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The bacterial effector HopZ1a acetylates the ZIP1 kinase to suppress *Arabidopsis* defence responses

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During the plant-pathogen interaction, disease or resistance are determined in the plant by a series of molecular events. The plant detects Pathogen-Associated Molecular Patterns (PAMPs), such as flagellin, triggering a defence response called PTI (PAMP-Triggered Immunity). Bacterial pathogens can in turn suppress such defence response through the translocation into the plant cell cytosol of virulence proteins, called effectors, via a Type Three Secretion System (T3SS). In resistant plants, intracellular receptors known as R proteins recognize these effectors, triggering a second line of defence, more specific and intense, called ETI (Effector-Triggered Immunity), which usually leads to programmed cell death known as HR (Hypersensitive Response). *Pseudomonas syringae* is a phytopathogenic bacterium whose virulence depends on a T3SS and its effector repertoire. Some strains include HopZ1a, an unusual effector which is able to suppress in *Arabidopsis* both local (PTI and ETI), and systemic (SAR, for Systemic Acquired Resistance) defences, by means of its acetyltransferase activity. In resistant *Arabidopsis* plants, HopZ1a acetylates the ZED1 pseudokinase, which is proposed to function as a decoy mimicking HopZ1a target in the plant: ZED1 modification activates an R-protein (ZAR1) to trigger HopZ1a-dependent ETI. None of the *Arabidopsis* proteins proposed to date as HopZ1a targets is a kinase, nor fully explains the effector´s defence suppression abilities. In this work we identify an *Arabidopsis* kinase that functions as a positive regulator of PTI, ETI and SAR, which interacts with HopZ1a and is acetylated by this effector in lysine residues essential for its kinase activity. Further, HopZ1a can specifically suppress the defence phenotypes resulting from ZIP1 expression in *Arabidopsis*. We propose that ZIP1 acetylation by HopZ1a interferes with its kinase activity, and consequently with positive defence signalling.