Proposition d'un sujet de stage au M2 ADAM (2020) -

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Titre

Analysis of *Arabidopsis thaliana root cellular* response to *Ralstonia solanacearum* in a global warming context

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Résumé

In the current climate change scenario, heat stress is one of the major abiotic stresses that can affect plant growth, development and defense mechanisms induced following pathogen attack (1). Bacterial wild is one of the most devastating disease induced by the soil born bacteria *Ralstonia solancearum* in 250 plant species belonging to more than 50 botanical families, including economically important crop (2). We recently demonstrated the Arabidopsis immunoreceptor pair RPS4/RRS1-R -mediated resistance to *R. solanacearum,* activated upon perception of the PopP2 effector (3) to be inhibited at elevated temperature (4). Surprisingly, the cellular processes associated with sensitivity and resistance mechanics against pathogen at the root level are poorly characterized (Miller et al. 2010) and the impact of elevated temperature is not known. To identify resilient resistance mechanisms to *R. solanacearum* a Gene Wide Association (GWA) mapping was perfomed. Natural accession of *Arabidopsis thaliana* were used to identify Quantitative Trait Loci (QTLs) associated with natural variations of plant response to the *R. solanacearum* GMI1000 strain at two different temperatures, 27°C and 30°C.(3). One QTL identified at 30°C underlies a gene (*At5g05170*) encoding the a cellulose synthase subunit 3 (CESA3) which together with CESA1, -2, -5, and -6 compose the CESA complex synthetizing cellulose microfibrils for the primary cell wall formation. *CESA3* has been validated as a susceptibility gene involved in a strong Quantitative Disease Resistance mechanisms to *R. solanacearum* remaining efficient at 30°C through the study of two allelic knock-down mutants in the Col0 background. However, these mutants also harbor a strong pleiotropic developmental phenotype. Therefore, to further characterize the role of CesA3 in thermostable resistance, a sequencing strategy was developed to identify the molecular mechanisms involving a specific CESA3 haplotype in wild accessions without any phenotype.

In this context, the objective of the internship will be to study molecular mechanisms involving specific haplotype of *CESA3* locus correlated to thermostable resistance. Identification of haplotypes will be realized in collaboration with the bioinformatics team at LIPM. The nature of the polymorphism identified will determine the strategy used to specify the mechanism involving CESA3. This material, together with other mutants available in the team (e.g. *rsp4, rrs1, rsp4/rrs1*) in Ws-2 genetic background resistant at 27°C that becomes susceptible at 30°C will be used to characterize the root cell mechanisms associated with the plant defense response under standard and elevated temperature conditions. Preliminary results should open up a thesis project aiming to study the localized immune responses in *Arabidopsis thaliana* roots to *R. solanacearum* and the effects of elevated temperature

Methods: Bioinformatics, DNA-RNA extractions, Rt-qPCR, cloning, plant transformation, Molecular biology, microbiology, cellular biology, fluorescent markers pathogen infection assay, confocal microscopy,

References

1)Desaint et al., 2020 *New Phytologist* (in press). **2)Mansfield** et al., 2015 *Mol. Plant Pathol.* 13: 614–629 **3)Le Roux** et al., 2015 *Cell* 21;16 1074-1088. **4)Aoun** et al**.**, 2017 *Front Plant Sci* 22;8:1387.

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