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^{COI1}) 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.

'JAZ repressor proteins are emerging as central targets in the rewiring of the hormone-regulated signaling circuitry that regulates growth and defense.'

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

New Phytologist

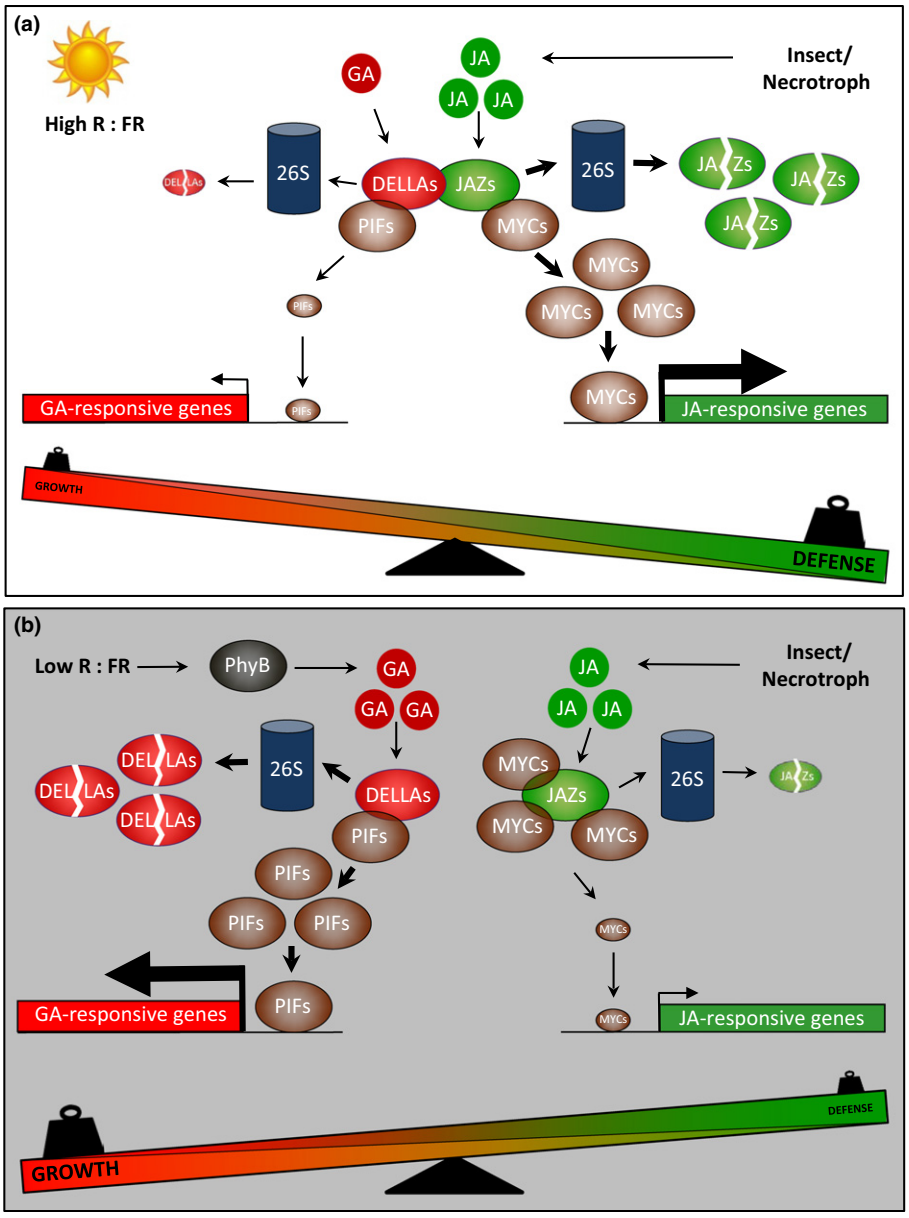

Fig. 1 Simplified model of how interactions between JASMONATE ZIM-domain proteins (JAZs) and DELLA proteins mediate crosstalk between shade-triggered elongation growth and jasmonate (JA)-dependent defenses. (a) Under conditions of high red : far-red (R : FR) light ratios (no shade), gibberellin (GA) concentrations are relatively low, allowing DELLAs to interact with Phytochrome Interacting Factors (PIFs) and JAZs. As a result, the cellular capacity of JAZs to inhibit the activity of MYCs and other transcription factors is relatively low. Induction of JA biosynthesis, for example, upon insect herbivory or infection by a necrotrophic pathogen, results in degradation of JAZs via the 26S proteasome, leading to a relatively high level of JA-dependent defenses against insect herbivores and necrotrophic pathogens. (b) During growth of plants in dense vegetation stands, the resulting low R : FR ratios of the light spectrum are sensed by the phytochrome photoreceptors (predominantly phyB), which initiate GA biosynthesis and signaling. Consequently, DELLAs are degraded by the 26S proteasome. On the one hand, this leads to the release of PIF transcription factors that activate GA-responsive growth-promoting genes that are associated with rapid elongation growth towards the light, which helps the plant to outcompete its neighboring vegetation; and on the other hand, JAZ–DELLA protein complexes become cured from DELLAs, resulting in enhanced binding of MYCs and other transcription factors to JAZs. In addition, JAZs become stabilized while MYCs are more rapidly turned over. Consequently, JA-induced defenses are suppressed, resulting in enhanced susceptibility to insect herbivores and necrotrophic pathogens.

(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 DELLAs plays a central role in redirecting resource allocation from defense to rapid elongation. Their results support the notion that shade-triggered degradation of DELLAs 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 DELLAs, which initiates GA-mediated elongation growth and cures DELLAs 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 DELLAs, 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 DELLAs 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-Ibanez *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.

Acknowledgements

The authors of this review are supported by ERC Advanced Grant no. 269072 of the European Research Council (C.M.J.P.) and VIDI grants nos 11281 (S.C.M.V.W.) and 86412003 (R.P.) of the Netherlands Organization of Scientific Research.

Corné M. J. Pieterse[1]*, Ronald Pierik[2] and Saskia C. M. Van Wees[1]

[1]Plant-Microbe Interactions, Department of Biology, Faculty of Science, Utrecht University, PO Box 800.56, 3508 TB, Utrecht, the Netherlands;
[2]Plant Ecophysiology, Department of Biology, Faculty of Science, Utrecht University, PO Box 800.56, 3508 TB, Utrecht, the Netherlands
(*Author for correspondence: tel +31 30 253 6887;
email: c.m.j.pieterse@uu.nl)

References

Browse J. 2009. Jasmonate passes muster: a receptor and targets for the defense hormone. *Annual Review of Plant Biology* **60**: 183–205.

Cerrudo I, Keller MM, Cargnel MD, Demkura PV, De Wit M, Patitucci MS, Pierik R, Pieterse CMJ, Ballaré CL. 2012. Low red/far-red ratios reduce Arabidopsis resistance to *Botrytis cinerea* and jasmonate responses via a COI1-JAZ10-dependent, salicylic acid-independent mechanism. *Plant Physiology* **158**: 2042–2052.

Chico J-M, Fernández-Barbero G, Chini A, Fernández-Calvo P, Díez-Díaz M, Solano R. 2014. Repression of jasmonate-dependent defenses by shade involves differential regulation of protein stability of MYC transcription factors and their JAZ repressors in Arabidopsis. *Plant Cell* **26**: 1967–1980.

Chini A, Fonseca S, Fernandez G, Adie B, Chico JM, Lorenzo O, Garcia-Casado G, Lopez-Vidriero I, Lozano FM, Ponce MR *et al.* 2007. The JAZ family of repressors is the missing link in jasmonate signalling. *Nature* **448**: 666–671.

De Wit M, Spoel SH, Sanchez-Perez GF, Gommers CMM, Pieterse CMJ, Voesenek LCJ, Pierik R. 2013. Perception of low red:far-red ratio compromises both salicylic acid- and jasmonic acid-dependent pathogen defences in Arabidopsis. *Plant Journal* **75**: 90–103.

Djakovic-Petrovic T, De Wit M, Voesenek LACJ, Pierik R. 2007. DELLA protein function in growth responses to canopy signals. *Plant Journal* **51**: 117–126.

Feng SH, Martinez C, Gusmaroli G, Wang Y, Zhou JL, Wang F, Chen LY, Yu L, Iglesias-Pedraz JM, Kircher S *et al.* 2008. Coordinated regulation of *Arabidopsis thaliana* development by light and gibberellins. *Nature* **451**: 475–479.

Fernandez-Calvo P, Chini A, Fernandez-Barbero G, Chico JM, Gimenez-Ibanez S, Geerinck J, Eeckhout D, Schweizer F, Godoy M, Franco-Zorrilla JM *et al.* 2011. The Arabidopsis bHLH transcription factors MYC3 and MYC4 are targets of JAZ repressors and act additively with MYC2 in the activation of jasmonate responses. *Plant Cell* **23**: 701–715.

Gimenez-Ibanez S, Boter M, Fernandez-Barbero G, Chini A, Rathjen JP, Solano R. 2014. The bacterial effector HopX1 targets JAZ transcriptional repressors to activate jasmonate signaling and promote infection in Arabidopsis. *PLoS Biology* **12**: e1001792.

Grant MR, Jones JDG. 2009. Hormone (dis)harmony moulds plant health and disease. *Science* **324**: 750–752.

Hou X, Ding L, Yu H. 2013. Crosstalk between GA and JA signaling mediates plant growth and defense. *Plant Cell Reports* **32**: 1067–1074.

Hou X, Lee LYC, Xia K, Yen Y, Yu H. 2010. DELLAs modulate jasmonate signaling via competitive binding to JAZs. *Developmental Cell* **19**: 884–894.

Jiang S, Yao J, Ma K-W, Zhou H, Song J, He SY, Ma W. 2013. Bacterial effector activates jasmonate signaling by directly targeting JAZ transcriptional repressors. *PLoS Pathogens* **9**: e1003715.

Leone M, Keller MM, Cerrudo I, Ballaré CL. 2014. To grow or defend? Low red: far-red ratios reduce jasmonate sensitivity in Arabidopsis seedlings by promoting DELLA degradation and increasing JAZ10 stability. *New Phytologist* **204**: 355–367.

de Lucas M, Daviere JM, Rodriguez-Falcon M, Pontin M, Iglesias-Pedraz JM, Lorrain S, Fankhauser C, Blazquez MA, Titarenko E, Prat S. 2008. A molecular framework for light and gibberellin control of cell elongation. *Nature* **451**: 480–484.

Moreno JE, Tao Y, Chory J, Ballaré CL. 2009. Ecological modulation of plant defense via phytochrome control of jasmonate sensitivity. *Proceedings of the National Academy of Sciences, USA* **106**: 4935–4940.

Navarro L, Bari R, Achard P, Lison P, Nemri A, Harberd NP, Jones JDG. 2008. DELLAs control plant immune responses by modulating the balance of jasmonic acid and salicylic acid signaling. *Current Biology* **18**: 650–655.

Pieterse CMJ, Van der Does D, Zamioudis C, Leon-Reyes A, Van Wees SCM. 2012. Hormonal modulation of plant immunity. *Annual Review of Cell and Developmental Biology* **28**: 489–521.

Pieterse CMJ, Zamioudis C, Berendsen RL, Weller DM, Van Wees SCM, Bakker PAHM. 2014. Induced systemic resistance by beneficial microbes. *Annual Review of Phytopathology.* **52**: 347–375.

Plett JM, Daguerre Y, Wittulsky S, Vayssieres A, Deveau A, Melton SJ, Kohler A, Morrell-Falvey JL, Brun A, Veneault-Fourrey C *et al.* 2014. Effector MiSSP7 of the mutualistic fungus *Laccaria bicolor* stabilizes the *Populus* JAZ6 protein and represses jasmonic acid (JA) responsive genes. *Proceedings of the National Academy of Sciences, USA* **111**: 8299–8304.

Thines B, Katsir L, Melotto M, Niu Y, Mandaokar A, Liu GH, Nomura K, He SY, Howe GA, Browse J. 2007. JAZ repressor proteins are targets of the SCF^{COI1} complex during jasmonate signalling. *Nature* **448**: 661–665.

Van der Does D, Leon-Reyes A, Koornneef A, Van Verk MC, Rodenburg N, Pauwels L, Goossens A, Körbes AP, Memelink J, Ritsema T *et al.* 2013. Salicylic acid suppresses jasmonic acid signaling downstream of SCFCOI1-JAZ by targeting GCC promoter motifs via transcription factor ORA59. *Plant Cell* **25**: 744–761.

Vos IA, Pieterse CMJ, Van Wees SCM. 2013. Costs and benefits of hormone-regulated plant defences. *Plant Pathology* **62**: 43–55.

Wasternack C, Hause B. 2013. Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. *Annals of Botany* **111**: 1021–1058.

Wild M, Daviere JM, Cheminant S, Regnault T, Baumberger N, Heintz D, Baltz R, Genschik P, Achard P. 2012. The Arabidopsis DELLA *RGA-LIKE3* is a direct target of MYC2 and modulates jasmonate signaling responses. *Plant Cell* **24**: 3307–3319.

Yan Y, Stolz S, Chetelat A, Reymond P, Pagni M, Dubugnon L, Farmer EE. 2007. A downstream mediator in the growth repression limb of the jasmonate pathway. *Plant Cell* **19**: 2470–2483.

Yang DL, Yao J, Mei CS, Tong XH, Zeng LJ, Li Q, Xiao LT, Sun TP, Li JG, Deng XW *et al.* 2012. Plant hormone jasmonate prioritizes defense over growth by interfering with gibberellin signaling cascade. *Proceedings of the National Academy of Sciences, USA* **109**: E1192–E1200.

Key words: DELLA respressor proteins, gibberellin, hormone crosstalk, jasmonate, JAZ respressor proteins, plant defense, shade avoidance syndrome.