Transcriptional regulation of effector-triggered immunity (ETI) in plants: from tissue to cells

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Stellingen
Behorende bij het proefschrift

Transcriptional regulation of effector-triggered immunity (ETI) in plants: from tissue to cells

1. The EDS1–PAD4-ADR1 signaling node plays a central role in mediating ETI-induced growth arrest. (This thesis)

2. Plants with quantitatively reduced immunity retain the capacity for immune priming and enhanced disease resistance. (This thesis)

3. The *cbp60g sard1* double mutant provides a powerful genetic model to dissect the relationship between disease resistance and the hypersensitive response (HR). (This thesis)

4. All leaf cell types possess the capacity to activate a shared core immune transcriptional program, while retaining a distinguishable cell-type-specific transcriptional responses during effector-triggered immunity (ETI). (This thesis)

5. Cell-surface and intracellular immune pathways operate within an integrated and mutually reinforcing plant immune network. (Ngou et al., 2021; Tian et al., 2021; Yuan et al., 2021)

6. EDS1-dependent immune signaling is functionally diversified through a modular architecture that enables the genetic separation of immune outputs such as defense activation and cell death. (Wagner et al., 2013; Wu et al., 2018; Lapin et al., 2019; Saile et al., 2020; Sun et al., 2021)

7. Synthetic immune activation systems enable causal dissection of signaling pathways by uncoupling immune activation from pathogen-derived inputs. (Ngou et al., 2020; Ngou et al., 2021)

8. Master transcription factors such as CBP60g and SARD1 function as regulatory hubs that coordinate both amplification and attenuation of immune responses. (Sun et al., 2015)

9. Scientific progress is accelerated by effective teamwork and coordination rather than individual effort alone.

10. There is no substitute for hard work, but its impact can be maximized through strategic and efficient execution.

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