Microbe-Associated Molecular Patterns

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Plants depend on both preformed and inducible defence responses to defend themselves against biotic stresses stemming from pathogen attacks. In this regard, plants perceive pathogenic threats from the environment through pattern recognition receptors (PRRs) that recognise microbe-associated molecular patterns (MAMPs), and so induce plant defence responses against invading pathogens. Close to thirty PRR proteins have been identified in plants, however, the molecular mechanisms underlying MAMP perception by these receptors/receptor complexes are not fully understood. As such, knockout (KO) of genes that code for PRRs and co-receptors / defence-associated proteins is a valuable tool to study plant immunity. The loss of gene activity often causes changes in the phenotype of the model plant, allowing in vivo studies of gene function and associated biological mechanisms. Here, we review the functions of selected PRRs, brassinosteroid insensitive 1 (BRI1) associated receptor kinase 1 (BAK1) and other associated defence proteins that have been identified in plants, and also outline KO lines generated by T-DNA insertional mutagenesis as well as the effect on MAMP perception - and triggered immunity (MTI). In addition, we further review the role of membrane raft domains in flg22-induced MTI in Arabidopsis, due to the vital role in the activation of several proteins that are part of the membrane raft domain theory in this regard.

Keywords: BAK1 ; innate immunity ; KO ; MAMPs ; membrane raft ; MTI ; PRRs ; T-DNA

1. Well-Studied MAMP Perception System(s)

1.1. Bacterial MAMPs

1.1.1. Flagellin Perception

Flagellin is the main protein component of bacterial flagella, that acts as a MAMP in both plants and animals. Plants have a notably sensitive perception system for the highly conserved domain in the N-terminus of eubacterial flagellin [1]. The perception thereof, and particularly the N-terminus flagellin peptides, flg22 and flg15 from *Pseudomonas syringae* pv. *tabaci*, lead to the alkalinisation of the culture medium in suspension-cultured cells of *Lycopersicon peruvianum* (a wild relative of tomato), *A. thaliana*, potato and tobacco respectively [1]. Flagellin from the rice-incompatible strain, N1141 of *P. avenae*, was also shown to induce the hypersensitive cell death and accumulation of EL2 mRNA (elicitor-responsive gene) in cultured rice cells [2]. As mentioned, FLS2 is a LRR-RLK involved in the perception of the bacterial flagellin and immune responses in *A. thaliana* [3][4]. Furthermore, the recognition of bacterial flg22 by Arabidopsis FLS2 induces a FLS2-BAK1 complex formation and triggers defence signalling [4][5]. By using chemical cross-linking and immunoprecipitation techniques, Chinchilla et al. [6] showed that the specificity of flagellin perception and immune responses is mediated by the binding of FLS2 to flg22 in *A. thaliana*. The *fls2* mutants carrying T-DNA insertion in the flagellin receptor gene *FLS2* were more susceptible to *Pst* DC3000 compared to the wildtype (WT) [7]. Upon flg22 treatment, *FLS2* mutants (*fls2-101*), generated by the insertion of T-DNA at the third *FLS2* exon, showed impaired binding of the flg22 and reduced seedling growth inhibition [8].

As stated, flg22 induces a FLS2-BAK1 complex in a ligand-dependent manner, and leads to defence responses in *A. thaliana* [4]. This study showed that although *BAK1* mutants (*bak1-3* and *bak1-4* with T-DNA inserted in 5th intron and 9th exon, respectively) were defective in an oxidative burst generation, they did not affect the binding of flg22 to FLS2. Furthermore, flg22 induces recruitment of the U-box E3 ubiquitin ligases (PUB12 and PUB13), previously mentioned, to the FLS2-BAK1 complex and this causes FLS2 degradation and the concomitant attenuation of immune signalling in Arabidopsis [9]. Here, there was a decrease in FLS2 ubiquitination and *Pst* DC3000 infection in mutant plants (*pub12-2*, *pub13* and *pub12/13* double knockout), compared to WT controls. A recent study has furthermore shown that flagellin-sensing 3 (FLS3) directly and specifically binds flgII-28 (a second flagellin epitope distinct from flg22), which enhances immune responses in tomato [10]. Finally, it is also important to note that BAK1 is involved in the defence signalling activities of both FLS2 and FLS3 [4][10].

1.1.2. EF-Tu Perception

Elongation factor-thermo unstable (EF-Tu), a prokaryotic elongation factor involved in the synthesis of proteins, is a bacterial MAMP that is recognised by the LRR-RLK, EFR, in Arabidopsis [11]. *N. benthamiana* normally lacks EF-Tu responsiveness, but achieved the ability to recognise the MAMP when EFR was transiently expressed in the plant [11]. Kunze et al. [12] showed that EFR binds directly to the acetylated N-terminus epitope of elf18 (the first amino acids of EF-Tu) and elicits innate immunity in Arabidopsis and other Brassicaceae species. The first acetylated 12 N-terminal amino acids (elf12) were, however, not able to elicit an immune response, but rather acted as a specific antagonist of elf18 [12]. Interestingly, even though EF-Tu induces MTI in rice, the elf18 peptide failed to trigger an immune response. However, another epitope, EFa50 (50-amino acid from central region of EF-Tu comprising Lys176 to Gly222) induced MTI responses, although the related PRR is still unknown [13]. Rice leaves treated with EFa50 induced early defence responses such as increased H_2O_2 and callose deposition, and triggered resistance to coinfection with pathogenic bacteria. In this regard, Arabidopsis *EFR* T-DNA insertion mutants (*efr-1* and *efr-2*), with both T-DNAs inserted in 1st exon, were insensitive to EF-Tu and susceptible to the bacterium *Agrobacterium tumefaciens* [11] (Table 1). In fact, *efr* mutants did not show typical growth inhibition as for the WT, increased oxidative burst and ethylene biosynthesis or induced resistance to *Pst* DC3000 upon EF-Tu treatment. Jeworutzki et al. [14] reported involvement of EFR and FLS2 in the Ca^{2+}-associated opening of plasma membrane anion channels during early bacterial flagellin and EF-Tu defence signalling in Arabidopsis mesophyll cells. Using electrophysiological approaches, they showed that the *efr-1* was defective in the membrane potential depolarisation, which is indispensable for cytosolic calcium influx in response to elf18 treatment.

1.1.3. LPS Perception

LPS is an amphipathic molecule found on the outer membrane of Gram-negative bacteria and protects the organism against antimicrobial compounds found in the environment [15][16]. This MAMP is involved in the bacterial adhesion and induction of defence-related responses in both mammals and plants [17]. In the former, LPS recognition is orchestrated by lipopolysaccharide binding protein (LBP), before recruitment into a complex comprising soluble myeloid differentiation protein 2 (MD-2), membrane attached cluster of differentiation 14 (CD14), and the transmembrane Toll-like receptor 4 (TLR4), thereby leading to mammalian defence activation against LPS [18][19]. LBP and bactericidal/permeability-increasing protein (BPI) thus play a vital role in regulation of immune responses against LPS in mammals [19][20]. As such, the mechanism by which LPS is recognised has been well-studied in animals, however, in plants, the mechanism of perception and recognised moiety/epitope(s) of LPS is still debateable.

In this regard, LPS, as well as its lipid A moiety from *Burkholderia cepacia, Xanthomonas campestris and P. syringae*, trigger the upregulation of genes involved in immunity and defence [21][22][23], with lipid A speculated to be the major elicitor in *A. thaliana* [24]. LPS has furthermore been shown to trigger defence responses, such as the generation of nitrogen oxide (NO), ROS, elevation of cytoplasmic Ca^{2+} concentration, stomatal closure and the expression of pathogenesis-related (PR) genes in Arabidopsis, tobacco and rice suspension cells [25][26][27][28][29]. Ranf et al. [25] earlier reported that LPS from *Xanthomonas* and *Pseudomonas* are sensed by the bulb-type (B-type) lectin S-domain (SD)-1 RLK lipooligosaccharide-specific reduced elicitation (LORE) and that *LORE* mutants showed defects in the LPS-induced elevation of cytosolic calcium, ROS and defence gene (*AtFRK1*) expression. Additionally, Shang-Guan et al. [24] reported the existence of two ROS production phases, characterised by a weak initial and second stronger ROS generation in *A. thaliana*. T-DNA insertion *LORE* mutants, however, showed little or no difference in second phase ROS production compared with WT, suggesting another LPS receptor(s). In contradiction to the original LORE study, a recent report implicated bacterial 3-hydroxy fatty acids in LORE-dependent induction of immune response in Arabidopsis [30]. Here, LORE could not sense LPS that was repurified to remove free 3-hydroxy fatty acids, indicating that LORE is not the receptor for LPS.

Using *B. cepacia* LPS-affinity capture strategies, Vilakazi et al. [31] and Baloyi et al. [32] identified BAK1 and other defence response proteins associated with the plasma membrane fraction in LPS-treated *A. thaliana*. Additionally, Arabidopsis LBP/BPI related-1 (AtLBR-1) and LBP/BPI related-2 (AtLBR-2) were shown to bind to both rough and smooth LPS, and regulate the expression of the *pathogenesis-related 1* (*PR1*) gene [23]. *AtLBR* T-DNA single mutants (*Atlbr-1*, *Atlbr-2-1*, *Atlbr2-2*) and (*Atlbr*-DKO) double-knockouts, generated by crossing *Atlbr-1* and *Atlbr-2-1* plants, were defective in ROS generation and in upregulation of LPS-induced *PR1* expression. A transcriptome analysis revealed that AtLBR-2 plays an indispensable role in the upregulation of 65 genes associated with defence responses upon *Pseudomonas* LPS treatment [33]. Furthermore, there was a defect in the upregulation of defence-related genes and salicylic acid (SA)-mediated signalling in the *Atlbr-2-1* mutants, compared to the WT after *Pseudomonas* LPS treatment.

Lastly, a recent study has reported OsCERK1, the chitin co-receptor, as an LPS receptor/co-receptor in rice, but not in *A. thaliana* [34]. This indicates a significant difference between LPS perception in rice and Arabidopsis.

1.1.4. Peptidoglycan Perception

Rice chitin elicitor-binding protein (CEBiP) homologs in Arabidopsis, LYM1 and LYM3 RLPs, bind in a ligand-specific manner to PGNs, heteropolymers that are part of the building blocks of the cell walls of Gram-negative and Gram-positive bacteria [35]. There was a significant expression of the immune marker gene, flagellin-induced receptor kinase *FRK1,* upon treatment of Arabidopsis with PGNs from Gram-negative *Pst* DC3000 [35]. Homozygous T-DNA insertional mutants of *LYM1* and *LYM3* showed a strongly reduced PGN-inducible marker gene expression and were more susceptible to *Pst* DC3000 bacterial infection than the WT (Table 1). The same authors further reported the involvement of AtCERK1 in the LYM1/LYM3 perception of PGN and subsequent immune response in *A. thaliana*. Thus, AtCERK1 is the additional protein that provides the cytoplasmic kinase domain that is lacking in the LYM1/LYM3 RLPs needed for the downstream transphosphorylation of PGN signalling. The study suggests that Arabidopsis senses PGNs in a LYM1/LYM3 and AtCERK1-dependent manner, similar to the chitin perception system in rice that uses OsCEBiP and OsCERK1 [36]. Furthermore, the direct interaction of PGN with AtLYM1 and AtLYM3 has been reported, but not with AtCERK1 [35]. In rice, OsCERK1 associates with LysM motif-containing proteins (LYP4 and LYP6) in PGN-induced defence responses [37][38][39]. Importantly, the putative ability of CERK1 to participate in the recognition and signalling of more than one MAMP supports the hypothesis of the capability of one PRR/co-receptor to recognise more than one MAMP, which favours the generation of transgenic crop plants with enhanced/altered recognition capabilities [40].

1.2. Fungal MAMPs

1.2.1. Chitin Perception

Chitin is a β(1→4)-linked polymer of N-acetylglucosamine, a major structural component in the exoskeleton of arthropods and cell walls of fungi [41]. Chitin and its fragments (chitooligosaccharides) are MAMPs recognised by plants PRRs, which elicit defence responses such as the oxidative burst, protein phosphorylation, transcriptional activation of defence-related genes and phytoalexin biosynthesis [41][42][43]. Kaku et al. [42] isolated and characterised a chitin elicitor binding protein (OsCEBiP), an RLP involved in the perception of chitin oligosaccharides in cultured rice cells. It was observed that OsCEBiP has two LysM motifs and a C-terminal transmembrane domain, however no intracellular kinase domain, suggesting that it requires additional protein-partner(s) to perform a signal transduction role. In rice, the LysM receptor, OsCEBiP, binds to the chitin oligosaccharide and forms a complex with OsCERK1 to trigger defence response [36]. Conversely, in *A. thaliana*, the LysM receptor, AtCERK1, directly binds to chitin, dimerises and triggers immune responses [44]. In the extracellular domain, OsCERK1 has one LysM motif, whereas AtCERK1 has three LysM motifs [36][45] that mediate the binding of chitin [46][47]. There are five genes encoding LysM receptor-like kinases (LYKs), which are comprised of *LYK1 (CERK1*) and *LYK2 - LYK5*, and three genes encoding LysM receptor-like proteins (LYPs) in the *A. thaliana* genome [48]. In this regard, LysM receptor-like kinase 1 (LysM RLK1) is involved in chitin signalling and fungal resistance in Arabidopsis [49]. A T-DNA insertion knockout mutant of *LysM RLK1* blocked the induction of chitooligosaccharide-responsive genes (CRGs) by chitooligosaccharides and increased susceptibility to fungal pathogens, but not bacterial pathogens. This was reversed when the mutant was complemented with the WT *LysM RLK1* gene using the cauliflower mosaic virus (CaMV) 35S promoter [44][49]. CERK1 KO mutants were unable to respond to chitin oligosaccharide elicitors when compared to the WT that exhibited rapid generation of ROS in Arabidopsis [44].

LYK4 is another LysM-RLK involved in chitin signalling and plant immunity in *A. thaliana*, and possibly in the chitin recognition receptor complex [50]. *LYK4* mutants were defective in the activation of chitin-responsive genes and were also susceptible to bacterial and fungal pathogen infection. Cao et al. [51] showed that AtLYK5 binds chitin with higher affinity and forms a chitin-induced complex with AtCERK1, thereby triggering immunity in *A. thaliana*. The higher affinity of AtLYK5 for chitin, compared to AtCERK1, also suggests the former to be the major chitin binding protein in Arabidopsis. These authors [51] further reported involvement of AtLYK5 in chitin-induced AtCERK1 phosphorylation and homodimerisation [51]. While *Atlyk5-2* was significantly impaired in chitin signal responses, *Atlyk4*/*Atlyk5-2* double mutant resulted in a complete loss of chitin response, indicating an overlap of signal function between AtLYK5 and AtLYK4.

The LysM domain-containing glycosylphosphatidylinositol-anchored protein 2 (LYM2), one of the three CEBiP homologs in Arabidopsis, binds chitin and mediates a reduction in molecular flux via the plasmodesmata [52]. AtCERK1 is not involved in the chitin-mediated regulation of plasmodesmata flux, thereby suggesting the presence of an alternative novel disease resistance mechanism in Arabidopsis [52][53]. Shinya et al. [54] showed that the Arabidopsis LYM2 can recognise chitin oligosaccharides in a similar way as the rice OsCEBiP, but does not participate in chitin signalling. The KO mutant of *LYM2* (*lym2-1*) was shown to be incapable of chitin-induced plasmodesmata flux, and susceptible to fungal pathogens (*Botrytis cenerea* and *Alternaria brassicicola*), but exhibited chitin-induced MAPK activation and an oxidative burst when compared to the WT [52][54]. LysM RLK1-interacting kinase 1 (LIK1) interacts with CERK1 and regulates chitin-induced MTI in Arabidopsis [55]. *LIK1* mutants (*lik1-1*, *lik1-2*, *lik1-3* and *lik1-4*), with T-DNA insertions located in intron 2, 13, and exon

18, respectively, showed an enhanced response to both chitin and flagellin elicitors. Furthermore, the mutants were defective in the expression of genes involved in jasmonic acid (JA) and ethylene (ET) signalling pathways, that have been shown to mediate resistance to necrotrophic pathogens [56]. In *A. thaliana* a powdery mildew-resistant kinase 1 (PMRK1), a RLK that is localised in the plasma membrane, is responsible for early chitin-induced defence signals against fungal pathogens [57]. *PMRK1* KO mutant (*pmrk1*) was more susceptible to both *Golovinomyces cichoracearum* and *Plectosphaerella cucumerina* (Table 1). In fact, these numerous identified PPRs linked to chitin perception suggest the complexity of plant chitin defence signalling.

2. BAK1 and Other Associated Proteins in MAMP Signalling

Following MAMP perception, PRRs trigger downstream events involving protein association/dissociation. BAK1 was initially identified as a co-receptor in BRI1-mediated brassinosteroid (BR) signalling, which modulates plant growth and development [58][59]. Studies have shown that BAK1 and related somatic embryogenesis receptor kinases (SERK) proteins associate with other LRR-RLKs or LRR-RLPs, and regulate plant growth and immunity [4][60][61].

Table 2 outlines different BAK1 and other associated proteins, and the implication of their KOs in plant MTI. In *A. thaliana*, both FLS2 and EFR form a complex with the co-receptor BAK1 to elicit immune responses immediately upon flg22 or elf18 perception, respectively [4][5][62][63]. Plants carrying *BAK1* mutants (*bak1-3* and *bak1-4*), generated by T-DNA insertion, displayed abnormal early and late flagellin-triggered responses [4][5]. In this regard, there was a significant reduction in the oxidative burst triggered by elf26 in *BAK1* mutants, indicating that EF-Tu is also affected by the mutation in BAK1 [4]. Interestingly, *BAK1* mutants were not completely impaired to flg22 or elf18 perception, indicating that BAK1 was not the only rate-limiting component and therefore suggests additional regulatory protein(s), such as BKK1, that are part of the FLS2 and EFR receptor complexes [4][5][60]. BAK1-disrupted *N. benthamiana* plants displayed decreased induction of MTI responses by the csp22 peptide (part of bacterial cold-shock protein) and INF1 (an oomycete elicitor) [5]. Furthermore, Arabidopsis *BAK1* KO mutants exhibited increased susceptibility to necrotrophic fungal pathogens, such as *Botrytis cinerea* and *Alternaria brassicicola* [64]. These results suggest a central role for BAK1 in modulating other PRRs besides FLS2 and EFR in plant defence signalling. The exact mechanism by which BAK1 mediates defence signalling is, however, not resolved. A recent study also showed BAK1 involvement in the tomato FLS3 recognition of flgII-28 (another flagellin epitope) and resulting immune response signalling [10].

BAK1 and BAK1-LIKE1 (BKK1) have dual physiological roles by positively regulating a BR-dependent plant growth pathway, and negatively regulating a BR-independent cell-death [65]. Here, cell death-control mediated by BAK1 and BKK1 is SA-dependent [66]. Upon flagellin perception, BIK1 as a RLCK, associates with the FLS2-BAK1 receptor complex to initiate plant innate immunity and cell death [67]. There was a significant loss of flg22-induced resistance to *Pst* DC3000 infection in *BIK1* mutant seedlings, however, the mutation did not affect flg22-induced FLS2 and BAK1 association. On the other hand, *BIK1* mutants were susceptible to necrotrophic pathogens but were resistant to a virulent bacterial pathogen *Pst* DC3000 [68]. Chen et al. [69] demonstrated that the *bik1* mutant displayed a strong SA-dependent resistance to *Plasmodiophora brassicae*, an obligate biotroph protist that induces gall formation in cruciferous plants. *Bak1-4 bik1* double mutants exhibited increased expression of plant defence genes and cell death phenotypes compared to *BIK1* single mutant [70], highlighting the cooperativity of BIK1 and BAK1 influence in plant immunity.

BIR2, a novel LRR-RLK, interacts with BAK1 in a kinase-dependent manner, and negatively regulates BAK1-dependent MAMP-triggered immune signalling [71]. Upon ligand binding to FLS2, BAK1 is released from BIR2 and recruited to the FLS2 complex. Therefore, BIR2 inhibits autoimmune cell-death responses by keeping BAK1 under control. Gao et al. [72] showed that BIR1, a BAK1-interacting RLK, negatively regulates multiple plant resistance signalling responses, and suppresses cell death in Arabidopsis. *BIR1* KO mutants (*bir1-1*) showed activation of constitutive defence responses and extensive cell death. However, the LRR-RLK SUPPRESSOR OF BIR1-1 (SOBIR1) and BAK1 function as co-receptors for LRR-RLPs, BAK1, and not SOBIR1, acts a co-receptor for LRR-RLKs [63]. SOBIR1, a co-receptor/adaptor for LRR-RLPs recruits BAK1 to SOBIR1-RLP23 and SOBIR1-RLP30 complex upon nlp20 and Sclerotinia culture filtrate elicitor1 (SCFE1) perception, respectively, in Arabidopsis [73][74]. Here, *SOBIR1* mutant (*sobir1-12*) was more susceptible to fungal *Sclerotinia sclerotiorum* and *B. cineria* [75]. The dissociation of BIR1 upon MAMP recognition by PRRs allows BAK1 to form an active complex with SOBIR1, which triggers downstream cell death and defence signalling [76].

The Arabidopsis malectin-like LRR-RLK, IMPAIRED OOMYCETE SUSCEPTIBILITY1 (IOS1) associated with PRRs FLS2, EFR and CERK1 in BAK1-dependent and -independent MTI responses [77]. Arabidopsis *IOS1* mutant (*ios1-2*) showed perturbations in the latter, including defective chitin responses and delayed upregulation of the PTI marker gene *FLG22-INDUCED RECEPTOR-LIKE KINASE1* (*FRK1*), as well as reduced downy mildew infection [74]. The malectin-like RLK FERONIA (FER), facilitates the ligand-induced complex formation of PRRs in Arabidopsis [78][79]. As such, the EFR/FLS2-

BAK1 complex formation has been shown to be promoted by FER and inhibited by Rapid Alkalinization Factor 23 (RALF23) [77]. Furthermore, a *FER* mutant (*fer-4*) showed diminished ligand-induced EFR/FLS2 complex formation, with the co-receptor BAK1. In addition, AtFER is involved in the negative regulation of jasmonic acid (JA) and coronatine (COR) signalling [79]. In support, BAK1 and other defence-responsive proteins were identified in *A. thaliana* plasma membranes after *B. cepacia* and *E. coli* LPS treatments [31][32]. Here, proteins identified were similar to some previously implicated proteins upon flg22 elicitation, suggesting that LPS perception and signalling could likely resemble that of flg22.

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