



## INTERNSHIP PROPOSAL M2 RESEARCH 2022-2023

## TITLE: STUDY OF A CHROMATIN REMODELING FACTOR AS A CANDIDATE SUBSTRATE OF MPK3 IN ARABIDOPSIS THALIANA

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Number of PhD currently supervised: 0

Possibilty to give rise to a PhD proposal :	■YES	
Direct presentation of the proposal to the students	<u>∷</u> ∎YES	
■01	/09/20 (IPS2)	□02/09/20 (IJPB)

**NEW :** we plan to communicate, for information, the internship proposals to M1 students so that they can get an idea of the research themes of the field. Would you agree to have your proposal distributed to them?

■YES □ NO

INTRODUCTION, SCIENTIFIC CONTEXT :

The «Stress signalling» team of IPS2 studies cellular signalling in response to environmental stresses, and more specifically mitogen-activated protein kinases (MAPKs). MAPK modules are composed of MAPKKKs, MAPKKs and MAPKs that are activated by phosphorylation cascades. In plants, MAPK modules are involved in development and in response to biotic and abiotic stresses. A detailed knowledge of these MAPKs could thus be transposed to cultivated species to increase their resistance/tolerance to environmental stresses.

RESEARCH PROPOSAL :

Arabidopsis thaliana MPK3 is a key actor of immune signalling. MPK3 is indeed involved in the two layers of immunity: PTI mediated by membrane receptors (PRRs) and ETI mediated by intracellular receptors (NLRs). While the global role of MPK3 in immunity is now well known, the underlying molecular mechanisms are still largely unknown. To better decipher these mechanisms, we developed a line expressing a constitutively active version of MPK3 (MPK3-





CA) (1). MPK3-CA line displays an auto-immune phenotype, with notably the accumulation of defense molecules, cell death and dwarfism.

We recently identified a chromatin remodeling factor whose loss-of-function mutant partially suppresses the MPK3-CA auto-immune phenotype (unpublished data). Interestingly, this chromatin remodeling factor is a probable substrate of MPK3 <sup>(2)</sup>.

The research proposal thus consists in 1) confirming that the candidate protein is indeed a direct substrate of MPK3, 2) phenotyping in details the suppressor line of *MPK3*-CA. This project will thus allow a better understanding of the signalling downstream MPK3.

METHODOLOGIES :

Part 1) Production of recombinant proteins and *in vitro* kinase-assays; validation of protein-protein interactions, notably by BiFC and yeast two-hybrid.

Part 2) RT-qPCR of defense marker genes, immunoblotting of defense marker proteins, pathogen assays, etc.

REFERENCES (maximum 5)

(1). Genot, B. et al. Constitutively active arabidopsis MAP kinase 3 triggers defense responses involving salicylic acid and SUMM2 resistance protein. *Plant Physiol.* (2017) doi:10.1104/pp.17.00378.

<sup>(2).</sup> Rayapuram, N. et al. Chromatin phosphoproteomics unravels a function for AT-hook motif nuclear localized protein AHL13 in PAMP-triggered immunity. *Proc. Natl. Acad. Sci. U. S. A.* (2021) doi:10.1073/pnas.2004670118.