

PROJECT TITLE

Investigating Ca²⁺ signaling in priming and Systemic Acquired Resistance

CONSORTIUM

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SUMMARY OF THE REPORT

Ca²⁺ is a universal second messenger in all eukaryotes. Transient changes in cytosolic Ca²⁺ are rapidly generated upon a diverse range of stimuli such as drought, heat, wounding, and biotic stresses such as pathogen infection. To generate stimulus-specific signals that can elicit the appropriate responses, Ca²⁺ signals must contain unique information, and this will be decoded and transferred to downstream signaling cascades by Ca²⁺ sensor proteins such as calmodulins and Calcineurin B-like proteins (CBLs).

A genetically encoded Ca²⁺ reporter, GCaMP3, was introduced into *Arabidopsis* and *Nicotiana benthamiana* to measure changes in cytosolic Ca²⁺ upon treatment with two strains of the pathogen, *Pseudomonas syringae*, *Pseudomonas syringae* pv. *tomato* DC3000 (DC3000, for *Arabidopsis*) or *Pseudomonas syringae* pv. *tabaci* (*Pta*, for *N. benthamiana*) (deFalco et al., 2017)

In this project, several experiments were conducted to investigate:

a) the priming effect on immunity-related Ca²⁺ signals mainly using the priming agent pipecolic acid.

b) the role in immunity-related Ca²⁺ signaling of 1) Cyclic Nucleotide-gated Channels (CNGCs, using *Arabidopsis* knockout mutants), and 2) a set of newly identified immune signaling components, CBL-interacting protein kinases6 (Cipk6, de la Torre et al., 2013) and a set of Cipk6-interactors (using Virus-induced gene silencing (VIGS) in *Nicotiana benthamiana*).