Pipecolic acid – a central regulator of plant systemic acquired resistance and defense priming

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Systemic acquired resistance (SAR) is an inducible plant immune response that is initiated by a localized inoculation of leaves with virulent or avirulent phytopathogens. Plants with activated SAR exhibit enhanced resistance in the entire foliage towards infections by many biotrophic and hemibiotrophic pathogens, and are primed to more effectively activate defense responses to future microbial attack. SAR induced in *Arabidopsis thaliana* by the bacterial pathogen *Pseudomonas syringae* is accompanied with a strong transcriptional response systemically in the foliage that includes up-regulation of genes involved in multiple stages of defense signaling, enhanced expression of pathogenesis-related (PR) genes, and downregulation of photosynthetic and growth- related genes. In the course of SAR activation, the lysine-derived non-protein amino acid pipecolic acid (Pip) strongly accumulates in both *P. syringae*-inoculated and in non-inoculated distal leaves. Pip is generated in dependence of AGD2-LIKE DEFENSE RESPONSE PROTEIN1 (ALD1), an aminotransferase gene which is systemically up-regulated in the plant upon *P. syringae* inoculation. Pip has a widespread occurrence in Angiosperms and is generated to high levels after bacterial, fungal, or viral infection in many different plant species, including rice, potato, tobacco, soybean, and Arabidopsis. Notably, the systemic accumulation of Pip in the foliage is necessary for SAR establishment and the associated systemic elevations of the phenolic defense hormone salicylic acid (SA) (Návarová et al., 2012). Pip exerts its SAR-inducing capacity in dependence of FLAVIN-DEPENDENT-MONOOXYGENASE1 (FMO1). A second critical SAR-regulatory metabolite is SA, which is synthesized in plastids by ISOCHORISMATE SYNTHASE1 (ICS1) from chorismate. Recent findings from our laboratory indicate that Pip regulates SAR via a major, SA-dependent and a minor, SA- independent activation pathway (Bernsdorff et al., 2016). This is illustrated by the full absence of the normally observed transcriptional SAR response in *ald1* and *fmo1* knockout plants and a strongly diminished but not fully abrogated response in *ics1* plants after *P. syingae* inoculation. The two central SAR regulatory metabolites Pip and SA act synergistically in the induction of *PR* gene expression but also trigger separate signaling pathways that can function independently from each other. Moreover, Pip orchestrates SA-dependent and SA-independent priming of pathogen responses in a FMO1-dependent manner. Therefore, a Pip/FMO1-signaling module acts as an indispensable switch for the activation of SAR and associated defense priming events, and SA amplifies Pip- triggered responses to different degrees in the distal tissue of SAR-activated plants. The talk will highlight the interplay between the two critical signals Pip and SA in SAR and defense priming, involve novel aspects of pipecolic acid metabolism, and introduce a novel regulatory component downstream of Pip and SA in SAR activation.

Literature:

Návarová H, Bernsdorff F, Döring A-C, Zeier J (2012). Plant Cell 24: 5123-5141.
Bernsdorff F, Döring A-C, Gruner K, Schuck S, Bräutigam A, Zeier J (2016). Plant Cell 28: 102-129.