The Lectin Receptor Kinase-VI.2 is required for priming and positively regulates Arabidopsis pattern-triggered immunity

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Plant cells can be sensitized toward a subsequent pathogen attack by avirulent pathogens or by chemicals such as beta-aminobutyric acid (BABA). This process is called priming. Using a reverse genetic approach in Arabidopsis thaliana, we demonstrate that the BABA-responsive L-type lectin receptor kinase-VI. 2 (LecRK-VI.2) contributes to disease resistance against the hemibiotrophic Pseudomonas syringae and the necrotrophic Pectobacterium carotovorum bacteria. Accordingly, LecRK-VI.2 mRNA levels increased after bacterial inoculation or treatments with microbe-associated molecular patterns (MAMPs). We also show that LecRK-VI.2 is required for full activation of pattern-triggered immunity (PTI); notably, lecrk-VI.2-1 mutants show reduced upregulation of PTI marker genes, impaired callose deposition, and defective stomatal closure. Overexpression studies combined with genome-wide microarray analyses indicate that LecRK-VI.2 positively regulates the PTI response. LecRK-VI.2 is demonstrated to act upstream of mitogen-activated protein kinase signaling, but independently of reactive oxygen production and BOTRYTIS-INDUCED KINASE1 phosphorylation. In addition, complex formation between the MAMP receptor FLAGELLIN SENSING2 and its signaling partner BRASSINOSTEROID INSENSITIVE1-ASSOCIATED KINASE1 is observed in flg22-treated lecrk-VI.2-1 mutants. LecRK-VI.2 is also required for full BABA-induced resistance and priming of PTI. Our work identifies LecRK-VI.2 as a novel mediator of the Arabidopsis PTI response and provides insight into molecular mechanisms governing priming.

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