ORIGINAL ARTICLE

Association mapping for *mungbean yellow mosaic India virus* resistance in mungbean (*Vigna radiata* L. Wilczek)

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Abstract

The present study aimed to detect the marker-trait association of a selected diverse panel of 127 mungbean genotypes against *mungbean yellow mosaic India virus* (MYMIV). Virus-specific primers pairs viz., AC-abut/AV-abut and BC-abut/BV-abut confirmed the involvement of MYMIV in yellow mosaic disease development and the same was validated through restriction digestion analysis. 256 genome-wide microsatellite markers were screened on a test panel in which 93 polymorphic markers were used in association studies. Population structure analysis led to formation of six distinct subpopulations. 1097 alleles were detected among 127 test genotypes whereas number of alleles ranged 2–22 and PIC values ranged 0.27–0.92%, indicating ample amount of variation at genome level. 15 microsatellite markers were detected as associated with MYMIV resistance, among them three microsatellites explained 11–14% phenotypic variation. The specific regions close to CEDG293, DMB-SSR008 and DMB-SSR059 associated with MYMIV resistance were detected, located on linkage group 2, 4 and 9 and may prove useful in marker-assisted mungbean improvement programme for enhancing MYMIV resistance.

Keywords Marker-trait association · MYMIV · Simple sequence repeats · *Vigna radiata* · YMD

Introduction

Mungbean or green gram [*Vigna radiata* (L.) Wilczek] has a unique position in prominent cropping systems in the Indian subcontinent as well other countries of East and South-East Asia due to its shorter life cycle, high per day productivity and use in numerous food preparations (Singh et al. 2017). Development of disease resistant, short duration and photo-thermo insensitive varieties of mungbean during the last

three decades has further helped in expanding its cultivation to newer areas and cropping systems in the world. However, several biotic and abiotic stresses such as drought, fluctuating temperatures and pest and disease problems continue to impart a challenge towards realizing the true yield potential of the released mungbean cultivars (Ali et al. 2010). Among them, viral diseases have emerged as the major threats to its cultivation (Singh et al. 2019). Yellow mosaic disease (YMD) caused by begomoviruses (family Geminiviridae, genus *Begomovirus*) is the most serious disease which has been reported to cause tremendous yield losses (Varma et al. 1992; Maiti et al. 2011) as well as deterioration in the seed quality in mungbean.

Three species of Begomoviruses, i.e., *mungbean yellow mosaic India virus* (MYMIV), *mungbean yellow mosaic virus* (MYMV) and *horsegram yellow mosaic virus* (HgYMV) are known to cause YMD in mungbean (Malathi and John 2008). *Dolichos yellow mosaic virus* (DoYMV) is also noticed as very close at genome level to these three viruses but its infection is reported limited to *Dolichos* bean only (Naimuddin et al. 2016). In *Vigna,* the YMD causing viruses are bipartite having DNA-A and DNA-B components, approx 2.7–2.8 kb in size (Pant et al. 2001; Borah and Dasgupta 2012) which are about 2723 and 2675 nucleotides

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long, respectively (Qazi et al. 2007). The DNA-A component of MYMIV and MYMV share about 82% identity in their sequences and therefore justify their separation into distinct species. The second group of DNA-B component is more related to MYMIV (about 92%) than to MYMV. The DNA-B group represents MYMIV, MYMV and HgYMV which cause similar disease symptoms and it is extremely difficult to differentiate them on the basis of morphological symptoms on the plant. However, genome characterization of these three described viruses helps in identifying the virus causing YMV diseases (Akaram et al. 2015).

The conventional breeding approach coupled with molecular marker technology has proven to be effective in introgressing disease resistance in food legumes such as chickpea (Varshney et al. 2014; Pratap et al. 2017), which can help in accelerating mungbean improvement programmes also. In mungbean, molecular breeding now sounds more practicable after the availability of whole genome sequence (Kang et al. 2014). Earlier, several workers utilized the cross-transferable microsatellite markers from related legumes in developing mungbean maps for various important traits (Isemura et al. 2012; Kitsanachandee et al. 2013; Chankaew et al. 2014), which indicated availability of reliable markers for marker-assisted selection (MAS) and identification of quantitative trait loci (QTLs) for desired traits. Developing high yielding varieties with enhanced resistance by tagging and mapping of loci/gene(s) governing the specific disease resistance is more effective and efficient approach. Based on published reports it is amply clear that MYMIV resistance in mungbean is governed by QTLs suggesting the quantitative inheritance of MYMIV. To date, seven QTLs have been reported for MYMIV resistance through bi-parental linkage analysis (Kitsanachandee et al. 2013; Alam et al. 2014). However, linkage mapping has its own limitations due to bi-parental segregation. In recent years, exploring QTLs by association analysis has been one of the effective approaches in quantitative genetics, which performs rapid and fine-mapping of the target locus. In the present investigation, we focused on identification of novel QTLs/associated markers for MYMIV resistance through SSR-based genome-wide association mapping approach.

Materials and methods

Mapping panel, field experimentation and disease scoring

The plant materials comprised 127 mungbean genotypes including released varieties, advanced breeding materials, elite lines, and local and exotic collections (Supplementary Table 1). The materials were grown under natural field conditions in six different environments viz., the tropical climate

at ICAR-Indian Institute of Pulses Research, Kanpur, India (26.28° N, 80.24° E, 152.4 m above mean sea level) during Monsoon season (*Kharif*) of 2016 (E_1), Summer season of 2017 (E_2), Monsoon season of 2017 (E_3), New Research Farm, Kanpur during Monsoon 2016 (E_4), Summer season of 2017 (E_5) and Monsoon season of 2017 (E_6). In each environment, all test genotypes were grown in two rows of 4 m length. Four highly MYMIV susceptible genotypes viz., Co-5, Vamban-1, Barabanki local and DGGV-2 were grown after every five test genotypes for creating sufficient innoculum load to ensure field epiphytotic conditions. The recommended cultural practices without any plant protection measure such as chemical seed treatment, insecticidal spray, etc. were followed to raise the crop. Disease scoring was done on 0–9 scale (0 as highly resistant and 9 as highly susceptible) following Ahmed (1985) (Table 1). When infector genotypes recorded > 75% of yellow mosaic symptoms, which usually occurred at 40–45 days after sowing, severity of disease was calculated as per the following formula:

Disease severity (DS) = {sum of all disease ratings/(total number of plants × maximum grade)} × 100.

Pathogen identification, validation and microsatellite markers

The genomic DNA extracted from symptomatic leaves showing yellow mosaic infestation was subjected to rolling circular amplification using RPLI-g kit (Qiagen, India) as per manufacturer's instruction to enrich the copy of circular DNA. The amplified product was diluted @1:20 (DNA: H_2O) and used for detection of virus(es). Six primer pairs each for DNA-A and DNA-B of MYMIV, MYMV and HgYMV were used for detection of presence of virus species (Table 2). The identified virus species leading to YMD development was validated through restriction digestion analysis. Initially, 256 microsatellite markers were screened on a test panel of 20 genotypes comprising highly resistant to highly susceptible genotypes to detect the polymorphic

Table 1 Disease scoring for categorization of yellow mosaic disease in mungbean

Disease score	Disease reaction	Remarks
0	HR	No visible disease symptoms on leaves
1	R	< 5.0% disease symptoms on leaves
3	MR	5.1–15.0% disease symptoms on leaves
5	MS	15.1–30.0% disease symptoms on leaves
7	S	30.1–75.0% disease symptoms on leaves
9	HS	> 75.1% disease symptoms on leaves and sometimes may be on pods

HR Highly resistance, *R* resistance, *MR* moderately resistance, *MS* moderately susceptible, *S* susceptible, *HS* highly susceptible

Table 2 Primer pairs used for characterization of virus(es) species involved in disease development

Primer code	Sequence	Remarks	References
AC-abut/AV-abut	GTAAAGCTTTACGCATAATG AAAGCTTACATCCTCCAC	MYMIV Full length DNA-A	Islam et al. (2012)
BV-abut/BC-abut	CCAGGATCCAATGATGCCT ATTGGATCCTGGAGATTCA	MYMIV Full length DNA-B	
MYMV-CP	ATG GG (T/G) TCC GTT GTA TGC TTG GGC GTC ATT AGC ATA GGC AAT	MYMV/DNA A	Naimuddin et al. (2011a, b, c)
MYMV-MP	ATG GAG AAT TAT TCA GGC GCA TTA CAA CGC TTT GTT CAC ATT	MYMV/DNA B	
HYMV-CP	ATG CTT GCA ATT AAG TAC TTG CA TAG GCG TCA TTA GCA TAG GCA	HgYMV/DNA A	
HYMV-MP	ATG GAG CAT TAT TCC GGT GCA TTA CA(G/A) GGT TTT GTT TAC AGT	HgYMV/DNA B	

markers. The selected 93 genome-wide polymorphic microsatellite markers covering the whole genome were used to find out the strongly associated markers in the vicinity of loci conferring resistance to MYMIV (Supplementary table-2).

DNA extraction and PCR conditioning

Total genomic DNA was extracted from 15-day-old seedlings of all mungbean genotypes following the Cetyl Trimethyl Ammonium Bromide (CTAB) method (Doyle and Doyle 1990). The quality of the extracted DNA was equated on 0.8% agarose gel and the quantity was determined using Nanodrop spectrophotometer ND 1000 (Nanodrop Technologies, DE, USA). DNA samples were normalized at a concentration of 50 ng/µl for PCR amplification. The PCR amplification was carried out in 20 µl reaction mixture containing 10× Taq buffer A with 15 mM $MgCl_2$, 2 mM dNTPs and 1U of Taq DNA polymerase (Fermentas, Mumbai), 50 ng template DNA and 10 pmol each of forward and reverse primers (ILS, India) in a tetrad thermocycler (G-Strom, Somerset, UK). PCR conditions were programmed at initial denaturation for 3 min at 95 °C, followed by 35 cycles of denaturation for 1 min at 94 °C, annealing for 1 min at 45–55 °C (primer specific), extension at 72 °C for 1–3 min (primer specific) and final extension of 72 °C for 10 min. The PCR products thus obtained were resolved on 1–3% agarose gel (based on expected size of fragments) in 1× TAE buffer and stained with ethidium bromide. Gel images were taken using gel documentation system (Uvitech, Cambridge). Polymorphic microsatellite markers were identified on the basis of differences in amplicons by comparison with 100-bp DNA ladder.

Data generation and statistical analysis

The clear and fine bands were scored and their reproducibility was validated by repeated amplification with selected markers. The data were subjected to analysis using Power Marker version 3.25 (Liu and Muse 2005) to calculate the total number of alleles (NA), major allele frequency (MAF), gene diversity (GD) and polymorphic information content (PIC) value. Population genetic structure analysis was carried out using STRUCTURE version 2.3.4 (Pritchard et al. 2000). The number of presumed population (K) was set from 2 to 15 with admixture model. The Markov Chain Monte Carlo (MCMC) chains were run at 50,000 burn-in-period at fixed number of population. The final results were based on 1,00,000 and three iterations on this chain using a model allowing for admixture. The optimal value of K was determined by examining Delta K statistic and L (K) (Evanno et al. 2005) using Structure Harvester (Earl and Von-Holdt 2012) available free online (https://taylor0.biology.ucla.edu/structureHarvester).

The TASSEL software version 2.1 (https://www.maizegenetics.net) was used to establish marker-trait association between individual markers and MYMIV. Two approaches viz., general linear model (GLM) with Q (without consideration of kinship) and multiple linear model (MLM) with $Q + K$ (with consideration of population structure and kinship) were adopted to extract information on association of the genome-wide microsatellite markers with MYMIV resistance.

Results

Identification and validation of YMD-causing virus species

Rolling circular amplification (RCA) was performed to enrich the quantity of virus DNA followed by PCR analysis and restriction digestion with different enzymes for both DNA-A and DNA-B. PCR amplification was done with specific primers for MYMIV, MYMV and HgYMV to confirm

the presence of virus(es) leading to disease development. The MYMIV specific markers, AC-abut/AV-abut (GTAAAG CTTTACGCATAATG/AAAGCTTACATCCTCCAC) and BC-abut/BV-abut (CCAGGATCCAATGATGCCT/ATT GGATCCTGGAGATTCA), the MYMIV specific primers, gave the expected fragment size of about 2.7 kb (Fig. 1a), which indicated the presence of MYMIV leading to YMD development in mungbean, whereas primer pairs MYMV-CP, MYMV-MP (Fig. 1b), HgYMV-CP and HgYMV-MP (Fig. 1c) did not amplify.

The presence of MYMIV in the infected samples was also validated through the analysis of restriction pattern of host DNA with specific enzymes (Fig. 2). Restriction digestion with *XbaI* (Fig. 2a) indicated the absence of DoYMV, but it could not rule out the presence of the other three viruses in the sample. To check the spectrum of other YMD-causing virus species, samples was further subjected to restriction digestion with PmeI (Fig. 2b), a cutter of MYMIV but non-cutter of MYMV, which confirmed that MYMV was not

present in the samples. Likewise, the sample was again digested with non-cutter of MYMIV and one positive control of MYMV was digested with the cutter of MYMV, *Sma*I (Fig. 2c). The sample was further digested with *Hin*dIII (Fig. 2d), which is a single cutter of MYMIV and multiple cutter of HgYMV. Based on the digestion pattern with various restriction enzymes, it was validated that only MYMIV was present in the diseased leaf samples collected from different experimental sites.

MYMIV-phenotyping

MYMIV scores of test entries were recorded after reaching the disease severity > 75% in spreader rows. The sufficient number of white flies were also recorded (data not sown), whose spreaded the MYMIV over the population. Based on phenotypic score, the genotypes were grouped into six categories viz., HR, R, MR, MS, S and HS. The extent of disease severity of MYMIV was recorded upto 81.00% in E1, 88.89% in E2, 40.00% in E3, 88.33% in E4, 89.93% in E5, 93.89% in E6 and 81.02% in pooled over environments (Fig. 3). In all environments individually as well as in pooled over environments all the four susceptible genotypes exhibited more than 75% disease severity (data not shown). Accordingly, 37 (29.13%) mungbean genotypes were categorized in HR and 31 (24.41%) in R category, whereas rest of the 59 genotypes (46.10%) exhibited variable disease reactions ranging between moderately susceptible to highly susceptible.

Genetic and phenotypic variations

Ample amount of genetic variation was detected among the test genotypes in terms of major allele frequency (MAF), number of alleles (NA), gene diversity (GD) and polymorphic information content (PIC) presented in Table 3. Based on 93 polymorphic microsatellite loci, 1097 alleles were
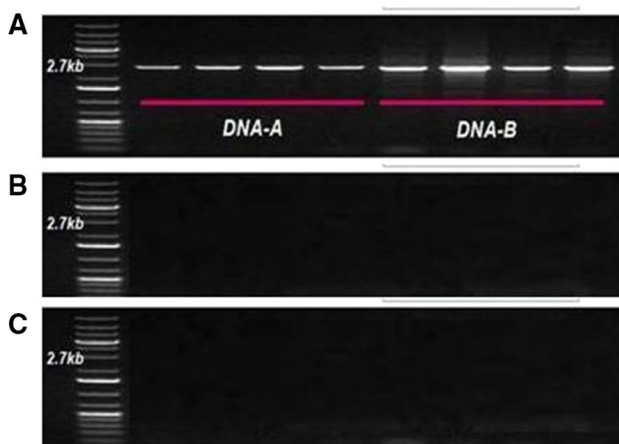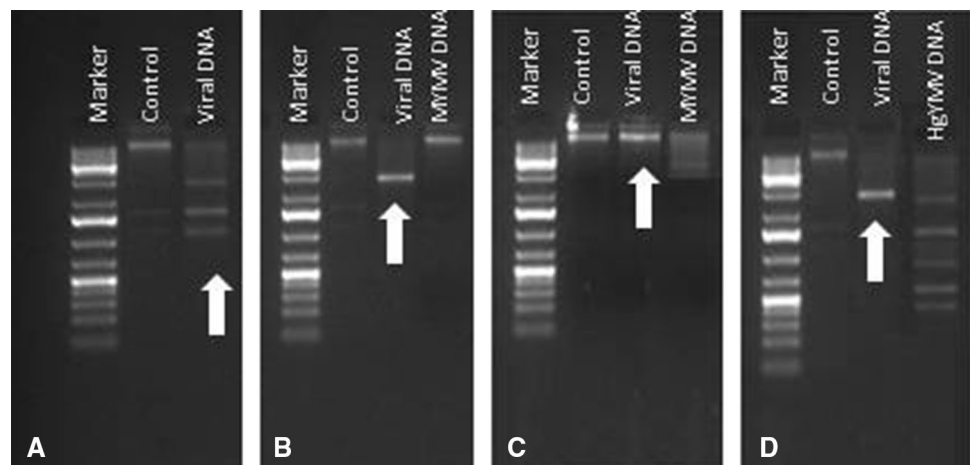

Fig. 1 Identification and confirmation of virus species **a** MYMIV, **b** MYMV, **c** HgYMV in development of yellow mosaic disease

Fig. 2 Validation of involvement of MYMIV in developing YMD through restriction digestion **a** XbaI, **b** PmeI, **c** SmaI, **d** HindIII
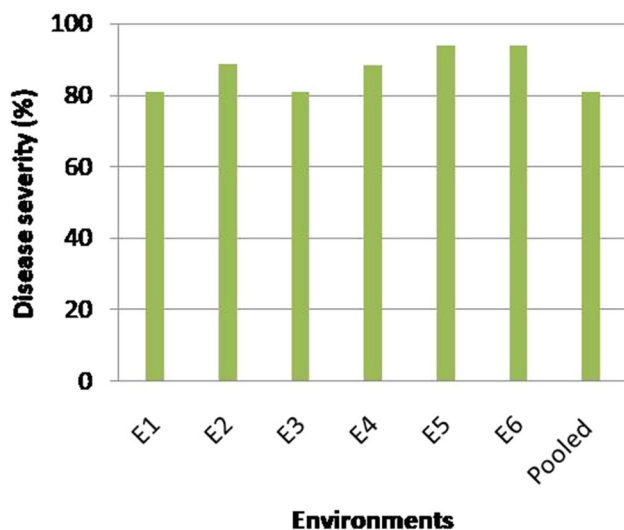
Fig. 3 The maximum percent MYMIV-disease severity exhibited in different environments

detected among the 127 test genotypes. Number of alleles ranged from 2 (CEDG244, cp08695) to 22 (CEDG254), with a mean of 11.72 alleles/locus. GD and PIC values ranged between 0.27 (cp05096) to 0.92 (CEDG100) and 0.25 (CDEG244) to 0.96 (CEDG097) with a mean value of 0.80 and 0.79, respectively. Locus CEDG097 was found most informative, whereas CDEG244 was the least. Any constant pattern of correlation was not observed between number of repeat units/number of alleles and GD or PIC. The structured data of population exhibited almost equal distribution of mungbean genotypes with differential MYMIV reactions.

Population genetic structure

Population genetic structure of 127 mungbean genotypes was carried out and $K = 6$ was found suitable and showed comparable or higher likelihoods than $K = 2–15$ (Fig. 4). Twenty (15.74%) out of 127 mungbean genotypes (Indore Mung, EC 520024, EC 520026, EC 520029, EC 520034, EC 520034-1, ML 818, ML 512, MG 331, OBGG 52, IC 296672, IPOI 539, Kopargaon, Ganga 8, SPS 5-1, Saptari, Asha Mung, EC 550831, PDM 139 and Sona) were categorized as admixtures with varying levels of membership shared among the three groups (Fig. 5). The subpopulation-I included 24 genotypes representing 18.89% of the elite breeding lines, whereas subpopulation-II was represented by 21 genotypes (16.54%). The subpopulation-III included 20 genotypes, subpopulation-IV, 02 genotypes, subpopulation-V included 19 genotypes and subpopulation-VI included 41 genotypes sharing 15.75, 1.5, 14.96 and 32.29% of all test genotypes, respectively. The subpopulation-I consisted of most of the exotic collection and some of the varieties released by GB Pant University of Agriculture and

Technology, Pantanagar and Banaras Hindu University, Varanasi. The subpopulation-II consisted of material developed by ICAR-Indian Agricultural Research Institute (IARI), New Delhi, Bhabha Atomic Research Centre (BARC), Mumbai, and Dr. Panjabrao Deshmukh Krishi Vidyapeeth (PDKV), Akola. The sobpopulation-III was characterized by advance breeding lines developed by ICAR-IIPR, Kanpur and other institutes. The subpopulation-VI was categorized as the largest subgroup which consisted majority of the released varieties and elite lines developed by ICAR-IIPR, Kanpur.

Marker-trait association

Association study was conducted using the MYMIV scores based on phenotypic evaluation and the allelic data of 93 genome-wide microsatellite markers. As a result, 15 marker-trait associations (MTAs) for MYMIV were observed over the environments using the most accepted maximum likelihood model (P3D mixed linear model with optimum compression) of the association analysis (Table 4). This association explained 0.41–14.45% of the total phenotypic variation through GLM whereas 0.58–13.26% of total phenotypic variation was explained by the markers through MLM. Our results showed a significant association of 15 microsatellite markers with MYMIV at P value ($P < 0.05$) in which five microsatellite loci viz., CEDG293, DMBSSR059, DMB-SSR008, CEDG211, and CEDG121 were strongly associated with MYMIV resistance which explained about > 5.00% phenotypic variation. Among them, CEDG211, CEDG121 and DMB-SSR008 were found within the earlier reported QTL region whereas two markers viz., CEDG293 and DMB-SSR059 were found outside the region and are considered as novel identified regions.

Discussion

Involvement of three different virus species in causing yellow mosaic disease in *Vigna* crops has been established leading to similar kind of morphological symptoms of YMD, thereby misleading us on the YMD-causing virus species. Therefore, detection of the particular virus species causing YMD is of upmost importance for its effective management. The characterization of the virus species is not possible at morphological level, as most of the molecular studies conducted earlier (Kumar et al. 2014; Singh et al. 2015; Gupta et al. 2015) also did not specified the exact species of virus leading to YMD in mungbean. Therefore, presence any of the three viruses and even a mixed infection could not be ruled out in these reports. However, now the identity of three different species of viruses leading to YMD development has been established at molecular level and the genome sequences of all these

Table 3 Diversity statistics of microsatellite marker in mungbean population

Marker	Major. Allele. Frquency	Allele No	Gene Diversity	PIC	Marker	Major. Allele. Frquency	Allele No	Gene Diversity	PIC
RGA 1F-CG/RGA 1R	0.64	8	0.52	0.46	CEDG 191	0.42	13	0.77	0.75
CEDG 036	0.16	12	0.73	0.73	CEDG 118	0.27	15	0.89	0.89
CEDG 003	0.1	11	0.73	0.73	CP1038	0.2	12	0.82	0.82
CEDG 053	0.31	11	0.73	0.71	CEDG 146	0.27	18	0.88	0.88
CEDG 204	0.5	10	0.73	0.72	CEDG 015	0.74	7	0.43	0.41
CEDG 256	0.23	13	0.76	0.84	GMES1823	0.14	13	0.81	0.81
CEDG 128	0.75	14	0.44	0.43	CP09781	0.15	13	0.84	0.84
CDEG 220	0.45	11	0.78	0.77	CEDG 225	0.16	11	0.82	0.82
CEDG 254	0.13	13	0.83	0.73	CDEG 244	0.85	2	0.27	0.25
CEDG 041	0.16	11	0.9	0.79	GMES 0211	0.09	16	0.86	0.86
GMES 2320	0.42	14	0.78	0.77	CEDG 064	0.15	15	0.82	0.82
CEDG 048	0.14	11	0.83	0.82	CEDG174	0.06	13	0.88	0.88
CEDG 293	0.17	8	0.82	0.82	MB-SSR 008	0.12	11	0.84	0.84
CEDG 136	0.26	21	0.88	0.87	CEDG186	0.33	11	0.86	0.85
CEDG 297	0.55	14	0.67	0.65	CEDG 218	0.39	11	0.81	0.8
CEDG 100	0.15	12	0.92	0.92	CEDG176	0.1	14	0.84	0.84
CP02662	0.33	12	0.87	0.86	CEDG 295	0.12	12	0.84	0.84
CEDG 225	0.18	11	0.9	0.89	CEDC 302	0.15	12	0.84	0.84
CDEG 244	0.16	13	0.84	0.83	CEDG 271	0.16	11	0.82	0.82
CEDG 050	0.11	12	0.84	0.84	CEDGAT 009	0.12	13	0.85	0.85
GMES 0211	0.4	10	0.81	0.8	CEDG 035	0.12	14	0.84	0.84
CP00361	0.15	8	0.82	0.82	VM 37	0.08	13	0.87	0.87
CEDG 296	0.15	12	0.83	0.82	CEDG 030	0.17	11	0.83	0.83
BM 146	0.33	13	0.86	0.85	CEDG 071	0.35	13	0.74	0.7
DMB SSR035	0.09	15	0.86	0.86	CEDG 247	0.66	8	0.54	0.51
CEDG 305	0.07	13	0.86	0.86	DMB-SSR 059	0.37	10	0.8	0.78
CEDG 254	0.17	22	0.81	0.81	CEDG 070	0.27	11	0.86	0.85
CEDG 159	0.06	11	0.86	0.86	CEDG 024	0.19	12	0.83	0.83
CEDG 084	0.11	12	0.86	0.86	CEDG 290	0.18	13	0.82	0.82
CEDC 139	0.37	11	0.78	0.76	VM 27	0.08	15	0.86	0.86
CEDC 055	0.12	11	0.85	0.84	CEDG 166	0.24	9	0.87	0.86
GMES0162	0.1	10	0.83	0.83	CEDG267	0.34	8	0.78	0.76
CEDG 185	0.15	9	0.83	0.82	BM 212	0.55	10	0.69	0.68
CEDG 088	0.21	7	0.79	0.88	CEDG 147	0.42	11	0.8	0.79
CEDC 033	0.13	13	0.85	0.85	CEDG113	0.12	10	0.83	0.83
GMES 0035	0.42	11	0.79	0.78	DMB-SSR043	0.19	13	0.9	0.89
DMB SSR008	0.27	11	0.82	0.89	CEDG 116	0.32	14	0.87	0.86
DMB-SSR199	0.11	13	0.84	0.84	CEDG097	0.11	11	0.86	0.96
DMB-SSR080	0.12	15	0.84	0.84	CEDG 150	0.33	14	0.8	0.78
DMB SSR024	0.26	8	0.89	0.88	CEDG 168	0.3	10	0.85	0.84
CEDG 008	0.08	10	0.87	0.87	CP00464	0.28	14	0.88	0.88
CEDG 115	0.1	11	0.85	0.85	CEDG 013	0.1	12	0.85	0.85
BM 170	0.19	11	0.83	0.83	CEDG 044	0.06	11	0.86	0.86
DMB SSR151	0.08	14	0.85	0.85	BM149	0.67	9	0.51	0.47
CEDG 121	0.23	10	0.9	0.89	CP 5096	0.27	11	0.84	0.83
CEDG 211	0.11	14	0.84	0.84	CP 08695	0.84	2	0.29	0.29
DMB SSR001	0.12	11	0.82	0.82	Mean	0.25	11.72	0.80	0.79

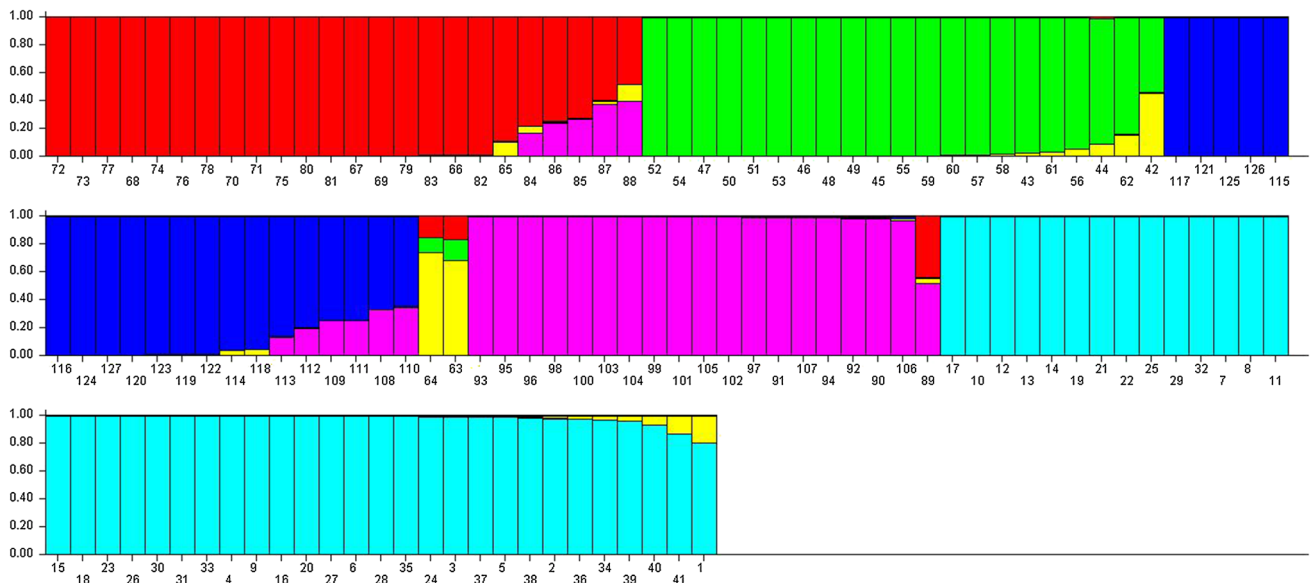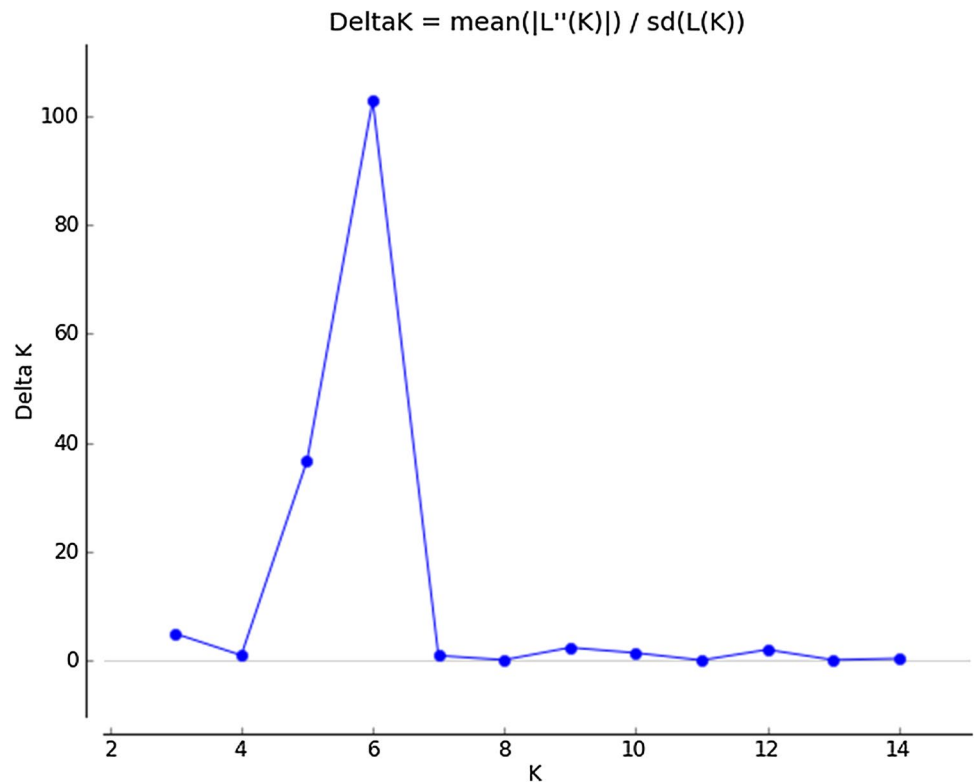**Fig. 4** Magnitude of Ln$P(D)$ and ΔK as a function of K

Fig. 5 Bar plot represented population genetic structure of 127 mungbean genotypes using the admixture model. Groups for each panel are represented by different colours

viruses are available. Therefore, the efforts should be directed towards their molecular detection which would be more precise to identify the virus specific resistance donors and develop the strategies for mungbean improvement. In our study, PCR analysis and restriction digestion were performed, which exhibited presence of MYMIV in YMD development at experimental sites.

Many of the important traits in crop plants such as yield, quality, resistance to biotic and abiotic stresses, etc. are likely to be controlled by additive genes. Nevertheless, the QTLs with minor contributions towards the trait and the

Table 4 Association of microsatellite markers with mungbean yellow mosaic virus resistance

Marker	LG	R2 (E1)	P value	R2 (E2)	P value	R2 (E3)	P value	R2 (E4)	P value	R2 (E5)	P value	R2 (E6)	P value	R2 (Pooled)	P value
General linear model (GLM)															
CEDG 293	2	14.45	0.008	13.14	0.015	12.2	0.783	13.26	0.016	12.42	0.016	12.35	0.641	12.12	0.100
CEDG 225	2	5.5	0.007	4.97	0.013	5.25	0.818	3.62	0.009	2.95	0.009	4.54	0.039	3.9	0.066
CP00361	2	4.1	0.000	3.34	0.001	2.68	0.845	3.2	0.005	1.97	0.005	4.07	0.015	2.99	0.003
CEDC 139	4	1.06	0.011	3.25	0.023	2.26	0.406	2.05	0.029	1.52	0.029	1.62	0.129	1.9	0.036
DMB SSR008	4	12.24	0.022	12.41	0.024	13.04	0.878	11.34	0.169	11.84	0.169	11.91	0.009	12.87	0.063
CEDG 121	6	3.25	0.010	1.87	0.072	2.23	0.960	1.79	0.187	2.28	0.187	1.2	0.200	1.87	0.193
CEDG 211	6	6.05	0.016	8.7	0.066	6.52	0.732	7.85	0.311	6.15	0.311	8.52	0.172	7.48	0.117
CEDG 191	6	4.72	0.004	4.72	0.028	8.89	0.850	7.7	0.095	6.33	0.095	5.43	0.146	5.02	0.068
CP1038	6	3.19	0.005	2.36	0.008	3.13	0.902	1.77	0.157	1.57	0.157	2.32	0.091	1.9	0.137
DMB-SSR 059	9	4.23	0.031	4.85	0.092	5.07	0.535	4.93	0.282	5.09	0.282	5.84	0.073	6.87	0.279
CEDG 070	9	4.04	0.020	5.48	0.002	6.24	0.501	3.05	0.042	1.4	0.042	2.1	0.090	2.31	0.083
CEDG 290	9	5.16	0.004	5.01	0.001	1.01	0.953	3.28	0.018	4.03	0.018	2.33	0.250	3.08	0.057
VM 27	9	2.06	0.054	2.25	0.039	2.26	0.967	1.56	0.003	2.16	0.003	1.62	0.227	1.84	0.073
CEDG 166	9	2.46	0.001	2.6	0.001	2.86	0.480	2.12	0.007	2.07	0.007	2.99	0.002	2.64	0.009
Multiple linear model (MLM)															
CEDG 293	2	13.26	0.008	12.04	0.015	11.84	0.7833	12.78	0.016	12.86	0.641	11.35	0.219	13.63	0.100
CEDG 225	2	2.43	0.007	2.18	0.011	1.29	0.818	1.89	0.009	1.17	0.039	1.7	0.044	1.79	0.066
CEDG 050	2	2.25	0.000	2	0.001	0.99	0.845	2.7	0.005	2.31	0.015	2.29	0.031	2.58	0.003
CEDC 139	4	2.3	0.011	1.92	0.025	0.45	0.406	1.54	0.029	1.68	0.129	1.6	0.012	2.01	0.036
DMB SSR008	4	11.54	0.010	11.48	0.075	12.66	0.960	11.37	0.187	11.36	0.200	11.54	0.192	11.35	0.193
CEDG 121	6	4.76	0.016	4.7	0.063	2.84	0.732	3.23	0.309	3.26	0.172	3.26	0.099	4.53	0.117
CEDG 211	6	5.09	0.004	5.66	0.023	5.09	0.850	7.73	0.093	4.22	0.146	6.12	0.025	5.18	0.068
CEDG 191	6	1.3	0.005	1.35	0.008	2.29	0.902	1.21	0.154	1.14	0.091	1.43	0.117	1.39	0.137
cp01038	6	1.63	0.029	1.42	0.079	0.83	0.535	1.44	0.275	0.58	0.073	0.84	0.285	1.03	0.279
DMB-SSR 059	9	12.45	0.021	13.08	0.002	12.66	0.501	12.64	0.043	14.03	0.090	12.85	0.031	3.45	0.083
CEDG 166	9	2.83	0.004	2.87	0.001	0.71	0.953	2.18	0.018	2.16	0.250	3.29	0.080	2.95	0.057
BM 212	9	2.48	0.001	2.26	0.000	2.3	0.480	1.92	0.007	1.18	0.002	2.04	0.037	2.2	0.009

sensitivity of a particular trait to the environment sometimes tend to mislead in identifying them. Molecular markers and advanced statistical methods together provide a platform to study the genetics of quantitative traits. Before the availability of mungbean genome sequence (Kang et al. 2014), work in mungbean at molecular level mainly focused on identification of transferable markers from other *Vigna* and related species for genetic analysis (Gupta et al. 2013; Pratap et al. 2014, 2016). Some of the transferable markers were successfully utilized in studying genetic diversity (Sangiri et al. 2007; Gwag et al. 2010; Gupta et al. 2013; Singh et al. 2014) and QTL identification for different traits by linkage mapping (Chankaew et al. 2014; Ismura et al. 2012; Alam et al. 2014). The earlier studies suggested the genetics of yellow mosaic disease resistance in mungbean and other *Vigna* crops to follow simple inheritance pattern and therefore it was thought to be relatively easier to manipulate in other backgrounds. Pal et al. (1991) suggested monogenic inheritance of yellow mosaic disease. However, exceptions of digenic interactions were also reported (Dwivedi and Singh 1986; Verma and Singh 1986). Even in other legumes like soybean, both single dominant monogenic (Bhattacharyya et al. 1999) and digenic recessive interaction (Singh and Mallick 1978) have been reported. Nevertheless, in most of these studies, there was an ambiguity on the species of the virus causing yellow mosaic disease. The contradictions in inheritance pattern could also be due to non-uniform distribution of whitefly population in the field, insufficient inoculum load and/or prevailing environmental conditions which might have led to inaccuracy in ascertaining yellow mosaic disease resistance, thereby leading to errors in selection of resistant genotypes. A relatively recent study suggested recessive monogenic resistance of MYMIV in blackgram (Gupta et al. 2013). CEDG180 was tagged with MYMIV resistance in blackgram which produced 136 and 163 bp amplicons in susceptible and resistance genotypes, located 12.9 cM away from the resistance gene. Likewise, linkage of SCAR-YMV1, SCAR-MYMV583, ISSR811 with yellow mosaic disease resistance in mungbean and urdbean have also been reported by Sauframanien and Gopalkrishnan (2006) and Binyamin et al. (2015). However in mungbean, no molecular evidence of monogenic control of yellow mosaic disease resistance is reported for the disease caused by MYMIV, whereas recent study on inheritance of yellow mosaic caused by MYMV clarify their monogenic inheritance pattern through SCAR markers and two SCARs namely CM9 and CM15 were found strongly associated with MYMV-resistance (Sai et al. 2017). By using the cross-transferable markers two QTLs namely qPMR1 (CEDG282-CEDG191) and qPMR2 (MBSSR238-CEDG166) with the phenotypic coverage of 20.10 and 57.81% have been identified indicating the reliability of these markers. Kitsanachandee et al. (2013) identified three QTLs for MYMIV resistance, one

each on linkage group 2, 4 and 9 and were designated as qMYMIV1 (CEDG100 and cp02662), qMYMIV2 (DMB-SSR008-VR113) and qMYMIV3 (CEDG166-CEDG304), which explained 9.33%, 10.61%, 12.55% of phenotypic variation. They also reported two more, environment-specific QTLs viz., qMYMIV4 (CEDG100-cp02662) and qMYMIV5 (CEDG121-CEDG191). Chen et al. (2013) identified DMB-SSR158, a SSR marker linked to major QTL controlling MYMIV resistance on LG2 between the markers interval of CEDG100 and CEDG108 (Kajonphol et al. 2012). Alam et al. (2014) constructed a linkage map comprising 11 linkage groups and identified a new QTL, i.e., qMYMIV7 (CEDG041-VES503) for YMD resistance. Because of multiple events covered in association mapping, it has been thus used in several crop plants to identify genomic regions associated with a trait of interest which could not have been picked up by analysing a bi-parental population. In this direction, exploring QTLs by association analysis is an effective approach to trace novel QTLs controlling quantitative traits. It is based on linkage disequilibrium and diversity analysis of trait of interest. It offers the advantages of natural population and detecting multiple alleles on the same locus, targeting single genes. Because of higher frequency of spurious associations in the natural population (Rosenberg and Nordborg 2006), the evaluation of population genetic structure is a pre-requisite of association studies. The accurate population structure analysis leads to higher genetic similarity within each group. Thus, the accuracy of the associated markers depends upon whether the population structure is appropriate or not. Therefore, we used model-based structure analysis to determine the population structure of studied genotypes and this information was used in marker-trait association to reduce the chance of false positives. Structured association analysis has been reported to be successfully deployed by Nie et al. (2016) in cotton, Kumar et al. (2014) in soybean, Kumar et al. (2017a) in lentil and Noble et al. (2018) in mungbean, to reduce the false discovery rate of the associated markers.

In our study, the mungbean population of 127 genotypes was classified into 6 subpopulations, which was reasonable to eliminate the spurious association effects in the mapping of MYMIV resistance by marker-trait association analysis. Overall, the nature of population structure in the present study suggested that it was unbiased within a specific sub-population and was appropriate for association analysis. Therefore, the marker-trait association recorded in the present study should be real and the chance of obtaining false positives would be rather less (Pritchard et al. 2000; Gupta et al. 2005; Ostrowski et al. 2006; Jaiswal et al. 2012). Beside this, use of diverse breeding lines makes association mapping more cost effective. The identified QTLs for trait(s) in the background of elite breeding materials can

be used directly in genomic-assisted selection (Kumar et al. 2017a, b).

In the present study 15 marker-trait associations are significantly associated with MYMIV resistance with the phenotypic coverage of 14.45 and 13.26%. However, it is not surprising to observe lower phenotypic variation in association mapping due to several reasons like including low LD and low allele frequency, less number of markers and/or accessions used in analysis (Yan et al. 2011). Based on MLM approach, three markers viz. CEDG293, CEDG225 and CEDG050 were located on LG2, which is likely to be associated with VrSSR112 loci. Likewise, CEDG139 and DMB-SSR008 (LG4); CEDG121, CEDG211, CEDG191 and cp01038 (LG6); DMB-SSR059, CEDG166 and BM212 (LG9) were found associated with their respective linkage groups and were observed to have a definite role in governing MYMIV resistance in mungbean. Three markers viz. CEDG293, DMB-SSR059 and DMB-SSR008 were found strongly associated with MYMIV in mungbean accounting to > 10.00% of the phenotypic variation. Among these the previously reported markers/QTLs involved in MYMIV resistance viz., DMB-SSR008 (Kitsanachandee et al. 2013) contributed 11.54% of phenotypic variation and rest two were detected as novel accounting to 13.26 and 12.45% of phenotypic variation, respectively. Previously, marker-trait association was conducted for a number of important traits in the legumes as well other crops (Kumar et al. 2017b; Zhang et al. 2014). These markers can be closely associated with the genes that control MYMIV resistance in mungbean and can be used in breeding program. Thus, results in the present study revealed that a number of genes/QTLs were responsible for controlling the MYMIV resistance in mungbean.

Association mapping has an ability to detect novel QTLs because it uses diverse germplasm that has more allelic diversity than bi-parental population. Therefore, marker loci associated with MYMIV resistance in the present study might be different from QTL/genes identified in earlier studies. One of the reasons for this may be that many QTLs do not segregated in the bi-parental population because only two alternative alleles are present for a trait (Jaiswal et al. 2012). In earlier studies, a marker associated significantly with a trait of interest explained low phenotypic variation (< 1%) in association mapping studies, for example, in cotton (Nie et al. 2016), and in linseed (Chandrawati et al. 2016). Kumar et al. (2014) detected two SSR markers associated with MYMIV resistance in soybean with coverage of < 1% phenotypic variation. In the present study, microsatellite markers could be associated with those genes that are involved in controlling MYMIV resistance in mungbean and two novel regions associated with MYMIV resistance on linkage group 2 and 9 were identified. The strong association and higher phenotypic coverage of CEDG293 and DMBSSR059 present on LG2 (3.3 cM) and LG9 (2.1 cM) as compared to the previously reported markers present in the QTL region, suggest their increased utility in detection of their allelic variants responsible for enhanced resistance to MYMIV. These markers can be deployed in mungbean improvement programs for introgression of MYMIV resistance.

Compliance with ethical standards

Conflict of interest It is to declare that there is no conflict of interest between the co-authors.

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