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HopAF1 associates with Arabidopsis heterotrimeric G-protein subunits

Javier Rueda Blanco
jrblanco@uma.es

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RESUMEN

Pseudomonas syringae is a bacterial plant pathogen that uses its Type III Secretion System to inject virulence proteins (type III effectors, T3Es) into the plant cell, interfering with the immune system to suppress defense. Some T3Es can be detected, triggering a defense response called ETI (Effector-Triggered Immunity). Sometimes ETI triggered by a given T3E is supressed by another, co-injected effector.

HopAF1 and HopZ3 are T3Es co-injected by P. syringae strain B728a. We have described that HopAF1 suppresses HopZ3-triggered ETI, and want to determine if this happens through direct interaction or rather an indirect molecular mechanism. Here we show by CoIP that HopAF1 interacts with HopZ3, suggesting both T3Es are closely associated within the plant cell. However our FRET-FLIM results suggest these T3Es do not interact directly.

HopAF1 and HopZ3 interact with plasma membrane-associated plant proteins that contribute to immune signalling. However, known HopAF1 interactors do not explain its ETI-suppressing ability. We propose that HopAF1 targets additional plant proteins.

HopAF1 has structural and sequence similarity with bacterial deamidases. T3Es from animal-infecting pathogens deamidate PM-associated heterotrimeric G proteins (with alpha, beta and gamma subunits), suppressing immunity. Arabidopsis expresses one G-alpha (GPA1), one G-beta (AGB1), three G-gamma (AGG-1/2/3) and three extra-large G-protein (XLG-1/2/3) subunits. Here we show by Co-IP that HopAF1 interacts with AtGPA1, AtGB1, AtAGG1, and AtXLG2. However our results from FRET-FLIM assays suggest these interactions are not direct.

We propose that HopAF1, HopZ3, and heterotrimeric G-proteins form part of the same membrane-associated macromolecular complex, where both T3Es interfere with plant immunity