

PEARLS

L-type lectin receptor kinases: New forces in plant immunity

Yan Wang^{1,2}, Klaas Bouwmeester^{1,3*}

1 Laboratory of Phytopathology, Wageningen University, Wageningen, The Netherlands, **2** Department of Plant Pathology, Nanjing Agricultural University, Nanjing, China, **3** Plant-Microbe Interactions, Department of Biology, Utrecht University, Utrecht, The Netherlands

* klaas.bouwmeester@wur.nl

Introduction

To halt pathogen invasion, plants—unlike animals—solely depend on an innate immune system. They possess an expanded arsenal of cell surface-localized pattern recognition receptors (PRRs) to perceive microbe- and damage-associated molecular patterns (M/DAMPs), here collectively termed invasion patterns [1]. Prominent examples are flagellin sensing 2 (FLS2) and elongation factor Tu (EF-Tu) receptor (EFR), 2 receptor-like kinases (RLKs) with leucine-rich repeat (LRR) ectodomains that initiate defense upon recognition of bacterial flagellin and EF-Tu, respectively [2]. Emerging key players in plant immunity are the lectin receptor kinases, RLKs that are subdivided in 3 distinct classes based on their extracellular lectin domains, i.e., G- (GNA-related or S-locus), C- (calcium-dependent), and L- (legume) type [3]. All 3 are omnipresent in plants but absent in animals, which deploy distinct C-type lectin receptors (CLRs) to initiate innate immunity [4]. Recent years have witnessed an accelerated interest in plant lectin receptor kinases. In this Pearl, we summarize our current knowledge on L-type lectin receptor kinases (LecRKs) in plant immunity.

Signatures of adaptive evolution

LecRKs are widespread in vascular plants, but only a few have been identified in lower land plants. It remains unclear whether green algae contain genuine LecRKs, because the few listed previously all lack an extracellular lectin domain [5]. The current wealth of plant genomic data enables identification of LecRKs on a genome-wide scale [6–10]. Phylogenetic analyses of LecRKs in 5 Brassicaceae species showed that all contain the 9 distinct clades (I to IX) as initially delineated in *Arabidopsis thaliana* [3,11]. In contrast, clade III orthologs are absent in closely related sister species, whereas outgroup species even lack multiple clades [11]. Clades I, II, and III are thus specific to Brassicaceae and potentially harbor genes with lineage-specific traits. Evolutionary analysis revealed that gene expansion within these lineage-specific clades is driven by a complex interplay of polyploidy and local duplication events [11]. In comparison to the genome-wide average, *Arabidopsis* LecRKs are significantly enriched in duplicates, with an overrepresentation of tandem duplicates, of which most share whole-genome and gene-transposed duplication events. Moreover, LecRKs that evolved by tandem duplication show accelerated molecular evolution, suggesting strong diversifying or positive selection [11]. Similar dynamic evolution is not found in the nucleotide-binding leucine-rich repeat (NLR) resistance gene family or in a few other gene families analyzed in this manner. How this compares to other RLK gene families is as yet unknown.

Comparative analysis revealed that distant species also differ in clade composition. In comparison to cucumber, tomato lacks clade V homologs but has 7 more divergent LecRKs divided



OPEN ACCESS

Citation: Wang Y, Bouwmeester K (2017) L-type lectin receptor kinases: New forces in plant immunity. PLoS Pathog 13(8): e1006433. <https://doi.org/10.1371/journal.ppat.1006433>

Editor: Cyril Zipfel, THE SAINSBURY LABORATORY, UNITED KINGDOM

Published: August 17, 2017

Copyright: © 2017 Wang, Bouwmeester. This is an open access article distributed under the terms of the [Creative Commons Attribution License](https://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Funding: This work was supported by a STW-VENI grant from The Netherlands Organization for Scientific Research (KB), a NSFC grant 31501622 from the National Natural Science Foundation of China (YW), and the Food-for-Thought campaign from the Wageningen University Fund. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing interests: The authors have declared that no competing interests exist.

over 4 clades (Fig 1) [6,7]. These 4 are also found in *Nicotiana benthamiana* and are likely Solanaceae-specific [6]. In the more distant monocots, the clade composition is again different [8,9]. For example, rice has 4 clades in common with *Arabidopsis* but has a large species-specific clade of 36 LecRKs that largely expanded by tandem duplication [9]. It can thus be concluded that the LecRK superfamily underwent multiple lineage- and species-specific expansions, indicative of genetic adaptation to environmental factors during the course of evolution.

LecRKs in plant immunity

LecRKs are differentially expressed during plant growth and upon exposure to external stimuli, pointing to a wide array of functions. To date, few *LecRKs* are associated with functions in plant development or abiotic stress tolerance [12–16], and no altered morphology or abiotic stress sensitivity was found in a large set of *Arabidopsis* mutants representing 36 out of the 45 *LecRKs* [17]. Multiple studies, however, revealed roles in plant immunity (Fig 1), with the first one addressing *Arabidopsis* *LecRK-I.9* published in 2011 [18]. Mutants deficient in *LecRK-I.9* were found to be compromised in cell wall-associated defense and jasmonate signaling and showed increased susceptibility to the bacterium *Pseudomonas syringae* and the oomycete pathogens *Phytophthora brassicae* and *Phytophthora capsici* [18,19]. Accordingly, overexpression of *LecRK-I.9* enhanced resistance to these 3 pathogens. *Arabidopsis* *LecRK-VI.2* and *LecRK-V.5* were both found to function in stomatal immunity against bacterial pathogens, albeit via different mechanisms. *LecRK-VI.2* mutants are unable to inhibit bacterial invasion via stomatal closure and are therefore more susceptible to bacterial pathogens but not to fungi or oomycetes [17,20,21]. This *LecRK* was found to associate with the flagellin receptor FLS2, emphasizing its specificity in bacterial immunity [22]. Instead of open stomata, *LecRK-V.5* mutants have closed stomata. This is promoted by elevated levels of oxidative burst, and as a result, these mutants are resistant to bacterial pathogens [23,24]. In contrast, *LecRK-V.5* mutants are more susceptible to *Phytophthora* [17], suggesting a dual role for *LecRK-V.5* in defense to different pathogens. As yet, the molecular mechanisms that underlie this Janus-faced role in immunity are unknown. Other immunity-related *Arabidopsis* *LecRKs* were pinpointed by exposing *LecRK* mutants to different pathogens. Fourteen *Arabidopsis* *LecRKs* function in *Phytophthora* resistance, 5 of which also play roles in immunity to bacterial pathogens [17–24]. Five others are implicated in fungal resistance [16,17], including 1 that functions in the

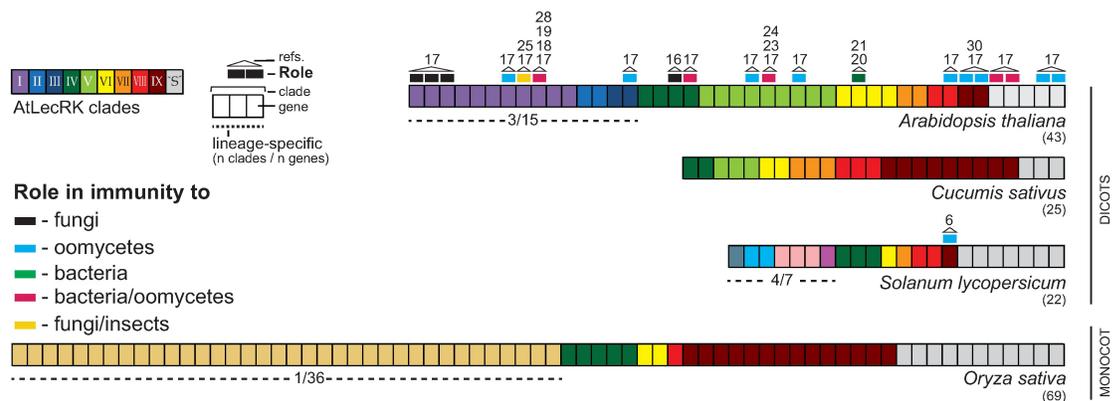


Fig 1. Clade composition of LecRK families in 4 different plant species. Lineage-specific clades are indicated with dashed lines. LecRKs involved in immunity are marked with colored pins. Numbers between brackets represent the amount of LecRKs with typical domain features, i.e., an extracellular lectin domain and an intracellular kinase domain. “S” represents singletons, LecRKs that do not belong to clades I–IX and group distantly.

<https://doi.org/10.1371/journal.ppat.1006433.g001>

perception of insect-egg deposition [25]. Homologs of clade IX LecRKs in tomato and *N. benthamiana* also function as immune receptors against *Phytophthora* [6], whereas a LecRK in pepper positively regulates immunity to diverse plant pathogens [26]. These data indicate that LecRKs have a conserved role in immunity throughout the plant kingdom.

Sensing invasion

The extracellular lectin domains of LecRKs have poorly conserved carbohydrate-binding residues but do compose a conserved hydrophobic cavity that is proposed to recognize hydrophobic ligands, e.g., complex glycans, hormones, and microbial invasion patterns [3]. Whether or not they bind monosaccharides or have agglutination activity similar to soluble legume lectins is unknown. So far, only ligands for 1 LecRK have been reported. *Arabidopsis* LecRK-I.9 was initially identified as a host target of the *Phytophthora infestans* effector IPI-O that interacts with LecRK-I.9 via the tripeptide Arg-Gly-Asp (RGD), a well-known cell attachment motif in mammalian cells [27]. Binding to IPI-O depends on 2 RGD-binding heptamers in the lectin domain [27]. The RGD-binding activity suggested a role for LecRK-I.9 in the maintenance of cell wall–plasma membrane (CW-PM) adhesions. Indeed, loss of LecRK-I.9 disrupts CW-PM integrity and host defense. This behavior as well as impaired resistance to *P. syringae* and *Phytophthora* pathogens was found to be phenocopied in *ipiO*-expressing *Arabidopsis* [18].

A second ligand is extracellular ATP (eATP). *LecRK-I.9* mutants are deficient in response to ATP and hence named “does not respond to nucleotides 1” (*dorn1*) [28]. In *in vitro* binding assays, the ectodomain of LecRK-I.9 has a relatively high binding affinity for ATP [28]. Mammalian purinoceptors that sense eATP were discovered many years ago and are well studied. Plants, however, have no homologs of purinoceptors. LecRK-I.9 is thereby the first identified eATP receptor in plants. Plants release eATP upon pathogen attack, and hence a model was proposed in which eATP acts as an invasion pattern that is recognized by LecRK-I.9 [28,29].

Launching defense

Our knowledge on immune signaling complexes is largely based on studies on the paradigmatic LRR-RLKs FLS2 and EFR [2]. Both heterodimerize with BRI1-associated receptor kinase 1 (BAK1)—a central coreceptor in defense signaling—in a ligand-dependent manner, and this leads to a cascade of phosphorylation events ultimately culminating in immunity. How do LecRKs activate defense and what are their downstream targets? Thus far, 2 LecRK interactors have been identified. One of them is ABCG40; a G-family ATP-binding cassette (ABC) transporter implicated in transport of the phytohormone abscisic acid (ABA). It was found to interact with *Arabidopsis* clade IX LecRKs that function in *Phytophthora* resistance and is required to mount adequate resistance [30]. Also, other LecRKs have links with ABA. LecRK-V.5 negatively regulates ABA-mediated stomatal closure, whereas LecRK-V.1 and clade VI LecRKs impact ABA responses during seed germination [14,15]. The second one is FLS2, which interacts with LecRK-VI.2 in a ligand-independent manner. It is remarkable, however, that loss of *LecRK-VI.2* does not affect FLS2-mediated association with BAK1 or phosphorylation of the receptor-like cytoplasmic kinase, botrytis-induced kinase 1 (BIK1) [22]. Since BIK1 is activated upon ligand binding, LecRK-VI.2 seems to function independently or downstream of BIK1. Interestingly, BAK1 is dispensable for *Arabidopsis* immunity mediated by lipooligosaccharide-specific reduced elicitation (LORE), a G-type LecRK that perceives bacterial lipopolysaccharides [31]. Whether LecRKs require coreceptors to mediate immunity remains to be determined.

Receptor dimerization is often a critical step in defense signaling [2]. As such, homo- or heterodimerization of LecRKs can also be hypothesized as well as the option that their lectin

domains dimerize with legume lectin-like proteins (LLPs). *Arabidopsis* LLPs are most homologous to clade VII and VIII LecRKs and also lack the essential residues for monosaccharide binding [11]. A role of these apoplastic LLPs in plant immunity is supported by the finding that overexpression of SA-induced legume lectin-like protein 1 (SAI-LLP1/LecP-I.7) in *Arabidopsis* potentiates defense to *P. syringae* [32]. Complex formation based on dimerization of extracellular domains has been observed for chitin elicitor receptor kinase 1 (CERK1), a lysin motif (LysM)-type lectin receptor kinase that perceives fungal chitin oligomers as an invasion pattern [2]. Upon chitin binding, *Arabidopsis* CERK1 forms homodimers, which activates intracellular signalling leading to fungal resistance. CERK1 also forms chitin-induced heterodimers with LYK5, an *Arabidopsis* LysM receptor kinase with an even stronger chitin-binding affinity. Interestingly, CERK1 also mediates immunity triggered by bacterial peptidoglycan [2]. CERK1 homodimers in rice, however, are unable to bind chitin directly and require heterodimerization with the chitin elicitor-binding protein (CEBiP), a membrane-spanning LysM protein that also lacks a kinase domain [2]. Another notable example is S-locus receptor kinase (SRK), a G-type LecRK that determines self-incompatibility in Brassicaceae by recognizing the pollen ligand S-locus cysteine-rich protein (SCR) during incompatible pollination [33]. Unlike CERK1, SRK can exist *in planta* as a homodimer prior to ligand binding. Structural studies recently revealed that SRC binding enhances homodimerization of SRK [34]. SRK also associates with S-locus glycoprotein (SLG), an apoplastic protein with a highly similar lectin domain, but whether SLG participates in a SCR-binding complex is still unknown [33]. In this respect, the multitude of LecRKs and LLPs (45 and 11 in *Arabidopsis*, respectively) may offer a wealth of combinatorial possibilities of receptor heterodimerization.

Exploiting LecRKs for crop resistance

LecRKs have several features that make them attractive as potential resistance components. They form a vast and diverse gene family, several of which confer broad-spectrum disease resistance. Another advantage is that LecRKs retain their function in plant immunity after interfamily transfer in a similar way as demonstrated for the LRR-RLK EFR [35]. This was shown for 3 *Arabidopsis* LecRKs when ectopically expressed in solanaceous plants [22,36,37]. As such, *LecRKs* have the potential to be introduced into distantly related species to increase disease resistance. A disadvantage, however, is that LecRK-mediated resistance is relatively mild in comparison to strain-specific resistance governed by intracellular NLR resistance proteins. Resistance can be enhanced by boosting *LecRK* expression, but caveats are pleiotropic effects on plant fitness [36,37]. For instance, ectopic expression of *LecRK-IX.1* can induce cell death. Fortunately, cell death in this case could be uncoupled from its resistance function [30,37], and this opens possibilities to minimize cell death without losing LecRK-mediated resistance. Exploiting LecRKs to improve crop resistance is within reach, especially when taking advantage of high throughput screening of TILLING populations or gene-editing tools such as CRISPR/Cas9. LecRKs can be used as a novel resource of cell surface receptors to boost immunity, and pyramiding LecRKs with classical intracellular NLRs may lead to a broader and more durable disease resistance.

Open questions

Our current understanding of how LecRKs function in plant defense is in its infancy, with many questions unanswered. Because LecRKs are largely diverse, they might recognize a variety of invasion patterns. Can the thus far detected immunogenic ligands be extrapolated to other LecRKs? Are there ligands that are specific for the lineage-specific LecRKs? Are other *Arabidopsis* LecRKs, besides LecRK-I.9, sensing eATP and RGD? And what is the eATP

receptor in plants that lack clade I LecRKs? The fact that LecRK-I.9 binds both RGD and eATP brings into question how these 2 processes are regulated. In this respect, it is of note that the mammalian eATP receptor P2Y₂ functions through interaction via its RGD motif with integrins that mediate cell integrity [38]. What type of components interact with the kinase domain of LecRKs? It remains elusive which components reside within LecRK signaling complexes. What is the role, if any, of common signaling partners, such as BAK1 and BIK1, and what is phosphorylated by LecRKs? It is also important to understand how microbial pathogens deal with LecRK-mediated immunity. Can pathogens compete via effectors for ligand binding to interfere with LecRK-mediated defense, or do they employ effectors that target LecRKs for degradation to promote host colonization? It is envisioned that integration of genetic, biochemical, and phylogenomics approaches will allow us to answer the abovementioned questions and support future resistance breeding.

Acknowledgments

We thank Francine Govers for constructive input on the manuscript and helpful suggestions, and we apologize to colleagues whose studies were not cited due to manuscript restrictions.

References

1. Cook DE, Mesarich CH, Thomma BPHJ (2015) Understanding plant immunity as a surveillance system to detect invasion. *Annu Rev Phytopathol* 53: 541–563. <https://doi.org/10.1146/annurev-phyto-080614-120114> PMID: 26047564
2. Couto D, Zipfel C (2016) Regulation of pattern recognition receptor signalling in plants. *Nat Rev Immunol* 16: 537–552. <https://doi.org/10.1038/nri.2016.77> PMID: 27477127
3. Bouwmeester K, Govers F (2009) Arabidopsis L-type lectin receptor kinases: phylogeny, classification, and expression profiles. *J Exp Bot* 60: 4383–4396. <https://doi.org/10.1093/jxb/erp277> PMID: 19773388
4. Dambuzza IM, Brown GD (2015) C-type lectins in immunity: recent developments. *Curr Opin Immunol* 32: 21–27. <https://doi.org/10.1016/j.coi.2014.12.002> PMID: 25553393
5. Lehti-Shiu MD, Shiu S-H (2012) Diversity, classification and function of the plant protein kinase superfamily. *Phil. Trans. R. Soc. B* 367: 2619–2639 <https://doi.org/10.1098/rstb.2012.0003> PMID: 22889912
6. Wang Y, Weide R, Govers F, Bouwmeester K (2015) L-type lectin receptor kinases in *Nicotiana benthamiana* and tomato and their role in *Phytophthora* resistance. *J Exp Bot* 66: 6731–6743. <https://doi.org/10.1093/jxb/erv379> PMID: 26248665
7. Wu T, Wang R, Xu X, He X, Sun B, Zhong Y, et al. (2014) *Cucumis sativus* L-type lectin receptor kinase (CsLecRK) gene family respond to *Phytophthora melonis*, *Phytophthora capsici* and water immersion in disease resistant and susceptible cucumber cultivars. *Gene* 549: 214–222. <https://doi.org/10.1016/j.gene.2014.07.058> PMID: 25065921
8. Shumayla, Sharma S, Pandey AK, Singh K, Upadhyay SK (2016) Molecular characterization and global expression analysis of lectin receptor kinases in bread wheat (*Triticum aestivum*). *PLoS ONE* 11: e0153925. <https://doi.org/10.1371/journal.pone.0153925> PMID: 27111449
9. Vaid N, Pandey PK, Tuteja N (2012) Genome-wide analysis of lectin receptor-like kinase family from Arabidopsis and rice. *Plant Mol Biol* 80: 365–388. <https://doi.org/10.1007/s11103-012-9952-8> PMID: 22936328
10. Van Holle S, Van Damme EJM (2015) Distribution and evolution of the lectin family in soybean (*Glycine max*). *Molecules* 20: 2868–2891. <https://doi.org/10.3390/molecules20022868> PMID: 25679048
11. Hofberger JA, Nsibo DL, Govers F, Bouwmeester K, Schranz ME (2015) A complex interplay of tandem- and whole genome duplication drives expansion of the L-type lectin receptor kinase gene family in the Brassicaceae. *Genome Biol Evol* 7: 720–734. <https://doi.org/10.1093/gbe/evv020> PMID: 25635042
12. Wan J, Patel A, Mathieu M, Kim SY, Xu D, Stacey G (2008) A lectin receptor-like kinase is required for pollen development in Arabidopsis. *Plant Mol Biol* 67: 469–482. <https://doi.org/10.1007/s11103-008-9332-6> PMID: 18392777
13. Vaid N, Pandey P, Srivastava VK, Tuteja N (2015) Pea lectin receptor-like kinase functions in salinity adaptation without yield penalty, by alleviating osmotic and ionic stresses and upregulating stress-responsive genes. *Plant Mol Biol* 88:193–206. <https://doi.org/10.1007/s11103-015-0319-9> PMID: 25863480

14. Xin Z, Wang A, Yang G, Gao P, Zheng ZL (2009) The Arabidopsis A4 subfamily of lectin receptor kinases negatively regulates abscisic acid response in seed germination. *Plant Physiol* 149:434–444. <https://doi.org/10.1104/pp.108.130583> PMID: 18987212
15. Deng K, Wang Q, Zeng J, Guo X, Zhao X, Tang D, et al. (2009) A lectin receptor kinase positively regulates ABA response during seed germination and is involved in salt and osmotic stress response. *J. Plant Biol* 52: 493–500.
16. Huang P, Ju HW, Min JH, Zhang X, Kim SH, Yang KY, et al. (2013) Overexpression of L-type lectin-like protein kinase 1 confers pathogen resistance and regulates salinity response in *Arabidopsis thaliana*. *Plant Sci* 203: 98–106. <https://doi.org/10.1016/j.plantsci.2012.12.019> PMID: 23415333
17. Wang Y, Bouwmeester K, Beseh P, Shan W, Govers F (2014) Phenotypic analyses of Arabidopsis T-DNA insertion lines and expression profiling reveal that multiple L-type lectin receptor kinases are involved in plant immunity. *Mol Plant Microbe Interact* 27: 1390–1402. <https://doi.org/10.1094/MPMI-06-14-0191-R> PMID: 25083911
18. Bouwmeester K, de Sain M, Weide R, Gouget A, Klamer S, Canut H, et al. (2011) The lectin receptor kinase LecRK-I.9 is a novel *Phytophthora* resistance component and a potential host target for a RXLR effector. *PLoS Pathog* 7: e1001327. <https://doi.org/10.1371/journal.ppat.1001327> PMID: 21483488
19. Balague C, Gouget A, Bouchez O, Souriac C, Haget N, Boutet-Mercery S, et al. (2016) The *Arabidopsis thaliana* lectin receptor kinase LecRK-I.9 is required for full resistance to *Pseudomonas syringae* and affects jasmonate signalling. *Mol Plant Pathol* <https://doi.org/10.1111/mpp.12457> PMID: 27399963
20. Singh P, Kuo YC, Mishra S, Tsai CH, Chien CC, Desclos-Theveniau M, et al. (2012) The lectin receptor kinase-VI.2 is required for priming and positively regulates Arabidopsis pattern-triggered immunity. *Plant Cell* 24: 1256–1270. <https://doi.org/10.1105/tpc.112.095778> PMID: 22427336
21. Singh P, Chien CC, Mishra S, Tsai CH, Zimmerli L (2013) The Arabidopsis LECTIN RECEPTOR KINASE-VI.2 is a functional protein kinase and is dispensable for basal resistance to *Botrytis cinerea*. *Plant Signal Behav* 8: 1–3. <http://doi.org/10.4161/psb.22611> PMID: 23221759
22. Huang PY, Yeh YH, Liu AC, Chien CP, Zimmerli L (2014) The Arabidopsis LecRK-VI.2 associates with the pattern-recognition receptor FLS2 and primes *Nicotiana benthamiana* pattern-triggered immunity. *Plant J* 79: 243–255. <https://doi.org/10.1111/tpj.12557> PMID: 24844677
23. Desclos-Theveniau M, Arnaud D, Huang TY, Lin GJC, Chen WY, Lin YC, et al. (2012) The Arabidopsis lectin receptor kinase LecRK-V.5 represses stomatal immunity induced by *Pseudomonas syringae* pv. *tomato* DC3000. *PLoS Pathog* 8: e1002513. <https://doi.org/10.1371/journal.ppat.1002513> PMID: 22346749
24. Arnaud D, Desclos-Theveniau M, Zimmerli L (2012) Disease resistance to *Pectobacterium carotovorum* is negatively modulated by the Arabidopsis lectin receptor kinase LecRK-V.5. *Plant Signal Behav* 7: 1070–1072. <https://doi.org/10.4161/psb.21013> PMID: 22899085
25. Gouhier-Darimont C, Schmiesing A, Bonnet C, Lassueur S, Reymond P (2013) Signalling of *Arabidopsis thaliana* response to *Pieris brassicae* eggs shares similarities with PAMP-triggered immunity. *J Exp Bot* 64: 665–674. <https://doi.org/10.1093/jxb/ers362> PMID: 23264520
26. Woo JY, Jeong KJ, Kim YJ, Paek K-H (2016) CaLecRK-S.5, a pepper L-type lectin receptor kinase gene, confers broad-spectrum resistance by activating priming. *J Exp Bot* 67: 5725–5741. <https://doi.org/10.1093/jxb/erw336> PMID: 27647723
27. Gouget A, Senchou V, Govers F, Sanson A, Barre A, Rougé P, et al. (2006) Lectin receptor kinases participate in protein-protein interactions to mediate plasma membrane-cell wall adhesions in Arabidopsis. *Plant Physiol* 140: 81–90. <https://doi.org/10.1104/pp.105.066464> PMID: 16361528
28. Choi J, Tanaka K, Cao Y, Qi Y, Qiu J, Stacey G (2014) Identification of a plant receptor for extracellular ATP. *Science* 343: 290–294. <https://doi.org/10.1126/science.1243618> PMID: 24436418
29. Choi J, Tanaka K, Liang Y, Cao Y, Lee SY, Stacey G (2014) Extracellular ATP, a danger signal, is recognized by DORN1 in Arabidopsis. *Biochem J* 463: 429–437. <https://doi.org/10.1042/BJ20140666> PMID: 25301072
30. Wang Y, Cordewener JHG, America AHP, Shan W, Bouwmeester K, Govers F (2015) Arabidopsis lectin receptor kinases LecRK-IX.1 and LecRK-IX.2 are functional analogs in regulating *Phytophthora* resistance and plant cell death. *Mol Plant Microbe Interact* 28: 1032–1048. <https://doi.org/10.1094/MPMI-02-15-0025-R> PMID: 26011556
31. Ranf S, Gisch N, Schäffer M, Illig T, Westphal L, Knirel YA, et al. (2015) A lectin S-domain receptor kinase mediates lipopolysaccharide sensing in *Arabidopsis thaliana*. *Nat Immunol* 16: 426–433. <https://doi.org/10.1038/ni.3124> PMID: 25729922
32. Armijo G, Salinas P, Monteoliva MI, Seguel A, García C, Villarroel-Candia E, et al. (2013) A salicylic acid-induced lectin-like protein plays a positive role in the effector-triggered immunity response of *Arabidopsis thaliana* to *Pseudomonas syringae* Avr-Rpm1. *Mol Plant Microbe Interact* 26: 1395–1406. <https://doi.org/10.1094/MPMI-02-13-0044-R> PMID: 24006883

33. Sanabria N, Goring D, Nürnberger T, Dubery I (2008). Self/nonself perception and recognition mechanisms in plants: A comparison of self-incompatibility and innate immunity. *New Phytol* 178: 503–514. <https://doi.org/10.1111/j.1469-8137.2008.02403.x> PMID: 18346103
34. Ma R, Han Z, Hu Z, Lin G, Gong X, Zhang H, Nasrallah JB, Chai J (2016) Structural basis for specific self-incompatibility response in Brassica. *Cell Res* 26: 1320–1329. <https://doi.org/10.1038/cr.2016.129> PMID: 27824028
35. Lacombe S, Rougon-Cardoso A, Sherwood E, Peeters N, Dahlbeck D, van Esse HP, et al. (2010) Inter-family transfer of a plant pattern-recognition receptor confers broad-spectrum bacterial resistance. *Nat Biotechnol* 28: 365–369 <https://doi.org/10.1038/nbt.1613> PMID: 20231819
36. Bouwmeester K, Han M, Blanco-Portales R, Song W, Weide R, Guo LY, et al. (2014) The Arabidopsis lectin receptor kinase LecRK-I.9 enhances resistance to *Phytophthora infestans* in Solanaceous plants. *Plant Biotechnol J* 12: 10–16. <https://doi.org/10.1111/pbi.12111> PMID: 23980842
37. Wang Y, Nsibo DL, Juhar HM, Govers F, Bouwmeester K (2016) Ectopic expression of Arabidopsis L-type lectin receptor kinase genes *LecRK-I.9* and *LecRK-IX.1* confers *Phytophthora* resistance in *Nicotiana benthamiana*. *Plant Cell Rep* 35: 845–855. <https://doi.org/10.1007/s00299-015-1926-2> PMID: 26795144
38. Erb L, Weisman GA (2012) Coupling of P2Y receptors to G proteins and other signaling pathways. *Wiley Interdiscip Rev Membr Transp Signal* 1: 789–803. <https://doi.org/10.1002/wmts.62> PMID: 25774333