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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_1s 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

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_1s 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 (F_2 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 (χ^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).

Table 1. Disease rating for MYMV on a 1 to 9 scale (Singh *et al*., 1988)

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_2 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_1 population, (F_1 x susceptible parents), all the progenies revealed susceptibility as that of F_1 hybrids whereas, in B_2 (F_1 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 Yellow Mosaic Virus in F_2 generation

Crosses	Different reaction of F2 Plants						
	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_2) generation involving F_1s and resistant genotypes

Crosses	Different reaction of B2 Plants						
	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 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, $R_{1-} r_{2r2,}$ the bigger leaf spots on foliage (MS) appeared, while, the other gene was in homozygous recessive, $r_1 r_{1,} R_{2-}$ 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 ($r_1 r_{1,} r_2 r_{2)}$ conditions showing complementary gene action and thus resulted in a highly resistant phenotype. The F_2 segregation, 9 ($R_{1,} R_2$) : 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.

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REFERENCES

Alam, A.M., Somta, P., Jompuk, C., Chatwachirawong, P. and Srinives, P. 2014. Evaluation of mungbean genotypes based on yield stability and reaction to mungbean yellow mosaic virus disease. *The Plant Pathology Journal*, **30**(3): 261-268. [Cross Ref]

Ammavasai, S., Phogal, D. S. and Solanki, I. S. 2004. Inheritance of resistance to mungbean yellow mosaic virus (MYMV) in greengram (*Vigna radiata* L. Wilczek). *Indian J. Genet*. **64**: 146.

Anjum, K.T., Gupta, S. and Datta, S. 2010. Mapping of Mungbean Yellow Mosaic India Virus (MYMIV) and powdery mildew resistant gene in black gram [*Vigna mungo* (L.) Hepper]. *Electronic Journal of Plant Breeding*, **1**(4): 1148-1152.

Basavaraja, T., Niranjana, M., Shashi, P. K. and Satheesh Naik, S. J. 2017. Inheritance of resistance to Mungbean Yellow Mosaic Virus (MYMV) in intra and inter-specific crosses of *Vigna*. *Journal of Food Legumes*, **30**(1): 15-19.

Bhanu, A. N., Singh, M.N. and Srivastava, K. 2018. Genetic analysis of gene-specific resistance to mungbean yellow mosaic virus in mungbean (*Vigna radiata*). *Plant Breeding.* **138**(2):202-206. [Cross Ref]

Dhole, V.J. and Reddy, K. S. 2012. Genetic analysis of resistance to mungbean yellow mosaic virus in

mungbean (*Vigna radiata*). *Plant Breeding.* **131**(3): 414-417. [Cross Ref]

Dikshit, H. K., Mishra, G. P., Somta, P., Shwe, T., Alam, A. and Bains, T. S. 2020. "Classical genetics and traditional breeding in ungbean," in The Mungbean Genome, Compendium of Plant Genomes. *Springer Nature*. 43–54. [Cross Ref]

Gupta, S., Kumar, S., Singh, R. A. and Chandra, S. 2005. Identification of a single dominant gene for resistance to Mungbean Yellow Mosaic Virus in blackgram (*Vigna mungo* (L.) Hepper). *SABRAO Journal of Breeding and Genetics*. **37**(2):85-9.

Jain, R. A., Lavanya, G. R., Reddy, P. A. and Babu, G. S. 2013. Genetic inheritance of yellow mosaic virus resistance in mungbean [*Vigna radiata* (L.) Wilczek]. *Trends Biosci.* **6**: 305-6.

Lekhi, P., Gill, R. K., Kunal. and Kaur, S. 2018. Identification of molecular marker linked to mungbean Yellow mosaic virus (MYMV) resistance in *Vigna radiata* (L.) Wilczek. *Electronic Journal of Plant Breeding,* **9** (3): 839-845. [Cross Ref]

Mahalingam, A., Satya, V. K., Manivannan, N., Narayanan, S. L. and Sathya, P. 2018. Inheritance of Mungbean Yellow Mosaic Virus Disease Resistance in Greengram (*Vigna radiata* L. Wilczek). *International Journal of Current Microbiology and Applied Sciences,* **7**(1): 880-885. [Cross Ref]

Malathi, V. C. and John, P. 2009. "Mungbean yellow mosaic viruses," in Desk encyclopedia of plant and fungal virology. Eds. M. Van Regenmortal and B. Mahy (*London: Academic press*), 217–226. [Cross Ref]

Marimuthu, T., Subramanian, C. L. and Mohan, R. 1981. Assessment of yield losses due to yellow mosaic infection in mungbean. *Pulse Crop Newsl*. 1: 104.

Modha, K., Patel, P., Sen, S., Shiv, A., Dhoot, R. and Patel, U. 2018. Epistatic Basis of Mosaic Resistance in Mungbean [*Vigna radiata* (L.) Wilczek]. *Int. J. Curr. Microbiol. App. Sci.,* **7**(4):3265-71. [Cross Ref]

Aski, M. S., Dikshit, H. K., Singh, D., Singh, A. and Prapti, P. 2015. Inheritance of resistance to Mungbean Yellow Mosaic India Virus (MYMIV) in mungbean (*Vigna radiata*). *Indian Journal of Agricultural Sciences.,* **85**(9):1144-7.

Nair, R. M., Pandey, A. K., War, A. R., Hanumantharao, B., Shwe, T. and Alam, A. 2019. Biotic and abiotic constraints in mungbean production-progress in genetic improvement. *Front. Plant Sci.,* **10**: 1340. [Cross Ref]

Nariani, T. K. 1960. Yellow mosaic of Mung (*Phaseolus aureus* L). *Indian Phytopath*., **13** (1): 24-29.

Nene, Y. L. 1972. A survey of viral diseases of pulse crops in Uttara Pradesh. A survey of viral diseases of pulse crops in Uttara Pradesh. **87**: 1-191.

Raj, K. R., Reshmi., Baisakh, B., Tripathy, S. K., Devraj, L. and Pradhan, B. 2020. Studies on inheritance of MYMV resistance in greengram [*Vigna radiata* (L.) Wilczek]. *Research Journal of Biotechnology.,* **15**:3.

Reddy, K. R. and Singh, D. P. 1993. Inheritance of resistance to mungbean yellow mosaic virus. *Madras Agricultural Journal,.* **80** (4):199-201.

Sai, C. B., Nagarajan, P., Raveendran, M., Rabindran, R. and Senthil, N. 2017. Understanding the inheritance of mungbean yellow mosaic virus (MYMV) resistance in mungbean (*Vigna radiata* L. Wilczek). *Molecular breeding*., **37**(5):1-5. [Cross Ref]

Salam, S. A., Patil, M. S. and Byadgi, A. S. 2011. Status of mungbean yellow mosaic virus disease incidence on green gram. *Karnataka J. Agric. Sci.,* **24** (2): 247–248.

Sandhu, T. S., Brar, J. S., Sandhu, S. S. and Verma, M. M. 1985. Inheritance of resistance to mungbean yellow mosaic virus in greengram. *J. Res. Punjab Agric. Univ*., **22**: 607-611.

Selvi, R., Muthiah, A. R. and Manivannan, N. 2006. Tagging of RAPD marker for MYMV resistance in mungbean (*Vigna radiata* (L.) Wilczek). *Asian J. Plant Sci.,* **5**: 277–280. [Cross Ref]

Shukla, G.P., Pandya, B.P. and Singh, D. P. 1978. Inheritance of resistance to yellow mosaic in

mungbean. *Indian J. Gene*., **38**: 357-360.

Singh, G., Kapoor, S., Singh, K. 1988. Multiple disease resistance in mungbean with special emphasis on Mungbean Yellow Mosaic Virus. In Mungbean Proceedings of the Second International Symposium held at Bangkok, Thailand, 16- 20 November 1987. AVRDC: 290-296.

Singh, S. K. and Singh, M. N. 2006. Inheritance of resistance to mungbean yellow mosaic virus in mungbean. *Indian Journal of Pulses research.,***19** (1): 21-23.

Sudha, M., Karthikeyan, A., Anusuya, P., Ganesh, N. M., Pandiyan, M., Senthil, N., Raveendran, M., Nagarajan, P. and Angappan, K. 2013. Inheritance of resistance to mungbean yellow mosaic virus (MYMV) in inter and intra specific crosses of mungbean (*Vigna radiata*). *American Journal of Plant Sciences.,* **4**: 1924-1927. [Cross Ref]

Thongmeearkom, P., Honda, Y., Saito, Y. and Syamananda, R. 1981. Nuclear ultra structural changes and

aggregates of virus like particles in mungbean cells affected by mungbean yellow mosaic disease. *Phytopathol*., **71**: 41–44. [Cross Ref]

Vadivel, K., Manivannan, N., Mahalingam, A., Satya, V. K., Vanniarajan, C. and Saminathan, V. R. 2019. Generation mean analysis for yield, yield components and mymv disease scores in blackgram [*Vigna mungo* (L). Hepper]. *Int. J. Curr. Microbiol. App. Sci*., **8**(5):1989-95. [Cross Ref]