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Evolutionary Genetics of Core Eudicot Inflorescence and Flower Development

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ABSTRACT

The genetic basis of flowering is best understood in the model core eudicot species *Arabidopsis thaliana* (Brassicaceae), and involves the genetic reprogramming of shoot apical meristems, ending in the production of flowers. Although inflorescences and flowers of core eudicots share a common ground plan, variation in architecture, shape and ornamentation suggests repeated modifications to this ancestral plan. Comparative studies, primarily in Brassicaceae and Leguminoseae (rosids), and Asteraceae, Plantaginaceae and Solanaceae (asterids), have revealed a common developmental framework for flowering across core eudicots. This serves as a basis for understanding genetic changes that underlie the diversification of inflorescence and floral form. Recent work is starting to reveal the relative importance of regulatory versus protein coding changes in genes involved in diversification of inflorescence and flower development across core eudicots. Furthermore, these studies highlight the importance of phylogenetic history for understanding functional conservation of duplicated genes.

Keywords: *Antirrhinum majus*, *Arabidopsis thaliana*, Asteraceae, cyme, flower development, inflorescence development, Leguminoseae, *Petunia hybrida*, raceme, Solanaceae

Abbreviations: ***AG***, *AGAMOUS*; ***ALF***, *ABERRANT LEAF AND FLOWER*; ***AP1***, *APETALA1*; ***AP2***, *APETALA2*; ***AP3***, *APETALA3*; ***BL***, *BLIND*; ***CRC***, *CRABS CLAW*; ***EVG***, *EVERGREEN*; ***DEF***, *DEFICIENS*; ***DOT***, *DOUBLE TOP*; ***FAR***, *FARINELLI*; ***FBP6***, *FLORAL BINDING PROTEIN 6*; ***FIM***, *FIMBRIATA*; ***FIS***, *FISTULATA*; ***FUL***, *FRUITFULL*; ***GLO***, *GLOBOSA*; ***LFY***, *LEAFY*; **MADS-box**, *MINICHROMOSOME MAINTENANCE1-*, *AG-*, *DEFICIENS-*, *SERUM RESPONSE FACTOR*-like transcription factors; ***PI***, *PISTILLATA*; ***PLE***, *PLENA*; **SAM**, shoot apical meristem; ***SEP***, *SEPALLATA*; ***SQUA***, *SQUAMOSA*; ***SUP***, *SUPERMAN*; ***TFL1***, *TERMINAL FLOWER1*; ***UFO***, *UNUSUAL FLORAL ORGANS*; ***WUS***, *WUSCHEL*

CONTENTS

INTRODUCTION

The switch from vegetative to reproductive development initiates with the genetic reprogramming of shoot apical meristems (SAMs), resulting in the production of flowers (Blázquez *et al*. 2006). Flowering is initiated in response to a combination of external and internal floral inductive signals, such as photoperiod and developmental age, which are perceived across the whole plant, but are integrated within the adult vegetative SAM (for review see Bäurle and Dean 2006; Running 2006). During floral induction, increased levels of cell division cause the vegetative SAM to elongate, producing the inflorescence meristem. Unlike the vegetative SAM, which produces a combination of stems, branches and leaves, the inflorescence meristem produces a combination of branches, bracts and flowers. Flower development commences with the production of either lateral or terminal floral meristems, which eventually give rise to the various floral organs.

The genetic basis of flowering is best understood in the model species *Arabidopsis thaliana* (Brassicaceae). In *A. thaliana*, the floral transition is under the control of four interconnected genetic pathways. Two of these pathways (photoperiod and vernalization) perceive and transport signals from the external environment, while the other two (autonomous and gibberellic acid) perceive and transport signals from the internal environment. The endpoints of these genetic pathways are the floral integrator genes that signal the SAM to transition to flowering (Baürle and Dean 2006). Upon floral transition, the SAM is reprogrammed to develop an inflorescence. Inflorescences of *A. thaliana* are indeterminate branching structures (racemose, open and monopodial), which initiate floral primordia spirally along the main axis (Reinhardt and Kuhlemeier 2002; Yamaguchi

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Invited Review

et al. 2007). Architecture of the *A. thaliana* inflorescence is controlled by the interplay of inflorescence meristem identity genes that either promote or repress floral meristem identity genes (for review see Running 2006; Benlloch *et al*. 2007). Once upregulated, the floral meristem identity genes promote determinacy within the lateral floral meristems. Floral determinacy results from the repression of branching, and the initiation of a suite of floral patterning and organ identity genes, which promote the development of floral organs.

Decades of work on *A. thaliana* have given us much insight into the genetic basis of flowering (for review see Amasino 2010; Irish 2010). However, to address the level of conservation of these developmental genetic pathways across major clades of angiosperms, comparable studies are required in other species. For example, studies have revealed some conservation in the inflorescence developmental genetic pathways between distantly related model eudicots (e.g. *A. thaliana*) and monocots (e.g. *Zea mays*) (e.g. Bomblies *et al*. 2003; Whipple *et al*. 2006; for review see Bommert *et al*. 2005; McSteen and Leyser 2005; Malcomber *et al*. 2006; Thompson and Hake 2009). This suggests a common ground plan for inflorescence development across the angiosperms, with subsequent modifications underlying morphological diversification. Here, I focus on recent evidence for genetic conservation of flowering in the core eudicots, and assess how this developmental genetic network may have been modified to affect diversification of form within this large and morphologically diverse group of angiosperms. To set the stage, I will briefly outline some major evolutionary transitions in core eudicot inflorescence and flower morphology.

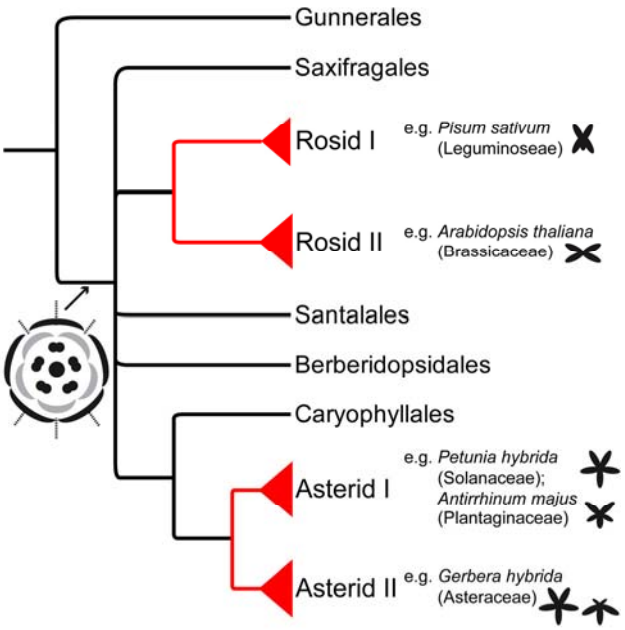

Fig. 1 Simplified hypothesis of phylogenetic relationships and flower evolution in core eudicots (based on APG II 2003; Soltis *et al*. 2005; Schönenberger and Balthazar 2006). It is hypothesized that, following the diversification of the Gunnerales, the ancestral flower of all other core eudicots had two distinct perianth whorls (sepals in black, petals in gray), few stamens, an inner gynoecium, and was polysymmetrical (dotted lines) (de Craene 2007). Polysymmetry has been lost and modified multiple times, as indicated by the floral diagrams for model species in the large rosid and asterid clades (red).

INFLORESCENCE AND FLOWER DIVERSITY IN CORE EUDICOTS

Core eudicots are a well-supported monophyletic clade of eudicots comprising seven subclades, Gunnerales, Caryophyllales, Santalales, Berberidopsidales, Saxifragales, rosids, and asterids (Soltis *et al*. 2000, 2003; APG II 2003) (**Fig. 1**). *A. thaliana* is a member of the rosid clade, which contains an estimated one-third of all angiosperm species (Magallón *et al*. 1999), including members of the pea (Leguminoseae) and rose (Rosaceae) families (**Fig. 1**).The other major clade in the core eudicots is the asterids, containing an estimated 25% of all angiosperm species (Bremer *et al*. 2001), including the snapdragon (Plantaginaceae), nightshade (Solanaceae), and sunflower (Asteraceae) families (**Fig. 1**). Most genetic studies on inflorescence and flower development have been conducted on model species in the Asteraceae, Brassicaceae, Leguminoseae, Plantaginaceae and Solanaceae. Accordingly, inflorescence development in rosids and asterids will be the main focus of this review.

Flowers of core eudicots are similar in overall organization (**Fig. 1**), but there is remarkable variation in both floral architecture (e.g. shape) and mode (e.g. specific adaptations to pollinators) (*sensu* Soltis *et al*. 2005). Most core eudicot flowers have a closed ground plan, with a fixed number of organs arranged in alternating whorls (Endress 2001; Soltis *et al*. 2003). Unlike early-diverging eudicots, the number of organs per whorl shows little variation; it has been hypothesized that genetic canalization has occurred for this trait, partly through neo-functionalization of core eudicot-specific floral organ identity genes (see later) (Endress 1990; Kramer and Irish 1999; Litt and Irish 2003; de Martino *et al*. 2006; but see Soltis *et al*. 2003). Additionally, the typical core eudicot flower has distinct sepals and petals (APG II 2003; Soltis *et al*. 2005; de Craene 2008). In most cases, the sepals are hardy structures that serve a protective function, whereas, the petals are short-lived delicate structures that are attractive to potential pollinators.

In contrast to organ number, flower shape and organ fusion is highly labile in core eudicots, particularly in the asterids. For example, floral zygomorphy (bilateral symmetry) is thought to have evolved at least 15 times independently in asterids, with approximately ten reversals to actinomorphy (Donoghue *et al*. 1998; Jabbour *et al*. 2008; Knapp 2010) (**Fig. 1**). Furthermore, ancestral character state reconstructions suggest that the evolution of zygomorphy may be contingent upon a fixed perianth organ number of five, and that zygomorphy may be required for the acquisition of both petal spurs and possibly fusion between petals and stamens (Soltis *et al*. 2005; Jabbour *et al*. 2008) (**Fig. 1**).

Inflorescence architecture is also highly variable in core eudicots, mainly as a result of differences in the number and size of bracts and flowers, the ratio of branches and flowers, and the position of organs on the inflorescence axis (phyllotaxy) (Weberling 1989; Reinhardt and Kuhlemeier 2002; Singer 2006) (**Fig. 2**). For example, the main inflorescence meristems of determinate (cymose, closed and sympodial) inflorescences (e.g. *Petunia hybrida* [Solanaceae]) terminate in a flower, while in indeterminate (racemose) inflorescences (e.g. *A. thaliana* and *Antirrhinum majus* [Plantaginaceae]) they do not (**Fig. 2**). Instead, flowers develop from lateral primordia in a specific phyllotaxy along the inflorescence axis. Inflorescences also differ in the presence (e.g. *A. majus*) or absence (e.g. *A. thaliana*) of floral bracts (leaf-like structures subtending flowers) and prophylls (leaf-like structures preceding flowers), and in the order of branching, which can be simple (e.g. *A. thaliana*) or compound (e.g. double racemes of *Pisum sativum* [Leguminoseae]) (Prenner *et al*. 2009) (**Fig. 2**). All this variation in inflorescence and floral traits make up the wealth of inflorescence diversity in the core eudicots, being restricted only by the nature of shared developmental programs and external selective pressures (Prusinkiewicz *et al*. 2007).

FLOWER DEVELOPMENT

Floral organ identity and patterning in *A. thaliana*

The production of whorled organ primordia from lateral floral meristems is achieved both by repression of genes that produce the floral meristem, and through activation of

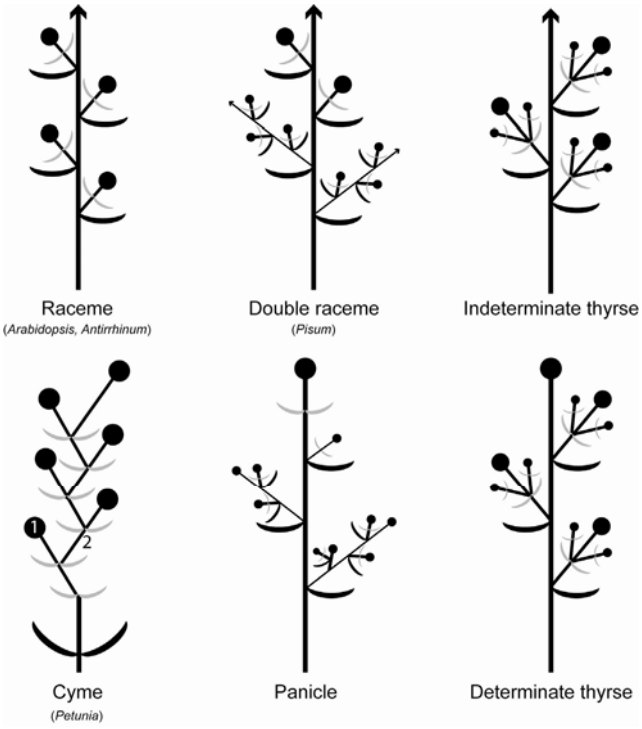

Fig. 2 Variation in core eudicot inflorescence architecture. Inflorescences are either indeterminate (upper diagrams) (e.g. *Antirrhinum majus*; Plantaginaceae) or determinate (lower diagrams) (e.g. *Petunia hybrida*; Solanaceae), and vary in orders of branching (e.g. simple versus double raceme) and the position of branches relative to bracts (black crescents) and prophylls (gray crescents). Lateral flowers develop in the axils of bracts in racemes and panicles, and in the axils of prophylls in cymes and thyrses (based on Prenner *et al.* 2009). Depending on the species, bracts and prophylls may be present (as shown here) or absent. Arrows, indeterminate meristems; circles, floral meristems; 1, apical meristem of a cymose inflorescence terminating in a flower; 2, lateral meristem of a cymose inflorescence giving rise to a bifurcating branch.

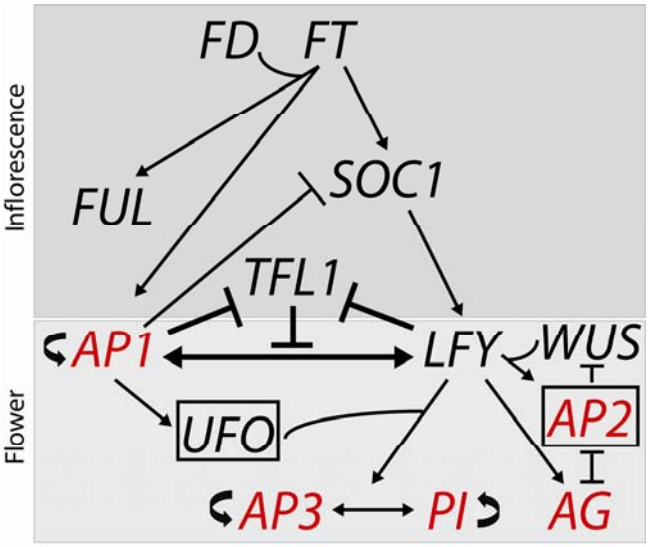

Fig. 3 Major genetic interactions regulating inflorescence (upper panel) and flower (lower panel) development in *Arabidopsis thaliana* (Brassicaceae). Inflorescence architecture is primarily defined by the negative interactions of the inflorescence meristem identity gene *TERMINAL FLOWER1* (*TFL1*) and the floral meristem identity genes *LEAFY* (*LFY*) and *APETALA1* (*AP1*) (thick lines). Arrowheads denote transcriptional activation, whereas blunt ended lines denote transcriptional repression. ABC class genes involved in specification of floral organ identity are in red. Genes known to be regulated by microRNAs are boxed.

genes that initiate and maintain organ development. In *A. thaliana*, floral organ development proceeds from the outer to inner whorl, resulting in the formation of four sepals, four petals, six stamens, and the inner two fused carpels. Many of the genes responsible for organ development also have an earlier role in the specification of floral meristem identity (see later) (**Fig. 3; Table 1**). Although not mutually exclusive, it is postulated that these dual roles can be explained through tissue specific interactions of flower deve-

Table 1 Key regulators of inflorescence and flower development in *Arabidopsis thaliana* and other model core eudicots.

***ARABIDOPSIS* GENE FUNCTION**	**SPECIES/GENES**[1,2]				
	Arabidopsis	***Pisum***	***Antirrhinum***	***Petunia***	***Gerbera***
Flowering meristem indeterminacy	*WUS*				
	STP			*EVG*	
	TFL1	*DET*, *LF*, *PsTFL1*	*CEN*		
Inflorescence meristem identity	*FUL*	*PsFUL*	*AmFUL*, *DEFH28*	*PhFBP26*, *PhPFG*, *PhFL*	
	SPL3,4,5		*SBP1,2*		
Floral meristem identity	*LFY*	*UNI*	*FLO*	*ALF*	
	UFO		*FIM*	*DOT*	
	*SVP**		*INCO*		
	*AGL24**				
Floral meristem and floral organ identity	*AP1*, *CAL*	*PM9*, *PIM*	*SQUA*	*PhFBP29*	*GSQUA1*
	AG	*PMADS3*, *FBP6*	*FAR*	*PhpMADS3*	*GAG1,2*
	SEP1,2	*PsSEP1,2*	*AmDEFH49*	*PhFBP5,9,23*	*GRCD1,2*
	SEP3	*PsMADS3*	*AmDEFH72,200*, *AmSEP3b*	*PhFBP2*	
	SEP4			*PhFBP4*	
	*AP2**		*LIP1,2*	*PhAP2A*	
Floral organ identity	*AP3*		*DEF*	*PhpMADS1*, *PhTM6*	*GDEF1,2*
	PI	*PsPI*	*GLO*	*PhGLO1,2*	*GGLO1*
Flowering gene silencing (microRNA)	miR156				
	miR169		*FIS*	*BL*	
	miR172				
Negative regulation of *AG**	*LUG*		*STY*		
	SEU				
	ANT				
Floral organ development	*CUC1,2*		*CUP*	*NAM*	
	SHP1,2	*PsSHP*	*PLE*	*PhFBP6*	
Flower symmetry	*TCP1*	*PsCYC1,2*	*CYC*, *DICH*		*GhCYC2,3,4*
Nectaries	*CRC*			*PhCRC*	

[1]References in text, supplemented with Leseberg (2008)
[2]Gaps represent missing data rather than gene loss

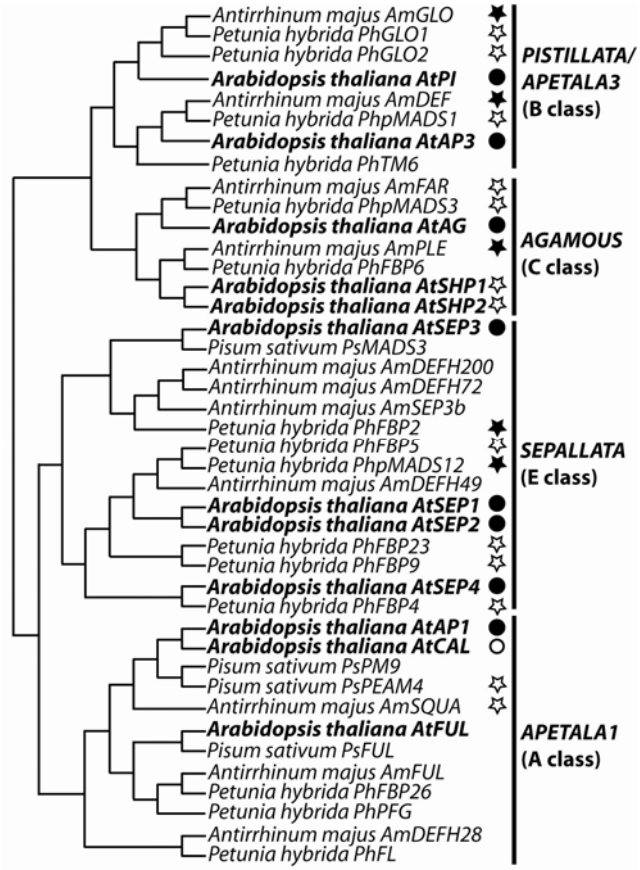

Fig. 4 Phylogenetic relationships among the *Arabidopsis thaliana* (Brassicaceae; rosid) (in bold) ABC and *SEP* (E) flower identity MADS-box genes and their homologues in the other core eudicot species *Pisum sativum* (Leguminoseae; rosid), *Antirrhinum majus* (Plantaginaceae; asterid), and *Petunia hybrida* (Solanaceae; asterid). Each major lineage is comprised of genes from both recent and ancient duplication events, resulting in multiple paralogous genes per individual species, which may or may not be strictly orthologous to related genes of other species. Genetic evidence suggests some instances of functional conservation between the *A. thaliana* organ identity genes (filled circles) and homologues of other species (filled stars), as well as instances of only partially functional conservation between the *A. thaliana* organ identity genes and homologues of other species (open stars). Likewise, developmental function can be conserved or only partially conserved (open circles) between *A. thaliana* organ identity genes and *A. thaliana* paralogues in the same gene lineage. Genes without symbols either have novel functions (discussed in the text) or their functions are unknown.

lopmental proteins (Castillejo *et al*. 2005; Gregis *et al*. 2008).

Central to our understanding of *A. thaliana* flower development is the ABC model, which proposes three classes of proteins that act in combination to specify the identity of the four floral organs (Coen and Meyerowitz 1991). A-class genes specify the identity of sepals and petals, B-class genes specify the identity of petals and stamens, and C-class genes specify the identity of stamens and carpels. The floral quartet model further predicts another class of proteins (SEPALLATA [SEP] and E-class) that act as cofactors of the ABC proteins (Pelaz *et al*. 2000; Honma and Goto 2001; Theissen 2001a, 2001b; Theissen and Saedler 2001; Ditta *et al*. 2004). Specific tetrameric complexes or 'floral quartets' composed of A, B, C, and SEP proteins are postulated to regulate the transcription of organ specific downstream targets (Honma and Goto 2001; Melzer *et al*. 2009; Melzer and Theissen 2009) (**Fig. 4**). Evidence for this comes from the transformation of leaves into petaloid and staminoid organs following ectopic expression of a specific subset of ABC and *SEP* genes, and *in vitro* binding assays (Honma and Goto 2001; Melzer and Theissen 2001; Melzer *et al*. 2009). Furthermore, according to these models, the A- and C-class genes are mutually antagonistic (Bowman *et al*. 1991b). Thus, A-class genes negatively regulate C-class genes in the first two whorls, and C-class genes negatively regulate A-class genes in the third and fourth whorls.

In addition to their role in floral meristem identity specification (see later), *APETALA1* (*AP1*) and *APETALA2* (*AP2*) are the putative *A. thaliana* A-class genes (Irish and Sussex 1990; Mandel *et al*. 1992; Weigel and Meyerowitz 1993) (**Fig. 3; Table 1**). Consistent with the ABC model, *ap1* and *ap2* mutants show abnormal development in the first two floral whorls. However, whereas *ap1* mutants develop leaf-like organs in the first whorl, and either lack organs or develop sepaloid petals in the second whorl, the first two whorl organs of *ap2* mutants are homeotically transformed to carpels and stamens, respectively (Irish and Sussex 1990; Mandel *et al*. 1992; Weigel and Meyerowitz 1993). The mutant phenotype of *ap2* is caused by ectopic expression of the sole C-class gene *AGAMOUS* (*AG)* in the first two whorls, consistent with antagonism between A- and C-class genes (Bowman *et al*. 1991a). Although *AP2* is expressed in all floral whorls, negative post-transcriptional regulation by microRNAs probably confines AP2 protein to the perianth whorls (Chen 2004). Indeed, it is becoming clear that microRNAs are critical regulators of transcription factors in both flower and inflorescence development (reviewed in Chuck *et al*. 2009; next section) (**Fig. 3**).

Direct negative regulation of *AG* has not been demonstrated for *AP1*. However, Gregis *et al*. (2008) recently hypothesized that, during early flower development, protein complexes of AP1, SHORT VEGETATIVE PHASE (SVP) and AGAMOUS LIKE 24 (AGL24) negatively regulate *AG* indirectly through the upregulation of *LEUNIG* (*LUG*) and *SEUSS* (*SEU*) (Sridhar *et al*. 2006) (**Table 1**). Slightly later in flower development, the authors suggest that AP1 proteins switch their protein partners from SVP and AGL24 to the newly present SEP1-4 proteins; evidence from yeast two- and three-hybrid studies suggest heterodimer formation of SEP-SEP-CAULIFLOWER (CAL)-AP1 in sepals and SEP-AP1-APETALA3 (AP3)-PISTILLATA (PI) in petals (Castillejo *et al*. 2005). Instead of negatively regulating *AG*, these organ specific complexes negatively regulate *SVP* and *AGL24*, acting as switches between floral meristem and floral organ development (Yu *et al.* 2004a; Gregis *et al*. 2008). Thus, similar to the closely related SEP proteins (**Fig. 4**), AP1 may not have a specific role in specifying organ identity. However, AP1 is likely important for the development of some floral organs, the identity of which are specified by other MADS-box proteins (e.g. petals by the B-class proteins AP3 and PI; discussed later) or represent the ground floral state of organs (e.g. sepals).

Unlike *AP1*, *AP2* and *AG*, *AP3* and *PI* do not have a role in floral meristem identity. Instead they function exclusively in the second and third whorl to initiate and maintain the development of petals and stamens (Jack *et al*. 1992; for review see Irish 2008) (**Fig. 3; Table 1**). In *ap3* and *pi* mutants, petals and stamens are homeotically transformed into sepals and carpels, respectively, consistent with their predicted B-class function (Bowman *et al*. 1989). Expression of both genes appears to be regulated temporally by the floral meristem identity proteins LEAFY (LFY) and AP1, and members of the gibberellic acid pathway, and spatially by both UNUSUAL FLORAL ORGANS (UFO) and AP3-PI dimers (Weigel and Meyerowitz 1993; Goto and Meyerowitz 1994; Lee *et al*. 1997; Blázquez *et al*. 1998; Zhao *et al*. 2001; Yu *et al*. 2004b) (**Fig. 3**). In addition to regulating *AP3* and *PI*, *UFO* is also a positive regulator of genes that define the boundaries between organs, such as *RABBIT EARS* (*RBE*) (Takeda *et al*. 2004; Krizek *et al*. 2006; for review see Irish 2008). *RBE* is exclusively expressed in young petal primordia, where it (in combination with *AP2* and the closely related gene *AINTEGUMENTA* (*ANT*), *SEU*, *LUG* and *STERILE APETALA* [*SAP*]) negatively regulates the expression of *AG* (Drews *et al*. 1991; Byzova *et al*. 1999; Krizek *et al*. 2000). It is hypothesized that petal dif-

ferentiation is established through the physical interaction of SEP3, AP1 and B-class proteins, which in turn upregulate genes required for petal development (Pelaz *et al*. 2000, 2001; Ditta *et al.* 2004; Castillejo *et al.* 2005). Genetic evidence suggests that petal size and abaxial identity are specified in a parallel pathway by *ANT* (Krizek *et al*. 2000); in early stages of flower development adaxial identity is specified by the TCP gene *TCP1* (Cubas *et al*. 2001).

Evidence that *AG* negatively regulates *AP1* in the inner two whorls comes from expression analyses of *ag* mutants. In addition to losing determinacy in the carpel whorl, partly due to the ectopic expression of the meristem gene *WUSCHEL* (*WUS*), *ag* mutants develop petals in place of stamens and sepals in place of carpels (Bowman *et al*. 1989; Laux *et al*. 1996; Lenhard *et al.* 2001). This phenotype is due both to the loss of AG protein *per se* and possibly the ectopic expression of *AP1* and/or *AP2* in the third and fourth whorls. In wild-type plants, *AP3* and *PI* are excluded from the fourth whorl by the action of *SUPERMAN* (*SUP*). *SUP* functions with *FLORAL ORGAN NUMBER1* (*FON1*), *CUP SHAPED COTYLEDON 1* (*CUC1*) and *CUC2* to repress floral meristem activity, promote organ identity, and define organ boundaries by reducing cell proliferation (Bowman *et al.* 1992; Aida *et al.* 1997; Huang and Ma 1997; Jacobsen and Meyerowitz 1997).

AG, and paralogous genes *SHATTERPROOF1* (*SHP1*) and *SHP2*, function upstream to the YABBY gene *CRABS CLAW* (*CRC*) to specify carpel identity (Alvarez and Smyth 1999; Bowman and Smyth 1999; Liljegren *et al.* 2000; Pinyopich *et al*. 2003; Lee *et al.* 2005a) (**Fig. 4**). Similar to other YABBY family genes, *CRC* promotes cell differentiation exclusively in the abaxial domain of the carpel, resulting in the control of carpel polarity (Eshed *et al*. 1999). A secondary role for *A. thaliana CRC* is in the formation of nectaries that develop at the base of stamens; in *crc* mutants, nectaries are entirely absent. In *A. thaliana*, constitutive expression of *CRC* does not result in ectopic nectary formation. This suggests that *CRC* is required but not sufficient for nectary development (Baum *et al*. 2001). Genetic analyses in different floral mutant backgrounds also suggest that *CRC*-dependent nectary production is independent of floral organ identity (Baum *et al*. 2001). Despite this, promoter analyses implicate floral organ identity MADS-box genes, including *SEP* genes, *AP2* and *LFY* in the spatial and quantitative regulation of *CRC* (Lee *et al*. 2005a).

Duplication and diversification of flower developmental genes in core eudicots

Although functional analyses in species other than *A. thaliana* have revealed broad conservation of the ABC and floral quartet models across core eudicots, both ancient and recent gene duplications impede simple extrapolation of the *A. thaliana* floral model across this plant lineage (**Fig. 4**). Furthermore, despite a similar floral ground plan, differences in shape and ornamentation suggest significant modification to the genetic basis of flower development in different core eudicot lineages. Evidence for both conservation and diversification of the developmental genetic basis of flowering come from comparative studies, particularly in the Asteraceae, Brassicaceae, Leguminoseae, Solanaceae and Plantaginaceae, some of which will be discussed here (for other reviews see Efremova *et al*. 2006; Rijpkema *et al*. 2006a; Teeri *et al*. 2006a).

Genetic evidence from *A. majus* suggests that B-function is specified by homologues of the *A. thaliana AP3* and *PI* genes (Saedler and Huijser 1993) (**Table 1**). As is the case for AP3 and PI, the *A. majus* B-class proteins DEFICIENS (DEF) and GLOBOSA (GLO) function as obligate heterodimers, and positively regulate their own transcription in the second and third whorls (Schwartz-Sommer *et al*. 1992; Tröbner *et al*. 1992). Mutations in either of these genes cause homeotic transformation of petals to sepals and stamens to carpels (Schwartz-Sommer *et al.* 1992). Although similar functional conservation has been found for *P. hybrida AP3/PI* genes as a whole, genetic studies have revealed lineage specific gene duplications followed by partitioning of function (subfunctionalization). Thus, both *P. hybrida* co-orthologues of *PI* (*PhGLO1* and *PhGLO2*) are required to fulfill *PI* function, and both the *P. hybrida AP3* orthologue (*PhDEF*) and paralogous *PhTM6* (which has been lost in *A. thaliana*) are required to fulfill *AP3* function (**Fig. 4; Table 1**) (van der Krol *et al*. 1993; Vandenbussche *et al.* 2004; Rijpkema *et al*. 2006b). A dual requirement of *TM6* and *AP3* genes has also been demonstrated for B-function in other Solanaceae species, including *S. lycopersicum*, and *Gerbera hybrida* (Asteraceae) (Yu *et al*. 1999; de Martino *et al*. 2006; Rijpkema *et al*. 2006b).

Similar to *AG*, the *A. majus* gene *PLENA (PLE)* conveys C-function to the flower; loss-of-function *ple* mutants show homeotic transformation of stamens to petals and carpels to sepals (Bradley *et al*. 1993). Interestingly, although *AG* and *PLE* are related MADS-box genes, they are not orthologous (**Fig. 4; Table 1**). Phylogenetic and genomic studies have demonstrated that the *A. majus* gene *FARINELLI* (*FAR*) is the orthologue of *AG*, and that the recently duplicated *A. thaliana* genes *SHP1* and *SHP2* are co-orthologous to *PLE* (Kramer *et al*. 2004; Causier *et al*. 2005). *FAR* (and its orthologues *pMADS3* in *P. hybrida* and *GAG1/2* in *G. hybrida*) and *SHP1/2* are involved in stamen and fruit development, respectively (Davies *et al*. 1999; Yu *et al*. 1999; Liljegren *et al*. 2000; Kapoor *et al*. 2002). It is hypothesized that the ancestor of these duplicated genes functioned in both male and female reproductive development, and that differential subfunctionalization of paralogues in the asterid and rosid lineages led to non-orthologous similarities in gene function (Causier *et al*. 2005). Similar divergent patterns of subfunctionalization have been found for *SEP*-like genes of *P. hybrida* and *G. hybrida* (Kotilainen *et al.* 2000; Vandenbussche *et al*. 2003; for review see Krizek and Fletcher 2005; Malcomber and Kellogg 2005; Teeri *et al*. 2006b).

Unlike B- and C-class genes, conservation of function between A-class gene orthologues of *A. thaliana* and core eudicots outside Brassicaceae is limited. For example, in addition to a role in meristem identity, the putative *A. majus* co-orthologues of *AP2 – LIPLESS1* (*LIP1*) and *LIP2 –* share a redundant role in the development of all four floral organs. Double *lip1:lip2* mutants have leaf-like sepals, petals that lack lips, reduced stamens, and female organs with low fertility (Keck *et al.* 2003). Similar to *AP2*, *LIP1* and *LIP2* are expressed in all four organs. However, unlike *AP2*, which is spatially regulated by an miR172 family microRNA, the *lip1:lip2* mutant phenotype suggests incomplete or absent post-transcriptional regulation in the inner two whorls. *LIP1* and *LIP2* also differ from *AP2* in their inability to negatively regulate the C-class gene *PLE*. Differences between *AP2* and *LIP1/LIP2* may be due to functional diversification following the divergence of *A. thaliana* and *A. majus*, or may reflect non-orthology between these genes (Litt 2007). Alternatively, differential partitioning of ancestral function between *AP2*- and *ANT*-like genes following divergence of asterids and rosids may explain functional differences, as in the case of C-class genes (Causier *et al*. 2005; discussed in Litt 2007). Better sampling of *AP2*-like genes for phylogenetic and functional studies is required to test these alternative hypotheses.

Comparison of mutant phenotypes also suggests functional differences between *A. thaliana AP1* (A-class) and *A. majus SQUAMOSA (SQUA)* genes. Although both genes share a conserved role in specification of floral meristem identity (see later), unlike *ap1* mutants, *squa* mutants are able to produce normal flowers. This either suggests that *SQUA* is not required for floral organ development, or that it is functionally redundant with other genes. Despite little evidence for specification of A-function *sensu stricto*, the latter hypothesis is partly supported. Firstly, the first few flowers of *squa* mutants are usually abnormal, often forming bract-like or petal-like sepals, leaf-like or sepal-like petals, and petaloid stamens (Huijser *et al.* 1992). Secondly,

double *squa:def* or *squa:glo* mutants have more abnormal floral organ phyllotaxy and organ number defects than do single mutants (Egea-Cortines *et al*. 1999). Yeast three-hybrid studies show that SQUA-DEF-GLO bind to CArG motifs, commonly found in promoters of MADS-box genes, with higher affinity than do DEF/GLO heterodimers or SQUA/SQUA homodimers (Egea-Cortines *et al.* 1999). This suggests that DEF/GLO heterodimers may be able to partially substitute for SQUA-DEF-GLO complexes under certain conditions, but that *SQUA* does have a role in proper floral organ development. Thirdly, *SQUA* and the *A. majus LFY* orthologue *FLORICAULA* (*FLO*) are actively upregulated by *STYLOSA* (*STY*) and the miR169 family microRNA *FISTULATA* (*FIS*) in the outer two whorls. Similar to the *STY* orthologue *LUG* in *A. thaliana*, this results in the negative regulation of the C-class gene *PLE* in these whorls (Motte *et al*. 1998; Navarro *et al*. 2004; Cartolano *et al*. 2007). Ectopic expression of *SQUA* in the inner two whorls of *ple* mutants also implicates *PLE* in the negative regulation of *SQUA* (Motte *et al*. 1998). Finally, *A. majus* has two more *SQUA*-like genes – *AmDEFH28* and *AmFUL* – that potentially share redundant function with *SQUA* (**Fig. 4**) (Müller *et al*. 2001; Litt and Irish 2003). Since *AmDEFH28* is not expressed in sepals, it is unlikely to function in these organs; *AmFUL* is expressed in both sepals and petals, but its function has not been determined (Preston and Hileman 2010).

As in *A. thaliana* and *A. majus*, expression of the legume *AP1/SQUA* orthologues *PROLIFERATING INFLORESCENCE MERISTEM* (*PIM*) of *Pisum sativum* (**Fig. 4; Table 1**) and *MTPIM* of *Medicago truncatula* is restricted to the outer two whorls of the flower (Berbel *et al*. 2001; Benlloch *et al*. 2006). Flowers of *pim* and *mtpim* mutants are either indeterminate or have organ identity defects (Taylor *et al*. 2002; Benlloch *et al*. 2006) in the first three whorls. Since petals and stamens develop from common primordia in these species, abnormal division of these organs suggests a role for *PIM* in both floral organ identity and organ boundary specification (Taylor *et al*. 2002; Benlloch *et al*. 2006). In *P. hybrida*, mutations at the *BLIND* (*BL*) locus cause homeotic transformation of first whorl organs to carpels and second whorl organs to stamens (Kater *et al*. 1998; Maes *et al*. 2001). This phenotype is due to ectopic expression of the *AG* orthologues *pMADS3* and *FLORAL BINDING PROTEIN 6* (*FBP6*).

The gene underlying the *BL* locus was recently cloned, and found to be the miR169 family microRNA miRBL (Cartolano *et al*. 2007). Members of the miR169 family negatively regulate *NF-YA* genes, which are known positive regulators of *AG*-like genes. In *P. hybrida*, miRBL is expressed in all whorls of the flower, but its indirect negative regulation of *AG* appears to be strongest in the first two whorls. In *A. majus*, a similar microRNA (miRFIS) was shown to underlie the *FIS* locus (Cartolano *et al.* 2007). Similar to *P. hybrida*, *fis* mutants resemble *ap2* mutants in having second whorl organs that are homeotically transformed to stamenoid petals. The similarity between *ap2* and *blind/fis* mutants appears to be due to convergent negative regulation of *AG* and *PhFBP6*/*PLE*, respectively. In *A. thaliana*, *AG* is negatively regulated by *AP2*, which itself is spatially restricted by the action of miR172 (Chen 2004). By contrast, the *AP2* orthologues of *A. majus* and *P. hybrida* do not negatively regulate *AG*. Rather, negative regulation of *PhFBP6* and *PLE* is mediated through the indirect action of miRBL and miRFIS, and the *UFO* orthologues *DOUBLE TOP* (*DOT*) and *FIMBRIATA* (*FIM*) (Cartolano *et al.* 2007).

In addition to floral organ identity, there is increasing evidence to suggest some conservation of floral organ boundary specification within core eudicots. However, as for homologues of the ABC and *SEP* genes, lineage specific gene duplications have led to different patterns of functional redundancy, and are possibly linked to interspecific differences in organ fusion (Weir *et al*. 2004). In *A. majus*, the *UFO* orthologue *FIM* regulates organ fusion and phyllotaxy by negatively regulating *CHORIPETALA* (*CHO*) and *DESPENTEADO* (*DESP*), themselves negative regulators of B-class genes in the first whorl (Simon *et al*. 1994; Egea-Cortines *et al*. 1999; Wilkinson *et al*. 2000). Similarly, *P. hybrida PhSUP* can partly complement the *A. thaliana sup* organ boundary mutant (Nakagawa *et al*. 2004). The NAC-family genes, *NO APICAL MERISTEMS* (*NAM*) of *P. hybrida* and *CUPULIFORMIS* (*CUP*) of *A. majus*, also specify organ boundaries. These genes are most closely related to *A. thaliana CUC2* (Weir *et al.* 2004). As predicted, *cup* mutants have supernumerary floral organs that are more highly fused than wild type (Weir *et al*. 2004). This phenotype is more severe than for *cuc2* mutants, an observation that can be explained by redundancy between *CUC2* and *CUC1*. On the other hand, the only floral phenotype of *nam* mutants is increased organ number, suggesting redundancy with other genes (Souer *et al*. 1996; Rijpkema *et al*. 2006a). The fact that *NAM/CUC* genes are important for reducing organ fusion makes these good candidates for variation in this trait. For example, corolla tubes are more common in asterids than rosids. Furthermore, flowers of *cup* mutants are less bilaterally symmetrical than wild type *A. majus* flowers. This is likely due to the role of *CUP* in the regulation of TCP family genes involved in cell division, such as TCP-Interacting with *CUP* (*TIC*), and possibly the dorsal flower identity genes *CYCLOIDEA* (*CYC*) and *DICHOTOMA* (*DICH*), the latter of which has not been tested (Weir *et al*. 2004).

The majority of Lamiales species (e.g. *A. majus*) have bilaterally symmetrical flowers; phylogenetic analyses strongly suggest that this trait has been independently derived multiple times in core eudicots (Ree and Donoghue 1999). In *A. majus*, bilateral flower symmetry is controlled by *CYC* and *DICH*, and the MYB transcription factors *RADIALIS* (*RAD*) and *DIVARICATA* (*DIV*) (Luo *et al*. 1996, 1999; Galego and Almeida 2002; Corley *et al*. 2005). Interestingly, despite independent origins of bilateral flower symmetry in Brassicaceae and Leguminoseae, a similar role in dorsal identity specification has recently been demonstrated for *CYC/DICH* homologues of *Iberis amara*, *Lotus japonicus* and *P. sativum*, respectively (Feng *et al.* 2006; Busch and Zachgo 2007; Wang *et al*. 2008; for review see Preston and Hileman 2009). Furthermore in *G. hybrida*, a species that has both bilaterally and radially symmetrical flowers within the same inflorescence, differential expression of *CYC*-like genes have been implicated in morphological differences in flower shape (Broholm *et al*. 2008) (**Fig. 1**). Since radially symmetrical flowers of *A. thaliana* show dorsal expression of the *CYC/DICH* homologue *TCP1* up until sepal initiation, but not later during floral organ differentiation, this suggests independent recruitment of *CYC*-like genes in bilateral symmetry from a dorsally regulated ancestral gene (Cubas *et al*. 2001).

In addition to flower shape and perianth fusion discussed above, presence or absence of floral nectaries – a variable trait across core eudicots – has important implications for pollinator attraction. In *A. thaliana*, nectar-bearing organs develop at the base of stamens and their development is regulated by *CRC* (Alvarez and Smyth 1999; Bowman and Smyth 1999). In *P. hybrida*, nectaries develop at the base of ovaries, and silencing of the *CRC* orthologue results in plants that lack nectaries (Lee *et al*. 2005b). It is unknown whether the ancestor of core eudicots had floral nectaries; ancestral character state reconstructions are equivocal. However, a simple explanation for *CRC* function in both asterid and rosid floral nectaries (as well as rosid extrafloral nectaries; Lee *et al*. 2005b) is that the ancestral gene was involved in nectary production, and was repeatedly modified spatially by modifications in upstream regulators (Lee *et al*. 2005b). Further analyses are required to more vigorously test this hypothesis.

INFLORESCENCE DEVELOPMENT

A genetic framework for inflorescence development in *A. thaliana*

Similar to the onset of flowering (for review see Bäurle and Dean 2006) and flower development, inflorescence development involves the interaction of two antagonistic pathways that at once repress and promote determinacy in the inflorescence meristem (**Fig. 3**). Although many genes are known to be involved, the inflorescence meristem identity gene *TERMINAL FLOWER1* (*TFL1*) and the floral meristem identity genes *LFY* and *AP1* are amongst the key regulators of this developmental transition (**Fig. 3**). Recent studies are starting to reveal that, in addition to the relative levels of expression, the location and timing of inflorescence developmental gene expression may have major implications for inflorescence architecture. In this section I briefly review the extensive literature on the developmental genetic basis of inflorescence development in *A. thaliana*, as a basis for comparison with other core eudicots (**Fig. 3; Table 1**). Other excellent reviews on this subject include Jack (2004), Blázquez (2006), and Benlloch *et al*. (2007).

Inflorescence development in *A. thaliana* is initiated when the floral integrator genes, *FLOWERING LOCUS T* (*FT*) and *FLOWERING LOCUS D* (*FD*), together activate a set of downstream activators of flowering, including the MADS-box transcription factors *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*), *FRUITFULL* (*FUL; AGL8*) and *AP1*, and *LFY* (Wigge *et al*. 2005) (**Fig. 3; Table 1**). Prior to the production of floral meristems, the conversion of the vegetative SAM to an inflorescence meristem is marked by the upregulation of *TFL1* and *TFL2*, *EMBRYONIC FLOWER 1* and *2* (*EMF1* and *EMF2*), *SVP*, *AGL24*, *SOC1*, *FUL*, and *LUMINIDEPENDENS* (*LD*). *TFL1* is closely related to *FT*, and both genes are recruited by *FD* to the promoter of floral genes (Wigge *et al*. 2005; Ahn *et al*. 2006). However, whereas *TFL1* represses the transcription of floral meristem genes *AP1* and *LFY*, *FT* acts redundantly with *LFY* to specify floral fate (Shannon and Meeks-Wagner 1991; Weigel *et al*. 1992; Ruiz-García *et al*. 1997; Samach *et al*. 2000; Wigge *et al*. 2005). *FT* and *LFY* are both activated by the photoperiod pathway integrator gene *CONSTANS* (*CO*) (Samach *et al*. 2000). Furthermore, *LFY* expression is rapidly upregulated by proteins involved in the autonomous (e.g. *SQUAMOSA-PROMOTER BINDING PROTEIN-LIKE 3* [*SPL3*] and *LD*) and gibberellic acid flowering pathways, evidencing its role as a key regulator of the floral transition (Simon *et al*. 1996; Blázquez 1997; Blázquez *et al*. 1998; Hempel *et al*. 1997; Aukerman *et al*. 1999; Wang *et al*. 2009; Yamaguchi *et al.* 2009) (**Fig. 3; Table 1**).

SVP and *AGL24* are closely related MADS-box genes in the StMADS11-clade, which, like *TFL1* and *FT*, have opposite roles in the floral transition. Ectopic expression of *SVP* causes late flowering, suggesting that it functions as a repressor of flowering (Hartmann *et al*. 2000; Lee *et al*. 2007). This function appears to be partly mediated through the repression of *FT*. By contrast, ectopic expression of *AGL24* causes early flowering, consistent with its known function as a promoter of flowering (Yu *et al*. 2002; Michaels *et al*. 2003; Yu *et al.* 2004b). Similar functional divergence has been found for orthologous StMADS11-clade genes in different species (Hartmann *et al*. 2000; Mao *et al*. 2000; Masiero *et al*. 2004; Szymkowiak and Irish 2006; Fornara *et al*. 2008; Gregis *et al.* 2008; next section).

Inflorescence mutant studies have shown that the maintenance of indeterminate stem cells in the primary inflorescence apex is partly controlled by expression of genes that repress floral meristem identity genes, particularly *TFL1* (**Fig. 3**), but also *TFL2, EMF1* and *EMF2* (Chen *et al.* 1997; Larsson *et al*. 1998; Liljegren *et al*. 1999; Calonje *et al*. 2008). Although less is known about the direct positive downstream targets of these genes, indeterminacy in the central zone of the SAM is maintained by the meristem structure KNOX-gene *SHOOTMERISTEMLESS* (*STM*) (Clark *et al*. 1996; Lenhard *et al*. 2002; reviewed in Ragni *et al*. 2007). Interestingly, despite its role in floral meristem identity, *LFY* is also implicated in the maintenance of inflorescence meristem indeterminacy, as inflorescence branches of strong *lfy* mutants lose indeterminacy, terminating in one or more carpels (Huala and Sussex 1992). However, this phenotype is probably an indirect effect, as many non-fertile mutants show loss of indeterminacy.

Racemes of *A. thaliana* develop flowers from lateral floral meristems that are formed from aggregates of cells in the peripheral zone of the inflorescence meristem (**Fig. 2**). Indeterminancy in the floral meristem is initially maintained by *WUS* (Laux *et al*. 1996; Lenhard *et al*. 2001; Lohmann *et al*. 2001). Genes such as *CLAVATA1-3* (*CLV1-3*), *ANT* and *FILAMENTOUS FLOWER* (*FIL*) then mark the transition from indeterminate to determinate growth (Clark *et al*. 1996; Fletcher *et al*. 1999; Sawa *et al*. 1999; Noel-Wilson and Krizek 2006; Goldshmidt *et al*. 2008).

The specific identity of axillary floral meristems is specified by a set of floral meristem identity genes, including *AP1*, *UFO*, *LFY* and *CAL* (Bowman *et al*. 1989; Weigel *et al*. 1992; Ingram *et al*. 1995; Mizukami and Ma 1995; Ferrándiz *et al*. 2000; Ditta *et al*. 2004) (**Fig. 3; Table 1**). Determinacy in the floral meristem is primarily achieved through a negative feedback loop involving *WUS, LFY* and *AG* (Laux *et al*. 1996; Lenhard *et al*. 2001; Lohmann *et al*. 2001) (**Fig. 3**). In addition to its role in stem cell maintenance, *WUS* works with *LFY* to upregulate *AG*. *AG* then negatively regulates *WUS* expression, reinforcing determinancy in the floral meristem. Floral meristem identity is also achieved by the repression of flowering time genes, such as *SOC1* by *AP1* (Liu *et al*. 2007), and *TFL1* by both *AP1* and *LFY*; and promotion of other floral meristem and floral organ identity genes, such as *LFY* and *AP1* by *AP1*, and *AP1* by *LFY* (Liljegren *et al*. 1999; Wagner *et al*. 1999) (**Fig. 3; Table 1**).

AP1, *CAL* and *FUL* are closely related MADS-box genes derived from two duplication events, one at the base of core eudicots (giving rise to *FUL* and the ancestor of *AP1/CAL*), and another at the base of Brassicaceae (Litt and Irish 2003) (**Fig. 4**). Mutations in *AP1* result in the partial loss of floral meristem identity, causing a highly branched inflorescence that eventually produces flowers (Irish and Sussex 1990; Kempin *et al*. 1995). By contrast, *cal* mutants show no abnormal phenotype, and *ful* mutants are defective in leaf and fruit morphology, and are late flowering (Bowman *et al.* 1993; Gu *et al*. 1998). Similar to strong *lfy* mutants, inflorescences of triple *ap1:cal:ful* mutants lack flowers entirely (Schultz and Haughn 1991; Ferrándiz *et al*. 2000). Since *FUL* is normally excluded from floral meristems by *AP1*, the triple mutant phenotype is due both to the loss of inflorescence and floral meristem identity by *FUL* and *AP1/CAL*, respectively (Bowman *et al*. 1993; Mandel and Yanofsky 1995; Hempel *et al*. 1997).

Unlike *LFY* and *FUL*, *AP1* and *CAL* are excluded from inflorescence meristems by the negative regulators *TFL1* and *TFL2* (Larsson *et al*. 1998; Liljegren *et al*. 1999) (**Fig. 3**). *AP1* and *CAL* are restricted to floral meristems where their expression is controlled by *LFY*, *FT*, *LATE MERISTEM IDENTITY1* (*LMI1*), and the SBP-box gene *SPL3*, which physically binds to elements in the *AP1* and *FUL* promoters and causes a late flowering phenotype when overexpressed (Cardon *et al*. 1997; Ferrándiz *et al*. 2000; Saddic *et al*. 2006; Wu and Poethig 2006; Gandikota *et al*. 2007; Wang *et al.* 2009; Yamaguchi *et al*. 2009). *SPL3* and its closely related paralogues, *SPL4* and *SPL5*, are further examples of genes whose expression is negatively regulated by microRNAs in the miR156/miRNA157 family (Wu and Poethig 2006; Gandikota *et al*. 2007; Wang *et al.* 2009; Yamaguchi *et al*. 2009).

In addition to patterns of determinacy, architecture of the *A. thaliana* inflorescence is also shaped by the phyllotaxy of determinate (floral) meristems, presence of pedicels, and absence of floral bracts. In wild type plants, floral

meristems are formed spirally along the primary inflorescence axis, concurrent with increased levels of the growth regulator auxin (for review see Golz 2006). Mutations in auxin transport or auxin-responsive genes, such as *PIN-FORMED1* (*PIN1*) and *PINOID* (*PID*), result in altered phyllotaxy. Both *pin1* and *pid* mutants have aberrant expression of *ANT* and *LFY*, suggesting that auxin transport is essential for both cell proliferation and the upregulation of genes that provide positional information to the floral homeotic genes (Weigel *et al*. 1992; Vernoux *et al*. 2000). Inflorescence phyllotaxy is also disrupted by mutations in genes (e.g. *SERRATE* [*SE*], *FASCIATA1* [*FAS1*], and *DISTORTED ARCHITECTURE1* [*DAR1*]) that affect the size and shape of the inflorescence meristem (Para and Sundas-Larsson 2003). Patterns of auxin transport may also be important for pedicel growth (Yamaguchi *et al*. 2007). For example, the auxin-responsive KNOX gene *BREVIPEDICELLUS* (*BP*) and *ERECTA*-like genes induce growth of the pedicel through increased cell proliferation, whereas the auxin-activated *CORYMBOSA1* (*COR1*) causes an increase in cell expansion (Ruegger *et al*. 1997; Douglas *et al*. 2002; Yamaguchi *et al*. 2007). Basal expansion of the flower pedicel may develop in response to floral identity, as pedicels of *lfy* and *ap1* mutants lack this outgrowth (Douglas and Riggs 2005). Furthermore, *lfy* mutants fail to suppress floral bracts, resulting in inflorescences that are highly branched and bushy (Huala and Sussex 1992).

Conservation and diversification of inflorescence developmental genes in core eudicots

Decades of genetic work on *A. thaliana* has not only provided a framework to test conservation and diversification of the inflorescence developmental pathway in other species, but has also revealed candidate genes whose functional evolution may underlie interspecific modifications in inflorescence architecture. Comparable studies in species other than *A. thaliana* suggest a common genetic network for inflorescence development in core eudicots. However, recent studies are starting to elucidate modifications to this inflorescence plan, through both regulatory and functional evolution of key developmental genes (**Table 1**). Some of these studies are discussed below.

Despite a wide phylogenetic distance between them (**Fig. 1**), *A. thaliana* and *A. majus* have similar indeterminate inflorescences that develop lateral branches, which then terminate in flowers (**Fig. 2**). Indeed, many homologues of *A. thaliana* inflorescence developmental genes have been found to function similarly in *A. majus*. For example, indeterminacy in the *A. majus* inflorescence meristem is controlled by the *TFL1* homologue *CENTRORADIALIS* (*CEN*), and floral meristem identity is controlled by the *LFY*, *AP1*, *AP2* and *UFO* orthologues *FLO*, *SQUA*, *LIP1/2*, and *FIM*, respectively (Carpenter and Coen 1990; Huijser *et al*. 1992; Ingram *et al*. 1995; Keck *et al*. 2003). Mutations in the *A. majus SQUA* gene cause the complete conversion of flowers to inflorescence shoots, suggesting a loss of floral meristem identity. However, although it shares many of the regulators of *ap1*, such as the *SPL3-5* homologues *SQUAMOSA-PROMOTER BINDING PROTEIN 1* (*SBP1*) and *SBP2*, the mutant phenotype of *squa* is more severe than for *ap1* (Huijser *et al*. 1992; Mandel *et al*. 1992; Klein *et al*. 1996; Preston and Hileman 2010). Like *A. thaliana*, *A. majus* has other *AP1/FUL* genes (*AmFUL* and *DEFH28*), one of which (*DEFH28*) appears to have been lost in the Brassicaceae lineage (**Fig. 4**) (Müller *et al*. 2001; Litt and Irish 2003). It is unclear exactly how functional redundancy between these paralogues may differ from redundancy between *AP1*, *CAL* and *FUL*. However, complete loss of floral meristem identity in the *AP1/SQUA* mutant *pim* of *P. sativum* suggests low redundancy for these genes outside Brassicaceae (Taylor *et al*. 2002).

Similar to *A. thaliana*, *A. majus* inflorescences lack floral prophylls. However, unlike *A. thaliana*, flowers of *A. majus* are subtended by bracts. In *A. majus*, lack of floral prophyll development appears to be the result of organ suppression, rather than incorporation into the sepal whorl. Mutations in the StMADS11-clade MADS-box gene *INCOMPOSITA* (*INCO*) result in *A. majus* inflorescences that develop paired lateral prophylls on the pedicel (Masiero *et al*. 2004). Since similar phenotypes have not been found for other StMADS11-clade genes, such as *A. thaliana SVP* and *S. lycopersicum JOINTLESS*, this suggests cooption of *INCO* in prophyll suppression within the asterids (Hartmann *et al*. 2000; Mao *et al*. 2000; Szymkowiak and Irish 2006; Gregis *et al*. 2008). Thus, differences in the expression and/or function of *INCO* orthologues may explain the presence of floral prophylls in close relatives of *A. majus*, such as *Gratiola officinalis* and *Digitalis purpurea*; this remains to be tested (Preston *et al*. 2009). Functional divergence in organ suppression is also evident in the *LFY* gene clade. Mutant analyses support a role for *LFY* in bract suppression of *A. thaliana*, but not for *LcrLFY* in closely related *Leavenworthia crassa* (Brassicaceae) or *FLO* in *A. majus* (Coen *et al.* 1990; Weigel *et al*. 1992; Yoon and Baum 2004).

Available evidence from various Leguminoseae and Solanaceae species further suggest that *LFY*-like genes are functionally conserved in floral meristem identity specification across core eudicots (Hofer *et al.* 1997; Souer *et al*. 1998; Molinero-Rosales *et al*. 1999). However, alterations in the transcriptional regulation of these genes may have been important for interspecific differences in inflorescence architecture. In *L. crassa* inflorescences develop in both the terminal and axillary positions. Expression of *LFY* and *LcrLFY* controlled by the *LcrLFY* promoter in *lfy* mutants of *A. thaliana* results in a similar reduction in apical dominance, suggesting a change in the spatial regulation of *LFY* and *LcrLFY* due to differences in their promoters. This is further evidenced by quantitative differences in the negative regulation of these genes by *TFL1,* due to differences in both promoter and protein coding regions (Yoon and Baum 2004; Maizel *et al*. 2005; Sliwinski *et al*. 2006).

Evolution of *LFY*-like gene expression has also been demonstrated for *FALSIFLORA* (*FA*) and *ABERRANT LEAF AND FLOWER* (*ALF*) in determinate (cymose) inflorescences of *S. lycopersicum* and *Petunia hybrida*, respectively (**Fig. 2**). Unlike *LFY* and *FLO,* which are expressed in lateral floral meristems, *FA* and *ALF* are strongly expressed in apical inflorescence meristems (designated 1 in **Fig. 2**), correlating with the development of terminal flowers (Souer *et al*. 1998; Molinero-Rosales *et al*. 1999). Recent studies have shown that this difference in *LFY*-like gene expression between racemose and cymose inflorescences (**Fig. 2**) is likely due to upstream regulators of both *LFY*-like genes and other regulators of meristem determinacy (Lippman *et al*. 2008; Rebocho *et al*. 2008; Souer *et al* 2008).

Key regulators of cymose inflorescence architecture in Solanaceae include the WUS-like protein EVERGREEN (EVG) of *P. hybrida* and its orthologue COMPOUND INFLORESCENCE (S) in *S. lycopersicum* (**Fig. 2; Table 1**). Similar to the closely related *A. thaliana* genes *STIMPY* (*STP*) and *WUS*, *EVG* and *S* share a conserved function in the maintenance of meristem indeterminacy. However, whereas *STP* is expressed in the central zone of vegetative SAMs, resulting in the maintenance of stem cell populations via upregulation of *WUS*, *EVG* and *S* are both expressed in the peripheral zone of inflorescence meristems (Wu *et al*. 2005; Lippman *et al*. 2008; Rebocho *et al*. 2008). This change in expression strongly correlates with evolution of developmental function in cymose inflorescences (Lippman *et al*. 2008; Rebocho *et al*. 2008) (**Fig. 2**).

In wild type *P. hybrida* and *S. lycopersicum*, inflorescence architecture is defined when the inflorescence meristems bifurcate into two meristems, the upper (apical) one forming a flower, and the lower (lateral) one forming a secondary bifurcating branch (Prusinkiewicz *et al*. 2007; Souer *et al*. 2008; Prenner *et al*. 2009) (**Fig. 2**). Inflorescence meristems of *evg* mutants rarely bifurcate, resulting in inflorescences with few branches and flowers (Rebocho *et al*.

2008). This suggests that *EVG* is required for the development of both lateral secondary inflorescence and apical floral meristems.

Rebocho and colleagues (2008) recently proposed a model to explain the dual role of *EVG*. Similar to their *A. thaliana* orthologues, *UFO* and *LFY*, the *P. hybrida* genes *DOT* and *ALF* determine floral meristem identity (Weigel *et al.* 1992; Ingram *et al*. 1995; Lee *et al*. 1997; Souer *et al*. 1998, 2008). According to the model, in *P. hybrida*, *DOT* and *ALF* are negatively regulated in the center of the inflorescence meristem by a non-cell autonomous factor that is expressed in the peripheral zone of the inflorescence meristem. As the inflorescence meristem elongates, upregulation of *EVG* in the peripheral zone results in bifurcation of the inflorescence meristem, cutting off the negative repression of the floral meristem identity genes. In contrast to *A. thaliana UFO* and *LFY*, which are expressed in lateral inflorescence meristems, this results in the expression of *DOT* and *ALF* in the upper meristem, producing a terminal flower, and the expression of *EVG* in the lower meristem, producing an indeterminate branch (Wu *et al*. 2005; Lippman *et al*. 2008; Rebocho *et al*. 2008; Souer *et al.* 1998, 2008) (**Fig. 2**). Although the identity of the negative repressor is yet unknown, candidate genes include *EXTRAPETALS* (*EXP*) and *HERMIT* (*HER*). Consistent with this, mutations in both these genes result in the production of solitary flowers (Souer *et al*. 1998; Rebocho *et al*. 2008).

CONCLUSIONS

This review highlights the importance of comparative and phylogenetic studies to both determine the extent of genetic conservation in the inflorescence and flower developmental pathways, and to identify genetic changes that may underlie inflorescence and floral diversification. The first major conclusion of this review is that homologues of the *A. thaliana* inflorescence and developmental regulators show broad levels of functional conservation across core eudicots. For example, *LFY*- and *AG*-like genes have been found to specify floral organ and/or floral meristem identity in rosid and asterid species alike. However, repeated changes in the regulation or specific interactions of these genes, and their protein products, probably underlie interspecific differences in inflorescence form. In the case of inflorescence architecture, despite several examples of regulatory modifications within a common genetic pathway, it remains unclear whether similar independent transitions are caused by comparable mutations in the same genes. Independent recruitment of *CYC*-like genes has been convincingly demonstrated for multiple evolutionary origins of floral bilateral symmetry. Thus, similar analyses of gene function and interactions will be critical to address the importance of parallel versus convergent evolution in the multiple origins of similar inflorescence traits in phylogenetically distinct species.

A second major conclusion from this review is that gene duplication and subsequent diversification has probably been important for the subtle modification of inflorescence and floral form. Although some of the important inflorescence development genes (e.g. *LFY*) are single copy in core eudicots, many other of these genes have a history of extensive gene duplication. Two major examples are the MADS-box genes and the *CYC*-like TCP genes. Recent studies suggest that the tendency of duplicated genes to lose ancestral function, or gain new function, in related lineages is not necessarily paralogue-specific. In other words, paralogues from different species may be more functionally similar than their orthologous counterparts. Thus, to better understand the degree of functional conservation among specific lineages of genes in distantly related species, thorough isolation and functional characterization of all related gene homologues must be carried out. In addition to phylogenetically targeted studies, the increasing availability of genomic data will be important to address these issues.

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