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Review

Gene-regulatory networks controlling inflorescence and flower development in *Arabidopsis thaliana*☆

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ABSTRACT

Reproductive development in plants is controlled by complex and intricate gene-regulatory networks of transcription factors. These networks integrate the information from endogenous, hormonal and environmental regulatory pathways. Many of the key players have been identified in Arabidopsis and other flowering plant species, and their interactions and molecular modes of action are being elucidated. An emerging theme is that there is extensive crosstalk between different pathways, which can be accomplished at the molecular level by modulation of transcription factor activity or of their downstream targets. In this review, we aim to summarize current knowledge on transcription factors and epigenetic regulators that control basic developmental programs during inflorescence and flower morphogenesis in the model plant *Arabidopsis thaliana*.
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1. Introduction

Successful reproduction in plants requires the coordinated transition from vegetative growth to reproductive development. The floral transition in angiosperms is triggered by environmental factors, such as photoperiod, temperature and nutrient conditions, as well as by endogenous cues [1]. The exact environmental conditions that initiate flowering are variable between species and show a high degree of adaptation to local habitats of natural populations (see, e.g. [2–4]). External signals are typically perceived in leaves and roots, and long-distance transport of signaling compounds to the shoot meristem then triggers its switch from vegetative to inflorescence meristem identity [1]. These changes in meristem identities result in alteration of internode growth, branching and the identities of organs that are produced by these meristems. In the course of flower development, the floral meristem becomes terminated and consumed during organ outgrowth.

While floral transition and inflorescence development are highly variable among different species of flowering plants, the generic flower bauplan with distinct perianth, male and female reproductive organ types is evolutionarily conserved. Angiosperm flowers are typically characterized by sterile perianth organs in the outer whorls, and by male (stamens) and female (carpels) reproductive organs in the inner whorls, respectively. The model plant species *Arabidopsis thaliana* (Arabidopsis, thale cress) has a differentiated perianth consisting of sepals and petals that is typical for eudicots. The specification of floral organ identities is controlled by floral homeotic genes, which have been identified to encode transcription factors (TFs) about 25 years ago [5–7]. According to the classical ABC model [8] that is based on phenotypes of homeotic mutants in Arabidopsis and *Antirrhinum majus*, floral organ identities are specified by 3 functional gene classes that act together in a combinatorial fashion: class A genes specify the identities of sepals, and together with class B genes determine petal identity. Class

Abbreviations: TF, transcription factor; MADS, **M**CM1, **A**GAMOUS, **D**EFICIENS, **S**RF; SEP, SEPALLATA; ChIP-seq, chromatin immunoprecipitation followed by deep sequencing; PcG, Polycomb group; H3K27me3, histone 3 lysine 27 tri-methylation; PRC, polycomb repressive complex; trxG, trithorax group; SAND domain, **S**p100, **A**IRE-1, **N**ucP41/75, **D**EAF-1 domain; ULT, ULTRAPETALA; miRNA, microRNA; AP2, APETALA2; FLC, FLOWERING LOCUS C; SOC1, SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1; FT, FLOWERING LOCUS T; FD, FLOWERING LOCUS D; IM, inflorescence meristem; AGL24, AGAMOUS-LIKE 24; FM, floral meristem; LFY, LEAFY; AP1, APETALA1; CUC2, CUP-SHAPED COTYLEDON 2; NAC, **N**AM, **A**TAF, **C**UC; BP, BREVIPEDICELLUS; KNAT, KNOTTED1-LIKE HOMEOBOX GENE from *Arabidopsis thaliana*; BLH, BEL1-LIKE HOMEODOMAIN; PNY, PENNYWISE; BOP, BLADE-ON-PETIOLE; ATH1, ARABIDOPSIS THALIANA HOMEOBOX GENE 1; PNF, POUNDFOOLISH; CAL, CAULIFLOWER; PIN1, PIN-FORMED 1; MP, MONOPTEROS; ARF, AUXIN RESPONSE FACTOR; ANT, AINTEGUMENTA; AIL6, AINTEGUMENTA-LIKE 6; SWI/SNF, SWITCH/SUCROSE NONFERMENTING; LMI, LATE MERISTEM IDENTITY; GA, gibberellic acid; SPL, SQUAMOSA PROMOTER BINDING PROTEIN-LIKE; ELA1, EUI-LIKE P450 A1; TOE, TARGET OF EAT; WUS, WUSCHEL; SVP, SHORT VEGETATIVE PHASE; SEU, SEUSS; AG, AGAMOUS; PI, PISTILLATA; AP3, APETALA 3; TALE-HD, three amino acid loop extension homeodomain; BEL1, BELL1; BRM, BRAHMA; SYD, SPLAYED; UFO, UNUSUAL FLORAL ORGANS; HEC, HECATE; CRC, CRABSCLAW; IAA, INDOLE-3-ACETIC ACID INDUCIBLE; GA2OX, GIBBERELLIN 2-OXIDASE; JA, jasmonic acid; GNC, GATA; NITRATE INDUCIBLE, CARBON METABOLISM INVOLVED; GNL, GNC-LIKE; KNU, KNUCKLES; PAN, PERIANTHIA; AGO, ARGONAUTE; SQN, SQUINT; UIF, ULTRAPETALA1 INTERACTING FACTOR1; SUP, SUPERMAN; Cas9, CRISPR-associated protein 9.

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B genes together with class C genes specify stamens. Class C genes alone determine carpel identity. The model was later extended by class E genes which are important for specification of all types of floral organs [9,10], while the 'A class' is debated and a new definition combining class A and E functions has been proposed as to set a floral 'ground state' [11]. With the exception of the classical A class, the functions of homeotic genes are widely conserved across angiosperms, and some evolutionary changes in basic flower morphology can be explained by modifications in the ABC(E) model [12].

Reproductive meristem specification and organ differentiation require orchestrated changes in expression of many genes. The characterization of the underlying gene-regulatory networks and molecular mechanisms has made tremendous progress in recent years, in particular in model plant species such as Arabidopsis and rice. In this review, we attempt to provide an overview on gene-regulatory factors and processes controlling reproductive development in the best studied model plant Arabidopsis. We do not attempt to comprehensively cover the floral transition process, which has recently been extensively reviewed elsewhere [1,2,13,14].

2. Gene-regulatory networks: what are the 'players'?

Developmental gene regulation in plants, like in other eukaryotic organisms, is controlled by the interplay of TFs, epigenetic mechanisms and posttranscriptional processes. The Arabidopsis genome encodes at least 2300 transcriptional regulators, at least 360 of which have annotated roles in development [15–17]. For example, at least 17 MADS (**M**CM1, **A**GAMOUS, **D**EFICIENS, **S**RF)-box genes and 23 homeobox TF genes have clear roles in flower development (Table 1). Some TF families appear to have predominant roles in individual processes, such as Myb-domain TFs which have been mainly described to act in stamen development (Table 1). Forward and reverse genetic approaches in the past 25 years have boosted our knowledge on the functions of developmental TF genes. It has also become clear that genetic redundancy is a major confounding factor in elucidating developmental roles of TFs. For example, style and stigma development in the gynoecium is controlled by four largely redundantly acting *NGATHA* genes that belong to the B3 TF family [18,19]. Another example is the four redundantly acting *SEPALLATA* (*SEP1–4*) MADS-box TFs which together control floral meristem and organ specification by constituting the so-called 'E class' [9,10,20]. Classical genetic analyses have described reproductive development as hierarchically organized genetic network with sequentially acting genes. Linked with genome-wide TF target gene identification, it has now become clear that gene-regulatory networks are inherently complex, with multiple feedback and feedforward loops not all of which have been fully understood to date [21]. Genome-wide target gene identification has been enabled by technologies such as chromatin immunoprecipitation followed by deep sequencing (ChIP-seq) or hybridization to tiling arrays (ChIP-chip) [21] together with gene perturbation and genome-wide expression analyses [22].

Next to TFs, also major epigenetic regulators have been shown to act in plant reproductive development. In general, epigenetic regulation is required for cellular memory of gene expression states across cell divisions. Switches in stage- and tissue-specific gene expression programs during development, and developmental plasticity in response to environmental signals require changes in epigenetic states of gene bodies and promoters [21,23,24]. The maintenance of gene repression across cell divisions, termed'epigenetic memory' is mediated by Polycomb group (PcG) factors, which deposit repressive histone 3 lysine 27 trimethylation (H3K27me3) marks (see [25,26] for recent review). PcG mediated regulation is not limited to flower development, but affects thousands of genes (see, e.g. [27–29]), and multiple mechanisms contribute to the recruitment of PcG proteins to their target gene promoters [30–34]. During vegetative development, floral homeotic genes are epigenetically repressed by PcG protein complexes. Gene regulation by Polycomb Repressive Complex (PRC) 1 and PRC2 is achieved by depositing H3K27me3 and histone 2A ubiquitination marks at target genes and subsequent chromatin compaction (for review, see [25]). Mutations in PRC components often show decreased H3K27me3 levels and precocious expression of floral homeotic genes in the vegetative phase [35,36]. Proteins that counteract the repressive function of PcG proteins are considered as trithorax group (trxG) factors. Some trxG proteins display histone methyl transferase activity, others assist in that function or are involved in chromatin remodeling (reviewed by [24,26]). For example, the SAND (**S**p100, **A**IRE-1, **N**ucP41/75, **D**EAF-1) domain proteins ULTRAPETALA (ULT) 1 and ULT2, are considered to function as trxG proteins influencing histone 3 lysine 4 tri-methylation [37–40]. Furthermore ULT1 acts as an anti-repressor, similarly counteracting PcG action on thousands of target genes including floral homeotic genes [41]. ULT1 and ULT2 have dual roles in floral stem cell accumulation and the establishment of apical-basal polarity in the gynoecium [38,40].

Next to TFs and other proteins, the role of regulatory RNAs in the control of developmental gene expression has also been widely appreciated now [42]. Most well-known are microRNAs (miRNAs), which are in their mature state approximately 21-nucleotides in length. MiRNAs originate from stem-loop regions of long primary transcripts and are processed by a Dicer-like enzyme [42,43]. They either function in degradation of complementary target mRNAs or in translational inhibition [43]. An example for a well-known family of miRNAs with roles in flower development is the miR172 family, which regulates several APETALA2 (AP2) class TFs thereby controlling flower initiation, patterning and floral stem cell activity [44–48]. Linked with the availability of large-scale sequencing approaches, long non-coding RNAs have been identified systematically more recently [49,50]. The best known example is the long non-coding antisense RNA *COOLAIR*, which regulates the epigenetic status of the central repressor of flowering, *FLOWERING LOCUS C (FLC)* [4,51,52]. However, roles of long non-coding RNAs after the transition to flowering are still largely unexplored.

3. Gene-regulatory networks controlling inflorescence development

Inflorescence development starts with the activation of the MADS-box TF *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*) in the shoot apex of Arabidopsis [53,54], mainly by the 'florigen' FLOWERING LOCUS T (FT) and its interaction partner FD. In the IM, *SOC1* constitutes a major hub in controlling the expression of many other genes involved in floral transition, meristem identity and other functions [55,56]. *SOC1* acts in concert with *AGAMOUS-LIKE 24* (*AGL24*) and *FRUITFULL*, which also encode MADS-domain TFs, and triple mutants show a drastic late-flowering 'perennial' phenotype with floral reversion events and prolonged indeterminate growth [57]. FT/FD furthermore activate the expression of the floral meristem (FM) identity genes *LEAFY* (*LFY*) and *APETALA1* (*AP1*) gene in the laterally emerging meristems [58–60].

Besides TFs that control IM identity, other transcriptional regulators, in concert with phytohormone signaling pathways, control inflorescence morphology, e.g. by affecting branching or phyllotaxis (reviewed in [61–63]). For example, the boundary gene *CUP-SHAPED COTYLEDON 2* (*CUC2*), which encodes a NAC (**N**AM, **A**TAF, **C**UC) family TF, plays a role in elongation of stem internodes and therefore in establishing the spiral inflorescence phyllotaxis [64]. Inflorescence architecture is furthermore controlled by a network of homeobox TFs. The KNOTTED1-LIKE HOMEOBOX GENE FROM ARABIDOPSIS THALIANA (KNAT) TF BREVIPEDICELLUS (BP) and the interacting BEL1-LIKE HOMEODOMAIN (BLH) protein PENNYWISE (PNY) have partially redundant roles in internode and radial patterning during inflorescence growth (see [65] for recent review). Their role is indicated by the enhanced double mutant phenotype with highly irregular internode length and increased branching [66]. BP and PNY act by restricting the expression of boundary genes such as *KNAT6*, *BLADE-ON-PETIOLE (BOP) 1 and BOP2*, and *ARABIDOPSIS THALIANA HOMEOBOX GENE1 (ATH1)* [67,68]. *PNY* acts redundantly with another BELL1 family member, *POUNDFOOLISH* (*PNF*) to promote flowering and to establish the FM, since *pny pnf* double mutants

Table 1
Examples of transcription factors with known roles in flower development in *Arabidopsis*.

TF-family	Gene	Identifier	Function	Selected recent references/reviews
AP2/ERF	DRNL	AT1G24590	Stamen development, organ founder cells	[179,183]
	AP2	AT4G36920	FM establishment and floral patterning	[44,46,123,182,184]
	ANT, AIL5, AIL6	AT4G37750, AT5G57390, AT5G10510	Organ morphology and growth, FM initiation, ovule development, flower primordium initiation	[185]
	SHN1, SHN2	AT1G15360, AT5G25390	Organ morphology	[217]
	TOE3	AT5G67180	Determinacy	[158]
B3 ARF	ARF5/MP	AT1G19850	Flower primordium formation, ovule development	[77,78,187]
	ARF6, ARF8	AT1G30330, AT5G37020	Organ maturation, carpel development	[135–137]
	ARF3/ETT, ARF4	AT2G33860, AT5G60450	Gynoecium development, determinacy, organ polarity	[170,171,188]
	ARF2	AT5G62000	Organ size	[138,180,189]
B3 RAV	NGA1, NGA2, NGA3, NGA4	AT2G46870, AT3G61970, AT1G01030, AT4G01500	Style development	[19,188]
bHLH	MYC2, MYC3, MYC4, MYC5	AT1G32640, AT5G46760, AT4G17880, AT5G46830	Stamen development	[176]
	BPE	AT1G59640	Petal growth	[133–135]
	bHLH010, bHLH089, bHLH091	AT2G31220, AT1G06170, AT2G31210	Anther development	[190]
	DYT1, AMS	AT4G21330, AT2G16910	Anther development	[190,191]
	SPT, ALC	AT4G36930, AT5G67110	Gynoecium development	[192]
	HEC1, HEC2, HEC3	AT5G67060, AT3G50330, AT5G09750	Gynoecium development	[192,193]
BPC	BPC1, BPC2, BPC3, BPC4, BPC6	AT2G01930, AT1G14685, AT1G68120, AT2G21240, AT5G42520	Organ number, morphology, meristem size	[175]
bZIP	TGA9, TGA10	AT1G08320, AT5G06839	Stamen development	[194,195]
bZIP	PAN	AT1G68640	Organ number, determinacy	[153,154,194]
C2C2-GATA	GATA15, HAN, HANL2, GNC, GNL	AT3G06740, AT3G50870, AT4G36620, AT5G56860, AT4G26150	Stamen growth, organ number, boundary formation	[138,196–198]
C2C2-YABBY	INO	AT1G23420	Ovule development	[199]
	CRC	AT1G69180	Gynoecium development, determinacy	[163,172,199]
	FIL	AT2G45190	Organ number, size, morphology, boundary formation, FM identity	[186]
C2H2	JAG, NUB	AT1G68480, AT1G13400	Organ growth and patterning	[127]
	SUP	AT3G23130	Boundary formation, floral patterning, determinacy, ovule development, petal and stamen development	[173,174]
	RBE	AT5G06070	Petal development, boundary formation	[200]
	KNU	AT5G14010	Gynoecium development, determinacy	[30,34,152,178]
GARP	UIF1	AT4G37180	Organ number, organ boundary	[167]
	KAN1, KAN2, KAN4/ATS	AT5G16560, AT1G32240, AT5G42630	Gynoecium development, organ polarity	[170,201]
HB	STM	AT1G62360	Shoot meristem activity, carpel development	[97,202]
	KNAT2	AT1G70510	Carpel development	[202]
	PFS2/WOX6	AT2G01500	Ovule development, petal development	[203]
	WUS	AT2G17950	Basic shoot meristem activity	[145,146,149,150,203]
	PHB, PHV, REV, CNA	AT2G34710, AT1G30490, AT5G60690, AT1G52150	Ovule development, meristem activity and determinacy, organ number	[204]
	WOX1, PRS/WOX3	AT3G18010, AT2G28610	Sepal and petal development	[203]
	HDG1, HDG2, HDG5, HDG12, PDF2	AT3G61150, AT1G05230, AT2G46680, AT1G17920, AT4G04890	Stamen development, petal and stamen development	[205]
	JAB	AT4G17460	Determinacy, gynoecium development	[206]
	BLR/PNY, PNF	AT5G02030, AT2G27990	Organ number and specification of petals, stamen and carpels, inflorescence structure	[69,202]
	BEL1	AT5G41410	Ovule development	[120]
LFY	LFY	AT5G61850	FM identity, floral patterning	[79,86,126]
MADS	CAL	AT1G26310	Floral meristem initiation	[83]
	AP1	AT1G69120	A-class gene, organ identity, meristem identity	[87,96,182,207]
	AP3, PI	AT3G54340, AT5G20240	B-class gene, petal and stamen identity	[124,182,207]
	SHP1, SHP2, STK	AT3G58780, AT2G42830, AT4G09960	Carpel development, ovule development	[120,207]
	AG	AT4G18960	Floral homeotic C-class gene, determinacy	[125,182,207]
	AGL24, SVP	AT4G24540, AT2G22540	Floral transition, floral meristem identity	[80,102,207]
	SEP1, SEP2, SEP3, SEP4	AT5G15800, AT3G02310, AT1G24260, AT2G03710	Floral homeotic *E*-class, organ identity, meristem identity	[20,106,207]
	FUL	AT5G60910	Floral transition, carpel development	[57,207]
MYB	MYB65, MYB33	AT3G11440, AT5G06100	Flower maturation, anther development	[208]
	MYB21, MYB24, MYB57	AT3G27810, AT5G40350, AT3G01530	Flower maturation; stamen development	[176,209]
	PWR	AT3G52250	Determinacy, carpel development, petal development	[48]
	LMI2	AT3G61250	Meristem identity	[85]
NAC	CUC1, CUC2, CUC3	AT3G15170, AT5G53950, AT1G76420	Boundary formation, gynoecium development, ovule development	[64,179,199]
SBP	SPL3, SPL8	AT2G33810, AT1G02065	FM identity, gynoecium development, anther development	[210]
SPL/NZZ	SPL/NZZ	AT4G27330	Anther development, ovule development; stamen identity,	[186]
SRS	STY1, STY2	AT3G51060, AT4G36260	Stamen development, gynoecium development	[192]
TCP	TCP3, TCP4, TCP5, TCP10, TCP13, TCP17, TCP15	AT1G53230, AT3G15030, AT5G60970, AT2G31070, AT3G02150, AT5G08070, AT1G69690	Petal morphology, petal growth, carpel development, flower maturation	[211,212]
Trihelix	PTL	AT5G03680	Petal development, boundary formation	[200]

fail to accumulate transcripts of the FM identity genes *LFY*, *AP1* and *CAULIFLOWER* (*CAL*) [69]. Recently it was shown that *BOP1/2* are directly repressed by PNY [70]. It has been proposed that BOP1/2 promote directly the expression of *ATH1*, and by that indirectly *FLC*, that act as repressors of *FD* [70–72]. This may provide a mechanistic explanation for the additional function of *PNY* in the induction of flowering.

4. Gene-regulatory networks controlling flower initiation

In the flanks of the Arabidopsis IM, FMs are established as lateral meristems in an auxin-dependent manner. The requirement for auxin signaling is shown by the 'naked pin'-like phenotype of *pin-formed 1* (*pin1*) and *monopteros* (*mp*) mutants, which essentially fail to initiate FMs [73,74]. *PIN1* encodes a major auxin efflux carrier involved in polar auxin transport [75], while MP/ARF5 encodes an auxin-dependent AUXIN RESPONSE FACTOR (ARF) TF [76]. During the earliest stages of flower development, differentiation is temporarily suppressed in the FM in order to generate a sufficient amount of cells for formation of the inner floral whorls. MP directly activates the FM identity gene *LFY* as well as the AP2-type TF genes *AINTEGUMENTA* (*ANT*) and *AINTEGUMENTA-LIKE 6* (*AIL6*), which in turn also up-regulate *LFY* and stimulate FM and organ proliferation [77] (Fig. 1). MP activates these genes by switching chromatin state via recruitment of SWITCH/SUCROSE NONFERMENTING (SWI/SNF) -type nucleosome remodelers in an auxin-dependent manner [78].

LFY is a key regulator in the network establishing FM identity and is activated by flowering time genes *AGL24*, *SVP* and *SOC1* [79,31,80,81]. It is expressed prior to FM outgrowth (floral stage 1; for flower developmental stages see Fig. 2) and first activates *LATE MERISTEM IDENTITY1* (*LMI1*) and, in combination with LMI1, up-regulates the expression of the MADS-box gene *CAL* [82] (Fig. 3). The MADS-domain TF AP1 acts in a partly redundant manner with its close paralog CAL in controlling FM identity [83]. CAL and LFY, partly in combination with LMI2, activate *AP1* that in turn can increase *LFY* expression in a positive feedback loop in order to maintain FM identity [84–88]. Comparison of the ChIP-seq data generated for the FM identity regulators LFY and AP1 [86,87,89] shows that approximately one third of AP1 or LFY bound genes are also targeted by the other TF, respectively [90]. Genes involved in gibberellic acid (GA) homeostasis are for instance targeted by LFY as well as AP1.

GA is a phytohormone that positively regulates floral transition but inhibits FM establishment. Molecular mechanisms underlying these antagonistic roles have only recently started to be elucidated. According to the current model, GA initially up-regulates the expression of *SQUAMOSA PROMOTER BINDING PROTEIN LIKE* (*SPL*) TF genes as well as *LFY* in a concentration-dependent manner by degrading DELLA proteins, transcriptional co-regulators, via the 26S proteasome [91,92]. It is suggested that LFY then promotes a reduction of GA levels in part by promoting expression of *EUI-LIKE P450 A1* (*ELA1*) [93], a cytochrome p450 enzyme involved in GA catabolism [94]. Decreasing the GA concentration leads to the accumulation of GA-sensitive DELLA proteins, which in turn are required for up-regulation of *AP1*. This is mediated by binding of DELLA proteins recruited by SPL9 to the *AP1* promoter [93]. Activation of AP1 increases *ELA1* expression in a positive feedback loop, thus enforcing a more rapid switch from vegetative to reproductive phase [93].

In order to stabilize the switch to FM identity, AP1 downregulates repressors of its own expression, such as the AP2 TF genes *TARGET OF EAT* (*TOE*) *1*, *TOE3*, *SCHNARCHZAPFEN* and the AP1 antagonist *TERMINAL FLOWER 1* [87]. AP1 also establishes FM identity by repressing genes involved in the shoot developmental program and genes that control onset of flowering, like *FD*, *FD PARALOG* and *SPL9* [87]. In addition to that, AP1 prevents the establishment of meristems in the axils of sepals in order to suppress axillary secondary flower formation [95]. Recently, it has been shown that AP1 mediates this function by suppressing cytokinin accumulation via direct repression of the cytokinin biosynthetic gene *LONELY GUY1* and activation of the cytokinin degradation gene *CYTOKININ OXIDASE/DEHYDROGENASE3* [96]. Direct regulation is in agreement with binding of AP1 to these loci [87,96]. By this, *AP1* counteracts the function of *SHOOTMERISTEMLESS* (*STM*), a homeodomain TF essential for shoot apical meristem formation and maintenance that upregulates cytokinin biosynthesis [97]. Changes in cytokinin levels affect the activity and downstream functions of the stem cell regulator WUS (see, e.g. [98]).

During early flower development, *AP1* is considered to be an FM identity gene. Later, around stage 3 of flower development, when sepal primordia arise (Fig. 2, stages according to Smyth et al. [99]),

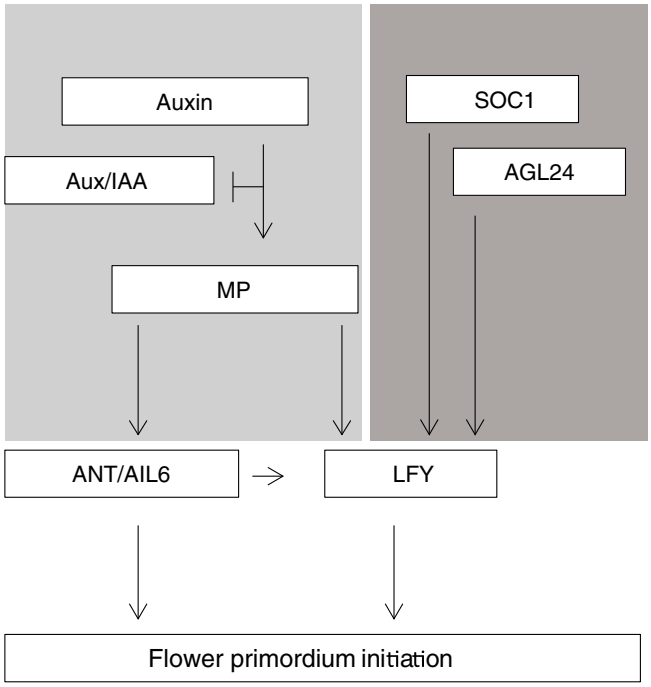

Fig. 1. Auxin mediated floral primordium initiation. At high levels of auxin, repressive Aux/IAA proteins are released from the Auxin Response Factor ARF5/MP. MP then activates expression of *LFY* as well as *ANT* and *AIL6*, which act in parallel with *MP* to promote *LFY* induction. Up-regulation of *LFY* and *ANT/AIL6* activate the gene regulatory networks that initiate the formation of floral primordia.

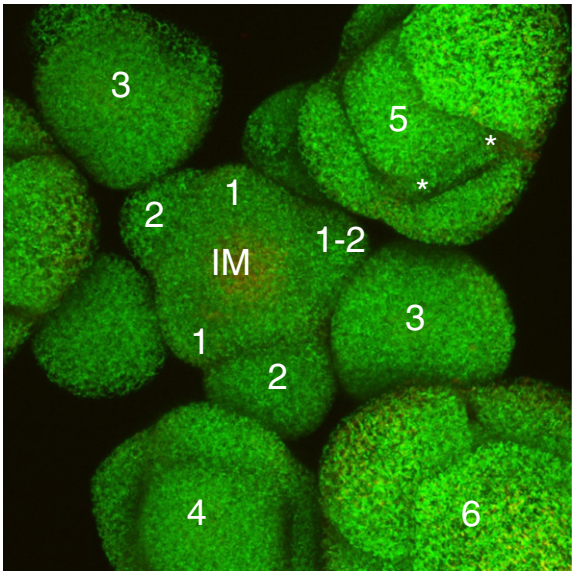

Fig. 2. Confocal microscopic analysis of early flower bud stages. Overview of an inflorescence meristem and early flower meristem stages 1–6. The early stages are defined as: flower buttress arises (stage 1), flower primordium forms (stage 2), sepal primordia arise (stage 3), sepals overlie floral meristem (stage 4), petal (*) and stamen primordia arise (stage 5), sepals enclose the buds (stage 6). Stages according to [99]. Confocal image shows ubiquitously expressed endomembrane-localized GFP reporter gene.

AP1 expression becomes restricted to the outer whorls, where it specifies sepal and petal identity [95]. A molecular mechanism for the functional change of AP1 could be different protein-protein interactions (Fig. 3): AP1 dimerizes with the MADS-domain TFs SHORT VEGETATIVE PHASE (SVP), AGL24 and SEP3 [100,101]. The expression of AGL24 and SVP is mostly restricted to flower developmental stages 1 and 2, before sepal primordia arise, and decreases afterwards [101,102]. Genetic data, ChIP experiments and protein-protein interaction studies suggest that SVP, AGL24 and AP1 act redundantly in maintaining the FM in a meristematic state by preventing precocious expression of floral homeotic genes and probably do so by forming complexes with each other and recruiting additional factors for *SEP3* repression [31,80,102]. In *svp agl24* double mutants, *SOC1*, which is usually not expressed in FMs, can take over their function [31,102]. Gregis et al. showed that AP1/SVP or AP1/AGL24 complexes interact with SEUSS (SEU) and LEUNIG [101], which form a transcriptional co-repressor complex [103,104]. Furthermore, *SEP3* repression by SVP was proposed to be mediated via recruiting the PcG factor TERMINAL FLOWER 2/LIKE HETEROCHROMATIN PROTEIN 1 and changes in H3K27me3 deposition, whereas AGL24 and SOC1 act via recruitment of proteins with histone deacetylase activity in the absence of SVP [31]. Repression of *SEP3* in the first stages is crucial for meristem maintenance, since SEP proteins directly activate the B and C class genes in concert with LFY and AP1 [31,105,106]. De-repression of *SEP3* in the early stage 3 allows the formation of AP1/SEP3 complexes leading to activation of homeotic gene expression and as a result of that floral organ identity specification, while flowering time genes are kept in a repressed state [31,102,105–107]. Interestingly, approximately 80% of the putative AP1 target genes are down-regulated in early FM development, but at later stages AP1 together with SEP3 preferentially act as activators of gene expression [87,89]. SEP3 as well has been shown to interact with SEU [108], indicating that recruitment of the co-repressors occurs more frequently by different floral MADS-domain TF complexes.

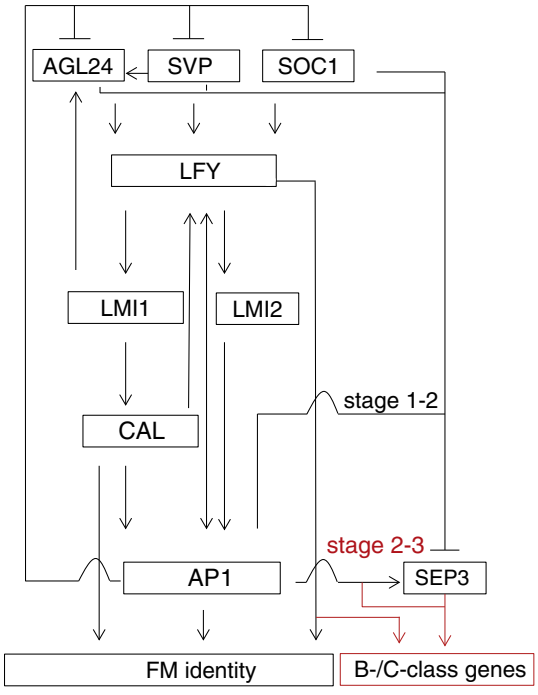

Fig. 3. LFY-mediated pathway for the establishment of floral meristem identity. During the transition from IM to FM (stages 1–2), LFY activates *AP1*, possibly by interacting with LMI2. Indirect activation of *AP1*, by *LFY* is achieved through *LMI1* and *CAL*. Up-regulation of *AP1* results in a positive feedback loop triggering *LFY* up-regulation in order to stabilize the switch from IM to FM. AP1 and LFY upregulate *SEP3* expression, which is required for strong activation of homeotic B- and C-class genes around stage 3 of flower development.

Genome wide identification of SVP target genes in inflorescences identified 265 putative targets that are common to SVP and AP1 [80]. Those genes are enriched for TF genes, including *KANADI1*, *PHABULOSA* and *ARF3* [80]. Furthermore, SVP binds to genes involved in auxin, GA, cytokinin and jasmonic acid (JA) signaling, and to genes with roles in posttranslational regulation in the FM [80]. SVP, SOC1 and AGL24 also promote *LFY* expression while LFY may be an indirect negative regulator of *SVP* and *AGL24* [109].

At later stages of FM development, AGAMOUS, AP1 and SEP3 directly repress *SVP*, *AGL24* and *SOC1* in a regulatory feedback loop to allow floral organ patterning and to restrict meristem activity [80,87,101,109,110]. At the same time, homeotic B and C class genes become activated allowing meristem differentiation and floral organ initiation.

5. Gene-regulatory networks controlling floral organ specification

After initiation of the FM, the distinct floral whorls are established resulting in the formation of the different types of floral organs. FM differentiation is initiated around floral stages 2–3, when the expression of floral homeotic B and C class genes commences in a spatially restricted whorled pattern within the meristem (Fig. 2). At stage 3, sepal primordia appear, followed by the organ primordia of the inner whorls in stage 5 and sepal outgrowth enclosing the buds at stage 6 [99]. With one exception, all classical floral homeotic genes of the ABCE model encode for members of the MADS domain TF family. Their combinatorial interactions in floral organ specification were proposed to be based on formation of organ-specific higher-order protein complexes, as suggested by the 'floral quartet model' [10,111]. According to this model, the exact composition of those complexes determines the identity of the organ to be formed. In Arabidopsis, sepal identity specification is mediated by A-class protein AP1 in combination with E-class proteins (SEP1–4), while AP1 together with the B-class proteins APETALA3 (AP3), PISTILLATA (PI) and SEP proteins specify petal identity. Stamens are defined by interactions of the C-class protein AG with AP3 and PI, which is mediated by SEP proteins, whereas carpels are specified by AG/SEP complexes [10,112]. SEP proteins were found to act as mediators of higher-order complex formation and, similar to AP1, possess strong transcriptional activation potential [112]. Only the combined overexpression of the different floral homeotic gene classes leads to the conversion of vegetative leaves to specific types of floral organs [112,113], suggesting that the heteromeric floral homeotic protein complexes are not only necessary, but also sufficient to specify floral organ identity.

In planta immunoprecipitation coupled to mass spectrometry of endogenously expressed MADS-domain TFs further supported the interactions that were proposed in the floral quartet model [114]. The floral homeotic protein complexes can also be assembled *in vitro* [114–116]. Higher-order complex formation can induce short-range looping of the DNA which can be relevant for recruitment to target gene promoters and for target gene regulation [116,117]. Although protein-protein interactions within the MADS-domain TF family are well studied [100,118], the exact molecular mechanisms of action and determinants of functional specificity are still not well understood. The stabilization of DNA-binding by two TF binding sites at (complex-)specific distances may be one aspect [119]. It is also possible that higher-order complexes show specific characteristics in the recruitment of co-factors or interactions with Non-MADS-TFs. Indeed, floral homeotic MADS-domain TFs have been found to interact with members of other TF families. For example, the Three Amino acid Loop Extension Homeodomain (TALE-HD) TF BELL1 (BEL1) was found to interact with MADS-domain protein complexes consisting of SEP3 and of AG-like proteins [120]. The interaction with BEL1 is important for ovule specification, and the interaction is thought to modify the activity of the SEP-AG complex that otherwise specifies carpel identity. Interestingly, TFs of different families have been identified in AP1-complexes as well: TALE-HD TF BLH1, PNY and KNAT3 as well as ARF2 and SPL8 have been enriched in protein complex

isolation experiments [114]. Incorporating additional factors into ternary complexes may have impact on target gene specificity and adds options for fine-tuning gene expression responses. The protein complex isolation experiments have identified additional complex partners of floral homeotic proteins, which provide clues on how these factors may regulate target gene activity. Most remarkably, the chromatin-remodeling factors BRAHMA (BRM), SPLAYED (SYD) and PICKLE were identified as components of MADS-domain TF complexes indicating an important role during flower development [114,121]. BRM and SYD are regarded as trxG factors and have been shown to reverse Polycomb repression [121]. Loss of BRM activity results in the change of H3K27me3 marks at hundreds of genes [122]. SYD and BRM are important for *AP3* and *AG* activation during flower patterning and are directly recruited to their promoters by LFY and SEP3 [121]. A further link to epigenetic gene regulation of MADS-domain proteins has been revealed by studying the impact of AP1 and SEP3 DNA-binding on chromatin accessibility. Pajoro et al. [89] found that the binding of these TFs to genomic regions precedes changes in chromatin accessibility, in particular 'opening' of the chromatin. Therefore, these factors provide the first candidates of "pioneer factors" in plants. Changes in chromatin accessibility induced by AP1 and SEP3 then would allow binding of other TFs to genomic regions thereby mediating spatio-temporal control of gene expression in flower development.

The question how major regulatory TFs of flower initiation and differentiation exert their functions has started to become elucidated by the identification of their target gene networks. Target gene identification has been achieved for TFs involved in FM or floral organ specification such as AP1 [87,89], AP2 [123], AP3 and PI [124], AG [125], LFY [86,126], SEP3 [89,106], as well as for the downstream acting growth regulator JAGGED [127]. For example, target gene analyses of the B-class proteins AP3 and PI, which form obligate heterodimers [128,129], have helped elucidate control of petal and stamen development. *AP3* transcripts accumulate in the petal and stamen whorls from stage 3 onwards until expression levels decrease at stage 9, when stamens have formed and petal primordia stalk at the base [130]. *PI* also shows expression from stage 3 onwards [131]. Due to their expression during different stages of flower development it is assumed that AP3 and PI fulfill stage-specific functions by targeting distinct sets of genes: This is based on perturbation experiments where most dramatic effects on organ identity can be seen when *AP3* downregulation occurs in the very early stages, while later disturbances mainly affect stamen maturation [124]. Genome-wide identification of target genes at stage 5, when petal and stamen primordia arise, confirms autoregulatory binding to the *AP3* locus [124]. The F-box gene *UNUSUAL FLORAL ORGANS* (UFO), which encodes a LFY cofactor and thereby up-regulates *AP3* expression [132], is also a direct AP3 target. Furthermore, central players of petal and stamen differentiation like *RABBIT EARS*, *PETAL LOSS*, *SPOROCYTELESS/NOZZLE* are directly regulated by AP3/PI [124]. Moreover, genes involved in carpel development, like *HECATE* (*HEC*) *1* and *HEC2*, *CRABSCLAW* (*CRC*) or *SHATTERPROOF 2* are directly repressed in order to prevent ectopic formation of carpels. B class proteins are also involved in hormone signaling: Especially factors involved in auxin (*INDOLE-3-ACETIC ACID INDUCIBLE (IAA) 2*, *IAA18*, *IAA27*, *ARF3*, *ARF8*), JA (*JASMONATE-ZIM-DOMAIN PROTEIN (JAZ) 9*, *JAZ10*) and GA (*GIBBERELLIN 2-OXIDASE* (*GA2OX*) *1*, *GA2OX3*, *GA2OX4*) signaling are among their downstream targets [124]. Those hormones play a crucial role in determining the size and morphology of floral organs. *ARF8* for example limits cell expansion in later stages of petal development in combination with *BIG PETAL* via combined down-regulation of auxin responsive genes [133–135]. In *arf6 arf8* double mutants, organ elongation in perianth organs and stamen is delayed, probably due to reduced JA levels [136,137]. Feeding experiments suggest that exogenous JA application can promote petal elongation and anther dehiscence, but does not complement other phenotypes, like vascular patterning and changes in petal cell morphology related to *arf6 arf8* mutations [136,137]. Growth defects in stamens often result in stigma protrusion phenotypes, which are related to decreased fertility due to an imbalanced size ratio of carpel and stamens. This phenotype can be observed in other mutants as well, for example in *arf2* mutants. ARF2 has been shown to be a repressor of GATA TF genes *GATA*, *NITRATE INDUCIBLE*, *CARBON METABOLISM INVOLVED* (*GNC*) and *GNC-LIKE* (*GNL*), and *gnc gnl* loss of function in *arf2* mutant background suppresses the *arf2* stamen growth defect [138]. *GNC* and *GNL* have been shown to be regulated by auxin and GA and act downstream of AP3/PI, thus showing that gene-regulatory networks of organogenesis, growth and hormone signaling are tightly interwoven. GNC and GNL are important regulators of different physiological processes, including nitrogen metabolism, sugar sensing and photosynthesis [138–141].

Comparison of genes directly bound by different classes of homeotic TFs show a high overlap [125,142], supporting the idea that they act as part of protein complexes on common sets of target genes. A fraction of the genes appears to be regulated by the different organ-specific complexes in an antagonistic manner, suggesting that the different homeotic protein complexes show partly overlapping DNA-binding activities, but differences in regulatory effects [125,142]. Time-series ChIP-seq experiments of AP1 and SEP3 showed that these proteins are recruited to their target gene promoters in a partly stage-specific manner, supporting the idea that homeotic proteins regulate their genes in a dynamic fashion [89]. The stage-specific differences in promoter occupancy and target gene regulation may depend on availability of cofactors or epigenetic state. However, the detailed molecular mechanisms underlying the stage- and organ-specificity of target gene selection by homeotic proteins remain to be elucidated.

6. Termination of stem cell activity in floral meristems

Stem cell activity in shoot and root meristems is controlled by the complex interplay of TFs, hormonal and cellular signaling pathways (see [143,144] for recent reviews). The homeobox TF WUS is required for the maintenance of the stem cell populations in shoot meristems [145,146], while meristem size is determined by feedback regulation with the CLAVATA signaling pathway, involving movement of WUS within meristems from the organizing center, where it is expressed, to the neighboring stem cells [147]. The stem cell niche in FM is only transiently maintained and a prolonged FM activity in the center of the flower can be observed in several mutants with a compromised determinacy (reviewed by [148]).

Floral determinacy is achieved by exhausting the pool of stem cells in a FM and is tightly linked with shutting down WUS mediated stem cell maintenance in a WUS-AG-feedback loop. During stage 3 of flower development, WUS, together with LFY, promotes *AG* expression in the center of the flower [149,150], which might be at least partly facilitated by non-cell autonomous activity of WUS [151]. Once AG is activated, it directly and indirectly represses *WUS* expression. On the one hand, AG directly binds to the promoter of *WUS* from stage 3 on modifying histone H3 with H3K27me3 marks in a CURLY LEAF dependent manner [30]. On the other hand, AG induces the expression of C2H2-zinc finger TF gene *KNUCKLES* (*KNU*) that in turn directly represses *WUS* from stage 6 onwards, where no *WUS* mRNA is detectable anymore [30,34,152]. Interestingly *KNU* itself shows H3K27me3 marks and the activation of its expression was shown to be cell division-dependent. There is evidence showing that AG binding to the *KNU* promoter interferes with PcG binding, causing a dilution of H3K27me3 marks in a cell division-dependent manner by incorporating unmodified histone H3 [34]. Therefore, AG has a dual and separable function in carpel specification and floral meristem determinacy [152].

The dosage-dependent role of AG in regulating floral determinacy becomes clear from mutants with decreased AG protein levels. For example, the basic leucine zipper TF PERIANTHIA (PAN) is a direct regulator of *AG* in a whorl specific manner, with a putative repressive function in whorl 1 and a promoting function in whorl 4. Mutations in *pan lfy* and *pan seu* show an increased indeterminacy phenotype [153,154]. In addition to transcriptional regulation of *AG*, posttranslational processing of

AG pre-mRNA impacts floral determinacy as well. Consistent with this, posttranscriptional regulators of *AG* show loss of determinacy and compromised *WUS* shutdown (see, e.g. [155–157]). Furthermore, miRNA processing is related to floral stem cell termination: the AG repressors *AP2* and *TOE3* are targeted by miR172, and overexpression of miRNA resistant proteins or mutations in the miR172 sequence result in indeterminate flowers [48,158]. Mutations affecting components of the miRNA processing machinery as *DICER-LIKE 1*, *ARGONAUTE (AGO) 1* or *AGO10* show a determinacy phenotype, partly because of their impact on miR172 activity [159–161]. Another factor in the AGO1 pathway is SQUINT (SQN), which is a cyclophillin 40 homolog and has been identified to enhance weak indeterminacy phenotypes of *crabs claw* (*crc*, see below) or *ag* [162–164]. SQN promotes the activity of AGO1 in plants via physical interaction with Heat shock protein 90 [165]. In addition to its role in floral meristem termination, Prunet et al. [166] propose an additional role for SQN in stem cell homeostasis via the CLAVATA signaling pathway.

The trxG protein ULT1 is a negative regulator of stem cell activity, indicated by supernumerary floral organs in *ult1* mutants, and it restricts *WUS* expression to the organizing center [37,38,163]. At the same time ULT1 interacts with ARABIDOPSIS HOMOLG OF TRITHORAX1 in order to reduce repressive H3K27me3 marks at the *AG* locus [39]. Recently, the Myb-domain TF ULTRAPETALA1 INTERACTING FACTOR1 (UIF) has been identified to interact with ULT1 [167]. UIP1 is able to bind *WUS* and *AG* genomic sequences *in vitro*, thereby creating the potential to recruit ULT1 to these loci.

In an enhancer screen using the weak *ag*-10 mutant allele, novel regulators of floral meristem determinacy have been identified recently. One of these identified genes encodes TOPOISOMERASE1α, which appears to have a general role decreasing nucleosome density in the genome, thereby mediating PcG recruitment to the *WUS* locus via an unknown mechanism [168]. In the same screen, *ARF3*, which has well-known roles in polarity establishment within leaves and carpels [169,170], has been identified as well [171]. *ARF3* confers its role via repression of *WUS* activity, and this partly depends on AG. *ARF3* is a target of AP2, and contributes to mediating its function in floral determinacy [171].

Moreover, target genes of AG may contribute to AG's function in floral determinacy, as has been shown in the case of the YABBY TF CRC. The *crc* single mutant has no defect in floral determinacy, but when combined with heterozygous *ag* or other mutants that act in floral determinacy, supernumerary whorls of floral organs can be observed [163,172]. *CRC* is a direct target of AG [125], but the exact molecular role of CRC in determinacy has not been unraveled yet. Another downstream target of AG is *SUPERMAN* (*SUP*), which encodes a C2H2-zinc finger TF that plays a role in boundary maintenance between 3rd and fourth whorl, indicated by supernumerary stamen on the expense of carpels in *sup* [173,174]. However, the exact molecular role of *SUP*, as in the case of *CRC*, in FM determinacy remains to be elucidated.

7. Understanding the networks underlying inflorescence and flower development: current status and outlook

Many important regulatory genes with roles in inflorescence and flower development have been identified over the last two decades. Basic genetic and molecular interactions have been characterized for some of these factors, largely boosted by the technical progress in genome-wide experimental analyses. It has now become clear that gene regulation in plant development is achieved at multiple levels: transcriptional regulation by TFs, epigenetic factors and (long) non-coding RNAs, posttranscriptional regulation by miRNAs, and regulation at the level of protein activity or stability. In particular the regulation at posttranslational level, e.g. the roles of posttranslational modifications of TFs or the assembly of regulatory complexes, is still poorly understood. On the other hand, it has become clear that there is a tight interplay between TFs and epigenetic regulation in the control of developmental transitions in plants, such as the floral transition and switches in meristem identity. A nice example is the conditional auxin-mediated recruitment of SWI/SNF2-type remodelers by MP, which triggers FM initiation [77]. It is also becoming clear that regulation of most developmentally controlled genes requires a combinatorial input of multiple TFs and other transcriptional regulators (see, e.g. [85,114,120,175,176]). The knowledge on genes and genetic interactions underlying flower development has enabled mathematical modeling approaches, which are able to generate novel, experimentally testable hypotheses on underlying mechanisms [177]. Live imaging using confocal microscopy generates quantitative data that can be used for computational morphodynamics approaches [213]. However, quantification of levels of regulatory molecules and their turnover at cellular resolution *in planta* remains an important challenge. This type of information will help us to understand the complex regulatory interactions that can be observed in gene-regulatory and hormonal signaling networks governing development.

While we do have a good knowledge of important regulatory processes in inflorescence and flower development, we still need to learn more about the impact of environmental fluctuations on specific gene-regulatory interactions. Genetic and molecular analyses are often performed under standardized environmental conditions, while in nature, plants are exposed to significant short and long term fluctuations, e.g. in temperature, light intensity/quality and water supply. It is clear that floral transition is highly adaptive in response to environmental factors, but how environmental robustness of inflorescence and flower development is achieved is largely unclear. Next to Arabidopsis, genes controlling inflorescence and flower development are now being systematically identified and analyzed in other model and crop plant species. Among the well-studied crop species are tomato (*Solanum lycopersicum*) and rice (*Oryza sativa*) [181,214,215]. Systematic evolutionary comparison of gene-regulatory networks provides rich information on conserved and non-conserved regulators and regulatory pathways, which has important impact on transferring knowledge from model to crop plant species. For instance, homologs of *UFO* and *LFY* have been implicated in inflorescence patterning in Solanum, along with other regulatory factors [181]. Comparative studies are therefore highly valuable in understanding the diversity and evolution of gene-regulatory networks.

In order to discern mechanisms of regulatory evolution, the comparison of closely related species and ecotypes may be particularly interesting (see, e.g. [216]). Here, the combination of genetic analyses (facilitated by novel genome editing tools such as RNA guided Cas9) with genome-wide functional genomics techniques (e.g. ChIP-seq) is likely to provide a new level of understanding of the evolution of inflorescence and flower development in angiosperms in the near future.

Transparency document

The Transparency document associated with this article can be found, in online version.

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