

Rice sheath blight: Reappraising the host-pathogen interplay and breeding strategies in the Omics Era

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*Rice sheath blight is caused by *Rhizoctonia solani* Kühn. The characteristic symptoms include root, crown, hypocotyl, pod and belly-rot, sheath- and leaf-blight, banded leaf, brown patch and canker. Chemical management of this disease is largely unsustainable and non-viable thus underpinning the importance of resistant varieties. Although many QTLs governing tolerance to rice sheath blight have been mapped but robust, easy-to-use QTL without linkage drag is lacking. The present-day omics-based understanding of mechanisms, identification of novel donors, and development of varieties offer great scope. The present article summarizes the understanding of sheath blight disease development in rice and its possible management through tolerant varieties.*

Keywords: Disease resistance, Rice sheath blight, *Rhizoctonia solani*, Tolerant rice varieties

RICE Sheath blight (ShB) caused by basidiomycetes necrotroph *Rhizoctonia solani* Kühn [teleomorph – *Thanatephorus cucumeris* Frank (Donk)] is the second most devastating disease of rice globally after rice blast. The estimated yield loss ranges from 20 to 50% depending on crop growth stage, environment, fungal load, and management practices. Of late the incidence of disease increased owing to indiscriminate usage of nitrogenous fertilizers, high crop densities, humid canopies, and usage of high-yielding semi-dwarf rice varieties. *R. solani* is a polyphagous and soil-water borne fungus causing root-, crown-, hypocotyl-, pod- and belly-rot, sheath- and leaf- blight, banded leaf, brown patch and canker. Among all the anastomosis groups of this fungus, AG1-IA has widely been accepted as the causative organism of rice sheath blight. It may infect rice plants at any growth stage however, early maturing, semi-dwarf, high-tillering and

compact stature rice cultivars are ideal hosts. Its current management precisely relies on fungicides which are largely unsustainable and leave greater environmental footprints. Therefore, biological strategies focusing on understanding the host-pathogen interplay seem promising. Unfortunately, the occurrence of broad host range, lack of germplasm with adequate level of resistance, and overwintering ability of pathogen during unfavourable environments (serves as an inoculum of the disease) appear as the major roadblocks for this. Furthermore, the advances in genomic technologies have provided a better understanding of the pathogenesis and identification of genomic regions pertaining to sheath blight resistance. Novel biotechnological approaches have offered opportunities to tailor rice genotypes with resistance to sheath blight. This article is primarily focused on the elucidation of the mechanism underlying

pathogenesis and modern breeding strategies to manage sheath blight in rice.

Knowing the fungus, disease, and disease development

Rice sheath blight was first reported in Japan in 1910 and in India at Gurdaspur, Punjab in 1954. ShB pathogen typically infects leaf sheath and leaf blade, however emerging panicles may also be affected depending on the disease severity. Symptoms may be visualized between 24 to 72 hours succeeding the infection depending on the environmental conditions. Albeit, the rice crop at any stage is vulnerable to the sheath blight pathogen but disease progression is rapid at tillering stage. The fungal mycelium is a key determinant of the size and shape of the lesions in infected plant parts. The typical symptoms of the disease are the appearance of spherical to ellipsoidal water-soaked lesion having greenish-grey centre and



Fig. 1. Rice sheath blight symptoms and the pathogen.

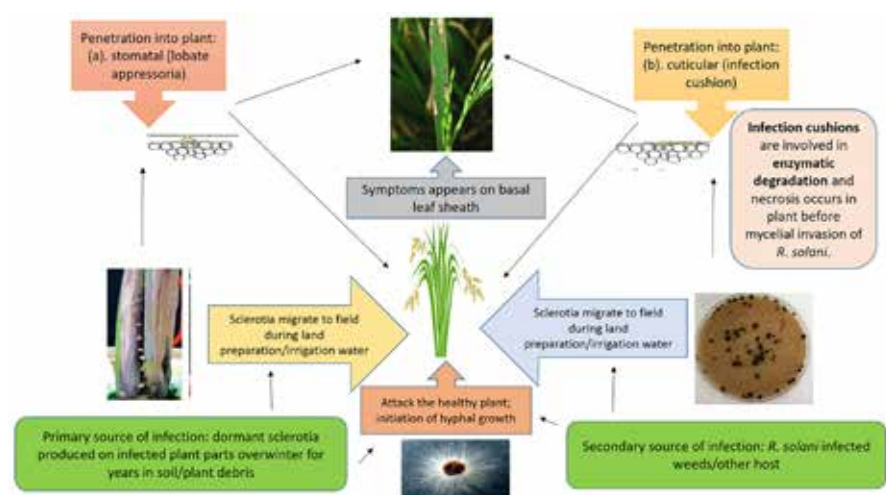


Fig. 2. Schematic representation of sheath blight infection in rice caused by *Rhizoctonia solani*.

brown margin on the leaf sheath near the water line (Fig. 1). The disease is vernacularly known as 'snake skin disease', 'mosaic foot stalk', and 'rotten foot stalk'. At the field level, the infection usually affects the plant in a typical circular pattern referred to as Bird's nest. The pathogen penetration may be stomatal or cuticular via lobate appressoria or infection cushion respectively (later being more frequent). The schematic representation of the disease cycle is presented in Fig. 2.

Molecular process involved in Rice-*Rhizoctonia solani* interplay

Rhizoctonia solani germinate along the veins of rice leaf and form infection cushion during disease establishment. These infection cushions are involved in enzymatic degradation leading to necrosis in plants prior to mycelial invasion. Metabolic changes such as a decline in photosynthesis, cellular respiration, formation of secondary metabolites, and phytohormones,

and accumulation of reactive oxygen species (ROS) inside the host cell lead to cell death.

The Pathogen's perspective

Diverse strategies such as effectors, secondary messengers, and secondary metabolites are employed by the fungi to colonize and establish itself in host to dampen the host defense machinery (Table 1). The cell wall presents the first barrier to fungal invasion; the fungi employ 223 different kinds of *carbohydrate-active enzymes* (CAZ) which include *glycosyl transferases* (GTs), *glycoside hydrolases* (GHs), *polysaccharide lyases* (PLs), *carbohydrate esterases* (CEs), *non-catalytic carbohydrate-binding modules* (CBMs), and *cell wall-degrading enzymes* (CWDEs). These primarily mediate degradation of cellulose, hemicellulose and chitin, and are thus important in penetration and infection. *Polygalacturonase* (PG), which are known to hydrolyse pectins and induce cell death is also known to play a role in pathogenesis. Three potential effector families viz. cytochrome C oxidase assembly protein CtaG/cox11 domain, glycosyltransferase GT family 2 domain and peptidase inhibitor I9 domain of *R. solani* AG1-1A were validated that could trigger crop defence responses in the form of cell death.

Table 1. List of effectors/secondary metabolites and their role in pathogenesis

Effector/secondary metabolites	Chemical properties	Role in pathogenesis
α -1,3-glucan	Polysaccharide	Mask chitin of the fungal cell wall to escape identification by PRR of the host
AG1IA_091612	Glycosyltransferase GT family 2 domain	Attachment of fungal pathogen and cell wall degradation
AG1IA_05310	Cytochrome C oxidase assembly protein CtaG/cox11 domain	Cell death phenotype
AG1IA_07795	Peptidase inhibitor I9 domain	Programmed cell death
<i>RsPG3</i> and <i>RsPG4</i>	Polygalacturonase; Cell wall degrading enzymes (CWDEs)	Release of reducing sugar, induce host tissue necrosis
<i>Host specific toxins</i> (<i>RS toxin</i>)	Carbohydrate consisting mannose, glucose, N-acetyl glucosamine, N-acetyl galactosamine	Act directly on living host protoplast and kill the cell of the plant
<i>Methyl-thio-propionic acid</i> (<i>MTPA</i>)	Secondary metabolite	Mycelial production and promote disease progression
<i>Oxalic acid</i>	Secondary metabolite	Inhibit synthesis of various phenolic substances and act synergistically with PGs, cause pH instability and cell wall degradation. Triggers programmed cell death
AG1IA_04727	Polygalacturonase	Role in pathogenesis

Once the extracellular signal is perceived by the pathogen, various signal transduction pathway is activated to induce pathogenicity. *Heteromeric G* protein constituted of $G\alpha$, $G\beta$, and $G\gamma$ regulates the cAMP pathway, MAPK pathway, and calcium calcineurin pathway which are major players in pathogenesis. The fungus utilizes α -1,3-glucans to escape chitin-triggered immunity of the host plant. The application of omics to study pathogenesis indicated that the induction of genes encoding extracellular protease, ABC transporter, and transcription factors are notable during the establishment phase. Contrastingly, during the necrotrophic phase, CAZymes, sugar transporters, cellular metabolism, and protein degradation-related genes are prominently induced. Furthermore, identification of the virulence gene of *R. solani* remains unaccomplished as the genetic transformation technique of the fungi is not yet standardized.

The Host's perspective

When a pathogen invades, penetrates, and colonizes the host plant, an arsenal of metabolic pathways and defence mechanisms are activated (Fig. 3) in the host to counteract the pathogen. Once the pattern recognition receptor identifies a foreign pattern (chitin) rather weak immune response PAMP triggered immunity (PTI) is initiated to invade the pathogen and restrict colonization. Further, direct or indirect interaction between resistance (R) proteins and avirulence (*avr*) effector induce a strong hypersensitive response known as effector triggered immunity (ETI). The involvement of jasmonic acid (JA), salicylic acid (SA) and ethylene mediated signal transduction pathways are also advocated to be involved in the resistance mechanism against *R. solani* in rice. Pathogenesis-related proteins (PR2,3,4,5) having antimicrobial properties are key players in host defence responses

against sheath blight in rice. Further, application of omics to study defense responses elucidated upregulation of genes for glycosyl hydrolase, secondary metabolite biosynthesis, cytoskeleton, and membrane integrity, the glycolytic pathway, and maintaining photosynthesis in resistance genotypes (Fig. 4).

Tug of war between host-pathogen

A tough battle is fought between the pathogen and host during establishment and progression of the disease. An arsenal of defense chemicals such as phytoalexin (sakuranetin), phytocassane A, B, C, D, and momilactone A, B are induced in the host plant upon sensing the attack. Higher synthesis of lignin, polyphenolpropanoid by the host reinforces the defense system. Detoxification of phytoalexins to other less toxic chemicals such as sakuranetin-40-O-b-d-xylopyranoside, naringenin and naringenin-7-O-b-d-xylopyranoside is employed by the pathogen. Secretion of host-specific

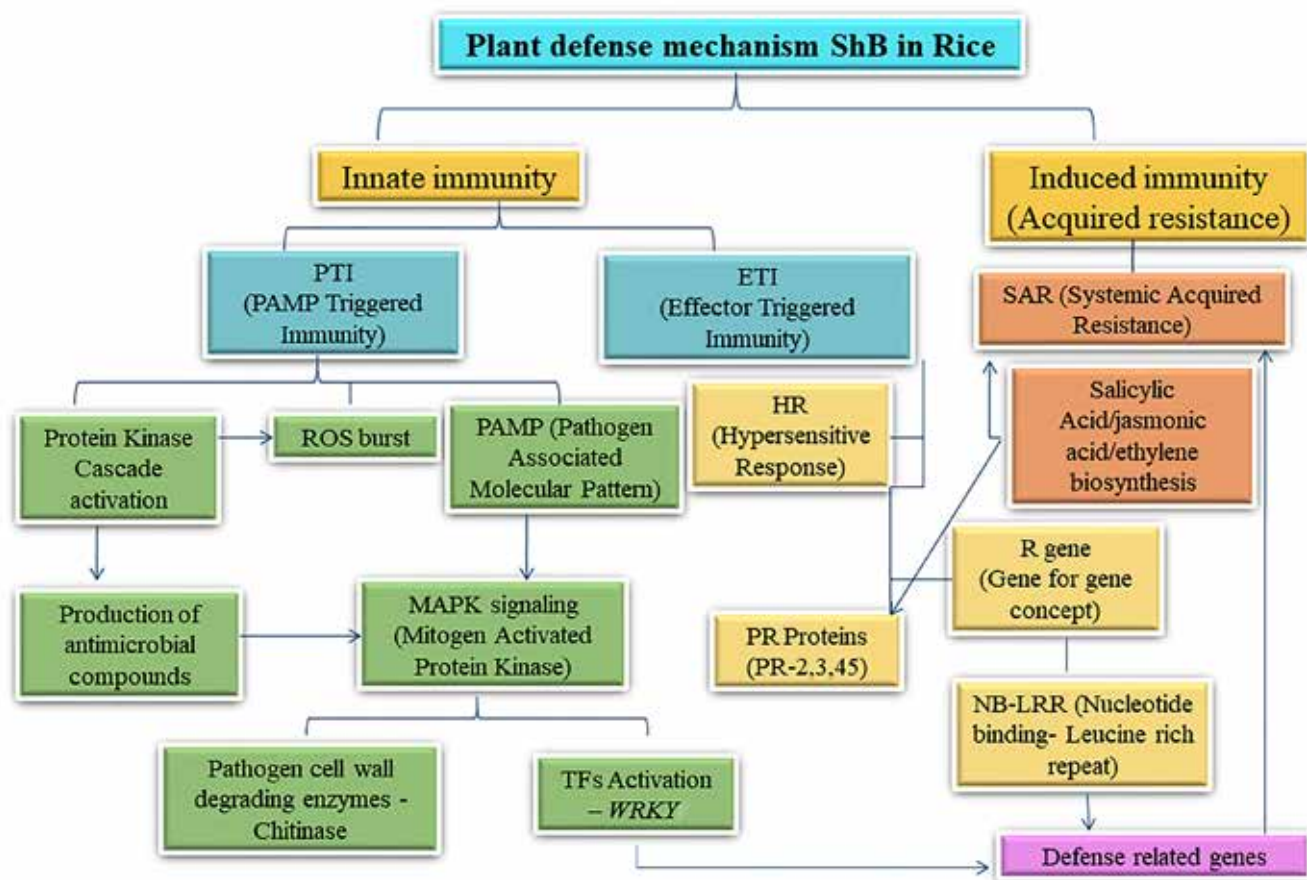


Fig. 3. Various plant defense mechanism against infection of rice sheath blight pathogen along with plant response.

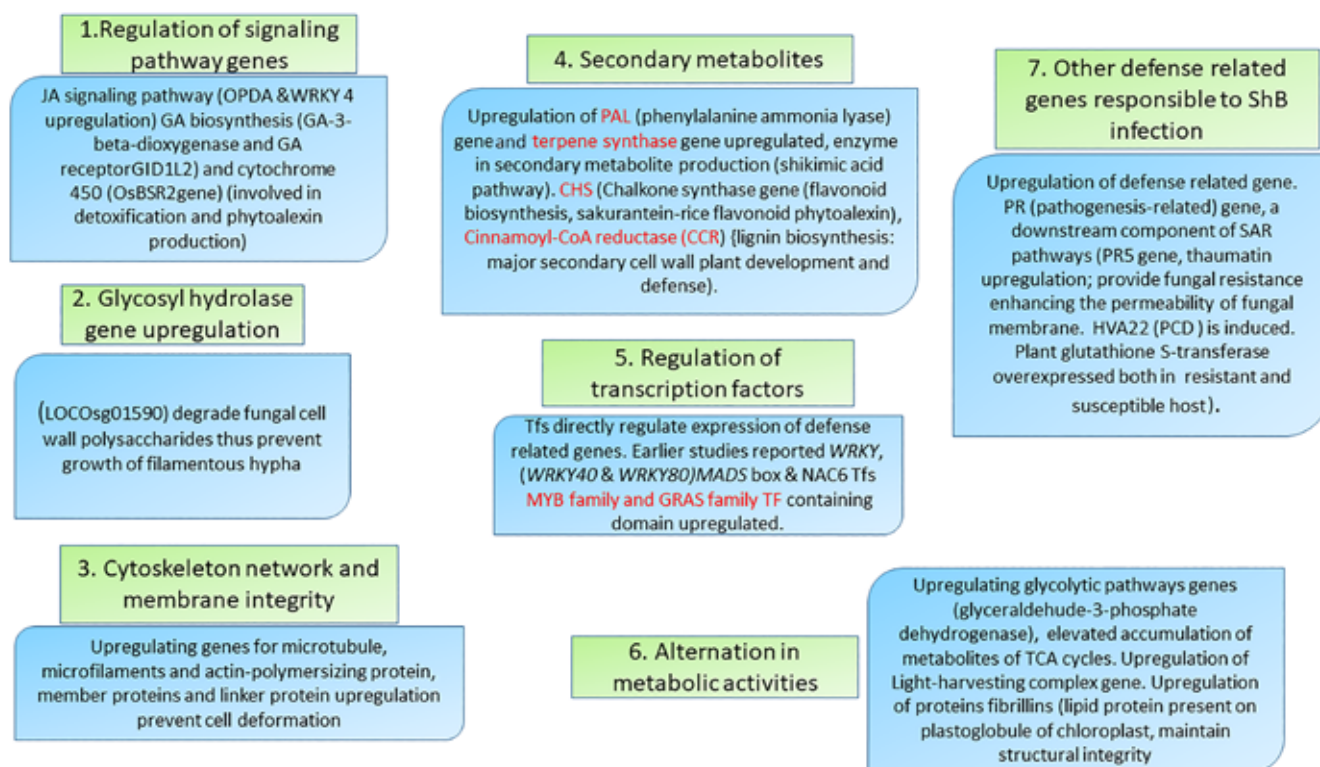


Fig. 4. Various cellular functional and molecular response in rice for rice sheath blight pathogen.

RS toxin composed of glucose, mannose, N-acetyl galactosamine and N-acetylglucosamine in more virulent isolates also appears as a counter-measure to dampen host defense.

Breeding strategies for sheath blight in rice

The standard protocol for screening for disease resistance is based on relative lesion height (RLH) which is calculated in the percentage or ratio of lesion height to plant height.

Conventional breeding strategies against *R. solani*

Breeding for resistance against sheath blight in rice is mainly based on disease escape and disease resistance. Disease escape strategy is mainly based on plant ideotypes such as plant height, heading date, and stem thickness. Conventional breeding for sheath blight resistance in rice is limited due to the direct association of RLH with plant height. Hence, the intervention of molecular and biotechnological techniques is plausible in resistance breeding against *R. solani*. Identification of resistance genotypes appears to be a preliminary step in resistance

Table 2. List of rice germplasms having moderate to high levels of tolerance against Rice Sheath Blight (ShB) disease

Rice species	Rice genotypes identified as sources of resistance to sheath blight disease
<i>Oryza sativa</i>	Teqing, Tetep, Jasmine 85, Dudsor, NC 678, Bhasamanik, Jaya, IR24, IR26, IR29, Mashoori, Jagganath, Tapoo cho Z, Tetep, Bharati Rohini, BPT-6, BogII, MTU 3, MTU 3642, MTU7, MTU13, Saket, Arkavati, Aduthurai, Jarjan, Nepal 8, Nepal 555, Thichang Native1, Phougak, Gumdhan, Ngnololasha, Wazuhophek, SM 801, 10-3
<i>Oryza nivara</i>	IRGC 104443, IRGC 104705, IRGC100898
<i>Oryza officinalis</i>	IRGC 105979,
<i>Oryza meridionalis</i>	IRGC 105306, IRGC105608
<i>Oryza barthii</i>	IRGC 100223,
<i>Oryza latifolia</i>	DRW 37004, WR 106

breeding, researchers identified resistant rice genotypes against ShB. In general, *indica* cultivars show more resistance than *japonica* cultivars. Moreover, among rice wild complex, *O. rufipogon*, *O. nivara*, *O. meridionalis*, and *O. barthii* are resistant to sheath blight (Table 2).

Identification of genomic regions harbouring *shb* resistance in rice

Numerous earlier studies elucidated the quantitative nature and polygenic inheritance of resistance against sheath blight in rice. Attempts were made to map the genomic regions; >50 major and minor QTLs were identified

across all the 12 chromosomes in rice. However, among all mapped QTLs, qShB9-2 and qSBR11-1, were reported as the major loci contributing 25% and 14% of total phenotypic variation, respectively. These QTLs were consensually mapped in several biparental QTL mapping studies representing many donor parents. Major QTLs with high contributions to resistant phenotype are qSBR9-2, qSBR11-1, qSB-9TQ and qSB-11LE. Also, genome-wide association studies have been attempted to identify genomic regions harbouring resistance marker-trait associations has been identified.

Marker-Assisted breeding (MAB) for ShB resistance in rice

Mapping and validation of QTLs appear a preliminary step for employing them in MAB programs. Most of the reported QTLs imparting resistance to ShB in rice explains <20% of phenotypic variance and also resistance through QTLs may be diluted with different genomic backgrounds, environments and other factors. Therefore, multiple additive QTLs pyramided will give more stable and durable resistance across the environment and genomic background. Pyramiding efforts dedicated to breeding ShB resistance rice are tabulated in Table 3.

Biotechnological approach for tailoring ShB-resistant rice

Recent advances in isolation and characterization of Pathogen-related (PR) genes, anti-fungal genes, antimicrobial genes, and master genes for defense responses affecting *R. solani* growth. Additionally, the unavailability of a stable source of resistance against ShB in rice germplasm makes genetic engineering a potentially viable option for designing ShB tolerance in rice. A schematic representation of potential candidate genes utilized for development of transgenics along with their functions are presented in Fig. 5. Glucanase and Chitinase are most widely utilized genes for engineering ShB tolerance in rice. The choice of promoter indeed remains a critical determinant for the success of any genetic engineering program; Rice actin 1, maize ubiquitin and CaMV35S has been widely used for constitutive expression of transgenes for ShB tolerance. Small interfering RNA (siRNA) mediated host-induced gene silencing/ RNA interference targeting pathogenesis-related Polygalacturonase and MAP kinase gene has also been utilized for tailoring ShB tolerance in rice. Apart from this, current gene editing technologies such as CRISPR/Cas can also be employed to modify or target candidate genes (Glucanase and Chitinase).

CONCLUSION

The green revolution has paved

Table 3. List of rice lines with sheath blight QTLs transferred through marker assisted breeding strategy to confer tolerance to rice sheath blight (Modified from Senapati *et al.* 2022)

Recipient parent	Donor parent	QTL introgressed
ZD88, WLJ1	IL55, IR24, Lemont	Sheath Blight (<i>qSB9^{TO}</i>), Tiller angle (<i>TAC1^{TO}</i>)
Pusa Basmati 1	Tetep	Sheath Blight (<i>qSBR11-1</i>), Blast (<i>Pi-54</i>)
CO51	Tetep	<i>qSBR11-1</i> , <i>Pi-54</i>
ASD 16 and ADT 43	Tetep and IRBB60	Bacterial leaf blight (<i>xa5 + xa13 + Xa21</i>), Blast (<i>Pi54</i>) and sheath blight (<i>qSBR7-1 + qSBR11-1 + qSBR11-2</i>)
CB14004 and CB14002	Tetep	Sheath Blight (<i>qSBR11-1</i>)

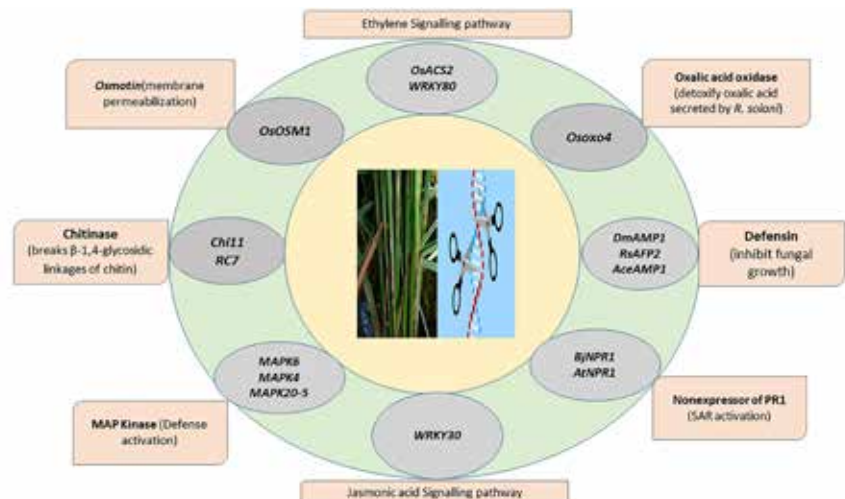


Fig. 5. A schematic representation about various classes of genes which can be a potential target for editing for development of sheath blight resistant varieties.

the way for the popularisation of high nitrogen-responsive semi-dwarf rice varieties which are increasingly susceptible to sheath blight of rice. Sheath blight has threatened rice production globally and may continue to be a menace. The management of this disease using resistant variety is definitely the most viable and sustainable approach but suffers from the following bottlenecks:

- Identification of virulence gene in *R. solani* remains unsuccessful till date thus making it difficult to understand the behaviour of pathogen.
- None of dominant resistance genes or loci have yet been discovered, cloned, characterized, and analysed.
- Sheath blight QTL are found to be positively correlated to plant height and days to heading thus creates severe linkage drag.
- No major QTL with very high phenotypic variance has not been detected.
- Broad host range and unavailability of identified germplasm with an adequate level of resistance.

However, a better understanding of the molecular mechanism of pathogenesis and defence mechanisms in the pathogen and host will help fasten the strategies for the development of resistance breeding. Moreover, an alternative and precise sheath blight scoring system independent of plant height is needed to avoid complexities. Future research needs to be focussed on the identification of the virulence gene. CRISPR-mediated genome-editing, and associated technologies to identify, study, and validate, the virulence gene can be potentially useful. Further, to utilize omics technologies, standardization of transformation technique of the pathogen needs to be in place. The breeding program focused on marker-assisted introgression of QTLs into popular cultivars coupled with the pyramiding of transgene(s) and multiple biotic and abiotic stress-tolerant genes will be helpful.

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