Electronic Journal of Plant Breeding

Research Article



Cross-species amplification and genetic variation among blackgram genotypes using SSR markers developed from mungbean DNA sequence scaffolds harbouring putative resistance genes

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Abstract

The cross species amplification of 97 mungbean derived resistance gene-SSR markers were investigated for diversity analysis in a set of 44 blackgram genotypes. A total of 68(70%) SSR markers showed amplification in blackgram. Our of 68 markers, thirty randomly selected markers were used to study the genetic variation among 44 blackgram genotypes varying for yellow mosaic disease (YMD) and powdery mildew disease (PMD) reaction. Thirty SSR primers collectively amplified 90 alleles in blackgram with an average of three alleles/locus. The polymorphic information content (PIC) of the SSR markers ranged from 0 to 0.86 with an average of 0.43. Cluster analysis based on UPGMA neighbour-joining method grouped the 44 genotypes into seven clusters. The genotypes NDU-1 and PU-19 were observed to be highly dissimilar with similarity coefficient of 0.27 in comparison to other genotypes. YMD and PMD resistant and susceptible genotypes could be differentiated by three (MRGSSR 12, MRGSSR 56, MRGSSR 77) and four SSR markers (MRGSSR12, MRGSSR 32, MRGSSR56 and MRGSSR65), respectively. Two of these markers *viz.*, MRGSSR12 and MRGSSR56 were mutually effective in differentiating YMD and PMD resistant genotypes. These were located in mungbean scaffolds JJMO01002369 and JJMO01001477 and exhibited homology with TMV resistance protein N and DNA damage-repair/toleration protein DRT100, respectively.

Keywords: Blackgram, Cross species amplification, Resistance genes, Genomic-SSR, Polymerase chain reaction, Diversity analysis.

INTRODUCTION

Blackgram, commonly known as urdbean is grown in India for its protein rich seeds. It is a self-pollinating, annual diploid (2n = 2x = 22) crop with a genome size of approximately 574 Mbp (Arumuganathan and Earle, 1991). The major yield-limiting factors in blackgram are various biotic (viruses, fungi, bacterial pathogens, and insects) and abiotic (salinity, drought, etc.) stresses (Souframanien *et al.*, 2017). Among the biotic constraints, yellow mosaic virus disease (YMD) transmitted by white fly and powdery mildew disease (PMD) caused by *Erysiphe polygoni* are major threats causing yield losses upto 85% (Varma and Malathi, 2003) and 40-90% (Channaveeresh *et al.*, 2014), respectively. Both YMD and PMD resistance in blackgram were reported to be under the control of single recessive gene (Reddy and Singh, 1995; Singh *et al.*, 1998; Kaushal and Singh, 1989).The disease screening becomes complicated due to rapid evolution of yellow mosaic viruses leading to emergence of new strains with wider host range and difficulties in screening breeding population for powdery mildew disease; especially when weather conditions are unfavourable for strong fungal growth and hot spots of natural epidemics are not always available (Channaveeresh *et al.*, 2014).Therefore, development of elite cultivars with durable resistance requires pyramiding of resistance genes from several sources. This necessitates development of molecular markers for as many resistance genes as possible for their reliable introgression and marker assisted selection.

During the course of evolution, plants have developed complex defense mechanisms to counteract pathogens (Staskawicz et al., 1995) through PAMP (Pathogen associated molecular pattern) Triggered Immunity (PTI) and/or Effector Triggered Immunity (ETI). In ETI, resistance (R) genes products recognize products of avirulence genes of the pathogens (Scofield et al., 1996) and evoke defense responses such as hypersensitive reaction, strengthening of the cell wall, phytoalexin production etc. (Dangl et al., 1996). These R genes are classified into four structurally distinct classes based on protein domains they encode (Ellis et al., 2000). Molecular characterization of these R genes reveal their highly conserved nature among plant species and presence of conserved domains/motifs such as nucleotide binding sites (NBSs), leucine-rich repeats (LRRs), transmembrane domains (TMs) and Toll/ interleukin-1 regions (TIR). These domains are known to be involved in the detection of diverse pathogens, including bacteria, viruses, fungi, nematodes, insects and oomycetes (McHale et al., 2006). This information has been exploited for exploring resistance gene analogues (RGA) in several crops such as blackgram (Basak et al., 2004), common bean (Lopez et al., 2003) and peanut (Yuksel et al., 2005). Degenerate primers derived from R genes conserved motifs have been used for targeting RGAs and profiling of different cultivars of potato, tomato, barley, and lettuce (Van der Linden et al., 2004). Furthermore, some of the RGAs have been transformed into molecular markers such as dCAPS (Derived Cleaved Amplified Polymorphic Sequence) and CAPS (Cleaved Amplified Polymorphic Sequence) to detect the presence of SNPs (Single Nucleotide Polymorphism) and their subsequent mapping on to the mapping populations of faba bean, pea, and chickpea (Palomino et al., 2009; Torres et al., 2010).

Although, RGH (resistance gene homologues) markers are much more effective than random markers, they rarely correspond to functional genes due to interference of large numbers of pseudo-genes, less expressed RGHs were easily lost when amplified from cDNA due to the interference of highly expressed RGHs in random cloning (Ren *et al.*, 2014). EST-derived RGHs can overcome these disadvantages but require EST database which are limited for blackgram. Moreover, some nonresistance genes like the R genes also harbour NBS-LRR (Nucleotide binding site-Leucine rich repeat) motifs and therefore, necessitates further confirmation of target sequence amplified through sequencing or gPCR (Yuksel et al., 2005). Thus, there is a need to search for other ways for exploring R genes rather than completely dependent on targeting RGHs through degenerate primers. Blackgram is assumed to be closely related to mungbean because both originated from the Indian subcontinent (Zukovaskij, 1962). This relatedness can be exploited for transferability of molecular markers such as simple sequence repeats (SSRs) from one species to other as reported in several legume crops such as blackgram (Gupta and Gopalakrishna, 2009; Souframanien and Gopalakrishna, 2009) and Glycine (Peakall et al., 1998). SSRs are the markers of choice because of their ease to use, high reproducibility, hyper variability, locus specificity, and co-dominant nature. Moreover, Studies on disease resistance genes have indicated a high level of polymorphism and presence of SSRs at certain loci (Yu et al., 1996). In the present study, 23 putative disease resistance genes identified by Kang et al., (2014) from mungbean whole genome shotgun sequencing were exploited for developing genomic resources in blackgram with the following objectives: 1) Developing SSR markers from mungbean scaffolds homologous to resistance genes, 2) Analyzing cross-species amplification of developed SSR markers and 3) Studying genetic variation in blackgram genotypes differing in YMD and PMD reactions.

MATERIALS AND METHODS

A total of 44 blackgram genotypes [EC-168200, PUSA-3, IPU02-43, KU96-3, KU96-7, IPU07-3, DPU88-31, TU94-2, Azad-1, LBG-752, LBG-17, LBG-693, LBG-623, TAU-1, Trombay Wild (TW), LBG-703, LBG-20, T-9, Nayagarh, Pant-U19, PU31, PLU-1, TU-43-1, TU-55-1, NDU-1, TU-67, WBG-17, WBG-57, WBG-13, COBG-653, PLU-710, Sharda mash, EC168058, LBG-685, LBG-709, Sheela, EC168234, EC168242, EC168243, IPU-02-6, IPU-99-247, SPS-30, ANU-11, IPU-99-40] including diverse cultivars, landraces, and one wild accession differing for disease reaction(YMD and PMD) were used in the study. Disease reactions of each of the genotypes were considered based on the Annual report of Mungbean, urdbean, lentil, lathyrus, rajmash and fieldpea (MULLaRP) and published literature (Table 1). Total genomic DNAs were extracted from young seedlings using Dellaporta method (Dellaporta et al., 1983). The quantity and quality of DNA were checked using Nanodrop ND 1000 spectrophotometer (Thermo Scientific, USA). The working DNA samples were diluted to a standard concentration of 15ng/µl.

The present study was carried out at Nuclear Agriculture & Biotechnology Division, Bhabha Atomic Research Centre, Mumbai, during 2019. A total of 23 putative

Table 1. Blackgram genotypes used in the study with their disease reaction to YMV and powdery mildew disease

S. No.	Genotypes	Reaction to YMV	Reaction to PMD	References
1	EC-168200	R	NA	Gupta <i>et al</i> ., 2015
2	PUSA-3	R	NA	Gupta <i>et al</i> ., 2015
3	IPU02-43	R	R	Bandi, 2018; Aktar <i>et al</i> ., 2014; Gupta <i>et al</i> ., 2013
4	KU96-3	R	R	Gupta <i>et al</i> ., 2015
5	KU96-7	R	NA	
6	IPU07-3	R	R	Gupta <i>et al</i> ., 2015
7	DPU88-31	R	S	Gupta <i>et al</i> ., 2013
8	TU94-2	R	MR	Bandi, 2018; Gupta <i>et al</i> ., 2015
9	Azad-1	R	NA	Anonymous, 2022
10	LBG-752	MS	R	Bandi, 2018; Priyanka <i>et al</i> ., 2018
11	LBG-17	S	R	Bandi, 2018; Srivastava <i>et al</i> ., 2011
12	LBG-693	S	NA	
13	LBG-623	S	R	Bandi, 2018; Priyanka <i>et al</i> ., 2018
14	TAU-1	S	S	Gupta <i>et al</i> ., 2015
15	Trombay Wild	S	NA	Gupta <i>et al</i> ., 2015
16	LBG-703	S	NA	
17	LBG-20	S	R	Gupta et al., 2013; Srivastava et al., 2011 Priyanka et al., 2018
18	T-9	S	MR	Bandi, 2018; Srivastava <i>et al</i> ., 2011
19	Nayagarh	R	NA	Gupta <i>et al</i> ., 2015
20	Pant-U19	R	R	Gupta <i>et al.</i> , 2015
21	PU31	R	R	Bandi, 2018; Aktar <i>et al</i> ., 2014
22	PLU-1	R	NA	Gupta <i>et al</i> ., 2013
23	TU-43-1	R	MR	
24	TU-55-1	R	MR	
25	NDU-1	R	NA	
26	TU-67	S	NA	
27	WBG-17	S	NA	
28	WBG-57	S	S	Basandrai <i>et al</i> ., 1999
29	WBG-13	S	NA	
30	COBG-653	S	S	Equbal <i>et al</i> ., 2015
31	PLU-710	S	NA	
32	Sharda mash	S	S	
33	EC168058	S	NA	Gupta <i>et al.</i> , 2015
34	LBG-685	S	MR	Bandi, 2018
35	LBG-709	S	R	
36	Sheela	NA	NA	
37	EC168234	NA	NA	
38	EC168242	NA	NA	
39	EC168243	NA	NA	
40	IPU-02-6	NA	NA	
41	IPU-99-247	NA	NA	
42	SPS-30	NA	NA	
43	ANU-11	R	R	
44	IPU-99-40	NA	NA	

R: Resistant; S: Susceptible; MR: Moderately resistant; MS: Moderately susceptible; NA: Not available

resistance proteins identified in mungbean (Vigna radiata var. radiata cultivar:VC1973A) from whole genome shotgun (wgs) sequences (Kang et al., 2014) available in NCBI database (Accession: PRJNA243847, ID: 243847) was used in this study. Amino-acid sequence of proteins were downloaded from uniprot (The UniProt Consortium 2017, https://doi.org/10.1093/nar/gkw1099). All 23 proteins were searched for sequence homology with scaffolds assembled from mungbean whole genome sequencing (Vigna radiata var. radiata, taxid:3916) with the help of tBLASTn algorithm. Significant mungbean scaffolds were searched for SSRs and primer-pairs were designed with the help of websat (http://purl.oclc. org/NET/websat/) online software (Martins et al., 2009). Thirty randomly chosen SSR primers were used to study the genetic variation among 44 blackgram genotypes. PCR reactions were carried out in a 25 µl reaction volume in an Eppendorf Master Cycler (Eppendorf, Hamburg, Germany) with following composition: 75 ng of genomic DNA,10 mM Tris-HCI (pH 8.3), 50 mM KCI, 2.5 mM MgCl₂, 0.08% Nonidet P40, 0.2 mM dNTPs, 1.5 pmoles of forward and reverse primers, and 0.5 unit of Taq DNA polymerase (Fermentas Life Sciences). The amplification conditions were initial denaturation at 94°C for 3 min,5 cycles of: 94°C for 30 s, 56 to 46°C (-1°C each cycle), 72°C for 1 min, and followed by 35 cycles of: 94°C for 30 s, 46°C for 1 min, 72°C for 1 min and ended up with a final extension at 72°C for 7 min. PCR products were resolved on 3% agarose gels in TBE buffer at 80 V and images were captured in a gel documentation system (Syngene, U.K).

Genotyping was done as presence (1) or absence (0) of bands for each allele of the marker regardless of their intensity. Polymorphic information content (PIC) was calculated by the formula of Anderson *et al.* (1993): PIC = $1-\Sigma(P_{ij})^2$, where P_{ij} is the frequency of the jth allele for the ith locus. Genotypic data was analyzed through NTSYS-pc version 2.0 software (Rohlf *et al.*, 1998) and dendrogram was generated using Jaccard's similarity coefficient.

RESULTS AND DISCUSSION

A total of 97 primer-pairs were designed for SSRs lying in mungbean scaffolds harbouring R genes, of which 74, 17, 2, and 4 primers were designed targeting di-nucleotides, tri-nucleotides, tetra-nucleotides and penta-nucleotides, respectively. Out of 97 SSR primers, 68 SSR primers (70%) showed amplificationin blackgram. Thirty of the primers showing amplification were randomly selected for genetic variation analysis in 44 blackgram genotypes differing in disease reaction to YMD and PMD. These 30 primers belonging to different scaffolds of mungbean (**Table 2**) harboured TMV resistance protein N, DNAdamage-repair/toleration protein DRT100, probable disease resistance protein At4g33300, protein suppressor of npr1-1, constitutive 1, putative disease resistance protein RGA4, and different putative resistance proteins. All 30 SSR primers collectively amplified 90 alleles in blackgram genotypes with an average of 3 alleles/locus. Twenty-eight of the thirty primers screened were found to be polymorphic with PIC ranging from 0.01 to 0.86 with an average of 0.43 (Table 2). The PIC values ranged from 0 to 0.86 and 0.11 to 0.67 for di-nucleotide and trinucleotide repeat motifs, respectively. Representative DNA amplification of blackgram genotypes using mungbean sequence derived SSR marker MRGSSR 118 is shown in Fig.1. The transferability of mungbean based SSR markers to blackgram was found to be 70% which is high compared to other similar reports such as 50% for cowpea unigene-SSR markers (Souframanien et al., 2017) and 68% collectively for azukibean, common bean, cowpea and mungbean (Souframanien and Gopalakrishna, 2009). The extent of transferability of SSR markers depends on evolutionary relationship between the species and conservation of PCR primer binding sites flanking the SSR motifs (Souframanien et al., 2017). The cross-species amplification of SSR markers from mungbean indicates that the sequence flanking the SSRs are conserved and between mungbean blackgram. Similarly, microsatellite markers were reported to be transferable in Phaseolus (Gaitan-Solis et al., 2002) and major pulses (Pandian et al., 2000). High transferability rate and less frequent null alleles observed in this study in comparisonto other reports could be due to use of genomic SSR markers which are not associated with problems of disrupted priming sites due to intron splice sites, large introns and additionally markers used in this study were developed from mungbean which is more closely related to blackgram compared to other Vigna species (Zukovaskij, 1962).

In the present study, allelic variation at 30 SSR loci with an average PIC of 0.43 which is comparable to genomic SSR markers from other Vigna species (Souframanien and Gopalakrishna, 2009) and supports utilization of these resistance genes based genomic-SSR markers in blackgram. MRGSSR12 and MRGSSR110 designed for di-nuleotide repeats $(AT)_{13}$ and $(AT)_7$ were found to be highly polymorphic with PIC values of 0.86 and 0.83, respectively. These highly polymorphic markers were derived from scaffolds homologous with TMV resistance protein N and putative disease resistance protein At4g11170. Di-nucleotides based primers were observed to exhibit high PIC value which is consistent with the earlier reports of such primers derived from cowpea (Souframanien and Gopalakrishna, 2009) and soybean (Hisano et al., 2007). Similarly, significance of variable repeat motifs can be comprehended by their positional effect. When present in the coding sequences or regulatory regions they could cause a frame shift, alteration of gene expression, inactivation of gene activity, and/or a change of function, and eventually phenotypic changes (Li et al., 2004).

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Table 2. Details of the 30 SSR markers developed from mungbeanWGS scaffolds harbouring nucleotide sequences homologous to putative resistance

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Matrix Putative function Registion Reposit Forward primer Putative function Rel JM00100053 (A)T ATGGAACCTGAACATGAACAT Prelini suppresson of npr1-1, constitutive 1 Rel JM00100053 (A)T ATGGAACTGAACATGAACAT Revease resistance protein N Rel JM00100053 (A)T ATGGAACTGACAACTGAACAT Revease resistance protein N Rel JM00100053 (A)TS ACTGCATCACACATGAACAT Revease resistance protein N Rel JM001000242 (A)TS ACTGCATCAGCAACTCACACAT Revease resistance protein N Rel JM001000242 (TA)S ACACCTTCACCACTCACACAT ACACCTACTCACACAT Revease resistance protein N Res JM001000242 (TA)S ACACCTTCACACATCACAT ACACCTACTCACACT Revease resistance protein N Res JM0010001242 (TA)S ACACCTACTCACACTCACAT ACACCTACTCACAT Revease resistance protein N Res JM001000124 (TA)S Revease resistance protein N Revease resistance protein N Res JM001000124 (TA)S REVCATATTCACACTCACAT								
JJM001000583 (aC)7 ATGTGGTGGTAGTGGTAGGTAGGTAG Protein su JJM001000248 (TTA)8 CCTTTGTGTTAAGTGGAAAA Probein su JJM001001488 (ATG)6 GCGACTTGTGTTTCAATGTATTCAATTAA TWV resis JJM001001488 (ATG)6 GCAACGTTGTGTTTAAGTGGGCTAAAGATTTCCACCTAACATTAA TWV resis JJM001001482 (ATC)8 CCTTTGTTAAAACGTACCAACGAAAA Probenie JJM001001482 (ATC)8 CCTTTGTTAAAACGTACCAACGAAAGTTAGGGGCT TWV resis JJM00100120369 (TA)8 CCCAATCCCTGCAACGTTAACGATGGGGCT TWV resis JJM00100120369 (TA)8 CCCAATCCCTCCACGACTTAAACTATAGGGGCTAAAGTTAAGGGGCAA TWV resis JJM0010012209 (TA)8 CCCAATCCCTCCACCAACGTTAACGAGGGCTAAGGTTATAGGGGCTAAAGTTAAGGGGCTAAAGTTAAGTCCCACGAAGTTAAGTGCTCCAAGGTTACGATGCTCTCAAGTTAAGGGGCTAAAGTTAAGGGGCTGAAAGTTAAGTGCACGAAGTTAAGTGCACGAAGTTAAGTGCACGAAGTTAAGTGCACGAAGTTAAGTGCACGAAGGTAAACGTACGAAGTTAAGTGCACGAAGGTAAAGTAAGT	Marker	Mungbean sequence ID	Repeat motif	Forward primer	Reverse primer	Putative function	No. of alleles	PIC
JM001000123(GA)7AGGGACTCTAGCAATIGGAAGAGAGAGAGAGAGAGAGAAGACGAAAProbableJM0010001483(TTA)8CCTTTCTCCCTTGACTTACACMV resisJM0010012389(AT)13CATACATCTGCCTTCACCRCCGAGAGATTTCCACCACGAGAGAGCACAAJM001002389(AT)3CATTCTTCCCTGACATGAAAATCGCCTCAAGTTGGCGCGAGAAGTGCGCJM001002389(AT)8CATTCTTCCCCACATGAAAATCGCTCAAGTTGGCGCGACAAJM001002389(TA)8CATTCTTCCCCGACTTCAACTCACCCCCAAGTTGGCGCGACAACTTACACATTWV resisJM001002389(TGA)7AAACGCGCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC	MRGSSR1	JJMO01000583	(AC)7	ATGTCGTGCATAGTCGTAGGTG	ATTGAAACAGGAGCTTCCAAGA	Protein suppressor of npr1-1, constitutive 1	2	0.01
JM001000647(TT)8CCTTTCTCCTGTCTTCATTMV resisJM001001488(MT)8CCTTTCTCCTGTGTCTTACATGG CCTGAAGTTTATMV resisJM001001488(MT)8GCAACACTTCTCCTTACATGG CCTGAAGTTTATMV resisJM001002289(TA)13CATTGTTAAACGGCACTCAATTMV resisJM001002289(TA)3CATTGTCACGCGCACTTAATACGGGGTAGGTGTCTProbableJM001002289(TA)8GCCCATTCCTCCCGGATTAATACGGGGTAGGTGTCTProbableJM001002399(TA)7AAGGGAAGGGAGGATAACGGGACAACProbableJM001001147(AT)8GCACATTCCTCCCCGCACTAAAACGGGCAGCACTTMV resisJM001001147(AT)8GCACATTCCTCCCCTCCAAGTAATGGATGGTGTCTProbableJM001001147(AT)8GCACATTCCTCCCCCCAAGAACGGGGAAGGGTAGGGTTATMV resisJM001001147(AT)8GCACATTCCTCCCCCCAAGAACCGGGAAGTTCATmresitalioJM001001147(AT)8GCTCAAACCTTCCAGGTAATGATGGGGTAGGGTTAGGGGTTAProbableJM0010001147(AT)8GCCCAAAACCACGCAACCTTAGGGTTAGGGGTTAGGGGTTProbableJM0010001147(AT)8GTCCAAACCTTCCAGGTTAGGGGTTAGGGGTTAGGGGTTProbableJM0010001147(AT)8GTCCAAACTTCCGGGTCAAGGAAGAAGGGGGGTGGGGTTProbableJM0010000122(TA)17AATTGTTCGGGGGTCAAGGAAGAAGGGGGGGGGGTGGGGTTProbableJM0010000122(TA)17AATTGTTCGGGGGTGGGGTGGGGTTProbableJM0010000122(TA)17AATTGTTCGGGGGGTGGGGGTGGGGTTProbableJM0010000122(TA)17AATTGTTGGGGGGGGGGGGGGGGGGGGGGGGGGGGGGG	MRGSSR3	JJMO01000123	(GA)7	ACGGACTCTAGCAAATGGAAAG	ATGGGAACCAAGAAGACAGAAA	Probable disease resistance protein At5g66900	2	0.33
JJM001001488 (ATG)6 GCAACATTGCTTTACATGS CATTACATGS CATTACATGS TW resis JM001001488 (ATG)8 CATTGTTAAACGTACCATGS TW resis JM001002289 (TAC)8 CATTGTTAAACGTACCATGTATGGGGTAAGCTC Probable JM001002289 (TAC)8 CACATTCCTCCCACTTAATACC AGGCTATGTCGGGTAAACGGGGTAGCTCA JJM00100289 (TG)7 AAAGGGAAGGGTTAACGCACATAW resis JJM00100283 (AG)7 AAAGGGAAGGGTTAACCAAACGGGCTAGGGGTAAGCAT JJM001001147 (AT)8 GCACATTGTCGCACCTCAAGGTTGAAAGCC JJM001000184 (AT)8 GCACCTTGTCGACCATCCTCCCGTATGATGGGGTAGGTTGAAAGC JJM001001147 (AT)8 GTAGAAGGGAAGGGTAACCCAAAGCACT JJM001000194 (AT)8 GTAGAAATGCTTCCTCCTCTCCG JJM001000194 (CT)7 GGGGTTGAAACGGGAAG JJM001000194 (TA)13 GTTCGACTGCACTGAAGCTTTAGGCCAACTTTAGGCCAACT TACAGGCACAACCCTTCGGGG JJM0010001147 (AT)6 GTCCAAGCTTCGCG JJJM001000147 (ATA)6 GTCCAAGCTTGCGCAGG JJJM001000147 (ATA)6 GTCCAAGCTTTGGGCACAACCTTTGGGGGTCT Putative d JJM001000147 (ATA)6 GTCCAAGCTTTGGGCACTGCAACCTTTGGGGGTCT Putative d JJM001000147 (ATA)6 GTCCAAGTTTGGGGTCTGGGGT CTCCAGGGGCCAACT JJJM001000147 (ATA)6 GTCCAAGCTTTGGGGCACAACCCAGGCACCTCCAATTG JJJM0010000122 (TA)17 AATTGTTGCGGTCTGGGGG AACCAGGCACAAGGAAGGGAGGGAGGAGGGGGGGT DNA-dar JJJM0010000122 (TA)17 AATTGTTGCGTGGTGG ACCAGGGCACCAAGGAGGGGGGT DNA-dar JJJM0010000122 (TA)6 GTCCAAGTTTCGGGGTGGAACAAGGGAAGAAGGGGGAGAA JJJM0010000122 (TA)6 GTCCAAGTTTCGGGGTGGGGGTG DNA-dar JJJM0010000122 (TA)6 GTCCAAGTTTCGGGGGGGGGGGGGGGGGGGGGGGGGGGG	MRGSSR9	JJMO01000647	(TTA)8	CCTTTCTCCCTGTCATCTTCAT	GCCAGAGATTTCCACCTACAAT	TMV resistance protein N	2	0.39
JJMO01002369 (AT)13 CATTGTTAAACGTACCACGGS AAATCGCCTCAAGTATGGGGA TW resis JJMO01001422 (ATC)8 AAATCACACACGCACTCAAT TW resis JJMO01002389 (TG)3 GCCTTATTGCGACTCACTCATCA TGGGTGAAGGGGTGAAGCT TW resis JJMO01002389 (TG)3 GCCCTTTCTCCCCACTCAAGTAGTGGGTGAAGCAT TW resis JJMO01001147 (AT)8 GCACTTCTCCCCACTCATCA AGAAGAAACAGGGGTGAAGCA TW resis JJMO01001147 (AT)8 GCACTTCTCCCACGCTCTTCAG AGAAGAAACAGGGGTGAAGCA TW resis JJMO01001147 (AT)8 GTCGAAGTGCTTCCTTCCAG JJMO01001147 (AT)8 GTCGAAGTGCTTCCAGGG GCACAATCCCACAGGGGTTCA Transitio JJMO01001147 (ATA)6 GCTTCAGCTCTTCGGG GCACAATCCCACACGGGGTTCA Transitio JJMO01001147 (ATA)6 GCTTCAGCTTCGGGG GCACAATCCAAACCAGGGGTTCA Transitio JJMO01001147 (ATA)6 GCTTCAGCTTCGGG GCACAATCCCAAACCAGGGGTTCA Transitio JJMO01001147 (ATA)6 GCTTCAGGCTCTCGG GCTTCAAACCTTTCGGCTTGGG GCTTCAAACCTTTGGGCTCGGG GCACAATCGGGGTTCA Transitio JJMO01001147 (ATA)6 GCTCATCTTGGGCTCGGGGTCACGGCCACTTGGGGTTCA Transitio JJMO01000121 (TA)13 TGTTGGGGTTGGCGGG GCACAACCCAGGGCACGCGGGCGACGCCCACCTTAAT DNA-dam JJMO01000122 (TA)13 TGTTGGGGTTGGCGGGCT ACCAACCTTTGGGCTGGGGGGGAGGGCCACCCACTTAAT DNA-dam JJMO01000122 (TA)13 TGTTGGGTTGGTGGGCACACCCACCTTAAT DNA-dam JJMO01000122 (TA)13 TGTTGGGTTGGTGGGAAACCCGGACCACCACGAGGGCTAAACCAGGGGTTAA JJMO01000022 (TA)13 TGTTGGGTTGGTGGGGAACCCGGGCTAAACCAGGGGTTAACGGGGTTAA JJMO01000022 (TA)13 TGTTGGGGTTGGCGGGTCAACCAGGGGTTAACGAGGGGTAAACGGGGTTAAC JJMO0100022 (TA)13 TGTTGGGGTTGGGGGTGAACGCGGCACACCAGGGGTTAACGAGGGTTAACGAGGGGTAAACGAGGGGTAAAGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAACGAGGGGTAAAGAGGGGTAAACGAGGGGTAAAGAGGGGGTAAAGAGGGGTAAAGAGGGGGTAAAGAGGGGGTAAAGAGGGGGTAAAGAGGGGTAAAGAGGGGGTAAAGAGGGGGTAAAGAGGGGTAAAGAGGGGGTAAAGAGGGGTAAAGAGGGGTAAAGAGGGGTAAAGAGGGGTAAAGAGGGGGTAAAGAGGGGAAAGAGGGGAAAGAGGGGTAAAGAGGGGTAAGAGGGGTAAAGAGGGGTAAGAGAGGGGTAAGAGGGGTAAGAGGGGGTAAGAGAGGGGTAAGAGGGGTAAGAGGGGTAAAGAGGGGGTAAGGGGGTAAGGGGGTAAGAGGGGTAAGAGGGGGTAAGAGAGGGGTAAGAGGGGTAAGAGAGGGGGG	MRGSSR10	JJMO01001488	(ATG)6	GCAACACTTCTGCTTTACATGG	CACTTACATGGCCTGGATTTTA	TMV resistance protein N	С	0.33
JJMOO1001492 (ATC)8 AATACACACACGCACGCACTAATAC GGCTATGTTCGAGTGCTC Disease r JJMO01002369 (TA)8 CCCAATCCCCGCACTAATACC AGGCTATGTTCGGATGCTCCT Disease r JJMO01002369 (TA)7 CCCAATCCCCCGCATTCAAATACCCCCCACAGGCACAA TWV resis JJMO01001147 (AT)8 GCACTTCTCCCTCCTCCAGGG AGCAAAAGC Protein su JJMO01001147 (AT)3 GTTCAAATGCTTCCCTTCCAGGG GCACAAACCCAAAGCC Putative d JJMO01001147 (AT)3 GTTCAAATGCTTCCCTTCCAGGG GCACAAAACCCAAATGCATTCAATTG AJJMO01001147 (AT)8 GTTCAAATGCTTCCCTCCCGTCAGGG CACAAAACCCAAATGCATCAATTG AJJMO01001147 (AT)8 GTTCAAATGCTTCAGGG GCACAAAACCCAAATGCAAACCAAAAGC Protein su JJMO01001147 (AT)6 GGGTTGGGATTGGTTGGGTGG AGCAATGCCAAACCAACTAAAGC JJMO01001147 (AT)6 GGGTTGGGATTGGTTGGGGATGAAACCAACTAAGGCAACTTTGGGCAACTAA CTTCAAATGCTCCCTCCCCCCAGTGA ACCAGCAACACCAACCAACTAA JJMO01000121 (TA)13 TGTTGAGAAATGGGCATGAAAAGC ACCAGCAACTAATGAAAGCA CTTCAAATGCTCCCTCCGGGCAAAATGGGCCCTGGGAAATGAG AAAGCAACAACAACTAAA JJMO01000121 (TA)13 TGTTGAGAAATGGGTCGGAATAA DNA-dam JJMO01000122 (TA)17 AATTATGTTGCGGTGG AGCAAACAACACAGGGGAAAAAGCA CCCCAAAACAACAACAACAACAACAACAACAACAACA	MRGSSR12	JJMO01002369	(AT)13	CATTGTTAAAACGTACCAACGG	AAATCGCCTCAAAGTATAGGGAC		7	0.86
JJMO01002209 (TA)8 CCCATCCCGACTTAATAC AGGCTATTCAGATGCTGCT Disease rd JJMO01002369 (TA)9 CCCATCCCCGACTTAATAC AGGCTAGTGCTGCA TWV resis JJMO0100136 (TG)7 CCCATCTCTCCCCTCTCAGGGCGACAA TWV resis JJMO01001147 (AT)8 GCACTTGCTCACCATCAGGGCGAGAAATCCCCCAAGCACTT TWV resis JJMO01001147 (AT)8 GTAGAATGCTTCCCCTTCAGG ATTCTTCCCCCAAACCCAGGTCA Translatio JJMO01001147 (AT)8 GTAGAATGCTTCCAGGCACTTCAGGGCTA TATAIGATTGGGCGAACT (AT)8 GTAGAATGCTTCCTCCTCTCAGG GCACAACCAGGCACTTAGAAACC JJMO01001147 (AT)8 GTAGAATGCTTCAGGGTCAACT Disease rd JJMO01001147 (AT)8 GTCCATCTTTAGCCATGG ATTCTTCCCCAAACCAGGCATT Divative d JJMO01001147 (TA)13 GTCGAGATTGGCTCAGG (AT)13 GTTGGAGAATGGCTCACG (AT)17 ATTTGTTGGCGTCAACT (TA)17 ATTTGTTGGCGTCATCA (TA)17 ATTTGTTGGCGTCAACT (TA)17 ATTTGTTGGCGTCAACTTTGGGCACTTAGGCCAACT DJMO0100012 (TA)17 ATTTGTTGGGTGATGT (TA)17 ATTTGTTGGCTCATCACGG (TA)17 ATTTGTTGGGTGATGTTGG (AG)9 GGGGGGGGTGGGGGGG (TA)22 (TA)17 ATTTGTTGGGGGGGTG (TA)17 ATTTGGTGGGTCATCACGGGCAACGGGCAGGGGTG PUABIWG (CT)7 (TA)32 ATCATTCTGGGTGGTG (TA)17 ATTTGGTGGGTCATCACGGGGAGGGGGGGGGGGGGGGGG	MRGSSR13	JJMO01001492	(ATC)8	AAATACACACGCGCACTCACAT	GTTGGGTGAAGGGTAAGCTC	Probable disease resistance protein At4g33300	-	0
JJMOO1002369 (Ta)9 GCACCTATGTTGAGATCCATGA AGAAACAGGGCAGA TMV resis JJMOO1002369 (TGA)7 ACACCTTCCTCCACTCCTTCA GAAGAAACAGGGCAGCAT TMV resis JJMOO1001147 (AT)8 GTAGAATGCTTCCTCCTTCCA ATTCTCTCCCCACAGGCACT TmV resis JJMO01001147 (AT)8 GTAGAATGCTTCCTCCTTCCA ATTCTTCTCCCCAGATCA Translatio JJMO01001147 (AT)8 GTAGAATGCTTCCTTCCA JJMO01001147 (AT)6 GTGCATGCTTCAGGG GCACATCCCAGAATCA Translatio JJMO01001147 (AT)6 GTGCATCTTTAGCCATGA GGGTTGGAATGGCTCAGGG GCACATCCAGGGGTCT Putative d JJMO01000121 (TA)13 TGTTGGAATGGCTTTAGCCATGA GTCCAACCCTTCAGGG GCACATCCCCGGATTCA Translatio JJMO01000121 (TA)13 GGGTTGGAATGGCTTGGGGGTCT Putative d JJMO01000121 (TA)13 TGTTGGAGATTGGCTTGGGGGTCT Putative d JJMO01000121 (TA)13 TGTTGGGAATGGCCATGGCAACTCTGGGGGTCT Putative d JJMO01000121 (TA)13 TGTTGGGAATGGCCATGGCAACTCCGGAATAA DNA-dam JJMO01000121 (TA)13 TGTTGGGAATGGCCATGGGGAAGGAATGGCT Putative d JJMO01000121 (TA)13 TGTTGGGAATGGCCATGGGGAAGGAATGGCT Putative d JJMO01000122 (TA)17 AATTATGTTGGGAGAGGAAGGAATGGGGTTG Putative d JJMO01000122 (TA)23 GGGAGGGGGGGGGGGGGGGGGGGGGGGGGGGGGGGGG	MRGSSR20	JJMO01002209	(TA)8	CCCAATCCCCGACTTAAATAAC	AGGCTATGTTTCAGATGCTGCT	Disease resistance RPP13-like protein 4	9	0.75
JJMOO1002369 (TGA)7 ACACCTTCCTCACTCCTTA GATCACCCACAGCACT TW resis JJMOO1001147 (AT)8 GTAGAATGCTTCCTCTCCACTCCATGGAGGTGAAGC Putative d JJMO01001147 (AT)8 GTAGAATGCTTCCTCCTTCCA JJMO01001147 (AT)8 GTAGAATGCTTCACTCAGGG GCACATCCAGAAGC Putative d JJMO0100147 (AT)8 GTGGATGGATTGGGGTTGGGGGTCT Putative d JJMO0100147 (AT)6 GGGTTGGATTGGTCGTGACTTTGGGGGTCT Putative d JJMO0100147 (TA)16 GTCCATGCTTTGGCACTCACACCAGGATCA Translatio JJMO0100147 (TA)16 GTCCAACCTTTGGCACTCACACCAGGATCA DNA-dam JJMO01000121 (TA)13 TGTTGGAGACTCACC TGCAGACCTTTGGGCACTCACTCATTAGGCCATCA JJMO01000121 (TA)13 TGTTGGAGACTCACC TGCAGACCTTGGGGCACTCATTAGGCCATTAG JJMO01000121 (TA)13 TGTTGGAGACTCACCACGCACTCTAAT DNA-dam JJMO01000121 (TA)13 TGTTGGAGACTCACC TGCAGACCTTGGGCACCTTGGGCA TCCCGGAACCCTTGGGCACGCTGGGGGAGGAGGACTGCATTAA DNA-dam JJMO01000122 (TA)13 TGTTGGGACACTCACCACGCACTCAATGCACT Putative d JJMO01000123 (TA)23 TCCACCTCTGGGGGAGGAGGAGGAGGAGGAGGAGGATGCG Putative d JJMO01000123 (TA)6 GTCAACCTTCGGCGGG GAGGAGGGGGGGGGGGGGGGGG	MRGSSR32	JJMO01002369	(TA)9	GCACCTATGTTGAGATCCATGA	AGAAAGAAAACAGGGCAGACAA	TMV resistance protein N	7	0.81
JJMO01000583(AG)7AAAGGGAAGGGTAACGGAAGProtein suJJMO01001147(AT)8GTAGAATGCTTCCTCCTCCAATTCTCTCCCGTATTGAAAGCPutative dJJMO01001466(AT)3CTTCACATGCTACATTCGGGGGCAATACCAAACCGATTCATranslatioJJMO01001466(CA)7GGGGTTGGGTATGGGGTTGGGGCTTAGGGGTTGPutative dJJMO01001477(AT)8GCGATCACTTTAGCCATGGTCCATTGGGGGCTTAGGGCTTAGGCCAACTDisease rJJMO01001477(AT)8GTCCAAACCTTTAGCCATGGTCCAGGCAACTTTAGGCCAACTDisease rJJMO01000121(TA)13TGTTGGGGAATGGGCCACCTTCCAGGCAACTTGGGCCAACTDisease rJJMO01000122(TA)13TGTTGGTGGCTCACCAGGGACCAGGAACCCAGCAATGGCCAACTDisease rJJMO01000122(TA)17AATTATGTTGGGGGCTGGGGGGGCAACTGGGCCAACTGGCAATGGCAATGGCAAGGGCGGGGGGGG	MRGSSR34	JJMO01002369	(TGA)7	ACACCTTCCTCACCACTCCTTA	GATCAAATACCCCCACAAGCACT	TMV resistance protein N	ო	0.67
JJMO01001147(AT)8GTAGAATGCTTCCTCTCCAATTCTCTCCCGTATTGAAAGCPutative dJJMO01001646(CA)7GGGGTTGGGATTCGTranslatioJJMO01001646(CA)7GGGGTTGGGGATTGGTranslatioJJMO01001646(CA)7GGGGTTGGGGATTGCAACCAACCAACCAACCAATGGGTTCATranslatioJJMO01001477(ATA)6GTCCAAACCATTTGGCCAACTDisease rJJMO0100121(TA)13GTCCAAACCTTCAGACCACCACCACCACCAATGGCTDNA-damJJMO01000122(TA)17AATTATGTTGGCGCACTGCAACTGGCCAACTGGCAACTDisease rJJMO01000122(TA)17AATTATGTTGCGGCACTGCAGCACCAGGCACTGGCAACTGGCAACTGGCAACTGGCAACTGGCAACTGGCAACTGGCAACGGCCACCAGGCAAGGGTGGCAACTGGCAACTGGCAACGGCACCAGGGCAACTGGCAAGGGCAGGGTGGGCGGGGGGGG	MRGSSR36	JJMO01000583	(AG)7	AAAGGGAAGAGGTAAACGGAAG		Protein suppressor of npr1-1, constitutive 1	2	0.37
JJMO01000120(AT)33CTTCACATGCTACATCAGGGTranslatioJJMO01001646(CA)7GGGTTGAGATTTGGTGAGATTGGTGAGGPutative dJJMO01001147(TJ)7GGGTTGAGACTTTAGCCATGATranslatioJJMO0100121(TJ)1GGGTTGAGACTCACCTACCAGCAACTTAGGCCAACTJJMO0100121(TJ)13TGTTGAGACTTTCAGACTCACTACCAGCAACTGAATAJJMO01000122(TJ)13TGTTGAGAATAGGACTCACGTACCAGCAACTGAATAJJMO01000122(TJ)13TGTTGAGGAATAGGACCAGGTACCAGCAACCAGGAATAJJMO01000122(TJ)13TGTTGGGGACATAGTTGAGGGTACCTCCAGATGACTAATJJMO01000122(TJ)13TGTTGGGGACATAGTTGAGGGTACCTCCAGATGACTJJMO010002047(TJ)2TTTGGTGGCGCTGGGGAGGGGGGGGGGGGGGGGGGGGGG	MRGSSR39	JJMO01001147	(AT)8	GTAGAAATGCTTCCTCCTTCCA	ATTCTCTCCCGTATTGAAAGC	Putative disease resistance protein RGA4	2	0.72
JJM001001646(CA)7GGGGTTGGAGATTGGTGTAGTPutative dJJM001000319(CT)7CATGATCCATCTTTAGCCATGATACCAGGAACTTAGGGCCAACTDisease rJJM001000121(TA)16GTCCAAACCTTTCAGACTCACGTACCAGCAACTTAGGCCAACTDisease rJJM001000121(TA)13TGTTGGGAAATGGGACCTCACGACCAGGAACCGGACTGGAATAADNA-damJJM001000121(TA)17AATTATGTTGGCGTCATCACGGACCAGGAACCGGACTGGAATAADNA-damJJM001000122(TA)17AATTATGTTGGCGTCATCACGGGACCAGGAACCCGGATGAADNA-damJJM0010002047(TA)32ATCATTCTTGATGTTGCGGAGGGAAAGCAAAGGAACCACGAGGGATProbable oJJM0010002047(TA)32ATCATTCTTGATGTTGGGAGGAGGAGAACGAGGGATProbable oJJM0010002047(TA)23TCAATCGTTGGGGGGGGGGAGGAGGGAGAACGAGGAGGAGGAGGAGGAG	MRGSSR45	JJMO01000120	(AT)33	CTTCACATGCTACACTTCAGGG	GCACAATACCAAACCAGATTCA	Translation factor GUF1 homolog, mitochondrial	2	0.26
JJM001000319(CT)7CATGATCCATCTTTAGCCATGATACCAGCAACTTTAGGCCAACTDisease reJJM00100121(TA)16GTCCAAACCTTTCAGACTCACCTACCAGCAACTTAGGCCAACTDisease reJJM00100121(TA)13TGTTGGGAAATAGGAACTCACGACCAGGAACTGGACTAATDNA-damJJM001000122(TA)17AATTATGTTGCTGCGTCATCAGGACCCGGAAACCGGACTAGGATAADNA-damJJM001000122(TA)17AATTATGTTGCTGGGGAAACCAGCAACTGCAATGGCTProbable oJJM001000122(TA)32ATCATTCTTGATGTTGGGGGGAACCAGCAACCACCAACGGGATADNA-damJJM0010000127(TA)32ATCATTCTTGGTGGGGGAACCACACCACCACCACCACAGGGTDNA-damJJM001000850(TA)23TCAACCATCCATGTGGGGGGGGGGGGGAATGGGGGTProbable oDNA-damJJM001000850(TA)23TCAACCATCCACCTGGGGGGGGGGGGGGGAATGGGGGGGG	MRGSSR48	JJMO01001646	(CA)7	GGGGTTGAGATTTGGTGTATGT	TCATTTGTGAGGCTTAGGGTCT	Putative disease resistance RPP13-like protein 1	ო	0.7
JJM001001477(ATA)6GTCCAAACCTTTCAGACTCACCTTGCAGACCTCAATDNA-damJJM001000121(TA)13TGTTGAGAAATAGGACCCTGGACCCGAAACCGCTAGATAADNA-damJJM001000122(TA)17AATTATGTTGCGTCATCACGGACCCGAAACCGGACTAGAATAADNA-damJJM001000122(TA)17AATTATGTTGCGTCATCACGGAAGCAAACCAGGACTAGAATGCTProbable oJJM001000122(TA)17AATTATGTTGCGTGATGTTGCGGGGAAGCAAACCAGGGACAGGGTProbable oJJM00100012047(TA)32ATCATTCTTGATGTGGGAGGGGGAAGCAAACCACAGGGGATGPutative dJJM001000772(AG)9GAGAGAGGCCTGGGGAGGAGGAGGAGGAATTGCGGGATDNA-damJuA-damJJM001000783(TA)23TCAACCATCCCAGGTGGGGGGGGGGGGGAGGAAATTGCGGGATDNA-damJuA-damJJM001000783(TA)23TCAACCATCCCAGGGAGGAGGAGGAGGAGGAGGAGGAAPutative dJuA-damJJM001001477(AT)38GTTCGTTCGGCTGCGGAGAAAGGCGAGGAGGAGGAGGAGGAGAAAPutative dJJM001001477(AT)38GTTCGTTCAAAGCCCACCAGGGCGAGGAAAPutative dJUM001001477ACGACTTCCACCACGGGATTGGPutative dJJM001001477(AT)38GTTCGTTCAAAGCCCACGGGATTGGGAGGCCTTTTGTTGTTGGGCGAGGAAAPutative dJUM001001477ACGACTTCCACCACGCGCTTCAAAGGCCGTTPutative dJJM001001477(AT)8GTTCGTTCCACCACGGGATTGGAGGCCTTTTGTTGTGGGCGGGGGGGGGGGGGGGGGGGGG	MRGSSR51	JJMO01000319	(CT)7	CATGATCCATCTTTAGCCATGA	TACCAGCAACTTTAGGCCAACT	Disease resistance protein RPP8	5	0.81
JJMO0100121(TA)13TGTTGAGAAATAGGACCTTGGAccontronoonAnthartGTTGCGTCACAGGDNa-damJJMO01000122(TA)17AntharGTTGCGTCATCAGGTTACTCCGGTTGCAAATGCTProbableJJMO01000102(CT)7TTTGGTGACATGGTTGGGGGCAGCCAAAGGGAAGGAATGGGGGTTGProbableJJMO01000102(TA)32ATCATTCTTGATGTTGGGGGGGAGCCAAAGGGAAGGAATGGGGGTTGProbableJJMO01000722(AG)9GAGGAGGAGGAGGAGGAGGAGGAGGAGGAGGAGGAGGATGGGGGTTProbableDNa-damJJMO01000722(AG)9GAGGAGGAGGAGGAGGAGGAGGAGGAGGAGGAGGAGGAG	MRGSSR56	JJMO01001477	(ATA)6	GTCCAAACCTTTCAGACTCACC	TTGCAGACACAGCCACTCTAAT	DNA-damage repair / toleration protein DRT100	9	0.32
JJMO01000122(TA)17AATTATGTTGCGTCATCACAGGTTACTCCAGTTTCCCAAATGCTProbableJJMO01000040(CT)7TTTGGTGACTAGTTGGGGGCAAAGCAAGGAAGGAAGGAGGGTTGPrutative dJJMO010002047(TA)32ATCATTCTTGATGTTGCTGGGGGAAGCAAGGAGGAGGAGGATGGGGGTTPutative dJJMO01000850(TA)23ATCATTCTTGATGTTGCGGAGGGGGGGAAGAGGAGGAGGATGCGGGATDNA-darrJJMO01000853(GA)7TCAACCATCCCAGGTTTTCTGAAACACACACACACACACACACACACACACACACACAC	MRGSSR57	JJMO01000121	(TA)13	TGTTGAGAAATAGGACCCTTGG	ACCCGAAACCCGACTAGAATAA	DNA-damage repair / toleration protein DRT100	2	0.55
JJMO0100040(CT)7TTTGGTGACATAGTTTGAGTTTGAGTTTGAGTTTGAGGTTGPutative dJJMO01002047(TA)32ATCATTCTTGATGTTGTGTGGGGTTGCAAGGGAGGAGGAGGGTPNA-darrJJMO01002047(AG)9GAGGAGGAGCCTGGGAGGAGGAGGAGCACAGAGGGGDNA-darrJJMO01000850(TA)23TCAACCATCCATCCAGGTGGGGGAAACACACACACACACAGAGGATDNA-darrJJMO01000853(GA)7TCAACCATCCAAGTTCTGAAACACACACACACACACACACACACACACACAPutative dJJMO01000853(TG)7TCTAATCAAATTCCATCGGCTGGGAGAAAGGACGAAGGAGAAAProtein suJJMO01000853(TG)7ATCAAGCAGCCCTGGCTGAAACACACACACACACAGAGAAPutative dJJMO01000452(TG)7ATCAAGCAGCCCTTGTTCCTTGGATGGGPutative dJJMO01001477(AT)38GTTCGTTCCCTTTCTTTGTTGAGGTGGATGGPutative dJJMO01001477(AC)8ACCAACTTCAAGGCCAATGTGAGGCCTCTTTGTTGAGGGTGGATGGGPutative dJJMO01001477(AC)8ACCAACTTCAAGGCCAATGTGAGGCCTCTTTGTTGGGGGGGGGGGGGGGGGGGGGGGGGG	MRGSSR65	JJMO01000122	(TAA)17	AATTATGTTGCGTCATCACAGG	TTACTCCAGTTTCCCCAAATGCT	Probable disease resistance protein At4g33300	4	0.11
JJM001002047(TA)32ATCATTCTTGATGTTGCTGGTGTTGCAGGGTCAAACACAGGGGTDNA-darrJJM001000772(AG)9GAGAGAGCCTGTGGAGGGGGATGTTACTGGAATTGCGGATDNA-darrJJM001000850(TA)23TCAACCATCCCAAGTTTTCTGAAACACACACACACACACACACACACACACACACACAC	MRGSSR77	JJMO01000040	(CT)7	TTTGGTGACATAGTTTGAGGCA	AAGCAAAGGAAGAATGAGGTTG	Putative disease resistance RPP13-like protein 1	2	0.63
JJM001000772(AG)9GAGAGAGCCTGTGGAGAGAGAGAGAGAGATTGCGGATDNA-darJJM001000850(TA)23TCAACCATCCAAGTTTTCTGAAACACACACACACACACACACADNA-darJJM001000853(GA)7TCTTAATCAAATTCCATCTCGCTCCAGCTACTATTCCCTCGAAPutative dJJM001000523(TA)6TCTAATCAAATTCCATTGCGAGAAGAGAGAGAGAAAProtein suJJM001000523(TA)738CCTTAATCAAATTCCATTGCTAAGCCTCTTTGTATAGACCACACACACACACACACACACA	MRGSSR82	JJMO01002047	(TA)32	ATCATTCTTGATGTTGCTGGTG	TTGCAGAGTCAAACACAGAGGT	DNA-damage repair / toleration protein DRT100	-	0
JJM001000850(TA)23TCAACCATCCAGTATTTCTGAAACACACACACACACACADNA-darrJJM001000853(GA)7CCTTAATCAAATTCCATCTGGCTCCAGCTACTATTCCCTGGAAPutative dJJM001000853(TG)7TCTATGATCCAATTCCATCGCCAGCACCACACACACACACACACACACAAPutative dJJM001000452(TG)7ATCAAGCAGCACCTTGTCTCAAGCCTCTTGTATAGACCGCTTPutative dJJM001001477(AT)38GTTCCTTCCTTGTCTTCTAAGCCTCTTGTAGGCTGGAAAPutative dJJM001001477(AC)8ACCAACTTCCAAAGCCAATGTGPutative dTCTCTTTCCTTGAGGGTTAGGCTAAGTGGPutative dJJM00100182368(GAT)8ACCAACTTCCACCACAGGGTTAGATTTAGTTTGTTGGGCGAGTGGPutative dJJM001002368(GAT)8AGTTCCACCACCAGGGTTAGAGTTGGCATCAATAGGCGPutative dJJM00100129(TTJ)9GCCAAGGAGGATGGAGGAGGAGGAGGAGGAGTGGPutative dTTTTTTTTTTGGCACCAATAGGAGPutative dJJM00100124(TTJ)9GCCAAGGATTGGAAGAGAGAGTGGPUtative dTTTTTTTTTTGGCACCAATAGGAGTPutative dJJM001000124(TTJ)9GCCAAGGATTGGAAGAAAAGTCCACCAACAATAGACGTPutative dJJM001000124(TTA)9GCCAAGGATTGGAAGAAAAAGTCCATCAATAGACGTPutative dJJM001000124(TTA)9GCCAAGGAGGAGAGAGAGAGAGAGAGAGAGAGAGAGAGA	MRGSSR84	JJMO01000772	(9G)	GAGAGAGCCTGTGGAGAGAG		DNA-damage repair / toleration protein DRT100	-	0
JJM001000853(GA)7CCTTAATCAAATTCCATCTCGGCTCAGCTACTATTCCCTCGAAPutative dJJM001000583(TA)6TCTATATGATCCTCGGCTGGGAGAAAGACGAAGGAAAProtein suJJM001000452(TG)7ATCAAGCAGAACCCTTGTCTCGAGGCCTCTTGTATGACGCAGTAPutative dJJM001001477(AT)38GTTCGTTCCCTTGTTCTTCTTATGGAGGTTAGGCTGGTGPutative dJJM001001477(AC)8ACCAACTTCAAGCCAATGTGPutative dTTTAGTTTGTTGAGGTGTGGTTGGPutative dJJM00100782(TA)6GTAAGCCTGCCTTCAAGCCAATGTGTTTAGTTTGTTGAGGTGTGGTGAATGGGPutative dJJM001000782(GAT)8AGTTCCACCACCAGGGATTAGAGTTGCATCAATAGGCGAGTGCPutative dJJM0010007286(GAT)8AGTTCCACCACCAGGGATTAGAGTTGCACCAATAGACGPutative dJJM001000129(TTA)9GCCAAGGATTGGAAGAAGAATAGTTGCACCAATAGACGPutative dJJM001000124(TTA)9GCCAAGGATTGGAAGAAATAGTTGCACCAACAATAGACGPutative dJJM001000124(TTA)9GCCAAGAATGGAAGAATAGGAATTTGGAACCAACAATAGACGPutative dJJM001000124(TTA)9GCCAAGAATGGAAGAAATAGACCATGAATTTGGAACCAACAATAGACGAPutative dJJM001000124(TTA)9GCCAAGAATGGAAGAAATAGACCATGAATTTGGAACCAACAATGGAACAATAGACGATAGPutative dJJM001000124(TTA)9GCCAAGAATGGAAGAATTTGGAACCAACAATTTGGAACCAACAATAGAACAATAATAAAAAAAA	MRGSSR86	JJMO01000850	(TA)23	TCAACCATCCCAAGTATTTCTG	AAAACACACACACACACACACA	DNA-damage repair / toleration protein DRT100	6	0.54
JJMO01000583 (TA)6 TCTATATGATCCTCTGGCTCGC GAGAAAGACGAAGAGAGAAGACGAAGAAA Protein su JJMO01000452 (TG)7 ATCAAGCAGACCCTTGTTCTC AAGCCTTTGTATAGACCCGTT Putative d JJMO01001147 (AT)38 GTTCGTTCCTTTCTTTGAGGTGGTGATTGG Putative d JJMO01001477 (AC)8 ACCAACTTCAAGCCAATGTG TCTTTTCCTTGAGGTGGTGG Putative d JJMO01000782 (TA)6 GTAAGCCTGCTTCATATTTC TTTAGTTTGTTGGGCGAGTGG Putative d JJMO01000782 (GAT)8 AGCTCCACCAGGGTTAG AGTTGGGCGAGTGC Putative d JJMO0100072368 (GAT)8 AGCTCCACCAGGGTTAG AGTTGGCATCAATAGACA Putative d JJMO01000129 (AT)7 TTTTCATTCCACCAGGGTTAG AGTTGGCACCAACATAGACA Putative d JJMO01000129 (TTA)9 GCCAAGGATTGGAGAGAAA PCCATGAATTGGAACAACAACAACAACAACAACAACAACAACAACAACAA	MRGSSR91	JJMO01000853	(GA)7	CCTTAATCAAATTCCATCTCCG	CTCCAGCTACTATTCCCTCGAA	Putative disease resistance protein RGA3	2	0.5
JJM001000452 (TG)7 ATCAAGCAGACCCTTGTCTCC AAGCCTCTTTGTATAGACCCGTT Putative d JJM001001147 (AT)38 GTTCGTTCTCCTTTCTTTCTTTGAGGTGTGGATTGG Putative d JJM001001477 (AC)8 ACCAACTTCAAAGCCAATGTG TCTCTTTGCTTGAGGCATAAGTGG DNA-dam JJM001000782 (TA)6 GTAAGCCTGCCGTTCATATTTC TTTAGTTTGTTGGCGAGTGG Putative d JJM001000782 (GAT)8 AGTTCCACCACCAGGGATTAG JJM001000129 (AT)7 TTTCATTCCACCACCAGGGATTAG JJM001000129 (AT)7 GCCAGGACTGGCTCATCATGGCAGATGG Putative d JJM001000124 (TTA)9 GCCAGGACTGGAGTGGAGTGC Putative d	MRGSSR99	JJMO01000583	(TA)6	TCTATATGATCCTCTGGCTCGC	GAGAAAAGACGAAGGCAAGAAA	Protein suppressor of npr1-1, constitutive 1	2	0.32
JJMO01001147 (AT)38 GTTCGTTCTCCTTTCTT ATGGAGGTTAGGTGATTGG Putative d JJMO01001477 (AC)8 ACCAACTTCAAGCCAATGTG TCCTTTCCTTGAGCATAGTGG PNA-dam JJMO01000782 (TA)6 GTAAGCCTGCCGTTCATATTC TTTTAGTTTGTATGGCGAGTGC Putative d JJMO010002368 (GAT)8 AGTTCCACCACCAGGATTAG ACTTGCATCAATAGACA Putative d JJMO01000129 (AT)7 TTTTCATTCCACCACCAGGATTAG ACTTCTTCCGCCCAACAATAGACA Putative d JJMO01000124 (TTA)9 GCCAAGAATGGAAGAAAT AGACCATGAATTGGAACCAGT TMV resis	MRGSSR101		(TG)7	ATCAAGCAGACCCTTGTCTCTC	AAGCCTCTTTGTATAGACCCGTT	Putative disease resistance protein RGA4	2	0.04
JJMO01001477 (AC)8 ACCAACTTCAAGCCAATGTG TCTCTTTCCTTGAGCATAAGTGG DNA-dam. JJMO01000782 (TA)6 GTAAGCCTGCCGTTCATATTTC TTTTAGTTTGGTGGGCGAGTGC Putative d JJMO01002368 (GAT)8 AGTTCCACCACCAGGATTAG AGTTGGCATCAATAGACA Putative d JJMO01000129 (AT)7 TTTTCATTCCACCAGCAGGATTAG AGTTGGCACTCAATAGACA Putative d JJMO01000129 (TTA)9 GCCAAGACTGGATTGGAAGAAAT AGACCATGGAATTGGAACCAGT TMV resis.	MRGSSR102	JJMO01001147	(AT)38	GTTCGTTCTCCCTTTCCTTCTT	ATGGAGGTTAAGGTGTGATTGG	Putative disease resistance protein RGA4	2	0.04
JJMO01000782 (TA)6 GTAAGCCTGCCGTTCATATTC TTTTAGTTTGTATGGCGAGTGC Putative d JJMO01002368 (GAT)8 AGTTCCACCACCAGGATTAG AGTTGGCATCCATCATAGACA Putative d JJMO01000129 (AT)7 TTTTCATTCCACCCGGTCC TTTCTTTCTGCACCCAACATAG Putative d JJMO01000124 (TTA)9 GCCAAGACTGGATTGGAAGAAT AGACCATGAATTTGGAACCAGT TMV resis	MRGSSR103	JJMO01001477	(AC)8	ACCAACTTCAAAGCCAATGTG	TCTCTTTCCTTGAGCATAAGTGG	DNA-damage repair / toleration protein DRT100	-	0
JJMO01002368 (GAT)8 AGTTCCACCACCAGGGATTAG AGTTGGCATCCATCATAGACA Putative d JJMO01000129 (AT)7 TTTTCATTCCACCCGTCC TTTCTTTCTGCACCAACATAG Putative d JJMO01000124 (TTA)9 GCCAAGACAGATTGGAAGAAAT AGACCATGAATTTGGAACCAGT TMV resis	MRGSSR104	JJMO01000782	(TA)6	GTAAGCCTGCCGTTCATATTTC	TTTTAGTTTGTATGGCGAGTGC	Putative disease resistance protein RGA4	2	0.59
JJMO01000129 (AT)7 TTTTCATTCCACCCGTCC TTTCTTTCTGCACCCAACATAG Putative d JJMO01000124 (TTA)9 GCCAAGACAGATTGGAAGAAAT AGACCATGAATTTGGAACCAGT TMV resis	MRGSSR105	JJMO01002368	(GAT)8	AGTTCCACCACCACAGGATTAG	AGTTGGCATCCATCAATAGACA	Putative disease resistance protein RGA4	7	0.55
JJM001000124 (TTA)9 GCCAAGACAGATTGGAAGAAAT AGACCATGAATTTGGAACCAGT TMV resis	RGSSR110	JJMO01000129	(AT)7	TTTCATTCCACCCGTCC	TTTCTTTCTGCACCCAACATAG	Putative disease resistance protein At4g11170	ю	0.83
Total number of alleles and PIC average	MRGSSR118		(TTA)9	GCCAAGACAGATTGGAAGAAAT	AGACCATGAATTTGGAACCAGT	TMV resistance protein N	2	0.52
						Total number of alleles and PIC average	90	0.43

https://doi.org/10.37992/2023.1401.028



Fig. 1. PCR amplification using MRGSSR118 genomic-SSR marker in 44 blackgram genotypes. Lane M 100 bp marker. λ DNA Eco RI and Hind III double-digest marker. Lanes 1 – 44 blackgram genotypes as listed in materials and methods.

Amplification of 30 SSR primers in 44 blackgram genotypes differing in their disease reactions (YMD and PMD) were analysed. In terms of YMD, 3 SSR markers MRGSSR12, MRGSSR56 and MRGSSR77 were differentially amplified predominantly in YMD resistant genotypes in comparison to susceptible genotypes. These markers MRGSSR12 [(AT)₁₃], MRGSSR56 [(ATA)₆] and MRGSSR77 [(CT),] were designed from mungbean scaffolds JJMO01001477, JJMO01002369 and JJMO01000040 exhibiting homology with TMV resistance protein N, DNA damage repair/toleration protein DRT100 and putative disease resistance RPP13-like protein 1, respectively. Similarly, YMD resistant and susceptible genotypes were differentiated using resistance gene analogues derived SSR and ISSR markers, in mungbean and blackgram (Maiti et al., 2011; Gupta et al., 2015; Souframanien and Gopalakrishna, 2009). While in case of PMD, four markers namely MRGSSR12, MRGSSR 32. MRGSSR56 and MRGSSR65 differentiated resistant (IPU02-43, KU96-3, IPU-07-3, LBG-752, LBG-17, LBG-623, LBG-20, Pant U-19, Pant U-31, LBG-709, ANU-11) and susceptible genotypes (DPU-88-31, TAU-1, WBG-57, COBG-653, Sharda Mash). Two of these markers viz., MRGSSR 32 and MRGSSR 65 showed amplification in five and one resistant genotypes, respectively, out of 11 resistant genotypes and were not amplified in five susceptible genotypes studied.

Therefore, two SSR primers, MRGSSR12 and MRGSSR

56 derived from mungbean scaffolds having homology with putative disease resistance genes were identified in the present study that could differentiate both YMD and PMD resistant and susceptible genotypes. MRGSSR12, designed from mungbean scaffold which shared homology with TMV resistance protein N. TMV resistance protein N is a disease resistance protein having one TIR, one NB-ARC domains and six LRR repeats which guard the plants against pathogens through direct or indirect interaction with avirulence protein and triggers a defense system including the hypersensitive response, which restricts the pathogen's growth and spread (The Uniprot Consortium,https://doi.org/10.1093/nar/gkw1099).

The cross-species amplification of mungbean derived resistance gene-SSR markers were investigated for diversity analysis in the set of 44 blackgram genotypes comprising of 43 cultivars and one wild species. Cluster analysis based on neighbour-joining method grouped the 44 genotypes into seven clusters (**Fig. 2**). Cluster I with 3 sub-clusters (Ia, Ib and Ic) comprised of 14 blackgram genotypes. Cluster Ia comprised of 2 genotypes, EC168200 and LBG-17, which are resistant to YMD and PMD, respectively. Of the five genotypes grouped under cluster 1b the genotypes Pusa-3, LBG 752, and IPU02-043 are resistant to YMD. Three of the seven genotypes constituting cluster Ic are YMD resistant (DPU88-31, IPU07-3 and KU96-7). The YMD resistant cultivars NDU-1 and PU-19 are clustered separately in cluster VII with



Fig. 2. Dendrogram constructed using Jaccard's similarity coefficient and UPGMA clustering among 44 blackgram genotypes based on mungbean derived SSR markers.

a similarity index value of 0.27. The highest similarity coefficient was observed between LBG693 and IPU02-43 (0.81).

The genetic closeness between some of the cultivars could be explained due to common parents in their pedigree. For example, genotypes IPU07-3 and IPU02-43 although from the different crosses, DPU88-31 x PDU-1 and DPU88-31 x DUR-1, respectively, had one parent in common and were grouped together in cluster I. Moreover, both genotypes were resistant to YMD and PMD. DPU88-31 was grouped with one of its parent T9 in sub-cluster Ic of cluster I. Likewise genotypes Pusa3 and DPU88-31 both are grouped together in cluster I along with their one common parent, T9. In this study, grouping of blackgram genotypes based on disease resistance were observed in cluster I. Similar grouping of genotypes based on disease resistance was reported in blackgram (Souframanien and Gopalakrishna, 2009). Grouping of individuals based on disease reaction observed in this study could be due to presence of resistance gene in the mungbean scaffolds used for designing the markers. For example, TMV resistance protein N, Protein suppressor of npr1-1, constitutive 1, putative late blight resistance protein homolog R1B-8, and putative disease resistance protein At4g11170 exhibited homology within the same scaffold JJMO01000125.Clustering of resistance gene analogues was also reported in several

other species such as rice (Monosi *et al.*, 2004), tomato (Dickinson *et al.*, 1993) and other species (Sheperd and Mayo, 1972).

In the present study, 68 mungbean resistance genes harbouring WGS scaffolds derived genomic-SSR primers showed cross species amplification in blackgram. These transferable genomic-SSR markers would be a valuable resource for blackgram genetic analysis because resistance genes-based SSR marker polymorphism would represent the variation present in the resistance sources of blackgram genotypes. However, SSR primers which differentiated YMD and PMD blackgram genotypes need further confirmation and validation of their association with the resistance trait. These SSR markers derived from resistance proteins homologous sequences could be lying within either the coding sequences, untranslated regions or regulatory regions of resistance genes, and would offer an opportunity to investigate the consequences of SSR polymorphisms on gene functions and regulations associated with disease resistance. These SSR markers would be helpful in the selection of appropriate genotypes in breeding programmes aiming at developing multiple stress tolerant cultivars.

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