Double Repression in Jasmonate-Mediated Plant Defense

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The functions of jasmonic acid (JA) in plant defense and development are controlled by the JAZ family of repressor proteins. In this issue of Molecular Cell, Hu et al. (2013) identified JAV1 as a repressor protein in the JA pathway that enables the plant to defend against necrotrophic pathogens and herbivorous insects apparently without influencing plant growth and development.

Methyl jasmonate (MeJA) is a volatile compound that was originally isolated from jasmine oil of the plant Jasminum grandiflorum. JA and its derivatives are cyclic fatty acid-derived regulators structurally similar to the animal defense regulator prostaglandins. They function as phytohormones important for plant defense against herbivorous insects and necrotrophic pathogens. They also regulate plant growth and development, including fertility of reproductive organs, root growth, anthocyanin accumulation, and trichome initiation (Wasternack and Hause, 2013). JA synthesis is induced in response to wounding and developmental cues. In the elicited cells, JA-Ile (the active, isoleucine conjugated form) is recognized by COI1, an F-box protein in SCF ubiquitin E3 ligase complex, and mediates the interaction between COI1 and the JAZ family of repressor proteins, leading to the ubiquitination of JAZs. Ubiquitinated JAZs are then degraded by the 26S proteasome (Figure 1). Destruction of JAZs releases target transcription factors from repression and activates downstream JA responses. The degradation of transcriptional repressors to rapidly activate transcriptional programs is a widely used mechanism for hormone sensing and signaling in plants (Pieterse et al., 2012). Similar to JA, other phytohormones such as gibberellic acid (GA) and indole-3-acetic acid (IAA) are also sensed by the ubiquitination machinery to trigger the degradation of transcriptional repressors (Robert-Seilaniantz et al., 2011).

In JA responses, the JAZs are master regulators, and their destruction leads to the activation of various transcriptional programs controlled by Myc and Myb transcription factors, enabling the plant to defend against defense-related pathways and to alter its growth and development (Browse, 2009; Pauwels and Goossens, 2011; Wasternack and Hause, 2013). In this issue, Hu et al. (2013) identified a new repressor protein, JAV1, in JA responses. It appears that JAV1 is another master controller in JA-mediated defense responses and enables the plant to defend against necrotrophic pathogens and herbivorous insects apparently without sacrificing plant growth and development. This interesting finding may lead to further deciphering of the JA pathway and to the improvement of crop resistance to pests and pathogens.

The isolation of insect-resistance genes in plants has been hindered by the lack of effective screening systems (Wu and Baldwin, 2010). In this study, an elegantly designed screen of a JA-inducible RNAi library for altered resistance to necrotrophic pathogens in Arabidopsis led to the identification of a plant-specific protein, Jasmonate-associated VQ motif protein, namely JAV1. No obvious growth and developmental defects were observed in JAV1-RNAi plants. JAV1-RNAi plants exhibit enhanced resistance to not only necrotrophic fungal pathogens but also to chewing and phloem-feeding insects. Insects that fed on JAV1-RNAi plants performed poorly. Downregulation of JAV1 may result in accumulation of certain metabolites that inhibit insect growth or reduce the attractiveness of JAV1-RNAi plants to insects.

At the molecular level, defense-related genes are induced more strongly by JA and wounding treatments in JAV1-RNAi plants. The induced genes (PDF1.2, THIONIN2.1, PR4, PR5, and VSP1) fall into two different branches of the JA pathway but are not part of the salicylic acid (SA) pathway, which also functions in pathogen defense (Pieterse et al., 2012). Most SA-responsive genes remain unchanged in JAV1-RNAi plants. These results indicate a specificity of JAV1 function in JA-mediated defense. Mechanical wounding, however, does not fully mimic insect attack, especially attacks by phloem-feeding aphids. Moreover, defense against chewing insects and phloem-feeding insects may use different mechanisms. An herbivore-associated molecular pattern (HAMP), similar to a microbe (or pathogen)-associated molecular pattern (MAMP or PAMP), may elicit defense responses (Wu and Baldwin, 2010). Therefore, comparison of the transcript profiles between pathogen-attacked or insect-attacked JAV1 RNAi plants and wild-type plants would provide additional useful data concerning JAV1 function in defense.

JAV1 is similar to JAZ in several ways. Both are upregulated at the transcript level by JA treatments. Both have repressive roles in JA-mediated defense responses and are degraded via the 26S proteasome in a COI1-dependent manner. However, unlike JAZs, JAV1 controls defense responses but has no apparent roles in plant growth and development. In addition, JA facilitates a direct interaction between JAZs and COI1, but
Unlike JAZs, JAV1 lacks the JAS and ZIM domains that are required for JA-mediated direct interaction between JAZs and COI1 or between JAZs themselves (Wager and Browse, 2012). Therefore, it is not surprising that JAV1 does not directly interact with either JAZs or COI1, although it is unclear whether JAV1 proteins may interact with each other to form dimer or multimer and whether multimerization of JAV1 is required for JAV1 function. JA-mediated degradation of JAV1, however, is still COI1-dependent, suggesting that an important gap remains to be bridged. As speculated in Hu et al., JA may trigger COI1 to activate an as-yet-unidentified E3 ligase to interact with and mediate the ubiquitination and degradation of JAV1 (Figure 1). It is likely that interacting partners of JAV1 could constitute a novel branch of the JA pathway to connect the perception of JA to the activation of JA-mediated responses specifically repressed by JAV1.

Transcriptional derepression systems allow plants to rapidly mount a defense against attackers while in peace time to use all available resources for normal growth and development. An intriguing question is how JAV1 fine tunes trade-offs between growth and defense. According to current knowledge, trade-offs between growth and defense may be subject to regulation on at least three levels. First, the pathogen elicitor or phytohormone may share the same coreceptor. For example, the effects of brassinosteroid (BR) hormone on growth and defense are fine tuned by the availability of the coreceptor BAK1 (Wang, 2012). Second, crosstalk between JA and GA regulates hormone level and generates synergistic or antagonistic interactions between repressors, or between repressors and their targets. For example, in Arabidopsis and rice, JA prioritizes defense over growth by reducing DELLA protein levels and by interfering with the interaction between DELLA proteins with the growth-related transcription factors (Yang et al., 2012). Third, repressors target multiple downstream transcription factors that differentially control growth- or defense-related gene-expression programs. For example, JAZs target transcription factors for JA-mediated regulation of growth, development, and defense (Pauwels and Goossens, 2011). Initial screening for JAV1 interactors led to the identification of two transcription factors, WRKY28 and WRKY51. Understanding of the function of WRKY28 and WRKY 51 in the JAV1 branch of the JA pathway could provide insight into the mechanism used by plants to control defense in a timely and cost-effective manner. In addition, dissecting the crosstalk between JA and IAA and between JA and GA signaling would help clarify the apparently defense-specific function of JAV1.

REFERENCES


