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REVIEW

Advances in breeding, biotechnology, and nanotechnological approaches to combat sheath blight disease in rice

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Abstract

Sheath blight, caused by the fungus *Rhizoctonia solani*, is a major problem that significantly impacts rice production and can lead to substantial yield losses. The disease has become increasingly problematic in recent years due to the widespread use of high-yielding semi-dwarf rice cultivars, dense planting, and heavy application of nitrogenous fertilizers. The disease has become more challenging to manage due to its diverse host range and the lack of resistant cultivars. Despite utilizing traditional methods, the problem persists without a satisfactory solution. Therefore, modern approaches, including advanced breeding, transgenic methods, genome editing using CRISPR/Cas9 technology, and nanotechnological interventions, are being explored to develop rice plants resistant to sheath blight disease. This review primarily focuses on these recent advancements in combating the sheath blight disease.

Keywords Rice · *Rhizoctonia solani* · Disease resistance · CRISPR/Cas9 · Nanoparticles

Introduction

Rice stands as one of the most vital staple foods, nourishing over half of the global population. As the population elevates in countries where rice is a staple crop, it is anticipated that there will be an increased demand for rice in the future. Despite tremendous advancements in agricultural technology over the past fifty years, a significant proportion of the global population still faces starvation and undernourishment. Hunger and malnutrition are caused by an imbalance between crop production and population for food leading to starvation and malnutrition, especially among children. Despite the effective implementation of many scientific advancements in the cultivation of rice crops, pests and diseases continue to exist, so preventative measures should be taken to minimize yield loss [1].

Sheath blight (ShB), caused by *Rhizoctonia solani* Kuhn [teleomorph - *Thanatephorus cucumeris Frank* (Donk)], is one of the most serious diseases of rice worldwide, which poses a significant threat to rice production. Despite the effective control of bacterial blight and rice blast through advanced resistance breeding, managing ShB remains challenging due to the limited understanding of its underlying molecular resistance mechanisms. In the early stages of the disease, water-soaked lesions appear, which later develop into a rattlesnake-like pattern with greyish-white cents and dark brown margins. As the infection becomes severe, the fungus produces sclerotia interwoven mycelial masses coated in hydrophobic layers. These sclerotia, measuring 1 to 3 mm in diameter, initially appear white and gradually turn brown or dark brown, forming on the surface of infected rice. These remains dormant in the soil between crops for many years and regains its their infectivity under favourable conditions [2]. ShB causes yield loss of up to 50% at the field level, whereas artificially inoculated plots show 20–42% yield loss [3].

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R. solani is a soil-borne necrotrophic fungal pathogen that infects plants of over 32 taxonomic families. *R. solani* isolates had fourteen different anastomosis groups (AGs). AGs are a classification system based on hyphal fusion reactions. These groups are crucial for understanding the genetic diversity in this plant pathogenic fungus, indicating its great genetic variability [4]. The first thirteen groups were called AG1–AG13, while the 14th group, AGB1, is a bridging isolate. Bridging isolate is a specific isolate that can undergo hyphal fusion with isolates from different AGs, making it important for understanding the genetic diversity and taxonomic connections within the *R. solani* species complex. Several anastomosis groups are further subdivided into intraspecific groups (ISGs). *R. solani* AG1 isolates are classified into three ISGs, viz., IA, IB, and IC, based on host origin, symptoms, cultural characteristics, DNA sequence homology and sclerotia morphology [5]. *R. solani* AG1-IA is generally accepted to be the cause of ShB disease in rice. Pathogens enter plants by lobate appressoria, infection cushions, or both. *R. solani* runner hyphae form convoluted hyphal clusters, known as infection cushions. Infection typically occurs through direct cuticular penetration via an infection cushion, while stomatal penetration *via* lobate appressoria is less common [2].

The pathogen is difficult to manage due to its broad host range, high genetic variability, and lack of natural resistance in available rice germplasm. Finding solutions to combat the pathogen is crucial for reducing rice yield losses and ensuring global food security. This review outlines breeding methodologies, focusing on the molecular mechanisms of defense-related genes, the use of genome editing to target negative regulators, and the roles of nanoparticles in enhancing ShB resistance.

Virulence factors involved in *R. solani* infection in rice

The pathogen *R. solani* infiltrates the host through multiple mechanisms, while rice counters with innate and systemic acquired resistance (SAR) immunity. Effector proteins are utilized by pathogens to infect host plants and can cause disease. *R. solani* is known to produce a variety of effector molecules with different functions that enable successful colonization. Domains of three potential secreted effectors of *R. solani* AG1-1 A: glycosyltransferase GT family 2, cytochrome C oxidase assembly protein CtaG/ cox11, and peptidase inhibitor I9 have been identified to induce cell death in rice during *R. solani* invasion [6]. Genomic analysis of virulent Indian strains identified additional effectors, including histone acetyltransferase, MDR transporter, polygalacturonase, and pectin lyase [7]. The polygalacturonase gene significantly contributes to *R. solani* pathogenesis [8].

R. solani secretes a variety of secondary metabolites, such as host-selective toxins and biologically active molecules, which enhance pathogen virulence by breaking down host physical barriers and disrupting normal physiological functions of the host plant [6]. Biologically active molecules produced by *R. solani* include oxalic acid (OA), 3-methylthiopropionic acid (MTPA), phenylacetic acid (PAA) and its derivatives [9]. OA produced by necrotrophic pathogens is an essential virulence factor for successful infection. During *R. solani* infection, OA inhibits the synthesis of various phenolic substances and degrades the cell wall for effective penetration [10].

Fungal plant pathogens typically secrete various types of carbohydrate-active enzymes (CAZymes), for successful invasion, including cell wall-degrading enzymes (CWDEs) viz., cellulases, pectinases, and hemicellulases to breach plant cell wall components. *R. solani* evades plant immunity by masking its cell wall chitin with α-1,3-glucan. The combined action of these CWDEs allows *R. solani* to effectively penetrate and spread within the host plant tissues, contributing to the pathogen's ability to cause disease. Understanding the role of CWDEs is a crucial strategy to combat *R. solani* infection [11].

Factors influencing ShB pathogenesis

Sheath Blight pathogenesis is influenced by various factors, including hormones, sugars, and nitrogen sources. Hormones play a crucial role in regulating plant responses to pathogens. Sugars and nitrogen sources are essential nutrients utilized by pathogens for their growth and development during host-pathogen interactions [12]. Understanding and effectively managing these factors are critical for controlling and mitigating the spread of ShB disease. Comprehending how hormones, sugars, and nitrogen sources interact with *R. solani* and the host plant is crucial for reducing disease incidence and enhancing crop yield.

Hormones

Plant hormones such as auxin, ethylene (ET), salicylic acid (SA), jasmonic acid (JA), brassinosteroids (BRs), gibberellin (GA), abscisic acid (ABA), strigolactone (SL), and cytokinin (CTK) intricately regulate rice defense responses against *R. solani* [12]. Auxin, essential for rice growth, facilitates transport *via* PIN-FORMED 1a (*OsPIN1a*) and enhances resistance to ShB when overexpressed, suggesting its role in modulating plant-pathogen interactions [13].

Ethylene, traditionally associated with fruit ripening, also significantly enhances rice resistance to pathogens by activating defense responses such as reactive oxygen species (ROS) and phytoalexin production, mediated by *OsACS2* and *OsEIL1* [14]. Salicylic acid and jasmonic acid, two distinct defense-related hormones, play differing roles in rice immunity: SA primarily acts against biotrophic, while JA defends against necrotrophic pathogens. Both hormones contribute positively to rice resistance against ShB through pathways involving NPR1-mediated SA signaling and *OsWRKY30*-mediated JA responses [15]. Beyond SA and JA, BRs, GA, ABA, SL, and CTK also modulate rice's defense strategies. BRs, typically associated with growth promotion, negatively regulate ShB resistance [16]. GA's influence on immunity involves complex interactions with JA signaling and developmental processes [17]. ABA acts as a negative regulator of rice immunity, contrasting with CTK's role in promoting defense responses against pathogens. These hormones collectively illustrate the intricate balance and interplay required for rice to effectively combat ShB, underscoring the complexity of plant-pathogen interactions [12].

Sugar

Sugars produced through photosynthesis are essential for plant growth and serve as vital nutrients utilized by *R. solani* during the invasion. SWEET proteins, such as *OsSWEET11/Os8N3*, mediate sugar transport across cell membranes, facilitating pathogen access to extracellular sugars [18]. Modulating sugar levels impacts rice susceptibility to ShB, linking sugar metabolism directly to disease outcomes. Effector protein AOS2 interact with host proteins (e.g., WRKY53, GT1) to activate sugar transporters (e.g.,*OsSWEET2a*, *OsSWEET3a*), influencing the rice-ShB interaction and resistance mechanisms [19].

Nitrogen fertilizer

Nitrogen (N) fertilizer has played a crucial role in boosting rice yields since the Green Revolution, particularly with the development of semi-dwarf varieties, such as those carrying the *sd1* allele, which enhance the yield but often exhibit poor nitrogen use efficiency (NUE) [20]. While high nitrogen levels support rice growth and yield, they also correlate with increased susceptibility to ShB disease [21]. Recent studies have highlighted specific genes influencing nitrogen's impact on ShB resistance. For example, *OsAMT*1;1, a rice ammonium transporter, has been identified as crucial for enhancing ShB resistance by promoting the accumulation of nitrogen metabolites and activating the ethylene signaling pathway [22]. Additionally, *OsDEP1*, associated with both NUE and ShB resistance, regulates susceptibility to ShB; silenced plants and mutants exhibit enhanced resistance, whereas overexpression aggravates susceptibility [23]. These findings underscore the complex interplay among nitrogen management, yield enhancement, and disease resistance strategies in rice.

Management of ShB disease in rice

Management of ShB disease is challenging because of their wide host range, genetic variability, fungicidal resistance and lack of durable resistance cultivars in rice. The excessive use of fungicides harms beneficial microorganisms and human health. In this context, there is an urgent need for alternative and sustainable management strategies.

Biological and chemical control

Biological control methods offer promising strategies for managing *R. solani.* Plant growth-promoting actinomycetes, particularly *Streptomyces* sp. exhibit strong microbial antagonism against *R. solani*. Bacterial biocontrol agents such as *Pseudomonas* and *Bacillu*s not only suppress fungal pathogens but also promote plant growth through various mechanisms such as nutrient solubilization and phytohormone synthesis [24]. Timely application of fungicides between panicle differentiation and heading stages is a crucial factor in overcoming resistance development, especially in susceptible varieties [25]. However, the continuous use of single fungicides can lead to resistance by *R. solani*, necessitating combination formulations like Azoxystrobin + Difenoconazole that will delay resistance development [26]. Despite their efficacy, chemical methods pose environmental risks. Hence, the use of non-chemical methods, which include modern breeding strategies such as QTLs, genome editing, and nanotechnological aspects, to develop viable resistance against ShB is indispensable.

Breeding strategies for ShB resistance

Breeding strategies aimed at enhancing ShB resistance in rice elite cultivars involve several key approaches. ShB resistance may be attributed to two main mechanisms: disease escape and physiological resistance. Disease escape relies heavily on crop architecture, with morphological traits such as plant height, heading date, and stem thickness showing positive correlations with resistance. Physiological resistance, on the other hand, is linked to processes that reduce the efficiency of one or several stages of the pathogen's infection cycle [27].

Utilizing host plant resistance

Host plant resistance is considered the most sustainable method for managing ShB disease. When a plant recognizes a pathogen, signal transduction pathways collaborate to establish a complex network that triggers defence responses. During pathogen invasion, certain genes, known as disease resistance genes, respond by changing their expression or protein modifications. These include R (Resistance) genes and HPRR (host pattern recognition receptor) genes, which help detect and counteract the pathogen. In rice, disease resistance is either qualitative or quantitative. Qualitative resistance, controlled by a single R gene, is race-specific and offers strong, targeted protection against specific pathogens [28].

Quantitative resistance is controlled by multiple genes or quantitative trait loci (QTL), which confers partial resistance to various pathogens. A plethora of reports elucidates that quantitative resistance can work against different strains and even different species of the pathogen, which offer broad-spectrum resistance from annual to perennial crops under conducive environmental conditions [28]. ShB resistance in rice *Indica* cultivar was commonly controlled by numerous minor genes, which exhibit broad-spectrum disease resistance comparable with *Japonica* cultivars. In addition, wild relatives such as *Oryza rufipogon*, *Oryza nivara*, *Oryza meridionalis*, and *Oryza barthii* have shown resistance traits against ShB [4, 29].

Resistant cultivars against ShB

The use of ShB-resistant rice varieties is the most economical and effective strategy to combat ShB disease. Identifying ShB-resistant germplasm and mapping resistance genes are essential to develop resistant varieties. Still, none of the genotypes with absolute resistance are identified [30]. The list of rice varieties with variable resistance is noted in Table 1.

Table 1 List of Rice varieties recognized for their resistance to ShB disease

Moderately resistant variety	Disease score	Subpopulation / Origin	Reference
Jasmine 85	4.26 ± 0.08	*indica*	[31]
Teqing	5.56–5.57(DPAA-based disease index)	*indica*	[31]
YSBR1	2.86 ± 0.10	*indica*	[32]
Pecos	6.33–6.56 (DPAA-based disease index)	United States	[33]
Koshihikari	2.72 ± 0.37	*temperate japonica*	[34]
C418	3.84 ± 0.09	*temperate japonica*	[34]
Tetep	4.89–6.44 (DPAA-based disease index)	*temperate japonica*	[35]

Marker-assisted breeding

Marker-assisted breeding is pivotal for integrating identified resistance QTLs into popular rice cultivars. By identifying and integrating genes that offer resistance to ShB, breeders can develop rice varieties with reduced vulnerability to the disease. Researchers have employed advanced backcross methods and double haploid (DH) populations to map rice's QTLs responsible for ShB resistance. They've discovered potential genetic markers for breeding resistant varieties, from both wild rice species like *O. minuta and O. rufipogon*, as well as cultivated varieties through backcrossing with species like *O. officinalis* [36]. These QTLs, notably located on chromosome 9, contribute to the complex genetic basis of ShB resistance, with studies indicating its polygenic nature influenced by multiple genes. This approach aids in reducing linkage drag during the introgression of genomic regions and facilitates the pyramiding of resistance genes. By utilizing molecular markers, breeders can precisely select rice plants carrying desired ShB resistance genes, thereby accelerating the breeding process [37]. While individual R genes have proven effective against ShB disease in rice, the persistent challenge of rapid pathogen evolution exists. To tackle this issue, integrating genetic diversity of wild rice species and utilizing breeding methods to incorporate advantageous ShB resistance QTLs into *japonica* cultivars shows significant potential for enhancing resistance. Ultimately, sustainable management strategies for ShB disease in rice can be achieved through the integration of various resistance mechanisms and genetic resources.

QTL mapping

Sheath blight resistance in rice is a quantitative trait controlled by multiple genes, making QTL identification, mapping, validation, and characterization crucial for developing resistant varieties [38]. Using diverse mapping populations and molecular markers, numerous QTLs for ShB resistance have been detected on all 12 rice chromosomes [2]. RIL (Recombinant Inbred Line) and DH mapping populations have largely replaced F2-derived populations due to lower recombination and their ephemeral nature [39]. Despite their potential, wild relatives of cultivated varieties have rarely been used for QTL detection due to crossability barriers [40]. Due to environmental influences, accurate disease phenotyping remains a significant challenge for fine mapping of ShB-resistant loci. Resistance to rice ShB is

governed by polygenes [41, 42] although some studies suggest control by major genes in certain varieties [31]. Over the two decades, numerous quantitative trait loci (QTLs) contributing to ShB resistance have been identified. Notable QTLs for sheath blight resistance in rice were mapped on chromosomes 2, 3, 7, 9, 11, and 12, with additional QTLs detected by various studies [36, 41]. However, these QTLs have yet to be exploited in developing ShB-resistant cultivars, and their breeding potential remains unassessed.

Introducing desirable ShB resistance QTLs into Japanese cultivars involves overcoming the predominance of such QTLs in *indica* rice. Most ShB resistance QTLs identified originate from *indica* rice, with few from *japonica* rice. Notably, qShB-7TQ and qShB-9TQ, originating from *indica* rice Teqing (TQ), have been located on chromosomes 7 and 9, respectively. Research indicates that qShB-9TQ provides significant resistance to ShB, reducing disease ratings by 1.0. (using the qShB-9TQ rating scale). However, Japanese rice varieties lack qShB-9TQ.To address this, breeding strategies involving crossing and backcrossing *indica* varieties with *japonica* varieties have been employed. This approach aims to introduce qShB-9TQ into *japonica* varieties, thereby enhancing their resistance to ShB [38]. Crossing the moderately-resistant CR 1014 with the susceptible Swarna-Sub results in identification of three QTLs (qShB-1.1, qShB-1.2, qShB-1.3) on chromosome 1, with qShB-1.1 consistently showing a high Logarithm of odds (LOD) score. This QTL co-located with qShB1 from *Oryza nivara*, includes potential candidate genes LOC_Os01g65650 and LOC_Os01g65900. Near-isogenic lines of Swarna-Sub1 carrying qShB-1.1 exhibited a 27.8% reduction in lesion height [43]. Most of the QTLs have been summarized in previous studies [27]. Only a few QTLs discussed in later studies are covered in the following section.

Role of major and minor QTLs

Among the identified QTLs for ShB resistance in rice, qShB9-2 and qSBR11-1 stand out as major contributors, explaining 25% and 14% of the phenotypic variation, respectively [44, 45]. qShB9-2 encompasses candidate genes such as β-1,3-glucanase and *OsWAK91* and has been fine-mapped to a 146-Kb region [46]. Similarly, qSBR11-1, derived from the partially resistant line Tetep, spans a 0.85 Mb region on chromosome 11, featuring a tandem array of eleven class III chitinase genes and LOC_Os11g47510, which confer tolerance in susceptible cultivars like Taipei 309 [47]. Despite extensive studies, no novel candidate genes for breeding or genetic engineering have emerged from these QTLs. In contrast, qSB12YSB, originating from rice variety YSBR1, has been mapped to a 289-Kb region on chromosome 12. Through the use of 150 BC4 backcross inbred lines and 34 chromosomal segment substitution lines, researchers identified 18 candidate genes associated with qSB12YSB, including those implicated in secondary metabolite biosynthesis and ROS scavenging systems according to KEGG analysis. Field trials confirmed qSB12YSB's efficacy, showing significant resistance in commercial rice cultivars NJ9108, NJ5055, and NJ44 under severe ShB conditions, reducing yield losses by up to 13.5% in the Lemont background. These findings underscore qSB12YSB's potential in rice breeding programs aimed at developing new, resistant varieties to combat ShB [48].

Minor QTLs in rice refer to genetic loci with moderate effects on phenotypic variation and lower LOD scores compared to major QTLs. In ShB resistance studies, these QTLs play a role in fine-tuning resistance traits across varying environmental conditions [49]. Exploring these minor QTLs is crucial for uncovering genes that could significantly enhance ShB resistance in rice breeding programs.

Pyramiding of genes

Pyramiding is a process of combining major and minor resistant genes to enhance and prolong resistance against ShB in rice varieties. Traditional breeding methods have struggled to produce ShB-resistant rice varieties due to the quantitative nature of the trait [38]. Marker-assisted selection has not been used to incorporate ShB resistance QTL (qShB)s into commercial rice varieties despite its widespread use in disease resistance breeding [47]. The introduction of qShB-9TQ and qShB-3TQ into Lemont could potentially reduce ShB loss by 15% [42].

Overexpressing a single defense-related protein may not be highly effective in enhancing resistance. Combining multiple ShB resistance QTLs can increase resistance to ShB. Combinatorial expression of defense genes has shown better results. Examples include combinations like MOD1 and RCH10, Chi11 and thaumatin-like protein, Chi11 and β-1,3-glucanase, DmAMP1 and RsAFP2, Chi11 and ap24, RCH10 and AGLU1, Oxalate oxidase 4 and Chi11, NPR1 and Chi11 [50, 51]. Studies indicate that dual-gene cassettes are more effective for ShB resistance than single-gene cassettes [50]. Additionally, combining glycoside hydrolase genes from *Trichoderma atroviride* (ech42, nag70, and gluc78) enhances pathogen tolerance [2, 52]. By combining these strategies, breeders can develop rice cultivars with enhanced tolerance to ShB disease in rice.

Omics approach to understanding the ShB pathogenesis

Omics is a comprehensive field of study focused on understanding the relationships among various molecules, particularly interactions between plants and pathogens. Recently,

research in this area has provided deeper insights into these interactions through transcriptomic, and metabolomic analyses etc. Whole-genome sequencing on 13 inbred rice lines in this study revealed over 200 candidate genes, encompassing a total of 333 nonsynonymous single nucleotide polymorphisms (SNPs), distinguishing between susceptible and resistant genotypes to ShB [53]. Comparative transcriptome analysis has revealed that alternative splicing of key pathogenic genes in *R. solani* AG1-1 A plays significant roles during its infection of rice, soybean, and corn plants [54]. Comparative transcriptomics between ShB-susceptible (Lemont) and tolerant (Teqing) rice cultivars identified 4806 differentially expressed genes (DEGs) [55]. Further investigations into the proteome and metabolome of rice lines pre- and post-ShB infection uncovered 38 differentially expressed proteins and 40 differentially accumulated metabolites, underscoring the significance of energy and carbohydrate metabolism in the plant's response to *R. solani* [56]. Recently identification of 23 putative candidate rice miRNAs that could potentially be involved in the defense mechanisms against *R. solani* [57]. Genomic, transcriptomic, proteomic, and metabolomic studies have identified key genes, proteins, metabolites, and miRNAs involved in rice and *R. solani* host-pathogen interactions.

Biotechnological approaches

Transgenic approaches

In rice, conventional breeding has attained partial resistance to ShB. Genetic engineering techniques give a promising foundation for further improvement to get complete resistance. Transgenic approaches have been adopted by researchers to facilitate the introduction of desired genes, aiming to achieve comprehensive resistance in a relatively condensed timeframe compared to traditional breeding methods. This strategy not only reduces dependability on chemical pesticides but also enhances key agronomic parameters, effectively addressing challenges such as sexual incompatibility. Moreover, transgenic technology has the potential to introduce novel traits from disparate systems into the target organism, amplifying its versatility in crop improvement [58].

In a transgenic strategy, researchers develop resistance against ShB disease in rice by elevating innate immune responses such as pathogenesis-related genes and introducing several foreign genes. Transgenic plants were developed in elite *indica* cultivars viz., Pusa Basmati, ASD16, ADT38, and IR50 with co-expression of pathogenesis-related (PR) genes such as chitinase (chi11) and thaumatin-like protein (TLP) reveals enhance resistance through synergistic activity against ShB [59]. Subsequently, the conclusive evidence on transgenic rice plants harbouring rice chitinase *chi11* gene, belonging to a PR-3, confers ShB resistance. Overexpression of the *OsACS2* gene, a crucial ET synthesis enzyme, is controlled by the vigorous pathogen-responsive promoter (PBZ1). Besides enhancing their resistance to *R. solani*, transgenic lines also contribute to maintaining crop productivity. These findings underscore the potential of manipulating ethylene levels to fortify rice's defence mechanisms against fungal pathogens [14].

Recently, 352 differentially expressed genes in six diverse rice genotypes resistant to ShB disease caused by *R. solani* AG1-IA are identified. Among 352 genes, *Oschib*1, a class III chitinase, was significantly overexpressed and cloned from the resistant variety Tetep. Overexpression of *Oschib1* in the susceptible variety Taipei 309 conferred resistance to *R. solani*, it demonstrates dose-dependent enzyme activity [60]. The list of transgenes utilized to develop resistance is listed in Table 2.

Genome editing through CRISPR technology

Earlier, techniques like EMS (Ethyl methane sulfonate) mutagenesis and T-DNA (Transfer-DNA) insertion induce random mutations in the genome. ZFN and TALEN represent advancements over earlier genome editing methods. CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats) is a natural defense mechanism in bacteria against phages, now widely utilized as a precise genome editing tool, surpassing the limitations of ZFN and TALEN. Among CRISPR/Cas systems, Type II CRISPR/Cas9 is commonly used, consisting of RNA-guided Cas9 endonuclease and single-guide RNA (sgRNA). In plants, the endogenous repair system employs two mechanisms to repair double-strand breaks: NHEJ (non-homologous end-joining), which is error-prone, and HDR (homology-directed repair), which can lead to extensive insertion or fragment replacement [85]. To enhance precision and minimize unintended effects, protein engineering techniques have been employed to modify the Cas9 nuclease, resulting in variants such as Cas9D10A for more accurate editing [86].

The utilization of CRISPR/Cas9 technology will facilitate resistance creation against *R. solani* infection by targeting the negative regulators. The knocking out of genes like *OsMESL*, *OsEIL2*, and *OsSLR* in rice enhances resistance against *R. solani* by reducing ROS accumulation and downregulating GA signaling pathways [87–89]. Emerging shreds of evidence on CRISPR-Cas9 knockout of the microRNA *Osa-miR444b.2* elucidate slower lesion expansion, reduced grain weight (GW), and smaller panicles with increased tillering and plant height. Subsequently, gene expression analysis indicated that *Osa-miR444b.2* regulates plant hormone signaling pathways, which play a pivotal

Table 2 List of transgenes utilized to develop ShB resistance

S.No	Gene	Source of gene	Function	Promoter	Level of Resistance	Reference
Group of Chitinase Genes						
1	*OsCHI11*	*Oryza sativa*	Degrades chitin by breaking β -1, 4 linkages	CaMV35S	More than 50% resistance	[61]
				Maize ubiquitin	40% resistance	[8]
2	*OsCHI12*	*Oryza sativa*	Degrades chitin by breaking β -1, 4 linkages	Maize ubiquitin	2-fold increase in resistance	[60]
3	*Oschib1*					
4	*OsRC7*					
5	*OsRCH10*			*rbcS* and *Act1* promoters	Significant symptom reduction	[62]
				CaMV35S	Significant resistance	[63]
6	*TAeCH42*	*Trichoderma atroviride*	Cell Wall Degrading Enzyme (CWDE)	Act1	Resistance	[52]
7	*TAeCHT42*	*Trichoderma virens*	Degrades chitin by breaking β -1, 4 linkages	CaMV35S	62% resistance	[64]
8	*McCHIT1*	*Momordica charantia*	Class 1 secretory endochitinase	Maize ubiquitin	25–43% resistance	[64]
Group of thaumatin-like proteins						
9	*OsTLP-D34*	*Oryza sativa*	Fungal xylanase inhibition &membrane permeabilization	CaMV35S	Enhanced resistance	[65]
				Maize ubiquitin	Enhanced resistance	[66]
Group of sweet transporters						
10	*OsSWEET11*	*Oryza sativa*	Sugar transporter	Rubisco promoter	Less susceptible	[67]
11	*OsSWEET14*	*Oryza sativa*	Sugar transporter	-	Less susceptible	[68]
Group of antimicrobial peptides						
2	*TaPIN A*, *TaPIN B*	*Triticum aestivum*	Fungal lipid membrane disruption	Maize ubiquitin	11–22% resistance	[69]
13	*Ace-AMP1*	*Allium cepa*	Effective antimicrobial protein homologous to ns-LTPs	PAL promoter and ubiquitin	67% resistance	[70]
14	*Dm-AMP1*,	*Dahlia merckii*	Effective antimicrobial protein homologous to ns-LTPs	Maize ubiquitin	72% resistance	[71]
15	*RS-AFP2*	*Raphanus sativus*	Antifungal plant defensin	Maize ubiquitin	45% resistance	[72]
16	*OsWRKY30*	*Oryza sativa*	Positively regulated defence response	Maize ubiquitin	Enhanced resistance	[73]
Group of osmotin genes						
17	*OsOSM1*	*Oryza sativa*	Osmotin protein belonging to the PR 5 family, positive regulator	Maize ubiquitin	Remarkable decrease in susceptible	[74]
18	*Ntap24*	*Nicotiana tabacum*	Plant defence response and Permeability stress	CaMV35S	Score 3 out of 9(IRRI scale)	[8]
Group of polygalacturonase (PG) Inhibiting proteins (PGIP)						
19	*OsPGIP1*	*Oryza sativa*	Inhibiting fungal polygalacturonase (PG) activity	CaMV35S	Enhanced resistance	[75]
20	*OsPGIP2*	*Oryza sativa*		Maize Ubiquitin-1	Enhanced resistance	[74]
21	*ZmPGIP3*	*Zea mays*		Ubiquitin	Enhanced resistance	[76]
Mitogen-Activated Protein (MAP) Kinases						
22	*OsMAPK20-5*	*Oryza sativa*	Plant development and adaptive response to biotic and abiotic stresses	-	Moderately susceptible	[77]
23	*OsACS2*	*Oryza sativa*	Overexpression of ethylene leads to resistance	PBZ1	Enhanced resistance	[14]
Group of non-expression of pathogenesis-related genes						
24	*AtNPR1*	*Arabidopsis thaliana*	Induce the SAR pathway	Rice PD54O-544	30% resistance	[78]
25	*BjNPR1*	*Brassica juncea*	Regulator of Systemic Acquired Resistance	CaMV35S	Enhanced resistance	[79]
Group of acyl-coa-binding proteins						
26	*OsACBP5*	*Oryza sativa*	Overexpression leads to resistance	(RTLP1) promoter	2.5-fold reduction	[80]
DNA-Binding One Finger (DOF)						

Table 2 (continued)

S.No	Gene	Source of gene	Function	Promoter	Level of Resistance	Reference
27	*OsDOF11*	*Oryza sativa*	Activation of DOF leads to resistance	-	Less susceptible	[68]
Group of probenazole responsive proteins						
28	*OsRSR1*	*Oryza sativa*	Enhanced disease resistance via NBS-LRR	CaMV35S	Enhanced resistance	[81]
29	*OsPP2A-1*	*Oryza sativa*	Protein Phosphatase Overexpression leads to resistance	Maize ubiquitin	Enhanced resistance	[82]
30	*OsIMPA 2*	*Oryza sativa*	Non-host resistance gene Importin alpha (IMPA) 2 provides immunity	-	Moderate resistance	[83]
31	*OsNYC3*	*Oryza sativa*	Chlorophyll degradation gene Gene suppression leads to resistance	Maize ubiquitin	2.86–0.86 score (IRRI scale)	[84]

Table 3 List of gene knockout studies using CRISPR against ShB disease in rice

S.No	Gene	Function	Reference
1	*SWEET11*	Sucrose transporter	[67]
2	*SWEET2a*	Sucrose transporter	[67]
3	*OsMESL*	Methyl esterase family protein It affects ROS accumulation	[88]
4	*OsTrxm*	Thioredoxin protein. Involved in chloroplast redox regulation	[88]
5	*OsNYC3*	Non-yellow colouring gene Regulates chlorophyll degradation	[84]
6	*OsERF65*	Act as a Transcription factor Modulate ROS homeostasis	[91]
7	*Osa-miR444b.2*	Involved in plant hormone signaling pathways	[90]
8	*OsEIL2*	Involved in ethylene and salicylic acid signaling and interacting with other defense-related genes	[89]
9	Os*SLR1*	Negative regulator of gibberellic acid (GA) signaling in rice	[87]
10	IDD3	PIN auxin transporter genes	[92]
11	*OsZF8*	Post-transcriptional regulator	[93]

role in disease resistance [90]. Several other genes are also employed to develop a resistance against ShB in rice, and they are listed in Table 3.

Role of pathogenesis-related genes

PR proteins play an important role in rice disease resistance response. During *R. solani* infection, activation of the SAR pathway in rice induces PR genes (PR-3, PR-5, PR-9, PR-10, PR-12 and PR-13) and PAL, enhancing resistance [94]. Subsequently, specific PR-5 genes (*TLP-D-34* and *OsOSM1*) give promising evidence for improving ShB resistance [65, 74]. Moreover, overexpression of ethylene biosynthetic genes such as PR1b and PR5 elevates resistance [14].

Signaling related genes

The nuclear localization of *NPR1* (Nonexpressor of pathogenesis related genes 1) is essential for activating the expression of PR genes. *BjNPR1* and *AtNPR1* show enhanced resistance to ShB in rice by activating the SA-mediated SAR pathway [78, 79]. In addition, transgenic rice lines expressing the *ACS2 (1-aminocyclopropane-1-carboxylic acid synthase)* gene for ethylene synthesis exhibit increased resistance to *R. solani* [14].

Antimicrobial peptides

The Glycine and cysteine-rich antimicrobial peptides (AMPs) like thionin (Thi3.1), defensin (PDF1.2) and lipid transfer proteins (LTPs) will defend by forming membrane pores, causing ion leakage and cell death [95]. AMP1 from *Dahlia merckii* and *Allium cepa*, AFP2 from *Raphanus sativus*, puroindoline (*pinA and pinB*) from wheat, Snakin-1, Stomoxyn ZH1, Purothionin, Cecropin B, D4E1, and Phor21 expressed either through transgenic methods or by intrinsic inhibitory properties, contribute to elevate resistance against *R. solani* [96].

Host-induced gene silencing

Host-induced gene Silencing (HIGS) leverages RNA interference (RNAi) to boost plant resistance by targeting pathogen genes. It relies on host plants by constitutively expressing dsRNA constructs that transfer siRNA complementary to the virulence factors of the pathogen. Recent

studies in rice *R. solani* pathogen system have successfully targeted key pathogenicity genes like MAP kinase (PMK) and polygalacturonase (PG), significantly reducing disease susceptibility [2]. Moreover, the Grassy tiller 1 (GT1) gene in rice, induced by *R. solani*, increases susceptibility to ShB by activating *SWEET2a* and *SWEET3a* genes. GT1 RNAi plants and mutants for *sweet2a* and *sweet3a* show reduced susceptibility to the disease compared to wild-type [97].

Nanotechnological approaches

Nanotechnology is the study of understanding the matter at nano dimensions. It involves building, controlling and structuring nanomaterials. So far, we broadly use chemical fungicides to manage ShB disease in rice. These chemicals are expensive and have residual effects. To overcome these things nowadays farmers use nanomaterials to combat ShB disease in rice [98]. Foliar application of lanthanum-based nanomaterials, particularly $La_{10}Si_6O_{27}$ nanorods, suppresses ShB in rice. NR (nanorods) are more advantageous because of their good bioavailability, slower dissolution, and silicon nutrient benefits. Simultaneously, they activate the CAM (Crassulacean acid metabolism) pathway, enhances PAL, leading to the production of SA, lignin, and antioxidants. SA, which upregulates the SAR, lignin acts as a physical barrier, and ROS scavenges through antioxidants. These comprehensively result in ShB resistance in rice [99]. Figure 1 illustrates the mechanisms by which nanomaterials combat ShB disease.

Silver nanoparticles (SNPs) are employed to combat ShB disease in rice, targeting the crucial factor of sclerotia germination. SNPs form an antimicrobial layer around rice plants, and penetrate the fungal cell membrane to eliminate pathogens. This process inhibits sclerotia formation and germination. The eco-friendly nature of SNPs, with fungistatic, bacteriostatic, and plasmonic properties, positions them as environmentally conscious inhibitors against plant pathogens, contrasting with synthetic fungicides [100]. The use of Boro gold also reduces the severity of ShB in rice. Boro gold (SNSp) is a combination of nanosilver particles

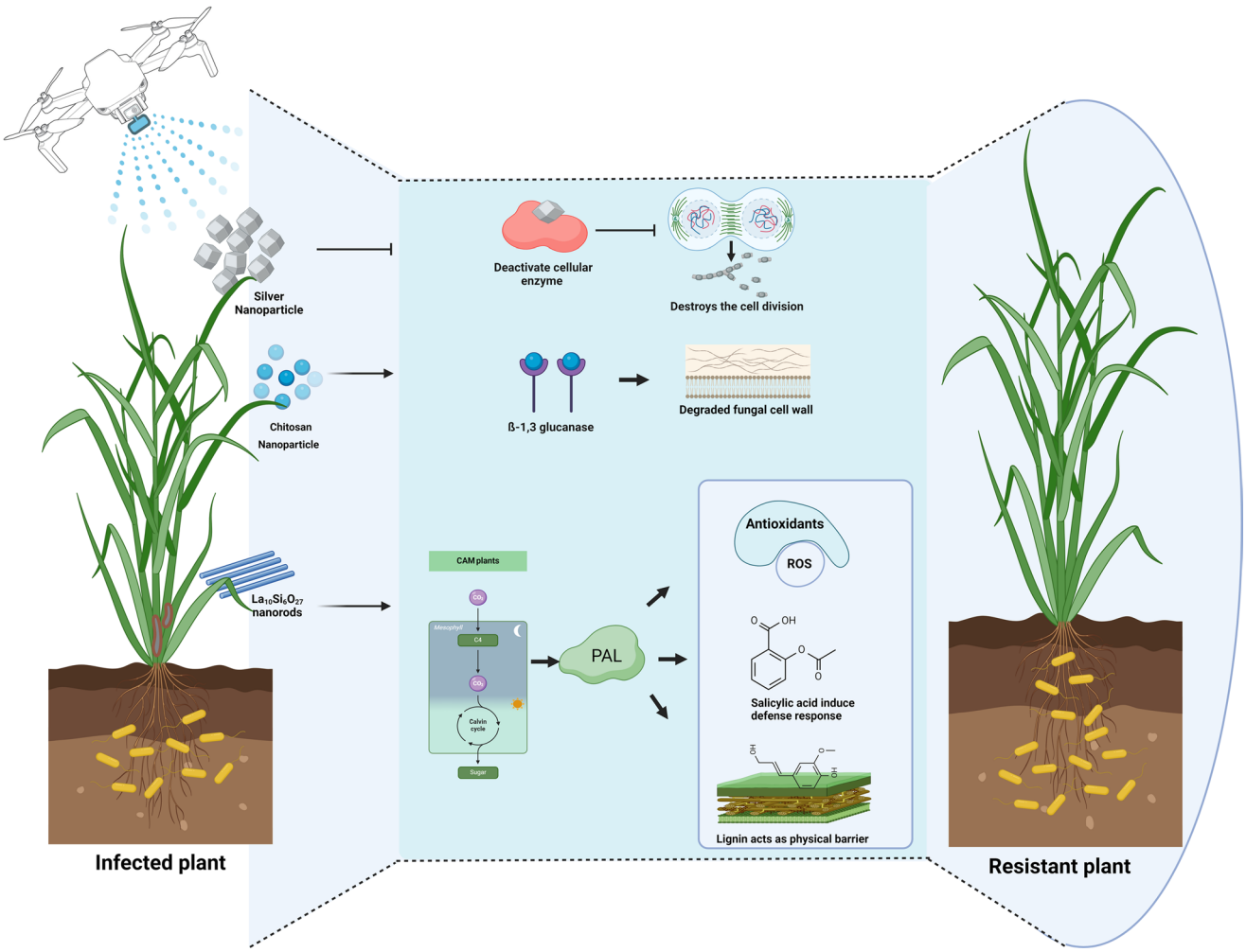

Fig. 1 Mechanism of various nanomaterials that develop resistance against ShB in rice plants. (Figure created with www.Biorender.com)

and peroxy acid. This can be administered through root dipping for 24 h followed by 2–3 times spray when it is needed [101].

Chitosan Nanoparticle (ChNP), partially or fully deacetylated chitin, is a hydrolytic enzyme and a pathogenesis-related (PR) protein. It is a non-toxic, biodegradable biopolymer and a powerful enhancer of plant immunity. Chitin is a primary component of the cell walls of fungi, insects, and crustaceans. Insoluble chitosan hinders agricultural use, but water-soluble chitosan nanoparticles overcome this, exhibiting enhanced efficacy. Shrimp shell waste, sourced for chitosan extraction, is transformed into nanoparticles through ionic gelation with polyanion tripolyphosphate. Increased peroxidase in ChNP-treated plants defends against oxidative stress during pathogen invasion. PAL activates phenylpropanoid pathways, and ß-1,3 glucanase breaks down fungal cell wall components. Chitosan nanoparticles trigger plant resistance by prompting diverse defence responses, including the formation of structural barriers. Chitosan nanoparticles can penetrate plant cells deeply, integrating into the plant's defence mechanism systemically. They serve as powerful inducers of systemic resistance in rice against ShB disease [102].

Foliar application of selenium nanomaterials (Se^0 NMs) significantly reduced ShB severity in rice by 68.8% at 5 mg/L, outperforming Se ions and Thifluzamide. Se^0 NMs' controlled release increased bioavailability and promoted SA and JA-dependent resistance pathways. Additionally, Se^0 NMs improved rice yield by 31.1%, enhanced nutritional quality by 6.4–7.2%, raised organic Se content by 44.8%, and decreased arsenic and cadmium levels by 38.7% and 42.1%, respectively. They also increased Se bio-accessibility by 22.0% and reduced As and Cd bio-accessibility by 20.3% and 13.4%, respectively, suggesting a sustainable strategy for better food quality and security [103].

Enzyme-responsive AZOX-AFS-Pec nanoparticles (NPs) use iron-based mesoporous materials and pectin as carriers to combat rice ShB. These NPs showed high AZOX loading capacity and released the fungicide selectively under acidic conditions in the presence of pectinase. They exhibited superior wetting and adhesion on rice blades, enhanced fungicidal activity against ShB, and promoted rice growth by releasing Fe ions. Moreover, the NPs increased SA levels in rice plants, bolstering disease resistance while reducing toxicity to earthworms compared to AZOX suspension [104]. Table 4 lists nanoparticles used to manage ShB.

Applying silver nanoparticles deactivates cellular enzymes that destroy pathogen cell division, enhancing plant resistance. Chitosan nanoparticles activate ß-1,3 glucanase, which breaks down fungal cell walls. Lanthanide nanorods activate the CAM pathway, enhancing PAL activity and producing SA, lignin, and antioxidants. SA upregulates SAR, lignin acts as a physical barrier, and antioxidants scavenge ROS accumulation. Collectively, these effects confer ShB resistance in rice.

Table 4 List of nanoparticles used against ShB in rice

S.No	Nanoparticle's Name	Function of Nanoparticle	% of resistance	Reference
1	$La_{10}Si_6O_{27}$ nanorods	Bioavailability, slower dissolution, and silicon nutrient benefits. Simultaneously, they activate the CAM pathway	62.4%	[99]
2	Silver nanoparticles (SNPs)	Inhibits sclerotia formation and germination	85–92%	[100]
3	Boro gold (SNSp)	Reduced ShB severity	60.94	[101, 102]
4	Chitosan Nanoparticle (ChNP),	Hydrolytic enzyme and a pathogenesis-related (PR) protein	75%	[102]
5	Selenium nanomaterials Se^0 NM	Increased bioavailability & promoted SA & JA dependent resistance pathways	68.8%	[103]
6	AZOX-AFS-Pec nanoparticles (NPs)	Enhance Delivery Efficiency and elevate SA levels	-	[104]

Key databases related to rice ShB

KRiShI - A Knowledgebase for Rice Sheath Blight Information, a comprehensive, manually curated platform, integrates dispersed unstructured scientific data on the rice ShB disease into an easy-to-use interface for effective mining, visualisation and search. In addition to offering comprehensive information on host resistance, gene expression, proteins, metabolites, resistance genes, pathways and OMICS studies (http://www.tezu.ernet.in/krishi/) [105]. RSIADB - Rice Sheath Blight Information and Analysis Database, is a dedicated resource offering extensive information on rice ShB disease. It includes data on pathogens, host interactions, resistance mechanisms, and related research findings (http://genedenovoweb.ticp.net:81/rsia/index.php) [106].

Conclusion

ShB stands out as an emerging concern among various rice diseases, capable of severely disrupting rice production and yield. Cultural and biological methods represent sustainable approaches for mitigating the severity of ShB disease. Promising bio-agents of *Pseudomonas* and *Bacillus* play a significant role in growth promotion and suppression

of hyphal invasion [30]. In extending context, the role of systemic fungicides in the management of ShB at the field level is inevitable. Even though, the multifaceted necrotrophic pathogen naturally has a wide host range, making its management is challenging till date. The lack of the R gene in available rice germplasm and wild weeds has not yet been elucidated. However, marker-assisted breeding aids in mapping QTL, offer a promising approach to identifying and utilising genes associated with ShB resistance. It underscores the necessity for continued exploration and innovation in breeding and biotechnological approaches to uncover and utilize potential R genes for enhancing disease resistance. In recent eras, emerging molecular techniques have created opportunities to explore host-pathogen interaction by utilizing CRISPR/Cas-mediated genome editing. It allows precise modifications of the rice genome by enabling scientists to successfully delve into targeting candidate negative regulators viz., *OsSWEET*, *OsMSEL*, *OsNYC3* and *Osa-miR444b.2* etc., associated with pathogenesis [67, 84, 88, 90]. These Transgene-free SDN-1 type of mutants will hold a great potential for widespread commercial adoption and mark a significant advancement in developing resilient cultivars. Despite genetic resistance, growing evidence leads to uncovering the utilization of nanomaterials in and along with fungicides such as SNPs, ChNP and AZOX-AFS-Pec, etc., triggers resistance against ShB [100, 102, 104]. To combat the devastating impact of sheath blight disease and ensure stable and sustainable agricultural practices in the future, there is an increasing focus on integrating conventional, nanotechnological, and novel approaches, including CRISPR-mediated genome editing of susceptible genes.

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Declarations

Ethics approval and consent to participate This article does not contain any studies with human or animal subjects.

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