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Genetics of Cercospora Leaf Spot Resistance in Mungbean [*Vigna radiata* (L.) Wilczek] through Generation Mean Analysis

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ABSTRACT

Background: Cercospora leaf spot (CLS) is a fungal disease of mungbean [*Vigna radiata* (L.) Wilczek] caused by *Cercospora canescens* and now emerged as an important biotic stress. A better understanding of the genetics of CLS resistance will help in formulating efficient breeding procedures in mungbean.

Methods: The present investigation focused on genetics of CLS resistance through generation mean analysis (six parameter model) in two intra-specific mungbean crosses namely, Kopergaon × HUM12 and Kopergaon × ML1720. Four quantitative disease resistance components, *viz*., area under disease progress curve (AUDPC), incubation period (IP), latent period (LP) and degree of sporulation (SP) were studied.

Result: A high correlation of AUDPC with latent period (r = -0.68 to -0.79, P < 0.0001) and SP (r = 0.72 to -0.81, P<0.0001) advocated that both are main contributor for CLS disease development. High heterosis along with high heritability in terms of AUDPC (>0.09) indicated the importance of genetic factor(s) in controlling CLS resistance. Generation mean analysis of both the crosses revealed duplicate epistatic interaction and involvement of two genes for CLS resistance in terms of AUDPC. This study supports oligogenic nature of inheritance, advocating AUDPC along with IP, LP and SP as important disease indicator for selection of CLS resistance in mungbean.

Key words: AUDPC, *Cercospora*, Epistasis, Heritability, Heterosis, Mungbean.

INTRODUCTION

Pulses are one of the most important components of dietary food crops after cereals in Indian diet especially vegetarian diet. Mungbean [*Vigna radiata* (L.) Wilczek] is an Indian origin legume crop (de Candole, 1886; Zhukovsky, 1950; Bailey, 1970) whose most of the genetic potentials still remains to be exploited. It has a number of benefits over other crop species and ranked as an international crop worldwide in recent decades. At present, mungbean attracts multidisciplinary research to dissect genetic potential to different attributes mainly yield, nutrition and stresses. Cercospora leaf spot (CLS) caused by a fungal pathogen *Cercospora canescens* Ellis and Martin is one of the biotic stresses in mungbean that significantly compromises the yield from 23 to 96% (Chand *et al.,* 2013; Nair *et al.,* 2019). This biotic stress is of emerging importance especially in humid tropical regions, like India where high humidity prevails during the growing season of the crop (Grewal *et al.,* 1980). Although, effective control of the disease with the fungicide application (Singh and Singh, 1978; Khunti *et al.,* 2005) is popular yet the highly recommended, economically feasible and ecologically viable disease management can be achieved only by exploiting host plant resistant and several CLS resistant or moderately resistant mungbean sources have been reported (Raje and Rao, 2002; Singh and Singh, 2014; Akhtar *et al.,* 2014; Bhasker, 2017; Nair *et al.,* 2019) but, the present scenario of climate change, population explosion, pressure of nutritional security along with soil health makes it inevitable to breed such CLS resistant mungbean varieties.

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Studies on CLS resistance in different crop species indicated a diverse mechanism of inheritance. Inheritance of CLS diseases resistance is worked out as polygenic in nature in *C. beticola* / sugar beet (*Beta vulgaris*) interaction (Smith and Campbell, 1996) while monogenic expressed as either recessive, complete or incomplete dominance in soybean (*Glycine max*) and cowpea (*Vigna unguiculata*) (Castro *et al.,* 2003; Booker and Umaharan, 2008). Similarly, Inheritance of resistance to CLS in corn (*Zea mays*) is quantitative, associated with additive, dominant, recessive and epistatic effects (Saghai *et al.,* 1996; Coates and White, 1998) wheras under a monogenic dominant gene control in Faba bean (Kimber and Paull, 2011) and by two genes in

southern pea (Fery *et al.,* 1976; Fery and Dukes, 1977). Studies on genetic analysis to CLS resistance in mungbean, using different resistant sources revealed that the resistance is controlled by either a single dominant gene (Thakur *et al.,* 1977; Lee, 1980; Chauhan and Gupta, 2004), a single recessive gene (Mishra *et al.,* 1988) or quantitative genes (Chankaew *et al.,* 2011). Hence, characterization of genetic basis of CLS resistance largely depended on the different resistant and susceptible sources used in a study (Castro *et al.,* 2003).

Plant breeding relies on estimates of gene effects for improvement of the trait in question. Three major factors which are crucial in the analysis of quantitative genetic variation has been suggested by Sprague (1966) are the number of genes involved, the type of gene action and the genotype × environment interaction. Additive-dominance model or simply additive model with simpliûed interpretation of genetic variation for the estimation of different genetic effects (Kearsey and Pooni, 2004) basically ignores the epistatic or non-allelic interactions. Generation mean analysis (GMA) is a biometrical tool which provides information on the relative importance of average effects of the genes (additive effects), dominance deviations and effects due to non-allelic genetic interactions such as additive × additive, dominance × dominance and additive × dominance genetic effects (Mather and Jinks, 1982). Genetic studies for yield and its component as well as plant habit have been studied extensively (Singh *et al.,* 2007; Rehman *et al.,* 2010; Iqbal *et al.,* 2015) and ample documentations are available, but genetic studies for CLS resistance breeding requires an impetus in mungbean breeding programs.

The progress in disease resistance breeding relies on an accurate and precise estimate of disease (Montes *et al.,* 2007; Bock *et al.,* 2010). Components of quantitative resistance or partial resistance in different crops to CLS have been identified (Aquino *et al.,* 1995, Ricker *et al.,* 1985) while in mungbean it still requires extensive studies. Longer latent periods, lower lesion number and reduced capacity for sporulation have been identified as components of rate reducing resistance against *Cercospora* and other similar leaf spot diseases in various crops (Parlevliet, 1979; Nevill, 1981; Ricker *et al.,* 1985). Therefore, this study incorporated the components of CLS resistance *viz*., incubation period (IP), latent period (LP) and degree of sporulation (SP) along with disease severity *i.e*., area under disease progress curve (AUDPC) to understand the mode of inheritance, the magnitude of gene effects, their mode of action, heritability and number of effective genes governing the CLS resistance in mungbean using two intraspecific crosses.

MATERIALS AND METHODS

Plant material and planting

The plant material consisted of the six generations (P_1, P_2, F_1, F_2, BC_r and BC_s) derived from two intraspecific crosses of mungbean, *viz*., Kopergaon × HUM12 (Cross I) and Kopergaon × ML1720 (Cross II). Kopargaon is a widely adopted cultivar, susceptible to CLS. Whereas, HUM12 is highly adapted and high yielding cultivar and ML1720 is a promising breeding line, both are resistant to CLS. The crossing procedure was carried out in polyhouse while final scoring for all the disease components were performed at the Agriculture Research Farm, Institute of Agricultural Sciences, Banaras Hindu University, during the successive growing seasons of 2016-17, 2017-18 and 2018-19. In 2016-17 season, the crosses were made among the parents to produce F_1 seeds; in 2017-18 season, F_1 plants were selfed to produce F_2 seeds and backcrossed to the respective parents to produce BC_r and BC_s seeds also fresh F_1 were consecutively developed in 2017-18 so that all the six generations from each cross were sown in a single season for an unbiased disease scoring. In 2018-19, the six generations of the two crosses were grown in field conditions in such a way that 42 plants in two rows for each of the parents and F_1s, 45 plants in two rows for back cross and 64 plants in four rows for the F_2 population were raised. Rows were 2 m long and 30 cm apart and plant to plant spacing was 20 cm. The experiment was done under epiphytotic conditions for CLS disease. The soil was fertilized and recommended agronomic package was followed with hand weeding in the field.

Artificial inoculation of CLS

A pathogenic strain of *C. canescens* 'MTCC-10835' has been used in the present study. Mass culturing and artificial inoculation was done by following protocol of Chand *et al* (2013). The inoculum (spore suspension) for artificial inoculation was prepared from 25 days old colonized sorghum grains (200 g) by soaking in 1 liter of sterilized water for 5 minutes. These grains were agitated thoroughly in water to dislodge the spores and filtered through two-fold muslin cloths. The inoculum (10^4 spore ml^{-1}) was delivered on mungbean leaves at flowering stage by spraying with a knapsack sprayer between 16.00 and18.00 hours (Chand *et al.,* 2013). The following morning at 07.00 hours field was irrigated to maintain high humidity. The field was irrigated after every two days in case of no rain, to maintain humidity.

Data collection

CLS disease severity was first scored after inoculation when around twenty percentage of leaf area were covered with leaf spots on susceptible parent Kopergaon and most of the lines showed the disease symptoms, second, third and fourth disease scoring was performed at 5, 4 and 3 day interval respectively by adopting the 1-9 scale (where, 1= no infection, 2 = upto 10%, 3 = 10.1-20%, 4 = 20.1-30%, 5 = 30.1-40%, 6 = 40.1-50%, 7 = 50.1-65%, 8 = 65.1-80% and 9 = >80% disease severity) and the Area Under Disease Progress Curve (AUDPC) was calculated using formula given by Shaner and Finnay (1977):

$$\sum_{i=1}^{n} [\{Yi + (Yi + 1)\}/2\{(ti + 1) - ti\}]$$

Where,
Yi = Disease level at time ti.
(ti+1)-ti = Time (days) between two percentage diseased area scores.
n = Number of observations (score).

Incubation period (IP) by subtracting the inoculation day from day to appearance of the first lesion (Aquino *et al.,* 1995); Latent period (LP) by subtracting inoculation day from day to appearance of first sporulating lesion (Aquino *et al.,* 1995); Degree of Sporulation (SP) by manual counting of sporulated lesion was done 30 days after inoculation on tagged leaves of each plant (Smith, 1980).

Statistical analysis

The statistical analysis for analysis of variance, correlation and regression was carried out using the statistical software SAS 9.3. To select the most suitable model for generation mean analysis (GMA), the Scaling test of Mather (1949) and joint scaling test of Cavalli (1952) were employed. Six parameter model of GMA was performed using the data obtained for six generations *viz*., P_1, P_2, F_1, F_2, BC_r and BC_s using Mather and Jinks method (1982). In this method, the mean of each character is indicated as follows:

$$Y=m+\alpha d+\beta h+\alpha 2[i]+2\alpha\beta[j]+\beta^2[1]$$

Where
Y = Mean of one generation.
m = Mean of all generations (population mean).
d = The sum of additive effects.
h = Total dominance effects.
i = Additive × additive effect (complementary).
j = Additive × dominance effects.
l = Dominance × dominance effect (duplicate).
α, $2\alpha\beta$ and β^2 = Coefficients of the model genetic parameters.

The genetic parameters (*m*, [*d*], [*h*], [*l*], [*j*], [1]) were tested for significance using a *t*-test. The type of epistasis was determined only when dominance (*h*) and dominance × dominance (*l*) effects were found to be significant. When these effects had the same sign, the gene effect was referred to as complementary whereas, the different signs indicated duplicate gene interaction (Kearsey and Pooni, 1996). Broad-sense (H_b) heritability was estimated as per Warner (1952).

Estimation of number of gene(s)

Number of genes segregating for CLS resistance was estimated using F_2 generation according to Wright's formula (Wright, 1968).

$$n = GR^2 \times [1.5 - 2h(1 - h)]/\{8[VF_2 - (VP_s + VP_r + 2VF_1)/4]\}$$

Where
n = Number of genes segregating.
GR = Genotypic range.
*V*Pr = Variance of resistant parent.
*V*Ps = Variance of susceptible parent.
VF_1 = Variance of F_1 generation.
VF_2 = Variance of F_2 generation.

$$h = (MF_1 - MP_r)/(MP_s - MP_r)$$

in which
MF_1 = Mean of F_1 population,
MP_r = Mean of resistant parent.
MP_s = Mean of susceptible parent.

Genotypic range was estimated by using the phenotypic range of segregating population, which does not assume that segregating genes come from a single parent; thus, it can be applied to resistant × resistant crosses as well as to resistant × susceptible crosses. Genotypic variance was estimated by subtracting environmental variance from phenotypic variance of F_2 population.

RESULTS AND DISCUSSION

Analysis of variance revealed significant differences for all the four traits (AUDPC, IP, LP and SP) studied in the two crosses (Kopergaon × HUM 12 and Kopergaon × ML 1720), indicating the existence of genetic variation and possibility of selection for CLS disease resistance in mungbean (Supplementary Table 1). The means and standard error of different traits for each generation are summarized in Table 1. Mean AUDPC of 849.17 has been observed on susceptible parent 'Kopergaon' whereas, resistant parent duo *i.e*., HUM 12 and ML 1720 exhibited least AUDPC of 88 and 85.79, respectively (Table 1). This huge difference between AUDPC values of resistant and susceptible parents suggested that the disease pressure was sufficiently high to reveal genetic

Supplementary Table 1: ANOVA for CLS resistance components in two mungbean crosses.

Trait	Source	DF	Kopergaon × HUM12		Kopergaon × ML 1720	
			SS	MS	SS	MS
AUDPC	Treatment	5	599524.40**	119904.88**	612780.817**	122556.16**
	Replication	1	15.377	15.377	0.003	0.003
IP	Treatment	5	79.99**	16.00**	92.52**	18.50**
	Replication	1	0.006	0.006	0.001	0.001
LP	Treatment	5	109.42**	21.88**	106.98**	21.40**
	Replication	1	0.000	0.000	0.145	0.145
SP	Treatment	5	8971.36**	1794.27**	8926.44**	1785.30**
	Replication	1	0.472	0.472	37.868	37.868

**Significant at p<0.001; *Significant at p<0.01.
AUDPC = Area under disease progress curve; IP = Incubation period (Days); LP = Latent period (Days) and SP = Degree of sporulation.

Table 1: Mean ± SE value of CLS resistance components in different generations of two mungbean crosses.

Cross	Kopergaon × HUM12				Kopergaon × ML1720			
generations	AUDPC	IP	LP	SP	AUDPC	IP	LP	SP
P1	849.17±5.15	7.74±0.26	16.86±0.24	95.17±2.4	849.17±5.15	7.74±0.26	16.86±0.24	95.17±2.4
P2	88.08±2.38	15.33±0.27	26.14±0.26	9.38±1.57	85.79±2.26	16.67±0.24	26.86±0.22	5.31±0.75
F_1	409.88±5.93	15.24±0.26	25.14±0.26	25.10±2.35	560±4.13	10.38±0.23	20.64±0.29	52.02±4.61
F_2	361.80±14.13	12.69±0.23	22.51±0.21	48.81±3.50	421.36±16.85	10.85±0.23	20.87±0.25	41.03±2.81
BCs	432.68±11.01	11.96±0.25	20.85±0.25	48.94±2.10	548.40±6.22	10.19±0.23	20.89±0.26	61.19±3.68
BCr	473.24±33.02	11.26±0.34	22.61±0.30	63.25±3.89	469.83±23.98	12.12±0.42	22.63±0.43	48.50±4.35

AUDPC = Area under disease progress curve; IP = Incubation period (Days); LP = Latent period (Days) and SP = Degree of sporulation.
BCs = F_1 × Susceptible parent.
BCr = F_1 × Resistant parent.

difference between resistance and susceptibility. Resistant parent HUM12 and ML1720 exhibited lowest mean value for SP (9.38, 5.31) and highest mean value for IP (15.33, 16.67) and LP (26.14, 26.86) in cross I and cross II, respectively. Mean values for other generations, *viz*., F_1, F_2, BC_s and BC_r lies between mean values of either parent for all the traits, revealed no transgressive segregant was observed in these generations for any of the trait studied.

Association studies were made among all the four traits in the F_2 generation of the two crosses. All the four traits were found to be significantly correlated with each other (Table 2). A significantly high and negative correlation was noticed for IP and LP with AUDPC in both the crosses (r = -0.90 to - 0.975; P<0.0001) while, correlation between SP and AUDPC was found to be significantly positive in both the crosses. Similarly, LP and IP exhibited significant positive correlation with each other but both these traits were negatively associated with SP. So, correlation studies underline the importance of these traits in breeding for CLS resistance in mungbean.

The values of individual estimates of gene effects *viz*., *m*, *d*, *h*, *i*, *j* and *l* for four different traits in the two crosses were estimated (Table 3). Highly significant values for scaling test and chi-square confirmed the presence of inter-allelic interaction in cross for all the traits studied (Supplementary Table 2). In the present study, Generation mean analysis revealed that a simple additive-dominance model was not adequate to explain the variation among the generations for resistance traits and indicated the presence of non-allelic interaction (s) for the CLS resistance. A six-parameter model was fitted to the data. The mean parameter (*m*) for all the traits studied indicated that the contribution due to the overall mean plus the locus effects and interaction of the fixed loci was signiûcant for both the crosses (Table 3). Significant additive gene effect (*d*) estimates were observed for IP in cross I and for SP in cross II. Significant dominant gene effect (*h*) was estimated for AUDPC in cross I, while for AUDPC and SP for cross II. Duplicate epistatic interaction was observed for AUDPC in both the crosses and SP in cross II. Furthermore, the additive and additive × additive components were together larger than the dominant component for all the traits in both the crosses (Table 3). The dominant component was larger than the additive and additive × additive components, although they were both in the same direction for AUDPC in cross II. By ignoring the sign, magnitude of dominance × dominance (*l*) was greater than additive × additive (*i*) for almost all the traits in both crosses.

Heritability in broad-sense (*Hb*), heterosis over mid-parent (HMP) and better parent (HBP) and number of effective genes in F_2 generation are estimated (Table 4). *Hb* was higher than 0.90 for AUDPC in both the crosses. While, moderate heritability was observed for IP, LP and SP ranging from 0.55 to 0.89 in both the crosses. The estimates of *Hb* suggested that selection in early generations can be effective for the CLS resistance in these crosses. The percentage of heterosis with respect to better parent (BP) and mid-Parent (MP) illustrated that heterosis over mid-parent was positive

for IP and LP in cross I while for AUDPC and SP in cross II. Heterosis over better parent was positive for AUDPC and SP for both the crosses. For AUDPC and SP, better parent is the one with lower values (Table 4). In the present study, F_2 plants showed continuous variation for AUDPC and no discrete segregation was observed. Hence, the quantitative method was used to estimate gene number. The number of genes segregating for CLS resistance was estimated using F_2 generation in both the crosses (Table 4). A minimum of two resistant genes were appeared to be segregating in both the crosses for CLS resistance in terms of AUDPC. Whereas, 3-8 genes were estimated to govern CLS resistance through LP, IP and SP in both the crosses (Table 4).

The present investigation was undertaken to estimate the nature and magnitude of gene actions, heritability in broad sense, heterosis over mid-parent and better parent and number of genes segregating for four CLS disease components, *viz*., AUDPC, LP, IP and SP among two intra-specific mungbean crosses, *viz*., Kopergaon × HUM 12 and Kopergaon × ML 1720.

The correlation results where IP and LP showed significant negative correlation with AUDPC in both the crosses but a significant positive association with SP were in agreement with that obtained by Aquino *et al* (1995) in *C. personatum*)/ groundnut interaction where, AUDPC values were highly correlated with latent period (r = –0.68 to –0.79, P <0.01). Longer latent period and incubation periods, reduced sporulation, smaller lesion diameter and reduced leaf area damage and disease score have been identified as resistance components for *Cercospora* early and late leaf spot disease in groundnut (Waliyar *et al.,* 1993; Dwivedi *et al.,* 2002). The correlation results also indicated that different CLS resistance components are under the same genetic control *i.e.,* the genomic regions controlling these components are either co-localized or pleiotropic in nature, which will only be deciphered after detailed analysis of CLS resistance in the mungbean.

In the present study, the scaling test and chi square value showed that both the crosses have significant epistatic gene effects for the all the traits, *viz*., AUDPC, IP, LP and SP, showing inadequacy of additive-dominance model (*m*, *d* and *h*) and presence of non- allelic interaction and hence, six parameter model was extended to estimate the gene effects. GMA results for CLS disease resistance were not in consensus with results of Duangsong *et al* (2018) on Yardlong bean, where a simple additive-dominance model

Table 2: Estimates of correlation coefficients among CLS resistance components in two mungbean crosses.

Cross	Kopergaon × HUM12			Kopergaon × ML1720		
Trait	AUDPC	IP	LP	AUDPC	IP	LP
IP	-0.94***			-0.960***		
LP	-0.90***	0.85***		-0.975***	0.962***	
SP	0.80***	-0.75***	-0.76***	0.541***	-0.498***	-0.506***

***Significant at p<0.0001.

AUDPC = Area under disease progress curve; IP = Incubation period (Days); LP = Latent period (Days) and SP = Degree of sporulation.

Table 3: Estimates of genetic parameters in generation mean analysis for CLS resistance components in two mungbean crosses.

Trait	Kopergaon × HUM12				Kopergaon × ML1720			
	AUDPC	IP	LP	SP	AUDPC	IP	LP	SP
[*m*]	361.797**	12.688**	22.508**	48.805**	421.356**	10.852**	20.867**	41.031**
[*d*]	-40.563	0.694*	-1.764**	-14.306**	-78.576**	-1.931**	-1.736**	12.694
[*h*]	305.888**	-0.603	0.528	1.992	443.56**	-0.589	2.345	57.05**
[*i*]	364.632**	-4.306**	-3.115	29.17*	351.036**	1.233	3.559*	55.264**
[*j*]	-842.208**	8.984**	5.758**	-114.397**	-606.228**	5.067**	6.528**	-64.468**
[*l*]	-419.44*	11.409**	9.484**	-98.821**	-332.542	-0.705	-5.587**	-70.129*
Epistasis	Duplicate	-	-	-	Duplicate	-	-	Duplicate

**Significant at p<0.001; *Significant at p<0.01.

AUDPC = Area under disease progress curve; IP = Incubation period (Days); LP = Latent period (Days) and SP = Degree of sporulation.

Supplementary Table 2: Scaling test values along with chi-square values for CLS resistance components in two mungbean crosses.

Trait	Kopergaon × HUM12					Kopergaon × ML1720				
	A	B	C	D	χ^2	A	B	C	D	χ^2
AUDPC	393.70**	-448.51**	309.83**	-182.32**	706.08**	312.36**	-293.87**	369.53**	-175.52**	1107.20**
IP	-0.94	8.044**	2.80	2.15**	239.6**	-2.27**	2.79**	1.76**	-0.62	67.15**
LP	0.31	6.06**	3.25**	1.56	152.45**	-4.28**	2.25**	1.53	-1.78**	129.01**
SP	22.37**	-92.02**	-40.48	-14.59	324.28**	24.80**	-39.67**	40.40*	-27.63**	80.21**

**Significant at p<0.001; *Significant at p<0.01.

AUDPC = Area Under Disease Progress Curve; IP = Incubation Period (Days); LP = Latent Period (Days) and SP = Degree of sporulation.

Table 4: Heritability in broad sense (Hb), heterosis over mid-parent (HMP) and better parent (HBP) and number of effective genes segregating in F_2 generation for CLS resistance components in two mungbean crosses.

Cross	Kopergaon × HUM12				Kopergaon × ML1720			
Trait	AUDPC	IP	LP	SP	AUDPC	IP	LP	SP
Hb	0.96	0.56	0.55	0.88	0.98	0.64	0.66	0.62
HMP	-12.54	32.10	16.94	-51.99	19.79	-14.93	-5.56	3.55
HBP	365.34	-0.62	-3.83	167.51	552.79	-37.72	-23.14	879.74
No. of effective genes in F_2	2.2	8.16	5.06	2.76	2.43	3.88	4.25	4.26

AUDPC = Area under disease progress curve; IP = Incubation period (Days); LP = Latent period (Days) and SP = Degree of sporulation.

was adequate to explain the genetic control CLS disease resistance. Both additive and non additive gene effects played significant role and, duplicate type of epistasis was found for AUDPC in both the crosses. In this case, mild selection intensity should be applied in the early generation followed by imposing intense selection intensity in the later generations that will result in selection of disease resistance. GMA studies on yield and related agro-morphological traits in mungbean, indicated that gene effects varied with cross and traits (Hegde *et al*., 1994; Azizi *et al*., 2006).

Contrary to the resuts of Duangsong *et al*. (2018) where they reported that the average number of major genes controlling the CLS resistance in yardlong bean was 1.05 and 0.92 for *C. canescens* and *P. cruenta*, respectively advocating that the resistance to CLS disease caused by *C. canescens* and *P. cruenta* in grain cowpea 'IT90K-59-120' is governed by a single major recessive gene, our result on inheritance of CLS resistance in mungbean were in accordance with results by Chankaew *et al*. (2011) advocating quantitative genetic control of resistance to CLS in mungbean as well as others like CLS resistance in sugar beet (*Beta vulgaris*) caused by *C. beticola* and resistance to *C. zeae-maydis* in corn (*Zea mays*) which advocates quantitative nature of disease and polygenic nature of resistance, associated with additive, dominant, recessive and epistatic effects indicating the difficulty inbreeding for resistance while maintaining yield (Smith and Campbell, 1996; Saghai *et al.,* 1996; Coates and White, 1998). It is worth to mention that number of genes segregating for CLS resistance is varying in both the crosses, it indicated that both the resistant parents (HUM12 and ML1720) are having different source of resistance as, the susceptible parent (Kopergaon) was common in both the crosses. QTL mapping effort will be able to brief about the genomic regions controlling the of CLS resistance in these crosses using all the four traits (AUDPC, IP, LP and SP) studied for CLS resistance in the present study.

The scope for exploitation of hybrid vigour depends upon the direction and magnitude of heterosis and type of gene action involved. Although advantages of hybrid vigour cannot be exploited commercially in highly self-pollinated crops like mungbean, heterotic F_1s can be used to isolate higher frequency of transgressive segregants in their later generations. High heterosis along with high heritability estimates for the resistance in terms of AUDPC (>0.09) indicated that the resistance is mainly controlled by genetic factor(s).

CONCLUSION

In conclusion, the present study provides valuable information on the gene effects and genetics of CLS resistance in mungbean. CLS resistance is quantitative in nature and so often would be complex in inheritance. Genetics of such traits and knowledge on interactions would help to develop a suitable breeding strategy. It can also be concluded that the nature and magnitude of gene effects differ with different crosses and showed importance of both additive and non-additive gene effects in the inheritance of CLS resistance traits studied. Further, duplicate type of epistasis was found to be commonly operated and thus, the success of different resistance breeding methods would be effective either in form of recurrent selection and diallel selective mating given by Jensen (1970) or biparental mating in early segregating generations. AUDPC along with IP, LP and SP can be used as disease indicator for selection of CLS resistance in mungbean.

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Conflicts of interest/Competing interests

The authors declare that they have no conflict of interest.

Ethics approval (N/A)

Availability of data and material

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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