ALD1 Regulates Basal Immune Components and Early Inducible Defense Responses in *Arabidopsis*

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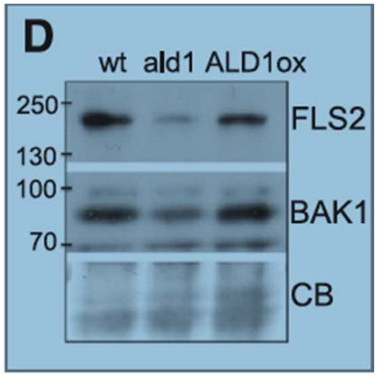
Funding Source: NSF grant IOS 0957963 and DOE Office of Biological and Environmental Research, Genomic Science Program

Background

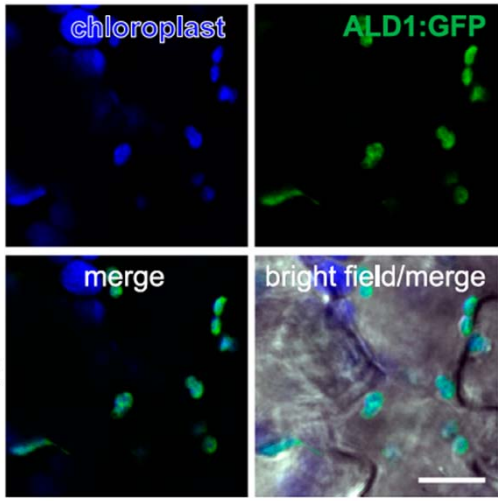
- Plant innate immunity has several mechanisms that enable the recognition of microorganims through different types of immune receptors
- We address the role of ALD1 in mediating defense amplification as well as the levels and responses of basal defense machinery in plant immunity.

Basal FLS2 and BAK1 levels in total extracts of transgenic plants.



Science

- Transgenic *Arabidopsis* plants were produced that overexpressed ALD1 and resistance levels to virulent *P. syringae* increased.
- During basal state conditions, very rapid defense responses to PAMP flg22 (reactive oxygen species (ROS) burst) was observed in these plants when compared to WT, therefore, ALD1 controls microbial-associated molecular pattern (MAMP) receptor levels and responsiveness.
- Pipecolic acid (Pip), an ALD1-dependent product, which allows for pathogen-induced salicyclic acid (SA) accumulation, did not confer increased flg22 responses to WT or over-expressing ALD1 plants, however exudates from these plants could confer disease resistance.



ALD1 localization in chloroplasts

Significance

•Salicyclic acid (SA), a central player for defense induction and important for resistanc to virulent *P. syringae*, is regulated by ALD1.

•ALD1 localizes to the chloroplast, which has a great impact in defense metabolic pathways.

•ALD1 affects earlier defense events than were previously described, therefore, the metabolites produced by ALD1 affect basal and early defenses, which may be more beneficial to the plant's innate immunity.

Cecchini NM et al. 2015. ALD1 Regulates Basall Immune Components and Early Inducible Defense Responses in Arabidopsis. MPMI (2015) 28(40: 455-466, doi: 10.1094/MPMI-06-14-0187-R

Molecular Plant-Microbe Interactions 2015, 28(4): 455-466

ALD1 Regulates Basal Immune Components and Early Inducible Defense Responses in Arabidopsis

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Abstract

Robust immunity requires basal defense machinery to mediate timely responses and feedback cycles to amplify defenses against potentially spreading infections. AGD2-LIKE DEFENSE RESPONSE PROTEIN 1 (ALD1) is needed for the accumulation of the plant defense signal salicylic acid (SA) during the first hours after infection with the pathogen *Pseudomonas syringae* and is also upregulated by infection and SA. ALD1 is an aminotransferase with multiple substrates and products in vitro. Pipecolic acid (Pip) is an ALD1-dependent bioactive product induced by *P. syringae*. Here, we addressed roles of ALD1 in mediating defense amplification as well as the levels and responses of basal defense machinery. ALD1 needs immune components PAD4 and ICS1 (an SA synthesis enzyme) to confer disease resistance, possibly through a transcriptional amplification loop between them. Furthermore, ALD1 affects basal defense by controlling microbial-associated molecular pattern (MAMP) receptor levels and responsiveness. Vascular exudates from uninfected ALD1-overexpressing plants confer local immunity to the wild type and ald1 mutants yet are not enriched for Pip. We infer that, in addition to affecting Pip accumulation, ALD1 produces non-Pip metabolites that play roles in immunity. Thus, distinct metabolite signals controlled by the same enzyme affect basal and early defenses versus later defense responses, respectively.