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Molecular and physiological interplay between *Rhizoctonia solani* pathogenesis and rice defence: Recent advances and future outlook

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Abstract

Rice sheath blight, caused by the necrotrophic fungus *Rhizoctonia solani,* remains a major constraint on global rice production, affecting 15–20 million hectares annually and causing yield losses of up to 50 % under epidemic conditions. The pathogen's broad host range, extensive genetic and pathogenic variability and the absence of durable host resistance make disease management particularly challenging. *Rhizoctonia solani* induces host tissue necrosis through cell wall-degrading enzymes, toxins, oxalic acid and effector molecules, disrupting photosynthesis, oxidative balance and carbon metabolism. In response, rice deploys a multilayered defence network involving pattern recognition receptors, jasmonic acid and ethylene mediated signalling, redox homeostasis, transcriptional reprogramming and cell wall reinforcement, reflecting the dynamic interplay between pathogen aggressiveness and host defence that determines disease outcome. Genetic studies have identified over 100 quantitative trait loci linked to sheath blight resistance and functional analyses have revealed key resistance-associated genes, such as *SBRR1-R* from indica germplasm, a promising candidate for molecular breeding that still awaits functional validation and field-level confirmation across diverse genetic backgrounds. Building on this foundation, emerging strategies including clustered regularly interspaced short palindromic repeats/CRISPR-associated (CRISPR/Cas) based engineering of susceptibility genes, host-induced gene silencing, defence priming, beneficial microbe-mediated induced systemic resistance and genome-assisted breeding offer environmentally sustainable avenues for durable disease management. This review synthesises recent advances in the molecular and physiological mechanisms underlying the rice-*R. solani* pathosystem and outlines emerging strategies for engineering durable sheath blight resistance.

Keywords: cell wall-degrading enzymes; disease resistance; host-pathogen interaction; *Rhizoctonia solani*; rice; sheath blight

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1. Introduction

Rice (*Oryza sativa* L.) belongs to the family *Poaceae* and is one of the world's most important cereal crops and a staple food for nearly half the global population. Its inflorescence is a terminal panicle and its fruit is a caryopsis, in which the pericarp is fused to the seed coat. Asia alone produces and consumes almost 90 % of the world's supply, making Asian rice production central to global food security. Global production reached approximately 542 million metric tonnes in 2024/2025, with the global rice market valued at USD 305–324 billion and projected to exceed USD 380 billion by 2033. These figures highlight both the scale of rice based agriculture and the economic stakes of any threat to its productivity (1). Sheath blight, caused by the soil borne necrotrophic pathogen *Rhizoctonia solani*, affects 15–20 million hectares of irrigated rice fields in Eastern Asia and causes annual losses of approximately 6 million tonnes of grain, comparable to rice blast (2, 3). Its incidence has risen with the adoption of high-yielding, semi-dwarf varieties grown under high nitrogen input and narrow spacing, conditions that favour a humid field microclimate conducive to infection (4). *Rhizoctonia solani* infects over 32 plant families, persists across seasons via dormant sclerotia and shows high genetic variability. Complete resistance sources remain unavailable, making resistant cultivar development difficult (5).

Unlike classical plant-pathogen interactions, which are dominated by salicylic acid (SA)-mediated hypersensitive responses effective against biotrophs, *R. solani*'s necrotrophic strategy, including infection cushion formation, secretion of cell wall-degrading enzymes and phytotoxins and effector-driven induction of host cell death, subverts these same defence outputs to its own advantage. Consequently, rice resistance relies predominantly on jasmonic acid (JA)/ethylene (ET) signalling rather than SA, reinforced by SA-JA/ET antagonism (6, 7). This host-pathogen conflict drives extensive physiological reprogramming (reduced photosynthetic efficiency, altered carbon metabolism, oxidative stress) alongside a coordinated defence response involving pattern recognition, mitogen activated protein kinase (MAPK) signalling and transcriptional activation of pathogenesis-related (PR) proteins and phytoalexins. Despite this, resistance remains quantitative, governed by numerous minor effect loci and no complete resistance source has been identified (8).

While genomic, transcriptomic, proteomic and secretome studies have substantially advanced our understanding of this pathosystem, their findings remain fragmented across disciplines. The mechanistic links between pathogen virulence factors, host signalling and the ultimate physiological transition from defence to susceptibility remain poorly integrated. In particular, the mechanisms by which effector- and enzyme-driven reprogramming of carbon metabolism and hormonal crosstalk convert host defence signalling into susceptibility remain insufficiently understood. This review addresses this gap by providing an integrated physiological, molecular, genetic and omics-based perspective on the rice-*R. solani* interaction, with particular emphasis on recent advances including the *SBRR1-R* resistance allele, transcriptomic and proteomic insights, emerging evidence for epigenetic defence memory and the potential of cross-kingdom RNAi and host-induced gene silencing for sheath blight management.

Scope and methodology

This review aims to provide an integrated assessment of the molecular and physiological interactions between rice and *R. solani*, with special reference to recent progress in host defence mechanisms, pathogen virulence strategies, rice resistance genetics and disease management in the rice-*R. solani* pathosystem. Data were retrieved through a systematic search of the major scientific databases: Web of Science, Scopus, PubMed, Google Scholar and ScienceDirect. The literature search was completed on 31 May 2026. We considered publications from January 2000 to May 2026 and included earlier foundational studies where necessary to provide historical and conceptual context. The search strategies included combinations of the following keywords: "*Rhizoctonia solani*, rice sheath blight, rice defence mechanisms, plant pathogen interactions, cell wall degrading enzymes, effector proteins, reactive oxygen species, jasmonic acid, ethylene signalling, quantitative trait loci, *SBRR1*, transcriptomics, proteomics, epigenetics, RNA interference, defence priming and induced systemic resistance.

Peer-reviewed research articles, review articles, book chapters and relevant conference proceedings were screened for relevance based on predefined inclusion and exclusion criteria. The inclusion criteria comprised studies focusing on the molecular biology, physiology, genetics, genomics, host-pathogen interactions and disease management of rice sheath blight, as well as studies reporting recent advances in multi-omics technologies, epigenetic regulation, host-induced gene silencing, defence priming and resistance-associated loci. We placed particular emphasis on recent findings on newly identified resistance loci, including *SBRR1-R*. Exclusion criteria included publications lacking sufficient experimental or scientific evidence, studies unrelated to rice or *R. solani*, studies focusing on other pathosystems without direct relevance to rice sheath blight and duplicate publications or reports. The selected literature was synthesised to provide a comprehensive and updated perspective on the mechanisms underlying sheath blight pathogenesis and resistance. Several reviews have summarised various aspects of the rice-*R. solani* pathosystem, including resistance breeding, host-pathogen interactions, genomics-assisted improvement and disease management strategies. However, most previous reviews have examined these aspects largely in isolation, with limited integration of pathogen virulence, host physiological responses, molecular defence signalling, genetic resistance and emerging technologies. The unique scientific contribution of this review is establishing an interconnected host-pathogen framework that links pathogen virulence mechanisms to rice physiological and molecular responses and further connects these responses to resistance genetics, emerging omics approaches and practical disease management. Rather than providing a topic-wise summary, this review critically examines how these biological processes interconnect and how molecular insights can be translated into resistance breeding and sustainable sheath blight management. In addition, recent advances in resistance-associated loci, defence priming, epigenetic regulation and molecular approaches are evaluated for their potential contribution to durable resistance. Thus, the review provides a mechanistic-to-application perspective, bridging fundamental understanding of rice-*R. solani* interactions with strategies for developing durable and sustainable sheath blight management approaches. To clarify this review's scope and contribution relative to earlier reviews, Table 1 presents a comparative assessment of selected reviews. The comparison considers the extent to which key components of the rice-*R. solani* pathosystem, including host physiology, pathogen effectors, *SBRR1-R*, susceptibility genes and epigenetic regulation and defence priming, have been addressed. Based on this synthesis, Fig. 1 presents the conceptual framework adopted in this review to illustrate the

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interconnected processes underlying rice-*R. solani* interactions. The framework links pathogen virulence factors with host perception and downstream hormone signalling, physiological responses, genetic regulation and emerging omics-based approaches, integrating the major components of the rice-*R. solani* pathosystem that collectively contribute to durable resistance strategies. Comparison of recent reviews on the rice-*R. solani* pathosystem and the unique contribution of the present review is provided in Table 1. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram for the systematic review is illustrated in Fig. 2.

Table 1. Comparison of recent reviews on the rice-*Rhizoctonia solani* pathosystem and the unique contribution of the present review

Main focus	Host physiology	Pathogen virulence and effectors	SBRR 1-R	Susceptibility genes	Epigenetic regulations and priming	Host defence and signalling	Resistance gene/ QTLs	Integrated host-pathogen framework	Omics and defence priming
Resistance breeding and QTLs	Limited	Limited	No	No	No	Moderate	Comprehensive	Limited	Limited
Molecular interactions between rice and *Rhizoctonia solani*	No	Moderate	No	No	No	Comprehensive	Limited	Moderate	Moderate
Hormonal regulation and defence-related genes	No	No	No	Limited	No	Comprehensive	Limited	Limited	No
Major QTLs and defence genes	Limited	No	No	No	No	Moderate	Comprehensive	Limited	Moderate
Resistance mechanisms	Limited	No	No	No	Moderate	Moderate	Comprehensive	Moderate	Moderate

*"No" indicates that the respective aspect was not addressed in the review; "Limited" indicates brief or peripheral discussion of the aspect; "Moderate" indicates that the aspect was discussed with some relevant evidence but without extensive or integrated analysis; and "Comprehensive" indicates substantial and focused discussion supported by relevant evidence and critical synthesis. QTLs: quantitative trait loci.

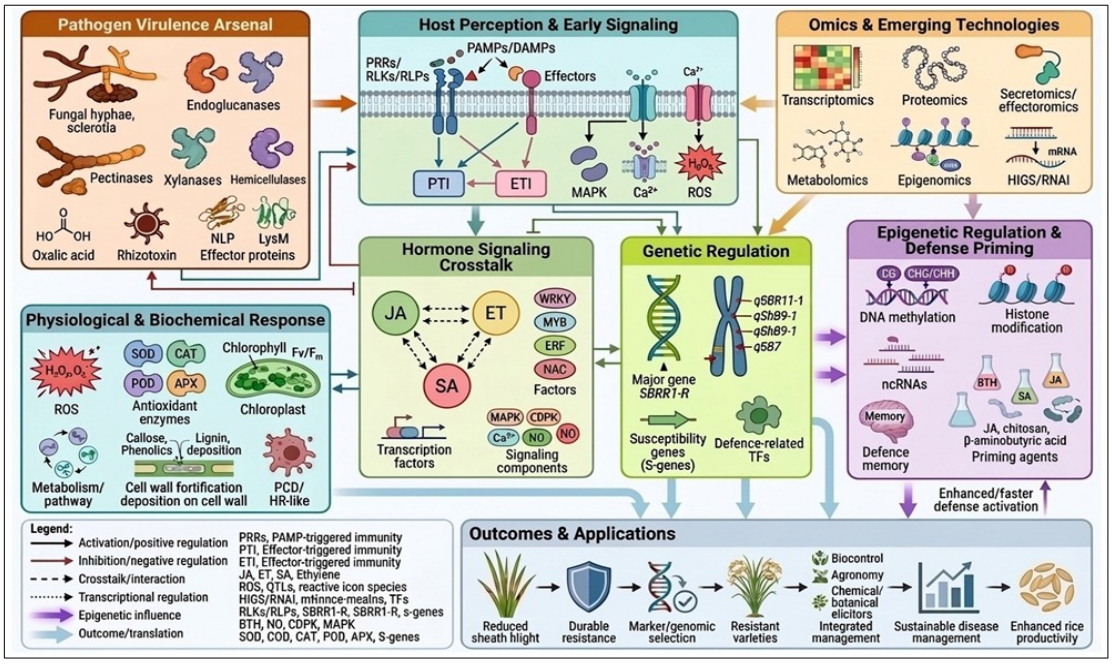


Fig. 1. Conceptual framework illustrating the major components of the rice-*Rhizoctonia solani* pathosystem, integrating pathogen virulence factors, host perception, phytohormone signalling, physiological responses, genetic regulation and emerging omics approaches that collectively contribute to durable resistance strategies. *The present review uniquely integrates all components into a single framework connecting pathogen virulence, host physiology, molecular defence, genetics and translational management strategies.

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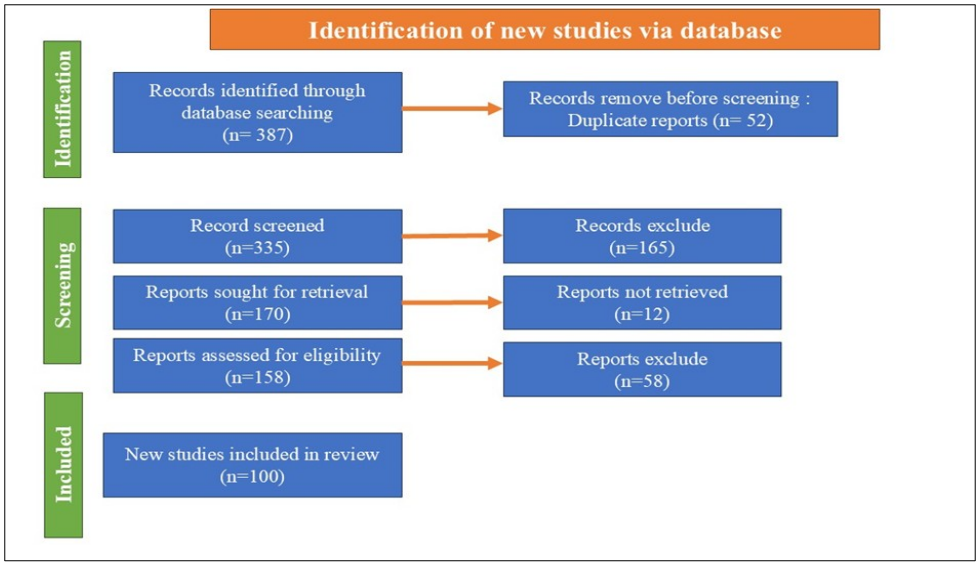


Fig. 2. PRISMA-style flow diagram illustrating the literature search and study selection process used in this review. *A total of 387 records were identified from Scopus, Web of Science, PubMed and Google Scholar Databases. After the removal of 52 duplicate records, 335 records were screened, of which 165 were excluded based on title and abstract screening. Of the remaining 170 reports sought for retrieval, 12 could not be retrieved. Subsequently, 158 full-text articles were assessed for eligibility and 58 articles were excluded following full-text evaluation, resulting in the inclusion of 100 studies in the final review. PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

Biology, current status and pathogenicity of *Rhizoctonia solani*

Taxonomy, anastomosis groups and host range

The genus *Rhizoctonia* belongs to the kingdom Fungi, phylum Basidiomycota, subphylum Agaricomycotina, class Agaricomycetes, order Cantharellales and family Ceratobasidiaceae, with *Thanatephorus cucumeris* (Frank) Donk recognised as the teleomorph of *R. solani* (9). This mitosporic fungus produces no asexual or sexual spores; instead, it relies on sclerotia as both its primary inoculum and principal survival structure (10). The mycelium is septate and multinucleated and characterised by a basal constriction at each hyphal branch. It exhibits right-angled branching and dolipore septa with colouration ranging from creamy white to light brown (11). Based on hyphal fusion compatibility, *R. solani* isolates are classified into 14 anastomosis groups (AG1–AG13 and AG-BI), of which AG1 comprises four intraspecific subgroups (AG1-IA, AG1-IB, AG1-IC and AG1-ID); AG1-IA is the causal agent of rice sheath blight. Comparative studies show AG1-IA is consistently more pathogenic than closely related subgroups such as AG1-IB and its dominance is attributed to a distinct toxin profile and higher production of cell-wall-degrading enzymes, which together drive faster lesion development and more severe yield loss than other anastomosis group (AG) groups infecting rice (12). Genomic analyses of AG1-IA isolates reveal expanded gene families associated with plant cell wall degradation, secondary metabolite biosynthesis and secretion of pathogenicity-related proteins, reflecting evolutionary adaptations toward aggressive necrotrophic colonisation of monocot hosts such as rice. This expanded enzymatic and metabolic arsenal likely underlies rapid lesion development and broad tissue maceration and helps explain why single-gene resistance has largely failed against this pathogen. It lacks a typical biotrophic effector repertoire but possesses a broad array of enzymes and candidate effectors that facilitate host tissue maceration and nutrient acquisition. Population-level studies further indicate high genetic variability within AG-IA, driven by somatic recombination, mutation and parasexual cycles, despite the absence of a well-defined sexual stage under field conditions. Moreover, considerable variation is observed among isolates in terms of virulence, mycelial growth rate, sclerotia production, enzymatic activity and sensitivity to fungicides, reflecting the high pathogenic and genetic diversity within *R. solani* populations. This isolate-level diversity complicates durable resistance breeding and fungicide reliability alike, since cultivars or treatments effective against one isolate population may fail against another. It explains why rice resistance has relied on multiple broad-spectrum quantitative trait loci (QTLs) rather than single genes and why candidate resistance genes must be validated across diverse field populations. *Rhizoctonia* uses a mass of monilioid and undifferentiated hyphal cells to form loose sclerotia (13). These structures are transported over long distances by irrigation water, where they germinate and form mycelium under suitable environmental conditions. This enables the fungus to survive in weed hosts during the offseason. The sclerotia can persist long-term in soil and stubble and reinfect the crop. The sclerotia and runner hyphae serve as the disease inoculum, but on rare occasions, basidiospores act as the inoculum. The biological characteristics of *R. solani* collectively hinder effective management of rice sheath blight. Its broad host range allows the pathogen to survive and maintain inoculum on alternative crops and weed hosts, facilitating persistence between rice-growing seasons. Additionally,

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substantial genetic and pathogenic diversity within *R. solani* populations leads to differential virulence and disease responses among rice genotypes, complicating the identification and deployment of stable, broadly effective host resistance. The pathogen's capacity to produce persistent sclerotia further enhances its survival in soil and infected crop residues, providing a recurrent source of primary inoculum for subsequent crops. Combined with favourable environmental conditions and intensive rice production practices, these factors diminish the effectiveness of single-component management strategies. Therefore, effective and durable sheath blight management necessitates integrating resistant cultivars with cultural, biological and chemical measures to reduce pathogen survival, inoculum and disease development. Climate change influences the epidemiology, distribution and severity of rice sheath blight by altering temperature, rainfall, relative humidity, crop-canopy microclimate and soil moisture. Elevated temperatures within the pathogen's optimal range enhance mycelial growth, infection and colonisation. Extended periods of high humidity and sheath wetness facilitate disease development and dissemination. Variations in rainfall patterns, such as intense rainfall events and irregular precipitation, modify soil moisture and canopy humidity, thereby affecting *R. solani* survival and activity. Increased atmospheric CO_2 indirectly affects disease by promoting rice biomass and canopy density, which can create a warmer, more humid microenvironment conducive to sheath blight. However, this response varies depending on rice genotype and environmental conditions. Additionally, climate-induced changes in cropping seasons, irrigation practices and crop growth further influence pathogen activity and extend periods favourable for infection (14, 15).

Current status, geographic distribution and yield losses

Previously, rice sheath blight was considered a minor disease but, in the wake of Green Revolution, it became one of the most serious rice diseases with the introduction of high yielding semi dwarf rice varieties, higher nitrogen application and higher planting density (16). Sheath blight of rice was first reported in Japan (17). Later reports confirmed its presence across temperate and tropical rice-growing regions worldwide, spanning Asia, America, Europe, Africa and Australia. In India, it was reported for the first time in the district of Gurdaspur, Punjab and was found to be a major production constraint in several states such as Punjab, Haryana, Uttar Pradesh, Uttarakhand, Bihar, West Bengal, Odisha, Chhattisgarh, Andhra Pradesh, Tamil Nadu, Karnataka, Kerala, Jammu and Kashmir, Madhya Pradesh, Assam, Manipur and Tripura (18). This disease causes a significant decrease in rice production through the effect on grain filling, panicle exertion, lodging and 1000-grain weight (19, 20). Severe infections at the booting and heading stages severely impair grain filling, leading to yield losses of 10–50 % during epidemics (21, 22). Rice sheath blight incidence has risen in recent years due to the intensification of rice cultivation, dense planting, high nitrogen fertiliser application and monoculture of high-yielding semi-dwarf varieties, compounded more recently by climate change, as elevated temperature, humidity and CO_2 levels favour pathogen growth and increase disease severity (11, 12). Given its widespread geographic spread and large associated losses, the molecular mechanisms of the disease and sustainable, genomics based management strategies need to be better understood. Fig. 3 illustrates the global distribution of rice sheath blight disease across temperate and tropical rice-growing regions.

Infection structures, hyphal aggregation and sheath colonisation

Rhizoctonia solani has a necrotrophic infection strategy, which involves forming specialised infection structures, aggregating hyphae and aggressively colonising the rice sheath. *Rhizoctonia solani* does not form haustoria, but penetrates by mechanical force and cell wall enzymatic degradation in direct contact with the host, using infection cushions and lobate appressoria as specialised penetration structures. Infection starts with hyphal adhesion to surfaces, which is enhanced by extracellular matrix components such as polysaccharides and hydrophobin-like proteins that promote surface recognition and adhesion (23). When the fungus detects signals from the host, it produces infection cushions, which consist of many cells and are made up of swollen hyphae used as penetration organs. They produce localised pressure and concentrated lytic enzymes at sites of infection. Their development is regulated by several conserved pathways controlling their cytoskeletal rearrangements and pathogenic differentiation, such as mitogen-activated protein (MAP) kinases and cyclic adenosine monophosphate (cAMP) (cAMP) dependent signalling pathways. The major event in *R. solani* pathogenesis is hyphal aggregation, which is a highly interwoven hyphal network on the sheath surface. The infection efficiency is improved by these aggregates because they allow simultaneous penetration at multiple locations. The network connectivity is further enhanced by hyphal anastomosis, which enables the redistribution of nutrients (24). At this stage, the pathogen produces a wide range of cell wall degrading enzymes, including polygalacturonases, cellulases, hemi-cellulases and pectin-modifying enzymes, which help macerate host tissues and promote fungal growth and colonisation (25). *Rhizoctonia solani* penetrates rice tissues and colonises both intercellular and intracellular spaces. This dual colonisation strategy facilitates rapid pathogen spread and host tissue damage. Intercellular growth enables the fungus to spread through the apoplast between cells, whereas intracellular invasion provides access to host cellular contents, thereby promoting nutrient acquisition and accelerating tissue necrosis. This colonisation is accompanied by the secretion of necrosis-inducing proteins and oxalic acid, which lead to host cell death, along with effectors that disrupt redox homeostasis and suppress host defence responses (26).

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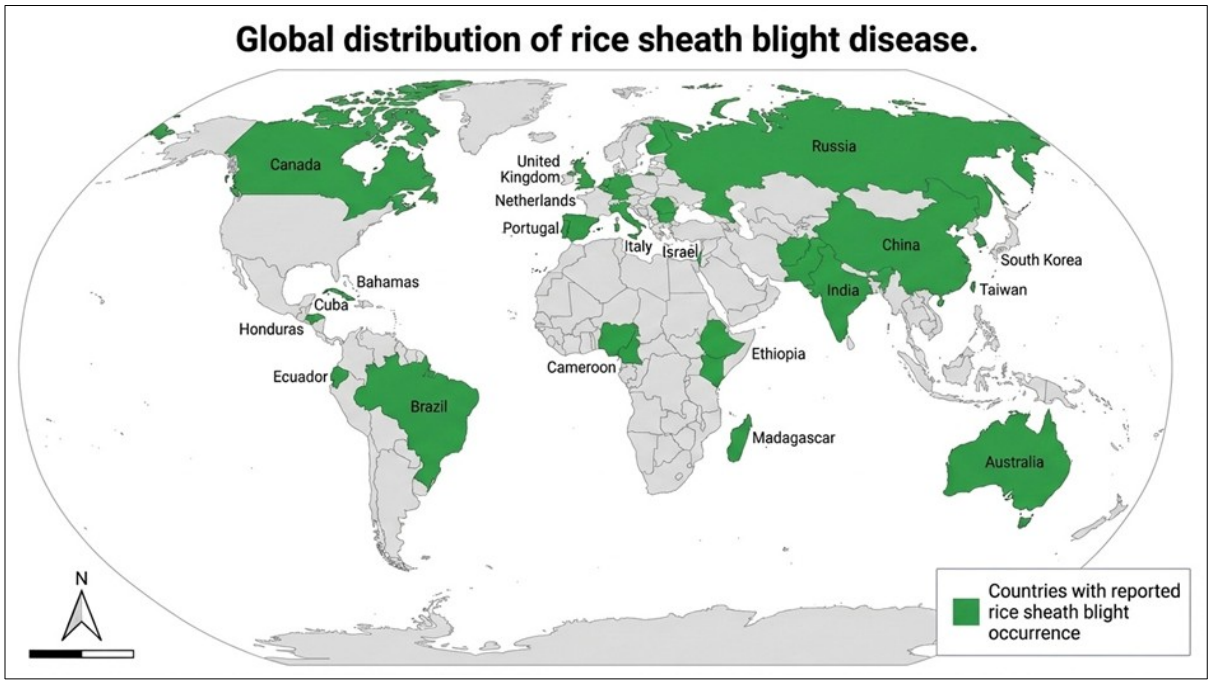


Fig. 3. Global distribution of rice sheath blight disease across temperate and tropical rice-growing regions.

Molecular and physiological mechanisms of pathogenesis

The pathogenesis of *R. solani* is a highly coordinated mechanism of physiological adaptations and a molecular arsenal of pathogenicity factors that enables rapid host invasion, immune suppression and necrotrophic colonisation. Recent multi-omics integrative studies have improved our understanding of the pathogenesis of *R. solani.* The temporal regulation of pathogenicity factors, from early penetration to tissue necrosis, is a critical determinant of disease progression. Premature or mistimed expression of cell wall-degrading enzymes and necrosis-inducing effectors may activate host defence responses before the pathogen has established sufficient biomass to withstand them. In contrast, coordinated, stage-specific deployment of these factors may enable *R. solani* to minimise host recognition during penetration and colonisation, followed by aggressive tissue destruction after nutrient acquisition has been established.

Cell wall-degrading enzymes

Successful colonisation of rice tissues by *R. solani* begins with the disruption of the cell wall, the primary structural barrier that restricts pathogen ingress. *Rhizoctonia solani* AG1-IA is a necrotrophic fungus that produces a plethora of cell wall-degrading enzymes (CWDEs) that attack the major components of the rice sheath cell wall. The pathogen encodes a robust arsenal of carbohydrate-active enzymes (CAZymes) that includes glycoside hydrolases, polysaccharide lyases and carbohydrate esterases, all of which are involved in the degradation of cellulose, hemicellulose, pectin and compounds associated with lignin (27). The cellulolytic activity of enzymes like endoglucanases, exo-glucanases and β-glucosidases breaks cellulose fibres to small soluble sugars, which reduces the rigidity and also provides nutrients for the colonisation of the fungus. Similarly, hemicellulose-degrading enzymes such as xylanase, mannanase and arabinofuranosidases break the cross-linked matrix, which surrounds the cellulose microfibrils, thus reducing the stability of the wall and increasing the vulnerability of the host tissue (28). In particular, pectin-degrading enzymes are important in the early stages of infection among the various CWDEs. The enzymes polygalacturonases, pectate lyases and pectin methyl esterases work together to break down pectic substances in the middle lamella, freeing up adjacent plant cells and facilitating tissue maceration. Up regulation of these enzyme-encoding genes has been associated with higher virulence and fast colonisation of the sheaths. In addition, transcriptome-based studies indicate that production of the CWDE is highly coordinated in the course of disease development, with pectinases being strongly induced during early stages of penetration and cellulase and hemi-cellulase activity during later stages of the necrotrophic expansion (29). Furthermore, the action of lignin-modifying enzymes like laccases and peroxidases helps break down or modify the lignin of secondary cell walls, allowing deeper penetration of fungal tissue into the mature tissues. Some xylanases have the ability to trigger the host defence system and serve as elicitor molecules, demonstrating that CWDEs not only play a role in tissue degradation but also in host-pathogen interactions (30). Consistently, proteomic and RNA-seq studies have found that during active colonisation, there is increased expression of glycoside hydrolases, endoglucanases and β-1,4-xylanases, which indicates their involvement in disease progression and nutrient acquisition (31).

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Besides assisting the physical invasion, CWDEs are also important in influencing rice-*R. solani* interaction with defence mechanisms. Damage-associated molecular patterns (DAMPs), such as oligogalacturonides, are released during cell wall degradation and may trigger pattern-triggered immunity (PTI) by inducing downstream defence signalling in rice. *Rhizoctonia solani* can, however, modulate these responses by controlled secretion of CWDEs and other virulence-associated molecules that disrupt host immunity. For example, the LysM effector *RsLysM* binds chitin oligomers released from the fungal cell wall during infection, thereby preventing their recognition by host chitin receptors and suppressing chitin-triggered immune signalling (32). *Rhizoctonia solani* has a relatively high and diverse number of CAZymes, which has been demonstrated through comparative genomic analyses, indicating that it has a necrotrophic lifestyle. Enzymes are further stimulated by processes of acidification (acidifying effect of oxalic acid) and oxidative stress induction, which result in a favourable environment for colonisation of tissues. Rice plants, on their part, work to strengthen themselves by lignifying the cell wall, depositing callose, producing an oxidative burst and synthesising polygalacturonase-inhibiting proteins (PGIPs). Although these defence mechanisms occur, the powerful enzymatic arsenal of *R. solani* frequently allows host colonisation and the sheath necrosis does not extend far, resulting in a decrease in photosynthetic efficiency and considerable yield losses (33).

Toxin and oxalic acid production

Among the diverse arsenal of virulence factors produced by *R. solani* during the colonisation of rice, phytotoxins and oxalic acid occupy a central position. These chemical weapons act ahead of or alongside mycelial invasion to destabilise host physiology, suppress defence signalling and facilitate tissue necrosis (34). The major toxins produced by *R. solani* are phenylacetic acid (PAA), m-hydroxyphenylacetic acid, 3-methylthiopropionic acid and many phenolic and organic acids. Phenylacetic acid is a key virulence associated toxin in sheath blight pathogenesis. It triggers electrolyte leakage, chloroplast disorganisation, membrane lipid peroxidation and programmed cell death. It also interferes with photosynthetic efficiency and mitochondrial functions, leading to the weakening of the host's physiological defence activities during the early pathogenic events. Comparative evidence demonstrates that highly virulent isolates generally produce a greater amount of PAA than less virulent strains. This suggests a positive correlation between toxin accumulation and aggressiveness. Toxin-mediated disruption of plasma membrane permeability enhances the penetration efficiency of infection cushions and facilitates the diffusion of fungal enzymes into the host tissues (35).

Oxalic acid (OA) is one of the principal virulence determinants during sheath blight pathogenesis through the enzymatic activity of oxaloacetate acetyl hydrolase (OAH), which catalyses the cleavage of oxaloacetate into oxalate and acetate. During the early stages of infection, *R. solani* secretes OA, which acidifies host tissues, chelates calcium ions from the cell wall and promotes the formation of calcium oxalate complexes, thereby weakening cell wall integrity and facilitating the action of cell wall-degrading enzymes. Virulent isolates generally produce higher levels of OA than less virulent isolates, indicating a positive association between OA production and pathogenicity. Rice oxalate oxidase (*OsOXO4*)-mediated oxalate metabolism counteracts these effects by degrading OA into carbon dioxide and hydrogen peroxide (H_2O_2). The resulting H_2O_2 functions as a defence signal that triggers downstream immune responses, primarily by oxidising and activating redox sensitive transcription factors and kinase cascades (e.g., MAPK signalling), which in turn activate the hypersensitive response, systemic acquired resistance (SAR), phytoalexin biosynthesis and pathogenesis-related (PR) gene expression, thereby restricting further fungal colonisation. Transgenic rice plants overexpressing *OsOXO4* exhibit enhanced oxalate degradation, elevated expression of defence-associated genes such as phenylalanine ammonia-lyase (PAL) and RC24 and significantly improved resistance to sheath blight under controlled greenhouse conditions, highlighting the importance of oxalate metabolism in modulating rice-*R. solani* interactions. At later necrotrophic stages, OA triggers programmed cell death and inhibits phenolic defence compound synthesis, facilitating tissue maceration and nutrient acquisition. The biphasic role of OA, early suppression followed by promotion of necrosis, represents a sophisticated manipulation of rice physiology that distinguishes highly virulent from weakly virulent strains (36).

Effector proteins and virulence factors

Pathogenicity of *R. solani* AG1-IA relies on a well-coordinated arsenal of effector proteins and virulence-associated determinants which enable invasion of the host, immune evasion, tissue degradation and colonisation. *Rhizoctonia solani* is a necrotroph and exploits host cell death as a nutritional source so it produces effectors that target host physiological and molecular defence mechanisms to establish the disease. The genomic and secretome analyses have identified a wide variety of candidate effectors, such as proteins with tissue necrosis-inducing activity, *LysM*-domain proteins, cell wall degrading enzymes, proteases, transporters and oxidative stress-responsive enzymes, which are activated during infection cushion formation and invasive growth in rice sheath tissues. These virulence determinants are coordinately expressed, allowing the pathogen to quickly colonise host tissue and promote the rate of sheath blight (37). During *R. solani* invasion, the major effectors that are produced are the necrosis- and ethylene-inducing peptide-like proteins (NLP), mainly *RsNLP1* and *RsNLP2,* which act as potent cytotoxic effectors during host colonisation. These secreted proteins bind to the glycosylinositol phosphoryl ceramides in the plasma membrane and cause membrane permeability, electrolyte leakage, chloroplast leakage and chloroplast

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disintegration and programmed cell death such as seen in infected rice tissue. The induction of *RsNLP* expression was correlated with the growth of lesions and disease severity during infection, indicating their role in necrotrophic pathogenesis. *Rhizoctonia solani* also produces *LysM*-domain effector proteins that bind free chitin oligosaccharides released from the fungal cell wall during infection, effectively sequestering these fragments and preventing their recognition by host chitin receptors such as plant *LysM* receptor kinases (e.g., *CEBiP/CERK1*). By outcompeting the host receptors for chitin binding and in some cases directly shielding hyphae from host chitinases, these effectors block the initiation of pattern-triggered immunity (PTI) and dampen downstream defence signalling, including MAPK activation, the oxidative burst and defence-gene expression (38). Recent research demonstrates that *R. solani* employs complex strategies of immune suppression during infection. Recent studies have shown that the effector AGLIP1 from *R. solani* AG1-IA contains a lipase domain and causes cell death in *Nicotiana benthamiana* Domin and rice protoplasts. The signal peptide and conserved catalytic residues of lipases were found to be key to the necrosis activity based on functional analysis. Furthermore, expression of AGLIP1, a glycoside hydrolase-family effector secreted during *R. solani* infection, is capable of inhibiting pathogen associated molecular pattern (PAMP)-triggered immunity (PTI) in rice; heterologous expression of AGLIP1 in *Arabidopsis thaliana* (L.) Heynh. was shown to strongly suppress the induction of pattern-triggered immunity (PTI) marker genes normally triggered by flg22 and chitin, thereby dampening basal defence signalling and facilitating disease progression. Additionally, *AGLIP1* transgenic plants were found to be more susceptible to bacterial infection, corroborating *AGLIP1* as a virulence-associated effector that facilitates disease development by inducing cell death in the host while suppressing basal immune responses (39).

Oxidative stress management and signal transduction pathways further play indispensable roles in the virulence strategy of *R. solani*. Rice plants produce oxidative bursts, which consist of hydrogen peroxide and superoxide radicals, as part of basal defence mechanisms against infection. To overcome these responses, *RsCAT1*, *RsSOD1* and *RsGST1* are induced by *R. solani,* producing reactive oxygen species (ROS) detoxification enzymes that help to maintain fungal redox homeostasis during invasion. At the same time, infection cushion differentiation, expression and secretion of virulence-associated proteins of pathogens are under the control of both MAP kinase and cAMP-dependent signalling pathways (40). These effectors and virulence factors, acting together, form an integrated pathogenicity network which helps *R. solani* to breach host immunity and manipulate host cellular physiology, allowing aggressive necrotrophic colonisation in rice. The interaction between *R. solani* and rice during infection is governed by a dynamic molecular crosstalk, in which both partners actively deploy and respond to a suite of biochemical signals. On the pathogen side, *R. solani* secretes cell wall-degrading enzymes to breach host physical barriers, releases toxins and secondary metabolites that compromise host cellular integrity, generates reactive oxygen species (ROS) that contribute to necrotrophic killing of host tissue and deploys effector proteins that modulate host immune signalling. In parallel, rice mounts a coordinated defence response, marked by an oxidative burst, activation of phytohormone signalling pathways (including salicylic acid, jasmonic acid and ethylene-mediated defence circuits), large-scale transcriptional reprogramming of defence-related genes and the biosynthesis of antimicrobial phytoalexins. The reciprocal and simultaneous nature of these fungal and host responses underscores that sheath blight pathogenesis is not a unidirectional process but rather an outcome of continuous molecular negotiation between pathogen virulence strategies and host defence mechanisms. A schematic representation of the molecular and physiological interactions occurring during rice-*R. solani* interactions is illustrated in Fig. 4.

Physiological responses of rice to infection

Changes in photosynthesis and carbon metabolism

The photosynthetic efficiency and primary carbon metabolism of rice are severely altered by infection by *Rhizoctonia solani* even before the onset of sheath blight disease symptoms, resulting in significant physiological alterations that play an important role in sheath blight symptom formation and yield loss. The pathogen invades the sheath tissues and causes considerable chlorosis, necrosis and expansion of lesions, which leads to disruption of chloroplast structure and photosynthetic machinery, resulting in a reduced ability to assimilate carbon. It has been found that *R. solani*-infected rice plants show significantly reduced chlorophyll content, stomatal conductance, transpiration rate and net photosynthetic rate compared to healthy plants. These physiological declines have been most extensively documented in susceptible rice genotypes, whereas resistant genotypes show comparatively smaller reductions in chlorophyll content and photosynthetic gene suppression, correlating with their enhanced disease tolerance (41). Further ultrastructural investigations showed the disorganisation of thylakoid membranes, swelling of chloroplasts and degradation of grana stacks after *R. solani* infection, which are signs of significant damage to the photosynthetic apparatus. These changes affect the efficiency of light harvesting and electron transport and lead to a decrease in the production of adenosine triphosphate (ATP) and nicotinamide adenine dinucleotide phosphate (NADPH) necessary for normal metabolic functions. Loss of photosynthetic performance in sheath blight infection is also linked to oxidative damage caused by the pathogen and disruption of the integrity of chloroplasts due to the formation of toxins. Virulence-associated metabolites stimulate lipid peroxidation and disorder of chloroplast membranes, resulting in faster induction of senescence-like symptoms in infected tissues. At the same time, ROS build-up during host-pathogen interaction causes damage to photosystem II (PS II) reaction centres and reduces the

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photochemical efficiency. Transcriptomics studies of infected rice tissues have shown that genes associated with photosynthesis, such as those involved in carbon fixation and the assembly of photosystems and genes involved in chlorophyll biosynthesis, are under expressed. Infected plants, as a result, have a negative effect on the production of carbohydrates and have less energy for growth and defence responses (42).

Rice defence-associated metabolic pathways are closely related to the changes in carbon metabolism. It is important to note that substantial amounts of carbon are needed for enhanced phenylpropanoid metabolism, lignin biosynthesis and production of defence-related secondary metabolites during infection. Hence, resistant rice varieties frequently have more efficient metabolic reprogramming systems, able to manage the activation of defence response while maintaining photosynthesis. Contrast is made with susceptible cultivars, which usually experience a high level of photosynthetic suppression, premature senescence and uncontrolled carbon loss as disease progresses. Hence, the role of maintaining photosynthetic efficiency and metabolic homeostasis is gaining increasing recognition as a key aspect of quantitative resistance in sheath blight. A mechanistic view of the physiological and molecular basis of photosynthetic disruption and carbon-metabolism reconfiguration could create new possibilities to enhance rice stress tolerance through metabolic engineering and breeding approaches. For instance, engineering rice to stabilise chloroplast integrity and delay chlorophyll degradation during infection, such as through overexpression of chloroplast protective genes like *OsCSP41b* or silencing of chlorophyll catabolic genes like *OsNYC3,* has been shown to sustain photosynthetic gene expression and enhance sheath blight resistance without compromising yield (43).

Oxidative stress and antioxidant responses

Oxidative stress is the first and most primary physiological reaction in sheath blight pathogenesis. Rice cells recognise pathogens rapidly and then, within a few minutes, they produce reactive oxygen species (ROS) such as superoxide radical, hydrogen peroxide, hydroxyl radical and singlet oxygen, using plasma membrane localised NADPH oxidases. The oxidative burst acts as an important part of basal defence, directly inhibiting pathogen growth by strengthening cell walls and by activating downstream immune signalling pathways. However, high levels of ROS can actually increase disease susceptibility. While ROS is a central component of pattern-triggered immunity (PTI)/effector-triggered immunity (ETI)-associated defence signalling against biotrophic pathogens, necrotrophs exploit the host's own oxidative burst to their advantage: excessive ROS accumulation triggers host programmed cell death and tissue necrosis and since necrotrophic fungi feed on dead and dying tissue, this cell death and tissue necrosis effectively creates a nutrient-rich, colonisable environment that favours fungal establishment and spread rather than restricting it. Therefore, the interaction of rice with *R. solani* is greatly dependent on the balance between oxidative damage and ROS-mediated defence (44). Excessive ROS accumulation during sheath blight progression leads to serious oxidative damage to cell components such as lipids, proteins, nucleic acids and membranes. In infected rice tissues, malondialdehyde, an index of membrane destabilisation and oxidative damage, is frequently observed at elevated levels. Chloroplasts are especially sensitive to ROS toxicity and this leads to a decreased permeability of the thylakoid membranes that culminates in accelerated senescence and collapse of plant tissue surrounding the infection sites. During the necrotrophic stage of the infection, necrosis-inducing proteins, toxins and oxalic acid secreted by *R. solani* further exacerbate ROS-caused cell damage. Rice plants develop an elaborate antioxidant defence system consisting of enzymatic and non-enzymatic scavenging systems in response to oxidative stress. The key antioxidant enzymes are superoxide dismutase (SOD), which converts superoxide radicals to hydrogen peroxide; catalase (CAT) and ascorbate peroxidase (APX), which detoxify hydrogen peroxide into water and oxygen; and peroxidases (POX) and glutathione reductase (GR), which maintain cellular redox homeostasis. These enzymes have been consistently found to be enhanced in resistant or moderately resistant rice cultivars following infection with *R. solani* and these enzymes are thought to play an important role in limiting oxidative damage and spread of *R. solani*. Also, in the case of pathogen challenge, non-enzymatic antioxidants like ascorbic acid, glutathione, flavonoids, phenolics and carotenoids play a role in ROS scavenging and membrane stabilisation (45).

Transcriptomic and proteomic analyses show that there is a high level of reprogramming of redox related pathways during rice sheath blight infection. Pathogen infection results in significant induction of genes related to ROS detoxification, glutathione metabolism, thioredoxin systems and stress-responsive transcription factors. At the same time, *R. solani* has an efficient antioxidant system including fungal catalases, superoxide dismutase and glutathione-S-transferases, which help the pathogen to withstand host-generated oxidative stress during colonisation. So, the rice-*R. solani* interaction is complex in nature and it is an oxidative duel in which the interdependence of both host and pathogen is capable of regulating ROS levels to support their survival. Controlled ROS signalling and effective antioxidant activity are now emerging as significant factors in quantitative resistance to sheath blight and could be good targets for breeding and molecular improvement of rice defence mechanisms. For instance, *OsERF65*, a stress-responsive transcription factor that represses peroxidase gene expression and thereby limits ROS scavenging capacity, has been identified as a promising target; knockout of *OsERF65* enhances antioxidant enzyme activity and significantly improves grain yield, making it an attractive candidate for molecular approaches (46).

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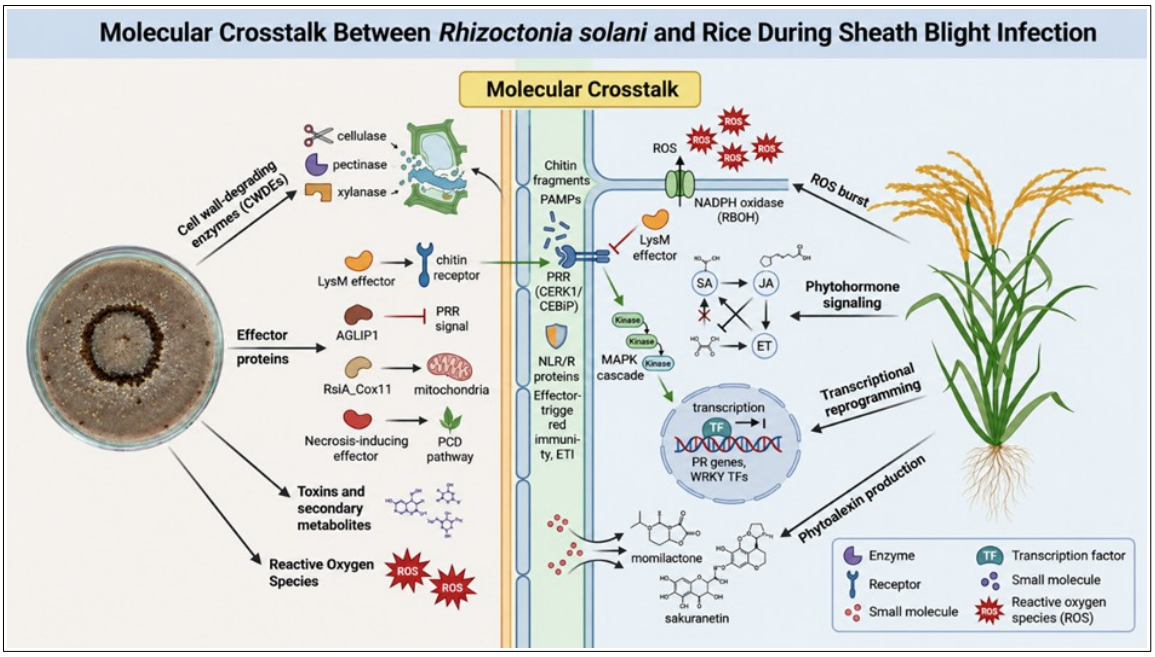


Fig. 4. Schematic representation of the molecular and physiological interactions occurring during rice-*Rhizoctonia solani* interactions. *The pathogen produces cell-wall-degrading enzymes, toxins, reactive oxygen species and effector proteins that promote host colonisation. Rice responds through oxidative burst, phytohormone signalling, transcriptional reprogramming and phytoalexin production. The dynamic crosstalk between pathogen virulence factors and host defence responses ultimately determines disease progression and resistance outcomes.

Cell wall fortification

Fortification of cell walls is a key structural defence response in rice when infected by *Rhizoctonia solani* and is crucial to limit pathogen penetration and colonisation. *Rhizoctonia solani* is a necrotrophic fungus and the cell wall degrading enzymes (CWDEs) play a major role in its tissue invasion. Therefore, cell wall reinforcement serves as a crucial defence mechanism to restrict fungal spread. Upon pathogen recognition, rice plants quickly trigger defence-related pathways leading to the deposition of structural polymers at sites of infection. Callose is rapidly deposited beneath the plasma membrane at the pathogen contact site, forming papillae that provide an immediate, localised physical barrier to slow initial fungal penetration, whereas lignin deposition occurs later and acts more durably, reinforcing and rigidifying the host cell wall and restricting pathogen spread beyond the infection site. Together with phenolics and hydroxyproline-rich glycoprotein, which further cross-link and strengthen the cell wall matrix, these structural reinforcements collectively slow fungal ingress and containment during the early stages of *R. solani* colonisation. Such changes enhance cell wall strength, decrease enzymatic activities and decrease the ability of the pathogen to mechanically penetrate hyphae and the cushions of hyphae produced during the initial stages of invasion. A key response to sheath blight infection is that the phenylpropanoid pathway is activated, producing lignified tissues. The activities of enzymes, such as phenylalanine ammonia-lyase (PAL), peroxidases (POX) and cinnamyl alcohol dehydrogenase (CAD), regulate the biosynthesis and polymerisation of lignin that occurs around infected tissues (47). Lignin deposition is important to increase the strength of secondary cell wall structures and to create physical barriers to fungal growth. Lignification is often correlated with sheath blight resistance, as the resistant rice genotypes tend to have earlier and stronger lignification than susceptible cultivars. Lignin and other phenolic components can also provide structural support for the cell and may have antimicrobial activity that contributes to their ability to inhibit fungal growth (48).

Another defence mechanism of rice against *R. solani* involves callose deposition in the cell wall. Callose, which is rich in β-1,3-glucan, is deposited throughout the plant in high amounts at papillae and around penetration sites upon pathogen attack, thus limiting pathogen expansion within host tissues and the entry of hyphae. Callose accumulation is closely linked to ROS signalling, influx of calcium ions and activation of defence-responsive transcription factors during early immune responses (49). At the same time, rice plants induce cross-linking of cell wall proteins and hydroxycinnamic acid derivatives, thereby further strengthening the rigidity of the cell wall and lowering its susceptibility to CWDE activity of fungi. *Rhizoctonia solani*, however, has developed a wide spectrum of polygalacturonases, cellulases, xylanases and pectin methyl esterases that are able to break down the reinforced components of the cell wall and cause tissue maceration. In resistant cultivars, genes that encode cellulose synthases, biosynthetic enzymes for lignin and callose synthases. Silicon deposition underneath the cuticle has also been reported to promote mechanical resistance to pathogen penetration, forming a rigid

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silica-cuticle double layer that acts as a physical barrier against fungal ingress. In addition, silica accumulation in rice tissues has been reported to increase cell wall fortification by polymerising into amorphous silica within the cell wall matrix and cross-linking with pectin and other wall components, thereby increasing wall rigidity and resistance to enzymatic degradation by fungal CWDEs. Silicon has also been shown to prime host defence responses, including enhanced phenolic and phytoalexin accumulation at infection sites. The cell wall fortification, hence, is a multi-layered and dynamic defence mechanism involving structural reinforcement, biochemical modification and defence signalling to curb sheath blight development. Breeding, silicon nutrition and molecular engineering of these mechanisms could play an important role in the development of lasting resistance to *R. solani* in rice (50).

Phytohormone signalling

The interaction between rice and *R. solani* is regulated through phytohormone signalling to control immune activation, oxidative balance, metabolic reprogramming and defence-associated cell death, which are all pivotal in determining the outcome of the interaction. Unlike immunity that is normally triggered by salicylic acid (SA) signalling in the rice immunity pathway against biotrophic pathogens, rice resistance against *R. solani* is mainly mediated by jasmonic acid (JA) and ethylene (ET) signalling. But if the JA responses are activated for extended periods, then the metabolic costs are likely to be high, suggesting that the temporal regulation of JA signalling is crucial for balancing defence and growth. JA-mediated defences and ethylene signalling work synergistically and play an important role in resistance to *R. solani* (51). When infection occurs, the genes of 1-aminocyclopropane-1-carboxylic acid (ACC) synthase (ACS) and ACC oxidase (ACO) are activated and ethylene is produced in infected tissues. Ethylene induces the expression of defence-related genes, ROS signalling and strengthens stress-related pathways that inhibit fungal colonisation. However, during the later phases of infection, an overproduction of ethylene can promote chlorosis, senescence and programmed cell death; because *R. solani* is a necrotroph that feeds on dead and dying host tissue, this ethylene-driven breakdown of cell integrity and release of nutrients from senescing cells ultimately benefits necrotrophic growth by providing a more favourable nutritional environment and facilitating further fungal colonisation (52). However, accumulating evidence indicates that the contribution of JA and ET is not simply proportional to their magnitude of activation. Early and transient induction of JA-ET signalling appears beneficial because it promotes rapid defence deployment while limiting cellular damage. In contrast, sustained or excessive activation may impose substantial metabolic costs and exacerbate tissue senescence, chlorosis and programmed cell death, thereby creating conditions that favour necrotrophic growth. Consequently, resistance cannot be interpreted as the consequence of maximal JA-ET signalling, but rather as the ability to achieve precise temporal regulation of these pathways. Resistant genotypes frequently exhibit rapid but tightly controlled JA-ET responses coupled with efficient antioxidant systems, whereas susceptible cultivars often display delayed, prolonged or poorly coordinated hormonal responses that culminate in extensive necrosis and lesion expansion (53).

The role of salicylic acid (SA) signalling in sheath blight remains considerably more contentious. Several studies suggest that salicylic acid (SA) contributes to basal defence during the early stages of *R. solani* infection. However, accumulating evidence indicates that sustained SA accumulation may enhance susceptibility by antagonising JA-dependent defence pathways that are essential for resistance against necrotrophic pathogens. From this perspective, SA signalling may function as a transient early response but become detrimental when maintained at elevated levels. Such observations underscore the importance of hormonal crosstalk and suggest that inappropriate activation of biotroph-associated defence programs can compromise effective resistance to *R. solani*. Beyond JA, ET and SA other hormones further modulate sheath blight development. Auxin signalling positively regulates resistance, as exogenous auxin application and auxin-responsive gene activity have been shown to reduce disease severity, whereas disruption of auxin homeostasis increases susceptibility. Brassinosteroid (BR) signalling, in contrast, tends to negatively regulate resistance. The transcription factor *RAVL1* paradoxically also activates ET signalling, which promotes resistance, indicating that these hormone pathways can act antagonistically within the same regulatory node. Abscisic acid (ABA) has a less clearly defined role specifically in *R. solani* pathogenesis, but based on its well-characterised function in other rice-pathogen interactions, it is thought to act largely as a negative regulator of defence by antagonising SA- and JA-dependent signalling, while also mediating stress adaptation and growth- defence trade-offs under the abiotic stress conditions that often accompany sheath blight infection. These observations collectively indicate that hormonal regulation during sheath blight is highly context-dependent and cannot be adequately explained by individual pathways operating in isolation. Taken together, current evidence supports a conceptual shift in our understanding of sheath blight resistance. Rather than being governed by a single dominant resistance gene, as observed in gene-for-gene interactions with biotrophic pathogens, resistance to *R. solani* appears to arise from the combined, quantitative effects of multiple interacting hormonal pathways, each contributing incremental and context-dependent effects on defence gene expression, ROS homeostasis and growth-defence allocation. Because these pathways cross-regulate and sometimes antagonise one another, the net resistance phenotype reflects the balance of the hormonal network rather than the action of any single pathway. This helps explain why sheath blight resistance is inherited as a polygenic, quantitative trait involving multiple minor-effect QTLs rather than a single major resistance gene. Quantitative resistance to

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R. solani is increasingly viewed as the capacity of the host to maintain hormonal equilibrium, restrict excessive self-damage and coordinate defence signalling in a temporally appropriate manner. Identification of the regulatory nodes governing hormone crosstalk and signalling plasticity therefore represents a promising avenue for molecular breeding, genome editing and defence-priming strategies aimed at achieving durable resistance against rice sheath blight (54).

Molecular defence responses of rice

Pattern-triggered immunity

In rice, pattern-triggered immunity (PTI) is the initial level of inducible immunity that is triggered after the recognition of *Rhizoctonia solani*. Plasma membrane-localised pattern recognition receptors (PRRs) detect conserved pathogen-associated molecular patterns (PAMPs) of fungi such as fungal chitin fragments, β-glucans or cell surface derived oligosaccharides, which trigger PTI. The *LysM*-containing receptor-like proteins, namely chitin elicitor binding protein (*CEBiP*) and chitin elicitor receptor kinase 1 (*OsCERK1*) are mainly responsible for chitin recognition in rice. Upon binding chitin oligosaccharides to these receptors, the receptor complexes are formed and phosphorylation cascades lead to activation of downstream immune signalling pathways. Early recognition event is extremely crucial for preventing the establishment of the pathogen and is also important to determine basal resistance of *R. solani* (55). Pattern-triggered immunity is able to trigger several defence-associated cellular reactions within a few minutes, which are collectively called PAMP-triggered defence reactions. These are calcium ion influx, depolarisation of the membrane, activation of mitogen-activated protein kinase (MAPK) cascades and generation of reactive oxygen species (ROS) and transcriptional reprogramming of defence-related genes. Calcium signalling is a second messenger system in early steps which activates defence responses via calcium-dependent protein kinases (CDPKs) and calmodulin (CaM). Simultaneously, MAPK signalling cascades involving MAP kinase kinase kinases (MAPKKKs), MAP kinase kinases (MAPKKs) and MAP kinases (MAPKs) amplify immune signals and regulate downstream transcription factors, particularly WRKY proteins, leading to activation of defence-related genes. The NADPH oxidases and apoplastic peroxidases are also sources of antimicrobial H_2O_2 production, which can cross-link structural proteins in the cell wall and promote lignification. In rice-*R. solani* interactions, secreted apoplastic peroxidases are commonly upregulated following pathogen recognition and function as key executors of PTI by driving the apoplastic oxidative burst. The H_2O_2 they generate is directly toxic to invading hyphae, while also acting as a substrate for oxidative cross-linking of cell wall glycoproteins and for lignin polymerisation, thereby reinforcing the cell wall as a physical barrier to fungal penetration and slowing pathogen advance during the early stages of infection. Up-regulation of genes encoding the phenylpropanoid metabolism, callose biosynthesis and production of phytoalexins helps to reinforce physical and biochemical barriers to fungal invasion. *Rhizoctonia solani*, however, is a necrotrophic fungus with a wide repertoire of virulence factors and infection is likely to be successful if the PTI responses are suppressed or evaded. The pathogen deploys *LysM*-domain effector proteins that bind chitin fragments and shield them from the rice immune receptors, to reduce the strength of chitin-triggered signalling and the activation of basal immunity. Moreover, secreted toxins and CWDEs can affect the cellular homeostasis of the host and immune-signalling pathways during infection. Transcriptomic and functional genomic studies of resistant cultivars of rice have revealed that resistant cultivars are able to induce PTI earlier and to a greater degree than susceptible cultivars. Sheath blight severity has often been correlated with both an increase in the levels of *OsCERK1*, *CEBiP*, MAPK-associated genes, ROS-generating enzymes and defence-responsive transcription factors. However, resistance to *R. solani* is quantitative and complex and PTI alone is often not enough to provide complete resistance. Because *R. solani* actively secretes effectors, CWDEs and toxins to suppress PTI signalling and to induce host cell death for its own nutritional benefit, the pathogen can effectively counteract or exploit PTI -associated responses rather than being restricted by them. Unlike biotrophic pathogens where single dominant resistance genes can confer strong gene-for-gene resistance, no such major resistance gene (R gene) has been identified against *R. solani* and resistance instead depends on the additive, quantitative contribution of multiple minor-effect genes and hormonal pathways, making complete resistance difficult to achieve through PTI activation alone. In this regard, the current research is increasingly focused on designing newer molecular breeding, receptor engineering and genome editing strategies to reinforce the initial immune perception and downstream defence signalling in the context of necrotrophic pathogens to improve the efficiency, stability and coordination of PTI (56).

Defence signalling pathways

Defence responses to *Rhizoctonia solani* in rice are multi-layered and very coordinated, involving both perception of the pathogen, Ca+ ion fluxes, ROS, mitogen-activated protein kinases (MAPKs) and phytohormone-mediated transcriptional reprogramming. Fungal PAMPs like chitin fragments are recognised by pattern recognition receptors like *OsCERK1* and *CEBiP*, which triggers rapid cytosolic calcium influx that activates calcium-dependent protein kinases, calmodulin-mediated pathways and causes oxidative burst and amplifies immune signalling. At the same time, MAPK signalling modules composed of *OsMPK3*, *OsMPK6* and upstream MAPKKKs bring immune signals from the extracellular environment to the level of transcription activation of a set of immune-responsive genes. These signalling pathways control the expression of genes involved in phytoalexin synthesis, phenylpropanoid metabolism, antioxidant defence and cell wall reinforcement which limit fungal penetration and lesion growth. Generally, resistant rice genotypes have much stronger and earlier activation of the signalling

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components involving MAPKs than the susceptible cultivars, indicating that efficient signal transduction is a key factor in determining quantitative sheath blight resistance (57).

As *R. solani* is a necrotrophic fungus, jasmonic acid (JA) and ethylene (ET)-mediated pathways are the major hormonal signalling pathways regulating the defence responses of rice. Following pathogen perception, JA biosynthesis is rapidly induced through the lipoxygenase-allene oxide synthase-allene oxide cyclase (LOX-AOS-AOC) pathway, resulting in the accumulation of bioactive jasmonates. These signals, activate downstream transcription factors including *OsMYC2*, *OsWRKY30*, *OsWRKY80* and *NAC* family proteins which collectively reprogram defence responses by enhancing antimicrobial metabolite production, stimulating phenylpropanoid metabolism, maintaining ROS homeostasis and reinforcing cell walls to limit pathogen invasion. These regulators orchestrate the expression of defence-related genes and the production of antimicrobial metabolites, lignification, ROS detoxification and stress adaptation in the downstream tissues (58).

By contrast, the salicylic acid (SA)-dependent pathway is only partly involved in sheath blight resistance and may interact with JA-dependent pathway through hormonal crosstalk pathways to dampen resistance. Early during infection, moderate induction of SA-responsive genes (e.g., pathogenesis-related protein 1 (PR1), pathogenesis-related protein 5 (PR5) and non-expressor of pathogenesis-related genes 1 (NPR1)) occurs. However excessive SA accumulation suppresses immune response to necrotrophs and increases susceptibility. Other hormone pathways also contribute to disease outcome. Auxin signalling generally supports resistance, with alterations in auxin homeostasis shown to increase susceptibility to *R. solani*. Brassinosteroid signalling, mediated in part by the transcription factor *RAVL1*, tends to suppress resistance, although *RAVL1* simultaneously activates ET signalling, reflecting cross-pathway antagonism. Abscisic acid (ABA) is not yet directly characterised in rice-*R. solani* interactions, is broadly known to antagonise SA- and JA-dependent defences in other rice pathosystems and likely contributes to disease outcome by modulating ROS homeostasis and mediating trade-offs between growth and defence under the concurrent abiotic stress that often accompanies infection. Recent transcriptomic analyses suggest that resistant rice cultivars have highly coordinated signalling networks characterised by prompt activation of MAPKs, JA/ET-responsive regulators, antioxidant systems and defence-associated transcription factors, while susceptible cultivars show delayed signalling responses that allow for extensive fungal colonisation. Multiple signalling components activated rapidly and in the correct sequence to restrict *R. solani* before it establishes necrotrophic growth, the overall resistance phenotype depends on the additive and synergistic contribution of many genes with small individual effects, rather than a single major gene; this explains why sheath blight resistance is typically mapped as multiple minor-effect QTLs rather than a discrete resistance locus and why resistant cultivars differ from susceptible ones more in the speed and coordination of their response than in possessing a unique defence pathway altogether (59).

Transcriptional factors orchestrating defence

The transcription factors (TFs) are the key regulatory elements that coordinate rice defence responses against *Rhizoctonia solani* through bringing together immune signalling pathways with downstream transcriptional reprogramming. After pathogen recognition and activation of the MAPK pathway, pathogen-induced activation of calcium, ROS and phytohormone-signalling pathways triggers the induction of multiple families of TFs that regulate pathogenicity-related, oxidative stress response-related, cell wall reinforcement-related and metabolic-adaptation-related genes. Of these, the members of the WRKY, NAC, myeloblastosis (MYB), basic leucine zipper (bZIP), AP2/ethylene-responsive factor (ERF) and myelocytomatosis (MYC) transcription factor families are important during sheath blight resistance. In particular, basic leucine zipper (bZIP) transcription factors contribute through at least two distinct mechanisms: *TGAP1*, a bZIP factor, is activated downstream of chitin elicitor perception and the MAP kinase kinase 4 (MMK4)-mitogen-activated protein kinase 3/6 (MPK3/MPK6) signalling cascade to induce biosynthesis of the phytoalexins momilactone and phytocassane, while *bZIP23* has been shown to physically interact with the NAM, ATAF1/2 and CUC2 (NAC) transcription factor *NAC028* to modulate resistance against *Rhizoctonia solani*, with *NAC028* itself regulating lignin biosynthesis via cinnamyl alcohol dehydrogenase 8B (*CAD8B*). In general, resistant rice cultivars tend to have higher and earlier expression of defence-associated TFs than do susceptible cultivars, which underscores the role of these TFs in regulating quantitative resistance to this necrotrophic pathogen. The WRKY transcription factor family is one of the most well studied rice immunity against *R. solani* regulators. *OsWRKY30*, *OsWRKY4* and *OsWRKY80* are highly induced after infection and act as positive regulators of jasmonic acid (JA) and ethylene (ET) defence pathways. Ectopic expression of *OsWRKY30* causes the accumulation of endogenous JA, increasing plant lignification, ROS-scavenging ability and defence-related gene expression, thereby boosting resistance against fungal pathogens (60). Likewise, *OsWRKY80* regulates signalling pathways that involve WRKY-dependent pathways, resulting in the induction of pathogenesis-related proteins and phenylpropanoid biosynthesis. Other NAC transcription factors such as *ONAC122* and *ONAC131* also control stress-related genes, such as those involved in cell wall fortification, ROS detoxification and accumulation of secondary metabolites during infection. Additional MYB and bZIP transcription factors are involved in the regulation of phenylpropanoid metabolism, antioxidant defence systems and hormone-responsive signalling pathways for sheath blight resistance (61).

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Ethylene-responsive factors (ERFs) modulate downstream defence activation by modulating pathogenesis-related (PR) protein activation, ROS homeostasis and stress responsive pathways. *Rhizoctonia solani* is a necrotrophic pathogen and over- or over prolonged activation of some TF-regulated pathways can stimulate senescence and necrosis that are beneficial to pathogen colonisation. Thus, resistance relies on temporal coordination and interaction between multiple TF networks and not just on activation of individual defence regulators. The recent transcriptomic and functional genomic analyses reveal that resistant cultivars of rice have very well-coordinated transcriptional networks that can quickly upregulate genes involved in defence response without causing metabolic imbalance and oxidative damage that paradoxically favour the necrotrophy by balancing rapid defence activation with tight negative regulation and return to homeostasis, resistant cultivars sustain protective signalling without tipping into the self-damaging, necrotrophy-favouring state characteristic of susceptible or over-responsive genotypes, making this balanced network architecture, rather than any single resistance gene, the basis of durable, broad-spectrum resistance to sheath blight. This coordination contributes to durable resistance because it allows the plant to mount an effective, timely response to *R. solani* while avoiding the excessive and prolonged activation of defence pathways that would otherwise trigger the senescence-associated cell death and oxidative damage The defence-regulating transcription factors are thus great candidates for molecular breeding, transgenic optimisation and genome engineering for durable sheath blight resistance in rice (62).

Epigenetic and post-transcriptional regulations

Rice's resistance to *Rhizoctonia solani*, beyond transcriptional regulation by the action of transcription factors, is also controlled by epigenetic and post-transcriptional regulatory mechanisms, that offer an extra layer of control and adaptability when challenged with sheath blight infection. The latter are dynamic mechanisms that regulate expression of defence-associated genes without changing the underlying DNA sequence and play a crucial role in coordinating rapid responses to immune activation, while minimising the metabolic costs of sustaining defence responses for long periods of time. The key epigenetic events associated with rice defence include DNA methylation, histone acetylation, histone methylation and chromatin remodelling which impact the transcriptional accessibility of immune-responsive loci. In the presence of *R. solani* infection, there is extensive chromatin level reprogramming in genes of jasmonic acid signalling, phenylpropanoid metabolism, ROS detoxification, phytoalexin biosynthesis and pathogenesis-related proteins. Under non-inflammatory conditions, histone deacetylases tend to suppress unwanted immune responses while histone acetyltransferases help to promote activation of those genes. Epigenetic plasticity is important to quantitative resistance to sheath blight, with resistant rice cultivars showing faster and more synchronised chromatin remodelling. In addition, epigenetic changes in response to stress can play a role in defence priming and immune memory, thereby increasing immunity to future challenges (63).

Post-transcriptional regulation further fine-tunes rice defence signalling by the activity of microRNAs (miRNAs), small interfering RNAs (siRNAs) and by RNA interference pathways, alternative splicing and by the stabilisation of mRNA. A few miRNAs, such as miR395a, have been functionally characterised in rice defence against *R. solani*; *miR395a* targets a pentatricopeptide repeat (PPR) protein-encoding transcript and its infection-induced upregulation leads to cleavage of this target, contributing to chloroplast-associated necrosis during sheath blight progression. These miRNAs have been identified to modulate the expression of transcription factor genes, including WRKY, NAC, MYB and bZIP, that are involved in signalling pathways related to jasmonic acid signalling, antioxidant response and secondary metabolite accumulation during fungal invasion. Small RNA-mediated regulation also regulates ROS homeostasis and the defence-growth balance, thereby avoiding excessive immune activation, which may lead to increased necrosis that benefits the necrotrophic lifestyle of *R. solani*. A few miRNAs are also implicated in sheath blight resistance for example Osa-miR1320-5p shows preferential expression during *R. solani* infection across multiple rice genotypes, while others such as *Osa-miR156d*, *Osa-miR159b*, *Osa-miR820c* and *Osa-miR1876* are differentially regulated between susceptible and resistant cultivars, with the majority of miRNAs showing downregulation during the early phase of infection (24–72 hr post-inoculation (hpi)) coinciding with the period of maximal host-pathogen molecular activity. Emerging evidence also points to cross-kingdom RNA interference (siRNA) during the rice-*R. solani* relationship, can involve RNA small molecules that suppress host immunity or fungal-derived small molecules that target virulence-associated genes. Therefore, the mechanism of host-induced gene silencing (HIGS) of some effectors, CWDEs and pathogenicity genes of *R. solani* have attracted huge interest as a powerful molecular approach to sheath blight control. Host delivered RNA interference (RNAi) targeting fungal genes conferred enhanced resistance in transgenic rice, a tobacco rattle virus-host-induced gene silencing (TRV-HIGS) screening system validated 29 of 33 candidate pathogenicity genes as contributing to necrosis and silencing of the effector gene *AGLIP1* reduced disease severity, while the most recent work has extended HIGS to core metabolic virulence targets, such as *RsEPSP* synthase in the shikimate pathway, showing that its silencing compromises fungal growth and virulence in stable transgenic rice lines. Despite this progress, most studies remain at the proof-of-concept or transient-expression stage and stable, field-validation HIGS rice lines with durable sheath blight resistance are still limited, indicating that translating these findings into deployable resistant cultivars remains an active area of

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ongoing research. Epigenetic and post-transcriptional regulatory mechanisms, together, closely interact with defence signalling and transcriptional networks to regulate rapid, coordinated and energy-efficient immune responses against *R. solani* in rice (64).

Genetic basis of host resistance

Quantitative trait loci and resistance loci

Resistance to rice sheath blight is genetically complex and predominantly quantitative with multiple quantitative trait loci (QTLs) exerting minor to moderate additive effect instead of single major genes for vertical resistance. The pathogen's broad host range with more than 200 host species, its necrotrophic lifestyle which avoids cell death-induced immunity and its high genetic and pathogenic variation within anastomosis groups (AGs), especially the AG1A, AG 1 IB and AG 1 IC that infect rice, are the reason for the absence of qualitative resistance. Sheath blight resistance is therefore polygenetic and includes genes involved in pathogen recognition via pattern-triggered immunity (PTI), cell wall reinforcement by lignification and suberisation, phytohormone signalling crosstalk, detoxification of reactive oxygen species (ROS) and defence-associated metabolic pathways such as phenylpropanoid and diterpenoid phytoalexin biosynthesis. Over the past two decades, more than 100 QTLs have been mapped across the 12 rice chromosomes using biparental populations, chromosome segment substitution lines (CSSLs), multi-parent advanced generation inter-cross populations and genome-wide association studies (GWAS) in a variety of *indica* and *japonica* germplasm collections with chromosomes 1,2,3,6,9 and 11 being most enriched with resistance associated loci (65). A number of important sheath blight associated QTLs have been consistently confirmed by different studies and genetic backgrounds, making them reliable candidates for marker-assisted selection (MAS). Among these, qSB-9tq and qSB-7TQ are consistently associated with reduced lesion length and delayed pathogen spread through enhanced lignification and ROS scavenging capacity. qSBR11-1 and qSB-11LE represent particularly significant loci enriched with defence- responsive gene clusters, including receptor like kinases, WRKY transcription factors and genes involved in phenylpropanoid and terpenoid secondary metabolism. Fine -mapping has narrowed these regions to intervals containing candidate genes linked to jasmonic acid signalling amplification and structural defence pathway activation (66). The qShB9-2 and qShB6-1 have been associated with enhanced tolerance through modulation of cell wall integrity and pathogen perception. Notably, chromosome 11 harbours multiple resistance-associated regions that co-localise with QTLs for plant height and tiller angle, reflecting the pleiotropic relationship between morphological disease escape mechanisms and true physiological resistance. This genetic correlation complicates QTL dissection and necessitates conditional QTL mapping to differentiate resistance from disease avoidance. Despite this complexity, pyramiding of validated QTLs such as qSB-9TQ and qSBR 11-1 through marker-assisted breeding has demonstrated synergistic effects, achieving resistance levels superior to single -QTL introgression lines and approaching practical field tolerance (67).

Recent advances in high-resolution genomics, transcriptomics and clustered regularly interspaced short palindromic repeats (CRISPR)-based functional validation have accelerated the identification and mechanistic characterisation of candidate resistance genes underlying sheath blight QTLs. The *OsWRKY30*, localised within the Qsb-9TQ region, functions as a positive regulator of JA and abscisic acid signalling, directly binding to W-box elements in promoters of defence genes to amplify transcriptional responses against *R. solani OsCERK1*, a coreceptor with OsCEBiP for chitin-triggered immunity, represents a PTI component whose allelic variants influence basal resistance; enhanced *OsCERK1* expression correlates with stronger MAPK activation and ROS burst upon chitin perception (68). *OsPAL1-3* within resistance QTLs catalyse the committed step in phenylpropanoid metabolism, driving lignin and flavonoid accumulation that reinforces cell wall barriers against fungal penetration. Comparative transcriptomics analyses between resistant and susceptible genotypes reveal that resistant lines exhibit stronger and faster induction of genes encoding receptor-like kinases, peroxidases, laccases and diterpenoid phytoalexin biosynthesis enzymes. However, the individual QTLs typically confer only 10–30 % phenotypic variance, are strongly influenced by environmental conditions and show QTL and environment interactions that complicate predictive breeding. Future strategies for durable resistance must therefore integrate QTL pyramiding with genomic selection (GS), clustered regularly interspaced short palindromic repeats-associated protein 9 (CRISPR-Cas9) editing of susceptibility factors and functional characterisation of resistance associated loci through map-based cloning and transgenic validation approaches that collectively promise to overcome the limitations of single QTL resistance and address the persistent challenge of *R. solani* genetic plasticity in rice production systems (69). In addition to the functionally characterised loci discussed above, several other QTLs have been mapped across the rice genome and associated with quantitative resistance to sheath blight, differing in their chromosomal location and reported mechanism of action, ranging from reduced lesion height and limited pathogen spread to stable resistance across field environments (Table 2). Despite this genetic diversity, as noted earlier, only a small subset of these QTLs (e.g., *qSBR11-1*) has been fine-mapped to the level of a functionally validated candidate gene, underscoring the gap between QTL discovery and practical breeding application. Major quantitative trait loci (QTLs) associated with rice sheath blight resistance were identified through linkage mapping, genome-wide association studies (GWAS) and QTL validation studies is provided in Table 2.

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Table 2. Major quantitative trait loci (QTLs) associated with rice sheath blight resistance were identified through linkage mapping, genome-wide association studies (GWAS) and QTL validation studies

QTL	Chromosomal location	Reported role in resistance	References
qSB-9TQ	Chr. 9	Major QTL associated with reduced lesion height and enhanced partial resistance to sheath blight	(71)
qSB-7TQ	Chr. 7	Contributes to quantitative resistance by limiting pathogen spread and disease severity	(72)
qSB-11LE	Chr. 11	Associated with stable field resistance and reduced sheath colonisation	(73)
qSBR11-1	Chr. 11	Confers stable resistance across environments; linked with defence-related responses	(74)
qSB-1	Chr. 1	Associated with suppression of lesion expansion under field conditions	(75)
qSB-12	Chr. 12	Enhances sheath blight resistance in combination with other QTLs	(76)
qSBR7-1	Chr. 7	Involved in partial resistance and delayed lesion development	(77)

*QTL: quantitative trait locus; Chr.: chromosome. Chromosomal positions are based on published mapping studies. Individual QTL effects may vary among genetic backgrounds and environments.

Although numerous QTLs associated with sheath blight resistance have been reported, very few loci with strong and practical breeding potential have been functionally characterised. This gap largely reflects the inherently quantitative and polygenic nature of sheath blight resistance. Individual QTLs typically confer only small, minor-effect contributions to resistance, are often detected inconsistently across different genetic backgrounds, environments and rice growth stages and are frequently linked to undesirable agronomic traits through linkage drag, complicating their direct use in breeding. Furthermore, most QTLs have been mapped only to broad chromosomal intervals without fine-mapping or candidate gene identification, so the causal gene and its underlying molecular mechanism remain unknown for the majority of reported loci, limiting their translation into marker-associated selection or gene editing targets for practical cultivar development. Recently, the identification of *SBRR1-R* (sheath blight resistance receptor-like kinase 1) allele represented a major breakthrough in sheath blight resistance research. It is a novel resistance associated locus discovered through genome wide association studies (GWAS) and natural germplasm analysis. *SBRR1* encodes a G-type lectin receptor-like protein kinase involved in rice defence against *R. solani*. The resistant allele, designated as *SBRR1-R*, was predominantly identified in indica rice germplasm and was associated with enhanced resistance to sheath blight due to the presence of 256-base pairs (bp) promoter insertion that increases pathogen-responsive gene expression. Functional characterisation revealed that *SBRR1-R* confers significantly stronger resistance compared to the susceptible allele (*SBRR1-S*). The resistant allele was introgressed into susceptible japonica cultivars through marker-assisted selection, resulting in near isogenic lines exhibiting improved sheath blight resistance without noticeable yield penalty. Under severe disease pressure, these introgressed lines showed reduced yield loss, demonstrating the practical breeding value of the locus. The study further established a defence signalling module involving "bHLH57-*SBRR1-R-SIP1-Chit3/4*", in which the transcription factor basic helix-loop-helix (bHLH) 57 activates *SBRR1-R* expression, leading to downstream induction of chitinase genes associated with antifungal defence responses. The discovery of *SBRR1-R* represents an important advance in sheath blight resistance research because it provides a naturally occurring resistance-associated locus with potential utility for resistance breeding. Under severe disease pressure, these lines showed reduced yield loss, demonstrating the practical breeding value of the locus. Since indica rice varieties are extensively cultivated in India, the presence of *SBRR1-R* in indica germplasm highlights its potential relevance for indica rice improvement programs targeting sheath blight resistance. Furthermore, the availability of a co-segregating 256 bp insertion marker provides opportunities for efficient marker-assisted breeding and pyramiding with other resistance QTLs to enhance durable resistance against sheath blight (70). A recently characterised example illustrating receptor-kinase-mediated resistance is the regulation of *SBRR1* expression. The transcription factor bHLH57 differentially activates two *SBRR1* transcript variants, *SBRR1-S* and *SBRR1-R*, which in turn regulate the expression of chitinase genes *Chit3* and *Chit4*. Activation via the *SBRR1-R* variant leads to high chitinase activity and enhanced resistance to *R. solani*, whereas activation via *SBRR1-S* results in low chitinase activity and increased susceptibility, demonstrating how alternative transcriptional regulation of a single receptor-like kinase locus can determine the resistance outcome. Proposed regulatory model of the *SBRR1-R* resistance allele during rice defence against *Rhizoctonia solani* is illustrated in Fig. 5.

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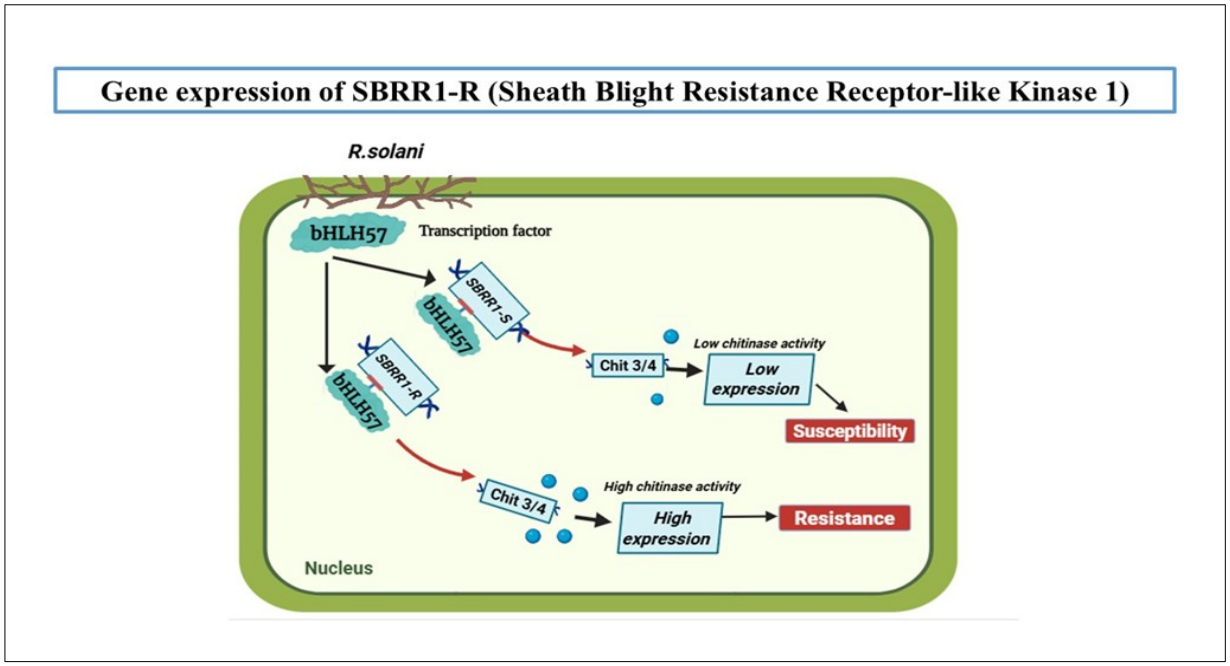


Fig. 5. Proposed regulatory model of the SBRR1-R resistance allele during rice defence against *Rhizoctonia solani*. *The resistant allele activates downstream signalling through the bHLH57-SBRR1-R-SIP1-Chit3/4 module, resulting in enhanced chitinase expression and improved sheath blight resistance.

Susceptibility genes

Besides resistance-associated loci, susceptibility (S) genes are also important in the rice-*Rhizoctonia solani* interaction as they enable pathogen colonisation, host immunity suppression, or the establishment of a favourable physiological environment for disease development. Susceptibility genes are host genes that the pathogen exploits or manipulates to make it easier to infect and cause the disease. *Rhizoctonia solani* is a necrotrophic fungus and resistance is frequently linked to an over activation of senescence pathways, hormonally unbalanced, oxidative stress and deregulation of defences. Thus, the identification of S genes has become an important thrust in their research to understand sheath blight susceptibility and the development of durable resistance strategies via gene editing and molecular breeding approaches (78). Multiple rice genes linked to hormone signalling, transcription regulation and defence suppression are suggested to be involved in sheath blight susceptibility. Some members of the *WRKY* transcription factor family are negative regulators of immunity; *OsWRKY62* was shown to inhibit the expression of defence-related genes and reduce resistance when over-expressed and related regulators such as *OsWRKY53* and *OsMAPK5* similarly modulate JA/SA crosstalk or dampen defence-associated signalling to suppress resistance. Differently, deregulation of ethylene-induced senescence genes can also contribute to the increase in tissue necrosis beneficial for fungus proliferation. Other genes might be involved in susceptibility too, promoting lesion expansion at later stages of infection, because of excessive ROS accumulation, chlorophyll degradation and programmed cell death; receptor-like proteins such as *OsRMC* have been implicated in this ROS-linked susceptibility mechanism. Furthermore, changes in genes involved in regulating cell wall architecture and carbohydrate metabolism, including the sugar transporters *OsSWEET11* and *OsSWEET14* and canopy architecture genes such as *DEP1*, can make cells more susceptible to fungal CWDE activity and nutrient exploitation (79, 80).

Recent transcriptomic and functional genomic analyses have identified several candidate S genes that are differentially expressed in susceptible cultivars of rice after *R. solani* infection have been identified. These involve genes related to negative regulation of immunity, suppression of ROS detoxification, hormonal imbalance and metabolic reprogramming for pathogen growth. Several sugar transporter genes and nutrient remobilisation pathways might also play a role in fungal colonisation by enhancing carbon availability in infected tissues. Thus, susceptibility gene manipulation has become a new concept and has attracted much interest in the management of rice diseases. Knockout or modification of the sheath blight resistance genes (S) with editing technologies like CRISPR/Cas seems to provide potential opportunities for enhancing sheath blight resistance while maintaining high yields. Many susceptibility-associated genes, however, are also involved in normal plant growth and development and hence, detailed functional characterisation is crucial to prevent unwanted pleiotropic effects. Thus, quantitative resistance breeding would be a viable approach to gain durable resistance against *R. solani* when combined with S gene editing (81). Major candidate susceptibility genes associated with rice sheath blight and their proposed roles in disease development is provided in Table 3.

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Table 3. Major candidate susceptibility genes associated with rice sheath blight and their proposed roles in disease development

Gene	Full Name / Family	Role	Mechanism / Function	Reference
OsSWEET11	Sugar transporter	Promotes susceptibility	Facilitates sugar efflux that may support pathogen nutrition	(82)
OsSWEET14	Sugar transporter	Putative S gene	Alters carbohydrate allocation during infection	(83)
OsWRKY53	WRKY transcription factor	Negative regulator of resistance in some contexts	Modulates JA/SA crosstalk and suppresses defence balance	(84)
OsWRKY62	WRKY TF	Susceptibility-associated	Suppresses defence gene activation	(85)
OsMAPK5	Mitogen-activated protein kinase	Can negatively regulate resistance	Dampens defence-associated signalling pathways	(86)
DEP1	Dense and erect panicle 1	Associated with sheath blight susceptibility	Dense canopy architecture favours disease progression	(87)
OsRMC	Receptor-like membrane protein	Susceptibility-associated	Alters ROS and defence signalling	(88)

*S genes denote host genes whose activity facilitates pathogen colonisation or suppresses defence responses. Several genes listed remain candidate susceptibility factors requiring further functional validation.

Transcriptomics and proteomics of resistant and susceptible genotypes

Transcriptomic and proteomic approaches have greatly contributed to the understanding of molecular mechanisms involved in differential resistance responses of rice genotypes against *Rhizoctonia solani*. The comparison of resistant and susceptible cultivars shows that many genes and proteins involved in pathogen perception, defence signalling, regulation of oxidative stress, secondary metabolism and cell wall strengthening are reprogrammed during infection with sheath blight. Resistant genotypes typically show earlier, more robust and more integrated responses to immune activation while susceptible cultivars exhibit a delayed or dysregulated immune response, allowing for rapid fungal colonisation. The complexity of quantitative resistance mechanisms can be demonstrated by the thousands of differentially expressed genes (DEGs) that have been identified after infection with *R. solani* by high throughput RNA sequencing (RNA-seq) analyses (89).

Several studies using transcriptomic analysis have shown that resistant rice cultivars are consistently able to induce genes known to play roles in pattern triggered immunity (PTI), jasmonic acid (JA) and ethylene (ET) signalling, MAPK cascades, ROS detoxification and phytoalexin biosynthesis. Receptor-like kinases (RLK), defence associated transcription factors (*OsWRKY30*, *OsWRKY80*, *NAC* protein) and antioxidant enzymes (catalases, peroxidases and glutathione-S-transferases) are highly upregulated in resistant interactions. Resistant genotypes also exhibit higher expression of genes involved in lignin biosynthesis, phenylpropanoid metabolism, callose deposition and pathogenesis-related proteins, leading to better structural and biochemical defence barriers. In contrast, susceptible cultivars often show higher levels of senescence-associated genes, susceptibility-related pathways and metabolic dysfunctions upon stress that promote the necrotrophic colonisation of *R. solani*. The transcriptomic profile of *R. solani* AG1-IA during sclerotia development gave valuable insights into the molecular mechanism involved in sclerotia formation and development. The study compared the changes in gene expressions between three developmental stages (mycelial stage, sclerotia initiation, sclerotia maturation) using RNA-Seq analysis. The number of clean reads assembled to 15100 unigenes > 300 million, with 12575 genes showing differential expression, suggesting a high level of transcriptional reprogramming during sclerotial morphogenesis. Genes related to melanin biosynthesis such as polyketide synthase (*PKS*), laccase-1 (*LAC1*), tyrosinase (*TYR*) and polyketide cyclase (*PKC*) showed dynamic expression patterns and this confirmed the importance of melanisation during sclerotial maturation and stress adaptations (90). The complementing protein dynamics of proteins and changes in post-translational modification and stress-responsive metabolism during infection are also revealed by proteomic analyses, which complement transcriptomic findings. A number of protein differences have been observed between resistant and susceptible rice varieties in terms of differential accumulation including proteins related to photosynthesis, carbohydrate metabolism, ROS scavenging, defence signalling and protein folding. Resistant cultivars tend to have high levels of antioxidant enzymes, chaperone proteins, PR proteins and enzymes concerned with secondary metabolite biosynthesis under pathogen stress, while susceptible cultivars experience severe disruption of photosynthetic proteins and energy metabolism. Integrative transcriptomic-proteomic approaches have also resulted in the identification of a number of candidate biomarkers and defence-associated pathways associated with quantitative sheath blight resistance. All these omics studies provide important insights into the molecular architecture of rice resistance to *R. solani* and can serve as important resources for functional genomics, molecular breeding

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and development of durable resistance strategy by genomic assisted selection and gene editing approaches (91).

Induced resistance and priming

Chemical elicitors of defence

Chemical elicitors of defence can be useful for increasing the resistance of rice to *Rhizoctonia solani* by inducing resistance before pathogen attack. They work by mimicking pathogen-induced signals or by triggering a plant's own immune response pathways, putting the plant on guard for quicker and stronger immune reactions when it's attacked. Defence elicitors do not directly inhibit pathogen growth, but rather activate signalling networks related to jasmonic acid (JA), salicylic acid (SA), ethylene (ET), reactive oxygen species (ROS) and phenylpropanoid metabolism, which enhances host defence capacity. Elicitor-induced resistance has gained significant attention in rice sheath blight management as an environmentally sustainable strategy and feasibility with the integrated disease management practices (91). Elicitors reported in rice fall broadly into two mechanistic groups. The first acts primarily by driving transcription of pathogenesis-related (PR) proteins and phytoalexin biosynthetic genes. Methyl jasmonate and benzothiadiazole (BTH) fall in this group, upregulating PR proteins alongside antioxidant enzymes and phenylalanine ammonia-lyase (PAL), which restricts fungal colonisation. The second group acts more through structural reinforcement and priming of downstream defence rather than constitutive activation. Chitosan triggers pattern-triggered immunity (PTI), a ROS burst, callose deposition and lignification; silicon supplementation deposits silica beneath the epidermis while also activating antioxidant and phenylpropanoid pathways; and β-aminobutyric acid (BABA) primes defence signalling to confer broader-spectrum resistance (92). Mechanistically, both groups converge on transcriptional reprogramming of defence genes MAPK signalling components, WRKY transcription factors, ROS detoxification machinery, antimicrobial compound biosynthesis and cell wall reinforcement. Critically, most elicitors work through priming rather than constitutive activation, which keeps metabolic costs low in the absence of a pathogen challenge. However, elicitor efficacy depends heavily on concentration, timing of application relative to infection pressure, rice genotype and environmental conditions and elicitors alone are generally insufficient under high disease pressure. Their practical value therefore lies in combination with resistant varieties, biocontrol agents and precision agronomy rather than as a standalone control measure (93). Given the quantitative, polygenic nature of sheath blight resistance discussed above, no single intervention is likely to provide complete or durable protection against *R. solani*. Instead, an integrated disease management strategy combining genetic, chemical and biological approaches is increasingly recognised as the most viable path toward sustainable sheath blight control. Genetic strategies such as QTL pyramiding and genomic selection aim to combine multiple minor-effect resistance loci to achieve additive protection, while CRISPR/Cas based editing of susceptibility genes offers a complementary route to reducing host vulnerability without compromising yield. In parallel, chemical priming and induced systemic resistance strategies, using the elicitors discussed earlier, can enhance the speed and magnitude of host defence responses, while bicontrol agents contribute an additional layer of protection by directly anatagonising the pathogen in the rhizosphere and phyllosphere.

Integrated disease management strategies for rice sheath blight combining host resistance, defence priming, biological control agents, induced systemic resistance, chemical elicitors and genomics-assisted breeding approaches for sustainable disease management are illustrated in Fig. 6.

Biocontrol agents and plant growth-promoting rhizobacteria-mediated induce systemic resistance

Biological control agents and plant growth-promoting rhizobacteria (PGPR) are being used more and more to induce systemic resistance (ISR) against *R. solani*, in the sustainable management of rice sheath blight. Direct chemical inhibition, as opposed to ISR, results from microorganisms that directly inhibit pathogen growth, whereas ISR stimulates the plant's natural immune system, making it more resistant to subsequent pathogen attacks. *Pseudomonas, Bacillus, Trichoderma* and *Streptomyces* species, with their ability to provide mycoparasitism, antibiosis, competition, hydrolytic enzyme production and activation of host defence mechanisms, have exhibited a high level of antagonistic activity against *R. solani*. These beneficial microorganisms suppress pathogen growth and improve the stress tolerance of the rice plant, while also boosting its vigour under field conditions (94).

The ISR effect mediated by PGPR is mainly controlled by jasmonic acid (JA) and ethylene (ET) signal transduction pathways, which are not involved with salicylic acid-mediated systemic acquired resistance (SAR), thus providing a way to prevent necrotrophs like *R. solani*. Application of PGPR to rice roots causes low-level activation of defence signalling pathways and thus causes a ‘priming' of immune responses at minimal cost to the host. Primed plants have enhanced and accelerated responses to pathogen challenge: ROS production, pathogenesis-related proteins, phenylpropanoid metabolism and callose deposition, as well as antioxidant defence systems. The activities of the enzymes like phenylalanine ammonia-lyase (PAL, peroxidase (POX), polyphenol oxidase (PPO), chitinases and β-1,3-glucanases in rice challenged with *R. solani* were found to be enhanced by the application of beneficial bacteria like *Pseudomonas fluorescens* and *Bacillus subtilis*. Similarly, *Trichoderma* spp. activates defence-responsive transcription factors, biosynthesis of phytoalexins and lignification as well as they produce antifungal metabolites and CWDEs that target the pathogen (95).

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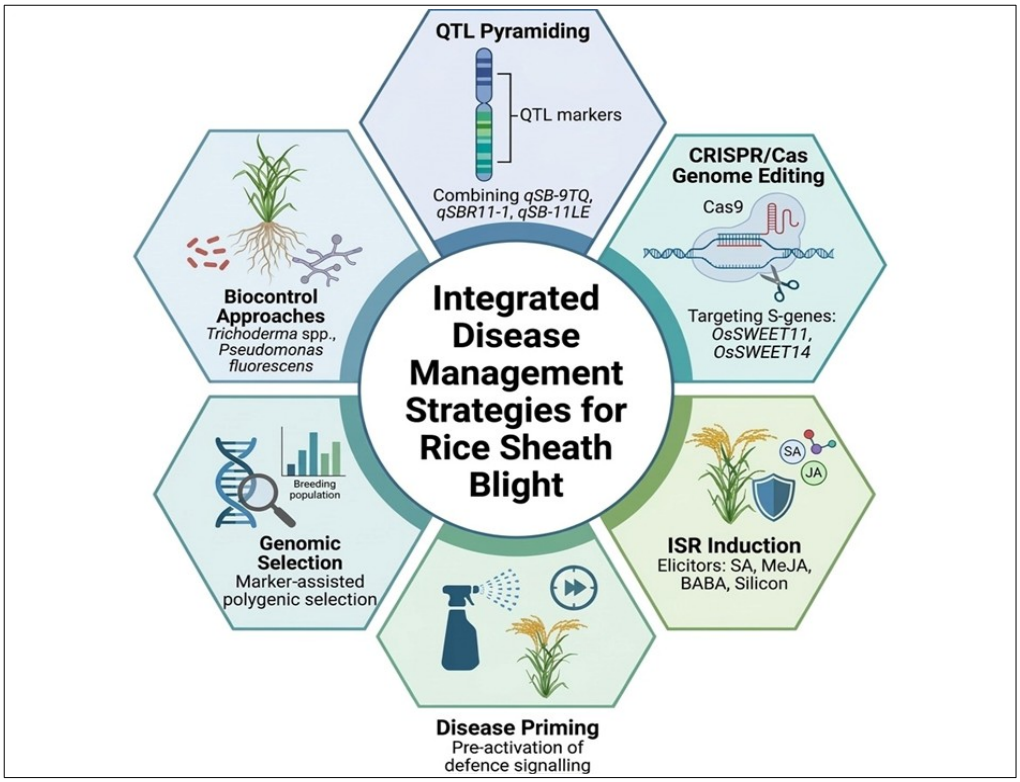


Fig. 6. Integrated disease management strategies for rice sheath blight combining host resistance, defence priming, biological control agents, induced systemic resistance, chemical elicitors and genomics-assisted breeding approaches for sustainable disease management.

Induced systemic resistance is characterised at the molecular level by extensive transcriptional and metabolic reprogramming of the cells, as well as increased readiness for immune functions. During infection, PGPR and fungal biocontrol agents regulate MAPK signalling pathways, WRKY transcription factors, ROS homeostasis and phytohormone crosstalk to enhance the efficiency of defence activation. Some beneficial microbes also secrete molecules such as volatile organic compounds (VOCs), siderophores, lipopeptides and quorum-sensing molecules that impact pathogen suppression and plant immunity. Recent research indicates that ISR may also play a role in long-term adaptation and defence priming, which could include epigenetic changes linked to immune memory. Field performance of biocontrol-mediated ISR is, however, affected by the nature of the environment, the composition of the soil microbiome, the genotype of the rice plant and the efficiency of microbial colonisation. Incorporating PGPR-based ISR with resistant cultivars, defence elicitors and precision agronomic practices is thus recommended as a solution for sustainable and environmentally friendly management of rice sheath blight. However, field -level deployment of PGPR-mediated ISR faces several practical limitations: efficacy is often inconsistent and highly dependent on strain-environment interactions, with variable soil type, temperature, moisture and native microbial competition affecting rhizobacterial colonisation and persistence; formulation and shelf life constraits can reduce inoculant viability during storage and transport and greenhouse-validated strains frequently underperform under field conditions compared with controlled settings, meaning results are not always reproducible across locations and seasons. As a result, PGPR-based approaches are generally most effective when integrated with, rather than substituted for, other management strategies such as resistant cultivars and reduced-rate fungicide application (96).

Priming memory and epigenetic basis

Priming defence is an adaptive immune reaction in which rice plants have been previously exposed to biotic or abiotic stimuli, allowing them to react more quickly and strongly to a second exposure to *R. solani*. In contrast to the activation of defence pathways, priming does not involve high metabolic costs, but keeps the plant in a state of physiological alertness until a pathogen challenges the plant. In the case of primed rice plants, the production of reactive oxygen species (ROS), pathogenesis-related proteins, phytoalexin biosynthesis, cell wall fortification and production of jasmonic acid (JA) and ethylene (ET) were accelerated upon infection. Inducing defence priming against sheath blight is known with the use of chemical elicitors like β-aminobutyric acid (BABA), analogues of salicylic acid, silicon and beneficial microorganisms like *Pseudomonas* spp*., Bacillus* spp*. and Trichoderma* spp. The primed rice genotypes have been observed to have higher level of the activation of defence genes in their transcription, less lesion expansion and greater tolerance to necrotrophic colonisation by *R. solani* when compared with unprimed plants (97, 98).

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The epigenetic modifications contribute to defence against *R. solani* in rice, through the regulation of expression of immunity-related genes associated with ROS homeostasis, hormone signalling and secondary metabolism. To promote defence responses quickly during sheath blight infection, a dynamic process of chromatin modifications involving changes in histone acetylation, histone methylation, DNA methylation and chromatin remodelling are essential. These changes affect the expression of genes involved in antioxidant enzymes, phenylpropanoid biosynthesis, pathogenesis related (PR) proteins and jasmonic acid (JA) and ethylene (ET)-mediated signalling pathways that play key roles in resistance to the necrotrophic pathogen *R. solani*. It has been reported that the application of priming agents like salicylic acid, β-aminobutyric acid (BABA), silicon and beneficial rhizobacteria creates a transcriptionally permissive chromatin state, resulting in faster and stronger activation of defence genes when challenged with pathogenic microbes. Susceptible cultivars, on the other hand, might show less epigenetic reprogramming, so that defence may be activated later, causing more disease development. Stress-induced chromatin modifications lead to defence memory in rice plants, which helps them to respond better to repeated attacks by *R. solani* and may enable sustained or transgenerational improvement of sheath blight resistance. Priming-associated defence memory also occurs during the interaction of rice and *R. solani* and is further influenced by post-transcriptional regulatory mechanisms. Primed immune responses involve the regulation of expression of transcription factors, phytohormone signalling components and antioxidant pathways by small RNAs, particularly microRNAs (miRNAs) and small interfering RNAs (siRNAs). In addition, priming increases the efficiency of MAPK signalling, the transcriptional regulation of WRKY and accumulation of secondary metabolites in response to pathogen challenge. Immune memory, however, must be regulated tightly, for long-term immunity can have adverse effects on plant growth and productivity. Hence, elucidation of the molecular and epigenetic mechanisms of priming becomes more significant for the development of sustainable management strategies to control sheath blight. The use of defence elicitors, beneficial microbes and epigenetic engineering can be effective ways to exploit memory and improve resistance of rice against *R. solani* in a lasting and environment-friendly manner. Most of the evidence supporting priming-based defence memory in the rice–*R. solani* system originates from controlled greenhouse or growth-chamber studies, where elicitor doses and challenge timing are precisely managed. However, the extent to which primed states persist and provide effective protection under the variable conditions of the field remains less clear. Priming responses differ considerably among rice genotypes and can be inconsistent across seasons and locations, complicating the extrapolation from single-cultivar research. Because priming lowers the activation threshold for defence, overly primed or long-lasting immune states may divert resources from growth and yield. The duration of primed states following a single elicitor or microbial treatment and whether ongoing reinforcement is necessary, remain poorly understood for *R. solani*. Similarly, definitive evidence distinguishing true transgenerational (epigenetically inherited) priming from within-generation memory is lacking. Many studies infer chromatin-level priming from bulk transcriptomic or enzyme activity data rather than direct epigenomic profiling techniques such as chromatin immunoprecipitation sequencing (ChIP-seq) or bisulfite sequencing. As a result, causal links between specific chromatin modifications and resistance are mostly correlational. Addressing these uncertainties through multi-environment field trials, advanced epigenomic analyses and detailed growth-defence trade-off assessments is crucial for establishing priming as a reliable, deployable strategy rather than a promising but inconsistently expressed phenomenon (99).

Challenges, limitations and economic considerations

Despite their mechanistic promise, induced resistance strategies face several practical constraints that limit large-scale adoption in rice sheath blight management. Synthetic elicitors such as BTH and purified BABA are typically more expensive per hectare than conventional fungicides, which can make them economically unattractive for smallholder rice systems where sheath blight is often most severe. Biocontrol-based ISR approaches (e.g., *Trichoderma* or *Bacillus* formulations) are generally cheaper to produce but still require investment in mass production, quality control and distribution infrastructure. Live microbial biocontrol agents are inherently less stable than synthetic chemistries; viability during storage and transport, especially under the heat and humidity typical of many rice-growing regions, remains a major bottleneck. Elicitor compounds such as chitosan and silicon-based products also face formulation challenges around solubility, uniform application and compatibility with existing spray or seed-treatment equipment. Environmental variables (temperature, humidity, soil type, native microbiome composition) and the timing of application relative to disease pressure strongly modulate the efficacy of both chemical elicitors and PGPR-mediated ISR. This translates into inconsistent field performance compared with the more predictable efficacy reported in controlled trials, which is a recurring concern for extension recommendations. Unlike a single curative fungicide spray, induced resistance strategies generally require correct timing before infection onset, which demands more precise disease forecasting and farmer training than reactive treatments. This raises the practical barrier to adoption, particularly where extension support and diagnostic infrastructure are limited. Biocontrol agents and novel elicitor formulations often face slower or less standardised regulatory approval pathways than conventional agrochemicals in many rice-producing countries, which can delay commercialisation even when efficacy data are favourable. Taken together, these constraints suggest that induced resistance and priming strategies are best

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positioned as components of integrated disease management, combined with resistant cultivars and, where necessary, targeted fungicide use, rather than as standalone replacements; future research should also report cost-per-hectare and field-consistency data alongside efficacy to support adoption decisions (100).

Knowledge gap and future perspectives

Despite substantial advances in understanding the rice-*R. solani* interaction, three critical gaps continue to limit durable resistance development. First, the *R. solani* effector repertoire remains largely uncharacterised. Unlike biotrophs such as *Magnaporthe oryzae*, few effectors have been functionally validated and how the pathogen manipulates hormonal crosstalk, ROS homeostasis and programmed cell death remains poorly resolved a challenge compounded by the fungus's broad genetic diversity and multinucleate nature, which complicate identification of conserved virulence targets. Second, resistance itself is quantitative and environmentally unstable: most known QTLs confer only partial, environment-dependent resistance, frequently overlap with architectural traits (plant height, heading date) that confound true resistance with disease escape and no major resistance (R) gene has yet been identified. Third, emerging areas such as epigenetic regulation, cross-kingdom RNA interference and microbiome-mediated immunity remain underexplored and biocontrol/ISR strategies that show promise experimentally lack field-level consistency.

Addressing these gaps requires five research priorities, namely genome-wide functional validation of candidate *R. solani* effectors to build a validated effector catalogue; discovery of major-effect resistance and susceptibility genes via pan-genomic resequencing, GWAS and mutant screens, prioritising loci separable from plant architecture; multi-environment, multi-isolate QTL fine-mapping to generate stable, pleiotropy-free markers for breeding; CRISPR/Cas-based editing of susceptibility genes and negative defence regulators, with field-level testing of durability and yield trade-offs and systems-level modelling that integrates multi-omics data to predict disease outcomes under variable climates.

Realising these priorities will require coordinated, multi-institutional consortia (analogous to international wheat rust or blast genomics initiatives) to pool isolates, germplasm and omics data; funding calls prioritising effector and susceptibility-gene discovery; sustained support for long-term, multi-location field networks to capture genotype-by-environment effects; open-access, artificial intelligence/machine learning (AI/ML)-ready phenotyping datasets for model benchmarking; and translational grants bridging gene discovery with precision breeding and gene-editing pipelines, including regulatory pathways for CRISPR-edited lines. Collectively, this agenda, anchored in effector and susceptibility-gene discovery, systems-level modelling and coordinated funding, offers a path toward sustainable, durable sheath blight resistance.

Artificial intelligence and machine learning in resistance breeding

Artificial intelligence (AI) and machine learning (ML) approaches offer considerable, largely untapped potential to accelerate sheath blight resistance breeding. Specific applications include: (i) ML-based genomic selection models trained on multi-environment phenotypic and genotypic datasets to improve prediction accuracy for a quantitative, environmentally sensitive trait such as sheath blight resistance; (ii) deep learning-based image analysis and high-throughput phenomics (e.g., convolutional neural networks applied to drone or field-based red-green-blue (RGB)/hyperspectral imagery) for rapid, non-destructive and scalable disease-severity scoring, thereby replacing labour-intensive visual rating scales; (iii) natural language processing and knowledge-graph approaches to mine existing multi-omics and literature datasets for effector-target and gene-gene interaction predictions, thereby prioritising candidates for functional validation; and (iv) AI-assisted design of CRISPR guide RNAs and prediction of off-target effects to improve the efficiency of susceptibility-gene editing. By coupling these AI/ML pipelines with the systems biology framework described above, breeders can substantially shorten the breeding cycle by narrowing candidate gene lists and prioritising field trials most likely to succeed.

4. Conclusion

The interaction between *Rhizoctonia solani* and rice is governed by a dynamic interplay between pathogen virulence and host immunity, in which disease outcome is ultimately determined by the balance between coordinated pathogen attack through cell wall-degrading enzymes, necrosis-inducing effectors, toxins and signalling-associated virulence factors and the multilayered defence response of rice, spanning pattern-triggered immunity, phytohormone signalling, antioxidant and cell wall reinforcement systems, antimicrobial proteins and transcriptional reprogramming, further shaped by epigenetic regulation, post-transcriptional control, defence priming and microbiome-associated immunity. The central insight emerging from this review is that sheath blight resistance is not governed by any single mechanism but is an integrated, systems-level property arising from the interdependence of pathogen virulence, host immune signalling and resistance genetics; recognising this integration is essential for moving breeding and engineering strategies beyond single-gene or single-pathway approaches toward a more holistic framework for durable resistance. Despite considerable progress in genomics, transcriptomics, proteomics and functional biology, major challenges continue to constrain the development of durable resistance. Sheath blight resistance remains largely quantitative, polygenic and strongly influenced by environmental conditions and the molecular functions of most predicted *R. solani* effectors and host susceptibility factors remain uncharacterised. Bridging this gap will require a shift from descriptive, single-omics studies

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toward integrative, systems-level analyses that combine multi-omics data, functional genomics, epigenetics and host-microbiome interactions to resolve the regulatory networks underlying resistance and susceptibility.

Looking ahead, the convergence of genome editing, RNA-based technologies, genomics-assisted breeding and defence-priming strategies offers a realistic path toward engineering resistance that is both durable and sustainable. Realising this potential will depend on translating mechanistic insight into precision interventions that can withstand pathogen variability and shifting climatic pressures. As these technologies mature and converge with a systems-level understanding of host–pathogen interaction, the long-term goal of durable, broad-spectrum resistance to rice sheath blight, once considered elusive, is becoming an increasingly attainable target for safeguarding global rice productivity.

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Authors' contributions

VM conducted the literature survey, synthesised the information and prepared the original manuscript. SJ provided the core concepts and intellectual guidance for the review. TM, JHS and KPA contributed to manuscript refinement and content development. RN, KC and JV critically reviewed the manuscript and contributed to improve its clarity. SMS performed the final revision. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest

Authors do not have any conflict of interests to declare.

Ethical issues

None

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used Grammarly for language editing and proofreading purposes. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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