Review

Plant innate immunity: An updated insight into defense mechanism

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Plants are invaded by an array of pathogens of which only a few succeed in causing disease. The attack by others is countered by a sophisticated immune system possessed by the plants. The plant immune system is broadly divided into two, viz. microbial-associated molecular-patterns-triggered immunity (MTI) and effector-triggered immunity (ETI). MTI confers basal resistance, while ETI confers durable resistance, often resulting in hypersensitive response. Plants also possess systemic acquired resistance (SAR), which provides long-term defense against a broad-spectrum of pathogens. Salicylic-acid-mediated systemic acquired immunity provokes the defense response throughout the plant system during pathogen infection at a particular site. Trans-generational immune priming allows the plant to heritably shield their progeny towards pathogens previously encountered. Plants circumvent the viral infection through RNA interference phenomena by utilizing small RNAs. This review summarizes the molecular mechanisms of plant immune system, and the latest breakthroughs reported in plant defense. We discuss the plant–pathogen interactions and integrated defense responses in the context of presenting an integral understanding in plant molecular immunity.

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1. Introduction

Phyto-pathogenesis is a global problem, posing a serious threat to food security. Phytopathogens are broadly divided into those that kill the host and feed on the nutritive materials (necrotrophs), those that require a living host to continue their life cycle (biotrophs) and those which require a living host initially, but kill at later stage of infection (hemibiotrophs) (Hammond-Kossack and Jones 2000). Bacteria and fungi adopt either a biotrophic or necrotrophic mode of infection while viruses are ideal biotrophs although viral infection can consequently result in host cell death (Dangl and Jones 2001). Among the 7100 classified bacterial species, roughly 150 species cause diseases to plants (Buonaurio 2008), about 8000 species of fungi and fungal-like organisms are phytopathogens (Ellis *et al.* 2008) and there are 73 genera and 49 families of plant pathogenic viruses (Zaitlin and Palukaitis 2000).

Since plant innate immunity is capable of recognizing potential invading pathogens and mount successful defences using sophisticated mechanisms, all the pathogens which invade the host could not cause disease, although disease outbreak is likely only when the pathogens are able to evade recognition or suppress host defense mechanism or both (Hammond-Kossack and Jones 2000). Interactions of pathogens with plants can either be incompatible or compatible. The former occurs when the pathogen encounters a non-host plant (non-host resistance) or a resistant host plant (cultivar-specific resistance), while the latter occurs when the pathogen infects susceptible host plants resulting in the development of disease symptoms (Heath 2000).

Although recent studies on the plant–pathogen interactions and integrated defense response has (1) helped in elucidating the signalling mechanisms by which the plant cell cope with a stress situation, (2) provided many sustainable practical solutions for the control of diseases in agricultural crops and (3) lead to the discovery of how the organisms from different kingdoms communicate with one another, a majority of the mechanism underlying in defense response still remains elusive.

Keywords. Effector-triggered immunity; microbial-associated molecular-patterns-triggered immunity; plant immunity; RNAi; systemic acquired resistance; trans-generational immune memory

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Although the topic is broad, with abundance of published research and reviews (Dangl and Jones 2001; Cohn *et al.* 2001; Chisholm *et al.* 2006; Jones and Dangl 2006; Dodds and Rathjen 2010), this review summarizes the recent reports and precise findings on the defense strategies employed by the plant to tackle pathogenesis and offers an integral understanding of plant molecular immunity.

2. Pathogen entry and infection sites

Microbial entry into the host tissue is a vital step in causing infection. The plant interior should be invaded by the pathogens to establish their virulence. This is achieved either by direct penetration of the plant surface or else by entry through physical injuries or natural openings such as stomata. Although pathogens can specifically colonize leaves, roots, fruits or particular cell types such as root epidermal or phloem cells of the vasculature, their entry into the host often takes place at a distant site (Lefert and Robatzek 2006).

Bacteria move towards the host by aerotaxis and chemotaxis (Chet *et al.* 1973; Raymundo and Ries 1980) and enter into the plant through trichomes (Layne 1967), lenticels (Fox *et al.* 1971), stomata (Getz *et al.* 1983; Melotto *et al.* 2006), hydathodes (Mew *et al.* 1984), lateral root and wounds (Huang 1986), and once inside the plant system, bacteria inhabit in the apoplast (Jones and Dangl 2006). Phytopathogenic fungi use modified hyphae as infection structures, dedicated for the invasion of plant tissues. The fungi adhere to the cuticle and direct the growth of a germ tube on the plant surface followed by the formation of appressoria. The appressoria develops a higher turgor pressure to support the penetration process (Howard *et al.* 1991). A penetration peg arises from a pore in the middle of the appressorial base (Mendgen *et al.* 1996). Supported by enzymes that soften the host cell wall, the hypha enters the leaf epidermal cell and gets differentiated into bulbous and lobed infectious hypae, which grow intra- and intercellularly (Nakao *et al.* 2011). A few biotrophic fungi such as *Cladosporium fulvum* do not form haustoria but instead grow exclusively in apoplast, subsisting on leaked nutrients (Thomma *et al.* 2005).

Viral entry is only possible through physical injuries induced either by environmental factors or by vectors. Once inside the cell, the virus mobilizes locally and systematically through intracellular (symplastic) movement through the plasmodesmata, which may occur in either virion or non-virion form. Movement proteins (MPs) and other virus-encoded factors assist virus movement inside the plant system in a coordinated and regulated manner, by acting together with the host components (Niehl and Heinlein 2010). Vector-mediated viral infection is facilitated by sap-sucking species of arthropods, which can deliver virus particles directly into the vasculature and thus rapidly disseminating the virus throughout the plant (Hammond-Kossack and Jones 2000).

3. Plant defense mechanisms

Entry of pathogens and the subsequent activation of inducible defense responses or disease symptom development is primarily prevented by performed barriers on the plant surface such as wax layers, rigid cell walls, cuticular lipids (Reina-Pinto and Yephremov 2009), antimicrobial enzymes (Habib and Fazili 2007) or secondary metabolites (Ahuja *et al.* 2012; Bednarek 2012). Pathogens which overcome these defensive layers (*as discussed in the previous section*) are systematically encountered and defense response is elicited by two interconnected mechanisms, namely, microbial (or pathogen)-associated molecular patterns (MAMP/PAMP)-triggered immunity (MTI/PTI) and effector-triggered immunity (ETI). Since the term PAMPs was misleading as this line of defense does not distinguish between mutualistic and parasitic symbiosis, Staal and Dixelius (2007) reinstated the term PAMPs with MAMPs (MTI), which will be followed throughout this review. An overall schematic representation of the plant defense mechanisms was given in figure 1.

3.1 *Microbial-associated molecular patterns (MAMP)-triggered immunity (MTI)*

Precisely, MTI (formerly called basal or horizontal immunity) involves the recognition of conserved, indispensable microbial elicitors called microbial-associated molecular patterns (MAMPs) by a class of plasma-membrane-bound extracellular receptors called pattern recognition receptors (PRRs) (Dodds and Rathjen 2010; Beck *et al.* 2012) and the activation of these PRRs results in active defense responses (Hammond-Kosack and Jones 1996), which ultimately contribute to halt infection before the microbe gains a hold in the plant.

3.1.1 *MAMPs/DAMPs:* MAMPs are essential structures or components that are conserved throughout the whole classes of pathogens (Felix *et al.* 1993). This includes oligogalacturonides (Galletti *et al.* 2008), ergosterol (Granado *et al.* 1995), bacterial flagellin (Felix *et al.* 1999), Pep-13 (Brunner *et al.* 2002), xylanase (Belien *et al.* 2006), cold-shock protein (Felix and Boller 2003) and lipopolysaccharides (LPS) (Dow *et al.* 2000). Recognition of particular MAMPs and subsequent elicitation of immune responses are confined to plant species belonging to a single family (Felix and Boller 2003; Ron and Avni 2004). For instance, EF-Tu recognition is reported only in the *Brassicaceae* family (Kunze *et al.* 2004). Conversely, other MAMPs including chitin, LPS and flagellin trigger immune responses in various host

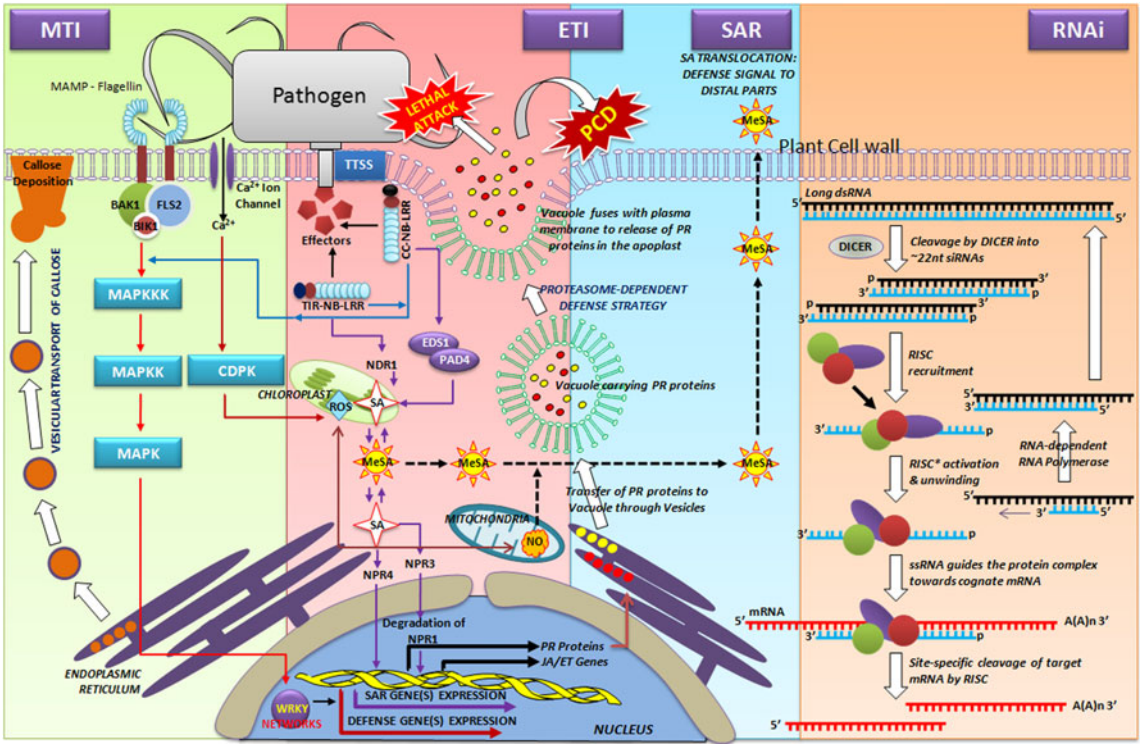

Figure 1. Schematic representation of all the four modes of plant immunity: (a) MAMP-triggered immunity (MTI); (b) effector-triggered immunity (ETI); (c) systemic acquired resistance (SAR); (d) gene silencing. ETI illustration includes the events involved in cell-autonomous immune system based on membrane fusion for attacking intercellular bacteria and inducing local necrosis (hypersensitive response-programmed cell death).

species, even if there is some degree of specificity and perception efficacy for a plant family or species (Zipfel *et al.* 2006).

Many plant pathogens produce lytic enzymes to infringe the structural barriers of plant tissues. The products such as cell wall fragments (Darvill and Albersheim 1984), cutin monomers (Kauss *et al.* 1999) and peptides (Boller 2005; Huffaker *et al.* 2006), which are generated as a consequence, can function as endogenous elicitors called damage-associated molecular patterns (DAMPs). These DAMPs characteristically emerge in the apoplast and serve as danger signals to induce innate immunity similar to MAMPs (Henry *et al.* 2012).

3.1.2 *Pattern recognition receptors:* Plant receptors perceiving MAMPs/DAMPs can be divided into surface and intracellular receptors. The latter function exclusively in ETI and are therefore detailed in the next section (ETI). The surface receptors called PRRs are known to detect both MAMPs and effectors. The PRR family includes receptor-like kinases (RLK) and receptor-like proteins (RLP). RLK resides in plasma membrane and comprises of a putative extracellular ligand-binding domain, a single transmembrane domain (Shiu and Bleecker 2001), and an intracellular serine/threonine kinase domain. Plant RLKs are structurally related to animal receptor-tyrosine kinases (RTKs) (Shiu and Bleecker 2001). Similarly, RLP consists of an extracellular domain and a membrane-spanning domain. As they lack an intracellular activation domain, and consequently, they require interaction with adaptor molecules for signal transduction (Zipfel 2008). RLPs are structurally reminiscent of the toll-like receptors (TLR) that mediate MAMP recognition in animals (Kopp and Medzhitov 2003).

3.1.3 *MAMP recognition:* Although various MAMPs and their corresponding PRRs have been reported, only a few are well characterized (table 1). Some of the best exemplified MAMP-PRR recognitions are Flg22:FLS2, EF-Tu:EFR and Ax21:Xa21. FLS2 directly interacts with flg22, a 22-amino-acid peptide derived from the amino terminus of flagellin (Gómez-Gómez and Boller 2000; Chinchilla *et al.* 2006), EFR specially perceives the elf18 peptide, the first 18 amino acid sequence of the N-terminus of EF-Tu (Kunze *et al.* 2004), and Xa21 recognizes Ax21, a sulfated 17-amino acid peptide derived from the amino terminus of *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) type I secreted protein (Lee *et al.* 2009).

Lysin motif receptor kinases (LysM-RKs) are a kind of RLKs with extracellular LysM and intracellular Ser/Thr kinase domain. These LysM-RKs play a critical role in

Table 1. MAMPs/DAMPs and their corresponding PRRs

Source	MAMPs/DAMPs	Epitope	PRR	PRR Type	References
Bacteria	Flagellin	flg22	FLS2	LRR-RLK	Gómez-Gómez *et al.* 2001
	Cold-shock proteins	RNP-1 motif	*unidentified*	*unidentified*	Felix and Boller 2003
	Xoo derived	*unidentified*	Xa26	LRR-RLK	Sun *et al.* 2004
	Ef-Tu	elf8	EFR	LRR-RLK	Zipfel *et al.* 2006
	Peptidoglycan hairpin	HrpZ	*unidentified*	*unidentified*	Erbs *et al.* 2008
	Ax21	axY^{s}22	Xa21	LRR-RLK	Lee *et al.* 2009
	lipopolysaccharides	*unidentified*	*unidentified*	*unidentified*	Erbs and Newman 2011
	Peptidoglycan	PGN	LYM1 & LYM3	LysM-RLP	Willmann *et al.* 2011
Fungi	Ergosterol	*unidentified*	*unidentified*	*unidentified*	Granado *et al.* 1995
	Cerebrosides	sphingoid base	*unidentified*	*unidentified*	Umemura *et al.* 2002
	Xylanase	TKLGE pentapeptide	LeEIX2	LRR-RLP	Ron and Avni 2004
	Necrosis-inducing proteins (NLP)	*unidentified*	*unidentified*	*unidentified*	Mattinen *et al.* 2004
	Chitin	chitin oligosaccharides	CERK1	LysM-RLK	Shimizu *et al.* 2010
	β-glucan	β-heptaglucan	GEBP	*unidentified*	Kishimoto *et al.* 2011
Oomycetes	β-glucan	hepta-β-glucoside	GnGBP	*unidentified*	Klarzynski *et al.* 2000
	Lipid-transfer proteins (elicitins)	*unidentified*	*unidentified*	*unidentified*	Osman *et al.* 2001
	Transglutaminase	Pep13	*unidentified*	*unidentified*	Brunner *et al.* 2002
	Cellulose-binding elicitor lectin (CBEL)	*unidentified*	*unidentified*	*unidentified*	Gaulin *et al.* 2006
DAMPs	Prosystemin	systemin	SR160	LRR-RLK	Scheer and Ryan 2002
	PEPR1	Pep1	PEPR1	LRR-RLK	Krol *et al.* 2010
	Homoglacturonan	oligogalacturonides	WAK1	EGF-RLK	Brutus *et al.* 2010

perception of fungal cell wall component, chitin. A representative example is chitin elicitor receptor kinase (CERK1) of *Arabidopsis* with three extracellular LysMs (Miya *et al.* 2007). The AtCERK1 directly binds with the chitin oligosaccharide elicitor (Iizasa *et al.* 2010; Petutschnig *et al.* 2010) and gets activated through chitin-induced dimerization (Liu *et al.* 2012b). An *Arabidopsis* CERK1 homolog, OsCERK1, is also reported to be essential for chitin signalling in rice (Shimizu *et al.* 2010). Recently, two homologous LysM-RKs, namely LYP4 and LYP6, functional in sensing both fungal chitin and bacterial peptidoglycan (PGN), were reported in rice (Liu *et al.* 2012a).

Plant PRRs can also recognize DAMPs in an approach similar to MAMPs and activate the defense signalling cascade. *Arabidopsis* PEP receptor 1 (PEPR1) was the earliest receptor for DAMPs perception reported, which belongs to the LRR-RLK family. AtPEPR1 can specifically recognize *Arabidopsis* Pep1, a 23-amino-acid peptide derived from the C-terminus of a pathogen/wound-induced gene product, PROPEP1, and activate the downstream signalling cascade (Krol *et al.* 2010).

3.1.4 *Signal transduction:* MAMPs-induced rapid heteromerization of PRRs is the earliest event in activating the MTI signalling downstream. Being functional kinases, phosphorylation events in PRRs are regarded as important regulatory mechanisms in MTI signalling. In Flg22:FLS2 interaction, the kinase domain of FLS2 is rapidly phosphorylated by stimulation with flg22 peptide (Gómez-Gómez *et al.* 2001) and the phosphorylated FLS2 instantaneously dimerizes with brassinosteroid intensitive 1 (BRI1)-associated kinase (BAK1) and *Botrytis*-induced kinase 1 (BIK1) (Chinchilla *et al.* 2007; Heese *et al.* 2007; Schulze *et al.* 2010). The rapid FLS2-BAK1 association (in less than 2 min) proposes the existence of BAK1 in a preformed complex at the membrane, weakly associated with FLS2. Mutual transphosphorylation of the kinase domains of BIK1 and FLS2/BAK1 (Lu *et al.* 2010; Schulze *et al.* 2010) results in conformational changes and ultimately phosphorylated BIK1 is released to activate signalling component downstream (Laluk *et al.* 2011; Wang 2012; Belkhadir *et al.* 2012).

In *Arabidopsis,* the discharged BIK1 activates two simultaneous MAPK cascades downstream, MKK4/MKK5-MPK3/MPK6 and MEKK1/MKK1/MKK2-MPK4 (Asai *et al.* 2002; Meszaros *et al.* 2006; Suarez-Rodriguez *et al.* 2007; Gao *et al.* 2008), leading to the activation of WRKY family of transcription factors (Pandey and Somssich 2009). WRKYs are of approximately 60 amino acids in length, with a conserved

region of WRKYGQK along with a unique zinc-finger pattern of Cys and His residues (Rushton *et al.* 1996). Being a DNA binding domain, these WRKYs interacts with the W-box (TTGACC/T) motif present in promoters of defense-associated genes (Navarro *et al.* 2004) and activates the expression of early defense-related genes (Ishihama and Yoshioka 2012).

3.1.5 *Defense response:* The initial defense response elicited by plant cell cultures in response to MAMPs is the alkalinization of the growth medium. Occurring 0.5 to 2 min after elicitation, this event relies on drastic changes in fluxes of H^+, K^+, Cl_2 and Ca^{2+} ions across the plasma membrane (Jabs *et al.* 1997; Garcia-Brugger *et al.* 2006). Elevation of cytoplasmic Ca^{2+} levels is a critical step in MTI response and is mediated by accumulation of Ca^{2+} in the thylakoid lumen of chloroplasts followed by increased Ca^{2+} influx in the cytoplasm (Nomura *et al.* 2012). This was accomplished by activation of a plasma membrane Ca^{2+}-conducting channel through PRR phosphorylation, G-protein molecular switch and the activation of cyclic nucleotide gated channels (Ali *et al.* 2007; Qi *et al.* 2010). Changes in $[Ca^{2+}]_{cyt}$ is perceived by calcium-binding proteins such as calmodulin, calcium-dependent protein kinases, and calcineurin B-like proteins (Reddy and Reddy 2004). Ca^{2+} elevation in cytosol plays a pivotal role in mediating other plant immune processes, including control of reactive oxygen species (ROS), salicylic acid (SA) production and stomatal closure (Chiasson *et al.* 2005; Kotchoni and Gachomo 2006; Takabatake *et al.* 2007; Nomura *et al.* 2008; Ogasawara *et al.* 2008; Wang *et al.* 2009; Du *et al.* 2009). Recently, calcium-dependent protein kinases (CDPKs), acting as Ca^{2+} sensor protein kinases, were reported to be major mediators of the early MTI immune signalling (Boudsocq *et al.* 2010).

The production of reactive nitrogen intermediates (RNIs) and ROS at the cell surface, known as the nitrosative burst and oxidative burst respectively, is one of the earliest events detectable attributing to plant defense. The ROS synthesis pathway has been deciphered but the sources of RNI remain elusive. Yun *et al.* (2011) reported a covalent attachment of the nitric oxide (NO) moiety to a protein cysteine thiol to form an *S*-nitrosothiol (SNO), a redox-based post-translational modification, which proceeds through a cascade of biochemical pathways to produce RNI. During higher concentrations of *S*-nitrosothiols, the NO directs a negative feedback loop mediated by *S*-nitrosylation of the NADPH oxidase (at Cys 890), thus restricting the HR by inhibiting the synthesis of ROS. The cys 890 was reported to be evolutionarily conserved and exclusively *S*-nitrosylated in both human and *Drosophila* NADPH oxidase, signifying that this system may govern immune responses in both plants and animals (Yun *et al.* 2011). ROS are potentially toxic analogous of reduced oxygen forms, such as the superoxide anion and hydrogen peroxide. They are considered to exert antimicrobial action through in direct microbicidal actions, strengthening of the cell wall through oxidative cross linking of glycoproteins, induction of intracellular signalling pathway such as the synthesis of SA and activation of MAPK cascade, or activation of SAR associated with systemic propagation of the oxidative burst (Lamb and Dixon 1997; Nuhse *et al.* 2007).

The accumulation of callose, a plant β-1,3-glucan polymer synthesized between the cell wall and the plasma membrane to limit the penetration of pathogens, is a conventional indicator of MTI (Bestwick *et al.* 1995; Brown *et al.* 1998). The rapid MAPK cascade triggered by Flg22 recognition stimulates the generation of RBOHD-dependent ROS, which subsequently promote deposition of PMR4-dependent callose (Zhang *et al.* 2007). The report on regulation of Flg22-induced callose synthesis by glucosinolate metabolites adds a novel layer to signalling pathways controlling MTI response (Clay *et al.* 2009). The signalling and biochemical cascade taking place during callose synthesis varies in accordance to type and property the MAMPs. It was also reported that multiple signalling pathways regulates callose deposition rather than one conserved downstream pathway (Luna *et al.* 2011).

MTI also includes closure of stomata in response to bacterial pathogen (Melotto *et al.* 2006). The mechanism involves the elevation of free cytosolic Ca^{2+}(Ca^{2+}_i) via a CAS signalling pathway. A high extracellular calcium (Ca^{2+}_o) level induces H_2O_2 and NO accumulation in guard cells, which further triggers Ca^{2+}_i transients and finally results in stomatal closure (Zhang *et al.* 2008; Wang *et al.* 2012). The decrease in photosynthesis in response to infection is another defense strategy employed by plants to dodge the pathogens (Bolton 2009). Göhre *et al.* (2012) demonstrated that activation of defense by MAMPs leads to a rapid decrease in non-photochemical quenching (NPQ), thus limiting carbon source availability for the pathogen.

MTI also involves the biosynthesis of salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) (Mishina and Zeier 2007; Tsuda *et al.* 2008), which are indispensable for both local and systemic acquired resistances (Durrant and Dong 2004). This response showed great similarity to *R*-gene-mediated defense (ETI) (Navarro *et al.* 2004), substantiating the fact that MTI and ETI extensively share downstream signalling machinery mediated by an integrated signalling network (Tsuda and Katagiri 2010; Göhre *et al.* 2012).

3.2 *Effector-triggered immunity (formerly called R-gene-based or vertical immunity)*

Successful pathogens have evolved strategies to challenge the MTI and consequently promote pathogenesis by injecting a battery of effector proteins across the plant cell wall

into the cytoplasm through the type III secretion system (TTSS) (Cornelis and van Gijsegem 2000; Grant *et al.* 2006; Deslandes and Rivas 2012). A virulent bacterium delivers about 15–30 type III secreted effectors (TTSEs) into host cells, which contribute pathogen virulence by (1) acting as transcription factors that directly activate transcription in host cells, (2) affecting histone packing and chromatin configuration and/or (3) directly targeting host transcription factor activity and ultimately promoting the release of nutrients required for the survival of pathogen (Feng and Zhou 2012). These effectors were identified originally not by their promotion of virulence but rather by their 'avirulence' activity (Collmer *et al.* 2000). Moreover, they are extremely diverse with little amino acid sequence similarity among them (White *et al.* 2000). Typical examples are *Pseudomonas syringae* TTSEs AvrPto, AvrPtoB, HopF2 and HopAI1, which suppress the FLS2-mediated MTI by directly targeting different sites in Flg22:FLS2 signalling cascade (Zhang *et al.* 2007; Rosebrock *et al.* 2007; Xiang *et al.* 2008; Göhre *et al.* 2008; Wang *et al.* 2010a).

The effectors (Avr proteins) are recognized in a highly specific fashion by the products of host disease resistance (*R*) genes. *R* genes have been shown to control the outcome of plant-pathogen interactions in a great diversity of hosts against an extensive list of pathogens (Martin *et al.* 2003).

3.2.1 *Resistance proteins:* Like most of the PRRs belong to the RLK family, a majority of R proteins belongs to the intracellular nucleotide-binding leucine-rich repeat (NB-LRR) protein family (Jones and Dangl 2006), which have striking similarities with animal NOD-like receptor or CATERPILLER proteins (Inohara and Nunez 2003; Rairdan and Moffett 2007). Plant NB-LRRs contain a C-terminal LRR domain, a varying N-terminal effector domain, and a central NB domain. The latter regulates the conformation and signalling activity of these proteins (Takken *et al.* 2006). Based on the N-terminal architecture, the NB-LRRs are divided into two subclasses: coiled-coil (CC) motif and toll/interleukin 1 receptor domain (TIR) (Eitas and Dangl 2010).

A second major class of R proteins belong to the extracellular LRR (eLRR) proteins group, which was classified into three subclasses according to their domain structures (Fritz-Laylin *et al.* 2005). These subclasses comprise RLP (receptor-like proteins; extracellular LRR and transmembrane [TM] domain), RLK (extracellular LRR, TM domain, and cytoplasmic kinase) and PGIP (polygalacturonase inhibiting protein; cell wall LRR) (Wang *et al.* 2010b). Comprehensive data of effector molecules, their sources, targets, corresponding R-proteins and R-protein structure is tabulated in table 2.

3.2.2 *Effector recognition and signalling:* NB-LRRs recognize effectors by either of the two modes: (1) direct physical interaction between the receptor and effector or (2) indirect interaction mediated by accessory-proteins that the immune receptor associates with and scrutinizes for effector-induced modifications. These indirect recognition-mediating accessory proteins may either be genuine virulence targets of the effector (the guard model) or decoy proteins that the plant has evolved to mimic its respective effector targets (the decoy model) (Dangl and Jones 2001; Hoorn and Kamoun 2008; Dodds and Rathjen 2010).

In the absence of effectors, NB-LRRs maintain an ADP-binding inactive state through intra-molecular interactions between their different domains and/or extra-molecular interaction with other host protein(s). During direct interaction, the effectors induce primary conformational changes of NB-LRRs, which is prone to ADP/ATP exchange. Nucleotide exchange then triggers a second conformational change that enables the NB-LRRs' N-terminus (TIR, CC) to interact with and activate downstream targets (Takken and Tameling 2009).

Indirect recognition of effectors by NB-LRRs was tacit on examining the role of conserved plant protein RIN4. *Arabidopsis* RIN4 is targeted by multiple bacterial effectors (AvrRpt2, AvrRpm1, AvrB and HopF2) and is monitored for effector-induced modification by two NB-LRRs (RPS2 and RPM1) (Leister *et al.* 1996; Mackey *et al.* 2002; Kim *et al.* 2005). AvrRpm1 and AvrB induce phosphorylation of RIN4; and AvrRpt2, being a cysteine protease, cleaves RIN4 and induces RIN4 degradation. The phosphorylation and proteolysis of RIN4 then respectively activate RPM1- and RPS2-mediated ETI (Mackey *et al.* 2003; Axtell and Staskawicz 2003; Wilton *et al.* 2010). The sites of RIN4 binding with and modified by AvrRpt2, AvrRpm1 and AvrB overlap in a short C-terminal nitrate-induced domain, including amino acids 142–176. Recently, it was reported that the phosphorylation of RIN4 at a position of threonine 166 is necessary for AvrB-triggered RPM1 activation (Chung *et al.* 2011; Liu *et al.* 2011a).

Various reports demonstrate that the activated NB-LRRs exert their activities in the nucleus (Burch-Smith *et al.* 2007; Shen *et al.* 2007). The CC-NB-LRR MLA10 (barley) and TIR-NB-LRRs RRS1-R (*Arabidopsis*), N (tobacco), RPS4 (*Arabidopsis*) and SNC1 (*Arabidopsis*) need nuclear localization and accumulation for complete activation of immunity (Deslandes *et al.* 2003; Wirthmueller *et al.* 2007; Shen *et al.* 2007; Burch-Smith *et al.* 2007; Cheng *et al.* 2009). The involvement of NB-LRRs in directly regulating the defense gene expression is evidenced by the presence of a WRKY DNA-binding domain in RRS1-R along with MLA10 and N with WRKY and SPL transcription factors, respectively (Caplan *et al.* 2008). These findings suggest that NB-LRR signalling might not employ many components, but rather take place immediately in the nucleus (Altenbach and Robatzek 2007; Cheng *et al.* 2009).

Table 2. Effector molecules, their sources, targets, corresponding R-proteins and R-protein structure

Pathogen	Effector	Target	R-protein	R-protein structure	References
Blumeria graminis	Avr10	*unidentified*	MLA10	CC-NB-LRR	Ridout *et al.* 2006
Cladosporium fulvum	Avr2	RCR3	*unidentified*	*unidentified*	Rooney *et al.* 2005
Cladosporium fulvum	Avr4	Chitinase	*unidentified*	*unidentified*	van den Burg *et al.* 2006
Hyaloperonospora parasitica	ATR13	*unidentified*	RPP13	CC-NB-LRR	Allen *et al.* 2004
Hyaloperonospora parasitica	ATR1	*unidentified*	RPP1	TIR-NB-LRR	Rehmany *et al.* 2005
Magnaporthe grisea	AvrPi-ta	*unidentified*	Pi-ta	CC-NB-LRR	Jia *et al.* 2000
Melampsora lini	AvrL567	*unidentified*	L5/L6/L7	TIR-NB-LRR	Dodds *et al.* 2006
Phytophthora infestans	Avr3a	CMPG1	*unidentified*	*unidentified*	Bos *et al.* 2010
Potato virus X	Coat protein	*unidentified*	Rx	CC-NB-LRR	Rairdan and Moffett 2007
Pseudomonas syringae	AvrPtoB	FLS2, BAK1, FEN, CERK1	Prf	CC-NB-LRR	Göhre *et al.* 2008
Pseudomonas syringae	AvrPphB	PBS1	RPS5	CC-NB-LRR	Zhang *et al.* 2010
Pseudomonas syringae	AvrRpt2	RIN4	RPS2	CC-NB-LRR	Coaker *et al.* 2005
Pseudomonas syringae	AvrRpm1	RIN4	RPM1	CC-NB-LRR	Liu et al. 2011a
Pseudomonas syringae	AvrB	RIN4	RPG1-B/TAO1	TIR-NB-LRR	Eitas *et al.* 2008; Liu *et al.* 2011a
Pseudomonas syringae	AvrRps4	*unidentified*	RPS4	TIR-NB-LRR	Hinsch and Staskawicz 1996
Pseudomonas syringae	HopAI-1	MPK3, MPK6	*unidentified*	*unidentified*	Zhang *et al.* 2007
Pseudomonas syringae	HopU1	GRP7	*unidentified*	*unidentified*	Jeong *et al.* 2011
Pseudomonas syringae	HopM1	AtMIN7	*unidentified*	*unidentified*	Nomura *et al.* 2006
Pseudomonas syringae	HopAO1	Downstream of PTI signalling	*unidentified*	*unidentified*	Underwood *et al.* 2007
Pseudomonas syringae	HopO1–1	Chloroplast protein	*unidentified*	*unidentified*	Fu *et al.* 2007
Pseudomonas syringae	HopO1–2	Chloroplast protein	*unidentified*	*unidentified*	Fu *et al.* 2007
Pseudomonas syringae	HopU1	AtGrp7, AtGrp8	*unidentified*	*unidentified*	Fu *et al.* 2007
Ralstonia solanacearum	PopP2	*unidentified*	RRS1-R	TIR-NB-LRR	Deslandes *et al.* 2003
Tobacco mosaic virus	P50	*unidentified*	N/NRG1	TIR-NB-LRR	Burch-Smith *et al.* 2007
Xanthomonas campestris	AvrBs2	*unidentified*	Bs2	NB-LRR	Mudgett *et al.* 2000
Xanthomonas campestris	AvrAC	BIK1, RIPK	*unidentified*	*unidentified*	Feng *et al.* 2012
Xanthomonas campestris	AvrXv4	Cytoplasmic target	*unidentified*	*unidentified*	Roden *et al.* 2004
Xanthomonas campestris	AvrBs3/PthA	UPA-box	*unidentified*	*unidentified*	Kay *et al.* 2007
Xanthomonas oryzae	AvrRxo1	*unidentified*	Rxo1	TIR-NB-LRR	Zhao *et al.* 2004

3.2.3 *Defense responses:* The downstream ETI response events partially overlap with MTI response, including activation of downstream MAPK cascade and activation of WRKY transcription factors. This subsequently induces rapid transcriptional activation of a string of pathogenesis-related (PR) genes in and around the infected cell for the biosynthesis of salicylic acid (SA), jasmonic acid (JA), ethylene (ET), cell wall strengthening, lignifications, production of various antimicrobial compounds in endoplasmic reticulum and secretion into vacuoles (Iwai *et al.* 2006; Nomura *et al.* 2012; Schäfer and Eichmann 2012). Salicylic acid thus accumulated in the infected areas binds to the receptor NPR3 (NONEXPRESSOR OF PR GENES3) with low affinity and mediates the degradation of cell-death suppressor NPR1 (Fu *et al.* 2012), thus leading to the development of hypersensitive response (HR) (Pennell and Lamb 1997; Hayward *et al.* 2009). The HR is a form of programmed cell death (PCD) characterized by cytoplasmic shrinkage, chromatin condensation, mitochondrial swelling, vacuolization and chloroplast disruption (Coll *et al.* 2011).

Two main classes of PCD were described by van Doorn (2011), namely autolytic-PCD and non-autolytic PCD. Autolytic-PCD involves a rapid cytoplasm clearance after tonoplast rupture due to the release of hydrolases from the

vacuole, which degrades the cytoplasm and brings about a localized cell death. This autolytic-PCD is associated with increased caspase-like activities (van Doorn 2011). Caspases are a family of cysteine proteases that cleave their substrates after an aspartic acid residue activates other caspases and degradative enzymes or proteins involved in necrosis. In *Arabidposis*, two metacaspases of type I, namely AtMC1 and AtMC2, were reported to control PCD antagonistically (Coll *et al.* 2010). Non-autolytic PCD involves the absence of a rapid clearance of the cytoplasm even in the persistence of increased permeability of tonoplast. This increased permeability or even rupture of tonoplast does not result in considerable discharge of hydrolases, which instantaneously clear the remaining cytoplasm. The PCD mechanism, as described by Hatsugai *et al.* (2009) was non-autolytic PCD, in which the plasma membrane fuses with the membranes of a large central vacuole mediated by proteasome subunit, resulting in the release of vacuolar antibacterial proteins in the apoplast. The extracellular fluid that was discharged had both antibacterial activity and cell-death-inducing activity, and thus promotes local necrosis and ultimately obstructing the pathogen growth.

3.3 *Systemic acquired resistance*

A systemic defense response in the distal plant parts is activated by the defense mechanism elicited at the infection spot to guard the unharmed tissues against succeeding invasion by the pathogen (Durrant and Dong 2004). This long-lasting and broad-spectrum induced disease resistance is referred to as systemic acquired resistance (SAR) (Durrant and Dong 2004), which requires endogenous accumulation of SA, resulting in transcriptional reprogramming of a battery of genes encoding PR proteins (van Loon *et al.* 2005; Park *et al.* 2007). The SA produced in the infected site as methyl-SA (MeSA) moves cell to cell via plasmodesmata or through the phloem to the rest of the plant (Kiefer and Slusarenko 2003; Park *et al.* 2007). Shulaev *et al.* (1997) suggested that airborne (volatile) MeSA may also participate in long-distance signalling during SAR in tobacco. Once inside the cell, SA binds to the high-affinity receptor NPR4 instead of binding to low-affinity NPR3 and averts the degradation of NPR1. This favours cell survival and expression of systemic immunity-related genes (Fu *et al.* 2012). NPR1 has also been reported to participate in cross talk between SA- and JA-dependent defense pathways, thus facilitating plants to generate suitable immune response, depending on the nature of the pathogen and the stage of infection (Spoel *et al.* 2003; Koornneef and Pieterse 2008; Luna *et al.* 2012).

Although methyl-SA was believed to be the long-distance signal in SAR because of its presence reported in the phloem sap and exudates collected from SAR-induced cucumber and tobacco leaves respectively, other studies using cucumber (Rasmussen *et al.* 1991) and grafting experiments with transgenic tobacco, *Arabidopsis* with reduced levels of methyl-SA strongly suggest that SA is not the mobile signal (Vernooij *et al.* 1994; Attaran *et al.* 2009). This led to the discovery of glycerolipids (Chaturvedi *et al.* 2008), azeleic acid (Jung *et al.* 2009) and glycerol-3-phosphate (Chanda *et al.* 2011) as signalling molecules. As a plausible explanation, Liu *et al.* (2011b) elucidated the participation of at least two mobile signals, namely MeSA, and a complex formed between the lipid transfer protein DIR1 and glycerolipid or lipid derivatives in controlling SAR. Role of MeSA in SAR was light-regulated, where the presence of MeSA and its metabolizing enzymes were reported in SAR elicited during late in the day. Conversely, absence of MeSA was reported in SAR induced in the morning. These findings revealed the multitude of transiently expressed signalling networks mediating the onset of SAR, which were divergent corresponding to the environmental conditions.

The genes activated downstream of SA are classified into two classes, namely, immediate-early genes and genes induced later. The immediate-early genes were provoked within 30 min of SA treatment (Horvath *et al.* 1998), whereas the genes induced later include the SA-marker gene *PR-1* (Lebel *et al.* 1998). Gene expression profiling experiments have illustrated the multifaceted patterns of regulation for a string defense-related genes which are induced or repressed relatively early or late after SA treatment and pathogen infection (Bartsch *et al.* 2006; Blanco *et al.* 2009).

3.4 *RNAi-mediated defense against viruses*

Plants can circumvent viral infection by specifically degrading viral RNA following a preliminary period of infection. This is achieved through RNA interference (RNAi) resulting in gene silencing. Plants exhibit two distinct gene silencing phenomena, namely transcriptional gene silencing (TGS) and post-transcriptional gene silencing (PTGS) (Al-Kaff *et al.* 1998; Lu *et al.* 2003; Padmanabhan *et al.* 2009; Sahu *et al.* 2012a), which uses small regulating RNAs (sRNAs) to specifically target and inactivate invading nucleic acids (Sharma *et al.* 2012).

3.4.1 *Post-transcriptional gene silencing:* The first step of PTGS is initiation in which dsRNA is synthesized from the viral genome either by the RNA-dependent RNA polymerase (RDR) of RNA viruses or by host RNA polymerase II in case of DNA viruses. The dsRNA serves as the substrate for Dicer (DCL), an endoribonuclease (RNase) enzyme, which generates a pool of siRNA (~21–24 nt) by recognizing the ends of dsRNA with its PAZ (Piwi/Argonaute/Zwille) domain and then cleaves the dsRNA with RNase III domains (Berstein *et al.* 2001). The processed siRNAs are carried to the effector component called RNA-induced silencing

complex (RISC). RISC is a ribonucleoprotein complex with an active component termed Argonaute (AGO) proteins, which cleave the target viral mRNA strand complementary to their bound siRNA in the middle of siRNA–mRNA duplex. AGO proteins comprise two homology domains, namely, the PAZ and Piwi domain. Piwi domain possesses the RNaseH activity, which cleaves the target single stranded RNA (Baulcombe 2004; Lindbo and Dougherty 2005) initiated by the elimination of 3′ poly A tail followed by degradation involving major cellular 5′–3′ exonuclease-like Xrn1p (Orban and Izaurralde 2005). Thus, the invading viral RNAs or the transcripts of viral DNA are eliminated through PTGS (Sharma *et al.* 2012). In our recent study, we evidenced a higher accumulation of 21 and 24 nt siRNA corresponding to replication associated proteins gene (Rep) region of the *Tomato leaf curl New Delhi virus* (ToLCNDV) and is responsible for resistance against the virus (Sahu *et al.* 2010; 2012a), which substantiates the existence of PTGS phenomena in tomato cultivars.

3.4.2 *Transcriptional gene silencing:* DNA cytosine methylation at carbon 5 of the pyrimidine ring [5-methylcytosine (5-meC)] is a prime epigenetic event in the defense response towards viruses (Lister *et al.* 2008). During RNA-directed DNA methylation (RdDM), the production of 24-nt heterochromatic siRNA involves Pol IV, a specialized polymerase evolved from Pol II which generates single-stranded transcripts from the viral genome (Huang *et al.* 2009). These ssRNA are converted into dsRNA by RDR2 and subsequently diced by DCL3 to generate 24-nt siRNA. These siRNAs are incorporated into AGO4-containing RNA-induced transcriptional silencing (RITS) complex and act as a guiding strand for heterochromatin formation and methylation. The AGO4 imparts chromatin modification either by cytosine methylation or by histone methylation. In plants, it has been reported that the viral genome is targeted for methylation through 24-nt siRNAs during infection. Various reports highlighted the existence of TGS, mediated by the methylation in the intergenic region of *Mungbean yellow mosaic India virus* (Yadav and Chattopadhyay 2011) and ToLCNDV (Sahu *et al.* unpublished).

The discovery of dsRNA being the inducer of RNA silencing in plants has led to the development of a more robust and effective inverted-repeat transgene system for engineering virus resistance in plants (Lu *et al.* 2003; Burch-Smith et al. 2006; Godge *et al.* 2007; Yamagishi and Yoshikawa 2010; Ma *et al.* 2012; Sahu *et al.* 2012b). Moreover, it is interesting to note that the RNA silencing signals spread from cell to cell through plasmodesmata and over long distances via the phloem (Ruiz-Medrano *et al.* 2004), similar to SAR.

3.4.3 *Viral counterparts of silencing suppression:* Although RNA silencing has evolved to be a potential antiviral defense strategy, most of the plant viruses encode at least one suppressor protein to circumvent the defense mechanism. Thus, viruses intrude with the host silencing machinery, resulting in increased viral replication and/or repression of systematic silencing (Raja *et al.* 2010). These viral suppressors (VSRs) interfere with either single or multiple steps in silencing pathway to enhance virus replication, eventually restraining the production of sRNA. For instance, the V2 protein of *Tomato yellow leaf curl virus* inhibits the generation of dsRNA by binding to SGS3 (cofactor of RDR6). This ultimately distracts the siRNA production and increases the susceptibility of tomato plant (Glick *et al.* 2008). *Cucumber mosaic virus* protein 2b binds with the AGO1 and blocks its cleaving activity (Goto *et al.* 2007). *Tobacco mosaic virus* VSRs HcPro and P122-kDa replicase inhibits HEN1-mediated methylation and initiates the degradation of siRNA (Lozsa *et al.* 2008). VSR binding to the dsRNA/siRNA is also an important mechanism of suppression. *Cymbidium ringspot tombusvirus* produces P19 silencing suppressor, which targets the 21- to 25-nt dsRNAs and eventually deteriorates the PTGS effector complexes. *Flock house virus* B2 protein has an affinity toward both siRNAs and longer dsRNAs, thus inhibiting the siRNA production by shielding dsRNA from cleavage by DCL (Chao *et al.* 2005). Plants utilize TGS mediated by RdDM to defend DNA viruses, but these viruses possess inhibitor proteins that suppress TGS. *Cauliflower mosaic virus* P6 protein binds to nuclear protein DRB4 (double-stranded RNA binding protein), which is essential for the functioning of DCL4 and inactivates it, thus suppressing TGS (Haas *et al.* 2008). Moreover, recent reports have revealed that the impact of VSRs on endogenous pathways is more complex and profound than had been estimated, and hence intense research is required for understanding the replication, subcellular localization and regulation of the expressions of viral genes, including VSRs (Burgyán and Havelda 2011).

4. Trans-generational immune memory

In the past decade, numerous findings suggested the phenomena of trans-generational immune memory in plants, where the environmental stresses challenged by a generation, can lead to effective adaptations to those stresses in the next generation. This is achieved by memorizing the acquired immune potential in a form and is disseminated through mitotic and meiotic divisions, even during the nonexistence of stress, thus equipping the progenies with an acquired immune power. Epigenetic changes in genetic material, such as modifications in DNA methylation patterns, chromatin remodelling and histone modification, can heritably and reversibly modify the expression of genes and have an impact on plant immunity (Molinier *et al.* 2006; Jaskiewicz *et al.* 2011). In addition, the role of small interfering RNAs

(siRNAs) in transmitting the immune memory to subsequent generation was also reported (Chitwood and Timmermans 2010).

Slaughter *et al.* (2012) compared the reactions involved in trans-generational memory of *Arabidopsis* that had been either primed with β-amino-butyric acid (BABA) or with an avirulent isolate of the bacteria *Pseudomonas syringae* pv *tomato* (*PstavrRpt2*). In the progenies of primed plants, they found an immediate and increased accumulation of transcripts of genes participating in SA signalling pathway along with an enhanced disease resistance upon challenging with pathogenic *P. syringae*. These trans-generationally primed progenies were exposed to an additional priming treatment, where their descendants were found to exhibit a much stronger primed phenotype (Slaughter *et al.* 2012). This suggested the ability of plants to inherit a sensitization for the priming event.

Luna *et al.* (2012) reported the prevalence of trans-generational SAR over one stress-free generation, indicating an epigenetic basis of memory in *Arabidposis* under disease pressure by *Pseudomonas syringae* pv *tomato* DC3000 (PstDC3000). They also discovered that the P1 progeny from the NPR1 mutants seldom developed trans-generational defense phenotypes, thus substantiated a critical role for NPR1 in expression of trans-generational SAR. Their study proposed that transmittance of trans-generational SAR is accomplished by hypomethylated genes which perform in direct priming of SA-dependent defenses in the subsequent generations (Luna *et al.* 2012).

5. The undeciphered modules in immunity

Amidst the captivating discoveries, the field still retains many enduring challenges and mysteries, including identification of *avr* genes of haustoria-forming fungal pathogens, immune mechanisms of roots, molecular mechanisms by which nuclear effectors from different microbes impose their transcriptional signature in host cells and its successful plant colonization, production of ROS/RNI members and their role in regulation of cellular activity and gene expressions through fine-tuning of the signalling processes. These concealments will undoubtedly make the field of plant innate immunity a hottest in research, in the future.

6. Conclusions

Plants have evolved innate immune systems that recognize the presence of pathogens and initiate effective defense responses, whereas successful pathogens have evolved effector proteins that can suppress host immune responses. Furthermore, effectors can themselves act as elicitors and can be disabled by the host. The studies on plant–pathogen interactions had described several interesting information such as unique immune strategies adopted by plants against highly pathogenic strains, R-protein-mediated surveillance and memory of immunity in SAR. Considerable progresses have been made in understanding the role and mechanism of RNA silencing, substantiating the fact that RNA silencing operates as an innate antiviral defense in higher plants. The development of powerful RNAi *in vitro* assays, the isolation and further characterization of RNA silencing mutants, and the investigation of viral-encoded silencing suppressors provide an exciting and fascinating research ground for the future. Obviously, a better understanding of plant–pathogen interactions and the molecular details of how plants are able to heritably protect their offspring against potential enemies in their environment without making changes in their DNA sequence will pave the way for the improvement of disease resistance in economically important crops, thereby ensuring food security, an issue of global importance.

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