



How Plants Respond to Pathogen Attack: Interaction and Communication

20

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Abstract

Plants are exposed to a plethora of microorganisms in their environment. A number of these microorganisms are plant pathogens. In order to defend themselves against pathogen attack, plants have evolved specialized sensory receptors to recognize some of the conserved molecular features (PAMPs, DAMPs, HAMPs, and NAMPs) as well as secreted effector molecules of pathogens. A cascade of signal transduction events are triggered which causes transcriptional rewiring leading to activation of defense responses. Closure of stomata, strengthening of cell wall along with accumulation of secondary metabolites, and induction of a hypersensitive response (HR) and pathogenesis-related (PR) proteins are some of the key defense strategies of the host. Interestingly, through secretion of volatile organic compounds (VOCs), plants have the ability to induce defense responses in uninfected tissues as well as surrounding plants. In this chapter, we elaborate on the mechanisms by which plants perceive pathogen attack and transduce the signal to downstream signaling molecules, culminating in the activation of defense responses.

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20.1 Introduction

Plants are constantly exposed to a diverse array of microorganisms. Among them some are pathogenic on the host, whereas others grow in harmony with the host without causing any damage. Plants possess a proficient and dynamic sensory system to distinguish between them. In case of beneficial interactions, plants have adapted to harbor these microorganisms in specialized compartments, thus maintaining a suitable niche inside their tissue (Oldroyd 2013; Jones et al. 2007). However, in case of a negative interaction, the microorganism tries to forcefully colonize to obtain nutrients from the host plant. Plants being sessile cannot evade from such harmful interactions but possess several robust defense mechanisms to inhibit the growth of such pathogenic organisms.

The first step in mounting an immune response lies in the ability of host to perceive the pathogen attack, and this is achieved via a wide array of specialized extracellular receptors that are present on the plant cell membrane. Generally, plants recognize bacterial pathogens by conserved structural components such as flagellin, lipopolysaccharides (LPS), peptidoglycans (PG), etc. or bacterial molecules such as EfTu or RaxX that are released into the extracellular milieu (Couto and Zipfel 2016). Fungal pathogens are sensed by the recognition of chitin or fungal secreted proteins such as NLPs (NEP1 like proteins) (Kaku et al. 2006). These conserved microbe-specific molecules are known as PAMPs/MAMPs (pathogen-/microbe-associated molecular patterns). Herbivory is perceived by the presence of certain herbivore-associated molecular patterns (HAMPs) present in the oral secretion of the insect at the time of attack (Mithofer and Boland 2008). Nematodes also secrete molecules that are known to elicit plant defense responses, and these molecules are known as nematode-associated molecular patterns (NAMPs) (Mendy et al. 2017). Besides these signals, plants can also sense molecules that are released from their own cells as a consequence of pathogen attack and use them as cues to mount an immune response (Bacete et al. 2018). These molecules are known as DAMPs (damage-associated molecular patterns). Classic examples of DAMPs are degradation products that are released following the action of microbial enzymes on various components of the plant cell wall. Also, plants have cytoplasmic receptors to sense effector molecules secreted by potential pathogens to mount a robust immune response (Schreiber et al. 2016).

Plants possess a two-tiered detection system against pathogens (Zipfel 2014). The first tier comprises of receptors present on the surface of a cell called PRRs (pattern recognition receptors) that recognizes PAMPs, DAMPs, HAMPs or NAMPs. The PRRs can broadly be classified under two types, receptor-like kinases

(RLKs; comprising of a ligand-binding ectodomain, a transmembrane domain, and a cytoplasmic kinase domain) and receptor-like proteins (RLPs; comprising of a ligand-binding ectodomain and a transmembrane domain). The immune responses that are mounted upon recognition of the pathogen by PRRs are referred to as pathogen-triggered immunity (PTI). Moreover, the immune responses that are induced following recognition of DAMPs are known as DAMP-triggered immunity (DTI). The second tier of the pathogen recognition system comprises of intracellular immune receptors that can sense secreted pathogenic effectors either directly or indirectly. The immune responses that are mounted upon recognition of these effectors are referred to as effector-triggered immunity (ETI). The receptors are classified into two types: nucleotide-binding site leucine-rich repeat (NBS-LRR) proteins and Toll-like receptor (TLR) proteins. The major difference in the signaling events during PTI and ETI is the duration and amplitude of the defense response, which is more in ETI as compared to PTI. In this chapter, we elaborate on how plants recognize various phytopathogens (bacteria and fungi) as well as herbivores and nematodes. We have described various players involved in the signal transduction events associated with pathogen perception and how the perceived signal is transduced to regulate host defense response pathways in host.

20.2 Perception of Pathogen Attack

Perception of danger is a key step in the activation of immune responses. However, induction of immune responses is an energy-consuming process that involves activation/deactivation of many molecular pathways, synthesis of new molecules, and alterations in basic metabolic processes (Andolfo and Ercolano 2015; Duan et al. 2013). Hence, it is crucial for plants to distinguish between a potential pathogen/pest and a random visitor to mount an appropriate immune response (Table 20.1).

20.2.1 Recognition of Bacterial Pathogens

Plants can recognize various structural components of bacteria or their secreted compounds to mount an immune response. Flagellin-Sensing 2 (FLS2), a LRR repeat domain-containing receptor-like kinase in Arabidopsis can recognize a 22-amino acid long peptide named flg22 derived from the flagellin of *Pseudomonas syringae* (Gómez-Gómez and Boller 2000). The flg22 peptide binds to the extracellular N-terminal domain of FLS2 and acts as a molecular glue between FLS2 and its co-receptor somatic embryogenesis receptor kinase 3 (SERK3) [also called as BAK1 (BRI1-associated receptor kinase 1)] (Meindl 2000; Sun et al. 2013). This complex phosphorylates downstream interacting partners and activates the immune response (Couto and Zipfel 2016). Interestingly, different plant species have evolved diverse receptors to recognize different epitopes on flagellin. Solanaceous plants such as pepper, potato and tomato recognize flgII-28 (the flagellin peptide derived from *Pseudomonas syringae*) by another type of LRR receptor, FLS3 (Hind et al. 2016).

Table 20.1 List of some known receptors of MAMPs, DAMPs and effectors perceived by plants

Source of ligand	Receptor	Plants	Ligand	Type of receptor	Type of Immune Response	Reference
Bacteria	FLS2	Arabidopsis	Flg22 (flagellin)	LRR-RLK	PTI	Gómez-Gómez and Boller (2000)
	FLS3	<i>Solanaceae</i>	FlgII-28 (flagellin)	LRR-RLK	PTI	Hind et al. (2016)
	EFR	Brassicaceae	Eif18 (EF-Tu)	LRR-RLK	PTI	Zipfel et al. (2006)
	Xa21	Rice	RaxX (putative)	LRR-RLK	PTI	Pruitt et al. (2015)
	OsLYM4/ OsLYM6	Rice	Peptidoglycan (bacterial cell wall)	LysM-RLP	PTI	Liu et al. (2013a, b)
	AtLYM1/ AtLYM3	Arabidopsis		LysM-RLP	PTI	Willmann et al. (2011)
	AtLORE (Putative)	Arabidopsis	Lipooligosaccharide (bacterial cell wall)	Lectin-RLK	PTI	Ranf et al. (2015)
	OsCEBiP	Rice	Chitin	LysM-RLP	PTI	Kouzai et al. (2014)
	AtCERK1/ AtLYK5	Arabidopsis		LysM-RLK	PTI	Cao et al. (2014)
	LeEix1/LeEix2	Tomato	EIX (fungal xylanase)	LRR-RLP	PTI	Rotblat et al. (2002)
	RLP23	Arabidopsis	Nlp20 (various pathogens)	LRR-RLP	PTI	Albert et al. (2015)
	RGAS5	Rice	AVR-Pia (magnaporthe effector)	NB-LRR	ETI	Ortiz et al. (2017)
	RPP1	Arabidopsis	ATR1 (<i>Hyaloperono-spora arabidopsidis</i>)		ETI	Krasileva et al. (2010)
L5/L6	Flax	AvrL567 (<i>Metampsora lini</i>)		ETI	Ravensdale et al. (2012)	
Nematodes	NILR1	Arabidopsis	Ascarosides	LRR-RLK	PTI	Mendy et al. (2017)
	WAK1	Arabidopsis	Oligogalacturonides (OG)	EGF-RLK	DTI	Brutus et al. (2010)
	DORN1	Extracellular ATP	eATP	Lectin-RLK	DTI	Choi et al. (2014)
	PEPR1/PEPR2	Arabidopsis	pep	LRR-RLK	PTI	Krol et al. (2010)

Plants can also recognize peptidoglycan (PG) and lipopolysaccharide (LPS) that are major components of either bacterial cell wall or the outer membrane, respectively. Exogenous treatment with either LPS or PG activates plant immune responses (Erbs et al. 2010; Gust et al. 2007). PG is a polymer of N-acetylglucosamine and N-acetylmuramic acid linked by oligopeptides (Gust et al. 2007). Plants possess LysM domain (lysine motif)-containing proteins that can recognize glycans in N-acetylglucosamine (Gust et al. 2012). In Arabidopsis, PG is recognized by receptor-like proteins AtLYM1 and AtLYM3 where chitin receptor AtCERK1 serves as a key component in PG recognition (Willmann et al. 2011). In rice, OsLYP4 and OsLYP6 are known to interact with both chitin oligomers as well as peptidoglycan (Liu et al. 2012). Additionally, OsCERK1 appears to be a key receptor/co-receptor for LPS perception in rice (Desaki et al. 2017). In Arabidopsis, bulb-type (B-type) lectin S-domain (SD)-1 containing RLK protein LORE (lipooligosaccharide-specific reduced elicitation) is thought to be the putative LPS receptor (Ranf et al. 2015). However, the physical interaction between LPS and putative receptors are yet to be established.

Plants can also sense various bacterial secreted proteins/peptides. Elongation factor Tu (Ef-Tu) is an abundant bacterial protein that is released upon cell lysis. Ef-Tu acts as an elicitor of immune responses in various plant species (Kunze 2004). Members of the Brassicaceae family recognize a conserved 18-aa long peptide (elf18) present at the N-terminal of EF-Tu by the LRR-RLK Ef-Tu receptor (EFR) (Zipfel et al. 2006). Rice recognizes EFa50, comprising of a 50aa long peptide sequence from the middle of Ef-Tu amino acid sequence (Furukawa et al. 2014). Another secreted peptide recognized by plants is RexX21-sY, a sulfated peptide secreted by *Xanthomonas oryzae* pv. *oryzae* (Xoo) type 1 secretion system (Pruitt et al. 2015). This is recognized by rice LRR-RLK receptor Xa21. Here it is worth mentioning that Xa21 has been widely used to breed rice for bacterial blight resistance (Williams et al. 1996).

20.2.2 Recognition of Fungal/Oomycete Pathogens

Chitin, a polymer of N-acetyl-D-glucosamine (GlcNAc), is a major component of fungal cell walls. Plants can identify chitin oligomers by 40aa long globular LysM motif-containing receptor proteins (Kaku et al. 2006; Miya et al. 2007; Wan et al. 2008). In Arabidopsis, AtCERK1/AtLYK1 (chitin elicitor receptor kinase) recognizes chitin oligomers and mounts defense responses. Binding of 7–8-residue long chitin oligomer with *AtCERK1* causes receptor homodimerization and transphosphorylation that lead to activation of defense signaling cascade (Liu et al. 2012). Rice recognizes chitin by a GPI-anchored RLP protein, OsCEBiP (chitin elicitor binding protein), that contains three extracellular LysM domains but lacks an intracellular kinase domain (Kaku et al. 2006; Kouzai et al. 2014). Ligand (GlcNAc)₈ binding causes homodimerization of OsCEBiP and OsCERK1 leading to the formation of a GlcNAc₈-2CEBiP-2CERK1 complex which in turn activates immune responses (Hayafune et al. 2014). Other plant receptors such as AtLYK4 (RLK),

OsLYP4 and OsLYP6 (both RLP) can also recognize chitin (Liu et al. 2012; Petutschnig et al. 2010; Wan et al. 2012).

Some plants can sense presence of fungal xylanases to mount immune responses. A fungal protein ethylene-inducing xylanase (EIX) was found to activate plant immune responses in various host species (Bailey et al. 1990, 1993; Fuchs et al. 1989; Ron et al. 2000). In tomato, LeEIX is recognized by LRR-RLP LeEix2 leading to activation of immune responses (Bar and Avni 2009; Bar et al. 2009). Similarly, in Arabidopsis, LRR-RLP receptor AtRLP42 recognizes fungal endopolygalacturonases (PGs) and activates its immune responses (Zhang et al. 2014).

20.2.3 Recognition of Herbivores

The plants are exposed to different insects, some of which feed upon plant parts by a process known as insect herbivory. Herbivorous insects can activate plant defense mechanisms either through mechanical wounding caused during the process of chewing or by their oral secretions. Mechanical wounding caused during herbivory induces either the activation of defense mechanisms or secretion of plant volatiles. Production of chemical factors or relaying of electrical signals across distal parts of the host tissues are some of the early plant responses generated immediately after wounding (Maffei et al. 2007).

The herbivore-associated molecular patterns (HAMPs) that are present in the oral secretions of insects are recognized by plants (Mithofer and Boland 2008). Some orally secreted compounds like fatty acid amino conjugates (FACs) act as elicitors in priming of plant defense responses (Bonaventure et al. 2011). Perception of FACs induces a MAPK signaling cascade including SIPK (salicylic acid-induced protein kinase) and WIPK (wound-induced protein kinase) along with activation of NPR1 signaling (Wu et al. 2007; Seo et al. 2007; Bonaventure and Baldwin 2010), culminating in the activation of defense responses.

20.2.4 Recognition of Nematodes

Plants are continuously exposed to a plethora of microorganisms surrounding their rhizosphere. The different varieties of root exudates secreted by the plants may either attract or deter away these microorganisms. Plants secrete flavonoid compounds that can attract symbiotic microbes like Rhizobia in case of beneficial interactions, phytoalexins to deter pathogen growth or allelopathic phenolic compounds to alter the growth of other plants (Hirsch et al. 2003). However, plant parasitic nematodes like root-knot nematode and potato cyst nematode can sense these host-derived signals. Following penetration inside the host tissue, the nematode migrates to its feeding site inside the root, wherein it feeds upon the host nutrients resulting in altered root architecture and reduced crop yield. Since long it had been speculated that plants could also mount a PTI response against nematodes, however not much was known about the compounds which elicit plant defense response.

Recently, a nematode pheromone, ascaroside has been identified that is perceived by host plants as a NAMP to mount a PTI response including activation of MAP kinase cascade, upregulation of plant defense hormones such as salicylic acid and jasmonic acid, and induction of defense responses (Manosalva et al. 2015; Holbein et al. 2016; Choi et al. 2016). Moreover, a nematode immune receptor NILR1 (nematode-induced LRR-RLK 1) belonging to the LRR-RLK has been identified in *Arabidopsis* that perceives NAMP and mounts PTI responses (Mendy et al. 2017).

20.2.5 Recognition of DAMPs

The plant cell wall serves as a formidable barrier against pathogens. Pathogen secretes various proteins to degrade different components of the plant cell (Jha et al. 2005). Moreover, plants have evolved the ability to sense this damage by recognition of the cell wall degradation products. Treatment of *Arabidopsis* with cellulose degradation products such as cellobiose, cellotriose, etc. or cellulose synthesis inhibitors (Engelsdorf et al. 2017) activates the host immune responses (Souza et al. 2017). Similarly, the treatment of plant tissue with pectin degradation products such as oligogalacturonides (OG) can activate the host immune responses (Ferrari 2013). In *Arabidopsis*, wall-associated kinases (AtWAK1 and AtWAK2) can perceive pectin and pectin degradation products (OG) (Brutus et al. 2010; Decreux and Messiaen 2005; Decreux et al. 2006). The activation of immune responses by WAKs has also been reported in other plant species such as rice and maize (Delteil et al. 2016; Zuo et al. 2015; Hu et al. 2017).

In response to pathogen/damage perception, plants secrete various peptides and nucleotides in their apoplast to amplify the immune response and trigger an elaborate defense mechanism in their neighboring cells (Boutrot and Zipfel 2017). Release of plant elicitor peptides (Peps, also known as danger peptides) derived from PROPEPs (precursor proteins) has been reported in *Arabidopsis* upon pathogen attack (Bartels et al. 2013; Klauser et al. 2015). *Arabidopsis* secretes 23aa long endogenous elicitor peptides known as *AtPep1*, which are recognized via LRR-RLK PEP receptor (PEPR) (Krol et al. 2010). Moreover, it has been reported that extracellular ATP (eATP) can act as a DAMP in *Arabidopsis* (Weerasinghe et al. 2009; Wu et al. 2008). The eATP is recognized by a lectin receptor kinase-I.9 (LecRK-I.9) named DORN1 (Does not Respond to Nucleotides 1) in *Arabidopsis* which activates downstream defense-responsive genes (Choi et al. 2014).

20.2.6 Recognition of Effectors

PTI and DTI form the first layer of plant immune responses. Pathogens can suppress these immune responses by secreting effector molecules directly into plant cells via the type-III-secretion system (Alfano and Collmer 2004). However, plants have evolved *R* gene-encoded proteins to recognize effector proteins to activate effector-triggered defense (ETD) response (Dodds and Rathjen 2010). Plants can

either directly recognize effector molecules via NB-LRR (or NLR) domain-containing receptor proteins or can indirectly sense their presence by monitoring their activity (Kourelis and van der Hoorn 2018). In both cases, plants mount a robust immune response that usually culminates in a hypersensitive response and localized death of plant tissue to limit spread of the pathogen. The NLR receptor proteins are usually comprised of either coiled-coil (CC) domain or toll/interleukin-1 receptor (TIR) domains at their N-terminal (Cui et al. 2015; Schreiber et al. 2016). However, there are exceptions, wherein certain effector proteins are not directly recognized by receptor proteins, instead are recognized when bound to an accessory protein (guardee). The guard model has been proposed to explain this phenomenon (Dangl and Jones 2001). Further, a modification of this hypothesis has been proposed as a decoy model, wherein certain effector targets have evolved to function as decoys (co-receptor) which bind to the effectors and cause activation of the defense response (van der Hoorn and Kamoun 2008). Due to a few limitations in the decoy model, an improved bait-and-switch model was proposed. In this model, a two-step recognition has been proposed wherein the accessory protein (bait) associated with the receptor protein interacts with the effector protein to mount a defense response (Collier and Moffett 2009). The current hypothesis states that the receptor protein instead of recognizing the accessory protein directly recognizes the effector protein only when it is bound with its accessory protein (Dodds and Rathjen 2010). We will now provide an outline of the different effector molecules that are secreted in different pathosystems and how plants are able to recognize them.

20.2.6.1 Bacterial Effector Recognition

The AvrPto and AvrPtoB (also known as HopAB2) effectors secreted by pathogenic strains of *P. syringae* (Abramovitch et al. 2003; Ronald et al. 1992) are recognized by plants to mount immune responses. AvrPto and AvrPtoB bind to various PTI receptors and suppress immune responses. For example, AvrPto binds to various PTI receptors like FLS2 and EFR while AvrPtoB binds to FLS2, BAK1 and LysM receptor kinases and suppress immune responses (Cheng et al. 2011; Gimenez-Ibanez et al. 2009; Göhre et al. 2008; Shan et al. 2008; Xiang et al. 2008; Zeng et al. 2012). Prf/Pto protein complex recognizes the presence of both of these effector molecules, whereas Pto has binding sites for both AvrPto and AvrPtoB as well as Prf. Prf acts as a positive regulator of ETI. In the native state, Pto binds to Prf along with some other kinases to form a large macromolecular complex that keeps Prf in its inactive state (Ntoukakis et al. 2013). In presence of cognate effectors, Pto binds to the effector, gets released from Prf/Pto complex and in turn activates ETI (Abramovitch et al. 2003; Dong et al. 2009; Mathieu et al. 2014).

Rin4 (RPM1 interacting protein 4) is a membrane-localized protein that lacks any functional domain but is a part of many PRR complexes (Selote and Kachroo 2010). Rin4 can activate as well as suppress PTI depending on the phosphorylation status of the protein (Chung et al. 2014). Pathogens have evolved effector molecules such as AvrB, AvrRpt2, AvrRpm1, and HopF2 to directly or indirectly target Rin4 to suppress PTI

(Lee et al. 2015; Russell et al. 2015; Wang et al. 2010; Wilton et al. 2010). In response, plants have also evolved R genes such as RPS2 (resistance to *P. syringae*) and RPM1 (resistance to *P. syringae* pv. *maculicola*) to sense the activity of effectors on Rin4 and mount defense responses (Chung et al. 2014; Coaker et al. 2005; Kim et al. 2005).

20.2.6.2 Fungal Effector Recognition

Fungal pathogens are also known to produce effector molecules which can either be secreted into the host cytoplasm or localized into the apoplastic space (Giraldo et al. 2013; Stotz et al. 2014). The recognition of apoplastic effectors is mediated by integral membrane proteins (RLPs) containing an extracellular leucine-rich repeat (eLRR) (Stergiopoulos and de Wit 2009). Induction of RLPs has been reported in tomato, apple, and oilseed rape against fungal pathogens like *Cladosporium fulvum*, *Venturia inaequalis*, and *Leptosphaeria maculans*, respectively (Rouxel and Balesdent 2013; Belfanti et al. 2004). However cytoplasmic effectors secreted by pathogens like *Blumeria graminis*, *Bremia lactucae*, *Puccinia striiformis*, *Magnaporthe grisea*, and *Phytophthora infestans* are recognized by NBS-LRR receptors that are present in the cytoplasm of respective host species (Bozkurt et al. 2010; Bai et al. 2012; Bonardi et al. 2012; Larkan et al. 2013; Rooney et al. 2005).

20.2.6.3 Nematode Effector Recognition

Plants utilize NB-LRR immune receptors to recognize effectors secreted from root or cyst nematodes to activate host defense responses. Some common examples of immune receptors against nematodes are Gpa2, Gro1-4 and Hero (Goverse and Smant 2014). It has been observed that root-knot nematodes secrete a diffusible compound called NemF that is very similar to NF (nodulation factor) secreted by symbiotic bacteria. The NemF signal is perceived by the plant through primary receptor kinases NFR1 and NFR5 along with secondary receptor kinase SYMRK. Signal perception leads to root hair branching and waviness which in turn facilitate nematode penetration (Weerasinghe et al. 2005). Plants also encode R genes to recognize effector proteins secreted by herbivores (Hogenhout and Bos 2011). Examples of R genes which confer resistance against herbivores are Mi-1.2 (Meloidogyne 1.2), Vat (Virus aphid transmission resistance) and Bph14 (Brown planthopper 14).

20.2.6.4 Miscellaneous

Necrosis and ethylene-inducing peptide 1-like proteins (NLPs) are plant immunogenic proteins with cytotoxic activity produced by a vast variety of bacterial, fungal, and oomycete species (Oome et al. 2014). Plants belonging to Brassicaceae family can recognize a conserved 20aa long fragment of NLP called nlp20 to activate their immune responses (Böhm et al. 2014; Oome et al. 2014; Oome and Van den Ackerveken 2014). In Arabidopsis, the LRR-RLP AtRLP23 recognizes nlp20 and activates immune responses by making a tripartite complex with two LRR-RLK, BAK1 (brassinosteroid insensitive 1 (BRI1)-associated kinase) and SOBIR1 (Albert et al. 2015).

20.3 Players Involved in Transduction of a Perceived Signal

The PRR proteins present on the plant cell surface can recognize pathogen attack and mount a defense response against the pathogen. However, induction of defense responses involves an intricate signaling network that transduces the signal to downstream molecular players to trigger immune responses. These signaling molecules include protein kinases (CDPKs, MAPKs), Ca^{2+} burst, ROS burst, NO, lipids, 14-3-3 proteins and various phytohormones (such as SA, JA and ethylene) (Bigeard et al. 2015).

20.3.1 Phosphorylation Events

Phosphorylation and dephosphorylation of proteins by kinases and phosphatases play an important role in the signal transduction process. After ligand binding, conformational changes in protein/binding with co-receptors lead to phosphorylation of the receptor. Somatic embryogenesis receptor kinase (SERK) family usually works as a co-receptor for many receptor kinases such as FLS2, EFR, BRI1, Xa21, PEPR, PSKR, etc. (Ma et al. 2016). In Arabidopsis, SERK3 [also called bri1-associated receptor kinase 1 (BAK1)] is a key co-receptor for many receptor kinases and is required for proper induction of immune responses (Ma et al. 2016). In rice, OsSERK2 interacts with Xa21, Xa3 and FLS2 (Chen et al. 2014) and is required for receptor-mediated resistance against Xoo. SERKs are also involved in RLP-mediated activation of immune responses such as nlp20-triggered immunity in Arabidopsis, csp22-triggered immunity in Nicotiana, Avr4- and Avr9-induced HR in tomato (Albert et al. 2015; Postma et al. 2016; Saur et al. 2016).

20.3.1.1 MAP Kinases

Mitogen-activated protein kinases (MAPKs) form signaling modules, which translate extracellular stimuli of pathogen attack into appropriate defense responses. MAPK cascade typically contains three sequential kinases (Rasmussen et al. 2012):

- MAP kinase kinase kinase (MAPKKK or MEKK)
- MAP kinase kinase (MAPKK or MKK)
- MAP kinase (MAPK or MPK)

Usually receptor/co-receptor phosphorylates MAPKKK that phosphorylates MAPKK which phosphorylates MAPK. MAPK then phosphorylates downstream signaling components such as transcription factors and modulates defense responses (Meng and Zhang 2013). In a recent study, it has been shown that phosphorylation of OsMKK3-OsMPK7-OsWRKY30 leads to transcriptional activation of defense responses against *X. oryzae* in rice (Jalmi and Sinha 2016). Interestingly, in order to suppress PTI response, pathogens have evolved effector molecules that majorly target MAPK modules due to their primary role in defense signaling of plants (Feng et al. 2012).

20.3.1.2 CDPKs

Calcium-dependent protein kinases (CDPKs) have a serine/threonine protein kinase domain at their N-terminal and CaM-like domain with EF-hand calcium-binding sites at their C-terminal (Boudsocq and Sheen 2013). They act as Ca^{2+} sensors and decode the signal to generate a swift response to the external stimulus (Seybold et al. 2014, 2017). CDPK response was found to be associated with changes in host physiology such as transcriptional reprogramming, ROS accumulation, and alteration of phytohormone levels. CDPKs together with MAPKs have been found to orchestrate the transcriptional regulation of defense genes under pathogen attack (Boudsocq et al. 2010). Another group of kinases called AGC kinases, comprising of cAMP-dependent protein kinase 1 (PKA) and cGMP-dependent protein kinase (PKG) along with protein kinase C (PKC), have been shown to regulate MAPK signaling cascade upon pathogen attack (Garcia et al. 2012).

20.3.1.3 14-3-3

14-3-3 proteins act as phosphosensors which bind to phosphorylated proteins and regulate their functions. 14-3-3 proteins aid in phosphorylation of proteins thereby activating them (Chevalier et al. 2009). They play a crucial role in strengthening plant defense mechanisms by interacting with MAPKK proteins involved in the defense signal transduction pathway (Oh et al. 2010; Oh and Martin 2011). Induction of 14-3-3 proteins was found primarily in the penetration stage and upper epidermis of barley infected with *Blumeria graminis* suggesting its involvement in early signaling events (Lozano-Durán et al. 2015). 14-3-3 proteins have been found to interact with plant immune-responsive proteins such as receptor kinase BAK1 and WRKY transcription factor along with few R genes (Chang et al. 2009). 14-3-3 proteins have also been reported to regulate phytohormone levels in infected plants culminating in enhanced immune responses. (Chang et al. 2009; Camoni et al. 2018).

20.3.1.4 Heterotrimeric G proteins

G proteins have been found to play a critical role in defense signaling in animals. However plants lack the canonical G protein structure as observed in animals (Urano and Jones 2014). G proteins are known to activate plant defense signaling responses mediated by the action of multiple RLKs (Liu et al. 2013a, b; Maruta et al. 2015). The signals received from RLKs by G proteins are transduced downstream to different MAPKs and ROS signaling genes (Nitta et al. 2015; Cheng et al. 2015). Studies have revealed direct physical association between the $\text{G}\alpha$, $\text{G}\gamma 1$, and $\text{G}\gamma 2$ subunits and RD-type kinases CERK1, BAK1, and BIR1 to activate the plant defense network (Aranda-Sicilia et al. 2015).

20.3.2 Regulation of Immune Responses

Plant immune responses are metabolically costly affair; plants regulate the processes in a tight manner to avoid non-specific activation and dampen the responses when they are no longer required. This is usually achieved by dephosphorylation or

degradation of receptors. After activation of immune responses, protein phosphatases (PP) such as PP2C and PP2A dephosphorylate the receptor and other intermediate kinases to negatively regulate immune responses (Durian et al. 2016; Fuchs et al. 2013). Some examples of PP2C involvement in immunity include kinase-associated protein phosphatase (KAPP), PLL4 and PLL5 of Arabidopsis, and XB15 of rice (Holton et al. 2015; Park et al. 2008).

Another approach to regulate immune response is via vesicle-mediated internalization of activated receptors or degradation of the receptor/signaling intermediate (Wang et al. 2016a, b). These proteins are polyubiquitinated by E3 ubiquitin ligases and degraded by 26S proteasomes. Some examples of this pathway include XB3 of rice and PUB12 and PUB13 of Arabidopsis (Lu et al. 2011; Wang et al. 2006).

20.3.3 Transcriptional Regulation

Activation of immune responses involves rapid transcriptional and translational changes (Li et al. 2016). Transcriptional events are modulated by transcription factors (TFs) which get activated by MAP kinases, Ca²⁺ signaling or hormonal response (Kang et al. 2015; Li et al. 2016). Some key TF families involved in defense responses include WRKY, MYC, TCP, ZIP, MVQ, AP2/ERF, etc. (Birkenbihl et al. 2017). TFs enhance expression of various defense genes such as PR genes, secondary metabolism, and hormone biosynthesis as well as regulation of related genes.

20.3.4 Secondary Signaling Molecules

Many non-proteinaceous molecules are key signaling intermediates in plant innate immunity. These molecules include Ca²⁺, ROS, NO, etc.

20.3.4.1 Burst of Ca²⁺

Ca²⁺ ions play an important role in defense signaling during pathogen attack. Ca²⁺ burst occurs when MAMPs/DAMPs are perceived and Ca²⁺ from the extracellular milieu is transported into the cytoplasm (Jeworutzki et al. 2010; Ranf et al. 2011). The permeability of plasma membrane to Ca²⁺ is mediated by elicitor responsive ion channels. The calcium levels accumulate in distinct signature patterns and generate a particular defense response pathway against the pathogen (Lecourieux et al. 2006). Influx of Ca²⁺ is followed by opening of other membrane ion transporters such as H⁺, K⁺, Cl⁻, and NO₃⁻ channels which lead to alkalization of extracellular space and membrane depolarization (Jeworutzki et al. 2010).

EF-hand motif-containing proteins are known to bind with calcium and serve as sensors of Ca²⁺ concentration (Schulz et al. 2013). These proteins mainly include Ca²⁺-dependent protein kinases (CDPK) and calmodulin (CaM). Ca²⁺ binding causes conformational changes in structure of these proteins leading either to phosphorylation or binding with downstream signaling intermediates (Ishida and Vogel 2006; Wernimont et al. 2010).

20.3.4.2 ROS Burst

Production of extracellular reactive oxygen species (ROS) also referred to as ROS burst has been found to be associated with pathogen attack (Ranf et al. 2011; Chinchilla et al. 2007; Nühse et al. 2007). MAMP perception is often associated with ROS production by respiratory burst oxidase homolog D (RBOHD), a member of NADPH oxidase family in Arabidopsis (Bigeard et al. 2015). ROS can be present in membranes as impermeable superoxide (O_2^-) or as permeable hydrogen peroxide (H_2O_2) and it can be readily translocated from one cell to another. Also it is often associated with elevated Ca^{2+} levels in the cytosol (Ranf et al. 2011; Bigeard et al. 2015). ROS signaling is accompanied by alteration in plant defense hormone levels such as JA, SA, and ethylene indicating a complex crosstalk between different pathways (Baxter et al. 2014).

20.3.4.3 NO Signaling

Nitric oxide (NO) along with its derivatives has also been involved in signal transduction pathway upon perception of pathogen attack. The role of NO in activating plant defense was first reported in tobacco mosaic virus infection wherein increase in NO synthase (NOS) resulted in activation of several downstream defense genes (Klessig et al. 2000). Interestingly, NO together with ROS plays a synergistic role in activation of plant defense responses (Domingos et al. 2015). NO can cause a rapid change in cellular glutathione levels in the cell associated with accumulation of SA and activation of NPR1-mediated defense responses (Kovacs et al. 2015).

20.3.4.4 Lipid Signaling

Lipid-based signaling molecules are also known to play a crucial role in defense signaling upon pathogen attack. These lipid molecules are produced as a result of degradation/destabilization of the cell wall upon pathogen attack. For example, phosphatidic acid (PA) and ceramides have been found to be involved in signal transduction upon pathogen infection (Okazaki and Saito 2014). PA is also involved in release of other signaling intermediates such as DAG, free fatty acids, and lysoPA which in turn induce downstream defense signaling (Wang 2004). Phospholipase A (PLA) which catalyzes the hydrolysis of phospholipids is involved in release of free fatty acids which are utilized during biosynthesis of defense hormone jasmonic acid (Shah 2005).

20.3.4.5 Hormonal Signaling

Major phytohormones such as jasmonic acid (JA), salicylic acid (SA) and ethylene have been found to play an important role in coordinating cell-to-cell communication during perception of pathogen attack. Each of these phytohormones activates its own downstream targets which lead to diverse immune and signaling events. There are also reports that other phytohormones such as auxin, cytokinin, abscisic acid, gibberellins and brassinosteroids are involved in plant immunity. There is a complex crosstalk among different phytohormones occurring at the cellular level that tailors a specific defense response upon attack by a specific pathogen. Here we summarize the role of some key defense-related phytohormones.

20.3.4.5.1 Salicylic Acid

SA is a phenolic hormone that is synthesized from chorismate via phenylalanine ammonia pathway (PAL) or isochorismate synthase (ICS) pathway (Chen et al. 2009). It is a key component of PTI as well as ETI and is known to enhance tolerance against various biotrophic, hemi-biotrophic, and viral infections (Dodds and Rathjen 2010; Malamy et al. 1990; Shigenaga and Argueso 2016). It is also necessary for the activation of various PR genes. Arabidopsis ICS1 mutant (*ics1*), also called SA deficient 2 (*sid2*), was compromised in SA-mediated immune response (Dewdney et al. 2000; Wildermuth et al. 2001). Interestingly, the non-expressor of PR genes 1 (NPR1) acts as a transcription co-activator and plays a key role in SA-mediated immune responses (Cao 1994). Generally at normal SA levels, NPR1 is localized in the cytoplasm in oligomeric form (Mou et al. 2003). However, at elevated SA level, the NPR1 binds to SA, adopts monomeric form, and gets transported to the nucleus (Kinkema et al. 2000; Mou et al. 2003). In the nucleus, NPR1 binds to TGA transcription factors and activates expression of defense-related genes including PR genes (Kesarwani et al. 2007). Infection studies on *ics1* mutant (that fails to increase SA level), NahG (salicylate hydroxylase that degrades SA) expressing transgenic lines (that fail to accumulate SA), and *npr1* mutant (that does not respond to SA) indicate that although SA can enhance tolerance towards biotrophic and hemi-biotrophic pathogens, it reduces resistance towards necrotrophic pathogens (Delaney et al. 1994; Glazebrook et al. 1996; Thomma et al. 1998). It is worth noting that phytopathogens utilize various effectors (such as HopJ, HaRxL44, HopM1 and PsIcs1) to target SA signaling pathway during host colonization (Caillaud et al. 2013; DebRoy et al. 2004; Liu et al. 2014).

20.3.4.5.2 Jasmonic Acid

JA is a lipid-derived hormone that is involved in many developmental and defense response pathways (Santino et al. 2013; Carvalhais et al., 2017). JA is synthesized by oxygenation of α -linolenic by lipoxygenase (Lox) enzymes and is converted into JA-Ile (JA-isoleucine; the active form of JA) by JA amido synthetase (JAR1) (Staswick 2004; Wasternack and Hause 2013). Coronatine insensitive 1 (COI1), an E3 ubiquitin ligase, is a receptor of JA, and a transcription factor jasmonate ZIM domain 1 (JAZ1) is a negative regulator of JA pathway (Sheard et al. 2010; Yan et al. 2009). At low JA levels, JAZ1 represses JA-responsive genes (Pauwels et al. 2010). After perception of pathogen attack, JA-Ile binds to COI1, which ubiquitinates JAZ1 leading to degradation of JAZ1. Degradation of JAZ1 leads to enhanced expression of JA-responsive genes (Thines et al. 2007).

JA and SA are believed to play antagonistic roles against each other in very complex plant defense response-activating pathways depending on the nature of the pathogen (Thaler et al. 2012; Robert-Seilaniantz et al. 2011). Pathogens have evolved mechanisms to utilize this crosstalk to suppress plant immune responses (Pieterse et al. 2012). A well-studied example is synthesis of the JA mimic molecule coronatine (COR) by *Pseudomonas* sp. COR activates the JA pathway and suppresses SA pathway leading to increased susceptibility towards biotrophic and hemi-biotrophic pathogens including *Pseudomonas* (Zheng et al. 2012). Interestingly

a hemi-biotrophic pathogen, i.e., *Pseudomonas*, utilizes effector molecules such as HopZ1 and HopX1 to induce JA pathway during pathogenicity process (Gimenez-Ibanez et al. 2014; Jiang et al. 2013).

20.3.4.5.3 Ethylene

Ethylene is a gaseous plant hormone known for its role in fruit ripening. However, it is also known to be involved in plant defense responses. ET and JA phytohormones work in a synergistic manner (Robert-Seilaniantz et al. 2011). The activation of JA pathway leads to enhanced expression of ET pathway genes (Penninckx et al. 1998; Zhu et al. 2011). Alike JA, ET also enhances tolerance towards necrotrophic pathogens but increases susceptibility towards biotrophic pathogens (Lawton et al. 1994, 1995). Similar to other phytohormones, ET pathway is also targeted by pathogens to overcome immunity. For example, AvrPto and AvrPtoB effectors of *Pseudomonas* sp. and XopD effector of *Xanthomonas* sp. have been found to alter the ET pathway (Cohn and Martin 2005; Kim et al. 2013).

20.4 Plant Defense Responses

Upon perception of pathogen attack, plants mount a strong immune response to restrict the spread of pathogen/predator. These immune responses involve strengthening of the cell wall, localized cell death, production of antimicrobial compounds, etc. The strength of the immune response depends upon the type of danger. Many pathogens have evolved mechanisms to suppress PTI/ETI directly by secreting effector molecules into the plant cell. This is known as effector-triggered susceptibility (ETS). However, recognition of effectors by host R genes leads to activation of ETI that includes robust defense responses such as programmed cell death to restrict the growth of the pathogen at the site of infection.

20.4.1 Stomatal Closure

Several phytopathogens use stomata to enter inside the host. Closure of stomata is one of the early defense responses used by the host to prevent pathogens from colonization. Upon perception of pathogen cues (flg22, elf18, elf26, LPS, chitin, oligogalacturonan, etc.), plants close their stomata (Arnaud and Hwang 2015; Murata et al. 2015). This process involves various signaling events including activation of MAP kinase pathway, synthesis of hormones, Ca²⁺ influx, ROS and NO production, etc. (Desclos-Theveniau et al. 2012; Melotto et al. 2006, 2017). SA and ABA pathways are known to promote stomatal closure while JA-Ile serves as a negative regulator of stomatal closure.

However, successful phytopathogens have evolved various mechanisms to avoid plant stomatal closure. For example, *P. syringae* secretes various effectors such as

HopM1, HopF2, HopZ1, HopZ1a, Hopx1 and AvrB to suppress closure of stomata (Gimenez-Ibanez et al. 2014; Hurley et al. 2014; Jiang et al. 2013; Lozano-Durán et al. 2014; Zhou et al. 2014, 2015). XopR, a Xoo-secreted effector, suppresses flg22-induced stomatal closure in rice (Wang et al. 2016a, b). On the other hand, some of the bacterial pathogens secrete phytotoxins to open stomatal pores to assist colonization. Some of the notable phytotoxins used by bacterium to open stomata are coronatin (COR) (Bender et al. 1999) and syringolin A secreted by *P. syringae* (Groll et al. 2008), plant natriuretic peptide-like (Gottig et al. 2008) and diffusible signaling factor (DSF) (Gudesblat et al. 2008) molecules secreted by *Xanthomonas* species.

20.4.2 Cell Wall Strengthening

Cell wall serves as a key barrier to phytopathogens. Pathogens need to degrade the cell wall to gain access to nutrients that are inside the plant cell. Strengthening of the cell wall is achieved by deposition of callose (β -1,3 glucan) and lignin (phenolic polymers). This is one of the basic mechanisms used by the host plant to suppress the growth of pathogen (Malinovsky et al. 2014). Treatment with various MAMPs, DAMPs or avirulent pathogen strains causes callose deposition in the infected tissues (Luna et al. 2011). Synthesis of callose usually leads to papillae formation that contains antimicrobial compounds such as thionins, H_2O_2 , etc. (McLusky et al. 1999; Thordal-Christensen et al. 1997; Voigt 2016). Besides callose, lignin is also deposited at the secondary cell wall to provide mechanical strength (Malinovsky et al. 2014). Loss of function mutations in various genes involved in lignin synthesis pathway makes the plants more susceptible to pathogens (Miedes et al. 2014).

20.4.3 Pathogenesis-Related Proteins

Expression of pathogenesis-related (PR) proteins is upregulated in plants after pathogen infection. These proteins are key components of plant immune responses. Many PR proteins are also observed to be upregulated after MAMP and DAMP treatment, wounding, ETI activation, and treatment with immune response-associated hormones (Sels et al. 2008). PR genes encode diverse classes of proteins which can be classified under 17 different families (van Loon et al. 2006). Most of the PR proteins have antimicrobial activities. PR3, PR4, PR8 and PR10 are chitinases which can degrade fungal cell wall, while PR2 proteins are β -1,3-glucanases. PR1 is a most common PR protein accumulated in various plant species upon pathogen attack and is known to have antimicrobial activity (Ménard et al. 2005; Segarra et al. 2013; Song et al. 2015). Some PR genes encode small peptides such as the PR6 family containing proteinase inhibitor peptides (Green and Ryan 1972), PR12s are cysteine-rich defensins (Terras 1995), PR13 encodes thionins (Epple et al. 1995) and PR14 codes for lipid transfer proteins (LPT) (García-Olmedo et al. 1995).

Interestingly, AtPR1, AtPR2, and AtPR5 are SA-responsive genes known to provide resistance against biotrophic and hemi-biotrophic pathogens in Arabidopsis. AtPR3 and AtPR4 are JA-responsive genes and provide tolerance against necrotrophic pathogens and herbivores (van Loon et al. 2006).

20.4.4 Secondary Metabolites

Plants produce various types of secondary metabolites upon infection by phytopathogens (Piasecka et al. 2015). These metabolites usually have antimicrobial activity and have a toxic effect on phytopathogens. One type of secondary metabolites that are constitutively produced are called phytoanticipins (VanEtten 1994). These are usually produced in an inactive form and are activated by hydrolysis after perception of danger. Plants produce various kinds of phytoanticipins including saponins such as α -tomatine and avenacin, glucosinolates, cyanogenic glucosides and benzoazinone glucoside compounds (Faizal and Geelen 2013; Halkier and Gershenzon 2006; Burkhardt et al. 1964; Papadopoulou et al. 1999; Sandrock and VanEtten 1998). Secondary metabolites that are de novo synthesized in response to biotic stress are called phytoalexins. The major types of phytoalexins include camalexins, phenylalanine-derived phytoalexins, and terpenoids (VanEtten 1994). Mutations in secondary metabolite synthetic genes make plants more susceptible to pathogens (Toyomasu et al. 2014; Xu et al. 2012).

20.4.5 Hypersensitive Response

Sometimes, plants undergo programmed cell death in the infected area to restrict the spread of a pathogen. This process is called hypersensitive response (HR). PCD is generally involved in developmental processes and stress responses including tolerance towards biotic stress (Bozhkov and Lam 2011; Pennell 1997). Upon pathogen perception by the host R gene, an intricate signaling cascade is triggered that leads to HR. This signaling cascade involves MAP kinase activation, SA production, ROS production, NO accumulation, cytosolic Ca^{2+} increase, membrane depolarization, etc. (Kadota et al. 2004; Kärkönen and Kuchitsu 2015; Kurusu et al. 2011). HR can be observed as the lesion phenotypes during infection or elicitor treatment or as lesion mimic phenotype if the immune response is constitutively activated (Coll et al. 2011; Lorrain et al. 2003). Although HR is a strong immune response, sometimes it can act like a double-edged sword for plants as necrotrophic pathogens that flourish on dead plant tissues have evolved various pathways to utilize this immune response of plants to colonize host tissues (Mukhtar et al. 2016). These pathogens modulate plant signaling to enhance ROS production and induce HR (Shetty et al. 2008).

20.4.6 Secretion of Volatile Compounds

Upon pathogen attack, plants often emit gaseous compounds known as VOCs (volatile organic compounds). Emission of volatile derivatives of certain plant hormones such as jasmonic acid and ethylene have been found to be responsible for the systemic activation of plant defense responses (Champigny and Cameron 2009; Fiers et al. 2013; Wasternack and Kombrink 2010; Tamogami et al. 2008). Plants also secrete volatile components that can attract predators or parasitoids such as parasitic wasps to forage upon the feeding insects or induce a systemic defense response in distal uninfected plant parts (Heil 2008; Heil and Silva Bueno 2007; Frost et al. 2007). Volatile compounds that are thus secreted are known as herbivore-induced plant volatiles (HIPV). Lima bean plants secrete certain compounds which are not only involved in attraction of predatory insects (natural enemies of herbivores) but also in production of certain extrafloral nectars (EFN) which serve as a food source for these predatory insects (Choh and Takabayashi 2010). Apple plants have been found to emit certain VOCs upon infection by the bacterial pathogen *Erwinia amylovora* which can activate the defense responses even in surrounding healthy uninfected plants (Cellini et al. 2018). Interestingly, it has been observed that VOCs produced upon infection by fungal pathogen *Colletotrichum lindemuthianum* in resistant bean plants can trigger defense responses in neighbouring susceptible plants (Quintana-Rodriguez et al. 2015).

20.5 Conclusion

Plants have specialized receptors to sense pathogen attack and mount potent defense responses. Various conserved structural components, damaged cell wall products or effector molecules produced by the pathogens are recognized by these receptors. Generally, these receptor proteins are maintained in a dephosphorylated inactive state and get activated at the time of pathogen attack. Various signaling intermediates like MAPKs, CDPKs, 14-3-3 and heterotrimeric G proteins participate in interception, amplification and transduction of the signal from the receptor to the target defense genes. Also several secondary messengers such as ROS, Ca²⁺, NO, lipids, and hormones help in the relay of signal (Fig. 20.1). Interestingly, the induction of defense responses is not merely restricted to the infected tissue but it is also elaborated in uninfected as well as distal parts of the plant. Interestingly, phytohormones such as salicylic acid, jasmonic acid and ethylene play a significant role in activation of immune responses.

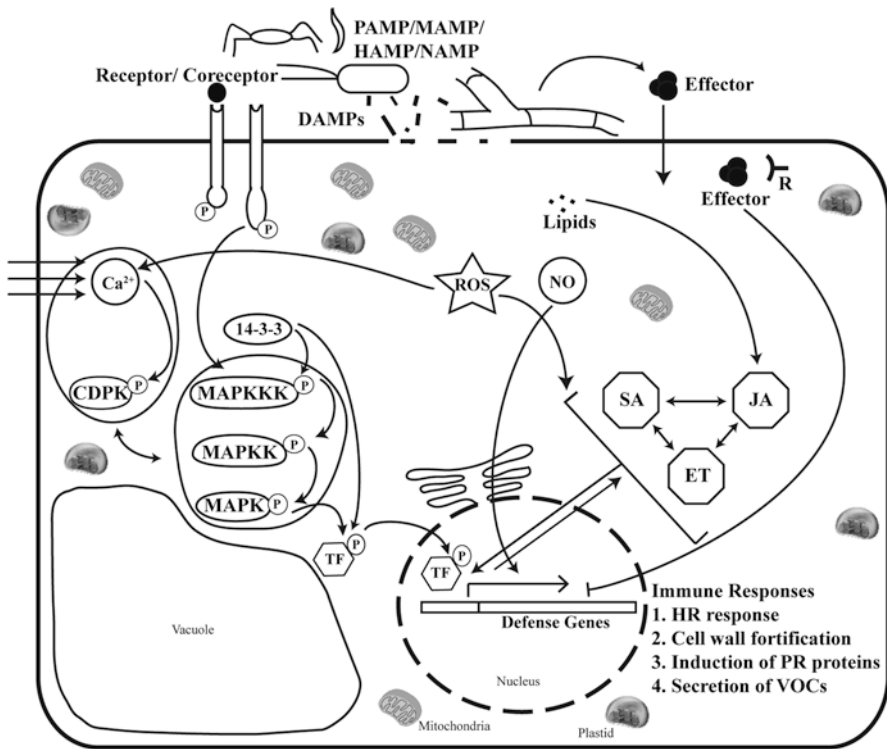


Fig. 20.1 Schematic overview of cellular responses induced in host upon perception of pathogen attack. The pathogen possesses certain conserved structural components (MAMPs, DAMPs, HAMPs, NAMPs, etc.) which are recognized by cognate receptors present in host plant. Upon signal perception, a cascade of signal transduction events including induction of phosphorylation events (involving MAP kinases, CDPKs, etc.) as well as secondary signaling molecules (such as calcium, NO, ROS, etc.) are triggered. All these events culminate in activation of potent immune responses which combat most of the potential pathogens to cause disease. Notably, several phytohormones such as SA, JA, ET, etc. also play important roles in elucidation of plant defense responses. Additionally, pathogens secrete effector molecules to inhibit plant immune responses, but plants have evolved resistance genes (R genes) to directly or indirectly recognize them and mount a strong defense response

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Authors have known the Editor who is the Chairman of the Scientific Advisory Committee of NIPGR.