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EXPLORATION OF DURABLE RESISTANCE IN MUNGBEAN AGAINST MUNGBEAN YELLOW MOSAIC DISEASE

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Mungbean yellow mosaic disease is most damaging disease of mungbean in all of its growing areas. Genetic resistance is a viable and environment friendly solution, for which resistance source is prerequisite. Total 83 mungbean genotypes were experimented for their resistance potential against disease for two consecutive years 2014 and 2015 during summer season. Infection percentage and disease severity based scoring scales were compared for their discrimination potential for screening. Disease severity based scoring scale was found superior to infection percentage based scale because it categorized genotypes into five groups while only two groups were generated by later. Infection %age ranged from 33.33 to 100% while disease severity index varied from 10 to 75%. Only one genotype (NM 6-68-2) was found comparatively tolerant (D.I. 10%) during both the years. Biplot analysis depicted that behavior of some genotypes showed instability in the second year while most of the genotypes showed a stable resistance/susceptibility levels during both the years. Accession NM 1-32-1 was highly susceptible genotype during both the years. Disease severity showed maximum correlation with chlorophyll-*a* reduction followed by 100 seed weight while it had high level of association with yield. Analytical dissection of correlation among traits revealed that direct effect of disease on yield reduction was negligible but it caused yield reduction via reduced harvest index and 100 seed weight. It is concluded that resistance sources are very scarce and resistance level is very low but still these sources can be exploited in breeding programs. For the improvement of genetic variability induced mutation could be another option.

Keywords: Screening, Mungbean, correlation, path analysis, MYMD, biplot, disease severity, path analysis

INTRODUCTION

Mungbean is an important legume crop of Asia which produces almost 90% of the total global production (Sudha *et al*., 2013). It is a nutritive crop with high nutrient value which contains 20-24% protein and 60-65% carbohydrates. During the year 2013-2014 it was grown on an area of 130.9 thousand hectare with production of 92.9 thousand tones in Pakistan (Anonymous, 2014). It is sown in two seasons i.e. spring (Feb-March) and summer (June-July) but major growing season is summer. It is heavily infested with whitefly during summer season which acts as a vector for a viral disease known as mungbean yellow mosaic disease; therefore, its cultivation in summer season is hindered. Virus is only transmitted by whitefly and is neither seed born nor transmitted mechanically. Disease is characterized by yellow specks and spots on leaves, stunted growth, reduced flowers and shriveled seeds. Almost all available varieties are vulnerable to disease and damage may approach up to 100% yield losses in severe epidemics (Sudha *et al*., 2013; Mohan *et al*., 2014). The disease is a major threat to mungbean cultivation in different countries including India, Sri-Lanka, Pakistan, Bangladesh, Papu New Guinea, Philippines and Thailand (Honda *et al*., 1983; Chenulu and Verma, 1988). The disease was first time reported in India on lima bean in 1940 (Capoor and Varma, 1948), in Dolichos (Capoor and Varma, 1950) and in mungbean (Nariani, 1960). In Pakistan (not independent at that time) the disease was first detected in field of chickpea during 1942 near Faisalabad (Vasudeva, 1942).

Crop can be managed by controlling the whitefly attack or by limiting the crop to spring season (no whitefly). But its cultivation in spring season is not economical because in Pakistan it will compete with wheat for cultivation area. Hence its cultivation in summer season is inevitable and whitefly control seems to be an effective option. Chemical control of whitefly requires a massive application of insecticides which is not economically and environmentally accepted. In short genetic resistance against the disease is the only viable option to combat the mentioned issue. A

massive research has been conducted to find out resistance source by different researchers (Singh *et al.,* 1996; Shad *et al.,* 2006; Habib *et al*., 2007; Sudha *et al*., 2013; Mohan *et al*., 2014**)** which identified either no resistance source (Singh *et al.,* 1996; Shad *et al.,* 2006; Habib *et al*., 2007) or relatively resistant source (Sudha *et al*., 2013; Mohan *et al*., 2014). No highly resistant mungbean variety is available in Pakistan. So there is a need of a durable resistance against aforementioned disease. Keeping this in view we have evaluated our local germplasm against mungbean yellow mosaic disease using severity based scoring scale (Akhtar *et al*., 2009) which is very effective as compared to previously used scales which are based on infection percentage. Another important feature of resistance source is its durability and stability under different years and locations. Any genotype which shows resistance at one location or time while become susceptible at another location or time is useless. Hence stable behavior of genotype is a prerequisite for breeding purpose.

MATERIALS AND METHODS

Present study was conducted at experimental area of the Department of Plant Breeding and Genetics, University of Agriculture Faisalabad. Total 83 mungbean genotypes, collected from National Agricultural Research Centre (NARC) Islamabad, Nuclear Institute for Agriculture and Biology (NIAB) and Ayub Agricultural Research Institute (AARI) Faisalabad, were evaluated against mungbean yellow mosaic disease for two consecutive years (2014 and 2015). Sowing was done in mid of June in both the years. Seed were sown following triplicated randomized complete block design (RCBD) with 30 cm row to row and 10 cm plant to plant distances. A well-known susceptible check "kabuli (name of mungbean variety)" was sown surrounding experimental field and one row was repeated after every two test entries to enhance the disease intensity. All recommended agronomic practices were followed for better crop stand establishment. No insecticide was sprayed to support a high whitefly population (vector). Genotypes were scored for disease severity using arbitrary scale (Table 1) given by Akhtar *et al*. (2009). For comparison, another scoring scale (Table 2) given by Bashir *et al*. (2005) and based on infection percentage of plants was followed. A similar set of experiment was conducted under protected field condition by covering the area with fly net (Hdpe mesh 40*60 per inch) for creation of normal/ disease free

Table 1. Disease scale based on disease severity (Akhtar *et al*., 2009).

Symptoms	Disease severity	% Disease Index*	Disease reaction
Complete absence of symptoms	0	0	Highly Resistant
Few small yellow specks or spots on few leaves seen after careful observations.	1	0.01-10	Resistant
Bright yellow specks or spots common on leaves, easily observed and some coalesced.	2	10.01-25	Moderately Resistant
Mostly coalesced bright yellow specks or spots common on leaves, but no or minor reduction in yield.	3	25.01-40	Moderately Susceptible
Plants showing coalesced bright yellow specks or spots on all leaves, with no or minor stunting and set fewer normal pods.	4	40.1-60	Susceptible
Yellowing or chlorosis of all leaves on whole plant followed by necrosis, shortening of internode, severe stunting of plants with no yield or few flowers & deformed pods produced with small, immature and shriveled seeds.	5	>60.01	Highly Susceptible

*The percentage disease index was calculated as: (Sum of all disease ratings/total number of plants) ×20

Table 2. Disease scale based on infection percentage (IP) (Bashir *et al.*, 2005).

Disease Severity	Percent Infection	Infection Category	Reaction Group
0	All plants free of disease symptoms	Highly resistant	HR (Highly resistant)
1	1 – 10% Infection	Resistant	R (Resistant)
2	10.01 -20% infection	Moderately resistant	MR (Moderately resistant)
3	20.01-30% infection	Moderately susceptible	MS (Moderately susceptible)
4	30.01-50% infection	Susceptible	S (Susceptible)
5	More than 50%	Highly susceptible	HS (Highly susceptible)

condition to calculate reduction percentage over control. At physiological maturity five plants (out of 20 plants) from each plot in each replication of protected (healthy) and unprotected (diseased) trial were randomly selected and data were recorded for plant height, pods per plant, pod length, seeds per pod, 100 seed weight, yield per plant, harvest index, chlorophyll *a* contents, chlorophyll *b* contents and β-carotenoid. Genotypic correlation of disease severity was calculated with both absolute values of traits as well as their percentage change over control. For the purpose of correlation and path coefficient analysis, the analysis of variance, the components of variances viz. genotypic variance (σ^2 g), phenotypic variance (σ^2 p) and environment variance (σ^2 e) and covariance between all possible pairs of characters separately were estimated following the methods as described by Singh and Chaudhary (1977). Genotypic correlation of disease with yield was dissected into direct and indirect effects using path coefficient analysis by using formula (Wright, 1921) extended by Dewey and Lu, (1959). Durability or stability of genotypes across the years or locations can be examined by using GGE-biplot method (Yan *et al*., 2000, 2001) which provides a more well-designed and useful presentation of multi environmental trial (MET) data. It efficiently enlightens both the difference of mega environments and genotype selection for a given mega environment based on performance and stability. In addition, it provides interpretation of genotype and environment interactions (Yan and Hunt, 2001). Hence, we have evaluated durability/stability of genotypes across two years by biplot analysis

RESULTS

After germination (almost two weeks after sowing) due to high population of whitefly (vector), disease symptoms started first on susceptible check. Mild to severe yellow specks were first observed on young leaves. Within next 2 weeks, these specks increased, coalesced and turned into yellow and green patches. The severity of disease increased with the passage of time. After 30 days of sowing all the genotypes were showing disease symptoms. While there was no disease symptom inside the protected area (Figure 1).

Protected field condition

Unprotected field condition

Figure 1. Pictorial view of both field conditions (20 days after sowing).

***Comparisons of scales*:** We used both types of scales for scoring and made comparison between them. According to infection %age scale, only three genotypes were scored as susceptible while remaining 80 genotypes were scored as highly susceptible during both years. There was not a single genotype in any resistant category i.e. HR, R or MR. In contrast to this, disease severity based scale differentiated the genotype into five groups i.e. R, MR, MS, S and HS (Table 3). So it showed that disease severity based scoring is superior to infection %age based scale because it had power to differentiate the genotypic differences more accurately. Secondly later scale dose not bother about extent of severity, that is, severely disease and mildly diseased plants are not differentiated in this scale. So, it is important finding that scoring methods are significantly different from each other and impose a drastic effect on decision of scoring a genotype as resistant or susceptible.

***Field evaluation*:** In year 2014 infection percentage (IP) ranged from 33.33 to 100% whereas it varied from 40 to 100% during 2015. Similarly, Disease index (DI) was also variable during both years, ranging 10 to 75% in 2014 and 8.8 to 77.7% during 2015. Not a single genotype was found to be highly resistant (HR) during both years. We preferred scoring scale based on severity index, so these screening results are presented according to this scale. During 2014 out of 83 genotypes, one genotype was found resistant, eight moderately resistant, 46 moderately susceptible, 27 susceptible and one highly susceptible while one resistant, seven moderately resistant, 39 moderately susceptible, 33 susceptible and 3 highly susceptible were found during 2015

Table 3. Response of Mungbean germplasm against MYMD during 2014 and 2015 with two different scales.

Genotypes	2014				2015				Genotypes	2014				2015			
	Infection percentage		Disease severity		Infection percentage		Disease severity			Infection percentage		Disease severity		Infection percentage		Disease severity	
	IP	DR	SI	DR	IP	DR	SI	DR		IP	DR	SI	DR	IP	DR	SI	DR
NM 5153	100	HS	45.7	S	93.75	HS	48.7	S	NCM 23	100	HS	44.00	S	100	HS	53.3	S
NARC-2013	100	HS	40	MS	100	HS	40.0	MS	NM 0183	50	S	15.00	MR	50	S	15.0	MR
6163 B-4	90.9	HS	34.5	S	100	HS	47.5	S	NM 05-6-18	100	HS	42.86	S	100	HS	45.0	S
NM 08	100	HS	37.7	S	100	HS	48.7	S	NM 6231	100	HS	42.86	S	94.44	HS	37.7	MS
NM 252-1	100	HS	48.8	S	100	HS	48.8	S	NM 42-4-3	87.5	HS	22.5	MR	100	HS	24.0	MR
11-12	100	HS	33.3	MS	87.5	HS	33.7	MS	NM 13124	60	HS	20.00	MR	85.71	HS	22.8	MR
NM 12	85.7	HS	25.7	MS	94.44	HS	36.6	MS	NCM 258-10	100	HS	31.43	MS	100	HS	38.4	MS
NM 13	100	HS	40	MS	100	HS	40	MS	NM 05-1-2	100	HS	42.00	S	100	HS	37.7	MS
NCM 257-10	100	HS	50	S	100	HS	43	S	MMH 42-15-104	75	HS	35.00	MS	100	HS	38.3	MS
97019	100	HS	30	MS	92.31	HS	40	MS	NM46-5-21	100	HS	40.00	MS	100	HS	46.0	S
NM 046	66.6	HS	33.3	MS	91.67	HS	40.0	MS	NCM 252-2	87.5	HS	42.5	S	100	HS	43.3	S
NCM 254-7	100	HS	34.2	MS	100	HS	38.82	MS	E 96	100	HS	45.00	S	100	HS	44.0	S
NM 04-1-11	100	HS	40	MS	93.33	HS	36.00	MS	D-173-B-10	88.8	HS	33.33	MS	100	HS	40.0	MS
V2/07009	100	HS	41.8	S	100	HS	43.00	S	C 2-94-3-11	100	HS	40.00	MS	100	HS	51.4	S
NM 15-11	100	HS	37.5	MS	100	HS	37.50	MS	NM 04-2-38	100	HS	33.33	MS	91.67	HS	31.6	MS
NCM 254-3	100	HS	40	MS	100	HS	61.43	HS	NM 04-2-29	100	HS	34.29	MS	100	HS	38.8	MS
NIFA Mung-2	66.67	HS	26.6	MS	100	HS	36.00	MS	NM 06	100	HS	28.00	MS	88.89	HS	32.2	MS
207	100	HS	42.8	S	100	HS	56.84	S	C 2-94-4-36	100	HS	40.00	MS	88.89	HS	36.6	MS
NM 3960-88-31	100	HS	50	S	100	HS	47.69	S	NCM 254-2	100	HS	42.5	S	100	HS	50.0	S
NM 1-32-1	100	HS	75	HS	100	HS	77.77	HS	NCM 252-10	100	HS	40.00	S	100	HS	52.2	S
NM 04-1-12	100	HS	40	MS	100	HS	40.00	MS	NCM 251-13	100	HS	32.5	MS	100	HS	40.0	MS
NM 46-5-215	100	HS	35.56	S	100	HS	55.38	S	DERA AZRI-01	88.8	HS	35.56	MS	100	HS	43.5	S
NCM 251-16	100	HS	43.33	S	100	HS	55.29	S	NCM 257-11	100	HS	50.00	S	100	HS	58.5	S
BRM 311	100	HS	37.14	MS	100	HS	35.38	MS	NCM 21	100	HS	40.00	MS	100	HS	35.0	MS
NM 04-1-5	90	HS	40	MS	100	HS	35.00	MS	NIFA MUNG 3	77.7	HS	24.44	MR	100	HS	25.0	MR
97007	100	HS	40	MS	100	HS	40.00	MS	V1/08009	100	HS	37.78	MS	100	HS	38.7	MS
NIA MUNG 1	100	HS	37.78	MS	100	HS	37.33	MS	NM 9	100	HS	42.22	S	100	HS	40.0	MS
D-173-B-10	88.89	HS	35.56	MS	100	HS	36.36	MS	V3/07006	100	HS	42.22	S	100	HS	42.2	S
NM 04-3-1	100	HS	32	MS	92.86	HS	40.00	MS	NM 05-6-7	100	HS	51.43	S	90.91	HS	47.2	S
NM 013	100	HS	35.56	MS	100	HS	49.33	S	NM 04-1-3	100	HS	42.22	S	100	HS	36.3	MS
NM 6-60-2	100	HS	53.33	S	100	HS	53.33	S	NCM 15-11	100	HS	25.00	MS	100	HS	28.0	MS
10-10	100	HS	37.78	MS	100	HS	37.78	MS	NM 5254104	100	HS	46.67	S	100	HS	42.0	S
NM 4-2-11	88.89	HS	42.22	S	100	HS	42.00	S	NM 05-6-17	60	HS	20.00	MR	60	HS	20.0	MR
NM 0115	100	HS	36.67	MS	100	HS	36.67	MS	NM 6-68-2	33.3	S	10.00	R	44	S	8.88	R
NM 36-15-3	100	HS	44	S	100	HS	44.00	S	NCM 251 4	100	HS	45.00	S	100	HS	48.8	S
NM 6-29-1	88.89	HS	31.11	MS	88.89	HS	31.11	MS	NCM 255-2	100	HS	43.33	S	100	HS	43.3	S
NCM 252-3	100	HS	40	MS	100	HS	50.53	S	E-136	87.5	HS	35.00	MS	100	HS	38.5	MS
AZ MH 2	75	HS	20	MR	100	HS	24.00	MR	AZ MH 1	100	HS	40.00	MS	100	HS	40.0	MS
NM 9800	100	HS	50	S	94.12	HS	32.94	MS	NM 57X	100	HS	56.00	HS	100	HS	70.0	HS
NM 05-1-7	40	S	12	MR	40	S	12.00	MR	NM-2011	75	HS	35.00	MS	100	HS	34.2	MS
NCM 257-3	90.9	HS	32.73	MS	100	HS	34.6	MS									

Table 4. Analysis of variance for disease severity.

Source	DF	SS	MS	F
Block	2	5.9	2.966	
Years	1	950.5	950.490	69.62
Error block*years	2	27.3	13.653	
Genotypes	82	51765.7	631.289	35.70*
Years*genotypes	82	6572.2	80.148	4.53*
Error block*years*genotypes	328	5799.4	17.681	
Total	497	65121.0		

(Table 3). It showed that there was a difference among the behavior of genotypes during both years; some genotypes showed different levels of resistance or susceptibility in both years. The genotype namely "NM 6-68-2" was found to be resistant and "NM 1-32-1" highly susceptible during both years while NM-57 and NCM-254-3 showed susceptible and moderately susceptible reaction respectively in 2014 but highly susceptible reaction in 2015.

***Biplot analysis*:** A stable behavior of genotype against any disease is very important character because it ensures durability of resistance both in spatial as well as temporal terms. Data of disease severity were subjected to analysis of variance (Table 4). Results presented in Table 4 showed that there were significant genotypic differences for disease severity. Genotype x year interaction was also significant. Hence stability analysis of genotypes was also carried out. For this purpose scoring was done in two years 2014 and

2015 and data was subjected to biplot analysis. Biplot (Figure 2) depicted that both the years had different effects and genotypes behaved differently. Some genotypes were proved very stable in their behavior while others unstable. Genotypes NM-6-68-2, NM-05-1-17, NM-0183 and NM-5254104 were found toward resistant side and therefore, declared as stable ones because their distance from vectors of both the years was same. Similarly genotype NM-1-32-1 was found at susceptible extreme having very stable behavior during both the years. In contrast, genotypes NCM-254-3, NM-9800, NCM-257-10 and NM-46-5-215 were observed as most unstable genotypes. NM-9800 and NCM-257-10 were relatively more susceptible genotypes during 2014 while less susceptible during 2015 whereas NCM-254-3 and NM-46-5-215 were found relatively more susceptible during 2015 and less susceptible during 2014. Under unstable resistant category, genotypes NM-12 and NIFA MUNG-2 showed relatively high resistance during 2014 while others were found susceptible during 2015.

***Correlation and path coefficient analysis*:** Correlation between disease and plant traits under disease condition was calculated and presented in Table 5. Disease severity showed significant negative correlation with plant height, pod length, 100 seed weight and yield per plant while it had non-significant correlation with harvest index, pods per plant, chlorophyll-*a*, chlorophyll-*b*, β-carotenoids and seeds per plant. This proves that MYMD has a significant negative effect on yield and yield related traits of mungbean. But linkages between disease and absolute values of traits are unable to explain the effect of disease on mungbean yield and growth exactly. For this purpose correlation was computed between MYMD severity and reduction percentage of various traits. Correlation in this case was positive and significant for plant height, harvest index, chlorophyll-*a*, chlorophyll-*b*, β-carotenoids, 100 seed weight and yield per plant while non-significant with seeds per pod, pods per plant and pod length.

Genotypic correlation of disease severity and yield was partitioned into direct and indirect effects for both reduction percentage and absolute values (Figure 3, 4). Direct effect of disease on yield in both conditions was negligible but with different signs. In absolute values direct effect was negative with the result that increase in disease intensity reduced yield while in case of reduction percentage direct effect was positive, that is, increasing disease severity increased reduction percentage of yield. In case of reduction percentage data, disease posed positive indirect effect via all the traits under study except plant height, pod length and chlorophyll-*b*. Indirect effect via harvest index and 100 seed weight was maximum in positively contributing parameters while indirect effect via plant height was maximum for negatively contributing parameters. In case of absolute data, disease posed positive direct effect on yield only through seeds per pod while negative through plant height, harvest index, pods per plant, chlorophyll-*a*, chlorophyll-*b*, β-carotenoids, 100 seed weight, seeds per pod and pod length.

Table 5. Genotypic correlations of disease severity with various traits and reduction percentage of the traits.

Pant traits	Reduction percentage	Absolute values
Plant height	0.274*	-0.241*
Harvest index	0.233*	-0.153
Number of Pods per plant	0.055	-0.062
Chlorophyll-*a*	0.984**	0.043
Chlorophyll-*b*	0.420**	0.034
β-carotenoids	0.526**	0.136
Pod length	0.173	-0.307**
Seeds per pod	0.185	-0.182
100 Seed weight	0.695**	-0.303**
Seed yield per plant	0.267*	-0.318**

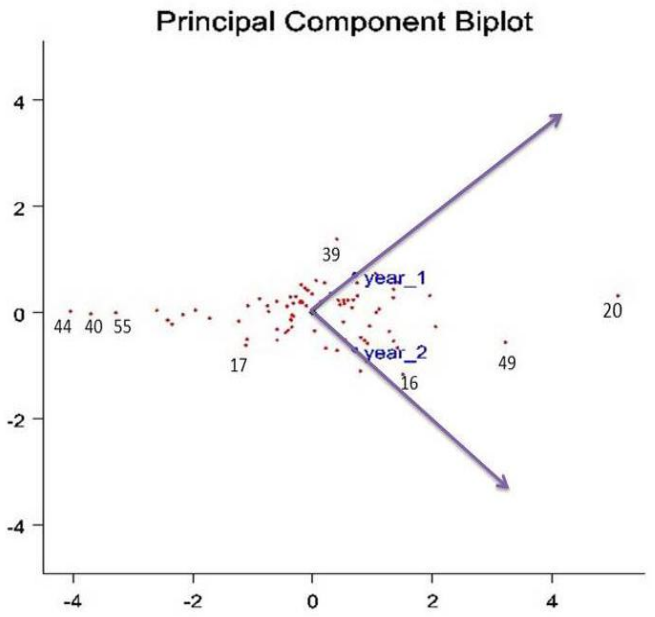

Figure 2. Biplot for MYMD resistance during 2014 and 2015.

Where Year-1= 2014, Year-2= 2015, Codes: 44 (NM 6-68-2), 40 (NM 05-1-7), 55 (Nm 0183), 17 (NIFA MUNG-2), 39 (NM 9800), 16 (NCM 254-3), 49 (NM 57X), 20 (NM 1-32-1)

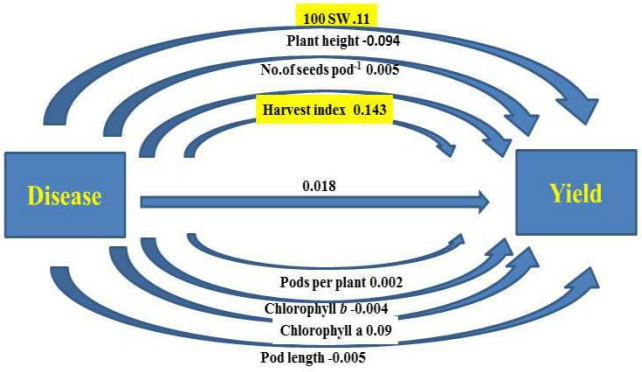

Figure 3. Direct and indirect effects of MYMD on percent change of yield.

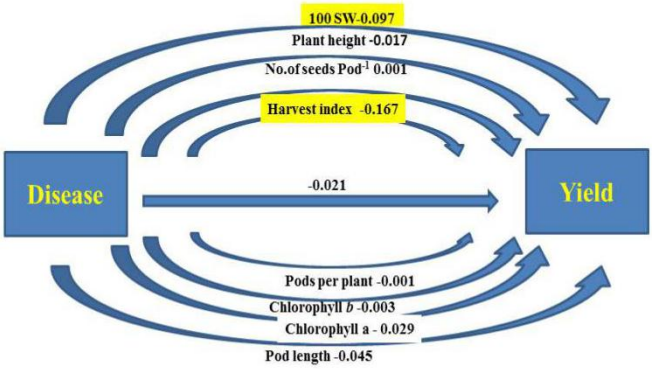

Figure 4. Direct and indirect effects of MYMD on yield.

DISCUSSION

In our study, out of 83 only one genotype was found to be resistant against the disease during both the years while 8 and 7 genotypes were moderately resistant during 2014 and 2015, respectively. It shows that mungbean germplasm is deficient of natural resistance against mungbean yellow mosaic disease. This study confirmed the findings that the resistance against mungbean yellow mosaic virus is very scarce (Ahmad, 1975; Sahoo and Hota, 1991; Singh *et al*., 1996; Saleem *et al*., 1998; Bashir, 2005; Shad *et al*., 2006; Akhtar *et al*., 2011; Iqbal *et al*., 2011). This scarcity of resistant source is actually due to low proportion of recessive alleles (Sudha *et al.,* 2013) in natural gene pool which also implies that recessive allele is mutated form of wild gene conferring susceptibility. Reported resistant genotype can be used as resistant source in breeding programs for the development of high yielding and mungbean yellow mosaic disease resistant varieties. Because of scarcity of resistance it is recommended that resistance should be hunted from wild species. There was no immune genotype which implied that all genotypes allowed viral replication however, there was difference in symptoms development rate and extent in addition to differential effect on yield losses among genotypes. The resistance levels found in this study may be sufficient to improve the resistance levels of already developed cultivars with good agronomic characteristics. A more detailed study of world mungbean genotypes will be needed to find more sources of resistance to broaden the genetic basis of MYMD-resistant germplasm.

From breeding point of view durability and stability of resistance is prerequisite. The genotype which remains resistant across different seasons and locations is desirable. Therefore, genotypes should be evaluated for their resistance behavior before declaring them resistant. The difference of behavior of genotypes across the years or locations may be due to variability of vector population, environmental conditions or genotype x environmental interaction. Hence germplasm was evaluated for two consecutive years to confirm that resulting resistance is only due to genotypic basis not due to lack of vector attack or any other environmental condition. The two-year evaluation discovered high level of genetic diversity among the genotypes for MYMD resistance and variable behavior of genotypes across the seasons and years (Akhtar *et al*., 2011).

An important aspect in breeding for disease resistance is accurate measurement of disease or disease resistance indicators (Akhtar and Khan, 2002) because it ensures the identification of a real resistance source. If there is problem in this step then upcoming efforts will end in nothing. As a matter of fact in case of MYMD, utilization of wrong disease scoring scale is most misleading step. Until now most of researchers utilized the disease scoring scales which are based on percentage of infected plants (Ahmad *et al*., 1975; Bashir, 2005; Bashir *et al*., 2006; Khattak *et al*., 2008) but do not account for severity of disease. It implies that this type of scale is unable to differentiate a severely affected plant from mildly affected plant. While another scoring scale based on severity of disease, was developed by Akhtar *et al*. (2009). We evaluated our germplasm on the basis of both of disease scoring scales. Scale based on the infection percentage was able to differentiate the genotypes only into two groups while five groups of genotypes were made on the basis of disease severity index. Another notable point is that pure line or self-pollinated variety is always homogeneous so at specific time or place either all plants of that variety should be affected or disease free. Without difference of environmental conditions or vector population this differential behavior within genotype is neither expected nor acceptable. So with respect to laws of genetics disease scoring scales based on infection percentage of plants are not justifiable because genetically similar plants have to behave alike under similar conditions.

Plant improvement requires pyramiding combination of desirable traits in a single plant. This needs the knowledge of

correlation of different plant traits among themselves. Correlation of disease severity with plant traits was measured under diseased condition. We found that there was significant negative correlation of disease severity with yield per plant, 100 seed weight, plant height and pod length. It implies that development of resistant genotypes can improve yield and yield contributing traits because the susceptibility has a negative association with all these traits. Although correlation of disease severity with absolute values of traits provides reasonable information of relations. But linkage between disease and absolute values of traits are unable to explain the effect of disease on mungbean yield and growth exactly. For this purpose correlation of MYMD severity was measured with reduction percentage of various traits. Correlation in this case was positive and significant for plant height, harvest index, chlorophyll-*a*, chlorophyll-*b*, β-carotenoids, 100 seed weight and yield per plant. It shows that increase of disease severity enhanced losses in aforementioned traits. Chlorophyll contents are very important with respect to photosynthetic capacity of plant and subsequently in yield per plant through assimilate production. We reported maximum association of disease severity with chlorophyll-*a* which implies that observed losses in chlorophyll-*a* were caused by disease. Losses/ destruction of chlorophyll contents due to MYMD result in symptom development of disease. Reduced photosynthetic capacity of plant cannot provide enough assimilates for development of healthy seeds in pods. This statement is reinforced by our observation that 100 seed weight was second most affected trait by MYMD. So, as a result yield reduction was also in line with disease severity resulting in positive significant correlation. So, it is evident that correlation for reduction percentage is more important than that of absolute values. It supports the idea that all plant traits are negatively affected by disease. Khattak *et al.* (2000) reported significant correlation of disease severity with plant height and 100 seed weight.

In case of correlation there was significant correlation among disease severity and yield per plant. We dissected this genotypic correlation into its contributor to determine the ways through which disease posed negative effect on yield per plant. It was interesting to know that there was negligible direct effect of disease on yield per plant and also on reduction percentage of yield. Yield per plant was only affected by disease via other yield contributing parameters. It suggests that significance of genotypic correlation among disease and yield was only due to other plant parameters. So, from both of the conditions it is concluded that disease does not affect yield directly but its effect on yield is via other yield contributing traits. Decrease in harvest index and 100 seed weight are the major causes of yield reduction due to disease; this endorsed the findings of Alam *et al.* (2014) who reported 100 seed weight as main component of yield improvement. It is concluded that these traits should be given proper consideration while breeding for MYMD resistance breeding.

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