## **Electronic Journal of Plant Breeding**

### **Research Article**



# Inheritance of resistance to Mungbean Yellow Mosaic Virus (MYMV) in mungbean [*Vigna radiata* L. Wilczek]

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#### Abstract

Six basic populations ( $P_1, P_2, F_1, F_2, B_1$  and  $B_2$ ) of 12 crosses involving two resistant and six susceptible mungbean genotypes were screened to study the inheritance pattern of MYMV resistance. All the 12  $F_1$ s were susceptible indicating that the susceptibility is dominant over resistance. The  $F_2$ 's of each of the 12 crosses exhibited a segregation ratio of 9S: 3MS: 3MR:1R indicating the involvement of two recessive genes ( $r_1$  and  $r_2$ ) in the inheritance of MYMV resistance. When both genes were present together in a homozygous dominant state,  $R_1, R_2$  plants were highly susceptible (S). In contrast, when both the genes were in homozygous recessive condition,  $r_1r_1r_2r_2$  the resistant reaction was obvious. However, when one gene was in homozygous recessive ( $R_1r_2r_2$ ) condition, moderate susceptibility (MS) was observed while, when the other gene was in homozygous recessive ( $r_1r_1R_2$ ) condition, moderate resistant (MR) reaction was observed. Thus, the  $F_2$  segregation, 9 ( $R_1R_2$ ): 3 ( $R_1r_2r_2$ ): 3 ( $r_1r_1R_2$ ): 1 ( $r_1r_1r_2r_2$ ), was explained on the basis of phenotypic expression, 9S: 3MS: 3MR:1R, which was further confirmed in a back cross generation.

Key words: Mungbean, Inheritance, MYMV, Resistance

#### INTRODUCTION

Mungbean (*Vigna radiata* L. Wilczek) also known as greengram is an indigenous crop of India and is the third most important grain legume. The seeds are cheap and important source of proteins (24%), thus playing an imperative role in alleviating protein malnutrition (Selvi *et al.*, 2006). It is rich in iron (40-70 ppm), easily digestible and with low flatulence. The foliage has importance in feed, fodder and hay. In addition to these health benefits, greengram also has importance in improving soil fertility by fixing the atmospheric nitrogen by *Rhizobium*. Globally, greengram is occupying an area of seven million hectares, yielding up to 3.5 million tons of

grains, especially from Asia (Nair *et al.*, 2019). Worldwide, India is the major producer, accounting for 2.17 million tons of grains from 4.32 million hectares. However, average productivity of 502 kg/ha was obtained, which is low when compared to other legumes. One of the reasons for low productivity is due to infection by MYMV (Mungbean Yellow Mosaic Virus) that drastically reduces the yield.

The earliest report of MYMV was made at Indian Agricultural Research Institute, New Delhi on *Vigna radiata* by Nariani (1960). Besides mungbean, YMV

https://doi.org/10.37992/2021.1203.114

also affects other leguminous crops *viz.*, blackgram (*Vigna mungo*), Lima bean (*P. lunatus*), mothbean (*Vigna aconitifolia*), French bean (*Phaseolus vulgaris*), pigeonpea (*Cajanus cajan*), cowpea (*Vigna unguiculata*), horsegram (*Macrotyloma uniflorum*), Dolichos (*Lablab purpureus*) and soybean (*Glycine max*) (Anjum *et al.*, 2010; Dikshit *et al.*, 2020). The yield penalty ranges from 10 to 100 per cent depending upon the genotype and growth stage of infection by MYMV (Marimuthu *et al.*, 1981).

MYMV is spread by white fly (Bemisia tabaci) an insect vector. Its spread has been reported throughout the world; however, its incidence is heavy in countries like India, Pakistan and Bangladesh (Salam et al., 2011). When a white fly feeds on the cell sap of the host, the virus gains its entry into the phloem cells and the viral aggregates are seen roughly two days prior to the appearance of the symptoms (Thongmeearkom et al., 1981). Early symptoms on leaves are visible as scattered yellow spots, which later turns into a yellow mosaic pattern, ultimately resulting in complete yellowing and senescence of the leaves. Later stage infection (pods), results in a reduction in pod size and photosynthetic efficiency, which is ultimately manifested as a severe yield penalty (Malathi and John, 2009). Since white fly is the vector that rapidly spreads the virus, its population control can reduce the yield losses. Several insecticides are being employed for controlling the vector (white fly), however, it is not an eco-friendly approach. The most effective approach is to develop genetically resistant cultivars.

In order to develop resistant cultivars, the information on the inheritance of resistance to MYMV and its source is very important. In the past, several studies were conducted on the inheritance of resistance to MYMV in greengram by using different resistant sources but the results were contradictory. The inheritance studies revealed that the resistance is controlled by a single recessive gene (Basavaraja et al., 2017; Sai et al., 2017), monogenic dominant gene (Sandhu et al., 1985; Gupta et al., 2005; Lekhi et al., 2018), two recessive genes (Ammavasai et al., 2004; Singh and Singh 2006; Dhole and Reddy, 2012; Alam et al., 2014; Aski et al., 2015; Bhanu et al., 2018) and complementary recessive genes (Mahalingam et al., 2018; Vadivel et al., 2019). The use of different sources of MYMV resistance for genetic studies and infection by different strains of the virus might have led to these conflicting results. Thus, a more extensive study is needed in order to finalize the mode of inheritance of the resistance to MYMV and to help the plant breeders in employing a suitable breeding strategy and selection procedures for developing high yielding and stable MYMV resistant variety. Therefore, the present investigation was carried out to understand the genetics of resistance to MYMV in greengram.

#### MATERIALS AND METHODS

The MYMV reaction was studied in 12 F1 crosses, namely Pusa Vishal x Pusa 0672, Pusa Vishal x HUM 8, LGG 460 x Pusa 0672, LGG 460 x HUM 8, ML 5 x Pusa 0672, ML 5 x HUM 8, ML 717 x HUM 8, ML 717 x Pusa 0672, K 851 x Pusa 0672, K 851 x HUM 8, HUM 12 x Pusa 0672 and HUM 12 x HUM 8) and corresponding  $F_2$ ,  $B_1$  and  $B_2$  along with six MYMV susceptible (Pusa Vishal, LGG 460, ML 5, ML 717, K 851 and HUM 12) and two MYMV resistant (Pusa 0672 and HUM 8) genotypes of greengram.

Six basic populations ( $P_1$ ,  $P_2$ ,  $F_1$ ,  $F_2$ ,  $B_1$  and  $B_2$ ) of 12 crosses were grown one plot each in the compact family block design with three replications at the Agricultural Research Farm, Institute of Agricultural Sciences, Banaras Hindu University, and Varanasi, during *Kharif*, 2016 season. Each plot consisted of a single row of three-meter length with a spacing of 30 and 10 cm between and within rows, respectively.

Under the infector row technique, one row of highly susceptible spreader line, Co 5 (Urdbean variety, highly susceptible to MYMV) was planted after every two rows of the test entries, besides planting two rows of spreader around the experimental sites. The insecticide spray was not applied in order to attract whiteflies for enhancing the infection of MYMV. In addition, artificial inoculation of the individual plant was also done in each of the parents and F<sub>1</sub>s using specially designed insect proof transparent plastic picule pots with screw caps (Nene, 1972). Mass inoculation of 18-20 plants at a time was also done in segregating (F2 and back crosses) progenies, using muslin cloth covered iron cage of 60 × 90 × 120 cm size (Reddy and Singh, 1993). In both cases, viruliferous whiteflies were released inside the cage at the rate of 8-10 flies per plant.

When the infector rows showed > 90 per cent MYMV infection, the individual plants of the parents,  $F_1$ 's,  $F_2$ 's, and back crosses were scored for disease reaction (**Table 1**). The resistant plants did not show any mosaic symptoms on leaves or pods during the entire growth period, while the susceptible plants showed various grades of yellowing depending on the stage at which infection occurred (Singh *et al.*, 1988). There was a wide range of variation in the incidence of disease on the plants of each parent,  $F_1$ ,  $F_2$  and back cross generations. The goodness of fit to the expected ratios in  $F_2$  and Back crosses was tested using the chi-square ( $\chi^2$ ) test.

#### **RESULTS AND DISCUSSION**

All the plants of susceptible parents and 12  $F_1$  hybrids showed highly susceptible reactions (S) whereas, no symptoms could be observed in resistant parents indicating that susceptibility was dominant over resistance. Similar results of dominant behavior of susceptibility in  $F_1$  were also reported by Shukla *et al.* (1978) and Singh and Singh (2006).

Scale	% Of infection	Disease reactions
1	No infection (Completely free)	Resistance
3	Upto10.0% (Trace of necrotic mottle)	Moderately resistant
5	10.1-25.0% foliage is covered with bigger leaf spots	Moderately susceptible
7	25.1% - 40% (Restricted yellow mottle)	Susceptible
9	Above 40% (Completely yellow mottle)	Highly susceptible

The plants in the segregating generation will be grouped into 4 classes, *viz.*, resistance (R) encompassing scale 0 to 3, Moderately resistant (MR) scale 3 to 5, Moderately susceptible (MS) scale 5 to 7 and susceptible (S) including scale 7 to 9.

In F<sub>2</sub> populations, a segregation ratio of 9S: 3MS: 3MR:1R was observed indicating the involvement of two recessive genes in governing the expression of resistance to MYMV in mungbean (Table. 2). In the B, population, (F, x susceptible parents), all the progenies revealed susceptibility as that of F, hybrids whereas, in B<sub>2</sub> (F<sub>1</sub> x resistant parents) population, four categories of segregation ratio of 1S: 1MS: 1MR:1R, were noticed, which further supports that the resistance to MYMV is controlled by digenic recessive genes (Table. 3). Similar reports of digenic recessive inheritance of MYMV was given by Ammavasai et al. (2004), Singh and Singh (2006), Dhole and Reddy (2012), Alam et al. (2014), Aski et al., 2015 and Bhanu et al. (2018). In contrast, Jain et al. (2013), Sudha et al. (2013), Sai et al. (2017) and Raj et al. (2020) reported single recessive gene (3:1) was involved in the inheritance of MYMV resistance in mungbean. On the other hand, Gupta et al. (2005) and lekhi et al. (2018) reported monogenic dominant gene and Mahalingam et al. (2018) and Modha et al. (2018) reported duplicate

recessive inheritance for resistance (15: 1). The probable reasons for such contradictory results in the inheritance of MYMV might be due to differences in the genetic makeup of the resistant genotypes involved in the studies or infection by different strains of the virus or due to the variable interaction between genotype and viral strain. In addition, the environmental factors may also show some influence on the expression of disease and thereby resulting in differences in the inheritance pattern.

Despite meagre work on record regarding inheritance of MYMV resistance, the phenotypic expression of each gene was not studied independently. Consequently, research was attempted to explain the role of each gene on the basis of phenotypic expression and development of disease symptoms. The  $F_2$  population were grouped into four classes based on phenotypic disease development symptoms at different growth stages. On this ground, besides resistant and susceptible, two more reactions were observed as follows: (a) plants showing traces of necrotic

Table 2. Segregation for resistance	to Mungbean Yello	ow Mosaic Virus in F <sub>2</sub>	generation
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	Different reaction of F2 Plants							
Crosses	Number of F2 Plants scored	S	MS	MR	R	χ2 9:3:3:1	P Value	
Pusa Vishal x Pusa 0672	144	78	30	25	11	1.04	0.79	
Pusa Vishal x HUM 8	140	81	22	28	9	0.88	0.83	
LGG 460 x Pusa 0672	145	79	29	26	11	0.67	0.88	
LGG 460 x HUM 8	138	80	24	24	10	0.56	0.90	
ML 5 x Pusa 0672	140	76	29	25	10	0.62	0.89	
ML 5 x HUM 8	135	77	23	24	11	1.08	0.78	
ML 717 x Pusa 0672	142	77	30	25	10	0.77	0.85	
ML 717 x HUM 8	140	77	27	24	12	1.46	0.69	
K 851 x Pusa 0672	138	76	28	23	11	1.18	0.75	
K 851 x HUM 8	142	83	23	26	10	0.77	0.85	
HUM 12 x Pusa 0672	145	80	25	29	11	0.74	0.86	
HUM 12 x HUM 8	140	77	28	24	11	0.93	0.81	

S: Susceptible, MS: Moderately Susceptible, MR: Moderately Resistant, R: Resistant S: Susceptible, MS: Moderately Susceptible, MR: Moderately Resistant, R: Resistant

Table.3. Segregation for resistance to MungbeanYellow Mosaic Virus in Back cross (B<sub>2</sub>) generation involving F<sub>1</sub>s and resistant genotypes

	Different reaction of B2 Plants						
Crosses	Number of B2 Plants scored	S	MS	MR	R	χ2 1:1:1:1	P Value
(Pusa Vishal x Pusa 0672) x Pusa 0672	28	5	8	6	9	1.43	0.69
(Pusa Vishal x HUM 8) x HUM 8	25	4	7	8	6	1.40	0.70
(LGG 460 x Pusa 0672) x Pusa 0672	26	6	7	5	8	0.77	0.85
(LGG 460 x HUM 8) x HUM 8	26	8	5	5	8	1.38	0.71
(ML 5 x Pusa 0672) x Pusa 0672	24	4	7	8	5	1.67	0.64
(ML 5 x HUM 8) x HUM 8	25	5	7	5	8	1.08	0.78
(ML 717 x Pusa 0672) x Pusa 0672	25	5	7	5	8	1.08	0.78
(ML 717 x HUM 8) x HUM 8	26	6	7	5	8	0.77	0.85
(K 851 x Pusa 0672) x Pusa 0672	26	5	8	5	8	1.08	0.78
(K 851 x HUM 8) x HUM 8	28	5	8	6	9	1.43	0.69
(HUM 12 x Pusa 0672) x Pusa 0672	27	8	5	6	8	1.00	0.80
(HUM 12 x HUM 8) x HUM 8	27	8	6	8	5	1.00	0.80

S: Susceptible, MS: Moderately Susceptible, MR: Moderately Resistant, R: Resistant

mottle (Moderately Resistant); (b) foliage is covered with bigger leaf spots (Moderately Susceptible). On the basis of these observations, it is suggested the involvement of two recessives genes  $(r_1 \text{ and } r_2)$  for governing the resistance to MYMV. Based on the segregation ratio in the present piece of investigation, it is concluded that when both dominant genes  $(R_1, R_2)$  were present together, plants were highly susceptible (S) as MYMV symptoms appeared on both leaves as well as pods. When one gene was homozygous recessive,  $R_{1\text{-}}\,r_{\text{2r2,}}$  the bigger leaf spots on foliage (MS) appeared, while, the other gene was in homozygous recessive, r1r1, R2 condition, minor traces of necrotic mottle (MR) was observed. However, highly resistant plants exhibited no symptoms on either leaves or pods when both recessive genes are present together in homozygous  $(r_1r_1, r_2r_2)$  conditions showing complementary gene action and thus resulted in a highly resistant phenotype. The  $F_2$  segregation, 9 ( $R_1R_2$ ) : 3  $(R_1 r_2 r_2) : 3 (r_1 r_1 R_2) : 1 (r_1 r_1 r_2 r_2)$ , was explained on the basis of phenotypic expression, 9S: 3MS: 3MR:1R, which was further confirmed by back cross generation as1S: 1MS: 1MR:1R.

Since two recessive genes were involved in governing resistance to MYMV in the two resistant donors (Pusa 0672 and HUM 8) studied in the present case, it is suggested that in resistance breeding programmes, a large number of segregating populations should be raised involving aforesaid resistant donors to recover enough resistant plants coupled with other desirable traits.

#### ACKNOWLEDGEMENT

The author is very grateful to the Department of Genetics and Plant Breeding, Institute of Agricultural Sciences,

Banaras Hindu University, Varanasi, India for providing facilities to conduct this experiment.

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