



Identification and fine-mapping of novel major locus for yellow mosaic disease resistance in *Glycine soja*

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Abstract

Key message This is the first report on the identification and fine-mapping of yellow mosaic disease resistance locus, *qMYMIV14.1*, on chromosome 14 using integrative genomic approaches in interspecific soybean populations.

Abstract Yellow mosaic disease (YMD) is a major viral threat to soybean production in Asian and Southeast Asian countries. Field screening of the disease was performed at Ludhiana; a YMD hot spot and its causative agent, mungbean yellow mosaic India virus (MYMIV, *Begomovirus vignaradiataindiaense*) was detected and validated as the causal agent through PCR and sequence analysis. Genetic assessment was conducted on 1784 F₂ plants derived from a cross between the susceptible cultivated soybean (*Glycine max* cv. ‘NRC 94’) and a resistant wild accession (*Glycine soja* accession ‘PI 393551’). The segregation ratio indicated that YMD resistance is controlled by four dominant genes, three of which confer resistance, while one inhibitory gene suppresses this resistance. Test of allelism performed on direct and reciprocal crosses [SL 958//JS 335/PI 393551 (BC₅F₆)] across F₁, F₂, and F₃ generations revealed that genes in cultivated and wild soybean were non-allelic with no maternal effect. Bulk segregant analysis (BSA) initially identified eight markers (Satt157, BARCSOYSSR_14_0441, BARCSOYSSR_14_0445, BARCSOYSSR_14_0448, BARCSOYSSR_14_0455, BARCSOYSSR_14_1416, BARCSOYSSR_14_1417 & BARCSOYSSR_14_1516) linked to resistance. Traditional QTL mapping revealed three novel QTLs on chromosome 14. Combined results from QTL-seq, a genome-wide association study, and QTL mapping consistently identified a major and stable locus, *qMYMIV14.1*, spanning the 46.55–48.70 Mb region on the long arm of chromosome 14. This is the first QTL detected from an interspecific cross which explained for 27.81–68.01% of phenotypic variance. Two candidate genes, *Glyma.14G173300* and *Glyma.14G173600*, encoding leucine-rich repeat proteins, were identified within this locus. These findings provide valuable genomic resources for marker-assisted selection and breeding of soybean cultivars with durable YMD resistance.

Keywords Soybean · *Glycine soja* · Yellow mosaic disease · Resistance · QTL-seq · QTL

Introduction

Soybean is pre-disposed to around 70 viral diseases (Gaiwad et al. 2021), among which yellow mosaic disease (YMD) is one of the most destructive in Asian and Southeast Asian countries. Historically the disease was confined to legumes (Mishra et al. 2020), but its recent occurrence in tomato (Agnihotri et al. 2019; Shahid et al. 2019) exhibits its expanding host range. The disease produces characteristic yellow and golden mosaic symptoms (Mishra et al. 2020). It

leads to an enormous loss in yield and seed quality in India (Yadav et al. 2015). Based on etiology, mungbean yellow mosaic India virus (MYMIV, *Begomovirus vignaradiataindiaense*) causes infection in Northern and Central India and is naturally transmitted by whitefly (Ramesh et al. 2019). Controlling its vector population by use of insecticides is one way to manage the disease. However, complete eradication of the vector using insecticides is not feasible as the presence of a single viruliferous whitefly can transmit the virus to healthy plants, making management of YMD practically unattainable by the use of insecticides (Naveed et al. 2023). Consequently, the cultivation of disease-resistant varieties remains the most effective, economical, and eco-friendly strategy for disease management (Kumar et al.

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2014), making it apparent that the incorporation of YMD resistance into elite soybean cultivars is a necessity.

Wild soybean, *Glycine soja* (Seib. and Zucc.), the progenitor of cultivated soybean, shares the same number of chromosomes ($2n=40$) and readily hybridizes with *G. max*, resulting in fertile progenies (Singh and Hymowitz 1988). This genetic compatibility makes *G. soja* a valuable reservoir of alleles for improving agronomic traits in cultivated soybean. Wild soybean harbors numerous beneficial genes associated with enhanced oil and protein content, tolerance to abiotic stresses, and resistance to several pests and diseases (Taliencio et al. 2024; Chen et al. 2025) including durable resistance to YMD (Rani et al. 2018). Despite this, its use in breeding has been limited due to its linkage with undesirable wild characteristics such as vining growth habit, lodging, pod shattering, delayed leaf abscission, small seed size, and dark seed coat (Wang et al. 2016).

For the development of a resistant variety, the first prerequisite is the complete understanding of the inheritance and genetic basis of resistance. However, despite the considerable threat posed by YMD, its underlying genetic architecture remains poorly characterized, as most studies have concentrated only on cultivated soybean germplasm, with limited investigations exploring the potential of wild or interspecific sources of resistance. Classical breeding for disease resistance often relies on the identification of resistance genes from wild relatives or landraces, which harbor valuable alleles lost during domestication (Zhou et al. 2015). In this context, wild soybean offers a promising yet underutilized genetic reservoir for YMD resistance.

Moreover, mobilizing resistance (R) genes or quantitative trait loci (QTL) into elite cultivated genotypes within a reasonable breeding timeframe relies heavily upon the prior identification and validation of tightly linked molecular markers associated with resistance loci (Rani et al. 2018). Traditionally, bulked segregant analysis (BSA) has served as an efficient approach for the rapid detection of markers linked to disease resistance traits. The resolution and accuracy of such mapping efforts can be significantly enhanced by increasing both the population size and the number of genetic markers (Karthikeyan et al. 2017). The advent of next-generation sequencing (NGS) technologies has transformed plant genomics by enabling high-throughput discovery of high-density single nucleotide polymorphisms (SNPs), providing a powerful means to identify resistance loci with precision (Sonah et al. 2013) through methods like genotyping-by-sequencing (GBS) and whole-genome sequencing (WGS).

Moreover, as plant viruses continue to evolve rapidly and expand their host range under changing ecological conditions, the deployment of resistance derived from a single source may prove inadequate (Khosla et al. 2021). Therefore, the identification and introgression of novel resistance locus

from wild soybean through integrative genomic approaches could facilitate the development of soybean cultivars with durable resistance to YMD. The present study was focused on fine-mapping of the genomic regions associated with YMD resistance and identification of candidate genes using mapping populations derived from an interspecific cross between *G. max* and *G. soja*.

Materials and methods

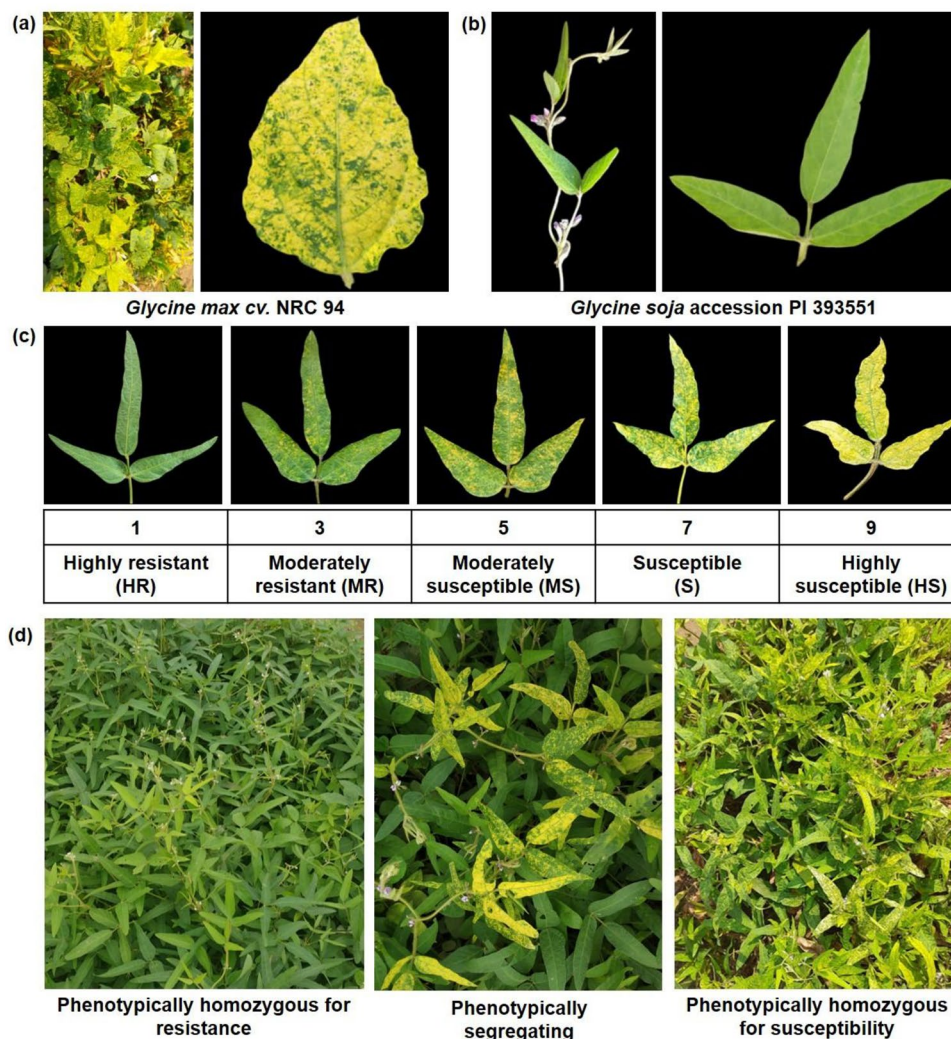
Plant material

G. max cultivars, ‘NRC 94’ and ‘JS 335’, both of which are high-yielding but highly susceptible (HS) to YMD, cultivar ‘SL 958’, a high-yielding and YMD-resistant variety developed at Punjab Agricultural University (PAU), Ludhiana (Gill et al. 2015), and *G. soja* accession ‘PI 393551’ which is highly resistant (HR) against YMD (Fig. 1a, b) were utilized. For Chi-square (χ^2) analysis, F_2 and $F_{2,3}$ populations of NRC 94/PI 393551 were evaluated, while only the F_2 generation was used for mapping. To test allelism, the SL 958 variety was chosen as the cultivated soybean source of resistance, while the cross ‘JS 335/PI 393551 (BC_5F_6)’ was selected as the wild source derivative. Direct and reciprocal crosses of ‘SL 958/JS 335/PI 393551 (BC_5F_6)’ in three different generations viz., F_1 , F_2 , and F_3 were evaluated. For genome-wide association study (GWAS), backcross inbred lines (BILs), BC_1F_6 generation of NRC 94/PI 393551/NRC 94 were evaluated. All the crosses were developed at Pulses Research Farm, Department of Plant Breeding and Genetics, PAU, Ludhiana (30°54′ N, 75°47′ E) which is a major hot spot of YMD with high disease pressure (Kitsanachandee et al. 2013).

Screening for YMD

Development of crosses started from 2014 and subsequent off-season generation advancement was carried out under controlled conditions in an incubator (25 ± 2 °C with a 16-h photoperiod & ~70% relative humidity) with seedlings initially raised in a cocopeat:sand mixture (3:1) and later transplanted at the two-leaf stage in a polyhouse. Populations were evaluated in the fields during 2021, except for the F_2 generation of NRC 94/PI 393551, which was evaluated across two consecutive years (2020 and 2021). The trials were laid out using the progeny rows of their original F_2 generation. To ensure uniform disease pressure, the infector row technique was adopted, with the susceptible cultivar ‘JS 335’ planted as border rows and after every three rows of test entries. Disease scores were assigned as per 1–9 scale (Fig. 1c) described by Parihar et al. (2017) when 90% of infector rows displayed typical YMD symptoms. Plants with

Fig. 1 Symptoms of yellow mosaic disease (YMD) on soybean. Plant and leaves of **a** susceptible parent *Glycine max* cv. ‘NRC 94’; **b** symptomless resistant parent *Glycine soja* accession ‘PI 393551’; **c** F_2 generation showing disease reactions and scores (1–9) against YMD infection; **d** progeny rows of $F_{2:3}$ generation from a cross between NRC 94 and PI 393551, differentiating phenotypically segregating and non-segregating lines against YMD infection



scores up to 3 were classified as resistant, whereas those scoring ≥ 4 were considered susceptible. The per cent disease incidence in BILs was calculated using the formula provided by Delp et al. (1986). All recommended agronomic practices were followed except for the spraying of pesticides to maintain the natural whitefly population (Anonymous 2025).

Isolation of total genomic DNA and survey of parental polymorphism

Genomic DNA (gDNA) was extracted from 1784 F_2 , 60 BILs, and three parental genotypes viz., NRC 94, PI 393551, and SL 958 using the cetyltrimethylammonium bromide (CTAB) method as described by Saghai-Marooft et al. (1984) with minor modifications. Quantity and purity were assessed using a NanoDrop Spectrophotometer (Thermo Fisher Scientific, USA), while integrity was confirmed via electrophoresis on 0.8% agarose gels (Lonza, Switzerland)

and visualized using a gel documentation system (Alpha Imager, USA).

Parental polymorphism was performed using 75–100 ng/μl gDNA with 352 simple sequence repeats (SSRs; Cregan et al. 1999; Song et al. 2010; Meghana 2021) covering all 20 chromosomes (Table S5). Polymerase chain reaction (PCR) was performed in a 96-well microtiter plate in a MiniAmp Plus Thermal Cycler (Applied Biosystems, USA) according to Khosla et al. (2021) with minor modifications.

Bulked segregant analysis

Resistant and susceptible DNA bulks were constructed based on the phenotypic data of F_2 and $F_{2:3}$ generations of NRC 94/PI 393551 cross to ensure homozygosity (Table S4). Two contrasting bulks viz., resistant and susceptible were prepared by pooling the equimolar DNA concentrations (0.5 μg/μl) of nine F_2 plants each from phenotypically extreme disease scores of 1 and ≥ 6 , respectively, for each bulk in accordance with Michelmore et al. (1991).

Sequencing

Two DNA bulks and parental lines (NRC 94 and PI 393551) were subjected to WGS. While the set of 60 BILs and parental lines (NRC 94 and PI 393551) was genotyped using GBS approach. The paired-end sequencing was performed on the Illumina NovaSeq 6000 platform at NGB Diagnostics, New Delhi.

Whole-genome sequencing and QTL-seq analysis

The quality of raw sequencing reads of bulks and parents was assessed using FastQC v012.1 (Wingett & Andrews 2018). Adapter and low-quality reads were trimmed with Trimmomatic v0.36 (Bolger et al. 2014). The clean reads were aligned to *Glycine max* cv. Williams 82 (Wm82.a6.v1) using BWA 0.7.18 (Li and Durbin 2010). The BAM files were sorted and indexed using SAMtools (Li et al. 2009). Picard (<https://broadinstitute.github.io/picard/>) was used to remove duplicate reads. Variant calling was performed using HaplotypeCaller in Genome analysis toolkit (GATK; McKenna et al. 2010). Variant filtering was done using VCFtools (Danecek et al. 2011). QTL-seq analysis and data visualization were conducted using the QTLseqr package in R (Mansfeld and Grumet 2018). Both the $\Delta(\text{SNP-index})$ and G' prime (G') statistical methods were utilized. A sliding window approach with a window size of 1 Mb was applied to calculate smoothed $\Delta(\text{SNP-index})$, G' values, and $-\log_{10}(p\text{-value})$ across the genome and identify candidate genomic regions (Magwene et al. 2011; Takagi et al. 2013).

Detection of virus

To confirm the identity of the causal virus, PCR was performed on 1000 ng/ μl gDNA of both parents (NRC 94 and PI 393551), F_2 plants with disease scores of 1, 3, 5, 7, and 9, and a mock without template. The MYMIV specific primer *NC_AVI* (forward: ACATCACTCACCCCAGAA GG; reverse: CACTTTGTACTGCCCGGATT) was used to target a 125 bp fragment of the *AVI* gene which encodes the viral coat protein (Kuppuraj 2024). The PCR was performed in the MiniAmp Plus Thermal Cycler (Applied Biosystems, USA) as per Kuppuraj (2024). In addition, the remaining unmapped reads obtained after aligning to Wm82.a6.v1 were analyzed using BLAST against 148,687 virus assemblies available on the NCBI Virus nucleotide database (<https://www.ncbi.nlm.nih.gov/labs/virus/vssi/#/>).

Genotyping by sequencing and GWAS analysis

Analysis of 60 BILs and parents was performed using the GBS pipeline described by Sonah et al. (2013). The imputation was performed by Beagle 5.3 (Browning et al. 2021). A

genome-wide association study was performed in R using the GAPIT package version 3.4.0 (Wang and Zhang 2021). Six models viz., fixed and random model circulating probability unification (FarmCPU), Bayesian-information and linkage-disequilibrium iteratively nested keyway (BLINK), maximum likelihood estimation of modal model (MLMM), maximum likelihood method (MLM), generalized linear model (GLM), and settlement of MLM under progressively exclusive relationship (SUPER) were utilized.

QTL mapping

QTL analysis was performed using QTL IciMapping version 4.2.53 (Meng et al. 2015) independently on two mapping populations, a selected set of 517 F_2 plants and 60 BILs. Initially, single marker analysis (SMA) was conducted to identify markers associated with the trait of interest. Subsequently, a linkage map was created with default parameters using the inclusive composite interval mapping (ICIM-ADD) function. Since the physical positions of SSR markers were unavailable for the latest reference genome Wm82.a6.v1, the primer sequences were aligned using the BLAST tool on the Phytozome database (Goodstein et al. 2012) to obtain physical coordinates based on the highest alignment score, identity percentage, and E -value. Further linkage and physical maps were drawn using MapChart version 2.32 (Voorrips 2002).

Prediction of candidate genes

A 100 kb genomic interval upstream, downstream, and within the identified QTL regions and significant SNPs was examined to identify candidate genes. Functional annotations were retrieved from the Phytozome database, and genes with disease resistance mechanisms were identified as potential candidates.

Results

Detection and confirmation of MYMIV

The 125 bp amplicon specific to the *AVI* gene of MYMIV was observed in all plants including NRC 94, excluding mock and resistant parent (PI 393551), confirming the presence of the virus (Fig. S1). In addition, unmapped reads obtained from WGS data of parents and bulks were subjected to BLAST. The analysis revealed matches to several viral sequences, with a maximum of 666 MYMIV assemblies detected in both resistant and susceptible bulks. The top hit showed 99.213% read identity to the DNA-B and 97.143% to the DNA-A of MYMIV Ur1_Ludhiana (GenBank accession: MW717942.1 and MW717971.1), confirming that

the natural infection in the present study was caused by the Ludhiana strain of MYMIV.

Polygenic nature of YMD resistance

From the year 2014 to 2021, the parental lines, NRC 94 and PI 393551, consistently showed disease scores of 9 (HS) and 1 (HR), respectively (Fig. 1a). A total of 1784 F_2 plants were assessed, among which 1325 plants displayed a disease score of 1 (HR), while only 18 plants showed a score of 9 (HS). The remaining plants exhibited intermediate disease scores (Table S1). A significantly higher number of resistant plants compared to susceptible ones suggests the presence of the dominant nature of YMD resistance in the interspecific cross of soybean. The F_2 population showed a goodness of fit in a 225 resistant (R):31 susceptible (S) segregation ratio (Table 1). The data indicated that YMD resistance is dominant in nature and is controlled by four genes. Disease reactions of 426 $F_{2,3}$ families showed 237 resistant, 160 segregating, and 29 susceptible lines. Although a clear Mendelian ratio could not be determined due to the limited number of families within each genotypic class and the complexity of the generation, the distribution pattern supported the dominant and polygenic nature of YMD resistance.

Presence of different genes in cultivated and wild soybean

Test of allelism was performed on direct and reciprocal crosses of ‘SL 958//JS 335/PI 393551 (BC_5F_6)’ in three different generations viz., F_1 , F_2 , and $F_{2,3}$ (Table S2). The disease scores of the F_1 generation in direct cross ranged from 1 to 9 in all the 24 plants, while in its reciprocal cross, all the seven plants were found to be resistant with a disease

score of 1. This variability in disease reactions observed in reciprocal crosses is not attributed to any maternal effect but rather to the small number of F_1 plants. A total of 1182 F_2 plants obtained from both direct and reciprocal crosses showed segregation with variable disease scores ranging from 1 to 7. Besides, the number of 38 susceptible plants observed was again far less than that of 1144 resistant plants (Table S2). In 146 $F_{2,3}$ families of both the direct and reciprocal crosses showed either homozygous resistant or segregating lines for YMD, with the complete absence of any susceptible progenies (Table S2).

Mapping of chromosome 14

A total of 352 SSR markers (Table S3) covering all the 20 chromosomes were employed to assess parental polymorphism between NRC 94 (HS) and PI 393551 (HR). Of these, 191 polymorphic markers (Table S3a) were amplified on both the parents and two extreme bulks (Fig. S2). Based on BSA, eight markers were found to be polymorphic and linked to YMD resistance (Figs. 2, 3e, S6, Table S3b). Among these, one marker, Satt157 (BARC-SOYSSR_02_0492), was located on chromosome number 2 at 9.28 Mb, while the rest of the seven markers viz., BARC-SOYSSR_14_0441 (7.86 Mb), BARC-SOYSSR_14_0445 (7.90 Mb), BARC-SOYSSR_14_0448 (8.02 Mb), BARC-SOYSSR_14_0455 (8.19 Mb), BARC-SOYSSR_14_1416 (50.71 Mb), BARC-SOYSSR_14_1417 (50.76 Mb), and BARC-SOYSSR_14_1516 (52.08 Mb) were present on chromosome 14 (Fig. 3e, Table S3b).

A set of randomly selected 517 F_2 individuals was genotyped with these eight polymorphic markers (Fig. 2). All the markers were found to be co-dominant and linked to YMD resistance. The linkage map for chromosome 14 was constructed with a total length of 294.90 cM (Fig. 3e). Three major and novel QTLs were determined using the ICIM-ADD approach which were designated as *qMYMIV1*, *qMYMIV2*, and *qMYMIV3* (Table 2). At the chromosomal level, *qMYMIV1* lies on the short arm, while the other two loci are located on the long arm of chromosome 14. Since only one marker was identified on chromosome 2, the linkage map could not be constructed. However, SMA revealed that it was responsible for 3.21% of the phenotypic variance explained (PVE). Combined with Satt157, these three QTLs

Table 1 Segregation and Chi-square test of F_2 population from NRC 94 (HS) and PI 393551 (HR)

Total number of plants			Goodness of fit 225(R):31(S)	
Disease reaction	Observed	Expected	χ^2 value	P value
Resistant	1552	1568	0.65	0.42
Susceptible	232	216		

HS highly susceptible; HR highly resistant; R resistant; S susceptible

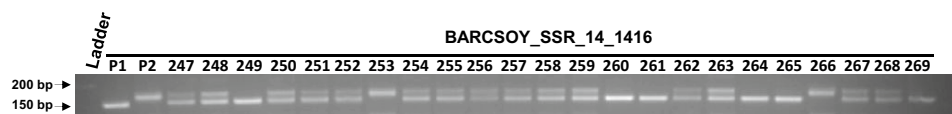


Fig. 2 Segregation pattern of linked SSR marker BARCSOYSSR_14_1416 on parents and F_2 population (plant numbered 247–269). P1: NRC 94, P2: *G. soja* accession ‘PI 393551’

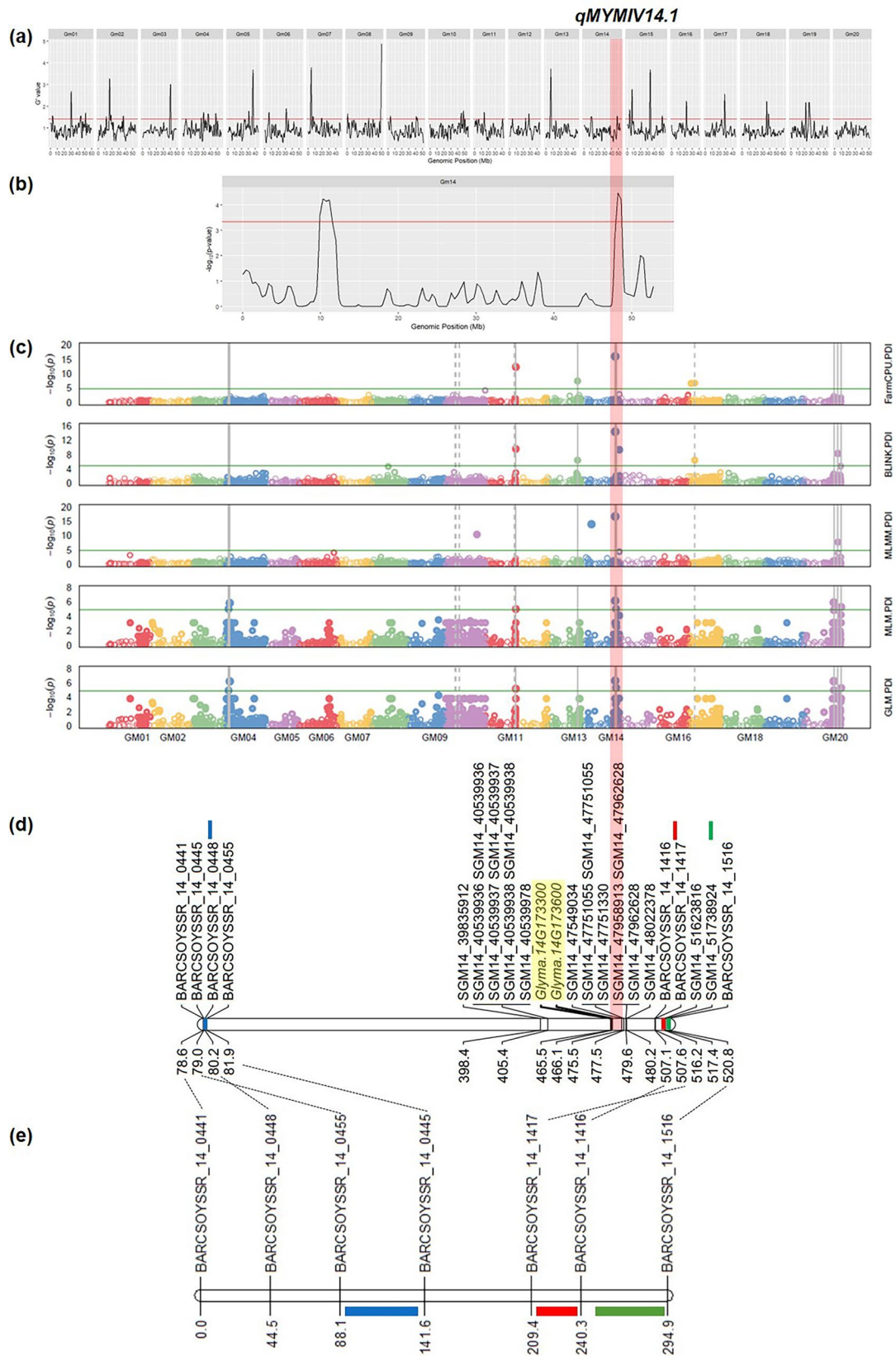


Fig. 3 Integrated identification of *qMYMIV14.1* genomic region associated with yellow mosaic disease (YMD) resistance in interspecific crosses of soybean with light red highlight indicating the candidate genomic interval. **a** G' values and **b** $-\log_{10}(p\text{-value})$ plots from QTL-seq highlighting significant regions on chromosome 14. **c** Detection of significant SNP (SGM14_46580701) across multiple GWAS models. **d** Physical map of chromosome 14 showing linked SSR markers, key SNPs identified (highlighted in yellow) from QTL mapping of backcross inbred lines (BILs), and candidate genes (scale=0.1 Mb). **e** Linkage map of chromosome 14 developed from F_2 population displaying SSR markers linked to YMD resistance (scale=centimorgans). Blue, red, and green bars depict *qMYMIV1*, *qMYMIV2*, and *qMYMIV3*, respectively

accounted for 95.91% of the phenotypic variation observed for YMD resistance.

Identification of *qMYMIV14.1* locus

Whole-genome sequencing of NRC 94, PI 393551, susceptible and resistant bulks generated 82.16, 78.87, 111.13, and 111.94 million raw reads, respectively. The guanine-cytosine (GC) content ranged from 36–43% across all samples. The average read depth for the parental lines was approximately 22 $\times$, while it was 30.5 $\times$ for bulks. Initial SNP calling yielded 12,274,035 variants between the bulks. After filtering for quality, missing data, and minor allele frequency, 359,321 high-confidence SNPs were retained for QTL-seq analysis. A total of 49 regions were observed across all chromosomes except for chromosome 20 (Fig. 3a, Table S5). Based on prior mapping results, particular attention was given to chromosome 14, which detected two significant genomic intervals between 9.89–11.53 Mb and 47.95–48.70 Mb (Fig. 3b). Of these, the latter was identified as an important QTL region and was designated as *qMYMIV14.1*, which spanned across 0.74 Mb with 285 SNPs. The peak $\Delta(\text{SNP-index})$ and G' values were recorded at 48.63 Mb and 48.21 Mb positions, respectively, which notably lie adjacent (~ 2.5 Mb) to the locus *qMYMIV2* determined through conventional QTL analysis, marking it as a potentially important genomic region contributing to YMD resistance.

Genome-wide association study conducted on 60 BILs (NRC 94/PI 393551/NRC 94) and both parents (NRC 94 & PI 393551) retrieved a total of 20,68,150 SNPs after alignment to Wm82.a6.v1. High-quality 5,99,941 SNPs were selected; among these, 65,299 SNPs were found to be polymorphic between the parents (including missing values). After excluding SNPs with missing values, only 3885 SNPs were retained for further analysis (Table S6). Sixty-one unique marker-trait associations (MTAs) were obtained from all the models (Table S7). Several of these SNPs were consistently identified across different models, indicating a strong association with YMD resistance (Fig. 3c). However, based on the quantile–quantile (Q–Q) plots of all the models FarmCPU, BLINK, and MLM were the best fit for

the identification of significant SNPs (Fig. S3). The most significant SNP, i.e., SGM14_46580701 explained 68.01% of phenotypic variation was detected through five models (FarmCPU, BLINK, MLM, MLM, and GLM) with a minor allele frequency of only 0.03 showing a strong effect for YMD resistance. Its physical position was mapped at 46.5 Mb (Table S8) and it lies in close proximity (1.37 Mb) to the locus *qMYMIV14.1* already detected by QTL-seq.

In addition, traditional QTL mapping was also performed with 169 SNPs present on chromosome 14, which led to the identification of nine QTLs associated with YMD resistance (Tables S9, S10). Among these, four closely spaced QTLs were delineated between SNP intervals SGM14_47549034–SGM14_47751055, SGM14_47751055–SGM14_47751330, SGM14_47958913–SGM14_47962628, and SGM14_47962628–SGM14_48022378, exhibiting LOD scores of 9.52, 9.50, 3.79, and 3.76, respectively (Table S10). Upon closer inspection due to the adjacency of these QTL, this region was considered a single locus that accounted for 27.81% PVE (Table S10). Besides, the physical positions of these loci span around the 47.54–48.02 Mb region, which overlaps with the *qMYMIV14.1* locus previously identified through QTL-seq (Fig. 3).

Identification of candidate R genes

Nine genes located either within the QTL or around their flanking regions were identified (*Glyma.14G179600*, *Glyma.14G179800*, *Glyma.14G179900*, *Glyma.14G180100*, *Glyma.14G180300*, *Glyma.14G173200*, *Glyma.14G174100*, *Glyma.14G173300*, & *Glyma.14G173600*). Only *Glyma.14G173300* and *Glyma.14G173600* were regarded as candidate R genes as they code for leucine-rich repeat proteins (Fig. 3d, Table 3). The rest coded for zinc-finger related proteins, CYCLIN-D4-1 protein, cytochrome C and mitochondrial ATP synthase subunit with two genes having unknown functions.

Discussion

Globally, resistance to YMD in soybean has traditionally relied on the cultivated source ‘UPSM 534 (PI 171443)’. However, dependence on a single source could lead to narrowing of the genetic base, which makes the exploration of wild germplasm imperative for the development of resistant cultivars. As early as 1974, *G. soja* has been recognized as a source of YMD resistance (Singh et al. 1974). Over the past 50 years, *G. soja* accession ‘PI 393551’ has consistently demonstrated resistance to YMD (Ramesh et al. 2019). However, its direct use in soybean breeding poses a major challenge due to linkage drag with undesirable agronomic

Table 2 Quantitative trait loci detected for YMD resistance from a cross between NRC 94 (HS) and PI 393551 (HR)

QTL	Left marker	Right marker	LOD	PVE (%)
<i>qMYMIV1</i>	BARCSOYSSR_14_0455	BARCSOYSSR_14_0445	7.94	27.84
<i>qMYMIV2</i>	BARCSOYSSR_14_1417	BARCSOYSSR_14_1416	11.37	31.48
<i>qMYMIV3</i>	BARCSOYSSR_14_1416	BARCSOYSSR_14_1516	13.24	33.38

HS highly susceptible; HR highly resistant; LOD: logarithm (base 10) of the odds; PVE phenotypic variation explained

Table 3 Details of candidate genes identified

Gene	Start position (bp)	End position (bp)	Pfam entry	GO description	Function
<i>Glyma.14G173300</i>	46551719	46555639	PF08263 (LRRNT_2)	Protein binding	Leucine-rich repeat protein
<i>Glyma.14G173600</i>	46613432	46616934	PF08263 (LRRNT_2)	Protein binding	Leucine-rich repeat protein

Pfam protein family; GO gene ontology

traits. Therefore, from a breeding perspective, using pre-breeding lines (advanced backcross) derived from *G. soja* represents a more desirable and practical alternative to direct crosses with wild accessions. However, Rani et al. (2018) speculated that successive backcrossing with the recurrent parent (*G. max*) may lead to the inadvertent loss of resistance genes derived from *G. soja*. This highlights the importance of understanding the inheritance of YMD resistance in *G. soja* before its effective use in breeding. In this context, F₂ populations directly derived from crossing PI 393551 were studied to understand the genetic architecture of YMD resistance as the F₂ generation allows to capture maximum recombination events and segregation pattern of parental alleles for effective mapping of resistance loci (Salomé et al. 2012).

Field screening at Ludhiana, a hot spot for YMD has been proved to be as effective as agro-inoculation for disease evaluation, as demonstrated by Ramesh et al. (2019). The 225(R):31(S) segregation ratio indicated that YMD resistance is a complex polygenic trait, dominant in nature and controlled by at least four genes. Of these, three genes provide resistance, while one gene with inhibitory action produces susceptible phenotype. Khosla et al. (2021) and Mandahal et al. (2024) also showed the presence of inhibitory gene action in the case of cultivated soybean. When a single resistant gene is present, the inhibitory (*I*) gene suppresses the effect of the resistance gene, resulting in a susceptible phenotype. However, when two or three resistance genes are present, they produce a resistant phenotype, overcoming the susceptibility conferred by the inhibitory gene. Based on the four gene model, genotypes for parents, F₁, and F₂ generations have been proposed (Table 4). The susceptible parent (NRC 94) is hypothesized to carry three recessive genes for resistance and one dominant inhibitory gene, all in the homozygous state. Contrary to this, the resistant parent (PI 393551) is thought to possess three genes imparting

resistance and one inhibitory gene in a recessive state; all the genes have homozygous alleles. The F₁ progeny was a heterozygote for all four genes (Table 4).

The classification of 256 genotypes into 16 distinct classes, out of which 11 classes showed resistance while the rest 5 showed susceptible disease reaction, further validates the four gene model with one inhibitory gene (Table 4). The present results deviate from the previous studies on inter-specific crosses conducted by Bhattacharyya et al. (1999) who reported single dominant gene action in 120–480 F₂ plants, whereas Rani et al. (2018) determined duplicate dominant gene action in 310–406 F₂ plants for YMD resistance. The increased resolution provided by the larger population size might have led to the detection of a greater number of resistance-associated genes in PI 393551 in our findings. Besides, the investigation led by Vales et al. (2005) showed the importance of a large population size for stripe rust in barley as the use of small population sizes to detect genomic regions associated with the trait of interest often leads to an underestimation of the number of identified regions. Moreover, differences in genetic ratios can be due to the involvement of completely different genes owing to their ancestry in all the crosses studied or different viral species/strains causing disease or disease escape due to low disease pressure (Aski et al. 2015).

To determine the genetic relationship between different resistance sources, a test of allelism was performed. The segregation observed in the F₂ and F_{2:3} progenies indicated that the resistance genes present in cultivated and wild sources for YMD resistance are different. The variety SL 958 was developed from a cross between SL 525 and SL 706. Notably, SL 525 is derived from PK 1023, a progeny of UPSM 534, highlighting the repeated use of UPSM 534 as a predominant source of YMD resistance in cultivated soybean. This reinforces the need to broaden the resistance gene pool through the introgression of diverse alleles from

Table 4 Proposed genotypes and YMD disease reaction in F₂ generation of a cross between NRC 94 (HS) and PI 393551 (HR)

Generation	Proposed genotype	Expected frequency	Disease reaction	Expected ratio 225(R):31(S)
NRC 94 (susceptible parent)	<i>r₁r₁r₂r₂r₃r₃II</i>	—	S	—
<i>G. soja</i> accession ‘PI 393551’ (resistant parent)	<i>R₁R₁R₂R₂R₃R₃ii</i>	—	R	—
F ₁	<i>R₁r₁R₂r₂R₃r₃Ii</i>	—	R	—
F ₂	<i>R₁—R₂—R₃—I—</i>	81	R	225
	<i>R₁—R₂—R₃—ii</i>	27	R	
	<i>R₁—R₂—r₃r₃I—</i>	27	R	
	<i>R₁—R₂—r₃r₃ii</i>	9	R	
	<i>R₁—r₂r₂R₃—I—</i>	27	R	
	<i>R₁—r₂r₂R₃—ii</i>	9	R	
	<i>R₁—r₂r₂r₃r₃ii</i>	3	R	
	<i>r₁r₁R₂—R₃—I—</i>	27	R	
	<i>r₁r₁R₂—R₃—ii</i>	9	R	
	<i>r₁r₁R₂—r₃r₃ii</i>	3	R	
	<i>r₁r₁r₂r₂R₃—ii</i>	3	R	
	<i>R₁—r₂r₂r₃r₃I—</i>	9	S	31
	<i>r₁r₁R₂—r₃r₃I—</i>	9	S	
	<i>r₁r₁r₂r₂R₃—I—</i>	9	S	
	<i>r₁r₁r₂r₂r₃r₃I—</i>	3	S	
	<i>r₁r₁r₂r₂r₃r₃ii</i>	1	S	

HS highly susceptible; HR highly resistant; R resistant; S susceptible

the wild gene pool. Such a non-allelic relationship was also observed for salt tolerance in an interspecific cross of soybean involving *G. soja* (Lee et al. 2009). The identification of non-allelic resistance genes offers a perfect opportunity to pyramid these genes for the development of varieties with durable YMD resistance. Such an approach is particularly important for countering the rapidly evolving nature of the virus. Furthermore, the absence of maternal effects confirms the nuclear inheritance of YMD resistance, thereby facilitating more efficient introgression into elite cultivars.

First, BSA was performed on parents and bulks where disease scores from F₂ and F_{2:3} generations ensured that the plants selected for creating bulks were strictly homozygous for resistance and susceptibility, as their progeny rows did not segregate in the F_{2:3} generation which could not have been determined at the F₂ level as described by Micheltore and Paran (1991). Subsequently, eight linked markers were identified that could be effectively employed to conduct marker-assisted selection. However, only seven markers identified on chromosome 14 were used for the construction of a genetic linkage map and the identification of three QTL. Among these, the *qMYMIV2* locus contributed 31.48% towards phenotypic variation which was flanked by the same markers that were reported by Rani et al. (2018) to be associated with YMD resistance. Notably, this region also harbors the *R_{SC4}* gene which confers resistance towards soybean mosaic virus, another important disease of soybean (Wang

et al. 2011). Thus, selecting this genomic region could provide dual resistance to two important viral diseases of soybean.

To take advantage of the recent advances in sequencing technologies that provide genome analysis at a much larger scale with higher resolution, WGS of both parents and bulks was performed and subjected to QTL-seq analysis which identified *qMYMIV14.1* on the long arm of chromosome 14 at the 47.95–48.70 Mb interval. This region lies approximately 2.5 Mb adjacent to the *qMYMIV2* region determined through conventional QTL analysis, marking it an important region associated with YMD resistance. Although marker Satt157 and locus *qMYMIV1* showed an association in BSA, it might have been missed due to the statistical power limits and stringent confidence thresholds inherent to QTL-seq. In addition, a significant SNP, SGM14_46580701 located in the 46.5 Mb region of chromosome 14 with high PVE of 68.01%, was consistently detected across five GWAS models. These models were reported to be extremely powerful and accurate in predicting multi-locus associations, making them highly effective for identifying complex trait associations (Wang and Zhang 2021).

Traditional QTL mapping of chromosome 14 using SNP data again detected 47.54–48.02 Mb intervals to be associated with YMD resistance. Thus, an integrated approach combining QTL-seq, GWAS, and linkage mapping, led to the identification of a novel, consistent, and major genomic

region associated with YMD resistance on the long arm of chromosome 14. This region, designated as *qMYMIV14.1*, spans approximately 2.2 Mb (46.55–48.70 Mb) and represents a robust locus consistently detected across all methods. Similar integrative approaches have been successfully employed to identify resistance genes against phytophthora blight in sesame (Asekova et al. 2021), bean leaf crumple virus (Ariza-Suarez et al. 2023), and wheat yellow mosaic virus (Liu et al. 2023).

Both phenotypic and molecular data establish that YMD resistance is polygenic in nature. Moreover, two candidate genes, *Glyma.14G173300* and *Glyma.14G173600*, were identified within the QTL region code for leucine-rich repeat proteins, a well-established class of R genes in plants (Kourelis and Van Der Hoorn 2018). These genes are different from several other candidate genes previously identified for YMD resistance in cultivated soybean, *Glyma.18G025100* (Yadav et al. 2015; Rahman et al. 2023), *Glyma.13G194500* (Rahman et al. 2023), *RNA-dependent RNA polymerase* (Khosla et al. 2021), and *Suppressor of Gene Silencing* (Khosla et al. 2021; Mandahal et al. 2024). The different set of genes identified through molecular analysis further substantiates the test of allelism, which suggested that distinct genetic loci confer YMD resistance in the cultivated and wild soybean sources. This molecular evidence reinforces the hypothesis that the resistance mechanisms in the two parental lines are genetically independent. Therefore, the utilization of wild soybean would not only broaden the genetic diversity of cultivated soybean but also introduce novel resistance alleles.

Interestingly, although the resistant parent PI 393551 and F₂ plant with a disease score of 1 appeared phenotypically symptomless, variable PCR amplification was obtained using virus-specific markers. Specifically, no amplification was observed in PI 393551, suggesting an absence of detectable viral load. However, WGS confirmed the presence of both DNA-A and DNA-B components of MYMIV in the PI 393551 sample, indicating that the virus persists in a latent or asymptomatic state (Ilyas et al. 2022). The absence of PCR amplification, despite the detection of viral sequences in WGS, likely suggests a low-titer infection that was below the detection threshold of the conventional PCR method. While the introgression of resistant QTL can help in breeding YMD-resistant soybean varieties, YMD resistance is a complex interplay between the host and pathogen, with the vector serving as a critical third factor that significantly influences disease dynamics which must be carefully considered.

Conclusion

The study provides insights into the genetic architecture of YMD resistance, revealing it as a complex polygenic trait governed by epistatic interactions and lacking any

maternal effects. The identification of a stable locus on chromosome 14 using an integrated approach can enhance our understanding of the molecular mechanisms underlying YMD resistance. The two LRR protein-encoding genes were identified within the locus, supporting its likely role in conferring disease resistance. The tightly linked molecular markers identified can serve as valuable tools for marker-assisted selection and pyramiding of resistance genes into elite soybean cultivars. The introgression of this novel resistance locus from wild soybean into the cultivated background will broaden the genetic base and contribute to the development of high-yielding and YMD-resistant soybean varieties. Moreover, cultivation of inherently resistant soybean cultivars will reduce dependence on chemical control and promote a sustainable disease management strategy.

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Data availability The raw sequencing data generated in this study have been deposited in the NCBI Sequence Read Archive (SRA) under BioProject accession numbers, PRJNA1289852 (WGS) and PRJNA1284629 (GBS).

Declarations

Conflict of interest The authors declare no conflict of interest.

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