Induction and suppression of rice innate immunity

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Abstract

The rice plant is infected by a number of pathogens which cause significant losses of yield. Plants possess inducible innate immunity by which they can perceive danger and mount defense responses. Recognition of the pathogen is a crucial step in induction of plant immune responses. Plants can recognise a wide category of molecules related either to conserved components of pathogen structures, pathogen secreted molecules or plant damage-associated molecules. Recognition of these molecules/elicitors by receptors initiates a signal transduction cascade which includes phosphorylation of various intermediate proteins, influx of calcium ions, production of reactive oxygen species and synthesis of phytohormones. The signaling intermediates also activate transcription factors leading to enhanced expression of genes related to defense associated functions. As immune responses are energy intensive processes, they are tightly regulated through phosphorylation/dephosphorylation events or through degradation of signalling intermediates. The activation of plant innate immunity suppresses multiplication and spread of pathogen within the host tissues. In this review we discuss about key molecular players involved in rice immune responses.

Keywords: Rice, rice diseases, plant immune responses, plant cell signaling, rice innate immunity

Introduction

Rice (*Oryza sativa* L.) is a staple food for more than half of the world's population. The rice crop is affected by various bacterial, fungal and viral pathogens. Some major bacterial diseases of rice crop are bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), bacterial leaf streak caused by *Xanthomonas oryzae* pv. *oryzicola* (*Xoc*) and sheath brown rot caused by *Pseudomonas fuscovaginae*. The common fungal diseases of rice include sheath blight caused by *Rhizoctonia solani*, blast disease caused by *Magnaporthe oryzae* (*M. oryzae*) and brown leaf spots caused by *Bipolaris oryzae* (Gnanamanickam 2009). Tungro disease is the most common viral disease of rice and is caused by a combination of two viruses namely the rice tungro baciliform virus (RTBV) and the rice tungro spherical virus (RTSV). Leaf hopper pests generally transmit these viruses. Another viral disease of rice is rice stripe virus disease (RSVD) caused by rice stripe virus (RSV) which is mainly spread by the brown plant hopper.

Unlike animals, plants are sedentary in nature and cannot run away from danger. Also, plants do not have specialised immune cells to combat pathogens. Instead, plants have evolved different but very effective strategies to defend themselves against pathogens. Plants can perceive presence of potential pathogens and induce strong immune responses. In the apoplast, plants can recognise presence of specific signature sequences of pathogens that could be structural components of pathogens or pathogen secreted molecules (Jones and Dangl 2006). These molecules elicit plant immune responses and are the so-called pathogen associated molecular patterns (PAMPs) and the immune responses triggered by PAMPs are called PAMP-triggered immunity (PTI) (Bigeard et al. 2015). Plant cell wall forms a formidable barrier for pathogens and potential pathogens secrete cell wall degrading enzymes that degrade different components of the cell wall. Plants perceive the cell wall degradation products as a mark of an infection and mount immune responses (Walton 1994). In addition, extracellular ATP and certain peptides that are released from

damaged cells also elicit immune responses. These host derived molecules that elicit/trigger plant immune responses are called damage associated molecular patterns (DAMPs) and such immunity is called DAMP-triggered immunity (DTI) (Wu et al. 2012). Potential pathogens have evolved molecules that are delivered directly into the cytoplasm of plant cells to suppress PTI/DTI and cause infection. In case of Gram-negative bacteria, this delivery of effectors is mediated by the bacterial type three secretion system (T3SS). *Xoo* secretes two types of effectors into rice cells, transcription activator like (TAL) effectors that modulate gene expression of plants and non-TAL effectors [also called as Xanthomonas outer proteins (Xop)] that suppress immunity by binding to various signaling intermediates (Kay and Bonas 2009). Plants also have evolved functions that can identify presence of effector molecules in the cytoplasm and this leads to induction of stronger immune responses to negate spread of pathogen. This is referred as effector triggered immunity (ETI) (Spoel and Dong 2012). The outcome of a plant-pathogen interaction depends on whether a pathogen possesses the functions required to suppress immune responses of its host plant. The ability to suppress innate immunity leads to onset of disease symptoms. In this review, we discuss the various steps that are associated with induction and suppression of innate immunity in rice. These steps include signal perception, elaboration of signal through intermediate steps, execution and regulation of immune responses.

Signal perception in rice immune response

Pathogen recognition is crucial for induction of plant immune responses. Plants have evolved various strategies to detect the presence of potential pathogens. In the extracellular milieu, plants can recognise the conserved components of pathogen structures (eg., chitin, flagellin, lipopolysaccharides, peptidoglycans etc.), pathogen secreted molecules

(e.g., EfTu, RaxX), and plant-damage associated molecules namely cell wall degradation products [(eg. oligogalacturonan (OG)] (Choi and Klessig 2016; Saijo et al. 2018) (Table 1). PAMPs/DAMPs are recognised by membrane localised receptor kinases. Most of the receptors that perceive PAMPs belong to leucine rich repeat containing receptor-like kinases (LRR-RLKs) while the carbohydrate-derived ligands are generally recognised by Lysine-motif (LysM) containing proteins. Based on the source of the molecules that are recognised as ligand, danger perception in rice can be catagorised in three categories: i) Perception of bacterial PAMPs, ii) Perception of fungal PAMPs, and iii) Perception of plant cell wall damage.

Perception of bacterial PAMPs

Plants can perceive presence of various bacteria specific molecules. Rice Xa21 protein is one of the first identified receptors that plays a key role in plant innate immunity (Song et al. 1995). The Xa21 gene is being used extensively in rice breeding to develop bacterial blight resistant rice lines. Recently it has been shown that a sulfated protein RaxX that is secreted by the bacterial type 1 secretion system is the ligand that is perceived by Xa21 (Pruitt et al. 2015). It was also observed that sulfation of tyrosine-41 (Y41) of RaxX is necessary to trigger immune response in rice. *Xoo* strains that encode an alternate allele of raxX evade recognition by Xa21. Bacterial flagellin is one of the most well studied PAMPs. In *Arabidopsis*, a receptor named Flagellin sensing 2 (AtFLS2) binds to a 22 amino acid long peptide (flg22) that is derived from flagellin of *Pseudomonas syringae* pv. *tomato* (*Pst* DC3000) (Gomez-Gomez and Boller 2000). In rice, *OsFLS2* can also perceive flg22 derived from *Pst* DC3000 or *Acidovorax avenae*, an important rice pathogen (Wang et al. 2015). Elongation factor-Tu (EF-Tu) is a bacterial protein that is released from bacterial cells, possibly due to cell lysis (Kunze et al. 2004). Rice can recognise presence of EF-Tu by perception

Table 1. List of PAMPs and their cognate receptors identified in rice plants

Gene family	Receptor	Ligand	Source of ligand	Reference
LRR-RLK	Xa21	RaxX-sY	Bacterial	(Pruitt et al. 2015)
	FLS2	flg22 (flagellin)	Bacterial	(Wang et al. 2015)
LysM-RLK	CERK1	Lipopolysaccharides	Bacterial	(Desaki et al. 2018)
	CEBiP1	Chitin	Fungal	(Kouzai et al. 2014)
	LYP4/LYP6	Peptidoglycan and chitin	Bacterial and fungal	(Liu et al. 2012)

RLK = Receptor-like kinases, LRR= Leucine rich repeat containing proteins, flg22 = Flagellin 22, LysM = Lysine-motif, FLS2 = flagellin sensing 2, CERK1= Chitin elicitor receptor kinase 1, CEBiP1 = Chitin-elicitor binding protein 1, and LYP = Lysin motif-containing proteins

of a 50-amino acid long peptide of EF-Tu called EFa50 and mount immune responses (Furukawa et al. 2014).

Peptidoglycan and lipopolysaccharides are major components of the bacterial cell wall. Rice LysM domain containing proteins OsLYP4 and OsLYP6 are known to interact with peptidoglycan (Liu et al. 2012). Some reports indicate OsCERK1 as a receptor/co-receptor for perception of lipopolysaccharides and peptidoglycan in rice (Ao et al. 2014; Desaki et al. 2018). A few *Xoo* strains have evolved to suppress this induction of CERK1 mediated immunity by suppressing its downstream target OsRLCK185 via T3SS secreted effector XopY (Xoo1488) (Yamaguchi et al. 2013). *Xoo* non-TAL effectors XopZ, XopN, and XopV can also suppress peptidoglycan induced immune response signaling in rice protoplast cells (Long et al. 2018). It is reported that *Xoo* secreted exopolysaccharides can also help in evasion of lipopolysaccharide induced immunity (Girija et al. 2016).

Perception of fungal PAMPs

Plants can also perceive structural components of fungal pathogens as PAMPs and mount immune responses. Chitin is a polymer of N-acetyl-D-glucosamine (GlcNAc) and is a major component of the fungal cell wall. Rice recognises chitin by a GPI-anchored protein called chitin elicitor binding protein (OsCEBiP), a receptor like protein (RLP). OsCEBiP contains 3 extracellular LysM domains but it lacks an intracellular domain for downstream signaling activation (Kaku et al. 2006). Ligand binding leads to homodimerization of OsCEBiP and its heterodimeri-zation with OsCERK1 that leads to activation of immune responses (Shimizu et al. 2010; Hayafune et al. 2014). *M. oryzae* has evolved strategy to avoid chitin perception in rice by secreting a chitinase MoChia1 that binds to chitin and avoids chitin triggered immunity (Yang et al. 2019). To counteract this strategy, rice cells secrete a tetratricopeptide repeat protein (OsTPR1) in the apoplast that competitively binds to MoChia1 leading to free chitin that activates PTI.

Perception of plant cell wall damage

Rice can also perceive the activity of pathogen secreted cell wall degrading enzymes on the cell wall and mount immune responses (Jha et al. 2005). *Xoo* secretes a battery of cell wall degrading enzymes such as cellulases, xylanases, and lipase/esterase to degrade different components of the cell wall and

treatment of rice tissue with any one of these purified cell wall degrading enzymes leads to activation of plant immunity (Rajeshwari et al. 2005; Jha et al. 2007). Rice cells likely sense the cell wall damage by perception of cell wall fragments (DAMPs) as heat inactivation or mutations in active site residues of the enzyme that lead to loss of biochemical activity result in loss of the ability of enzyme to induce immunity (Jha et al. 2007; Aparna et al. 2009). So far, no receptor for any DAMP is reported in rice but members of wall associated kinase (WAK) gene family are predicted to be putative receptors of cell wall damage (Zhang et al. 2005). Xa4, a major resistance QTL of rice that is known to enhance resistance against *Xoo* is reported to be a WAK gene family member (Hu et al. 2017). Our recent observation also indicates a wall associated kinase as a putative receptor for a *Xoo* lipase/esterase induced cell wall damage in rice (unpublished data). *Xoo* secreted T3SS effectors XopN, XopX, XopQ and XopZ can suppress cell wall damage induced immunity indicating evolution of specific functions to suppress DTI (Sinha et al. 2013).

Signaling intermediates in rice immune responses

Perception of danger by receptors leads to initiation of signaling events that induce immune responses that are conveyed into the cell via many signaling intermediates. These signaling intermediates involve diverse sets of molecules such as proteins, hormones or small ions such as calcium (Ca^{2+}) and reactive oxygen species (ROS). Here we discuss about few signaling intermediates reported in rice innate immunity.

Somatic embryogenesis receptor kinase (SERK)

SERK is an LRR-RLK which transduces signals by forming dimers (homodimers or heterodimers) with other receptor-like kinases (RLKs), in response to ligand binding. Dimerization leads to phosphorylation of kinase domains of the RLKs, which then activates signalling cascade. Over expression of *OsSERK1* in rice plants leads to enhanced tolerance to *M. oryzae* (Hu et al. 2005). Rice *OsSERK2* has been shown to play a critical role in XA21, XA3, and OsFLS2-mediated immunity by directly binding to these receptors (Chen et al. 2014).

Mitogen-activated protein kinases (MAP Kinases or MAPKs)

Upon PAMP/DAMP treatment, the induced signal is associated with activation of a MAPK cascade. This

activation is observed as early as two minutes after treatment and disappears within an hour (Meng and Zhang 2013). Classically, an activated MAPKKK (MAP kinase kinase kinase) phosphorylates MAPKK (MAP kinase kinase) which in turn activates MAPK (MAP kinase) by phosphorylation (Rasmussen et al. 2012). The activated MAPK can interact and phosphorylate a wide variety of signaling molecules (transcription factors, 14-3-3 proteins, E3 ubiquitin ligases, VQ proteins, cellular enzymes, etc.) which eventually leads to the activation of immune responses (Bigeard et al. 2015). There are 74, 8 and 17 genes encoding for MAPKKKs, MAPKKs and MAPKs, respectively in rice (LP et al. 2006; Reyna and Yang 2006; Rao et al. 2010). *OsMPK6* plays contrasting roles during *Xoo* infection; local resistance is positively affected by *OsMPK6* but systemic acquired resistance activated post *Xoo* infection is negatively regulated by *OsMPK6* (Yuan et al. 2007a; Shen et al. 2010). Rice resistance to *Xoo* is negatively regulated by OsMAPKKK1 (aka *OsEDR1*) (Shen et al. 2011). Phosphorylation of OsMPK1 and OsMPK5 was observed after treatment with peptidoglycan isolated from *Xoo* (Long et al. 2018).

14-3-3 proteins

14-3-3 proteins are adaptor proteins which play diverse roles in signaling events. For interaction, 14-3-3 proteins are sensitive to phosphostatus of their client proteins. Binding of target protein with a 14-3-3 protein can lead to diverse molecular events (Lozano-Duran and Robatzek 2015).14-3-3 genes *GF14b*, *GF14c*, *GF14e* and *GF14f* were induced when rice was exposed to either *Xoo* or *M. oryzae* (Chen et al. 2006). 14-3-3 proteins in rice, GF14b and GF14f interact with BIMK1 (MAP kinase) and induce systemic resistance against *M. oryzae* (Cooper et al. 2003).

E3 ubiquitin ligases

Ubiquitination is a multi-step and multi-enzyme process. Three proteins referred to as E1, E2 and E3 are involved in this process. The E1 protein is an ubiquitin activating enzyme which uses ATP to activate ubiquitin. The E1 protein transfers ubiquitin to the E2 protein which is an ubiquitin conjugating enzyme. The E3 protein is the ubiquitin ligase which is involved in the transfer of ubiquitin from E2 to the target protein. The type of ubiquitination dictates the fate of the target protein. Proteins are marked for degradation by 26S proteasome, if they are polyubiquitinated with K48 (Lysine at 48th position of the amino acid sequence of ubiquitin protein) linkages, whereas polyubiquitination with linkages other than K48 and monoubiquitination do not cause degradation but bring out other effects such as internalization and endocytosis of membrane receptors, histone modification, etc. (Zhou and Zeng 2017). Suppression of the ubiquitin ligase *XB3* compromises Xa21-mediated *Xoo* resistance in rice and it is accompanied by a drop in Xa21 protein levels (Wang et al. 2006). But how XB3 regulates XA21 protein levels and what is the downstream target of XB3 are areas that need to be further explored. OsPUB44 positively regulates peptidoglycan and chitin-induced immune responses and tolerance to *Xoo* in rice. XopP, an *Xoo* secreted effector, suppresses OsPUB44's E3 ligase activity and compromises rice immune responses (Ishikawa et al. 2014).

Calcium influx

PAMP/DAMP treatment induces Ca^{2+} influx within thirty seconds of treatment and it peaks by six minutes. Two-pore channel-1 (TPC1, calcium channel) and glutamate receptor-like channels (GLRs) are responsible for Ca^{2+} influx. Ca^{2+} influx induces opening of other membrane channels (such as those for proton, chloride ion, potassium ion and nitrate ion) which ultimately leads to membrane depolarisation (Lecourieux et al. 2006; Szechyñska-Hebda et al. 2017). Calcium level sensor proteins like calmodulin (Bellincampi et al. 2014) or Ca^{2+} dependent protein kinases (CDPKs) perceive changes in cytoplasmic Ca^{2+} levels and translate it by activating downstream signaling to induce defense responses. CDPKs play a key role in translating the pathogen induced signal to change in the levels of Ca^{2+} concentration which would transiently lead to activation of plant defense responses. In rice, CDPK gene family is predicted to comprise around 29 members.

Reactive oxygen species (ROS) production

ROS burst is observed as early as 2-3 minutes after PAMP treatment (Spoel and Dong, 2012). The ROS production in response to PAMP/DAMP treatment is through activation of a plasma membrane-situated NADPH oxidase enzyme, respiratory burst oxidase homolog D (RBOHD). In rice, recent observations indicate that treatment of rice suspension cells with chitin or chitin derived oligosaccharides triggers ROS production (Hayafune et al. 2014; Yang et al. 2019).

Plant hormones

Salicylic acid (SA) and jasmonic acid (JA) are phytohormones which have been implicated in rice defense responses triggered by pathogens. Unlike

Arabidopsis, rice plants exhibit higher basal levels of salicylic acid. Exogenous applications of JA or SA have been reported to impart resistance to *Xoo* infection in rice (Babu et al. 2003; Yamada et al. 2012). OsJAZ8, a repressor of JA signaling in rice, negatively regulates JA-mediated resistance to *Xoo* infection (Yamada et al. 2012). SA-deficient NahG rice plants show more susceptibility to oxidative stress caused by *M. oryzae* infection (Yang et al. 2004). Overexpression of *OsNH1*, a key SA-responsive gene, leads to constitutive expression of defense-related genes and enhanced resistance to *Xoo* (Yuan et al. 2007b; Chen et al. 2014). In SA accumulation deficient *NahG* transgenic rice plants, this *OsNH1*-mediated resistance to *Xoo* infection is compromised indicating involvement of SA signaling in tolerance against *Xoo* (Yang 2009).

Transcription factors involved in rice defense

The early signaling events (calcium, ROS, MAPK mediated signaling) and/or the phytohormone-mediated signaling bring about transcriptional reprogramming (TR) (Tsuda and Somssich 2015; Li et al. 2016). The kind of transcriptional reprogramming that is observed is highly variable and it varies with the pathogen, type of elicitor, period of exposure, developmental state of the plant, etc. Broadly, the reprogramming that takes place during pathogen attack involves downregulation of normal cellular activity like photosynthesis and growth-related processes. Expressions of genes involved in pathogen defense are upregulated. The key molecules that execute transcriptional reprogramming are transcription factors (TFs) and chromatin modulators. The transcription factors that are majorly involved in the regulation of plant immunity belong to WRKY, AP2/ERF, bHLH and MYB gene families. WRKY transcription factors have the so called WKRY domain [consisting of four conserved amino acids: tryptophan (W), lysine (K), glutamate (R), and tyrosine (Y)] and a zinc finger motif. WRKY transcription factors bind to DNA at W-box consensus sequences (TTGACT/C) (Thomas Eulgem 2000; Agarwal et al. 2010). A number of WRKY proteins are regulators of plant defense responses (Zhao 2012). *AtWRKY33* and *OsWRKY45* are marker genes for activation of plant immunity in Arabidopsis and rice respectively. Over expression of *OsWRKY45-2* leads to enhanced tolerance to bacterial and fungal infection in rice (Shimono et al. 2012). Other WRKY proteins, *OsWRKY71* and *OsWRKY22* function as positive regulators of rice immunity (Liu et al. 2007; Abbruscato et al. 2012). Studies have revealed that protein-protein interaction between the same or different WRKY

transcription factors is required for many WRKY proteins for their function in transcription and chromatin remodelling (Chi et al. 2013). *OsWRKY62* and *OsWRKY76* are reported to form homo- and heterodimers. The splice variants of these two WRKY transcription factors have different roles in plant defense. Over expression of *OsWRKY62.1* and *OsWRKY76.1* leads to enhanced tolerance against *Xoo* and *M. oryzae* infection while the shorter variants, *OsWRKY62.2* and *OsWRKY76.2*, act as repressors (Liu et al. 2016) of the immunity.

Another class of transcription factor which are involved in controlling responses to abiotic and biotic stresses are the AP2/ERF proteins (Sharoni et al. 2011). Overexpression of *OsEREB1* and *OsAP2/ERF152* primes rice plants against subsequent *Xoo* infection (Jha et al. 2010; Jisha et al. 2015). Several MYB proteins are also reported as positive regulators of plant immunity (Lotkowska et al. 2015). In rice, the *OsMYB30*, *OsMYB55* and *OsMYB110* functions are reported as inducers of immune responses against bacterial and fungal pathogens likely by activating synthesis of secondary metabolites that have antimicrobial activity (Kishi-Kaboshi et al. 2018). In rice, the NAC transcription factor *OsNAC066* positively regulates tolerance against blast and bacterial blight while *OsNAC60* enhances tolerance against rice blast disease (Liu et al. 2018).

Regulation of rice immune responses

Immune responses are energy consuming processes; therefore, they are tightly regulated and the signaling cascade is quickly shut-off soon after activation (Yang et al. 2012). This suppression is attained through dephosphorylation of active site residues, phosphorylation of alternate sites that inhibit active site residue phosphorylation or degradation of signaling intermediates such as receptors, intermediate kinases or transcription factors (Park et al. 2012). Xa21 physically interacts with XB24, an ATPase that enhances *in vitro* autophosphorylation of XA21. Xa21 can also bind to XB15, a protein phosphatase 2C that can dephosphorylate XA21 (Park et al. 2008; Chen et al. 2010). Experiments with XB24 and XB15 indicate that XB24 keeps Xa21 inactive in the resting state by phosphorylation. After signal perception by Xa21, structural changes in the intracellular kinase domain promote autophosphorylation at an alternate site that leads to dissociation from XB24 and phosphorylation of downstream signaling intermediate. After some time of activation, XB15 dephosphorylates Xa21 when the

immune response is not required (Park et al. 2008; Chen et al. 2010).

Ubiquitin ligases negatively regulate plant defense through degradation of the activators of plant defense responses. For example, OsSPL11 and EBR1 (enhanced blight and blast resistance 1) are E3 ubiquitin ligases of rice, which suppress programmed cell death (Zeng et al. 2004; You et al. 2016). Calcium signaling can also regulate rice immune responses. OsCDPK12 was reported to negatively affect ROS production and resistance to blast disease in rice (Asano et al. 2012). Rice CPK18 was reported to directly phosphorylate and activate rice MPK5 and this CPK18-MPK5 pathway was found to negatively regulate rice immunity against blast disease (Xie et al. 2014). There are many examples of transcription factors that act as negative regulators of plant immunity. *OsWRKY62, OsWRKY42, OsWRKY13* and *OsWRKY24* negatively regulate defense responses in rice (Mao et al. 2007; Cheng et al. 2015). A *de novo* gene with no known function specific to rice plant, *Defense Related 10* (*OsDR10*) acts as suppressor of plant defense against *Xoo* infection.

Execution of rice immune response

Activation of plant immune responses can check the entry of pathogens into plant cells. Many pathogens enter a plant system through natural openings such as stomata or hydathodes (Gustavo e. Gudesblat 2009). Recognition of PAMP/DAMP leads to closure of stomata in order to prevent the entry of pathogens. The plant cell wall is the next physical barrier that a pathogen encounters. Lignification is one of the strategies of strengthening of the rice cell wall to prevent pathogen-mediated cell wall disruption (Jha et al. 2007; Kishi-Kaboshi et al. 2018). Another important way of cell wall fortification is by callose deposition or papillae formation at the site of pathogen entry. Callose is a β (1-3) glucan polymer which along with antimicrobial molecules forms the papillae. Treatment of rice tissue with either PAMPs or cell wall degrading enzymes leads to enhanced callose deposition (Jha et al. 2010; Liu et al. 2013; Furukawa et al. 2014).

On the induction of an immune response, plants release ROS that can play a role in signaling and in directly inhibiting the pathogen (Waszczak et al. 2018). Secondary metabolites produced by plants act as antimicrobial compounds (Scala et al. 2013; Kushalappa et al. 2016). These molecules include terpenoids, alkaloids, vitamins and phenolic compounds. Protein defense molecules include defensins, amylase, and proteinase inhibitors which target different enzymes of pests and pathogens. Plants also produce lytic enzymes that act upon the cell wall of pathogens. A number of the PR genes induced upon PAMP recognition, are hydrolytic enzymes that attack microbial cell walls. There are seventeen PR protein families identified in plants (van Loon et al. 2006). In rice, the expression of several PR proteins (PR1, PR2, PR3, PR4b, PR5, PR8, PR16 and PR-pha) is enhanced after *Xoo* infection (Hou et al. 2012).

Defense response associated programmed cell death (PCD) is induced in plants, including rice, upon recognition of elicitors and effectors (van Doorn and Woltering 2005; Jha et al. 2010; Dickman and Fluhr 2013; Tyler et al. 2013; Wang and Bayles 2013). This response is believed to prevent further growth and movement of pathogens within plant tissues. This is especially the case for biotrophic and hemi-biotrophic pathogens. However, many necrotrophic pathogens have evolved strategies to utilise this immune response to kill plant cells. Thus, the decision of triggering an appropriate type of PCD is also a crucial event in plant defense.

Conclusion

Like other plants, rice possesses a robust immune response that can ward of most potential pathogens. Understanding the mechanisms by which these defense response pathways work will be helpful not only in terms of providing new knowledge about how plants interact with their pathogens but can also help in the development of new strategies for reducing crop losses. In this review, we discussed some of the signaling events and intermediates that are involved in the elaboration and execution of immune responses. Although, a large number of R-genes and resistant germplasm are available for many rice diseases, the same is not the case for several others. This makes it important to understand the molecular mechanisms involved in rice immune responses as this might help in the development of new strategies for controlling such diseases. This might also be helpful where resistance sources are currently available but which may become ineffective due to emergence of newer races of pathogens.

Declaration

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References

Abbruscato P., Nepusz T., Mizzi L., Del Corvo M., Morandini P., Fumasoni I., Michel C., Paccanaro A., Guiderdoni E., Schaffrath U., Morel J.-B., Piffanelli P. and Faivre-Rampant O. 2012. OsWRKY22, a monocot WRKY gene, plays a role in the resistance response to blast. Mol. Plant Pathol., **13**: 828-841.

Agarwal P., Reddy M. P. and Chikara J. 2010. WRKY: its structure, evolutionary relationship, DNA-binding selectivity, role in stress tolerance and development of plants. Mol. Biol. Rep., **38**: 3883-3896.

Ao Y., Li Z., Feng D., Xiong F., Liu J., Li J. F., Wang M., Wang J., Liu B. and Wang H. B. 2014. OsCERK1 and OsRLCK176 play important roles in peptidoglycan and chitin signaling in rice innate immunity. The Plant Journal : For Cell and Molecular Biology, **80**: 1072-1084.

Aparna G., Chatterjee A., Sonti R. V. and Sankaranarayanan R. 2009. A cell wall-degrading esterase of Xanthomonas oryzae requires a unique substrate recognition module for pathogenesis on rice. Plant Cell, **21**: 1860-1873.

Asano T., Hayashi N., Kobayashi M., Aoki N., Miyao A., Mitsuhara I., Ichikawa H., Komatsu S., Hirochika H., Kikuchi S. and Ohsugi R. 2012. A rice calcium-dependent protein kinase OsCPK12 oppositely modulates salt-stress tolerance and blast disease resistance. The Plant J., **69**: 26-36.

Babu R. M., Sajeena A., Samundeeswari A. V., Sreedhar A., Vidhyasekaran P., Seetharaman K. and Reddy M. S. 2003. Induction of systemic resistance to *Xanthomonas oryzae* pv. *oryzae* by salicylic acid in *Oryza sativa* (L.). J. Plant Dis. Prot., **110**: 419-431.

Bellincampi D., Cervone F. and Lionetti V. 2014. Plant cell wall dynamics and wall-related susceptibility in plant pathogen interactions. Frontiers in Plant Science, **5**.

Bigeard J., Colcombet J. and Hirt H. 2015. Signaling Mechanisms in Pattern-Triggered Immunity (PTI). Mol. Plant, **8**: 521-539.

Chen F., Q L., L S. and Z H. 2006. The rice 14-3-3 gene family and its involvement in responses to biotic and abiotic stress. DNA Research, **13**: 53-63.

Chen X., Chern M., Canlas P. E., Ruan D., Jiang C. and Ronald P. C. 2010. An ATPase promotes autophosphorylation of the pattern recognition receptor XA21 and inhibits XA21-mediated immunity. Proc. Nat. Acad. Sci., **107**: 8029-8034.

Chen X., Zuo S., Schwessinger B., Chern M., Canlas P. E., Ruan D., Zhou X., Wang J., Daudi A., Petzold C. J., Heazlewood J. L. and Ronald P. C. 2014. An XA21-Associated Kinase (OsSERK2) Regulates Immunity Mediated by the XA21 and XA3 Immune Receptors. Mol. Plant, **7**: 874-892.

Cheng H., Liu H., Deng Y., Xiao J., Li X. and Wang S. 2015. The WRKY45-2 WRKY13 WRKY42 Transcriptional Regulatory Cascade Is Required for Rice Resistance to Fungal Pathogen. Plant Physiol., **167**: 1087-1099.

Chi Y., Yang Y., Zhou Y., Zhou J., Fan B., Yu J.-Q. and Chen Z. 2013. Protein–Protein Interactions in the Regulation of WRKY Transcription Factors. Mol. Plant, **6**: 287-300.

Choi H. W. and Klessig D. F. 2016. DAMPs, MAMPs, and NAMPs in plant innate immunity. BMC Plant Biol., **16**: 232.

Cooper B., Clarke J. D., Budworth P., Kreps J., Hutchison D., Park S., Guimil S., Dunn M., Luginbuhl P., Ellero C., Goff S. A. and Glazebrook J. 2003. A network of rice genes associated with stress response and seed development. PNAS, **100**: 4945-4950.

Desaki Y., Kouzai Y., Ninomiya Y., Iwase R., Shimizu Y., Seko K., Molinaro A., Minami E., Shibuya N., Kaku H. and Nishizawa Y. 2018. OsCERK1 plays a crucial role in the lipopolysaccharide-induced immune response of rice. The New Phytologist, **217**: 1042-1049.

Dickman M. B. and Fluhr R. 2013. Centrality of Host Cell Death in Plant-Microbe Interactions. Ann. Review Phytopathol., **51**: 543-570.

Furukawa T., Inagaki H., Takai R., Hirai H. and Che F. S. 2014. Two distinct EF-Tu epitopes induce immune responses in rice and Arabidopsis. Mol. Plant Microbe Interact., MPMI, **27**: 113-124.

Girija A. M., Kinathi B. K., Madhavi M. B., Ramesh P., Vungarala S., Patel H. K. and Sonti R. V. 2016. Rice Leaf Transcriptional Profiling Suggests a Functional Interplay Between Xanthomonas oryzae pv. oryzae Lipopolysaccharide and Extracellular Polysaccharide in Modulation of Defense Responses During Infection. Mol. Plant Microbe Interact., **30**: 16-27.

Gnanamanickam S. S. 2009. Major Diseases of Rice. In: Biological Control of Rice Diseases, (Ed. S.S. Gnanamanickam), Dordrecht: Springer Netherlands, pp. 13-42.

Gomez-Gomez L. and Boller T. 2000. FLS2: an LRR receptor-like kinase involved in the perception of the bacterial elicitor flagellin in Arabidopsis. Mol. Cell, **5**: 1003-1011.

Gustavo e. Gudesblat P. S. T. a. A. A. v. 2009. Stomata and pathogens Warfare at the gates. Plant Sig. and Behav., 12: 1114-1116.

Hayafune M., Berisio R., Marchetti R., Silipo A., Kayama M., Desaki Y., Arima S., Squeglia F., Ruggiero A., Tokuyasu K., Molinaro A., Kaku H. and Shibuya N. 2014. Chitin-induced activation of immune signaling by the rice receptor CEBiP relies on a unique sandwich-type dimerization. Proc. Nat. Acad. Sci., (USA), **111**: E404-413.

Hou M., Xu W., Bai H., Liu Y., Li L., Liu L., Liu B. and Liu G. 2012. Characteristic expression of rice pathogenesis-related proteins in rice leaves during interactions with Xanthomonas oryzae pv. oryzae. Plant Cell Rep., **31**: 895-904.

Hu H., Xiong L. and Yang Y. 2005. Rice SERK1 gene positively regulates somatic embryogenesis of cultured cell and host defense response against fungal infection. Planta, 222.

Hu K., Cao J., Zhang J., Xia F., Ke Y., Zhang H., Xie W., Liu H., Cui Y., Cao Y., Sun X., Xiao J., Li X., Zhang Q. and Wang S. 2017. Improvement of multiple agronomic traits by a disease resistance gene via cell wall reinforcement,: 17009.

Ishikawa K., Yamaguchi K., Sakamoto K., Yoshimura S., Inoue K., Tsuge S., Kojima C. and Kawasaki T. 2014. Bacterial effector modulation of host E3 ligase activity suppresses PAMP-triggered immunity in rice. Nature Commun., **5**: 1-11.

Jha G., Rajeshwari R. and Sonti R. V. 2005. Bacterial Type Two Secretion System Secreted Proteins: Double-Edged Swords for Plant Pathogens. Mol. PlantMicrobe Interact., **18**: 891-898.

Jha G., Rajeshwari R. and Sonti R. V. 2007. Functional interplay between two Xanthomonas oryzae pv. oryzae secretion systems in modulating virulence on rice. Mol. Plant Microbe Interact., **20**: 31-40.

Jha G., Patel H. K., Dasgupta M., Palaparthi R. and Sonti R. V. 2010. Transcriptional Profiling of Rice Leaves Undergoing a Hypersensitive Response Like Reaction Induced by Xanthomonas oryzae pv. oryzae Cellulase. Rice, **3**: 1-21.

Jisha V., Dampanaboina L., Vadassery J., Mithofer A., Kappara S. and Ramanan R. 2015. Overexpression of an AP2/ERF Type Transcription Factor OsEREBP1 Confers Biotic and Abiotic Stress Tolerance in Rice. PLoS One, **10**: e0127831.

Jones J. D. G. and Dangl J. L. 2006. The plant immune system. Nature, **444**: 323.

Kaku H., Nishizawa Y., Ishii-Minami N., Akimoto-Tomiyama C., Dohmae N. and Takio K. 2006. Plant cells recognize chitin fragments for defense signaling through a plasma membrane receptor. Proc. Nat. Acad. Sci., (United States of America), 103.

Kay S. and Bonas U. 2009. How Xanthomonas type III effectors manipulate the host plant. Current Opinion Microbiol., **12**: 37-43.

Kishi-Kaboshi M., Seo S., Takahashi A. and Hirochika H. 2018. The MAMP-Responsive MYB Transcription Factors MYB30, MYB55 and MYB110 Activate the HCAA Synthesis Pathway and Enhance Immunity in Rice. Plant Cell Physiol., **59**: 903-915.

Kouzai Y., Nakajima K., Hayafune M., Ozawa K., Kaku H., Shibuya N., Minami E. and Nishizawa Y. 2014. CEBiP is the major chitin oligomer-binding protein in rice and plays a main role in the perception of chitin oligomers. Plant Mol. Biol., **84**: 519-528.

Kunze G., Zipfel C., Robatzek S., Niehaus K., Boller T. and Felix G. 2004. The N Terminus of Bacterial Elongation Factor Tu Elicits Innate Immunity in Arabidopsis Plants. The Plant Cell, **16**: 3496-3507.

Kushalappa A. C., Yogendra K. N. and Karre S. 2016. Plant Innate Immune Response: Qualitative and Quantitative Resistance. Critical Reviews in Plant Sciences, **35**: 38-55.

Lecourieux D., Ranjeva R. and Pugin A. 2006. Calcium in plant defence-signalling pathways. The New Phytologist, **171**: 249-269.

Li B., Meng X., Shan L. and He P. 2016. Transcriptional Regulation of Pattern-Triggered Immunity in Plants. Cell Host & Microbe, **19**: 641-650.

Liu B., Li J. F., Ao Y., Qu J., Li Z. and Su J. 2012. Lysin motif-containing proteins LYP4 and LYP6 play dual roles in peptidoglycan and chitin perception in rice innate immunity. Plant Cell, **8**.

Liu J., Chen X., Liang X., Zhou X., Yang F., Liu J., He S. Y. and Guo Z. 2016. Alternative splicing of rice WRKY62 and WRKY76 transcription factor genes in pathogen defense. Plant Physiol., pp. 01921.02015.

Liu Q., Yan S., Huang W., Yang J., Dong J., Zhang S., Zhao J., Yang T., Mao X., Zhu X., and Liu B. 2018. NAC transcription factor ONAC066 positively regulates disease resistance by suppressing the ABA signaling pathway in rice. Plant Mol. Biol., **98**: 289-302.

Liu W., Liu J., Ning Y., Ding B., Wang X., Wang Z. and Wang G. L. 2013. Recent progress in understanding PAMP- and effector-triggered immunity against the rice blast fungus *Magnaporthe oryzae*. Mol. Plant, **6**: 605-620.

Liu X., Bai X., Wang X. and Chu C. 2007. OsWRKY71, a rice transcription factor, is involved in rice defense response. J. Plant Physiol., **164**: 969-979.

Long J., Song C., Yan F., Zhou J., Zhou H. and Yang B. 2018. Non-TAL Effectors From Xanthomonas oryzae pv. oryzae Suppress Peptidoglycan-Triggered MAPK Activation in Rice. Front. Plant Sci., **9**.

Lotkowska M. E., Tohge T., Fernie A. R., Xue G.-P., Balazadeh S. and Mueller-Roeber B. 2015. The Arabidopsis transcription factor MYB112 promotes anthocyanin formation during salinity and under high

light stress. Plant Physiol., pp. 00605.02015.

Lozano-Duran R. and Robatzek S. 2015. 14-3-3 proteins in plant-pathogen interactions. Mol. Plant Microbe Interact., **28**: 511-518.

LP H., MC N., S S., MJ M., M E., J E., N B., D B. B. K., J L., G M., J M., Y O., D S., J S., T X., S Z., A S. and BE E. 2006. Ancient signals: comparative genomics of plant MAPK and MAPKK gene families. Trends Plant Sci., **11**: 192-198.

Mao P., Duan M., Wei C. and Li Y. 2007. WRKY62 Transcription Factor Acts Downstream of Cytosolic NPR1 and Negatively Regulates Jasmonate-Responsive Gene Expression. Plant Cell Physiol., **48**: 833-842.

Meng X. and Zhang S. 2013. MAPK Cascades in Plant Disease Resistance Signaling. Annu. Rev. Phytopathol., **51**: 12.11-12.22.

Park C. J., Caddell D. F. and Ronald P. C. 2012. Protein phosphorylation in plant immunity: insights into the regulation of pattern recognition receptor-mediated signaling. Front. Plant Sci., **3**: 177.

Park C. J., Peng Y., Chen X., Dardick C., Ruan D., Bart R., Canlas P. E. and Ronald P. C. 2008. Rice XB15, a protein phosphatase 2C, negatively regulates cell death and XA21-mediated innate immunity. PLoS Biology, **6**: e231.

Pruitt R. N., Schwessinger B., Joe A., Thomas N., Liu F., Albert M., Robinson M. R., Chan L. J., Luu D. D., Chen H., Bahar O., Daudi A., De Vleesschauwer D., Caddell D., Zhang W., Zhao X., Li X., Heazlewood J. L., Ruan D., Majumder D., Chern M., Kalbacher H., Midha S., Patil P. B., Sonti R. V., Petzold C. J., Liu C. C., Brodbelt J. S., Felix G. and Ronald P. C. 2015. The rice immune receptor XA21 recognizes a tyrosine-sulfated protein from a Gram-negative bacterium. Sci. Adv., **1**: e1500245.

Rajeshwari R., Jha G. and Sonti R. V. 2005. Role of an In Planta-Expressed Xylanase of Xanthomonas oryzae pv. oryzae in Promoting Virulence on Rice. Mol. Plant Microbe Interact., **18**: 830-837.

Rao K. P., Richa T., Kumar K., Aghuram B. and Sinha A. K. 2010. *In Silico* Analysis Reveals 75 Members of Mitogen-Activated Protein Kinase Kinase Kinase Gene Family in Rice. DNA Research, **17**: 139-153.

Rasmussen M. W., Roux M., Petersen M. and Mundy J. 2012. MAP kinase cascades in Arabidopsis innate immunity. Front. Plant Sci., **3**: 1-6.

Reyna N. S. and Yang Y. 2006. Molecular Analysis of the Rice MAP Kinase Gene Family in Relation to *Magnaporthe grisea* Infection MPMI, **19**: 530-540.

Saijo Y., Loo E. P. and Yasuda S. 2018. Pattern recognition receptors and signaling in plant-microbe interactions. Plant J., **93**: 592-613.

Scala A., Allmann S., Mirabella R., Haring M. and

Schuurink R. 2013. Green Leaf Volatiles: A Plant's Multifunctional Weapon against Herbivores and Pathogens. Inter. J. Mol. Sci., **14**: 17781-17811.

Sharoni A. M., Nuruzzaman M., Satoh K., Shimizu T., Kondoh H., Sasaya T., Choi I.-R., Omura T. and Kikuchi S. 2011. Gene Structures, Classification and Expression Models of the AP2/EREBP Transcription Factor Family in Rice. Plant Cell Physiol., **52**: 344-360.

Shen X., Yuan B., Liu H., Li X., Xu C. and Wang S. 2010. Opposite functions of a rice mitogen-activated protein kinase during the process of resistance against *Xanthomonas oryza*. The Plant J., **64**: 86-99.

Shen X., Liu H., Yuan B., Li X., Xu C. and Wang S. 2011. OsEDR1 negatively regulates rice bacterial resistance via activation of ethylene biosynthesis. Plant Cell Environ., **34**: 179-191.

Shimizu T., Nakano T., Takamizawa D., Desaki Y., Ishii-Minami N. and Nishizawa Y. 2010. Two LysM receptor molecules, CEBiP and OsCERK1, cooperatively regulate chitin elicitor signaling in rice. The Plant Journal : For Cell and Molecular Biology, **64**.

Shimono M., Koga H., Akagi A. Y. A., Hayashi N., Goto S., Sawada M., Kurihara T., Matsushita A., Sugano S., Jiang C.-J., Kaku H., Inoue H. and Takatsuji H. 2012. Rice WRKY45 plays important roles in fungal and bacterial disease resistance. Mol. Plant Pathol., **13**: 83-94.

Sinha D., Gupta M. K., Patel H. K., Ranjan A. and Sonti R. V. 2013. Cell wall degrading enzyme induced rice innate immune responses are suppressed by the type 3 secretion system effectors XopN, XopQ, XopX and XopZ of *Xanthomonas oryzae* pv. oryzae. PloS One, **8**: e75867.

Song W. Y., Wang G. L., Chen L. L., Kim H. S., Pi L. Y., Holsten T., Gardner J., Wang B., Zhai W. X., Zhu L. H., Fauquet C. and Ronald P. 1995. A receptor kinase-like protein encoded by the rice disease resistance gene, Xa21. Science (New York, N.Y.) 270, 1804-1806.

Spoel S. H. and Dong X. 2012. How do plants achieve immunity? Defence without specialized immune cells. Nature Reviews Immunology, **12**: 89-100.

Szechyñska-Hebda M., Lewandowska M. and Karpiñski S. 2017. Electrical Signaling, Photosynthesis and Systemic Acquired Acclimation. Front. Physiol., **B8**: 684.

Thomas Eulgem P. J. R., Silke Robatzek and Imre E. Somssich. 2000. The WRKY superfamily of plant transcription factors. Trends Plant Sci., **5**: 199-206.

Tsuda K. and Somssich I. E. 2015. Transcriptional networks in plant immunity. New Phytologist, **206**: 932-947.

Tyler B., Kabbage M., Williams B. and Dickman M. B. 2013.

Cell Death Control: The Interplay of Apoptosis and Autophagy in the Pathogenicity of Sclerotinia sclerotiorum. PLoS Pathogens, **9**: e1003287.

van Doorn W. G. and Woltering E. J. 2005. Many ways to exit? Cell death categories in plants. Trends Plant Sci., **10**: 117-122.

van Loon L. C., Rep M. and Pieterse C. M. 2006. Significance of inducible defense-related proteins in infected plants. Annu. Rev. Phytopathol., **44**: 135-162.

Walton J. D. 1994. Deconstructing the Cell-Wall. Plant Physiol., **104**: 1113-1118.

Wang J. and Bayles K. W. 2013. Programmed cell death in plants: Lessons from bacteria? Trends Plant Sci., **18**: 133-139.

Wang S., Sun Z., Wang H., Liu L., Lu F., Yang J., Zhang M., Zhang S., Guo Z., Bent Andrew F. and Sun W. 2015. Rice OsFLS2-Mediated Perception of Bacterial Flagellins Is Evaded by Xanthomonas oryzae pvs. oryzae and oryzicola. Mol. Plant, **8**: 1024-1037.

Wang Y.-S., pi L.-Y., Chen X., Chakrabarty P. K., Jiang J., Leon A. L., Liu G.-Z., Li L., Benny U., Oard J., Ronald P. C. and Song W.-Y. 2006. Rice XA21 Binding Protein 3 Is a Ubiquitin Ligase Required for Full *Xa21*-Mediated Disease Resistance. The Plant Cell, **18**: 3635-3646.

Waszczak C., Carmody M. and Kangasjärvi J. 2018. Reactive Oxygen Species in Plant Signaling. Ann. Review Plant Biol., **69**: 209-236.

Wu K., Heil M., Ibarra-Laclette E., Adame-Álvarez R. M., Martínez O., Ramirez-Chávez E., Molina-Torres J, and Herrera-Estrella L. 2012. How Plants Sense Wounds: Damaged-Self Recognition Is Based on Plant-Derived Elicitors and Induces Octadecanoid Signaling. PLoS One, **7**: e30537.

Xie K., Chen J., Wang Q. and Yang Y. 2014. Direct Phosphorylation and Activation of a Mitogen-Activated Protein Kinase by a Calcium-Dependent Protein Kinase in Rice. The Plant Cell, **26**: 3077-3089.

Yamada S., Kano A., Tamaoki D., Miyamoto A., Shishido H., Miyoshi S., Taniguchi S., Akimitsu K. and Gomi K. 2012. Involvement of OsJAZ8 in Jasmonate-Induced Resistance to Bacterial Blight in Rice. Plant Cell Physiol., **53**: 2060-2072.

Yamaguchi K., Yamada K., Ishikawa K., Yoshimura S., Hayashi N., Uchihashi K., Ishihama N., Kishi-Kaboshi M., Takahashi A., Tsuge S., Ochiai H., Tada Y., Shimamoto K., Yoshioka H. and Kawasaki T. 2013. A Receptor-like Cytoplasmic Kinase Targeted by a Plant Pathogen Effector Is Directly Phosphorylated by the Chitin Receptor and Mediates Rice Immunity. Cell Host Microbe, **13**: 347-357.

Yang C., Yu Y., Huang J., Meng F., Pang J., Zhao Q., Islam A., Xu N., Tian Y. and Liu J. 2019. Binding of the Magnaporthe oryzae chitinase MoChia1 by a rice tetratricopeptide repeat protein allows free chitin to trigger immune responses. The Plant Cell, tpc. 00382.02018.

Yang D. L. 2009. The phytohormonal signaling pathways in rice immune responses and Jasmonate signaling pathway represses Gibberellin signaling pathway. Ph.D. Thesis, Chinese Acad. Sci., D2009-D2118.

Yang D. L., Yao J., Mei C. S., Tong X. H., Zeng L. J., Li Q., Xiao L. T., Sun T. P., Li J., Deng X. W., Lee C. M., Thomashow M. F., Yang Y., He Z. and He S. Y. 2012. Plant hormone jasmonate prioritizes defense over growth by interfering with gibberellin signaling cascade. Proc. Natl. Acad. Sci., U S A, **109**: E1192-1200.

Yang Y., Qi M. and Mei C. 2004. Endogenous salicylic acid protects rice plants from oxidative damage caused by aging as well as biotic and abiotic stress. The Plant J., **40**: 909-919.

You Q., Zhai K., Yang D., Yang W., Wu J., Liu J., Pan W., Wang J., Zhu X., Jian Y., Liu J., Zhang Y., Deng Y., Li Q., Lou Y., Xie Q. and He Z. 2016. An E3 Ubiquitin Ligase-BAG Protein Module Controls Plant Innate Immunity and Broad-Spectrum Disease Resistance. Cell Host Microbe, **20**: 758-769.

Yuan B., Shen X., Li X., Xu C. and Wang S. 2007a. Mitogen-activated protein kinase OsMPK6 negatively regulates rice disease resistance to bacterial pathogens. Planta, **4**: 953-960.

Yuan Y., Zhong S., Li Q., Zhu Z., Lou Y., Wang L., Wang J., Wang M., Li Q., Yang D. and He Z. 2007b. Functional analysis of rice *NPR1*-like genes reveals that *OsNPR1/NH1* is the rice orthologue conferring disease resistance with enhanced herbivore susceptibility. Plant Biotechnol. J., **5**: 313-324.

Zeng L.-R., Qu S., Bordeos A., Yang C., Baraoidan M., Yan H., Xie Q., Nahm B. H., Leung H. and Wang G.-L. 2004. *Spotted leaf11*, a Negative Regulator of Plant Cell Death and Defense, Encodes a U-Box/Armadillo Repeat Protein Endowed with E3 Ubiquitin Ligase Activity. The Plant Cell, **16**: 2795-2808.

Zhang S., Chen C., Li L., Meng L., Singh J. and Jiang N. 2005. Evolutionary expansion, gene structure, and expression of the rice wall-associated kinase gene family. Plant Physiol., 139.

Zhao M. 2012. WRKY transcription factor superfamily: Structure, origin and functions. African J. Biotechnol., 11.

Zhou B. and Zeng L. 2017. Conventional and unconventional ubiquitination in plant immunity. Mol. Plant Pathol., **18**: 1313-1330.