

INHERITANCE STUDY OF RESISTANCE TO MUNGBEAN YELLOW MOSAIC VIRUS DISEASE IN MUNGBEAN (*Vigna radiata* L. Wilczek)

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ABSTRACT

The objectives of this study were screening of 50 genotypes of mungbean [*Vigna radiata* (L.) Wilczek] against MYMV disease during two seasons of *kharif* 2015 and *rabi* 2015-16 under natural field condition at Research cum Instructional Farm, College of Agriculture, Indira Gandhi Krishi Vishwavidyalaya, Raipur, Chhattisgarh and to determine the inheritance of MYMV resistance in F₁ and F₂ generation and to identify the role of each resistance gene. During *kharif* season 2015 two genotypes *i.e.* AKM 1210 and Samrat were found to be highly resistant against MYMV disease, 12 genotypes were resistant, 15 genotypes were moderately resistant, eight genotypes were moderately susceptible, 11 genotypes were susceptible and two genotypes were highly susceptible. Same set of 50 genotypes screened during *rabi* 2015-16 revealed that none of the entries were highly resistant, 11 genotypes to be resistant, nine genotypes were moderately resistant, 12 were moderately susceptible, 14 genotypes were susceptible and four genotypes showed highly susceptible reaction against MYMV disease indicating the role of weather conditions in expression of disease. Six crosses between resistant and susceptible genotypes were attempted. Goodness of fit test pertaining to MYMV resistance confirmed that all the F₁ cross combinations *viz.*, IGKM -08 x TM 96-2, Ling MS Thung M-1 x HUM -12, TM 2000-1 x PUSA 9072 and SGC -20 x Malviya Jyoti were resistance to MYMV clearly indicated the resistance nature was dominant over susceptibility, while the F₁s of crosses DDGV 2 x IPM 409-4 and IGKM -0642 x PUSA 1471 were susceptible to MYMV, indicating that resistance is recessive. Thus the investigation revealed that MYMV resistance was digenically controlled in the five crosses with various types of interaction *i.e.*, duplicate interaction (IGKM 0642 x PUSA 1471, F₂ having two recessive genes in complementation for resistance), complementary interaction (IGKM-08 x TM 96-2 and TM 2000-1 x PUSA 9072, F₂ having two complementary dominant genes for resistance) and inhibitory interaction (DGGV-2 x IPM 409-4, F₂ having one recessive and one dominant gene in complementation and Ling MS Thung M-1 x HUM 12, F₂ having two independent genes one dominant gene and other recessive gene for resistance) and monogenic controlled in cross SGC 20 x Malviya Jyoti, F₂ having one independent dominant gene for resistance.

KEYWORDS: Gene, Inheritance, Mungbean, Mungbean Yellow Mosaic Virus, Resistance, Screening, Susceptible.

INTRODUCTION

Mungbean [*Vigna radiata* (L.) Wilczek] is an ancient and well known pulse crop of Asian countries. It is one of the thirteen food legumes grown in India and third most important pulse crop of India after chickpea and pigeonpea. It is a rich source of essential amino acid lysine, proteins (22-28%), 60-65% carbohydrates, 1-1.5 % fat, 3.5-4.5% fibers, minerals and vitamins providing higher calories. Mungbean is easy to cook and easily digestible. Mungbean sprouts can provide additional iron which is deficient in South Asian diets. Mungbean also improves soil fertility through symbiotic nitrogen (30-74 kg/ ha) fixation (Malik 1994). Historically, India has been the largest global producer and consumer of mungbean. However, productivity in the country is still low and there is a need for improvement.

In Indian sub-continent Mungbean Yellow Mosaic Virus (MYMV) is one of the serious diseases common to many leguminous crops and causes tremendous losses in production of mungbean crop (Chenulu and Verma 1988). Among the various diseases limiting mungbean productivity, mungbean yellow mosaic virus (MYMV) is the most destructive and widely distributed, causing 10-100% yield losses depending on the crop stage at which the plants becoming infected (Nene 1972, Chenulu *et al.*, 1979, Marimuthu *et al.*, 1981). First of all this disease was reported in 1955 on mungbean crop growing at the Indian Agricultural Research Institute (IARI), Delhi (Nariani 1960). This disease has been reported to be transmitted by an insect vector white fly (*Bemisia tabaci* Genn.) and not by seed, soil or mechanical inoculation (Ahmad and Harwood 1973; Nair and Nene 1973). The

control of yellow mosaic disease is often based on limiting the vector population by using insecticides, which are 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 mungbean. Use of resistant genotypes is the most effective alternative to mitigate the yield loss.

Knowledge of the mode of inheritance to MYMV in mungbean is useful for incorporation of resistance into agronomically poor, but desirable genetic resources. MYMV control is often based on limiting the vector population with insecticides, which are ineffective under severe whitefly infestations. The use of host resistance is the most efficient and ecologically friendly approach to reduce MYMV damage in mungbean. For breeding resistant cultivars, information on the inheritance of resistance genes and its source is very important. Several resistance sources have been reported for MYMV disease. The monogenic recessive inheritance for MYMV has been reported by Singh and Patel (1977), Malik *et al.* (1986 and 1988), Malik (1992), Saleem *et al.* (1998) and Khattak *et al.* (2000) a dominant gene (Sandhu *et al.*, 1985), two recessive genes (Verma and Singh 1988, Pal *et al.*, 1991, Amavasai *et al.*, 2004) and complementary recessive genes (Shukla and Pandya 1985). The present investigation was carried out to understand the mode of inheritance of resistance to MYMV in mungbean.

MATERIALS AND METHODS

Screening for MYMV disease resistance

The 50 genotypes of mungbean (Table-1) were evaluated against MYMV disease under natural field condition during *kharif* 2015 and *rabi* 2015-16 (Fig-1 and 2). The seed of these genotypes were procured from IIPR, Kanpur and MULLaRP, IGKV, Raipur. Each genotype was represented by single row of 4.0 m length with three replications at row spacing of 30 cm and 5-10 cm between plants. General cultural practices were followed to maintain the experiment except that no insecticides were sprayed to discourage whitefly population for spread of the disease. After emergence, the crop was regularly observed for development of yellow mosaic disease. Diseases severity was recorded at weekly intervals using 0-5 rating scoring Scale (Bashir 2005) (Table-2). Plants were exposed to natural infection. Per cent disease incidence (PDI) of MYMV disease (Table-3) in each genotype/ variety was recorded by counting the number of infected plants out of the total number of plants.

Development of F₁s and evaluation of F₂ population

The present investigation was conducted at Research cum Instructional Farm, College of Agriculture, Indira Gandhi Krishi Vishwavidyalaya, Raipur, Chhattisgarh during *rabi* 2015-16 and *kharif* 2016. This study was planned to determine the mode of inheritance of resistance in six selected genotypes *viz.*, IPM 409-4, TM 96-2, HUM -12, PUSA 1471, PUSA 9072 and Malviya Jyoti. These six resistant genotypes were crossed with susceptible genotypes *viz.*, DDGV -2, IGKM -08, Ling MS Thung M-1, IGKM -0642, TM 2000-1 and SGC -20 respectively. A total of six crosses (F₁s) were made during *rabi* 2015-16 *viz.*, DDGV 2 x IPM 409-4, IGKM -08 x TM 96-2, Ling MS Thung M-1 x HUM -12, IGKM -0642 x PUSA 1471, TM 2000-1 x PUSA 9072 and SGC -20 x Malviya Jyoti to build up F₂ generations for scored yellow mosaic disease during *kharif*, 2016. The plants showing yellow in mosaic symptoms were considered 'susceptible' failing which were considered 'resistant'. The goodness of fit to Mendelian segregation ratio has been tested in the F₂ segregating population by Chi-square test (Harnett 1970).

RESULTS AND DISCUSSION

The 50 genotypes of mungbean were evaluated against MYMV disease under natural environmental condition during *kharif* 2015 and *rabi* 2015-16. The seed of these genotypes were procured from IIPR, Kanpur and MULLaRP, IGKV, Raipur. Each genotype was represented by single row of 4.0 m length with three replications at row spacing of 30 cm and 5-10 cm between plants. General cultural practices were followed to maintain the experiment except that no insecticides were sprayed to discourage whitefly population for spread of the disease. After emergence, the crop was regularly observed for development of yellow mosaic disease. Diseases severity was recorded at weekly intervals using 0-5 rating scoring Scale (Bashir 2005). Plants were exposed to natural infection. Per cent disease incidence (PDI) of MYMV disease in each genotype/ variety was calculated based on disease scale and ratings.

Among all the 50 genotypes of mungbean evaluated under natural field condition during *kharif* 2015, two genotypes *viz.*, AKM 1210 and Samrat were found to be highly resistant against yellow mosaic virus disease with score of zero, 12 genotypes *viz.*, SML 1815, Pairy Mung, HUM 12, KM 2328, IPM 409-4, MH 421, MH 934, RMG 1030, VBN (Gg) 2, VGG 10-008, LGG 544 and CO 6 exhibited resistant reaction with score of one, 15 genotypes *viz.*, LGG 460, HUM 16, Pusa Visal, Malviya Jyoti, Beliya, TM 96-2, Pragya, PKV AKM 4, PUSA 1471, DGG 3, COGG 11-02, PUSA 9072, PUSA 15161, IPM 16 and IPM 19 showed moderate resistant reaction with score of two, eight genotypes *viz.*, IGKM 08, Ling MS Thung M-1, BM 4, VGG 05-006, PM 9-11, SGC 20, IPM 5-07 and Meha were found to be moderately susceptible, 11 genotypes showed susceptible reaction *viz.*, HUM 27, KM 2342, MH 810, NVL 641, ML 2056, IGKM 0642, TARM 1, TM 2000-1, RM 03-

$$\text{Percent Disease Incidence (PDI)} = \frac{\text{Number of infected plants}}{\text{Total number of plants observed}} \times 100$$

71, RM 03-79 and Pratiksha and two genotype DGGV 2 and RMG 1028 were highly susceptible against yellow mosaic virus disease.

Same set of 50 genotypes of mungbean was further screened during *rabi* 2015-16. Among all the genotypes none of them was found to be highly resistant, 11 genotypes *viz.*, SML 1815, Pairy Mung, HUM 12, KM 2328, IPM 409-4, MH 421, RMG 1030, AKM 1210, VBN (Gg) 2, VGG 10-008 and Samrat revealed resistant reaction having range of 1-10 per cent infection against MYMV disease, nine genotypes *viz.*, LGG 460, TM 96-2, MH 934, PKV AKM 4, PUSA 1471, PUSA 9072, PUSA 15161, IPM 16 and IPM 19 were moderately resistant having range of 11-20 per cent infection against MYMV disease, 12 genotypes *viz.*, HUM 16, Pusa Visal, Malviya Jyoti, Beliya, Ling MS Thung M-1, Pragya, VGG 05-006, DGG 3, COGG 11-02, CO 6, SGC 20 and IPM 5-07 were moderately susceptible having range of 21-30 per cent infection against MYMV disease, 14 genotypes *viz.*, IGKM-08, BM 4, PM 9-11, HUM 27, KM 2342, ML 2056, LGG 544, IGKM 0642, TARM 1, TM 2000-1, RM 03-71, RM 03-79, Meha and Pratiksha were susceptible having range of 31-50 per cent infection against MYMV disease and rest of the four genotypes *viz.*, DGGV 2, RMG 1028, MH 810 and NVL 641 were found as highly susceptible with >50 % infection against yellow mosaic virus disease in mungbean.

The resistant and highly susceptible genotypes could be utilized as parents for the development of mapping population to validate the molecular markers which can facilitate marker-assisted selection (MAS) for the development of MYMV resistant breeding lines for future crop improvement programmes. Earlier many researchers have screened mungbean germplasm against the MYMV disease and reported similar findings. Ahmad *et al.* (2017) identified six genotypes, Suman *et al.* (2015) screened two genotypes, Iqbal *et al.* (2011) found four lines and Salam *et al.* (2009) reported three genotypes resistant to the MYMV disease in mungbean. On the other hand, Habib *et al.* (2007) and Shad *et al.* (2006) screened mungbean germplasm and reported no resistance sources.

To decipher the inheritance pattern of MYMV resistance six mungbean cross combinations were evaluated and Chi-square test was worked out to ascertain the expected deviation from the Mendelian segregation ratio of segregating generation (F_2) and the results are presented in Table-5. All the available information pertaining to MYMV resistance confirmed that the F_1 of all the crosses *viz.*, IGKM -08 x TM 96-2, Ling MS, Thung M-1 x HUM -12, TM 2000-1 x PUSA 9072 and SGC -20 x Malviya Jyoti were resistance to MYMV clearly indicated the resistance nature of all the F_1 of each cross combination was dominant over susceptibility, while the F_1 s of crosses DGGV 2 x IPM 409-4 and IGKM -0642 x PUSA 1471 were susceptible to MYMV, indicating that resistance is recessive.

The cross IGKM -0642 x PUSA 1471, the F_1 shown susceptible reaction depicting recessive nature of disease reaction in PUSA 1471. The F_2 segregation ratio of 1 resistant: 15 susceptible shows that resistance is governed by two recessive gene in complementation. Similar results have been shown by Shukla *et al.* (1978), Singh (1980), Verma and Singh (1989), Murugan and Nadarajan (2011), Prasad *et al.* (2015) and Thamodhran *et al.* (2016).

The F_1 of the crosses IGKM -08 x TM 96-2 and TM 2000-1 x PUSA 9072 were resistant, indicating that resistance is dominant and the F_2 segregation for MYMV appeared to be governed by digenic complementary epistasis as seen from the ratio of 9:7 (resistant: susceptible). The digenic complementary epistasis reaction for resistance to MYMV disease in mungbean is also reported by Verma and Singh (1980), Sandhu *et al.* (1985), Shukla and Pandya (1985), Verma and Singh (1988), Amavasai *et al.* (2004), Singh and Singh (2006), Murugan and Nadarajan (2011), Prasad *et al.* (2015) and Thamodhran *et al.* (2016).

The F_1 of cross DGGV-2 x IPM 409-4 was susceptible showing recessive resistance reaction in IPM 409-4 is governed by recessive gene. The F_2 segregation ratio of 3 resistant: 13 susceptible reaction reveals presence of digenic inhibitory epistasis where in resistance is governed by recessive gene in presence of complementary dominant gene in other locus. Similar results were shown by Solanki *et al.* (1982), Verma and Singh (1986) and Prasad *et al.* (2015).

Similarly F_1 of cross Ling MS Thung M-1 x HUM -12 was resistance showing dominance nature resistance reaction. Further the F_2 segregation ratio of 13 resistant: 3 susceptible reveal digenic reaction where in resistance is governed in HUM -12 by two independent dominated recessive genes in other locus. Similar results have been shown by Reddy and Singh (1993), Murugan and Nadarajan (2011), Prasad *et al.* (2015).

In cross SGC -20 x Malviya Jyoti, resistant reaction in F_1 indicated that resistance is dominant and the F_2 segregated in 3 resistant: 1 susceptible ratio showing monogenic dominance reaction for resistance to MYMV. The monogenic dominance reaction for resistance to MYMV disease in mungbean is also reported by Singh and Patel (1977), Malik *et al.* (1986), Malik (1992), Saleem *et al.* (1998), Khattak *et al.* (2000), Dhole and Reddy (2012), Yogeesh *et al.* (2012), Sudha *et al.* (2013) and Vinoth and Jayamani (2014).

The female plants in the crosses were susceptible and their F_1 were always highly resistant. Resistance was therefore, transferred through the pollen and was not cytoplasmically inherited. The dominant gene for MYMV resistance is inherited through the nuclear genome.

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Table: 1. Details of experimental material used

S.No.	Genotypes	Pedigree	Source
1	LGG -460	Lam M2 x ML 267	RARS, Lam
2	SML -1815	-	PAU, Ludhiana
3	DGGV -2	Chninamung x TM 98-50	MARS, Dharwad
4	HUM -16	Pusa bold-1 x HUM 8	BHU, Varansi
5	PaityMung	TARM 1 x J 781	IGKV, Raipur
6	MalviyaJyoti	BHUM 1 x Pant U 30	BHU, Varansi
7	Pusa Vishal	Selection from NM 92	IARI, New Delhi
8	HUM -12	HUM 5 x DPM 90-1	BHU, Varansi
9	Beliya	Sel. from Deobhoggermplasm	IGKV, Raipur
10	KM -2328	KM 2241 x HUM 16	CSA, Kanpur
11	IPM 409-4	PDM 288 x IPM 03-1	IIPR, Kanpur
12	MH -421	Muskan x BDYR 2	CCS HAU, Hisar
13	IGKM -08	Selection from Bastargermplasm	IGKV, Raipur
14	TM 96-2	Kopergaon x TARM-2	BARC, Mumbai
15	RMG -1028	MUM 2 x RMG 62	ARS Durgapur
16	Ling MS Thung M-1	-	China
17	Pragya	Selection from germplasm	IGKV, Raipur
18	MH -934	MH 96-1 x 2 KM 117	CCS HAU, Hisar
19	BM -4	Mutant of T 44	ARS, Badnapur
20	RMG -1030	RMG 275 x RMG 492	ARS, Durgapur
21	PKV AKM 4	BM 4 x PS 7	PDKV, Akola
22	PUSA 1471	-	IARI, New Delhi
23	VGG 05-006	VRM 1 x (Gg) x Vellore Local	Vamban
24	PM 9 -11	PM 5 x BinaMung	Pantnagar
25	HUM -27	Selection from HUM-12	BHU, Varansi
26	KM -2342	KM 132 x KM 2241	CSA, Kanpur
27	AKM 1210	AKM 9911 x BM 2003-2	PDKV, Akola
28	MH -810	MH 96-1 x 2 KM 114	CCS HAU, Hisar
29	NVL- 641	NVL-605 x NVL- 501	Pachora
30	DGG -3	GG 4 x TM 98-50	UAS, Dharwad
31	ML 2056	ML 1165 x ML 1191	Ludhiana
32	SGC -20	AAU 34 x T-44	Shillongani
33	IPM 5-07	EC 398885 x PDM 139	IIPR, Kanpur

34	VBN (Gg) 2	VGG 4 x MH 309	NPRC, Vamban
35	COGG 11-02	COGG 912 x IPM 02-3	Coimbatore
36	VGG 10-008	PDM 139 x BB 2664	NPRC, Vamban
37	PUSA 9072	PUSA 106 x 10-215	IARI, New Delhi
38	LGG -544	TM 96-2 x AKM 8802	Lam
39	CO 6	VGG 37 x CO 5	Coimbatore
40	IGKM -0642	BELIYA x TARM 1	IGKV, Raipur
41	TARM -1	RUM 5 x TARM 18	BARC, Mumbai

S.No.	Genotypes	Pedigree	Source
42	PUSA 15161	PDM 96-267 x PUSARATNA	IARI, New Delhi
43	TM-2000-1	TARM 1 x J 781	BARC, Mumbai
44	RM-03-71	TM 96-2 x VC 6370-71	IGKV, Raipur
45	RM-03-79	TM 96-2 x VC 6370-79	IGKV, Raipur
46	Meha	PM 3 x APM 36	IIPR, Kanpur
47	Pratiksha	VC6370-92 x (VC2768A x V6601)	Nepal
48	IPM-16	PDM-139 x EC 398884	IIPR, Kanpur
49	IPM-19	(PM-3 x AMP-36) x EC 398897	IIPR, Kanpur
50	Samrat	ML 20/19 x ML 5	PAU, Ludhiana

Table: 2. Mungbean yellow mosaic virus disease scoring (0 to 5 scale) (Bashir 2005)

Score	Disease infection	Reaction
0	0	Highly Resistant (HR)
1	1-10	Resistant (R)
2	11-20	Moderately Resistant (MR)
3	21-30	Moderately Susceptible (MS)
4	31-50	Susceptible (S)
5	> 50	Highly Susceptible (HS)

Table: 3. Mungbean yellow mosaic virus disease scores of different seasons

S.No.	Genotypes	Disease Score		
		<i>kharif</i> 2015	<i>rabi</i> 2015-16	Mean
1	LGG -460	2.0	2.0	2.0
2	SML -1815	1.0	1.0	1.0
3	DGGV -2	5.0	5.0	5.0
4	HUM -16	2.0	3.0	2.5
5	Pairy Mung	1.0	1.0	1.0
6	Malviya Jyoti	2.0	3.0	2.5
7	Pusa Vishal	2.0	3.0	2.5
8	HUM -12	1.0	1.0	1.0
9	Beliya	2.0	3.0	2.5
10	KM 2328	1.0	1.0	1.0
11	IPM 409-4	1.0	1.0	1.0
12	MH -421	1.0	1.0	1.0
13	IGKM -08	3.0	4.0	3.5
14	TM 96-2	2.0	2.0	2.0
15	RMG -1028	5.0	5.0	5.0
16	Ling MS Thung M-1	3.0	3.0	3.0
17	Pragya	2.0	3.0	2.5
18	MH -934	1.0	2.0	1.5
19	BM 4	3.0	4.0	3.5
20	RMG -1030	1.0	1.0	1.0
21	PKV AKM 4	2.0	2.0	2.0
22	PUSA 1471	2.0	2.0	2.0
23	VGG 05-006	3.0	3.0	3.0
24	PM 9-11	3.0	4.0	3.5
25	HUM -27	4.0	4.0	4.0
26	KM 2342	4.0	4.0	4.0
27	AKM 1210	0.0	1.0	0.5
28	MH -810	4.0	5.0	4.5
29	NVL -641	4.0	5.0	4.5

30	DGG -3	2.0	3.0	2.5
31	ML -2056	4.0	4.0	4.0
32	SGC -20	3.0	3.0	3.0
33	IPM 5 -07	3.0	3.0	3.0
34	VBN (Gg) 2	1.0	1.0	1.0
35	COGG 11-02	2.0	3.0	2.5
36	VGG 10-008	1.0	1.0	1.0
37	PUSA 9072	2.0	2.0	2.0
38	LGG -544	1.0	4.0	2.5
39	CO 6	1.0	3.0	2.0
40	IGKM -0642	4.0	4.0	4.0
41	TARM -1	4.0	4.0	4.0
42	PUSA 15161	2.0	2.0	2.0
43	TM 2000-1	4.0	4.0	4.0
44	RM 03-71	4.0	4.0	4.0
45	RM 03-79	4.0	4.0	4.0
46	Meha	3.0	4.0	3.5
47	Pratiksha	4.0	4.0	4.0
48	IPM -16	2.0	2.0	2.0
49	IPM -19	2.0	2.0	2.0
50	Samrat	0.0	1.0	0.5

Table: 4. Details of the parents used in the inheritance study

S.N	Genotypes	Parentage	Place of origin	MYMV Reaction
1	DGGV-2	Chninamung × TM 98-50	MARS, Dharwad	HS
2	Malviya Jyoti	BHUM 1 x Pant U 30	BHU, Varansi	MR
3	HUM -12	HUM 5 x DPM 90-1	BHU, Varansi	R
4	IPM 409-4	PDM 288 x IPM 03-1	IIPR, Kanpur	R
5	IGKM -08	Selection from Bastar germplasm	IGKV, Raipur	MS
6	TM 96-2	Kopergaon x TARM-2	BARC, Mumbai	MR
7	Ling MS Thung M-1	-	China	MS
8	PUSA 1471	-	IARI, New Delhi	MR
9	SGC -20	AAU 34 x T-44	Shillongani	MS
10	PUSA 9072	PUSA 106 x 10-215	IARI, New Delhi	MR
11	IGKM -0642	BELIYA x TARM 1	IGKV, Raipur	S
12	TM-2000-1	TARM 1 × J 781	BARC, Mumbai	S

Table: 5. MYMV reaction in F₁ and F₂ generations derived from cross of susceptible x resistant genotypes

S.N	Crosses	Reaction of F ₁	Reaction of F ₂ population		Expected ratio	Calculated X ²	X ² t _{5%}
			Resistant	Susceptible			
1	DGGV-2 x IPM 409-4	S	61	250	3:13	0.152	3.84
2	IGKM-08 x TM 96-2	R	252	173	9:7	1.600	3.84
3	Ling MS Thung M-1 x HUM 12	R	300	73	13:3	0.165	3.84
4	IGKM 0642 x PUSA 1471	S	26	281	1:15	2.580	3.84
5	TM 2000-1 x PUSA 9072	R	178	146	9:7	0.226	3.84
6	SGC 20 x Malviya Jyoti	R	291	111	3:1	1.463	3.84

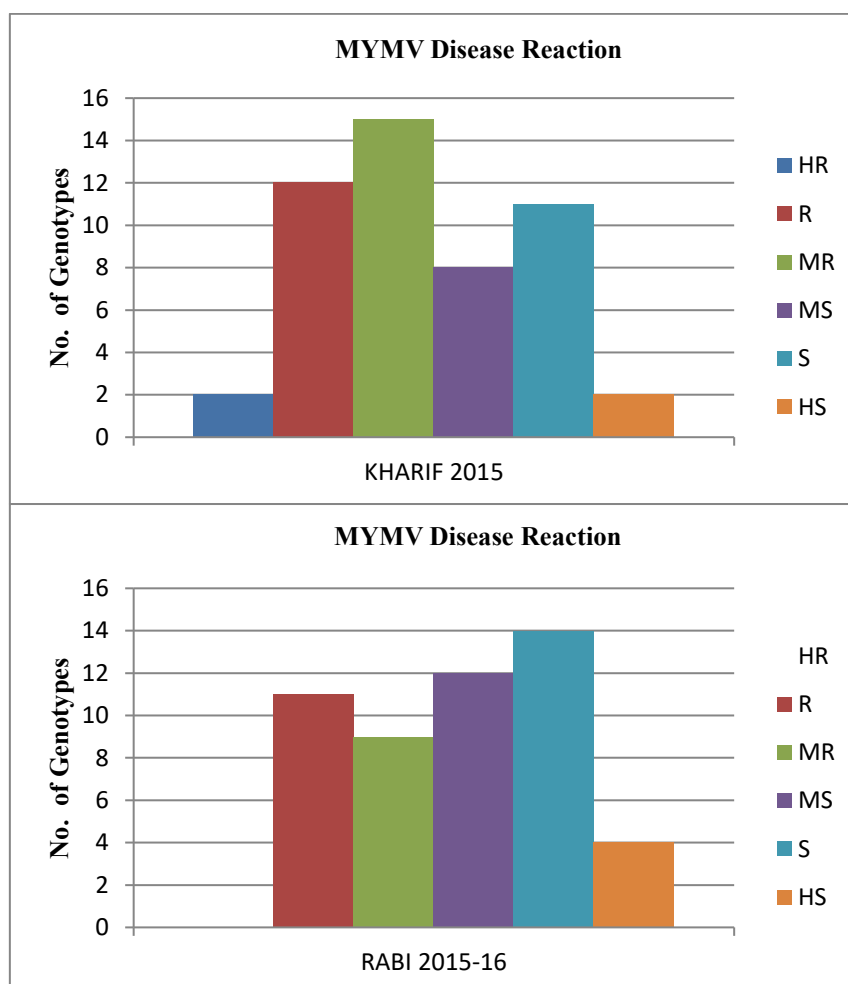


Fig: 1 and 2. 50 genotypes of mungbean categorized in six classes for their reaction to MYMV under natural conditions during *kharif* 2015 and *rabi* 2015-16