Understanding sheath blight resistance in rice: the road behind and the road ahead

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Summary

Rice sheath blight disease, caused by the basidiomycetous necrotroph Rhizoctonia solani, became one of the major threats to the rice cultivation worldwide, especially after the adoption of high-yielding varieties. The pathogen is challenging to manage because of its extensively broad host range and high genetic variability and also due to the inability to find any satisfactory level of natural resistance from the available rice germplasm. It is high time to find remedies to combat the pathogen for reducing rice yield losses and subsequently to minimize the threat to global food security. The development of genetic resistance is one of the alternative means to avoid the use of hazardous chemical fungicides. This review mainly focuses on the effort of better understanding the host–pathogen relationship, finding the gene loci/markers imparting resistance response and modifying the host genome through transgenic development. The latest development and trend in the R. solani–rice pathosystem research with gap analysis are provided.

Introduction

Future rice demand is speculated to be higher due to the increasing trend in the consumption of rice and the rising world population. Total growth in rice consumption may even outplay population growth if the recent uptrend in per capita consumption continues in the big three countries (China, India and Indonesia) (Mohanty, 2013). The situation is more aggravated by the shrinkage of agricultural land and the annual loss of crops worth billions of dollars due to different diseases caused by pathogens.

Sheath blight (ShB), caused by Rhizoctonia solani Kühn [teleomorph – Thanatephorus cucumeris Frank (Donk)], is one of the highly destructive diseases of rice, considered as a globally significant one, second-most prevalent to the blast disease. The estimated yield reduction from sheath blight ranged from 20% to 42% in artificially inoculated field plots (Cu et al., 1996). The use of high doses of nitrogen fertilizer and the introduction of semi-dwarf high-yielding varieties (HYV) caused a sharp rise in the incidence of sheath blight disease (Savary et al., 1995). Higher crop densities and resultant humid canopies have long been advocated as important factors which favour increasing sheath blight incidence (Kannayia and Prasad, 1983).

The pathogen is generally soil-waterborne, infectious to a broad range of plants from ~32 taxonomic families (Gangopadhyay and Chakrabarti, 1982). The pathogen is highly variable genetically; a total of 14 different anastomosis groups (AG) have been assigned to the isolates of R. solani (Carling et al., 2002a; Carling et al., 2002b). First, 13 groups were named as AG1 to AG13, whereas the 14th group, AGB1, is a bridging isolate. Based on the DNA-sequence homology and morphology of sclerotia, R. solani AG1 isolates have been further subdivided into three subgroups, including IA, IB and IC (Sneh et al., 1991). However, the existence of other subgroups/intraspecific groups like ID, IE and IF was proposed and debated based on isozyme comparison, rDNA-ITS sequence RFLP and fatty acid analysis (Liu and Sinclair, 1993; Priyatmojo et al., 2001). The widely accepted view is that the R. solani AG1-IA is the causal organism of sheath blight disease in rice. In addition to the broad host range and variability of the pathogen, the main obstacle in managing sheath blight disease is the lack of an identified germplasm with an adequate level of resistance for using in the resistance breeding programme (Bonman et al., 1992).

Characteristic symptoms of the disease are water-soaked, spherical to oval-shaped or irregularly elongated discoloured, greyish to light brownish lesions with brown margin on the leaf sheath and blades (Figure 1). The disease is also vernacularly called ‘snake skin disease’ as the symptoms sometimes resemble the skin of a snake. A typical inoculum of the disease is either sclerotia or runner hyphae from the infected plants. In a rare occasion, basidiospores act as inoculum. These sclerotia may remain dormant for over the years in the soil and stubbles and can re-infect healthy rice plants in the subsequent crop season (Figure 2). By virtue of their buoyancy, sclerotia can float long distances in the field with irrigation water and aid in the spreading of the disease. The pathogen penetrates the plant by means of lobate appressoria or infection cushions or both (Marshall and Rush, 1980). Infection cushions are convoluted...
hyphal aggregates developed from the runner hyphae of R. solani (Molla et al., 2013). For infection, direct cuticular penetration mediated by infection cushion is a frequent method, whereas stomatal penetration mediated by lobate appressoria is a less frequent method (Marshall and Rush, 1980). A complete disease cycle is schematically depicted (Figure 2).

Control of the disease is highly dependent on chemical fungicide, and cultural practices since resistance breeding remain unsuccessful till the date owing to the inability to identify any resistance resources from the available rice germplasm. Moreover, high genetic variability, extensive host compatibility and the ability of the pathogen to survive form one crop season to next by forming dormant sclerotia made additional difficulties in controlling the pathogen. Review articles on cultural, chemical and biological control have been published (Singh et al., 2019; Yellareddygari et al., 2014; Taheri and Tarighi, 2011).

Plants have evolved several strategies to defend themselves from pathogen attack. Complex molecular mechanisms are involved in executing those strategies. A great deal of previous research elucidated the plant’s molecular defence apparatus for many diseases (Andersen et al., 2018; Hammond-Kosack and Parker, 2003; Staskawicz et al., 1995). Despite its economic importance, the molecular basis of rice sheath blight disease and rice–R. solani interaction has not been studied well (Molla et al., 2019). However, recent advances in molecular, biotechnological and sequencing technologies have led the researcher to focus on investigating the genetics of ShB tolerance, decoding molecular features and studying pathogenesis mechanisms. Although recent review articles discussing management practices and crop improvement for sheath blight resistance (Singh et al., 2019; Yellareddygari et al., 2014) have been written, no comprehensive review has been published focusing on both genetics and molecular biology of the disease.

In this current review, we report a comprehensive up-to-date synthesis on the recent advancement of the understanding of rice and R. solani interaction in the postgenomics era. The progress made regarding the identification of genetic regions, quantitative trait loci (QTLs) and molecular markers associated with sheath blight resistance has also been analytically reviewed. We also provide a thorough and critical discussion on the deployment of disease resistance genes from rice and nonrice sources for developing sheath blight-resistant transgenic rice.

Molecular interplay between Rice and R. solani

On pathogen attack, plants protect themselves by activating highly complex interacting signalling pathways. Salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) are the three vital role players in most of the pathogen-responsive signalling pathways (Glazebrook, 2005; Kunkel and Brooks, 2002; Nagarajkumar et al., 2005). Although Rhizoctonia solani is widely described as a necrotrophic fungal pathogen, the possibility of the existence of a combination of necrotrophic and hemibiotrophic behaviour on its compatible host cannot be excluded (Garcia et al., 2006). In a significant advance in 2018, Kouzai et al. suggested a hemibiotrophic nature of R. solani. In general, SA-mediated signalling induces resistance against biotrophic pathogens, while JA-mediated signalling induces resistance against the necrotrophs (Browse, 2009; Glazebrook, 2005; Nagarajkumar et al., 2005; Oka et al., 2013). The pathogen R. solani uses diverse strategies to successfully colonize and infect rice plant, while in turn rice plants activate different signalling pathways and produce antimicrobial molecules to fight against them (Figure 3). We discuss the molecular interplay section in three distinct segments, a perspective from the pathogen, an angle from the host plant and the chemical battle between host and pathogen.

Perspective from the pathogen

Effectors

Secreted fungal effector molecules favour fungal colonization on host plants through subduing plant defence (Lo Presti et al., 2015). Three potential secreted effectors (viz. cytochrome C oxidase assembly protein CtaG/cox11 domain, glycosyltransferase GT family 2 domain and peptidase inhibitor I9 domain) of R. solani AG1-1A were validated that could trigger crop defence responses in the form of cell death phenotype (Zheng et al., 2013). Similarly, inhibitor I9 containing proteins have been...
abundantly detected in the predicted secretome of ShB pathogen (Anderson et al., 2017). In a recent genomic analysis of two virulent Indian strains of *R. solani* AG1-1A, several putative candidate effectors such as histone acetyltransferase, histone deacetylase inhibitor, MDR transporter, O-antigen biosynthesis protein, O-methyl sterigmatocystin oxidoreductase, polygalacturonase and pectin lyase have been predicted (Ghosh et al., 2019). A total of 44 putative effector transcripts were found to be up-regulated in *R. solani* AG1-1A from different hosts, including rice (Xia et al., 2017). More specifically, up-regulation of two putative effectors, AG1IA_03245, a kinase-domain-containing protein, and AG1IA_08303, a peroxidase, has been observed in the pathogen from rice (Xia et al., 2017).

Like many other fungi, *R. solani* AG1-1A secretes oxalate as one of its virulence factors; and virulent isolates secrete more oxalate than less virulent one (Nagarajakumar et al., 2005). The ability of transgenic rice plants overexpressing oxalate oxidase to break down oxalate has been proved to be effective to enhance resistance against the pathogen (Molla et al., 2013), indicating the involvement of oxalate and oxalate oxidase in *R. solani* pathogenesis and rice plant defence, respectively. Recently, an RNA seq analysis identified *R. solani* genes that are induced during infection and post-penetration phase in rice plants (Ghosh et al., 2018). Few putative effector genes were also found to be up-regulated in the study. Two polygalacturonase genes (RsPG3 and RsPG4) have been cloned from *R. solani* and demonstrated to induce rice sheath tissue necrosis and subsequent release of reducing sugar, indicative of their role as one of the important virulence factors of the pathogen (Chen et al., 2017). Recently, a polygalacturonase (AG1IA_04727) gene has been shown to play a significant role in *R. solani* pathogenesis (Rao et al., 2019).

Unlike other fungal pathogens, genetic transformation of *R. solani* is not established mostly because of its multinucleate nature. Therefore, identification of virulence genes of *R. solani* remains unsuccessful to date as genetic approaches are challenging to apply.

According to an interesting finding by Fujikawa et al. (2012), *R. solani* uses a stealthy tactic to avoid the plant’s innate immunity. For a successful infection, *R. solani* uses α-1, 3-glucan to mask its cell wall chitin from being recognized by PRR (pattern recognition receptor) (Fujikawa et al., 2012). Therefore, transgenic expression of α-1,3-glucanase in rice plants to degrade the α-1, 3-glucan mask may be proved as an efficient strategy to expose fungal chitin PAMP (pathogen-associated molecular pattern) and to subsequently activate plant’s innate immunity.

**Secondary messenger**

Heteromeric G protein, made up of α, β, and γ subunits, is an important signalling component which plays a significant role in the virulence and pathogenesis of filamentous fungi (Regenfelder et al., 1997). Interestingly, a study has demonstrated that the disruption of *R. solani* gene *Rga1*, encoding a G protein α subunit, negatively affects the growth, pathogenicity and the sclerotia forming ability (Charoenopharath et al., 2008). Nine putative G protein subunits, 13 G protein-coupled receptors like genes, 22 homologous genes of MAPK pathway, 15 genes of the calcium–calcineurin pathway and 5 genes of the cAMP pathway have been predicted from *R. solani* AG1-1A genome sequence (Zheng et al., 2013) pointing out that those secondary signalling molecules may have important role in *R. solani* pathogenesis. In a global protein–protein interaction network model study, the interaction of *R. solani* Gβ subunit with Gγ subunit, RACK1 homolog (WD domain containing) and with a molecular chaperone (T complex protein) has been made evident (Lei et al., 2014). Gβ subunit alone or synergistically with Gγ subunit plays a vital role in the regulation of MAP kinase, adenylate cyclase (responsible for cAMP synthesis) and ion channels (Li et al., 2007), each of which in turn may play an essential role in the pathogenesis of *R. solani*. Interestingly, the WD domain and Gβ repeat domain-containing protein from rice also have been predicted to play a role in the ShB resistance (Silva et al., 2012). Three protein genes, viz. RS_P1 (panvinulin subfamily of peptide prolyl cis/trans isomerase (PPIC) protein), RS_P3 (NFU like protein) and RS_P4 (V-SNARE like protein), have been shown to exhibit significant up-regulation during in planta growth of *R. solani* in rice (Ghosh et al., 2014).

**Application of Omics technologies to understand the pathogenesis**

Omits, the collective term which broadly includes genomics, transcriptomics and proteomics technology, is used to study the role of various molecules and their relationship and actions in a specific biological and disease processes. Because of the rapid development in high-throughput sequencing technology and its cost-effectiveness, omics (genomics, RNA seq/transcriptomics, and miRNAseq) became popular to gain insight into a particular interaction study. Zheng et al. (2013) reported the first draft of the *R. solani* AG1-1A genome (36.94-Mb) sequence and annotated 6156 genes, including 257 genes specific for host–pathogen interaction. They identified 25 candidate effector genes from their transcriptomic study involving samples collected from 18- to 72-h infection stages. Recently, the draft genome sequence of a Malaysian isolate has been published (Nadarajah et al., 2017). However, a smaller genome size of about 28.93 Mb is reported (Nadarajah et al., 2017). Comparative genomics between the different sequenced genome of *R. solani* anastomosis groups further revealed detail about AG1-1A specific genes and putative virulence factor/effector genes (Ghosh et al., 2014; Hane et al., 2014). Similarly, a study of whole-genome sequencing of 13 inbred rice lines was performed to identify SNPs and candidate genes for sheath blight resistance (Silva et al., 2012). The study identified more than 200 candidate genes containing a total of 333 nonsynonymous ‘single nucleotide polymorphisms’ (SNPs) between the SHB-susceptible and SHB-resistant genotypes. Transcriptome profile of *R. solani* isolated from infected sheath enlisted the putative pathogenesis genes (Ghosh et al., 2018). Interestingly, alternative splicing of important pathogenic genes of *R. solani* AG1-1A has been documented to play significant roles during infection of rice, soybean and corn plants as evidenced by comparative ecotype transcriptome analysis (Xia et al., 2017). In rice–*R. solani* interaction, differentially expressed genes (DEGs) were found to be significantly involved in aldehyde dehydrogenase [(NAD(P)H]-activity, acetaldehyde catabolism and in the extracellular region, which differed greatly from the DEGs in maize–*R. solani* and soybean–*R. solani* interaction (Xia et al., 2017). The finding of Xia et al. (2017) might be helpful to find the answer to the age-old question – what makes *R. solani* infectious to almost every crop plants.

Construction of small RNA libraries from the fungal hyphae and their sequencing enabled identification of a total of about 177 ‘miRNA-like small RNAs’ (miRNAs), which includes 15 predicted candidate pathogenic novel miRNAs (Lin et al., 2016).
For analysis and comparison of genome and transcriptome, a user-friendly database RS1ADB has been developed for *R. solani* AG1-1A, which is accessible through the URL – http://geneden.ovoweb.ticp.net:81/rsia/index.php (Chen et al., 2016).

An angle from the host plant

**Involvement of several signalling systems: a coordination**

The involvement of JA, LOX (lipoxygenase), and octadecanoid signal transduction pathways in resistance response to *R. solani* was demonstrated (Taheri and Tariigh, 2010; Vidhyasekararan et al., 1997). A JA-deficient rice mutant, *hebiba*, exhibited enhanced susceptibility to the sheath blight disease (Taheri and Tariigh, 2010), suggesting the involvement of JA in resistance response. The transcript of LOX, an important gene in the JA biosynthetic pathway, is elevated on *R. solani*-infected rice plant (Sayari et al., 2014; Taheri and Höfte, 2006). *WRKY30* overexpression in transgenic rice plants has been shown to enhance ShB tolerance by activating jasmonate biosynthesis-related genes and subsequent increase of endogenous JA accumulation (Peng et al., 2012). Similarly, *WRKY80* was found to confer ShB tolerance through the activation of *WRKY4* and JA/Et signalling pathway (Peng et al., 2016). Recent studies indicated the involvement of JA and phenylpropanoid metabolism in ShB disease resistance (Ghosh et al., 2017; Zhang et al., 2017).

Increasing pieces of evidence suggest that ethylene may be a significant role player in disease response signalling against *R. solani*. Transgenic rice plants overexpressing an ethylene biosynthetic gene have been demonstrated to increase the expression of PR1b and PR5 and subsequent resistance to *R. solani* (Helliwell et al., 2013). The increased resistance may also be attributed to the effect of cyanide, a toxic by-product of the ethylene biosynthesis pathway (Peiser et al., 1984). An ethylene-insensitive mutant (*sickle*) exhibited high susceptibility to the pathogen *R. solani* AG8, whereas overexpression of ethylene response factors (ERFs) in *Medicago* enhanced resistance to the pathogen (Anderson et al., 2010; Varma Penmetsa et al., 2008).

Contrastingly, as an indication of involvement of the SA-mediated pathway, higher expression of *PR1b* gene, a SAR marker, and *PBZ1* gene was found to be related to the ShB disease development in rice (Zhao et al., 2008). Similarly, the *PR5* gene (*TLPD-34*) was induced in rice plants infected with *R. solani* (Velazhahan et al., 1998). It is a well-established notion that the PR genes become activated as a result of the induction of SAR pathway. The expression levels of six PR genes, viz. PR-3, PR-5, PR-9, PR-10, PR-12 and PR-13, and PAL gene were found to be induced in rice at different time points of *R. solani* infection, indicating the involvement of SAR activation (Sayari et al., 2014).

In a previous study, we demonstrated the elevation of the *RC24*, a PR gene and the PAL, a SAR marker gene, in ShB-infected transgenic rice plants, further supporting the possible involvement of SAR pathway in rice-*R. solani* interaction (Molla et al., 2013). It has also been showed that the activation of mornilactone A ‘A’ (a rice phytoalexin) biosynthesis, induction of PR proteins (β-1,3-glucanases and exochitinases) and increasing activity of the phenylalanine ammonia lyase (PAL) in rice plants act as a multicomponent coordinated defence response against *R. solani* (Bera and Purkayastha, 1999).

External application of SA and overexpression of a SAR regulatory *AtNPR1* gene (Karmakar et al., 2019; Karmakar et al., 2017; Molla et al., 2016) have conferred resistance to the sheath blight pathogen further proposing the involvement of SA. Studies have furnished evidence on the existence of similar kind of disease resistance signalling pathway in rice as the Arabidopsis NPR1 pathway (Chern et al., 2014; Chern et al., 2001; Yuan et al., 2007). Interestingly, a most recent study showed that pretreatment with SA induces sheath blight resistance (Kouzai et al., 2018). Further, rice plant expressing SA hydroylase (NahG) gene, able to cause SA deficiency, were found to be more ShB-susceptible than the wild-type plants, indicating the involvement of SA-dependent immunity (Kouzai et al., 2018). All those recent studies are pointing towards the involvement of SA-mediated defence response against *R. solani*.

Interestingly, along with some other gibberelin (GA) receptor kinase, the GA receptor GID1L2 (Os09g28690.1) was highly up-regulated in resistant rice cultivar compared to in susceptible one (Yuan et al., 2018).

**Secondary messengers**

Unfortunately, the direct involvement of well-known secondary messenger signalling molecules has not been deduced so far in rice–*R. solani* interaction. A recent study reported that upon ShB infection, the rice Gf8 subunit (RGB1) gets induced but not Gf7 subunit (RRG1) (Swain et al., 2019). The study also reported improved sheath blight tolerance in transgenic rice plants overexpressing the RGB1 gene. The involvement of secondary messengers of rice in immune responses to *R. solani* has been reported in some comparative transcriptome and proteomic studies (Karmakar et al., 2019; Zhang et al., 2017). Several calcium/calmodulins signalling pathway genes (*OsCPK10*, *OsCML16*, *OsCML26*, *OsCML27*, *OsCML31*, *OsCam1-1*) were differentially regulated at different time points after infection in response to ShB infection (Zhang et al., 2017) indicating the involvement of the secondary messengers, Ca2+ and calmodulin. Wall-associated kinases (WAKs) are known to bind several types of pectin, including oligomer generated by pathogen-mediated cell wall degradation, and involved in pathogen-responsive signal transduction pathways (Kohorn and Kohorn, 2012). Six different WAK proteins were up-regulated in reaction to *R. solani* infection in resistant cultivar (Yuan et al., 2018). The full-length protein product of *OsWAK91* seemed to be related to ShB resistance (Al-Bader et al., 2019). A recent proteomic study revealed that protein 14–3–3GF14f is highly stimulated in infected transgenic rice plants (Karmakar et al., 2019). 14-3-3 proteins are vital in many signal transduction pathways since they act as an adapter for phosphorylated proteins (Chen et al., 2006). Another important secondary messenger, MAP kinase 6 (MPK6), was found to be highly accumulated in transgenic rice plants in response to ShB disease (Karmakar et al., 2019).

**Metabolic alteration in the host**

For interaction study, another critical area is the investigation of adaptive alterations in the metabolic profile of rice plants upon *R. solani* infection. Studies have been conducted to reveal the involvement of metabolic changes during the period of rice response to the *R. solani* attack, the majority of which were focused on carbohydrate metabolism. For example, a study has shown that the enzymes of the glycolytic pathway and pentose phosphate pathway activated to a greater extent in response to *R. solani* in resistant line than a susceptible line (Danson et al., 2000). The association of activation of the glycolytic pathway, oxidative pentose phosphate pathway (OPPP), secondary metabolism, tricarboxylic acid cycle (TCA) cycle and reduced level of starch synthesis in rice plant infected with *R. solani* has been
demonstrated (Mutuku and Nose, 2010). Further studies from the same group provided clues regarding the involvement of glycolytic regulation as a response to R. solani infection. Two important regulatory enzymes (pyrophosphate-fructose-6-phosphate phosphotransferase and 6-phosphofructokinase) of the glycolytic pathway have been activated on pathogen infection (Mutuku and Nose, 2012a). In addition, all glycolytic metabolite contents, activities of all glycolytic enzymes and activities of transketolase (TK), PAL and peroxidase have been shown to increase in R. solani-infected resistant rice plants suggesting the interconnection of glycolytic regulation with phenylpropanoid pathway in response to pathogen attack (Mutuku and Nose, 2012b). These results indicated that the up-regulation of glycolysis in R. solani-infected rice plants led to an increased synthesis of glyceraldehyde-3-phosphate (GAP), which might be utilized in the generation of erythrose-4-phosphate (E-4-P) by TK. The subsequent combination of phosphoenolpyruvate (PEP) and E-4-P may lead to the enhanced production of lignin as the first line of defence response to R. solani (Mutuku and Nose, 2012b).

Similarly, another metabolite profiling study also showed the accumulation of glycolysis and TCA cycle intermediates (Ghosh et al., 2017). A recent study indicated the involvement of canavanine, a non-protein amino acid, as a response of rice plants to R. solani infection (Suharti et al., 2017). Besides, R. solani-infected rice plants also exhibited an accumulation of proteinogenic amino acids (Ghosh et al., 2017; Suharti et al., 2016). In a proteomic approach, the induction of enzyme GAPDH in R. solani inoculated resistant rice line further supports the involvement of the glycolytic pathway in the defence response (Lee et al., 2006). The induction of 3-β-hydroxysteroid dehydrogenase/isomerase (3-β-HSD) has been observed in the same study, indicating the defensive role of steroids.

**Omics studies to understand host defence**

Omics offers an attractive tool to get an overview of the collective response of genes in a host plant and how it reacts to a pathogen attack. Comparative transcriptomics has been performed to understand the extent and pattern of the differentially expressed genes (DEGs) between SHB-susceptible (Lemont) and tolerant (Teqing) rice cultivars, wherein 4806 DEGs were identified (Zhang et al., 2017). Metabolic alterations and responsive transcripts during susceptible rice–R. solani interaction have been unravelled in a recent metabolomic and transcriptomic study (Ghosh et al., 2017). Most recently, Karmakar et al. (2019) investigated the proteometabolic alterations of AtnPnp1-transgenic rice lines in response to SHB infection. On comparing the proteome and metabolome of rice lines before and after the SHB infection, the study detected a total of 38 differentially expressed proteins and 40 differentially accumulated metabolites (Karmakar et al., 2019). From both the proteome and metabolome profiles obtained, it is indicative that energy and carbohydrate metabolism is the primary area where the plant adjusts themselves on R. solani infestation (Karmakar et al., 2019). A similar result was obtained by Suharti et al. (2016) in a metabolomics study. Lin et al. (2016) predicted 23 putative candidate rice miRNAs possibly involved in defence against R. solani.

**The chemical battle between Rice and R. solani**

Always there is a tough fight between pathogen and its host plant goes on to win over each other. After sensing the attack of the pathogen, plants produce several defence-related chemicals such as phytoalexin resultant of secondary metabolism. Rice plants infected with R. solani have been reported to produce phytoalexins, phytocassane A, B, C, D and momilactone A and B (Bera and Purkayastha, 1999; Koga et al., 1995). A biZAP transcription factor, TGA1 and signalling kinases like MKK4, MPK3 and MPK6 were found to be induced by fungal elicitor (chitin oligomer) to produce phytoalexin momilactone and phytocassane (Kishi-Kaboshi et al., 2010a; Kishi-Kaboshi et al., 2010b; Okada et al., 2009). A picture could be visualized from those studies that the breakdown product of fungal cell wall component chitin acts as an elicitor to induce signalling pathways, which in turn could regulate the synthesis of rice diterpene phytoalexins. Chlorogenic acid, an ester related to polyphenol family and lignin biosynthetic pathway intermediate, has been found to be accumulated in a higher amount in the tolerant cultivar than susceptible one (Suharti et al., 2016). Similarly, sakuranetin, a flavonoid phytoalexin, has been found to exhibit antifungal activity against R. solani (Park et al., 2014).

It is a matter of apprehension that fungus is also rapidly developing various methods to detoxify phytoalexin. For example, R. solani has been reported to detoxify the cruciferous phytoalexin camalexin into a less toxic form (Pedras and Ahiahonu, 2005). Interestingly, a recent study showed that R. solani can detoxify the rice phytoalexin sakuranetin to another less toxic chemicals such as sakuranetin-4′-O-β-d-xylopyranoside, naringenin and naringenin-7-O-β-d-xylopyranoside (Katsumata et al., 2018). The study also revealed that detoxification by xylosylation is unique to R. solani (Katsumata et al., 2018).

On the other hand, the pathogen R. solani secretes host-specific RS toxin as one of its weapons to counteract rice defence. RS toxin, composed of glucose, mannose, N-acetylgalactosamine and N-acetylglucosamine, was found to be secreted in a higher amount by highly virulent isolates than less virulent isolates (Vidhyasekaran et al., 1997). The sensitivity of rice plants to the toxin has been reported to be correlated with the sheath blight disease susceptibility (Brooks, 2007). The enzyme α-glucosidase from Trichoderma viride was found to be able to degrade the RS toxin (Shanmugam et al., 2001; Srim et al., 2000), indicating possible existence of α-glucosidase-mediated resistance mechanism in partially resistant rice variety. Osmotin, a cysteine-rich cytotoxic PR-5 protein, shows significant antifungal activity (Hakim et al., 2018). A resistant rice variety exhibited elevated expression of rice osmotin gene, OSM1 in response to ShB infection (Xue et al., 2016), indicating the production of osmotin protein might be a part of the chemical arsenal of rice against the pathogen.

**Quantitative Trait Loci (QTL)-based resistance**

Like many other traits, sheath blight resistance in rice is also considered to be a quantitative trait controlled by the collective effect of multiple genes (Pinson et al., 2005; Zuo et al., 2014a). Therefore, identification of QTLs, mapping, validation and their subsequent characterization could greatly expedite the map-based or positional cloning of important resistance genes, which in turn could help in the development of sheath blight-resistant rice varieties. In a genetically segregating or variable population, QTL is a statistically significant association of allelic variation at a particular locus with a phenotypic trait that exhibits a continuous variation (St. Clair, 2010). Since 1995, many QTLs for ShB resistance trait have been detected on all over the 12 chromosomes of rice genome using different mapping populations and different types of molecular markers. Although identified ShB
QTLs were summarized in two previous studies (Jia et al., 2009; Srinivasachary et al., 2011), up-to-date detailed tabular presentation of the identified QTLs, respective molecular markers, LOD value, the population used is given in Table 1. For a particular study, only identified new QTLs were kept in the table, while the re-established QTLs were excluded. The identified major and minor QTLs are physically mapped on different rice chromosomes, and putative candidate genes predicted are highlighted (Figure 4). Molecular markers such as RFLP and SSR were extensively used in the mapping studies for ShB QTL, whereas STS (Sato et al., 2004; Zuo et al., 2013), Indel (Jia et al., 2012; Zuo et al., 2007; Zuo et al., 2014a) and CAPS (cleaved amplified polymorphic sequence) (Zuo et al., 2013; Zuo et al., 2014a) were also used. In some cases, morphological markers were also employed in the mapping studies using RIL population derived from Teqing X Lemont (Pinson et al., 2005). It is clear from the present review that RIL and DH mapping population largely replaced the mapping populations derived from segregating F2 generation (Table 1). It might be due to the lower degree of recombination in the F2-derived population and their ephemerality (Schneider, 2005). Cultivars belong to indica subspecies were reported to exhibit higher level of resistance to sheath blight than those belong to japonica subspecies (Jia et al., 2012; Sharma et al., 2009; Zuo et al., 2008). This fact is reflected in the selection of donor and recurrent parent for mapping population development in the majority of the studies. Indica cultivars have been used as donor (partial ShB resistance) parents in 22 (81.5%) studies out of the total 27. To detect resistance QTLs in a broad genetic base, the majority (66%) of the studies have been performed using mapping populations developed from intersubspecific crosses (indica X japonica). Teqing and Jasmine 85 are the two indica cultivars that have been used as donor parents in the majority of the studies (Table 1). Wild relatives of cultivated varieties could be proved as an important source of ShB resistance to develop resistant varieties (Jena and Khus, 2000; Prasad and Eizenga, 2008). Despite the fact, to date, only a single study has been reported for ShB QTL detection using two accessions (IRGC100898; IRGC104705) of wild progenitor species Oryza nivara as donor parent (Eizenga et al., 2013), owing to the crossability barrier. Since ShB disease phenotyping is greatly influenced by canopy density, plant height, fertilizer application and environmental condition, proper disease phenotyping is the major hurdle for fine mapping of ShB-resistant loci.

Major QTLs and underlying candidate genes

Among all the ShB QTLs identified, two QTLs, qShB9-2 (Liu et al., 2009) and qSBR11-1 (Channamallikarjuna et al., 2010), were reported as the major loci contributing 25% and 14% of total phenotypic variation in ShB, respectively. These two QTLs are discussed here, as many other studies have verified them. It is noteworthy that the major QTL, qShB9-2, was first identified by Li et al. (1995) and later redetected and confirmed in several other studies (Han et al., 2002; Liu et al., 2013; Pinson et al., 2005; Sharma et al., 2009; Tan et al., 2005; Yadav et al., 2015; Yin et al., 2009; Zuo et al., 2014b) across different field locations and in different rice varieties revealing an almost complete dominance effect. In an earlier study, the qShB9-2 region was found to contain β-1,3 glucanase like defence gene (Yadav et al., 2015). Silva et al. (2012) identified ten candidate genes within qShB9-2 from resistant lines Jasmine 85, Teqing and MCR010277. Recently, four differentially expressed candidate genes between susceptible and resistant genotypes were detected from the vicinity of qShB9-2 (Al-Bader et al., 2019). Among the four, a C>T SNP in the OsWAK91 sequence has been shown to be associated with the resistant phenotype (Al-Bader et al., 2019). Interestingly, a recent study, dedicated to demarcating the physical region of qSB9-TQ, has mapped it successfully to a 146-Kb region flanked by the markers CY85 and Y86 (Figure 4) (Zuo et al., 2014a). The accessibility of high-quality reference genome sequence (IRG5, 2005) and fine mapping of QTLs have the cumulative potential to enhance the prediction or annotation of genes that underlie the loci. Zuo et al. (2014a) have detected 12 candidate genes in the qSBR-TQ region.

Another major QTL, qSBR11-1, derived from the rice line Tetep (partially resistant), was fine mapped to 0.85 Mb on the long arm of chromosome 11, flanked by SSR markers K39512-sbq33 (Figure 4) (Channamallikarjuna et al., 2010). On the same chromosome 11, another QTL qSBR11-LE (derived from susceptible Lemont cultivar) was identified (Tan et al., 2005; Zou et al., 2000) and fine mapped to a 78.87-Kb region between two CAPS marker Z22-27C and Z23-33C (Figure 4) (Zuo et al., 2013). Three genes are predicted to be defence-related candidate genes from a total of twelve predicted putative genes underlying the qSBR11-LE region (Zuo et al., 2013). Among the twelve genes, a lipase-like gene and two receptor-like protein kinase 5 precursor were assumed to be the most likely candidate genes for the qSBR11-LE (Zuo et al., 2013). A total of about 154 genes were anticipated to be present within the qSBR11-1 region, and among them, 26 genes were found as disease responsive (Channamallikarjuna et al., 2010). Interestingly, among the disease responsive genes, a tandem array of eleven class III chitinase, well-known antifungal gene, have been annotated (Channamallikarjuna et al., 2010). Further, a chitinase gene (LOC_Os11g47510) underlying qSBR11-1 region has been expressed in ShB-susceptible cultivar Taipei 309 and shown to confer a varying level of tolerance as evidenced by detached leaf bioassay (Richa et al., 2017). Unfortunately, no novel candidate gene worth of use in breeding/genetic engineering is identified from the mapped sheath blight QTLs.

Minor QTLs are not less important

The majority of the identified ShB QTLs exhibiting low LOD values, a small proportion of phenotypic variation and inconsistency across the environment, year and mapping populations are considered as minor QTLs. These minor QTLs could be of great significance as they may be involved in minor adjustment or fine-tuning of the traits (Chen et al., 2014a). For example, the genes for heading date minor QTLs were isolated/tagged (Lin et al., 2003; Yamamoto et al., 1998) and characterized (Wu et al., 2013) in rice, revealing their importance and possibility of candidate gene isolation from minor QTLs. However, minor ShB QTLs are less explored and no candidate genes have been identified till date. Therefore, minor ShB QTLs should not be undermined as resistance genes might be located at those loci (Zeng et al., 2014). Otherwise, it could impede the identification of genes having the potential to provide a higher level of resistance.

Relationship of resistance trait with morphological trait

Albeit the QTLs identified for plant height (PH), and heading date (HD) are not considered in this review, PH and HD are the two traits found to be correlated with ShB resistance in many of the QTL studies (Eizenga et al., 2013; Fu et al., 2011; Kunihiro et al., 2002; Li et al., 1995; Liu et al., 2014; Nelson et al., 2012; Pan
Table 1 A summary of quantitative trait loci detected for sheath blight resistance. For QTL study before 2011, see the review by Srinivasachary et al. (2011) and Jia et al. (2009). For a study, identified new QTLs are tabulated, while the re-established QTLs are excluded. Superscript I, indica rice genotype; superscript J, japonica rice genotype; BIL, backcross inbred line; RIL, recombinant inbred line; DH, doubled haploid; CSSL, chromosomal segment substitution lines.

| Name of QTLs | Chromosome | Flanking markers/sharest marker | Mapping population (resistant x susceptible) | Molecular markers (number) | LOD value/Phenotypic variance | Associated character | Remarks | Reference |
|--------------|------------|---------------------------------|---------------------------------------------|---------------------------|-----------------------------|---------------------|---------|-----------|
| qShB1.1      | 1          | RM151-RM12253                   | 210 F2 (ARC10531 x BPT-5204²)             | SSR (70)                  | 10.7                        | Percentage relative lesion height | 32 candidate genes identified in the region qShB9.2 | Yadav et al. (2015) |
| qShB7.1      | 7          | RM81-RM615                      |                                             |                           |                             | Sheath blight resistance is correlated with plant height | Wen et al. (2015) |
| qShB7.2      | 7          | RM10-RM2169                     |                                             |                           |                             | No correlation was found between LH and PH | Zeng et al. (2015) |
| qShB8.1      | 8          | RM21792-AB310                   |                                             |                           |                             | Sheath blight resistance fine mapped (146 Kb covering region), 12 genes were annotated | Zuo et al. (2014a) |
| qDR-4        | 4          | RM1155-RM575                    | 155 RIL F8:11 (RSBO2 X HH1B)               | SSR (163)                 | 2.71                        | DR – disease rating, LL – lesion length, LH – lesion height, RLL – relative LL, RLH – relative LH | Epistasis and QTL x environment (QE) interaction were studied | Liu et al. (2014) |
| qRL-4        | 4          | RM1155-RM575                    |                                             |                           |                             | Sheath blight resistance fine mapped (79 Kb covering region), 11 genes were annotated | Zuo et al. (2013) |
| qRTL3        | 3          | RM570                           | BIL (JarjanI x KoshihikariI)               | SSR (151)                 | 3.5                         | RTL – Rate of tillers with lesions | Taguchi-Shiobara et al. (2013) |
| qRTL5        | 5          | RM5724                          |                                             |                           |                             |                     |
| Name of QTLs | Chromosome | Flanking markers/nearest marker | Mapping population (resistant x susceptible) | Molecular markers (number) | LOD value/Phenotypic variance | Associated character | Remarks | Reference |
|--------------|------------|---------------------------------|---------------------------------------------|---------------------------|-------------------------------|----------------------|--------|-----------|
| qRTL6        | 6          | RM6395                          |                                             |                           | 5.8                           |                      |        |           |
| qRTL9        | 9          | RM3353                          |                                             |                           | 3.8                           |                      |        |           |
| qShB2.1-AR   | 2          | RM279-RM71                      | 216 RIL (jasmine 85' X Lemont')             | SSR (199)                 | 3.7                           | ShB resistance       | The major QTL qShB9-2 was reconfirmed based on the field data | Liu et al. (2013) |
| qShB7 LA     | 7          | RM5711-RM2                      |                                             |                           | 4.0                           |                      |        |           |
| qShB11-1-TX  | 11         | RM7203-RM536                    |                                             |                           | 3.2                           |                      |        |           |
| qShB11-1-2-TX| 11         | RM536-RM229                     |                                             |                           | 3.3                           |                      |        |           |
| qShB6 (wild 1-field 2009) | 6          | RM3431-RM3183                   | 252 Wild-1 and 253 Wild-2 BC2F2 (Oryza nivara X Bengal/O. sativa) | SSR (131)                 | 7.8                           | ShB resistance       | Colocalization of qShB6 with qDH1 and qShB1 with qPH1 revealed the influence of heading date and plant height on resistance | Eizenga et al. (2013) |
| qShB6 (wild 2-field 2009) | 6          | RM253-RM3431                    |                                             |                           | 21.2                          |                      |        |           |
| qShB6 (wild 2-field 2008) | 6          | RM253-RM3431                    |                                             |                           | 11.1                          |                      |        |           |
| qShB1 (wild 2-field 2008) | 1          | RM431-RM1361                    |                                             |                           | 4.7                           |                      |        |           |
| qShB6-mc (wild 1-microchamber) | 6          | RM3183-RM541                    |                                             |                           | 3.3                           |                      |        |           |
| qSBR2-1      | 2          | RM5340-RM521                    |                                             |                           | 3.1                           |                      |        |           |
| qSBR2-2      | 2          | RM110-osr14                     |                                             |                           | 5.2                           |                      |        |           |
| qSBR2-3      | 2          | RM7245-RM5303                   |                                             |                           | 3.3                           |                      |        |           |
| qSBR4        | 4          | RM3825                          |                                             |                           | 3.8                           |                      |        |           |
| qSBR5-2      | 5          | RM7446-RM3620                   |                                             |                           | 4.8                           |                      |        |           |
| qSBR7        | 7          | RM1132-RM473                    |                                             |                           | 3.3                           |                      |        |           |
et al., 1999; Pinson et al., 2005; Sato et al., 2004; Sharma et al., 2009). On the contrary, few studies have demonstrated disassociation of sheath blight resistance trait with other morphological traits (Channamallikarjuna et al., 2010; Taguchi-Shiobara et al., 2013; Zeng et al., 2015; Zou et al., 2000). As the sheath blight rating system is highly dependent on the lesion height of the symptom relative to the plant height, there is a high possibility of influenced result towards resistance with increasing plant height. Similarly, change in heading date (early or late maturity) qualifies plants to grow in an altered environment, which may or may not be favourable for the pathogen growth. These two facts might be the cause of the association of PH and HD with ShB resistance. Zeng et al. (2014) reviewed the frequent colocalization of PH-QTLs with ShB QTLs. They recommended not to target these ShB QTLs for utilization, as some of the PH-QTLs are mistaken as ShB QTLs and have pleiotropic effects. Moreover, plant compactness and leaf angle are the two other morphological characters that were found to be significantly correlated with sheath blight resistance in previous studies (Han et al., 2003; Hossain et al., 2016). Plant compactness may influence the microclimate (change in relative humidity and temperature) of the canopy, which significantly influences pathogen growth and sheath blight incidence.

### QTL introgression and varietal development

In addition to the identification of ShB QTLs, it is vital to analyse or evaluate their potential of utilization to develop sheath blight resistance in rice. Reports in this aspect are reviewed here. Introduction of QTL-qSBR11-1 into ‘Improved Pusa Basmati 1’ from the donor parent Tetep employing marker-assisted selection has enhanced resistance against virulent sheath blight pathogen (Singh et al., 2012). The resultant breeding line (Pusa1608) exhibited moderate resistance with ShB score 3 compared to wild-type control plant with score 7 (Singh et al., 2012). The study to determine resistance effect of two QTLs, qSB9-2 and qSB12-1 (from Teqing), has shown a higher level of resistance from the combinatorial impact of two QTLs than their individual effect (Wang et al., 2012). Wang et al. (2012) estimated the average improvement in ShB resistance from the combined introgression of the two QTLs (qSB9-2 and qSB12-1) is 0.77.

In a similar approach, the pyramiding effect of three QTLs (qSB7Tq, qSB9Tq and qSB11Le) in resistance to ShB disease has been demonstrated (Yin et al., 2008). Interestingly, the introgression of QTL-qSB-9Tq (Teqing) into nine different japonica cultivars from different ecological regions decreased ShB score by 0.5–1.3 (Zuo et al., 2008). Notably, three rice lines TIL (Teqing-into-Lemont backcross introgression lines): 455, TIL:514 and TIL:642 containing 8 introgressed ShB resistance alleles have been released jointly by the USDA-ARS, the International Rice Research Institute and the Louisiana State University Agricultural Center in 2007 (Pinson et al., 2008). These TIL lines showed significantly improved ShB resistance in 4 of the 5 year-location combinations. Pyramiding of two QTLs, qSB-7-TQ and qSB-9-TQ in susceptible japonica varieties, has been demonstrated to achieve an average 14% yield loss reduction in severe disease condition (Chen et al., 2014b).

### Association mapping

Association mapping, a high-resolution and less time-consuming consuming method, has been used in ShB-resistant loci identification. Unlike the traditional biparental way, association mapping does not require any crossing between contrasting parents (Zhu et al., 2007; Pinson et al., 2005; Sato et al., 2004; Sharma et al., 2009). On the contrary, few studies have demonstrated disassociation of sheath blight resistance trait with other morphological traits (Channamallikarjuna et al., 2010; Taguchi-Shiobara et al., 2013; Zeng et al., 2015; Zou et al., 2000). As the sheath blight rating system is highly dependent on the lesion height of the symptom relative to the plant height, there is a high possibility of influenced result towards resistance with increasing plant height. Similarly, change in heading date (early or late maturity) qualifies plants to grow in an altered environment, which may or may not be favourable for the pathogen growth. These two facts might be the cause of the association of PH and HD with ShB resistance. Zeng et al. (2014) reviewed the frequent colocalization of PH-QTLs with ShB QTLs. They recommended not to target these ShB QTLs for utilization, as some of the PH-QTLs are mistaken as ShB QTLs and have pleiotropic effects. Moreover, plant compactness and leaf angle are the two other morphological characters that were found to be significantly correlated with sheath blight resistance in previous studies (Han et al., 2003; Hossain et al., 2016). Plant compactness may influence the microclimate (change in relative humidity and temperature) of the canopy, which significantly influences pathogen growth and sheath blight incidence.

### Table 1. Continued

| Name of QTLs | Chromosome | Flanking marker/yeast | Mapping population (resistant x susceptible) | Molecular markers (number) | LOD value/Phenotypic variance | Associated character | Remarks |
|--------------|------------|-----------------------|---------------------------------------------|----------------------------|-----------------------------|---------------------|---------|
| qSBR8        | 8          | RM43264-RM1109         | 251 DH (bagyngui X maybeale)                | 227 SSR (227)              | 4.2                         | Sheath blight resistance | Xu et al. (2011) |
| qSBR9        | 9          | RM23889-RM3505         | 350 DH (bagyngui X maybeale)                | 121554                     | 5.0                         | Sheath blight resistance | Xu et al. (2011) |
| qSBR11 (2007) | 2          | RM174-RM145           | 350 DH (bagyngui X maybeale)                | 150545                     | 3.96                        | Sheath blight resistance | Xu et al. (2011) |
| qSBR11 (2008) | 2          | RM174-RM145           | 350 DH (bagyngui X maybeale)                | 150545                     | 3.96                        | Sheath blight resistance | Xu et al. (2011) |
| qSBR13 (2007) | 3          | RM135-RM145           | 350 DH (bagyngui X maybeale)                | 150545                     | 3.42                        | Sheath blight resistance | Xu et al. (2011) |
| qSBR13 (2007) | 3          | RM135-RM146           | 350 DH (bagyngui X maybeale)                | 150545                     | 3.42                        | Sheath blight resistance | Xu et al. (2011) |
| qSBR11Le     | 5          | RM18872-RM121         | 350 DH (bagyngui X maybeale)                | 150545                     | 4.35                        | Sheath blight resistance | Xu et al. (2011) |
| Rice cultivar          | Transformation method | Promoter used | Gene  | Origin   | Comments                                                                                      | References          |
|------------------------|-----------------------|---------------|-------|----------|-----------------------------------------------------------------------------------------------|---------------------|
| Chinsurah Boro II      | Protoplast            | CaMV35S       | chi1  | Rice     | A class 1 chitinase a PR3 protein                                                             | Lin et al. (1995)   |
| Chinsurah Boro II', IR72' and IR51500 | Protoplast and biolistic | CaMV35S       | Tlp-D34 | Rice     | Rice thaumatin-like protein-a PR3 protein                                                      | Datta et al., (1999b) |
| Basmati 122, Tulsi and Vaidehi | Agrobacterium mediated | CaMV35S       | chi1  | Rice     | A class 1 chitinase                                                                          | Datta et al. (2000) |
| IR72, IR64, IR688998, MH63 and Chinsurah Boro II | Biolistic          | CaMV35S       | RC7   | Rice     | Class 1 chitinase                                                                            | Datta et al. (2001) |
| M202                   | Biolistic             | Maize ubiquitin | pinA  | Wheat    | Puroindoline-antimicrobial peptides                                                            | Krishnamurthy et al. (2001) |
| Swarna                 | Biolistic to anther-derived calli and embryo | CaMV35S       | chi1  | Rice     | Chitinase                                                                                    | Baisakh et al. (2001) |
| IR72                   | Pyramiding of transgenes using marker-assisted selection | CaMV35S       | chi1, xa21 and cry gene | Rice     | Rice chitinase, receptor kinase-like protein and BT toxin protein                             | Datta et al. (2002) |
| Kenfong                | Biolistic             | Rice rbcS promoter for MOD1 and act1 promoter for RCH10 | MOD1 and RCH10 | Maize and rice | Maize ribosome-inactivating protein and rice basic chitinase                                 | Kim et al. (2003)   |
| Pusa Basmati 1         | Agrobacterium mediated | Maize ubiquitin | chi1  | Rice     | Chitinase                                                                                    | Kumar et al. (2003) |
| Pusa Basmati 1         | Agrobacterium mediated | Maize ubiquitin | chi1  | Rice     | Chitinase                                                                                    | Sridevi et al. (2003) |
| Ishikari-shiroge       | Agrobacterium mediated | Act1          | ech42, nag70 and gluc78 tlp and chi11 | Rice     | Endochitinase, exochitinase and exo-1,3-β-glucanase                                           | Liu et al. (2004)   |
| ADT38, ASD16, IR50 and Taipei 309 | Biolistic          | Maize ubiquitin | tlp and chi11 | Rice     | Rice thaumatin-like protein and chitinase                                                     | Kalpana et al. (2006) |
| Pusa Basmati 1         | Agrobacterium mediated | PAL promoter and ubiquitin | Ace-AMP1 | Allium cepa | A nonspecific lipid transfer protein with antimicrobial property                              | Patkar and Chattoo (2006) |
| Pusa Basmati 1, Co43, white Ponni and ADT38 | Agrobacterium mediated | Maize ubiquitin | RC7   | Rice     | Chitinase                                                                                    | Nandakumar et al. (2007) |
| ASD16, ADT38, IR72, IR64, and White Ponni | Agrobacterium mediated | Maize ubiquitin for tlp and chi11, Native promoter for xa21 | chi1, chi11 and xa21 | Rice     | Rice thaumatin-like protein, chitinase and serine-threonine kinase                            | Maruthasalam et al. (2007) |
| Pusa Basmati 1         | Agrobacterium mediated | Maize ubiquitin for chi11 and CaMV35S for glucanase | chi1, β-1,3-glucanase chi11 | Rice and tobacco | Rice chitinase and tobacco b-1,3-glucanase                                                    | Sridevi et al. (2008) |
| Pusa Basmati 1         | Agrobacterium mediated | Maize ubiquitin | Dm-AMP1 | Dahlia merckii | Defensin protein                                                                             | Jha et al. (2009)   |
| Pusa Basmati 2         | Agrobacterium mediated | Maize ubiquitin | Dm-AMP1 | Dahlia merckii | Defensin protein                                                                             | Jha et al. (2009)   |
| JinHui 35              | Agrobacterium mediated | Maize ubiquitin 1 | McCHIT1 | Momordica charantia | A class 1 secretory endochitinase                                                             | Li et al. (2009)    |
| Pusa Basmati 1         | Agrobacterium mediated | Maize ubiquitin 1 | Dm-AMP1 and Rs-AFP2 | Dahlia merckii and Raphanus sativus | Both are plant defensin antimicrobial protein                                                  | Jha and Chattoo (2009) |
| Pusa Basmati 1         | Agrobacterium mediated | CaMV35S       | chi42  | Rice     | Endochitinase                                                                                | Shah et al. (2009)  |
Table 2. Continued

| Rice cultivar            | Transformation method | Promoter used          | Gene          | Origin       | Comments                                 | References        |
|-------------------------|-----------------------|------------------------|---------------|--------------|------------------------------------------|-------------------|
| Pusa Basmati 1          | Agrobacterium mediated| Maize ubiquitin        | Rs-AFP2       | Raphanus sativus | Plant defensin protein                   | Jha and Chattoo (2010) |
| Pusa Basmati 1          | Agrobacterium mediated| Maize ubiquitin for chi11 and CaMV35S for ap24 | chi11 and ap24| Rice and tobacco | Chitinase and osmotin                     | Rao et al. (2011)   |
| Xiushui 11              | Agrobacterium mediated| Maize ubiquitin        | OsWRKY30      | Rice          | Transcription factor gene                 | Peng et al. (2012)  |
| Kitaake                 | Agrobacterium mediated| PBZ1                   | OsACS2        | Rice          | 1-Amino cyclopropane-1-carboxylic acid synthase | Helliwell et al. (2013) |
| Pusa Sugandhi-2         | Biolistic              | Rice P540-544          | Osoxo4        | Rice          | Nonexpressor of pathogenesis-related gene 1 | Molla et al. (2013) |
| Chaitanya and Samba Mahsuri | Agrobacterium mediated | CaMV35S               | BNPRI       | Brassica juncea | Nonexpressor of pathogenesis-related gene 1 | Molla et al. (2016) |
| Taipei 309              | Agrobacterium mediated| CaMV35S               | RCH10 and AGLU1 | Rice and alfalfa | Basic chitinase and β-1,3-glucanase      | Mao et al. (2014)   |
| Zhonghua 11             | Agrobacterium mediated| CaMV35S               | OsPGIP1       | Rice          | Polygalacturonase-inhibiting proteins     | Wang et al. (2015)  |
| Pusa Sugandhi-2         | Biolistic              | Rice P540-544          | AINPR1        | Arabidopsis thaliana | Nonexpressor of pathogenesis-related gene 1 | Molla et al. (2016) |
| Xiushui 11              | Agrobacterium mediated| Maize ubiquitin        | OsWRKY80      | Rice          | Transcription factor                     | Peng et al. (2016)  |
| BR-29                   | Agrobacterium mediated| Rice P540-544 and maize PEPC | OsOXO4 and OsCHIT1 | Rice          | Rice oxalate oxidase 4 and rice chitinase 11 | Karmakar et al. (2016) |
| Xudao 3                 | Agrobacterium mediated| Maize Ubiquitin        | OsOMS1        | Rice          | Rice osmotin                             | Xue et al. (2016)   |
| Jaldi-13                | Agrobacterium mediated| Rice P540-544 and maize PEPC | AINPR1 and OsCHIT1 | Arabidopsis thaliana and rice | Nonexpressor of pathogenesis-related gene 1 and rice chitinase 11 | Karmakar et al. (2017) |
| Taipei 309 (TP-309)     | Biolistic              | CaMV35S               | Chitinase Bacsusbin | Rice | B. subtilis strain BS-916 | Rice chitinase Oxalate decarboxylase | Richa et al. (2017) |
| Nipponbare              | Agrobacterium mediated| CaMV35S               | OsASR2        | Rice          | Absciscic acid, stress and ripening 2 protein | Li et al. (2018)    |
| Zhonghua 11             | Agrobacterium mediated| Maize Ubiquitin 1 promoter | OsASR2       | Rice          | Cytochrome P450 protein (CYP78A family) | Maeda et al. (2019) |
| Nipponbare              | Agrobacterium mediated| Maize Ubiquitin 1 promoter | OsASR2       | Rice          | Cytochrome P450 protein (CYP78A family) | Maeda et al. (2019) |

et al. (2017), has been used in ShB-resistant loci identification. A recent study of association mapping in 217 subcore rice entries with 155 markers covering the whole genome revealed a significant association of ten marker loci with the response of rice to ShB pathogen (Jia et al., 2012). Similar kinds of studies detected an association of ShB resistance with 17 marker loci (Eizenga et al., 2006) and with three marker loci (Eizenga et al., 2009). Despite the availability of high-quality genome sequence, reduced cost of high-throughput sequencing and the existence of large rice germplasm, association mapping is poorly explored to identify the region associated with the trait. To understand the genetic basis of a complex trait like rice blight, it is vital to utilize this high-resolution method efficiently.

Transgenic approach

Besides crop rotation and integrated pest management (IPM), developing resistant rice varieties either by genetic engineering or by conventional breeding would be one of the best options for sustainable agriculture (Datta, 2000). Generating host plant resistance against ShB could alone result in 7.9%–8% rice yield gain (Willocquet et al., 2004). The generation of transgenic plants is now becoming a preferred way of expressing the gene of interest for a particular desired trait. Due to the absence of resistant germplasm of rice against the pathogen Rhizoctonia solani Kuhn, traditional breeding for this trait is not yet successful. Many potential genes involved in fungal disease resistance have been isolated and characterized from rice as well as from several other plant species in recent times. The availability of suitable genetic engineering methods for many rice genotypes opens up possibilities for examining the effects of expression of essential defence genes, including pathogenesis-related (PR) genes with antifungal activity (Datta and Muthukrishnan, 1999). Several antifungal genes of plant origin, antimicrobial genes, master switch genes of defense response and genes capable of inhibiting the fungal enzyme and virulence factor have been used for the
production of rice lines with enhanced resistance against the sheath blight fungus (Table 2 and Figure 5). We review here the type of genes utilized, the strategies adopted for overexpression of those genes and their efficacy in providing ShB resistance.

Pathogenesis-related (PR) genes

On pathogen infection, one of the major generalized plant defence responses is the accumulation of pathogenesis-related (PR) proteins (Datta et al., 1999a). Expression of the glycoside hydrolase protein, which can degrade or lyse the fungal cell wall and membrane, has been the most used strategy to develop rice transgenics (Table 2). As the most abundant polymer of the fungal cell wall is chitin and β-1,3-glucan, chitinase which breaks down the β-1,4-glycosidic linkages of chitin and β-1,3-glucanase which breaks the β-1,3-linkages of glucan polymer have been utilized from different sources. The first report of transgenic expression of class I chitinase gene (chi11) in rice plant for sheath blight resistance showed that the level of resistance of the transgenic plants correlates with the level of chitinase expression (Lin et al., 1995). Transgenic plants have been shown to confer enhanced ShB tolerance when overexpressed with rice chi11 (Baisakh et al., 2001; Datta et al., 2000; Kumar et al., 2003; Rao et al., 2011; Sridi et al., 2003; Sripriya et al., 2008), rice RC7 (Datta et al., 2001; Nandakumar et al., 2007), Momordica charantia CHIT1 (Li et al., 2009) and Trichoderma virens cht42 (Shah et al., 2009). The enzyme β-1,3-glucanase has not been expressed alone, but they were used in combination with other defence genes (see Pyramiding of genes). PR-5 proteins, thaumatin-like proteins (TLP) and/or osmotins are known to permeabilize the fungal membrane. The rice PR-5 genes, TLP-D-34 (TLP-P subfamily) and OSM1 (TLP-PA subfamily), have been expressed for enhanced ShB tolerance (Datta et al., 1999b; Xue et al., 2016).

Antimicrobial peptides (AMP)

AMPs are glycine or cysteine-rich small peptides that function in host innate defence and found in both animal and plant kingdoms. Thionin, defensin and lipid transfer protein are the three widely known plant AMPs that aid in defence by pore formation in membrane resulting in ion/metabolite leakage and cell death (Navrrot et al., 2014; Pelegrini et al., 2011). Transgenic expression of different plant AMPs from various sources showed enhanced protection of rice plants against R. solani. Ectopic expression of AMP1 from Dahlia merckii (Jha et al., 2009) and Allium cepa (Patkar and Chattoo, 2006), AFP2 from Raphanus sativus (Jha and Chattoo, 2010) and puroindoline (pinA and pinB) from wheat (Krishnamurthy et al., 2001) have been demonstrated to enhance the ShB resistance in rice. Snakin-1, an AMP, has been shown to confer resistance to R. solani when expressed in transgenic potato (Almasia et al., 2008). Some AMPs like stomoxyn ZH1, purothionin, cecropin B, D4E1 and phor21 exhibited in vitro inhibitory activity on R. solani AG-1A (Elhag

Figure 2 Disease cycle of rice sheath blight caused by Rhizoctonia solani AG1-1A. Sexual reproduction through basidiospores is ignored.
Figure 3  Schematic representation of rice–
*R. solani* molecular interaction and signalling
pathways involved. The blue outer circle symbolizes the pathogen, *R. solani*, and the central oval-shaped figure signifies the rice plant. The pink half of the diagram consists of the rice defence strategies to counteract the pathogen, while the yellow coloured half consists of various pathogenesis mechanisms. SA, salicylic acid; JA, jasmonic acid; ET, ethylene; OxO, oxalate oxidase; AC, adenylate cyclase; cAMP, cyclic adenosine monophosphate; MPK, MAP kinase; MKK, MAPK kinase; GT, glycosyltransferase; PR-I9, peptidase inhibitor I9 domain; PR, pathogenesis related; MeJA, methyl jasmonate; ACS2, 1-
aminocyclopropane-1-carboxylic acid synthase 2; GGPP, geranyl geranyl pyrophosphate; GAP, glyceraldehyde 3-phosphate; E4P, erythrose-4-phosphate; PEP, phosphoenolpyruvate; WD, tryptophan-aspartic acid repeat domain-containing protein. Dotted arrow signifies that the connection is not experimentally evidenced.

Figure 4  Physical map of different ShB QTLs on different rice chromosomes. QTLs are depicted on the right side of the bar, while the markers are shown on the left. Putative candidate genes (CGs) assigned to major QTLs are highlighted. Unit: Megabase (Mb). Length of segments on chromosomes represent physical intervals of corresponding QTLs. Abbreviation, RLPK, receptor-like protein kinase; Ch, chromosome.
et al., 2017; Oard et al., 2004). It would be interesting to see the effect of transgenic expression of the genes encoding snakin-1 and other AMPs mentioned in enhancing ShB disease resistance.

Signalling gene

After plants sense the presence of a pathogen, they retaliate via the activation of signalling pathways, which ultimately terminate with the synthesis of defence proteins. Therefore, manipulation of upstream master signalling genes and transcription factor genes is a sound idea to stimulate the pathway in advance leading to enhanced protection from pathogens (Gurr and Rushton, 2005). The ‘non-expressor of pathogenesis-related genes 1’ (NPR1) has been identified as a master controller of SA-mediated activation of the SAR pathway in Arabidopsis (Cao et al., 1997). Introduction of Brassica juncea NPR1 (BjNPR1) gene in rice has been shown to display an elevated level of resistance to sheath blight (Sadumpati et al., 2013). In our recent studies, we have demonstrated the effectiveness of AtNPR1 gene in rice sheath blight tolerance via the activation of several downstream defence genes (Karmakar et al., 2019; Karmakar et al., 2017; Molla et al., 2016). Overexpression of transcription factor genes, WRKY30 and WRKY80 caused improved resistance to ShB by activating JA or JA/ET signalling pathway (Peng et al., 2012, 2016). Similarly, a transgenic plant expressing the ACS2 (1-aminoacyclopropane-1-carboxylic acid synthase) gene, essential for the synthesis of signalling molecule ethylene, showed increased resistance against R. solani (Helliwell et al., 2013).

Genes which act against pathogen effector

Fungi secrete virulence factors or effectors for their infection, while plants try to degrade them upon recognition (Stergiopoulos and de Wit, 2009). Overexpression of oxalate oxidase 4 (Molla et al., 2013) and oxalate decarboxylase (Qi et al., 2017), capable of degrading the virulence factor oxalate, has been shown to exhibit improved tolerance to the ShB pathogen. Interestingly, in our study, we observed oxalate oxidase-mediated degradation of oxalate resulted in the generation of H2O2, which in turn might activate the plant’s defence system (Molla et al., 2013). Similarly, overexpression of OsPGIP (polygalacturonase-inhibiting protein), capable of counteracting the pectin degrading enzyme polygalacturonase, caused ShB tolerance (Wang et al., 2015). It is evident from those studies that degradation or inhibition of fungal virulence factors or cell wall-degrading enzymes is an efficient strategy to impede the pathogen.

Pyramiding of genes

It might not be the finest way to strengthen resistance by the overexpression of a single defence-related protein (Hammond-Kosack and Parker, 2003). Substantial efforts have been made for combinatorial expression of defence genes, such as the maize ribosome inactivating protein gene MOD1 and RCH10 (Kim et al., 2003), rice chitinase (chi11) and thaumatin-like protein (Kalpana et al., 2006; Maruthasalam et al., 2007), chi11 and β-1,3-glucanase (Sridive et al., 2008), DmAAMP1 and RsAFP2 (Jha and Chattoo, 2009), chi11 and ap24 (Rao et al., 2011), rice basic chitinase gene (RCH10) and the alfalfa β-1,3-glucanase gene (AGLU1) (Mao et al., 2014), rice oxalate oxidase 4 and rice chitinase 11 (Karmakar et al., 2016), and Arabidopsis NPR1 and rice chitinase 11 (Karmakar et al., 2017) genes for greater level of resistance over the resistance conferred by single gene. Our comparative study indicated that dual gene cassette is more effective in providing ShB resistance than the individual effect of single gene cassette (Karmakar et al., 2017).
Host-induced gene silencing (HIGS)

HIGS, an RNA interference (RNAi)-based strategy, involves the expression of a suitable RNAi construct targeting the pathogen effector gene/s in the host plant, transfer of the double-stranded RNA (dsRNA) or small interfering RNA (siRNA) into the pathogen during interaction, and subsequent silencing of the target gene and inhibition of virulence. The success of HIGS largely depends on the existence of a functional RNAi silencing system in the pathogen. Only recently, two studies showed the successful implication of HIGS in rice–R. solani pathosystem and indicated the presence of the RNA interference system in ShB pathogen (Rao et al., 2019; Tiwari et al., 2017). To employ HIGS for controlling ShB, Tiwari et al. (2017) targeted pathogenicity map kinase (PMK) genes, while Rao et al. (2019) silenced polygalacturonase (PG) gene in R. solani. Interestingly, effective silencing of the pathogen genes has been shown to suppress the ShB disease in the transgenic rice plant carrying the RNAi constructs.

Choice of promoter

Precise selection of promoter is a critical factor in any genetic engineering programme. In the majority of transgenic studies for ShB tolerance, transgenes have been expressed constitutively using different promoters like CaMV35S, maize ubiquitin and rice actin 1 (Table 2). However, in many cases, it has been reported that the constitutive expression of the master signalling gene increases the metabolic load and develops undesirable phenotype. For instance, constitutive expressions of AtNPR1 developed lesion mimic phenotype (LMP) (Fitzgerald et al., 2004), and OsNH1 developed dwarfing and LMP under two different growth conditions (Chern et al., 2005). Similarly, a pyramiding of RCH10 and AGLU1 in transgenic rice resulted in low seedling vigour and diminished germination rate (Mao et al., 2014). As green tissues are the area of infection by R. solani, it is better to target the expression of transgenes only to green tissues. Our recent report showed green tissue-specific expression of master controller AtNPR1 gene in rice enhances ShB resistance avoiding the phenotypic cost (Molla et al., 2016). Recently, Xu et al. (2017) devised another strategy to avert fitness costs due to AtNPR1 expression. The study demonstrated that the translational control of NPR1 gene by the uORFSTBF1 cassette, which comprises of an immune-inducible promoter and two pathogen-responsive upstream open reading frames, could engineer plant for disease resistance without compromising fitness penalty. Only a few studies have reported the use of green tissue-specific promoters such as rice rbcS (Kim et al., 2003), rice D540-544 (Molla et al., 2013) and maize PEPC (Karmakar et al., 2016, 2017) to express transgenes for ShB tolerance. It should also be taken into consideration that the strength of the tissue-specific promoter needs to be on par with the constitutive one. Alternatively, inducible promoters are useful for temporal regulation of the transgene since they initiate the expression of genes only after the pathogen attack. The genes AceAMP1 and OsACS2 have been expressed effectively in rice under the control of pathogen-inducible PAL (Patkar and Chattoo, 2006) and PBZ1 promoter (Hellwell et al., 2013).

Conclusive remarks and future prospects

The ShB disease and the causal agent R. solani are convincingly growing as a significant threat to rice cultivation throughout the world. The versatile nature of R. solani and its tremendous ability to attack almost all types of tissues on a wide range of plants made it as an ‘enigmatic pathogen to control’ even after 100 years of its discovery. IRRI scientists have screened more than 30,000 rice germplasms, and except few wild relatives, none found with a reliable level of sheath blight resistance (https://irri.org). As no R gene has been reported against sheath blight to date, it is not unusual to see assumptions in the scientific community that quantitative loci might solely confer the resistance. Although quite a plethora of reports have been published on the identification and mapping of ShB QTLs, major QTLs with high phenotypic variance have not been detected to date. Despite many mapping studies, not a single study reported the cloning and functional validation of the vital regions underlying a QTL. As a result, no notable progress has been made towards sheath blight resistance breeding utilizing those QTLs. Many minor effect QTLs have been identified across the studies, which may play a significant role in the complex sheath blight resistance trait. Pyramiding several favourable consistent minor QTL alleles into a single cultivar could clarify their possible role in the resistance mechanism. However, a question arises here – is it feasible to introgress all consistent minor QTLs into a single background? The task becomes more difficult with a greater number of QTLs. To achieve the goal, one approach is the marker-assisted recurrent selection (MARS) in which ‘the frequency of favorable QTL alleles in the population can be increased through cycles of MAS for multiple QTLs and intermating of the selected individuals in the population in a recurrent selection scheme’ (Bernardo, 2008; St. Clair, 2010). ‘Relative lesion height’ based ‘standard evaluation system’ (SES) for sheath blight disease is highly influenced by plant height. So, it is not uncommon to misinterpret the tolerance level, which in turn affects the detection of QTLs. An alternative and precise sheath blight scoring system independent of plant height is needed to avoid the complications.

With the advancement of sequencing technology and increasing affordability, it became easier to generate enormous genomic resources that can be explored to find natural variation in defence-related genes among genotypes, which can be further validated for significant association with disease resistance. In this regard, all landraces, farmer’s variety, weedy rice and wild relatives could also be exploited to find hidden treasure in the form of sheath blight resistance.

Identification of rice genes for negative regulator of defence against R. solani or the rice genes which support the infection (a.k.a. susceptibility genes) (van Schie and Takken, 2014) of R. solani utilizing the expanding amount of omics data from infected plants would pave the way to modulate the host defence by knocking out/knocking down. Since the study of Kouzai et al. (2018) indicated the involvement of SA-mediated immunity, it can be speculated that SA catabolism might be one of the strategies of R. solani to overcome the immunity. Rice plants with ectopically expressed SA hydroxylase gene became more susceptible to ShB disease than the wild type (Kouzai et al., 2018). Search for the up-regulated SA-catabolizing genes (van Schie and Takken, 2014) in rice in response to R. solani infection may lead to the identification of a susceptibility gene.
Identification of pathogenic determinants, effectors or virulence factor genes from *R. solani* utilizing their recently available genome sequence would greatly facilitate to unravel pathogenesis mechanism. Newly developed CRISPR-mediated genome editing and associated technologies have great potential to identify, study and functionally validate fungal genes linked to the pathogenesis (Molla and Yang, 2019; Pickar-Oliver and Gersbach, 2019). To utilize the technology, a well-standardized genetic transformation system of *R. solani* needs to be in place.

Although the secretome of *R. solani* AG1-1A has been in silico predicted (Anderson et al., 2017), no in vitro study has been performed to characterize its secretome. Analysis of secretome may significantly contribute to a better understanding of pathogenesis and subsequently to develop management strategies. It is true that the secretome analysed from using synthetic media may not be enough to get a picture close to the secretome *R. solani* uses during rice plant infection. Since protein expression and secretion are mostly reliant on the nature of the substrate (Suárez et al., 2005), it is challenging to unravel the real secretome. One possible solution is to use rice straw decoction as the substrate.

Improved understanding of plant–fungal interaction and the identification of an increasing number of antifungal proteins have facilitated the development of different transgenic strategies to generate rice plants with enhanced sheath blight tolerance. However, the development of tolerance to sheath blight could not be achieved yet to a commercially useful level. It has become progressively clear that introgression of single candidate genes is not enough to provide an adequate level of resistance (Melchers and Stuiver, 2000). Precise designing of transformation construct and finding a suitable combination of genes to stack are vital for further transgenic development. Combining a master controller gene (like NPR1) with a PR gene or integrative approach to combine QTL with qualitative resistance gene/genomes may be proved as a good line of future action.

Emerging pieces of evidence suggest that microRNAs (miRNAs) play a pivotal role in different plant developmental processes, biotic/abiotic stress adaptation, as well as in host–pathogen interaction mostly via post-transcriptional gene regulation. Except in few recent studies (Campos et al., 2013; Guo et al., 2012; Li et al., 2014), the role of miRNA in rice disease resistance/susceptibility is poorly explored. Identification of rice miRNAs differentially regulated by *R. solani* infection is an initial step towards investigating their role in host immunity (Li et al., 2014). Not only miRNA but also the study of global expression profiles of small RNA (sRNA) is also equally essential to identify infection-related sRNAs from rice and *R. solani*. Fungal sRNA effectors are transported to host cells during infection to suppress host immunity-related genes (Weiberg et al., 2014). The exploration of any role of cross-kingdom RNA interference in virulence and pathogenesis, as well as host resistance, would significantly enhance the understanding of the pathosystem.

The circadian clock system is likely to be involved in effector-triggered immunity and pathogen-associated molecular pattern-triggered immunity (Sharma and Bhatt, 2015). Accumulation of salicylic acid upon pathogen infection is sensed by the master regulator NPR1, which simultaneously up-regulates both defence genes and circadian clock genes showing how plants gate their immune responses towards the morning to anticipate infection (Zhou et al., 2015). Since SA-mediated immunity seemed to play a role in ShB resistance (Kouazi et al., 2018), the study of the circadian oscillation system and their possible involvement in the modulation of defence system could be done in rice–*R. solani* pathosystem to gain novel insight. Regardless of many studies, scientists are stumbling into a quagmire to find a durable means to control sheath blight disease of rice. Therefore, the study of any possible factors or hopes should not be left untouched to get an answer in the near future.

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**Conflict of interest**

Authors declare no conflict of interest.

**Author contribution**

SKD, KD and KAM conceived the idea. KAM, SK, SKD and KD planned and outlined the review. KAM and SK collected materials. KAM wrote the manuscript. KAM and SK created the figures. JM, PV and RKV created the physical map of ShB QTLs. KAM and SK prepared the tables. SK, KD, RKV and SKD edited the manuscript. All authors reviewed and finalized the manuscript.

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