

# Structure and dynamics of the plant immune signaling network in plant–bacteria interactions

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## Abstract

Plants are equipped with immune receptor proteins on the cell surface or inside the cell to sense microbial invasions. Perception of microbe-associated molecular patterns (MAMPs), which are derived from conserved molecules in a class of microbes such as bacterial flagellin, triggers pattern-triggered immunity (PTI). While pathogens deploy a repertoire of effectors that dampen PTI, plants detect the presence or actions of effectors and induce effector-triggered immunity (ETI) to stop pathogen infection. PTI and ETI activate shared signaling components such as reactive oxygen species, mitogen-activated protein kinases (MAPKs) and phytohormones to reprogram the cell state toward defense responses. It has become increasingly clear that these signaling components interact with each other to form an intricate network<sup>1</sup>.

The immune phytohormones jasmonic acid (JA) and salicylic acid (SA) accumulate concomitantly during PTI. I discovered a signaling network where JA enables tunable and robust SA accumulation: JA suppresses SA accumulation to mitigate SA-dependent growth retardation but promotes SA accumulation when a network component, PAD4, is perturbed by pathogen effectors or high temperature. I also uncovered that plant immune signaling networks are exploited by bacterial pathogens: the phytotoxin coronatine produced by various pathovars of the bacterial pathogen *Pseudomonas syringae* activates JA signaling to inhibit not only SA accumulation but also MAPK activation. Interestingly, however, plants can counteract these virulence actions of coronatine and reestablish disease resistance during ETI.

Overall, my research achievements have shed light on the molecular mechanisms underlying the structure and dynamics of plant immune signaling networks that operate behind plant–bacteria interactions. I anticipate that these studies provide a foundation for further understanding of plant–pathogen interactions from a network perspective, which will eventually allow us to manipulate plant immune signaling networks for designing disease resistance according to pathogen virulence mechanisms and environmental conditions in agricultural fields.

## References

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