Molecular mapping of sheath blight resistance QTLs from the wild rice Oryza rufipogon accession CR100438

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Research Article

Keywords: Rhizoctonia solani, O. rufipogon, Sheath blight, QTL mapping, SNP markers

Posted Date: July 20th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10326973/v1

Additional Declarations: No competing interests reported.

Abstract

Background

Sheath blight (ShB), caused by the necrotrophic fungus *Rhizoctonia solani* Kühn (AG1 IA), is one of the most destructive diseases of rice (*Oryza sativa* L.). Breeding resistant cultivars is constrained by the absence of complete resistance in cultivated germplasm and the quantitative nature of resistance. Therefore, exploiting wild rice germplasm is essential for developing ShB-resistant cultivars.

Results

The BC_2F_5 and BC_2F_6 populations derived from PR114/*O*. *rufipogon* accession CR100438 were evaluated against sheath blight. Seven disease-associated traits, namely plant height, lesion height, relative lesion height, disease score, number of tillers, number of infected tillers, and heading date, exhibited continuous variation, confirming the quantitative nature of ShB resistance. A subset of 118 progenies with consistent performance across seasons was selected for genotyping-by-sequencing. A total of 3,531 high-quality SNP markers were used for QTL mapping. Composite interval mapping identified 26 QTLs distributed across chromosomes 2, 3, 5, 6, 7, 8, 9, 10, 11, and 12. Six major QTLs (*qLH6*, *qLH11*, *qLH12*, *qRLH6*, *qRLH7*, and *qRLH12*) explained 9.10-14.26% of the phenotypic variance. A QTL hotspot containing *qLH6, qRLH6*, and *qDS6* was identified on chromosome 6. Pre-breeding lines with relative lesion height less than 25 percent and desirable agronomic traits such as days to flowering, plant height etc. have been identified for further use in breeding program.

Conclusions

This study identified promising QTLs with superior pre-breeding lines derived from *O*. *rufipogon* CR100438. These findings provide valuable genetic resources for fine mapping, marker-assisted breeding, and development of sheath blight-resistant rice cultivars.

Introduction

Rice (*Oryza sativa* L.) is the staple food for more than half of the world's population. It plays a vital role in food and nutritional security, particularly across South and Southeast Asia [1]. Global food demand is expected to increase as the world's population is projected to reach 10 billion by 2050 [2]. Sustaining rice productivity under increasing biotic stress is therefore a major challenge. Despite advances in breeding and crop management, insect pests and diseases continue to cause substantial yield losses in major rice-growing regions [3]. Global rice production during 2024-25 is estimated at 540.93 million metric tons. India contributes 149.07 million metric tons, accounting for 27.6% of the global production. Punjab alone produces approximately 14.43 million metric tons which represents 10% of total rice production of India.

Sheath blight (ShB), caused by the soil-borne basidiomycete *Rhizoctonia solani* Kühn, is one of the most destructive diseases of rice worldwide. In tropical Asia, it is second only to rice blast in terms of economic importance. Under favorable conditions, yield losses may reach up to 50% [4]. The pathogen survives as sclerotia and mycelial fragments in soil and infected crop residues between cropping seasons. Infection begins as water-soaked lesions on the leaf sheaths which later develop into grayish-white necrotic lesions with dark brown

margins. The long-term survival of the pathogen in soil makes disease management difficult [5]. In addition, the broad host range of *R. solani* and the high genetic diversity among its anastomosis groups further complicate the development of durable resistance [6]. Integrated disease management, including fungicide application, balanced nitrogen fertilization, optimum planting density, irrigation management, and crop residue removal can reduce sheath blight incidence. However, these approaches increase production costs, raise environmental concerns, and may promote reduced fungicide sensitivity in pathogen populations [7]. Therefore, the development of ShB-resistant cultivars remains the most sustainable and economically viable strategy for long-term sheath blight management. So far, no rice cultivar with complete resistance to sheath blight is available in the cultivated gene pool [8]. Most cultivated varieties possess only partial resistance, which is quantitatively inherited and strongly influenced by genetic background and environmental conditions [5].

Resistance to ShB is governed by multiple minor-effect quantitative trait loci [9]. Their expression is influenced by allelic heterogeneity, host-pathogen specificity, epistasis, and environmental conditions [1, 9]. To date, more than 110 loci associated with ShB resistance have been reported. These loci are distributed across all 12 rice chromosomes with enrichment on chromosomes 1, 3, 7, 9, 11, and 12 [1, 9]. However, most QTLs span broad genomic intervals which limit their direct use in marker-assisted breeding (MAB). Among these, only a few loci are stable, major-effect, and fine-mapped. These include *qSB-11*^{LE} [10], *qSB9-2*^{TQ} [11], *qSBR11-1* [12], and *qSB12*^{YSB} [13], *qShB-1.1* [14] that have been consistently detected. Among these, *qSB-11LE*, *qSB9-2*^{TQ}, *qSBR11-1*, *qSB12*^{YSB} and *qShB-1.1* have been fine-mapped to relatively small genomic intervals, facilitating candidate gene identification and marker development. Only a few rice varieties and breeding lines such as Tetep, Lemont, Teqing, Jasmine 85, and YSBR1 have been identified as reliable sources of sheath blight resistance across environments [6, 8]. Despite these advances, only a limited number of validated QTLs are currently available for routine deployment in breeding programmes. Therefore, identifying novel and stable resistance loci from diverse germplasm remains a priority for developing durable sheath blight-resistant rice cultivars [15].

Despite the significant economic impact of sheath blight caused by *Rhizoctonia solani*, the genetic potential of wild *Oryza* species for improving sheath blight resistance remains largely underexploited. To date, most reported quantitative trait loci (QTLs) for sheath blight resistance have been identified from cultivated rice germplasm, whereas only a few studies have investigated *Oryza rufipogon* and other wild *Oryza* species as donors of novel resistance alleles. As the wild progenitor of cultivated rice, *O. rufipogon* possesses the AA genome and is readily cross-compatible with *O. sativa*. Therefore, it represents an ideal donor for the identification and introgression of favorable alleles into elite cultivars. Hence, systematic exploration of *O. rufipogon* provides a promising opportunity to discover novel QTLs for sheath blight resistance, broaden the genetic base of resistance, and facilitate the development of durable sheath blight-tolerant rice cultivars.

Extensive germplasm screening at Punjab Agricultural University (PAU), India, has identified the wild rice *O. rufipogon* acc. CR100438 as a stable and reproducible donor of ShB tolerance [15]. A population of 118 backcross introgression lines (BILs) was subsequently developed using the susceptible cultivar PR114 as the recurrent parent and CR100438 as the donor. Therefore, the present study was undertaken to identify and map quantitative trait loci (QTLs) associated with sheath blight (ShB) resistance from *O. rufipogon* acc. CR100438 and to generate breeding resources for the genetic improvement of sheath blight tolerance in elite rice cultivars.

Materials and methods

Plant Material

The wild rice *O. rufipogon* accession CR100438 which consistently exhibited moderately resistance during five years of field screening at PAU, Ludhiana, was used as donor parent. The susceptible elite rice cultivar Punjab Rice 114 (PR114) was used as the recurrent parent. The cross was made between PR114 as female and *O. rufipogon* acc. CR100438 as male. A cross was made using PR114 as the female parent and *O. rufipogon* acc. CR100438 as the male parent. The F_1 plants were backcrossed twice with PR114 followed by selfing to develop the BC_2F_5 generation. In the present study, BC_2F_5 and BC_2F_6 progenies were screened against sheath blight and subsequently used to map QTLs underlying sheath blight resistance.

Screening for sheath blight disease reaction

The fungus *R. solani* was isolated from sheath blight-infected rice plants exhibiting disease symptoms. The infected leaf sheaths were collected, washed under running tap water, cut into small segments, surface-sterilized with 0.1% mercuric chloride solution, and rinsed repeatedly with sterile distilled water. The sterilized sheaths were placed on potato dextrose agar (PDA) and incubated at 26 ± 2°C. After 5–7 days, actively growing sclerotia/hyphal tips were sub-cultured on fresh PDA to obtain pure cultures. The isolate used for artificial inoculation belonged to the AG-1 IA anastomosis group (source/confirmation: Rice Pathology Laboratory, PAU). For mass multiplication, the pathogen was cultured on maize meal-sand (1:3 w/w) medium enriched with 20g sucrose, then autoclaved at 121°C (15 psi) for 30 minutes for 3 consecutive days [16]. These inoculated slants were transferred to a BOD incubator maintained at 26 ± 20°C for maturation up to 7 days.

The mature sclerotia cultures can be stored at 4°C until further multiplication. These cultures were used for mass multiplication on a maize meal-sand (1:3 w/w) medium enriched with 20g sucrose, which was then autoclaved at 121°C (15 psi) for 30 minutes for 3 consecutive days. The culture was used after 10 days of mature hyphal growth. The inoculated medium was incubated at 25 ± 20°C for 10 days to allow uniform colonization [16]. Artificial inoculation was performed at the maximum tillering stage (approximately 40 days after transplanting). Approximately 5g of colonized inoculum was placed at the center of each hill near the basal sheath region. A total of 1030 plants consisting of five plants/individual progeny of BC_2F_5 and three plants/individual progeny of BC_2F_6 populations were inoculated during kharif 2022 and 2023, respectively.

Disease assessment

The inoculated plants were assessed for disease after 21 days under field conditions, with PR114 as a susceptible check. Disease symptoms initiated on lower leaf sheaths and progressed upward, producing water-soaked greenish-grey lesions that later enlarged and coalesced into greyish-white centers bordered by irregular brown margins. Various parameters are used to record the disease index. In the present study, 7 disease variables were observed and recorded for BC_2F_5 and BC_2F_6 progenies. It included Plant Height (PH), Lesion Height (LH), Relative Lesion Height (RLH %), Number of Tillers (NT), Number of Infected Tillers (NIT), Heading Date (HD), and Disease Score (DS). The PH was measured from the base to the tip of the highest panicle, LH represents the maximum height at which the lesion appears, and RLH% is calculated from LH/PH×100. The data recorded for tillers included the total number of tillers per plant (NT) and the number of infected tillers (NIT). The heading date of a plant suggests the number of days to flowering. The severity of disease was assessed using a

0–9 scale based on the standard evaluation system (SES) for sheath blight. Disease scores of 0, 1, and 3 represent immune, resistant, and moderate resistance, respectively. Descriptive statistics (minimum, maximum, mean, standard deviation, skewness, and kurtosis) and Pearson's correlation coefficients among disease-related traits were computed using SAS, and significance was tested at $P < 0.05$.

Genotype-by-sequencing (GBS) analysis

High-quality DNA of BC_2F_6 progeny, along with parental lines PR114 and *O. rufipogon* CR100438, was extracted following the CTAB method [17]. These samples were processed for genotyping using double-digest restriction site-associated DNA sequencing (ddRAD-seq) by AgriGenome. The restriction enzymes used were *SphI* and *MlucI* (non-methylation-sensitive) and sequencing was performed on the Illumina HiSeq 2000/2500 platform. The reads obtained were aligned to the Nipponbare reference IRGSP 1.0 genome in fastq format to identify Single Nucleotide Polymorphisms (SNPs) using Bowtie2 [18]. The alignments were converted to SAM (Sequence Alignment/Map) format and then combined into a single BAM (Binary Alignment Map) using SAMtools 0.1.18. These BAM files were then used to call SNPs with Genome Analysis Toolkit (GATK). The major filtrations included a threshold for SNP call rate ≥ 0.7, minor allele frequency (MAF) ≥ 0.05, and SNP markers with < 10% missing data were used for the construction of the genetic map, and SNP markers with a Chi-square value > 10 were considered distorted and excluded from the linkage analysis.

Linkage mapping and QTL analysis

For marker ordering, the RECORD (REcombination Counting and ORDering) software was used with the default functions, the Kosambi mapping function, and a 30% recombination rate [19]. When multiple SNPs showed similar segregation patterns, all markers were placed in the same genetic bin, and only one representative marker was used for final map construction. Further, for linkage map construction, Map-Chart 2.0 software was used [20]. The QTL cartographer software was used to perform QTL analysis for sheath blight tolerance using the composite interval mapping (CIM) function with a window size of 10 cM (http://statgen.ncsu.edu/qtlcart/WQTLCart.htm). The threshold logarithm of the odds (LOD) scores was determined using 1000 permutations, and the observed phenotypic variance (R2) exhibited by the QTL was estimated [21].

Results

Screening of genotypes for sheath blight resistance

Evaluation of 118 BC_2F_5 and BC_2F_6 progenies across two growing seasons revealed substantial quantitative variation for all seven disease-associated traits (Supplementary table 1). Plant height (PH) ranged from 78.67 to 99.80 cm with a mean of 92.48 cm whreas lesion height (LH) varied between 21.25 and 30.90 cm (mean = 25.41 cm). Relative lesion height (RLH) showed a continuous distribution ranging from 22.21% to 34.06%. Density plot showed that majority of genotypes clustered within the moderately resistant category and a smaller proportion in the moderately susceptible category. The average number of tillers (NT) ranged from 6.34 to 16.42, while the number of infected tillers (NOIT) varied bteween 2.00 to 6.38. Disease score (DS) values ranged from 2.30 to 5.00 across population. The frequency distribution of all evaluated traits were continuous and approximately normal

that indicates quantitative nature of inheritance (Fig. 1). The estimate of skewness and kurtosis were low to moderate for all ShB-related traits (Table 1).

Table 1
Differential statistical parameters of mapping populations against sheath blight

Disease Variables	Statistical parameter						
	Minimum	Maximum	Range	Mean	Standard Deviation	Kurtosis	Skewness
Plant Height	78.67	99.80	21.13	92.48	3.54	1.20	-0.92
Lesion Height	21.25	30.90	9.65	25.41	2.05	-0.29	0.49
Relative Lesion Height	22.21	34.06	11.85	27.55	2.47	-0.17	0.46
No. of Tillers	6.34	16.42	10.08	10.04	1.95	0.71	0.68
No. of Infected Tillers	2.00	6.38	4.38	4.30	0.90	-0.28	0.12
Heading Date	2.30	5.00	2.70	3.39	0.53	-0.01	0.80
Score	65.00	89.00	24.00	79.80	6.82	-0.31	-0.94

Pearson's correlation analysis

Pearson's correlation analysis based on the pooled mean values of BC_2F_5 and BC_2F_6 progenies revealed significant association among several ShB-associated traits (Fig. 2). Significant relationships were observed the seven evaluated disease variables ($P < 0.01$ or $P < 0.0001$). LH showed a strong positive correlation with RLH ($r = 0.89$, $P < 0.0001$) and DS ($r = 0.85$, $P < 0.0001$), suggesting that lesion height is reliable indicator of overall disease development in the present study. In contrast, PH was negatively correlated with RLH ($r = -0.45$, $P < 0.0001$) and DS ($r = -0.28$, $P < 0.01$). Additionally, HD exhibited a weak positive correlation with PH ($r = 0.30$, $P < 0.01$). The NT and NOIT showed weak and non-significant correlation with most of the evaluated traits. Overall, LH and RLH showed strongest associations with DS, indicating their close relationship with sheath blight severity in the mapping population.

Genotyping and linkeage map construction

GBS of 118 BC_2F_5 and BC_2F_6 progenies with their parental lines generated 2,187,892 raw sequence variants comprising SNPs and INDELs (Supplementary table 2). Stringent quality filtering was applied to remove markers with ≥ 10% missing data, heterozygous SNPs, monomorphic loci, and INDELs. A set of 269,460 high-quality polymorphic SNPs were retained for linkage analysis. Based on Chi-sqaure analysis, markers exhibiting significant segregation distortion were subsequently excluded. The final dataset comprising 3531 polymorphic SNPs were subjectd to linkage mapping analysis. Only one representative marker was kept if more than two markers showing same segeragation pattern. Of these, 3,504 markers were successfully assigned to linkage groups corresponding to 12 rice chromosomes.

The genetic map spanned a total length of 704.175 cM with individual linkage group ranging from 53 cM to 79 cM (Supplementary table 3). The largest likeage groups was chromosome 3 (79 cM), whereas the smallest linkage group was 10 (53 cM). Marker density varied among linkage groups. The highest marker density was observed for chromosome 11 (360 markers) with an average marker distance of 0.15 cM.

QTL mapping for sheath blight resistance-associated traits

Composite interval mapping identified 26 QTLs governing seven sheath blight-associated traits, distributed across chromosomes 2, 3, 5, 6, 7, 8, 9, 10, 11, and 12 (Table 2). All detected loci exceeded the threshold LOD score of 2.5 and explained 5.14–14.26% of the phenotypic variance (PVE). Three QTLs controlling plant height namely *qPH2*, *qPH7*, and *qPH8*, were identified on chromosomes 2, 7, and 8, respectively. Among these, *qPH8* exhibited the highest LOD score (10.66) and explained 8.34% of PVE, followed by *qPH2* (LOD = 10.59; PVE = 8.04%) and *qPH7* (LOD = 6.0; PVE = 5.56%). Notably, *qPH7* and *qPH8* co-localized with tiller nuber QTLs *qNT7* and *qNT8*, respectively. These results indicate that overlapping genomic intervals influence both plant height and tillering ability. Five QTLs associated with lesion height (*qLH5*, *qLH6*, *qLH10*, *qLH11*, and *qLH12*) were mapped on chromosomes 5, 6, 10, 11, and 12 with PVE ranged from 5.16–12.60. Among these, *qLH6* exhibited the highest PVE of 12.60% with LOD 6.52, followed by *qLH12* (LOD = 7.64; PVE = 10.89%) and *qLH 11* (LOD = 9.32; PVE = 10.48%). The *qLH6* co-localized with QTLs *qRLH6*, *qDS6*, and *qHD6* which forms a major QTL cluster associated with several disease parameters. A similar QTL cluster was observed on chromosome 10 where *qLH10* coincided with *qRLH10* and *qDS10*.

Table 2
QTLs mapped for tolerance against sheath blight in *O. rufipogon* (CR100438) derived populations

Trait Name	LGs	QTLs	Position	Flanking markers	LOD Value	PVE (%)	Putative Candidate gene
Plant Height	2	*qPH2*	1636	2:9780219-2:11255113	10.5943	8.04	-
	7	*qPH7*	2607	7:5273211-7:26501958	6.0115	5.56	-
	8	*qPH8*	286	8:13638337-8:9711042	10.6634	8.34	-
Lesion Height	5	*qLH5*	357	5:1603843-5:1603843	3.1688	6.02	*LOC_Os05g03690* (Retrotransposon protein)
	6	*qLH6*	668	6:987759-6:22497899	6.529	12.60	-
	10	*qLH10*	1596	10:21258620-10:17555834	5.1647	5.16	*LOC_Os10g38100* (Thaumatin-like protein) *LOC_Os10g39520* (MLO domain containing protein)
	11	*qLH11*	1908	11:10371515-11:10371344	9.3293	10.48	*LOC_Os11g18366* (Cycloartenol synthase)
	12	*qLH12*	1607	12:663331-12:663254	7.6432	10.89	*LOC_Os12g02170* (Retrotransposon protein)
Relative Lesion Height	3	*qRLH3*	949	3:13462926-3:26029188	3.7403	10.83	-
	5	*qRLH5*	2275	5:119507755:21774564	5.5488	8.00	-
	6	*qRLH6*	2232	6:24827970-6:15689215	2.5645	9.10	-
	7	*qRLH7*	529	7:164990807:2343927	2.768	6.41	-
	10	*qRLH10*	1723	10:1298960110:8516188	7.7592	14.26	-
	11	*qRLH11*	825	11:17982161-11:15667654	5.7018	8.22	-
	12	*qRLH12*	355	12:22869469-12:3990596	6.7658	9.96	-
No. of Tillers	7	*qNT7*	383	7:11926466-7:8677651	2.8234	7.73	-
	8	*qNT8*	473	8:7182240-8:12259527	2.7346	5.14	-
	9	*qNT9*	49	9:7929974-9:7835155	2.8839	8.06	-
	11	*qNT11*	297	11:25677271-11:25677253	4.5644	5.82	-
No. of Infected Tillers	9	*qNOIT9*	2551	9:4914338-9:12696996	3.4003	6.60	-

Trait Name	LGs	QTLs	Position	Flanking markers	LOD Value	PVE (%)	Putative Candidate gene
	10	*qNOIT10*	861	10:2318348-10:12737888	4.4235	7.12	-
Heading date	6	*qHD6*	1117	6:21274055-6:10660354	3.4716	8.16	-
	11	*qHD11*	3045	11:18765388-11:28122332	4.52	5.49	-
Score	2	*qDS2*	2540	2:32339218-2:30105	3.2985	5.51	-
	6	*qDS6*	215	6:14872222-6:25478495	3.0361	5.06	-
	10	*qDS10*	1037	10:15253044-10:15704671	3.5474	5.85	*LOC_Os10g29620* (Tyrosine protein kinase domain containing protein)

Table 3
Promising BC_2F_6 progenies identified for sheath blight resistance

Genotype	PH (cm)	LH (cm)	RLH (%)	NT	NOIT	DS	HD (days)	QTLs
7193	93.85 ± 1.69	22.85 ± 0.65	24.24 ± 0.28	10.50 ± 1.36	4.75 ± 0.16	2.80 ± 0.45	88.00 ± 1.50	*qRLH5*, *qLH5*,*qNT11*, *qDS2, qDS6*
7198	97.10 ± 2.98	21.50 ± 0.84	22.21 ± 1.00	11.50 ± 0.49	5.09 ± 0.35	2.30 ± 0.25	84.50 ± 1.30	*qRLH11*, *qLH6*, *qDS2, qDS6*
7199	95.40 ± 4.59	22.80 ± 0.17	23.91 ± 1.01	12.75 ± 0.21	5.50 ± 0.30	3.00 ± 0.06	85.50 ± 1.05	*qRLH11, qLH6*, *qNT11*, *qNT8*
7200	98.00 ± 3.49	23.00 ± 0.27	23.46 ± 0.57	12.34 ± 0.97	4.84 ± 0.64	3.00 ± 0.28	84.50 ± 2.33	*qRLH5*, *qRLH12*
7203	96.15 ± 1.82	23.85 ± 0.38	24.82 ± 0.18	12.00 ± 0.82	5.00 ± 0.35	3.00 ± 0.52	86.50 ± 2.67	*qRLH10*, *qLH5, qLH11*
7205	92.15 ± 2.74	21.25 ± 0.75	22.98 ± 0.13	10.75 ± 0.23	4.88 ± 0.33	2.60 ± 0.28	83.50 ± 2.25	*qRLH10*, *qLH11, qPH8*
7207	92.05 ± 2.67	22.55 ± 0.48	24.47 ± 0.49	12.75 ± 0.61	3.63 ± 0.38	3.00 ± 0.28	83.50 ± 1.22	*qRLH5*, *qRLH7*, *qLH5,qNT11, qNOIT9*
7222	96.60 ± 3.36	23.80 ± 0.93	24.65 ± 1.19	11.00 ± 0.68	4.67 ± 0.39	3.00 ± 0.12	82.00 ± 0.66	*qLH5, qDS6*
7236	95.0 ± 2.26	22.65 ± 1.01	23.80 ± 0.85	10.25 ± 1.06	3.88 ± 0.25	3.00 ± 0.52	85.50 ± 1.57	*qRLH10*, *qPH8*, *qNOIT9*, *qDS6*
7238	97.38 ± 2.92	24.25 ± 0.48	24.94 ± 1.11	9.34 ± 1.15	4.75 ± 0.31	3.00 ± 0.26	86.00 ± 2.24	*qRLH6*,*qRLH7*, *qLH6*
7241	94.95 ± 1.84	22.80 ± 0.96	23.95 ± 1.50	11.42 ± 0.95	4.50 ± 0.16	2.80 ± 0.23	86.50 ± 2.25	*qRLH12*, *qRLH10*, *qLH11*,*qNT11*, *qDS2*
7257	93.40 ± 3.11	22.70 ± 1.27	24.31 ± 2.11	10.84 ± 0.38	5.25 ± 0.28	3.00 ± 0.36	78.00 ± 2.27	*qRLH7*,*qNT7*
7273	96.40 ± 1.81	22.60 ± 0.64	23.44 ± 0.84	12.33 ± 0.75	5.67 ± 0.33	3.00 ± 0.08	67.00 ± 1.75	*qRLH3*, *qRLH12*
7277	95.60 ± 1.31	23.00 ± 1.39	24.12 ± 1.15	11.09 ± 0.91	5.75 ± 0.22	2.60 ± 0.04	80.00 ± 2.35	*qRLH3*, *qRLH5*, *qHD11*
7279	94.80 ± 0.43	23.35 ± 0.82	24.67 ± 0.98	10.50 ± 0.99	5.75 ± 0.09	2.80 ± 0.12	82.00 ± 1.13	*qNT8*
PR114	**100.36 ± 0.87**	**69.07 ± 0.79**	**68.82 ± 1.16**	**8.78 ± 0.853**	**7.25 ± 0.34**	**9.0 ± 0.29**	**109.03 ± 1.66**	-

**PH-Plant Height; LH-Lesion Height; RLH-Relative lesion height; NT-Number of tiller; NOIT-Number of infected tiller; DS-Disease severity HD-Heading date; Data represents the mean of three replicates.*

Seven QTLs associated with relative lesion height were detected on chromosomes 3, 5, 6, 7, 10, 11, and 12. Among these, *qRLH10* had the highest LOD (7.75) with 14.26% PVE, followed by *qRLH3* (LOD = 3.7; PVE = 10.83%)

and *qRLH12* (LOD = 6.7; PVE = 9.96%). Four QTLs controlling number of tillers (*qNT7*, *qNT8*, *qNT9*, and *qNT11*) were mapped to chromosomes 7, 8, 9, and 11, respectively, each accounting for 5.14–8.06% of PVE. Two QTLs for number of infected tillers (*qNOIT9* and *qNOIT10*) were identified on chromosomes 9 and 10, explaining 6.60% and 7.12% PVE, respectively. Three QTLs governing disease score (*qDS2*, *qDS6*, and *qDS10*) were identified on chromosomes 2, 6, and 10, each contributing approximately 5–6% PVE. In addition, two QTLs (*qHD6* and *qHD11*) for heading date were mapped to chromosomes 6 and 11, explaining 8.16% and 5.49% of PVE, respectively.

Promising genotypes for varietal development

To identify superior introgression lines, BC_2F_6 progenies were selected based on key disease parameters including RLH (≤ 25%), LH (≤ 25%), and DS (≤ 3.0). with least PH (≤ 100 cm) and HD (≤ 90 days). Fifteen progenies were identified as promising sources of sheath blight resistance (**Table XX**). Among these, genotype 7198 emerged as the most promising line which exhibits the lowest DS (2.30) and RLH (22.21%) with a desirable plant height of 97.10 cm. Genotype 7205 also displayed a superior combination of disease tolerance (DS = 2.60; RLH = 22.98%) and agronomic performance. Based on their consistently low disease severity and favorable plant architecture, these two lines were classified as highly promising. Five additional progenies (7273, 7200, 7236, 7199, and 7241) were categorized as very promising. These lines show RLH values ranging from 23.44 to 23.95% and DS between 2.80 and 3.00. These lines maintained plant heights between 94–98 cm and exhibited moderate to high tillering ability. The remaining eight progenies (7277, 7193, 7257, 7207, 7222, 7279, 7203, and 7238) were classified as promising which showed RLH values between 24.12 and 24.94% and DS ranging from 2.60 to 3.00. Notably, several of these lines combined reduced disease severity with favorable agronomic traits such as desirable plant height and adequate tiller number. Overall, the selected progenies exhibited a favorable combination of sheath blight tolerance and agronomic performance. These lines represent valuable pre-breeding materials for the development of sheath blight-tolerant rice cultivars and provide potential donors for validation and deployment of the identified QTLs in marker-assisted breeding programs.

Discussion

Sheath blight caused by *R. solani* is one of the most destructive diseases of rice that poses a serious threat to the global rice productivity. Despite extensive screening, complete resistance has not been identified in cultivated rice germplasm [9, 13]. Therefore, the present study aimed to identify novel QTLs conferring resistance to sheath blight from the wild progenitor *O. rufipogon.* The backcross population developed from PR114 and *O. rufipogon* acc. CR100438 exhibited continuous phenotypic variance for disease-related traits. The identification of 26 QTLs across ten chromosomes further confirmed highly polygenic nature of sheath blight resistance. Previous studies also reported that sheath blight resistance is controlled by multiple minor- and moderate-effect QTLs [14, 22–25]. Such quantitative resistance is often considered more durable because it imposes lower selection pressure on pathogen populations and is therefore less prone to breakdown by virulent strains [12, 13].

O. rufipogon as a valuable donor of pathogen and insect resistance

Wild rice species are inavaluable reservoirs of novel alleles for many useful agronomic traits. They harbour extensive genetic diversity that has largely been lost during domestication. Among them, *O. rufipogon* is the immediate progenitor of cultivated rice which has been widely exploited for enhancing yield, disease resistance, and adaptation to environmental stresses [26–29]. Many QTLs conferring resistance to economically important diseases, including bacterial blight, blast, and bacterial streak disease have been identified from *O. rufipogon* and

successfully transferred into elite cultivars [29–33]. Wild rice *O. rufipogon* also served as a valuable donor for resistance to destructive insect pests such as brown planthopper, white-backed planthopper, and other pests [34, 35]. Earlier studies involving *O. rufipogon*, *O. nivara*, and other wild rice species have consistently demonstrated that wild introgressions harbor novel QTLs for sheath blight resistance [36–38].

In the present study, the donor accession CR100438 constantly exhibited resistance to sheath blight over two years. The continuous distribution of disease-related traits was observed among BCF_5 and BC_2F_6 progenies that indicated the resistance in *O. rufipogon* is polygenic in nature. These findings further establish CR100438 as a valuable donor for broadening the genetic base of sheath blight resistance in elite rice breeding programmes.

Identification of QTL clusters governing sheath blight resistance

A total of 26 QTLs associated with disease and asscoated agronomic traits were identified across ten rice chromosomes. Most QTLs explained moderate phenotypic variance (5.06–14.26%), confirming that sheath blight resistance in *O. rufipogon* acc. CR100438 is governed by multiple minor-effect loci. Similar genetic architecture has been reported in cultivated and wild rice populations. Our previous study also identified twenty QTLs explaining 3–10% of the phenotypic variance, further supporting the quantitative inheritance of this trait [6, 24]. The QTL intervals identified in this study were relatively broad and each contained several annotated genes. Broad confidence intervals are common in interspecific mapping populations because recombination is frequently suppressed within introgressed donor segments [39, 40]. In addition, *O. rufipogon* harbors many sterility- and gamete-killer loci that cause segregation distortion [41, 42]. Stringent filtering of these distorted markers combined with moderate population size, further reduced mapping resolution. As a result, only the relatively smaller QTL intervals were considered for candidate gene analysis.

A major finding of this study was the identification of two QTL hotspots. The chromosome 6 interval (9.8–24.8 Mb) harboured *qLH6*, *qRLH6*, *qDS6*, and *qHD6* whereas the chromosome 10 interval (8.5–21.2 Mb) contained *qLH10*, *qRLH10*, and *qDS10*. However, present mapping resolution does not allow us to distinguish between pleiotropy and tight linkage among closely linked loci. The chromosome 6 hotspot overlaps with previously reported QTLs, including *qShB6*, *qShB-mc*, *qNOL6.1*, *qPH6.1*, and *qRTL6* which have been mapped within the same genomic region [6, 24, 25]. Independent mapping studies have repeatedly identified this region, suggesting that chromosome 6 harbours conserved loci governing quantitative sheath blight resistance. [23, 25]. The second hotspot on chromosome 10 conataining *qLH10*, *qRLH10*, and *qDS10* appeared particularly interesting because this region has reported less frequently than hotspot on chromosome 6. This hotspot spanned 8.5–21.70 Mb on rice genome and contained numerous putative disease resistance candidate genes.

Besides the major QTL hotspots on chromosomes 6 and 10, some additional QTLs were detected on chromosomes 2, 3, 5, 7, 11, and 12. Among these, chromosome 11 and 12 repeatedly harboured QTLs controlling lesion height and relative lesion height. Particularly noteworthy were *qLH11* and *qRLH11*, which mapped to the 10.3–17.9 Mb region of chromosome 11. This interval overlaps with previously reported QTLs including *qShB11-2*, *qSBR11-2*, and *qSBR11-3*, whereas the adjacent region also contains the widely validated major loci *qSBR11-1* and *qSB11LE* [10, 12]. These QTLS have subsequently been pyramided into elite cultivars through marker-assisted breeding [43, 44]. Similarly, *qLH12* and *qRLH12* co-localized with previously reported QTLs *qLH12.2*, *qDS12.2*,

qSB12, *qDR12*, and *qRTL12* [13, 22, 24]. Detection of such conserved loci across independent studies represent promising targets for marker-assisted breeding.

Among the candidate genes, the *qLH10* contained LOC_Os10g38100 encoding thaumatin-like protein (TLP). TLP belongs to a family of pathogenesis-related proteins (PR-5) that exhibit antifungal activity through inhibition of fungal growth and induction of defence responses. Thaumatin-like proteins have been frequently detected in controlling ShB resistance [6, 15, 24]. The antifungal role of TLP has been widely demonstrated in sheath blight resistance in rice [45]. Another candidate within the same interval is LOC_Os10g39520 which encodes an MLO-domain protein. The MLO proteins play an important role in plant–pathogen interactions. Loss-of-function in alleles of these genes often confers broad-spectrum resistance against powdery mildew in crop species [46]. Whether LOC_Os10g39520 contributes to quantitative resistance against *R. solani* remains unknown. LOC_Os10g29620 which encodes a protein kinase, is another potential candidate gene underlying *qDS10*. Receptor-like and serine/threonine protein kinases are key components in pathogen recognition and activation of immune signalling pathways in rice [47]. Despite their reported roles in plant immunity, direct evidence linking these genes to sheath blight resistance in *O. rufipogon* is still lacking and warrants validation through high-resolution mapping, expression profiling, and targeted genome editing.

Identification of promising ShB resistant lines for pre-breeding

Beneficial alleles from *O. rufipogon* have previously been deployed for improving yield, nutrient-use efficiency and resistance to biotic and abiotic stresses [48]. In the present study, fifteen BC_2F_6 introgression lines combined reduced sheath blight severity with desirable agronomic performance. Among these, genotypes 7198 and 7205 exhibited the most favorable combination of reduced lesion development, low disease score, moderate plant height, and acceptable maturity duration. Genotype 7198 exhibited the lowest disease score (2.3), together with reduced lesion height and desirable plant height. It carried favourable alleles at *qLH6*, *qRLH11*, *qDS2*, and *qDS6*, indicating that pyramiding QTLs controlling different components of disease severity substantially improved resistance. Likewise, genotype 7205 combined *qRLH10* and *qLH11* with the plant height QTL *qPH8*, resulting in low disease severity without compromising plant architecture. The pyramiding of major- and minor-effect QTLs were attempted for breeding rice cultivars resistance to ShB [43, 44, 49, 50]. Similar accumulation of multiple small-effect QTLs has been shown to provide more durable and stable resistance than single-QTL deployment [22, 23]. Such QTL combinations are particularly valuable because they improve disease resistance without compromising agronomic performance.

Interestingly, some promising lines possessed QTL combinations originating from different chromosomes. For example, genotype 7241 carried resistance QTLs for LH and RLH on chromosomes 10, 11, and 12 together with *qNT11*, whereas 7193, 7199, and 7207 combined lesion-related QTLs with tillering QTLs (*qNT8* or *qNT11*). These results demonstrate that favourable wild introgressions can simultaneously improve sheath blight resistance and agronomic performance. The co-introgression of disease-resistance and agronomic QTLs is particularly advantageous because plant height, tiller number, and heading date strongly influence canopy architecture, disease development, and grain yield. Similar pyramiding of multiple minor-effect QTLs has been shown to produce more durable and stable resistance than deployment of individual loci [6, 24, 25]. These introgression lines provide valuable pre-breeding material for validating major QTL clusters, developing near-isogenic lines, and pyramiding sheath blight resistance loci into elite rice cultivars.

Conclusion

The present study demonstrates that *Oryza rufipogon* accession CR100438 is a valuable source of novel alleles for improving sheath blight resistance in rice. Stable genomic regions associated with sheath blight resistance were identified, including two prominent QTL hotspots on chromosomes 6 and 10. These regions provide promising targets for fine mapping and candidate gene validation. The study also identified superior introgression lines combining improved sheath blight resistance with desirable agronomic performance, highlighting the potential of wild rice for broadening the genetic base of cultivated rice. Together, these genomic regions and pre-breeding lines provide valuable resources for marker-assisted breeding and QTL pyramiding aimed at developing rice cultivars with durable sheath blight resistance.

Abbreviations

ShB	Sheath blight
QTL	Quantitative trait locus
GWAS	Genome-wide association study
SNP	Single nucleotide polymorphism
RIL	Recombinant inbred line
PVE	Phenotypic variance explained
LOD	Logarithm of odds
CIM	Composite interval mapping
MAS	Marker-assisted selection
LH	Lesion Height
RLH	Relative Lesion Height
PH	Plant Height
DS	Disease Score
HD	Heading Date
NOIT	Number of Infected Tiller

Declarations

Acknowledgements

We extend our sincere gratitude to the International Rice Research Institute (Philippines) and the Central Rice Research Institute (Cuttack) for generously providing wild rice accessions.

Funding

The authors are thankful to the Indian Council for Agricultural Research (ICAR), New Delhi for providing funding under the Project “Incentivizing Research in Agriculture project-IV-Molecular genetic analysis of

resistance/tolerance to different stress (Rice) CS/F. No. 16-8/17-IAIV (ICAR-65)" and Molecular breeding for improvement of tolerance to biotic and abioticstresses, yield, and quality traits in crops-Rice (ICAR-64).

Competing Interest

The authors have no relevant financial or non-financial interests to disclose.

Authors' contribution

KN- Conceived the idea; KN, SJ, AB, SR- Screening of the germplasm, phenotypic and genotypic data analysis; JSL- Provided the sheath blight inoculum for screening. KT, KK, KSD-Performed QTL mapping and Manuscript writing ; KN,YV, RK- Handling and maintenance of the germplasm, KN, KK- Writing and proofreading of the manuscript. All the authors read the manuscript carefully and approved it.

Ethics approval

This article does not contain any studies with human participants or animals performed by any of the authors.

Consent to participate

Not applicable.

Consent for publication

Informed consent was obtained from all individual participants included in this study.

Availability of data and materials

All the data generated and analysed in this study are available in this article as supplementary materials.

References

1. Senapati M, Tiwari A, Sharma N et al (2022) Rhizoctonia solani Kühn Pathophysiology: Status and Prospects of Sheath Blight Disease Management in Rice. Front Plant Sci 13:881116. https://doi.org/10.3389/fpls.2022.881116
2. Hickey LT, Hafeez N, Robinson A H, et al (2019) Breeding crops to feed 10 billion. Nat Biotechnol 37:744–754. https://doi.org/10.1038/s41587-019-0152-9
3. Chen J, Xuan Y, Yi J et al (2023) Progress in rice sheath blight resistance research. Front Plant Sci 14:1141697. https://doi.org/10.3389/fpls.2023.1141697
4. Zheng A, Lin R, Zhang D et al (2013) The evolution and pathogenic mechanisms of the rice sheath blight pathogen. Nat Commun 4:1424. https://doi.org/10.1038/ncomms2427
5. Li D, Li S, Wei S, Sun W (2021) Strategies to Manage Rice Sheath Blight: Lessons from Interactions between Rice and Rhizoctonia solani. Rice 14:21. https://doi.org/10.1186/s12284-021-00466-z
6. Neelam K, Aggarwal SK, Kumari S et al (2024) Molecular Mapping and Transfer of Quantitative Trait Loci (QTL) for Sheath Blight Resistance from Wild Rice *Oryza nivara* to Cultivated Rice (Oryza sativa L). Genes 15:919. https://doi.org/10.3390/genes15070919

7. Singh P, Mazumdar P, Harikrishna JA, Babu S (2019) Sheath blight of rice: a review and identification of priorities for future research. Planta 250:1387–1407. https://doi.org/10.1007/s00425-019-03246-8
8. Feng Z, Gao P, Wang G et al (2025) Natural variation in SBRR1 shows high potential for sheath blight resistance breeding in rice. Nat Genet 57:2004–2016. https://doi.org/10.1038/s41588-025-02281-4
9. Molla KA, Karmakar S, Molla J et al (2020) Understanding sheath blight resistance in rice: the road behind and the road ahead. Plant Biotechnol J 18:895–915. https://doi.org/10.1111/pbi.13312
10. Zuo S, Yin Y, Pan C et al (2013) Fine mapping of qSB-11 LE, the QTL that confers partial resistance to rice sheath blight. Theor Appl Genet 126:1257–1272. https://doi.org/10.1007/s00122-013-2051-7
11. Zuo S, Zhang Y, Yin Y et al (2014) Fine-mapping of qSB-9 TQ, a gene conferring major quantitative resistance to rice sheath blight. Mol Breed 34:2191–2203. https://doi.org/10.1007/s11032-014-0173-5
12. Channamallikarjuna V, Sonah H, Prasad M et al (2010) Identification of major quantitative trait loci qSBR11-1 for sheath blight resistance in rice. Mol Breed 25:155–166. https://doi.org/10.1007/s11032-009-9316-5
13. Wang Y, Sun Q, Zhao J et al (2023) Fine mapping and candidate gene analysis of qSB12YSB, a gene conferring major quantitative resistance to rice sheath blight. Theor Appl Genet 136:246. https://doi.org/10.1007/s00122-023-04482-z
14. Bal A, Samal P, Mukherjee AK et al (2026) High-resolution mapping of stable sheath blight resistance QTL qShb-1.1 from rice cultivar CR 1014. https://doi.org/10.1007/s13237-026-00669-z. Nucleus
15. Aggarwal SK, Malik P, Neelam K et al (2022) Genome-wide association mapping for identification of sheath blight resistance loci from wild rice *Oryza rufipogon*. Euphytica 218:144. https://doi.org/10.1007/s10681-022-03091-z
16. Lore JS, Hunjan MS, Singh P et al (2013) Phenotyping of Partial Physiological Resistance to Rice Sheath Blight. J Phytopathol 161:224–229. https://doi.org/10.1111/jph.12055
17. Saghai-Maroof MA, Soliman KM, Jorgensen RA, Allard RW (1984) Ribosomal DNA spacer-length polymorphisms in barley: mendelian inheritance, chromosomal location, and population dynamics. Proc Natl Acad Sci USA 81:8014–8018. https://doi.org/10.1073/pnas.81.24.8014
18. Langmead B, Salzberg SL (2012) Fast gapped-read alignment with Bowtie 2. Nat Methods 9:357–359. https://doi.org/10.1038/nmeth.1923
19. Van Os H, Stam P, Visser RGF, Van Eck HJ (2005) RECORD: a novel method for ordering loci on a genetic linkage map. Theor Appl Genet 112:30–40. https://doi.org/10.1007/s00122-005-0097-x
20. Voorrips RE (2002) MapChart: Software for the Graphical Presentation of Linkage Maps and QTLs. J Hered 93:77–78. https://doi.org/10.1093/jhered/93.1.77
21. Churchill GA, Doerge RW (1994) Empirical threshold values for quantitative trait mapping. Genetics 138:963–971. https://doi.org/10.1093/genetics/138.3.963
22. Pinson SRM, Capdevielle FM, Oard JH (2005) Confirming QTLs and Finding Additional Loci Conditioning Sheath Blight Resistance in Rice Using Recombinant Inbred Lines. Crop Sci 45:503–510. https://doi.org/10.2135/cropsci2005.0503
23. Yadav S, Anuradha G, Kumar RR et al (2015) Identification of QTLs and possible candidate genes conferring sheath blight resistance in rice (*Oryza sativa* L). SpringerPlus 4:175. https://doi.org/10.1186/s40064-015-0954-2

24. Rasool S, Babbar A, Kumari S et al (2025) Molecular mapping and transfer of sheath blight resistance QTLs from PAU-shb8 to cultivated rice PR-121. Mol Genet Genomics 300:21. https://doi.org/10.1007/s00438-024-02220-8
25. Thesiya MR, Lore JS, Bhatia D et al (2025) QTL mapping and candidate gene identification for sheath blight resistance in rice (*Oryza sativa*) F3 progenies using QTL-seq analysis. Trop plant pathol 50:63. https://doi.org/10.1007/s40858-025-00739-8
26. McCouch SR, Sweeney M, Li J et al (2007) Through the genetic bottleneck: *O. rufipogon* as a source of trait-enhancing alleles for O. sativa. Euphytica 154:317–339. https://doi.org/10.1007/s10681-006-9210-8
27. Roy SC, Shil P (2025) Comprehensive genetic diversity revealed in the pre-breeding RILs (*O. sativa* × *O. rufipogon)* with enhanced yield and pigmented grain quality. Front Genet 16:1659937. https://doi.org/10.3389/fgene.2025.1659937
28. Sridhar M, Gireesh C, Balakrishnan D et al (2025) Wild species *Oryza rufipogon* as a potential donor for development of interspecific backcross population in the background of Samba Mahsuri (BPT5204) and detection of yield enhancing QTLs. Plant Physiol Rep 30:846–865. https://doi.org/10.1007/s40502-025-00904-w
29. Eizenga GC, Grunden QP-H, Jia MH et al (2026) Resistance to rice blast disease identified among progeny of a Presidio × *Oryza rufipogon* advanced backcross population. Crop Sci 66:e70304. https://doi.org/10.1002/csc2.70304
30. He W, Huang D, Li R et al (2012) Identification of a Resistance Gene bls1 to Bacterial Leaf Streak in Wild Rice *Oryza rufipogon* Griff. J Integr Agric 11:962–969. https://doi.org/10.1016/S2095-3119(12)60087-2
31. Zheng C, Jiang N, Zhao X et al (2020) Identification of the blast resistance gene *Picl(t)* from Chaling common wild rice (*Oryza rufipogon* Griff). J Phytopathol 168:211–219. https://doi.org/10.1111/jph.12883
32. Huang F, He N, Yu M et al (2023) Identification and fine mapping of a new bacterial blight resistance gene, *Xa43(t)*, in Zhangpu wild rice (*Oryza rufipogon*). Plant Biol J 25:433–439. https://doi.org/10.1111/plb.13502
33. Bhatia SK, Vikal Y, Kaur P et al (2024) Introgression and Mapping of a Novel Bacterial Blight Resistance Gene *xa49(t)* from *Oryza rufipogon* acc. CR100098A into *O. sativa*. Phytopathology® 114:2412–2420. https://doi.org/10.1094/PHYTO-02-24-0061-R
34. Guo X, Wang X, Luo L et al (2022) Identification of resistance of Guangxi (Oryza rufipogon Griff.) to white-backed planthopper with stem evaluation method. Genet Resour Crop Evol 69:2345–2359. https://doi.org/10.1007/s10722-022-01376-9
35. Kaur P, Neelam K, Sarao PS et al (2022) Molecular mapping and transfer of a novel brown planthopper resistance gene bph42 from *Oryza rufipogon* (Griff.) To cultivated rice (Oryza sativa L). Mol Biol Rep 49:8597–8606. https://doi.org/10.1007/s11033-022-07692-8
36. Senapati M, Tiwari A, Vinod KK et al (2024) High-density single nucleotide polymorphism-based linkage mapping reveals novel quantitative trait loci for rice (*Oryza sativa*) sheath blight resistance from wild rice (*Oryza rufipogon*)-derived introgression line. Plant Breeding 143:828–839. https://doi.org/10.1111/pbr.13210
37. Eizenga GC, Prasad B, Jackson AK, Jia MH (2013) Identification of rice sheath blight and blast quantitative trait loci in two different *O. sativa*/*O. nivara* advanced backcross populations. Mol Breed 31:889–907. https://doi.org/10.1007/s11032-013-9843-y
38. Eizenga GC, Li D, Jia MH et al (2022) Identification of sheath blight QTL in a LaGrue x Oryza nivara rice advanced backcross population. Euphytica 218:158. https://doi.org/10.1007/s10681-022-03101-0

39. Xiao J, Li J, Grandillo S et al (1998) Identification of Trait-Improving Quantitative Trait Loci Alleles From a Wild Rice Relative, Oryza rufipogon. Genetics 150:899–909. https://doi.org/10.1093/genetics/150.2.899
40. Wang S, Cao M, Ma X et al (2017) Integrated RNA Sequencing and QTL Mapping to Identify Candidate Genes from *Oryza rufipogon* Associated with Salt Tolerance at the Seedling Stage. Front Plant Sci 8:1427. https://doi.org/10.3389/fpls.2017.01427
41. Koide Y, Shinya Y, Ikenaga M et al (2012) Complex genetic nature of sex-independent transmission ratio distortion in Asian rice species: the involvement of unlinked modifiers and sex-specific mechanisms. Heredity 108:242–247. https://doi.org/10.1038/hdy.2011.64
42. Yang Y, Pu Q, Lv Y et al (2025) Identification and Genetic Analysis of Collinearity Loci for Interspecific Hybrid Sterility in Genus *Oryza*. Rice 18:43. https://doi.org/10.1186/s12284-025-00803-6
43. Ramalingam J, Raveendra C, Savitha P et al (2020) Gene Pyramiding for Achieving Enhanced Resistance to Bacterial Blight, Blast, and Sheath Blight Diseases in Rice. Front Plant Sci 11:591457. https://doi.org/10.3389/fpls.2020.591457
44. Sangeeta S, Barik SR, Mishra A et al (2026) Pyramiding QTLs for durable sheath blight resistance in the popular rice variety. 'Maudamani ' Sci Rep 16:19875. https://doi.org/10.1038/s41598-026-50068-2
45. Liu J-J, Sturrock R, Ekramoddoullah AKM (2010) The superfamily of thaumatin-like proteins: its origin, evolution, and expression towards biological function. Plant Cell Rep 29:419–436. https://doi.org/10.1007/s00299-010-0826-8
46. Kusch S, Panstruga R (2017) *mlo* -Based Resistance: An Apparently Universal Weapon to Defeat Powdery Mildew Disease. MPMI 30:179–189. https://doi.org/10.1094/MPMI-12-16-0255-CR
47. Tang D, Wang G, Zhou J-M (2017) Receptor Kinases in Plant-Pathogen Interactions: More Than Pattern Recognition. Plant Cell 29:618–637. https://doi.org/10.1105/tpc.16.00891
48. Jain S, Kaur M, Kumar K et al (2026) Marker-assisted introgression of superior haplotypes of Phosphorus Starvation Tolerance 1 (PSTOL1) gene from *Oryza rufipogon* imparts enhanced phosphorus uptake efficiency. Genet Resour Crop Evol 73:181. https://doi.org/10.1007/s10722-026-02807-7
49. Singh AK, Singh VK, Singh A et al (2015) Introgression of multiple disease resistance into a maintainer of Basmati rice CMS line by marker assisted backcross breeding. Euphytica 203:97–107. https://doi.org/10.1007/s10681-014-1267-1
50. Hossain MK, Islam MR, Sundaram RM et al (2023) Introgression of the QTL qSB11-1TT conferring sheath blight resistance in rice (Oryza sativa) into an elite variety, UKMRC 2, and evaluation of its backcross-derived plants. Front Plant Sci 13:981345. https://doi.org/10.3389/fpls.2022.981345

Figures

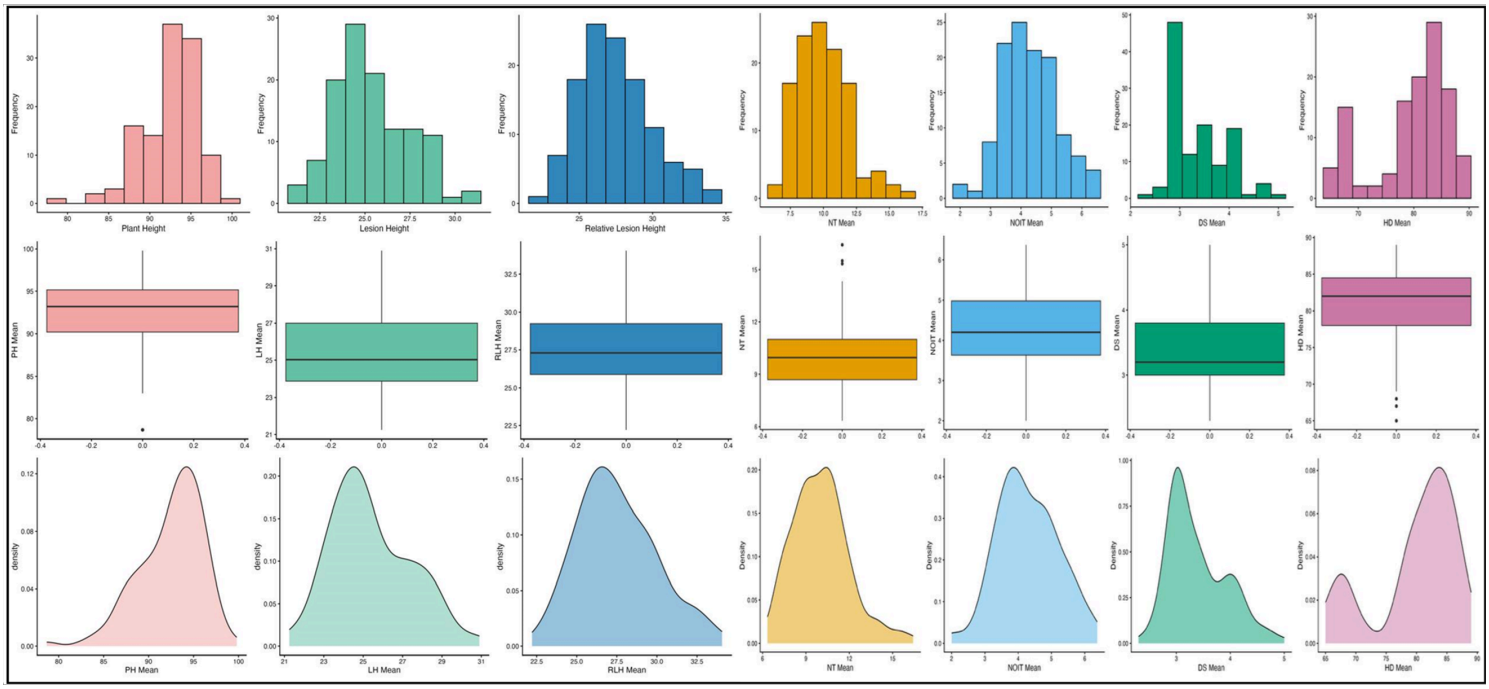

Figure 1

Frequency distribution of seven sheath blight disease resistance contributing traits, based on the pooled mean values of 118 BC_2F_5 and BC_2F_6 progenies.

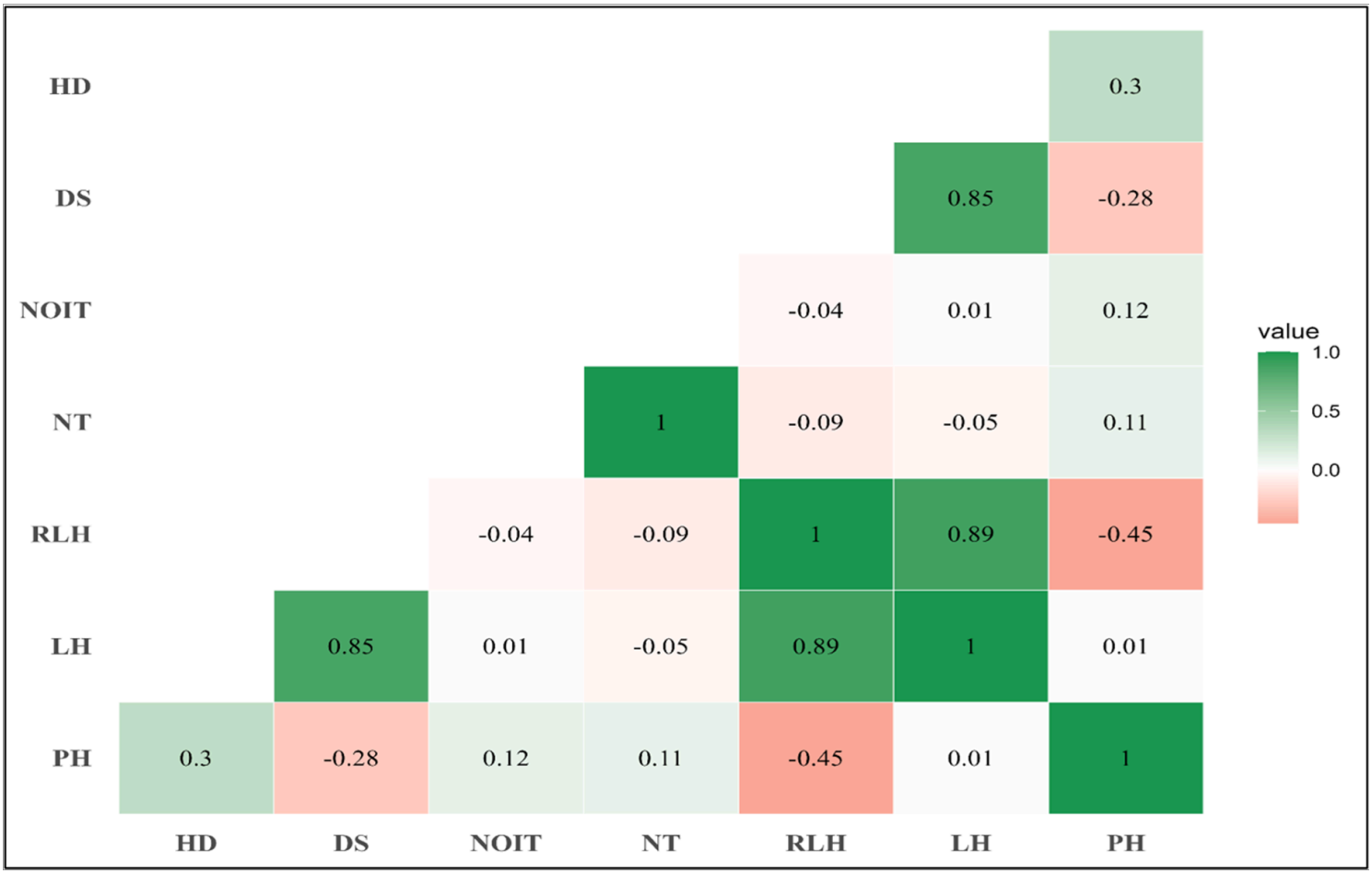

Figure 2

Heat map showing Pearson’s correlation coefficients between plant height (PH), lesion height (LH), relative lesion height (RLH %), number of tillers (NT), number of infected tillers (NIT), heading date (HD) and disease score (DS).

Supplementary Files

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