Int.J.Curr.Microbiol.App.Sci (2018) *7*(1): 880-885

International Journal of Current Microbiology and Applied Sciences
ISSN: 2319-7706 **Volume 7 Number 01 (2018)**
Journal homepage: http://www.ijcmas.com

Original Research Article https://doi.org/10.20546/ijcmas.2018.701.107

Inheritance of Mungbean Yellow Mosaic Virus Disease Resistance in Greengram [*Vigna radiata* (L.) Wilczek]

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Keywords

Greengram, Mungbean yellow mosaic virus, Inheritance

Article Info

Accepted:
10 December 2017
Available Online:
10 January 2018

A B S T R A C T

Mungbean Yellow Mosaic Virus (MYMV) disease is one of the most devastating diseases of greengram causing 85 – 100 per cent yield loss and transmitted by the white fly, *Bemisia tabaci.* This has become increasingly serious because of the lack of resistance in the existing cultivars. Ten greengram genotypes were screened for MYMV disease resistance at hotspot of National Pulses Research Centre, Vamban during *kharif* season. Among them, SML 1815, MH 421 showed resistant reaction whereas, VBN (Gg) 3, VBN (Gg) 2, LGG 460, RMG 10-28 and TM 96-2 showed susceptible reaction. Three crosses were made between resistant and susceptible genotypes. The three F_2 populations were sown along with blackgram CO5 as infector row for evaluation against MYMV resistance. The F_2 population segregated in the ratio of 15R:1S and 13R:3S, suggesting the involvement of two dominant genes in imparting resistance against MYMV disease.

Introduction

Yellow Mosaic Virus Disease (YMVD) inflicts heavy yield losses in five economically important food legumes including blackgram (*Vigna mungo*), soybean (*Glycine max*), mungbean (*Vigna radiata*), frenchbean (*Phaseolus vulgaris*) and mothbean (*Vigna aconitifolia*). Greengram, which is mainly cultivated in India, Myanmar, Thialand, Philippines and Pakistan are highly prone to YMVD. The disease is caused by representative species of the genus *Begomovirus*. Based on several studies, it has been confirmed that at least two virus species causing YMVD are prevalent in Indian sub-continent. One of these species, mungbean yellow mosaic India virus (MYMIV) is commonly occurring in northern part of Indian sub-continent while Mungbean Yellow Mosaic Virus (MYMV) is mostly confined to peninsular region of India (Varma and Malathi, 2003; Malathi and John, 2008). These two virus species can easily be distinguished on the basis of nucleotide sequence identity (Fauquet *et al.*, 2003).

The losses due to Mungbean Yellow Mosaic Virus Disease (MYMVD) have been observed from 60 to 100 %. Since the virus

transmission is attributed by the vector-whitefly (*Bemisia tabaci*), control of MYMVD based upon limiting the vector population by using insecticides is ineffective under severe whitefly infestations. Further, this is also not an eco-friendly approach. The most effective way to prevent the occurrence of this disease is to develop genetically resistant cultivars of greengram. There are conflicting reports about the genetics of resistance to MYMVD, claiming both resistance and susceptibility to be dominant. Monogenic dominant nature of resistance was reported by Dahiya *et al.*, (1977), Kaushal and Singh (1988) and Gupta *et al.*, (2005) while it was reported to be digenic recessive by Singh (1980), Dwivedi and Singh (1985), Verma and Singh (1986). Monogenic recessive control of yellow mosaic resistance has also been reported (Pal *et al.*, 1991; Reddy and Singh, 1995).

Information on the inheritance of resistance to MYMVD disease is useful in breeding for resistant cultivars. Inheritance of resistance to MYMVD in mungbean has been studied extensively using different resistant sources but results were contradictory. Therefore the present investigation was conducted to understand the inheritance pattern of resistance to MYMVD in the segregating material as mentioned below.

Materials and Methods

The present investigation was conducted at National Pulses Research Center, Vamban a hot spot for MYMVD screening in India. A total of ten mungbean genotypes (Table 1) were screened for their resistance against MYMVD during *Kharif* 2015. Based on the screening results two resistant genotypes *viz*., SML 1815 and MH 421 and two susceptible genotypes *viz*., VBN (Gg) 2 and VBN (Gg) 3 (Table 2) were used to produce three cross combinations *viz*., VBN(Gg) 2 x SML 1815, VBN(Gg) 3 x SML 1815, VBN (Gg) 3 x MH 421 during *Kharif* 2015. These F_1s were raised along with parents during *rabi* 2015-16 and true F_1 plants were identified to build up F_2 population. All the three F_2 populations were evaluated during *kharif* 2016 along with highly susceptible cultivar CO 5 of blackgram (*Vigna mungo* (L.) Hepper) as infector row. For each four rows of F_2 populations, an infector row of CO 5 was raised to intensify the MYMVD pressure. Insecticides were not sprayed during the cropping period in order to maintain the natural whitefly population in the field. Individual plants of the parents and F_2 were scored after 100 per cent of the plants in infector rows showed MYMV disease.

Based upon the MYMVD score, the mungbean plants were classified into five categories, resistant (R), moderately resistant (MR), moderately susceptible (MS), susceptible (S) and highly susceptible (HS). Plants that are moderately susceptible (MS), susceptible (S) and highly susceptible (HS) were included in susceptible group while the resistant (R), moderately resistant (MR) were included in resistant group. Mendelian segregation ratio for MYMVD (resistance: susceptible) in the segregating population was tested through Chi-square test to confirm goodness of fit of each cross.

Results and Discussion

In India, the major bottleneck experienced in the expansion of mungbean cultivation has been attributed to its susceptibility to mungbean yellow mosaic virus which is most threatening disease of mungbean. Cultivation of resistant varieties is the cheapest and most effective method of combating mungbean yellow mosaic virus. For developing high yielding, yellow mosaic virus resistant varieties, it is prerequisite to identify the stable resistant source as well as to understand its nature of inheritance.

Ten mungbean genotypes, namely SML 1815, MH 421, IPM 409-4, IPM 410-3, LGG 460, RMG 10-28, TM 96-2, Gam 5, VBN (Gg)2 and VBN (Gg)3 were screened for their resistance against MYMVD during *kharif* 2015 season. Among these genotypes, LGG 460, TM 96-2, RMG 10-28, VBN (Gg)2 and VBN(Gg) 3 clearly exhibited susceptibility whereas SML 1815 and MH 421 showed resistance reaction to mungbean yellow mosaic virus (MYMV) (Table 1).

To decipher the inheritance pattern of MYMV resistance, three greengram cross combinations were effected *viz*., VBN(Gg) 2 x SML 1815, VBN(Gg) 3 x SML 1815, VBN (Gg) 3 x MH 421 and evaluated during *rabi* 2015-16 season (Table 2).

The rating scale suggested by Singh *et al*., (1988), was adopted as given below

Scale	Percentage of plant foliage affected	Reaction
1	Mottling of leaves covering 0.1 to 5.0% of the leaf area	Resistant
3	Mottling of leaves covering 5.1 to 10.0% of the leaf area	Moderately resistant
5	Mottling and yellow discolouration of 10.1 to 25.0% of the leaf area	Moderately susceptible
7	Mottling and yellow discolouration of 25.1 to 50.0% of the leaf area	Susceptible
9	Severe yellow mottling on more than 50.0% and up to 100% of the leaf area	Highly Susceptible

Table.1 Details of the mungbean genotypes and their reaction against MYMV disease

Sl. No	Variety / Genotype	Total No. of plants screened	Disease scale	Reaction
1	VBN (Gg) 3	30	7	Susceptible
2	VBN (Gg) 2	30	5	Moderately susceptible
3	MH 421	30	1	Resistant
4	IPM 410-3	30	3	Moderately resistant
5	IPM 409-4	30	3	Moderately resistant
6	LGG 460	30	7	Susceptible
7	TM 96-2	30	5	Moderately susceptible
8	Gam 5	30	3	Moderately resistant
9	RMG 10-28	30	7	Susceptible
10	SML 1815	30	1	Resistant

Table.2 Chi square test for inheritance of MYMVD resistance in greengram

Sl. No	Generation	Disease reaction		Total number of plants	Expected ratio	χ^2 Value	χ^2 Table value (0.05)
		Resistance	Susceptible				
1	VBN(Gg) 2 x SML 1815	131	10	141	15:1	0.17	3.841
2	VBN(Gg) 3 x SML 1815	123	26	149	13:3	0.16	3.841
3	VBN (Gg) 3 x MH 421	133	15	148	15:1	1.46	3.841

Fig.1 Screening of F_2 population of VBN (Gg) 2 x SML 1815 cross combination against MYMV disease

In the present study, the inheritance of MYMVD resistance in the cross combinations *viz*., VBN (Gg) 2 x SML 1815, VBN (Gg) 3 x MH 421 reveals a F_2 segregation ratio of 15:1 (Resistant: Susceptible), indicating the involvement of two dominant genes (Fig. 1). Whereas in VBN (Gg) 3 x SML 1815 cross combinations the F_2 result revealed a ratio of

13:3 (Resistant: Susceptible) which indicate the presence of inhibitory gene action. The present result was also in accordance with earlier report of Murugan and Nadarajan (2012), Durga Prasad *et al.,* (2015) and Thamodharan *et al.,* (2016).

From the above discussion it was found that, though in two crosses same female parent VBN (Gg) 3 was involved there exist two types of gene interactions *viz*., duplicate dominant interaction(15:1) in VBN (Gg) 3 x SML 1815 and inhibitory dominant interaction(13:3) in VBN (Gg) 3 x MH 421. The differences in gene interaction may be attributed to the presence of modifier genes in male parents. From the above results it has been concluded that the resistance of MYMVD is under the influence of digenic dominant interaction with some modifier genes. Hence, it may appropriate to suggest the recombination breeding and delayed selection may be effective to enhance the MYMVD resistance in greengram.

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How to cite this article:

Mahalingam, A., V.K. Satya, N. Manivannan, S. Lakshmi Narayanan and Sathya, P. 2018. Inheritance of Mungbean Yellow Mosaic Virus Disease Resistance in Greengram [*Vigna radiata* (L.) Wilczek]. *Int.J.Curr.Microbiol.App.Sci.* 7(01): 880-885.
doi: https://doi.org/10.20546/ijcmas.2018.701.107