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Screening of mungbean germplasm against mungbean yellow mosaic virus under natural condition

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Abstract

Mungbean [*Vigna radiata* (L.) Wilczek] is an important legume which can be grown in all three crop seasons viz., *kharif, rabi* and spring/summer in India, as sole or inter crop for grain and green manure. The production of mungbean is severely affected by mungbean yellow mosaic virus (MYMV) caused by begmoviruses transmitted by white fly, *Bemesia tabaci*. The absence of resistant/tolerant sources against MYMV disease leads to tremendous crop yield losses, with the aim of identifying the resistant source we carried out an experiment at UAS Dharwad, screened one hundred seven genotypes of mungbean germplasm lines against MYMV during summer season under field conditions. The germplasm was categorized in to resistant and susceptible depending upon severity of disease. The differential response of mungbean accessions to MYMV was determined and most of the genotypes studied were categorized as susceptible to moderately susceptible. In spite of the variable response to MYMV, the genotypes HUM 1, KM 15, KM 16, KM 24, KM 28, KM30, KM 36, KM 39, KM45, KM 47, KM 49, KM 51, KM 60, KM70, KM78 and KM 79 exhibited resistant reaction against the MYMV disease fourteen were moderately resistant and 30 were moderately susceptible. Remaining 30 accessions were classified as susceptible and 43 as highly susceptible accessions.

Keywords: Mungbean, resistant, screening, mungbean yellow mosaic virus

Introduction

Mungbean (*Vigna radiate* L. Wilczek) is under cultivation since prehistoric time in India. It is also known as green gram and serve are a major source of dietary protein for the vast majority of people. In India these crops are cultivated in three different seasons, viz., *kharif, rabi* and *summer*. Among several constraints for mungbean production, Mungbean Yellow Mosaic Virus (MYMV) disease occupies prime position and is the most destructive and devastating viral disease. It was first reported in India in 1955 and is transmitted by the insect vector, White fly (*Bemisia tabaci*) and it is not spread by mechanical inoculation or by seed. (Shad *et. al.*, 2005) ^[13]. The virus initially develops yellow patches then progressively turn the entire leaf yellow and the affected plants flower sparsely and the pod contain shriveled seeds. MYMV can cause yield loss of about 75–100% depending on disease incidence, virus strains, mungbean genotypes and interaction between these factors (Singh 1980).

Controlling MYMV incidence is only possible by the way of reducing the vector viz., whitefly population using insecticides which are ineffective under severe infestations. The chemical management of the vector is not cost-effective since numerous sprays of insecticides are required to control whitefly. Recurrent sprayings also lead to health danger and ecological effluence (Sana *et al.* 2007) ^[12]. So, in this regard, identification of varieties having vector as well as virus resistance served as economical and feasible approach in alleviation of disease severity and placed a prominent value in breeding programmes. Resistance in mungbean germplasm against MYMV has been recognized earlier by different workers by using scale based on disease severity (Ahmad, 1975, Murtza *et al.*, 1983; Ghafoor *et al.*, 1992; Bashir & Zubair 2002, Bashir 2005, Bashir *et al.*, 2006, Khattak *et al.*, 2008, Pathak *et al.*, 2006) ^[2]. In this study, mungbean germplasm were screened against mungbean yellow mosaic virus under natural field conditions in order to identify resistant genotypes which could be helpful in the improvement of breeding efforts.

Materials and Methods

The experiment consisted of 107 mungbean germplasm lines were screened under natural condition to Mungbean yellow mosaic virus at college of agriculture Dharwad. Each entry was sown in an augmented design with spacing 30 x 15 cm, PUSA 9591; SML-134, KKM4 and HUM 1 were used as checks. Summer whiteflies are the source of the virus in the field, so no insecticide was sprayed in order to maintain the natural whitefly population in the experimental field. When 80% of the plants showed MYMV symptoms, scoring of the test materials was done. Disease incidence was recorded. Percentage Disease Incidence was worked out using the formula

Number of Plants infected in a row Percentage Disease Incidence (PDI) = $\xrightarrow{} \times 100$ Total number of plants in a row

The genotypes were categorized using (0-9) arbitrary scale (Mohan *et. al.* 2014) ^[8] as Immune (I), Resistant (RR), Moderately Resistant (MR), Susceptible (S) and Highly Susceptible (HS) based on disease incidence

 Table 1: Disease Scoring Scale (0-9) for MYMV based on Percentage Disease Incidence (PDI)

Scale	Description	Category
0	No plants showing any symptoms.	Immune
1	Plants exhibiting less than one per cent infection.	Resistant
3	1-10 per cent plants exhibiting symptoms.	Tolerant
5	11-20 per cent plants exhibiting symptoms.	Moderately susceptible
7	21-50 per cent plants exhibiting symptoms.	Susceptible
9	51-100 per cent plants exhibiting symptoms.	Highly susceptible

Table 2: The disease reaction of	f green gram g	enotypes to Mungbean	Yellow Mosaic Virus.
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Reaction to MYMV	Disease score	Genotypes
Resistant	1	HUM1 KM16, KM15, KM78, KM60, KM59, KM51, KM48, KM49, KM47, KM45, KM39, KM36, KM30, KM28, KM24
Tolerant	3	SML668, HUM12, SML348, M108, KM70
Moderately susceptible	5	VGG4, KM6, KM8, SML134, PUSA9072, PDM11, MDV3156, PM1, KM27, KM31, KM35, KM37, KM38, KM46, KM40, KM41, KM42, KM 43, KM67
Susceptible	7	PS16, K851, PB1, TAP7, TARM2 PSA9591, KKM3, MAVT836, NP36, NDM1, PM2, PRATHAP, KAPORGAO, KA851, PUSA9531, PUSAVISHAL, PM5, AKM8804, KM1, KM2, KM5, KM9, KM11, KM12, KM13, KM14, KM17, KM18, KM19, KM 20, KM21, KM22, KM 23, KM25, KM26, KM29, KM32, KM34, KM44, KM52, KM53, KM54, KM55, KM56, KM58, KM61, KM62, KM63, KM34, KM65, KM66, KM68, KM69, KM72, KM75, KM77
Highly susceptible	9	LM182, KM3, KM4, KM10, KM17, KM73, KM74, KM71KM57, KM33



Fig 1: Number of mungbean genotypes categorized in different disease reaction against MYMV

Results

One hundred and seven genotypes of mungbean were sown under natural environmental conditions on 2015 summer in augmented design. Among the 107 genotypes HUM 1, KM 15, KM 16, KM 24, KM 28, KM30, KM 36, KM 39, KM45, KM 47, KM 49, KM 51, KM 60, KM70, KM78 and KM 79 had resistance towards MYMV". Five of the mungbean genotypes viz., SML668, HUM12, SML348, M108 and KM70 exhibited a tolerant reaction against MYMV with a disease score of "3". Other genotypes VGG4, PM1, MDV3156, SML134, PUSA9072, PDM11, KM6, KM 8, KM27, KM31, KM35, KM37, KM38, KM40, KM41, KM42, KM43, KM46, KM50 and KM67 shows the moderate susceptible with a disease score 5. Genotypes PS16, K851, PB1, TAP7, TARM2, PSA9591, KKM3, MAVT836, NP36, PM103, NDM1, PM2, PRATHAP, KAPORGAON,

KA851,PUSA9531, PUSA VISHAL,PM5,AKM8804, KM1, KM2, KM5, KM7, KM9,KM11, KM12, KM13, KM 14, KM18, KM19, KM20, KM21, KM22, KM23, KM25, KM26, KM29, KM32, KM34, KM44, KM48, KM52, KM53, KM54, KM55, KM56, KM58, KM61, KM62,, KM63, KM64, KM65, KM66, KM68, KM 69 and KM 75 shows the susceptible disease reaction with a disease score 7. The genotypes LM182, KM3, KM4, KM10, KM17, KM33, KM57, KM71, KM73 and KM74 exhibit the highly susceptible to MYMV disease with disease score 9. Based on the observed result, only few genotypes appeared to be as resistant, which indicated the existence of small amount of resistance in genotypes against MYMV.

Discussion

Viral diseases including MYMV drastically reduce the yield of numerous legume crops. Screening of mungbean genotypes in response to MYMV under field conditions determines the greater susceptibility of genotypes to MYMV, which may perhaps be associated to favorable environmental conditions for the disease development owing to the presence of enormous vector population in the field. The susceptible check lines after every two test entries resulted in enhanced vector population Iqbal et al. (2011)^[5, 6]. The MYMV vector, whitefly (Bemisia tabaci Genn) appeared to inhabit plant soon after the emergence and remained till maturity and with the passage of time, disease severity increased significantly. In summer season, high temperature favored vector dispersion, since it required opportunity of multiplication on host crop (Shakoor et al., 1977) [14] Shad et al., (2006) [9] found no immunity or resistance in 254 lines; all lines were susceptible to highly susceptible to the virus. Bashir et al. (2006)^[2] reported to have found as high as 85 lines to be highly resistant and 14 lines to be resistant to MYMV out of 110 lines screened in mungbean. The differential response of mungbean accessions to MYMV were observed by Iqbal at al., 2011^[5, 6], and identify the 43 highly susptible accession and no resistance accessions were found. Kooner et al. (2005) ^[7] found just two genotypes were resistant to MYMV out of the fifty seven genotypes of mungbean. Ghuge et al (2018) identified 74 genotypes of mungbean eight were found resistant, nineteen genotypes were moderately resistant, thirty seven were susceptible, five genotypes were moderately susceptible and five were highly susceptible to yellow mosaic virus of mungbean.

Among the one hundred and forty six lines were screened only one line was found to be resistant against virus, which showed that this virus is a severe problem (Akhtar et al., 2009). For the identification of resistance source against MYMV, continuous screening during every year is required. The disease incidence is more in summer this is due to high vector population and other environmental factors as the temperature during that period was suitable for vector and MYMV multiplication. As environmental data given shows that the average temperature during the above mentioned period was highly suitable for the vector multiplication. Humidity level during that period is also optimal for the spread of disease. Findings of Habib et al. (2007)^[4] also indicated that the crop is more sensitive to MYMV during the initial period of crop development. The chances of infections are reduced with maturity of the crop and the current findings were similar with results of Shad et al., (2006)^[9]. The results showed that there were 56 susceptible and 10 highly susceptible genotypes of mungbean. Great variation in genotype response to MYMV represents variability in their genetic makeup. It was generally reported that two recessive genes took part in the control of resistance mechanism in mungbean (Shukla & Pandya, 1985), whereas susceptibility was controlled by single recessive gene. So it becomes evident that susceptibility is dominant over resistance. Lack of resistant varieties necessitates the development of virus resistant varieties through genetic engineering and biotechnology in future.

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