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Sheath blight resistance in rice (*Oryza sativa*): A comprehensive review

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ABSTRACT

The growing global population and the rising trend of rice consumption are expected to drive up future rice (*Oryza sativa* L.) demand. If India, China, and Indonesia's recent pattern of rising per capita consumption of rice continues, the overall increase in rice consumption might possibly outpace the population rise. The reduction of cultivable area and the yearly crop loss valued at billions of dollars as a result of sheath blight disease and changing climate aggravate the issue. The pathogen's extremely wide host range, great genetic variability, and the lack of any discernible natural resistance in the existing rice germplasm make it difficult to control. It is imperative to discover countermeasures against the disease in order to minimize the threat to global food security and reduce losses in rice yield. In this review, details on the sheath blight symptoms, pathogen character, disease cycle, host range, QTLs, genome wide association study (GWAS), genomic selection approach, key genes related to sheath blight resistance are summarized.

Keywords: Disease resistance, GWAS, Management, QTL, *Rhizoctonia*, Rice, Sheath blight

Rice (*Oryza sativa* L.) is considered as most essential crops in the world, as it feeds over 50% of the world's population. In India, rice occupies an area of 47 million ha with a production and productivity of 132 million tonnes and 4.2 t/ha during 2023–24 (https://ipad.fas.usda.gov). Rice is grown in diverse conditions ranging from plains to coasts and hills. Rice is affected by many factors, such as fungi, bacteria, virus, nematode and nutrient deficiencies. The main limiting factors influencing rice productivity are biotic stresses caused by plant pathogens. The major diseases infecting rice which cause heavy yield losses are blast (*Magnaporthe grisea*), false smut [*Ustilaginoidea virens* (Cke.) Tak], sheath blight (*Rhizoctonia solani*), bacterial leaf blight (*Xanthomonas oryzae* pv. *oryzae*) and viral disease, rice tungro virus. Among these, sheath blight (ShB) disease caused by *Rhizoctonia solani* AG1–1A is the second most important disease after blast disease. Sexual stage of *Rhizoctonia solani* is *Thanatephorus cucumeris* (A B Frank) Donk. It survives in soil as saprophyte for many years by forming sclerotia. Though, it has minimum movement from field to field, it has broad host range and considerable genetic heterogeneity, it is highly challenging to control. Different factors such as local climatic conditions, variety, early detection and presence of favourable host are playing a major role in influencing the disease development. Monocropping of rice and use of semi dwarf varieties increased the ShB incidence in rice cultivation areas. Different approaches are followed for the control of the ShB incidence in rice (Satya *et al.* 2007, Bashyal *et al.* 2017, Bashyal *et al.* 2022). Management of ShB disease is generally achieved through use of fungicidal spray, but it causes environmental hazards, pesticide residue in grains, development of resistance and also increases the cost of production. Biocontrol agents activate the plant defence mechanism which in turn inhibit the pathogen development. But, some virulent pathogen overcome the plant defence by avoiding the resistance gene activation. Host plant resistance is another way to control ShB disease which includes mapping of rice population to identify the loci with trait of interest through quantitative trait loci (QTL), genome selection and genome wide association study. After few years, the disease resistance break down due to development of new strains or race of pathogen. Various QTLs governing resistance loci of ShB were reviewed by Li *et al.* (2019). Till date, only three QTL loci (qSB-9^{TQ}, qSB-11^{LT} and qSBR-11–1) for ShB resistance was well mapped and utilized in development of disease resistant varieties. Biotechnological approaches are also used for the management of the ShB disease in rice. It includes host delivered, RNA interference (HD-RNAi),

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development of transgenic plants with desirable gene and genome editing. Many reviews have been made earlier on management approaches and mapping of genes governing ShB resistance. This review comprises recent developments in resistance mechanism for ShB disease in rice.

Symptoms

Fungus produces water-soaked lesions with brown edges that form on the stalks and leaf sheaths near the water level. The lesions enlarge and spread upwards and downwards become irregular with grey centre and dark brown margin which looks like snake skin (Fig. 1). During severe cases, it leads to rotting of leaf sheaths and drying of whole leaf and spreads to aerial parts of the plants resulting grain discoloration (Hollier *et al.* 2009 and Singh *et al.* 2016). Brown coloured mustard like sclerotia seen on the affected portions. When the rice canopy is at its densest during flowering, the illness spreads swiftly and creates an environment that is conducive to pathogen growth (Brooks 2007). Depending on the portion of the plant afflicted, *Rhizoctonia solani* can infect seeds to fully grown plants, resulting in mild to considerable yield losses. Large lesions on lower rice leaf infected sheaths may cause the stem to become softer and cause stem lodging (Nagarajkumar *et al.* 2005).

Fig. 1 Symptoms of sheath blight in rice showing larger brown lesion with grey centre.

Characters of Rhizoctonia solani

Mycelium is septate, multinucleate mycelium, constriction at the base of the hypha where point of branching, formation of dolipore septum. They also differ in their capacity to infect different host plants, such as rice (Zeng *et al.* 2011). Guleria *et al.* (2007), Thind and Aggarwal (2008) and Khodayari *et al.* (2009) reported growth rate data of *Rhizoctonia solani* isolates that were identical. But, significant variation was seen in the mycelial properties of the *Rhizoctonia solani* isolates, which ranged in colour from creamy white to dark brown. There were also differences in the mycelial look from fluffy to flat and in the regularity of the margins from regular to irregular. The fresh and dry mycelial weights of the isolates varied significantly, suggesting significant variations in their growth strategies. The sclerotia of the *Rhizoctonia solani* isolates also showed variation in size, colour (light brown to dark brown), texture (fine to coarse), shape (globose to irregular), and formation patterns (scattered, central, concentric). Similar results were published by Sharma *et al.* (2009), who noted a 4.6–6.3 mg sclerotial weight range on PDA medium.

Anastomosis groups and genetic variability

Anastomosis grouping was proven to be the most crucial criterion in separating isolates that are identified as belonging to the species. Based on hyphal fusion of isolates which differed in phenotypic, biochemical, genotypic and pathogenic characters, they were categorized into various groups. Totally 14 anastomosis groups (AGs) are there in *Rhizoctonia solani* (Carling *et al.* 2002a). First 13 groups were named as AG1 to AG13, whereas, the 14th group was named as AGB1. They are AG-1–1A, AG-1–1B, AG-1–1C, AG-1–1D, AG-1–1E, AG-1–1F (Kuninaga *et al.* 2002), AG-2 (1, t, Nt, 2IIIB, 2IV, 2LP, 3, 4) (Carling *et al.* 2002a), AG-3 (TB, PT, TM) (Johnk *et al.* 1993), AG-4 (HGI, HGII, HGIII) (Johnk and Jones 2001), AG-5 (Ogoshi 1972), AG-6 (HG-I, GV) (Kuninaga and Yokosawa 1984), AG7 (Homma *et al.* 1983), AG-8-ZG^a (1, 2, 4, 5) (MacNish and Sweetingham 1993), AG-9 (TP, TX) (Carling *et al.* 1987), AG-10 (MacNish *et al.* 1995), AG-11 (Carling *et al.* 1994), AG-12 (Carling *et al.* 1999), AG-13 (Carling *et al.* 2002b) and AG-BI (bridging isolate) (Kuninaga *et al.* 1978). AGs are having some specific characters, some of them prevalent in specific geographical region (AG-3 is most prevalent in Pakistan). Even though, the relationship between the AGs and host specificity is not yet clear, some of them causing specific disease in particular host (AG-3 causing black scurf and stem canker in potato). While breeding for resistance against sheath blight the above said points have to be taken into consideration.

Singh *et al.* (2024) studied genetic diversity among 35 isolates of *Rhizoctonia solani* from rice and other crop hosts. High variations in morphological characters, viz. colony colour, mycelial growth pattern, sclerotial formation and number of sclerotia were observed, may be due to environmental conditions. Genetic diversity was also studied using various markers and ITS marker formed one major cluster and six sub clusters, whereas, SSR marker formed six clades and universal random primer formed three clades.

Host range

Rhizoctonia solani infects wide range of host other than rice and express different kinds of symptoms. It includes maize, rice, wheat, barley, oat, soybean, peanut, dry bean, alfalfa, chickpea, lentil, field pea, tobacco, potato, sugar beetroot, coffee, cotton, canola, pothos, ficus, lettuce and flax. Various kinds of symptoms produced by *Rhizoctonia solani* on different hosts are stem canker, black scurf, crown rot, seedling blight, stem rot, hypocotyl rot, limb rot, pod rot and pre- and post-emergence damping off.

Disease cycle

Teleomorph stage is *Thanatephorus cucumeris*. *Rhizoctonia solani* is spread through dormant mycelium and sclerotia present in seed and soil. The disease is spread into new areas through seeds. At the time of harvest, sclerotia in the infected plants fall on the ground surface which serves the primary source of inoculums. At the time of last puddling, sclerotia come out, float on the water surface and contact the rice leaf sheath. At the point of contact, sclerotia germinate, produce mycelium and enter into the leaf sheath leads to irregular grey lesion with dark brown margin (Savary *et al.* 1995, Sivalingam *et al.* 2006). Numerous sclerotia produce and fed into the soil. Under favourable condition, the sclerotia germinate and produce basidia and basidiospores which carry by the wind and spread the disease into new field or new area by causing aerial blight (Fig. 2).

Epidemiology

A dense crop canopy with a high frequency of tissue contact and excessive nitrogenous application contribute to severe incidence. Disease development is very faster when the 25–30°C of temperature and 80–100% relative humidity in the forenoon, leaf wetness along with high ambient air temperature. The presence of rice tungro virus, root knot nematode and brown plant hopper intensify the disease severity and yield loss.

Disease resistance

Quantitative trait loci (QTL) mapping

Loci governing quantitative traits are quantitative trait loci. QTLs are defined as any chromosomal region linked to a marker and a quantitative trait (Xu 2002). ShB resistance is considered as a quantitative trait, like growth and yield parameters. It is controlled by many genes (Pinson *et al.* 2005, Zuo *et al.* 2014). Finding QTLs, mapping, validation, and followed by characterization accelerate the process of positioning or map-based cloning significant resistance genes, which could aid in the development of rice cultivars resistant to ShB.

Molecular markers such as restriction fragment length polymorphism (RFLP), single nucleotide polymorphism (SNP) and simple sequence repeat (SSR) were widely used in the mapping research to identify the QTL for ShB resistance (Molla *et al.* 2020). InDel polymorphic molecular marker, sequence-tagged site (STS), cleaved amplified polymorphic sequence (CAPS) were also used (Jia *et al.* 2012, Zuo *et al.* 2013, Zuo *et al.* 2014). Different types of mapping populations have been commonly used for detecting QTL. They are F2 populations (Yadav *et al.* 2015a), F2:3 (Bal *et al.* 2020), F4 (Li *et al.* 1995), backcrossed inbred lines (Zuo *et al.* 2008, Taguchi-Shiobara *et al.* 2013, Eizenga *et al.* 2015), near-isogenic lines (NILs) (Loan *et al.* 2004, Yin *et al.* 2009), recombinant inbred lines (RILs) (Han *et al.* 2003, Pinson *et al.* 2005, Channamallikarjuna *et al.* 2010, Liu *et al.* 2014, Goad *et al.* 2020), and chromosome segment substitution lines (CSSLs) (Zuo *et al.* 2013, Zhu *et al.* 2014, Zuo *et al.* 2014) and double haploids (DHs) (Kunihiro *et al.* 2002, Xu *et al.* 2011, Nelson *et al.* 2012). Using a variety of mapping population techniques, >200 QTLs for resistance to ShB distributed overall 12 chromosomes have been found (Channamallikarjuna *et al.* 2010, Molla *et al.* 2020). For QTL mapping of ShB resistance, rice doubled haploid lines and RILs have been widely utilized.

Channamallikarjuna *et al.* (2010) was studied ShB resistance in rice over four years in three locations using a mapping population made up of 127 recombinant inbred lines produced from a cross between rice cultivars HP2216 (susceptible) and Tetep (resistant). QTL contributing to ShB resistance was found on the long arm of chromosome 11 based on ShB phenotypes and a genetic map containing 126 equally dispersed molecular markers. On chromosome 11, between the marker intervals RM1233 (26.45 Mb) and sbq33 (28.35 Mb), the QTL qSBR11–1 for ShB resistance was found. This study also discovered a significant QTL for ShB resistance at 116.2 cM (between 28.0–28.1 Mb), which is home to a sizable cluster of 14 defence response genes, 11 of which are chitinases. Two mapping populations, viz. F2 and BC1F2 derived from cross of BPT-5204/ARC10531 were utilized to map the QTL for ShB resistance. Nine QTLs were identified

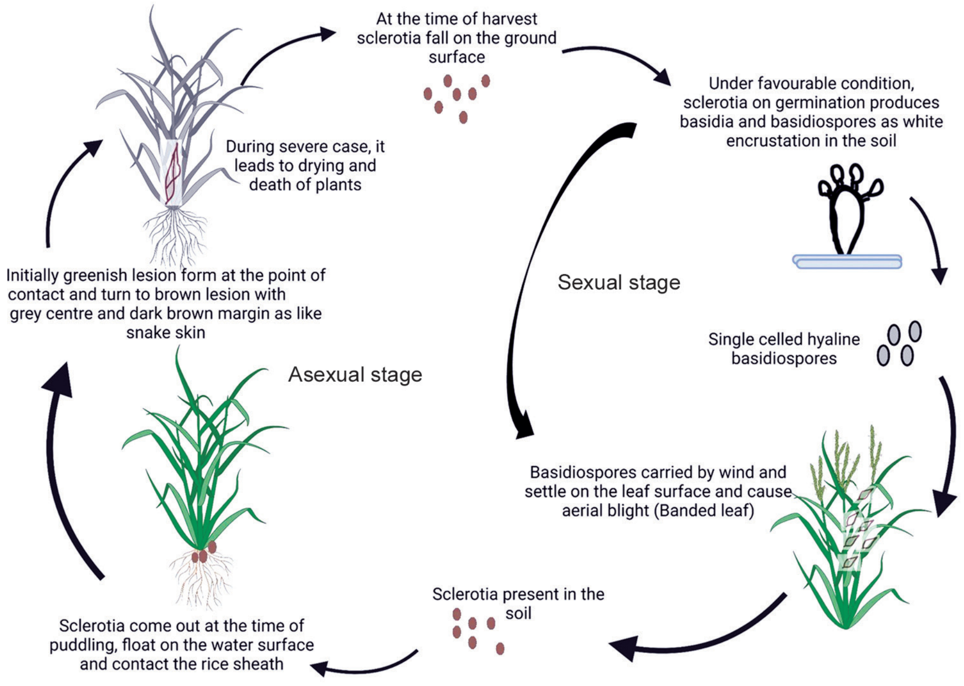

Fig. 2 Disease cycle of sheath blight pathogen in rice.

on chromosomes 1, 6, 7, 8 and 9 with phenotypic variance ranging from 8.40–21.76%. It was discovered that the significant QTLs qshb7.3 and qshb9.2 were strongly related to two SSR markers, RM336 and RM205, respectively. QTLs for ShB resistance in rice are summarized in Table 1 (Updation of Senapati *et al.* 2022 and Chen *et al.* 2023). Even though so many QTLs were identified in rice for ShB resistance, only three QTLs, viz. qSB-9^{TQ}, qSB-11^{LT} and qSBR-11–1 were mapped well and utilized for resistance breeding programme.

Defence mechanism

The existence of certain receptors and signalling cascades enables plants to detect and generate a defence response against sheath blight disease in response to pathogen attack. Early defensive signalling events such as the activation of MAPKs, variations in ion fluxes, salicylic acid (SA), abscisic acid (ABA), jasmonic acid (JA), nitric oxide (NO), hydrogen peroxide (H_2O_2) and reactive oxygen species (ROS) are triggered when membrane Pattern Recognition Receptors (PRRs) called R genes detect Pathogen Associated Microbial Patterns (PAMPs) or pathogen effectors or avirulence gene in pathogen. These early occurrences subsequently trigger intermediate and late defence responses, such as defence gene activation, cell wall strengthening, synthesis of phytoalexin, hypersensitivity reaction, and induced resistance.

Auxin plays a major role in sheath blight resistance. Sheath blight resistance in rice can be increased by pre-treating it with auxin, which alters the plant's natural auxin balance. Auxin efflux carrier PIN1a controls rice's ability to withstand sheath blight. Kinesin-like protein stimulates the production of PIN1a, raising the concentration of auxin locally and fostering resistance to sheath blight through its interaction with the transcription factor LPA1. Small interfering RNA, siR109944 makes the rice plant susceptibility to sheath blight by affecting the auxin balance. Whereas siRNA targets a putative auxin receptor (*FBL55* gene) encoding transport inhibitor response 1 (TIR1)-like protein increases the resistance to sheath blight (Qiao *et al.* 2020). Indeterminate domain proteins, LPA1 and IDD13 regulate the sheath blight resistance by enhancing the expression of auxin efflux carrier PIN1a (Sun *et al.* 2020).

Ethylene plays a major role in sheath blight resistance. Pathogenesis related genes, viz. PR1, PR5 and PR10 were expressed in rice during application of ethylene exogenously. These PR proteins enhanced ShB resistance. OsEIL1, the main player in the rice ethylene signalling pathway, positively controls rice resistance to ShB by controlling the expression of ethylene-responsive genes (Yuan *et al.* 2018). Ammonium transporter, AMT1 is responsible for ammonium uptake in rice. AMT1 stimulates ethylene signalling, as evidenced by the greater expression levels of ethylene signalling genes (EIL1, EIL2, and EIN2) and biosynthetic genes (ACO2 and ACO3) in AMT1;1 OX and decreased expression levels of AMT1 RNAi in comparison to the wild-type plants. In order to confirm the significance of ethylene signalling in AMT1-mediated ShB resistance, AMT1;1 OX plants were treated with ethylene biosynthesis and signalling inhibitors, and the expression of ethylene signalling genes was investigated. AMT1;1 OX ShB resistance was markedly reduced by the inhibitor treatment, indicating that ethylene signalling is essential for AMT1-dependent rice resistance to ShB (Li *et al.* 2024).

Jasmonic acid (JA) and salicylic acid (SA) are key phytohormones regulating plant defence, often exhibiting antagonistic interactions. While SA predominantly counters biotrophic pathogens, JA is effective against necrotrophs like *Rhizoctonia solani*, which thrive on host cell death (Pieterse *et al.* 2009). Crosstalk between SA and JA pathways allows plants to balance fitness costs and fine-tune defence mechanisms. JA signaling plays a critical role in rice defence against *Rhizoctonia solani*. The chitin pathway, regulated by JA, enhances chitinase expression, strengthening resistance (Karmakar *et al.* 2016). Overexpression of transcription factors like WRKY30 enhances JA-related gene expression, including *LOX* and *AOS2*, and increases JA accumulation, thereby improving resistance to *Rhizoctonia solani* (Peng *et al.* 2016). Similarly, the OsWRKY80-OsWRKY4 module positively regulates resistance, while WRKY4 overexpression elevates JA/ethylene-responsive pathogenesis-related (PR) genes such as *PR1a*, *PR1b*, *PR5*, and *PR10/PBZ1* (Wang *et al.* 2015, Peng *et al.* 2016).

ROS plays as a signal molecule in activating the defense mechanism against sheath blight resistance. Pathogen attack may cause susceptible plants to accumulate excessive amounts of reactive oxygen species (ROS), which can lead to cell death and facilitate the growth and colonisation of necrotrophic fungi. Thus, ROS may serve as a signal for *Rhizoctonia solani* to transition from the establishment to the necrotrophic stage. According to Oreiro *et al.* (2020), a quantitative trait locus qLN1128 that is abundant in defence-related genes helps activate the genes linked to the ROS redox pathway and reduces ROS accumulation in rice cells, which delays the colonisation of *Rhizoctonia solani*. In rice, hub gene network contains the catalase OsCATC and chloroplast glutathione peroxidase, which scavenge ROS, may be crucial in balancing normal ROS levels in rice during *Rhizoctonia solani* infection. It activates the L-phenylalanine ammonia-lyase mediated disease resistance by involving H_2O_2 and serves as a vital enzyme in phenylpropanoid metabolism. Through the activity of PAL, it provides precursor for biosynthesis of lignin in the shikimate pathway and provide resistance to ShB by increasing the production of plant antioxidants, cell wall lignification and cell wall thickening. It induces the synthesis of a varied natural products including flavonoids, condensed tannins, hydroxycinnamic acids, stilbenes and coumarins and those compounds performed as signal molecules and phytoalexins (Shi *et al.* 2020).

Key genes related to sheath blight resistance

Identification and characterization of genes involved in ShB resistance are used in the development of resistant

Table 1 QTLs identified in rice for sheath blight resistance

S.No.	QTL	Chromosome	Marker interval	Phenotypic variance	Reference
1.	qSB-1	1	RG532x	8	Pinson *et al.* 2005
2.	qSBR1-1		Hvssr68-RM306	15.01	Channamallikarjuna *et al.* 2010
3.	qSBR1-1		RM1232-Hvssr68	8.13	
4.	qSBR1-1		RM5389-RM3825	3.2	Fu *et al.* 2011
5.	qShB1		RM431-RM12017	5.18–8.03	Xu *et al.* 2011
6.	qSBR1		RM11229	9.5	Jia *et al.* 2012
7.	qShB1		RM431–RM1361	4.7	Eizenga *et al.* 2013
8.	qShB1.1		RM151-RM12253	12.18	Yadav *et al.* 2015
9.	qSBR1–1		SNP03790–1	19.1	Mahantesh *et al.* 2022a
10.	QRh1		SNP101TP5–001	5.4	
11.	qShB.1–2		RM306	8.16	Naveenkumar *et al.* 2023
12.	qSBR-2	2	RG171-G243A	11.2	Kunihiro *et al.* 2002
13.	qSB-2		C624x	7	Pinson *et al.* 2005
14.	qSBR2–1		RM5340-RM521	3.1	Fu *et al.* 2011
15.	qSBR2–2		RM110-osr14	5.2	
16.	qSBR2–3		RM7245-RM5303	3.3	
17.	qShB2		RM174-RM145	3.96	Xu *et al.* 2011
18.	qsbr_2.1		RM8254-RM8252	3.4–29.7	Nelson *et al.* 2012
19.	qsbr_2.2		RM3857-RM5404	2.9–37.8	
20.	qShB2–1		RM279–RM71	3.7	Liu *et al.* 2013
21.	qSBR2–1		SNP101VHF-001	11.6	Mahantesh *et al.* 2022a
22.	qSBR2–2		SNP101W2M-001	5.2	
23.	qSB-3	3	R250–C746	26.5	Zou *et al.* 2000
24.	qSBR-3		G249-G164	10.5	Kunihiro *et al.* 2002
25.	qSBR3–1		RM251-RM338	9.96	Channamallikarjuna *et al.* 2010
26.	qShB3		RM135-RM186	3.42	Xu *et al.* 2011
27.	qSB-3		SNP101WMR-001	5.5	Mahantesh *et al.* 2022a
28.	qShB.3–2		RM16	10.33	Naveenkumar *et al.* 2023
29.	qShB.3–6		RM85	16.54	
30.	qShB.3–1		RM1350	5.69	
31.	qSBR4	4	RM3288-RM7187	3.8	Fu *et al.* 2011
32.	qSB-5	5	Y1049	6	Pinson *et al.* 2005
33.	qSBR5–2		RM7446-RM3620	4.8	Fu *et al.* 2011
34.	qShB5		RM18872-RM421	4.35	Xu *et al.* 2011
35.	qSBR5–1		SNP101XV7-001	7.7	Mahantesh *et al.* 2022a
36.	qSBR5–2		SNP01177–1	7.7	
37.	qShB.5–1		RM13	11.44	Naveenkumar *et al.* 2023
38.	qShB.5–3		RM334	5.08	
39.	qShB.5–1		RM13	9.11	
40.	qShB.5–2		RM178	8.06	
41.	qShB6	6	RM3431–RM3183	7.8	Eizenga *et al.* 2013
42.	qShB6		RM253–RM3431	21.2	
43.	qShB6		RM253–RM3431	11.1	
44.	qShB6-mc		RM3183–RM541	3.3	

Contd.

Table 1 (*Concluded*)

S.No.	QTL	Chromosome	Marker interval	Phenotypic variance	Reference
45.	qRTL6		RM6395	5.8	Liu *et al.* 2013
46.	qShB6.1		RM400-RM253	13.25	Yadav *et al.* 2015a
47.	qSBR6–1		SNP05385–1	10	Mahantesh *et al.* 2022a
48.	qSBR6–2		SNP101YH1–001	5.5	
49.	qSBR-7	7	RG511-TCT122	15.5	Kunihiro *et al.* 2002
50.	qSBR7–1		RM3691-RM336	10.02	Channamallikarjuna *et al.* 2010
51.	qSBR7–1		RM5481-RM3691	26.05	
52.	qSBR7		RM1132-RM473	3.3	Fu *et al.* 2011
53.	ARqShB7-AR		RM5711-RM2	4.0	Liu *et al.* 2013
54.	qShB7 LA		RM5711-RM2	6.0	
55.	qShB7.1		RM81-RM6152	10.52	Yadav *et al.* 2015a
56.	qShB7.2		RM10-RM21693	9.72	
57.	qShB7.3		RM336-RM427	21.76	
58.	qSBR7–1		SNP13515–001	15.6	Mahantesh *et al.* 2022a
59.	qSBR8–1	8	RM210-Hvssr47	8.37	Channamallikarjuna *et al.* 2010
60.	qSBR8		RM8264-RM1109	4.2	Xu *et al.* 2011
61.	qShB8.1		RM21792-RM310	10.52	Yadav *et al.* 2015a
62.	qSBR8–1		SNP10208P-001	13.5	Mahantesh *et al.* 2022a
63.	qShB.8–2		RM5428	13.51	Naveenkumar *et al.* 2023
64.	qSB-9	9	RZ404	6	Pinson *et al.* 2005
65.	qShB9–1		RM409-RM257	5.4	Liu *et al.* 2009
66.	qShB9–2		RM215-RM245	24.3	Liu *et al.* 2009
67.	qSBR9–1		Hvssr9-27-RM257	9.19	Channamallikarjuna *et al.* 2010
68.	qSBR9		RM23869-RM3769	5.0	Xu *et al.* 2011
69.	qRTL9		RM3533	3.8	Liu *et al.* 2013
70.	qShB9.1		RM257-RM242	8.40	Yadav *et al.* 2015a
71.	qShB9.2		RM205-RM105	19.81	
72.	qShB9.3		RM24260-RM 3744	12.58	
73.	qSB-9		SNP102151–001	5.3	Mahantesh *et al.* 2022a
74.	qShB.9–1		RM257	10.09	Naveenkumar *et al.*2023
75.	qSBR10	10	SNP101U6D-001	5.5	Mahantesh *et al.* 2022a
76.	qSBR-11	11	CT224-CT44	9.5	Kunihiro *et al.* 2002
77.	qSBR11–1		sbq11-RM224	11.99	Channamallikarjuna *et al.* 2010
78.	qSBR11–1		sbq1-RM224	13.99	
79.	qSBR11–1		sbq11-RM224	21.59	
80.	qSBR11–1		RM224-K39516	13.38	
81.	qSBR11–2		RM3428-RM209	7.81	
82.	qSBR11–3		RM536-RM202	21.59	
83.	qSBR11		RM7203	1.9	Jia *et al.* 2012
84.	qShB11–1		RM7203-RM536	3.2	Liu *et al.* 2013
85.	TXqShB11–2-TX		RM536-RM229	3.3	
86.	qSB-11–1		SNP101UHW-001	5.5	Mahantesh *et al.* 2022a
87.	qSBR11–1		SNP101UWB-001	8.5	
88.	qSBR11–2		SNP07570-1	6.4	
89.	qsBR_12.1	12	RM3747-RM27608	49.1	Nelson *et al.* 2012

varieties (Singh *et al.* 2019). Yadav *et al.* (2015) reported that β,1–3 glucanase like defence gene occur in the ShB QTL qShB9-2 region which breaks the β-1,3-linkages of glucan polymer present in the fungal cell wall resulting lysis of fungal cell wall.

Chitinase breaks the β-1,4-glycosidic linkages of chitin to degrade the fungal cell wall. Chitinase (chi11) gene has increased resistance to ShB in rice (Baisakh *et al.* 2001, Datta *et al.* 2001). Class III chitinase genes mapped in QTL qSBR11–1 of rice line Tetep were found to be effective in reduction of mycelial growth of *Rhizoctonia solani* due to chitinolytic activity (Richa *et al.* 2016). Osmotin belongs to PR5 family thaumatin like proteins are involved in the permeability stress and defence response in plants.

WRKY transcription factor plays a key role as plant immune response regulator under a biotic stress (Cui *et al.* 2019). Phukan *et al.* (2016) reported that WRKY genes play a critical role in either directly or indirectly interacting with PAMPs/effector proteins or regulating MAPK to suppress or enhance plant defence responses. ShB resistance in rice augmented by transcription factors, viz. OsWRKY4,13,30 and 80 (Wang *et al.* 2015a, Peng *et al.* 2016, Lilly and Subramanian 2019).

Zuo *et al.* (2013) reported 12 predicted putative genes in qSB11-LE. Among the 12 genes, three defence related candidate, two receptor like protein kinase 5 precursors and one lipase like gene responsible for ShB resistance. Key genes involved in ShB resistance are summarized in Table 2.

Xie *et al.* (2023) demonstrated the role of OsERF65 in ShB resistance and reported that OsERF65, a rice transcription factor, inhibits cell death to negatively modulate resistance to ShB. When *Rhizoctonia solani* infection occurred in the susceptible cultivar Lemont, OsERF65 was markedly elevated and expressed strongly in the leaf sheath. While the knockout mutant (oserf65) showed dramatically improved resistance to ShB, OsERF65 overexpression lowered rice resistance by suppressing the expression of peroxidase genes and modulating reactive oxygen species homeostasis.

RNA silencing

Silencing of fungal pathogenicity genes by making transgenic plants with small RNA (sRNA). RNAi is the mechanism which associated with many regulatory processes includes protection against viral pathogens, gene expression control, genome stability, etc. Transgenic rice plants produce sRNA and double stranded RNAs which silence the pathogenicity genes in *Rhizoctonia solani*, there by symptom production will be arrested (Huang *et al.* 2019). Tiwari *et al.* (2017) reported that RPMK1–1 and RPMK1–2 are homologues of pathogenicity MAP Kinase 1 in *Rhizoctonia solani* were silenced by host delivered RNAi (HD-RNAi) which in turn increased the resistance against sheath blight. Rao *et al.* (2019) reported that HD-RNAi was silenced the polygalacturonase encoded by the gene AG1IA_04727 resulting resistance to sheath blight pathogen in rice. Delivery of dsRNA targeting interest gene onto the plant reduced the fungal infection by altering the morphology or inhibiting spore production or inhibiting mycelial growth. Qiao *et al.* (2021) was prepared dsRNA targeting the gene DCTN1, SAC1 and polygalacturonase

Table 2 Key genes involved in sheath blight resistance

S.No.	Gene name	Gene description	Reference
1.	PR-5	Thaumatin-like protein	Datta *et al.* 1999
2.	OsRC7, OsCHI11	Chitinase gene	Datta *et al.* 2001
3.	chi11, TLP, Xa21	Chitinase, thaumatin-like protein and serine-threonine kinase	Maruthasalam *et al.* 2007
4.	Dm-AMP1	Antifungal plant defensin	Jha *et al.* 2009
5.	OsWRKY30	WRKY transcription factor	Peng *et al.* 2012
6.	OsWRKY45	WRKY transcription factor	Shimono *et al.* 2012
7.	OsPBZ1	PR (pathogen-related) gene	Helliwell *et al.* 2013
8.	OsACS2	Ethylene biosynthetic genes	
9.	OsOXO4	Oxalate oxidase 4	Molla *et al.* 2013
10.	AtNPR1	Regulates systemic acquired resistance pathway	Sadumpati *et al.* 2013
11.	TLP-D34	Thaumatin-like protein	Shah *et al.* 2013
12.	RCH10, AGLU1	Chitinase and Alfalfa β-1,3-glucanase gene	Mao *et al.* 2014
13.	OsPGIP1	Inhibits activity of polygalacturonase, resistance against ShB	Wang *et al.* 2015b
14.	OsWRKY4	WRKY transcription factor	
15.	OsOXO4, OsCHI11	Oxalate oxidase and chitinase gene	Karmakar *et al.* 2016
16.	BjNPR1	Non-expressor of pathogenesis related gene	Molla *et al.* 2016
17.	OsWRKY80	WRKY transcription factor	Peng *et al.* 2016
18.	OsOSM1	PR 5, defence against ShB in rice	Xue *et al.* 2016

Contd.

Table 1 (*Concluded*)

S.No.	Gene name	Gene description	Reference
19.	OxDC	Oxalate oxidase	Qi *et al.* 2017
20.	Os11g47510	Chitinase gene	Richa *et al.* 2017
21.	chi11, ap24	Rice chitinase and Tobacco osmotin	Sripriya *et al.* 2017
22.	RPMK1–1, RPMK1–2	Pathogenicity Map Kinases	Tiwari *et al.* 2017
23.	OsSWEET11	Sugar transporter	Gao *et al.* 2018
24.	OsEIL1	Ethylene responsive gene	Yuan *et al.* 2018
25.	OsPGIP2L233F	Polygalacturonase inhibiting proteins (PGIP)	Chen *et al.* 2019a
26.	OsWRKY13	WRKY transcription factor	Lilly and Subramanian 2019
27.	OsMAPK20–5	MAP kinase, defence response	Liu *et al.* 2019
28.	OsPIN1a	Auxin efflux carrier	Sun *et al.* 2019
29.	ZmPGIP3	PGIP	Zhu *et al.* 2019
30.	OsWRKY53	WRKY transcription factor negatively regulated	Peng *et al.* 2020
31.	OsFBL55	A putative auxin receptor	Qiao *et al.* 2020
32.	OsGSTU5	Defence associated protein	Tiwari *et al.* 2020
33.	OsACBP5	Acyl-CoA-binding protein	Panthapulakkal *et al.* 2020
34.	KSP	Kinesin like protein	Chu *et al.* 2021
35.	OsSWEET2a	Sugar transporter	Gao *et al.* 2021
36.	OsSWEET14	Sugar transporter	Kim *et al.* 2021
37.	DOF11	DNA-binding one finger	
38.	PP2A-1	Protein Phosphatase	Lin *et al.* 2021
39.	OsRSR1	Probenazole responsive protein	Wang *et al.* 2021
40.	OsNYC3	Chlorophyll degradation gene	Cao *et al.* 2022
41.	IMPA 2	Non-host resistance gene	Parween and Sahu 2022
42.	Os05t0572000	DNA binding transcription factor and ERF/AP2 domain-containing protein	Naveenkumar *et al.* 2023
43.	Os05t0566400	Group D MAPK, regulation of overreactions to defences	
44.	Os06t0667900	Plant immune response	
45.	Os08t0193700	Encodes a subunit of the proteasome complex, ubiquitin-dependent protein degradation	
46.	Os08t0155900	Similar to pathogen-induced defence-responsive Protein	
47.	Os09t0458300	Leucine-rich repeat (LRR), N-terminal domain-containing Protein	
48.	Os10t0478300	Myb DNA binding domain, Transcription factors with eukaryotic-type helix-turn-helix and tryptophan clusters	
49.	Os10t0195000	Multi-antimicrobial extrusion protein	
50.	Os01t0750400	LRR domain-containing protein	
51.	Os01t0629900	Known as mitogen-activated protein kinase 10	
52.	Os01t0899800	DNA-binding domain transcription factor	
53.	Os02t0769800	Mus musculus proteasome involves in protein degradation	
54.	Os02t0759700	LRR 2 containing protein	
55.	Os03t0848700	Contain disease resistance N-terminal domain, plant defence against plant pathogens	
56.	Os03t0600600	Similar to beta-1,3-glucanase	
57.	Os03t0184400	LRR	
58.	Os03t0860100	Plant hormone signal transducer	
59.	Os04t0119800	LRR domain-containing protein	
60.	Os04t0122000	LRR, N-terminal domain-containing protein	
61.	Os05t0246600	LRR domain-containing protein	

(PG) responsible for vesicle trafficking pathway genes and virulence factor using *in vitro* MEGA script®RNAi Kit reduced the sheath blight incidence in rice.

Differential gene expression in resistant and susceptible variety

Level of gene expression was studied between the resistant 'Shennong 9819' and susceptible cultivar 'Koshihikari' in response to inoculation of ShB pathogen in rice through high-throughput sequencing. A total of 6630 and 14151 differentially expressed genes (DEGs) found in resistant and susceptible cultivars, respectively and 3505 DEGs expressed commonly in both cultivars. Expression of DEGs in the leaf sheath of Koshihikari inoculated with ShB pathogen is increased first and then declined; also, there were noticeably more DEGs than in the resistant cultivar. Interruption or specificity in metabolic pathway by the upregulated genes during *Rhizoctonia solani* infection in both resistant and susceptible cultivars was analysed through Kyoto Encyclopedia of Genes and Genomes (KEGG) enrichment analysis. The plant hormone signal transduction, tropane, phenylalanine metabolism, pyridine, isoquinoline alkaloid biosynthesis pathways, tyrosine, piperidine, glycine, serine, threonine and beta-alanine pathways were markedly enhanced in Shennong 9819 and not in the Koshihikari, whereas, in the case Koshihikari, ascorbate, aldarate, linoleicAcid, propanate and 2-Oxocarboxylic acid metabolism pathway and valine, leucine and isoleucine degradation pathway was markedly enriched. OsPR1b expression was consistently greater in Shennong 9819 than in Koshihikari. On the other hand, at most time points, Koshihikari's OsPR1a expression level was higher than Shennong 9819's, suggesting that the two populations might have distinct resistance mechanisms. Expression of OsWRKY30 was higher in Shennong 9819, whereas, expression of OsWRKY70 was higher in Koshihikari. The overexpression of the transcription factor OsWRKY70 may be the cause of Koshihikari's reduced resistance to *Rhizoctonia solani* when compared to the resistant cultivar Shennong 9819 (Yang *et al.* 2022).

Comparative transcriptomic analysis of the resistant cultivar YSBR1 and the susceptible cultivar Lemont post-*Rhizoctonia solani* infection identified 7624 differentially expressed genes (DEGs) across one or more time point. Gene ontology (GO) analysis revealed that genes encoding cell wall-modifying enzymes, glycosyl-degrading enzymes, and antimicrobial proteins were prominently induced in YSBR1 as early as 6 hours post-inoculation (hpi). MapMan analysis further highlighted the upregulation of DEGs related to cell wall fortification, β-glucanases, respiratory burst, phenylpropanoids, and lignin biosynthesis in YSBR1, suggesting enhanced defence-related pathways. In addition, receptor-like kinases and jasmonic acid signaling pathways appeared to play critical roles in YSBR1's resistance mechanism (Yuan *et al.* 2018). In the context of YSBR1 and Lemont, contrasting expression patterns were observed underlining their potential role in resistance.

Genome wide association study (GWAS)

With the development of biotechnology, GWAS has gained popularity as a way for rice breeding. Single nucleotide polymorphisms (SNPs) in the genome and the population's linkage disequilibrium (LD) form the foundation of GWAS. It is an analytical technique to determine the association between target qualities and genetic markers/candidate genes within a population by combining population structure, genome-wide LD level, and phenotypic data. High accuracy, quick processing, and no population-based mapping building are some of its benefits. GWAS offers an effective technique and strategy for researching the genetic basis of rice disease resistance and identifying putative genes for disease resistance (Li *et al.* 2017, Bhandari *et al.* 2020).

Many GWAS have been performed for ShB resistance. Jia *et al.* (2012) reported that substantial correlation of 10 marker loci was found in 217 sub-core rice entries containing 155 markers using GWAS on rice ShB. Chen *et al.* (2019b) identified 11 single-nucleotide polymorphism (SNP) sites that are strongly linked to ShB resistance by employing 44,000 SNP markers and 299 distinct rice types using GWAS. Zhang *et al.* (2019) performed GWAS with 563 rice accessions and 2,977,750 SNPs and detected association of 132 SNP loci with lesion height, 562 SNP loci with relative lesion height and 75 SNP loci with culm length. Oreiro *et al.* (2020) performed GWAS with phenotypic of 228 rice accessions using 700,000 SNPs for ShB resistance. GWAS was performed with Ting's core collection along with 150 land race accessions of rice for studying ShB resistance by using 5,173,707 SNPs through EMMAX method (Fu *et al.* 2022). Significant levels (P<0.00001) were found for SNPs (chr09_21,587,781) and SNPs (chr11_26,875,550) in the main QTLs qShB9–2 and qSBR11–1, respectively. Li *et al.* (2022) was analyzed and identified 18 QTLs for ShB resistance from 417 accessions of the Rice Diversity Panel 1 (RDP1) through GWAS. Among 18 QTLs, one QTL, *qShB4–1* was identified as new QTL governing ShB resistance.

Genomic selection approach

Conventional breeding for disease resistance is a time consuming process as it takes 10 years to develop a varieties. Though, marker assisted selection (MAS) consumes less time period, it is associated with QTL and it is highly possible for the traits which are governed by one or few major genes. If the quantitative traits associated with many minor genes, MAS is not suitable or ideal. Genomic selection is the best approach for such quantitative traits and it predicts the genomic estimated breeding values (GEBV) of an offspring in a population by using genome wide DNA markers and phenotypes of specific traits. So many models are used in genomic selection and are represented in Fig. 3.

Mahantesh *et al.* (2022b) evaluated the effective models for genomic selection for predicting the ShB resistance in rice. In that, three models, viz. Bayesian A, Bayesian B and Bayesian CPi were used with 1545 Recombinant inbred lines

(RILs) derived from eleven crosses (Jasmine 85 × TN1, Jasmine 85 × Swarna-Sub1, Jasmine 85 × II32B, Jasmine 85 × IR54, Tetep × TN1, Tetep × Swarna-Sub1, Tetep × II32B, Tetep × IR54, MTU 9992 × TN1, MTU 9992 × II32B and MTU 9992 × IRBB4) made between resistant parents (Tetep, Jasmine 85 and MTU 9992) and susceptible parents (IR 54, II32B, IRBB4, TN1 and Swarna Sub1) and 6564 SNP markers to predict the genomic estimated breedings values. The prediction accuracy of tenfold cross validation ranged from 069 to 0.72 across models. The highest prediction accuracy was observed with Bayesian B model, whereas, lowest prediction accuracy was observed with Bayesian A model. As the prediction accuracy was consistent with Bayesian B across tenfold, the model Bayesian B was the best to predict the ShB resistance in rice (Mahantesh *et al.* 2022b). Mahantesh *et al.* (2022c) analysed the best model for calculating the GEBV and marker effects with the five models of rrBLUP, BayesA, BayesB, BayesCPi and GBLUP and 1545 RILs. From the analysis, GBLUP was identified as the best model for calculating the GEBVs when compared to other models tested, as it uses the genetic relationship coefficients and BayesB was on par with this GBLUP.

Conclusion

The *Rhizoctonia solani*, causative agent of ShB disease is increasingly posing a credible danger to rice farming worldwide. Even after a century since its discovery, *Rhizoctonia solani* remains a "enigmatic pathogen to control" due to its adaptable nature and capacity to infect nearly all sorts of tissues on a broad spectrum of crop plants. Though numerous methods are available currently for combating this disease, though many varieties are available most of them confer moderate resistance to the pathogen. QTLs offer greater scope for developing highly resistant varieties against the pathogen. More than 200 QTLs have been mapped and identified so far in rice against ShB. But, these QTLs have not been functionally characterized clearly, if they are properly validated and characterized for their desirable disease resistance which can be used in resistance breeding. Once their functional characteristics have been established, the discovered resistant donors with desirable alleles may be employed to confer resistance against ShB in rice. The major QTLs governing ShB resistance has to be utilized for development of resistant varieties by gene pyramiding. Till date, no R gene against ShB has been identified. The interaction between the pathogenicity genes in *Rhizoctonia Solani* and the host genetic factors involved in ShB resistance has to be studied in detail and identification of target gene or defence gene is most essentially needed for resistance breeding or the genes is most responsible for disease susceptibility. Many elite cultivars, traditional rice varieties, wild cultivars may be explored to find out the genes responsible for ShB resistance. High throughput sequencing of various rice genotypes will delineate the function and variation of different defence genes present in various genotypes. On identifying the target gene, the same can be employed in RNAi to improve or incorporate the resistant characters through transgenic transformation. As a component of IDM, several fungicide is being recommended and used for the management of ShB which are hazardous to environment and results in development of not only resistance but also evolves new strains of pathogen. CRISPR-Cas mediated genome editing receives more attention now a days and through this validation of pathogenicity genes functionally responsible for symptom production can be done.

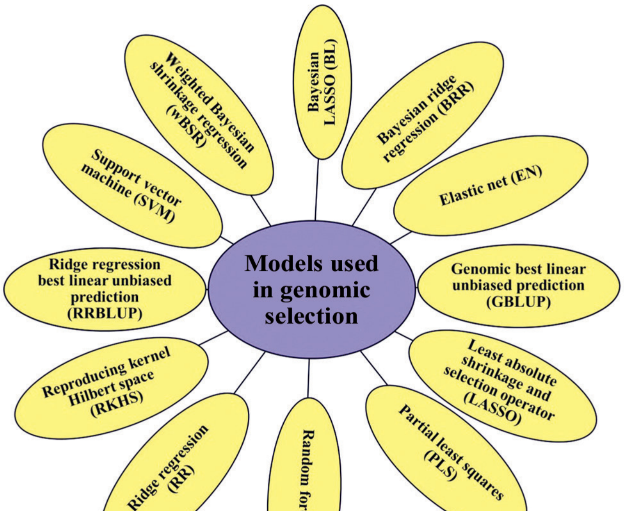

Fig. 3 Different models used in genomic selection in rice.

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