



Inheritance and Gene Effect of Resistance to Mungbean Yellow Mosaic Virus (MYMV) in Mungbean [*Vigna radiata* (L.) Wilczek]

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ABSTRACT

Background: Mungbean Yellow Mosaic Virus is one of the major constraints in mungbean production. Knowledge of mode of inheritance and gene effects of MYMV resistance is very useful and effective for the development of genotypes resistant to disease or incorporation of resistance into the desirable promising genotypes which lack of disease resistance.

Methods: In order to estimate inheritance pattern of MYMV disease resistance in mungbean, the study was conducted in summer season (2019) under natural condition. Six generations such as P₁, P₂, F₁, BC₁, BC₂ and F₂ of six combinations [two resistant genotypes (7639 and 10266) and three susceptible genotypes (7621, 10257 and R-021018)] were studied inheritance pattern of resistance to MYMV in segregation population.

Result: Based on the result from mode of inheritance pattern of MYMV resistance, it can be concluded that single recessive gene is controlled the resistance of MYMV and susceptible behavior indicated as dominant over resistant. Additive gene action was the major role for the selection of MYMV resistance. Some differences in the expression of gene contributing for MYMV resistance from others findings might be attributed due to the sources of resistant genotypes which have different nature of resistant gene used in this investigation.

Key words: Inheritance, Mungbean, MYMV, Resistance.

INTRODUCTION

Mungbean Yellow Mosaic Virus (MYMV) is one of the most serious disease and severely occur in the sowing time of February, March and April in mungbean (Nay *et al.* 2008). White fly, *Bemisia tabaci*, is the vector which transmit the MYMV and insecticides application is ineffective under severe insect population (Malathi and John, 2008) and yield losses was found to the extent of 10-100% depending disease severity and crop growth stage (Nay *et al.* 2008 and Khattak *et al.* 2000).

The knowledge on inheritance pattern of resistance on MYMV is very important and useful to the identification of genes that will lead to the suitable breeding techniques and selection procedure for the varietal improvement program of mungbean. The inheritance pattern of MYMV resistance has been reported by many researcher in mungbean and described by a single recessive gene (Reddy, 2009 and Basak *et al.* 2004), a dominant gene (Sandhu *et al.* 1985), two recessive genes (Ammavasai *et al.* 2004 and Pal *et al.* 1991), complementary recessive genes (Shukla and Pandya, 1985) and two recessive genes governed MYMV resistance (Pal *et al.* 1991). However, the study of the selected parents lacking information on controlling gene action for MYMV resistance. Estimation of genetic effects or knowledge of gene action *viz.* additive, dominance and epistatic helps in the selection of parent to know the genetic cause of variability and to frame an efficient breeding strategy to be followed for the improvement of traits (Yadav *et al.* (2017); Narasimhulu *et al.* (2016); Khattak *et al.* (2001).

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Generation mean analysis (Mather and Jinks (1982); Mather and Jinks (1971); Hayman (1958); Jinks and Jones (1958) provides the information about the relative importance of effects of different gene actions *viz.*, additive and dominance with an unambiguous test for epistasis. Moreover, generation mean analysis is a simple and useful technique for the estimation of type of epistasis *viz.*, additive × additive, additive × dominance and dominance × dominance operating in the inheritance of a character. Therefore, this study was conducted to investigate the inheritance pattern of resistance to MYMV in segregation population and determine types of gene action for the improvement of mungbean breeding program.

MATERIALS AND METHODS

The study was conducted at Food Legumes Section, Department of Agricultural Research (DAR), Yezin, Myanmar

(19°51' N latitude and 96°7' E longitude at 97 m altitude) in summer (February-May, a hot spot season for MYMV disease), 2019. The materials of this study were consisted of five mungbean genotypes, viz. two resistant genotypes (7639 and 10266) and three susceptible genotypes (7621,

10257 and R-021018) to develop F_1 , F_2 , BC_1 (F_1P_1) and BC_2 (F_1P_2). Origin, disease reaction and some good characters of parental genotypes were presented in Table 1. These resistant genotypes and susceptible varieties were crossed by the procedure of Khattak *et al.* (1998) and each cross combination (F_1) raised as F_2 , BC_1 (F_1P_1) and BC_2 (F_1P_2). The six basic populations, namely P_1 , P_2 , F_1 , BC_1 , BC_2 and F_2 of each combination will be grown in compact family block design with three replications. Each plot consisted of 3-m rows with inter and intra row spacing of 30 and 10 cm, respectively. The parents (P_1 , P_2), F_1 , BC_1 ($F_1 \times P_1$) and BC_2 ($F_1 \times P_2$) were sown in single row with 3m long (30 plants) and 5 row (150 plants) in F_2 for one family. All recommended cultural practices were followed, except insecticide is not spraying to allow the growth and development of vector *Bemecia tabaci*. Disease reaction of mungbean yellow

Table 1: Origin and disease reaction of parental genotypes.

Genotype	Origin	Name of Cultivar	MYMV disease reaction
7621	AVRDC	VC-1973-A	HS
10257	Myanmar	Shwewar, Kyaikto	HS
R-021018	Seed Bank, DAR	-	HS
7639	Philippine	M-28	HR
10266	Myanmar	Pedishwewar, Salin	HR

Table 2: Reaction to mungbean yellow mosaic virus (MYMV) in six generations of six mungbean crosses.

Generation	Resistant			Susceptible			Ratio	Observed		χ^2	Prob
	HR	R	MR	MS	S	HS		R	S		
7621	0	0	0	0	0	89			89		
7639	84	1	1	2	1	0		86	3		
F_1	0	0	0	0	3	98			101		
F_2	87	1	20	5	31	293	1:3	108	329	0.02	0.89
BC_1	6	0	0	0	1	104		6	105		
BC_2	45	0	5	3	7	55	1:1	50	65	1.96	0.16
10257	0	0	0	0	0	69			69		
7639	56	0	0	0	0	2		56	2		
F_1	0	0	0	0	0	72			72		
F_2	52	0	12	2	20	160	1:3	64	182	0.14	0.71
BC_1	16	0	1	1	1	55		17	57		
BC_2	33	0	1	0	6	27	1:1	34	33	0.01	0.90
R-021018	0	0	0	0	0	45			45		
7639	57	0	0	0	0	0		57			
F_1	0	0	0	0	0	53			53		
F_2	35	0	5	6	20	158	1:3	40	184	6.09	0.01
BC_1	0	0	0	0	0	48			48		
BC_2	28	0	2	3	4	31	1:1	30	38	0.94	0.33
7621	0	0	0	0	0	47			47		
10266	48	0	0	0	0	0		48			
F_1	0	0	1	0	0	47			47		
F_2	35	0	1	3	22	121	1:3	36	146	2.64	0.10
BC_1	1	0	0	0	1	54		1	55		
BC_2	24	0	0	0	4	24	1:1	24	28	0.32	0.56
10257	0	0	0	0	0	89			89		
10266	86	0	1	0	0	0		87			
F_1	2	0	0	0	0	112		2	112		
F_2	76	0	13	9	49	254	1:3	88	313	2	0.16
BC_1	1	0	0	0	0	93		1	93		
BC_2	37	0	2	0	10	31	1:1	39	41	0.05	0.82
R-021018	0	0	0	0	0	75			75		
10266	57	0	0	0	0	0		57			
F_1	0	0	0	0	1	77			78		
F_2	51	0	9	11	27	194	1:3	60	232	3.08	0.08
BC_1	0	0	0	0	0	84			84		
BC_2	27	0	2	1	2	33	1:1	29	36	0.75	0.38

mosaic virus was recorded at 60 days after sowing (when 95% of the spreader rows (Yezin-9) showed MYMV incidence) under field conditions. The tested populations were classified into six reactions that is, highly susceptible (HS), susceptible (S), moderately susceptible (MS), moderately resistant (MR), resistant (R) and highly resistant (HR) on the basis of disease scores and type of symptoms by estimating the average score for the top 5 leaves of each plant within each lines at 60 days after planting by Nair *et al.* (2017). The plants in the F_2 and back cross generations will be classified as resistant (1-3) and susceptible (4-6) following Reddy and Singh (1993).

Statistical Analysis

The chi-square test was performed to determine the goodness of fit of observed segregation for MYMV disease reaction in F_2 and test cross generations for the inheritance pattern of MYMV. The generation mean analysis was performed for the estimation of genetic components by scaling test for A, B, C and D scales as suggested by Mather and Jinks (1971) and Hayman and Mather (1955) and joint scaling test by Cavalli (1952) and estimation of gene effects by the model of the model of Jinks and Jones (1958). The minimum number of gene or number of effective factors controlling resistance was estimated by three methods: Method 1 was proposed by Wright (1968); Methods 2 and 3 were proposed by Lande (1981).

RESULTS AND DISCUSSION

The number of plants, disease reaction and chi square value of parent 1, parent 2, F_1 , F_2 , BC_1 and BC_2 of six crosses were presented in Table 2. According to the results, the F_1 of all six crosses obviously showed that susceptibility to MYMV was dominant over resistance and no maternal effect on the inheritance pattern of MYMV in mungbean. This finding was very close agreement with the research of Khattak *et al.* (2000) and Shukla *et al.* (1978) in mungbean. Moreover, the segregation ratio of F_2 and BC_2 was observed with 1R:3S and 1R:1S, respectively and all these crosses verified that it is a single recessive gene inherited character with dominance effect of susceptibility over resistance to MYMV. The pattern of monogenic recessive inheritance for MYMV has been reported by Sandhiya and Saravanan

(2020); Basavaraja *et al.* (2017); Sai *et al.* (2017); Jain *et al.* (2013); Reddy, (2009). There are, however, many research findings that documented with monogenic dominant gene (Gupta *et al.* (2005); Sandhu *et al.* (1985), two recessive genes (Alam *et al.* (2014); Singh *et al.* (2013); Dhole and Reddy (2012), complementary recessive genes (Thamodhran *et al.* (1988) and Shukla and Pandya (1985) and two dominant genes (Mahalingam *et al.* (2018) and Murugan and Nadarajan (2012).

The result of scaling and joint scaling test by Mather (1949) and Cavalli (1952) for the present or absence of gene interaction was shown in Table 3. The research revealed that the scaling test A, B, C and D were not significant from zero in all crosses except from 10257 × 7639 and R-021018 × 7639. This indicated that the presence of additive × dominance interaction in 10257 × 7639 and dominance × dominance interaction in 21018 × 7639, respectively. However, when the results were subjected to confirm for the scaling test by joint scaling test, all the crosses were found with non-significant values of chi square test. The range of chi square values from the estimate of MYMV was 0.74 and 6.74, respectively. All the values were less than the 0.05 at $df = 3$ (7.81). Therefore, this is clearly indicated that resistance of MYMV was controlled by allelic gene action with no epistatic gene interaction and revealed the adequacy of the simple additive-dominance model which is appropriate to describe the inheritance of MYMV in the respective cross.

Genetic effects *viz*, m, d and h for resistance of MYMV in six mungbean crosses were presented in Table 4 according to the three parameter model by Jinks and Jones (1958). The mid parent value was significant in all crosses and expressed with the value of 3.58, 3.78, 4.19, 3.96, 3.77 and 3.45 in the cross of 7621 × 7639, 10257 × 7639, R-021018 × 7639, 7621 × 10266, 10257 × 10266 and R-021018 × 10266, respectively. This finding pointed that this MYMV resistance was quantitatively inherited. The three parameter model pronounced that only additive gene effect was found in all crosses and more pronounced significant with the value of 2.42, 2.40, 2.48, 2.44, 2.47 and 2.48 in the cross of 7621 × 7639, 10257 × 7639, R-021018 × 7639, 7621 × 10266, 10257 × 10266 and R-021018 × 10266, respectively while the no significant dominance gene effects

Table 3: Estimate of scaling and joint scaling test for MYMV resistance in six crosses of mungbean.

Cross	Scaling test				Joint scaling test (χ^2)
	A	B	C	D	
762 × 7639	-0.5± 0.59	0.49±0.46	0.01±0.73	0.008±0.34	6.47 ^{ns}
10257 × 7639	-0.17**± 0.09	-0.34±0.62	-0.31±0.54	0.10±0.40	4.66 ^{ns}
R-021018 × 7639	-0.02± 0.05	0.40±0.59	1.06**±0.49	0.34±0.38	5.42 ^{ns}
7621 × 10266	-0.09± 1.84	0.23±3.47	0.60±5.77	0.23±3.30	1.41 ^{ns}
10257 × 10266	0.03± 0.12	0.04±0.54	0.35±0.41	0.14±0.33	0.74 ^{ns}
R-021018 × 10266	0.03± 0.03	0.55±0.61	0.51±0.45	-0.03±0.37	2.74 ^{ns}

A, B, C and D = Scales used to identify the presence/absence of epistasis; ns = non-significant; ** = significant and highly significant at 0.01 probability levels, respectively.

Table 4: Gene effect for MYMV resistance in six crosses of mungbean by three parameter model by Jinks and Jones (1958).

Cross	m	d	h
7621 × 7639	3.58**±0.68	2.42**±0.04	2.36±1.75
10257 × 7639	3.78**±0.80	2.40**±0.06	1.47±2.08
R-021018 × 7639	4.19**±0.76	2.48**±0.01	1.48±1.99
7621 × 10266	3.96**±0.91	2.44**±0.03	1.67±2.41
10257 × 10266	3.77**±0.67	2.47**±0.02	1.92±1.81
R-021018 × 10266	3.45**±0.75	2.48**±0.01	3.14±2.01

m = mid parent value, d = Additive effect, h= Dominance effect and ** = significant and highly significant at 0.01 probability levels, respectively.

Table 5: Effective factor of gene for resistance of MYMV in six mungbean crosses.

Effect	7621×7639	10257×7639	R-021018×7639	7621×10266	10257×10266	R-021018×10266
EF1	1.09	1.05	1.38	1.18	1.20	1.27
EF2	0.75	0.74	0.92	0.81	0.86	0.86
EF3	0.43	0.49	0.54	0.46	0.52	0.53
Mean	0.75	0.76	0.95	0.82	0.86	0.89

were observed in all crosses. The plus sign in the additive gene effect implies that P_1 contributes positively to the trait as compared to P_2 and vice versa. This finding was more closely related with the results of gene effects for powdery mildew in mungbean and late leaf spot in groundnut by Wambi *et al.* (2014) and Sorajjapinun *et al.* (2005) who observed that additive gene action was a major role in controlling powdery mildew resistance in mungbean and late leaf spot in groundnut. The estimates of effective factor or minimum number of gene for resistance of MYMV in six mungbean crosses ranged from 0.43-1.09, 0.49-1.05, 0.54-1.38, 0.46-1.18, 0.52-1.20 and 0.53-1.27 in the cross of 7621 × 7639, 10257 × 7639, R-021018 × 7639, 7621 × 10266, 10257 × 10266 and R-021018 × 10266, respectively (Table 5). The average number of effective factor was found with below one in all crosses of this study. Therefore, the resistance of MYMV was governed by only one gene. However, MYMV resistance in mungbean was controlled by at least two genes according to the findings of Alam *et al.* (2014) and Singh *et al.* (2013), respectively.

CONCLUSION

Based on the result of this study, it can be concluded that additive gene action was the major role for the selection of MYMV resistance. Susceptible behavior indicated as dominant over resistant and single recessive gene that controlled for the resistance of MYMV. Therefore, the knowledge of mode of inheritance and gene effects of MYMV resistance is very useful and effectively for the development of genotypes resistant to disease or incorporation of resistance into the desirable promising genotypes which have lack of disease resistance. The expression of gene contributing for MYMV resistance from others findings might be attributed due to the sources of resistant genotypes which have different nature of resistant gene used in this investigation.

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