Jasmonoyl---isoleucine catabolic pathways provide new insights into jasmonate homeostasis

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Jasmonates (JAs) are well---known regulators of plant defense responses to external cues and mediate also developmental processes like fertility. Since the demonstration that the conjugate jasmonoyl---isoleucine (JA---Ile) rather than jasmonic acid (JA) is the conserved hormonal signal, elucidation of JA---Ile turnover mechanisms has attracted interest.

We and others have characterized biochemically and genetically two JA---Ile catabolic pathways that are stress---inducible in leaves and developmentally regulated in flowers. The first pathway consists in Arabidopsis in a group of 3 co---regulated cytochromes P450 of the CYP94 subclade that define a two---step JA---Ile ω---oxidation process. CYP94B3 is the main enzyme catalyzing JA---Ile turnover/inactivation through hydroxylation upon mechanical leaf wounding. In contrast, upon fungal infection or in maturing stamens, the JA signatures in tissues reflect the predominant expression of CYP94C1. CYP94C1 exhibits peculiar properties as performs further oxidation to 12COOH---JA---Ile, a totally inactive derivative. All 3 enzymes oxidize JA---Ile and some less abundant JA---amino acid conjugates, and CYP94C1 additionally generates aldehyde intermediates. Unexpectedly, single or multiple mutations in CYP*94* genes, while increasing the amount and half---life of JA---Ile at the expense of hydroxy--- and carboxy---derivatives, has negligible consequences on defense responses amplitude or antifungal resistance. This suggests that mechanisms may exist, including JAZ repressor hyperinduction, that desensitize signaling under high JA---Ile levels. In contrast, ectopic overexpression of CYP94B3 or CYP94C1 reduces JA---Ile levels and shuts down induced defenses and associated resistance.

The second pathway is defined by IAR3 and ILL6 amido---hydrolases that additionally contribute to hormone homeostasis by cleaving JA---Ile and 12OH---JA---Ile conjugates, providing an indirect route for the formation 12OH---JA and its derivatives. Catabolic pathways redefine a complex metabolic grid, where JA---Ile positions as a hub initiating the formation of many derivatives through oxidation and deconjugation. These metabolic circuits may reveal additional conversion routes with potential regulatory functions.