INTRODUCTION

Plants face threats from various pathogenic microbes and resist attacking pathogens through both constitutive and inducible defenses (Jones and Dangl, 2006). The pattern-triggered immunity (PTI) defense response represents the front line of plant innate immunity. PTI is activated upon recognition of pathogen- or microbe-associated molecular patterns (PAMPs or MAMPs) via pattern recognition receptors (PRRs; Jones and Dangl, 2006; Zipfel, 2009; Trach and Katagiri, 2010; Zhang and Zhou, 2010). Examples of MAMPs comprise the lipopolysaccharide envelope of Gram-negative bacteria, peptidoglycans from Gram-positive bacteria, eubacterial flagellin, eubacterial elongation factor (EF), methylated bacterial DNA fragments, and fungal cell wall derived glucans, chitins, and proteins (Girardin et al., 2002; Cook et al., 2004; Ausubel, 2005; Röll and Felix, 2009). MAMP perception results in PTI activation which includes downstream defense responses such as production of reactive oxygen species (ROS), activation of mitogen-activated protein kinases, changes in gene expression, and production of defense compounds together leading to broad resistance to pathogens (Cook et al., 2004; Ausubel, 2005; Röll and Felix, 2009). In addition, MAMP perception at stomatal guard cells induces stomatal closure, thus activating stomatal innate immunity (Melotto et al., 2006; Zeng et al., 2010).

Pathogen entry into host tissue is a critical, first step in causing plant infection. Stomata at the leaf epidermis are natural openings that bacteria use to enter into leaves. Typically, Arabidopsis stomata close when in contact with bacteria, thus functioning as innate immunity gates to actively prevent bacteria entry into plants (Melotto et al., 2016b, 2008; Schultz-Lefert and Robatzek, 2006; Zeng et al., 2010; Faulkner and Robatzek, 2012). Usually, 1 h after exposure to Pseudomonas syringae pv. tomato strain DC3000 (Ptr DC3000) bacteria, Arabidopsis stomata close as a result of stomatal innate immunity activation. Virulent bacteria such as Pst DC3000 can re-open Arabidopsis Col-0 stomata 3–4 h after infection through the action of the chemical effector coronatine (COR) suggesting that plant pathogens have evolved virulence factors to suppress innate immunity functions of stomata (Melotto et al., 2006; Schultz-Lefert and Robatzek, 2006). The ability of COR to inhibit stomatal closure is dependent on the COR gene (Melotto et al., 2006) and the priming compound beta-aminobutyric acid (BABA) blocks the COR-dependent re-opening of stomata during Pst DC3000 and Pectobacterium carotovorum subsp. carotovorum (Pcc) infection (Tsai et al., 2011; Po-Wen et al., 2013). Stomatal closure in response to treatments with fgl22, a peptide representing the most conserved domain of bacterial flagellin, is dependent on the flagellin receptor FLS2 (FLAGELLIN SENSITIVE2), demonstrating that perception of bacterial MAMPs through PRRs leads to closure of Arabidopsis stomata (Ziegler et al., 2004; Zeng and He, 2010). The chloroplastic enzyme ASPARTATE OXIDASE that catalyzes de novo biosynthesis of nicotinamide adenine dinucleotide is also a critical player during activation of stomatal innate immunity in response to Pst infection (Mucho et al., 2012). In addition, both salicylic acid (SA) and abscisic acid (ABA) signaling pathways are required during bacteria- and MAMP-induced stomatal closure in Arabidopsis (Melotto et al., 2006; Zeng et al., 2010). Recent works emphasized the lectin receptor kinases in plant innate immunity. In this review, we will thus focus on the role of this emerging family of receptor kinases in plant innate immunity, with highlights on stomatal innate immunity.

LECTIN RECEPTOR KINASES IN PLANT DEFENSE

In plants, perception and transduction of environmental stimuli are largely governed by receptor-like kinases (RLKs; Mahajan and Tuteja, 2005). RLKs belong to a vast protein family found in...
higher plants that is represented by 610 genes in the Arabidopsis genome (Shiu and Bleecker, 2001, 2003). Lectin receptor kinases are RLKs characterized by an extracellular lectin motif. These lectin receptor kinases are classified into three types: G, C, and L (Bouwmeester and Govers, 2009; Vaid et al., 2012). G-type lectin receptor kinases are known as S-domain RLKs and are involved in self-incompatibility in flowering plants (Kusaba et al., 2001; Sherman-Broyles et al., 2007). C-type (calcium-dependent) lectin motifs can be found in a large number of mammalian proteins that mediate immune immune responses and play a major role in pathogen recognition (Cambi et al., 2005), but are rare in plants. Arabidopsis has only a single gene encoding a protein with a C-type lectin motif but so far its function has not been elucidated (Bouwmeester and Govers, 2009). Arabidopsis contains 45 L-type lectin receptor kinases (LecRKs) that are characterized by an extracellular legume lectin-like domain, a transmembrane domain and an intracellular kinase domain (Herve et al., 1996; Barre et al., 2002; Bouwmeester and Govers, 2009). LecRKs were suggested to play a role in abiotic stress signal transduction (Garcia-Hernandez et al., 2006; Nishiguchi et al., 2002; Riou et al., 2002; He et al., 2004; Drag et al., 2009; Joshi et al., 2010). Notably, LecRK members of the Arabidopsis LecRK-VI clade (Bouwmeester and Govers, 2009), are redundant negative regulators of the ABA response during seed germination (Xin et al., 2009).

Due to the resemblance of the extracellular domain with lectin proteins known to bind to fungal and bacterial cell wall components, lectin receptor kinases are predominantly hypothesized to participate in biotic stress tolerance (Bouwmeester and Govers, 2009). Some lectin receptor kinases were indeed reported to be involved in plant resistance to pathogens. For example, Fr-4-2, a G-type lectin receptor kinase from rice, provides resistance against the fungal pathogen Magnaporthe grisea, the causal agent of rice blast (Chen et al., 2006). In tobacco, the expression of another G-type lectin receptor kinase was recently shown to be up-regulated by lipopolysaccharides (Sanabria et al., 2012). In Nicotiana benthamiana, the LecRK NILR1K1 was suggested to be a component of the N. benthamiana protein complex that recognizes the Phytophthora infestans INFI elicitor and mediates INFI-induced cell death (Kanazaki et al., 2008).

Like few other RLK proteins, such as PERK (proline-rich extensin-like receptor protein kinase), WAK (wall-associated kinase) and CaRLK (Catharanthus roseus-like RLK), LecRK-I.9 mediates cell wall–plasma membrane (CW–PM) continuum (Bouwmeester and Govers, 2009). The maintenance of structural CW–PM continuity is a critical factor that governs plants response to various stimuli and is essential for defense against pathogens (Bouwmeester and Govers, 2009; Bouwmeester et al., 2011). The association of RGD (arginine–glycine–aspartic acid) motif containing proteins with cellular proteins is a key mechanism that maintains the structural integrity of CW–PM contacts (Gouget et al., 2006). The RGD motif present in IPI-O (in plants induced-O), a secreted effector protein of the oomycete pathogen Phytophthora infestans, disrupts CW–PM adhesions upon interaction with a variety of cellular proteins, including LecRKs (Gouget et al., 2006). Further analysis revealed that deficiency in LecRK-I.9, earlier found to interact with RGD motif containing proteins (Gouget et al., 2006), leads to a gain of susceptibility phenotype toward the oomycete Phytophthora brassicae (Bouwmeester et al., 2011). These results imply that LecRKs may be involved in protein–protein interactions with RGD-containing proteins as potential ligands, and may play a structural and signaling role at the plant cell surfaces upon pathogen infection.

LecRK-VL2 is critical for resistance against hemibiotrophic Pst DC3000 and necrotrophic Pcc bacteria (Singh et al., 2012). Increased susceptibility of the transferred DNA (T-DNA) insertion mutant line lecrk-VL2-1 is correlated with defective bacteria and MAMP-induced MPK3 (Mitogen-activated protein kinase 3) and MPK6 (Mitogen-activated protein kinase 6) activities, PTI-responsive gene expression, and callose deposition (Singh et al., 2012). Transcriptome analysis of a LecRK-VL2 over-expression line revealed transcription up-regulation of numerous genes responsive to virulent or avirulent bacteria, the MAMP flag2, or to the SA functional analog benzothiadiazole further suggesting a role for LecRK-VL2 in the Arabidopsis PTI response (Singh et al., 2012). BAK1 (Brassinosteroid insensitive1-associated kinase 1) and FLS2 association, RIK1 (BOTRYTIS-INDUCED KINASE1) phosphorylation, and ROS production that are usually considered as early PTI responses (Zipfel and Bobatzik, 2010), were not compromised in the mutant lecrk-VL2-1. These data suggest that LecRK-VL2 positively modulates PTI signaling upstream of MPK3 and MPK6 and downstream of FLS2 (Singh et al., 2012). In addition, LecRK-VL2 is a key modulator of BABA-mediated priming and BABA-induced resistance (Singh et al., 2012). Further analyses of the function of LecRK-VL2 revealed that LecRK-VL2 possesses a functional kinase domain and is not critical for resistance to the necrotrophic fungal pathogen Botrytis cinerea (Singh et al., 2013). By contrast, over-expression of the plasma membrane-localized L-type lectin-like protein kinase 1, ATLPK1 (LecRK-IV.5) induces Arabidopsis resistance to B. cinerea (Huang et al., 2013).

Lectin receptor kinases are also critical for plant resistance to insects. The lectin receptor kinase 1 (LecRK1) is important during herbivory by Manduca sexta larvae to suppress insect-mediated inhibition of jasmonic acid-induced defense responses in Nicotiana attenuata (Gildardoni et al., 2011). Importantly, reduction of LecRK1 expression in N. attenuata induces increased Manduca sexta folivory (Gildardoni et al., 2011). The insect-induced accumulation of protease inhibitors, as well as the expression of the gene encoding threonine deaminase, two critical defense responses were also several fold reduced in N. attenuata with a silenced LecRK1 when compared to non-silenced controls (Gildardoni et al., 2011). Inhibition of SA accumulation through the expression of nahG in silenced lecrk plants restores wild-type levels of resistance against Manduca sexta herbivory, suggesting that LecRK1 inhibits the accumulation of SA during herbivory (Gildardoni et al., 2011). More recently, LecRK-1.8 was suggested to be important for the perception of insect egg-derived elicitors in Arabidopsis (Gouvïer-Ducimont et al., 2013).

LecRK-VL2 AND LecRK-V5 IN Arabidopsis STOMATAL INNATE IMMUNITY

In addition to positively regulating apoplastic PTI, LecRK-VL2 is also critical for Arabidopsis stomatal innate immunity
Arnaud, D., Desclos-Theveniau, M., Ausubel, F. M. (2005). Are innate immunity sensors defective in the mutant lecrk-V-5? A re-assessment of PTI marker genes are not affected in lecrk-V-5 mutants. COR mutants lacking a functional LecRK-V-5 are resistant to *Pst* DC3000 and *Pcc* surface inoculation, but are normally sensitive to infiltration inoculation (Arnaud et al., 2012; Desclos-Theveniau et al., 2012). These observations suggest that disruption of LecRK-V-5 affects early Arabidopsis defenses by restricting bacterial entry into leaves and point to a role of LecRK-V-5 in stomatal innate immunity (Desclos-Theveniau et al., 2012). Analyses of stomatal apertures in *lecrk-V-3* indeed revealed that this mutant possesses constitutively closed stomata (Desclos-Theveniau et al., 2012). Transgenic lines over-expressing LecRK-V-5 are less resistant to *Pst* DC3000 COR and this is correlated with a re-opening of stomata in *lecrk-V-5* over-expression lines even in the absence of COR. These observations suggest the existence of a stomatal re-opening mechanism positively modulated by LecRK-V-5 (Desclos-Theveniau et al., 2012). Interestingly, LecRK-V-5 over-expression lines are also defective in MAMP-induced stomatal closure. Together these data indicate that LecRK-V-5 negatively regulates Arabidopsis resistance to bacteria through fine-tuning of stomatal innate immunity (Desclos-Theveniau et al., 2012). Localized expression of LecRK-V-5 upon PTI activation at stomatal guard cells further supports a role for LecRK-V-5 in stomatal innate immunity (Desclos-Theveniau et al., 2012). Similarly to the *sci* mutant that shows a defective stomatal innate immunity but exhibits wild-type apoplastic immunity (Zeng et al., 2011), apoplastic PTI responses such as flg22-triggered oxidative burst, bacteria-mediated callose deposition and up-regulation of PTI marker genes are not affected in lecrk-V-5 mutants. COR treatments re-open closed stomata in lecrk-V-5 mutants (Desclos-Theveniau et al., 2012), suggesting that LecRK-V-5 acts upstream of COR. lecrk-V-3 mutants accumulate high levels of ROS in guard cells and chemical inhibition of ROS accumulation in lecrk-V-5 guard cells re-opens closed stomata (Desclos-Theveniau et al., 2012). By contrast, treatments with PAMPs increase guard cell ROS levels in wild-type, but no increase of ROS production was observed in Arabidopsis over-expressing LecRK-V-5 (Desclos-Theveniau et al., 2012). Since ROS induce stomatal closure, high levels of ROS and defective ROS accumulation may explain constitutive stomatal closure in lecrk-V-5 mutants and deficient stomatal closure in LecRK-V-5 over-expression lines, respectively. In addition, lines over-expressing LecRK-V-5 demonstrate a compromised ABA-mediated stomatal closure (Desclos-Theveniau et al., 2012), thus LecRK-V-5 functions in guard cell ABA signaling pathway downstream of MAMP perception. LecRK-V-5 may thus act at a specific branch involving ABA for the control of stomatal innate immunity and may negatively regulate ABA-mediated stomatal responses (Desclos-Theveniau et al., 2012). Negative regulation of stomatal innate immunity may have evolved in order to avoid the deleterious effects of a prolonged inhibition of photosynthesis that would be caused by decreased CO2 availability following prolonged stomatal closure.

**CONCLUSION**

Although new knowledge about lectin receptor kinases function and signaling has emerged recently, many questions still remain unanswered. For example, what are the potential ligands and downstream partners that modulate lectin receptor kinase-dependent innate immunity responses are critical points that need to be solved. Importantly, the unraveling of the mechanisms regulating ligands perception by lectin receptor kinases will provide further insights into how LeCRKs affect the plant response to pathogens. This may clarify whether these receptor kinases function as PRRs. Knowledge derived from such studies could lead to novel methods for managing plant disease resistance.

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