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Review

Unlocking Durable Sheath Blight Resistance in Rice: The Central Role of GWAS in Genetic Dissection and Trait Enhancement

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ABSTRACT

Sheath blight (ShB), caused by *Rhizoctonia solani* AG1-IA, is a globally devastating rice disease resulting in 20–50% yield losses. Its management is challenged by the pathogen's genomic complexity, quantitative host resistance, and trade-offs with agronomic traits. We first discuss the critical foundation of reliable phenotyping, spanning standardized field assays to emerging high-throughput phenomics, which is essential for accurate genetic dissection. This review also synthesizes advances in genetic and molecular dissection of ShB resistance, emphasizing the transformative role of Genome-Wide Association Studies (GWAS). We highlight how GWAS has overcome limitations of traditional QTL mapping, enabling high-resolution discovery of major resistance loci (e.g., *SBRR1*), defense pathways (e.g., ROS-antioxidant systems), and novel candidate genes across diverse germplasm. Integration of GWAS with gene editing (e.g., *dep1*-cys), overexpression strategies (e.g., *OsCSP41b*), and transcription factor networks (e.g., *OsbHLH34, OsBZR1*) reveals pathways to break yield-resistance trade-offs. Future progress hinges on multi-omics-augmented GWAS, epistasis modeling, and deploying genomic selection for durable resistance. This work underscores GWAS as a cornerstone for accelerating ShB-resistant rice breeding.

Keywords: *Rhizoctonia solani*, Quantitative Trait Loci (QTLs), Genomic Selection, Haplotype Breeding, Transcription Factors, Disease Resistance, Signaling pathway

INTRODUCTION

Rice (*Oryza sativa* L.) stands as a cornerstone of global food security, feeding over half of the world's population. Yet, its productivity is perpetually threatened by a multitude of pathogens, among which sheath blight (ShB), caused by the necrotrophic fungus *Rhizoctonia solani* Kühn AG1-IA, ranks as the second most devastating disease after blast. This soil-borne pathogen inflicts staggering yield losses, routinely estimated at 20-50% under conducive conditions, posing a severe threat to rice production systems worldwide (Feng *et al.*, 2025; Thesiya *et al.*, 2025).

Alarmingly, the incidence and severity of ShB are intensifying, driven largely by modern agricultural practices. The widespread adoption of semi-dwarf, high-yielding varieties, coupled with high nitrogen fertilization and increased planting densities, creates dense canopies and humid microclimates that are highly favorable for *R. solani* infection and spread (Molla *et al.*, 2020).

Developing durable genetic resistance to ShB remains an elusive goal and a formidable challenge for rice breeders and pathologists, starkly contrasting the significant progress made against diseases like blast and bacterial blight. This difficulty stems from a confluence of factors inherent to both the pathogen and the host. *R. solani* AG1-IA exhibits high genetic variability (classified into 14 anastomosis groups), possesses an exceptionally broad host

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range (infecting plants from ~32 families), and persists robustly between seasons through long-lived sclerotia in soil and crop debris, complicating management strategies (Molla *et al.*, 2020)

Crucially, resistance in rice is inherently quantitative and polygenic, controlled by numerous small-effect quantitative trait loci (QTLs) rather than single, dominant resistance (R) genes (Li *et al.*, 2022; Molla *et al.*, 2020; Wang *et al.*, 2021). Also, resistant plants often have traits like shorter height, fewer tillers, or earlier maturity, which are not ideal for farming. This makes it hard to tell whether the plant is truly resistant to the disease or just avoiding it because of its shape or growth pattern (Jia *et al.*, 2012; Li *et al.*, 2022).

Traditional biparental QTL mapping, while valuable, suffers from limited mapping resolution due to the relatively low number of recombination events captured within a narrow genetic base. In this complex landscape, Genome-Wide Association Studies (GWAS) have emerged as a transformative and robust approach for dissecting the genetic architecture of ShB resistance. By leveraging naturally diverse germplasm panels encompassing a wide spectrum of genetic variation, GWAS offers significantly higher resolution for detecting marker-trait associations (MTAs) compared to biparental mapping populations (Rabbi *et al.*, 2021; Rabbi *et al.*, 2021; Yu & Buckler, 2006). This power stems from the historical recombination events captured across diverse accessions. Consequently, GWAS has proven exceptionally effective in identifying novel resistance QTLs, pinpointing causal genes and alleles, and uncovering key defense pathways implicated in the rice-*R. solani* interaction.

Critically, the discoveries facilitated by GWAS directly enable advanced breeding strategies, including haplotype-based breeding, marker-assisted selection (MAS) for pyramiding favorable alleles, and the development of genomic selection (GS) models for complex traits like ShB resistance. Therefore, the primary objective of this review is to synthesize and critically evaluate the substantial progress made in unraveling the genetic and molecular basis of ShB resistance in rice, with a particular emphasis on the pivotal contributions of GWAS. We will:

1. Establish the critical role of robust and scalable phenotyping protocols, from traditional field screens to high-throughput phenomics, as the essential foundation for accurate genetic discovery and germplasm evaluation.
2. Detail how GWAS has overcome limitations of traditional mapping, leading to the discovery of major resistance genes (e.g., *SBRR1*), the elucidation of key defense pathways (e.g., the antioxidant system), and the identification of numerous novel QTLs and promising candidate genes.
3. Highlight how GWAS findings are integrating with and complementing breakthroughs achieved through other cutting-edge approaches, such as targeted gene editing (e.g., *DEP1*), overexpression strategies (e.g., *OsCSP41b, IDD14*), and the characterization of transcription factor networks (e.g., *OsbHLH34, OsBZR1*), all aimed at breaking the resistance-yield trade-off.
4. Discuss the exciting future prospects for GWAS, including enhancing resolution through larger, more diverse panels and improved phenomics, integrating multi-omics data (eQTL, metabolomics), modeling epistatic interactions, exploring non-coding regulatory variation, and directly feeding into genomic selection and precision breeding programs.

By providing a comprehensive overview of these advances, particularly the central role of GWAS, this review aims to illuminate the path forward for developing high-yielding rice cultivars endowed with robust and durable ShB resistance, a critical imperative for safeguarding global rice production.

Screening Protocols for ShB: From Field to Phenomics

A reliable and repeatable screening protocol is the cornerstone of identifying genetic resistance

to ShB. The most common methods involve artificial inoculation at the maximum tillering stage, with the "hill inoculation" technique being a standard in field evaluations (Sharma *et al.*, 1990). This method involves placing inoculum, often mycelial plugs or colonized substrates like typha bits, into the leaf sheath of plants, followed by maintaining high humidity to promote disease development (Bashya *et al.*, 2017; Parveen & Ali, 2018). Disease assessment is typically quantified using the Relative Lesion Height (RLH), which measures the vertical spread of the lesion as a percentage of total plant height, and is scored on a standardized 0-9 scale (IRRI, 1996) as per the Standard Evaluation System (SES) for rice (Kumar *et al.*, 2019).

The strength of these traditional field and greenhouse protocols lies in their direct relevance to agronomic conditions. However, their reliability can be influenced by environmental variability. To address this, controlled-environment methods have been developed to enhance repeatability. Techniques such as the microchamber (Jia *et al.*, 2007), mist-chamber (Liu *et al.*, 2009), and detached sheath inoculation (Venu *et al.*, 2007) provide more uniform conditions by standardizing humidity, temperature, and inoculum pressure (Jia *et al.*, 2013; Park *et al.*, 2008). For instance, the use of liquid-cultured mycelial balls inserted into the sheath and covered with aluminum foil to maintain high humidity has been shown to achieve a 100% infection rate, ensuring consistent and severe disease pressure for discriminating between resistant and susceptible lines (Park *et al.*, 2008). The detached sheath method, conducted in a laboratory setting, offers high throughput and requires minimal space and plant material, making it suitable for rapid preliminary screening. The reliability of these methods is evidenced by their successful use in identifying stable quantitative trait loci (QTLs) and in gene expression studies, underscoring their value for genetic dissection (Jia *et al.*, 2013; Liu *et al.*, 2009).

Looking forward, high-throughput phenotyping technologies are emerging to augment traditional scoring. Hyperspectral imaging, combined with advanced algorithms, has demonstrated remarkable accuracy (over 95%) in automatically detecting ShB scabs at the pixel level, offering a non-destructive, objective, and rapid alternative to visual disease assessment (Zhang *et al.*, 2021). This integration of digital phenomics with established pathological methods promises to further improve the precision, scalability, and repeatability of ShB resistance screening.

Pathogen Complexity and Infection Challenges

R. solani exhibits high genetic variability, classified into 14 anastomosis groups (AGs), with AG1-IA being the primary causal agent of rice ShB (Carling *et al.*, 2002a; Carling *et al.*, 2002b). Its extensive host compatibility, infecting plants from ~32 taxonomic families, and survival between seasons via buoyant sclerotia in soil and stubble significantly complicate control (Molla *et al.*, 2020). Primary inoculum (sclerotia or runner hyphae) initiates infection mainly via infection cushions penetrating the cuticle directly, or less frequently, via lobate appressoria entering stomata (Molla *et al.*, 2013).

Molecular Defense Mechanisms and Pathogen Countermeasures

Rice activates complex defense signaling pathways upon R. solani infection. Key phytohormones involved are salicylic acid (SA), jasmonic acid (JA), and ethylene (ET), modulating defenses against pathogen lifestyles (Glazebrook, 2005). Although traditionally considered necrotrophic, evidence suggests *R. solani* AG1-IA may exhibit hemibiotrophic traits, blurring defense pathway distinctions (Kouzai *et al.*, 2018). The pathogen deploys an arsenal of virulence factors: validated effectors include cytochrome C oxidase assembly protein (CtaG/cox11), glycosyltransferase GT2, and peptidase inhibitor I9 domains (Anderson *et al.*, 2017; Zheng *et al.*, 2013). Genomic studies predict numerous other candidates (e.g., polygalacturonases like RsPG3, RsPG4, AG1IA_04727; pectin lyases, histone modifiers) (Ghosh *et al.*, 2019; Rao *et al.*, 2019).

Oxalate secretion is a key virulence factor, countered by transgenic rice expressing oxalate oxidase (Molla *et al.*, 2013; Nagarajkumar *et al.*, 2005). A significant immune evasion tactic involves α-1,3-glucan masking of chitin, preventing recognition by plant PRRs; disrupting this mask via α-1,3-glucanase expression enhances resistance (Fujikawa *et al.*, 2012).

The Quantitative Genetic Architecture of ShB Resistance

Resistance to ShB is inherently polygenic, controlled by numerous small-effect quantitative trait loci (QTLs) rather than single dominant R genes (Wang *et al.*, 2021). Phenotyping is complex, typically involving lesion height (LH), relative lesion height (RLH), disease score (DS), percent disease index (PDI), and area under the disease progress curve (AUDPC) (Aggarwal *et al.*, 2022; Naveenkumar *et al.*, 2023). Resistance often exhibits negative correlations with plant height (PH), tiller number, and maturity time, making it crucial to disentangle true physiological resistance loci from morphological confounders (Jia *et al.*, 2012).

GWAS: Revolutionizing the Discovery of ShB Resistance Genes

Genome-Wide Association Studies (GWAS) have emerged as a powerful tool for dissecting the complex genetics of ShB resistance, leveraging diverse germplasm to achieve higher resolution than traditional QTL mapping. Key contributions include:

High-Resolution QTL Identification: Multiple GWAS have identified significant marker-trait associations (MTAs) for ShB resistance across all 12 rice chromosomes (Aggarwal *et al.*, 2022; Chen *et al.*, 2019; Naveenkumar *et al.*, 2023; Wang *et al.*, 2021). Studies consistently reveal higher inherent resistance in Aus, aromatic, and wild rice (*O. rufipogon, O. nivara*) accessions compared to elite japonica cultivars (Aggarwal *et al.*, 2022; Bhatia *et al.*, 2024; Chen *et al.*, 2019; Goad *et al.*, 2020).

Germplasm Screening and the Identification of Resistance Sources: Comprehensive screening of diverse germplasm collections has been a global endeavor to identify sources of ShB resistance, consistently confirming the scarcity of immune genotypes. Evaluations of cultivated varieties, landraces, and wild relatives have identified several sources of partial resistance. For example, international screenings have identified cultivars like Tetep and Jasmine 85 as consistent sources of moderate resistance, which are frequently used in breeding programs (Park et al., 2008). Wild relatives have proven particularly valuable, with accessions of *Oryza rufipogon* (IC336719 and IC336721) and *O. nivara* (IRGC104705, IRGC100898, and IRGC104443) being identified as resistant, exhibiting smaller lesions and reduced fungal penetration structures (Bashya *et al.*, 2017; Prasad & Eizenga, 2008).
In Bangladesh, the Bangladesh Rice Research Institute (BRRI) has been active in screening its genebank accessions to identify promising donors for rice breeding programs. A significant screening effort of 57 rice germplasm accessions identified the local cultivar 'Orgoja' (Acc. No. 5310) as a standout resistant source. Orgoja consistently exhibited the lowest Relative Lesion Height (8.33% - 11.66%) and a minimum SES score of 1 in both field and detached sheath inoculation methods, demonstrating stable resistance across different environments. Another accession, 'Gopal ghosh', was categorized as moderately resistant (Parveen & Ali, 2018). These findings are critical for BRRI's breeding objectives, as Orgoja provides a locally adapted and genetically distinct source of resistance for introgressing ShB resistance into high-yielding BRRI varieties such as BR11, which is highly susceptible. The ongoing research at BRRI thus bridges the gap between traditional germplasm screening and modern genetic improvement, aiming to pyramid resistant QTLs from donors like Orgoja into elite genetic backgrounds to develop durable ShB resistant rice varieties for Bangladeshi farmers.

Major Gene Discovery - *SBRR1*: GWAS enabled the landmark discovery of Sheath Blight Resistance Receptor-like Kinase 1 (*SBRR1*) (Feng *et al.*, 2025). They identified a 256-bp insertion in the *SBRR1* promoter (the *SBRR1*-R allele), prevalent in *indica* varieties from ShB-prone regions. This insertion creates a binding site for transcription factor bHLH57, driving high expression. SBRR1-R localizes to the plasma membrane via interaction with SIP1, requires phosphorylation, and activates downstream chitinase genes (*Chit3, Chit4*) to confer significant resistance without yield penalty under disease pressure. This represents the first major gene characterized with high breeding potential identified through GWAS (Table 1).

Unraveling Defense Pathways - Antioxidant System: GWAS across rice developmental stages identified 653 associated genes (Wang *et al.*, 2021). Functional validation focused on two key candidate genes: *OsRSR1*, encoding an RPM1-like disease resistance protein, and *OsRLCK5*, encoding a receptor-like cytoplasmic kinase. *OsRSR1* interacts with serine hydroxy-methyltransferase 1 (*OsSHM1*) to modulate ROS burst, while *OsRLCK5* interacts with glutaredoxin (*OsGRX20*), implicating the glutathione-ascorbate (GSH-AsA) antioxidant system as a crucial component of ShB defense (Table 1).

Novel Loci and Candidate Genes: GWAS continually identifies novel QTLs and candidate genes. Examples include the identification of 30 MTAs (including 8 novel QTLs) with candidate genes such as *Os05t0566400* (defense-related) (Naveenkumar *et al.*, 2023), and 22 significant SNPs in *O. rufipogon*, with loci on chromosomes 3 and 9 associated with multiple disease traits (Aggarwal *et al.*, 2022). A novel locus, *qShB6* (Chr6: 0.81–4.27 Mb), enriched for LRR-RLKs and transcription factors, was also identified using QTL-seq in a biparental population (Thesiya *et al.*, 2025), consistent with GWAS findings.

Beyond GWAS: Diverse Genetic Strategies Yield Breakthroughs

While GWAS has been pivotal, other approaches have also identified key resistance genes and strategies:

Precise Gene Editing (*DEP1*): Using CRISPR/Cas9 to create a truncated *DEP1* allele (dep1-cys) improved both ShB resistance and yield, avoiding the drawbacks of a full gene knockout. The *dep1* cys variant weakens its interaction with *IDD14*, which enhances PIN1a activity, and strengthens its interaction with IDD10, which suppresses ETR2 expression. Together, these changes reduce disease susceptibility (Zhu *et al.*, 2025) (Table 1).

Overexpression Strategies: Overexpression of *OsCSP41b* (chloroplast transcript stabilizer) enhances ShB, drought, and salt tolerance without yield penalty (Zhao *et al.*, 2025). Moderate IDD14 overexpression also boosts resistance without compromising yield (Cui *et al.*, 2022; Sun *et al.*, 2020) (Table 1).

Transcription Factor Networks: Recent studies have highlighted the importance of transcription factor (TF) networks in regulating rice resistance to pathogens. Key players include *OsbHLH34*, a positive regulator that enhances ethylene biosynthesis and defense responses by activating *OsERF34*. Plants with *OsbHLH34* knockouts are highly susceptible, while overexpression confers resistance (Zhai *et al.*, 2025) (Table 1). Similarly, *OsBZR1*, a central component of the brassinosteroid (BR) signaling pathway, has been shown to play a positive regulatory role in disease resistance; knockdown mutants exhibit increased susceptibility (Chen *et al.*, 2024) (Table 1). Members of the WRKY family also contribute to resistance modulation. For instance, *WRKY36* negatively regulates resistance by suppressing the sugar transporter *SWEET11* (Gao *et al.*, 2018), while *WRKY53* functions within BR signaling to fine-tune *SWEET2a* expression, also contributing to negative regulation of resistance (Gao *et al.*, 2021) (Table 1).

Other Genes Controlling the Resistance: Beyond transcriptional control, several favorable alleles and gene variants have been identified that enable resistance enhancement without compromising yield. The *idd10* mutant enhances ethylene signaling and resistance while maintaining normal yield levels (Li *et al.*, 2025). The *UMP1R2115* allele confers broad-spectrum resistance with no adverse effects on agronomic performance (Hu *et al.*, 2023) (Table 1). Similarly, *AMT1*;1, involved in nitrogen uptake and ethylene signaling, improves resistance while preserving yield stability (Li *et al.*, 2025; Wu *et al.*, 2022) (Table 1). Other genes such as *BGL2* (*β*-glucanase) and *GELP77* (a GDSL lipase) not only bolster resistance but also contribute to improved tillering and yield outcomes (Li *et al.*, 2025; Zhang *et al.*, 2024) (Table 1). GT1, which regulates sugar partitioning and tillering, maintains or slightly enhances resistance without reducing yield (Yang *et al.*, 2024) (Table 1). The gene-edited variant *RBL1Δ12* also increases resistance while sustaining normal growth and yield (Sha *et al.*, 2023). At the *Pigm* locus, *PigmR* provides strong resistance, while *PigmS* supports yield, collectively achieving a resistance–yield balance (Deng *et al.*, 2017). Additional positive regulators include GRF6, which modulates auxin and jasmonate signaling to enhance both yield and bacterial blight resistance (Yuan *et al.*, 2024), and IPA1, which boosts immunity and yield through phosphorylation-mediated mechanisms (Wang *et al.*, 2018). Finally, the *ROD1* allele serves as a dynamic modulator of the defense–growth trade-off, contributing to balanced plant performance under stress (Gao *et al.*, 2021) (Table 1).

Future Scope and Role of GWAS

GWAS has proven indispensable but its potential is far from exhausted. Key future directions include:

Enhanced Resolution & Power: Utilizing larger, more diverse panels (especially under-represented Aus and wild relatives), high-density genotyping (haplotypes, SV calling), and improved, standardized phenotyping (including image-based) will increase power to detect smaller-effect loci and rare alleles (Aggarwal *et al.*, 2022; Bhatia *et al.*, 2024; Mahantesh *et al.*, 2021).

Multi-Omics Integration: Combining GWAS with transcriptomics (eQTL), proteomics, metabolomics, and epigenomics will provide deeper mechanistic insights into gene function, regulatory networks (e.g., downstream of *SBRR1, OsbHLH34*), and gene-by-environment interactions (Wang *et al.*, 2021).

Elucidating Gene Networks & Epistasis: GWAS data is crucial for modeling epistatic interactions between resistance loci (e.g., how *SBRR1* interacts with antioxidant genes or hormone pathways) and for prioritizing candidates within QTL regions identified by both linkage and association mapping (e.g., *qShB6*) (Thesiya *et al.*, 2025).

Feeding Genomic Selection (GS) and Breeding: GWAS findings directly inform the development of robust GS models by identifying predictive markers. Validated genes and alleles (*SBRR1-R, dep1-cys*, favorable *IDD10, UMP1, AMT1;1* alleles) are prime targets for marker-assisted selection (MAS), gene pyramiding, and gene editing in elite backgrounds (Feng *et al.*, 2025; Mahantesh *et al.*, 2021; Zhu *et al.*, 2025).

Exploring Non-Coding Variation: GWAS can identify regulatory variants (like the *SBRR1* promoter insertion) controlling gene expression, offering novel targets for manipulation (Feng *et al.*, 2025).

Table 1. Molecular Determinants of Sheath Blight Resistance in Rice: Genes, Functions, and Mechanisms.

Gene	Full Name	Function	Reference
SBRR1	Sheath Blight Resistance Receptor-like Kinase 1	Plasma membrane-localized receptor-like kinase. The SBRR1-R allele (promoter insertion) drives high expression, activates chitinase genes (*Chit3, Chit4*), conferring significant resistance without yield penalty.	(Feng *et al.*, 2025)
OsRSR1	Rice Starch Regulator 1	Interacts with serine hydroxymethyltransferase 1 (*OsSHM1*) to modulate the ROS burst, implicating the glutathione-ascorbate (GSH-AsA) antioxidant system in defense.	(Wang *et al.*, 2021)
OsRLCK5	Receptor-Like Cytoplasmic Kinase 5	Interacts with glutaredoxin (*OsGRX20*), implicating the glutathione-ascorbate (GSH-AsA) antioxidant system in defense.	(Wang *et al.*, 2021)
DEP1	Dense and Erect Panicle 1	Edited allele (*dep1*-cys) weakens interaction with *IDD14* (enhancing PIN1a activation) and strengthens interaction with *IDD10* (suppressing ETR2 expression), reducing susceptibility and increasing yield.	(Zhu *et al.*, 2025)
OsCSP41b	Chloroplast Stem-Loop Binding Protein of 41kDa b	Chloroplast transcript stabilizer. Overexpression enhances ShB, drought, and salt tolerance without yield penalty.	(Zhao *et al.*, 2025)
IDD14	INDETERMINATE DOMAIN 14	Transcription factor. Moderate overexpression boosts resistance without compromising yield.	(Sun *et al.*, 2020)
OsbHLH34	Basic Helix-Loop-Helix 34	Transcription factor. Positive regulator activating *OsERF34* (ethylene biosynthesis/defense); knockouts are susceptible, overexpressors are resistant.	(Zhai *et al.*, 2025)
OsBZR1	BRASSINAZOLE-RESISTANT 1	Transcription factor central to brassinosteroid (BR) signaling; knockdown increases susceptibility, indicating a positive role in resistance.	(Chen *et al.*, 2024)
WRKY36	WRKY DNA-binding protein 36	Transcription factor. Suppresses SWEET11, negatively regulating resistance.	(Gao *et al.*, 2018)
WRKY53	WRKY DNA-binding protein 53	Transcription factor. Via BR signaling, balances SWEET2a expression, negatively regulating resistance.	(Gao *et al.*, 2018)
IDD10	INDETERMINATE DOMAIN 10	Transcription factor. Mutant (*idd10*) modulates ethylene signaling, increasing resistance without affecting yield.	(Li *et al.*, 2025).
UMP1	Proteasome Maturation Factor UMP1.	Provides strong resistance against a wide range of diseases while maintaining normal growth and yield characteristics.	(Hu *et al.*, 2023)

Gene	Full Name	Function	Reference
AMT1;1	AMMONIUM TRANSPORTER 1;1	Involved in nitrogen uptake and ethylene signaling. Specific alleles increase resistance and maintain yield stability.	(Li *et al.*, 2025; Wu *et al.*, 2022)
BGL2	β-1,3-GLUCANASE 2	β-glucanase. Confers increased resistance and promotes tillering/yield.	(Li *et al.*, 2025; Zhang *et al.*, 2024)
GELP77	GDSL LIPASE 77	GDSL lipase. Promotes JA accumulation, conferring broad-spectrum resistance and increased yield.	(Zhang *et al.*, 2024)
GT1	**GRAIN TILLERING 1**	Affects sugar partitioning and tillering. Can maintain or increase resistance without compromising yield.	(Yang *et al.*, 2024)
RBL1	RESISTANCE TO BLAST1.	The edited allele (*RBL1Δ12*) increases resistance without affecting growth or yield.	(Sha *et al.*, 2023)
PigmR	Pigm Resistant allele	Resistance allele at the *Pigm* locus. Pyramiding with *PigmS* (susceptibility allele) allows sustained blast resistance without yield penalty.	(Deng *et al.*, 2017)
GRF6	GROWTH-REGULATING FACTOR 6	Modulates auxin (IAA) and jasmonic acid (JA) pathways. Increases yield and bacterial blight resistance.	(Yuan *et al.*, 2024)
IPA1	IDEAL PLANT ARCHITECTURE 1	Increases immunity and yield via phosphorylation mechanisms.	(Wang *et al.*, 2018)
ROD1	RESISTANCE OF RICE TO DISEASES 1	Allele dynamically balances defense and growth.	(Gao *et al.*, 2021)

CONCLUSION

Research on rice ShB resistance has accelerated rapidly, shifting from a historical scarcity of strong resistance genes to the identification of concrete molecular targets and pathways. This represents a pivotal advance in our capacity to manage the disease. GWAS has played a central role in this transformation, enabling the landmark discovery of *SBRR1*, implicating key defense pathways such as the GSH–AsA antioxidant system, and continually revealing new loci and candidate genes. Its power to dissect polygenic traits has fundamentally reshaped the field.

These GWAS-driven findings are reinforced by diverse genetic approaches. Precise gene editing (e.g., *dep1-cys*), targeted overexpression strategies (*OsCSP41b, IDD14*), and the characterization of major transcription factor networks (*OsbHLH34, OsBZR1*) offer practical routes to enhance resistance, with several studies demonstrating the potential to overcome the long-standing resistance–yield trade-off. Together, these advances create momentum toward more durable and agronomically sound resistance.

Looking ahead, progress will depend on strengthening the pipeline from discovery to deployment. Refining high-throughput digital phenotyping will be essential for generating accurate, scalable datasets that can power advanced GWAS and validate resistance under field conditions. Equally important is the functional validation of priority GWAS-derived

genes through knockout, overexpression, and allele-editing studies, supported by multi-omics integration to uncover regulatory networks, epistatic interactions, and non-coding variation underlying ShB resistance. Finally, translating these insights into breeding outcomes will require coordinated use of marker-assisted selection, allele pyramiding, and genomic selection to deliver high-yielding cultivars with robust and durable resistance. Advancing this agenda is critical not only for scientific understanding but also for safeguarding global rice production and ensuring long-term food security under evolving pathogen and climate pressures.

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