Commentary

Different shades of JAZ during plant growth and defense

Ever since their discovery as key regulators of the jasmonate (JA) signaling pathway (Chini et al., 2007; Thines et al., 2007; Yan et al., 2007), repressor proteins of the JASMONATE ZIM-domain (JAZ) family have been rising stars in research on hormonal regulation of plant growth and defense. In plant cells, JAZ repressor proteins interact with an E3 ubiquitin ligase complex (SCF^COI^) that together function as a JA receptor. In resting cells, JAZs block the activity of transcriptional regulators of JA responses by physically binding to them. Upon perception of bioactive JAs, JAZ proteins are rapidly degraded via the ubiquitin/26S proteasome-dependent proteolytic pathway. This releases the JAZ-bound transcription factors, resulting in the activation of downstream JA responses (Fig. 1a). JAs play a dominant role in regulating defense responses against herbivorous insects and necrotrophic pathogens, and in adaptive responses to beneficial soilborne microbes (Wasternack & Hause, 2013; Pieterse et al., 2014). In addition, JAs have a signal function in a myriad other processes, including abiotic stress reactions and plant growth responses to environmental cues (Wasternack & Hause, 2013).

The JA pathway functions in the context of a complex network of hormone-regulated signaling pathways that, depending on the environmental or developmental condition, can act antagonistically or synergistically on each other to finely balance resource allocation between growth and defense and minimize fitness tradeoffs (Pieterse et al., 2012; Vos et al., 2013). In the process of balancing plant growth and defense, gibberellins (GAs) have emerged as dominant antagonists of the JA signaling output (Hou et al., 2013). GAs regulate different aspects of plant growth via DELLA repressor proteins that block the activity of transcriptional regulators of GA responses by physically binding to them. Analogous to the role of JAZs in the JA pathway, DELLAs are degraded upon perception of GAs, resulting in the activation of downstream growth responses (Fig. 1a). Interestingly, DELLAs also interact with JAZs, thereby mutually limiting the cellular binding capacity to their cognate transcription factors (Hou et al., 2010; Yang et al., 2012). Consequently, GA-mediated degradation of DELLAs enhances the cellular binding capacity of JAZs to their cognate transcription factors, thus reducing the potential JA signaling output (Hou et al., 2013). This GA-mediated antagonistic effect on the JA pathway becomes apparent during the shade-avoidance response of plants that grow in dense vegetation stands. Shade-intolerant plant species respond to competition for light by increasing apical dominance and accelerating stem and petiole elongation. These growth and developmental responses occur in response to a drop in the red : far-red (R : FR) light ratio that is sensed by the phytochrome photoreceptors, predominantly phyB, and are GA-dependent through GA-mediated degradation of DELLA proteins (Djakovic-Petrovic et al., 2007). This allows them to outgrow neighboring plants, but at the cost of a reduced defensive capacity against necrotrophic pathogens and insect herbivores (Moreno et al., 2009; Cerrudo et al., 2012; De Wit et al., 2013) (Fig. 1b).

In this issue of New Phytologist, Leone et al. (pp. 355–367) zoomed in on the specific role of the Arabidopsis thaliana (Arabidopsis) JAZ10 protein in this process and shed light on the involvement of DELLAs in GA–JA crosstalk during the shade-avoidance response. Besides growth-related hormones, effector proteins of pathogens and plant growth-promoting mycorrhizal fungi have also recently been shown to target JAZ repressor proteins, thereby changing the defense-related signaling circuitry for their own benefit. Hence, JAZ repressor proteins are emerging as central targets in the rewiring of the hormone-regulated signaling circuitry that regulates growth and defense.

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GA vs JA: to grow or to defend

In Arabidopsis, a total of 12 JAZs (JAZ1–JAZ12) and five DELLAs (RGA, GAI, RGL1, RGL2 and RGL3) have been identified with overlapping and distinct roles in JA- and GA-mediated plant responses, respectively (Chini et al., 2007; Browse, 2009; Hou et al., 2013). Both the JAZs and the DELLAs specifically interact with basic helix–loop–helix (bHLH)-type transcription factors, thereby preventing them from regulating their downstream target genes under conditions without a stimulus. JAZs interact with MYC2, MYC3 and MYC4, which are essential in the activation of JA-dependent defenses (Chini et al., 2007; Thines et al., 2007; Fernandez-Calvo et al., 2011). DELLAs interact with Phytochrome Interacting Factors PIF3 and PIF4, which regulate light-controlled genes involved in hypocotyl elongation (Feng et al., 2008; de Lucas et al., 2008). DELLAs also interact with JAZs, thereby competing with their cognate MYC transcription factors and modulating the JA signal output under JA-inducing conditions (Hou et al., 2010). In addition, the DELLA protein RGL3 was shown to act as a positive transcriptional regulator of MYC2 and, as such, is required for full activation of JA-induced responses.
(Wild et al., 2012), adding another layer of regulation of crosstalk between the GA and JA signaling pathways.

By using a quintuple *della* mutant in which all five *DELLA* genes are impaired and by monitoring *DELLA* protein turnover, Leone et al. demonstrate that under conditions of competition for light, GA-mediated degradation of *DELLA*s plays a central role in redirecting resource allocation from defense to rapid elongation. Their results support the notion that shade-triggered degradation of *DELLA*s increases the cellular capacity of JAZs to bind and thereby inactivate MYC-type transcription factors that are required for JA-induced responses (Hou et al., 2010, 2013). Moreover, they provide evidence that the shade-triggered negative effect on JA signaling is specifically targeted at JAZ10. Initiation of the shade avoidance response by plant exposure to low R : FR light conditions significantly increased the stability of JAZ10, resulting in the suppression of JA-induced responses. Importantly, this dominant negative effect of JAZ10 on the JA response was abolished in jaz10 mutant and RNAi lines, suggesting that the DELLA-mediated antagonistic effect on the JA signaling output is specifically targeted at JAZ10. However, recent findings by Chico et al. (2014) indicate that low R : FR light ratios also stabilize several other JAZs under JA-inducing conditions. Moreover, the latter study showed that the transcription factors MYC2, MYC3, and MYC4 have a higher turnover under low R : FR conditions, which further contributes to the suppression of JA-inducible responses (Chico et al., 2014). Hence, under conditions of competition for light, Arabidopsis reconfigures its resource allocation strategy to favor rapid elongation over defense by degrading *DELLA*s, which initiates GA-mediated elongation growth and cures *DELLA*s from *DELLA*-JAZ complexes, resulting in an enhanced cellular capacity of JAZs to bind and inactivate MYC transcription factors; stimulating the stability of JAZs, in particular JAZ10; and reducing
the stability of MYC2, MYC3, and MYC4, which are required for the activation of JA-dependent defenses.

Use or abuse: hijacking JAZs to rewire the defense signaling network

In analogy to the shade-mediated repressive effect on JA-dependent defenses, certain pathogens have evolved ways to interfere with the JA signaling output through activation of the GA/DELLA signaling pathway. For instance, several necrotrophic fungi have been shown to produce GAs themselves, possibly as a virulence factor to stimulate DELLA degradation and consequently suppress effective JA-dependent defenses that would impair growth of the pathogen in the host (Grant & Jones, 2009). Conversely, general microbe-associated molecular patterns (MAMPs), such as flagellin, have been shown to stabilize DELLA proteins, resulting in enhanced JA-dependent defenses and elevated resistance against necrotrophic pathogens (Navarro et al., 2008). Another dominant antagonist of the JA signaling pathway is the defense hormone salicylic acid (SA) (Pieterse et al., 2012). In general, the SA signaling pathway regulates defense responses that are effective against pathogens with a biotrophic lifestyle. The SA and JA pathways act antagonistically on each other and provide the plant with a mechanism to fine-tune its defense response depending on the lifestyle of the enemy. Although the antagonistic effect of SA on JA signaling functions independently of JAZ protein stability (Van der Does et al., 2013), GA/DELLA/JAZ-mediated modulation of JA signaling does affect SA-dependent defenses. It has been postulated that the flagellin-mediated stabilization of DELLA proteins and the resulting positive effect on JA signaling is a way for biotrophic pathogens to suppress effective SA-dependent defenses that would otherwise limit growth of the pathogen (Navarro et al., 2008; Pieterse et al., 2012). This crosstalk mechanism is, however, not involved in low R : FR-mediated repression of the JA response, as both JA- and SA-mediated defense responses are suppressed under conditions of low R : FR (De Wit et al., 2013).

In addition to low R : FR ratios, microbially produced GAs, and MAMPs such as flagellin, effector proteins produced by successful pathogens have also recently been shown to affect the stability of specific JAZ proteins. As a result, the JA pathway is rewired to the benefit of the microbe. For instance, the bacterial pathogen Pseudomonas syringae produces effector proteins such as HopX1 and HopZ1a that specifically interact with, and promote, the degradation of JAZ proteins. Consequently, JA responses are activated while SA-dependent defenses are antagonized, resulting in enhanced susceptibility of the host (Jiang et al., 2013; Gimenez-Ibabeit et al., 2014). In addition to pathogens, the symbiotic ectomycorrhizal fungus Laccaria bicolor also produces an effector (MiSSP7), which is necessary for the establishment of symbiosis and acts by binding to the PtJAZ6 protein in its host poplar (Plett et al., 2014). Binding of MiSSP7 to PtJAZ6 stabilizes the JAZ protein to suppress JA-dependent defenses that would otherwise attenuate the symbiosis.

Exciting developments in research on the role of JAZs in the regulation of plant growth and defense highlight that plants are capable of displaying different shades of JAZ action. Regulation of

the stability of this central regulator emerged as a common theme in plant responses to their environment. Induced changes in the cellular capacity of JAZs to bind and inactivate MYC transcription factors modulate the JA signaling output, to prioritize elongation growth over defense, or to finely tune the hormone-regulated immune signaling network to facilitate defense, symbiosis, or pathogenesis.

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