Probing the Link Between EDS1-PAD4 and Transcriptional Reprogramming in *Arabidopsis thaliana* Immunity

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Abstract

Plants deploy cell-surface and intracellular receptors to trigger innate immune responses against pathogens. Receptor proteins at the plasma membrane recognize pathogen-derived molecules to elicit pattern-triggered immunity (PTI), whereas intracellular receptors detect microbial effectors inside cells to confer effector-triggered immunity (ETI). PTI and ETI rely on the transcriptional activation of similar sets of genes and transcriptional reprogramming is a hallmark of immune activation, which can ultimately lead to pathogen restriction and/or host cell death. In *Arabidopsis*, a core signaling hub for immune-triggered transcriptional reprogramming consists of the Enhanced Disease Susceptibility 1 (EDS1) - Phytoalexin-Deficient 4 (PAD4) heterodimer, onto which surface- and intracellular- receptor immune signaling converges. Despite recent advances in understanding the molecular mode of action of EDS1-PAD4, it remains unknown whether EDS1-PAD4 directly controls immune transcriptional reprogramming and how the latter connects to pathogen restriction.

From previously published proteomic datasets, I identified several transcriptional factors (TFs) that interact with PAD4 upon ETI activation. Among these TFs, Calmodulin-binding Transcription Activator 3 (CAMTA3) was of particular interest because it is involved in regulating (by activating or repressing) the expression of genes related to plant defense. In this study, the connection between EDS1-PAD4 and CAMTA3 was further investigated. I confirmed in *Arabidopsis thaliana* that the interaction between PAD4 and CAMTA3 is enhanced upon ETI activation and the association requires the presence of *Arabidopsis* EDS1. Furthermore, I observed an EDS1-PAD4 dependent re-localization or stabilization of CAMTA3 in the nucleus following an ETI trigger. Chromatin Immunoprecipitation-sequencing (ChIP-seq) analyses performed in CAMTA3 transgenic *Arabidopsis* lines indicated CAMTA3 binding to the promoter regions of target immune-related genes, and that EDS1-PAD4 might inhibit this process to relieve CAMTA3 suppression. Indeed, RNA-seq analysis of the same tissues revealed down-regulation of CAMTA3-targeted immune-related genes upon ETI activation in the absence of EDS1 or PAD4. This study fills a knowledge gap in understanding the role of EDS1-PAD4-CAMTA3 in plant ETI. I propose that EDS1-PAD4 inhibits transcriptional repression of CAMTA3 at promoters of immune-related genes by interacting with CAMT