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Genetic control of leaf curl virus disease, horticultural and biochemical traits in chilli (*Capsicum annuum* L.)

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Abstract

Two genotypes MS-341 (susceptible) and S-343 (resistant) were used to develop six generations (P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2). These were screened for leaf curl virus disease (LCVD) and phenotyped at fruiting stage. The data was recorded for percent disease index (PDI) of LCVD, horticultural and biochemical traits of chilli. The genetic analysis revealed that most of the studied traits revealed higher magnitude of dominance effects which were also confirmed by the average degree of dominance which was more than unity. The traits like PDI, plant height, plant spread, yield per plant, fruit number, fruit length, fruit diameter, coloring matter in powder and oleoresin content showed duplicate type of epistasis. There was a preponderance of dominance × dominance (*l*) interactions for most of the traits. Therefore, an importance of both additive and non-additive gene effects was realized with the preponderance of non-additive gene effects, hence population improvement approaches such as recurrent selection and bi-parental mating would be suggested.

Keywords: Dominance, genetics, heritability, horticultural traits, leaf curl disease

Introduction

Chilli pepper or hot pepper (*Capsicum annuum* L., 2n=2x=24) is an important spice and vegetable crop of family Solanaceae. It is indigenous to South America and was first introduced in India from Brazil by Portuguese towards the end of 15th century. India is considered to be the secondary center of diversity for chilli, especially of *C. annuum*, the most important cultivated species (IBPGR, 1983) [17]. Capsicum species are usually self-compatible (Onus and Pickers gill 2004) [30] and *C. annuum* is a partially self-pollinating crop (Rylski, 1986) [32]. India is the largest producer, consumer and exporter of chillies in the world. According to an estimate for 2017, in India, green chillies were cultivated on 3.16 lakh hectare with a total production of 36.34 lakh metric tonnes and dry chillies were cultivated on 8.40 lakh hectare with a production of 29.6 lakh metric tonnes (NHB, 2016-17) [29].

Chilli is susceptible to a number of diseases caused by various pathogens, including chilli leaf curl virus disease (ChiLCVD). ChiLCVD causes huge production losses and in severe cases, 100 percent losses of marketable fruit have been reported (Senanayake *et al.,* 2012) [33]. It is transmitted by whitefly *B. tabaci* in a persistent manner. The typical symptoms consisting of leaf curling, rolling and puckering, blistering of interveinal areas, thickening and swelling of the veins, shortening of internodes and petioles, crowding of leaves and stunting of whole plant.

In India breeding for resistance in chilli was started in the late sixties (Mishra *et al.,* 1963, Dhanraj and Seth, 1968) [27-13] but most screening has been done under field conditions, assessing disease incidence and disease severity. Some multiple virus-resistant varieties and hybrids have been developed at Punjab Agricultural University, Ludhiana (reviewed by Thakur *et al.,* 2018) [38] Punjab Sindhuri and Punjab Tej (Dhaliwal *et al.,* 2013) [7], CH-27 (Dhaliwal *et al.,* 2015) [8], Saurian 2010, Perennial and Japani Loungi (Ahmad *et al.,* 2016) [1], S-343, SL 475 and SL 456 (Jindal, 2014, Thakur *et al.,* 2017 and Thakur *et al.,* 2019) [18, 37, 39]. Despite efforts by various research groups, it was not possible to establish genetic control of the resistance gene(s).

Success of any crop improvement program is mainly dependent upon the information regarding nature and magnitude of disease resistance and gene effects controlling economic

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quantitative traits. Since yield is a complex character depending upon number of other characters and their interactions, knowledge about the associations of these characters with yield will greatly help a breeder in his selection work with more precision and accuracy (Deb and Khaleque, 2009) [6]. There are very few reports about genetics of ChiLCVD associated with horticultural and biochemical traits in chilli. Considering this, an investigation was undertaken to evaluate the mode of gene action such as additive and dominant gene effects, non-allelic gene interaction (epistasis), genetic parameters and components of variation in a cross including susceptible (MS-341) and resistant parent (S-343) using generation mean analysis in chilli.

Material and Methods
Development of populations
Two genotypes MS-341 (susceptible) and S-343 (resistant) were used to develop six generations (P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2). The susceptible line (MS-341) was crossed as a female parent with highly resistant line S-343 to generate F_1. The F_1 plants were grown in March 2017, selfed to produce F_2 and backcrossed simultaneously to both the parents to produce backcross generations. Seedlings of MS-341, S-343, F_1, F_2 and both backcrosses (BC_1P_1 and BC_1P_2) were grown in protrays and screened artificially inside insect proof cage as described above, during September-October 2017.

Artificial screening
All the six generations (P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2) were screened in 2017 using artificial screening method by challenging with viruliferous whiteflies and phenotyped at nursery stage (Sharma *et al.* 2018) [34]. The Non-viruliferous

whitefly culture was maintained on cotton (*Gossipium hirsutum* L.) grown in 12×8 cm size plastic pots in the insect-proof glass house. The inoculum of ChiLCVD was maintained on susceptible cultivar MS-341 (10 pots) in a separate insect proof cage. The non-viruliferous whiteflies maintained on cotton were left frequently on these susceptible plants throughout the screening procedure. The test material to be screened was sown in plug trays filled with Cocopeat, vermiculite and perlite mixture in 3:1:1 proportion and maintained in the same cage. Virus infected plants (inoculum) were shaken 2-3 times a day for a uniform spread of the virus by whiteflies. All the six generations were transplanted in the field and phenotyped again at fruiting stage to avoid any escape using standard screening procedure developed at PAU, Ludhiana.

Recording of data
The experiment was transplanted in randomized complete block design in three replications in the month of February, 2018. The inoculated seedlings (10 plants of each parent and F_1, 20 plants of backcross generations, 70 plants of F_2 generation) were transplanted per replication in the field. Disease reactions for each plant were scored after 90 days of transplantation on a symptom severity grade of 0 to 4. The adult plants with severity grade 0, 1, 2 were considered as resistant and plants with rating 3 and 4 as susceptible, as per disease rating scale of Banerjee and Kalloo (1987) [3]. Individual plants of all the generations were categorized into resistant and susceptible groups. For percent disease index (PDI) individual plants of all the six generations were rated (0-4) on the basis of disease severity scale given by Banergee and Kalloo (1987) [3].

Table 1: Different phenotypic classes for ChiL CVD severity grade (Banerjee and Kalloo, 1987) [3]

Symptoms	Symptom severity grade	Response value	Coefficient of infection	Percent Disease Index	Disease reaction
Symptoms absent	0	0	0-4	0	HR
Very mild curling (up to 25% leaves)	1	0.25	5-9	Less than 5	R
Curling, puckering of 26-50 % leaves	2	0.50	10-19	6-25	MR
Curling, puckering of 51-75 % leaves	3	0.75	20-39	26-50	MS
Severe curling, puckering of >75% leaves	4	1.00	40-69	51-75	S
			70-100	More than 75	HS

HR: Highly Resistant, R: Resistant, MR: Moderately Resistant, MS: Moderately Susceptible, S: Susceptible, HS: Highly Susceptible

The single plant data for horticultural traits was recorded randomly from each generation in all the three replications. The data was recorded from each replication using 5 plants of P_1, P_2 and F_1, 25 plants of F_2 and 15 plants of each back cross generation excluding border plants. Percent Disease Index (PDI) was calculated for all the test materials following McKinney (1923) [26], where PDI= Σ [(s × n)/(S × N)] × 100 %, with s= disease rating scale, n= number of plants with each disease rating, N= total number of plants and S= highest disease rating scale. The coefficient of infection was calculated by multiplying the PDI with response value assigned to each severity grade. The data was recorded for following horticultural traits: plant height (cm), plant spread (cm), total yield per plant (g), number of fruits per plant, fruit weight (g), fruit length (cm), fruit width (mm) and pericarp thickness (mm). The data was recorded for following biochemical traits: dry matter content (%), capsaicin in powder (%), oleoresin content (%), coloring matter in powder (ASTA), capsaicin in oleoresin (%), coloring matter in oleoresin (ASTA).

Statistical analysis
To test the adequacy of additive-dominance model the scaling tests given by Mather (1949) [24] and Hayman and Mather (1955) [16] were used. Estimation of various genic effects and test of fitness of appropriate genetic model was done according to joint scaling test of Cavalli (1952) [5], as described in detail by Jinks and Jones (1958) [19]. Variance components viz., additive (D), dominance (H), environment (E) and correlation between D and H (F) were estimated as described by Mather and Jinks (1982) [25]. Genetic advance was calculated as per Johnson *et al.*, (1955) [20]. The statistical analysis were carried out by using 'Windostat Version 9.2' software programme developed by 'Indostat services, Hyderabad'.

Results and Discussion
Reaction of six generations for chilli leaf curl virus disease (ChiLCVD)
Reaction of six generations for ChiLCVD with respect to coefficient of infection and per cent disease index are

presented in Table 2. The line MS-341 showed a highly susceptible reaction with a PDI of 87.75 and CI of 70.20. The line S-343 showed a highly resistant reaction with PDI of 6.00 and CI of 0.6. The F_1 (MS-341 × S-343) also showed resistant reaction with a PDI of 7.18 and CI of 1.44. The backcross (F_1 × MS-341) showed a moderately susceptible reaction with a PDI of 39.89 and CI of 23.93. The back cross with S-343 showed a resistant reaction with PDI of 10.26 and CI of 2.04. The first symptoms appeared on MS-341, 13.66 days after inoculation, then F_2 (17), BC_1P_2 (20.66), BC_1P_1 (21.66), F_1 (24) and S-343 after 29.33 days.

Table 2: Reaction of six generations for chilli leaf curl virus disease with respect to coefficient of infection and per cent disease index

Generation	Symptom severity grade	Response value	Percent disease Index	Coefficient of infection	Days to symptom appearance	Reaction
MS-341 (P_1)	4.0	1.00	87.75 ± 0.87	87.75	13.66 ± 1.20	HS
S-343 (P_2)	0.1	0	6.00 ± 0.25	0	29.33 ± 2.90	HR
F_1	1.0	0.25	7.18 ± 0.18	1.79	24 ± 1.52	HR
F_2	2.0	0.50	26.11 ± 0.27	13.05	17 ± 1.15	MR
BC_1P_1	3.0	0.75	39.89 ± 0.14	29.91	21.66 ± 2.33	MS
BC_1P_2	1.0	0.25	10.26 ± 0.21	2.56	20.66 ± 2.02	HR

HS: Highly Susceptible, HR: Highly Resistant, MR: Moderately Resistant, MS: Moderately Susceptible, HR: Highly Resistant

Potency of genes from generation means

For per cent disease index (PDI) of ChiLCVD, the mean PDI of F_1 plants was intermediate between both the parents but it was skewed too much toward the better parent (S-343) and was almost equal to it, which suggests the complete dominance of S-343 for lower PDI in F_1 plants (Table 3). The role of dominance for leaf curl virus score 45 days after inoculation was also reported by Anandhi and Khader (2011) [2] in cross "Mavelikkara Local' × 'Jwalasakhi".

The mean values of cross MS-341 (susceptible parent) × S-343 (resistant parent) for all the horticultural traits have been presented in Table 3. The average value of F_1 plants surpasses both the parents for following traits viz., yield per plant, number of fruits per plant and fruit length which suggests the presence of over dominance for these traits. Joshi and Nabi (2018) [21] reported more yield of F_1 than both the respective parents used in three of the crosses studied by them, which shows the role of over dominance in inheritance of this trait. Similarly a role of over dominance have also been reported by Hasanuzzaman and Golam (2011) [15] for number of fruits per plant and fruit length. The traits viz., plant height, fruit diameter and pericarp thickness showed partial dominance toward the better parent i.e., S-343 because the value of F_1 mean was intermediate between both the parents but it was higher than the mid parent value. The role of partial dominance for plant height and fruit diameter was also reported by Joshi and Nabi (2018) [21] and Patil (2011) [31], respectively. The average fruit weight of F_1 population was intermediate between both the parents but it was skewed too much toward the better parent (S-343) and was almost equal to it, which suggests the complete dominance of S-343 for this trait. These results are not in line with Bento et al., (2016) [4], who has reported absence of dominance for average fruit weight.

Table 3: Generation means and standard errors of six generations for horticultural traits

Generation	Plant height (cm)	Plant spread (cm)	Yield per plant (g)	Number of fruits per plant	Fruit weight (g)	Fruit length (cm)	Fruit diameter (mm)	Pericarp thickness (mm)
P_1 (MS-341)	73.60 ± 0.17	57.26 ± 0.61	307.33 ± 0.67	175.73 ± 2.44	1.64 ± 0.00	1.74 ± 0.01	8.83 ± 0.05	0.82 ± 0.00
P_2 (S-343)	118.07 ± 0.46	77.20 ± 0.76	810.0 ± 2.00	217.93 ± 2.26	3.88 ± 0.06	6.30 ± 0.04	13.01 ± 0.12	1.43 ± 0.00
F_1	104.57 ± 0.40	65.26 ± 0.28	934.50 ± 2.21	244.33 ± 3.15	3.61 ± 0.03	6.76 ± 0.03	11.56 ± 0.03	1.20 ± 0.00
F_2	92.40 ± 0.72	59.36 ± 0.27	767.93 ± 1.94	262.80 ± 2.30	2.92 ± 0.02	4.55 ± 0.02	10.54 ± 0.04	1.03 ± 0.00
BC_1P_1	69.40 ± 0.42	59.56 ± 0.13	658.00 ± 1.62	281.97 ± 6.16	2.25 ± 0.03	3.88 ± 0.04	10.78 ± 0.01	0.93 ± 0.01
BC_1P_2	89.93 ± 0.57	70.38 ± 0.22	961.11 ± 1.56	249.40 ± 1.47	3.79 ± 0.03	6.62 ± 0.02	12.11 ± 0.05	1.24 ± 0.00

Table 4: Generation means and standard errors of six generations for biochemical traits

Generation	Dry matter (%)	Capsaicin content (%)	Coloring matter in powder (ASTA)	Oleoresin content (%)	Capsaicin in oleoresin (%)	Coloring matter in oleoresin (ASTA)
P_1 (MS-341)	25.12±0.27	0.86±0.02	141.74±1.15	12.32±0.21	2.30±0.10	596.51±0.10
P_2 (S-343)	18.98±0.30	0.59±0.01	171.89±2.12	10.99±0.03	2.11±0.05	562.21±1.98
F_1	25.86±0.33	0.87±0.01	235.76±1.81	15.24±0.12	2.74±0.04	567.55±1.54
F_2	23.66±0.09	0.67±0.01	140.35±1.12	13.97±0.05	1.73±0.03	563.56±1.28
BC_1P_1	24.83±0.13	0.76±0.00	144.33±0.53	13.53±0.03	1.78±0.01	578.14±1.71
BC_1P_2	21.32±0.25	0.71±0.01	212.91±0.90	12.80±0.07	2.27±0.02	557.78±0.91

The mean values of all the six generations for biochemical traits have been presented in Table 4. The mean value of F_1 surpasses both the parents for following traits viz., capsaicin in powder, coloring matter in powder, oleoresin content, capsaicin in oleoresin and dry matter content, which showed the presence of over dominance for these traits. These results are not in agreement with Dhall and Hundal (2005b) [10] for capsaicin content, Dhall and Hundal (2005a) [9] for coloring matter and Dhall and Hundal (2009) [12] for dry matter content as these authors had reported preponderance of additive gene action for these traits. The F_1 mean was intermediate between both the parents for coloring matter in oleoresin but it was lesser than the mid parent value which revealed the presence of partial dominance toward the lower value parent. The differing results in these studies may be due to the different germplasm used and prevailing climatic conditions in these two studies.

Genetic analysis of means for PDI of ChiLCVD

The significance of all the four scaling tests A, B, C and D indicates the failure of additive-dominance model and revealed the presence of all the three types of non-allelic interactions *viz.,* additive × additive (*i*), additive × dominance (*j*) and dominance × dominance (*l*). Also the significant values of chi square in joint scaling test for three parameters represents the presence of all the three types of non-allelic interactions (Table 5). In the best fit model of joint scaling test, both the additive (*d*) and dominance (*h*) gene effects were significant. Moreover, the magnitude of (*h*) component was higher than the (*d*) component. This suggests the presence of dominance along with additive effects for inheritance of this trait.

Among the epistatic effects, all the three types of interactions were significant which shows that the adequacy of best fit model could not be tested for the digenic interactions. The magnitude of (*l*) gene effects were higher than the other two gene effects, which indicated the role of dominance × dominance type of interaction. Moreover, the opposite signs of (*h*) and (*l*) effects indicated the presence of duplicate type of epistasis. The positive sign of (*l*) effects indicated higher frequencies of increaser alleles. So, it can be concluded that disease incidence is controlled by dominance, additive and epistatic effects. Anandhi and Khader (2011) [2] also reported significant levels of all types of gene actions (additive, dominance and epistasis) for yield and virus resistance. So to improve this trait methods like recurrent selection, multiple cross, or diallel selective mating system may be adopted in chilli improvement programmes.

Genetic analysis of means for horticultural traits
For almost all the traits the four scales A, B, C and D were found significant and therefore a simple additive-dominance model could not be satisfactorily account for the variation observed in this cross. The failure of additive-dominance model revealed the presence of all the three types of non-allelic interactions viz., additive × additive (*i*), additive × dominance (*j*) and dominance × dominance (*l*). Also the significant values of chi square in additive dominance model of joint scaling test revealed the presence of all the three types of non-allelic interactions (Table 5 and 6).

Table 5: Estimates of scaling tests and genic effects for PDI and horticultural traits

Parameters	Percent disease index	Plant height (cm)	Plant spread (cm)	Yield per plant (g)	Number of fruits per plant	Fruit weight (g)	Fruit length (cm)	Fruit diameter (cm)
A	-15.16* ± 0.92	-39.37* ± 0.93	-3.40* ± 0.73	74.17* ± 3.96	143.87* ± 12.93	-0.75* ± 0.09	-0.77* ± 0.07	1.18* ± 0.09
B	7.35* ± 0.51	-42.76* ± 1.32	-1.70 ± 0.92	177.71* ± 4.32	36.53* ± 4.87	0.12 ± 0.10	0.163* ± 0.08	-0.34 ± 0.18
C	-3.61* ± 1.46	-31.20* ± 3.17	-27.55* ± 1.55	85.40* ± 9.18	168.85* ± 11.64	-1.08* ± 0.15	-3.37* ± 0.18	-2.84* ± 0.25
D	2.09* ± 0.61	25.47* ± 1.63	-11.21* ± 0.59	-83.23* ± 4.49	-5.78 ± 7.83	-0.21* ± 0.08	-1.40* ± 0.05	-1.83* ± 0.13
Joint scaling test (three-parameter model)								
M	41.07* ± 0.20	92.98* ± 0.22	64.28* ±. 28	576.42* ± 0.94	205.19* ± 1.55	2.74* ± 0.02	3.86* ± 0.02	10.78* ± 0.05
[d]	33.02* ± 0.17	-20.62* ± 0.23	-9.99* ± 0.21	-268.45* ± 0.91	-18.85* ± 1.51	-1.08* ± 0.02	-2.13* ± 0.02	-1.37* ± 0.05
[h]	-33.38* ± 0.31	1.35* ± 0.43	-0.02 ± 0.49	407.74* ± 2.08	68.70* ± 3.14	0.73* ± 0.04	2.40* ± 0.03	0.95* ± 0.06
$\chi^2_{(3\,df)}$	543.13*	2483.70*	413.73*	1794.65*	287.51*	130.42*	1480.88*	451.42*
Six-parameter model								
M	51.05* ± 1.30	146.76* ± 3.28	45.09* ± 1.28	392.17* ± 9.02	196.83* ± 1.66	2.20* ± 0.05	1.24* ± 0.12	7.24* ± 0.26
[d]	40.87* ± 0.44	-22.23* ± 0.24	-10.63* ± 0.21	-251.32* ± 1.04	-21.10* ± 1.66	-1.10* ± 0.02	-2.26* ± 0.02	-2.10* ± 0.06
[h]	-55.83* ± 3.00	-175.27* ± 7.32	36.90* ± 2.96	960.68* ± 20.93	220.35* ± 9.50	1.38* ± 0.07	7.70* ± 0.34	8.86* ± 0.62
[i]	-4.16* ± 1.22	-50.93* ± 3.26	22.27* ± 1.17	166.48* ± 8.96	11.55 ± 15.65	0.55* ± 0.06	2.77* ± 0.11	3.67* ± 0.25
[j]	-22.52* ± 1.03	3.40* ± 1.52	-1.69 ± 1.10	-103.55* ± 4.97	100.61* ± 9.39	-0.88* ± 0.12	-0.93* ± 0.11	1.51* ± 0.20
[l]	11.96* ± 1.78	133.07* ± 4.21	-16.73* ± 1.81	-418.36*± 12.84	-172.85* ± 10.30	-0.19 ± 0.28	-2.18* ± 0.21	-4.51* ± 0.39
$\chi^2_{(6-p\,df)}$	--	--	2.34	--	0.53	0.50	--	--
Type of interaction		D	D	D	D	-	D	D

For almost all the traits the dominance component (*h*) was of higher magnitude than the additive component (*d*), which showed the preponderance of dominance gene action for these traits. The role of dominance have been reported earlier by Marame *et al.,* (2008) [23] for plant height, Hasanuzzaman and Golam (2011) [15] for plant spread, Joshi and Nabi (2018) [21] for yield per plant, Dhall and Hundal (2006) [11] for number of fruits per plant, Anandhi and Khader (2011) [2] for fruit length and Navhale *et al.,* (2014) [28] for fruit diameter. The traits like pericarp thickness, revealed the presence of additive gene action as only the (*d*) component was significant. The preponderance of additive gene action for these traits was also revealed by Gueddes *et al.,* (2015). The opposite signs of dominant (*h*) and dominance × dominance (*l*) component showed the presence of duplicate type of epistasis for the traits viz., plant height, plant spread, yield per plant, fruit number, fruit length and fruit diameter. Duplicate type of epistasis have also been reported by Dhall and Hundal (2006) [11], Patil (2011) [31] and Hasanuzzaman and Golam (2011) [15] for fruit length, fruit width and fruit number. Therefore, an importance of both additive and non- additive gene effects was realized with the preponderance of non-additive gene effects along with duplicate type of epistasis for most of the traits, therefore population improvement approaches such as recurrent selection and bi-parental mating would be more effective to accumulate desirable genes or to break undesirable genes.

Genetic analysis of means for biochemical traits
The four scales A, B, C and D were found significant and therefore a simple additive-dominance model could not be satisfactorily account for the variation observed in this cross. The failure of additive-dominance model revealed the presence of all the three types of non-allelic interactions. Also the significant values of chi square in additive dominance model of joint scaling test revealed the presence of all the three types of non-allelic interactions (Table 6). Two traits viz., dry matter and coloring matter in oleoresin revealed the presence of additive gene action as only the (*d*) component was significant for these traits. The preponderance of additive gene action for dry matter content is in agreement with Dhall and Hundal (2009) [12]. The traits viz., capsaicin content,

oleoresin content, coloring matter in powder and capsaicin in oleoresin, revealed higher magnitude of dominance effects than the additive, which showed importance of dominance in inheritance of these traits. For the traits viz., dry matter, oleoresin content and coloring matter in oleoresin, the dominance × dominance (*l*) type of interaction was significant and of higher magnitude, while for capsaicin content, coloring matter in powder and capsaicin in oleoresin the additive × additive (*i*) type of interaction was significantly higher. The opposite signs of dominant (*h*) and dominance × dominance (*l*) component showed the presence of duplicate type of epistasis for the traits viz., coloring matter in powder and oleoresin content. While the complementary type of epistasis was revealed only by the trait capsaicin in oleoresin. Therefore, it can be concluded that the biochemical traits are controlled by additive and non-additive gene effects, so it would be difficult to improve these through direct selection. So, to improve these traits selection should be delayed up to reduction of dominance in later segregating generations (F_4 and F_5) followed by population improvement in early segregating generations.

Table 6: Estimates of scaling tests and genic effects for biochemical traits

Parameters	Pericarp thickness (mm)	Dry matter (%)	Capsaicin content (%)	Oleoresin content (%)	Coloring matter in powder (ASTA)	Capsaicin in oleoresin (%)	Coloring matter in oleoresin (ASTA)
A	-0.18* ± 0.02	-1.29* ± 0.50	-0.20* ± 0.01	-0.52 ± 0.27	-88.81* ± 2.40	-1.50* ± 0.10	-7.78* ± 3.90
B	-0.15* ± 0.01	-2.22* ± 0.68	-0.05* ± 0.01	-0.62* ± 0.22	18.16* ± 3.32	-0.28* ± 0.09	-14.21* ± 3.10
C	-0.47* ± 0.02	-1.15 ± 0.84	-0.50* ± 0.03	2.12* ± 0.43	-223.70* ± 6.28	-2.98* ± 0.19	-39.58* ± 6.36
D	-0.08* ± 0.02	1.19* ± 0.33	-0.13* ± 0.00	1.63* ± 0.14	-76.52* ± 2.47	-0.60* ± 0.06	-8.80* ± 3.21
Joint scaling test (three-parameter model)							
M	0.97* ± 0.01	21.82* ± 0.17	0.67* ± 0.01	11.49* ± 0.05	156.73* ± 0.98	1.59* ± 0.04	575.89* ± 0.90
[d]	-0.30* ± 0.01	3.02* ± 0.15	0.08* ± 0.01	0.48* ± 0.07	-41.18* ± 0.77	-0.45* ± 0.02	19.71* ± 0.88
[h]	0.21* ± 0.01	3.48* ± 0.36	0.11* ± 0.01	3.83* ± 0.13	32.18* ± 1.87	0.83* ± 0.06	-14.09* ± 1.82
$\chi^2_{(3\,df)}$	657.17*	16.61*	165.58*	125.28*	2844.66*	291.33*	42.93*
Six-parameter model							
M	0.98* ± 0.01	22.89* ± 0.16	0.47* ± 0.02	14.95* ± 0.31	3.75 ± 5.11	0.97* ± 0.12	561.85* ± 1.409
[d]	-0.30* ± 0.01	3.12* ± 0.15	0.13* ± 0.01	0.71* ± 0.07	-14.96* ± 1.19	0.10 ± 0.13	18.04* ± 0.91
[h]	0.05 ± 0.10	-3.35 ± 1.84	0.39* ± 0.03	- 4.15* ± 0.80	322.77* ± 2.80	1.27* ± 0.29	1.39 ± 15.99
[i]	0.14* ± 0.01	-0.97* ± 0.26	0.25* ± 0.01	-3.26* ± 0.28	156.59* ± 1.18	1.20* ± 0.12	16.92* ± 1.93
[j]	-0.03 ± 0.03	0.92 ± 0.72	-0.15* ± 0.01	0.11 ± 0.28	-107.49* ± 3.11	-0.01* ± 0.04	6.31 ± 4.29
[l]	0.20* ± 0.01	2.87* ± 0.45	-0.01 ± 0.05	4.44* ± 0.56	-87.02* ± 4.21	0.48 ± 0.19	5.64* ± 2.49
$\chi^2_{(6\text{-}p\,df)}$	3.52	4.96	0.004	0.14	0.54	2.97	2.16
Type of interaction	-	--	--	D	D	--	--

Variance components and heritability estimates
The variance component estimates for all the traits are presented in Table 7. Large variations were observed for both additive and dominant components of variance with additive variance (D) ranging from -2022.56 to 674.98 and dominance variance (H) from -406.48 to 5213.72. The dominant component of variance was higher than the additive for the traits viz., percent disease index, plant spread, fruit number, fruit weight, fruit length, and dry matter, capsaicin in powder, coloring matter in powder, oleoresin content and capsaicin in oleoresin. The traits plant height, yield per plant and fruit diameter revealed higher additive variance while these were showing dominance in the genetic analysis in means. This difference in these findings may be due the presence of duplicate type of epistasis for these traits. So, selections in the later generations (F_4-F_5), recurrent selection and bi-parental mating would be more effective to improve these traits. This delay in selection permits a loss in non-additive gene effects. Heritability is the expression of the extent to which the genotype of an individual determines its phenotype. The broad sense heritability reflects all the genetic contributions to a population's phenotypic variance and includes gene effects due to additive variance, dominance variation and the variation which acts epistatically. Narrow sense heritability includes gene effects due to additive gene effects. In the present study almost all the traits showed low magnitude of additive and environmental variances, revealing higher estimates of broad and narrow sense heritability's. Considerable differences were observed in broad sense and narrow sense heritability's for all the traits. The broad sense heritability varied from 0.02 to 0.94 (Table 7). The narrow sense heritability's were less than the broad sense heritability for the traits viz., fruit number, fruit weight, fruit length, pericarp thickness, dry matter and coloring matter in oleoresin. The lesser narrow sense heritability for fruit weight and fruit diameter was also reported by Marame *et al.,* (2008) [23] and Bento *et al.,* (2016) [4]. The negative value of heritability may be due to the negative sign of phenotypic variance. The negative heritability may arise due to random noise acting on estimates of genuinely positive heritability, but it may also arise from misspecication of the standard additive mechanism that is supposed to justify the statistical procedure. It may be a possibility that negative heritability estimates could reflect a real physical feature of the biological process from which the data were sampled (Steinsaltz *et al.,* 2018) [36].

Table 7: Components of genetic variance, degree of dominance and heritability estimates for all the traits

Traits	Additive variance (D)	Dominance variance (H)	Environmental variance (E)	Broad sense heritability (h²b)	Narrow sense heritability (h²n)	Genetic Advance	√H/D	F
Plant height (cm)	115.60	-77.41	2.02	0.94	1.42	12.44	0.81	-7.57
Plant spread (cm)	16.07	-31.09	5.18	0.05	1.47	0.23	1.39	-1.38
Yield per plant (g)	674.98	-406.48	46.61	0.83	1.19	28.90	0.77	7.77

Fruit number	-2022.56	5213.72	104.84	0.73	-2.54	30.20	1.60	1607.20
Fruit weight (g)	-0.03	0.26	0.02	0.69	-0.22	0.37	2.94	0.001
Fruit length (cm)	-0.13	0.31	0.01	0.49	-2.77	0.16	1.52	0.01
Fruit diameter (cm)	0.32	-0.31	0.11	0.42	0.82	0.38	0.98	-0.17
Pericarp thickness (mm)	-0.02	0.02	0.00	-2.50	-21.84	-0.11	1.00	0.01
Capsaicin content (%)	0.01	-0.02	0.00	0.02	1.40	0.00	1.40	-0.001
Coloring matter in powder (ASTA)	281.46	-366.47	45.96	0.52	1.47	10.37	1.14	-23.02
Oleoresin content (%)	0.39	-1.07	0.35	-0.26	0.71	-0.36	1.66	-0.23
Capsaicin in oleoresin (%)	0.17	-0.46	0.07	-0.48	1.67	-0.23	1.63	-0.01
Dry matter (%)	-5.52	7.70	1.41	-1.43	-4.73	-2.25	1.18	-2.27
Coloring matter in oleoresin (ASTA)	147.74	49.96	36.52	0.70	0.60	16.04	0.58	95.63
Disease incidence (%)	17.00	-28.10	4.23	0.25	1.48	1.27	1.28	-1.01

Also the heritability estimates are influenced by the type of genetic material, sample size, method of sampling, and conduct of experiment, method of calculation and effect of linkage.

The rate of genetic advance is connected with heritability (Mather and Jinks, 1982) [25]. It is influenced by the genetic variability, heritability and selection intensity (Sharma et al., 2003) [35]. The genetic advance was moderate for yield per plant (28.90), fruit number (30.20) and coloring matter in oleoresin (16.04) and while all the other traits revealed very low genetic advance (Table 7). These results are contradictory to Manju and Sreelatha kumary (2006) [22], who have reported high genetic advance for almost all the traits studied. The lower genetic advance in our study may be due to the presence of duplicate type of epistasis.

The perusal of values of average degree of dominance ($\sqrt{H/D}$) from Table 7 revealed that average dominance ratio was more than unity for the traits viz., disease incidence, plant spread, fruit number, fruit weight, fruit length, dry matter, capsaicin in powder, coloring matter in powder, oleoresin content and capsaicin in oleoresin which showed the importance of the dominance gene effects which is in agreement with the low narrow sense heritability in some of the traits like fruit number, fruit weight, fruit length and dry matter. Average degree of dominance more than unity was also reported by Marame et al (2008) [23] for number of fruits per plant, fruit weight and fruit length. F is an indicator of correlation between additive and dominance variance if F is zero or in positive direction it means that dominant genes are in the parent with high performance while negative F denotes that dominant genes are in the low performing parent.

Conclusion
The results showed that, percent disease index for ChiLCVD and most of the yield contributing traits were controlled by both additive and non-additive effects. However, the magnitude of non-additive effects was much higher than the additive effects in almost all the traits. Most of the traits also showed the presence of duplicate type of epistasis. The heritability estimates and genetic advance were also less which supports the role of non-additive gene action controlling these traits. The role of dominance effects controlling these traits was also supported by degree of dominance which was more than unity for most of the traits. So, to improve all these traits in a chilli breeding programme recurrent selection and bi-parental mating would be more effective to accumulate desirable genes or to break undesirable genes.

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