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GENETICS OF MUNGBEAN [*VIGNA RADIATA* (L.) WILCZEK] CERCOSPORA LEAF SPOT RESISTANCE: A HOST PLANT DISEASE RESISTANCE APPROACH TO IDM STRATEGY IN AGRICULTURE

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Abstract

The cercospora leaf spot (CLS) disease of mungbean [*Vigna radiata* (L.) Wilczek] is one of the important biotic stresses to the mungbean production. It is a fungal disease caused by *Cercospora canescens (Ellis & Mart.)*. Even though chemical management of plant disease is popular, exploitation of host resistance as management strategy is a step towards enhancing the resilience of agriculture to climate change. In this regard, the current study aimed on genetics of CLS resistance through generation mean analysis (six parameter model) in an inter-specific cross namely, Kopergaon (mungbean) × PU31 (urdbean). Four quantitative disease resistance components *viz*., Incubation period (IP), Latent period (LP), Lesion number count (LN) and degree of sporulation (SP) were studied. Area under disease progress curve (AUDPC) was calculated as measure of disease severity. Significant association of AUDPC with IP ($r = -0.91$, $P < 0.01$) and LP ($r = -0.87$, $P < 0.01$), suggested their important role in quantitative disease resistance influencing CLS disease development. High broad sense heritability (Hb) of AUDPC (=0.98), IP (0.89), LP (0.90) and SP (0.86) indicated the role of genetic factor(s) in regulating mungbean host plant CLS resistance. Multiple linear regression analysis revealed IP as the factor having maximum influence on AUDPC. Generation mean analysis studies revealed involvement of two genes for CLS resistance in terms of AUDPC. Present study supports oligogenic nature of inheritance, suggesting AUDPC along with IP, LP and SP as important factors for selection of CLS resistance in mungbean.

***Keywords*:** Mungbean, *Cercospora canescens*, AUDPC, Genetics and Inheritance.

Introduction

Cercospora leaf spot (CLS) of mungbean is known to significantly compromise the mungbean yield from 23 to 96% (Chand *et al*., 2013; Zhimo *et al*., 2013; Bhat *et al*., 2014), in humid tropical regions, like India where high temperature and humidity prevails during the growing season of the crop (Grewal *et al.*, 1980). It is caused by a fungal pathogen *Cercospora canescens* Ellis and Martin. Chemical management of mungbean CLS is popular but, in the present scenario of accumulating pressure of climate change, population explosion, soil health maintenance and nutritional security it becomes the need of hour to exploit host plant resistant as plant disease management strategy. The breeding program exploiting the host plant disease resistance depends on the knowledge of the type of gene action involved in its expression as it provides the basis for evaluation of selection methods. Inheritance studies on mungbean CLS resistance have proposed variety of mechanism, according to which, CLS resistance is controlled by either a single dominant gene (Thakur *et al*., 1977; Lee, 1980), a single recessive gene (Mishra *et al.,* 1988) or quantitative genes (Chankaew *et al*., 2011).

For the complex traits like host plant disease resistance, epistatic effects are important (Eta-Ndu and Openshow, 1999). Epistatic effects interact more strongly with the environment than additive and dominance gene effects (Adetimirin *et al*., 2001; Eta-Ndu and Openshaw, 1999). Presence of epistasis, may lead to biased estimates of genetic parameters causing erroneous expectations from selection (Eta-Ndu and Openshaw, 1999). Compared with other mating designs such as diallel, Generation mean analysis (Mather and Jinks, 1982) has an increased level of sensitivity through a decreased error rate (Hallauer and Miranda, 1988). Mather and Jinks (1982) model describes the phenotype in terms of the mid parental values [m], additive effects [d], dominance effects [h] and additive by additive [i], additive by dominance [j] and dominance by dominance [l] epistatic interaction effects (Mather and Jinks, 1982).

The host plant disease resistance breeding counts on an accurate and rigid estimate of disease (Montes *et al*., 2007; Bock *et al*., 2010). Longer latent periods, lower lesion number and reduced capacity for sporulation have been recognized as factors of rate reducing resistance against *Cercospora* leaf spot diseases in different crops (Parlevliet 1979; Nevill 1981; Ricker *et al*., 1985). Current study incorporated incubation period (IP), latent period (LP), lesion number (LN) and degree

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of sporulation (SP) as the components of CLS resistance *viz*., along with disease severity calculated as, area under disease progress curve (AUDPC).

Materials and Methods

Development of Planting material and sowing

The six generations (P1, P2, F1, F2, BCr and BCs) derived from an interspecific cross of mungbean *viz*., Kopergaon and urdbean *viz*., PU31 consisted of the planting material. Kopargaon, a CLS susceptible parent is a widely adopted cultivar whereas, PU31, immune to CLS, is highly adapted, high yielding cultivar. The crossing procedure was carried out in polyhouse whereas the final scoring for disease severity and 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 F1 seeds were produced by crossing the parents; in 2017-18 season, F2 seeds were produced by selfing the F1 plants and F1 plants were backcrossed to the respective parents to produce BCr and BCs seeds also. In 2017-18 season, fresh F1 were consecutively developed 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 21 plants in two rows for each of the parents and F1s, 23 plants in two rows for back cross and 42 plants in four rows for the F2 population were raised. Rows were 2 m long and 30 cm apart and plant to plant spacing was 20 cm. The experiment was conducted 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 inoculum

The present study utilized a pathogenic strain of *C. canescens* 'MTCC-10835'. The protocol by Chand *et al*. (2013) was followed for mass culturing and artificial procedure. The inoculums (spore suspension) for artificial inoculation utilized 25 days old colonized sorghum grains (200 g) soaked 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 flowering stage of mungbean plant was utilized for delivery of inoculum (10^4 spore ml^{-1}). Inoculum was delivered on mungbean leaves by spraying with a knapsack sprayer between 16.00 and18.00 hours (Chand *et al*., 2013). The field was irrigated the following morning at 07.00 hours to maintain high humidity. To maintain humidity, the field was irrigated after every two days in case of no rain.

Data recording

Disease score estimates were recorded as visual assessment of percentage necrotic area. 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. The second, third and fourth disease scoring was performed at 5, 4 and 3 day interval respectively. 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).

Recording of Incubation period (IP) was done 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); Total number of lesions was counted on 20^{th} day after first inoculation (LN) as adopted from Johnston *et al*. (1986). Degree of Sporulation (SP) by manual counting of sporulated lesion was done 30^{th} days after first inoculation on tagged leaves of each plant (Smith, 1980). All the data were recorded on five tagged fully open trifoliate leaves in two replications. Leaf area of Kopargaon leaves (average of tagged trifoliate leaves) were taken standard for Lesion numbers counts and Degree of Sporulation.

Statistical analysis

The statistical analysis for analysis of variance, correlation and regression was carried out using the statistical software SAS 9.3 (SAS 2014). Selection of the most suitable model for generation mean analysis (GMA) comprised of the Scaling test of Mather (1949) and joint scaling test of Cavalli (1952). Six parameter model of GMA was carried out Mather and Jinks method (1982) using the data obtained from six generations *viz*., P1, P2, F1, F2, BC1 and BC2. In this method, each character mean was indicated by the formulae:

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

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, 1 = dominance × dominance effect (duplicate) and α, 2αβ and β2 are the coefficients of the model genetic parameters. The significance of the scales and gene effects were tested by using the t-test at 0.05 and 0.01 level of significance. The type of epistasis can be determined only when dominance (h) and dominance × dominance (l) effects were found to be significant. The gene effect were considered complementary when these effects had the same sign, whereas, the different signs indicated duplicate gene interaction (Kearsey and Pooni, 1996). Broad-sense (Hb) heritability was estimated as per Warner (1952).

Estimation of number of gene(s)

Wright's formula (Wright, 1968) was utilized to estimate the number of genes segregating for CLS resistance using F2 generation. The formula is as under:

$$n = GR2 \times [1.5 - 2h(1-h)]/\{8[VF2 - (VPs + VPr + 2VF1)/4]\}$$

Where, n = number of genes segregating, GR = genotypic range, VPr = variance of resistant parent, VPs = variance of susceptible parent, VF1 = variance of F1 generation, VF2 = variance of F2 generation and h = (MF1 – MPr)/(MPs – MPr), in which MF1 = mean of F1 population, MPr = mean of resistant parent, and MPs = 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 F2 population.

Results and Discussion

The present investigation was undertaken to estimate the nature and magnitude of gene actions, association, heritability in broad sense, overdominance and number of genes segregating for four CLS disease resistance components, *viz*., LP, IP, LN and SP along with AUDPC in an inter-specific mungbean - urdbean cross, *viz*., Kopergaon × PU31.

Analysis of variance

Analysis of variance revealed significant differences among generations ($P < 0.01$) for AUDPC along with the all the four components (IP, LP, LN and SP) studied in the interspecific cross (Kopergaon × PU31), indicating the presence of genetic variation and prospects of selection for CLS disease resistance in mungbean.

Mean, standard error and variance

Mean and standard error estimates of five variables under CLS resistance study were found to be highly variable among the parents as well as in the segregating generations (Table 1). The susceptible parent 'Kopergaon' recorded the highest AUDPC Mean of 849.17, whereas, resistant parent PU31 recorded immunity by exhibiting no disease symptom (Table 1). This difference between AUDPC values of resistant and susceptible parents suggested optimum disease pressure to sufficiently reveal genetic difference between resistance and susceptibility. Mean values for other generations, *viz*., F_1, F_2, BC_s and BC_r lied between mean values of either parent for all the traits.

Correlation and multiple regression estimates

Correlation studies were made among AUDPC and all the four traits in the F_2 generation of the cross. All the traits were found to be significantly correlated with each other (Table 2). A significantly high and negative correlation was recorded for IP ($r = -0.91$, $P < 0.001$) and LP ($r = -0.87$, $P < 0.001$) with AUDPC while, correlation between SP and AUDPC was found to be significantly positive ($r = 0.73$, $P < 0.001$). Similarly, LP and IP displayed significant positive association with each other ($r = 0.90$, $P < 0.001$) but both these traits were negatively associated with LN ($r = -0.59$, $P < 0.001$; $r = -0.59$, $P < 0.001$) and SP ($r = -0.65$, $P < 0.001$; $r = -0.79$, $P < 0.001$). These correlation results were in agreement with that recorded by Aquino *et al.* (1995) in *C. personatum*)/ groundnut interaction where, AUDPC values were highly associated with LP ($r = –0.68$ to $–0.79$, $P < 0.01$). Longer LP and IP, reduced SP, smaller lesion diameter, and reduced leaf area damage and disease score have been recognized as factors of resistance for early and late *Cercospora* leaf spot disease in groundnut (Waliyar *et al.,* 1993; Dwivedi *et al.,* 2002). The correlation results also suggested that various CLS resistance factors are under the same genetic control *i.e.*, in nature the genomic regions regulating these components are either co-localized or pleiotropic, which can only be interpreted after comprehensive analysis of CLS resistance in the mungbean. So, correlation studies supported these variables as important components of host plant CLS disease resistance influencing the disease development. Multiple linear regression analysis identified IP as the component having maximum influence on AUDPC (Table 3).

Genetics and gene interaction

The values of individual estimates of gene effects *viz*., *m*, *d*, *h*, *i*, *j* and *l* for different traits in the cross were estimated (Table 4). 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 all the traits studied for the mungbean host plant CLS resistance extending to six parameter model to estimate the gene effects.. 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 significant for the cross (Table 4). Significant additive gene effect (*d*) estimates were observed for AUDPC, IP and LP whereas significant dominant gene effect (*h*) was estimated for AUDPC, LN and SP for the cross (Table 4). So, both additive and non-additive gene effects played significant role. Duplicate epistatic interaction was observed for LN alone suggesting predominantly dispersed alleles at the interacting loci (Jinks and Jones, 1958) for this trait. The dominant component was larger than the additive and additive × additive components, although they were both in the same direction for AUDPC in the cross. These GMA results for mungbean CLS disease resistance were not in agreement with those of Duangsong *et al.* (2018) working on Yardlong bean × grain cowpea cross, where a simple additive-dominance model was adequate to explain the genetic control CLS disease resistance.

Hb, h/d and number of effective genes

Heritability in broad-sense (*Hb*), over-dominance (h/d) and number of effective genes in F_2 generation are estimated (Table 5). High broad sense heritability (Hb) of AUDPC (=0.98), IP (0.89), LP (0.90), LN (0.89) and SP (0.86) indicated the influence of genetic factor(s) in regulating mungbean CLS resistance. Dominance gene effects (h) were found to be relatively more important with higher values than the additive (*d*) values in case of AUDPC. The dominance gene action would favor the production of hybrids (Edwards *et al*., 1975). In the present study, F_2 plants showed continuous variation for AUDPC and other disease resistance components 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 the cross (Table 5). A minimum of two resistant genes were appeared to be segregating in the cross for CLS resistance in terms of AUDPC. Whereas, 3-5 genes were estimated to govern host plant CLS resistance through IP, LN and SP in the cross (Table 5). Hence, current study supported oligogenic nature of inheritance, advocating AUDPC along with IP, LP and SP as important indicators for selection of CLS resistance in mungbean. Present results were contrary to those of Duangsong *et al.* (2018) where they reported single major recessive gene (1.05 and 0.92 for *C. canescens* and *P. cruenta*, respectively) governing the resistance to CLS disease caused by *C. canescens* and *P. cruenta* while working on yardlong bean. Current result were in consensus with results by Chankaew *et al*. (2011) advocating quantitative genetic control of resistance to CLS in mungbean. The result was in agreement with CLS resistance in other crop/phytopathogen interaction like in sugar beet (*Beta vulgaris*)/ *C. beticola* and corn (*Zea mays*)/ *C. zeae-maydis* where quantitative nature of disease and polygenic nature of resistance, associated with additive, dominant, recessive and epistatic effects have been suggested indicating the difficulty inbreeding for resistance while maintaining yield (Smith and Campbell, 1996; Saghai *et al.,* 1996; Coates and White, 1998). It is worth mentioning that number of genes segregating for mungbean host plant CLS resistance is varies with the crosses, indicating that the resistant parents may have different source of resistance.

Conclusion

The current study presents useful information on the gene effects and genetics of host plant resistance to CLS in mungbean. Mungbean CLS resistance is concluded to be quantitative in nature and complex in inheritance. Although it has been observed that the nature and magnitude of gene effects differ with different crosses, the knowledge on the genetics and gene interactions of such traits would help to develop a suitable mungbean CLS resistance breeding strategy. The nature and magnitude of gene effects (additive, dominance and epistatic types) varied with traits under study suggesting that appropriate breeding method is the one that can effectively exploit all the three types of gene effects simultaneously. Further, duplicate type of epistasis was found to operate for LN alone. Both additive and non-additive gene effects were important in the inheritance of CLS resistance traits proposing the use of reciprocal recurrent selection and diallel selective mating given by Jensen (1970) or Bi-parental 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. Furthermore, QTL mapping effortscan be exploited to decipher the genomic regions regulating the of mungbean host plant CLS resistance using all the traits (AUDPC, IP, LP, LN and SP) studied for CLS resistance in the present study.

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Table 1: Mean ± SE and variance (VAR) estimates of mungbean host plant CLS resistance components in different generation ofinterspecific cross (Kopergaon × PU31)

TRAITS	P1		P2		F1		F2		BCs		BCr	
	Mean ± SE	VAR	Mean ± SE	VAR	Mean ± SE	VAR	Mean ± SE	VAR	Mean ± SE	VAR	Mean ± SE	VAR
AUDPC	849.17±5.15	556.3	0	0	619.17±5.96	1640.45	496.67±22.41	21097.34	690.49±14.81	5045.73	383.48±29.07	19434.3
IP	7.74±0.26	1.418	0	0	8.91±0.16	0.53	12.82±0.38	6.05	9.98±0.50	5.67	12.74±0.77	13.53
LP	16.86±0.24	1.2	0	0	18.95±0.17	0.58	22.51±0.37	5.84	19.63±0.31	2.19	21.61±0.63	9.22
LN	95.17±2.40	120.44	0	0	119.5±2.61	142.99	85.94±3.93	647.72	103.54±6.43	950.74	86.02±9.11	1907.53
SP	7.16±0.04	0.04	0	0	93.24±3.56	266.09	47.57±4.70	926.25	60.5±5.70	746.57	62.65±6.57	988.54

AUDPC = Area under Disease Progress Curve; IP = Incubation Period (Days); LP = Latent Period (Days); LN = Lesion number count and SP = Degree of Sporulation.
BCs = F_1 × Susceptible parent
BCr = F_1 × Resistant parent

Table 2: Estimates of correlation coefficients among mungbean host plant CLS resistance components in interspecific cross (Kopergaon × PU31)

TRAITS	**AUDPC**	**IP**	**LP**	**LN**
IP	**-0.91****			
LP	**-0.87****	**0.90****		
LN	0.64**	-0.59**	-0.59**	
SP	**0.74****	-0.65**	**-0.79****	**0.71****

(**significant at $p < 0.001$)
Trait abbreviations as mentioned in Table 1.

Table 3: Estimates of Multiple regression analysis with AUDPC as dependent variable in interspecific cross (Kopergaon × PU31)

TRAITS	**Coefficients**	**S. E.**	**t-value**	**Significance**
IP	-45.399	9.286	-4.889	**0**
LP	6.445	12.374	0.521	0.597
LN	0.367	0.551	0.666	0.499
SP	-0.152	0.913	-0.166	0.866
Constant	2,609.78			

Trait abbreviations as mentioned in Table 1.

Table 4: Estimates of genetic parameters in generation mean analysis for mungbean host plant CLS resistance components in interspecific cross (Kopergaon × PU31)

Traits	**m**	**d**	**h**	**i**	**j**	**1**	**Epistasis**
AUDPC	496.667**	307.011**	355.851**	161.268	-235.145**	-221.703	-
IP	12.821**	-2.761**	-0.815	-5.851	-13.259**	-14.036**	-
LP	22.512**	-1.978**	2.954	-7.569**	-20.813**	-20.146**	-
LN	85.94**	17.522	82.321**	35.369	-110.051**	-30.403**	Duplicate
SP	47.571**	-2.152	101.673**	56.019	-99.471**	-20.68	-

Trait abbreviations as mentioned in Table 1.

Table 5: Heritability in broad sense (Hb), overdominance (h/d) and number of effective genes segregating in F_2 generation for mungbean host plant CLS resistance components in interspecific cross (Kopergaon × PU31)

Traits	**Hb**	**h/d**	**Effective genes (F2)**
AUDPC	0.98	1.08	2.44
IP	0.89	0.54	4.25
LP	0.90	1.22	-
LN	0.89	2.17	5.33
SP	0.86	6.87	3.25

Trait abbreviations as mentioned in Table 1.