**Rhizoctonia solani** Kühn
Pathophysiology: Status and Prospects of Sheath Blight Disease Management in Rice

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Sheath blight caused by necrotrophic fungus *Rhizoctonia solani* Kühn is one of the most serious diseases of rice. Use of high yielding semi dwarf cultivars with dense planting and high dose of nitrogenous fertilizers accentuates the incidence of sheath blight in rice. Its diverse host range and ability to remain dormant under unfavorable conditions make the pathogen more difficult to manage. As there are no sources of complete resistance, management through chemical control has been the most adopted method for sheath blight management. In this review, we provide an up-to-date comprehensive description of host-pathogen interactions, various control measures such as cultural, chemical, and biological as well as utilizing host plant resistance. The section on utilizing host plant resistance includes identification of resistant sources, mapping QTLs and their validation, identification of candidate gene(s) and their introgression through marker-assisted selection. Advances and prospects of sheath blight management through biotechnological approaches such as overexpression of genes and gene silencing for transgenic development against *R. solani* are also discussed.

**Keywords:** *Rhizoctonia solani*, rice sheath blight (ShB), biological control, disease resistance, transgenic rice, resistance QTLs

**INTRODUCTION**

Rice (*Oryza sativa* L.) serves as the primary diet for approximately 67% of the world population. In the Asian region, the demand for rice production is the highest in the world, due to the increased preference for rice among the population (Mohanty, 2013). Throughout the world, productivity of rice is affected by several biotic and abiotic factors. There are about 50 different biotic factors that can cause potential yield loss in rice including fungi, bacteria, viruses, nematodes and insects. Of the disease-causing organisms, fungal pathogens impose a greater challenge in sustaining rice production (Webster and Gunnell, 1992).

Among the fungal diseases causing significant yield loss in rice, sheath blight is ranked the second most important after rice blast (Pan et al., 1999). The sheath blight pathogen has two stages, *Rhizoctonia solani* Kühn, the anamorph stage and a teleomorph stage, *Thanatephorus cucumeris* (Frank) Donk. Belonging to the division Basidiomycota, *R. solani* is a necrotrophic fungus that produces sclerotia of varying sizes but with uniform texture, which can remain dormant for many years (Mukherjee, 1978). The disease causes a yield reduction ranging from 20 to 50% depending...
on the severity of infection (Groth and Bond, 2007; Margani and Widadi, 2018). In the recent past, sheath blight has become a major threat, especially under intensive rice cultivation. Monoculture of high-yielding semi-dwarf rice varieties, heavy doses of nitrogenous fertilizers and the favorable micro-environment facilitated by the crop density are implicated as the major factors favouring the sharp increase in the disease incidence (Savary et al., 1995; Cu et al., 1996). Reported for the first time in Japan in 1910 (Miyake, 1910), sheath blight disease had spread all across the world. *R. solani* is a very destructive pathogen. Taking advantage of the large host range (Kozaka, 1965), the pathogen often survives on the alternate hosts during hostile conditions, making the disease very difficult to manage. Besides, it can also survive in soil and dead plant debris by producing resting structures such as sclerotia.

To incite the disease in rice plants, the fungal inoculum should come in contact with the live host tissues in the field. The inoculum can be a runner hypha or a sclerotium and in rare cases basidiospores, often floating in the irrigation water. By this mode, inoculum can travel and spread to different locations in the field or from the irrigation canals where alternate hosts can supply sufficient inoculum. In rice, *R. solani* can infect the plant at any growth stage (Dath, 1990). The incidence of sheath blight is more severe in early maturing, semi-dwarf, highly tillering and compact cultivars (Bhunkal et al., 2015b). The disease severity and incidence increase with plant age (Singh et al., 2004). The resistance and susceptibility in the rice genotypes are distinct in mature plants as compared to seedlings (Dath, 1990). The sheath blight progression is slow in initial growth stages, while it is fast at tillering and later stages of growth (Thind et al., 2008).

Although several cultural, chemical and biological control strategies have been suggested to manage sheath blight disease of rice (Yellareddygari et al., 2014; Datta and Vurukonda, 2017), chemical control has been the most widely used method so far. However, this method is relatively less sustainable in crop production because of the increased cost of production, development of fungicide tolerance and apprehensions of residual toxicity. Biological strategies targeting host plant resistance have been advocated as the most viable solution, which includes mapping of gene(s) or quantitative trait loci (QTLs) governing disease resistance and introgression to elite cultivars through molecular breeding. Additionally, novel biotechnological approaches like RNAi, transgenics and genome-editing approaches can also be used to generate a new resistance spectrum against *R. solani*. There are several reviews made previously on the sheath blight tolerance in rice, but most of which provide relatively less focus on breeding for resistance. In the present review, we have made a comprehensive update on the understanding of the pathophysiology of *R. solani* keeping in view crop varietal improvement and biological management of the sheath blight disease in rice. The review also summarizes a critical analysis of the pathogen diversity, host range, pathogenicity and genetics of rice plant resistance. Various approaches adopted in managing the disease through development of resistant varieties have also been described including the novel biotechnological approaches.

**DIVERSITY OF R. SOLANI**

**Morphological Diversity Based on Anastomosis of Vegetative Hyphae**

Anastomosis is a key process for a large number of filamentous fungi that facilitates the fusion of cell walls, cytoplasm and nucleus between genetically similar groups. An anastomosis group (AG) is a collection of closely related isolates grouped based on the ability of vegetative hyphae to anastomose/fuse with one another (Parmeter et al., 1969). *R. solani* is classified into different AGs based on their hyphal capability to fuse with tester hyphal mycelium (Carling, 1996; Craven et al., 2008). The fungus is assigned with fourteen different AGs starting from AG1 to AG13 and AGB1 as a bridging group. The 14 AGs exhibit wide variation in morphology of mycelial colony, nutritional requirement, host range and pathogenic virulence (Carling et al., 2002a,b; Ajayi-Oyetunde and Bradley, 2018).

The anastomosis grouping of *R. solani* causing sheath blight of rice indicated that it belonged to AG1 group. Further grouping of AGs into different intraspecific subgroups (ISGs) have been carried out based on their DNA sequence and its homology, colony morphology, pathogenicity, isozyme pattern, rDNA-internal transcribed sequences and fatty acid composition. Classification of AG1 resulted in three subgroups, AG1-IA, AG1-IB, and AG1-IC, all causing blight (Ogoshi, 1987; Sneh et al., 1991; Carling, 1996). Among these, majority of the rice sheath blight pathogen belongs to the AG1-IA subgroup.

**GENETIC VARIABILITY IN R. SOLANI**

Considerable morphological, pathogenic and genetic diversity has been established within *R. solani* isolates obtained from different parts of the world (Shu et al., 2014; Yugander et al., 2015). Taheri et al. (2007) could group a set of 150 isolates of *R. solani* collected from different parts of India into 33 groups at an 80% genetic similarity level using amplified fragment length polymorphism markers. Twenty-nine isolates from Bangladesh were grouped into two clusters by Ali et al. (2004) while Moni et al. (2016) grouped 18 isolates into four clusters. However, there was no significant correlation between virulence variation and genetic groups identified based on random amplified polymorphic DNA (RAPD) markers (Yi et al., 2002). In China, 175 isolates of *R. solani* belonging to AG1-IA showed considerable variability in virulence (Wang et al., 2015c). They could classify the isolates into weakly virulent, moderately virulent and highly virulent classes based on disease severity, which represented 28.0, 63.4 and 8.6% of isolates, respectively. Further establishing the genetic variability, as many as 80 alleles were detected using RAPD markers from 25 *R. solani* isolates collected from different geographic regions of India (Singh et al., 2015). The number of alleles per locus varied from 1 to 7.

Initially, the genome size of *R. solani* was estimated to be between 36.9 and 42.5 Mb with 11 chromosomes ranging in size from 0.6 to 6 Mb (Keijer, 1996). Later, a draft genome sequence of *R. solani* AG1-IA strain with a size...
of 36.94 Mb was released using next-generation sequencing technology (Zheng et al., 2013). Subsequently, another draft genome sequence of *R. solani* AG1-IA strain, 1802/KB (GenBank accession number KF312465) isolated from a popular rice variety from Malaysia, was generated with a size of 28.92 Mb (Nadarajah et al., 2017). Besides, a web-based database, RSIADB was constructed using the genome sequence (10489 genes) and annotation information for *R. solani* AG1-1A to analyze its draft genome and transcriptome (Chen et al., 2016).

**Host Range**

*Rhizoctonia solani* is pathogenic against a diverse range of about 250 host plant species belonging to members of Poaceae, Fabaceae, Solanaceae, Amaranthaceae, Brassicaceae, Rubiaceae, Malvaceae, Asteraceae, Araceae, Moraceae, and Liliaceae (Chahal et al., 2003). As many as 188 plant species belonging to 32 families were found to be infected by this fungus in Japan (Kozaka, 1961). Tsai (1974) reported *R. solani* infection in 20 species of 11 families in Taiwan, while it was found to infect 10 types of grasses and a *Cyperus* spp. in Thailand (Dath, 1990). In India, it has been reported on 62 economically important plants and 20 families of weeds (Roy, 1993). Several weed plant species have been identified to act as collateral hosts for the pathogen in absence of rice plants (Acharya and Sengupta, 1998), and serve as inoculum and aid in further spread of the disease (Kannaiyan and Prasad, 1980; Srinivas et al., 2014).

**Disease Symptoms**

On infection, the fungus causes a range of symptoms including sheath blight, foliar blight, leaf blight, web-blight, head rots, bottom rot and brown patch in different crops. In rice, *R. solani* mainly attacks the leaf sheath and leaf blades and in severe cases, the whole plant including the emerging panicles may be affected (Rangaswami and Mahadevan, 1998). The disease symptoms on the infected plant can be visualized within 24–72 h after infection depending on the environmental conditions. Although the disease can occur at any growth phase, rice crop is most vulnerable at the tillering phase (Singh et al., 1988). Fungal mycelium determines the size and shape of lesions which are produced in patches of varying sizes (Ou et al., 1973). The typical symptom (Figure 1) is the appearance of greenish-gray water-soaked lesions on the leaf sheath near the water level that are circular, oblong or ellipsoid and about 1 cm long. These lesions enlarge and attain irregular shape, the center of which becomes gray white with brown margins. Lesions may appear on any part of the sheath and several lesions may coalesce to encircle the whole stem. Under favorable conditions, the infection may spread to upper leaf sheaths and leaf blades, which ultimately results in the rotting of leaf sheath and drying up of the whole leaf. In severe cases, the infection spreads to the panicle affecting grain filling and leading to the discoloration of seeds with brownish-black spots or black to ashy gray patches (Singh et al., 2016). In acute cases, the disease causes the death of the whole leaf, tiller and even the whole plant. At the field level, the infection usually affects the plants in a circular pattern referred to as 'bird’s nest' (Hollier et al., 2009).

**The Disease Cycle**

*Rhizoctonia solani* is a seed- and soil-borne pathogen, which survives through sclerotia and mycelia in infected seeds or soil in tropical environments. In soil, infected plant debris is the major carrier that may arise from rice or weed hosts (Figure 2). In temperate regions, soil and crop residue borne sclerotia act as the primary source of inoculum, which can spread through irrigation water from one field to another (Kozaka, 1970). Under favorable conditions, the sclerotia germinate to form mycelia, which on establishing contact with the rice plant surface grows and produces infection structures such as infection cushions and lobate appressoria. These infection structures aid mycelial penetration into the plant tissues. However, in some cases, infection occurs through stomata, where no infection structures are observed (Marshall and Rush, 1980). The pathogen spreads both vertically and horizontally with a horizontal spread of up to 20 cm/day under field conditions is reported (Savary et al., 1995). Plant to plant and field to field spread of the disease takes place through floating sclerotia and mycelia dispersed through rainfall and irrigation water runoff. Infected seeds are the primary source of inoculum for the spread of this disease to new areas. The seed infection and transmission of the pathogen from seed to seedlings in the form of lesions varies from 4.6–14.0% under field conditions (Sivalingam et al., 2006). Wind also helps in the secondary spread of the disease by dispersing the basidiospores to new fields. The basidia hymenium acts as a continuous source of secondary inoculum.

**GEOGRAPHICAL DISTRIBUTION OF R. SOLANI**

Since its first report in Japan in 1910, the pathogen has spread to most of the all the rice growing areas in the world (Figure 3). This disease is recognized as a serious problem in the top ten rice growing countries viz. China, India, Indonesia, Bangladesh, Vietnam, Thailand, Burma, Philippines, Pakistan and Brazil (Singh et al., 2016). Incidence of sheath blight disease of rice in India was reported for the first time from Gurdaspur in Punjab (Paracer and Chahal, 1963). Later on, the disease has become a major problem in rice producing areas of eastern Uttar Pradesh, Uttarakhand, Bihar, West Bengal, Haryana, Odisha, Chhattisgarh, Tamil Nadu, Kerala, Karnataka, Andhra Pradesh, Jammu and Kashmir, Madhya Pradesh, Assam, Tripura and Manipur. The disease incidence was particularly severe among the high yielding semi-dwarf rice varieties, owing to their narrow genetic base, high dependency on chemical fertilizers and favorable weather. Due to the widespread incidence, economic losses to the tune of up to 58% in rice yield have been reported (Chahal et al., 2003).

**Pre-disposing Factors Affecting the Epidemiology**

High ambient air temperature in combination with high relative humidity in the forenoon and wet leaves are major predisposing factors for sheath blight development in rice (Castilla et al., 1996; Biswas et al., 2011). Favorable temperature and evaporation rate results in 23.0 and 61.1% of disease incidence under field...
FIGURE 1 | Symptom of sheath blight disease in rice; left side shows the initial symptoms appear on leaf sheath starting from water level, and the right side shows the disease spread up to panicle.

FIGURE 2 | Disease cycle of sheath blight of rice caused by *Rhizoctonia solani* AG1-IA.
conditions, respectively (Lenka et al., 2008). The maximum progression of the disease is observed at the temperature range of 25°–30°C and relative humidity of 80–100% (Thind et al., 2008; Bhunkal et al., 2015a). The disease severity and yield loss increase with excess nitrogen application (Tang et al., 2007), and are accentuated in the presence of brown plant hopper and rice root-knot nematode, *Hirschmaniella oryzae* (Dath, 1990) and rice tungro virus (Sarkar and Chowdhury, 2007). Another factor under which severe incidence is seen is when the crop canopy is dense with high contact frequency between tissues (Huang et al., 2007). There is also a difference seen between the disease incidence among two sub-species of rice, *indica* and *japonica*, with the former having relatively higher tolerance than *japonica*. However, Lee and Rush (1983) reported that *japonica* cultivars with short and medium grains have higher resistance than long grain *indica* rice cultivar from the southern United States. Indicating the importance of nitrogen, Dath (1990) found a reduction in disease severity with the use of slow-release nitrogenous fertilizer such as Crotonylidene diurea (CDU) and Guanyl urea phosphate with the solo application of silica, phosphorus and potash. Increased dose of nitrogen and phosphorus reduces the incubation period as well as phenolic contents, leading to high disease severity, while application of K, Zn, S, and Fe reduce disease severity (Prasad et al., 2010). Application of soil amendments including neem cake, farm yard manure (FYM), vermicompost and rice husk (Senapoty, 2010) and spraying Ganoderma diethyl ester formulation (Sajeena et al., 2008) can reduce the disease incidence. Long-term field experiments revealed that *R. solani* sclerotia population and sheath blight disease severity remained low in conventional seeded plots as compared to stale seedbeds and no-till seedbeds (Cartwright et al., 1997). Minimal tillage also promotes sheath blight development (Rodriguez et al., 1999). Besides, the rate of infection was less in direct-seeded rice than in transplanted rice irrespective of spacing. Certain crop cycles can also influence the disease incidence pattern as seen with soybean in rotation with rice which leads to a heavy incidence of sheath blight (Rodriguez et al., 2003; Groth and Bond, 2007).

### Host-Pathogen Interaction Between Rice and *R. solani*

To colonize and establish the disease in rice plants, *R. solani* employs a variety of tactics. Effector proteins are used by pathogens to infect the host plant and cause disease. *R. solani* is known to produce several effector molecules (*Table 1*) with varying functions enabling successful colonization. The primary requirement for *R. solani* infection is the degradation of the plant cell wall. *R. solani* AG1-IA is predicted to produce as many as 223 carbohydrate-active enzymes (CAZymes) such as glycoside hydroxylases, glucosyltransferases, and polysaccharide lyases (Zheng et al., 2013). Polygalacturonase hydrolyses the pectin in the plant cell wall, which results in cell death (Chen et al., 2017). During the infection process, the pathogen secretes oxalate and transgenic rice plants overexpressing oxalate oxidase break oxalate and enhance resistance against sheath blight (Molla et al., 2013). *R. solani* has also been reported to use α-1,3-glucans to mask the chitin on its surface and evade the host defense mechanism (Fujikawa et al., 2012). When an extracellular signal is received, the fungi activate different signal transduction pathways for pathogenicity. One of them is the membrane-bound heterotrimeric guanine nucleotide-binding (G) protein-mediated signaling (Li et al., 2007). The Gα subunit of G protein upon activation regulates downstream effectors, such as adenylyl cyclase, phospholipase, ion transporters, and mitogen-activated protein kinase (MAPK) involved in various biological processes including pathogenicity (Neves et al., 2002). Li et al. (2007) reported that two G proteins (Gβ and Gγ) regulate pathogenesis...
TABLE 1 | List of effector molecules related to R. solani colonization in rice plant.

| Effector Molecules | Properties | Function | Defense response compromised in rice plant | References |
|-------------------|------------|----------|-------------------------------------|------------|
| AGLIP1            | Lipase     | Signal peptide and active sites of AGLIP1 play a role in inducing cell death in rice protoplasts | flg22- and chitin-triggered PR genes expression suppressed | Li et al., 2019 |
| RpPG2             | Polygalacturonase (Cell-wall degrading enzyme) | release of reducing sugar and induce rice sheath tissue necrosis | Hydrolysis of the α-1, 4-glycosidic linkage of β-galacturonic acid in pectin in the plant cell-wall | Chen et al., 2017 |
| AG1A_04727        | Polygalacturonase | α-1, 3-glucon polysaccharide α-1, 3-glucon mask cell wall chitin of R. solani which is non-degradable in plants | Pattern Recognition Receptors in rice do not recognize α-1, 3-glucon masked chitin | Rao et al., 2019 |
| CAZYmes (Cell-wall degrading enzymes) | Polysaccharide | cell wall degradation | Various glycoside hydrolases, glucosyl transferases, and polysaccharide lyases cause depolymerization of the host cell wall and colonization of the pathogen | Zheng et al., 2013; Ghosh et al., 2014 |
| AG1A_09161        | Glycosyltransferase GT family 2 domain | Attachment of fungal pathogen and cell wall degradation |  |  |
| AG1A_05310        | Cytochrome C oxidase assembly protein CtaG/cox11 domain | programmed cell death in host plant |  |  |

by monitoring the adenylate cyclase and MAP kinase pathway. Rga1, a G protein subunit gene, affects pathogenicity and its disruption decreased vegetative growth and pathogenicity of the rice sheath blight pathogen (Charoenphophat et al., 2008). The genome sequence of R. solani AG1-1A revealed that a group of secondary molecules including G protein-coupled receptors (GPCR), G protein subunits, MAPK pathway, cAMP pathway and calcium–calcineurin pathway genes may play a major role in pathogenesis (Zheng et al., 2013).

When a pathogen attacks a plant, the plant uses various pathways and defense mechanisms to prevent it from colonizing. On infection by R. solani, rice plants respond by activating various signaling pathways and producing antimicrobial compounds. The plant immune system is of two types, PTI (Pathogen-associated molecular triggered immunity) and ETI (Effector-triggered immunity). PTI is the first line of defense in plants, which is initiated when pattern recognition receptors (PRRs) recognize non-self molecular patterns from pathogens. PTI induces a relatively weak immune response that restricts colonization by invading organisms. ETI, the second line of defense, is initiated when a cognate resistance (R) protein directly or indirectly recognizes highly variable pathogen molecules called avirulence (Avr) effectors and induces a hypersensitive reaction (Liu W. et al., 2014). Pathogenesis related proteins (PR proteins) are produced by the host plant only in pathological or related stress situations. PR3 and PR4 families of chitinases that hydrolyze the β-1,4 linkages between N-acetylglucosamine residues of chitin, a structural polysaccharide of the cell wall of R. solani are differentially induced in rice plants. Chitin fragments are recognized by LysM receptor-like proteins (Gust et al., 2012). POC1, a cationic pathogen-induced peroxidase is upregulated in rice on R. solani infection (Taheri and Tarighi, 2010). Most PRs are induced by the action of salicylic acid (SA), Jasmonic acid (JA), or ethylene (ET), and possess antimicrobial activities. A JA-deficient rice mutant, Heibiba, exhibited enhanced susceptibility to the sheath blight disease (Taheri and Tarighi, 2010). It was found that transgenic plants overexpressing WRKY30 could improve disease resistance by accumulating more JA and conferred resistance to sheath blight by activating the JA/ET signaling cascade. Transcriptome analysis of sheath blight resistant and susceptible rice cultivars infected with R. solani led to the identification of 7624 differentially expressed genes (DEGs), mainly associated with cell wall, β-glucanase, respiratory burst, phenylpropanoids and lignin (Yuan et al., 2018; Molla et al., 2020).

MANAGEMENT OF SHEATH BLIGHT DISEASE

Currently, sheath blight disease of rice is largely managed through the use of fungicides, utilization of genetic resistance/tolerance, cultural practices and biological control are also strategically adopted in the integrated management. Although rice germplasm shows diverse responses to R. solani infection, yet, none of the rice varieties, landraces, weedy types or wild relatives have been identified as immune or completely resistant to this disease. However, some of the genotypes have been found to be partially resistant.

Chemical Control

In the absence of effective host plant resistance against sheath blight pathogen in rice, the management of sheath blight disease is mainly carried out through the use of chemicals (Naik et al., 2017). Foliar spray and seed treatment are the most popular method of fungicidal application against R. solani. Even though both systemic and non-systemic fungicides are used for chemical management, systemic fungicides offer better management...
of this disease (Naik et al., 2017). Timely application of selective fungicides between panicle differentiation and heading stage offers effective protection against this disease. Periodical monitoring of the rice field and application of fungicides at the initial stages of infection especially at booting stage is recommended for managing sheath blight in susceptible varieties (Singh et al., 2016; Uppala and Zhou, 2018).

Several chemical formulations are in use for the control of sheath blight in rice (Table 2). The major focus in the development has been on the identification of fungicides with novel target sites and diverse modes of action. Presently, the Strobilurin group of systemic fungicides are the most preferred chemical group to manage sheath blight disease in rice (Yellareddygari et al., 2014). Strobilurin group of fungicides are derivatives of β-methoxy acrylates and are obtained from forest-grown wild mushrooms (Strobilurus tenacellus). Azoxystrobin from this group is very effective for not only controlling the disease but also found to enhance yield as well (Groth and Bond, 2007). Triazole fungicides are also commonly used in sheath blight management. Application of other chemicals such as Flutolanil, Carbendazim, Iprobenfos, Mancozeb, Thifluzamide and Validamycin also offers effective control of this disease.

The use of a single chemical with the same mode of application for a prolonged time leads to the evolution of resistance in the fungus (Uppala and Zhou, 2018). Hence, a combinatory chemical formulation such as Azoxystrobin 18.2% + Difenoconazole 11.4% (Bhuvaneswari and Raju, 2012; Kumar et al., 2018); Propiconazole + Difenoconazole (Kandhari, 2007); Prothiocarbazole + Tebuconazole 240 g/kg SC (Chen et al., 2021). Captan 70% + Hexaconazole 5% (Pramesh et al., 2017); Trifloxystrobin 25% + Tebuconazole 50% (Shahid et al., 2014; Rashid et al., 2020); Carbendazim + Mancozeb (Prasad et al., 2006; Kumar et al., 2013); Carbendazim 25% + Flusilazole 12.5% SE (Sanjay et al., 2012) etc., are recommended to manage the disease. The chemical method of control is applicable for all areas, irrespective of varieties and has an advantage in a reduction in disease occurrence, spread and enhance yield. However, it has several disadvantages such as environmental hazards that could deteriorate soil health, and cause groundwater pollution. The toxic residue may enter the food chain affecting the health of both humans and animals. It is difficult for a new chemical to have a balancing role in disease management and environmental safety. Therefore, the use of non-chemical control options like cultural, biological, and development and use of resistant varieties offers a viable solution to sheath blight management.

### Table 2

| Chemical group     | Active ingredient (a.i.) | Trade name | Target site                           | Dosage* (g/ha) | References |
|--------------------|--------------------------|------------|---------------------------------------|----------------|------------|
| Strobilurin        | Azoxystrobin 23%EC       | Amistar    | Respiration: inhibition of Cytochrome bc1 at Quinone out site | 125            | Sanjay et al., 2012 |
|                    | Kresoxim-methyl          | Sovran     |                                       | 250            | Bag et al., 2016 |
|                    | Trifloxystrobin          | Flint      |                                       | 150            | FRAC, 2021 |
|                    | Fluoxastrobin            | Aftershock |                                       |                |            |
|                    | Pyraclostrobin           | Insignia   |                                       | 75–100         |            |
| Triazole           | Difenoconazole 25%EC     | Score      | Sterol biosynthesis in the cell membrane | 62.5–125       | Kandhari, 2007 |
|                    | Hexaconazole 5% EC       | Contaf     |                                       | 50             | Naik et al., 2017 |
|                    | Flusilazole 40%EC        | Cursor     |                                       | 120            | FRAC, 2021 |
|                    | Tebuconazole 25.9%EC     | Folcure    |                                       | 187.5          |            |
|                    | Propiconazole 25%EC      | Tilt       |                                       | 125            | Kumar et al., 2013 |
| Phenyl-benzamides  | Flutolanil               | Prostar    | Respiration: an inhibitor of Succinate dehydrogenase | 560            | Prasad et al., 2006; Kandhari, 2007 |
|                    | Carbendazim 50% WP       | Bavistin   | Cytoskeleton: assembling of β-tubulin during mitosis | 250            |            |
|                    | Iprobenfos 48%EC         | Kitazin    | Lipid synthesis: methyltransferase    | 240            | Kumar et al., 2013 |
|                    | Mancozeb 35%SC           | Dthane M-45| Multi-site contact activity            | 875            | Prasad et al., 2006; FRAC, 2021 |
|                    |                        |            |                                       |                |            |
| Carboxamide        | Thifluzamide 24% SC      | Spencer    | Respiration: NADH oxireductase        | 375            | Sunder et al., 2003 |
|                    | Fluxapyroxad             | Monoceren  | Inhibition pathogen mycelial growth   | 100            | Chen Y. et al., 2014 |
| Phenyliureas       | Pencycuron 22.9%SC       | Monoceren  | Cytoskeleton:—cell division           | 187.5          | Kumar et al., 2013 |
| Glucopyranosyl antibiotic | Valdimycin            | Sheathmar  | Inhibition of trehalose                | 60             | Miyagi, 1990 |
| Nano Particle -Fungicides | Halogen substituted Azomethines |          | Tested effective against sheath blight |                | Siddhartha et al., 2020 |
|                    | Silver and Gold Nanoparticle |          | Reduces the radial growth of pathogen |                | Das and Dutta, 2021 |

*Active ingredient (g/ha).
Cultural Practices

Historical records on varietal susceptibility, prior disease incidence, prevailing weather conditions and disease spread help in devising appropriate cultural practices for managing sheath blight disease of rice (Singh et al., 2019). Agro-morphological traits of rice including plant height, stem thickness and tiller angle, length and width of flag leaf, days to heading and planting density affect the susceptibility of rice to *R. solani*.

Plant height has been found to show a strong negative association between relative lesion length (Willocquet et al., 2012). Wider spacing reduces the sheath blight severity by improving the canopy thickness. Split application and use of slow-releasing nitrogenous fertilizers have been found to reduce sheath blight infection (Roy, 1986). The effect of dose of nitrogen fertilizer on disease spread has been higher than the effect of plant density (Zhang et al., 1995). Similar to nitrogen, higher doses of phosphorous fertilizers increase the disease incidence, while potassic fertilizers have been found to reduce it (Sarkar et al., 1991). Silicon application to rice fields through carbonized rice husk helps delay the disease spread without any negative effect on yield (Sabes et al., 2020). A waste product from charcoal production (Bamboo tar) was reported to inhibit multiple diseases including rice sheath blight (Maliang et al., 2021). Timely removal of weeds which are alternate host for *R. solani*, removal of plant debris, crop rotation with non-host crops reduces the sheath blight incidence by minimizing the primary inoculum sclerotia (Singh et al., 2019).

Biological Control

In addition to chemical and cultural control, biological control has been suggested as a very promising strategy to manage necrotrophic fungus. Plant extracts or botanicals are very effective in managing the disease. Extracts from garlic, ginger, neem leaf and clove inhibit more than 80% mycelial growth in *R. solani* (Chakrapani et al., 2020; Rajeswari et al., 2020). Microbial antagonism is a common property found between microorganisms and it is most predominant among soil microbes. This effect of antagonism between the pathogen and beneficial microbes in the soil will lead to a reduction in disease development to a greater extent. There are several biocontrol agents (BCAs) belonging to actinomycetes, fungi and bacteria. Actinomycetes colonize the plant roots and represent a greater portion of the rhizosphere microflora. Actinomycetes against *R. solani* in tomatoes could reduce the disease incidence by up to 63% (Singh et al., 2017). One of the most common actinomycetes, *Streptomyces* spp. is reported to reduce the growth of *R. solani* up to 50% and disease suppression up to 53.3% (Patil et al., 2010). Ethyl acetate extracted from *Streptomyces diastatochromogenes*, KX852460 have been found to inhibit mycelial growth, reduce sclerotia formation and suppress lesion length on *R. solani* AG3 (Ahsan et al., 2019). Another group of potential BCAs mostly used against *Rhizoctonia* is fungal antagonists. Many species of *Trichoderma, Corticium, Aspergillus* and *Gliocladium* have been used for managing sheath blight disease (Chinnaswami et al., 2021). For effective management, these BCAs are applied as a soil treatment, foliar spray and root dipping of seedlings. Different strains of *Trichoderma* have been reported to inhibit *Rhizoctonia* growth by up to 71% and reduce the sheath blight infestation by up to 59% (Mishra et al., 2020). *Trichoderma* can be applied alone or in combination with other BCAs like Vesicular arbuscular mycorrhiza, *Pseudomonas* and yeasts for both controlling the pathogen and supplementing growth factors (Mathivanan et al., 2005; Mohammed et al., 2020). Plant growth-promoting rhizobacteria (PGPR) are the most common group of bacterial BCAs used against a wide range of plant pathogens for disease reduction. PGPR also helps in increasing root growth, phosphate solubilization, nitrogen uptake, iron-chelating siderophores and phytohormone synthesis. Among the different PGPR, *Pseudomonas* and *Bacillus* provide an effective way of systemic resistance against sheath blight. Rice seedlings treated with different strains of *Pseudomonas fluorescens* helped to increase the chitinase activity responsible for the suppression of sheath blight disease (Radjacomare et al., 2004). *Bacillus* sp. having a broad range of antibiotic properties was also very useful in reducing the growth of *Rhizoctonia* (Abbas et al., 2019; Raj et al., 2019). The combination of *Bacillus subtilis* strain MBI600 with Azoxystrobin helps not only disease suppression but also increases the yield to 14% (Zhou et al., 2021). In a recent study, three strains of nitrogen-fixing cyanobacteria have been reported to significantly inhibit the growth of *R. solani* (Zhou et al., 2020). However, the effectiveness of BCAs in sheath blight is influenced by their ability to survive, multiply and control pathogens and also provide additional supplements promoting rice growth. Nanoparticles of Gold and Silver have antifungal activity against *R. solani* (Das and Dutta, 2021). Recently, silver nanoparticles from rice leaf extract have been reported to be very effective against *R. solani* infection in rice (Kora et al., 2020). Different biocontrol agents were screened against sheath blight for their timing of application in a greenhouse environment, treatment of these bio fungicides before pathogen inoculation has a great role against the disease (Tuyen and Hoa, 2022). Eugenol from clove (Syzygium aromaticum L.) has been found to control this pathogen by dehydrating the cell and increasing the cell membrane permeability (Zhao et al., 2021).

CROP IMPROVEMENT STRATEGIES AGAINST *R. SOLANI*

Theoretically breeding for sheath blight resistance is mainly based on two approaches, disease escape and disease resistance. Disease escape mainly consists of plant architectural traits including plant height, heading date and stem thickness (Sattari et al., 2014; Susmita et al., 2019). The standard protocol for screening for disease resistance is based on relative lesion length (RLH) which is calculated in the percentage of ratio lesion height to plant height (Sharma et al., 1990; IRRI, 1996). Conventional breeding is more difficult in this case because of the direct influence of plant height on RLH during its screening protocol. Hence marker assisted breeding is highly preferred for the introgression of identified resistance QTLs. Marker assisted breeding has several advantages over conventional breeding as it helps in accurate selection of desired genotypes, saves time during selection,
reduces linkage drag during introgression of genomic regions and helps in easier gene pyramiding.

**Donors for Resistance**

Development of resistant rice varieties through genetic improvement is a sustainable option for managing plant diseases. Since there are no genotypes with absolute resistance, identification of reliable resistance sources must be confined to the moderate to high levels of tolerance in the germplasm. There are several such genotypes reported (Table 3) that are being used in breeding sheath blight resistant cultivars. Among the cultivated species, the * indica* cultivars are reported to show better resistance than the * japonica* type (Liu et al., 2009; Willocquet et al., 2012). Additionally, some accessions of wild species such as *O. rufipogon*, *O. nivara*, *O. meridionalis* and *O. barthii* have been reported to be resistant to sheath blight disease (Prasad and Eizenga, 2008; Bashyal et al., 2017).

**Genetics and Analysis of Quantitative Resistance**

Several earlier studies indicate that the tolerance against sheath blight disease in rice is a quantitative trait governed by polygenes (Xu et al., 2011; Kosheriya et al., 2018). Therefore, it is essential to map the genomic regions governing quantitative variation for tolerance among the source germplasm. Attempts on mapping quantitative trait loci (QTLs) have been taken up in rice for sheath blight tolerance. One of the earliest attempts by Li et al. (1995) used RFLP markers in an *F4* population derived from Lemont/Teqing. Lemont was a highly susceptible * japonica* cultivar, while Teqing was a semidwarf high yielding Chinese * indica* variety with high tolerance to leaf blight. Since then a large number of QTLs governing resistance to sheath blight disease have been reported across all the 12 chromosomes of the rice genome (Table 4). A map showing the physical location of the reported QTLs and the linked markers is presented in Figure 4. Most of the earlier mapping populations were based on the partially resistant * indica* genotypes such as Teqing and Jasmine 85 and the susceptible * japonica* genotype, Lemont (Li et al., 1995; Pan et al., 1999; Wen et al., 2015). Using these mapping populations, a large number of QTLs governing sheath blight resistance have been mapped (Li et al., 1995; Zou et al., 2000; Liu et al., 2009; Eizenga et al., 2015). Eizenga et al. (2013, 2015) also identified resistance sources from wild accessions of *O. nivara* and *O. meridionalis*. QTLs for resistance have been mapped from weedy rice also (Goad et al., 2020; Jia et al., 2022). Goad et al. (2020) reported four QTLs from RIL populations generated by crossing the rice cultivar, Dee-Geo-Woo-Gen (DGWG) with two weed species (straw hull and black hull awned). Yuan et al. (2019) utilized a RIL population from Lemont/Yangdao4 to map 128 minor effect QTLs, most of which clustered around 17 stable loci across the rice genome.

**Genome Wide Association Studies**

Identification of genomic regions associated with sheath blight resistance has also been carried out using genome wide

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**TABLE 3** | Rice genotypes identified as sources of resistance to sheath blight disease.

| Source of resistance | References |
|----------------------|------------|
| Dudiosr, NC 678, Bhasamanik | Das, 1970 |
| Zenith, Chin-Kou-tsao, CO17 | Wu, 1971 |
| Lalatarkara | Roy, 1977 |
| ARC 18119, ARC15762 | Bhakta-Vatsalam et al., 1978 |
| Jaya, IR24, IR26, IR29, Mashoori, Jaggaranath | Rajan and Nair, 1979 |
| Tapachoor, Lakka, Bahagia | Cift et al., 1982 |
| Tapoo cho 2, Tetep, Bharati Rohini | Gokulapulap and Nair, 1983 |
| Chidion, Dholamula, Supherku, Taraboli 1 | Borthakur and Addy, 1988 |
| Tetep | Sha and Zhu, 1989 |
| BPT-6, Bogli, MTU 3, MTU 3642, MTU 7, MTU 13, Saket, Arkavati, Aduthurai | Channamalikarjuna et al., 2010 |
| LSBR 33, LSBR 5 | Ansari et al., 1989 |
| TIL 842, TIL 455, TIL 514 | Xie et al., 1992 |
| Teqing | Singh and Dodan, 1995 |
| Mairan KK2, As 93-1, Camor, Dodan, IR40, Chingdak | Li et al., 1995; Pinson et al., 2005 |
| Jasmine 85 | Marchetti et al., 1996 |
| Mairan, Panjasali, N-22, Chingdak, Upland 2, AS93-1 | Pan et al., 1999; Zou et al., 2000; Li et al., 2009 |
| Minghui 63 | Singh and Borah, 2000 |
| Zhaiqueqin 8, Jingei 17 | Han et al., 2002 |
| Xiangzaoxian 19 | Kunihiro et al., 2002 |
| WSS2 | Che et al., 2003 |
| O. latifolia; DRW 37004, WR 106, DRW 21009, DRW 24008 | Sato et al., 2004 |
| O. nivara; IRGC 104443, IRGC 104705, IRGC 100898 | Ram et al., 2008 |
| O. officinalis; IRGC 105979 | Prasad and Eizenga, 2008 |
| O. meridionalis; IRGC 105306 | Chen et al., 2009 |
| O. barthii; IRGC 100223 | Sharma et al., 2009 |
| C418 | Zu et al., 2009 |
| Pecos | Xu et al., 2011 |
| YSBR1 | Fu et al., 2011 |
| Balseyqiu | Jia L. et al., 2012 |
| RSB03 | Jia Y. et al., 2012 |
| GSOR 310389, GSOR 31147, GSOR 310475 | Nelson et al., 2012 |
| LJRIL103, LJRIL158, LJRIL186, LJRIL220 | Taguchi-Shiobara et al., 2013 |
| MCR10277 | Zhu et al., 2014 |
| Jarjan, Nepal 8, Nepal 555 | Dubey et al., 2014 |
| HUX74 | Liu Y. et al., 2014 |
| Kajrawha, BML 21-1, BPL 7-12, BML 27-1 | Eizenga et al., 2015 |
| RSB02 | Yadav et al., 2015 |
| O. meridionalis; IRGC105608 | Gaihre et al., 2015 |
| ARC10531 | Wei et al., 2015 |
| 2F18-7-32 (32R) | Wu et al., 2015; Yuan et al., 2019 |
| Yangdao 4 | Zeng et al., 2015 |
| TN1 | Dey et al., 2016 |
| Phouak, Gumsham, Nongkolasha, Wazuhophek, SM 801, 10-3 | Bashyal et al., 2017 |
| O. rufipogon; IC336719, IC336721 | Koshihikari et al., 2018; Mandal et al., 2018 |
| Dagad Deshi | Chen Z. et al., 2019 |
| Bico Branco, DOM Zard, Vary Vato462, T26, Peh-Kuh-Tsao, Bombilla, Koshihikari, PR304, Kaukau, Ghati Kmma Nangarhar | Mandal et al., 2018 |
| QTLs | Chr. | Linked markers | Mapping Population | Cross | References |
|------|------|----------------|-------------------|-------|------------|
| qSB1 | 1    | RG332X         | RIL               | Lemont/Tieqing | Pinson et al., 2005 |
| qSB1 |      | RM104          | RIL               | Lemont/Jasmine 85 | Liu et al., 2009 |
| qSB1 |      | RM1339         | F2:3              | Rosemont/Pecos  | Sharma et al., 2009 |
| qSB1-1 |     | HvSSR68       | RIL               | HP2216/Tetep | Channamallakkurjana et al., 2010 |
| qSBH1 |     | RM343-1-RM12017 | DH               | Maybelle/Baiyeqiu | Xu et al., 2011 |
| qSBH1-1 |   | RM5389-RM3825  | RIL               | HH1B/RSB03 | Fu et al., 2011 |
| qSH1-1 |     | RM1361-RM104   | BC2F1            | IRGC100896/Bengal | Ezenga et al., 2013 |
| qSBH1 |     | RM6703-RM5448  | F2                | 32R/Nipponbure | Zhu et al., 2014 |
| qSH1-1 |     | RM151-RM12253  | F2 and BC1 F2    | BPT5204/ARC 1053 | Gahre et al., 2015 |
| qSBH1-1 |   | HsSSR1-87     | RIL               | Danteshwari/Dagad Deshi | Yadav et al., 2015 |
| qSH1-2 |    | RM243          | RIL               | Danteshwari/Dagad Deshi | Koshraya et al., 2018 |
| qSB1-1 |     | RM5            | RIL               | Danteshwari/Dagad Deshi | Koshraya et al., 2018 |
| qSH1-2 |    | RM84           | RIL               | Danteshwari/Dagad Deshi | Mandal et al., 2018 |
| qSBH1-2 |   | SNP            | RIL               | Sh-W and BHAW/Dee-Geo-Woo-Gen | Mandal et al., 2018 |
| qSB2-2 |    | RM243          | F4                | Lemont/Tieqing | Li et al., 1995 |
| qSB2-3 |    | RM654-RZ260    | F4                | Jasmine 85/Lemont | Pan et al., 1999 |
| qSB2 |     | G243-RM29      | F2                | Zhai Ye Qing 8/Jing Xi 1 | Kunihro et al., 2002 |
| qSB2 |     | RM3685         | DH                | Rosemont/Pecos  | Sharma et al., 2009 |
| qSB2 |     | RM174-RM145    | F2:3              | Maybelle/Baiyeqiu | Xu et al., 2011 |
| qSB2 |     | RM5340-RM521   | RIL               | HH1B/RSB03 | Fu et al., 2011 |
| qSB2-1 |   | RM7245-RM5303  | RIL               | HH1B/RSB03 | Fu et al., 2011 |
| qSB2-3 |    | RM8254-RM8252  | RIL               | HH1B/RSB03 | Fu et al., 2011 |
| qSB2-1 |     | RM3857-RM5404  | DH                | MCR10277/Cocodrie | Nelson et al., 2012 |
| qSB2-2 |    | RM221-RM112    | DH                | MCR10277/Cocodrie | Nelson et al., 2012 |
| qSB3 |     | RG348-RG944    | F4                | Lemont/Tieqing | Li et al., 1995 |
| qSB3 |     | R250-C746      | F2                | Jasmine 85/Lemont | Pan et al., 1999 |
| qSB3q |    | RM347-1        | DH                | Zhai Ye Qing 8/Jing Xi 1 | Kunihro et al., 2002 |
| qSB3 |     | RM3856         | BC1 F1           | Hinohikari/WSS2/Hinohikari | Sato et al., 2004 |
| qSB3 |     | RM5626         | RIL               | Lemont/Jasmine 85 | Liu et al., 2009 |
| qSB3 |     | RM5117         | F2:3              | Rosemont/Pecos  | Sharma et al., 2009 |
| qSB3 |     | RM251          | RIL               | HP2216/Tetep | Channamallakkurjana et al., 2010 |
| qSHB3 |    | RM135-RM186    | DH                | Maybelle/Baiyeqiu | Xu et al., 2011 |
| qSHB3 |     | RM232-RM282    | BC2F1            | IRGC100896/Bengal | Xu et al., 2011 |
| qSB3 |     | RM4317-RM6080  | F2                | 32R/Nipponbure | Ezenga et al., 2013 |
| qSB3 |     | D328B-D331B    | F2 and F2:3       | Yangdao 4/Lemont | Gahre et al., 2015 |
| qSHB3 |    | RM232          | RIL               | Danteshwari/Dagad Deshi | Wei et al., 2015 |
| qSB4a |    | RG143-RG214    | F4                | Lemont/Tieqing | Li et al., 1995 |
| qSB4-1 |   | RG1094E        | RIL               | Lemont/Tieqing | Pinson et al., 2005 |
| qSB4 |     | RM3286-RM7187  | RIL               | HH1B/RSB03 | Fu et al., 2011 |
| qSB4 |     | RM3276-RM3843  | F2                | 32R/Nipponbure | Mandal et al., 2015 |
| qSB4-1 |   | RM273          | RIL               | Danteshwari/Dagad desii | Mandal et al., 2018 |
| qSHB4 |    | SNP            | RIL               | Sh-W and BHAW/ DGWG/ DGWG | Gahre et al., 2015 |
| qRsb |     | RM 393000      | F2                | 4011/Xiangzaoxian19 | Che et al., 2003 |
| qSB5 |     | Y1049          | RIL               | Lemont/Tieqing | Pinson et al., 2005 |
| qSB5 |     | RM13           | RIL               | Lemont/Jasmine 85 | Liu et al., 2009 |
| qSB5 |     | RM6545         | RIL               | Lemont/Tieqing | Pinson et al., 2005 |
| qSB5 |     | RM18872-RM421  | DH                | Maybelle/Baiyeqiu | Xu et al., 2011 |
| qSB5 |     | RM122-RM413    | BC2F1            | IRGC100896/Bengal | Ezenga et al., 2013 |
| qSB5 |     | RM1024-RM3419  | F2                | 32R/Nipponbure | Mandal et al., 2015 |
| qSB5-1 |   | HvSSR5-52     | RIL               | Danteshwari/Dagad Deshi | Mandal et al., 2018 |
| qSB5 |     | RM459          | RIL               | Danteshwari/Dagad Deshi | Mandal et al., 2018 |
| qSB6-2 |   | RZ508          | RIL               | Lemont/Tieqing | Pinson et al., 2005 |
| qSB6 |     | RM190          | RIL               | Lemont/Jasmine 85 | Liu et al., 2009 |
| qSB6-1 |   | HvSSR6-35     | RIL               | Danteshwari/Dagad Deshi | Mandal et al., 2018 |
| qSB6 |     | RM3183-RM341   | BC2F1            | IRGC100896/Bengal | Ezenga et al., 2013 |
| qSB6-1 |   | RM400-RM253    | F2 and BC1 F2    | BPT5204/ARC 1053 | Yadav et al., 2015 |
| qSB7 |     | RG30-RG477     | F2                | Zhai Ye Qing 8/Jing Xi 1 | Kunihro et al., 2002 |
| qSB7 |     | C285           | DH                | Lemont/Tieqing | Pinson et al., 2005 |

(Continued)
TABLE 4 | (Continued)

| QTLs | Chr. | Linked markers | Mapping Population | Cross | References |
|------|------|----------------|--------------------|-------|------------|
| qSBR7-1 | 3 | RM1132-RM473 | RIL | HP2216/Tetep | USDA | Sun et al., 2014 |
| qSBR7 | 4 | RM290-RM5111 | RIL | HH1B/RSB03 | USDA | Li et al., 2015 |
| qSHB7 | 5 | RM6728-RM214 | BC2F1 | IRGC100898/Bengal | USDA | Fu et al., 2011 |
| qSB7 | 7 | RM81-RM152 | F2 | 32R/Nipponbare | USDA | Ezengia et al., 2013 |
| qSHB7-1 | 8 | RM10-RM21693 | F2 and BC1F2 | BPT-5204/ARC 1053 | USDA | Gahre et al., 2015 |
| qSHB7-2 | 9 | RM336-RM427 | F2 and BC1F2 | BPT-5204/ARC 1053 | USDA | Yadav et al., 2015 |
| qSHB7-3 | 10 | D760-RM248 | F2 and BC1F2 | BPT-5204/ARC 1053 | USDA | Yadav et al., 2015 |
| qSL7 | 11 | RM5887-RM531 | | | USDA | | |
| qSBR8a | 12 | RM21792-RM510 | F2 and BC1F2 | Yangdao 4/Lemont | USDA | | |
| qSBR8-2 | 13 | RG20-RG1034 | F4 | Lemont/Teqing | USDA | Li et al., 1995 |
| qSBR8-1 | 14 | RG62 | RIL | Lemont/Teqing | USDA | Pinson et al., 2005 |
| qSBR8 | 15 | RM210 | RIL | HP2216/Tetep | USDA | Channamallakarjuna et al., 2010 |
| qSBR8 | 16 | RM8264-RM1109 | RIL | HH1B/RSB03 | USDA | Fu et al., 2011 |
| qSBR8 | 17 | RM5887-RM531 | F2 | 32R/Nipponbare | USDA | Ezengia et al., 2015 |
| qSHB8-1 | 18 | RM21792-RM510 | F2 and BC1F2 | BPT-5204/ARC 1053 | USDA | Gahre et al., 2015 |
| qSBR9a | 19 | RG9 106-RZ777 | F4 | Lemont/Teqing | USDA | Li et al., 1995 |
| qSBR9 | 20 | C979-G103 | F2 | Jasmine 85/Lemont | USDA | Zou et al., 2000 |
| qSBR9-2 | 21 | RG570-CS56 | F2 | Jasmine 85/Lemont | USDA | Zou et al., 2000 |
| qSBR9 | 22 | RM205-RM201 | F2 | Teqing/Lemont | USDA | Tan et al., 2005 |
| qSBR9 | 23 | RM205-RM201 | RIL | | USDA | Liu et al., 2009 |
| qSBR9 | 24 | RM5882 | F3 | Rosemont/Pecos | USDA | Sharma et al., 2009 |
| qSBR9 | 25 | RM257 | RIL | HP2216/Tetep | USDA | Channamallakarjuna et al., 2010 |
| qSBR9 | 26 | RM23869-RM3769 | RIL | HH1B/RSB03 | USDA | Fu et al., 2011 |
| qSBR9 | 27 | RM24070-RM3823 | DH | MCR102777/Cocodrie | USDA | | |
| qSBR9 | 28 | Nang08K18184- | BIL | Jarjan/Koshihikari/Koshihikari | USDA | Nelson et al., 2012 |
| qSBR9 | 29 | Nang08K18871 | BC | IRGC102608/Lemont | USDA | Taguchi-Shiobara et al., 2013 |
| qSBR9 | 30 | RM 257-RM107 | | | USDA | Ezengia et al., 2015 |
| qSBR9-1 | 31 | RM566-RM175 | | | USDA | Gahre et al., 2015 |
| qSBR9 | 32 | RM257-RM242 | F2 and BC1F2 | BPT-5204/ARC 1053 | USDA | Yadav et al., 2015 |
| qSBR9-2 | 33 | RM205-RM105 | F2 and BC1F2 | BPT-5204/ARC 1053 | USDA | Yadav et al., 2015 |
| qSBR9-3 | 34 | RM24060-RM 3744 | F2 | BPT-5204/ARC 1053 | USDA | Yadav et al., 2015 |
| qSBR9 | 35 | RM444 | RIL | Danteshwarz/Dagad desi | USDA | Mandal et al., 2018 |
| qSB10 | 36 | RGS61 | RIL | | USDA | Pinson et al., 2005 |
| qSB11 | 37 | G44-RG118 | F2 | Jasmine 85/Lemont | USDA | Zou et al., 2000 |
| qSB11 | 38 | RM167-R529 | F2 | Teqing/Lemont | USDA | Tan et al., 2005 |
| qSB11 | 39 | RM224 | RIL | HP2216/Tetep | USDA | Channamallakarjuna et al., 2010 |
| qSB11 | 40 | RM209 | RIL | HP2216/Tetep | USDA | Channamallakarjuna et al., 2010 |
| qSB11 | 41 | RM202 | RIL | HP2216/Tetep | USDA | Channamallakarjuna et al., 2010 |
| qSB11 | 42 | RM332-RM21 | BC2F1 | IRGC100898/Bengal | USDA | Ezengia et al., 2013 |
| qSB11 | 43 | InDel Markers | CSSL | HX574/Amid3 | USDA | | |
| qSB11 | 44 | D1103-RM26155 | F2 and F2.3 | Yangdao 4/Lemont | USDA | | |
| qSB12 | 45 | RG214a-RZ397 | F4 | Lemont/Teqing | USDA | Sato et al., 2004 |
| qSB12 | 46 | RM1880 | BC2F1 | Hinohikari/WSS2/Hinohikari | USDA | Pinson et al., 2005 |
| qSB12 | 47 | G1106 | RIL | Lemont/Teqing | USDA | Pinson et al., 2005 |
| qSB12 | 48 | RM3747-RM27608 | BC1F2 | MCR102777/Cocodrie | USDA | Nelson et al., 2012 |
| qSB12 | 49 | RM5746-RM277 | BC1F2 | IRGC100898/Bengal | USDA | Ezengia et al., 2013 |
| qSB12 | 50 | RM1246-D1252 | F2 and F2.3 | Yangdao 4/Lemont | USDA | | |
| qSB12 | 51 | RM260 | RIL | Danteshwarz/Dagad Deshi | USDA | Wen et al., 2015 |
| qSB12 | 52 | RM277 | RIL | Danteshwarz/Dagad Deshi | USDA | Koshariya et al., 2018 |
| qSB12 | 53 | RM20 | RIL | Danteshwarz/Dagad Deshi | USDA | Mandal et al., 2018 |

**SHW, Straw hull weeed; BHAW, Black hull awned weeed; DGWG, Dee Geo Woo Gen.**

association studies but on a limited scale. Jia L. et al. (2012) identified 10 marker-trait associations (MTAs) and three genotypes for resistance from a set of 217 core entries from USDA using 155 genome-wide simple sequence repeat (SSR) markers. Using a larger population of 456 rice accessions, Sun et al. (2014) identified 10 significant MTAs with 144 SSR markers. Chen Z. et al. (2019) reported 11 MTAs and two QTLs, qSB3 and qSB6 by screening 299 rice varieties.
with 44K SNPs. GWAS with 228 rice accessions genotyped with 700,000 SNPs identified two major MTAs associated with sheath blight resistance in rice (Oreiro et al., 2020). Zhang et al. (2019) identified 562 MTAs for lesion height, 134 for culm length and 75 MTAs for relative lesion height through GWAS on a set of 563 rice accessions genotyped
with 220,335 SNPs. GWAS was conducted using 259 diverse varieties and identified a regulation model against the disease (Wang et al., 2021).

**Fine Mapping of QTLs**

Although a large number of major and minor effect QTLs have been identified for sheath blight resistance in rice, efforts to fine-map these QTLs have been limited. Chromosome segment substitution lines (CSSLs) are a group of homozygous lines, each having a different chromosome segment from the donor species. Individually one CSSL has a donor segment that overlaps the other donor segment in the next CSSL. Altogether, CSSLs contain the whole genomic DNA of donor species in different segments. The CSSLs eliminate the genetic background effect, and enables, the fine mapping of QTLs (Eshed and Zamir, 1994). Channamalikarjuna et al. (2010) fine mapped a major QTL, qSB-11-1 for sheath blight resistance, which has been narrowed down to 0.85 Mb on chromosome 11. A set of 154 putative genes have been identified within this genomic region, out of which 11 chitinase genes in tandem repeats have been identified as candidate genes governing resistance to sheath blight disease. A major QTL qSB-11LE identified from the first QTL mapping effort (Li et al., 1995) and subsequent studies (Zou et al., 2000; Tan et al., 2005) has been fine mapped to a 78.8 kb genomic region, from which three candidate genes have been identified (Zuo et al., 2013). qSB-9TQ from Teqing has also been fine mapped to a region of 146 kb region using CSSLs (Zuo et al., 2014ab).

**Marker Assisted Breeding for Sheath Blight Resistance in Rice**

Mapping and validation of QTLs are essential for their utilization in marker assisted breeding. Teqing is one of the most frequent donors for the QTLs, qSB7Q, qSB9Q and qSB12Q. Marker assisted introgression of single or multiple of these QTLs were found to reduce the yield loss due to sheath blight disease (Wang et al., 2012; Chen Z. X. et al., 2014). Sheath blight resistance has been enhanced by the introgression of QTL qSB9Q along with QTL for tiller angle, TAG1T (Zuo et al., 2014ab). Yin et al. (2008) introgressed three main effect QTLs namely, qSB7Q, qSB9Q and qSB11Q into Lemont to develop sheath blight resistant genotypes. In India, Tetep has been widely used as a donor source for both sheath blight as well as blast resistance. A major QTL qSB11-1 using ‘Tetep’ was introgressed along with another gene, Pi54 governing blast resistance into a bacterial blight resistant Basmati rice variety, ‘Improved Pusa Basmati 1’ leading to the development of improved near isogenic lines (NILs) with resistance to virulent strains of R. solani (Singh et al., 2012). qSB11-1 and Pi54 have been pyramided into the high yielding variety, COS5 (Senthivel et al., 2021). Gene(s) for multiple diseases resistance including bacterial leaf blight (xa5 + xa13 + xa21), Blast (Pi54) and sheath blight (qSB7-1 + qSB11-1 + qSB11-2) have been pyramided into the background of popular cultivar ASD 16 and ADT 43 using, Tetep and IRBB60 as donors (Ramalingam et al., 2020). Raveendra et al. (2020) introgressed sheath blight resistance from Tetep into the background of bacterial blight resistant genotypes, CB14004 and CB14002.

**Biotechnological Approaches for Managing Sheath Blight Diseases of Rice**

Comparison of transcripts between resistant and susceptible cultivars in response to Rhizoctonia led to the identification of Ethylene-insensitive protein 2, trans-cinnamate-4-monooxygenase and WRKY 33 transcriptome factor (Shi et al., 2020). Rice is endowed with resources and techniques enabling the study of the expression of these pathogen-related (PR) genes, anti-fungal genes and master genes for defense response affecting R. solani growth.

In the absence of stable sources of sheath blight resistance, genetic engineering offers promise in developing novel resistance in rice. Several potential genes from various species have been identified as candidates for engineering resistance against Rhizoctonia solani in rice (Table 5). Chitinase and glucanase are the most widely used genes for engineering resistance against R. solani. Lin et al. (1995) were the first to generate a transgenic line with constitutive expression of a chitinase gene (Chi11) leading to resistance to sheath blight disease of rice. Since then, many studies have demonstrated the effect of overexpression of the chitinase gene in rice. Chitinase cleaves at the β-1,4-glycosidic linkage of N-acetyl-D-glucosamine and glucanase cleaves at the β-1,3 linkage of glucan polymer, arresting the fungal invasion of the host tissues. Recent studies on overexpression of genes like WRKY13 (Lilly and Subramanian, 2019), OsBR2 (Maeda et al., 2019), RGB1 and RGG1 (Swain et al., 2019), LPAI (Sun et al., 2019a, 2020) and OsGSTU5 (Tiwari et al., 2020) have demonstrated the effectiveness of these genes in managing sheath blight of rice. Overexpression of the genes from the WRKY gene family namely, OsWRKY4 (Wang et al., 2015a), OsWRKY13 (Lilly and Subramanian, 2019), OsWRKY30 (Peng et al., 2012) and OsWRKY80 (Peng et al., 2016) have been reported to reduce R. solani infection in rice. A schematic representation of genes being utilized in the development of transgenics with resistance to sheath blight disease along with their mode of action is presented in Figure 5. Constitutive expression of Chi11 and β-1,3 glucanase genes in a transgenic line, Pusa Basmati-CG27, helped to validate their role in conditioning sheath blight resistance, based on which these genes were used in marker assisted improvement of White Ponni (Kannan et al., 2017). Over expression of a basic helix–loop–helix transcription factor (OsbHLH057) with cis-acting AATCA has been reported to be effective against both sheath blight and drought (Liu et al., 2022). Recently, Dauda et al. (2022) identified a set of Cytokinin glucosyltransferases (CGTs) in rice with the plant secondary product glucosyltransferases (PSPG) motif of 44-amino-acid consensus sequence characteristic of plant uridine diphosphate (UDP)-glycosyltransferases (UGTs), the validation of which showed upregulation of four genes namely LOC_Os07g30610.1, LOC_Os04g25440, LOC_Os04g25490, and LOC_Os04g25800 specifically under R. solani infection.
### TABLE 5 | Genes reported for sheath blight resistance in rice.

| Group                        | Gene name                | Function                                                                 | References                        |
|------------------------------|--------------------------|--------------------------------------------------------------------------|-----------------------------------|
| Chitinase                    | OsCHI11                   | Degrades chitin by breaking β-1, 4 linkages                              | Lin et al., 1995                  |
|                              | OsRC7                    |                                                                          | Sridivi et al., 2003              |
|                              | RCH10                    |                                                                          | Datta et al., 2001                |
|                              | Os11g47510                |                                                                          | Kim et al., 2003                  |
|                              |                          |                                                                          | Richa et al., 2017                |
| Antimicrobial peptide        | Ace-AMP1                  | Plant defensin that inhibits pathogen growth                             | Krishnamurthy et al., 2001        |
|                              | Dm-AMP1, Rs-AFP2          |                                                                          | Patkar and Chattoo, 2006          |
|                              | RS-AFP2                  |                                                                          | Jha and Chattoo, 2009             |
|                              | snakin-1                 |                                                                          | Jha and Chattoo, 2010             |
| WRKY transcription factor    | OsWRKY30                 | Positively regulated defense response                                     | Peng et al., 2012                 |
|                              | OsWRKY4                  |                                                                          | Wang et al., 2015a                |
|                              | OsWRKY80                 |                                                                          | Peng et al., 2016                 |
|                              | OsWRKY13                 |                                                                          | Lilly and Subramanian, 2019       |
| Osmotin                      | ap24                     | Plant defense response and Permeability stress                            | De Yuan et al., 2020              |
| Osmotin                      | OsOSM1                   |                                                                          | Shimo no et al., 2012             |
| Oxalate oxidase              | Osixo4                   | Degrade oxalic acid (OA) and reduce the OA accumulation                   | Rao et al., 2011                  |
| Polyalacturonase (PG) inhibiting proteins (PGIP) | OsPGIP1 | Stabilizes the plant cell wall component Pectin | Wang et al., 2015b |
| Polyalacturonase (PG) inhibiting proteins (PGIP) | OsPGIP2C233F | | Chen X. J. et al., 2019 |
| Polyalacturonase (PG) inhibiting proteins (PGIP) | ZmPGIP3 | | Zhu et al., 2019 |
| Mitogen-activated protein (MAP) Kinas  | OsMAPK20-5               | Plant defense response                                                   | Li et al., 2019                    |
| Thaumatin-like protein       | Tlp-D34                  | Co-expression of Tlp with CH reduces disease index                       | Shah et al., 2013                 |
| Ethylene biosynthetic genes  | OsACS2                   | Overexpression of ethylene leads to resistance                            | Hellweil et al., 2013             |
| Non-expressor of pathogenesis related gene | AinPFR1 | Regulator of Systemic Acquired Resistance | Sadumpati et al., 2013 |
| Non-expressor of pathogenesis related gene | BjNPR1 | | Molla et al., 2016 |
| Sugar transporter            | OsSWEET11                 | Negatively regulated                                                      | Gao et al., 2018                  |
| Loose Plant Architecture (LPA) | OsSWEET2a                | Positively regulated                                                      | Gao et al., 2021                  |
| Defense associated protein   | OsGSTU5                  | Over expression                                                          | Kim et al., 2021                  |
| Acyl-CoA-binding protein     | OsACBP5                  | Overexpression of OsACBP5 leads to resistance                             | Panthapulakkal et al., 2020       |
| Kinesin like protein         | KSP                      | KSP overexpression is less susceptible to disease                        | Chu et al., 2021                  |
| DNA-binding one finger (DOF) Transcription factor | DOF11 | Activation of DOF leads to resistance                                    | Kim et al., 2021                  |
| Probenazole responsive protein | OsRSR1          | Enhanced disease resistance via NBS-LRR                                  | Wang et al., 2021                 |
| Protein Phosphatase                       | PP2A-1                   | Overexpression leads to resistance                                        | Lin et al., 2021                  |
| Non-host resistance gene      | IMPA 2                   | Importin alpha (IMPA) 2 provides immunity                                | Parween and Sahu, 2022            |
| Chlorophyll degradation gene  | OsNYC3                   | Gene suppression leads to resistance                                      | Cao et al., 2022                  |

Small RNAs (siRNAs and miRNAs) play a major role in regulating several processes in plants by switching genes on and off leading to resistance to biotic/abiotic stresses. Host-induced gene silencing or RNA interference (RNAi) strategy has been utilized against *Rhizoctonia* by targeting pathogenicity linked MAP kinase genes (Tiwari et al., 2017) and polygalacturonase genes (Rao et al., 2019). Overexpression of a siRNA (SiR109944) targeting a gene, F-Box domain and LRR-containing protein 55, has been found to increase the susceptibility of rice to sheath blight disease (Qiao et al., 2020). An ethylene signaling gene, *ELL1* has been found to positively regulate sheath blight resistance in rice (Sun et al., 2019b). Transcriptome
FIGURE 5 | Genes are being utilized for the development of transgenics and their mode of action in conferring resistance to sheath blight disease of rice. The blue circle indicates the genes, the details of their mode of action are given in Table 5.

analysis has revealed that the upregulation of genes controlling cytoskeleton, membrane integrity, and glycolytic pathway plays a major role in disease resistance (Samal et al., 2022). It is recently reported that lauric acid has a role against R. solani by modifying fatty acid metabolism leading to apoptosis (Wang et al., 2022).

CONCLUSION

Sheath blight is one of the diseases of major concern in rice with the potential to upset rice production and productivity. The causal agent, R. solani is a dynamic pathogen with a wide host range which enables it to overwinter and survive. R. solani has many anastomosis groups, among which AG1-IA is important as the rice sheath blight pathogen. Because of its versatility, the pathogen is very difficult to manage. Chemical control has been the most commonly used approach for management, which is not only environmentally unsafe but also leads to the evolution of novel virulent strains of the pathogen. Although there are other approaches such as cultural practices, and biological control to reduce the disease severity, utilizing host plant resistance is the most sustainable approach for managing this fungal disease. However, rice lacks absolute resistance to rice sheath blight, therefore moderate to high level of tolerance should be tapped as the source of resistance. There have been efforts to map QTLs among the tolerant lines, and many of them have been utilized in marker assisted breeding. However, the progress in molecular breeding has been slow as compared to other major diseases such as bacterial blight and blast diseases where effective genes have been widely available. Standard method of screening for sheath blight disease is based on relative lesion height (RLH) as given by IRRI. This RLH is directly influenced by plant height. Therefore, there is a need to develop a new method for screening against the disease with appropriate standardization. The breeding for sheath blight resistance also needs to focus on utilizing the QTLs through marker assisted introgression into popular cultivars. Several genes have been identified and some of them have been functionally characterized in rice and from other plant species, which provides an opportunity for the development of transgenics as well as genome-editing to create novel variations for managing the sheath blight of rice.

DATA AVAILABILITY STATEMENT

The original contributions presented in the study are included in the article-supplementary material, further inquiries can be directed to the corresponding author/s.
AUTHOR CONTRIBUTIONS
SK, AS, and KV proposed the idea. BB, MS, HB, and PB outlined the review. MS, AT, NS, and PC collected the materials and prepared the draft. RE, BB, SK, and KV edited the manuscript. All authors read and confirmed the final manuscript.

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