Title Ethylene signaling renders the jasmonate response of *Arabidopsis* insensitive to future suppression by salicylic acid

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Abstract

Cross-talk between jasmonate (JA), ethylene (ET), and Salicylic acid (SA) signaling is thought to operate as a mechanism to fine-tune induced defenses that are activated in response to multiple attackers. Here, 43 *Arabidopsis* genotypes impaired in hormone signaling or defense-related processes were screened for their ability to express SA-mediated suppression of JA-responsive gene expression. Mutant *cev1*, which displays constitutive expression of JA and ET responses, appeared to be insensitive to SA-mediated suppression of the JA-responsive marker genes *PDF1.2* and *VSP2*. Accordingly, strong activation of JA and ET responses by the necrotrophic pathogens *Botrytis cinerea* and *Alternaria brassicicola* prior to SA treatment counteracted the ability of SA to suppress the JA response. Pharmacological assays, mutant analysis, and studies with the ET-signaling inhibitor 1-methylcyclopropene revealed that ET signaling renders the JA response insensitive to subsequent suppression by SA. The APETALA2/ETHYLENE RESPONSE FACTOR transcription factor ORA59, which regulates JA/ET-responsive genes such as *PDF1.2*, emerged as a potential mediator in this process. Collectively, our results point to a model in which simultaneous induction of the JA and ET pathway renders the plant insensitive to future SA-mediated suppression of JA-dependent defenses, which may prioritize the JA/ET pathway over the SA pathway during multi-attacker interactions.