Journal of Integrative Plant Biology 2009, **51** (12): 1064–1070

· Invited Expert Review ·

Gibberellin and Jasmonate Crosstalk during Stamen Development

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Received 27 Aug. 2009 Accepted 2 Sep. 2009

doi: 10.1111/j.1744-7909.2009.00881.x

Abstract

Gibberellin (GA) and jasmonate (JA) are two types of phytohormones that play important roles during stamen development. For example, *Arabidopsis* plants deficient in either of GA or JA develop short stamens. An apparent question to ask is whether GA action and JA action during stamen filament development are independent of each other or are in a hierarchy. Recent studies showed that GA modulates the expression of genes essential for JA biosynthesis to promote JA production and high levels of JA will induce the expression of three *MYB* genes *MYB21*, *MYB24* and *MYB57*. These three *MYB* genes are crucial factors for the normal development of stamen filament in *Arabidopsis*.

Key words: *Arabidopsis*; gibberellin, jasmonate, stamen development.

Peng J (2009). GA and JA crosstalk during stamen development. *J. Integr. Plant Biol.* **51**(12), 1064–1070.

Available online at www.jipb.net

Flowering in *Arabidopsis* consists of three distinct phases: (i) floral initiation (transformation of the vegetative meristem into an inflorescence meristem); (ii) floral organ initiation (determination of the identities of different floral organs); and (iii) floral organ growth (growth and maturation of different floral organs). *Arabidopsis* flowers contain sepals, petals, stamens and pistils that are organized into four concentric whorls (Smyth et al. 1990). The male reproductive organ stamens form the third whorl. Stamen development, including stamen filament elongation and anthesis, is precisely controlled so that the maturation of stamens coincides with pistil development to determine fertility. Mutations causing faulted stamen development such as filament elongation, pollen maturation or anther dehiscence will result in male sterility (Chaudhury 1993; Taylor et al. 1998). Through studying male sterile mutants many genes have been identified to control stamen development (McCormick 2004; Nakayama et al. 2005). Stamen development is also subjected to hormonal control. This is evident from the facts that both severe GA-deficient (e.g. *ga1-3* mutant in *Arabidopsis*, which is caused by a mutation in the *ent-CDP synthase* gene encoding an enzyme that catalyzes a relatively early step in the biosynthesis of GA; Sun et al. 1992; Sun and Kamiya 1994) and JA-deficient (e.g. *opr3*) mutants are male sterile due to failure of stamen filament elongation and of completion of anthesis and anther dehiscence (Koornneef and van der Veen 1980; Stintzi and Browse 2000). This review will summarize recent studies on the role of crosstalk between GA and JA in regulating stamen development, especially the stamen filament growth.

GA Downregulates DELLA to Promote Floral Development

Gibberellin was first identified in 1935 in an extract from a culture of a fungus (*Gibberella fujikuroi*) which causes the rice "bakanae" disease and then was demonstrated to be an important endogenous plant growth regulator in the 1950s (Phinney 1983). GA regulates diverse developmental processes during plant growth, ranging from seed germination, leaf expansion, stem elongation, to floral initiation (Richards et al. 2001). Plants lacking GA (e.g *ga1-3* mutant) display retarded vegetative growth of shoots (Koornneef and van der Veen 1980; Peng and Harberd 1997; King et al. 2001) and roots (Fu and Harberd 2003). The *ga1-3* mutant plants are also impaired in the development of floral organs, especially petals and stamens, and are male-sterile owing to retarded anther development (Wilson et al. 1992; Goto and Pharis 1999). Application of exogenous GA can restore the fertility to the *ga1-3* mutant plants (Koornneef and van der Veen 1980), demonstrating the importance of GA during stamen development (Pharis and King 1985). Cheng et al. performed detailed anatomical analysis of the *ga1-3* stamens and found that the male sterile phenotype of *ga1-3* is due to arrestment of the stamen filament cell elongation and failure of the completion of anthesis (Cheng et al. 2004). Further genetic studies showed that arrestment of floral development in *ga1-3* is mediated by DELLA proteins (Cheng et al. 2004; Tyler et al. 2004). There are five DELLAs in *Arabidopsis* (GAI, RGA, RGL1, RGL2 and RGL3) and they belong to a subfamily of the plant GRAS family (Pysh et al. 1999; Richards et al. 2001). DELLAs are transcription factors that function as negative regulators of the GA response in diverse plant species including *Arabidopsis*, barley, rice, wheat etc. (Peng et al. 1997, 1999; Silverstone et al. 1998; Dill et al. 2004; Ikeda et al. 2001; Boss and Thomas 2002; Chandler et al. 2002; Lee et al. 2002). GA triggers DELLA degradation via the 26S proteosome pathway to activate the GA response (Silverstone et al. 2001; Itoh et al. 2002; Fu et al. 2002; McGinnis et al. 2003; Sasaki et al. 2003; Dill et al. 2004; Fu et al. 2004; Hussain et al. 2005). The role of DELLAs in controlling stamen filament development is implicated from the observation that transgenic expression of wild-type or mutant forms of GAI can retard stamen elongation and induce male sterility in tobacco and *Arabidopsis*, respectively (Huang et al. 2003; Hynes et al. 2003). Interestingly, mutants lacking the rice or barley DELLA proteins SLR1 or SLN1 also exhibited infertility due to impaired floral development (Ikeda et al. 2001; Chandler et al. 2002). In *Arabidopsis*, mutants lacking GAI, RGA, GAI and RGA, or RGL2 alone cannot suppress the *ga1-3* floral phenotype albeit *GAI*, *RGA*, *RGL1* and *RGL2* are all expressed in developing inflorescences (Dill and Sun 2001; King et al. 2001; Lee et al. 2002). Further detailed genetic studies by Cheng et al. showed that RGA, RGL2 and RGL1 act in combination to repress the petal and stamen development and GA triggers degradation of these DELLAs to promote floral development (Cheng et al. 2004; Tyler et al. 2004; Yu et al. 2004; Griffiths et al. 2006; Willige et al. 2007).

MYB21, *MYB24* and *MYB57* are DELLA-repressible GA-response Genes that Mediate Stamen Filament Growth

Apparently, the next key question is to identify GA-response genes that act downstream of DELLAs. Global gene expression profiling is an ideal way to identify downstream response genes. The floral development in the *ga1-3* mutant is retarded and this fact suggests that the transcriptome for floral development in the *ga1-3* mutant must be in a repressive state due to the high levels of DELLA repressors. In contrast, the *ga1-3* mutant co-current loss-of-function of GAI, RGA, RGL1 and RGL2 (*ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1*) can bolt and produce fertile flowers even in the absence of GA, suggesting that (i) DELLAs are the central signaling molecules in GA-mediated floral development pathway (Cheng et al. 2004; Tyler et al. 2004; Yu et al. 2004; Cao et al. 2006); and (ii) the transcriptomes for floral development are constitutively activated in this mutant line (Ogawa et al. 2003; Cao et al. 2006). Cao et al. compared the gene expression patterns among wild type (WT), the *ga1-3* mutant and the *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1* penta mutant. Data analysis identified 360 DELLA-repressed genes (named for genes whose expression is downregulated in the *ga1-3* but is apparently restored to the WT level in the *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1* penta mutant) and 273 DELLA-activated genes (named for genes upregulated in *ga1-3* but restored to the WT level in the *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1* penta mutant), which are essential for floral development (Cao et al. 2006). In the same report, Cao et al. also compared the expression patterns between *ga1-3* and *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1* during seed germination and then cross compared the DELLA-down and DELLA-up genes in pre-germinating seeds and in young flower buds. Surprisingly, they found that the set of DELLA regulated GA-response genes essential for seed germination is largely distinct from the set essential for floral development despite the fact that GA regulates similar cellular processes during seed germination and floral development (Cheng et al. 2004; Cao et al. 2005). This observation demonstrates that the *Arabidopsis* seed germination and floral development are mediated by distinct DELLA-dependent GA-response transcriptomes.

Cheng et al. went further to identify DELLA-repressed stamen-enriched genes based on their microarray data. Semi-quantitative reverse transcription-polymerase chain reaction (RT-PCR) examination of candidate gene expression in sepal, petal, stamen and pistil identified a total of 34 DELLA-repressed stamen-enriched genes (Cheng et al. 2009). Among these 34 stamen-enriched genes, there are three *MYB* genes, namely *MYB21*, *MYB24* and *MYB57*. MYB24 and MYB21 belong to the subgroup 19 of the R2R3-MYB family (Stracke et al. 2001) while

MYB57 shares high similarity with MYB24 and MYB21, and is also a member close to MYB21 and MYB24 phylogenetically (Kranz et al. 1998). These three *MYBs* are all expressed at very low levels in the young *ga1-3* flower buds but are restored to the WT levels in the *ga1-3 gai-t6 rga-t1 rgl1-1 rgl1-1* penta mutant (Cheng et al. 2009). Detailed examination of expression of these three *MYB* genes in four quadruple mutants, namely *ga1-3 gai-t6 rgl1-1 rgl1-1* (wild type for *RGA*), *ga1-3 gai-t6 rgl1-1 rga-t2* (wild type for *RGL2*), *ga1-3 rga-t2 rgl1-1 rgl1-1* (wild type for *GAI*) and *ga1-3 gai-t6 rga-t2 rgl1-1* (wild type for *RGL1*), revealed that RGA and RGL2, but not GAI nor RGL1, were the more effective DELLAs in repressing the expression of *MYB21*, *MYB24* and *MYB57*. This observation nicely correlates with the observation described in a previous publication showing that the *ga1-3 gai-t6 rgl1-1 rgl1-1* and *ga1-3 gai-t6 rgl1-1 rga-t2* two quadruple mutants are retarded in floral development while the *ga1-3 rga-t2 rgl1-1 rgl1-1* and *ga1-3 gai-t6 rga-t2 rgl1-1* two mutants produce normal fertile flowers (Cheng et al. 2004). The apparent question to ask is if these three *MYBs* are necessary for normal floral development. To answer this question, Cheng et al. took a genetic approach and isolated mutants corresponding to these three MYB genes. Based on analyzing *myb* single, double and triple mutants, Cheng et al. found that *MYB21* is the predominant gene in controlling stamen filament elongation while *MYB24* and *MYB57* work redundantly with *MYB21*. Analyzing two hexa mutants *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1 myb21-t1* and *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1 myb24-t1* and the hepta-mutant *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1 myb21-t1 myb24-t1* showed that the *myb21 myb24* mutations are epistatic to *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1* and the short stamen caused by the *myb21 myb24* mutations are due to the arrest of cell elongation rather than cell proliferation (Cheng et al. 2009), a fact also true for the short stamen phenotype conferred by the ga1-3 mutation (Cheng et al. 2004). Therefore, the authors conclude that MYB21 and MYB24 function downstream of DELLA proteins in the GA signaling pathway to control stamen filament growth (Cheng et al. 2009).

MYB21 and *MYB24* are also JA-response Genes

Jasmonic acids are cyclopentanone derivatives and are biosynthesized from linolenic acid by the octadecanoid pathway (Creelman and Mullet 1997; Reymond and Farmer 1998; Sasaki et al. 2001; Shan et al. 2007). JA biosynthesis is accomplished by a sequential biochemical reaction mediated by JA-biosynthesis genes including *DAD1*, *LOX1, 2*, *AOS*, *AOC1, 2, 3, 4* and *OPR3* and is regulated by 12-oxo-phytodienoic acid (OPDA) compartmentalization and a JA-mediated positive feedback loop (Sasaki et al. 2001). During plant development, the level of JA in plants varies in different tissues and cell types (Creelman and Mullet 1997). JA level also changes in response to several different environmental stimuli (Howe et al. 2000; Maucher et al. 2000; Ziegler et al. 2000). For example, JA levels increase rapidly in response to mechanical stress such as wounding and tendril coiling (Falkenstein et al. 1991; Creelman et al. 1992). JAs act as plant hormones that regulate diverse aspects of plant defense and development. The function of JAs includes regulation of seed germination, promotion of the accumulation of storage proteins and inhibition of insect attack and pathogen infection etc. (Anderson 1988; Pelacho and Mingo-Castel 1991; Feys et al. 1994; McConn and Browse 1996; Creelman and Mullet 1997; McConn et al. 1997; Rao et al. 2000; Sanders et al. 2000; Cheong and Choi 2003; Howe 2004; Shan et al. 2007; Wasternack 2007; Browse 2009). Significant progress has been made during last 15 years regarding JA perception and signaling. The best studied JA-signaling is that the isoleusine-conjugated form of JA (jasmonoyl–isoleucine, JA–Ile) can stimulate binding of COI1 (CORONATINE-INSENSITIVE 1, an F-box protein) to JAZ (JASMONATE ZIM DOMAIN PROTEINS) and induce the subsequent ubiquitin-dependent degradation of JAZ. Degradation of JAZ leads to transcription activation of JA-responsive genes (including some transcription factors) (Chini et al. 2007; Farmer 2007; Shan et al. 2007; Thines et al. 2007; Katsir et al. 2008).

In addition to the above mentioned JA functions, JAs also play important roles during floral development. It is reported that flowers and pericarp tissues of the developing reproductive organs contain high levels of JA (Creelman and Mullet 1997; Cheng et al. 2009), implying a role for JA during floral development. Indeed, researchers found that JA-deficient mutant *opr3* and -signaling mutant *coi1* both displayed retarded filament elongation, delayed anther dehiscence, and reduced pollen viability that leads to male sterility (Feys et al. 1994; Xie et al. 1998; Reymond et al. 2000; Stintzi and Browse 2000; Xu et al. 2002; Wang et al. 2005). To study the mechanism of JA controlling stamen development, Mandaokar et al. analyzed the gene expression profiles between *opr3* mutant treated with or without JA. They found a total of 821 genes were specifically induced by JA and 480 genes were repressed. Detailed analysis of the stamen-specific JA transcriptome allowed the authors to identify 13 transcription factors that may be key regulators of the stamen maturation processes triggered by JA. MYB21 and MYB24 are among these 13 JA-response transcription factors. Mandaokar et al. went further to analyze the *myb21* and *myb24* loss-off-function mutants and found that the *myb21* single mutant or *myb21 myb24* double mutant displayed shorter stamens that could not be restored by exogenous JA (Mandaokar et al. 2006). Interestingly, Cheng et al. found that overexpression of *MYB21* restored the stamen filament elongation and fertility to the *opr3* flowers. This observation strongly suggests that JA-mediated stamen filament growth is mainly through the MYB pathway (Cheng et al. 2009).

GA Promotes JA Biosynthesis and JA Upregulates *MYB21*, *MYB24* and *MYB57* Expression, which is Essential for Stamen Filament Growth

The independent findings of regulation of MYB21 and MYB24 expression by GA and JA respectively, prompted Cheng et al. to study whether GA-mediated and JA-mediated stamen development are via two parallel pathways or in a hierarchical manner to control stamen development. To address this question, Cheng et al. first tested the response of GA-deficient mutant *ga1-3 gai-t6 rga-t2 rgl1-1* (a quadruple mutant) to JA treatment and the response of JA-deficient mutant *opr3* to GA treatment. They observed that application of exogenous JA onto the *ga1-3 gai-t6 rgl1-1 rgl1-1* quadruple mutant flower buds could restore the expression of *MYB21*, *MYB24* and *MYB57*, whereas application of exogenous GA onto *opr3* mutant flower buds failed to do so. Apparently, it appears that JA acts downstream of GA pathway to modulate the expression of these three *MYBs*. There are two ways for JA to act downstream of GA. First, JA may modulate the stability or activity of DELLA proteins to induce the expression of the three *MYBs*. Alternatively, GA suppresses DELLA to promote JA production or modulate JA-signaling to induce the expression of the three *MYBs*. To sort the possibility out, Cheng et al. carried out various molecular analyses and showed that neither the level of RGL2 protein (data not shown) nor the expression patterns of three GA-response genes *GA2ox1*, *GA3ox1* and *GA20ox1* were obviously altered in the JA-treated *ga1-3 gai-t6 rga-t2 rgl1-1* plants although the three *MYBs* are apparently induced. This observation excludes the first possibility. On the other hand, the fact that JA application can induce the expression of the three *MYBs* in the GA-deficient background strongly suggests that JA biosynthesis is or partially impaired in the *ga1-3 gai-t6 rga-t2 rgl1-1* mutant. Indeed, measurement of JA contents showed that the JA content in the young flower buds of the *ga1-3 gai-t6 rga-t2 rgl1-1* quadruple mutant is much lower than that in the WT (Cheng et al. 2009). Cheng et al. went further to analyze the major genes involved in the JA biosynthesis (including *DAD1*, *LOX1, 2*, *AOS*, *AOC1, 2, 3, 4* and *OPR3*) in GA-related or JA-related mutants treated with or without GA or JA. They found that GA upregulates JA-biosynthetic genes *DAD1* and *LOX1*. DAD1 acts at the early step of JA biosynthesis, which limits the production of the initial substrates for JA biosynthesis in flowers. In the *dad1* null mutant, the JA levels in flowers were only 22% of that of WT (Ishiguro et al. 2001). *DAD1* was greatly downregulated in both *ga1-3* single and *ga1-3 gai-t6 rga-t2 rgl1-1* quadruple mutants, but partially restored to a relatively high level in the *ga1-3 gai-t6 rga-t2 rgl1-1 rgl1-1* mutant in the young flower buds. This expression pattern of *DAD1* is consistent with the JA levels found in these mutant lines. Based on these results the authors conclude that GA is required for the expression of *DAD1* to control the production of JA via repression of DELLA proteins (Cheng et al. 2009).

The next question to ask is whether GA induction of JA biosynthesis is prior to the induction of the three *MYB* expressions. Indeed, it is found that exogenous GA treatment of the *ga1-3 gai-t6 rga-t2 rgl1-1* induced the *DAD1* expression first then followed by the induction of *MYB* expression (Cheng et al. 2009). However, how the *DAD1* expression is controlled by the GA-DELLA pathway is still unknown. One report showed that *AGAMOUS* (*AG*) expression is downregulated in the *ga1-3* mutant (Yu et al. 2004), while it appears that AG can directly regulate *DAD1* expression (Ito et al. 2007). Therefore, it would be interesting to study whether there is a relationship among DELLAs, AG and DAD1 in the future. In addition to *DAD1*, the data obtained by Cheng et al. also clearly showed that expression of *LOX1* was downregulated in the *ga1-3* mutant and restored to the WT level in the penta mutant. Combining these observations suggests that GA may be one of the key endogenous signals involved in the regulation of JA biosynthesis genes (Grant and Jones 2009). Although exogenous GA induced *MYB21*, *MYB24* and *MYB57* expression in the *ga1-3 gai-t6 rga-t2 rgl1-1* quadruple mutant, the restoration of the

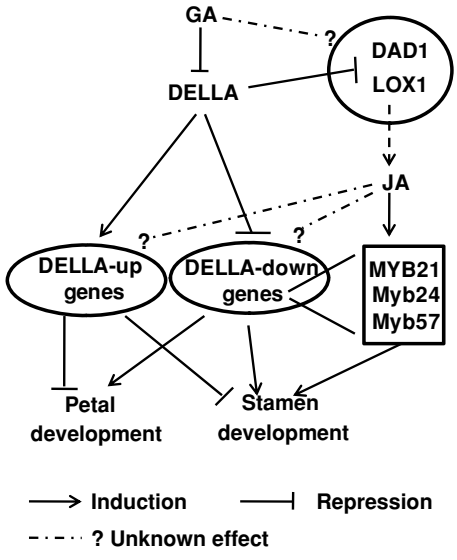

Figure 1. Diagram outlining the crosstalk between gibberellin (GA) and jasmonic acid (JA) during stamen and petal development.

Gibberellin triggers the degradation of DELLA proteins to release DELLA's repression on DELLA-down genes and JA-biosynthesis genes *DAD1* and *LOX1*. Activation of *DAD1* and *LOX1* expression promotes JA production. JA induces the expression of *MYB21*, *MYB24* and *MYB57* for normal stamen filament growth. GA controls stamen development also via a JA-independent pathway mediated by unknown GA-response factors. There is still no concrete evidence to show whether GA can regulate JA biosynthesis via a DELLA-independent pathway and if JA can regulate any other DELLA-up or DELLA-down genes in addition to *MYB21*, *MYB24* and *MYB57*.

expression of these *MYBs* is not enough to rescue the mutant flower phenotype (Cheng et al. 2009). Apparently, these three *MYBs* are necessary but not sufficient for GA-mediated floral development. In addition, Cheng et al. argued that modulation of the JA pathway may be only one of the branches of GA function in regulating stamen development because both *ga1-3* single and *ga1-3 gai-t6 rga-t2 rgl1-1* quadruple mutants display a more severe flower phenotype than does the *myb21-t1 myb24-t1 myb57-t1* triple mutant (Cheng et al. 2009).

In conclusion, all evidence in hand demonstrates that GA modulates JA biosynthesis gene expression (e.g. *DAD1* and *LOX1*) to promote the production of JA and high levels of JA will induce the expression of *MYB21*, *MYB24* and *MYB57* to promote stamen filament development (Figure 1). Therefore, we can envisage a hierarchical relationship between GA and JA in that modulation of the JA pathway by GA is one of the prerequisites for GA to control the normal stamen development in *Arabidopsis*.

Acknowledgements

I thank Dr Hui Cheng for her hard work on revealing the relationship between GA and JA during her PhD study in my lab, and also for her major contribution to the drawing of the diagram used here as Figure 1.

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(Co-Editor: Li-Jia Qu)