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Cytomolecular aspects of rice sheath blight caused by *Rhizoctonia solani*

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Abstract Sheath blight, caused by anastomosis group 1-IA of *Rhizoctonia solani* Kühn (teleomorph *Thanatephorus cucumeris* (Frank) Donk), is one of the most destructive rice diseases worldwide. The pathogen is able to infect plants belonging to more than 27 families, including many economically important monocots and dicots such as rice, wheat, alfalfa, bean, peanut, soybean, cucumber, papaya, corn, potato, tomato and sugar beet. It is a soil borne necrotrophic fungus that survives in plant debris as sclerotia, which are small brown-to-black, rocklike reproductive structures. The sclerotia can survive in the soil for several years and infect rice plants at the water-plant interface in the flooded field by producing mycelia. Management of rice sheath blight requires an integrated approach based on the knowledge of each stage of the disease and cytomolecular aspects of rice defence responses against *R. solani*. This review summarizes current knowledge on molecular aspects of *R. solani* pathogenicity, genetic structure of the pathogen populations, and the rice-*R. solani* interaction with emphasis on cellular and molecular defence components such as signal transduction pathways, various plant hormones, host defence genes and production of defence-related proteins involved in basal and induced resistance in rice against sheath blight disease.

Key words Genetic variability · *Oryza sativa* · Pathogenesis-related proteins · Signaling · *Thanatephorus cucumeris*

Abbreviations

IR	induced resistance
MAPK	mitogen activated protein kinase
PRs	pathogenesis-related proteins
SA	salicylic acid
JA	jasmonic acid

Introduction

Sheath blight, caused by *Rhizoctonia solani*, is an important fungal disease of rice ranking only after blast and often rivalling it (Banniza and Holderness 2001). The teleomorph of *R. solani*, *Thanatephorus cucumeris*, is classified in kingdom Fungi; subkingdom Eumycota; phylum Basidiomycota; class Heterobasidiomycetes; order Ceratobasidiales; and Family: Ceratobasidiaceae (Webster and Weber 2007). The disease occurs through rice production areas of the world, in both tropical and temperate climates, and it is most damaging in intensive protection systems. Widely adopted changes in rice cultivation that support disease development have helped to make sheath blight

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destructive and important worldwide. The damage caused by the disease has increased in rice-growing regions since the introduction of high-yielding compact semi-dwarf cultivars and the application of high levels of nitrogen fertilizers in rice fields. The main symptoms of sheath blight are lesions on the sheaths, at the base of the plants, or close to the water line in irrigated crops. The size, shape, and colour of the lesions may vary in different environmental conditions. Initially, the symptoms are green-gray, water-soaked lesions on the sheaths. The lesions can expand and the centres of the lesions might become pale green to white that are surrounded by an irregular purple-brown margin. The sheath blight lesions can expand rapidly on the plant, causing the corresponding leaf blade to wilt and die within a few days. Sheath blight triggers a number of damage mechanisms including a reduction in chlorophyll content and loss of phothosynthetically active leaf area due to the establishment of lesions on both leaf blades and leaf sheaths (Damodaram Nadu et al. 1981). Furthermore, an alteration of photosynthetic and respiratory processes occurs in the green tissues due to the activities of enzymes released by the pathogen, and accelerated senescence of leaves due to the wide array of lytic enzymes and phytotoxin produced by such an efficient necrotrophic pathogen (Savary and Mew 1996).

Control of rice sheath blight is difficult due to the low inherent level of resistance of rice cultivars against this disease, wide host range of the pathogen, its ability to survive in soil for a long time, and its high genetic variability (Taheri et al. 2007). Although partial genetic resistance to sheath blight in rice has been reported, no major gene responsible for resistance has been found (Kumar et al. 2003), and rice sheath blight is not efficiently controlled by resistance breeding. Most of the traditional cultivars, planted on over 90% of the rice-growing areas, are susceptible to disease. Therefore, for planning the best disease management strategies, it is necessary to understand the biology and genetic structure of the pathogen populations, cellular and molecular mechanisms involved in *R. solani*-rice interaction, and signal transduction pathways affecting basal and induced resistance in rice against this destructive pathogen.

Here, we present a concise and up-to-date review about the biology and genetic variability of *R. solani*, cytomolecular aspects of pathogenicity, and the interaction between *R. solani* and rice by reference to past literature and recent research.

Taxonomic history

The genus concept in *Rhizoctonia* was first established in 1815 by De Candolle (Sneh et al. 1991). The inclusion of a given taxa to the genus *Rhizoctonia* was based for a long time on the possession of certain vegetative characters such as brown pigmented hyphae, constrictions at branch points forming right angles, and absence of mitospores. Together with these characteristics, which are always present, there are other characteristics not constantly present in the entire species complex, but present in a large number of species, including the presence of moniloid cells and sclerotia in culture, fast rates of hyphal growth, and a complex dolipore septal apparatus.

The most important species of *Rhizoctonia*, *R. solani,* was originally described by Julius Kühn on potato in 1858 and is the most widely documented and the most important and destructive species of *Rhizoctonia* (Ogoshi 1996). In nature, usually *R. solani* has asexual reproduction and exists primarily as vegetative mycelium and/or sclerotia. The teleomorph of *R. solani*, *Thanatephorous cucumeris*, is classified in Basidiomycota (Webster and Weber 2007). *R. solani* is considered as a species complex rather than a single species (Adams 1988; Anderson 1982). The species complex has been divided into various homogeneous groups based on hyphal anastomosis. Hyphae of *R. solani* isolates can only anastomose with each other if they are within the same anastomosis group (AG) (Anderson 1982; Carling 1996; Sneh et al. 1991). Isolates of *R. solani* have been divided into 14 AGs designated as AG 1 through 13 and a bridging isolate (BI) group (Carling 1996; Carling et al. 2002). Several AGs are further subdivided into intraspecific groups (ISGs) based on cultural morphology, nutritional requirements, temperature effect on growth, host specificity, frequency of hyphal anastomosis, and pathogenicity (Sneh et al. 1991). Isolates of *R. solani* AG 1 have been subdivided into three subgroups including IA, IB, IC based on the size and shape of sclerotia and DNA base sequence homology (Sneh et al. 1991).

The causal agent of rice sheath blight was described for the first time by Yano (1915) as *Pellicularia sasakii* (Shirai) S. Ito (Hashiba et al. 1972). It has also been reported in the literature as *Corticium sasakii* (Shirai) Matsumoto, *C. vagum* Berk. & Curt., and *Hypochnus sasakii* Shirai (Lee and Rush 1983; Ogoshi 1987).

Researchers now generally accept *R. solani* AG1-IA as the sheath blight pathogen. The classification as *R. solani* AG1-IA is supported by morphological characterization (Sneh et al. 1991; Taheri et al. 2007), analysis of ribosomal DNA-internal transcribed spacer (rDNA-ITS) sequences (Guillemaut et al. 2003; Johanson et al. 1998; Taheri et al. 2007), and surveys on infection structures (Marshall and Rush 1980a, b; Eizenga et al. 2002). It is known that not only *R. solani* AG1-IA, but also isolates of IB and IC subgroups of AG1 are capable of causing sheath blight symptoms on rice (Taheri et al. 2007). However, AG1-IA subgroup is the predominant causal agent of sheath blight reported from rice growing regions worldwide (Bernardes-De-Assis et al. 2009; Gonzalez-Vera et al. 2010; Taheri et al. 2007).

Life cycle and epidemiology

The pathogen survives in soil within diseased plant material as mycelia or sclerotia during unfavourable environmental conditions for several years. It is transported in infested soil, irrigation water, or through movement of diseased plant tissues. Potential for seed-borne inoculum also exists. In favorable environmental conditions (high humidity and ±30°C), the pathogen is able to spread as mycelial strands from the lower parts to the upper parts of the plant, and also from one tiller to another in one plant, or between neighbouring plants. The rate of spread of the mycelial strands, and subsequently disease progress, can be very fast, as the horizontal spread of the disease has been estimated up to an average of 20 cm/day under field conditions (Savary et al. 1995).

Following infection of the rice plant by *R. solani*, sexual spores are formed on specialized structures called basidia. Four spores are produced on each basidium. Basidia are formed when enough moisture is available and sufficient growth of the fungus has occurred. Formation of basidiospores on diseased host plants in nature is rarely observed. Although basidiospores are wind-borne, their role in initiating disease has not been considered important in the case of sheath blight, but it can be important for foliar diseases such as web blight on bean under conditions of high humidity (Sneh et al. 1991). Each basidiospore has a single nucleus. Basidiospores can germinate only in high moisture and the hyphae produced by germinating these spores will fuse or anastamose with each other to form new hyphae with a mixture of different types of nuclei. When a host plant is in contact with the pathogen and environmental conditions are favourable, *R. solani* begins to colonize the surface of the host plant with long, unbranched hyphae called runner hyphae. Depending on the isolate and host species, the runner hyphae may branch and penetrate through stomata, produce structures called lobate appressoria, which are swollen hyphal tips, or produce infection cushions, which are aggregates of complex hyphae (Fig. 1). Infection cushions of *R. solani* form most frequently along the edges of the vascular bundles or over stomata. The infection cushions are involved in enzymatic degradation and physical penetration of the leaf surface, providing entrance into the plant for the pathogen (Groth and Nowick 1992).

The disease develops most rapidly during early heading and grain filling. Therefore, an accelerated senescence of tillers at these growth stages may lead to the destruction of potentially fertile tillers (Savary and Mew 1996). The widespread adoption of susceptible semi-dwarf cultivars, and the changes in cultural practices associated with these cultivars, favour development of the disease and have contributed to its rapid increase and high levels of yield losses in rice-growing areas. Because of the high yield of semi-dwarf compact rice cultivars, they need high soil fertility, particularly in nitrogen. The denser plant canopy of these cultivars creates a favourable microclimate with high humidity and enhances sheath blight development (Slaton et al. 2003; Taheri and Tarighi 2010).

Infection process

Histological studies of Marshall and Rush (1980a) first described the process of *R. solani* infection and development of sheath blight symptoms on rice. The pathogen survives as sclerotia on plant debris or soil for long periods. In favourable conditions, the sclerotia are capable of germination and formation of mycelia. After contact with the rice plant surface, the mycelia can growth and produce infection structures such as infection cushions (Fig. 2a and c) and/or lobate appressoria (Fig. 2b and d) which directly penetrate into the plant tissues by penetration

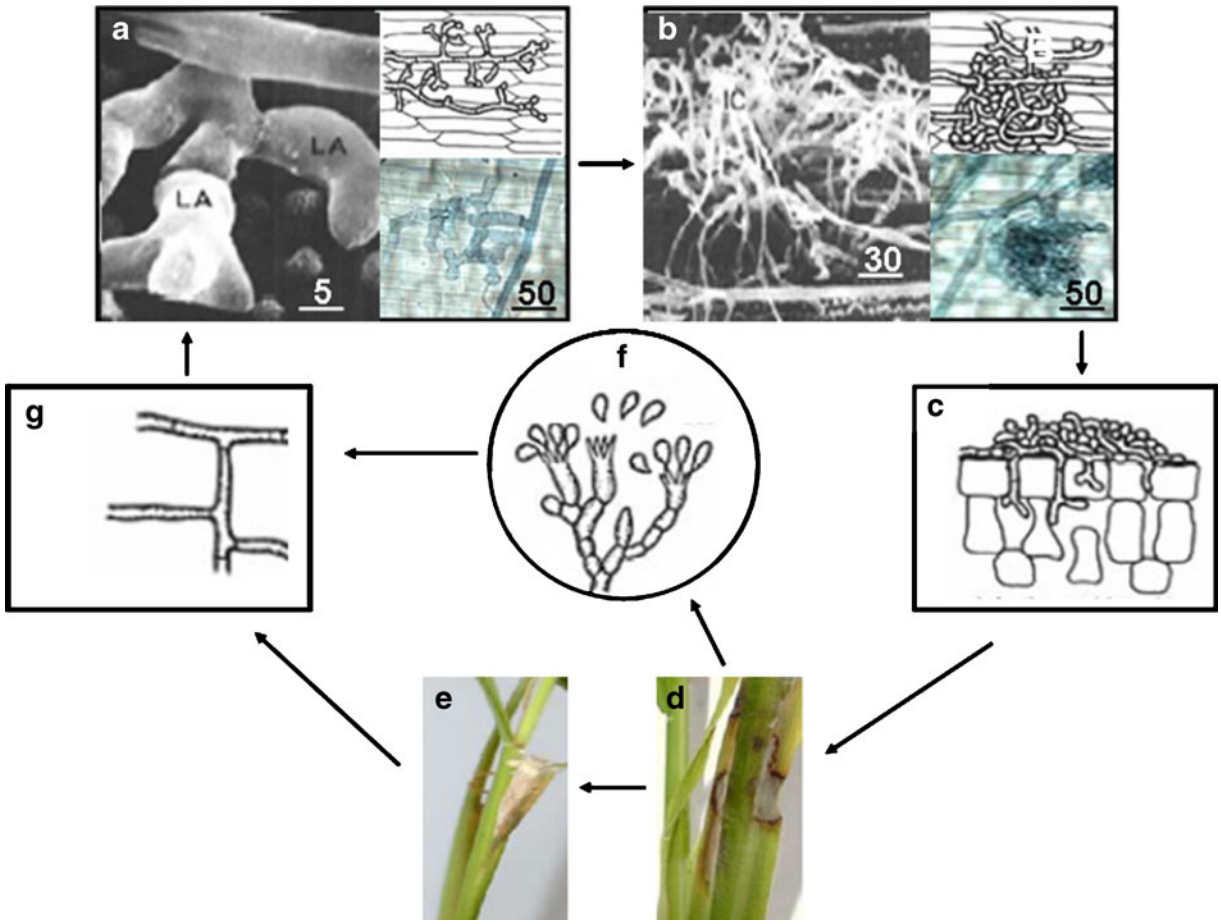

Fig. 1 Life cycle of *Rhizoctonia solani*. **a** Lobate appressoria and **b** infection cushions on rice leaf. **c** Infection cushion invading host tissue by formation of infection hyphae. **d** sheath blight symptoms on rice sheaths. **e** Formation of white circular sclerotia on infected rice plant which turn to brown in maturity. **f** Basidiospores, which are rarely observed in the nature, produced on infected tissue in favourable environmental conditions. **g** Germination of sclerotia or basidiospores leads to formation of mycelia with right angles which infects the host tissue. The black and white cycle is obtained from http://apsnet.org/education/lessonsPlantPath/Rhizoctonia/discycle.htm. Calibration bars are in micrometers

pegs (Fig. 2d). Stomatal penetration is infrequent and no infection structures are observed in this case (Marshall and Rush 1980b). Development of disease symptoms on the infected plant can be observed within 24–72 h after infection depending on the environmental conditions.

The penetration activities and lesion formation by *R. solani* are controlled via several factors. It is known that infection cushion formation can be due to a contact stimulus with the host surface, and it can be induced by host exudates. The results obtained by Marshall and Rush (1980a) revealed that the growth of *R. solani* on the rice sheath surface and formation of infection structures by this pathogen were not controlled by a contact stimulus. They made surface replicas of outer sheath surface of rice cultivars which were resistant or susceptible to the sheath blight pathogen. The infection structures were not formed on the replicas, suggesting no effect of contact stimulus in their formation.

The sheath blight resistance of rice cultivars is known to be highly correlated with the number and type of infection structures produced by the pathogen on the cultivar (Marshall and Rush 1980a). On resistant rice cultivars, only formation of lobate appressoria was observed; whereas on susceptible and intermediate cultivars, both infection cushions and lobate appressoria are formed (Marshall and Rush

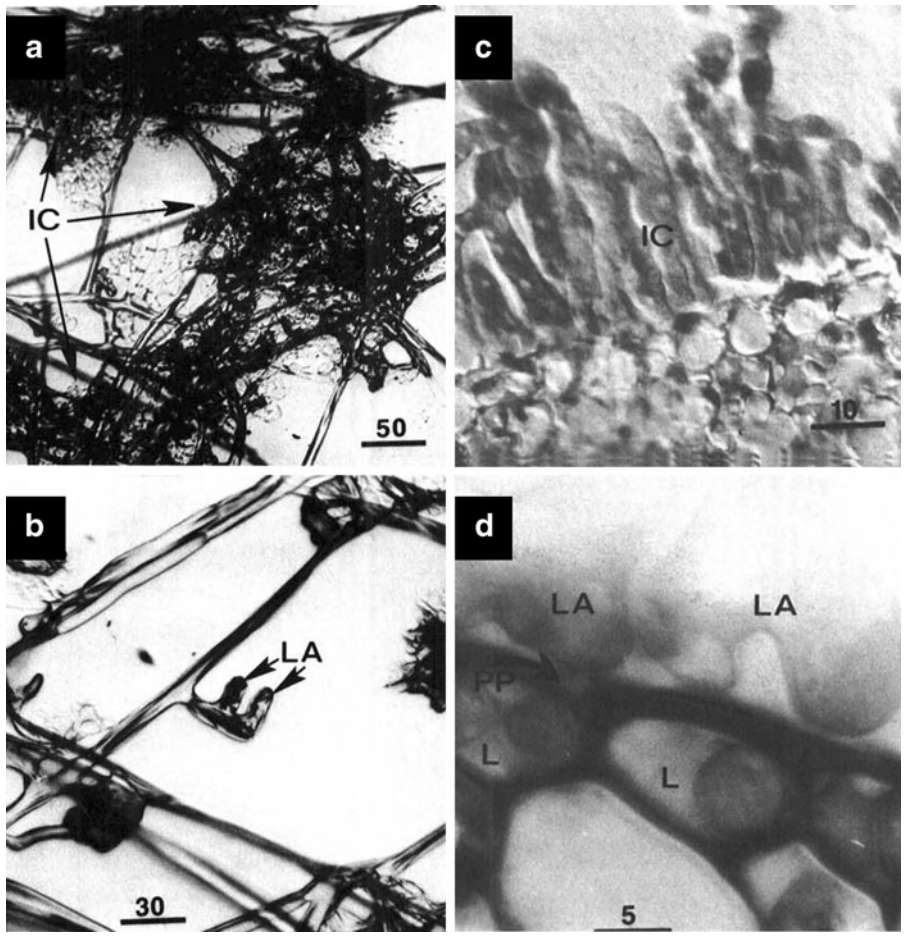

Fig. 2 Development of *Rhizoctonia solani* on artificial surfaces (**a** and **b**) and rice sheath surfaces (**c** and **d**). **a** *R. solani* infection cushions and **b**, lobate appressoria, produced on collodion membrane over rice sheath surface. **c** and **d** cross sections through **c**, infection cushions and **d**, lobate appressoria. **c** Showing cushion cells perpendicularly oriented to the sheath surface. **d** Showing a penetration peg and enlargement of hyphae in a cell lumen. *IC* infection cushion; *L* cell lumen; *LA* lobate appressorium; *PP* penetration peg. Calibration bars are in micrometers. Pictures in **a** and **b** are obtained from Marshall and Rush 1980a; **c** and **d** from Marshall and Rush 1980b

1980a). Formation of infection structures is controlled by the host exudates. Nutrition of *R. solani* on host tissue prior to penetration plays an important role in formation of infection structures and finally on pathogenesis. It has been demonstrated that exogenous application of both glucose and 3-O-methylglucose (MEG) reduce lesion development on rice plants. This reduction is correlated with the inhibition of infection cushion formation on various rice cultivars (Marshall and Rush 1980a; Weinhold and Bowman 1974).

A host-specific phytotoxin produced by *R. solani*, designated RS toxin, is well-known to be responsible for producing the sheath blight symptoms. The RS toxin appears to be a carbohydrate containing N-acetylgalactosamine, N-acetylglucosamine, glucose, and mannose. Virulence of *R. solani* isolates is correlated with the amount of toxin produced by each isolate, and the toxin is demonstrated to be in infected rice leaves (Vidhyasekaran et al. 1997). Investigating the association of toxin reaction with sheath blight progress revealed that there is a correlation between toxin sensitivity and disease susceptibility in various rice cultivars. Sensitivity to the phytotoxin has been shown to be conferred by two dominant genes (Brooks 2007). A map-based cloning strategy could be used to identify each sensitivity gene. This strategy would be an advancement toward mapping sheath blight resistance genes that previously were scored as quantitative trait loci (QTL). Also, the toxin is useful to identify detoxification proteins from plants and microorganisms. The studies of Shanmugam et al. (2001) revealed that the RS toxin could be inactivated using an extracellular protein, known as α-glucosidase enzyme, purified from a biocontrol fungus *Trichoderma viride*. The gene that codes this enzyme could be exploited to develop transgenic rice plants with enhanced resistance against sheath blight.

Molecular aspects of pathogenicity

Currently, very little is known about molecular aspects of *R. solani* pathogenicity on rice. The scarcity of molecular information on pathogenicity can be related to the relatively large genome size of the pathogen, estimated to be 37–71.5 Mbp (Cubeta et al. 2009; Keijer et al. 1996) and the lack of an efficient molecular transformation system. Isolates of *R. solani* have at least 11 chromosomes ranging in size from 0.6 to 6 Mbp (Keijer et al. 1996), but the location of pathogenicity genes on the chromosomes is not known yet. The genome sequence of *R. solani* AG1-IA, causing rice sheath blight, is not known yet. However, despite a late start, genome sequencing of *R. solani* AG3 is in progress (Cubeta et al. 2009). This fungus is an important pathogen of food crops in the plant family Solanaceae. The resulting databases will allow the comprehensive analysis of developmental processes that are characteristic of this fungus, including the molecular nature of pathogenicity. DNA databases support analyses of the fungal transcriptome, proteome, and metabolome. This combined information will contribute to our basic understanding of not only the mechanisms of infection but also the evolution of *R. solani* pathogenicity (Yoder and Turgeon 2001).

Fungi inevitably respond to extracellular signals or stimuli via a wide array of transduction pathways for pathogenicity. One of the most studied pathways in the filamentous fungi is the signalling cascade mediated by membrane-bound heterotrimeric G proteins, composed of Gα, Gß, and Gγ subunits (Li et al. 2007; Wendland 2001). The Gα subunit containing intrinsic GTPase activity is the key step in controlling the cellular response via the G protein signal transduction pathway. Upon receiving extracellular stimuli, a G protein-coupled receptor (GPCR) interacts with the G protein, inducing replacement of GDP in the Gα subunit by GTP which leads to dissociation of Gα from Gß and Gγ subunits. The released Gα subunit becomes activated and in turn regulates downstream effectors, such as adenylate cyclase, phospholipase, ion transporters, and mitogen activated protein kinase (MAPK) involved in numerous biological processes including pathogenicity (Neves et al. 2002).

Charoensopharat et al. (2008) demonstrated the biological function of the Gα subunit gene, *Rga1*, in the rice sheath blight pathogen by target gene disruption. The deduced primary structure of the *Rga1*-encoded protein of *R. solani* showed high identity to those of Gα subunits from other filamentous fungi. Disruption of *Rga1* led to decreased vegetative growth and pathogenicity of the rice sheath blight pathogen. The *Rga1* disruptant showed altered colony morphology. In addition, the sclerotia formation ability of the disruptant was completely lost. Therefore, they concluded that Gα subunit encoded by *Rga1* is involved in a signal transduction pathway in *R. solani* that controls growth, development and pathogenicity (Charoensopharat et al. 2008). Also, similar results have been observed for the genes encoding G protein subunits in other phytopathogenic fungi, such as *gpa3* in *Ustilago maydis* (Regenfelder et al. 1997), *cpg1* in *Cryphonectria parasitica* (Gao and Nuss 1996), and *fga1* in *Fusarium oxysporum* (Jain et al. 2002). Disruption of *fga1* in *F. oxysporum* causes a lower level of cAMP and reduction of pathogenicity (Jain et al. 2002). In *Cryphonectria parasitica*, however, disruption of Gα gene, *cpg1,* leads to an increase in cAMP level and loss of pathogenicity (Gao and Nuss 1996). Therefore, it seems that alteration of intracellular cAMP level, whether it is an increase or decrease in each fungal pathogen, is accompanied by reduction or loss of fungal pathogenicity. It is still unknown if the cAMP level is increased or decreased in the *Rga1* disruptant of *R. solani*, but it is possible that a changed cAMP level in the *Rga1* disruptant results in reduced pathogenicity. Further analysis of downstream effector molecules of this signal transduction pathway may provide a wider insight into molecular mechanisms correlated with *R. solani* pathogenicity.

Genetic structure of the pathogen populations

Analysis of population structure and genetic diversity in *R. solani* AG1-IA is important for understanding its ecology, pathology, and host specificity. Therefore, the knowledge of genetic variability within and among various populations of this phytopathogenic fungus will be useful in management of sheath blight disease. Isolates of AG1-IA associated with rice sheath blight have been the subject of different diversity and population studies, in which variation has been measured using intra-

and extracellular enzymes and proteins (Liu & Sinclair 1993; Matsuyama et al. 1978; Neeraja et al. 2002a), cellular fatty acids (Stevens Johnk and Jones 1994), and various molecular techniques such as restriction fragment length polymorphism (RFLP) (Banniza et al. 1999; Rosewich et al. 1999), simple sequence repeat polymerase chain reaction (SSR-PCR) or microsatellites (Banniza and Rutherford 2001; Bernardes-De-Assis et al. 2009; Gonzalez-Vera et al. 2010), random amplified polymorphic DNA (RAPD) markers (Neeraja et al. 2002b), repetitive element PCR (Rep-PCR) (Linde et al. 2005), and amplified fragment length polymorphism (AFLP) (Taheri et al. 2007). Previous investigations support the hypothesis that isolates of AG1-IA causing rice sheath blight display high genetic diversity (Banniza and Rutherford 2001; Rosewich et al. 1999). In a population genetic study of *R. solani* AG1-IA from India that was based on RFLP and Rep-PCR, results were consistent with small genetic distances among populations and high levels of gene flow (Linde et al. 2005).

Despite these studies, relatively little is known about genetic variability within populations, particularly among isolates of different ISGs of *R. solani* AG1 infecting rice. Analysis of molecular variance (AMOVA) using AFLP data revealed that the geographic region is the dominant force influencing the genetic structure of *R. solani* AG1-1A infecting rice, whereas host cultivar did not account for genetic variation (Taheri et al. 2007). In particular, pathogen populations should be monitored to determine if new genotypes have been introduced into a region over time. However, understanding of disease epidemiology, host-pathogen interactions, and subsequently successful management of sheath blight disease is really dependent on our knowledge concerning variability of the pathogen populations and the factors affecting genetic structure of these populations.

Genetics of *R. solani-Oryza sativa* interaction

The interaction of *R. solani* and rice plant has been investigated in various specific areas, two of which are discussed here including the role of defence-related proteins and signal transduction pathways activated in plant cells.

Defence-related proteins

Plants exhibit a variety of responses during infection by pathogens, insects, or abiotic stresses, many of which involve the activation of host defence genes. Activation of these genes leads to physical and biochemical changes in plant cells which are not favourable for damage progress in plant. Among the major biochemical changes is biosynthesis and the accumulation of inducible defence-related proteins. Most of these proteins correspond to pathogenesis-related proteins. These proteins are mostly of low molecular weight, preferentially extracted at low pH, resistant to proteolysis, and localized predominantly in the intercellular spaces of leaves. At first, the term "pathogenesis-related proteins" was abbreviated as PR-proteins to designate the "proteins coded by the host plant but induced only in pathological or related stress situations" (Van Loon et al. 1994; Van Loon and Van Strien 1999; Jayaraj et al. 2004). Then, it became a collective term for all microbe-induced proteins and their homologues to the extent that enzymes such as phenylalanine ammonia-lyase (PAL), peroxidase, and polyphenoloxidase, which are generally present constitutively and only increase during most infections (Van Loon et al. 2006) are included. Therefore, these defence-related proteins are often also referred to as PR-proteins (PRs). All PRs are of particular interest because they are part of the host plant's defence system.

On the basis of amino acid sequence data and biochemical functions, PR-proteins have been classified into 17 families so far (Van Loon et al. 2006). Not all families of PRs have been identified in each plant species. For instance in rice, only PR-1, -2, -3, -4, -5, -9, and -10 are known, suggesting that various plant species differ in the types of PRs expressed upon infection or stress situations (Table 1). The role of PR genes in rice sheath blight resistance has been suggested not only by the tight correlation between expression levels of PR genes and disease resistance, but also by the observation of enhanced resistance in the transgenic rice plants overexpressing certain PR genes. Therefore, we will continue this section with the following sub-headings: *involvement of rice PR genes in defence responses against R. solani*, and *transgenic rice overexpressing PRs or other antifungal peptides.*

Table 1 Recognized families of pathogenesis-related proteins in rice

Family	Gene name	Properties	Induced by	References
PR-1	*OsPR1a*, *OsPR1b*	Unknown	*Magnaporthe grisea, Rhizoctonia solani,* wounding	Agrawal et al. 2000a, b; Agrawal et al. 2001; Mitsuhara et al. 2008; Zhao et al. 2008
PR-2	*Gns1*	ß-1,3-glucanase	ethylene, cytokinin, salicylic acid, wounding, fungal elicitors (derived from *Sclerotium oryzae* or *Saccharomyces cereviseae*) *Rhizoctonia solani*	Anarutha et al. 1996; Simmons et al. 1992
PR-3	*RC24, RCH10, Rcht2*	Chitinase	*Rhizoctonia solani,* wounding	Anarutha et al. 1996; Kim et al. 1998; Xu et al. 1996; Zhu and Lamb 1991, Agrawal et al. 2003b
PR-4	*OsPR4 OsPR4-b*	Chitinase	jasmonic acid, abscisic acid, *Magnaporthe grisea*	
PR-5	*Pir2*	Thaumatin-like	*Rhizoctonia solani*	Reimmann and Dudler 1993; Velazhahan et al. 1998
PR-9	*POX8.1,*	*POX22.3, POC1*	Peroxidase	*Xanthomonas oryzae* pv. *Oryzae, Magnaporthe grisea, Rhizoctonia solani,* Riboflavin
	Chittoor et al. 1997; Hilaire et al. 2001;	Manandhar et al. 1999; Schweizer et al. 1997; Taheri and Hofte 2006		
PR-10	*PR10a, PR10b, PR10c*	Ribonuclease	*Magnaporthe grisea,* Jasmonic acid	McGee et al. 2001; Kim et al. 2003b, 2004, 2008

Involvement of rice PR genes in defence responses against *R. solani*

Differential induction of some of PRs has been investigated during infection of rice with the sheath blight fungus. Several studies revealed the increased expression of PR-1, 2, -3, -4, -5, -9 genes in rice plants after *R. solani* inoculation (Table 1). These findings suggest the involvement of PRs in basal defence responses of rice against this pathogen. As is shown in Table 1, the PR-2 family comprises ß-1,3-glucanases that can exert their antifungal activity in at least two different ways: either directly by degrading the cell walls of the fungal pathogen or indirectly by promoting the release of cell wall degradation products that can act as elicitors to trigger a wide range of defense responses (Leubner-Metzger and Meins 1999).

The PR-3 and -4 families are comprised of chitinases. They hydrolyze the ß-1,4 linkages between *N*-acetylglucosamine residues of chitin, a structural polysaccharide of the cell wall of many fungi, such as *R. solani*. The enzyme is linked with the thinning of the growing hyphal tips of fungi, followed by a balloon-like swelling that eventually leads to a bursting of hyphae. Furthermore, the degradation products of the fungal cell wall, especially the oligomers, could serve as resistance elicitors (Muthukrishnan et al. 2001). A combination of chitinase and -1,3-glucanase is known to be more effective than each enzyme alone against many fungi (Jayaraj et al. 2004; Van Loon et al. 2006).

The PR-5 or thaumatin-like proteins (TLPs) have a high degree of sequence similarity with each other and show immunological relationship with the sweet-tasting protein thaumatin, found in fruits of the West African shrub *Thaumatococcus daniellii* (Cornelissen et al. 1986). At high concentrations, TLPs can actively lyse fungal membranes, while at low concentrations, they affect membrane permeability which can cause leakage of cell constituents and increase the uptake of other antifungal compounds. There are two classes of TLPs in plants. The larger class includes proteins with a size of about 22–26 kDa, and members of the smaller class are about 16–17 kDa.

Rice has both classes of inducible TLPs; one is a 16 kDa TLP induced by *Pseudomonas syringae* (Reimmann and Dudler 1993). Infection of rice plants with the sheath blight pathogen resulted in the induction of two TLPs of 24 and 25 kDa within one to two days after infection (Velazhahan et al. 1998).

The PR-9 or peroxidases are key enzymes in the cell wall-building process, and it has been suggested that extracellular or wall-bound peroxidases would enhance rice resistance against various pathogens by the construction of a cell wall barrier that may hamper pathogen ingress and spread in plant cells. The PR-9 group contains a specific type of peroxidase that could act in cell wall reinforcement by catalyzing lignification (Passardi et al. 2004; Van Loon et al. 2006) and enhance resistance against multiple pathogens. For instance, POC1 is a cationic pathogen-induced peroxidase which is grouped in PR-9 family. Upregulation of the *POC1* gene has been observed not only after infection of rice plants by *R. solani* (Taheri and Höfte 2006), but also after infection with *Xanthomonas oryzae* pv. *oryzae,* causing bacterial leaf blight disease (Hilaire et al. 2001). Genes for peroxidases were also induced by infection with the rice blast fungus *Magnaporthe grisea* (Manandhar et al. 1999; Schweizer et al. 1997). Furthermore, the increased activity of ß-1,3-glucanase (PR-2) and upregulation of the corresponding gene have been observed in *R. solani*-infected and also in *Sclerotium oryzae*-infected rice plants (Simmons et al. 1992; Anarutha et al. 1996). Therefore, there is an overlap in rice defence mechanisms against various fungal and bacterial pathogens with different life cycles. This suggestion is also made by Zhao et al. (2008), who examined the expression of 100 rice genes induced by infection with *Magnaporthe oryzae*, *Xanthomonas oryzae* pv. *oryzae*, and *X. oryzae* pv. *oryzicola.* They found that 25 out of 100 genes were differentially expressed after sheath blight infection, indicating the overlap of rice defence responses to various fungal and bacterial pathogens.

Most PRs are induced through the action of the signalling compounds salicylic acid (SA), jasmonic acid (JA), or ethylene (ET), and possess antimicrobial activities in vitro through hydrolytic activities on cell walls, contact toxicity, and perhaps an involvement in defence signal transduction pathways in various pathosystems. In addition, induction of PRs is known to be associated with induced resistance, a phenomenon by which plants exhibit enhanced levels of resistance against pathogen attack by priming an efficient network of defence mechanisms that can be triggered by various biotic or abiotic stimuli (Ahn et al. 2005; Dong and Beer 2000; Taheri and Höfte 2006). This priming phenomenon results in a faster and stronger defence activation at the moment the plant is attacked (Conrath et al. 2006). In rice-*R. solani* pathosystem, exogenous application of the chemical priming agent riboflavin (vitamin B_2) has been demonstrated to induce resistance and activate defence responses in both intact and detached rice leaves in a manner similar to JA. Riboflavin and JA triggered various defence mechanisms, including post-invasive penetration mechanisms, such as lignin formation in the plant cells via involvement of defence related proteins including PRs. Riboflavin-induced resistanse in the pathosystem has been shown to be correlated with upregulation of *PAL* and *POC1* genes which leads to lignification in plant cells around the infection site and prevents further pathogen invasion (Taheri and Höfte 2006; 2007; Taheri and Tarighi 2010). A clear understanding of the roles of defence-related proteins would be possible through genetic engineering of plants, either by knocking out or by overexpressing corresponding genes in transgenic plants.

Sheath blight resistance in transgenic rice expressing PRs or other antifungal peptides

Genes encoding plant-derived PRs, antimicrobial peptides such as lectins and ribosome-inactivating proteins, and cell wall hydrolyzing enzymes of microbial origin have been used to develop transgenic plants with resistance to fungal pathogens (Punja 2006). Transgenic rice lines with increased resistance to sheath blight are listed in Table 2.

Antifungal protein genes of microbial origin offer a good resource of candidate genes for generating resistant transgenic plants (Lorito and Scala 1999). *Trichoderma* spp. are avirulent plant symbionts which are capable of parasitizing other fungi and serve as effective biocontrol agents against many phytopathogenic fungi including *R. solani* (reviewed by Harman et al. 2004). The 42 kDa endochitinase of *Trichoderma* is the key enzyme contributing to lysis of fungal cell wall (Lorito and Scala 1999). Purified 42 kDa endochitinase, obtained from *T. harzianum*, provided strong in vitro inhibition of *R. solani* and *R.*

Table 2 Transgenic rice lines with enhanced resistance to sheath blight.

Rice cultivar[a]	Gene name	Gene origin	Enzyme activity	Resistant to	Reference
Pusa Basmati1 (PB1)	*cht*42	*Trichoderma virens*	chitinase	*Rs*[b]	Shah et al. 2009
PB1	*Chi*11	rice	chitinase	*Rs*	Lin et al. 1995; Sridevi et al. 2003,
PB1	*Chi*11 and *gluc*	Rice, tobacco	Chitinase, ß-1,3-glucanase	*Rs*	Sridevi et al. 2008
JinHui35	*McCHIT1*	bitter melon	chitinase	*Rs*, *Po*[c]	Li et al. 2009
Chinsurah Boro II, IR72, IR51500	*tlp-D34*	rice	Thaumatinlike-protein	*Rs*, *Po*	Datta et al. 1999
PB1	*Dm-AMP1*	*Dahlia merckii*	defensin	*Rs*, *Po*	Jha et al. 2009
IR72, IR64, IR68899B, MH63, Chinsurah Boro II	*RC7*	rice	chitinase	*Rs*, *Po*	Datta et al. 2001
M202	*pinA* and/or *pinB*	wheat	puroindoline	*Rs*, *Po*	Krishnamurthy et al. 2001

[a] All of the rice cultivars mentioned above are indica type.

[b] *Rs*: *Rhizoctonia solani*

[c] *Po*: *Pyricularia oryzae*

oryzae (Wu et al. 2006). Rice transformation with the gene encoding this protein resulted in displayed resistance against sheath blight (Shah et al. 2009).

Antifungal protein genes of a plant origin have been successfully used for production of partially resistant rice lines against the sheath blight fungus. For instance, transgenic rice plants expressing genes encoding PR proteins, such as chitinases (Li et al. 2009; Lin et al. 1995; Shah et al. 2009; Sridevi et al. 2003), ß-1,3-glucanase (Sridevi et al. 2008), Defensin (Shah et al. 2009), and thaumatin-like protein (Datta et al. 1999) show a high level of resistance to sheath blight (Table 2). Chitinases from rice were effective against *R. solani* and *M. grisea* in rice (Datta et al. 2001; Lin et al. 1995). Transgenic rice lines and their progenies overexpressing a bitter melon gene (*McCHIT1*), encoding McCHIT1 chitinase, a class I secretory endochitinase, showed enhanced resistance to both sheath blight and blast pathogens (Li et al. 2009). Therefore, *McCHIT1*-transgenic rice confirmed the inheritance of the transgene. Furthermore, these findings revealed the overlap in rice defence responses against the two major fungal pathogens, *R. solani* and *M. grisea*. Also, this overlap has been reported by Shah et al. (2009) in defence responses of a transgenic rice line overexpressing a defensin gene (*Dm-AMP1*), obtained from a plant species (*Dahlia merckii*). Therefore, with the aid of genetic engineering, the generation of transgenic rice lines with broad spectrum resistance against various pathogens will be possible (Table 2).

Combined expression of both chitinase and b-1,3-glucanase genes was attempted to achieve higher levels of fungal resistance in many plant species. For instance, combined expression of rice chitinase (chi11) and tobacco ß -1,3-glucanase (*gluc*) genes in an elite indica rice cultivar Pusa Basmati 1 resulted in enhance resistance against *R. solani* (Sridevi et al. 2008). Since the cell wall of *R. solani* has both chitin and ß-1,3-glucan, combined expression of chitinase and ß-1,3-glucanase is capable of increasing sheath blight resistance to a higher level.

Many of the known antimicrobial proteins such as PRs are present in rice plants. Therefore, to use these proteins in transgenic plants to effectively limit pathogen growth, we would require overexpression of the protein. The expression of antimicrobial proteins in plants where they are not normally produced, may have greater potential to limit pathogen infection or growth. The puroindolines, endosperm-specific proteins involved in wheat seed hardness, are small proteins reported to have in vitro antimicrobial properties. Transgenic rice plants that constitutively express the puroindoline genes *pinA* and/or *pinB* throughout the plants showed significantly increased resistance to the most devastating fungal diseases of

rice, including blast and sheath blight (Table 2). Wheat puroindolines were effective as in vivo antifungal proteins in rice, which normally does not produce these proteins (Krishnamurthy et al. 2001). Therefore, the incorporation of puroindoline genes could be an effective tool in protecting rice against fungal diseases. Because the puroindolines normally are in wheat endosperm and thus are routinely ingested by humans and animals alike, their use in transgenic crops may be less unpleasant than gene products normally not in plants or in the edible portion of plants.

Signal transduction pathways

The plant defense responses to pathogen attack are regulated by highly coordinated sequential changes at the cellular and molecular levels. These changes are regulated by a complicated matrix of signal transduction pathways in which the plant hormones salicylic acid (SA), jasmonic acid (JA), ethylene (ET), and abscisic acid (ABA) are key signalling molecules (Adie et al. 2007; Grant and Lamb 2006; Ton et al. 2009). After pathogen attack, enhanced production of these phytohormones in plant cells leads to transcriptome changes and activation of distinct sets of defence-related genes. This phenomenon, which is a so-called signal signature, varies greatly in quantity, timing, and composition according to the type of plant-attacker combination and determines the defence mechanisms activated (Conrath et al. 2006; De vos et al. 2005; Glazebrook 2005). With some exceptions, it is generally accepted that SA acts as a central regulator of defence against (hemi) biotrophic pathogens, whereas jasmonic acid (JA) and ethylene (ET) are important signals in defence against necrotrophs (Glazebrook 2005; Thomma et al. 1998). In rice, involvement of JA signalling in basal and induced defence responses against *R. solani*, with a necrotrophic life style, has been demonstrated recently (Taheri and Tarighi 2010). Furthermore, JA signalling is involved in rice resistance to the blast fungus, *Pyricularia oryzae,* as a hemibiotrophic pathogen (Mei et al. 2006). These findings indicate the main role of JA in resistance of rice plants to sheath blight and blast diseases.

Plant defence responses against pathogen attack are known to be mediated by a complex regulatory network that connects various signalling pathways by synergistic or antagonistic effects on each other. Extensive cross-talk between various signal transduction pathways allows the plant to fine-tune its defence responses against different types of attackers. Although very little is known about synergistic and antagonistic cross-communication, a number of transcription factors and regulatory proteins have been characterized which are important in organizing the kinetics of various cellular processes, signal sensitivity and transduction in induced defence systems. For example, defence regulatory proteins such as NONEXPRESSER OF PR GENES1 (NPR1), WRKY53 and WRKY70 function as important transducers of the SA signalling and play a crucial role in the SA-mediated suppression of JA signalling (Li et al. 2004; Spoel et al. 2003; Miao and Zentgraf 2007; Ukler et al. 2007). However, contradictory results have been obtained by Zheng et al. (2006, 2007), indicating that WRKY25 and WRKY33 are effective supressors of SA responses.

Further molecular effectors in SA and JA signalling, as key pathways involved in plant defence, can be plastidial fatty acid-derived signals and/or glutaredoxins (Kachroo et al. 2003; Ndamukong et al. 2007; Koornneef and Pieterse 2008). In addition, mitogen-activated protein kinases (MAPKs) are important players in SA/JA cross talk (Frye et al. 2001). The MAPK signalling cascades are a group of protein kinases that play a central role in the intracellular transmission of extracellular signals. These cascades operate as major lines of communication within a complicated signalling network that regulates many cellular processes, including proliferation, differentiation, development, stress response, and programmed cell death in all eukaryotes. Besides MAPKs, this cascade is formed by MAPK kinases (MAPKKs) and MAPKK kinases (MAPKKKs). MAPKs are activated through phosphorylation by MAPKKs, which in turn are activated by MAPKKKs on serine and threonine/serine residues in the conserved SXXXS/T motif (Ligterink and Hirt 2001; Kim et al. 2003a, b; Schaeffer and Weber 1999; Widmann et al. 1999).

In rice plants treated with K252a as a protein kinase inhibitor, increased susceptibility to *R. solani* and suppression of the *LOX* gene expression, as a JA signalling marker, were observed. Furthermore, K252a suppressed the ability of riboflavin (vitamin B_2) to induce resistance in rice against the sheath blight pathogen via suppression of JA-mediated priming of lignification (Taheri and Höfte 2007; Taheri and Tarighi 2010). Therefore, MAPK cascades

are involved in riboflavin-IR in this pathosystem, which is in agreement with a previous report about the involvement of protein kinases in riboflavin-IR in tobacco and *Arabidopsis* against various biotrophic and necrotrophic pathogens (Dong and Beer 2000). The inhibitory effect of the protein kinase inhibitor, K252a, on expression of the phenylalanine ammonia-lyase *(PAL)* and peroxidase (*POC*), as SA-responsive genes, in addition to suppression of *LOX* expression as a JA responsible gene, might suggest that protein kinase cascades are upstream of both SA and JA signalling pathways (Taheri and Höfte 2007). These observations are in agreement with the findings on the effect of MAPK cascades on SA and JA signalling pathways in *Arabidopsis*. A protein kinase, known as MPK4, has been shown to be a negative regulator of SA signalling and a positive regulator of JA signalling in *Arabidopsis*, thereby functioning as a molecular switch between these mutually antagonistic pathways. Inactivation of MPK4 in an *Arabidopsis mpk4* mutant resulted in elevated SA levels and constitutive expression of SA-responsive PR genes, suppression of JA-responsive genes, and enhanced susceptibility to the necrotroph *A. brassicicola*. (Brodersen et al. 2006; Petersen et al. 2000). Some of the WRKY transcription factors can be phosphorylated by MPK4 (Andreasson et al. 2005), and may be downstream targets of MPK4 that contribute to the suppression of SA responses. For instance, overexpression of *WRKY25* and *WRKY33* resulted in decreased pathogen-induced PR-1 expression and enhanced susceptibility to *P. syringae* (Petersen et al. 2000; Zheng et al. 2006, 2007).

It is already known that a similar response is produced when the plant is attacked by a necrotrophic pathogen and/or after wounding (Pieterse and Van Loon 1999). Wound signalling is known to involve at least two major components including the JA signalling and the MAPK cascades in both dicots and monocots (Rakwal and Agrawal 2003), but our knowledge about the involvement of these mechanisms in plant defence responses against necrotrophic pathogens such as *R. solani* is still scarce. Application of JA on rice is reported to induce the MAPK cascade (Agrawal et al. 2003a); whereas to date, JA is not known to induce MAPKs in dicot plants. In Fig. 3, it is schematically presented how rice plants respond to a variety of environmental factors (including biotic and abiotic stresses) mainly by action of MAPKs and triggering the rapid production of jasmonates, the final products of JA signalling, followed by activation of downstream components and the cellular defence responses. As it is shown in this schematic, various WRKY transcription factors in *Arabidopsis* (*AtWRKY*s) have contradictory functions on SA and JA signalling. Since it is suggested that *WRKY* genes in rice and *Arabidopsis* are functionally likely to cooperate in the same signal transduction pathways (Berri et al. 2009), it will be interesting to find out the role of various orthologs of *AtWRKY* genes in rice, which are shown as *OsWRKY*s in Fig. 3, to provide a more in depth understanding of defence signalling pathways in rice plants.

Future perspectives

Although partial genetic resistance to sheath blight in rice has been reported, no major gene responsible for resistance has been found (Kumar et al. 2003). Therefore, absolute resistance to *R. solani* is not available in any of the rice germplasms grown worldwide. Most of the traditional cultivars, planted on over 90% of the rice-growing areas, are susceptible to this disease. However, it is known that resistance to sheath blight is controlled by polygenic quantitative trait loci in rice, making it difficult to evaluate the effect of individual genes on resistance. Fine mapping and cloning the genes involved in sheath blight resistance is an important step for understanding resistance mechanisms and increasing selection accuracy using marker-assisted selection (Liu et al. 2009). There are some rice lines such as Tetep, Tadukan, Teqing, Jasmine 85, ZYQ8, Minghui 63, LSBR-5 and LSBR-33 in which a high degree of resistance is available against this pathogen under field conditions. The mechanisms that provide quantitative resistance to *R. solani* in these rice lines is still not completely understood. It has been attributed to morphological and ecological characters other than mechanisms such as activation of host defence genes and production of PR-proteins upon pathogen infection (Channamallikarjuna et al. 2010).

The toxicity and negative environmental effects of fungicides and appearance of fungicide-resistant pathogen strains has created interest in using alternative protection strategies, such as biological or chemical induction of defence responses and adoption

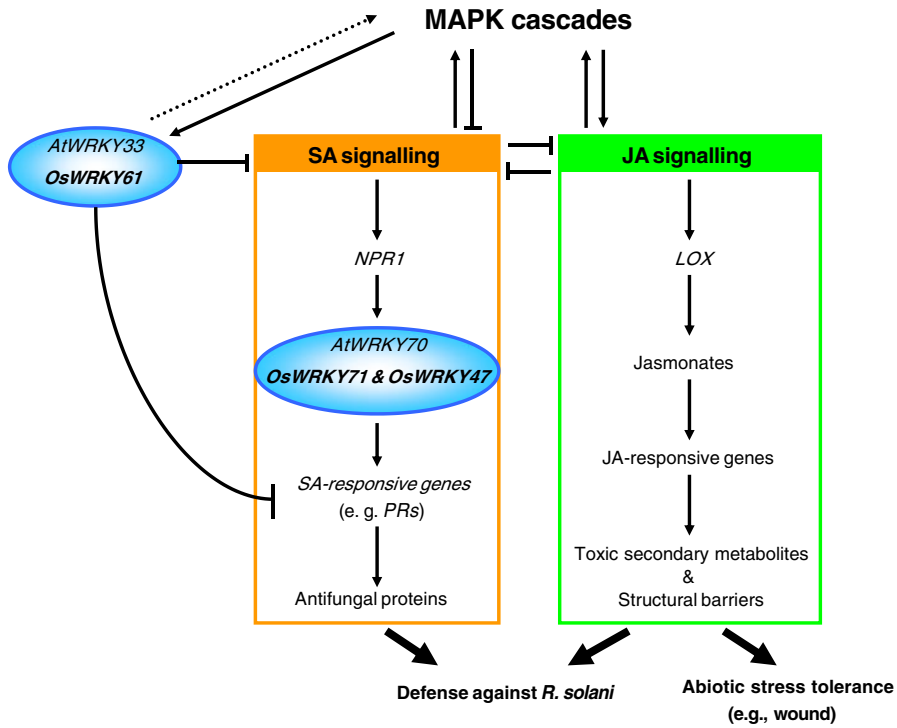

Fig. 3 A model illustrating how MAPK, SA, and JA signaling pathways and WRKY transcription factors are involved in rice defense responses against sheath blight disease and abiotic stress. The model is based on the results of Berri et al. (2009); Kim et al. (2003a, b); Li et al. (2004); Rakwal and Agrawal (2003); Taheri and Höfte (2007); Taheri and Tarighi (2010); Zheng et al. (2006, 2007). Sharp, solid lines represent synergistic effects, and blunted lines indicate antagonistic interactions. Gene names written in bold inside the circles (***OsWRKY***s) show the rice transcription factors, which are homologues of *Arabidopsis* transcription factor indicated in each circle. It is suggested that *OsWRKY*s might have the same function as their homologues in *Arabidopsis* (*AtWRKY*s), but it remains to be investigated in the future researches. Dotted line in the top of figure depicts the possibility of future studies to determine the interaction between *WRKY*s and MAPK cascades in rice and *Arabidopsis* model plants

of transgenic rice lines. Both of these management strategies rely on our knowledge concerning the genetic structure of the pathogen populations, cellular and molecular aspects of the rice-*R. solani* interaction, and defence signal transduction pathways involved in resistance against the pathogen. Although extensive progress has been made in our understanding of biotic and abiotic stress signalling and tolerance mechanisms in plants, the picture of defence signalling networks activated in rice plants after *R. solani* infection is still quite incomplete. Forward and reverse genetic studies using this monocot model plant continue to be critical for identifying unknown signalling components. Searching for superior alleles of key sheath blight resistance determinants such as *LOX* from naturally sheath blight-resistant rice lines will be very useful for engineering partially resistant cultivars. With more and more defence-related genes cloned, the big challenge for molecular biologists and crop breeders is how to sort out the most important genes and combine them in transgenic plants or use marker-assisted breeding to improve crop production under disease conditions. Many identified QTLs for sheath blight resistance have been reported in the rice genome which will facilitate identification of candidate genes and positional cloning of defence-related genes. Therefore, the sheath blight resistance QTLs, especialy the recently identified major QTL qSBR11-1, could be a preferred target for breeding durable disease resistance in rice cultivars (Channamallikarjuna et al. 2010). The markers closely linked to sheath blight resistance genes can be used for marker assisted selection in rice breeding programme. Overexpression of miRNA-resistant target genes will help overcome post-transcriptional gene silencing, and thus may lead to better expression of engineered trait in transgenic

plants which are partially resistant to sheath blight. Furthermore, the miRNA technique could be used for targeting pathogenicity genes of *R. solani* as the metagenomic draft of this destructive phytopathogen has already been known. Small RNA studies will shed more light on the molecular mechanisms of rice defence responses. Understanding the role of small RNAs and chromatin regulation in transcriptome homeostasis, cellular tolerance, phenological and developmental plasticity of plants infected with *R. solani* will be important for effective genetic engineering of high-level sheath blight resistance in rice plants.

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