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Review

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 (Kannaiyan 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 (García *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

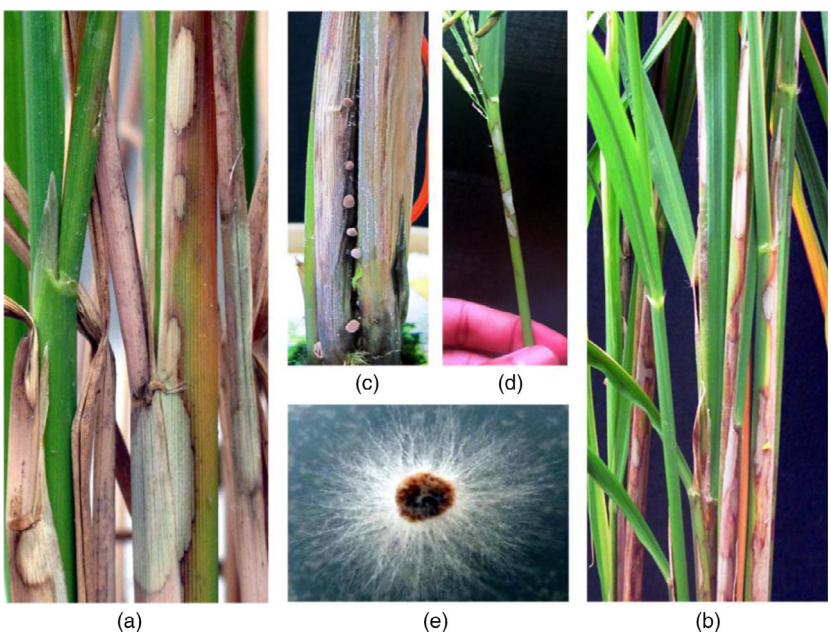

Figure 1 Rice sheath blight disease symptom and the pathogen. (a), (b) and (d) typical sheath blight disease symptoms. (c) Sclerotia formed on a heavily infected plant. (e) Growing mycelia from a sclerotium.

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 (Nagarajkumar *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 Gα, Gβ and Gγ 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 (Charoensopharat *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 [parvulin subfamily of peptide prolyl cis/trans isomerase (PPiC) protein], RS_P3 (NifU 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

Omics, 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)+] 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' (milRNAs), which includes 15 predicted candidate pathogenic novel milRNAs (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 Tarighi, 2010; Vidhyasekaran *et al.*, 1997). A JA-deficient rice mutant, *hebiba,* exhibited enhanced susceptibility to the sheath blight disease (Taheri and Tarighi, 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 also may 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 momilactone '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 hydroxylase* (*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 gibberellin (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 Gβ subunit (RGB1) gets induced but not Gγ subunit (RGG1) (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*, *OsCML*31, *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 *AtNPRI*-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 bZIP transcription factor, TGAP1 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 other 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; Sriram *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 Tequing 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 inter-subspecific 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 Khush, 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 (IRGS, 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 qSB9-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 qSB11-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 qSB11-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 qSB11-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/nearest 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^{I} X BPT-5204^{I})	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			8.8			
qshb7.2	7	RM10-RM2169			6.7			
qshb8.1	8	RM21792-RM310			4.2			
qSBL7 (E2)	7	D760-RM248	190 F2 (Yangdao 4^{I} X Lemont^{J})	SSR (52) and InDel (128)	3.12	DR – disease rating	Sheath blight resistance is correlated with plant height	Wen *et al.* (2015)
qSBPL-7 (E2)	7	D760-RM248			5.07	LH – lesion height		
qSBD-12-2 (E1)	12	RM1246-D1252			3.74	PL – percentage of lesion height		
qHZaLH3	3	RM143-RM514	116 DH (TN1^{I}X CJ06^{J})	SSR (214)		DR, LH	No correlation was found between LH and PH	Zeng *et al.* (2015)
qHZaLH6	6	WX-RM587						
qHZaDR8	8	RM1376-RM4085						
qHZaDR9	9	RM444-AGPSMA						
qHZbDR5	5	RM3321-RM3616						
qSB-9^{TQ}	9	CY-85 and Y86	235 CSSLs (BC6F3) (Teqing^{I} (TQ) X Lemont^{J})	InDel and CAPS (22)	–	ShB resistance	Fine mapped (146 Kb covering region), 12 genes were annotated	Zuo *et al.* (2014a)
qDR-4	4	RM1155-RM5757	155 RIL F8:11 (RSB02 X HH1B)	SSR (163)	2.71	DR – disease rating LL – lesion length	Epistasis and QTL x environment (QE) interaction were studied	Liu *et al.* (2014)
qRLL-4	4	RM1155-RM5757			5.84	LH – lesion height RLL – relative LL		
qRLH-4	4	RM1155-RM5757			4.77	RLH – relative LH		
qSB-11^{LE}	11	Z22-27C and Z23-33C	112 CSSLs (Teqing^{I} X Lemont^{J} (LE))	STS and CAPS (26)	–	ShB resistance	Fine mapped (79 Kb covering region), 11 genes were annotated	Zuo *et al.* (2013)
qRTL3	3	RM570	BIL (Jarjan^{I} x Koshihikari^{J})	SSR (151)	3.5	RTL- Rate of tillers with lesions		Taguchi-Shiobara *et al.* (2013)
qRTL5	5	RM5784			4.3			
qRTL6	6	RM1161			7.7			
qRTL9	9	RM6251			3.1			
qRTL3	3	RM16200			5.9			
qRTL6	6	RM2615			2.9			
qRTL12	12	RM7025			3.2			
qRTL5	5	RM3286			3.1			

Table 1. Continued

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	RM3533			3.8			
qShB2-1-	2	RM279–RM71	216 RIL (Jasmine 85[I] X Lemont[J])	SSR (199)	3.7	ShB resistance	The major QTL qShB9-2 was reconfirmed based on the field data	Liu *et al.* (2013)
ARqShB7-AR	7	RM5711–RM2			4.0			
qShB7 LA	7	RM5711–RM2			6.0			
qShB11-1-	11	RM7203–RM536			3.2			
TXqShB11-2-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[J](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 (wild2- 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			
qsbr_2.1	2	RM8254-RM8252	197 DH (MCR10277[I] X Cocodrie[J])	SSR (111)	3.4-29.7	SBF – sheath blight		Nelson *et al.* (2012)
qsbr_2.2	2	RM3857-RM5404			2.9-37.8	Disease severity in field, SBI – disease severity in microchamber		
qsbr_12.1	12	RM3747-RM27608			49.1	SBM – disease severity in mist chamber		
qSBR1	1	RM11229	217 core collection of USDA	SSR (154) and Indel (1)	9.5%	Sheath blight resistance		Jia *et al.* (2012)
qSBR11	11	RM7203			1.9%			
qSBR1-1	1	RM5389-RM3825	121 RIL (RSB03 X HH1B)	SSR (123)	3.2	DR – disease rating LL – lesion length		Fu *et al.* (2011)
qSBR2-1	2	RM5340-RM521			3.1	LH – lesion height		
qSBR2-2	2	RM110-osr14			5.2	RLL – relative LL		
qSBR2-3	2	RM7245-RM5303			3.3	RLH – relative LH		
qSBR4	4	RM3288-RM7187			3.8			
qSBR5-2	5	RM7446-RM3620			4.8			
qSBR7	7	RM1132-RM473			3.3			

Table 1. Continued

Name of QTLs	Chromosome	Flanking markers/nearest marker	Mapping population (resistant x susceptible)	Molecular markers (number)	LOD value/ Phenotypic variance	Associated character	Remarks	Reference
qSBR8	8	RM8264-RM1109			4.2			
qSBR9	9	RM23869-RM3769			5.0			
qShB1 (2007/2008)	1	RM431-RM12017	251 DH (Baiyeqiu[1] X Maybelle[h])	SSR (227)	5.18-8.03	Sheath blight resistance		Xu *et al.* (2011)
qShB2 (2008)	2	RM174-RM145			3.96			
qShB3 (2007)	3	RM135-RM186			3.42			
qShB5 (2007)	5	RM18872-RM421			4.35			

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

Table 2 Transgenic rice against sheath blight pathogen *Rhizoctonia solani*: a historical overview

Rice cultivar	Transformation method	Promoter used	Gene	Origin	Comments	References
Chinsurah Boro II	Protoplast	CaMV35S	*chi11*	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 PR5 protein	Datta *et al.*, (1999b)
Basmati 122, Tulsi and Vaidehi	*Agrobacterium* mediated	CaMV35S	*chi11*	Rice	A class 1 chitinase	Datta *et al.* (2000)
IR72, IR64, IR68899B, MH63 and Chinsurah Boro II	Biolistic	CaMV35S	*RC7*	Rice	Class 1 chitinase	Datta *et al.* (2001)
M202	Biolistic	Maize ubiquitin	*pinA pinB*	Wheat	Puroindoline–antimicrobial peptides	Krishnamurthy *et al.* (2001)
Swarna	Biolistic to anther-derived calli and embryo	CaMV35S	*chi11*	Rice	Chitinase	Baisakh *et al.* (2001)
IR72	Pyramiding of transgenes using marker-assisted selection	CaMV35S for *chi11*	*chi11, 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	*chi11*	Rice	Chitinase	Kumar *et al.* (2003)
Pusa Basmati 1	*Agrobacterium* mediated	Maize ubiquitin	*chi11*	Rice	Chitinase	Sridevi *et al.* (2003)
Ishikari-shiroge	*Agrobacterium* mediated	Act1	*ech42, nag70* and *gluc78*	*Trichoderma atroviride*	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	Biolistic	Maize ubiquitin for *tlp* and *chi11*. Native promoter for *xa21*	*tlp, 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*	*chi11* and *β-1,3-glucanase*	Rice and tobacco	Rice chitinase and tobacco b-1,3-glucanase	Sridevi *et al.* (2008)
Pusa Basmati 1	*Agrobacterium* mediated	Maize ubiquitin	*chi11*	Rice	Chitinase	Sripriya *et al.* (2008)
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	*cht42*	*Trichoderma virens*	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 $P_{D54O-544}$	*Osoxo4*	Rice	Rice oxalate oxidase	Molla *et al.* (2013)
Chaitanya and Samba Mahsuri	*Agrobacterium* mediated	CaMV35S	*BjNPR1*	*Brassica juncea*	Nonexpressor of pathogenesis-related gene 1	Sadumpati *et al.* (2013)
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 $P_{D54O-544}$	*AtNPR1*	*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 $P_{D54O-544}$ and maize PEPC	*OsOXO4* and *OsCHI11*	Rice	Rice oxalate oxidase 4 and rice chitinase 11	Karmakar *et al.* (2016)
Xudao 3	*Agrobacterium* mediated	Maize Ubiquitin	*OsOSM1*	Rice	Rice osmotin	Xue *et al.* (2016)
Jaldi-13	*Agrobacterium* mediated	Rice $P_{D54O-544}$ and maize PEPC	*AtNPR1* and *OsCHI11*	*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	Rice	Rice chitinase	Richa *et al.* (2017)
Nipponbare	*Agrobacterium* mediated	CaMV35S	*Bacisubin*	*B. subtilis* strain BS-916	Oxalate decarboxylase	Qi *et al.* (2017)
Zhonghua 11	*Agrobacterium* mediated	Maize Ubiquitin 1 promoter	*OsASR2*	*Rice*	Abscisic acid, stress and ripening 2 protein	Li *et al.* (2018)
Nipponbare	*Agrobacterium* mediated	Maize Ubiquitin 1 promoter	*OsBSR2*	*Rice*	Cytochrome P450 protein (CYP78A family)	Maeda *et al.* (2019)

et al., 2008), 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 sheath 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 defence 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; Sridevi *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 (Nawrot *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

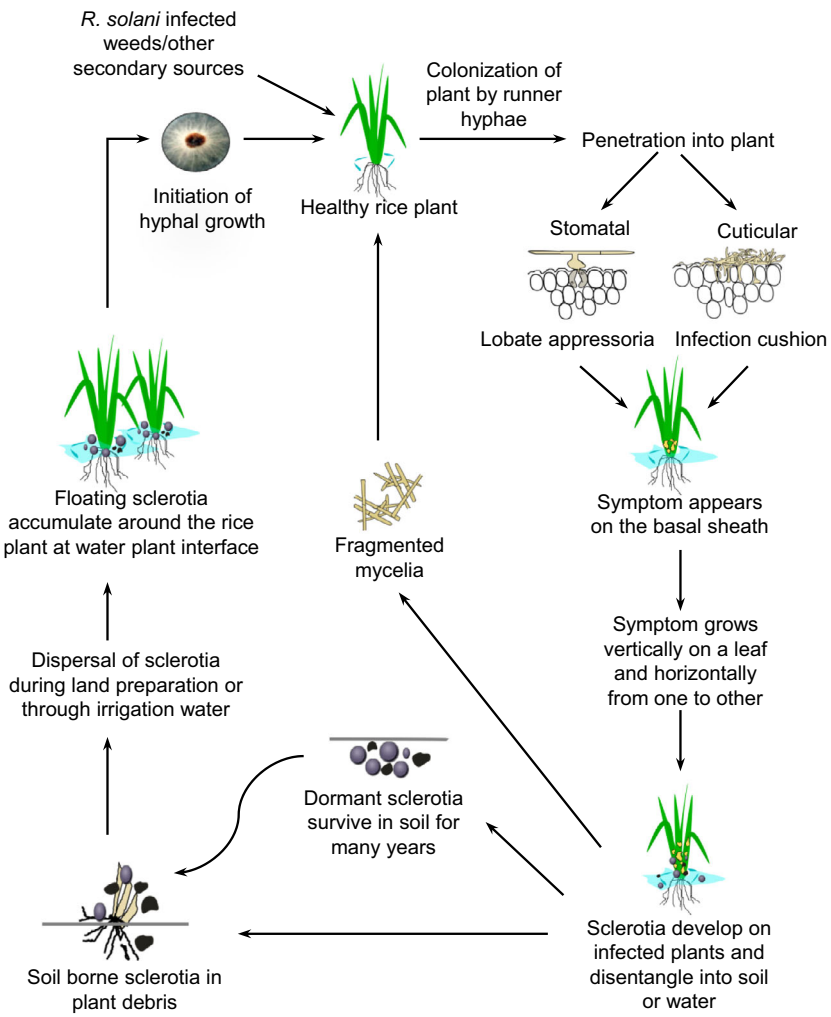

Figure 2 Disease cycle of rice sheath blight caused by *Rhizoctonia solani* AG1-1A. Sexual reproduction through basidiospores is ignored.

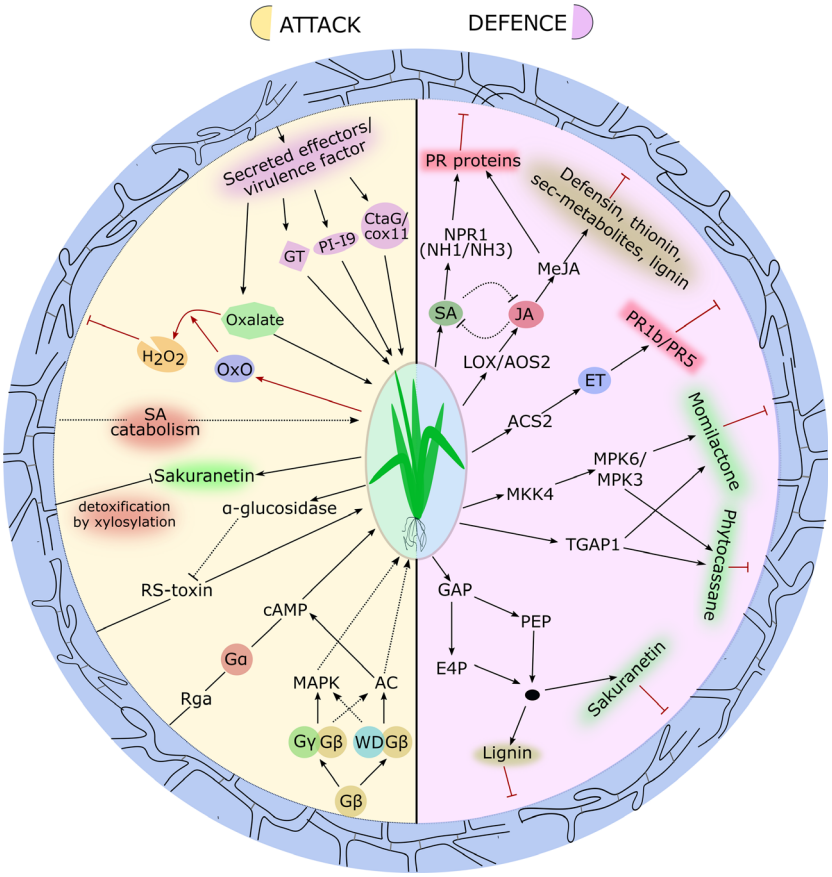

Figure 3 Schematic representation of rice–*R. solani* molecular interaction and signalling pathways involved. The blue outer circle symbolizes the pathogen, *R. solani,* and the central oval-shaped figure signifies the rice plant. The pink half of the diagram consists of the rice defence strategies to 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; PI-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.

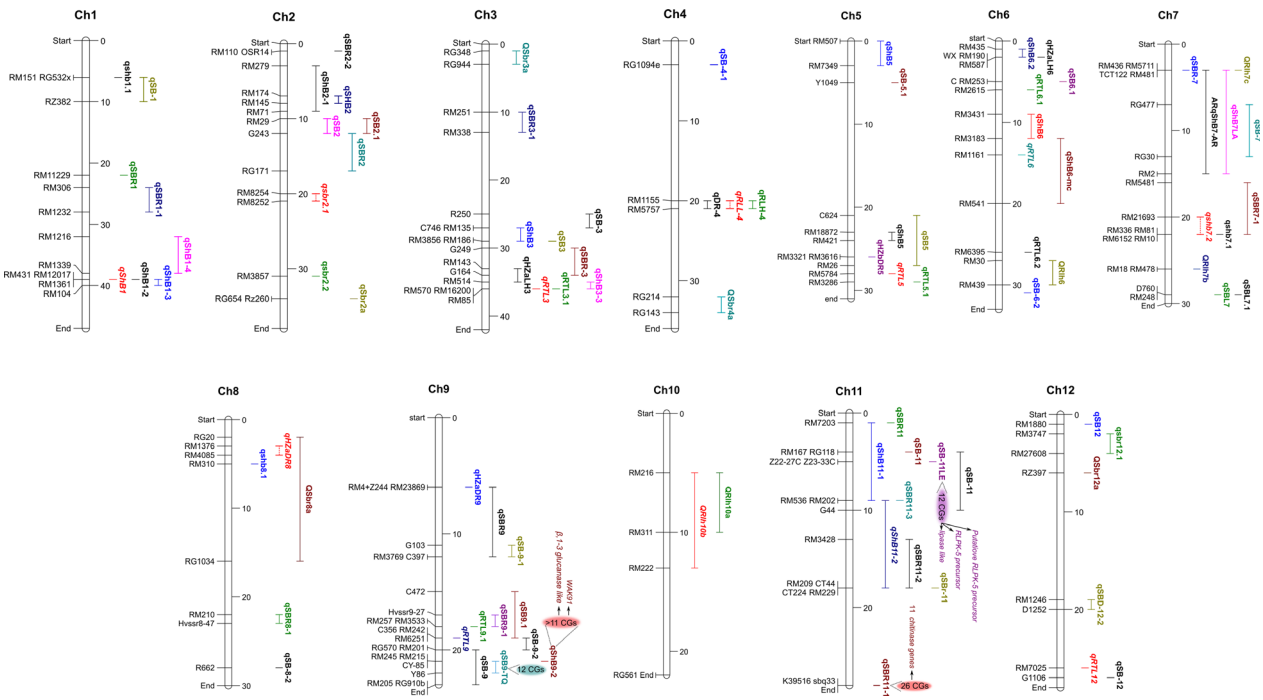

Figure 4 Physical map of different ShB QTLs on different rice chromosomes. QTLs are depicted on the right side of the bar, while the markers are shown on the left. Putative candidate genes (CGs) assigned to major QTLs are highlighted. Unit: Megabase (Mb). Length of segments on chromosomes represent physical intervals of corresponding QTLs. Abbreviation, RLPK, receptor-like protein kinase; Ch, chromosome.

Figure 5 Schematic model showing different genes utilized to raise rice transgenics and their mode of action conferring sheath blight tolerance. The outermost pink circle contains the gene names, and the middle yellow circle contains the respective mode of action to resist fungal pathogen. Os, *Oryza sativa*; Nt, *Nicotiana tabacum*; At, *Arabidopsis thaliana*; Mc, *Momordica charantia*; Rs, *Raphanus sativus*; Ace, *Allium cepa*; Dm, *Dahlia merckii*; Ta, *Triticum aestivum*; Zm, *Zea mays*; Bj, *Brassica juncea*.

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-aminocyclopropane-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 H_2O_2, 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* (Sridevi *et al.*, 2008), *DmAMP1* 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). Three glycoside hydrolase genes, *ech42* (*endochitinase*), *nag70*

(*exochitinase*) and *gluc78* (*exo-1,3-β-glucanase*), from *Trichoderma atroviride* have been pyramided in all possible combinations and shown to elevate tolerance level to the pathogen (Liu *et al.*, 2004).

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 D54O-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 (Helliwell *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/genes 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 (Campo *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 (Kouzai *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 drawn the figures. JM, PV and RKV drawn 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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