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Hormonal control of the floral transition: Can one catch them all?

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ABSTRACT

The transition to flowering marks a key adaptive developmental switch in plants which impacts on their survival and fitness. Different signaling pathways control the floral transition, conveying both endogenous and environmental cues. These cues are often relayed and/or modulated by different hormones, which might confer additional developmental flexibility to the floral process in the face of varying conditions. Among the different hormonal pathways, the phytohormone gibberellic acid (GA) plays a dominant role. GA is connected with the other floral pathways through the GA-regulated DELLA proteins, acting as versatile interacting modules for different signaling proteins. In this review, I will highlight the role of DELLAs as spatial and temporal modulators of different consolidated floral pathways. Next, building on recent data, I will provide an update on some emerging themes connecting other hormone signaling cascades to flowering time control. I will finally provide examples for some established as well as potential cross-regulatory mechanisms between hormonal pathways mediated by the DELLA proteins.

1. Introduction

When to flower is a key decision for plants, affecting the adaptability of species to any given environment. The floral transition marks a change in the shoot apical meristem (SAM), the growing tip of the shoot; the SAM generates rosette leaves separated by short internodes during the vegetative phase (V), and switches to produce flowers, fruits and seeds after the floral transition. Besides producing all lateral structures, the SAM generates the portion of stem which separates consecutive lateral structures (internodes). In addition, the SAM perpetuates itself, thus keeping its own identity, by maintaining a pool of undifferentiated stem cells (Huala and Sussex, 1993; Sussex, 1989). The switch to flowering occurs when the (vegetative) SAM receives appropriate signals (Bernier et al., 1993) and in *Arabidopsis* it precedes bolting (i.e. the elongation of the uppermost internodes of the stem). After the floral transition, the SAM enters the inflorescence phase (I) when flowers appear at the flanks of the SAM instead of leaves (Fig. 1). This alters the above-ground architecture of the plant (Coen and Nugent, 1994), and different mutants affected in the switch between the V and I developmental phases can be precisely identified and compared based on the number of vegetative leaves. Late-flowering and early-flowering mutants produce a greater and fewer number of vegetative leaves compared with wild-type plants, respectively (Koornneef et al., 1991).

Physiological and genetic studies of different flowering time mutants have led to the definition of four major flowering pathways in *Arabidopsis* (Martínez-Zapater et al., 1994). The photoperiodic and the vernalization pathways convey light and temperature information (Amasino, 2010; Andrés and Coupland, 2012; Bäurle and Dean, 2006; Kobayashi and Weigel, 2007). In contrast, the autonomous and the gibberellic acid (GA) pathways largely relay endogenous cues (Mutasa-Göttgens and Hedden, 2009; Simpson, 2004). During the past 15 years this genetic and physiological framework has been increasingly elaborated to include the plant age and ambient temperature pathways (Huijser and Schmid, 2011; Samach and Wigge, 2005). Additionally, it is now becoming apparent that in natural environments plants are able to recognize an even wider array of environmental information that, once integrated, give rise to developmental decisions (Brachi et al., 2012; Burghardt et al., 2016; Kenney et al., 2014; Kooyers, 2015; McKay et al., 2003). Because extreme environmental conditions ultimately challenge plant survival, the ability to modulate the flowering process plays an important role in the adaptation to different environments (Kazan and Lyons, 2016; Takeno, 2016).

Plant hormones constitute a major signaling network that relay external or internal variations and translate these into plant developmental responses (Santner et al., 2009; Wolters et al., 2009). It is thus not surprising that modulation of hormone signaling also contributes to the extraordinary plasticity of the flowering process. While GA is probably the best studied hormone in flowering, other hormones including abscisic acid (ABA), jasmonate (JA), salicylic Acid (SA), brassinosteroids (BRs), cytokinin (CKs), ethylene (ET) and nitric oxide (NO) have been reported to play a role in regulating the flowering

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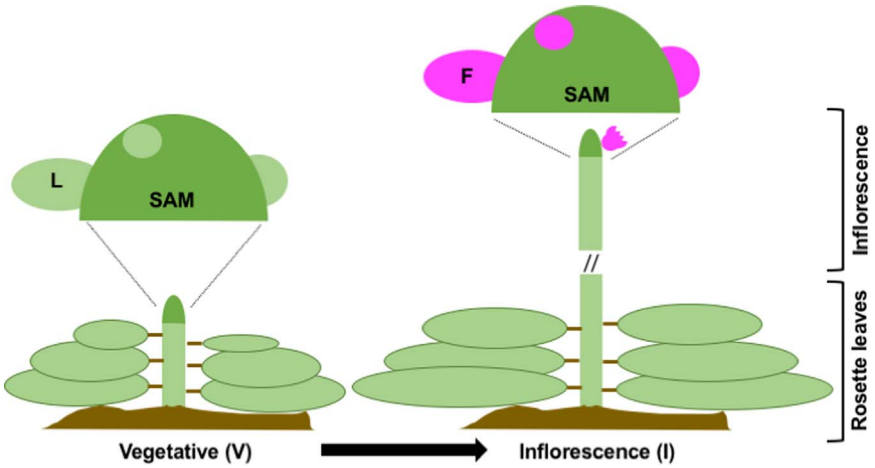

Fig. 1. *The floral transition occurs at the shoot apical meristem (SAM).* Graphical representation of the developmental switch occurring in Arabidopsis between the vegetative (V) and inflorescence (I) phases. During the V phase the SAM produces primordia which undergo a leaf fate (L, light green). After the floral transition, the SAM generates primordia that attain a floral identity (F, purple). Note that the number of vegetative leaves (composing the rosette) is generally directly related to flowering time (i.e. the duration of the switch between the V and I phases).

network (Davis, 2009; Kazan and Lyons, 2016). Furthermore, in addition to these well-established phytohormones, several diffusible molecules including sugars and other metabolites regulate flowering (Mattioli et al., 2008; Wahl et al., 2013). The role of sugar has been recently reviewed and will therefore not further discussed here (Bolouri Moghaddam and Van den Ende, 2013).

Our increasing knowledge of the different genetic components underlying hormone signaling allows us to better understand how these hormones affect flowering time. Interestingly, different hormones signaling cascades often converge to refine the expression of key floral genes under specific conditions. This observation emphasizes the importance of treating the various flowering pathways as part of an integrated structure, rather than the sum of insulated modules. In this review I discuss recent advances in the role of different hormone signaling pathways in the regulation of the floral transition, emphasizing their mode of integration with known floral genes. Although my discussion will be limited to *Arabidopsis*, it is likely that similar circuitries might exist in other species, including crops.

2. The floral network of *Arabidopsis*

Here I provide an overview of the basic structure of the different floral pathways, emphasizing the role of the photoperiodic pathway for its tight connection with different hormonal signals. I invite the reader to refer to recent exhaustive reviews to gain further details on each of these signaling modules.

2.1. The photoperiodic pathway

It has been long recognized that the length of the day (known as photoperiod) is a crucial environmental factor that controls flowering (Mozley and Thomas, 1995). The perception of the photoperiod occurs in the leaves and triggers the production of one or more mobile, graft-transmissible substances (florigens) which ultimately promote flowering at the shoot apex (Evans, 1971). The study of *Arabidopsis* mutants impaired in photoperiod perception has provided information about the molecular components required for proper photoperiod perception and signaling through the production of the florigenic substance (Andrés and Coupland, 2012; Golembeski and Imaizumi, 2015; Kobayashi and Weigel, 2007). As a facultative long day plant, *Arabidopsis* flowers much earlier under long days (LDs, typical of spring/summer) compared to short days (SDs, typical of autumn/winter). Mutants of *constans* (*co*), *gigantea* (*gi*), and *flowering locus t* (*ft*) flower late under LDs conditions but display little or no flowering defects under SDs (Fowler et al., 1999; Huq et al., 2000; Kardailsky et al., 1999; Kobayashi et al., 1999; Koornneef et al., 1998; Putterill et al., 1995). The molecular study of these mutants allowed for the identification of the mobile protein FLOWERING LOCUS T (FT) and its paralogue TWIN SISTER OF FT (TSF) as the main constituents of the florigen substance (Corbesier et al., 2007). The CO and GI proteins are required for the correct perception of photoperiod and the transcriptional activation of the florigen genes. *CO* encodes a zinc finger transcriptional regulator expressed in the phloem companion cells of the leaves (An et al., 2004; Putterill et al., 1995; Takada and Goto, 2003). The transcriptional activation of *CO* is daily regulated, with *CO* transcript levels being low in the morning and reaching a maximum in the night (Suarez-Lopez et al., 2001). GI is largely responsible to confer such daily fluctuations of *CO* transcripts. GI interacts with LIGHT OXYGEN VOLTAGE (LOV) domain-containing FLAVIN-BINDING, KELCH REPEAT F-BOX 1 (FKF1) blue light photoreceptor. Blue light stimulates the formation of the GI–FKF1 complex which targets a class of CO transcriptional repressors, the CYCLING DOF FACTORs (CDFs), for degradation in a specific temporal window in LDs (Fornara et al., 2009; Imaizumi et al., 2005; Sawa et al., 2007; Song et al., 2014). Following degradation of the CDF repressors, a poorly characterized series of events lead to the transcriptional activation of *CO*. Among the positive regulators of *CO* is *FLOWERING BHLH* (*FBH1*) and related group of bHLH transcription factors (Ito et al., 2012).

CO protein is specifically stabilized under LDs when the peak of *CO* mRNA peaks in the light phase at the end of the day (Suarez-Lopez et al., 2001). Several types of photoreceptors act at different parts of the day to control CO abundance. Ultimately, a peak of CO abundance occurs in coincidence with dusk under LDs (Jang et al., 2008; Lazaro et al., 2015; Liu et al., 2008; Song et al., 2012b; Valverde et al., 2004; Zuo et al., 2011). Photoperiod-stimulated CO is able to induce early flowering by activating *FT* and *TSF* in the phloem companion cells (Adrian et al., 2010; An et al., 2004; Jang et al., 2009; Michaels Scott et al., 2005; Yamaguchi et al., 2005; Yoo et al., 2005). In addition to CO, the transcriptional regulation of *FT* involves a complex interplay between different classes of transcription factors and three-dimensional chromatin conformations (Abe et al., 2015; Bratzel and Turck, 2015; Cao et al., 2014; Golembeski and Imaizumi, 2015; Liu et al., 2014). This complexity probably reflects the integrative role of *FT*, conveying a vast array of signaling pathways in addition to photoperiod (Pin and Nilsson, 2012). FT protein acts as a florigenic signal by moving long distance to the SAM through a regulated transport system (Corbesier et al., 2007; Jaeger and Wigge, 2007; Liu et al., 2012;

Mathieu et al., 2007; Notaguchi et al., 2008). In the SAM, FT forms a complex with the bZIP transcription factors FLOWERING LOCUS D (FD) and FD PARALOGUE (FDP) to activate another set of genes that trigger a floral fate in lateral primordia (Abe et al., 2005; Jaeger et al., 2013; Wigge et al., 2005).

2.2. The vernalization and the autonomous pathways

Both the autonomous and vernalization pathways activate flowering indirectly, by inducing and maintaining a state of epigenetic silencing at the *FLOWERING LOCUS C* (*FLC*) locus (Boss et al., 2004; Henderson et al., 2003; Kim et al., 2009; Michaels and Amasino, 1999). *FLC* encodes a MADS domain protein that represses key floral activators in the leaf and in the SAM (Searle et al., 2006). *Arabidopsis* accessions that have high *FLC* levels flower extremely late, unless they experience vernalization (i.e. a period of growth under cold conditions) (Shindo et al., 2006). In response to cold exposure, *FLC* expression is reduced as a result of epigenetic silencing occurring at the *FLC* locus (Amasino, 2004; Bastow et al., 2004; Sheldon et al., 2000; Sung and Amasino Richard, 2004). On return to warm conditions the silencing is maintained epigenetically so that plants are ready to respond to flowering inductive cues. Mutations in the autonomous pathway cause a delay in flowering irrespective of the photoperiod, so that these mutants flower late under any day length condition (Koornneef et al., 1998). Moreover, the late-flowering phenotype of autonomous pathway mutants can be reverted by vernalization (Simpson, 2004). Unlike the photoperiodic pathway, the autonomous pathway does not form a sequential cascade of events, but is rather composed of genetically separable modules (Koornneef et al., 1998; Michaels and Amasino, 2001; Simpson et al., 1999). Each of these modules is involved in the negative regulation of *FLC*.

2.3. Integration of flowering pathways in the SAM

The FT-FD activator complex reprograms different transcriptional networks in the SAM required for the specification of floral primordia. Here, another level of integration between various floral pathway occurs through the MADS domain family genes *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* (*SOC1*) and *FRUITFULL* (FUL) both early targets of the FT–FD complex (Abe et al., 2005; Borner et al., 2000; Jang et al., 2009; Lee et al., 2000; Melzer et al., 2008; Moon et al., 2003; Samach et al., 2000; Searle et al., 2006; Wang et al., 2009b; Wigge et al., 2005; Yamaguchi et al., 2009). These genes products contribute to the amplification of the FT-FD signal and activate the floral meristem identity genes. While the precise site of migration of FT in the SAM is still unknown, only the cells located in the peripheral zone of the SAM are able to acquire a floral fate, marked by the upregulation of the floral meristem identity gene *LEAFY* (*LFY*) and *APETALA1* (*AP1*) (Hempel et al., 1997, 2000; Schultz and Haughn, 1993; Weigel et al., 1992). The central portion of the SAM is not competent to activate a floral gene expression program due to the presence of the *FT* homologue *TERMINAL FLOWER 1* gene product, which antagonizes FT function (Bradley et al., 1997; Conti and Bradley, 2007; Hanano and Goto, 2011; Jaeger et al., 2013; Ratcliffe et al., 1999).

3. Hormonal regulation of the floral transition

Recent molecular studies delineate a more precise role for some hormones in the floral transition, and define their modes of interaction with known floral pathways. In broad terms these studies indicate that several hormonal signals affect flowering at two sites, the leaf and the SAM. Secondly, different hormones appear to co-ordinately converge on the transcriptional activation of a small number of floral integrator genes. Thirdly, while different hormonal pathways participate in the floral process (Davis, 2009; Kazan and Lyons, 2016; Mutasa-Göttgens and Hedden, 2009), the role of GA is probably the most dominant. Fourthly, the GA-signaling proteins DELLAs act as hubs for hormonal cross-regulation upstream of individual floral integrators.

3.1. GA is an important regulator of flowering of Arabidopsis

GA signaling constitutes one of the four major floral pathways initially identified in *Arabidopsis*. The GA signaling cascade is activated by bioactive gibberellins (GAs). GAs derive from a common diterpene precursor, whose structure is sequentially elaborated by a complex array of oxidative enzymes (Hedden and Kamiya, 1997; Yamaguchi, 2008). The cellular homeostasis of GAs is maintained by regulation of the *GA20-oxidase* (*GA20OX*) and *GA3-oxidase* (*GA3OX*) genes, that catalyze the final steps of GAs biosynthesis, and the *GA2-oxidases* (*GA2OX*), which contribute to GAs inactivation and turnover. Mutants impaired in GA biosynthesis (e.g. *ga1*, defective in the early steps of GAs production) are moderately late flowering under LDs but do not flower under SD conditions (Wilson et al., 1992). These phenotypic observations indicate an absolute requirement for GAs when the photoperiodic pathway is not active. They also suggest that GAs production is largely dispensable under LDs, presumably as a result of the activation of the photoperiodic pathway and consequent mobilization of FT in the apex.

Molecular studies coupled with a more precise knowledge of individual components of GA signaling have greatly helped elucidate the mode of action of GAs in the presence or absence of activated photoperiodic signaling (Galvão et al., 2012; Hou et al., 2014; Porri et al., 2012; Yu et al., 2012). GA signaling is largely mediated by a class of nuclear proteins, globally referred to as DELLA, which act as negative regulators of GA signaling (Harberd, 2003). There are five *DELLA* genes in *Arabidopsis*, with both specific and redundant functions (Daviere and Achard, 2013). All these DELLA proteins are regulated at the post-translational level by varying levels of GAs, which trigger their degradation through the ubiquitin-proteasome system. The proteolytic cascade initiates when GAs bind to the soluble receptor GID1 (Griffiths et al., 2006; Murase et al., 2008; Shimada et al., 2008; Ueguchi-Tanaka et al., 2005, 2007). GAs promote a conformational change in GID1 that increases its affinity for DELLA proteins, via direct binding to the DELLA domain (Feng et al., 2008; Griffiths et al., 2006; Hirano et al., 2010; Wang et al., 2009a; Willige et al., 2007). This interaction stimulates the binding of the E3 Ubiquitin ligase SLEEPY1 (SLY1) to DELLA, which activates its degradation (Dill et al., 2004; Silverstone et al., 1998, 2001). In line with a role for GA signaling in flowering, mutants affected in GA perception (*gid1*), DELLA ubiquitination (*sly1*), or mutants carrying a dominant, non-degradable form of the DELLA protein GAI (GA-INSENSITIVE, *gai*) display similar flowering phenotypes to the aforementioned *ga1* biosynthetic mutants (Galvão et al., 2012; Griffiths et al., 2006; Porri et al., 2012; Willige et al., 2007). In contrast, mutants carrying loss-of function alleles in the *DELLA* genes, display an early flowering phenotypes (Galvão et al., 2012).

Using transgenic approaches, it was possible to locate two major sites of GA action in flowering: the leaf and the SAM. These studies took advantage of available promoters active in the SAM or in the leaf, to locally impair either the accumulation of GAs or its signaling. The mis-expression of the GA catabolic enzyme *GA2OX7* in the leaf (via the *SUC2* promoter, active in the phloem companion cells) or in the SAM (via the *KNAT1* promoter) causes a general delay in flowering under LDs. However, under SDs, only the SAM-specific depletion of GAs causes a non-flowering phenotype, reminiscent of the phenotype of *ga1* mutants (Porri et al., 2012). Similar phenotypes arise by mis-expressing a non-degradable, constitutively active form of DELLA (ΔDELLA) in the SAM or in the leaf (Galvão et al., 2012; Yu et al., 2012). Several important conclusions can be drawn from these experiments. First, they support a role for GAs in the SAM which is crucial for flowering under SD conditions, but less so under LDs. Secondly, they demon-

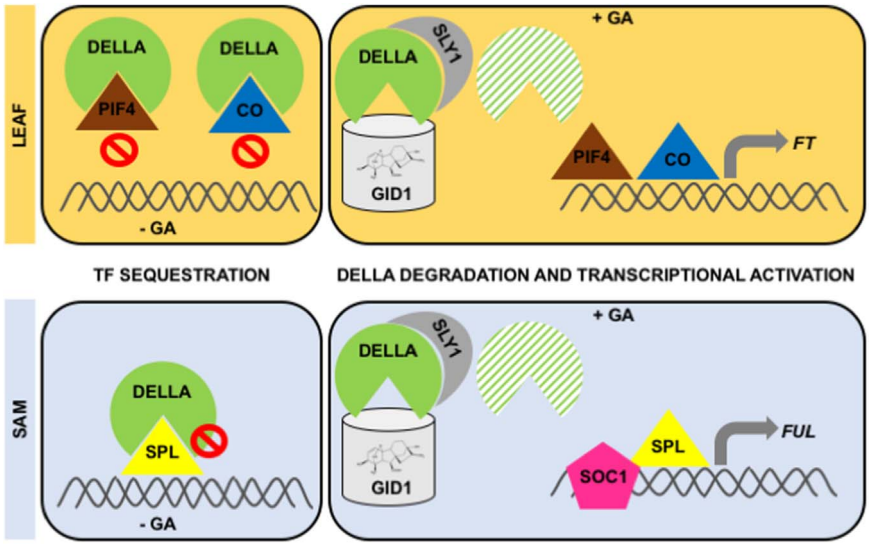

Fig. 2. *Cycles of DELLA sequestration and degradation modulate transcriptional events in the leaf and in the SAM*. Cartoon summarizing the role of DELLA in the control of flowering time at two sites of the plant, the leaf and the SAM. In the leaf, DELLA prevent positive regulators of *FT* including CO and PIF4 from binding to DNA. In the shoot, DELLA prevents SPLs factors from activating the transcription of floral integrators like *FUL*. In both cases GAs trigger DELLA degradation and subsequent release of the transcriptional regulator.

strate that DELLA degradation must occur to activate flowering. Thirdly, under LDs, GA accumulation in the leaf can promote flowering, in the same cells where the production of FT occurs. I will now illustrate how GAs activate gene expression and flowering by controlling DELLA accumulation starting with the role of GAs in the leaf (Fig. 2).

3.2. GA signals modulate the expression of the florigen genes in the leaf

Under LDs GAs promote the transcriptional activation of *FT*. Supporting this role, reduced levels of *FT* transcript are observed in GA-depleted lines or plants with impaired GA signaling, whereas increased *FT* levels are observed when GAs are applied exogenously or in mutants with activated GA signaling (Galvão et al., 2012; Hisamatsu and King, 2008; Hou et al., 2014; Porri et al., 2012; Yu et al., 2012). In contrast, foliar applications of GAs cannot activate *FT* transcriptionally in wild-type plants under SDs or in mutants of *co* under LDs (Hisamatsu and King, 2008; Wang et al., 2016). Thus, one critical question is to identify the GA-sensitive component(s) which regulate the expression of *FT* under LDs.

Recent reports describe multiple mechanisms through which GAs can regulate the expression of *FT*, all occurring downstream of the transcriptional activation of *CO* (Galvão et al., 2012; Hou et al., 2014; Porri et al., 2012; Yu et al., 2012). One such mechanism relies on the DELLA-dependent down-regulation of the *microRNA172* (*miR172*), which negatively regulates the *APETALA2* (*AP2*)*-like* genes *SCHLAFMUTZE* (*SMZ*), *SCHNARCHZAPFEN* (*SNZ*), *TARGET OF EAT1, 2* and 3 (*TOE1,2* and *3*), via translational inhibition (Aukerman and Sakai, 2003; Chen, 2004; Mathieu et al., 2009). The AP2-like proteins in turn negatively regulate the transcriptional activation of the florigen genes (as well as other floral integrators in the SAM) (Mathieu et al., 2009). The GA and the *miR172* pathways are interconnected through the DELLA and the SQUAMOSA PROMOTER BINDING PROTEIN-LIKE (SPL) transcriptional regulators (Yu et al., 2012). The SPLs are positive regulators of *miR172* and a particular *SPL* gene (*SPL3*) product also directly binds to and activates *FT* (Kim et al., 2012). DELLAs bind to SPL proteins and prevent their trans-activation function on target genes (Yu et al., 2012). As a result of this, when a constitutively active *ΔDELLA* allele is expressed under the *SUC2* promoter the accumulation of the *miR172* is significantly reduced (Yu et al., 2012), which leads to reduced accumulation of *FT* transcript. Supporting the physiological significance of this mechanism, the overexpression of *miR172* can rescue the late flowering of *SUC2:ΔDELLA* plants, suggesting that one role of DELLA is to enhance the transcriptional repression of *FT* via interfering with *SPL-miR172* regulation.

Besides indirectly activating a repressor of *FT*, DELLA also impairs the function of CO, the key transcriptional activator of *FT*. DELLA binds to the CO, CO-like, TOC1 (CCT) domain of CO, responsible for its interaction with the DNA (Tiwari et al., 2010; Xu et al., 2016a). Consequently, either the depletion of GAs or an increase in DELLA levels result in reduced transcript accumulations of *FT* and *TSF* at dusk, coincidently with the stabilization of CO (Porri et al., 2012; Wang et al., 2016). *In vitro* assays also indicate that DELLA prevents the interaction between CO and the NF-Y subunit B, which is required for the CO-mediated activation of *FT in vivo* (Kumimoto et al., 2008; Tiwari et al., 2010). The function of the CO/NF-Y complex has been proposed to maintain a specific chromatin conformation at the *FT* locus, which favors its transcriptional activation (Cao et al., 2014). Therefore, by sequestering CO, DELLA prevents the formation of a transcriptionally active chromatin conformation at the *FT* locus (Wang et al., 2016) (Fig. 2). Interestingly, since also DELLA interact with the NF-Y subunits B and C a more elaborated mechanism emerges whereby DELLA obstruct the formation of the NF-Y/CO complex by sequestering its different molecular components (Hou et al., 2014).

DELLA proteins are able to physically interact with a variety of transcriptional regulators. In many cases such interactions lead to the inhibition of the DNA-binding capacity of these transcription factors (TF) (Davière and Achard, 2016). Amongst the DELLA-regulated TFs is PHYTOCHROME INTERACTING FACTOR 4 (PIF4), which binds to the promoter of *FT* and contributes to its activation under warm ambient temperature in cooperation with CO (Fernández et al., 2016; Kumar et al., 2012). Following interaction with DELLA proteins, PIF4 can no longer bind to DNA (de Lucas et al., 2008; Feng et al., 2008) (Fig. 2). Therefore, GAs may broadly impact on how plants sense variations in temperature (which translates into changes in flowering time) through modulating the interaction between DELLA and PIF4 or other PIF-like TFs (Galvão et al., 2015) (Fig. 3).

In addition to sequestering TFs, DELLA can affect transcriptional events through other mechanisms (Davière and Achard, 2016). For example, a recent report extends the sequestration model to show that DELLA also triggers degradation of its bound proteins (Li et al., 2016a). Although this mechanism does not seem to apply to the regulation of CO (Wang et al., 2016; Xu et al., 2016a), it does affect

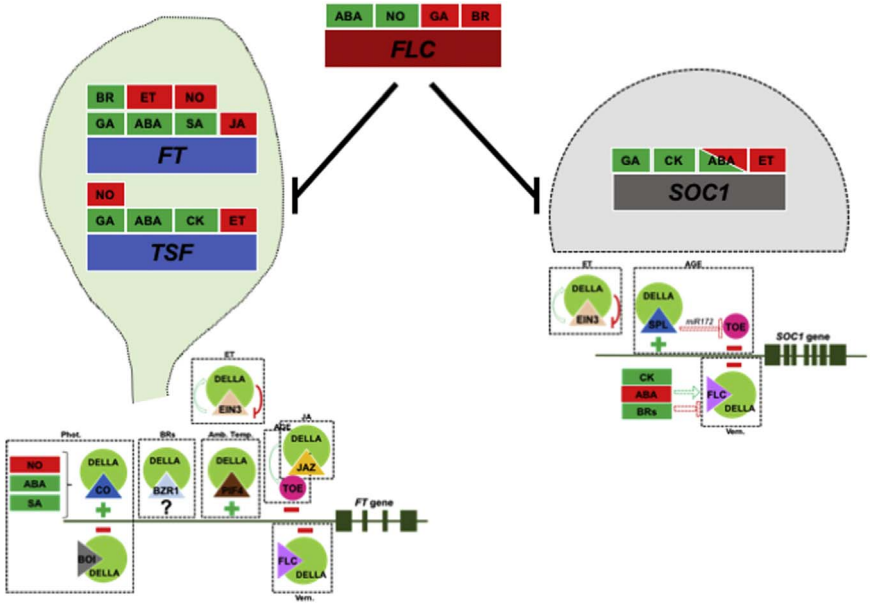

Fig. 3. *Hormonal regulation of the floral integrators and integrative roles of DELLA in the floral network.* Summary of the hormonal regulatory mechanisms operating upstream of floral integrators in the leaf and the SAM. Individual hormones can have positive (green), negative (red) or both (red and green) roles on the transcriptional activation of floral genes *FT*, *TSF* and *SOC1* in the leaf or in the SAM. *FLC* is also regulated by different hormones and negatively regulates floral integrators. DELLA proteins are connected to different floral and hormonal pathways as illustrated below in more details. DELLA is connected with the Age (by down regulating miR172, dotted green arrow), Ambient temperature (Amb. Temp., via PIF4), Photoperiodic (Phot., via CO and BOI) and Vernalization pathways (Vern., via FLC) in the leaf or in the SAM. Potential relation with the JA (via the JAZ) and BRs (via BZR) are also shown, although it is not clear whether JA itself acts as a flowering-inhibitory molecule, and how BZR1 activates *FT*. DELLA interacts with the ET pathway whereby EIN3 indirectly promotes DELLA accumulation (dotted green arrow), whereas DELLA directly inhibits EIN3 function (solid red line). Note that other hormones converge to regulate the photoperiodic pathways through regulating CO action or accumulation (see text.). Symbols (+ or -) indicate the positive or negative contribution of the indicated transcriptional regulators to gene expression. DELLA is connected to the age pathway in the SAM (through regulation of the SPLs-miR172 module), and, indirectly with the ethylene pathway. It is assumed that in the SAM, ABA antagonizes GAs by downregulating *SOC1* expression or signaling. This could be indirect, through the transcriptional activation of *FLC* (dotted green arrow) which in turn interacts with DELLA. BRs in turn negatively regulate *FLC* (dotted red line), whereas CKs might promote *SOC1* expression through an unknown mechanism.

other *FT* regulators like the PIFs. In other cases, DELLA proteins guide transcriptional repressors at specific genomic locations, including the *FT* locus. A class of four RING domain-containing proteins referred to as BOTRYTIS SUSCEPTIBLE1 INTERACTORs (BOIs) interact with DELLAs and act as repressors of flowering time (Park et al., 2013). With respect to the floral transition, the BOI genes are largely epistatic to *DELLA* suggesting that the activity of BOI is required for DELLA function. BOI and the DELLA protein REPRESSOR OF GA (RGA) are enriched at similar positions of the *FT* promoter, and the binding of BOI to these promoter regions is DELLA-dependent (Nguyen et al., 2015). Besides directly interacting with DELLA, BOI also interacts also with CO via its CCT domain, which probably interferes with the DNA binding activity of CO (Nguyen et al., 2015). Thus, one possibility is that DELLA, in addition to impeding CO access to the DNA, further obstructs the formation of the CO/NF-Y complex by recruiting BOI in chromatin positions normally occupied by CO. In a similar fashion, DELLA proteins bind to and recruit FLC to the *FT* (and *SOC1*) promoters, thus contributing to transcriptional repression (Li et al., 2016b) (Fig. 3).

Because of this huge diversity of DELLA- coordinated protein complexes that regulate *FT*, one would expect that GA production and/or signaling are temporally and spatially aligned with the expression of *FT*. From a spatial point of view, the accumulation of *GA3OX2* (catalyzing the last step of the GA biosynthetic pathway) is found in the vasculature of leaves, closely resembling the domain of *FT* expression (Mitchum et al., 2006). The expression of this gene is directly repressed by the functionally redundant *TEMPRANILLO* (*TEM*) *1* and *2* transcriptional regulators, which are also direct negative regulators of *FT* (Castillejo and Pelaz, 2008). *TEM1* and *2* are diurnally regulated, peaking at dusk, in coincidence with *FT* expression (Osnato et al., 2012). Therefore, the *TEMs* antagonize CO in two ways; by direct repression at the *FT* promoter, and by preventing the over-accumulation of GAs in the vasculature in coincidence with CO stabilization. Conversely, the MYB-type transcription factor *ASYMMETRIC LEAVES 1* (*AS1*) antagonizes TEM function in the phloem companion cells at two levels. Not only is AS1 a positive regulator of *FT* expression, but it also promotes the activation of *GA20OX1*, which contributes to GA accumulation (Song et al., 2012a). Thus, in the phloem companion cells, different transcriptional regulators coordinate GA accumulation and *FT* expression by directing transcriptional events at the promoters of the GA metabolic genes and *FT*.

From a temporal perspective, the pattern of accumulation of the DELLA protein RGA shows diurnal variations, with low DELLA proteins occurring at dusk (Wang et al., 2016). Such rhythmicity in DELLA accumulation may also derive from circadian regulation of the GA receptors *GID1A* and *B* (Arana et al., 2011). Thus, the timing of accumulation of CO protein broadly coincides with the GA-sensitive temporal window characterized by reduced DELLA levels. Furthermore, since the accumulation of GAs depends on various environmental conditions, GA signaling also relays external information onto *FT* regulation (Achard et al., 2006; Hisamatsu and King, 2008; Magome et al., 2008). In summary, GA signaling and production provide temporal, environmental and spatial information that, superimposed on activated photoperiod signaling, modulate the transcriptional activation of *FT*.

3.3. GAs promote flowering in the SAM

The SAM is the other important site of GA action in flowering (Figs. 2 and 3). In support of this conclusion, foliar applications of GAs cannot reactivate *FT* expression under SDs, yet they activate flowering of wild-type, *co* and, *ft tsf* mutant plants - albeit to a lesser extent compared with the wild type (Hisamatsu and King, 2008; Jang et al., 2009; Porri et al., 2012; Song et al., 2012a). In the light of the previously-described mis-expression studies, these data suggest that an excess of GAs in the leaf under non inductive conditions can trigger

flowering in the SAM, independent of the florigen genes. This can be due to transport of GAs from the leaf to the SAM or thorough activation of an *FT*-independent route to flowering (Eriksson et al., 2006). Although the precise dynamics of GA distribution within plants are still poorly understood, it is well known that GAs are actively transported from sites of synthesis to sites of action (Ragni et al., 2011; Regnault et al., 2015; Tal et al., 2016). If we consider flowering under continuous SDs, the levels of GA_4 (a bioactive and abundant GA isoform in *Arabidopsis*), increase dramatically in the shoot in coincidence with the floral transition. However, such an increase in GA_4 is not preceded by the transcriptional upregulation of the GA biosynthetic genes at the apex, suggesting that the pool of GA_4 originates from sources outside of the SAM itself (Eriksson et al., 2006). A critical regulator of GA homeostasis under SDs is the basic helix-loop-helix transcription factor NO FLOWERING IN SHORT DAY (NFL). *nfl* mutants display altered levels of GA metabolic and catabolic genes (reduced and increased, respectively), which is reflected in a broad perturbation of GA levels in the shoot apex. Intriguingly, unlike GA deficient mutants, *nfl* mutant plants do not display observable flowering defects under LDs, pointing to a photoperiod-dependent mechanism of regulation of NFL and its targets (Sharma et al., 2016).

Under LDs elevated expression of the GA metabolic gene *GA20OX2* can be observed in the rib region of the SAM in coincidence with the floral transition (Andrés et al., 2014). This pattern of *GA20OX2* accumulation requires the mobilization of FT in the SAM. Here, FT promotes the expression of *GA20OX2*, through the downregulation of *SHORT VEGETATIVE PHASE* (*SVP*), a floral repressor. Therefore, under LDs, one role of the systemic FT signal is to stimulate the production of GAs in the shoot which facilitates the floral transition. GAs also contribute to maintain their own production through feed-forward regulation that leads to the downregulation of *SVP* (Li et al., 2008). SVP is a central regulatory hub for several GA-related metabolic genes. This emerges from genome-wide studies employing chromatin immuno-precipitation followed by DNA sequencing (ChIPseq). Besides repressing *GA20OX2* (albeit indirectly), SVP regulates the expression of a network of GA metabolic and catabolic genes in association with FLC (Mateos et al., 2015). Among the major direct targets of the FLC/SVP complex are different *GA2OX* genes, which are GA catabolic enzymes. FLC/SVP also negatively regulates *TEM1* and positively regulates *TEM2*, encoding repressors of *GA3OX1* and *2*. Thus, the SVP/FLC complex regulates the GA homeostasis in the SAM (and probably in other tissues) by activating different sets of GA metabolic enzymes.

Modulation of GAs levels in the SAM - either through import or *de novo* local biosynthesis – affects the accumulation of DELLAs which orchestrate different pathways that collectively contribute to the switch to flowering. GAs, through a DELLA-dependent mechanism, activate the expression of *microRNA159* (*miR159*), which targets *MYB33* (also referred to as *GAMYB*), a direct activator of the floral meristem identity gene *LEAFY* (Achard et al., 2004; Blazquez et al., 1998; Blazquez and Weigel, 2000; Gocal et al., 2001). GAs also positively regulate the expression of an important integrator of flowering in the SAM, the MADS box genes *SOC1*, independent of the *miR159*/*MYB33* pathway (Achard et al., 2004; Moon et al., 2003). *SOC1* is also an important activator of *LFY* (Lee et al., 2000; Lee and Lee, 2010). Thus, GAs positively regulate *LFY* expression through *SOC1*, and at the same time, through an auto regulatory feedback loop, reducing *LFY* accumulation through the activation of *miR159*. There is a complex genetic interaction between GAs and *SOC1*. *SOC1* acts downstream of the GA pathway (Hou et al., 2014; Moon et al., 2003; Richter et al., 2013). However, *SOC1* levels are also positively regulated by the SPL factors, which are in turn negatively regulated by DELLA (Yu et al., 2012). On the other hand *SOC1* activates the expression of several *SPLs* in the SAM during the floral transition under LDs, which may provide an auto-regulatory feed-back loop (Jung et al., 2012; Torti et al., 2012).

In addition to GAs, under non-inductive SD conditions flowering is promoted by the age pathway, driven by *microRNA156* (*miR156*), which targets the *SPL* transcriptional regulators. The *miR156-SPL* module is evolutionarily conserved and active under all photoperiodic conditions (Huijser and Schmid, 2011; Wang, 2014). Its activation depends on an age-dependent decrease in *miR156* levels which results in an increase in *SPL* accumulation. *SPLs* have different targets in the leaf and in the SAM, including *miR172* (targeting *AP2-like* floral repressors, previously discussed), several MADS box genes (e.g. *SOC1, AP1* and *FUL*), and *LFY* (Wang et al., 2009b; Wu et al., 2009; Yamaguchi et al., 2009). The gradual decrease of *miR156* is required to enable GA-dependent responses. Plant over-expressing *miR156* (and therefore with reduced *SPL* accumulation) are extremely late flowering under SDs and this phenotype can only be marginally corrected by exogenous GA applications (Hyun et al., 2016; Yu et al., 2012). Thus, degradation of DELLA (as a result of GA applications) is insufficient to activate flowering in the absence of *SPLs*, suggesting a genetic interaction between *DELLA* and the *SPLs*. There is no evidence that the *SPLs* negatively affect GA accumulation in the SAM, or promote *DELLA* stabilization that may account for the late flowering of *miR156* (Yu et al., 2012). In contrast, DELLA affects the function of *SPLs* at two levels, transcriptional and post-transcriptional. At the transcriptional level, DELLA impairs the transcriptional activation of different *SPL* genes at the shoot apex (Galvão et al., 2012; Porri et al., 2012). The role of DELLA in negatively regulating the *SPL* genes is antagonized by the chromatin remodeler PICKLE (PKL) protein which acts as a global positive regulator of GA transcriptional responses (Park et al., 2017). DELLA opposes PKL function by direct binding, thus providing a molecular link between histone modifications at GA regulated transcriptional responses (Zhang et al., 2014). At the post-transcriptional level, as previously described, DELLA proteins physically interact with the SPLs and prevent their transactivation activity (Hyun et al., 2016; Yu et al., 2012). Several lines of evidence support the physiological relevance of the DELLA-SPL interaction in the shoot. First SPLs and DELLA regulate the floral transition in an opposite manner by acting on common downstream targets, including *FUL* and *SOC1* (Hyun et al., 2016; Yu et al., 2012). Second, the expression of a GA resistant *ΔDELLA* form can suppress the early flowering phenotype conferred by a constitutively active allele of *SPL9* (i.e. resistant to the *miR156*-dependent degradation) (Yu et al., 2012). Thus, in the SAM, DELLA impairs the activation of floral genes by interfering with the function of the *SPLs* (Figs. 2 and 3).

The phenotypic consequences of the SPLs-DELLA interaction are most evident under SDs, although they also contribute to flowering under LDs (Hyun et al., 2016; Schwab et al., 2005; Xu et al., 2016b; Yu et al., 2012). Recent data indicate that the *SPL15* is the key target of DELLA under SDs, since mutants of *spl15* show an extreme late flowering phenotype under SDs, similar to GA deficient mutants (Hyun et al., 2016). However, other observations indicate that the role of *SPL15* in flowering under SDs is not unique, and highly redundant with other *SPLs* (Xu et al., 2016b). *FUL*, an important floral integrator is among the direct targets of SPL15 in the SAM. Interestingly, DELLA is enriched at nucleotide positions occupied by SPL15 at the *FUL* promoter, and such enrichment is SPL15 - dependent. This suggests that SPL15 tethers DELLA to specific DNA sites and at these positions DELLA impairs the ability of SPL15 to activate transcription. In the presence of GAs, SOC1 proteins cooperatively interact with SPL15 to induce *FUL* expression, and that of other genes that orchestrate flowering in the SAM (Fig. 2). There appears to be a division of labor between SPL15 and SOC1 at the *FUL* promoter whereby each of these protein is responsible to independently recruit additional chromatin remodeling protein complexes to activate gene expression (Hyun et al., 2016). In a similar fashion, the SPL15/SOC1 module directly activates the expression of *miR172* at the shoot apex. As previously discussed, *miR172* targets the *AP2-like* floral repressors. The key role of GAs is thus to remove the DELLA-imposed block on the SPL factors which promotes reproductive competence to the SAM (Hyun et al., 2016).

Noticeably, when bound to SPL9, DELLA activates transcription at the *AP1* promoter in the floral meristem (Yamaguchi et al., 2014). Therefore, depending on the DELLA-SPL species and the regulatory DNA context, GAs exert different effects on the expression of the floral meristem identity genes.

4. Connections between GA and other hormonal pathways

A general theme emerging from the study of DELLA proteins is that GAs regulate flowering indirectly, often playing a permissive role on other signaling cascades, including hormones. Such an interplay between DELLA and various hormonal pathways is very well described especially during the control of cell growth and differentiation (Davière and Achard, 2016). In the context of the regulation of flowering time, the molecular targets responsible for the cross-talk between the GA/DELLA module and hormones jasmonate (JA), brassinosteroids (BR) and ethylene (ET) are just beginning to emerge. For other hormones (namely abscisic acid, ABA, citokinins, CK, nitric oxide, NO and salicylic acid, SA), which participate in the control of the floral transition, there are still little indications as to their molecular link with the DELLAs. With this in mind, I will describe recent advances on the role of different hormonal pathways in flowering, highlighting their possible connection with GAs (Fig. 3).

4.1. JA and the transition to flowering

JA is a fatty acid-derived molecule that orchestrates different plant-environment responses (mostly related to pathogen defense), as well as endogenous developmental processes (Browse, 2009; Stintzi and Browse, 2000). Central to JA signaling are the JASMONATE-ZIM domain (JAZ) family of transcriptional repressors that are targeted by the F-box protein CORONATINE-INSENSITIVE PROTEIN 1 (COI1) for degradation (Chini et al., 2007; Thines et al., 2007). JA acts as a molecular glue that brings these two proteins in contact. The function of JAZ proteins is to prevent the activity of TFs, including the bHLH-containing MYC2 protein, that orchestrate JA responses. Thus, by removing JAZ proteins, JA initiates the transcriptional reprogramming of the cell and the activation (de repression) of JA responses. Mutants of *coi1* are early flowering under both LDs and SDs, indicating that COI1-dependent signaling pathway delays flowering of *Arabidopsis* (Robson et al., 2010; Zhai et al., 2015). The genetic manipulation of JAZ signaling by overexpression of a non-degradable form of JAZ also leads to early flowering, supporting the role of the canonical JA signaling cascade in flowering (Zhai et al., 2015). Genetic and molecular data indicate that JAZ proteins positively regulate the expression of *FT*. The mechanism involved appears to be indirect, as a subset of JAZ proteins can interact with the AP2-like floral repressors TOE1 and 2, binding to the AP2 domain responsible for their interaction with the DNA (Zhai et al., 2015). Thus, one role of JA may be to modulate the accessibility of TOE1 and 2 proteins to the *FT* promoter, through degradation of JAZ repressors. JAZ proteins also link JA signaling to GAs (Hou et al., 2010). DELLAs interact with JAZs and reduce their inhibitory function on their key target MYC2. Although *myc2* mutants do not display flowering defects, it would be expected that, as a result of the sequestration of JAZ, DELLAs indirectly enhance the activity of TOE1 and 2. In addition, by down regulating *miR172*, DELLA also promotes the accumulation of TOE1 and 2 (Yu et al., 2012). Thus, as discussed earlier, the degradation of DELLA by GAs disengages multiple layers of repression at the *FT* promoter (Fig. 3). While the expression of several JA biosynthetic enzymes largely coincide with the site of accumulation of the *FT* transcript, no flowering phenotype is observed in mutants with disrupted expression of the JA biosynthetic gene ALLENE OXIDASE SYNTHESIS (AOS) (Chauvin et al., 2016; Zhai et al., 2015). It is therefore unclear what signal stimulates the COI1-JAZ module to repress flowering, and whether is related to JA or other fatty acid-derived molecules.

4.2. BRs and the floral transition

Mutants affected in BR biosynthesis or signaling are late flowering, suggesting a positive role for BRs in floral activation (Domagalska et al., 2007; Li et al., 2010). Interestingly, the late flowering phenotype of BRs defective mutants is dramatically enhanced in *Arabidopsis* backgrounds characterized by elevated expression of *FLC* (e.g. the autonomous pathway mutants). *FLC* levels are strongly increased in these double mutant plants, which could be related to increased levels of histone H3 acetylation at the *FLC* locus (which marks actively transcribed chromatin). These molecular studies indicate a role for BRs in maintaining a silenced epigenetic state at the promoter of *FLC*, thus contributing to its downregulation (Domagalska et al., 2007). The study of the GAs - BRs crosstalk provides additional clues about the mode of BR-induced flowering. First of all, GAs and BRs act synergistically in flowering, since augmenting endogenous BRs levels strongly enhances the early flowering phenotype conferred by the overexpression of *GA20OX1*, a rate limiting GA biosynthetic gene (Domagalska et al., 2010). GA applications also rescue the late flowering phenotype of BRs-insensitive mutants, indicating that at least some aspects of the BRs-dependent activation of flowering are dependent on GA availability (Unterholzner et al., 2015). Molecular studies have shown that DELLA negatively regulates BRs signaling through sequestering BRASSINAZOLE RESISTANT 1 (BZR1) (and related proteins), a class of bZIP transcription factors mediating BRs signaling (Bai et al., 2012; Gallego-Bartolomé et al., 2012; Li et al., 2012). BRs promote BZR1 activity in two ways; by phosphorylation and, indirectly, by stimulating GA production, through the transcriptional activation of GA biosynthetic genes (Unterholzner et al., 2015). Once released from DELLA, BR-activated BZR1 binds to DNA to elicit BR-dependent responses. Precisely how BZR1 activates the flowering process is still poorly understood. Some indications arise from the finding that the BZR1-related protein BRI1-EMS-SUPRESSOR 1 (BES1) can recruit two JmjN/C domain-containing proteins, EARLY FLOWERING 6 (ELF6) and RELATIVE OF EARLY FLOWERING 6 (REF6), to regulate target gene expression (Yu et al., 2008). ELF6 and REF6 regulate histone modifications and control flowering time at different levels; ELF6 is a repressor of *FT* whereas REF6 acts as a repressor of *FLC* (Jeong et al., 2009; Noh et al., 2004). While the link between BRs and *FT* regulation awaits confirmation, the BZR1/BES factors may control gene expression by guiding chromatin remodeling complexes at specific loci (Fig. 3).

4.3. ABA and the floral transition

The phytohormone ABA is generally regarded as drought stress-related hormone, coordinating several adaptive responses as a result of water deprivation (Shinozaki and Yamaguchi-Shinozaki, 2007). However, ABA clearly plays important roles in development, even in the absence of stress (Barrero et al., 2005; Liu et al., 2016). Three signaling components constitute the core ABA signaling pathway; these are the PYRABACTIN RESISTANCE (PYR)/ REGULATORY COMPONENT OF ABA RECEPTOR (RCAR), the PROTEIN PHOSPHATASE 2Cs (PP2Cs), and SNF1-RELATED PROTEIN KINASE 2s (SnRK2s) (Cutler et al., 2010). ABA is recognized by the PYR/PYL/RCAR receptor proteins. Binding of ABA stimulates the interaction of PYR/PYL/RCARs with group A PP2C protein phosphatases and consequent release of the SnRK2 protein kinases. In this model the PP2Cs and the SnRK2s act as negative and positive regulators of ABA signaling, respectively (Ma et al., 2009; Park et al., 2009). SnRK2s subsequently activate different substrates, including a complex network of TFs to coordinate ABA responses (Furihata et al., 2006; Umezawa et al., 2013; Wang et al., 2013a, 2013b; Yoshida et al., 2014).

The contribution of ABA signaling in the floral transition is still controversial, as both positive and negative roles of ABA have been reported (Conti et al., 2014a; Domagalska et al., 2010). ABA is emerging as a positive regulator of flowering under LDs, via activation of *FT* and *TSF* genes under LDs (Riboni et al., 2013, 2016). In support of this idea, mutants of *ABA1* or *ABA2*, defective in different enzymatic steps in ABA production, are late flowering under LDs, but present no flowering defects under SDs (Riboni et al., 2016, 2013). The phloem companion cells are the source of ABA production, overlapping with site of *FT* transcriptional activation (Kuromori et al., 2014). Other indications point to a role for ABA in controlling *FT* activation via an interaction with the photoperiodic pathway. The genetic manipulation of the ABA signaling cascade causes changes in *FT* accumulation at dusk, when *FT* levels increase in response to light-stabilized CO protein (Riboni et al., 2016). From a temporal perspective, ABA production is subject to a circadian regulation, with a peak occurring in the middle of the day in a 12 h photoperiod (Lee et al., 2006). The ABA responsive genes follow different patterns of diel accumulation, not necessarily coinciding with the peak of ABA accumulation (Covington et al., 2008; Seung et al., 2012). Therefore, the effects of ABA signaling extend beyond the peak of ABA accumulation to activate the florigen genes.

Mutants deficient in ABA production do not display diminished *CO* transcript accumulation suggesting that ABA affects *FT* expression mainly downstream of the transcriptional activation of *CO* (Riboni et al., 2016, 2014). Other reports based on the study of ABA signaling mutants also support a positive role for ABA in flowering, upstream of the transcriptional activation of *CO* (Koops et al., 2011; Riboni et al., 2016; Yoshida et al., 2014). This discrepancy could be due to the fact even severe ABA biosynthetic mutants still produce detectable amounts of ABA (20–30% compared with the wild type), which might be sufficient to drive transcriptional events upstream of *CO* (Léon-Kloosterziel et al., 1996). ABA signaling may thus promote the transcriptional activation of *CO* as well as its function. Some molecular details about the underlying mechanisms are beginning to emerge. Prime candidates involved in the ABA-mediated transcriptional activation of *CO* are a class of bZIP transcriptional regulators collectively known as ABRE-binding (AREB) proteins or ABRE-binding factors (ABFs) (Choi et al., 2000; Uno et al., 2000). ABA activates the ABFs transcriptionally and post-transcriptionally, via phosphorylation (Fujii et al., 2007; Fujita et al., 2009; Wang et al., 2013a). Mutants of *areb2 abf3 abf1* are late flowering compared with the wild type, supporting a role for these bZIP factors in the floral network (Yoshida et al., 2014). The transcript levels of *CO* are reduced in the *areb1 areb2 abf3 abf1* mutants, which may account for their late flowering. This could depend on reduced accumulation of the *FLOWERING BHLH 3* (*FBH3*) transcription factors, an upstream regulator of *CO*, in *areb areb2 abf3 abf1* mutants compared with the wild-type (Ito et al., 2012; Yoshida et al., 2014). However, adding further complexity to this model, similarly reduced levels of *FBH3* and *CO* are observed in mutants deficient in ABA-dependent phosphorylation, which display an extreme early flowering phenotype (Wang et al., 2009a; Yoshida et al., 2014). Thus, the precise role of the *ABFs* upstream of *CO* warrants further investigation.

ABA signaling also affects CO protein function or signaling (Riboni et al., 2016). Genetic and physiological data indicate that both *GI* and *CO* are required to mediate ABA-dependent signals upstream of *FT* under conditions that favor ABA accumulation. Although the underlying mechanism has no yet been elucidated, one can speculate that both GI and ABA may synergistically activate an additional component which is necessary to enhance the function of CO (Riboni et al., 2016). One potential ABA-dependent modulator of CO activity has been described, but its connection with GI and/or distribution in adult leaves is unknown. The ABA-related transcription factor *ABSCISIC ACID-INSENSITIVE 3* (*ABI3*) acts as a negative regulator of the floral transition, and may affect the accumulation of the florigen genes by impairing the function of CO through binding to its CCT domain (Kurup et al., 2001; Zhang et al., 2009). It is expected that once bound to ABI3, CO is no longer available for binding to DNA (Tiwari et al., 2010). ABA negatively regulates ABI3 by triggering its ubiquitination and subsequent proteasome-dependent degradation (Zhang et al., 2009). These data suggest that ABA might facilitate *FT* upregulation by CO, in part through ABI3 degradation. In summary, these observations support a role for ABA upstream of the florigen genes, and that ABA can have both transcriptional and post-transcriptional effects. Interestingly, the role of ABA in the leaf is parallel and/or synergic to GAs but it is unknown whether these two hormones converge to regulate a common component during the activation of *FT*.

Since ABA levels are usually related to variations in water availability, the different mechanisms discussed above further underlie the remarkable plasticity of *FT* expression under different environmental conditions. On the other hand, ABA is also involved in regulating flowering downstream of *FT*, but in a negative manner. Under non-inductive photoperiodic conditions, mutants with activated or impaired ABA signaling display late and early flowering phenotypes, respectively (Chandler et al., 2000; Riboni et al., 2016, 2013; Wang et al., 2013a, 2013b). These phenotypes may probably derive from a distinct mode of action of ABA in the SAM. Genetic evidences indicate that the negative role of ABA in flowering is exerted through *SOC1* (Riboni et al., 2016). Recent works offer some molecular insights into this negative role of ABA in flowering by showing that ABA directly activates *FLC* through the bZIP transcriptional factor *ABSCISIC ACID-INSENSITIVE 5* (*ABI5*) and the AP2/ERF domain-containing transcription factor *ABSCISIC ACID-INSENSITIVE 4* (*ABI4*) (Shu et al., 2016; Wang et al., 2013b). Thus, by activating *FLC* ABA might cause reduction in *SOC1* levels, causing a delay the floral transition. Because *ABI5* does not appear to contribute to flowering under SDs (Shu et al., 2016; Wang et al., 2013b), *ABI4* and perhaps other ABA-related mechanisms might be responsible for the regulation of *FLC* and *SOC1* under these conditions (Shu et al., 2016; Wang et al., 2013b). There are clearly other routes of ABA regulation on *SOC1*, as in some cases ABA promotes *SOC1* by inducing nuclear re-localization of the OXS2-type Zinc Finger transcription factors (Blanvillain et al., 2011). Furthermore, because *SOC1* is also positively targeted by GAs, ABA and GAs appear to have opposing roles in flowering, by differentially regulating *SOC1* expression and/or signaling. Recent reports describe a regulatory mechanism between ABA and GA in the context of seed germination. DELLA proteins form a protein complex with ABI3 and ABI5 which binds the promoter and activates the transcription of target genes (Lim et al., 2013). It is unknown whether this circuitry also operates in other tissues (e.g. the SAM), and contributes to the regulation of *SOC1* through the activation of *FLC*. It is also unknown whether other ABA-related bZIP might be involved (Fig. 3). A comprehensive understanding of the spatial and temporal interplay between the positive and negative roles of ABA in flowering is still lacking. Delineating a more precise pattern of ABA accumulation (and its related signaling components) in the SAM is an important goal if we are to understand the role of ABA in flowering and its interaction with other hormones.

4.4. Ethylene and flowering

In addition to ABA, other hormonal pathways enable plants to adapt their life-cycle appropriately with fluctuating environmental conditions. One such example is ethylene, which acts as floral repressor in *Arabidopsis* and is highly induced by salt stress, which delays flowering (Achard et al., 2006). Application of ethylene or mutant plants with constitutively-activated ethylene signaling are late flowering under LDs and, most dramatically, under SDs (Achard et al., 2007). The *ETHYLENE INSENSITIVE 3* (*EIN3*) and *EIN3-like* (*EIL*) transcription factors mediate ethylene transcriptional responses. These proteins are normally subject to continuous degradation by the ubiquitin/proteasome system, unless the ethylene signaling cascade

is activated (Guo and Ecker, 2003; Potuschak et al., 2003). Consistent with the negative role of ethylene being dependent on EIN3 function, mutants that confer EIN3 stabilization delay the floral initiation in SDs. Furthermore, EIN3 accumulation delays flowering by activating the *ETHYLENE RESPONSE 1* (*ERF1*) -related genes, belonging to the APETALA2 (AP2)/ethylene responsive element binding proteins family. The negative role of ethylene in flowering (through the EIN3-ERF1 axis) is broadly attributed to reduced bioactive GA levels, causing enhanced accumulation of DELLAs (Achard et al., 2007; Vriezen et al., 2004). Consistent with the idea that ethylene delays flowering by promoting the stabilization of DELLA, the late flowering of constitutive ethylene response mutants can be partly rescued by loss-of-function mutations in genes encoding the DELLAs (Achard et al., 2007). Interestingly, DELLA proteins inhibit ethylene signaling by binding EIN3 and various ERFs to prevent their binding to the DNA (An et al., 2012; Marín-de la Rosa et al., 2014). These physical interactions may confer an auto regulatory feedback mechanism to avoid over-accumulation of DELLA under adverse stress conditions.

4.5. The role of NO, SA and CKs in flowering

The role of NO, SA, and CKs in flowering is well documented but knowledge about their mode of integration with the floral network is currently very limited. Pathogen and stress-related hormones NO and SA have contrasting effects on flowering, with NO repressing flowering, and SA activating it (He et al., 2004; Martínez et al., 2004). NO exerts its negative role on flowering by targeting multiple floral mechanisms, impairing the activation of *CO* and at the same promoting *FLC* accumulation (He et al., 2004). In contrast, the levels of *FT* are increased following SA application, which is indicative of an integration of SA-dependent signals in the photoperiodic pathway. Genetic data indicate that to activate flowering, SA requires GI function but not CO under LDs. An additional component required for the SA-dependent activation of *FT* is the *PATHOGEN AND CIRCADIAN CONTROLLED 1* (*PCC1*) gene (Segarra et al., 2010). Physiological and molecular data place the function of *PCC1* downstream of *GI* and in parallel with *CO* in the cascade of events leading to *FT* activation. SA also activates flowering under SDs, but very little is known about its target (Martínez et al., 2004; Villajuana-Bonequi et al., 2014).

The application of CKs under SDs promotes flowering through the activation of *TSF* but not *FT*. Besides *TSF* also the *FD* and *SOC1* functions are required to for the CKs-mediated flowering (D'Aloia et al., 2011). Thus, a possible model emerges whereby CKs stimulates *TSF* expression, independent of *CO* or *GI*. Following its translocation in the SAM TSF binds to FD to induce a floral reprogram, possibly through activation of *SOC1*. Cytokinin responses are mediated by type-B ARABIDOPSIS RESPONSE REGULATOR (ARR) factors (Sakai et al., 2001). These proteins can bind to DELLA, but unlike the previous examples this interaction causes the re-localization of DELLAs to the target promoters, which leads to the activation of target genes (Marín-de la Rosa et al., 2015). Whether DELLAs participate as transcriptional co-activators in the CKs-mediated flowering is an interesting future question.

4.6. Concluding remarks

There is an extensive cross-talk amongst different hormonal pathways to modulate growth and differentiation processes, which might confer increased developmental flexibility to plants in an ever-changing environment (Depuydt and Hardtke, 2011). The evidence reviewed here also point to a general contribution of hormonal signals to modulate flowering. Hormonal signaling cascades affect the transcription of floral integrators, acting in the leaf or in the SAM (Fig. 3). However, gaps remain in our understanding of the regulatory logic of different hormonal pathways, their precise distribution in the different cell types and their temporal dynamics in flowering time. With respect to the regulation of flowering time, the role of DELLA as modulator of the photoperiodic and age pathway is now well-established. The available data also point to cross-regulatory mechanisms between hormonal pathways often mediated by DELLA proteins which act as keystones for the assembly of diverse protein complexes. In this sense, DELLA may help bridge together hormonal and floral signals upon floral integrators (Fig. 3). Adding further complexity to this integrative role for DELLAs, recent reports describe multiple post-translational modifications (PTMs) which confer different binding properties to DELLA proteins (Conti et al., 2014b; Zentella et al., 2016, 2017). Two related proteins, SPINDLY (SPY) and SECRET AGENT (SEC), regulate DELLA in an opposite manner, by competing for the attachment of monofucose and O-GlcNAc monosaccharide moieties, respectively (Zentella et al., 2017, 2016). These modifications alter the binding affinity between RGA and its interacting transcription factors PIF4 and BZR1 and possibly many others. Since the flowering phenotype of *spy* and *sec* mutants is opposite (early and late flowering, respectively) variations in the PTMs state of DELLA may similarly alter DELLA protein-protein interaction networks required for the regulation of flowering time (Jacobsen and Olszewski, 1993; Zentella et al., 2016). More work is needed to resolve the dynamics of these PTMs, their interdependence and/or whether they affect different pools of DELLA proteins. Nevertheless, PTMs clearly add a new dimension to GA signaling beyond the DELLA degradation-dependent mode of regulation.

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