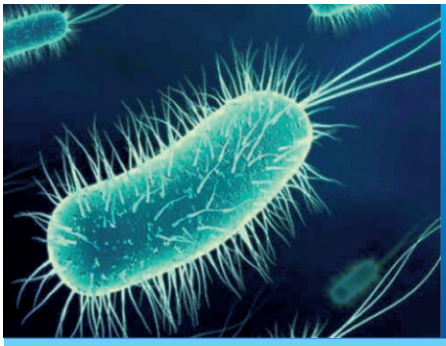

Autoacetylation of the *Ralstonia solanacearum* effector PopP2 targets a lysine residue essential for RRS1-R-mediated immunity in *Arabidopsis*

Céline Tasset, Maud Bernoux, Alain Jauneau, Cécile Pouzet, Christian Brière, Sylvie Kieffer-Jacquinod, Susana Rivas, Yves Marco and Laurent Deslandes

Plant and animal bacterial pathogens have evolved to produce virulence factors, called type III effectors, which are injected into host cells to suppress host defences and promote pathogen development. In plants, host proteins targeted by some effectors called avirulence proteins are surveyed by plant disease resistance proteins referred to as "guards". The *Ralstonia solanacearum* effector protein PopP2 triggers immunity in *Arabidopsis* following its perception by the RRS1-R resistance protein. In the present work, we demonstrated that PopP2 stabilizes and associates with RRS1-R in the plant nucleus. This study represents the first report of an Avr protein (PopP2) that interacts with its matching resistance protein (RRS1-R) in living plant cells.

Type III effectors often display enzymatic activities, mimicking an endogenous eukaryotic activity that target host signalling components. PopP2 belongs to the YopJ-like family of cysteine proteases that are predicted to act as acetyl-transferases on host targets. Here, we were able to show that PopP2 autoacetylates on a particular lysine residue well-conserved among YopJ-like family members. This lysine is a critical residue since its mutation prevents autoacetylation of PopP2 and abolishes its recognition by the host. PopP2 represents the first example of a T3E from phytopathogenic bacteria that displays an acetyl-transferase activity.

This study provides new clues on the activities displayed by bacterial type III effectors that may be used to target defense-related host components. Future work will focus on the identification of host proteins targeted by PopP2 activity and should contribute to the understanding of the molecular role(s) played by protein acetylation during plant innate immunity.

RRS1-R plants