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Current status and intervention in sheath blight disease resistance in rice through conventional, molecular, and transgenic approaches

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ABSTRACT

Sheath blight (ShB) of rice, caused by Rhizoctonia solani Kuhn (teleomorph Thanatephorus cucumeris [Frank] Donk), is now one of a very serious diseases in rice-growing countries. We reviewed the occurrence and spread of this disease on the different hosts; however, a brief discussion was made only on rice. The taxonomy of R. solani, classification of the pathogen, and strategies for disease management are briefly described in their host. Presently sheath blight disease is controlled by synthetic chemicals, however, this may create a problem in environments. Resistance variety is best option for the control of this disease, though only little/moderate resistance has been reported in few wild cultivars and in cultivated rice genotypes namely Jasmine 85, Tatep, and Tequing etc. High level of resistance against ShB have been conveyed in the developed transgenic rice stains. Identification and molecular characterization of resistance QTLs in the promising lines of rice will be a grateful effort for the further transfer in the high yielding varieties of rice to achieve the ShB resistance in future. To successfully mitigate the impact of sheath blight on rice production, it is crucial that we understand the barriers and advancements in sheath blight pathogen, their broad host range and management strategies under field condition. The present review primarily concentrates on the effort to improve findings related to the important gene loci, their related markers and transgenic development in different rice cultivars.

Keywords: *sheath blight, wild rice, soil-borne pathogen, resistance gene, quantitative trait loci, molecular, transgenic*

INTRODUCTION

Rice (*Oryza sativa* L.), affiliate of the family Graminae, is extensively grown in tropical and subtropical region. Roughly half of the total populace devours rice as their fundamental and staple food for nourishment (www.irri.org/). Rice is affected by various biotic and abiotic stresses. The biotic stresses caused by bacteria, fungus and others microorganisms are one of the most genuine restricting variables for rice production. About 90% of the world's rice is cultivated in the Asian continents and comprises a staple nourishment for 2.7 billion individuals around the world. It is disgraceful that such a significant important crop is attacked by numerous sorts of infections, of which sheath blight (ShB) caused by *R. solani* Kuhn is one of the most dangerous disease all through the world (107). Frequency of ShB in rice fields is reliant on the strategy for planting and plant population density. Examinations at farmers' fields and experimental fields showed that square technique for transplantation brought about ideal high return thickness, higher leaf territory record and dry issue generation.

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This strategy for transplantation likewise added to expanded ShB obstruction and higher grain yields (193). Though, sparse planting brought about lower ShB incident and notable lodging resistance in rice. The other significant impacts of sparse planting included less number of stems/m^2, more stems/slope, delay in date of most high tillering stage, heading time, aging time, more prominent number of spikelets per head, and more spikelets on secondary rachis-branches (170). Planting of rice seedlings distant from the bund showed reduced ShB incidence since bunds also have weed hosts of *R. solani*. . The ShB disease may cause severe losses in localized areas and may reduce grain yield up to 30%. *Rhizoctonia* root rot in barley and wheat, produced by the soil borne fungal pathogen *R. solani* AG-8, was initially diagnosed as a delinquent in direct-seeded wheat and barley in the PNW in themid-1980s. However, wheat is normally less affected severally than barley, and spring-seeded crops are more susceptible to ShB infection than fall-sown crops. Yield losses connected with *Rhizoctonia* root rot are particularly distinct in direct-seed systems.

In rice, sheath blight disease, caused by the fungal pathogen *R. solani* Kuhn, causes important yield damage and reduction in grain quality in the southern United States and other sections of the world (79, 145). Most of cultivated and wild rice relative are susceptible to *R. solani* and presently expensive fungicide applications are the crucial methods for control of this disease. Different examinations have demonstrated that response of various rice lines infected by *R. solani* and expressed partial

resistance (87). However, few studies showed moderate level of resistance that may be either incomplete, quantitative, field, or horizontal resistance (189). it is conducted in a research to exploit whole genome sequences of 13 rice (*O. sativa* L.) inbred lines to identify non-synonymous SNPs (nsSNPs) and candidate genes for resistance to ShB and Sanger sequencing conformed presence of 12 selected nsSNPs in two lines. "Resistant" nsSNP alleles were detected in two accessions of *O. nivara* that suggests sources for resistance occur in additional *Oryza* sp (161). ShB QTLs identified via association mapping in rice using 217 subcore entries from the USDA rice core collection, which were phenotyped with a micro-chamber screening method and genotyped with 155 genome-wide markers (54). Majority of the resistant entries that contained a large number of the putative resistant alleles belonged to indica, which is consistent with a general observation that most ShB resistant accessions are of indica origin. There have been several efforts for the identification of genes in rice that confer increased ShB resistance. However, till date, few major ShB resistance genes have been identified from either cultivated rice or wild rice accessions (112; 35; 153; 14).

SHEATH BLIGHT PATHOGEN

Upto 200 plant species are infected by *R. solani* Kuhn (teleomorph *Thanatephoruscucumeris* [Frank] Donk). This disease is one of the recorded common soil-borne pathogens in crop plants (40). *Rhizoctonia* belongs to the Basidiomycetes, with *R. solani* being multinucleate. *R. solani* species [teleomorph *T. cucumeris* (Frank)] represent a collective species (166), which has been divided into 13 anastomosis groups (AGs) (AG-1–AG-13) and AG-BI (the bridging isolate AG) (10; 9). The anastomosis bunch AG-1 can be additionally subdivided into three intraspecific gatherings dependent on malady indications, social qualities, rDNA similitude, and isozymes (91; 109; 86). The intraspecific gatherings are AG-1 IA (ShB on rice), AG-1 IB (web scourge), and AG-1 IC (damping off) (165). Rice ShB is an especially significant segment of the rice ailment complex, happening in most rice-creating regions, including India. From hyphal anastomosis responses, isolates are divided into AGs. Different types of strains of *R. solani* belong to at least 14 different, genetically defined populations of AGs determined by anastomosis between hyphae of strains belonging to the similar AG. The AGs themselves do not essentially give evidence on the genetic difference and taxonomic relationships within and between AGs. Among the different types of symptoms, ShB is the most prominent and common one. Because of its semi-saprophytic nature, *R. solani* has a wide range and uncharacterized pathogenicity mechanisms. Despite the fact that *R. solani* is causal organism of extensive range of economically significant diseases in different plant species. There are very few reports on the concerned gene(s) and their respective function in relation to pathogenicity (94). Encompassing the present attention on genomics to include *R. solani* would be of great utility for building up knowledge on genes and gene expression from this important plant pathogen. it is reported that high nitrogen (N) rate and dense planting were conducive to ShB development. Application of silicon fertilizer under high N rate failed to suppress the disease epidemic, especially when silicon concentration of the soil is high or there is enough plant-available silicon(183)

Mode of infection and transmission of *R. solani* and biochemical response by rice

The fungus survives either as sclerotia or mycelia in plant debris, which forms infection cushions surface in favourable condition, germinates, and forms infection cushions and/or lobate appressoria on the plant surface for infection (Fig. 1). After the initial infection, the pathogen moves up the plant by surface hyphae and develops new infection structures over the entire plant, causing significant necrotic damage (119). The infected rice seeds may produce 4–6.6% seedling infection in India (102; 119). But on transplantation, the infected seedlings were unable to develop disease (113). The disease cycle takes place predominantly through sclerotia in the humid tropics. Sclerotia, the dormant are shed before/or during the harvest operation and remain in the soil and survive for a long time. When the buoyant sclerotia tend to accumulate in undisturbed standing water at the plant–water interface, the aerobic fungus creeps up several centimeters in 24 h and the primary infections are caused in wetland rice. Rain water runoff and flood irrigation permit good dispersal of floating sclerotia (77) and consequently provide the primary foci of infection through the stretches of rice fields. Further, with the increasing size of sclerotia on their fragments, the number, and size of lesions also increased (30). The pathogen-induced lesions on leaf blades and leaf sheaths of infected plants. It produces sclerotia on both abaxial and adaxial leaf sheath surfaces but not in the tissue. The pathogen forms infection cushions and lobate appresoria on leaf sheath and directly penetrates the cuticle or through stomata (65). Once infection occurs, secondary spread takes place through direct contact (role of basidiospores uncertain). Sclerotia may move from one field to another through irrigation water, and during movement, they may produce mycelia and secondary or tertiary sclerotia.

Defense responsive proteins, including different enzymes that can directly act on pathogen components have been linked to basal resistance and this resistance governed by different quantitative traits both of which are associated with broad-spectrum resistance. Rice proteomics research has made considerable progress recently in providing functional information of proteins expressed in the various developmental stages, tissues, cells, and abiotic and biotic stress environments (Fig 2). it is studied that the defense response in transgenic Pusa Basmati 1 (PB1) rice lines engineered with rice chitinase gene (*chi11*) against the *R. solani*. After inoculation, with *R. solani* enhanced production of phenylalanine ammonia lyase, peroxidase, and polyphenol oxidase enzyme activities in resulted followed by reduced symptom development in transgenic rice lines in comparison to non-transgenic control plants. After infection with *R. solani*, loss of chlorophyll resulted in a non-transgenic line in comparison to transgenic rice line (149). In one study, the role of NH-1, several PR genes, phenylalanine ammonia-lyase, and lipoxygenase in the defense responses of rice against *R. solani* was observed, the causal agent of rice ShB disease. The induction of PR-5, PR-9, PR-10, PR-12, PR-13, and NH-1 was observed in the resistant and susceptible Iranian cultivars of rice Tarom and Khazar rice cultivars after infection by *R. solani* (151). Even though plant–pathogens, their hosts and the interactions between them have been studied using classical biochemical, genetic, molecular biological, and plant pathology approaches, systems biology approaches such as genomics and proteomics are essential to provide global information on the various cellular genomic and proteomic networks (159). Chitinases are an example of defense response enzymes that have been linked to

basal resistance. a specific 3-β HSD proteins was identified in resistant rice varieties LSBR-5 associated with response to infection by *R. solani*after2-dimensional gel electrophoresis and electrospray ionization quadrupole-time of flight mass spectrometry (ESI Q-TOF MS). Sixteen additional proteins identified in the above studied have been previously reported to be involved in antifungal activity, signal transduction, energy metabolism, photosynthesis, protein folding and degradation, signal transduction, and antioxidation (80).

Detection of ShB pathogen in rice

Rice sheath diseases caused by *Rhizoctonia* species are relatively difficult to diagnose by visual observation alone due to the similarity of the symptoms with those caused by other disorders. Moreover, various *Rhizoctonia* species have been isolated from rice sheaths showing similar symptoms. *R. oryzae*, the causal agent of "bordered sheath spot" and *R. oryzae-sativae*, the causal agent of "aggregate sheath spot" have been reported on rice from Eastern and South eastern Asia (47; 48). These pathogens produce very similar symptoms in the field. In India, scientific information on rice ShB and related diseases is scanty and the population diversity of the causal agents has not yet been surveyed. However, knowledge of the populations of pathogenic *Rhizoctonia* species is essential for integrated control strategies; along with the understanding of the influence of other characteristics, including pathogenicity, host range, and adaptability to environmental conditions. it is studied that a real-time, quantitative polymerase chain reaction (QPCR) assay to detect and quantify *R. solan i*AG-1 IA DNA from infected rice plants. A specific primer pair was designed based on the internal transcribed spacer region of the fungal ribosomal DNA. The specific detection of *R. solani* DNA was successful with quantities as low as 1 pg. The QPCR assay could be used for detecting the rice ShB pathogen, quantifying fungal aggressiveness, and evaluating the resistance level of rice cultivars (152). In a report, RAPD-PCR was used for identifying a specific fragment from which SCAR primers were developed and used for PCR detection of the subgroup AG 1-IB. The designed SCAR primer N18-rev/N18-for allowed the unequivocal detection of the specific DNA fragment of 324 bp from field-grown lettuce plants with bottom rot symptoms or artificially inoculated plant species and from different types of inoculated field soils. A specific diagnosis PCR assay for *R. solani* subgroup AG 1-IB was established, which can be used as a highly specific, reproducible, and applicable test system in plant disease diagnosis. The designed primer pair may have applications in a multiplex detection tool for *R. solani* or soil-borne pathogens (38). a conventional primer set (Rs1F2 and Rs2R1) was designed from the nuclear ribosomal internal transcribed spacer (ITS1 and ITS2) regions of *R. solani*. Following PCR amplification, a 0.5-kb product was amplified from DNA of all isolates of AG-3 using primers Rs1F2 and Rs2R1. No product was amplified when DNA from isolates belonging to a range of other *R. solani* AGs or from a selection of other potato pathogens was tested, confirming the specificity of the primers for AG-3 only. *R. solani*AG-3 was also detected in potato tissue with varying black scurf severity, and in soil inoculated with sclerotia of *R. solani* to a minimum detection level of 5×10^{-4} g sclerotia/g soil. In addition, specific primers RsTqF1 (based on the Rs1F2 sequence) and RsTqR1, and a TaqMan™ fluorogenic probe RQP1, were designed to perform real-time quantitative (TaqMan) PCR. The conventional PCR and real-time PCR assays were compared and combined with direct DNA extraction from soil and a seed-baiting method to determine the most reliable method for the detection and quantification of AG-3 in both artificially inoculated field soil and naturally infested soils. It was shown that direct DNA extractions from soil could be problematic, although AG-3 was detectable using this method combined with the real-time PCR assay (81). developed SYBR Green I-based real-time QPCR assays developed that is specific to internal transcribed spacers ITS1 and ITS2 of the nuclear ribosomal DNA of *R. solani* and *R. oryzae*. The assays were diagnostic for *R. solani*AG-2-1, AG-8, and AG-10, three genotypes of *R. oryzae*, and an AG-I-like binucleate *Rhizoctonia* species (117). Quantification was reproducible at or below a cycle threshold (C_t) of 33, or 2–10 fg of mycelial DNA from cultured fungi, 200–500 fg of pathogen DNA from root extracts, and 20–50 fg of pathogen DNA from soil extracts. However, pathogen DNA could be specifically detected in all types of extracts at about 100-fold below the quantification levels. Another study reported that AG1-IA specific genes and predicts important virulence determinants that might enable the pathogen to grow inside hostile plant environment (34). the gene responsible for the pathogenicity was identified through RNAseq analysis, Ghosh et al (34) identified a total of 65 and 232 *R. solani* (strain BRS1) genes to be commonly upregulated in three different rice genotypes (PB1, Tetep, and TP309) at establishment and necrotrophic phase, respectively. The induction of genes encoding extracellular protease, ABC transporter, and transcription factors were notable during establishment phase. While during necrotrophic phase, several CAZymes, sugar transporters, cellular metabolism, and protein degradation-related genes were prominently induced (33).

Disease Management by chemical

Inorganic nutrient management is a major factor determining rice ShB disease. it has been reported that plant variety and nitrogen fertilizers are the major factors influencing ShB disease and concomitant yield losses in rice, both during wet and dry seasons. Varieties with taller stature, fewer tillers, and lower leaf N concentration, such as IR68284H, generally had lower ShB lesion height (LH), ShB index, and consequently lower yield loss from the disease. Greenhouse and field studies with the fungicide Lustre (37.5SE) (fluconazole + carbendazim) against ShB revealed that the application of the triazole mix could reduce disease severity and increase yields (174). Further, it was proved that the test fungicide was a safe combination fungicide without any phytotoxic symptoms. Its prophylactic application gave better results than as a curative application (140). Certain new fungicidal formulations were also found effective against rice ShB. Among them, Amistar 25 SC@1.0 ml L-1 (30.6%) and RIL-010/FI 25 SC at 0.75 ml L-1 (30.1%) showed a high degree of efficacy in reducing the disease severity and were superior over the standard fungicides (validamycin at 2.5 ml L-1). Highest grain yields were also reported in these fungicide treatments (137). Details of different chemicals used for the successful control of sheath blight resistance has been applied in the farmer's field (Table 1). However, use of chemicals are harmful for the environment and farmers.

Biological Control

Leaf extracts of certain plant species were also used for the effective management of rice ShB. Among them, the leaf extract of *Pithecellobium dulce* was highly effective to inhibit the mycelia growth of test pathogen (2.5 cm over 8.9 cm in control). Both the leaf extracts of *P. dulce* and *Prosopis juliflora* were found equally effective in inhibiting sclerotial number, dry weight, and

germination of the ShB pathogen and controlling ShB with a disease incidence of 32.3% and 33.3%, respectively, over 76.2% in control (105).

Resistance sources in rice and their wild relatives

Disease resistance in plants can be classified into two major categories. Various terms have been used to describe the two categories of resistance, such as vertical versus horizontal resistance (178), qualitative versus quantitative resistance (118), and complete versus partial resistance (55; 157). There are different rice lines reported resistance like Brimful, Jasmine 85, LSBR-5, LSBR-33, Marsi, Minghui 63, Saza, Tetep, Tadukan, Teqing, Tauli, and ZYQ8, in which a high degree of quantitative resistance is found against ShB pathogen under field conditions (121; 150; 158). However, in most cases, qualitative resistance is modulated by direct or indirect interaction between the products of a major disease resistance (R) gene and an avirulence gene; this type of resistance is specific to pathogen race and is lifetime-limited in a particular cultivar due to the strong selection pressure against and the rapid evolution of the pathogen. In contrast, quantitative resistance is conferred by quantitative trait loci (QTLs) and is presumably race-nonspecific and durable (144; 103).).

Several groups have attempted to identify sources of ShB resistance by screening local accessions, cultivars, landraces, and/or advanced breeding lines (Table 2). Sources of ShB resistance have been sought for in different rice-growing regions by many different research groups. These studies resulted in the identification of genotypes with moderate-to-high levels of resistance. In rice, only partial resistance to rice ShB has been identified, as evidenced by a survey of 6000 rice cultivars from 40 countries from which no cultivar exhibiting a major gene for rice ShB resistance was identified (56;). Additional research suggests it is feasible to identify major genes conferring high levels of partial resistance (121), pyramid these genes, and achieve nearly complete ShB resistance. Screening has also been conducted at the Centro International de Agricultura Tropical (CIAT); 63% of the genotypes tested were found to be promising candidates as sources of ShB resistance. ShB resistance sources were also sought by Raj et al. (133, 42) . 282 accessions were tested recently in USA and reported that 25 showed high levels of resistance (78).

Both wild species and landraces of the *Oryza* genus possess under-exploited alleles that may have a strong potential for the improvement of Asian rice (*O. sativa* L.) and African rice (*Oryza glaberrima* Steud.). Wild rice accessions have been used to successfully develop resistance against many rice diseases (8). Over the years, a very large number of accessions from different species of *Oryza* have been tested at IRRI to identify sources for ShB resistance (Table 2). From a total of 233 accessions tested, 76 were found to contain a high level of resistance to ShB and 29 showed moderately resistance. The latter accessions belonged to the African rice, *O. glaberrima* (2n = 24 AA), a close relative of *O. sativa* (2n = 24 AA). The relatively high resistant accessions belonged to mixed genetic groups (48). In addition to the studies mentioned above, Wild accessions or their derivatives were evaluated against ShB resistance (2,75). In the USA, Prasad and Eizenega (130) evaluated 73 *Oryza* spp. accessions with three different screening methods and identified seven accessions (three *O. nivara*) Sharma and Shastry and one each of *O. barathi* A. Chev, *O. meridionalis* Ng, *O. nivara*, *O. sativa* L., and *O. officinalis* Wall ex Watt) that showed moderate resistance. Similar efforts were made by Ram et al. (136), who screened 22 accessions belonging to 11 different species of *Oryza*, identifying the accessions of *O. latifolia* (Desv.), *O. grandiglumis* (Doell) Prod, *O. nivara*, and *O. rufipogon* as having a higher level of resistance, and Shamim et al. (157) also reported two wild rice accessions *O. australiensis* and *O. grandiglumis*.Ram et al. (2008) screened 32 accessions belonging to 11 different species of *Oryza*, namely, 11 accessions of *O. rufipogon*, 8 of *O. nivara*, 3 of *O. eichingeri*, 2 each of *O. ojficinalis* and *O. latifolia*, and 1 each of *O. longistaminata*, *O. minuta*, *O. aha*, *O. meridionalis*, *O. punctate*, and *O. grandiglumis* against ShB along with susceptible variety of *O. saliva* (cv. Ajaya). Crosses of susceptible *O. sativa* (HM 36-6-4-F) and resistant *O. latifolia* (DRW 37004) and *O. sativa* (HM 36-6-4-F) × *O. punctata* (DRW 32002) and F_1s were produced using embryo rescue technique were screened. One BC_1F_1 plant of each *O. sativa* × *O. latifolia* and *O. sativa* × *O. punctata* showed high level of resistance with score 3 and one each from *O. sativa* × *O. latifolia* and *O. sativa* × *O. punctata* showed moderate level of resistance with score 5. Finally, they reported that the ShB resistance is heritable and there is scope for introgression of genes from distantly related species. The *O. rufipogon* accession DRW 22017-5 provides an important source for ShB resistance, which can be exploited to improve the modern high-yielding cultivars and pyramiding it with the genes for moderate resistance in cultivated germplasm would certainly increase the level of resistance. Shamim et al. (157,158) also reported that wild rice accessions, *O. australiensis* and *O. grandiglumis* have resistance source against *R. solani*, belonging to AG 1 IA anastomosis group.

RESISTANCE QTLS AGAINST SHEATH BLIGHT

Over the past decades, studies on resistance to ShB have been conducted by many researchers who have had diverse objectives, including screening the germplasm of cultivated rice/rice wild relatives, assessment of genetically engineered plants with genes for resistance, and phenotyping for QTL mapping or validation (Fig 3 and Fig 4and Table 3). To this end, a broad spectrum of methods has been employed, which can be described by four main components: the biological hierarchy level addressed (from organs to field plots), the inoculation method used, the incubation conditions, and the disease assessment methodology. The choice of these components is critical to the outcomes of the studies since it is underpinned by the (presupposed) biological processes involved in disease resistance. Methodological choices also have major consequences on the accuracy, precision, repeatability, and ultimately usefulness of the results. Quantitative resistance, in contrast with qualitative resistance, is generally considered as partial resistance in a particular cultivar (123). This type of disease resistance is controlled by multiple loci, referred to as QTLs, and does not comply with simple Mendelian inheritance. Thus, selecting for these QTLs is difficult. However, several studies have indicated that pyramiding resistance QTLs can achieve the same level or even a higher level of resistance than that conferred by an R gene (Khush, 1977 67; 30; 79; 145; 104 11 40; 143; 180; 198; 54; 161; 199; 179).

Six QTLs were identified against ShB resistance in an F_4 population of Teqing/Lemont, but one allele on chromosome 8 for the resistance contributed by Lemont could not be identified in our clonal population, of which one parental variety was also Lemont. On the other hand, a major resistance QTL, *qSB*-11, on chromosome 11, which explained 31.2% of the total phenotypic variation, was identified in our study, and Li et al. also indicated that there might have been a putative-resistant QTL in the same

interval of qSB-11, though they did not give any further information about the effect of this locus possibly due to its low LOD score (84). In addition, they identified three QTLs for heading date and four QTLs for plant height in the resistance loci interval and thus thought that the QTLs for ShB resistance were closely associated with the QTLs for heading date or plant height. The main QTL (QSbr4c) controlling LH and actual lesion length (ALL) associated with RM280, on chromosome 4, was located near the chromosomal region of resistance QTL and QSbr2a controlling LH and ALL associated with RM341, on chromosome 2, was approximately mapped on the same chromosomal region of qSB2 identified by Zou et al. (200). Approx. 266 Near Isogenic Introgression Lines was constructed with randomly introgressed Lemont segment of a cross between Lemont × Teqing. Further, 15 M-QTLs detected for LH and ALL over assessment times were mapped on seven chromosomes (1, 2, 3, 4, 5, 9, and 12), explaining 35.8%–93.8% of the phenotypic variation. The QTLs with high additive effects for most resistance traits were found at the markers RM341 (on chromosome 2), RM156 (on chromosome 3), and RM280 (on chromosome 4). The four QTLs, namely, QSbr1a, QSbr2a, QSbr4c, and QSbr9b that were found not associated with plant morphology or heading date are potentially useful in breeding programs for ShB resistance (93).

The introgression of the QTL, qSB-11LE was reported and observed reduced grain loss by 10.71% in Lemont background under severe disease infestation in field trials (201). The QTLs namely qSB-9TQ and qSB-3TQ could reduce the crop loss due to ShB by 15% when introduced into Lemont (127). The resistance QTLs were obtained by crossing the Lemont and Teqing (LT-ILs and TQ-ILs). Lemont further a total of 10 main-effect QTLs (M-QTLs) and 13 epistatic QTLs (E-QTLs) conferring ShB resistance (SBR) were mapped using data obtained from different years and genetic backgrounds. Among them, 6 M-QTLs detected in 2006 were verified in 2007, suggesting that these M-QTLs had reliable performance across the years. *QRlh4* was the only M-QTL expressed under the reciprocal backgrounds. On chromosome 10, *QRlh0a* between RM216 and RM 311 was detected in TQ-ILs and *QRlh0b* between RM222 and RM 216 was detected in LT-ILs and regarded as a different gene because their directions of additive effect were opposite. Most QTLs identified in TQ-ILs were not expressed in LT-ILs, indicating the presence of a significant effect of genetic background. By comparative mapping, 8 M-QTLs detected in this study were located in the same or near regions that were associated with SBR identified in the previous studies. These M-QTLs have great potential to be applied in rice breeding for SBR by marker-assisted selection (MAS), and M-QTLs expressed stably in different backgrounds are favorable for gene pyramiding in SBR improvement in rice (187).

The three resistance QTLs could significantly improve the resistance to rice ShB separately or jointly. A rice ShB resistance QTL *qSB7* (superscript Tq) on rice chromosome 7 of Teqing was confirmed by using the backcross between Teqing and Lemont. The effects and pyramiding effects of *qSB7* (superscript Tq), *qSB9* (superscript Tq) (a rice ShB resistance QTL mapped on chromosome 9 of Teqing) and *qSB11* (superscript Le) (a rice ShB resistance QTL mapped on chromosome 11 of Lemont) were studied by using a set of near-isogenic lines (NILs) under the background of Lemont (194). A population of 279 $F_{2:3}$ progeny derived from a cross between two tropical *japonica* U.S. rice cultivars, Rosemont (semi-dwarf, SB susceptible) and Pecos (tall, SB resistant), was used to map SB resistance (109).

Similarly, *qShB9*-2, a QTL for ShB, was mapped to a region at the bottom of chromosome 9 consisting of ≈1.2 Mbp flanked by SSR markers RM215 and RM245 (87). The majority of variants in qShB9-2 were classified as sSNPs (73%), a substantially smaller percentage as nsSNPs (26%), and the smallest fractions identified were insertions (1.0%) or deletions (0%). When the CV selection procedure was carried out to identify candidate nsSNPs for SB resistance within qShB9-2, relatively few selected nsSNPs (10) were found that mapped throughout most (»1.1 Mbp) of the QTL. The nsSNPs were detected in a total of 10 genes that were placed into seven groups based on gene ontology/gene function. The physical location of selected nsSNPs within qShB9-2 along with corresponding genes and QTL qShB9-2 explained »25% of the observed variation for SB resistance when Jasmine 85 was used as the resistant parent (Liu et al, 2009 87).

Around 127 recombinant inbred lines in seven environmental conditions at three locations across 4 years were examined, but the QTL with the largest effect was detected. One QTL, qSBR11-1, was detected commonly in three conditions (r^2 = 12–14%), but no QTL was detected in more than three (12). Zuo et al. (202) studied ShB resistance and its potential in breeding programs by using NILs and found the three different genotypes at the qSB-11LE locus and seven backcross populations sowed positive effect. Observation from the field disease evaluation data under artificial inoculation revealed that the inheritance of resistance of *qSB-11LE* to ShB is controlled by additive gene action and corresponding genes have a dosage effect on ShB resistance. Further in greenhouse evaluations, the resistance effect of *qSB-11LE* was expressed at 11 and 14 days after inoculation at the tillering stage. Finally, analysis of field resistance of six BC_1F_1 populations and one BC_2F_1 population, developed by backcrosses between Lemont as the donor parent and six commercial *O. indica* rice cultivars as recurrent parents, significantly indicated that *qSB-11LE* could be effectively used to enhance these cultivars against ShB resistance.

Three Teqing into Lemont backcross introgression lines (TILs) were selected with more resistant than their susceptible parent (Lemont). Further these QTLs were molecularly verified to contain Teqing alleles at *qSB92* and/or *qSB121*. By comparing the ShB resistance in micro-chamber evaluations and inoculated field plots, the phenotypic values of the QTL were measured. Under both study conditions, disease resistance ranked *qSB92* + *qSB121*>*qSB92*>*qSB121*> no QTL, with both *qSB92*and *qSB121* acting as dominant resistance genes. In micro-chamber studies, *qSB92 TQ* reduced disease with an average of 1.0 disease index units and *qSB121TQ* by 0.7 using a scale of 0–9. Field effects of *qSB92 TQ* and *qB121TQ* were less pronounced, with average phenotypic gains of 0.5 and 0.2 units, respectively. TIL:642 proved to contain *qSB92 TQ* in an introgression so small that it was tagged by just RM205 on the tip of chromosome 9. These studies verify that the *indica* introgression of *qSB92 TQ* or *qSB121 TQ* can measurably improve resistance to ShB disease in a highly susceptible tropical *japonica* cultivar, and fine mapped the *qSB92* locus (180). Pandian et al. (122) identified that Tetep carry 12 QTLs governing ShB resistance. Further, parental screening for the ShB resistance by using a highly virulent isolate Kapurtala was done. Evaluation of 186 advanced backcross inbred lines for ShB resistance revealed that 9 Pusa6B-derived inbred lines were resistant, 11 Pusa1460-derived inbred lines, and 12 PRR78-derived lines were moderately resistant to ShB. The varying quantum of resistance depicted by the field screening implies that, varying number of QTLs present in the residual donor segments of the ABLs.

The major ShB-QTL *qShB9-2* was confirmed based on the field data and also identified one new ShB-QTL between markers RM221 and RM112 on chromosome 2 in the RIL population derived from the cross of Lemont × Jasmine 85 (LJRIL) . Based on the field verification of ShB evaluations, the micro-chamber and mist-chamber assays were simple, effective, and reliable methods to identify major ShB-QTLs like *qShB9-2* in the greenhouse at early vegetative stages. The markers RM215 and RM245 were found to be closely linked to *qShB9-2* in greenhouse and field assays, indicating that they will be useful for improving ShB resistance in rice breeding programs using MAS (89). Three landraces were collected from the Himalayas, Jarjan, Nepal 555 and Nepal 8, with resistance to ShB. Further, they developed backcrossed inbred lines derived from a cross between Jarjan and the leading Japanese cultivar Koshihikari and further were used in QTL analyses, since later-heading lines showed fewer lesions. Eight QTLs were further identified, and only one QTL on chromosome 9 (between markers Nag08KK18184 and Nag08KK18871) was detected. Chromosome segment substitution lines (CSSLs) carrying it showed resistance in field tests. Thirty F_2 lines derived from a cross between Koshihikari and one CSSL supported the QTL (172).

Nine rice cultivars were selected and screened at greenhouse conditions. Results showed that Tetep and Teqing had the lowest disease ratings. UKMRC2, a new high yielding cultivar, was as recipient parent. Crosses between UKMRC2 and Teqing, and UKMRC2 and Tetep were made and confirmed. Subsequently four-way crosses between the two F_{1s} were performed to develop pyramidal lines (44). The major quantitative trait locus Qsb9 was repoted which confers significant resistance to rice ShB. However, the precise location has not yet been determined. They reported the fine mapped location of *qSB9 TQ*, the resistant allele(s) underlying qSB9 derived from *indica* rice variety Teqing (TQ). A population containing 235 CSSLs that integrated TQ donor segments specific to the qSB9 region in the Lemont genetic background were developed and studied. These CSSLs contained identical genetic backgrounds, as monitored with 111 molecular markers and showed similar morphologies except for TA. They also identified a gene controlling TA, *TAC1 TQ*, in the qSB9 region by comparing the TA phenotype and the genotype of each CSSL. Although *TAC1 TQ* only showed a very mild effect on SB resistance, it affected the accurate evaluation of the contribution of *qSB9 TQ*. The development of new molecular markers in this region and accurate determination of the SB resistance phenotypes of these 10 CSSLs by conducting both field and greenhouse tests allowed us to finemap*qSB9 TQ* to a 146-kb region defined by markers CY85 and Y86 (203).

Wen et al. (182) reported eight different QTLs for disease rating (four in E1, four in E2, and three in E3), six QTLs for LH (one in E1, three in E2, and two in E3), and seven QTLs for percentage of LH (one in E1, four in E2, and two in E3). Sixteen of the ShB-QTLs co-localized as six clusters on chromosomes 3, 7, 11, and 12. Four of the six clusters contained ShB-QTLs that were detected in two environments, while the other two clusters with ShB-QTLs were detected in one environment. Three ShB-QTLs (*qSBD-3-2*, *qSBL-3-1*, and *qSBPL-3-1*) were delimited to a 581-kb region flanked by markers D333B and D334 on chromosome 3. 40 different rice germplasm including 8 wild, 4 landraces, 26 cultivated, and 2 advanced breeding lines was studied for ShB resistance. Except two rice varieties, Tetep and ARC10531 expressed moderate level of resistance against ShB. Further two mapping populations (F_2 and BC_1F_2) were developed from the cross BPT-5204/ARC10531 for QTL mapping. With the utilization of composite interval mapping analysis, 9 QTLs were mapped to 5 different chromosomes with phenotypic variance ranging from 8.40% to 21.76%. Two SSR markers RM336 and RM205 were noted to be closely related with the major QTLs qshb7.3 and qshb9.2. A hypothetical β 1–3 glucanase with other 31 candidate genes were identified in-silico study by utilizing rice database RAP-DB (191).

A doubled haploid population developed that was constructed from a cross between a *japonica* variety CJ06 and an *indica* variety TN1 and analyzed the QTLs for SB resistance under three different environments. They identified QTLs for LH on chromosomes 1, 3, 4, 5, 6, and 8 and explained 4.35–17.53% of the phenotypic variation against ShB. The ShB resistance allele of *qHNLH4* from TN1 decreased LH by 3.08 cm and contributed to 17.53% of the variation at environment 1. The QTL for LH (*qHZaLH8*) detected on chromosome 8 in environment 2 explained 16.71% of the variation, and the resistance allele from CJ06 reduced LH by 4.4 cm. Eight QTLs for DR were identified on chromosomes 1, 5, 6, 8, 9, 11, and 12 under three conditions with the explained variation from 2.0% to 11.27%. The QTL for disease rating (*qHZaDR8*), which explained variation of 11.27%, was located in the same interval as that of *qHZaLH8*; both QTLs were detected. Yuan et al. (2019) detected a total of 128 minor effect QTLs were detected by multiple interval mapping. These QTLs explained less than 11.2% of the phenotypic variations individually, and 106 QTLs were clustered in 20 QTL-rich regions/putative loci. Significant QTLs by environment interactions were detected at three putative loci (qSBR11.1, qSBR11.2 and qSBR11.3), indicating that these 3 loci were not stable. The other 17 stable loci (qSBR1.1, qSBR1.2, qSBR2.1, qSBR2.3, qSBR3.1, qSBR3.2, qSBR3.5, qSBR3.6, qSBR5.1, qSBR7.1, qSBR8.1, qSBR9.1, qSBR9.2, qSBR9.3, qSBR12.1, qSBR12.2 and qSBR12.4) provided a foundation for marker-assisted selection in breeding. Further the study concluded that eight resistance alleles from four QTLs (qSBR7.1, qSBR8.1, qSBR9.3, and qSBR12.2) might be pyramided for enhancing sheath blight resistance. The identified QTLs are frequently used by the breeders for the detection of other rice locus in the different other rice genotypes. These QTLs will also be used for the stacking of resistance in the high yielding rice genotypes (197). Quantitative trait loci (QTL) associated with ShBresistance mapped using two F8 recombinant inbred line populations generated from crosses of an indica crop variety, Dee-Geo-Woo-Gen (DGWG), with individuals representing the two major US weed biotypes, straw hull (SH) and black hull awned (BHA). Nine ShB resistance QTL across both mapping populations were identified and two of these, qShB1–2 and qShB4, are different from previously identified ShB QTL and represent new candidates for further study in respect to sheath blight resistance (135).

TRANSGENIC RICE AGAINST ShB

There is several transgenic rice lines with different defense related gene with increased resistance to ShB (Table 4) have been reported (20; 85; 188; 98). Pathogenesis-related (PR) proteins are produced in response to an attack by a pathogen and are known to play key roles in the plant defense mechanisms 21; 23). Over-expression of PR proteins, including chitinase (PR-3), β,3-glucanases (PR-2), thaumatin-like proteins (PR-5), and other plant- or microbe-derived antifungal proteins have been used to develop transgenic plants against

fungal infection. Chitinases that hydrolyze the b-1,4 linkages of nacetyl glucosamine (chitin) have been well characterized. Over-expression of different chitinases in rice cultivars has been found to result in enhanced resistance against ShB (22). The expression of pinA and/or pinB 68), Ace-AMP1 (124), and Dm-AMP1 (52) resulted in not only enhanced resistance against ShB but also against other rice diseases. There have also been efforts to combine resistance genes to generate plants with increased resistance to ShB. These researchers suggested that Dm-AMP1 and Rs-AFP2 may be the best genes used to date in transgenic approaches. To date, more than 12 rice cultivars, including IR72, IR64, Chinsurah Boro II, Basmati 122, Swarna, and IR58, have been transformed with genes for ShB resistance The transgenic plant with a chitinase gene was reported under the control of the CaMV 35S promoter showed resistance to the ShB pathogen, *R. solani* (85). Transgenic elite indica rice cultivars with a PR-3 rice chitinase gene (RC7) showed higher resistance to rice ShB disease caused by *R. solani* (22). Transgenic rice was developed by introducing a basic chitinase gene (RC24) into the elite indica variety Zhuxian B and stably integrated in the genome of transgenic rice from R_0 generation to R_6 generation and expressed. Two transgenic strains, Zhuzhuan 68 and Zhuzhuan 70, and 43 zy transgenic lines were obtained, showing significantly higher resistance against rice blast and ShB (188). Transgenic rice, Zhongda 2, was developed by rice chitinase gene (*RC24*), showed high resistance to rice ShB (*R. solani*) in laboratory and a 2-year field experiment. The *R. solani* could invade sheath of Zhongda 2 and induce symptoms of the disease. No difference was noted in time of penetration or incubation period between Zhongda 2 and non-transgenic rice control, Zhuxian B, but the hyphae lysate could be observed earlier than control (188).

The different lines of elite indica rice cultivars such as ADT38, ASD16, IR50, and PB1 were engineered by constitutively overexpressing rice tlp encoding a thaumatin-like protein. The putative transformants and their progenies expressing tlp showed enhanced resistance against the ShB pathogen, *R. solani*, when compared to the non-transformed plants. The use of rice chi11, encoding a chitinase, as a cotransgene along with tlp produced a tlp–chi11 co-transformant that showed enhanced resistance against *R. solani* than the ones that express tlp or chi11 transgene alone (57). Indica rice cultivars were engineered with two genes rice chitinase (*chi11*) and a thaumatin-like protein (*tlp*) coexpression of chitinase and thaumatin-like protein in the progenies of a transgenic PB1 line revealed an enhanced resistance to the ShB pathogen, *R. solani*, as compared to that in the lines expressing the individual genes. The transgenic PB1 line pyramided with the genes*chi11*, *tlp*, and*Xa21*showed enhanced resistance against ShB and bacterial blight (101).

Transgenic *O. sativa* L. var. PB1 was developed by using *Agrobacterium tumefaciens*. The TDNA of the cointegrate vector pGV2260::pSSJ1 carried the hygromycin phosphotransferase (hph) and betaglucuronidase genes. The binary vector pCamchi11, without a plant selectable marker gene, harbored the rice chitinase (chi11) gene under maize ubiquitin promoter. Co-transformation of the gene of interest (chi11) with the selectable marker gene (hph) occurred in 4 out of 20 $T_{(0)}$ rice plants (20%). Segregation of hph from chi11 was accomplished in two (CoT6 and CoT23) of the four co-transformed rice plants in the $T_{(1)}$ generation. The selectable marker free lines C_0T_6 and C_0T_{23} contained single copies of chi11. The lines C_0T_6 and C_0T_{23} exhibited 38% and 40% reduction in ShB disease (169). A transgenic rice line with 42 kDa endochitinase (*cht*42) gene was constructed from the mycoparasitic fungus, *Trichoderma virens*. Eight different transgenic plants containing single copies of complete TDNA were identified by Southern blot analysis. Homozygous transgenic plants were further identified for five lines in the T_1 generation. Homozygous T_2 plants constitutively accumulated high levels of the *cht*42 transcript, showed 2.4–4.6-fold higher chitinase activity after infection with *R. solani*. Infection assays with *R. solani* showed up to 62% ShB disease index reduction (155). Transgenic rice (cv. White Ponni) with thaumatin like protein gene (*tlpD*34, PR5) combination with the chitinase gene (*chi*11, PR3) was developed. The homozygous T_2 plants harboring*tlpD*34 + *chi*11 genes showed 2.8–4.2-fold higher chitinase activity. Upon infection with *R. solani*, the disease index reduced from 100% in control plants to 65% in a T_3 homozygous transgenic line T_4 expressing the *tlpD*34 gene alone. Disease index reduced up to 39% in the T2 homozygous transgenic line CT22 co-expressing *tlpD*34 and *chi*11 genes (156).

Helliwell et al. (43) produced transgenic lines with inducible production of ET by expressing the rice ACS2 (1-aminocyclopropane-1-carboxylic acid synthase, a key enzyme of ET biosynthesis) transgene under control of a strong pathogen-inducible promoter. The OsACS2-overexpression lines showed significantly increased levels of the OsACS2 transcripts, endogenous ET and defense gene expression in comparison to wild rice, especially in response to pathogen infection. The transgenic lines further exhibited increased resistance to a field isolate of *R. solani*, as well as different races of *M. oryzae*. Transgenic rice lines was generated by overexpressing the rice *oxalate oxidase 4* (*Osoxo4*) gene in a green tissue-specific manner which breaks down oxalic acid (OA), the pathogenesis factor secreted by *R. solani*. Transgenic plants showed higher enzyme activity of oxalate oxidase (OxO) than nontransgenic control plants. Transgenic Pusa Sugandhi II plants showed a higher level of expression of other defence-related genes in response to pathogen infection (110).

Rice basic chitinase gene (*RCH10*) and the alfalfa β-1,3-glucanase gene (*AGLU1*) were tandemly inserted into transformation vector pBI101 under the control of 35S promoter with its enhancer sequence to generate a double-defense gene expression cassette pZ100. The pZ100 cassette was transformed into rice (cv. Taipei 309) by *Agrobacterium*-mediated transformation. More than 160 independent transformants were obtained and confirmed by PCR. Northern analysis of inheritable progenies revealed similar levels of both *RCH10* and *AGLU1* transcripts in the same individuals. Disease resistance to both ShB and blast was challenged in open field inoculation. Immunogold detection revealed that RCH10 and AGLU1 proteins were initially located mainly in the chloroplasts and were delivered to the vacuole and cell wall upon infection, suggesting that these subcellular compartments act as the gathering and execution site for these antifungal proteins (98). OsPGIP1 was used against the PGase from *R. solani* for the transformation purpose. In addition, the location of OsPGIP1 was also determined by subcellular localization and subsequently, over expressed OsPGIP1 in a rice cultivar Zhonghua 11 (*O. sativa* L. ssp. japonica). Field testing of *R. solani* inoculation showed that the ShB resistance of the transgenic rice was significantly improved. Furthermore, the levels of ShB resistance were in accordance with the expression levels of OsPGIP1 in the transgenic lines. The results revealed the functions of OsPGIP1 and its resistance mechanism against rice ShB (179).

Molecular and functional analysis of the resistance genes was conducted with the major *R. solani*-resistance QTL qSBR11-1in indica rice genotypes Tetep. Sequencing and further study revealed the presence of a set of 11 tandem repeats containing genes with a high degree of homology to class III chitinase defense-response genes. Comparison between the resistant Tetep and the susceptible HP2216 lines shows that the induction of the chitinase genes is much higher in the Tetep line (142). Recombinant protein produced in vitro for 6 of the 11 genes showed chitinolytic activity in gel assays, but we did not detect any xylanase inhibitory activity. All the six *in vitro* expressed proteins show antifungal activity with a clear inhibitory effect on the growth of the *R. solani* mycelium. The characterized chitinase genes can provide an important resource for the genetic improvement of *R. solani* susceptible rice lines for ShB resistance breeding. Overexpression of OsOSM1 (OsOSM1ox) in susceptible variety Xudao 3 significantly increases resistance to SB in transgenic rice. The OsOSM1 mRNA levels in different transgenic lines are found to be positively correlated with their SB resistance levels. Intriguingly, although extremely high levels of OsOSM1 were detrimental to rice development, appropriately elevated levels of OsSOM1 were obtained that enhanced rice SB resistance without affecting rice development or grain yield. The OsSOM1 protein is localized on plasma membrane. OsOSM1 is upregulated by jasmonic acid (JA); furthermore, JA-responsive marker genes are induced in OsOSM1ox lines (190). Transformed IR64 rice was transformed by *mASAL* gene and evaluated antifungal activity against *R. solani*. The developed transgenic lines against *R. solani* exhibited an average of 55 % reduction in sheath blight percentage disease index (31). Similarly, a transgenic ASD16 rice plants harbouring rice chitinase *chi11* gene, belonging to a PR-3 group of defense gene conferring sheath blight (*R. solani* Kuhn) resistance has been developed by Rajesh et al. (135).

First transgenic line was developed by overexpressing two defense genes namely; rice chitinase gene (OsCHI11); and Arabidopsis NPR1 (AtNPR1) gene leads to a improved and improved performance against sheath blight than that of a single specific gene. A novel rice chitinase gene, LOC_Os11g47510 was cloned from QTL region of *R. solani* tolerant rice line Tetep and used for functional validation by genetic transformation of ShB susceptible japonica rice line Taipei 309 (TP309) (61). The chitinase expression and number of lesions formed and lesion length caused by *R. solani* and further the chitinase gene overexpression in transgenic plants correlate directly with sheath blight resistance in otherwise susceptible rice line TP309 (141; 148). An experiment was conducted by Overexpressing and knockdown rice transgenic lines of the OsGSTU5 gene in rice. The results obtained after *R. solani* infection displayed that the lesion cover area and hyphal penetration were more in the knockdown line and lesser in the overexpression line. Analysis of reactive oxygen species (ROS) accumulation showed more spots of H2O2 and O_2^- in knockdown lines compared to overexpressed lines. Later, RS transcript level was analyzed in *R. solani*-infected transgenic lines, which manifested that the knockdown line had higher RS transcripts in comparison to the control line and least RS transcripts were observed in the overexpressed line. In conclusion, rice transgenic lines overexpressing OsGSTU5 were found to be more tolerant, while the knockdown lines were more prone to *Rhizoctonia* infection compared to control lines (176).

Sheath blight resistance

The role of GF14e was studied in rice disease resistance by suppressing its expression using an RNA interference (RNAi)-silencing approach. GF14e-silenced transgenic plants showed spontaneous HR-like lesions and enhanced resistance to a virulent strain of *Xanthomonas oryzae* pv. oryzae. The enhanced resistance correlates with the high expression of a rice peroxidase gene and the accumulation of ROS. Silencing GF14e also enhanced resistance to the necrotrophic ShB pathogen *R. solani* (97). Xia (185) studied a gene encoding, a nucleoporin, named as cloned from rice Nipponbare (*O. sativa* L. spp. *japonica a*, var. *nippobare*). Further, the expression of *OsSeh*1 gene was induced by salicylic acid or ShB agent *R.solani.* The highest expression of *OsSeh*1 was observed at 24 h as 3.5 times more expression, was treated with *R. solani*. RNAi rice lines of *OsSeh1* gene were more susceptible to *R. solani.* In the transgenic line of T1 generation, relative expression quantity of *OsSeh*1 was found 6–11 with significant resistance, which was higher than 2.47 in wild type rice Nipponbare. In the RNAi rice plants, relative expression quantity of *OsSeh*1 is 0.2–0.6 in with obvious susceptibility. The results showed that rice resistance to *R. solani* was positively correlated with *OsSeh*1 expression levels. for the study of Host Delivered RNA Interference (HD-RNAi) technology to target two PATHOGENICITY MAP KINASE 1 (*PMK*1) homologs, *RPMK*1-1 and *RPMK*1-2, from *R. solani* using a hybrid RNAi construct. *PMK*1 homologues in other fungal pathogens are essential for the formation of appressorium, the fungal infection structures required for penetration of the plant cuticle, as well as invasive growth once inside the plant tissues and overall viability of the pathogen within the plant (175). Evaluation of transgenic rice lines revealed a significant decrease in fungal infection levels compared to non-transformed controls and the observed delay in disease symptoms was further confirmed through microscopic studies. Relative expression levels of the targeted genes, *RPMK*1-1 and *RPMK*1-2, were determined in *R. solani* infecting either transgenic or control lines with significantly lower levels observed in *R. solani* infecting transgenic lines carrying the HD-RNAi constructs.

FUTURE DIRECTION AND CONCLUSION

Sheath blight in rice is increasing in warm and humid climatic conditions. Application of fungicide is still most common management practice for this disease in the field condition. However, chemical fungicide is not using with care and good management that has a negative impact on environment as well as human health. Use of tolerant and resistant rice varieties is alternative sustainable method for the control of sheath blight disease. Whereas QTL study has recognized few probable resistance loci. molecular methods i.e. transcriptomic and sequencing technique revealed important candidate genes Identification and their characterization. These studies used for basic mechanisms for the pathogenicity factor of *R. solani* and resistance identification in rice and other hosts are not well understood and should be carried out on top priority for resistance identification. Pathogenesis genes from *R. solani* associated for different phases of infection also validated for their phase-wise infection during infestation process on hosts, that will serve as a situational expression for developing new resistance varieties. New molecular tools used for the transgenic lines overexpressing pathogenesis-related genes and silencing of pathogen-related kinase revealed promising results. Various molecular breeding strategy i.e. mapping and

tagging tools will also be used towards sheath blight resistance breeding utilizing those QTLs. In this context some important minor effect QTLs already have been identified in the different rice varieties, which may play a significant role for resistance breeding. Therefore, in the future resistance breeding approaches for sheath blight, researcher can appropriately increase the resistance sources with the identification of more resistant/tolerant cultivars and identification of important QTLs. The discovery of new QTLs/genes and alleles may further open the possibility of introducing resistance alleles into high yielding commercial varieties to reduce yield losses sustained by the sheath blight disease in rice.

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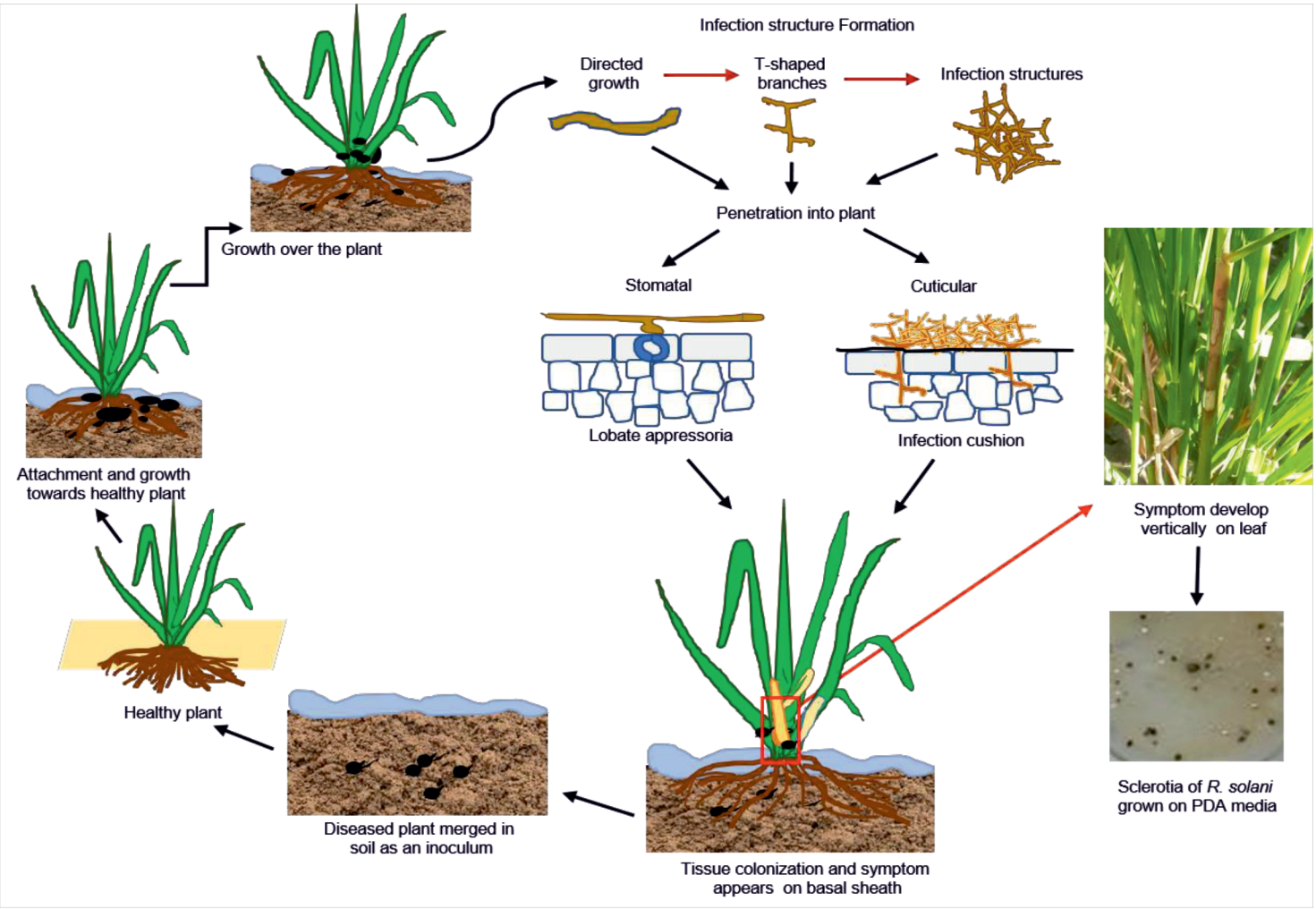

Fig. 1 Disease cycle and infection process in rice by Rhizoctonia solani showing different phases of sclerotia development and disease symptom

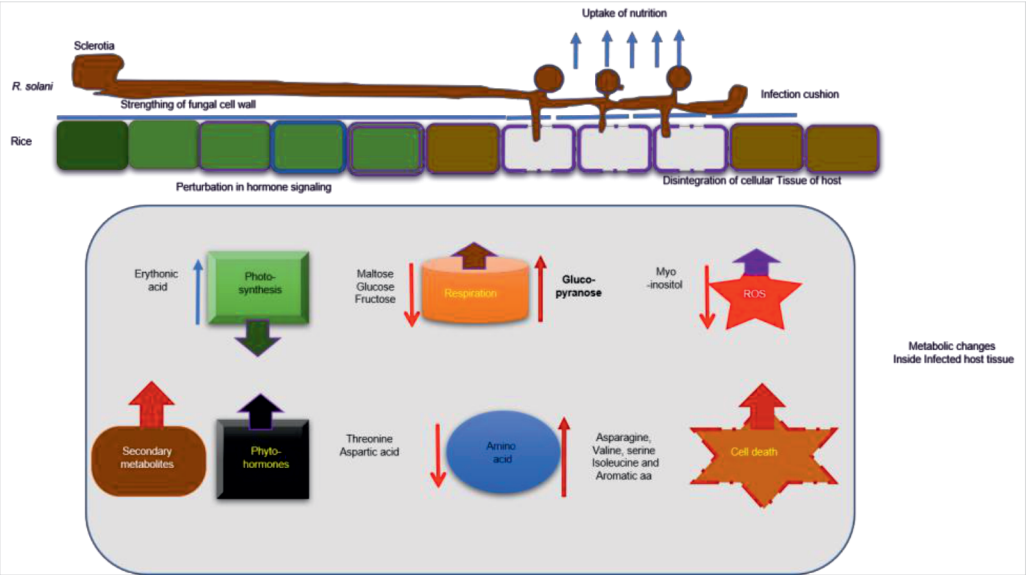

Fig 2. *R. solani*-Rice interaction during infection and biochemical interacion process. Upon inoculation *R. solani* sclerotia germinate along rice veins and form infection cushion during disease establishment. Downregulation of photosynthesis and increased respiration, secondary metabolism, phytohormones, ROS accumulation and cellular disintegration leading to host cell death were noteworthy changes during pathogenesis. Upward and downward arrow represents upregulation and downregulation of genes/processes, respectively (Adopted and modified Based on Ghosh et al. 2017)

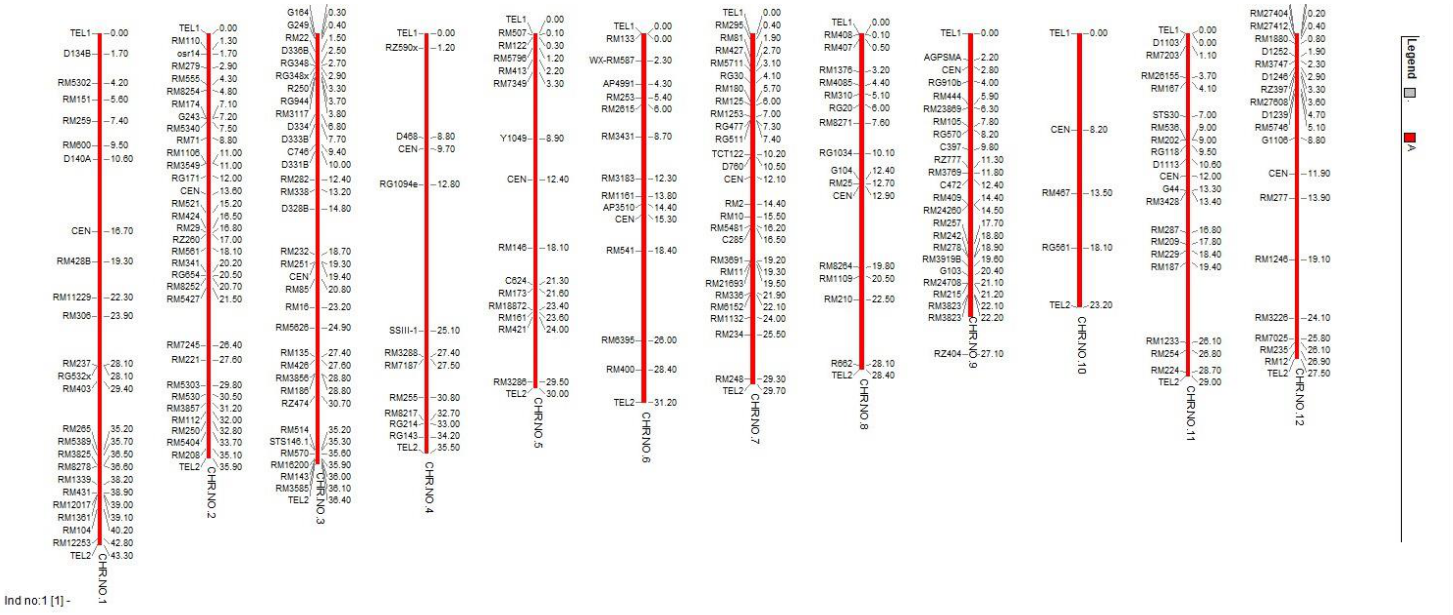

Fig 3. Location of different markers on rice chromosome for the assessment of sheath blight resistance mapping and marker assisted selection

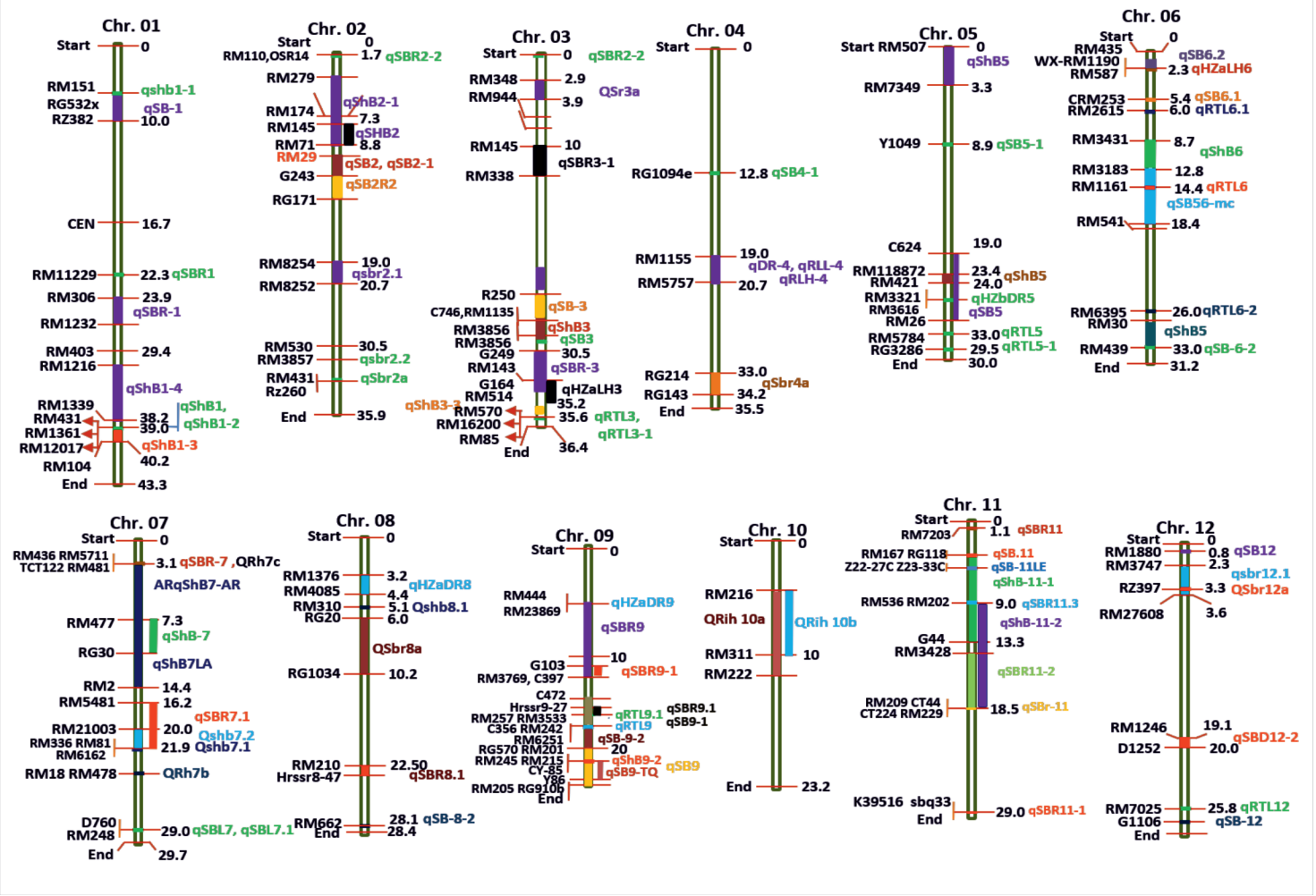

Fig 4. Location of different important QTLs for sheath blight resistance in rice

Table1. Some important chemical control of sheath blight in rice

S No.	Fungicide	Nature	Mode of Action	Formulation dosage/ha	References
1.	Carbendazim and iprodione	Systemic and contact fungicide	Inhibit mitosis in fungi and blocks the growth of the fungal	20-60 WP	Izadyar and Baradaran, 1989
2.	Benlate	Systemic fungicide	A microtubule-destabilising agent	50% EC	Premalatha Dath, 1990
3.	Topsin-M	Systemic fungicide	A microtubule-destabilising agent	70% EC	Das and Mishra, 1990
4.	Epoxiconazole	Systemic fungicide	Inhibits the metabolism of fungi cells (sterol biosynthesis inhibitor)	7.5EC	Kumar et al, 1997
5.	Metominostrobin	Systemic fungicide	By blocking mitochondrial respiration	20 %SC	Ichiba et al, 2000
6.	Thifluzamide and hexaconazole	Systemic fungicide	Inhibits succinate dehydrogenase in the tricarboxylic acid cycle./ inhibits ergosterol biosynthesis (steriod dimethylation inhibitor)	24% SC/3%SC	Sunder et al, 2003
7.	Propiconazole and tebuconazole	Systemic fungicide	Demethylation of C-14 during ergosterol biosynthesis/ inhibits spore germination, mycelial growth, and the spore production of fungi	250 EC/ 25.9% EC	Mian et al, 2004
8.	Carbendazim + mancozeb	Systemic fungicide/ contact fungicide	Acts by inhibiting development of germ tubes/ reduces the activity of enzymes in fungus which in turn reduces the energy production	50WP/75WP	Prasad et al, 2006
9.	Difenoconazole and validamycin	Systemic fungicide/systemic antibiotics	Sterol demethylation inhibitor which prevents the development of the fungus by inhibiting cell membrane ergosterol biosynthesis./ non-systemic antibiotic with fungicide action	25% EC/3% EC	Saha, 2003; Kandhari, 2007
10.	Bavistin	Systemic fungicide	Disrupt alpha tubulin assembly in mitosis of fungi and inhibits development of the germ tubes, formation of appressoria and the growth of mycelia	50%WP	Xiuguo et al 2009
11.	Azoxystrobin	Systemic fungicide	Inhibition of mitochondrial respiration in fungi.	2.08 SC/ 23%SC	Groth and Bond, 2007; Bag et al, 2016
12.	Pencycuron and azoxystrobin	Contact action/ systemic fungicide	Inhibiting mycelium growth of fungi/ inhibition of mitochondrial respiration in fungi.	250 SC (22.9% w/w)	Goswami et al, 2012
13.	Propiconazole	Systemic fungicide	Target succinate dehydrogenase complex II in respiratory chain and affect the fungal respiration	25% EC	Kumar et al, 2013; FRAC, 2017
14.	Score	Contact	Inhibits sterol demethylation, prevents the development of the fungus by inhibiting cell membrane ergosterol biosynthesis.	25% EC	Kumar et al, 2018

Table 2. Sheath Blight resistance cultivated wild rice accessions.

Sl. No.	Rice cultivar for sheath blight	References
1.	NC 678, Dudsor, Bhasamanik	Das, 1970
2.	Chin-kou-tsan, Zenith, CO.17, Dinominga, Puang Nahk 16, Baok, Toma-112, R.T.S.31, Kele Kala	Wu, 1971
3.	Lalsatkara	Roy, 1977
4.	ARC15762, ARC 18119, ARC 18275, ARC 18545	Bhaktavatsalam et al, 1978
5.	IR24, IR26, IR29, Jaya, Jaganath, Mashoori, Pankaj, Rajeshwari, Supriya, Sabari, TKM6	Rajan and Nair, 1979
6.	Nizersail, Rajasail, Tabend, Ta-poo-cho-z, Kattachambha, DA 29, ARC 5925, ARC 5943, ARC 14529, ARC 10572, ARC 10618, ARC 10836	Manian and Rao, 1979
7.	Tapoochoz, Bahagia, Laka	Crill et al, 1982
8.	Taraboli 1, Dholamula, Supkheru, Chidon	Borthakur and Addy, 1988
9.	Bharati, Rohini	Gokulapulan and Nair, 1983
10.	Bog II, Aduthurni, Chinese galendopuram, Arkavati, Saket-4, Neela, MTU-3, MTU-7, MTU-13, MTU-3642, BPT-6	Ansari et al, 1989
11.	Tetep, Tapoo-cho-z, Guyanal	Sha and Zhu, 190
12.	LSBR-5, LSBR-33	Xie et al, 1992)
13.	RU8703196, B82-761	Marchetti et al, 995; 1996
14.	KK2, Dodan, IR40 and Camor	Singh and Dodan, 1995
15.	Chingdar, As 93-1, Mairan, N–22, Panjasali, Up-52, Upland-2	Singha and Borah, 2000
16.	Yangdao 4	Pan et al, 2001
17.	TIL:455, TIL:514, TIL:642	Pinson et al, 2008
18.	MCR10277	Nelson et al, 2012
19.	WSS3, Jarjan, Nepal 555 and Nepal 8	Taguchi-Shiobara et al, 2013
20.	298 induced mutated (by gamma radiation) Pusa Basmati lines	Meena et al, 2013
21.	Moderately resistant rice cultivars, Teqing, Jasmine85, Tetep, Pecos, Azucena and Taducan	Hossain et al, 2014
22.	BPL 7-12, BML 27-1, BML 21-1 and Kajarahwa	Dubey et al, 2014
23.	Tetep and ARC10531	Yadav et al, 2015
24.	SM 801, 10–3, Ngnololasha, Wazuhophek, Gumdhan and Phougak and RP 2068-18-3-5	Dey et al, 2016
25.	Landrace Nizam shait (Resistance) Bidar local-2, Jigguvaratiga, NavaliSali, Jaddu (moderately resistance)	Lavale et al. 2018
26.	DagadDeshi	Koshariya et al. 2018
Wild rice for sheath blight resistance		
1.	*O. latifolia* (DRW 37004), *O. punctata* (DRW 32002), and *O. rufipogon* accession DRW 22017-5	Ram et al. 2008
2.	Seven Oryza spp. accessions moderately resistant, three were *O. nivara* accessions (IRGC104705, IRGC100898, and IRGC104443), *O. barthii* (IRGC100223), *O. meridionalis* (IRGC105306), O. nivara/O. sativa (IRGC100943), and *O. officinalis* (IRGC105979)	Prasad and Eizenega, 2008
3.	*O. australiensis* and *O. grandiglumis*	Shamim et al. 2014
4.	*O. nivara* accessions (IRGC81941A, CR100008 and CR100111B)	Aggarawal et al. 2019

Table 3. Identified rice ShB-QTL in different mapping populations and their association with other Traits

QTLs name	Position of QTLs on chromosome	Associated with	Resistance sources	Recipient	Genetic material/ mapping population(s)	References
qSB-2, *qSB-3*, *qSB-4*, *qSB-8*, *qSB-9*, and *qSB-12*	2–4, 8, 9, and 12	PH, HD, PH	Teqing (indica)	Lemont (tropical japonica)	255 F_4	Li et al, 1995
qSB-2, qSB-3, and qSB-7	2, 3, and 7	HD	Jasmine 85 (indica)	Lemont (tropical japonica)	F_2 clonal families	Pan et al, 1999
qSB-2 (2 years), qSB-3 (1 year), qSB-7 (1 year), qSB-9.1 (1 year), qSB-9.2 (1 year), and qSB-11 (2 years)	2, 3, 7, 9, and 11	NA	Jasmine 85 (indica)	Lemont (tropical japonica)	128 F_2 clonal families	Zou et al, 2000
qSBR-2, qSBR-3, qSBR-7, and qSBR-11	2, 3, 7, and 11	CL and ND	Zhai Ye Qing 8 (ZYQ8) (indica)	Jing Xi 17 (JX17) (japonica)	DH, 127 HD	Kunihiro et al, 2002
qSB-5 and qSB-9	5 and 9	ND	Minghui 63(indica)	Zhenshan 97 (indica)	RILs 240 lines	Han et al, 2002
Rsb 1,	5	ND	Xiangzaoxian19 indica)	4011a (indica)	1032 F2	Che et al, 2003
qSB-3 and qSB-12	3 and 12	CL	WSS2 (Tetep) (indica)	Hinohikari (japonica)	60 BC1F1	Sato et al, 2004
qSB-9 and qSB-11			Teqing (indica)	(tropical japonica)	115 F2 clonal population	Tan et al, 2005
qSB-1, qSB-2, qSB-3.1, qSB-3.2, qSB-4.1, qSB-4.2, qSB-5, qSB-6.1, qSB-6.2, qSB-7, qSB-8.1, qSB-8.2, qSB-9, qSB-10, and qSB-12	1–10 and 12	HD, PH	Teqing (indica)	(tropical japonica)	F10 and F11	Pinson et al, 2005
qSB-1, qSB-2, qSB-3, and qSB-9	1–3 and 9	PH and HD	Pecos (tropical japonica)	Rosemont (tropical japonica)	279 F2:3	Sharma et al, 2009
qSB-1 (both), qSB-2-1 (mist), qSB-2-2 (mist), SB-3-1 (both), qSB-3-3 (mist), qSB-5 (microch), qSB-6 (microch), qSB-9-1 (microch), and qSB-9-2 (both)	1–6 and 9	ND	Jasmine 85 (indica)	Lemont (tropical japonica)	250 F5 RILs	Liu et al, 2009
qSBR1-1, qSBR3-1, qSBR7-1, qSBR8-1, qSBR9-1, qSBR3-11-1, qSBR3-11-2, and qSBR3-11-3	1, 3, 7–9, and 11	NA	Tetep (indica)	HP2216 (indica)	127 RIL (F2:10), 96 varieties 192 F2 population Derived from Pusa Basmathi I/ Tetep	Channamallikarjuna et al, 2010
qSB-11LE	11		Backcross population	Lemont	Near-isogenic lines (NILs) six BC1F1 populations and one BC2F1	Zuo et al, 2011
qShB1, qShB2, qShB3, qShB5	1, 2, 3 and 5	NA	Baiyeqiu	Maybelle	double haploid (DH) population	Xu et al, 2011
qSBR1-1, qSBR1-2, qLL2-1, qSBR2-1, qHD1, qPH1-1, qPH1-2, qSBR2-2, qLL2-2, qSBR2-3, qRLL2-1, qRLL2-2, qHD2, qPH2, qPH3, qSBR4, qRLH4, qHD4, qSBR5-1, qSBR5-2, qLL5, qRLL5, qPH5 qLH6, qHD6, qSBR7, qLL7, qLH7, qRLL7, qRLH7, qHD7, qHD7, qSBR8, qLH8, qLL8, qRLL8, qRLH8, qHD8, qSBR9, qHD9 and qRLL12	1, 2, 3, 4, 5, 6, 7, 8, 9 and 12	DR, LL. LH and RRL	SRB03	HH1B	recombinant inbred line (RIL) population consisting of 121	Fu et al, 2011
qSB92 and *qSB121*	9 and 11		Teqing	Lemont	backcross introgression lines (TILs)	Wang et al, 2012
qShB9-2	9		Tetep	Pusa6B	backcross inbred lines	Pandian et al, 2012
qpht_2.1, qsbr_2.1, q sbr_2.1, qsbr_2.2, qsbr_2.2, qpht_3.1, qdth_5.1, qpht_5.1, qpht_6.1, qpht_6.2, qdth_8.1, qdth_9.1, qsbr_9.1, qsbr_9.1, qsbr_9.1, qdth_12.1, qsbr_12.1	2, 3, 5, 6, 8, 9 and 12	DTH and HT	MCR10277	Cocodrie	197 doubled-haploid lines	Nelson et al, 2012
qSB2.I-AR, qSB2.2-AR, qSB2.1-TX, qSB2.2-TX, qSB2-LA, qSB3-AR, qSB3-TX, qSB7-AR, qSB7-LA, qSB9-AR, qSB9-TX, qSB9-LA, qSBll.l-TX, qSB11.2-TX	2,3.7.9and 11	NA	Jasmine 85	Lemont	216 LIRILs	Liu et al, 2013
qSB9	9	NA	Jarjan	Koshihikari	BILs	Taguchi-Shiobara et al, 2013
qShB1 , qPH1, qPT1, qShB3, qDH3, qDH4, qShB6 , qDH6, qShB7 , qDH8, qPT9 , qShB11, qPH12	1, 3, 4, 6, 7, 8, 9 and 12	DH and PH	*O. nivara* acc. IRGC100898 and acc. IRGC104705	Bengal (PI561735)	backcross populations	Eizenga et al, 2013
qDR-1a, *qDR-1b*, *qDR-4*, *qDR-5*, *qDR-6*, *qDR-12*, *qLL-1a*, *qLL-1b* ,*qLL-3*, *qLL-9*, *qLH-1a*, *qLH-1b*, *qLH-1c*, *qLH-1d*, *qRLL-1a*, *qRLL-1b*, *qRLL-1c*, *qRLL-3*, *qRLL-4*, *qRLL-6a*, *qRLL-6b*, *qRLL-9*, *qRLH-1a*, *qRLH-1b*, *qRLH-1c*, *qRLH-2*, *qRLH-4*, *qRLH-6a*, *qRLH-6b*, *qPH-1a*, *qPH-1b*, *qPH-1c*, *qPH-3*, *qPH-7a*, *qHD-6*, and *qHD-7*	1–9 and 12		HH1B	RSB02	RIL $F_{8:11}$ population consisting of 155 lines	Liu et al, 2014
qSB-7 and qSB-9	7 and 9	HD and PH	Teqing	WLJ1	Advanced backcrossed lines	Chen et al, 2014
TAC1TQ and *qSB9TQ*	1 and 9	TA and GY	IL55	IR24	NILs	Zuo et al, 2014
qSB1-1^{HJX74} and qSB11^{HJX74}			Amol3(sona)	HuaJingXian74	chromosome segment substitution lines	Zhu et al, 2014
Qsb-1, qsb-2, qsb5-1 and qsb5-2	1, 2 and 5	NA	IR28	Dagundao	157 RIL Lines	Yang et al, 2015
qshb7.3 and qshb9.2	7 and 9		ARC10531	BPT-5204	mapping populations (F_2 and BC_1F_2)	Yadav et al, 2015
qSBD-1, qSBD-3-1, qSBD-3-2, qSBD-7, qSBD-11-1, qSBD-11-2, qSBD-12-1 and qSBD-12-2	1,3, 7, 11 and 12	LH, DR and PLH	Lemont	Yangdao 4	F_2 population	Wen et al, 2015
qHNPH1, qHZaLH1, qHNDR1, qHNPH2, qHNLH3, qHZaLH3, qHNPH3, qHNLH4, qHZbPH4, qHNPH5, qHNPH4, qHZbLH5, qHZbDR5, qHZaLH6, qHNPH6, qHNDR6, qHNLH6, qHZaDR8, qHNPH8, qHZaDR9, qHZbDR9, qHNPH9, qHZaPH10, qHZbPH10, qHNDR11 and qHNDR12	1, 2, 3, 4, 5, 6, 8, 9, 10, 11, 12	LH, DR	CJ06/TN-1	TN-1/CJ06	doubled haploid (DH) population	Zeng et al, 2015
T-1 and T-4 (on chr1), T-3 (on chr2), T-3 (on chr4), T-3 (on chr-5), T-3(2.5) (on chr 6), T-3(2,5 on chr 7), T-3, T-4, T-3(2.5) and T-5 (on chr 12)	1, 3, 4, 5, 6, 7 and 12	LW	Dagad Deshi	Danteshwari	Recombinant inbred line (RIL) population consisting of 122 lines	Koshariya et al, 2018
qSB-3 and qSB-6	3 and 6	DS	genome-wide association study (GWAS) of SB resistance using 299 varieties from the rice diversity panel 1 (RDP1		299 varieties	Chen et al, 2019
qShB1-1, qShB1-2, qShB1-3, qShB1-4, qShB3, qShB4, qShB6-1, qShB6-2 and qShB8	1, 4, 3, 6 and 8	PH and HD	AR-2001–1135-01, and RR9/ MS-1996-9, and RR20	AR-2001–1135-01, and RR9/ MS-1996-9, and RR20	F8 recombinant inbred line populations	Goad et al, 2020

Table 4. Transgenic rice developed against sheath blight by gene transfer

S. No.	Gene(s) transformed	Source of gene(s)	Features of concerned gene(s)	Transgenic rice developed	References
1.	*Bar*	Streptomyces hygroscopicus	Herbicide tolerance gene, reduced ShB infection when plants spayed with bialaphos or phosphinothricin	Yamahoushi, Nipponbare	Uchimiay et al, 1993
2.	*Chi 11*	Rice	Chitinase-containing rice genomic DNA (1.1 kb)	Chinsurah Boro II	Lin et al, 1995
3.	*TLP-D34*	Rice	Rice thamatin-like protein, a member of PR-5 group	Chinsurah Boro II, IR72, IR1500	Datta et al 1999b
4.	*RC 7*	Rice	Rice chitinase	IR64, IR72, IR688998, MH63 Chinsurah Boro II	Datta et al, 2000; 2001
5.	*pinA, pinB*	Wheat	Structural protein from *Triticum aestivum*	M202	Krishnamurthy et al, 2001
6.	*Chi 11*	Rice	Rice chitinase	Swarna	Baisakh et al, 2001
7.	*Chi, Xa21, Bt*	Rice	Chitinase, receptor-like kinase, and *Bt* toxin	IR72	Datta et al, 2002
8.	*MODI, RCH0*	Maize and rice	Modified maize ribosome-inactivating protein gene and basic chitinase	Kenfong	Kim et al, 2003
9.	*Chi11*	Rice	Chitinase	Pusa Basmati 1	Kumar et al, 2003
10.	*Chi 11*	Rice	Rice chitinase	Pusa Basmati 1	Sridevi et al, 2003
11.	*RC 24*	Rice	chitinase gene	zhongda 2,	Yuan et al, 2004
12.	ech42, nag70 and gluc78	Trichoderma atroviride	ndochitinase, exochitinase and exo-1,3-b-glucanase	Ishikari-shiroge	Liu et al, 2004
13.	*Chi 11, tip*	Rice	Enhanced resistance to both ShB and ShR	ADT38, ASD16, IR50 Pusa Basmati 1	Kalpana et al, 2006
14.	*Ace-AMP1*	Allium cepa	A non-lipid transfer protein with antimicrobial property isolated from Allium cepa showed enhanced resistance against ShB, Blast and BLB	Pusa Basmati 1	Patkar and Chattoo, 2006
15.	*RC 7*	Rice	Rice chitinase	Pusa Basmati 1, White Ponni ADT38, Co43	Nandakumar et al, 2007
16.	*Chi 11, tlp, Xa21*	Rice	Rice chitinase, thaumatin-like protein and serine-threonine kinase enhanced resistance to both ShB and BLB	ASD16, ADT38, IR72, IR64, White Ponni	Maruthasalam et al, 2007
17.	*Chi 11, b-1,3-glucanase*	Rice and tobacco	Rice chitinase and tobacco b-1,3-glucanase	Pusa Basmati 1	Sridevi et al, 2008
18.	*Chi 11*	Rice	Rice chitinase	Pusa Basmati 1	Sripriya et al, 2008
19.	*Rs-AFP2*	Dahlia merckii and *Raphanus sativus*	A defensin gene from *Raphanus sativus*	Pusa Basmati 1	Jha and Chattoo, 2009a
20.	*Dm-AMP1*	Dahlia merckii	A defense gene from *Dalia merkii*	Pusa Basmati 2	Jha et al, 2009
21.	*McCHIT*	Momordica charantia	A class I chitinase gene of bitter melon	JinHui35	Li et al, 2009
22.	*Cht 42*	Trichoderma virens	A chitinase gene from *Trichoderma* spp.	Pusa Basmati 1	Shah et al, 2009

23.	*Dm-AMP1, Rs-AFP2*	Raphanus sativus	Defensin genes from *D. merkii* and *R. sativus*, respectively	Pusa Basmati 1	Jha and Chattoo, 2010
24.	chi11 and ap24	Rice and tobacco	Chitinase and osmotin	Pusa Basmati 1	Rao et al, 2011
25.	OsWRKY30	Rice	Transcription factor gene	Xiushui 11	Peng et al, 2012
26.	*tlpD34, PR5, chi11, PR3*	Rice	Thaumatinlike protein gene (*tlpD*34, PR5) combination with the chitinase gene (*chi*11, PR3)	White Ponni	Shah et al, 2013
27.	*ACS2 (1-aminocyclopropane-1-carboxylic acid synthase*	Rice	Rice ACS2 (1-aminocyclopropane-1-carboxylic acid synthase, a key enzyme of ET biosynthesis)	Kitaake	Helliwell et al, 2013
28.	*oxalate oxidase 4 (Osoxo4)*	Rice	Overexpression of *oxalate oxidase 4* (*Osoxo4*)	Pusa Sugandhi II	Molla et al, 2013
29.	*BjNPR1*	*Brassica juncea*	Nonexpressor of pathogenesis-related gene 1	Chaitanya and Samba Mahsuri	Sadumpati et al, 2013
30.	*chitinase gene (RCH10) β1,3-glucanase gene (AGLU1)*	Rice and alfalfa	Rice basic chitinase gene (*RCH10*) and the alfalfa β1,3-glucanase gene (*AGLU1*)	Taipei 309	Mao et al, 2014
31.	*OsPGIP1*	Rice	Over expressed OsPGIP1	Zhonghua 11	Wang et al, 2015
32.	AtNPR1	*Arabidopsis thaliana*	Nonexpressor of pathogenesis-related gene 1	Pusa Sugandhi-2	Molla et al, 2016
33.	*OsWRKY80*	Rice	Transcription factor	Xiushui 11	Peng et al, 2016
34.	*OsOXO4* and *OsCHI11*	Rice	Rice oxalate oxidase 4 and rice chitinase 11	BR-29	Karmakar et al, 2016
35.	*OsOSM1*	Rice	upregulated by jasmonic acid (JA)	Xudao3	Xue et al, 2016
36.	*chi11*	Rice	Chitinase gene	ASD16	Rajesh et al, 2016
37.	*mASAL*	*Allium sativum*	mannose binding *Allium sativum* leaf agglutinin	IR64	Ghosh et al, 2016
38.	*OsCHI11 and AtNPR1*	*Arabidopsis thaliana* and rice	Chitinase and Arabidopsis NPR1	Jaldi-13	Karmakar et al, 2017
39.	*LOC_Os11g47510*	Rice	Tatep Novel Chitinase Gene	Taipei 309	Richa et al, 2017
40.	Oxalate decarboxylase protein Bacisubin	*B. subtilis* strain BS-916	Oxalate decarboxylase protein Bacisubin from *Bacillus subtilis*	Nipponbare	Qi et al, 2017
41.	OsASR2	Rice	Abscisic acid stress and ripening 2 protein	IRBB13 and Zhonghua 11	Li et al. (2018)
42.	*AG1IA_04727*	Pectin induced *R. solani* Wgl-2 RNA	Polygalacturonase (PG),	Taipei 309	Rao et al. (2019)
43.	*Chitinase11*	Rice	Chitinase	Pusa Basmati1	Sai et al. (2019)
44.	*OsBSR2*	Rice	Cytochrome P450 protein (CYP78A family)	Nipponbare	Maeda et al. (2019)
45.	*OsGSTU5* (*Os09g20220*	Rice	a tau class GST Glutathione-*S*-transferase	Nipponbare	Tiwari et a. (2020)

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