

## Mapping of quantitative trait loci for sheath blight resistance in rice

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Received : 18 May 2018

Accepted : 08 June 2018

Published : 27 June 2018

### ABSTRACT

Sheath blight is a fungal disease caused by *Rhizoctonia solani* Kühn, major diseases of rice and severely impairs both yields and quality. Quantitative trait loci (QTL) analysis of the sheath blight resistant using SSR markers was conducted in RIL population derived from the cross Danteshwari and Dagad deshi. Sheath blight resistant in this population and parents was screened by inoculation of fungus *Rhizoctonia solani* isolate in field condition. The sheath blight disease index of RIL was continuously distributed, as expected for a quantitative trait. On the basis of disease evaluations, quantitative trait loci (QTL) were identified for sheath blight resistance on chromosomes 1, 2, 3, 4, 5, 6, 8, 9, 11 and 12. The QTL, (qSBR1-1) for individual lesion length (ILL) mapped on chromosome 1. Two other QTLs, (qSBR1-1) and (qSBR1-2) for individual lesion width (ILW) were mapped to closely linked markers RM5 and RM84, respectively. The major QTL, (qSBR4-1) for total lesion length (TLL), Total lesion area (TLA) and (qSBR5-1, qSBR5-2) for total lesion area (TLA) were mapped on chromosome 4 and 5, respectively. Similarly, QTL (qSBR9-1) was mapped for individual lesion area (ILA) on chromosome 9 and (qSBR12-1) for individual lesion width (ILW) on chromosome 12, respectively. There was many minor effect QTLs also identified on various chromosome. These major QTLs will be good candidates for marker-assisted selection programme and fine mapping could lead to identification of novel resistance alleles.

**Key words:** Rice, sheath blight disease, QTL analysis, RIL population, SSR marker

### INTRODUCTION

Sheath blight caused by the fungus *Rhizoctonia solani* Kuhn, is one of the major foliar diseases of rice worldwide that severely impairs both grain yield and quality (Ou, 1985; Savary et al., 2006). It is becoming a major constraint to rice production, especially in the intensified cultivation system (Jayaprakashvel and Mathivanan, 2012). The disease was first reported in Japan in 1910 and subsequently reported to be widespread (Rush and Lee, 1992). The sheath blight infected leaves senesce or dry out and die more rapidly. Symptoms are oval or ellipsoidal greenish gray lesions, usually 1-3 cm long, on the leaf sheath, initially just above the soil or water level in the case of conventionally flooded rice. Under favorable conditions, these initial lesions multiply and expand to the upper part of the sheaths, leaves and then spread to

neighboring tillers belonging to different hills (transplanted rice) or plants (direct-seeded rice). Lesions on the leaves usually have irregular, often with gray-white centers and brown margin as they grow older (IRRI, 2017). Sheath blight is considered to be an important disease next to rice blast (IRRI, 2017). Furthermore, annual losses due to Sheath Blight (ShB) are estimated to be 10% in India and 20% in Thailand (Boukaew and Prasertsan, 2014). Loss in yield of rice may vary between 7-50% depending on the cultivar, environmental condition, stages at which the plants are infected and level of infection (Rice Knowledge Management Portal, DRR). The yield losses of 5-10% have been estimated for tropical lowland rice in Asia (Savary et al., 2000). In Japan, the disease has caused a yield loss of as high as 20% and affected about 120,000-190,000 hectares. A yield loss of 25% was reported if the flag leaves are infected. In the United

States, a yield loss of 50% was reported when susceptible cultivars were planted and also caused a yield loss of 6% in tropical Asia (IRRI, 2017). Breeding for sheath blight resistance has been difficult, mainly because of the lack of identified resistant donors in cultivated varieties (Bonmann et al., 1992). Rice genetic resources have not been comprehensively exploited for improvement of sheath blight resistance, although many cultivars and lines have been reported as promising sources of resistance (Srinivasachary et al., 2011). The identification of genes that affect complexly inherited trait is often difficult and is best approached through developing a genetic linkage map to identify quantitative trait loci (QTLs) (Tanksley and McCouch, 1997). More than 70 QTLs for sheath blight resistance have been reported in previous study (Fu et al., 2011).

The quantitative trait loci (QTLs) contributing to sheath blight resistance identified on all 12 rice chromosomes (Li et al., 1995; Pan et al., 1999; Zou et al., 2000; Kunihiro et al., 2002; Han et al., 2002; Che et al., 2003; Sato et al., 2004; Tan et al., 2005; Pinson et al., 2005; Xiang et al., 2007; Xie et al., 2008; Sharma et al., 2009; Liu et al., 2009; Li et al., 2009; Channamallikarjuna et al., 2010; Fu et al., 2011; Xu et al., 2011; Wang et al., 2012; Nelson et al., 2012; Eizenga et al., 2013; Liu et al., 2013; Taguchi-Shiobara et al., 2013; Liu et al., 2014; Wen et al., 2015; Gaihre et al., 2015; Yadav et al., 2015). The QTLs for sheath blight resistance have been often detected in the same region of chromosome of plant height (Zou et al., 2000; Pinson et al., 2005; Sharma et al., 2009). The QTL mapping can help for identification of genes responsible for resistance to sheath blight by fine mapping or gene cloning. Once, the tightly linked markers have been identified, the quantitative trait loci can be selected for breeding programs using marker-assisted selection (MAS) strategy. So, the objective of present study was to identify QTLs related to sheath blight resistance and its relation with plant height in rice.

## MATERIALS AND METHODS

### Plant materials and isolate

The mapping population of 122 F<sub>14</sub> RILs derived from a cross between cultivars Danteshwari (dwarf, sheath blight susceptible) and Dagad deshi (tall, sheath blight resistant) was used to analyze sheath blight resistance. The characteristic features of parents given in Table 1.

The local isolate of *Rhizoctonia solani* was isolated from ground soil of district Raipur, Chhattisgarh and used for screening. The fungus was maintained on oat meal agar medium for the production of sclerotia. The pure culture of *Rhizoctonia solani* isolate was maintained in petri dishes on potato dextrose agar medium and transfer in rice bran for mass multiplication.

### Evaluation of sheath blight resistance

The trial was conducted during wet season 2013 in Randomized Complete Block Design (RCBD) with three replications with each genotype having 2 rows of 1.5m length at research cum instructional farm of IGKV, Raipur, Chhattisgarh, India (21° 16' N and 81° 36' E at altitude of 289.6 meter above sea level). The RILs screened by inoculation of fungus *Rhizoctonia solani* isolate at first elongation stage (30 days after transplanting) during month of September, 2013. The observations of disease lesion were recorded by measuring lesion size in centimetre (cm) after 10th day of inoculation from randomly selected six plants and affected tillers per plant of each RIL. The greyish-green lesions enlarge and coalesce with other lesions, mostly on lower leaf sheath. The disease lesion length and width were measured with the scale from one end to another end covering whole infected region of the sheath tissue. The length and width of the biggest lesion were also taken for analysis (Channamallikarjuna et al., 2010).

### SSR analysis

The genomic DNA isolated from leaves of young succulent single plant of parents (Danteshwari & Dagad deshi) and 122 RIL population using MiniPrep method (Doyle and Doyle, 1987). The detail of DNA isolation method used as around 0.1g of leaf sample was grinded in a 2 ml eppendorf tube containing 0.4 ml of extraction buffer using MoBIO tissue lyzer. Then 0.4 ml of chloroform-isoamyl alcohol (24:1) mixture was added. Mixed well by vortexing. Centrifuged at 13000 rpm for 30 sec. Supernatant was collected and transferred to a new eppendorf tube. Then 0.8 ml of absolute ethanol was added and mixed properly by tube inversion. Centrifugation was done at 13000 rpm for 2 min. Supernatant was discarded and pellets were washed with 70% ethanol. Dried the pellets for 15-20 minutes.

**Table 1.** The characteristic features of parents.

S. No.	Parent	Pedigree	Salient features
1.	Danteshwari	Shamridhi × IR 8608-298	High yielding, dwarf, early and high tillering, resistant to gall midge, Early maturity of 105 days, Long slender grain
2.	Dagad deshi	Land race	Strong culm, tall, shy tillering, broad leaves, bold seeded, early maturity of 100 days

Pellets were dissolved in 50-100 µl (based on the size of pellet) TE buffer. The optimized PCR protocol was used for identifying the informative SSR markers on the basis of parental polymorphism. Polymerase chain reaction (PCR) amplification for SSR was performed in a total volume of 20 µl and the reaction mixture contained 10 X Assay buffer, 1 mM dNTP mix, 5 pM forward and reverse primers, 40 ng of template DNA and 1 unit Taq polymerase in 96 well veriti Applied Biosystems thermal cycler, USA. After an initial denaturation step of 95°C for 5 min, the amplification was carried out for 34 cycles comprising 1 min each of 94°C (denaturation), 55°C (annealing) and 72°C (extension). The final elongation step was extended to 7 min at 72°C followed by 4°C. After the PCR reaction was completed, 5 µl of 6 X loading dye was added to PCR amplicons and 7 µl (PCR product with dye) was loaded on 5 % PAGE in a vertical electrophoresis system (CBS scientific, model MGV-202-33, USA) with 180V for 1.5 hours. DNA fragments were then stained with ethidium bromide and visualized with a UV transilluminator Bio-rad XR+ manufactured from USA.

### Construction of linkage map and QTL mapping

The polymorphism survey was conducted between the parents Danteshwari and Dagad deshi by using 830 SSR markers randomly distributed on all 12 rice chromosomes. A total of 162 well distributed polymorphic SSR (RM and HvSSR) (McCouch et al., 2002; Singh et al., 2010) markers were used to construct a linkage map. The genotypic data was prepared for each line based on the banding patterns. All of 162 clearly polymorphic markers were used in segregation analysis of the 122 RILs. The linkage map was constructed using MapMaker/exp ver. 3.0 program (Lander et al., 1987). All pairs of linked markers were identified using the "group" command with an LOD value of 3.0. The marker order was determined using the "orders" and the "compare" commands and verified using the "ripple" command. The frequency of recombination between two markers was converted to

genetic distance using Kosambi map function (Kosambi, 1944). The genotypic and phenotypic data were further used in QTL mapping. The composite interval mapping (CIM) was performed by QTL cartographer (version 2.5) (Wang et al., 2007). The threshold log likelihood ratio (LOD) score was estimated empirically with 1000 permutations at 0.05 significant levels (Gaihre et al., 2015). The presence of putative QTLs declared if the LOD threshold was more than 3 for the traits. The proportion of phenotypic variation explained by each QTL was calculated on the basis of R<sup>2</sup> value.

## RESULTS AND DISCUSSION

### Distribution of sheath blight resistance in RIL population

The RILs population and parents were screened for sheath blight resistance. The RILs population exhibited significant phenotypic variance to support QTL mapping. The resistance segregation in this experiment varied continuously. The sheath blight disease index of RILs was continuously distributed, as expected for a quantitative trait (Fig. 1). The parents Danteshwari and Dagad deshi showed significant differences in their resistance level in the experiment. The average individual disease lesion area was obtained 1.62 cm<sup>2</sup> for Danteshwari and 1.05 cm<sup>2</sup> for Dagad deshi. Similarly the average Individual lesion length and Individual lesion width obtained 2.54, 0.64 cm for Danteshwari and 2.23, 0.47 cm for Dagad deshi, respectively. The parent Dagad deshi showed resistance for sheath blight and Danteshwari was highly susceptible under field condition. Some RILs displayed higher resistance compared to Dagad deshi and some lines were more susceptible than Danteshwari. The Danteshwari × Dagad deshi derived mapping population provides a good basis to study and to analyze genetically complex and polygenic forms of disease resistance known as "Quantitative trait loci" (QTL) for sheath blight in rice.

**Molecular linkage map**

There were 830 SSR markers screened for parent polymorphism on all 12 rice chromosomes but 162 (19.52%) found polymorphic. All of 162 SSR (RM and

HvSSR) markers were used to construct linkage map. The map spanned approximately 3972.8 cM of the genome, with an average marker interval of 24.52 cM. The number of markers per chromosome ranged from 8 (chromosome 10) to 23 (chromosome 1), with an

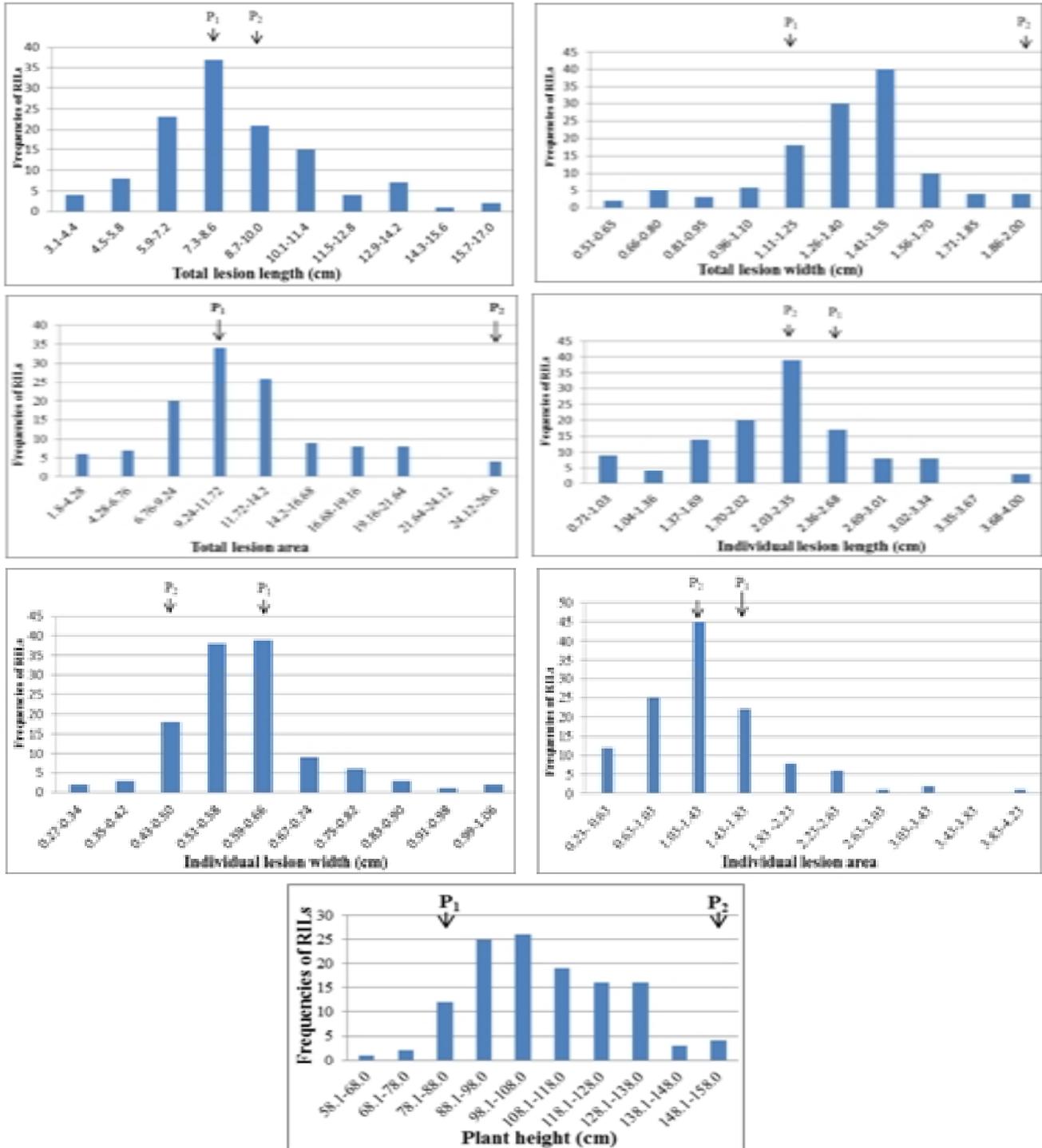


Fig. 1. Frequency distribution of RILs for sheath blight resistance and plant height.

average of 13.5 markers per chromosome. The chromosomes 1 and 5 found the longest linkage groups,

**Table 2.** QTL underlying sheath blight resistance mapped by QTL Cartographer 2.5

Trait	QTL	Chr.	Closely linked marker	Marker position (cM)	LOD	Additive effect	R <sup>2</sup> %
Total lesion length (TLL)	<i>qSBR4-1</i>	4	RM273	88.6	3.4072	-0.979	11.68
Total lesion area (TLA)	<i>qSBR4-1</i>	4	HvSSR4-35	86.2	3.0171	-1.9889	13.72
	<i>qSBR5-1</i>	5	HvSSR5-52	603.7	3.1464	1.0289	4.92
	<i>qSBR5-2</i>	5	RM188	625.7	3.1474	1.0286	5.00
Individual lesion length (ILL)	<i>qSBR1-1</i>	1	RM428	11.5	3.8432	0.1038	4.85
	<i>qSBR1-1</i>	1	RM5	232.7	15.047	-0.0293	5.53
Individual lesion width (ILW)	<i>qSBR1-2</i>	1	RM84	266.8	15.1095	-0.0385	5.18
	<i>qSBR2-1</i>	2	RM485	65.5	14.9972	-0.026	2.42
	<i>qSBR2-2</i>	2	HvSSR2-12	101.4	14.7277	-0.014	0.13
	<i>qSBR2-3</i>	2	RM174	133.3	13.2501	0.0089	0.40
	<i>qSBR2-4</i>	2	RM492	142.9	13.8959	-0.0007	0.07
	<i>qSBR3-1</i>	3	RM517	215.7	17.3122	-0.0083	0.43
	<i>qSBR3-2</i>	3	RM232	229.7	17.3098	-0.0083	0.42
	<i>qSBR3-3</i>	3	RM7	439.7	14.8935	0.0214	0.02
	<i>qSBR3-4</i>	3	HvSSR3-56	695.2	14.3093	-0.0127	0.02
	<i>qSBR3-5</i>	3	HvSSR3-85	705.2	13.4936	-0.0063	0.00
	<i>qSBR3-6</i>	3	RM55	712.6	13.8352	-0.0083	0.01
	<i>qSBR4-1</i>	4	RM307	8	14.754	-0.0055	0.51
	<i>qSBR5-1</i>	5	HvSSR5-23	19	15.6682	-0.0229	3.91
	<i>qSBR5-2</i>	5	HvSSR5-39	49.8	15.1734	-0.0272	3.35
	<i>qSBR5-3</i>	5	HvSSR5-39	141.8	14.7266	0.0097	0.18
	<i>qSBR5-4</i>	5	HvSSR5-52	434.7	17.6583	-0.0497	5.60
	<i>qSBR5-5</i>	5	RM188	766.7	14.7608	-0.0205	2.60
	<i>qSBR5-6</i>	5	RM274	840	14.0242	0.0041	0.89
	<i>qSBR5-7</i>	5	RM26	851.4	14.4522	0.0045	0.95
	<i>qSBR6-1</i>	6	HvSSR6-35	41.3	14.321	0.0082	0.24
	<i>qSBR8-1</i>	8	RM433	122.6	13.611	0.0065	0.70
	<i>qSBR8-2</i>	8	RM230	132.8	17.5287	0.0043	0.01
	<i>qSBR9-1</i>	9	RM444	10.8	15.6494	-0.0276	2.08
	<i>qSBR9-2</i>	9	RM296	46.5	15.8382	-0.0121	0.45
	<i>qSBR11-1</i>	11	HvSSR11-1	30	15.1529	-0.0528	0.01
	<i>qSBR12-1</i>	12	RM20	21	15.2806	0.04	4.09
	<i>qSBR12-2</i>	12	RM20	187	17.4974	-0.02	0.90
	<i>qSBR12-3</i>	12	RM511	193	17.9606	-0.0157	0.37
	<i>qSBR12-4</i>	12	HvSSR12-36	256.2	17.722	-0.0158	0.15
	<i>qSBR12-5</i>	12	RM277	266.1	17.7249	-0.0183	0.82
<i>qSBR12-6</i>	12	RM270	304.8	15.0655	-0.0128	1.86	
Individual lesion area (ILA)	<i>qSBR2-1</i>	2	RM492	137.9	3.9609	0.0164	0.01
	<i>qSBR3-1</i>	3	RM517	215.7	4.6655	-0.0706	1.32
	<i>qSBR3-2</i>	3	RM232	229.7	4.6619	-0.0706	1.32
	<i>qSBR3-3</i>	3	HvSSR3-56	694.2	3.4982	-0.1169	1.81
	<i>qSBR4-1</i>	4	HvSSR4-35	83.2	3.258	-0.1101	4.31
	<i>qSBR4-2</i>	4	RM273	91.6	3.7266	-0.1213	4.62
	<i>qSBR5-1</i>	5	HvSSR5-52	432.7	4.8309	-0.1405	7.62
	<i>qSBR8-1</i>	8	RM230	132.8	4.6803	-0.0007	0.05
	<i>qSBR9-1</i>	9	RM444	16.8	3.0496	-0.1621	7.89
	<i>qSBR12-1</i>	12	RM20	187	5.3081	-0.0998	1.84
	<i>qSBR12-2</i>	12	RM511	193	5.2966	-0.0951	1.41
	<i>qSBR12-3</i>	12	HvSSR12-36	257.2	5.0166	-0.0926	1.06
<i>qSBR12-4</i>	12	RM277	266.1	5.1945	-0.1271	2.76	
Plant height (PH)	<i>qPH1.1</i>	1	RM3825	476.5	11.7951	-13.2941	49.11
	<i>qPH1.2</i>	1	HvSSR1-87	484.4	10.7775	-14.5098	46.29

whereas chromosomes 10 and 7 were among the shortest. Assignment of linkage groups to the respective chromosomes was based on genetic maps developed by (McCouch et al., 2002), Gramene Annotated Nipponbare Sequence map (<http://www.gramene.org>) and Rice Genome Research Project (<http://rgp.dna.affrc.go.jp/>). The molecular linkage map is shown in Fig. 2.

### **QTL analysis for sheath blight resistance**

The QTLs for sheath blight resistance were designated as *qSBR*. The genotypic data and phenotypic data of field condition for sheath blight disease resistance was analysed by QTL cartographer 2.5. In the present investigation, we found many QTLs for sheath blight resistance. A total of 50 QTLs were identified for sheath blight disease resistance. The QTLs were found to be present on chromosomes 1, 2, 3, 4, 5, 6, 8, 9, 11 and 12. The QTLs along with their LOD score and R<sup>2</sup> value worked out through composite interval mapping given in Table 2 and Figure 2. There was several minor effect QTLs also identified on various chromosome but only high phenotypic variance QTLs considered for explanation. Their resistant alleles were derived from both the parents.

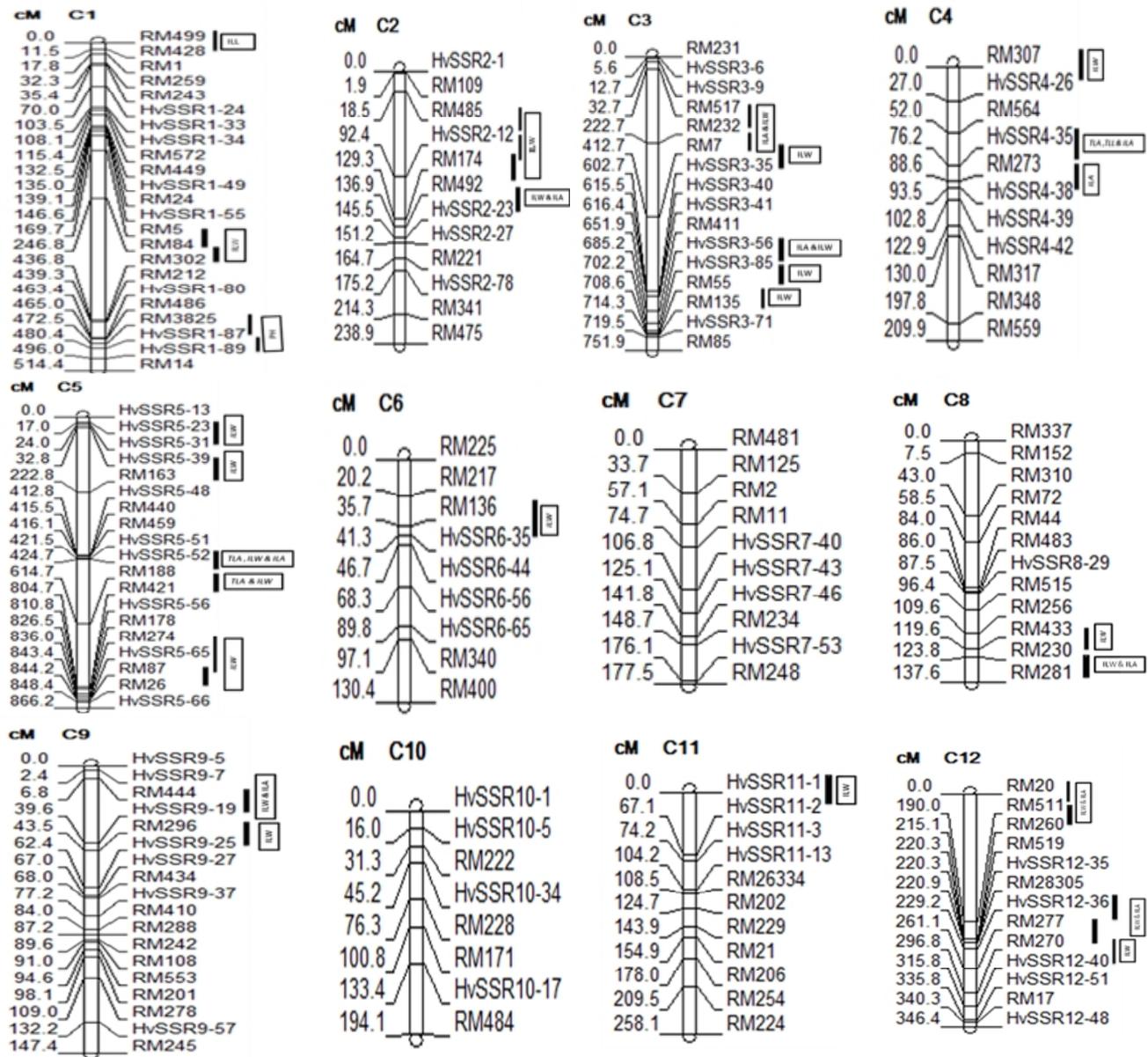
The QTL, *qSBR1-1* for Individual lesion length (ILL) mapped linked to marker RM428 on chromosome 1. Two QTLs, *qSBR1-1* and *qSBR1-2* for Individual lesion width (ILW) were mapped to closely linked markers RM5 and RM84 with high LOD values 15.047 and 15.1095, respectively. The QTLs for total lesion length (TLL) were located on chromosome 4. The major QTL, *qSBR4-1* was identified for total lesion length (TLL) between markers RM273 and HvSSR4-35 with LOD score of 3.4072, explained 11.68% of the phenotypic variation. The QTL showed negative additive effect means alleles from the susceptible parent Danteshwari. Another QTL, *qSBR4-1* for trait total lesion area (TLA) associated with sheath blight resistance also mapped on chromosome 4 in the same marker interval with LOD score 3.0171 under heavy infection condition. The percentage of phenotypic variance explained by this QTL was 13.72% and showed negative additive effect means that the allele from the susceptible parent Danteshwari. Both the QTLs also mapped for Individual lesion area (ILA) at the same marker interval.

The QTLs for total lesion area (TLA) were located on chromosome 5. The *qSBR5-1* and *qSBR5-2* were mapped for total lesion area (TLA) closely linked to marker HvSSR5-52 and RM188, respectively. The QTLs explained 4.92 and 5.0% of phenotypic variance, respectively. Both the QTLs showed positive additive effect, means allele carried from resistant parent Dagad deshi, acted to increase disease resistance. Both the QTLs also mapped for Individual lesion width (ILW) at the same markers interval but with negative additive effect. The *qSBR5-1* also mapped for Individual lesion area (ILA) between markers interval of HvSSR5-52 and RM188. The QTL, *qSBR9-1* was mapped for Individual lesion area (ILA) linked to marker RM444 on chromosome 9. The QTL showed negative additive effect with 7.89% of phenotypic variance. The same QTL also mapped for Individual lesion width (ILW) with lower phenotypic variance with same linked marker. Similarly many QTLs identified for sheath blight on chromosome 12 but most of them were with low phenotypic effect. The *qSBR12-1* was identified closely linked to marker RM20 for individual lesion width (ILW) and allele carried from resistant parent Dagad deshi. The other QTLs identified for Individual lesion width (ILW) on chromosome 12 also mapped as same position as of Individual lesion area (ILA).

### **QTL analysis for trait plant height**

The parent Dagad deshi is tall and Danteshwari is dwarf. Two QTLs for plant height mapped using same RILs population. The significant major QTLs, *qPHI.1* and *qPHI.2* mapped to closely linked marker RM3825 and HvSSR1-87, respectively for plant height on chromosomes 1. The percentage of phenotypic variance explained by these QTLs was 49.11 and 46.29%, respectively. The QTLs along with LOD score and R<sup>2</sup> value worked out through composite interval mapping also given in Table 2.

There was no gene identified rice researchers till date conferring true resistance to sheath blight (Li et al., 1995). However, few resistant varieties and lines such as Tetep, Jasmin 85, Teqing and Minghui 63 offer sufficient partial resistant to pathogen in field condition to be agriculturally useful (Pan et al., 1999; Zou et al., 2000; Kunihiro et al., 2002). The genetic nature of sheath blight has been found to be complex and



**Fig. 2.** The linkage map depicting location of QTLs for Sheath blight resistance and Plant height traits {Total lesion length (TLL), Total lesion area (TLA), Individual lesion length (ILL), Individual lesion width (ILW), Individual lesion area (ILA), Plant height (PH)}

controversial issue in the earlier studies (Loan et al., 2004). On the contrary, genetics studies on the quantitative resistance to *R. solani* in rice have shown both polygenes and major gene inheritance (Li et al., 1995; Zou et al., 2000). In the present study, the disease index of the RILs population for sheath blight response found continuously distributed as expected for a quantitative trait. Thus, QTLs might be involved in resistance to sheath blight. Similarly, the sheath blight response of 127 RIL population derived by single seed

descent method from a cross between HP2216 (susceptible to *R. solani*) and Tetep (having a high degree of resistance to *R. solani*) also reported continuously distributed (Channamallikarjuna et al., 2010). The mean sheath blight severity on a subset of 256 F<sub>5</sub> RILs from Lemont × Jasmine 85 (LJ RILs) in the micro chamber and mist-chamber assays were distributed normally, with the resistant and susceptible parents at the extreme ends (Liu et al., 2009). The frequency distributions of sheath blight response ratings

of 300 recombinant inbred lines (RILs) from the cross Lemont  $\times$  Teqing in a 2-year replicated field experiment exhibited continuous variation for SBR with skewing toward resistance both years (Pinson et al., 2005). In the other study, the  $F_{2,3}$  progeny population exhibited significant phenotypic variance for sheath blight disease scores were continuously distributed, as expected for a quantitative trait (Sharma et al., 2009). The frequency distributions of lesion height (LH), actual lesion length (ALL) and disease ratings by inoculation with *Rhizoctonia solani* were continuous, typical of quantitative traits from 266 Teqing near isogenic introgression lines (NILs) were developed by using Teqing as recurrent parent and Lemont as introgression parent (Loan et al., 2004). The disease ratings in the  $F_2$  clonal population were continuously distributed from total of 128  $F_2$  clonal families and their parents were used for genetic analysis of disease resistance (Zou et al., 2000).

### Comparisons of QTLs with previous studies

In the present study, RILs were used for mapping sheath blight resistance using phenotypic data from inoculated rice plant grown in field condition. The sheath blight resistant QTLs were identified in other studies using phenotypes collected from rice plants at an early vegetative stage under controlled greenhouse conditions (Eizenga et al., 2002; Jia et al., 2007; Liu et al., 2009; Yadav et al., 2015) and field conditions (Li et al., 1995; Zou et al., 2000; Sato et al., 2004; Pinson et al., 2005; Sharma et al., 2009; Channamallikarjuna et al., 2010; Gaihre et al., 2015). The linkage map showed QTLs position on chromosomes in this study is depicted in Fig. 2.

In the present study, many QTLs mapped for sheath blight resistance but with high phenotypic variance considered for explanation. The QTL, (*qSBR1-1*) for Individual lesion length (ILL) and (*qSBR1-1*, *qSBR1-2*) for Individual lesion width (ILW) were mapped on chromosome 1 associated with sheath blight resistance are important QTLs. The presence of QTLs on this chromosome was also reported by different researchers (Pinson et al., 2005; Sharma et al., 2009; Li et al., 2009; Liu et al., 2009; Channamallikarjuna et al., 2010; Gaihre et al., 2015; Yadav et al., 2015). Previously, Pinson et al. (2005) was identified qSB-1 on chromosome 1 with LOD value of 3.80 and 8.0%

of PVE and found associated with morphological character heading date. Similarly, the QTLs such as *qDR-1a*, *qLL-1a*, *qLH-1b*, *qLH-1d*, *qRLL-1b*, *qRLH-1a* and *qRLH-1b* also mapped on chromosome 1 (Liu et al., 2009). Channamallikarjuna et al. (2010) also identified *qSBR1-1* on chromosome 1 a peak marker HvSSR1-68 with LOD value 2.9-3 and 8.1-15.0% of PVE. A QTL, *qSBR1* was mapped on chromosome 1 (Gaihre et al., 2015). A QTL, *qShB1* was also mapped between marker RM1361-RM104 on chromosome 1 for year 2008 and 2009 (Eizenga et al., 2013). Yadav et al. (2015) was also identified qshb1.1 close to marker RM151 on chromosome 1. All these study, confirmed existence of identified QTLs and share the same chromosome 1 but different in position.

In this study, we identified major QTLs, *qSBR4-1* for total lesion length (TLL) and total lesion area (TLA) on chromosome 4 for sheath blight resistance with respect to different position as earlier reported QTLs. Li et al. (1995) reported a QTL, qSB-4 on chromosome 4 with a peak marker locus RG14-RG214 with 2.8 and 5% PVE. Similarly two QTLs, *qSB-4-1* and *qSB-4-2* reported on chromosome 4 with LOD value 3 with 5% PVE and 4.6 with 7% PVE, respectively (Pinson et al., 2005). Xie et al. (2008) also reported a QTL, *Qsh4* on this chromosome. The *qDR-4*, *qRLL-4* and *qRLH-4* were found to in the interval between RM1155 and RM5757 (Liu et al., 2014). A QTL, *qSBR4* was identified close to marker RM3276 on chromosome 4 (Gaihre et al., 2015). All these QTLs share different locations of the same chromosome 4. Similarly, QTLs, (*qSBR5-1* and *qSBR5-2*) were mapped for Total lesion area (TLA) on chromosome 5 and same also mapped for traits Individual lesion width (ILW) and Individual lesion area (ILA). Previously, many QTLs identified on this chromosome for sheath blight resistance with different positions by other researchers (Han et al., 2002; Pinson et al., 2005; Li et al., 2009; Liu et al., 2014; Eizenga et al., 2013). The *qSB-5* was mapped to marker intervals C624-C246 (Han et al., 2002), *QDs5* and *QRh5* close to marker RM161 (Li et al., 2009) and *qSB-5* close to marker Y1049 (Pinson et al., 2005). The *qShB5-mc* was identified on chromosome 5 between marker RM122-RM5796 for sheath blight resistance in microchamber in the greenhouse study of wild population 1 (Eizenga et al., 2013). Liu et al. (2014) also identified a QTL,

*qDR-5* on chromosome 5 between marker intervals RM1248-RM1182 with LOD value 5.26.

The QTL, *qSBR9-1* was mapped for Individual lesion area (ILA) for sheath blight resistance on chromosome 9, in this study. Many QTLs identified by other researchers share the same chromosome (Li et al., 1995; Han et al., 2002; Tan et al., 2005; Pinson et al., 2005; Liu et al., 2009; Channamallikarjuna et al., 2010; Liu et al., 2014; Yadav et al., 2015). The QTL, *Qsbr9a* was mapped (Li et al., 1995), *qSB-9-1* and *qSB-9-2* (Zou et al., 2000), *qSB-9* (Han et al., 2002), *qSB-9* (Pinson et al., 2005), *qSBR9-1* (Channamallikarjuna et al., 2010), *qLL-9* (Liu et al., 2014), *qshb9.1*, *qshb9.2* and *qshb9.3* on chromosome 9 (Yadav et al., 2015). The QTL mapped different in position and specific to this analysis. The *qSBR12-1* was mapped for Individual lesion width (ILW) and allele carried from resistant parent Dagad deshi. Previously, many QTLs identified on this chromosome for sheath blight resistance with different positions (Li et al., 1995; Sato et al., 2004; Pinson et al., 2005; Li et al., 2009; Nelson et al., 2011; Eizenga et al., 2013; Liu et al., 2014). The *Qsbr12a* was mapped in marker interval of RG214a-RZ397 (Li et al., 1995), *qSB-12* was linked to marker RM1880 (Sato et al., 2004), QRh12 linked to RM235 (Li et al., 2009), *qSB-12* (Pinson et al., 2005), QTL for sheath blight at marker interval RM3-RM2 (Nelson et al., 2011), *qShB12-mc* at marker interval RM5746-RM277 (Eizenga et al., 2013).

### Relationship between QTLs for sheath blight resistance and plant height

There were many investigations been conducted to know the nature of QTLs for correlated traits. In order to explain the true relationship between sheath blight resistance and other agronomic trait, we also mapped the QTLs for plant height using same population. By comparing, the location of different QTLs on chromosomes, it was found that QTLs for plant height did not co-localize with QTLs for sheath blight resistance. It is indicating no tight linkages between the genes controlling morpho-developmental and sheath blight resistance traits. Generally, partial resistance of sheath blight influence by morphological traits (Li et al., 1995; Zou et al., 2000; Kunihiro et al., 2002; Sato et al., 2004; Sharma et al., 2009; Srinivasachary et al., 2011). In similar study, Teqing/Lemont F<sub>4</sub> population,

Li et al. (1995) reported that a large proportion of the phenotypic variation in sheath blight resistance was explained by the morpho-developmental traits (mainly heading date, 42% and plant height, 4%). Similarly, plant height and heading date explained 43% of the sheath blight reaction in a mapping population derived from Pecos, a tropical japonica reported to be sheath blight resistance (Sharma et al., 2009). A total of 33 QTL associated with sheath blight resistance located on all 12 rice chromosomes have been reported, with ten of these colocalizing with QTL for morphological attributes, especially plant height, or for heading date (Srinivasachary et al., 2011).

### ACKNOWLEDGEMENT

We thank Department of Plant Molecular Biology and Biotechnology, Indira Gandhi Krishi Vishwavidyalaya, Raipur for providing the necessary facilities.

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