Current Status of Rice Breeding for Sheath Blight Resistance

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Introduction

Rice is one of the three major food crops in the world. It is the staple food for most of the people of South-East Asia. Rice productivity fluctuates significantly from region to region; season to season due to biotic and abiotic stress. Sheath blight is one of the major biotic constraints in rice cultivation. It is caused by Rhizoctonia solani Kuhn. This disease can cause yield reduction between 20-50% depending on the severity of infection. Several genotypes reported for sheath blight resistance but none of the genotypes were found with absolute resistance. Sheath blight resistance is controlled by polygenes or quantitative trait loci (QTLs) each with small effect. Pyramiding of such QTLs is expected to increase resistance to sheath blight in the cultivars. Genetic engineering of crops with plant pathogenesis-related (PR) genes may give a promising and long-lasting solution for sheath blight disease management.

Keywords
Rice, Sheath blight, Rhizoctonia solani, Resistance, QTLs

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rice-producing areas. It is second in importance next to rice blast in reducing both grain yield and quality (Webster and Gunnell, 1992).

**Sheath blight disease**

Sheath blight disease caused by *Rhizoctonia solani Kuhn*. It survives either as sclerotia or mycelia in host plants’ debris. Sclerotia can survive for 2 years in soil and spread during field preparation and flooding the field for irrigation (Webster and Gunnell, 1992; Brooks, 2007). During the infection process, the sclerotia germinate on rice sheaths forming infection cushions or appressoria. Then pathogen colonizes the entire plant through surface hyphae, developing new infection structures (Ou, 1985). According to Hashiba et al., (1982) secondary spread of disease depends exclusively on running hyphae that progress out from the initial lesions, from the lower part of the crop canopy towards its upper part along tillers and leaves, and across adjacent plant units (individual plants or hills). This has been commonly referred to as the ‘vertical’ and ‘horizontal’ spread process. The Canopy architecture and the associated microclimate have strong effects on both the mobilization of primary inoculum and the further spread of the disease (Savary et al., 1995). Canopy architecture depends on a number of factors like the crop establishment method (Willoquet et al., 2000), fertilizer input (Cu et al., 1996; Slaton et al., 2002; Tang et al., 2007), and the morphology of the rice genotype itself. Microclimate with high temperature (28-32°C) and relative humidity (more than 90%) facilitates the spread of this disease (Kaur et al., 2015).

At early stage disease symptoms appears as circular, oblong or ellipsoid, greenish-grey water-soaked spots about 1cm long that occur on leaf sheath near the water level. Later these lesions enlarge and become oblong and irregular in outline, the center of which become grey white with brown margins. Due to the semi-saprophytic nature and uncharacterized pathogenicity mechanism of *R. solani*, it infects nearly 50 species besides rice. Earlier it was considering as minor disease of rice, but with the introduction of modern, semi dwarf nitrogen responsive cultivars it converted to major disease. Rice sheath blight can cause yield reduction between 20-50% depending on the severity of infection (Rao, 1995). In India, the estimation of losses due to this disease has been reported up to 54.3 % (Chahal et al., 2003).

**Management of sheath blight**

Sheath blight disease management is very difficult due to its wide host range. There are different control measures available for sheath blight like host resistance, cultural control, chemical control and biological control. Among all these host resistance is most valid and eco-friendly choice for almost all type of plant stress.

**Host resistance**

Several groups have attempted to identify sources of sheath blight resistance by screening local accessions, cultivars, landraces, and/or advanced breeding lines. Sources of sheath blight resistance have been sought for different rice-growing regions by different research groups. These studies resulted in the identification of genotypes with moderate to high levels of resistance. Summary of important Sheath Blight resistance sources reported so far in literature is presented in Table 1.

Although several genotypes reported for sheath blight resistance but none of the genotypes were found with absolute resistance (Lee and Rush, 1983; Chen et al.,
2000; Eizenga et al., 2002; Jia et al., 2012; Dey et al., 2016) and their disease reaction is not consistent.

**QTLs associated with sheath blight**

Resistance to rice sheath blight is a complex, quantitative trait controlled by polygenes (Sha and Zhu, 1990; Li et al. 1995; Pinson et al., 2005). First QTL linked to molecular marker RG118 identified by Li et al. (1995) using F2-3 population of Lemont/Teqing. However, few researchers (Xie et al., 1992; Pan et al., 1999) proposed that sheath blight resistance in some rice varieties is controlled by only a few major genes. Over the past two decades, several sheath blight resistance quantitative trait loci (QTL) have been mapped and few of them are discussed here. Zou et al. (2000) identified six QTLs qSB-2, qSB-3, qSB-7, qSB-9-1, qSB-9-2 and qSB-11, contributing to sheath blight resistance, located on chromosomes 2, 3, 7, 9 and 11 respectively, using F2 clonal population of Jasmine 85/Lemont. Sato et al. (2004) also identified two QTLs for sheath blight resistance (qSB-3 and qSB-12) on chromosomes 3 and 12 from the cross Hinohikari/WSS2//Hinohikari. qSB-\(g^TQ\), a major QTL derived from Teqing was reported by Zuo et al. (2008). The QTL qSBR11-1 for sheath blight resistance was identified between the marker interval RM1233 (26.45 Mb) to sbq33 (28.35 Mb) on chromosome 11 from the population RILs of HP2216/Tetep (Channamallikarjuna et al., 2010).

Xu et al. (2011) detected four QTL (qShB1, qShB2, qShB3 and qShB5) using a double haploid (DH) population of ‘Maybelle. Zhu et al. (2014) identified two major rice sheath blight resistance QTLs, qSB1-I\(^{HJX74}\) and qSB1-I\(^{HJX74}\) using chromosome segment substitution lines. Two major QTLs, qshb7.3 and qshb9.2 positioned on the chromosome 7 and 9 also identified using BC\(_1\)F\(_2\) mapping populations from the cross BPT-5204/ARC10531 (Yadav et al., 2015). But so far, identified QTLs have not been utilized in development of sheath blight resistant cultivars and their breeding value has not been assessed. The reported QTLs for sheath blight resistance in rice are depicted in Table 2.

**Sheath blight breeding strategies**

Hypothetically sheath blight resistance may have two main groups of mechanisms viz., disease escape and physiological resistance (Sattari et al., 2014). Disease escape is strongly determined by crop architecture. Morphological traits like plant height (Li et al., 1995; Peng et al., 2003 and Willocquet et al., 2010), heading date (Shiobara et al., 2013; Li et al., 1995 and Park et al., 2008) & stem thickness (Dey et al., 2016) positively correlated with sheath blight resistance. Sharma et al. (2009) reported that the short stature at sd-1 semi-dwarfing locus was strongly linked to higher sheath blight infection. Physiological resistance correlated with physiological process that is associated with a decrease in efficiency of one or several of the infection stages of the pathogen.

As we discussed earlier sheath blight resistance is governed by quantitative traits, development of sheath blight resistant rice varieties is very difficult through traditional breeding method. Pyramiding of QTLs through marker-assisted selection may results stable and potential cultivars. Chen et al. (2014) improved japonica rice resistance to sheath blight by pyramiding qSB-\(g^TQ\) and qSB-7\(^{TQ}\) on chromosomes 9 and 7 respectively. Zuo et al. (2014) reported that NILs carrying both TAC1\(^{TQ}\) and qSB-\(g^TQ\) showed more resistance than the NILs containing only one of them.

Further, there are evidences which show better disease management by pyramiding
two or more disease resistance genes/QTLs. Singh et al (2012) developed multiple disease resistance basmati rice by transferring the blast resistance gene Pi54 and sheath blight resistance quantitative trait loci (QTL) from Tetep, qSBR11-1 to ‘Improved Pusa Basmati’.

Transgenic approach

Development of transgenic rice plants may provide a novel strategy to reduce yield losses caused by sheath blight disease. Plant pathogenesis-related (PR) genes like PR-3 chitinase (Datta et al., 2000) and PR-5 (thaumatin-like protein) (Datta et al., 1999) provide resistance against sheath blight disease. Instead of single PR gene, combination of two PR genes shows more efficient for conferring a higher level of sheath blight resistance. Some example of PR combination are barley chitinase and barley b-1,3-glucanase genes (Jach et al., 1995); maize ribosome inactivating gene MOD1 and rice basic chitinase gene RCH10 (Kim et al., 2003); CHI11 and thaumatin-like protein (Kalpana et al., 2006); rice chitinase (CHI11) and tobacco b-1,3-glucanase (gluc) (Sridevi et al., 2008); rice chitinase gene (OsCHI11) and Arabidopsis NPR1 (AtNPR1) gene (Karmakar et al., 2017). ASD16 has been reported as stable transgenic line against sheath blight (Rajesh et al., 2016). Shah et al (2009) reported that transgenic rice expressing an endochitinase gene (cht42) from Trichoderma virens showed up to 62% reduction in the sheath blight disease index.

Durable and broad-spectrum resistance cultivars can be obtained by the pyramiding of transgenes. Datta et al (2002) utilized Xa21 gene (resistance to bacterial blight), the Bt fusion gene (for insect resistance) and the chitinase gene (for tolerance of sheath blight) for gene pyramiding and identified stable elite rice lines resistant to disease and insect pests. Maruthasalam et al (2007) reported that a transgenic Pusa BasmatiI line pyramided with chi11, tlp and Xa21 showed an enhanced resistance to both sheath blight and bacterial blight.

It is concluded that, rice sheath blight is second in importance next to rice blast in reducing both grain yield and quality. Germplasm with absolutely resistant to the pathogen have not been discovered till now. To reduce yield loss due to sheath blight, development of sheath blight resistant cultivars is important. However, only moderately resistant genotypes are reported.

These genotypes show variable disease reaction from one season to another season, which limit their use in breeding programme. Many QTLs for sheath blight resistance have been reported, but only few of them have been fine mapped. Validation of these QTLs is required before being used for marker-assisted breeding (MAB).

It has been observed in several cases, resistance to sheath blight is a cumulative effect of several minor QTLs. Earlier efforts were focused on improvement of sheath blight resistance in elite susceptible cultivars. Employing genotypes possessing moderate resistance to sheath blight governed by minor effect QTLs in breeding programmes will only result in distribution of such QTLs in the segregating populations. Further, this also poses difficulty in retrieving the same phenotype in mapping populations as that of resistant parent phenotype making it difficult to establish marker-trait associations. Hence, breeding strategies have to be modified in the development of sheath blight resistant cultivars. Here, we propose a two step breeding strategy to deal with difficult and complex traits like sheath blight.
Table 1: List of promising genotypes for sheath blight resistance

| (Local accessions/varieties/cultivars/land races) | Reference |
|--------------------------------------------------|-----------|
| NC 678, Dudsor, Bhasamanik                       | Das, 1970 |
| Chin-kou-tsau, Zenith, CO.17, Dinominga, Puang Nahk 16, Baok, Toma-112, R.T.S.31, Kele Kala | Wu, 1971 |
| Lalsatkara                                        | Roy, 1977 |
| ARC15762, ARC 18119, ARC 18275, ARC 18545       | Bhaktavatsalam et al., 1978. |
| IR24, IR26, IR29, Jaya, Jaganath, Meshoori, Pankaj, Rajeshwari, Supriya, Sabari, TKM6 | Rajan and Nair, 1979 |
| Nizersail, Rajasail, Tabend, Ta-poo-cho-z, Kattachambha, DA 29, ARC 5925, ARC 5943, ARC 14529, ARC 10572, ARC 10618, ARC10836 | Manian and Rao, 1979 |
| Tapoochoz, Bahagia, Laka                         | Crill et al., 1982 |
| Bharati, Rohini                                  | Gokulapulan and Nair, 1983 |
| Taraboli 1, Dholamula, Supkheru, Chidon          | Borthakur and Addy, 1988 |
| BogII, Aduthurini, Chinese galendopuram, Arkavati, Saket-4, Neela, MTU-3, MTU-7, MTU-13, MTU-3642, BPT-6 | Ansari et al., 1989 |
| Tetep, Tapoochoz, Guyanal                       | Sha and Zhu, 1990 |
| LSBR-5, LSBR-33                                  | Xie et al., 1992 |
| KK2, Dodan, IR40 and Camor                      | Singh and Dodan, 1995 |
| RU8703196, B82-761                               | Marchetti et al., 1995 and Marchetti et al., 1996 |
| Chingdar, As 93-1, Panjasali, Up-52, Upland-2, Mairan, N-22 and 1/69-70 | Sinha and Borah, 2000 |
| TIL 455, TIL 514, TIL 642                       | Pinson et al., 2008 |
| Jarjan, Nepal 555 and Nepal 8                    | Shiobara et al., 2013 |
| BPL 7-12, BML 27-1, BML 21-1 and Kajarahwa     | Dubey et al., 2014 |
| Tetep and ARC10531                               | Yadav et al., 2015 |
| SM 801, 10–3, Ngnoolasha, Wazuhophek, Gumdhan and Phougak and RP 2068-18-3-5 | Dey et al., 2016 |
Table 2: List of reported QTLs for sheath blight tolerance in rice

| Chr. no. | Locus  | Marker or marker interval Nearest | Resistant parent | Susceptible parent | Mapping population | PV (%) | Reference |
|----------|--------|----------------------------------|------------------|--------------------|-------------------|--------|-----------|
| 3        | Qsbr3a | RG348–RG944                      | Teqing           | Lemont             | F₄ Bulk           | 27.7   | Li et al., 1995 |
| 9        | Qsbr9a | RG910b–RZ777                     | Teqing           | Lemont             | F₄ Bulk           | 9.4    |           |
| 2        | qSB-2  | G243-RM29 (RM29-RG171)           | Jasmine 85       | Lemont             | 14.4 (21.2)       |        | Zou et al., 2000 |
| 3        | qSB-3  | R250-C746                        | Jasmine 85       | Lemont             |                   | 26.5   |           |
| 7        | qSB-7  | RG30-RG477                       | Jasmine 85       | Lemont             |                   | 22.2   |           |
| 9        | qSB-9-1| C397-G103                        | Jasmine 85       | Lemont             |                   | 9.8    |           |
| 9        | qSB-9-2| RG570-C356                       | Jasmine 85       | Lemont             |                   | 10.1   |           |
| 11       | qSB-11 | G44–RG118                        | Jasmine 85       | Lemont             |                   | 20.5   |           |
| 2        | qSBR-2 | RG171–G243A                      | Jingxi 17        | Zhaiyeqing 8      | DH                | 11.2   | Kunihiro et al., 2002 |
| 3        | qSBR-3 | G249-G164                        | Jingxi 17        | Zhaiyeqing 8      | DH                | 10.5   |           |
| 7        | qSBR-7 | RG511-TCT122                     | Jingxi 17        | Zhaiyeqing 8      | DH                | 15.5   |           |
| 11       | qSBR-11| CT244-CT44                       | Jingxi 17        | Zhaiyeqing 8      | DH                | 9.5    |           |
| 5        | qSB-5  | C624-C246 (C246-RM26)            | Minghui 63       | Zhenshan 97B      | RILs              | 10.5 (9.5) | Han et al., 2002 |
| 9        | qSB-9  | C472-R2638 (RM257-RM242)         | Minghui 63       | Zhenshan 97B      | RILs              | 10.1 (6.9) |           |
| 5        | Rsb1   | RFLP+SSR                         | 4011             | XZX19             | F₂                | 11.2   | Che et al., 2003 |
| 3        | qSB-3  | RM3856                           | WSS2             | Hinohikari        | BC₁F₁             | 19.4   | Sato et al., 2004 |
| 1        | qSB-1  | RG532x                           | Teqing           | Lemont             | RIL               | 8      | Pinson et al., 2005 |
| 2        | qSB-2  | C624x                            | Teqing           | Lemont             | RIL               | 7      |           |
| 3        | qSB-3-1| RG348x                           | Teqing           | Lemont             | RIL               | 18     |           |
| 3        | qSB-3-2| RZ474                            | Teqing           | Lemont             | RIL               | 10     |           |
| 4        | qSB-4-1| RG1094c                          | Teqing           | Lemont             | RIL               | 5.0    | Pinson et al., 2005 |
| 4        | qSB-4-2| RZ590x                           | Teqing           | Lemont             | RIL               | 7.0    |           |
| 5        | qSB-5  | Y1049                            | Teqing           | Lemont             | RIL               | 6.0    |           |
| qSB  | RIL  | X    | RIL  |     |     |
|-------|------|------|------|-----|-----|
| 6     | qSB-6-1 | C    | Teqing | Lemont | RIL | 5.0 |
| 6     | qSB-6-2 | RZ508 | Teqing | Lemont | RIL | 7.0 |
| 7     | qSB-7   | C285  | Teqing | Lemont | RIL | 5   |
| 9     | qSB-9   | RZ404 | Teqing | Lemont | RIL | 6.0 |
| 10    | qSB-10  | RG561 | Teqing | Lemont | RIL | 5.0 |
| 12    | qSB-12  | G1106 | Teqing | Lemont | RIL | 9.0 |
|       | -      | -    | Rsb-2(t) | -    | -   | Xiang et al., 2007 |
| 9     | qSB-9q | Indel | Teqing | Lemont | BC₁F₁ | -   | Zuo et al., 2008 |
| 9     | qShB9-2 | RM245 | Jasmine 85 | Lemont | RIL | 24.3 | Liu et al., 2009 |
| 1     | -      | -    | -     | -    | -   | -   | - |
| 9     | qSB1   | HJX74 | Nag08KK18184-Nag08KK18871 | HuaJingXian74 | chromosome substitution lines | - | Zhu et al., 2014 |
| 11    | qSB11  | HJX74 | ZY7.7-1-5 | Amol3(sona) | HuaJingXian74 | - | - |
| 7     | qshb7.3 | RM 205 | ARC10531 | BPT-5204 | BC₁F₂ | 21.76 | Yadav et al., 2015 |
| 9     | qshb9.2 | RM 336 | ARC10531 | BPT-5204 | BC₁F₂ | 19.81 | - |
Fig. 1 Symptom of Rice Sheath blight
Firstly, genotypes with moderate resistance from the identified pool should be intermated for possible accumulation of several minor effect QTLs that would be evident from enhanced phenotypic effect in populations. Secondly, breeding line with increased resistance to sheath blight than either of it parents should be crossed to an elite susceptible cultivar. Further intermating in segregating populations would ensure retention of accumulated minor effect QTLs in elite background. Modified breeding strategy proposed here coupled with application of advanced genomic tools would widen the scope of development of high yielding elite cultivars with resistance to sheath blight.

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