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The leucine-rich repeat receptor-like kinase PpLRR1 enhances peach resistance to *Xanthomonas arboricola* pv. *pruni*

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Abstract

Plants have evolved sophisticated defense mechanisms to counter pathogen infections, among which the hypersensitive response (HR) mediated by leucine-rich repeat receptor-like kinases (LRR-RLKs) plays a key role in limiting pathogen spread. However, whether LRR-RLKs mediate HR to confer resistance against *Xanthomonas arboricola* pv. *pruni* (Xap) in peach (*Prunus persica*) remains unclear. This study identified the LRR-RLK gene family member, *PpLRR1*, which was significantly upregulated in response to Xap infection. Functional analysis revealed that *PpLRR1* is essential for HR signaling and enhances resistance to pathogen attack. Transient overexpression of *PpLRR1* significantly reduced disease symptoms, whereas its silencing aggravated infection. Further studies demonstrated that the MYB transcription factor PpLIMYB directly binds to the *PpLRR1* promoter and activates its transcription. These findings highlight the important role of the PpLIMYB-*PpLRR1* regulatory module in peach disease resistance. This study advances the understanding of disease resistance mechanisms in woody plants and identifies a potential target for breeding disease-resistant cultivars.

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Plants are continuously exposed to pathogen attacks in natural environments. Through evolution, they have developed a sophisticated immune system to combat pathogens[1]. The first layer, Pathogen-associated Molecular Patterns (PAMPs)-triggered immunity (PTI), is initiated when pattern recognition receptors (PRRs) such as leucine-rich repeat receptor-like kinases (LRR-RLKs) detect conserved PAMPs. When pathogens bypass PTI via effector secretion, plants activate effector-triggered immunity (ETI), typically mediated by nucleotide-binding leucine-rich repeat (NLR) proteins that recognize specific effectors[2]. Both PTI and ETI can trigger the hypersensitive response (HR)[3], a form of programmed cell death (PCD) that restricts pathogen spread by forming a barrier of dead cells at infection sites. LRR-RLKs not only initiate PTI but also modulate HR through phosphorylation cascades regulating reactive oxygen species (ROS) burst and defense gene expression[4], playing a central role in early immune signaling and resistance responses. In *Arabidopsis thaliana*, LRR-RLK genes such as *FLS2* and *EFR* detect bacterial flagellin and elongation factor EF-Tu, respectively[5]. However, the role of LRR-RLKs in resistance to *Xanthomonas arboricola* pv. *pruni* (Xap) in peach (*Prunus persica*) remains unclear. Xap causes bacterial spot disease, leading to leaf necrosis, fruit drop, and up to 30% yield loss[6]. Although chemical control can partially reduce symptoms, the lack of resistant cultivars remains a major limitation. Therefore, identifying resistance genes and elucidating their regulatory mechanisms is essential for peach breeding[7]. Here, an LRR-RLK gene, *PpLRR1*, that contributes to HR, was identified. Further analysis revealed that PpLIMYB transcription factor (TF) regulates *PpLRR1* expression. These findings offer insights into *PpLRR1*-mediated resistance and provide valuable genetic resources for breeding disease-resistant peach varieties.

Healthy leaves of 'Guanghetao (GHT, *Prunus mira*)' and 'Qiubaitao (QBT, *Prunus persica*)' were inoculated with Xap. Disease symptoms appeared at 2 d post-inoculation (dpi) and became more pronounced by 4 dpi (Fig. 1a). Leaves were sampled at 0, 2, and 4 dpi for transcriptome sequencing, which identified ten upregulated LRR-RLK differentially expressed genes (DEGs) (Fig. 1b). Of these, four DEGs Prupe.1G168500, Prupe.4G069400, Prupe.4G069600, and Prupe.7G262300 were selected for preliminary functional validation based on their significant expression in both GHT and QBT (Supplementary Fig. S1). Among these, Prupe.4G069400 and Prupe.4G069600 encoded identical proteins; therefore, only one was used in subsequent analyses. Transient expression of the three candidate genes in *Nicotiana benthamiana* revealed that only *PpLRR1* induced PCD (Fig. 1c), a response also observed in peach leaves (Fig. 1d; Supplementary Table S1). qRT-PCR analysis showed a significant upregulation of *PpLRR1* at 4 dpi (Fig. 1e), suggesting its involvement in hypersensitive response (HR)-mediated cell death during pathogen infection. Therefore, *PpLRR1* was chosen as the primary candidate for further study. Phylogenetic analysis (Supplementary Fig. S2) placed *PpLRR1* within the LRR-RLK-IX subfamily, clustering closely with *AtBARK1* from Arabidopsis and *NbBIR1* from tobacco[8]. Additional analyses of gene structure and physicochemical properties of LRR-RLK-IX subfamily members were performed (Supplementary Table S2). The *PpLRR1*-encoded protein contains a canonical LRR-TM-PK (leucine-rich repeat–transmembrane–protein kinase) domain architecture, consistent with its predicted function as a receptor-like kinase and its homology to known plant resistance genes (Supplementary Fig. S3). Subcellular localization assays confirmed PpLRR1 is primarily localized to the plasma membrane (Fig. 1f), supporting its role as a pattern recognition receptor (PRR) involved in pathogen perception[9].

To further validate the functional role of *PpLRR1* in disease resistance, the gene was co-expressed with the pathogen in tobacco leaves via *Agrobacterium*-mediated transformation. Trypan blue staining showed that *PpLRR1* significantly reduced disease severity (Fig. 1g), consistent with the effects of *LcPIP1* in *Litchi chinensis*[10], and *Xa21* in *Oryza sativa*[3]. The overexpression of *PpLRR1* significantly mitigated disease symptoms and reduced lesion area

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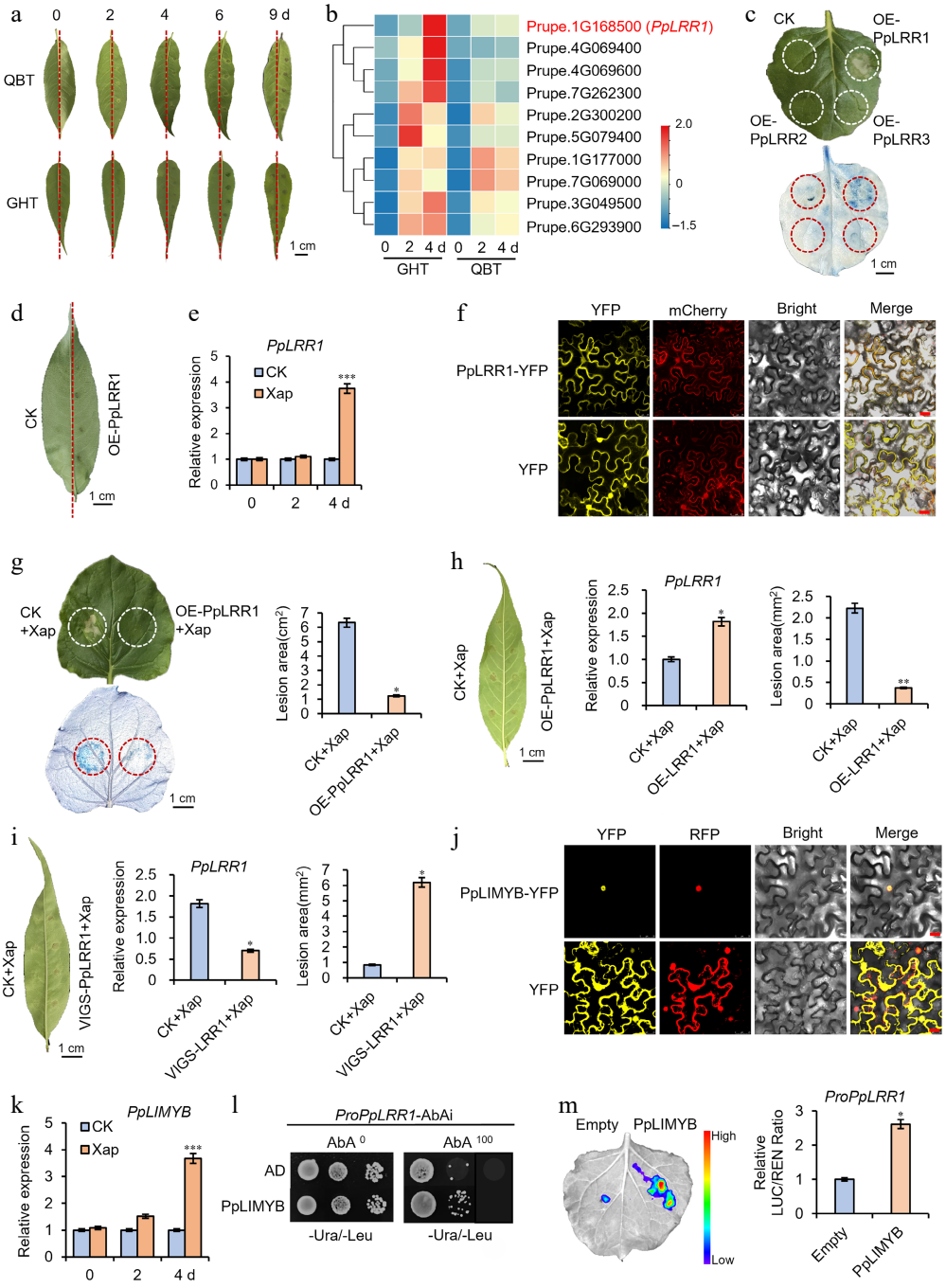

Fig. 1 Functional validation of *PpLRR1* in resistance to peach bacterial spot disease. (a) Phenotypic comparison of peach leaves from 'Qiubaitao (QBT, *Prunus persica*)' and 'Guanghetao (GHT, *Prunus mira*)' cultivars inoculated with *Xanthomonas arboricola* pv. *pruni* (Xap) vs control at 4 dpi. Transcriptome sequencing was conducted on leaf samples at 0, 2, and 4 dpi. Scale bar = 1 cm. (b) Heatmap of differentially expressed genes ($|\log_2 FC| > 1$, $p < 0.05$) identified from RNA-seq data. *PpLRR1* is highlighted in red. (c) Trypan blue-stained tobacco leaves transiently overexpressing *PpLRR1*, *PpLRR2*, or *PpLRR3* compared to the empty vector control at 3 dpi. (d) Hypersensitive response phenotype in peach leaves transiently overexpressing *PpLRR1* at 4 dpi. (e) Relative expression of *PpLRR1* in 'Mantianhong (MTH)' peach leaves at 0, 2, and 4 dpi, measured by qRT-PCR. Expression was normalized to the sterile water-injected control, set to 1. (f) Subcellular localization of PpLRR1-YFP fusion protein in *Nicotiana benthamiana* epidermal cells. Yellow fluorescence (YFP) co-localizes with the red fluorescent plasma membrane marker (mCherry). OD_{600} = 1.0. Cultivate for 48 h after injection. Scale bar = 25 μm. (g) Quantification of lesion area in tobacco leaves co-inoculated with Xap and overexpressing *PpLRR1* at 3 dpi (trypan blue staining). (h) Lesion area and relative *PpLRR1* expression in MTH peach leaves co-inoculated with Xap and overexpressing *PpLRR1* at 4 dpi. (i) Lesion area and *PpLRR1* expression in MTH peach leaves co-inoculated with Xap following *PpLRR1* silencing. TRV, TRV1 + TRV2; PpLRR1-TRV, TRV1 + PpLRR1-TRV2. (j) Subcellular localization of PpLIMYB-YFP fusion protein in transgenic *N. benthamiana* epidermal cells. Yellow fluorescent signal (YFP) co-localizes with RFP (red). TM–NR: tonoplast marker and red fluorescent nuclear marker (Nucleus–RFP). OD_{600} = 1.0. Cultivate for 48 h after injection. Scale bar = 25 μm. (k) Relative expression levels of *PpLIMYB* in peach leaves at 0, 2, and 4 dpi assessed by qRT-PCR. Expression was normalized to the sterile water-injected control, set to 1. (l) Yeast one-hybrid assay showing direct binding of PpLIMYB to the *PpLRR1* promoter. AbA^0: medium without AbA; AbA^{100}: medium with 100 ng/mL AbA. (m) Dual-luciferase reporter assay quantifying PpLIMYB-mediated activation of the *PpLRR1* promoter. Data represent mean ± SD of three biological replicates (*p < 0.05). Each treatment was performed in triplicate, and each replicate contained three to five peach leaves. Error bars indicate standard deviation. Statistical significance was assessed by one-way ANOVA and *t*-test: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

(Fig. 1h), while gene silencing led to enhanced susceptibility and increased lesion area (Fig. 1i), further supporting its role in resistance. Moreover, transgenic Arabidopsis plants overexpressing *PpLRR1* exhibited significantly reduced lesion areas and milder disease symptoms upon Xap inoculation compared to wild-type plants (Supplementary Fig. S4), confirming its functional contribution to peach disease resistance by mediating HR and limiting pathogen spread.

To investigate how PpLRR1 regulates disease resistance, its promoter region was analyzed (Supplementary Fig. S5), and multiple stress-responsive cis-elements were identified, including binding sites for 14 MYB (C/TAACT/CG/A), three MYC (CAA/TTTG), two WRKY (TTGACC/T), and two bHLH (G-box: CACGTG) TFs. Notably, MYB-binding sites were the most abundant. This is significant given the established role of MYB TFs as central regulators of plant immunity, including antiviral defense[11,12] and antibacterial responses[13]. Although several TF families may regulate *PpLRR1*, focus was placed on MYB TFs. Transcriptome analysis identified 17 MYB-related TFs that were both pathogen-responsive and significantly upregulated. Among these, PpLIMYB was identified via yeast one-hybrid (Y1H) assays. Subcellular localization confirmed its nuclear function, and qRT-PCR revealed strong upregulation at 4 dpi, mirroring the *PpLRR1* expression pattern (Fig. 1k). Additionally, co-overexpression of *PpLIMYB* and Xap in peach leaves led to simultaneous upregulation of *PpLRR1* (Supplementary Fig. S6), suggesting a regulatory relationship. Y1H (Fig. 1l) and dual-luciferase reporter assays (Fig. 1m) confirmed that PpLIMYB directly binds to the *PpLRR1* promoter and activates its transcription.

These findings reveal the important role of the PpLIMYB-*PpLRR1* module in peach immunity against Xap (Supplementary Fig. S7). Similar to Arabidopsis LRR-RLKs (e.g., *FLS*), which activate MAPK-mediated defense signaling[5], *PpLRR1* likely acts downstream of PpLIMYB to regulate defense genes (e.g., *PR1* and *PR5*). The plant immune response is a complex process that may also trigger other immune responses, such as the activation of downstream immune genes, calcium ion influx, callose deposition, and the production of phytoalexins, among other cascading reactions. This mechanism resembles *LcPIP1*-mediated resistance in litchi. The membrane localization of *PpLRR1* (Fig. 1e) suggests it perceives extracellular pathogen signals. Its activation of *PpLRR1* by PpLIMYB provides new evidence for a 'receptor-TF' regulatory module in plant immunity. Future studies should examine interactions between PpLRR1 and co-receptors[14,15], and evaluate the conservation of this module across peach cultivars. *PpLRR1* is strongly induced by Xap infection and confers resistance by activating downstream defenses. Therefore, these findings not only provide valuable genetic resources for breeding disease-resistant peach varieties but also offer an insight into the specific regulatory interactions in peach, a woody fruit tree with unique genetic and physiological characteristics. Further research will clarify the molecular functions of *PpLRR1* and its applications in resistance breeding.

Author contributions

The authors confirm their contributions to the paper as follows: study conception and design: An JP; data collection: Li MY, Zhao L, Di A, Li ZY; analysis and interpretation of results: Li MY, Liao L, Han Y, Luo CX; draft manuscript preparation: Li MY, An JP. All authors reviewed the results and approved the final version of the manuscript.

Data availability

All data generated or analyzed during this study are included in this published article and its supplementary information files.

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Conflict of interest

The authors declare that they have no conflict of interest.

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