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A Review

Genomics and transgenic approaches for sheath blight resistance development in rice

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Among the most devastating fungal diseases, rice sheath blight is brought on by *Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris*), and it significantly reduces rice grain output. Since there are no dependable resistance genes and nothing is known about the biology of the disease, rice sheath blight continues to be a significant global problem. Presently, fungicides are most frequently used to treat rice sheath blight. However, they harm the environment and are not advised. Genomics and transgenic approaches show promise for sheath blight control by identifying key insight genes and developing the host resistance plants. This review emphasizes comprehending the genetics underlying host-pathogen interactions, identifying gene loci/markers, and designing a transgenic host genome.

Keywords: *Rhizoctonia solani*, Sheath blight, Host-pathogen interaction, Genomics, Transgenics, RNAi, Genomic editing

Introduction

Rice is the main cereal crop that provides food for two-thirds of the world's population. Increases in rice production could significantly ease the pressure on world food production (Ricepedia). Production is under constraint due to the shrinkage of agricultural land, biotic and abiotic stresses, and climatic changes. Rice suffers heavy yield loss due to various biotic stresses like sheath blight, bacterial blight, blast, and tungro. Losses in rice crops due to all pathogens had been estimated to be 12-15%[1-3]. The plant protection measures adopted to control rice diseases and insect pests have always been a major challenge due to the evolution of pathogens and pests.

A soil-borne Basidiomycete fungus called *Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris*) causes the sheath blight disease (ShB) in rice[4]. The disease has been considered one of the major constraints for rice production. The fungus possesses a wide host range, thus infecting more than 32 plant families and 188 genera. Based on unique physiology, genetic makeup, and hyphal interactions, the pathogen *R. solani* Kühn is divided into 14 anastomosis groups (AG1 to AG13 and AGBI) (Table 1). The most devastating pathogen that causes sheath blight disease in rice is thought to be AG1 IA, an intra-specific member of the *R. solani* AG1 group[5].

In rice, characteristic symptoms of the disease appear in the form of spherical to water-soaked, oval-shaped, and greyish to light brownish lesions on the sheath of leaves and blades, with a brown margin. The disease's inoculums may be from the infected plants, either sclerotia or runner hyphae. These sclerotia can remain dormant in the soil for several years and can re-infect the plants when the conditions are conducive to fungal growth during the crop season (Fig. 1). A necrotrophic fungus designated *R. solani* develops its hyphae on the exterior of the hosts to produce the distinctive lobate appressoria, which develops into infectious hypha to cause disease[6]. It is one of the most devastating diseases, with an estimated 5.9 to 69% yield loss. Farmers count on chemical fungicides to manage the sheath blight disease, which results in pesticide run-off and environmental pollution because there are no viable resistant rice cultivars.

The development of rice cultivars resistant to sheath blight has been impeded by the scarcity of resistance in the existing rice germplasm, the polygenic nature of resistance, and the unknown complexity of resistance. Several thousand germplasm, landraces, and genotypes were screened by many to identify resistance lines to sheath blight fungus by groups, but in vain. Many investigators

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Table 1 — Details of hosts and the diseases caused by different anastomosis groups of *Rhizoctonia solani*

Plant	Disease	Disease symptoms	Anastomosis group
Rice	Sheath blight	Leaf	AG1-IA
Barely	Barely stunt disorder	Roots	AG3
Maize	Sheath blight	Leaf, sheath	AG2-2
Wheat	Root rot	Stem	AG-8
Potato	Black scurf Stem canker		AG3
Sorghum	Sheath blight	Stem	AG-1IA
Sugar beet	Root and crown rot	Root	AG2-2IV & AG2-2IIIB
Tobacco	Leaf spot and root rot	Stem	AG-2-2 & AG-3
Cucumber	Belly rot	Fruit	AG4
Soya bean	Seedling blight	Seedlings	AG2-2IIIB, AG4 & AG5
Coffee	Necrotic leaf spot	Leaves	AG1-ID
Oilseed rape and canola	Seedling damping-off, seedling root rot and basal stem or foot rot (brown girdling root rot) of adult plants	Seedlings and root	AG2-1 & AG4
Cauliflower	Damping off	Seedlings	AG2-1
Cotton	Root rot	Root	AG4 & AG7

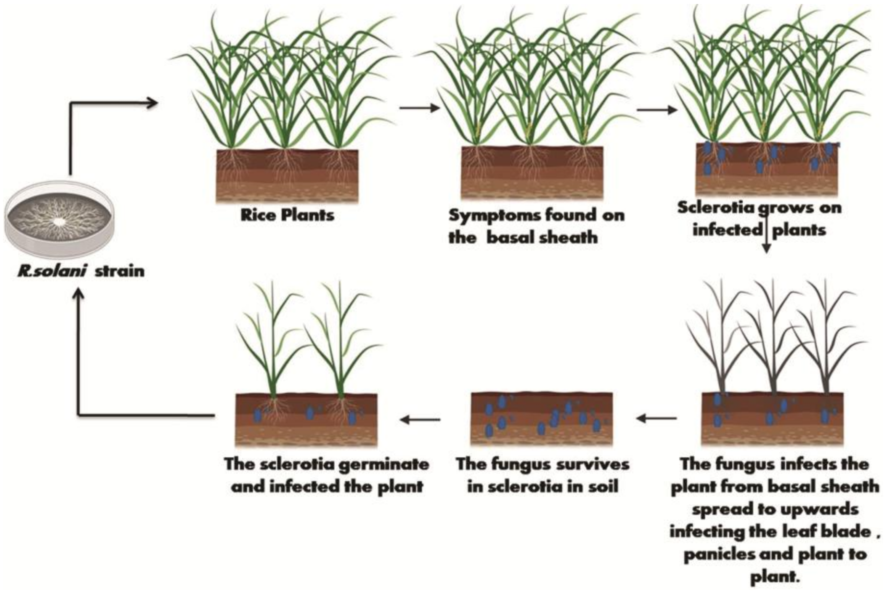

Fig. 1 — Rice sheath blight disease cycle

have tried to identify the QTLs and developed transgenic rice plants for disease resistance[7]. However, commercial cultivars with a desirable level of field resistance to sheath blight disease are not available. One of the major bottlenecks in developing resistance against this pathogen is the lack of a good understanding of the molecular mechanisms of host-pathogen interaction and the genes associated with the infection process and disease establishment. Therefore, substantial work is required to establish a reliable and durable resistance to the sheath blight disease in rice.

Though cultivars with varying degrees of resistance have been found, no rice cultivar has been shown to be genetically immune (with durable resistance) to *R. solani* to date[8]. Resistance to rice ShB is an intricate process, a quantitative trait controlled by polygenes[7]. Over the past 20 years, many ShB resistance quantitative trait loci (QTL) have been found, but the results of different studies haven't always been consistent. Using the Illumina platform, 13 inbred lines of rice (*Oryza sativa L.*) had their whole genomes sequenced in order to find candidate genes for resistance to ShB and non-synonymous

SNPs (nsSNPs)[7]. Today, breeding efforts do not exploit these QTLs or significant genes as resistance donors[8]. Similar measures were attempted to transfer resistance sources from recognized sources, such as Tetep, Teqing, and Jasmine 85, as well as from the *O. sativa* germplasm, which has been effective in a small number of cultivars with better resistance to ShB. Over all, breeding for ShB resistance was challenging due to the absence of resistant donors seen in the cultivated variety and inconsistent phenotyping across years and geographical areas.

Rice that has had antifungal genes inserted into its genetic makeup has demonstrated good resistance to ShB. The transgenic rice lines harboring *bar* different chitinases - chi11[9], RC7, chi, Cht42, basic chitinase, and bitter melon chitinase, thaumatin-like protein (tlp), pinA, pinB, ribosome-inactivating proteins, antimicrobial protein, β-1,3-glucanase and defensin genes suggested that a resistance level up to 50 to 70% was achieved in the *in-vitro* assays.

The use of genomics and transgenic approaches in many pathogenic fungi has been proven to be successful. However, relatively little work has been done to create ShB resistance. RNA sequencing is a promising technique that helps understand the functional components of the host and pathogen genomes during pathogen interaction and the progression of the disease. It offers a largely objective way to precisely and methodically define an organism's transcript fluctuations. The transcriptional profiles of microorganisms and plant pathogens can now be studied or understood with the use of this technology. RNA-seq can identify novel fungal pathogen transcripts[10] and capsule formation genes. RNA-seq could be a promising tool for understanding plant-pathogen interactions and to the extent that alternative control strategies and resistant cultivars are developed[11].

In the current review, we emphasize the comprehensive report on the recent advancement of understanding the host-pathogen interactions in the genomic era. The progress made through genomics and the transgenic approach was also reviewed.

Genomics

The objective of genomics is to describe, sequence, and study the entire genome's genetic makeup, as well as its forms, activities, and relationships. Bioinformatics, epigenomics, phenomics, transgenomics, genomic selection, proteomics, metabolomics, metagenomics, and system biology have all contributed to the growth and improvement of the subject. Genome sequencing has improved dramatically in terms of speed, capacity, and affordability thanks to next-generation sequence technologies. Furthermore, bioinformatics advancements have enabled hundreds of life-science databases and projects to support scientific research. These databases store and organize information that can be easily searched, compared, and analyzed[12].

Pathogen genomics

The development of next-generation sequencing technology makes it possible for us to sequence the genomes of several strains of various plant pathogenic species. These sequences are known as reference genomes, and many genomics investigations may use them as their foundation[12]. The technique helped analyze population genetics and evolutionary genetics and identify pathogen determinants, virulence factors, host association, and pathogenic mechanisms, in particular using reference genomes to pathogens genomes (Fig. 2).

Using RNA sequencing, numerous research teams have examined pathogen-host interactions in important plants like *Magnaporthe oryzae*, *Ustilago maydis*, and *Phytophthora infestans*[13]. Different pathogens, including *Colletotrichum*, *Postia placenta*, *Moniliophthora perniciousa*, *Xanthomonas*, and *Fusarium*, as well as their hosts, have been studied using transcriptomics analysis. Currently, *R. solani* AG1IA, AG1IB (isolate 7/3/14), AG3, AG8, and AG2-2IIIB have had their genomes sequenced. These organisms' genomes have also been compared. Comprehending the proteome of *R. solani* has helped us better understand the mechanisms through which it adapts to different plant hosts[10].

The functional mechanism of pathosystems was previously understood using transcriptome data, for example, a brown spot of *Zoysia japonica* and foliar blight of soybean caused by *R. solani* AG1-IA[11], rice blast disease caused by *Magnaporthe oryzae;* bottom-rot of lettuce caused by *R. solani* AG1-IB, white mold disease in brassica caused by *Sclerotinia sclerotiorum*, maize smut disease and other pathosystems caused by *Ustilago maydis*. Recent studies of *R. solani* gene expression profiles, transcriptome, and gene regulatory network definitions have greatly advanced our understanding of sheath blight illness and host-pathogen interaction[10].

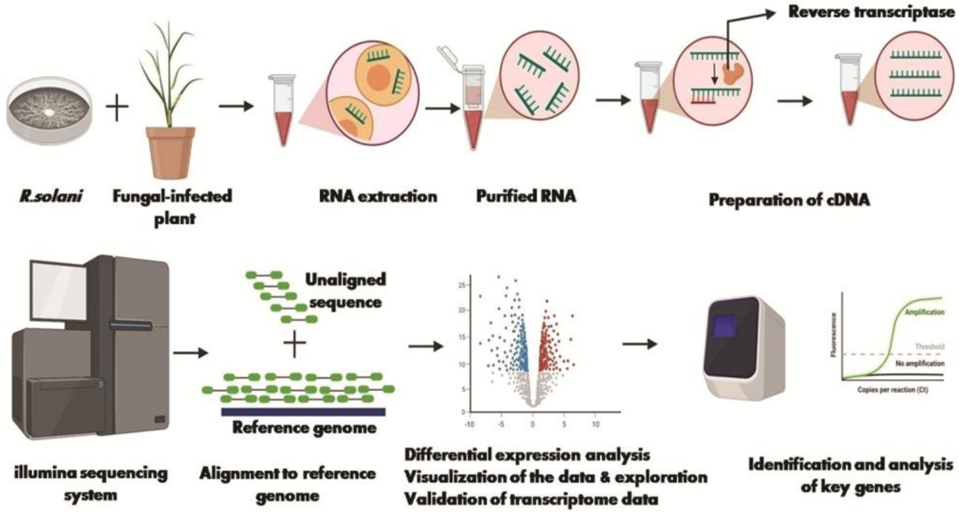

Fig. 2 — Analytical flowchart to study *R. solani* by RNA-Seq

Host genomics

Much work has gone into screening rice germplasm for ShB resistance during the past three decades. From 1995 onwards, many QTLs have been identified for sheath blight resistance using different mapping populations and molecular markers on all 12 chromosomes[8]. Though many groups have tried to map the QTLs for ShB resistance, it is very difficult to use them to develop fungal resistance, as polygenes control the QTLs. The research groups of Dr. Yulin Jia, USDA-ARS, made a breakthrough by mapping the qShB9-2, the major region controlling ShB resistance[14]. Also, Dr. Shannon Pinson, USDA ARS, Beaumont, has found that 18 chromosomal regions possess genes that can resist ShB while mapping recombinant inbred lines (RILs). This also included the qShB9-2 genetic region confirmed by Dr. Jia. In India, qSBR11-1 is the primary locus responsible for 25 and 14%, respectively, of the entire phenotypic variation in ShB. A β, 1-3 glucanase-like defense gene was discovered in the qShB9-2 region. Ten potential genes from the resistant lines Jasmine 85, Teqing, and MCR010277 were found in qShB9-2[7]. Although some partial resistance has been identified in some cultivars through breeding programs, these cultivars become obsolete in due course due to the emergence of virulent forms of the pathogen.

Based on studies performed with the indica and japonica subspecies, it was observed that the indica subspecies showed a higher resistance towards the sheath blight disease. Many other researchers also identified the qShB9-2 QTL region that contains ten candidate genes along with β,1-3 glucanase defense genetic factor. The QTL, qSBR11-1 obtained from the resistance rice cv Tetep, contains about 154 genes. Among them, it found that 26 genes were disease-responsive[15]. Twelve candidate genes were obtained from susceptible Lemont cultivars for the qSB11-LE. The most likely candidate genes for the qSB11-LE are two receptor-like protein kinase 5 precursors and a lipase-like gene. There are no cultivars with total immunity, nevertheless, and no significant genes that confer immunity to *R. solani* have been discovered.

Researchers have used RNA sequencing to identify the candidate genes in host plants, which aids in understanding the host plants' defense mechanisms. WRKY family members play a significant role in both positive and negative disease resistance modulation in plants. According to studies, OsWRKY4, OsWRKY13, OsWRKY30, and OsWRKY80 favorably influence ShB resistance. The PR (pathogens are related) genes are critical for a plant's defense against pathogens, and by being overexpressed, OsOSM1, a PR5 family member, may enhance resistance to ShB. Overexpression of OsACS2 enhances rice's defenses against ShB and rice blast fungus. It has been reported that OsFBDUF47 at LOC_Os09g37590 enhances tobacco disease resistance. Transcriptome analysis to examine rice's molecular defense system against ShB. OsMYB4, OsPR4b, OsPAL4, 14-3GF14f, OsASR2, and OsACS2 are a few previously known genes that contribute resistance to ShB. Furthermore, overexpression of rice PGIP strengthened resistance against *R. solani*[16].

Transgenic

Transgenic plants are those whose DNA has been modified through genetic engineering. Giving the plant a novel trait that doesn't occur naturally in the species is the aim. Transgenic plants carry a gene or genes that have been deliberately added. The inserted gene sequences, known as transgenes, can come from completely different species or unrelated plants. A plant's increased productivity and usefulness are the intended outcomes of introducing a particular gene combination[17-19]. Several groups have attempted the development of transgenic plants for ShB resistance in rice and, reportedly, have transformed various genes and evaluated their effectiveness against ShB under greenhouse conditions as well as in the field (Fig. 3). Some of them are listed in (Table 2).

Host genes

The build-up of PR genes is one of the primary defense mechanisms known to occur during pathogen infection. One method of developing transgenics has been to use the expression of the PR genes. When rice chi11 and rice RC7 are overexpressed in transgenics, it has been shown to increase tolerance to ShB. OSM1 (a member of the TLP-PA subfamily) has been expressed to improve tolerance to ShB[7], rice chitinase (chi11) and thaumatin-like protein, chi11 and β-1,3-glucanase, DmAMP1 and RsAFP2, ShB tolerance was brought on by the overexpression of OsPGIP. This protein inhibits the pectin-degrading polygalacturonase[16]. It has been demonstrated that increased tolerance to the ShB pathogen results from

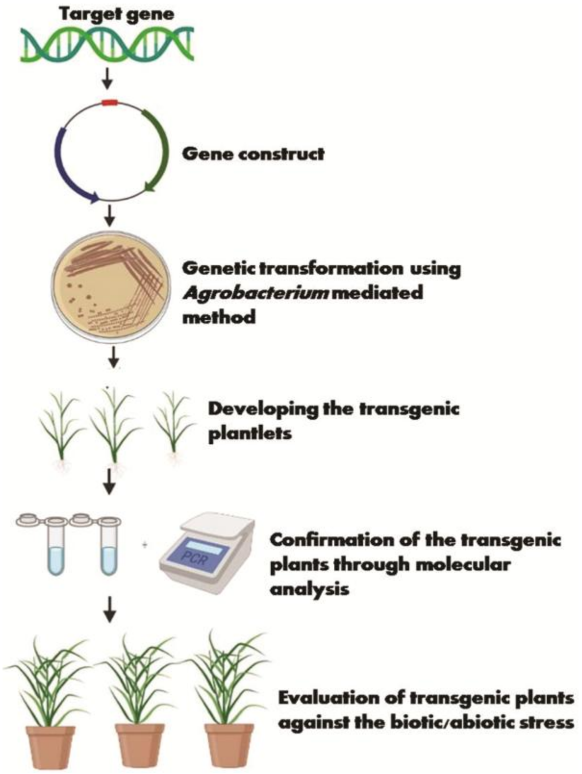

Fig. 3 — Schematic representation of transgenic development

Table 2 — Genes used for the development of ShB resistance in rice by the transgenic approach.

Gene	Source
Chi11	Rice
Tlp-D34	Rice
RC7	Rice
pinA, pinB	Wheat
chi11, xa21 & cry gene	Rice
MOD1 & RCH10	Maize & Rice
ech42, nag70& gluc78	*Trichoderma atorviride*
Tlp& Chi11	Rice
Ace-AMP1	*Allium cepa*
tlp, chi11 & Xa21	Rice & tobacco
chi11 & β-1,3-glucanase	Rice
Dm-AMP1	*Dalia merkii*
McCHIT1	*Momordica charantia*
Dm-AMP1& Rs-AFP2	*Dalia merkii & Raphanus* sativus
cht42	*Trichoderma virens*
RS-AFP2	*Raphanus sativus*
chi11 & ap24	Rice & tobacco
Cutinase	*Nicotiana tabacum, Glycine max, Solanum tuberosum*
CYP51A, CYP51B, and *CYP51C*	*Hordeum vulgare*
MAPK, cyclophilin (CYC1), and calcineurin (CNB) regulatory subunit gene	*Triticum* aestivum
BjNpr1	*Brassica juncea*
RCH10 & AGLU1	Rice & Alfalfa
AflR	*Zea mays*
aflS, aflR, aflC, pes1, aflep	*Arachis* hypogaea
Velvet and Fusarium transcription factor 1	*Musa* sp
Chitin synthase (Chs) 3b	*Triticum aestivum* L.
F-box protein Required for Pathogenicity 1 *(FRP1)*, *F. oxysporum* Wilt 2 *(FOW2)*, *12*-oxophytodienoate-*10,11*-reductase gene *(OPR)*	*Arabidopsis thaliana*
Hygrophobins1 *(VdH1)*	*Gossypium* sp.
AtNPR1	Rice
OsWRKY80	Rice
OsOXO4 and OsCHI11	Rice
AtNPR1 and OsCHI11	*Arabidopsis thaliana* and rice
Bacisubin	*Bacillus subtilis* strain BS-916
RPMK1-1 and RPMK1-2	Rice
OsASR2	Rice
OsBSR2	Rice

overexpression of the enzymes oxalate oxidase 4 and oxalate decarboxylase[20], both of which are proficient in degrading the virulence component oxalate. ShB resistance was increased as a result of the overexpression of the transcription factor genes WRKY30 and WRKY80.

Under the direction of the PBZ1 promoter and pathogen-inducible PAL, the AceAMP1 and OsACS2 have been successfully produced in rice. chi11 and ap24 rice oxalate oxidase 4 and rice chitinase 11, *Arabidopsis* NPR1 and rice chitinase 11, and the alfalfa b-1,3-glucanase gene (AGLU1) and rice basic chitinase gene (RCH10) genes for a higher level of confrontation over the resistance conferred by a single gene. *Trichoderma atroviride* ech42 (endochitinase), gluc78 (exo-1,3-b-glucanase) glycoside hydrolase, and nag70 (exochitinase) genes have been pyramided in all possible combinations and have been proven to increase pathogen tolerance. A single chitinase gene, LOC Os11g47510, was recently cloned from a QTL area (qSBR11-1) for ShB resistance in the *R. solani*-tolerant rice line Tetep. It was then verified by a gene transfer into the vulnerable rice strain Taipei 309. Transgenic plants have been shown to exhibit increased ShB tolerance as a result of the overexpression of multiple additional chitinase genes, such as OsCHI11 and RCH10. The levels of PGIP expression in the transformed rice lines correlated with disease resistance, and overexpression of rice PGIP significantly increased resistance against *R. solani* by preventing fungal polygalacturonases from degrading rice tissue[16]. Through the negative modulation of ShB resistance in rice, *R. solani* infection may initiate BR signaling to enhance SWEET2a expression *via* WRKY53. Sheath blight resistance was greatly enhanced by the OsABC9 gene, which is located in the well-known sheath blight resistance QTL qShB9-2.

Pathogen genes

The RNAi-based strategy involves silencing the target gene and preventing virulence by expressing an appropriate RNAi construct in the host plant that targets the pathogen effect or gene or genes. During the interaction, the pathogen receives the transferred double-stranded RNA (dsRNA) or small interfering RNA (siRNA). Major mycotoxin-producing fungi, including *Aspergillus flavus*, *Fusarium*, and *Penicillium* species, have genes that are well known and play important roles in the growth, development, production of secondary metabolites, pathogenicity, and survival of fungi[21]. It also contains genes that code for universal and pathway-specific regulators of fungal secondary metabolism, growth, and stress response, in addition to the enzymes required to produce toxic secondary metabolites. Based on the success of earlier studies employing synthetic siRNAs to inhibit important fungal genes responsible for producing toxins in *Aspergillus* and *Fusarium*, a hairpin RNA-based transgenic RNAi approach to decrease mycotoxigenic fungus in plants appears feasible.

To demonstrate the in vivo interference phenomena in the pathogenic fungus *Fusarium verticillioides*, reported that transgenic tobacco plants express siRNAs from a dsRNA representing the specific fungal transgene[22]. Additionally, the RNAi approach to combating plant-pathogenic fungus has been described in two U.S. patent applications (Nos. 20060247197 and 20080022423). By generating transgenic rice that expresses particular dsRNA generated from *M. grisea* genes, they were able to determine resistance to rice blast infection. In comparison to control leaves, these transgenics displayed lower infection rates and lesion sizes. In the second invention, the tubulin, *vATPase*, and *Pac1* genes of *S. sclerotiorum*, which are crucial for the fungus' survival, were silenced in tobacco, *Arabidopsis*, and soybean. This resulted in the development of a resistance response to the fungus**.** Recent research demonstrated the successful application of HIGS in the rice-*R. solani* pathosystem and suggests that the ShB pathogen contains an RNA interference system. The PMK (pathogenicity map kinase) genes were targeted. The polygalacturonase (PG) gene has been silenced in *R. solani*. In the transgenic rice plant carrying the RNAi constructs, it has been shown that effective pathogen gene silencing reduces the ShB disease.

RNAi

It has been almost 20 years since RNA interference (RNAi) in the nematode *Caenorhabditis elegans* was discovered. It is now clear that many eukaryotes share a conserved mechanism for gene silencing mediated by double-stranded RNA (dsRNA). The expression of dsRNA or siRNA targeted against particular insect and fungal target genes in transgenic plants has made it possible to produce a new generation of pest-resistant crops (Fig. 4). This strategy has a bright future because it opens up a variety of possible targets for the inhibition of gene expression in fungi and insects[23].

An early, successful application of the RNAi system using sense and antisense RNA for the harmful fungus *Cryptococcus neoformans* was reported. *Venturia inaequalis*, *Magnaporthe oryzae, Histoplasma capsulatum, Phytophthora infestans*, and *Blastomyces dermatitidis* were also used to demonstrate the effectiveness of RNAi. The GFP gene was expressed in the fungus before being silenced by RNAi[24].

Several RNA silencing pathway components were examined, and it was discovered that many of them had an impact on *Verticillium*. It is hypothesized that Verticillium-specific defensive responses are regulated in part by gene silencing mechanisms[25]. A ground-breaking work on insect control using dsRNA feeding trials was revealed. It has been demonstrated that feeding insects plant material that expresses hairpin dsRNA constructs against specific target genes can silence their genes. They also offered proof of the possible application of RNA interference (RNAi) in crop defense to mitigate insect pests.

In India, Praveen and associates started applying the RNAi technique to agriculture in 2007. They employed a range of hairpin RNA-mediated strategies, including antisense, self-complementary inverted repeats, and intron-spliced hairpin RNAs, and they reported the stable in vivo silencing of the tomato leaf curl virus by the virus' antisense replicase gene (AC1). The ability of virally generated RNAi constructs to silence the tomato leaf curl virus-AC4 gene was reported, and it proposed that dsRNA with a longer arm capable of generating a pool of diced siRNAs is more effective in silencing genes[26]. Additionally, when the target gene is present in a pool, the impact of off-targeting siRNA is reduced. Recent research has demonstrated positive (HIGS) effects in rice-*R. solani* and suggests the use of an RNAi pathway strategy. Two cloned polygalacturonase genes (RsPG3 and RsPG4) are present in *R. solani*. They have been demonstrated to be one of the pathogen's primary virulence factors by causing rice sheath tissue necrosis and the ensuing release of reduced sugar[27]. It has recently been shown that the polygalacturonase (AG1IA_04727) gene is essential to the pathophysiology of *R. solani.*

RNAi machinery in fungi

The primary components of the RNAi machinery are RNA-Induced Silencing Complex (RISC), Dicer, Argonaute, and RDRP. These components are essential to the gene-silencing process. Various fungi, including ascomycetes, basidiomycetes, and zygomycetes, most of which have numerous RNA silencing components in the genome, have RNAi machinery. A variety of fungus species and fungus-like organisms have been used RNAi to investigate gene function. These discoveries show that the RNAi mechanism may exist in a broad sense even though RNA silencing pathways seem to have undergone significant diversification. Though, it is just the beginning, a series of research papers appeared in recent years providing a strong base for the use of this technology against fungal pathogens. Also reported that based on proteins or enzymes, RNAi pathways significantly vary amongst different fungal species and undergo a variety of adaptations throughout their evolutionary history[28]. However, most of the silencing studies were done through either transformation of fungi or *in vitro* approach (Fig. 4).

Genomic editing

The genome-editing tool is a more sophisticated kind of molecular biology method that enables precise, effective, and targeted alterations at genomic loci. The development of clustered, regularly interspaced short palindromic repeats (CRISPR)/Cas systems that provide simplicity and ease of targeted gene editing has recently come under the spotlight. CRISPR/Cas system is simple, efficient, low cost and it can target multiple genes. Genome editing using zinc-finger nucleases (ZFNs) and transcription activator-like effector nucleases (TALENs) has been around for twenty years. Due to its potential to effectively combat both abiotic and biotic stresses, CRISPR/Cas9 has been widely used in plants (Fig. 5). Fungi are responsible for several diseases, including sheath blight, smut, rust, mildew, and rot, which leads to heavy yield loss[29]. Downy mildew resistant (DMR) 6 in Arabidopsis thaliana is a membZher of the superfamily of 2-oxoglutarate Fe(II)-dependent oxygenases, and its overexpression increased vulnerability. Downy and powdery mildews in tomatoes cause significant monetary losses. In Arabidopsis thaliana, downy mildew resistant 6 (DMR6), a member of the subfamily of 2-oxoglutarate Fe(II)-dependent oxygenases, contributes to salicylic acid homeostasis and increases sensitivity to downy mildew132. Investigators adopted the CRISPR-Cas9 method to deactivate DMR6 orthology in tomatoes and established that, without significant harmful effects, DMR6 mutants showed disease resistance to different pathogens, including *Phytophthora capsica*,

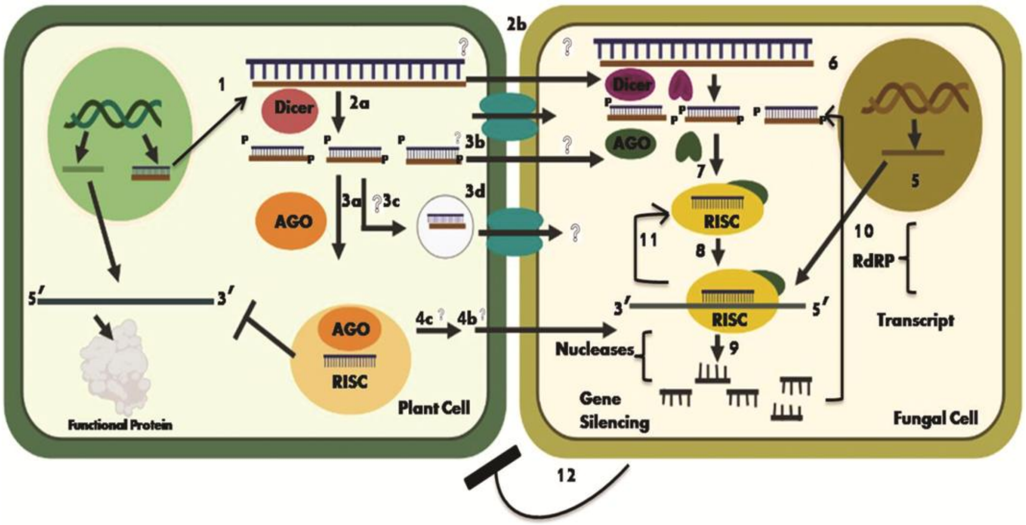

Fig. 4 — Schematic representation of the RNAi mechanism in Fungal. 1) Pathogen-specific double-stranded RNA generated in the host. 2) The fate of ds RNA a: Small siRNA duplexes produced from dsRNA by the action of host Dicer b: The long dsRNA is transported into the fungi cell and processed by fungal dicer to produce small siRNA. 3) a: Host-generated siRNA is processed by host silencing machinery and form the RISC complex b, c &d: Host-generated siRNA is transferred/transported to fungal cells and processed by pathogen Argonautes and formed an RNA-induced silencing complex (RISC). 4) a & b: Transport the guide RNA from the host to a pathogen. 5) Transcription of fungal DNA resulted in the functional mRNA. 6, 7 & 8) Processing of small RNA (obtained from both host and fungi) and form the RISC complex. 9) Cleavage of the fungal transcript by RISC complex-mediated through nucleases and inhibits the translation. 10 & 11). The components of cleaved mRNAs can be regenerated into siRNA duplexes through RNA-dependent RNA-polymerase

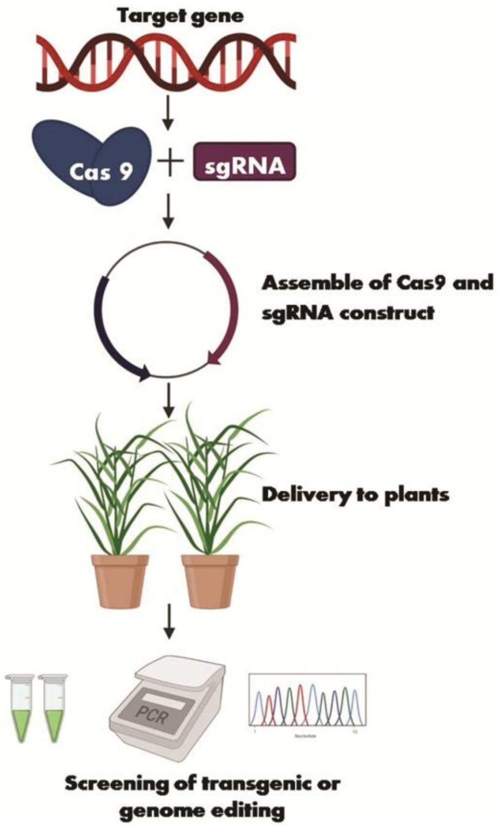

Fig. 5 — Generating a CRISPR/Cas-9- mutagenized plant line (Pathogen /insect resistance)

Pseudomonas syringae, and *Xanthomonas* spp. Mildew in A against *P. syringae*. The CRISPR-Cas9 method was used to develop tomato dominant JAZ2 repressors devoid of the domain connected to the C-terminal jasmonate (Jas) JAZ2Δjas. It is conceivable to adopt a CRISPR-Cas9-based fruit crop protection strategy in the field since these JAZ2Δjas repressors confer resistance to *P. syringae*[30]. This approach has been used to edit various crops, including rice, maize, wheat, soybean[31], watermelon, grapes, barley, sorghum, Camelina, tomato, flax, rapeseed, apple, cotton[32], lettuce, grapefruit, oranges, cucumber, and potato[13]. Genome editing plays an important role in increasing production and helps to meet the global demand for food.

Conclusion

R. solani Kühn is a significant group of soil-borne basidiomycete pathogens that affect a variety of plant species. The disease known as sheath blight can infect rice at the seedling, tillering, and booting stages, posing a serious threat to rice farming worldwide. Because commercial rice cultivars lacked the desired genetic resistance to this pathogen, researchers made great efforts to create sheath blight cultivars through breeding and transgenic techniques. Sadly, the efforts are ineffective due to a lack of a solid understanding of the molecular pathways involved in the onset and progression of disease. In situations where pathogen biology is fully understood, resistance breeding has proven more effective. This aids in the synthesis of chemical compounds that are more powerful when used as fungicides to suppress the infection. Through the use of genomes, transcriptomics, and proteomics techniques, attempts to study pathogen biology have increased recently. It is now easier to produce large genomic resources that can be examined to identify genetic variations in defense-related genes, which can then be further confirmed for a strong correlation with disease resistance. This is made possible by the advancement of sequencing technology and the reduction of costs. In this way, the secret treasure of sheath blight resistance could also be found by utilizing all landraces, farmers' varieties[33], and wild relatives. To achieve effective and affordable disease treatment, further approaches like CRISPR-mediated knock-out and the exploitation of recent alternative technologies like RNAi technology should be incorporated.

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Conflict of interest

There is no conflict of interest.

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