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REVIEW

The Hormonal Regulation of Flower Development

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Abstract Homeotic genes comprising the ABCE classes partly detail the genetic networks that control aspects of floral organ initiation, development, and architecture, but less is known about how these gene functions are translated into changes at the cellular level in growth and cellular differentiation that are involved in the formation of diverse floral organs with specific shapes and sizes. Hormones are the principal transducers of genetic information, and due to recent advances in understanding hormone function in floral development, it is timely to review some of these findings. Flower development is the result of a regulated balance between meristem size and coordination and organ initiation. Floral meristem size is regulated by cytokinin, gibberellin, and auxin, and auxin plays a major role in organ initiation and organogenesis. How hormones contribute to the development of each organ is partly known, with stamen development reliant on almost all hormones, petal development is affected by gibberellins, auxin, and jasmonic acid, and gynoecium development is predominantly regulated by auxin. Furthermore, the interconnections between genetic hierarchies and hormones are being elucidated, and as almost all hormone groups are implicated in floral development, points of hormone crosstalk are being revealed.

Keywords Flower development · Auxin · Floral meristem · Gibberellins · Inflorescence meristem · Genetic hierarchy

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Introduction

A huge variation in floral morphology exists among eudicots and between eudicots and monocots. *Arabidopsis* has become a model for flower development due to its stereotypical floral ontogeny, with four sepals, four petals, usually six stamens comprised of four medial and two lateral, and a gynoecium consisting of two fused carpels, in concentric whorls, with organs in each whorl being initiated sequentially from outer to inner whorls. Flower production is the result of sequential events involving different genetic networks (Jack 2004) (Fig. 1). First, floral induction, the phase change from vegetative to reproductive growth, is effected by an integrated network of flowering-time genes in response to the environment and endogenous factors. These genes converge on the activation of meristem identity genes which confer floral meristem (FM) identity, before floral organs are initiated by the coordinated spatial and temporal expression of transcription factors (Fig. 1). Finally, a raft of downstream target genes of the floral identity genes coordinate floral organogenesis.

Among these four hierarchic levels, genetic networks have been well established, for example, for flowering time (Mouradov and others 2002) and FM identity genes (see Krizek and Fletcher 2005). Work with floral homeotic mutations in *Antirrhinum* and *Arabidopsis* has led to the ABCE model (Bowman and others 1991; Coen and Meyerowitz 1991; Theissen 2001), a field model whereby the overlapping expression domains of different classes of transcription factors combinatorially specify organ identity in each whorl. Despite the diversity in eudicot and monocot floral morphology, there is evidence for conservation of homeotic genes, such as *AP3* in monocots (Whipple and others 2004) and in gymnosperms (Ambrose and others

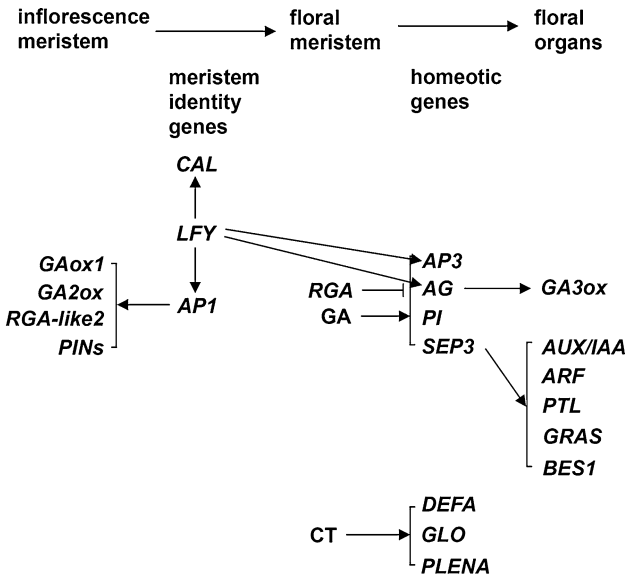

Fig. 1 Scheme showing the phase changes and sequential genetic events leading to flower formation and the different meristem identity and floral homeotic genes that are known to be involved in hormone pathways

2000), and the ABCE model has been extended for monocot flowers (Kanno and others 2003). As leaves can be converted into petals by the combined constitutive overexpression of *SEPELLATA3* (*SEP3*) with A and B class genes (Pelaz and others 2001), and floral organs can be converted into leaf-like structures by loss of *sep1 sep2 sep3 sep4* function (Ditta and others 2004), this suggests floral organs and leaves share a core developmental program probably from an ancestral organ (Sablowski 2010), a concept that might aid the identification of conserved key modules of development. As a further example, components of the *AGAMOUS* (*AG*) *SEPALLATA* (*SEP*) *APETALA3* (*AP3*) *PISTILLATA* (*PI*) complex can regulate genes involved in vegetative growth (Sablowski 2010), supporting the hypothesis that proteins involved in floral identity modulate the function of general developmental regulators.

How the genetic regulators and hierarchies of floral development translate developmentally by integrating into physiological and hormone pathways has been addressed for floral evocation (Bernier and Périlleux 2005; Davis 2009), but has only recently been addressed for floral organ development. Much data from physiological experiments on the role of hormones in the development of specific floral organs have also accumulated, and current goals are to synthesize this information and also to marry the role of hormones with the known genetic hierarchies in floral organ initiation and development. Therefore, a review of this area is appropriate.

Linking Meristem Identity and Floral Organ Identity Genes to Hormone Pathways

The link between FM or homeotic genes and hormone pathways has been addressed recently by studies investigating targets of *AG* (Gómez-Mena and others 2005), *SEP3* (Kaufmann and others 2009), and *APETALA1* (*AP1*) (Kaufmann and others 2010). Several lines of evidence support a link between these genes and gibberellins (GAs). It has long been known that GA regulates the transcriptional activity of the FM identity gene *LEAFY* (*LFY*) (Blázquez and others 1998) (Fig. 1), which in turn controls the floral homeotic genes *AP1* (Wagner and others 1999), *AG* (Busch and others 1999), *AP3* (Lamb and others 2002), and *CALUIFLOWER* (*CAL*) (William and others 2004). The transcription of *PI*, as well as of *AP3*, *PI*, and *AG*, is also positively regulated by GA in young *ga-1* mutant flowers after floral organ initiation and is repressed by the DELLA protein RGA (Yu and others 2004) (Fig. 1). AG also activates *GA3ox*, which encodes a key gibberellin biosynthesis enzyme (Gómez-Mena and others 2005) (Fig. 1), and overexpression of *AG* can partially rescue the floral phenotype of *ga-1* (Yu and others 2004). This GA feedback loop involving homeotic genes suggests a link to GA-regulated cell division and growth in the shaping of floral organs.

Induction of *AP1* activity in an *ap1 cal* mutant background to identify early floral-induced genes led to an upregulation of genes involved in GA homeostasis, including GA synthesis by *GA3ox* and degradation via *GA2ox* (Wellmer and others 2006; Kaufmann and others 2010) (Fig. 1), suggesting that feedback regulation of GA levels is important in the FM. In addition, four members of the *PIN* family were upregulated, indicating that polar auxin redistribution is important for floral organ induction, and *RGA-LIKE2*, an inhibitor of GA response, was repressed (Kaufmann and others 2010) (Fig. 1).

SEP3 targets represent multiple links to auxin signaling pathways, including an overrepresentation of transcription factors such as *PETAL LOSS* (*PLT*), which is involved in auxin homeostasis, or AUX-IAA and ARF family members, in addition to those in BES1 and GRAS families (Kaufmann and others 2009) (Fig. 1), implicating brassinosteroid and GA interactions. Dominant-negative repression of *SEP3* function, in addition to giving homeotic phenotypes, leads to phenocopies of *pin1*, *pid*, or *ettin* mutants, suggesting that *SEP* might regulate auxin pathways by interacting with various ARF proteins (Kaufmann and others 2009). These gene target studies are beginning to elucidate how FM and homeotic genes regulate hormone functions.

Cytokinin (CT) can also affect the activity of floral homeotic genes. For example, in tobacco, local increases in

CT concentration via *IPT* activity resulted in a decrease in transcripts of the *Antirrhinum* floral homeotic genes *DEFICIENS* (*DEFA*), *GLOBOSA* (*GLO*), and *PLENA* (Estruch and others 1993) (Fig. 1). Furthermore, application of benzylaminopurine to *Arabidopsis* flowers at different developmental stages resulted in an increase in the number of all floral organs and homeotic organ transformations and secondary floral buds within flowers, phenocopying known floral homeotic mutants (Venglat and Sawhney 1996). This supports the hypothesis that CTs regulate the activity of meristem and floral organ identity genes, either directly or as a result of an alteration in the balance of CT/auxin ratios. More recently, an approach to identify further gene targets of CT-regulated flower development expressed the *Arabidopsis IPT4* gene under control of the *AP1* promoter, and expression profiling showed that *CUC2* and *CUC3* were positively regulated by elevated FM CT levels and that CT signaling via *Arabidopsis* histidine kinase 2 and 3 was responsible for the increase in meristem size, number of flowers, and floral organs observed (Li and others 2010).

Meristem Development

Floral organogenesis is dependent on the activity of the inflorescence meristem (IM) in generating lateral organs, that is, flowers, and subsequently on the FM to provide a stem cell population for cell recruitment for floral organ formation. FM size is dependent on the size of the stem cell population, partly controlled by the *WUSCHEL* (*WUS*)/*CLAVATA* (*CLV*) feedback loop, which regulates organ number, with *clv* mutants having more organs (Clark and others 1993, 1995) and *wus* mutants having fewer organs due to premature meristem termination (Laux and others 1996). Several reviews have focused on the role of hormones in regulating meristem size and function (Hay and others 2004; Dodsworth 2009; Galinha and others 2009; Veit 2009), which will not be reviewed here. The major hormone determinants of meristem size are GAs, CT, and auxin. Briefly, low GA levels in meristems are maintained by *KNOX* genes (Hay and others 2002), and CTs positively regulate *Arabidopsis* shoot apical meristem (SAM) size (Li and others 2010) via feedback pathways between *WUS* and *CLV* (Lindsay and others 2006; Gordon and others 2009). CT signaling and biosynthesis can also affect meristem size in maize (Giulini and others 2004) and rice (Kurakawa and others 2007). The *WIGGUM/ENHANCED RESPONSE to ABA1* (*ERA1*) gene (Cutler and others 1996; Ziegelhoffer and others 2000) controls FM size, with mutants having a greater number of all floral organs. *WIGGUM/ERA1* encodes a farnesyltransferase that links response to ABA signal transduction to FM size.

The same pathways are thought to regulate the size of the SAM, IM and FM, although differences exist such as the antagonistic effect of CT on root meristem size (Dello Ioio and others 2008) compared with the CT promotion of size in aerial meristems. Here, it is relevant to point out only the unique features of IMs and FMs. The first difference is that FMs produce organs in whorls as opposed to the full range of phyllotactic patterns seen in vegetative and floral lateral organs. In addition, unlike the SAM, FMs show determinacy resulting from an imbalance between organ differentiation and meristem maintenance, and determinacy is vital to floral organ patterning. IMs can be either determinate or "closed," ending in a terminal flower, or indeterminate or "open" (Bull-Hereñu and Claßen-Bockhoff 2010) as for *Arabidopsis*. Determinacy in the FM is achieved by the inactivation of *WUS* transcription in the central FM cells by *AG* (Lenhard and others 2001; Lohmann and others 2001).

Auxin is an Instructive Signal for Lateral Organogenesis from the IM

Lateral organ initiation from the IM must be distinguished from floral organ initiation from the FM, although several common mechanisms affect both processes, including the role of auxin. Few detailed studies have examined the number of lateral organ founder cells necessary for organ initiation, and sector analysis has suggested that flowers arise from only four cells on the flank of the inflorescence apex (Bossinger and Smyth 1996). However, the role of auxin as an instructive signal in lateral organ initiation and positioning is well established; vegetative lateral organ outgrowth from the SAM is initiated at points of transient auxin maxima determined by the polar orientation of the PIN1 auxin efflux protein in tomato and *Arabidopsis* (Reinhardt and others 2000). *pin1* mutants produce no lateral organs (Gälweiler and others 1998), which can be induced from the IM by exogenous auxin application (Reinhardt and others 2003). PINOID (PID) is a serine/threonine protein kinase that directly controls PIN1 function (Kleine-Vehn and others 2009; Zhang and others 2010), and consequently, *pid* mutants also have fewer lateral floral organs than wild type before premature inflorescence termination (Bennett and others 1995). A further mutant with *pin*-type inflorescences is *naked pins in yuc mutant1* (*npy1*), an allele of *enhancer of pinoid 1* (*enp1*) (Treml and others 2005), and *macchi-bou 4* (*mab4*) (Furutani and others 2007) in combination with its redundant relative *npy5* (Cheng and others 2008). *NPY1* is a member of the *NON-PHOTOTROPIC HYPOCOTYL3* (*NPH3*) gene family, has an overlapping expression domain with both *PIN1* and *PID*, and controls its

abundance and activity (Cheng and others 2007; Furutani and others 2007). The phenocopying of *pid* and *pin1* phenotypes by *npy1 npy5* mutants and genetic interactions between *npy1* and *pid* mutants suggest that *NPY1* acts in the same pathway as *PID* to regulate polar auxin transport (Cheng and others 2007).

Mutations in *AUXIN RESPONSE FACTOR 5* (*ARF5*)/*MONOPTEROS* (*MP*) also phenocopy *pin1* in the production of naked inflorescences lacking or almost lacking lateral floral organs (Przemeck and others 1996) (Fig. 2). Expression levels of *PID*, *PIN1*, and *MP* are transcriptionally activated by auxin (Benjamins and others 2001; Peer and others 2004; Wenzel and others 2007), showing that feedback loops within auxin concentration gradients help to maintain directed auxin transport. These various mutants in different aspects of auxin biosynthesis, polar transport, and perception in *Arabidopsis* demonstrate the importance of auxin in lateral floral organ initiation.

Homologous or orthologous loci for *Arabidopsis* auxin synthesis and polar transport have been characterized in maize (McSteen and others 2007; Barazesh and McSteen 2008; McSteen 2010) and rice (Morita and Kyozuka 2007), and their mutation leads to defects in floral architecture/organ initiation, thereby showing a conservation of auxin biosynthesis, transport, and signaling for lateral organ initiation across widely diverging species.

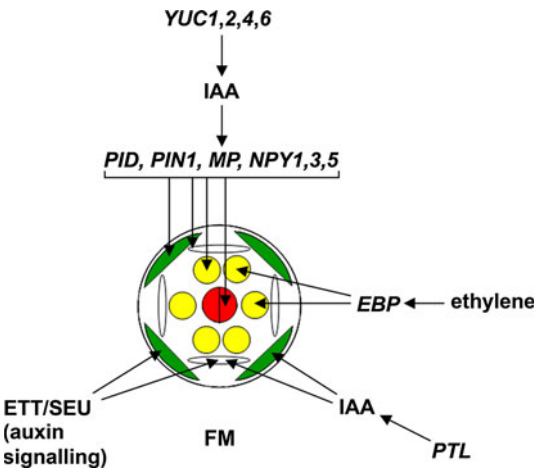

Fig. 2 A schematic diagram showing the effect of hormones on floral organ merosity. A representative *Arabidopsis* floral meristem is shown, with floral organ primordia depicted by symbols in concentric whorls, from outwards to inwards: sepals, petals, stamens, and carpels. Auxin biosynthesis and transport affect merosity of all four organs, and auxin signaling, via *ETT*, regulates perianth organ number. Perianth organ number is also affected by auxin homeostasis via *PTL*. Ethylene controls stamen number via the AP2 transcription factor *EBP*

Floral Organ Initiation and Growth: Making and Shaping Organs

The FM provides a stem cell pool for cell recruitment for organogenesis. Following initiation from the FM, floral organ development is dependent on the coordinated regulation of cell division and cell elongation, but elucidating how the tissue-specific and temporal regulation hormone signals coordinate and integrate transcription factor functions and how hormone networks combine to affect cell proliferation and expansion remains an important goal. Sector analysis in *Arabidopsis* has shown that each floral organ arises from a small, defined number of cells: petals arise from two cells in flower primordia, medial stamens from four, and sepals and carpels from eight (Bossinger and Smyth 1996). Although this sector analysis considered only L1 cells, floral organ initiation in *Arabidopsis* is initiated from division of a similar number of underlying cells, with divisions giving rise to sepals, petals, and stamens occurring in the L2 layer, and those for carpels from the L2 or L3 cells (Crone and Lord 1994).

Flower development consists of the spatial regulation of a considerable number of organ-specific genes. The prediction of organ-specific transcriptomes by comparing global expression profiles for *ap1*, *ap2*, *ap3*, *pi*, and *ag* and wild type has been performed to identify genes expressed predominantly in one organ type (Wellmer and others 2004). The majority of expressed transcripts were found in the reproductive organs instead of the perianth, reflecting the more complex anatomy of tissue and cell types within stamens and carpels and major developmental events such as ovule and pollen formation.

Another aspect of floral organ growth is the coordination and synchronization of organ outgrowth: a complete flower is derived from the integrated growth of different organs. Although organs are initiated more or less sequentially, from outer to inner whorls, in *Arabidopsis*, the medial stamen primordia are more visible at stage 5 than the more or less synchronously induced petal primordia (Smyth and others 1990). In addition, organ outgrowth rates in *Arabidopsis* are variable, with petal elongation occurring rapidly during stage 9 (Smyth and others 1990), and rapid filament elongation during stages 12 and 13 is coordinated with gynoecium maturity to effect fertilization (Smyth and others 1990). The signals that coordinate growth between developing floral organs are poorly understood. The *KLU* gene has recently been shown to be a growth-regulating gene encoding a cytochrome P450 (Eriksson and others 2010). Many cytochrome P450s are catalysts in the synthesis of plant growth regulators such as GAs, JA, and brassinosteroids (BRs) (Chapple 1998) and auxin (Mikkelsen and others 2000; Bak and others 2001), but *KLU* acts independently of these and may regulate a novel

mobile growth-regulating signal that controls organ size and correlates organ size within individual flowers (Eriksson and others 2010).

Final floral organ size is a coproduct of cell proliferation and cell size. There are few genes that *per se* specifically affect organ size without pleiotropically affecting whole-plant architecture (Weiss and others 2005). For example, the *ARGOS* gene positively regulates organ size mainly by cell number, and all aspects of plant architecture and lateral organs are larger than wild type, including leaves and all floral organs (Hu and others 2003). *ARGOS* acts in an auxin signaling pathway and may translate signals downstream of *AXR1* and upstream of *ANT* (Hu and others 2003). *BIGPETAL* (*BPE*) encodes a bHLH transcription factor that regulates petal size via cell expansion (Szécsi and others 2006) and is regulated post-transcriptionally by JA (Brioudes and others 2009), thereby linking JA to the control of floral organ size (Fig. 3b).

The morphological changes wrought by hormone action are partly effected by cell division and cell elongation; CTs and BRs activate cell division in *Arabidopsis* via activation of a D-type cyclin (reviewed in D'Agostino and Kieber 1999; Hu and others 2000). Auxins also promote cell division, for example, in lateral organ formation (Reinhardt and others 2000), and cell elongation by acid growth (Rayle and Cleland 1992) or by stimulating expansins, which loosen cell walls (Hutchison and others 1999), or they can inhibit cell elongation as in root gravitropism (Abas and others 2006). Cell expansion is also induced by BRs (Clouse and Sasse 1998) and GAs, which reorganize cortical microtubules (Inada and Shimmen 2001). The effect of ethylene to inhibit cell elongation often occurs via auxin synthesis and transport, as in *Arabidopsis* roots (Růžička and others 2007).

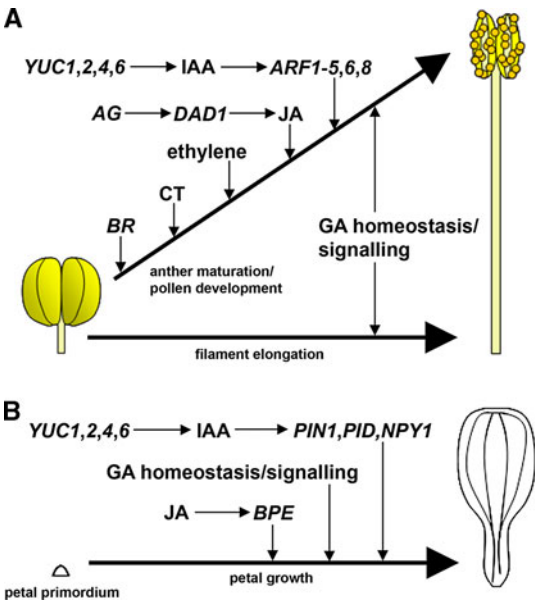

Fig. 3 Gene-hormone interactions known to be involved in anther (**a**) and petal (**b**) development. Brassinosteroids (*BRs*), cytokinin (*CT*), ethylene, jasmonic acid (*JA*), gibberellin (*GA*), and auxin affect anther and pollen development and GA controls filament elongation. Petal development is regulated by auxin and GA homeostasis and by JA via the transcription factor *BIGPETAL*

The Role of Different Hormones in Floral Organ Development

Auxin

Similar to lateral organ initiation, polar auxin transport (PAT) has a role in floral organ initiation from the FM. Weak *pin1* alleles (*pin1-5*) and *pid* produce flowers, but these are defective in organ number, with more or fewer organs produced than wild type (Cheng and others 2008) (Fig. 2), as are the flowers produced by double *npy1 npy5* mutant flowers (Cheng and others 2008). PAT also regulates organ development (Fig. 3b). In addition to correct auxin distribution by polar transport, local auxin biosynthesis is a key regulator of floral organ development. Although several different auxin biosynthesis pathways exist in plants, synthesis via the *YUCCA* (*YUC*) family of flavin monooxygenases is of predominant importance for floral organ initiation and development in *Arabidopsis* (Cheng and others 2006; Cheng and Zhao 2007) (Fig. 2), *Petunia* (*FLOOZY*) (Tobeña-Santamaria and others 2002), and maize (*SPARSE INFLORESCENCE*) (Gallivotti and others 2008), and *YUCCA* genes are also present in rice (Yamamoto and others 2007).

In *Arabidopsis*, *YUC1*, *YUC2*, *YUC4*, and *YUC6* have partly overlapping expression domains and redundant functions in floral development (Cheng and others 2006), with *yuc1yuc4* or *yuc2yuc6* double mutants and triple and quadruple *yuc* mutants showing floral patterning defects in both organ initiation and ontogeny (Fig. 3b). The tissue-specific expression patterns and higher-order mutant phenotypes of *yuc* mutants show that local auxin synthesis is essential for floral organ development. Because specific combinations of *yuc* mutants have organ-specific phenotypes, this shows that auxin from other floral organs cannot compensate for this tissue-specific deficiency, suggesting that different subsets of *YUC* genes precisely regulate auxin gradients in tissues, with minimal auxin redistribution occurring within flowers. The *FLOOZY* gene in *Petunia* acts non-cell autonomously (Tobeña-Santamaria and others 2002), a fact that contributes to the debate concerning the relevance of polar auxin redistribution compared with local *de novo* synthesis. Clearly, gradients within floral organs are maintained and are vital for

organogenesis, for example, in gynoecium development (see section below).

In addition to its biosynthesis (Fig. 3a), auxin response is important for stamen development because mutants in many different *Arabidopsis AUXIN RESPONSE FACTORS* (*ARFs*), which mediate auxin-regulated gene expression, show stamen development defects (Fig. 3a), including *ARF1* (Ellis and others 2005), *ARF2* (Ellis and others 2005; Schruff and others 2006), *ARF3*/*ETTIN* (*ETT*) (Sessions and others 1997), *ARF4* (Pekker and others 2005), *ARF5*/*MONOPTEROS* (*MP*) (Przemeck and others 1996), and *ARF6* and *ARF8* (Nagpal and others 2005). Auxin flow in anther filaments is also necessary for viable pollen development via mitosis (Feng and others 2006), and auxin synthesis in anthers is important for pollen maturation and dehiscence (Cecchetti and others 2008). Auxin signaling via *ETTIN*/*ARF3* also regulates the number of perianth organs (Sessions and others 1997) (Fig. 2).

Mutants for the *PETAL LOSS* (*PTL*) locus, which encodes a trihelix transcription factor, have perianth development defects, including fused sepals and a reduced number or complete absence of petals (Brewer and others 2004). *PETAL LOSS* overexpression disrupts auxin homeostasis in the flower (Li and others 2008), suggesting a role for auxin in perianth initiation and development (Fig. 2).

In addition to an instructive role in organ development, a second role for IAA in flower development is proposed to be the suppression of the growth of neighboring organs. For example, the removal of young stamens results in early petal elongation, suggesting that they exert a negative growth effect via high auxin concentrations (Aloni and others 2006). Auxin may therefore have a role in synchronizing development. Tissue-specific auxin concentration has been inferred in developing floral organs of *Arabidopsis* using the *DR5::GFP* auxin response monitor (Aloni and others 2006). This has shown that auxin accumulates in the tips of each developing floral organ, as in leaves, where free auxin production begins in the tips and spreads basipetally along the margins before appearing in the lamina (Aloni and others 2003), but it is also highly concentrated in young anthers and mature pollen grains whereas petals produce very low levels. In addition to free IAA pools, conjugated auxin is highly and ubiquitously expressed throughout young flower buds (Aloni and others 2006). Five auxin-conjugate hydrolases have been characterized, with overlapping but distinct floral expression domains in *Arabidopsis* (Rampey and others 2004), which potentially can locally produce free IAA from the conjugated pool. This raises the question of to what extent auxin accumulation in organs such as developing stamens and gynoecium is due to PAT, or how much is due to local auxin biosynthesis or conjugation hydrolysis. *SEUSS* (*SEU*) is a modifier of *ETT*/*ARF3* which influences the shape, number, and phyllotaxy of petals (Pfluger and Zambryski 2004) (Fig. 2).

Gibberellin

GAs are essential for the development of stamens (Fig. 3a) and petals (Fig. 3b) (Koornneef and van der Veen 1980), and many mutants affected in GA synthesis have underdeveloped floral organs (Olszewski and others 2002); for example, *gib-1*, a GA-deficient mutant of tomato flower bud development, is developmentally arrested (Jacobsen and Olszewski 1991), and the *Arabidopsis ga1-3* mutant shows reduced elongation of petals, stamens, and pistils (Wilson and others 1992). The role of GAs in regulating fertility, petal growth, and stamen filament elongation via cell elongation is opposed by DELLA protein repression (Cheng and others 2004). The importance of GA signaling in stamen development was demonstrated by a whole-genome microarray analysis to identify genes responsive to the activation of the DELLA protein RGA in a *ga1-3 rgl2 rga* mutant background. More than one third of RGA downregulated genes were specifically or mainly expressed in stamens (Hou and others 2008).

A number of genes, including enzymes at different points in GA biosynthesis, have been characterized, and studies of their floral expression patterns and mutant phenotypes allow inferences to be made concerning important biologically active GA pools during flower development, as little is known about how plants regulate GA levels developmentally. GA synthesis can be separated into early and late stages, with CPS (*ent*-copalyl diphosphate synthase) encoded by *GA1* acting in early synthesis and *GA20-oxidase* and *GA3-oxidase* in late steps. *GA1* expression is high in IMs, early floral primordia, the receptacle, mature anthers, and pollen (Silverstone and others 1997). Of the family of five *Arabidopsis GA20-oxidases*, *GA20ox1* and *GA20ox2* play redundant roles in stamen filament elongation (Fig. 3a) and fertility, and *GA20ox4* and *GA20ox5* are weakly expressed in mature pollen (Rieu and others 2008). *GA3-oxidases* comprise a multigene family in *Arabidopsis* and catalyze the final step of GA biosynthesis. Reporter gene analysis of four *GA3-oxidases* shows that in developing flowers, *GA3ox3* and *GA3ox4* are expressed in developing anthers, with activity reaching a maximum at stages 9–10; *GAox2* is weakly expressed in anthers; and *GAox1* is present in anther filaments and flower receptacles (Hu and others 2008). Therefore, GA catabolism by GA oxidases may regulate tissue-specific pools of active GA in floral development. GA is essential for the development of stamens, and *ga-1-3*, which is blocked in GA biosynthesis, has arrested stamens at floral stage 10, a lack of filament growth, and reduced pollen fertility (Cheng and others

2004), as have *gaox1*, *gaox3*, and *gaox4* higher-order mutants (Hu and others 2008). *De novo* GA synthesis and bioactive GAs synthesized in the stamens and/or flower receptacles and transported to petals are necessary for early stamen development (Hu and others 2008). In rice, microarray analysis has identified a considerable number of GA-regulated genes involved in anther development (Wang and others 2005).

Cytokinins

In addition to the effects of CT on meristem size and homeotic genes, CTs are also required for pollen development (Fig. 3a). CT depletion by overexpression of the CT oxidase gene *CKX1* in maize led to male sterility that could be overcome with exogenous CT application (Huang and others 2003).

Jasmonate

JA is a major regulator of anther development and pollen maturation (Fig. 3a): mutants deficient in JA response are male-sterile (Feys and others 1994), and sterility caused by mutants in JA biosynthesis can be overcome by JA treatment (Stinzi and Browse 2000). A large number of genes positively or negatively regulated by JA comprise a significant stamen-specific JA transcriptome in *Arabidopsis* (Mandaokar and others 2003, 2006). Furthermore, the floral organ identity gene *AG* controls late stamen development via JA biosynthesis by directly regulating *DEFECTIVE IN ANTHER DEHISCENCE1* (*DAD1*), which encodes the JA biosynthetic enzyme (Ito and others 2007). Many JA-regulated genes in rice have been implicated in anther development (Wang and others 2005).

Brassinosteroids

Pollen is rich in BRs (Grove and others 1979) and BRs control anther and pollen development (Fig. 3a), as the BR-related mutants *bri1*, *bin2*, and *cpd* are almost male-infertile due to reduced pollen number, viability, and impaired release (Ye and others 2010).

Ethylene

A possible role for ethylene in stamen initiation has been shown by mutation of *EBP*, an *Arabidopsis* ethylene response factor that produces a subtly reduced number of stamens (Ogawa and others 2007) (Fig. 2). Furthermore, ethylene regulates floral sex determination in cucumber (Kahana and others 1999; Duan and others 2008), with exogenous application or increased endogenous ethylene production causing femaleness by arresting anther development (Fig. 3a).

Focus on Gynoecium Development and Auxin

Study of early fruit development is one way to focus on the role of auxin in flower development. An auxin gradient in the gynoecium primordium has been postulated to control gynoecium ontogeny (Nemhauser and others 2000), with high levels in the tip specifying the style and stigma, medial levels patterning the ovary, and lowest basal levels promoting gynophore differentiation. Support for this model is based partly on high *DR5::GFP/GUS* expression in the gynoecium tip (Aloni and others 2006) and that genetically or chemically altering PAT disrupts the apical–basal pattern of gynoecium organ differentiation (Ståldal and Sundberg 2009) theoretically by altering the threshold boundaries of auxin necessary for each gynoecium tissue type. Furthermore, the mutation of several transcription factors involved in auxin signaling leads to female organ phenotypes, including *ETTIN* (*ARF3*), *SPATULA* (*SPT*), *STYLISH1* (*STY1*), *LEUNIG* (*LUG*), and *SEUSS* (*SEU*). Among pleiotropic phenotypes, *ett* mutants have elongated gynophores and smaller ovaries (Sessions and Zambryski 1995; Sessions and others 1997; Nemhauser and others 2000), and *ETT* functions upstream of *SPT* (Heisler and others 2001) (Fig. 4). *SEUSS* acts together with *ETT* to regulate auxin response genes (Pfluger and Zambryski 2004) (Fig. 4) and *LUG* interacts with *SEU* probably upstream of *STY1* (Kuusk and others 2006) (Fig. 4). *STY1*, which is expressed in the apical part of the gynoecium, binds to the promoter of *YUC4* (Ecklund and others 2010) and activates its transcription (Sohlberg and others 2006) (Fig. 4), and thereby is a candidate for directly regulating high local auxin synthesis in the apical gynoecium. *STY1* overexpression also leads to higher levels of auxin-regulated genes of the GH3 class (Fig. 4), that is, *YDK1* and *GH3.3* (Sohlberg and others 2006), which are involved in negatively regulating auxin homeostasis by catalyzing auxin-amide conjugation. Therefore, the control of gynoecium development by *STY1* has a functional basis via both local auxin synthesis and homeostasis. A link from auxin via *ETT* is provided to AG, which regulates the expression of *GIANT KILLER* (*GK*), which in turn controls *ETT* expression epigenetically (Ng and others 2009) (Fig. 4). The auxin gradient model of gynoecium development has been refined by the invocation of an antagonistic concomitant cytokinin gradient (Østergaard 2008), such that the respective antagonistic function of either hormone establishes three distinct developmental domains; however, experimental data to support this are lacking. It is clear, however, that valve development is sensitive to auxin

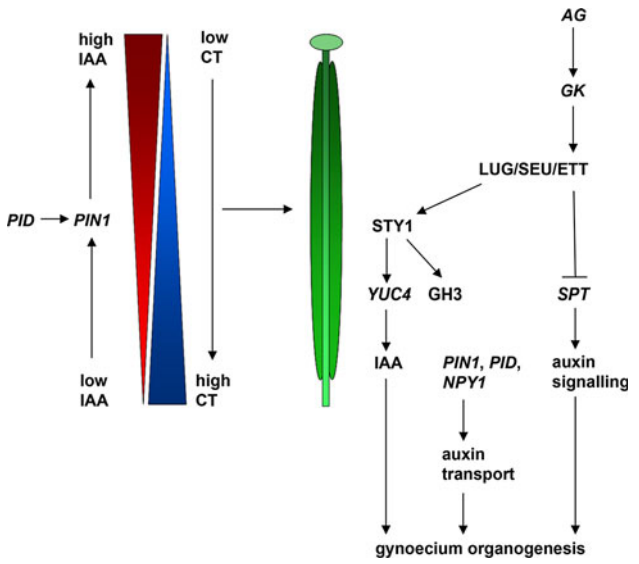

Fig. 4 The role of hormones in the developing gynoecium: an apical/basal gradient of auxin generated by PAT across the gynoecium prirmordium, possibly coupled with an antagonistic cytokinin gradient, is postulated to establish the apical, medial, and basal gynoecium domains. Shown are the transcription factor hierarchies known to interact with auxin in the developing gynoecium: STY1 physically binds the *YUC4* promoter to regulate auxin biosynthesis in the apical domain and possibly affects auxin homeostasis via *GH3* gene induction. LEU physically interacts with SEU, and SEU with ETT, thereby regulating downstream targets such as *SPT*, involved in auxin signaling, and auxin responsive genes. AG regulates *GK* upstream of *ETT*. Auxin polar transport is affected by *PIN1*, *PID*, and *NPY1*

concentration, as it is disrupted in *pid* mutants (Bennett and others 1995) (Fig. 4) and in dominant-negative *sty1* mutants, which act upstream of auxin biosynthesis (Ecklund and others 2010).

Crosstalk

Hormones do not control development via linear pathways but via complex interconnected webs of cross-regulation and crosstalk (Chandler 2009; Kuppusamy and others 2009). One way to dissect hormone crosstalk in floral development is to perform gene microarrays or CHipSeq using meristem or floral organ identity genes, to place these master transcription factors as nodes in hormone crosstalk networks. Meristem size is determined by hormone interplay, with CTs and auxin converging on the function of two type-A *ARABIDOPSIS RESPONSE REGULATORS ARR7* and *ARR15* (Zhao and others 2010). *KNOX* genes also integrate multiple hormone signals, promoting CT biosynthesis (Yanai and others 2005) in addition to acting in GA pathways (Hay and others 2002). However, examples of hormone crosstalk in floral organ development are currently not extensive and mainly involve male organ development, which is regulated by many hormones in concert. JA biosynthesis is promoted by GA biosynthesis during stamen filament and anther growth in *Arabidopsis* (Cheng and others 2009), which establishes a hierarchical relationship between two hormones known to affect male sterility and stamen development. By DELLA repression, GA activates the JA biosynthesis gene *DAD1*, which in turn regulates the transcription factors *MYB21*, *MYB24*, and *MYB57* (Cheng and others 2009). The phenotype of double *arf6 arf8* mutants of short stamen filaments, indehiscent anthers, and immature gynoecia is reflected in decreased JA production (Nagpal and others 2005) via a reduction in *DAD1* expression (Tabata and others 2010). Therefore, auxin response is intertwined with the JA regulation of floral organ elongation. During stamen development in *Arabidopsis*, GA signaling reveals an important point of cross-regulation with many other phytohormones: A whole-genome microarray to identify genes controlled by DELLA protein RGA (Hou and others 2008) showed that a proportion of these genes was also regulated by JA, and others by ABA, ethylene, and auxin. Furthermore, *KNAT2*, a member of the *KNOX* gene family, which in *Arabidopsis* positively regulates CT biosynthesis (Jasinski and others 2005; Yanai and others 2005), was also regulated by RGA. Therefore, DELLA regulation of floral organ development within GA signaling pathways is modulated by multiple phytohormones.

Many gene targets of *SEP3* are involved in signaling and homeostasis of several different hormones, including auxin, GA, and BR (Kaufmann and others 2009), making *SEP3* a node for hormone cross-regulatory networks. The tissue-specific impairment of GA signaling via *gai* expression in anthers and pollen of tobacco and *Arabidopsis* which leads to male sterility could be overcome by kinetin application (Huang and others 2003), demonstrating an interplay between GA and CT pathways in male development. *OsMADS1*, which controls floret development in rice, is one potential node of hormone cross-regulation because it affects many ARF and Aux/IAA genes as well as CT biosynthesis-encoding genes and CT response regulators (Yadav and Vijayraghavan 2008).

Conclusions and Perspectives

The characterization of the targets of floral organ identity genes such as *SEP*, *AG*, and *AP3* shows, as might be expected, that these targets regulate floral organ architecture by recruiting floral-specific transcription factors. Some progress has been made in marrying hormone pathways to master gene hierarchies of floral organ identity genes or floral-building genes; however, the identification of further targets of floral homeotic genes is needed to unravel more organ-specific connections with hormone functions. It is

clear that the reproductive organs contain a larger complement of organ-restricted transcripts than perianth organs (Wellmer and others 2004). Concomitantly, male development in particular is regulated by multiple hormones in concert, probably to a more complex degree than other organs. There are some data that show the interconnection of TF hierarchies through hormones, and the floral organ where this is best understood is the developing gynoecium in relation to auxin.

Many general features concerning the hormonal regulation of floral development have arisen from an understanding of auxin and GA function, including the importance of tissue-specific hormone homeostasis by local hormone synthesis, for auxin by flavin monooxygenases (Cheng and others 2006), and for GAs shown by reporter gene analysis of different GA biosynthetic-encoding genes (Rieu and others 2008). Tissue-specific homeostasis involving degradation and transport and feedback/feedforward loops, for example, GA synthesis, is feedback-regulated via *GA3ox* and *GA20ox* by GA (Cowling and others 1998; Xu and others 1998) is also an important regulatory component.

Auxin is of key importance in lateral organ initiation from the IM and in floral organ initiation from the FM and subsequent organogenesis, which is dependent on both local auxin biosynthesis and PAT. For auxin, redundancy is a key aspect of function, in genes encoding biosynthetic enzymes (Cheng and others 2006 for *YUCCA*), transport (Friml and others 2003 for PIN family), and signaling (ARF) (Hardtke and others 2004; Wilmoth and others 2005). For GA, biosynthetic enzymes encoded by multigene families function in parallel (Hedden 1999), which means that one feature of floral organ development must reside in the complement of tissue-specific biosynthesis or signaling genes, at least for GA and auxin. The role of auxin gradients in floral development is less clear: Dynamic auxin gradients are certainly established and drive development in other tissues such as roots (Grieneisen and others 2007) and the female gametophyte (Pagnussat and others 2009); and in as far as an auxin gradient can induce cell-type specification, auxin can also function as a morphogen (Bhalerao and Bennett 2003; Leyser 2005). An auxin gradient is apparently important for gynoecium development, but the exact interplay and importance between transport, concentration, and gradient establishment here and in other floral organs requires clarification. Aside from within-organ hormone functions, it is not clear to what extent hormones or signaling components are redistributed between developing organs. There is some evidence that auxin controls interorgan ontogeny (Aloni and others 2006), but the role of hormones in general in synchronizing development is unknown and should be the focus of future research. Further open questions concerning auxin that remain are to what extent it acts as a morphogen and how important are the relative contributions of local biosynthesis versus transport. Experiments to manipulate tissue- and temporal-specific hormone concentration at the biosynthetic, transport, and signaling levels will inform this debate.

In addition to organ outgrowth, hormones also have a role in limiting growth and regulating organ boundaries. One example is boundary formation in sepals and stamens of flowers by the *CUC* genes (Aida and others 1997; Vroemen and others 2003). *CUC2* and *CUC3* are positively regulated by CTs in FMs (Li and others 2010), providing a link between hormones and the transcription factor control of organ boundaries. Future research should address the extent of hormone cross-regulation for which little is currently known for floral development, including identifying cross-regulatory nodes. Finally, the extent to which floral organ regulation is conserved among the plant kingdom across plants of widely differing morphologies is also a relevant and exciting question. Exciting new developments may even be the identification of novel growth-regulatory signals.

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