# 32ND INTERNATIONAL CONFERENCE ON ARABIDOPSIS RESEARCH


# ABSTRACT BOOK

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# The bacterial effector HopAF1 interacts with *Arabidopsis* MKK4/5

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## ABSTRACT

*Pseudomonas syringae* is a phytopathogenic bacterium whose virulence depends on a Type III Secretion System and its effector (T3E) repertoire, which is translocated into the host cell cytosol suppressing both basal and effector-triggered Immunity (ETI). HopAF1 is a T3E that suppresses several plant immunity phenotypes, including flg22-induced ethylene (ET) production. HopAF1 is mainly located in the plasma membrane (PM), where it interacts with plant proteins MTN1/2 that participate in ET biosynthesis. However, PM localization is not essential for HopAF1 ability to repress ET accumulation.

While seeking for additional HopAF1 interactors in the plant, we have identified MAP kinase kinases MKK4/5. The MAP kinase module including MKK4/5 and MPK3/6 positively regulates 1-aminocyclopropane-1-carboxylic acid synthase (ACS) activity, a rate-limiting and major regulatory step in stress-induced ET production. MKK4/5-dependent phosphorylation directly stabilizes ACS2/6 and indirectly induces ACS transcription. We originally identified HopAF1-MKK4 interaction through an MS-based screening, and have now confirmed it by pull-down and BIFC, while obtaining variable results using FRET-FLIM. Moreover, we are monitoring ACS2/6 expression levels by qRT-PCR, in the presence and absence of HopAF1. We propose that HopAF1 interference with MKK4/5 is likely to contribute to the suppression of ET production, by altering MKK4/5-dependent regulation of ACS activity.