of variation (PCV) and genotypic coefficient of variation (GCV) values were obtained very high for cotton leaf curl virus. The distance among values of genotypic and phenotypic coefficient of variation did not change statistically, it proposed that resistance of cotton leaf curl virus (CLCV) disease is rarely affected with the environment. Hence, selection would be effective on the basis of phenotypic appearance. High heritability broad sense (h^{bs}) and very high genetic advance over mean percent was observed for cotton leaf curl virus. The valuation of heritability (h^{bs}) does not alone useful for predict subsequent effect for selection, because, it contain both additive and non-additive genes. So, heritability (h^{bs}) coupled with genetic advance would be more fruitful for selection. Therefore, high heritability (h^{bs}) along with high genetic advance over mean percent was perceived, it is suggested that inheritance of resistance to cotton leaf curl virus is usually controlled with additive genes. So that single plant selection would be fruitful or improvement through mass and progeny selection as well.

The Wr-Vr graphical analysis of cotton leaf curl virus in F_1 generation are depicted in Fig. 3. The regression coefficient significantly less than unity (1.0) indicated that the genetic system could be assumed to be an additive without the impediment of non-allelic interaction. The regression line intersected the Wr axis beyond the origin indicated genes are partial dominance as confirmed with degree of dominance $\sqrt{(H_1/D)}$. The graphical analysis further shown that genotype Mac-7 carried more dominant genes, as it was next towards the origin line. While, genotype USD16-3058 contained more recessive genes, as being utmost far from the origin. However, Rest of the parents had different constitution of dominant and recessive genes. However, in F_2 generation similar trend was observed Fig.4, the regression coefficient was significant and less than one, as the genetic system is expected to be additive, without the obstruction of non-allelic interaction. Alike regression line crossed the Wr axis beyond the

origin representing partial dominance as confirmed with degree of dominance $\sqrt{\frac{1}{4}(H_1/D)}$. In F_2 generation, parent Mac-7 was found as closet to origin line and possess maximum dominance genes. While, parent USD16-3058 was the farthest from origin that carried maximum recessive genes. Rest of the genotypes had variable genes different constitution for CLCV.

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(*Accepted for publication May 2021*)