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Inheritance Pattern of Mungbean Yellow Mosaic Virus (MYMV) Disease Resistance in Blackgram [*Vigna mungo* (L.) Hepper]

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ABSTRACT

Background: Mungbean yellow mosaic virus (MYMV) disease is the most destructive disease in blackgram. Development of MYMV resistant varieties is one of the best possible solutions to avoid the yield reduction in blackgram. There are conflicting reports on the genetics of resistance to MYMV disease claiming that it is controlled by both dominant and recessive genes. Hence the present study was aimed to understand the inheritance pattern of the MYMV disease resistance in eight crosses of blackgram.

Methods: Parents, F_1 and F_2 generation of eight cross combinations were raised during July - Sep, 2018 at National Pulses Research Centre, Tamil Nadu Agricultural University, Vamban, Tamil Nadu. An infector row of CO 5 was raised to intensify the MYMV disease pressure after every eight rows. Based on disease incidence on 60th day after sowing, two phenotypic classes were formed among F_2 plants with the scales of (1 to 3) as resistant phenotype and (4 to 9) as susceptible phenotype. The goodness of fit to Mendelian segregation ratio for MYMV disease resistance in the segregating population was tested by Chi square test (Stansfield, 1991).

Result: The MYMV disease incidence was tri-genically controlled with inhibitory gene action in four crosses *viz.,* MDU 1 x Mash 114, CO5 x Mash 114, MDU 1 x VBN 6 and CO 5 x VBN 6. Complementary gene action with two genes was observed in four crosses *viz.,* MDU 1 x Mash 1008, CO 5 x Mash 1008, MDU 1 x VBN 8 and CO 5 x VBN 8. Differences in number of genes were observed due to the presence of recessive inhibitory gene in both male and female parents of the crosses which had complementary gene action for MYMV disease. The putative gene symbols assigned for the six genotypes *viz.,* $S_1S_1S_2S_2ii$ (MDU 1 and CO 5), $s_1s_1s_2s_2II$ (Mash 114 and VBN 6) and $s_1s_1s_2s_2ii$ (Mash 1008 and VBN 8), respectively.

Keywords: Blackgram, Complementary gene action, Genetics, Inhibitory gene action, MYMV disease.

INTRODUCTION

Blackgram [*Vigna mungo* (L.) Hepper] is one of the important pulse crop of India. India is the largest producer and also consumer of blackgram. It is referred as the "king of the pulses" due to its delicious taste and numerous other nutritional qualities. Blackgram is superb combination of all nutrients, which contains proteins (25-26%), carbohydrates (60%), fat (1.5%), minerals, amino acids and vitamins. Among various biotic and abiotic yield limiting factors, yellow mosaic disease (YMD) caused by mungbean yellow mosaic virus (MYMV) is the most destructive limiting factor in blackgram. Infection of MYMV may cause up to 85–100% yield loss in blackgram (Singh *et al.,* 2011). Main pathogens causing YMD in India are mungbean yellow mosaic virus (MYMV) and mungbean yellow mosaic India virus (MYMIV) (Varma and Malathi, 2003; Malathi and John, 2008). The disease is caused by geminivirus with bipartite genomes transmitted by different species of whitefly belongs to the genus begomovirus and family geminiviridae. Infected plants are stunted in growth and usually mature late. They produce very few flowers and pods. Pods are curled and reduced in size with increased percentage of shrivelled seeds (Nariani 1960; Nene 1973). Since the virus transmission is attributed by the vector-whitefly (*Bemisia tabaci*), control of MYMV disease is based upon limiting the vector population by using insecticides. However, it is ineffective under severe whitefly infestations and also not an eco-friendly approach. The most effective way to prevent the occurrence of this disease is to develop genetically resistant cultivars of blackgram. There are conflicting reports about the genetics of resistance to MYMV disease, claiming both resistance and susceptibility is controlled by dominant gene. In blackgram, monogenic dominant nature of resistance was reported by Dahiya *et al.* (1977), Kaushal and Singh (1988) and Gupta *et al.* (2005), while it was

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reported to be digenic recessive by Singh (1980), Dwivedi and Singh (1985) and Verma and Singh (1986). Monogenic recessive control of yellow mosaic resistance was also reported by some authors (Pal *et al.* 1991; Reddy and Singh 1995). Hence, the present study was aimed to understand the inheritance pattern and the gene action governed by the MYMV disease resistance in eight crosses of blackgram.

MATERIALS AND METHODS

The material for the present investigation consists of six parents and their eight crosses in F_2 generation. Female parents *viz.,* MDU 1 and CO5 were highly susceptible to mungbean yellow mosaic virus (MYMV) disease. Male parents *viz.,* Mash 114, Mash 1008, VBN 6 and VBN 8 were resistant to MYMV disease. Eight crosses *viz.,* MDU 1 x Mash 114, CO 5 x Mash 114, MDU 1 x Mash 1008, CO 5 x Mash 1008, MDU 1 x VBN 6, CO 5 x VBN 6, MDU 1 x VBN 8, CO 5 x VBN 8 were generated during Dec 2017- Feb 2018 under field conditions at National Pulses Research Centre, Vamban. Parents, F_1 and F_2 generation of these cross combinations were raised during July - Sep, 2018 at National Pulses Research Centre, Tamil Nadu Agricultural University, Vamban, Tamil Nadu. Each progeny was raised in one row of 3 m length with a spacing of 30 x 10 cm. Susceptible genotypes *viz.,* CO 5 and MDU 1 were sown as disease spreader rows after every eight rows and also around the plots. All parents, F_1 and F_2 generations were screened for MYMV disease by adopting infector row method. An infector row of CO 5 was raised to intensify the MYMV disease pressure after every eight rows of test rows. Insecticides were not sprayed during the cropping period in order to maintain the natural whitefly population in the field. The MYMV disease incidence was recorded on all plants of F_2 populations and parents based on the visual scores on 60th day after sowing. Based on disease grade two phenotypic classes were formed among F_2 plants with the scales of (1 to 3) as resistant phenotype and (4 to 9) as susceptible phenotype. Grouping of phenotypic classes were carried out as reported earlier (Dahiya et al. 1977; Verma and Singh, 1986; Singh, 1980; Dwivedi and Singh, 1985; Verma and Singh, 1986; Kaushal and Singh, 1988; Pal *et al.* 1991; Reddy and Singh 1995; Gupta *et al.,* 2005; Murugan and Nadarajan, 2012). The rating scale suggested by Singh *et al.* (1988), was adopted as given below:

Grade	Description	Reaction
1	No visible symptoms on leaves	Free
2	Small yellow specks with restricted spread covering 0.1-5% leaf area	Highly Resistant (HR)
3	Mottling of leaves covering 6-10% leaf area	Resistant (R)
4	Yellow mottling covering 11-15% leaf area	Moderately resistant (MR)
5	Yellow mottling and discolouration of 15-20% leaf area	Moderately susceptible (MS)
6	Yellow coloration of 21-30% leaves and yellow pods	Susceptible (S)
7	Pronounced yellow mottling and discoloration of leaves and pods, reduction in leaf size and stunting of plants covering 30-50% 0f foliage	
8	Severe yellow discoloration of leaves covering 50-75% of foliage, stunting of plants and reduction in pod size	Highly susceptible (HS)
9	Severe yellowing of leaves covering above of foliage, stunting of plants and no pod formation	HS

The goodness of fit to Mendelian segregation ratio for MYMV disease resistance in the segregating population was tested by Chi square test (Stansfield, 1991).

Table 1: Details of parents involved in the genetic study.

Genotype	Parentage	MYMV Disease scale	Reaction
Female parents			
CO 5	Pureline selection from Musiri local	9	Highly Susceptible
MDU 1	ADB 2003 x VBG 66	9	Highly Susceptible
Male parents			
Mash 114	Mash 338 x RBI 1	1	Free
Mash 1008	SML-32 x Mash-1	1	Free
VBN 6	Vamban 1 x *V. mungo* var *sylvestris*	1	Free
VBN 8	Vamban 3 x VBG 04-008	3	Resistant

RESULTS AND DISCUSSION

The details of parental particulars were presented in Table 1. Among eight crosses *viz*., MDU 1 x Mash 114, CO 5 x Mash 114, MDU 1 x VBN 6 and CO 5 x VBN 6 were resistant to MYMV disease, while other four crosses were found to be susceptible. The parents MDU 1 and CO 5 were used as common female parents for all the crosses. The appearance of both resistance and susceptibility in the F_1s partly agreed with previous findings. Dominant nature of MYMV disease resistance was reported by Dahiya *et al.* (1977), Verma and Singh (1986), Kaushal and Singh (1988) and Murugan and Nadarajan (2012). While the recessive nature of MYMV disease resistance was reported by Singh (1980), Dwivedi and Singh (1985) and Verma and Singh (1986). The appearance of the various phenotypic expression of MYMV disease incidence among the F_1s clearly showed the contradiction on the inheritance pattern. The inheritance patterns of MYMV disease resistance in F_2 generation of cross combinations were studied and the results are presented in Table 2. The results of the crosses *viz.,* MDU 1 x Mash 114, CO 5 x Mash 114, MDU 1 x VBN 6 and CO 5 x VBN 6 indicated goodness of fit for trigenic inhibitory gene action for MYMV disease. These crosses had the common male parents *viz.,* Mash 114 and VBN 6. The trigenic inheritance pattern indicated the presence of a third gene that influences the masking effect of phenotypic expression. Trigenic inhibitory gene action was reported by several workers in blackgram (Solanki *et al.,* 1982; Verma and Singh, 1986; Reddy and Singh, 1995 and Murugan and Nadarajan, 2012). The results of crosses viz., MDU 1 x Mash 1008, CO 5 x Mash 1008, MDU 1 x VBN 8 and CO 5 x VBN 8 indicated goodness of fit for digenic complementary gene action for MYMV disease. These crosses had the male parents *viz.,* Mash 1008 and VBN 8. A similar type of digenic interaction for MYMV resistance in black gram was reported by Verma and Singh (1980), Sandhu *et al.* (1985), Shukla and Pandiya (1985) and Thamodhran *et al.* (2015). The differential expression of the inheritance pattern showed that the variation might be due the alleic pattern of parents. The digenic nature of inheritance of MYMV disease can be explained only if the third gene is recessive in both parents of a cross. Based on these findings, putative gene symbols for each parent were worked out (Table 3.). The female parents MDU1 and CO5 had similar allelic pattern ($S_1S_1S_2S_2ii$), which act in complementation. Male parents Mash 114 and VBN 6 had similar allelic pattern ($s_1s_1s_2s_2ii$), in which third gene was responsible for the inhibitory gene action. Hence, the appearance of resistant phenotype in the F_1 ($S_1s_1S_2s_2ii$) might be due the influence of the inhibitory gene in the third locus. The male parents Mash 1008 and VBN 8 had similar allelic form ($s_1s_1s_2s_2ii$), and showed the complimentary gene interaction in the crosses. Hence the F_1 ($S_1s_1S_2s_2ii$) obtained from the cross combination involving male parents Mash 1008 and VBN 8 had susceptible reaction.

Based on the foregoing discussion, it may be concluded that the MYMV disease resistance is governed by recessive genes in these eight crosses of blackgram. The presence of three genes is confirmed with the various types of interaction obtained in the study. However, the gene symbols allotted are subject to confirmation by allelic tests. The allelic tests may be conducted by intercrossing all the four male parents and studied the resistant pattern for MYMV disease incidence. Hence, recombination breeding with two or three

Table 2: Chi-square test for inheritance of MYMV disease resistance in blackgram.

Generation	F_1 Phenotype	F_2 Phenotype					Gene action
		Observed values		Expected ratio	÷2 values	Probability between	
		Resistant	Susceptible				
MDU 1 x Mash 114	Resistant	77	15	49:15	2.61 ns	20-30	Inhibitory gene action
CO 5 x Mash 114	Resistant	59	25	49:15	1.87 ns	30-50	Inhibitory gene action
MDU 1 x VBN 6	Resistant	81	36	49:15	3.50 ns	10-20	Inhibitory gene action
CO 5 x VBN 6	Resistant	51	17	49:15	0.09 ns	95-98	Inhibitory gene action
MDU 1 x Mash 1008	Susceptible	69	94	7:9	0.001 ns	90-95	Complimentary gene action
CO 5 x Mash 1008	Susceptible	33	50	7:9	0.21 ns	50-70	Complimentary gene action
MDU 1 x VBN 8	Susceptible	40	69	7:9	1.35 ns	20-30	Complimentary gene action
CO 5 x VBN 8	Susceptible	35	54	7:9	0.30 ns	50-70	Complimentary gene action

ns – not significant at 5 % probability.

Table 3: Gene symbols for parents involved in the study.

Parent	Reaction to MYMV	Gene symbol for MYMV disease incidence
CO 5	Highly Susceptible	$S_1S_1S_2S_2ii$
MDU 1	Highly Susceptible	$S_1S_1S_2S_2ii$
Mash 114	Free	$s_1s_1s_2s_2II$
VBN 6	Free	$s_1s_1s_2s_2II$
Mash 1008	Free	$s_1s_1s_2s_2ii$
VBN 8	Resistant	$s_1s_1s_2s_2ii$

cycles of recurrent selection may be a viable option to harness the MYMV disease resistance in these two susceptible female parents of blackgram.

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