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The poetry of reproduction: the role of *LEAFY* in *Arabidopsis thaliana* flower formation

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ABSTRACT For successful reproduction, angiosperms must form fertile flowers at the appropriate positions and at the appropriate times. The reproductive transition is especially important for monocarpic plants that only flower once. In the model annual plant *Arabidopsis thaliana*, this transition is controlled through regulation of a group of genes termed floral meristem identity genes, of which *LEAFY (LFY)* is arguably the most important. *LFY* orthologs are found throughout land plants and are essential for angiosperm reproduction. These genes have also been implicated in reproductive development in gymnosperms. *LFY* encodes a plant-specific transcription factor that can act as either an activator or repressor depending on context, including what co-factors it is interacting with. It controls multiple aspects of floral morphogenesis, including phyllotaxis, organ number, organ identity and determinacy. Much progress has been made in elucidating the molecular mechanisms through which LFY and its orthologs contribute to a precise switch to flowering. We discuss the current state of knowledge in *Arabidopsis*, with an emphasis on known target genes and co-factors of LFY.

KEY WORDS: *inflorescence, meristem, transcription factor, angiosperm, morphogenesis*

The flower is the poetry of reproduction.
It is an example of the eternal seductiveness of life.
Jean Giraudoux

Introduction

Reproduction is essential for all organisms. In angiosperms, the unit of reproduction is the flower. Plants, unlike animals, have indeterminate growth, which is mediated by meristems. Meristems are groups of undifferentiated stem cells that give rise to the plant body. At germination, seedlings contain two such meristems, the shoot apical meristem (SAM) and the root apical meristem (RAM). During vegetative growth, the SAM produces lateral organs at its flanks as well as producing those cells that form the plant stem. During the reproductive phase, lateral meristems will become flowers. Reproductive success depends on initiating flowering at the right time and maintaining reproductive fate until the plant successfully sets seeds. The precise timing of reproduction is especially important in plants that only flower once, such as annuals. This review will concentrate on reproduction, specifically flower formation, in *Arabidopsis thaliana*, a model annual angiosperm.

Transition to reproduction in *Arabidopsis*

Arabidopsis is a facultative long day plant and has much accelerated time to flowering under long days, although it will eventually flower under short day conditions. The reproductive phase of *Arabidopsis* is complex. The first event in the transition is the change of the SAM into an inflorescence meristem (IM). During the vegetative phase, *Arabidopsis* grows as a rosette, with little internode elongation. However, the transition to reproduction is accompanied by bolting (the elongation of the stem of the internodes; Fig. 1A). In addition, the IM produces several cauline leaves with associated branches before producing flowers that are not subtended by bracts, modified leaves associated with flowers, which are suppressed in *Arabidopsis* (Fig. 1A). Unlike shoots, flowers are determinate structures that give rise to a set number of floral organs and then cease growth. Floral meristems (FMs) initially have a growth phase during which they increase in size. They then

Abbreviations used in this paper: AP1, APETALA1; FM, floral meristem; IM, inflorescence meristem; LFY, LEAFY; RAM, root apical meristem; SAM, shoot apical meristem; SEP3, SEPALLATA3.

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begin to produce floral organs in a whorled pattern, starting at their flanks with four sepals followed by four petals, five to six stamens and two fused carpels in the center of the flower. The identity of the floral organs depends on the activity of floral homeotic genes, which can be divided into four classes, A, B, C and E (Fig. 1B; reviewed in (Krizek and Fletcher, 2005). The A, B and C genes act in a combinatorial manner to specify each organ type. The relatively recently identified *SEPALLATA* genes (*SEP1-4*) function with the other homeotic genes in specifying floral organs (class E; (Pelaz *et al.*, 2000; Ditta *et al.*, 2004). Class A genes function alone to specify sepal identity in the outermost whorl. Class A and B genes together specify petals in the second whorl. Class B and C genes specify stamens in the third whorl and class C alone functions to specify carpel identity in the inner most whorl. A and C genes also negatively regulate each other (Bowman *et al.*, 1991; Coen and Meyerowitz, 1991; Weigel and Meyerowitz, 1994). More recent work has shown that A genes may only function in *Arabidopsis* and its close relatives, as orthologs of the A genes from *Arabidopsis*, *APETALA1 (AP1*; Irish and Sussex, 1990) and *APETALA2 (AP2*; Kunst *et al.*, 1989), do not function in specification of the perianth (sepals and petals; reviewed in (Litt and Kramer, 2010). All the floral homeotic genes encode MADS box transcription factors (Irish, 2010; Sablowski, 2010) with the exception of *AP2*, which encodes a founding member of a plant specific group of transcription factors, the AP2/EREBP family (Riechmann and Meyerowitz, 1998).

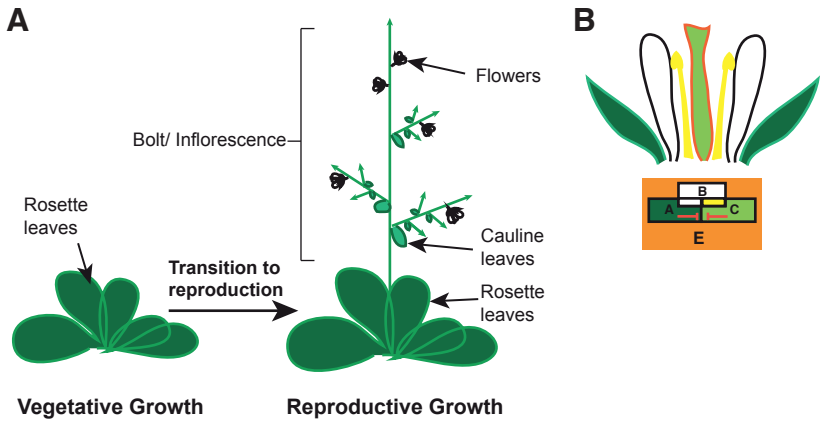

Fig. 1. The *Arabidopsis thaliana* reproductive transition and flower formation. (A) *During the vegetative phase, rosette leaves are produced without internode elongation. Upon transition to reproduction, internode elongation is activated to form the bolt and several branches with subtending cauline leaves are made before lateral meristems become flowers. Arrows indicate indeterminate growth.* **(B)** *The ABCE model for floral organ specification. Diagram of a flower showing the arrangement of sepals, petals, stamens and carpels in whorls 1-4, respectively. The ABCE genes act combinatorially to specify organ identity, as indicated below the flower. Barred lines indicate mutual repression between A and C class activities.*

As mentioned above, reproductive transition in *Arabidopsis* is characterized by two phases, one in which paraclades, composed of cauline leaves subtending flower-bearing branches, are produced and then one in which flowers are produced. Two models have been proposed to explain the determination of the inflorescence paraclades. The first model postulates that there are two transitions that occur in sequence from the base to the apex, the first to bolting and the second to flowering (Schultz and Haughn, 1993; Haughn *et al.*, 1995; Ratcliffe *et al.*, 1998). The other model holds that there is only one bidirectional transition responsible for both the base to apex progression of flowers and the apex to base progression of paraclades (Hempel, 1994). It has been suggested that the two models are not mutually exclusive and may depend on the strength and duration of flowering signals (Suh *et al.*, 2003; Pouteau and Albertini, 2009; Pouteau and Albertini, 2011). Regardless, both the bolting transition and the flowering transitions are important for reproduction in *Arabidopsis*.

The transformation of the SAM into an IM is tightly regulated by both endogenous and environmental factors that integrate to result in flowering (Parcy, 2005). Experiments beginning in the 1920s have demonstrated that different plants have varying requirements to trigger flowering (Garner and Allard, 1920). In *Arabidopsis*, a number of forward genetic screens have identified many genes that are involved in control of flowering time. Subsequent genetic analysis has defined at least five pathways in *Arabidopsis* that control this process: the photoperiod pathway, the vernalization pathway, the autonomous pathway, the gibberellic acid (GA) pathway and a developmental age pathway (Martinez-Zapater *et al.*, 1994; Araki, 2001; Mouradov *et al.*, 2002; Simpson and Dean, 2002; Bastow and Dean, 2003; Amasino, 2004; Boss *et al.*, 2004; Jack, 2004; Sung and Amasino, 2004). A recent review (Srikanth and Schmid, 2011) summarizes these five pathways in depth and they will not be discussed in detail here. These pathways converge on a set of genes that include floral meristem identity genes, which are discussed below.

The floral meristem identity genes

Once an IM is formed, it will begin generating FMs on its flanks after formation of 2-5 cauline leaves. Floral meristem identity genes are required to specify the lateral meristems as flowers. The floral meristem identity genes encode transcription factors and are involved in a complex network of mutual regulation (Fig. 2). Floral meristem identity proteins in *Arabidopsis* include LEAFY (LFY; (Weigel *et al.*, 1992; Blázquez *et al.*, 1997; Nilsson *et al.*, 1998), the related MADS box transcription factors AP1 (Mandel *et al.*, 1992; Bowman *et al.*, 1993; Irish and Sussex, 1990; Ferrandiz *et al.*, 2000), CAULIFLOWER (CAL; (Kempin *et al.*, 1995; Ferrandiz *et al.*, 2000) and FRUITFUL (FUL; (Ferrandiz *et al.*, 2000), the SEP MADS box transcription factors (SEP1-4), especially SEP3 and SEP4 (Ditta *et al.*, 2004; Castillejo *et al.*, 2005; Kaufmann *et al.*, 2009), the MADS box proteins AGAMOUS LIKE24 (AGL24) and SHORT VEGETATIVE PHASE (SVP; Gregis *et al.*, 2008), the class 1 HD-Zip transcription factor LATE MERISTEM IDENTITY1 (LMI1; (Saddic *et al.*, 2006) and the R2R3 class MYB transcription factor LATE MERISTEM IDENTITY2 (LMI2)/AtMYB17 (Pastore *et al.*, 2011). All of the genes encoding these proteins are expressed in FMs (Mandel *et al.*, 1992; Hempel *et al.*, 1997; Hartmann *et al.*, 2000; Pelaz *et al.*, 2000; Yu *et al.*, 2002; Ditta *et al.*, 2004; Saddic *et al.*, 2006; Pastore *et al.*, 2011). In *Arabidopsis*, LFY and AP1 are the two most important floral meristem identity regulators (Huala and Sussex, 1992; Weigel *et al.*, 1992; Bowman *et al.*, 1993; Mandel and Yanofsky, 1995; Ferrandiz *et al.*, 2000). In addition to these positive promoters of floral identity, there is a negative regulator

of floral fate, *TERMINAL FLOWER1 (TFL1)*, which is expressed in the IM and encodes a member of the CETS (CENTRORADIALIS/TFL1/FT) family of plant proteins that have similarities to Raf kinase inhibitory protein and a phosphatidylethanolamine-binding protein and function in transcriptional complexes (Liljegren *et al.*, 1999; Ratcliffe *et al.*, 1999). TFL1 prevents the IM from expressing floral meristem identity genes and becoming a flower, therefore maintaining its indeterminate nature.

The MADS box transcription factor-encoding gene *AP1* is a floral meristem identity regulator in *Arabidopsis* and its homologs in other species also appear to function in floral meristem identity and/or floral induction (reviewed in (Litt and Kramer, 2010). *AP1* is expressed throughout the very young FM before becoming confined to the outer two whorls at stage 3 of floral development (Mandel *et al.*, 1992). *AP1* expression is directly activated by the floral meristem identity genes LFY, LMI2, AGL24, SVP and SEP3 (Fig. 2; Wagner *et al.*, 1999; Kaufmann *et al.*, 2009; Grandi *et al.*, in press; Pastore *et al.*, 2011). AP1 activates genes promoting floral organ formation and represses flowering time genes to maintain the floral fate of the meristem (Hill *et al.*, 1998; Tilly *et al.*, 1998; Ng and Yanofsky, 2001; Yu *et al.*, 2004a; Liu *et al.*, 2007). In *ap1* mutants, extra cauline leaves are made before the formation of flowers. *ap1* flowers have leaf-like sepals and no petals, although stamen and carpel development are normal. In addition, ectopic flowers form in the axils of the leaf-like sepals, a phenotype that has been interpreted as a floral meristem identity defect (Irish and Sussex, 1990; Bowman *et al.*, 1993). The effect of loss of *AP1* function on floral identity is not as severe as loss of its orthologs in some other groups of angiosperms due to the presence of the *CAL* gene. *CAL* is a paralog of *AP1* found in Brassicas that is partially redundant with *AP1* (Kempin *et al.*, 1995). *ap1-1; cal-1* mutants show complete transformation of flowers into meristems, although loss of *CAL* alone has no phenotype (Bowman *et al.*, 1993). However, eventually the meristems of *ap1-1; cal1-1* plants will form differentiated flowers that resemble the flowers of *ap1* single mutants. This is due to the activity of *FUL*, one of the closest genes to *AP1/CAL* in the *Arabidopsis* genome. Loss of all three of these genes leads to a severe meristem identity defect (Ferrandiz *et al.*, 2000). AP1, CAL, FUL all activate transcription of *LFY*, directly or indirectly (Fig. 2; Ferrandiz *et al.*, 2000). The complex genetic interactions among these genes reflects their membership in the AP1 family, which in higher angiosperms consists of two clades, eu*AP1* (in which *Arabidopsis AP1* and *CAL* fall) and eu*FUL* (to which *FUL* belongs; Litt and Irish, 2003). The eu*FUL* clade underwent a duplication event to generate two subclades: eu*FUL1*, including *FUL*, and eu*FULII*, including *Arabidopsis AGL79*. This genetic and evolutionary complexity makes it difficult to determine the functional ortholog(s) of *AP1* acting in meristem identity in other angiosperms.

The SEP1-4 MADS box proteins have roles in floral meristem identity, floral organ identity and ovule identity (Pelaz *et al.*, 2000; Pelaz *et al.*, 2001a; Pelaz *et al.*, 2001b; Favaro *et al.*, 2003; Ditta *et al.*, 2004). They physically interact with other MADS box transcription factors to form ternary complexes that regulate gene expression (Honma and Goto, 2001; Jack, 2001). SEP3 has been shown to be especially important for floral meristem identity (Castillejo *et al.*, 2005) and regulates expression of other floral meristem identity genes, including itself, as well as *SEP4*, *CAL*, *AP1*, *LMI1*, *LMI2* and *LFY* (Kaufmann *et al.*, 2009). SEP3 interacts physically with AP1 and is present in transcriptional complexes with it (Sridhar *et al.*, 2006; Immink *et al.*, 2009). Not surprisingly, many targets of AP1 and SEP3 overlap.

Two other MADS box transcription factor encoding genes, *AGL24* and *SVP*, also function as floral meristem identity genes. These genes have been shown to be important for several aspects of reproduction in *Arabidopsis* (Hartmann *et al.*, 2000; Yu *et al.*, 2002; Michaels *et al.*, 2003; Yu *et al.*, 2004a; Lee *et al.*, 2007a; Lee *et al.*, 2007b; Liu *et al.*, 2007; Gregis *et al.*, 2008; Liu *et al.*, 2009; Grandi *et al.*, in press). Their roles in controlling flowering time are best known, where they play opposing roles, with *AGL24* promoting the floral transition while *SVP* acts to repress flowering (Hartmann *et al.*, 2000; Michaels *et al.*, 2003). More recent work has revealed that these two genes act redundantly to promote flower identity once the reproductive transition has occurred (Gregis *et al.*, 2008). *AGL24* and *SVP* act together with *AP1* in this process; the triple mutant *agl24-2; svp-41; ap1-10* has a phenotype reminiscent of that of *apl-1; cal1-1*, suggesting some redundancy in function among these genes. Similar genetic interactions between these genes and *LFY* have also been shown, further confirming the role of *AGL24* and *SVP* in promoting flower identity (Grandi *et al.*, in press). These transcription factors also directly activate *LFY* and *AP1* (Grandi *et al.*, in press). SVP and AGL24 also act redundantly with AP1 to repress B and C homeotic genes during stage 1 and 2 of flower development (Gregis *et al.*, 2006; Gregis *et al.*, 2008; Gregis *et al.*, 2009), important for proper floral patterning.

In addition to the genes discussed above, two

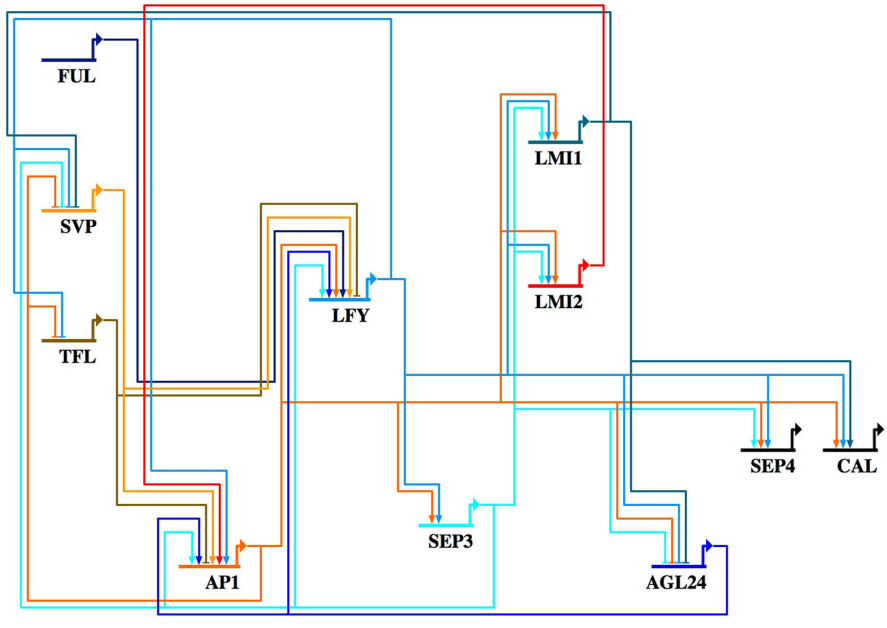

Fig. 2. Floral meristem identity genes mutually regulate each other. *The network was visualized using the BioTapestry program (Longabaugh* et al.*, 2009). LEAFY (LFY) activates transcription of many other floral homeotic genes and is regulated by them in turn.*

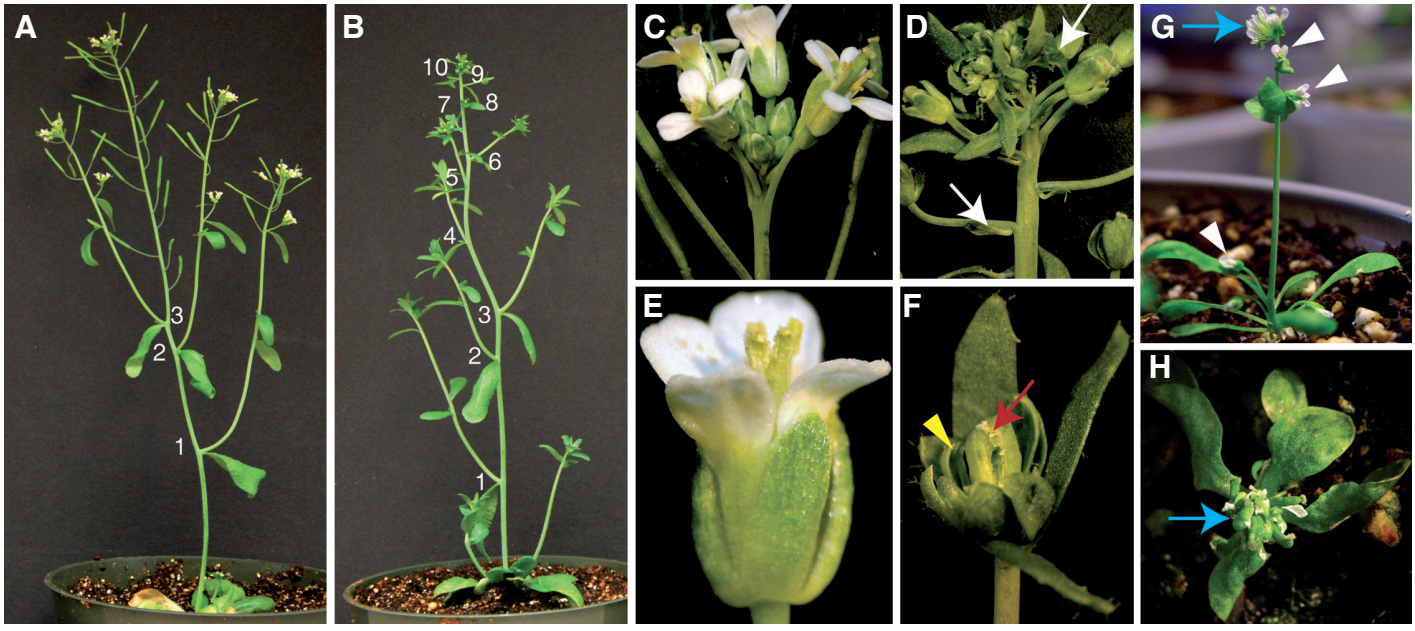

Fig. 3. *LEAFY (LFY)* is a floral meristem identity gene. *Micrographs of* Arabidopsis *plants and flowers.* **(A)** *Wild type plant.* **(B)** lfy-6 *plant. Numbers indicate cauline leaves with subtending branches along the primary stem.* **(C)** *Top of wild type inflorescence.* **(D)** *Top of* lfy-6 *inflorescence.* **(E)** *Wild type flower.* **(F)** lfy-6 *mutant flower. Note partially spiral phyllotaxy in (f).* **(G,H)** 35S::LFY *plants. White arrows indicate bracts, red arrows indicate partially fused carpel-like organs, yellow arrowhead indicates leaf-like sepals, blue arrows indicate terminal flowers and white arrowheads indicate single flowers formed in place of branches.*

genes originally identified as direct targets of LFY, *LMI1* and *LMI2*, also function in floral meristem identity. An *lmi1* mutant enhances the meristem defects of the weak *lfy-10* allele (Saddic *et al.*, 2006). *lmi1* mutants have very subtle meristem defects. LMI1 acts upstream of *CAL* and together with LFY regulates *CAL* expression directly (Fig. 2; Saddic *et al.*, 2006). Its expression is activated by both AP1 and SEP3 in addition to LFY (William *et al.*, 2004; Kaufmann *et al.*, 2009; Kaufmann *et al.*, 2010). *lmi2* mutants similarly enhance the phenotypes of the *lfy-10* allele. LMI2 acts together with LFY to activate expression of *AP1*, therefore operating in a feed forward loop to positively regulate floral meristem identity (Fig. 2; Pastore *et al.*, 2011). Similarly to *LMI1*, the transcription of *LMI2* is also dependent on SEP3 and AP1 (William *et al.*, 2004; Kaufmann *et al.*, 2009; Kaufmann *et al.*, 2010).

A very recent publication demonstrates the complex regulatory network among the floral meristem identity genes *AP1, CAL, AGL24, SVP, LMI1* and *LFY* (Fig. 2; Grandi *et al.*, in press). LFY acts to repress *AGL24* and *SVP* transcription, although this appears to be an indirect activity. LFY's direct target and co-factor LMI1 acts to positively regulate these two genes. AGL24 and SVP directly activate transcription of both *LFY* and *AP1*. Clearly, the interactions between floral meristem identity genes involve multiple feedback loops, both positive and negative.

In *Arabidopsis*, the IM remains indeterminate and does not form a terminal flower. This is due at least in part to the activity of *TFL1* in the IM. AP1 and LFY repress *TFL1* expression in floral meristems, suppressing IM fate (Fig. 2; Liljegren *et al.*, 1999; Parcy *et al.*, 2002). TFL1 in turn suppresses the expression of *AP1* and *LFY* in the IM (Ratcliffe *et al.*, 1998). The balance between these genes is what regulates shoot architecture (Bradley *et al.*, 1997). In fact, during the domestification of soybean (*Glycine max*), mutant alleles of the *GmTFL1* gene were selected because they conferred a determinate growth habit, an agronomically important trait (Tian *et al.*, 2010).

LEAFY: a master regulator of flowering

LFY was first recognized for its function in flower meristem development. Although expression can be detected weakly in leaves, *LFY* expression is highest in floral meristems, where it is found throughout the early primordium with earliest accumulation before cell groups have begun to separate from the IM (Weigel *et al.*, 1992). Later in floral development (starting at stage 3), expression begins to decline in the center of the flower. At stage 6, when the carpel primordia emerge, *LFY* is detected in incipient petals, stamens and pistil and persists until stage 9, after which it is not detected. *lfy* mutants are slightly late flowering, produce extra cauline leaves and have abnormal floral-like structures in which there is homeotic transformation of floral organs to leaf-like structures (Fig. 3 B,D,F; Schultz and Haughn, 1991; Huala and Sussex, 1992; Weigel *et al.*, 1992). Conversely, constitutive expression of *LFY* under the *35S* promoter causes the conversion of indeterminate lateral meristems into flowers and the conversion of the IM into a flower (Fig. 3 G,H; Weigel and Nilsson, 1995). *LFY* expression is directly regulated by AP1, AGL24, SVP and SEP3 (Fig. 2; Wagner *et al.*, 1999; Kaufmann *et al.*, 2009; Grandi *et al.*, in press; Winter *et al.*, 2011).

LFY encodes a plant specific transcription factor (Weigel *et al.*, 1992) with a DNA binding domain that is structurally related to helix-turn-helix domains (Hamès *et al.*, 2008). In addition to the conserved DNA binding domain, located at the C-termini of LFY-like proteins, an N-terminal domain of unknown function is also conserved (Fig. 4; Maizel and Weigel, 2004). Unlike many other transcription factors that have evolved by gene duplication

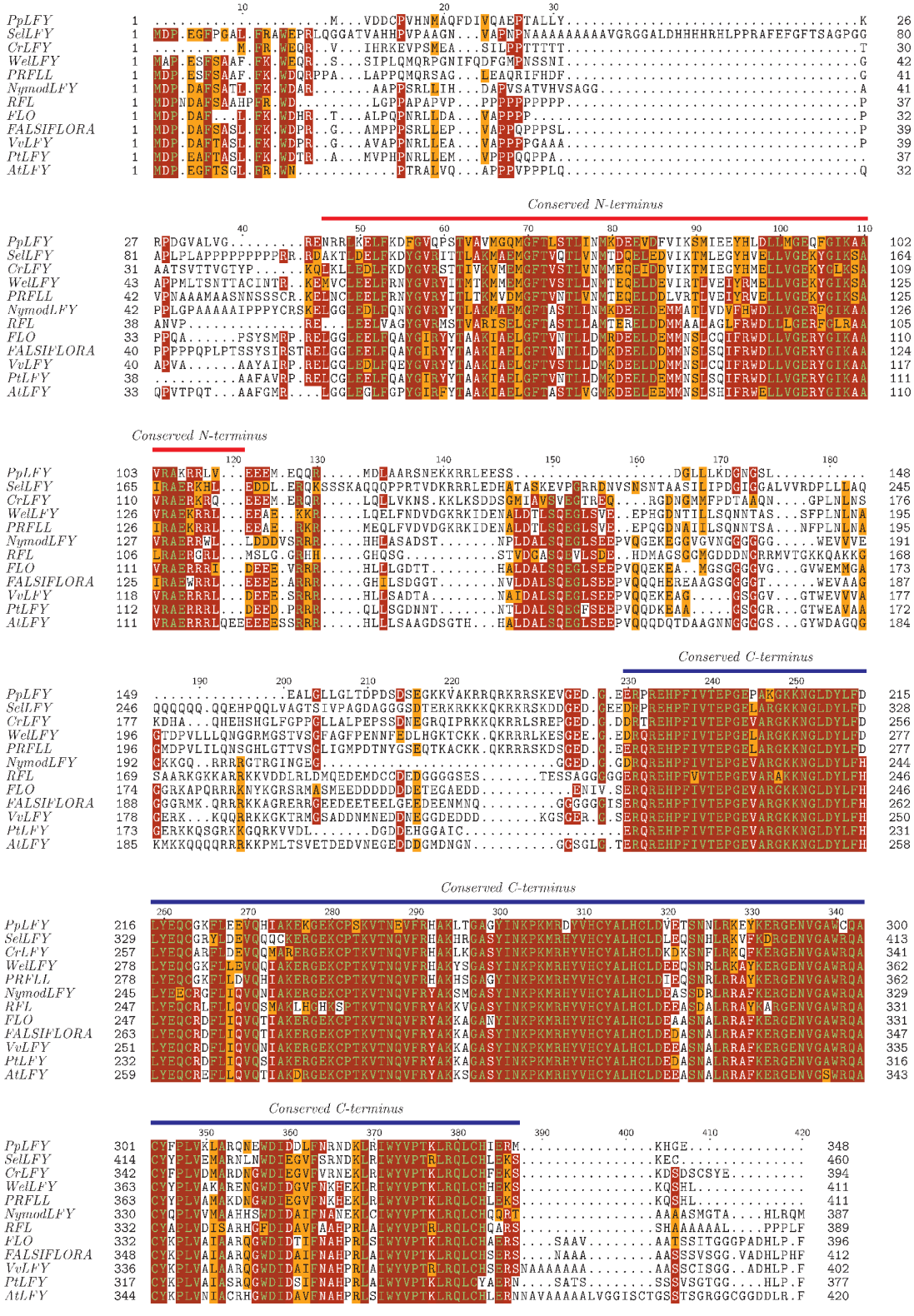

Fig. 4. LEAFY and its orthologs contain two conserved domains. *Amino acid alignment of selected LFY orthologs from across land plants. The conserved N-terminal region is marked with a red bar. The conserved DNA binding domain is marked with a blue bar. Dots indicate gaps introduced to optimize the alignment. Identical amino acids are indicated by red shading and similar amino acids by orange shading. The alignments were generated using the MUSCLE3.8.31 multiple alignment tool, using default settings (Edgar, 2004). At,* Arabidopsis thaliana*; Pt,* Populus trichocarpa*; Vv,* Vitis vinifera*; FALSIFLORA.* Solanum lycopersicon *LFY ortholog; FLO,* Antirrhinum majus *LFY ortholog; RFL,* Oryza sativa *LFY ortholog; Nymod,* Nymphea odorata*; PRFLL,* Pinus radiata *LFY ortholog; Wel,* Welwitschia mirabilis*; Cr,* Ceratopteris thalictroides*; Sel,* Selaginella moellendorfi*; Pp,* Physcomitrella patens.

to form multigene families (Riechmann and Ratcliffe, 2000), *LFY* is present as a single copy in most of the angiosperms. This makes LFY unique among transcription factors in plants. The *LFY* gene is conserved throughout land plant species, from bryophytes (the moss *Physcomitrella patens*) to flowering plants (Maizel *et al.*, 2005). Studies done using species across the land plants demonstrated that the ability to complement the *Arabidopsis lfy* mutant decreases as the evolutionary difference from *Arabidopsis* increases and that this is due to changes in the DNA binding specificity of the more distantly related proteins (Maizel *et al.*, 2005). In moss, the *LFY* orthologs *PpLFY1* and *PpLFY2* regulate the first division of the zygote (Tanahashi *et al.*, 2005). It is hypothesized that *LFY* -like genes had an ancestral role in controlling cell division activity and placement of new cells (Moyroud *et al.*, 2010). In gymnosperms and angiosperms, *LFY*-like genes are associated with reproductive structure formation (cones and flowers, respectively).

The importance of LFY in reproductive development across angiosperms and its expression in cones in gymnosperms has informed many evo-devo studies. In the gymnosperm *Pinus radiata* two paralogous *LFY*-like genes have been identified: *PRFLL* and *NEEDLY (NLY)* (Mellerowicz *et al.*, 1998; Mouradov *et al.*, 1998). The presence of two paralogs in most gymnosperms seems to be the rule, although at least one species (*Gnetum gnemon*) does not have a *NLY*-like gene; at the base of the angiosperms, the *NLY*-like gene disappeared and only the *PRLL*-like gene persisted (Albert *et al.*, 2002). In *P. radiata*, *PRFLL* expression is restricted to male cones while *NLY* expression is mostly confined to the female cones; both are also expressed in vegetative meristems (Mellerowicz *et al.*, 1998; Mouradov *et al.*, 1998). This difference in expression and lack of *NLY* orthologs in angiosperms lead to a hypothesis about the evolutionary origin of flowers from cones, termed the "mostly male theory" (Frohlich and Parker, 2000). This theory postulates that the bisexual flower arose from male cone-like structures bearing ectopic ovules in hypothetical ancestral plants. However, the *LFY* orthologs from *Gnetum parvifolium* and *Picea abies* are expressed in seed bearing cones (Shindo *et al.*, 2001; Carlsbecker *et al.*, 2004). In a broad survey across the gymnosperms it was found that both *LFY*-like and *NLY*-like genes are expressed in both pollen and seed cones (Vazquez-Lobo *et al.*, 2007). Thus, the presence or absence of *LFY* or *NLY* does not explain the development of bisexual flowers, although it does not disprove the "mostly male" theory. Other theories for the origin of the flower have postulated that spatial changes in B class MADS box genes (Theissen *et al.*, 2000; Theissen and Becker, 2004) or concerted changes of *LFY*, LFY co-factors and MADS box genes (Baum and Hileman, 2006) could underlie the transition to hermaphroditic flowers. The absence of *LFY*-like gene expression in apical meristems that are undergoing more sustained indeterminate growth in gymnosperms such as *Picea* and its presence in those meristem that form reduced numbers of ovule-bearing scales such as *Podocarpus* does suggest that *LFY*-like genes confer determinate growth, similar to its function in angiosperm flowers (Vazquez-Lobo *et al.*, 2007).

Although control of flower development is the core function of *LFY* genes in angiosperms, in some species additional roles have been acquired. Several of the gymnosperm *LFY*-like genes are expressed vegetatively, supporting the idea that non-reproductive functions maybe ancestral. Some of these functions include involvement in SAM development in tobacco (Ahearn *et al.*, 2001), compound leaf development in legumes (Hofer *et al.*, 1997) and tomato (Molinero-Rosales *et al.*, 1999) and panicle branching in rice (Kyozuka *et al.*, 1998). In addition, recent evidence suggests that *Arabidopsis* LFY functions during vegetative growth to regulate plant defense pathways (Winter *et al.*, 2011). A recent review has highlighted the diverse roles of LFY orthologs across the angiosperms (Moyroud *et al.*, 2009). We will concentrate on floral development in this review. LFY plays two main roles in flowering, which are both temporal and genetically separable (Parcy *et al.*, 1998). Firstly, LFY acts as a meristem identity regulator and activates other important floral meristem identity regulators. During this phase of activity, LFY regulates phyllotaxy in the flower as well as organ number. Secondly, LFY is necessary for activation of floral organ identity genes and genes involved in floral morphogenesis. *LFY* also is necessary to maintain FM identity.

As a DNA binding transcription factor, LFY can act as a transcriptional activator as well as a transcriptional repressor (Wagner *et al.*, 1999; William *et al.*, 2004; Winter *et al.*, 2011). However, LFY does not seem to have either activation or repression activity on its own (Parcy *et al.*, 1998; Busch *et al.*, 1999), suggesting that it is dependent on co-factors for its activity, as does the fact that LFY regulates some of its target genes in only a subset of its spatial and temporal expression domain (see below). LFY regulates gene expression by recognizing pseudopalindromic sequence elements (CCANTGT/G) in the promoters of its target genes (Parcy *et al.*, 1998; Busch *et al.*, 1999; Wagner *et al.*, 1999; Lohmann *et al.*, 2001; Lamb *et al.*, 2002). The crystal structure of the LFY C-terminus bound to DNA showed that the DNA binding domain has a compact fold composed of two short β-strands followed by seven helices connected by short loops showing base-specific contacts with both the major and minor grooves of the DNA (Hamès *et al.*, 2008). This has more accurately defined the LFY binding sequence as T/ANNNNCCANTGG/TNNNNT/A (with the center of the pseudopalindrome underlined; (Hamès *et al.*, 2008). This motif has the previously defined consensus as the core. Several recent papers have also observed this expanded consensus sequence (Moyroud *et al.*, 2011; Winter *et al.*, 2011).

LFY and floral meristem identity

LFY is necessary for flower formation; however, it is not essential for the reproductive transition and bolting. Flowering time and bolting is slightly delayed in *lfy* mutants, but this delay is relatively minor (Blázquez *et al.*, 1997). *LFY* expression is first detectable in leaf primordia at a very low level and increases until a certain threshold is reached; once the threshold is reached, the primordia are specified as flowers. In other words, the level of *LFY* in the plant is the trigger to produce flowers (Blázquez *et al.*, 1997; Hempel *et al.*, 1997). Thus, when the number of copies of LFY is altered, timing of flower formation is changed (Blázquez *et al.*, 1997). The level of *LFY* reflects the quantity and the quality of different flowering signals the plant perceives (Lee *et al.*, 2008). Previous studies done on flowering time mutants show that in many late flowering mutants, *LFY* expression is delayed, while in early flowering mutants, its expression is accelerated (Nilsson *et al.*, 1998). Since *LFY* has been shown to be downstream of all the known pathways that control flowering time (Blázquez *et al.*, 1998; Nilsson *et al.*, 1998; Aukerman *et al.*, 1999), expression of the other key meristem identity gene in *Arabidopsis*, *AP1*, is observed only after the floral transition has been initiated (Mandel *et al.*, 1992;

Simon *et al.*, 1996; Hempel *et al.*, 1997) and LFY is a direct activator of *AP1* transcription (Wagner *et al.*, 1999) as well as other meristem identity genes, it is thought that LFY is the key player in the floral transition (Fig. 2). However, AP1's role in *Arabidopsis* is obscured by the presence of CAL and FUL, as discussed above.

Loss of *LFY* function causes lateral meristems that would normally make flowers to instead produce cauline leaves and associated lateral shoots (Fig. 3B; Schultz and Haughn, 1991; Huala and Sussex, 1992; Weigel *et al.*, 1992). Eventually *lfy* plants will form structures that have both shoot-like and flower-like characteristics and consist of many leaf-like organs and abnormal carpels in a partially spiral phyllotaxy (Fig. 3F; Schultz and Haughn, 1991; Huala and Sussex, 1992; Weigel *et al.*, 1992). This is due to the fact that, in *Arabidopsis*, later-arising flowers have only a partial requirement for *LFY* because *AP1* can become activated independently of LFY (Huala and Sussex, 1992; Bowman *et al.*, 1993; Wigge *et al.*, 2005). *lfy* mutant flower-like structures are often subtended by bracts that normally are suppressed in *Arabidopsis*, demonstrating that LFY also controls this aspect of floral morphology in *Arabidopsis* (Fig. 3D; Schultz and Haughn, 1991; Huala and Sussex, 1992; Weigel *et al.*, 1992).

The role of LFY in specifying lateral meristems as flowers depends on its direct activation of the transcription of other floral meristem identity genes (Fig. 2 and Table 1). In addition, other targets of LFY are likely to be involved in floral specification and/or determining aspects of floral morphology such as a whorled arrangement of organs, pedicel (the stem that connects the flower to the inflorescence stem) length and orientation, correct organ number and suppression of internode elongation. For the purposes of this review, we have defined LFY target genes as those loci that have been identified in at least two independent experiments, including whole genome level chromatin immunoprecipitation (ChIP) and/or by at least two independent experimental techniques (such as ChIP and microarray, for example). LFY controls expression of a wide variety of genes, reflecting its roles in multiple aspects of floral architecture. LFY has recently been shown to be necessary for the reduced cortical cell elongation at the adaxial side of the pedicel base (Yamaguchi *et al.*, in press). This suppression is necessary to prevent *Arabidopsis* flowers from bending down. At least some of this function of LFY is mediated by its activation of the *ASYMMETRIC LEAVES2 (AS2)* gene (Table 1; Yamaguchi *et al.*, in press). An interesting category of LFY targets are those involved in auxin biosynthesis, transport and signaling (Table 1). Auxin flux is temporally and spatially correlated with FM development and its control is necessary for FM formation (Blázquez *et al.*, 2006; Heisler *et al.*, 2005; Liu *et al.*, 2009). LFY represses transcription of the *PIN4* gene, encoding an auxin efflux carrier (Table 1; Friml *et al.*, 2002). PIN proteins are the rate-limiting step in polar auxin transport (Petrasek *et al.*, 2006). Inhibiting auxin efflux from the incipient floral meristem would allow auxin accumulation and meristem outgrowth. In addition, disruptions in polar auxin transport have been shown to result in flowers with reduced numbers of floral organs as well as defective organs (Nemhauser *et al.*, 2000). LFY may control floral organ number in part by inhibition of *PIN4* expression. LFY also binds to the regulatory regions of *AINTEGUMENTA (ANT)* and *AINTEGUMENTA-LIKE6 (AIL6)*, encoding partially redundant AP2/ERF family transcription factors (Table 1). ANT and AIL6 are known to impact floral meristem initiation and floral meristem patterning through regulation of auxin physiology during floral development (Krizek, 2011). LFY also targets genes involved in organ growth and polarity, such as *GIF1* (Lee *et al.*, 2009) and *FILAMENTOUS FLOWER (FIL;* Sawa *et al.*, 1999a; Sawa *et al.*, 1999b). Tissue polarity is also known to be correlated with FM development (Blázquez *et al.*, 2006). *FIL* encodes a YABBY transcription factor expressed on the abaxial side of young FMs (Sawa *et al.*, 1999b; Siegfried *et al.*, 1999) and when mutations in this gene are combined with either *lfy* or *ap1* mutants, FM defects are enhanced (Sawa *et al.*, 1999a). A target of LFY transcriptional activation, *ETTIN (ETT;* Table 1), which encodes an AUXIN RESPONSE FACTOR (ARF3), is known to affect organ polarity through regulation of the abaxial fate promoting *KANADI* genes (Pekker *et al.*, 2005). Thus, LFY directly regulates components of auxin signaling, organ polarity and a factor that links both. In addition, LFY also targets the GA pathway, which is known to regulate *LFY* expression (Blázquez and Weigel, 1999; Eriksson *et al.*, 2006; Achard *et al.*, 2007) and be necessary for proper organ growth (Mutasa-Gottgens and Hedden, 2009). Another target of LFY, *AtTLP8/LMI5* (William *et al.*, 2004; Winter *et al.*, 2011), is enriched in the quiescent center of the root (Nawy *et al.*, 2005), suggesting it has general functions in stem cells.

Other floral meristem identity genes also regulate a number of LFY target genes, consistent with their molecular and genetic interactions. Expressing both LFY and SEP3 together outside of the flower can induce formation of floral organs, suggesting they act together (Castillejo *et al.*, 2005). Analysis of SEP3 target genes has revealed that this gene also regulates auxin homeostasis and that, furthermore, its targets have an enrichment of auxin response elements (ARF binding sites) in their regulatory region (Kaufmann *et al.*, 2009). This is consistent with the phenotypic consequences of expression of a SEP3-EAR fusion protein that represses target gene expression. In the flowers of these plants there are fewer, smaller organs. One interesting common target of SEP3 and LFY is *ETT* (Table 1). LFY and SEP3 were shown to physically interact using *in vitro* GST-immunoprecipitation assay (Table 2; Liu *et al.*, 2009). This suggests that LFY and SEP3 act together in common transcriptional complexes to regulate gene expression and that some of the targets of these complexes are auxin-related genes. AP1 also shares a number of target genes with LFY and SEP3 (Table 1), including other floral meristem identity genes such as *LMI1* and *LMI2* and *SEP3* (Fig. 2 and Table 1) as well as auxin and GA related genes and those involved in organ polarity. SEP3 has been shown to physically interact with AP1 in so-called MADS box protein quartets (Honma and Goto, 2001; Jack, 2001; Pelaz *et al.*, 2001a), suggesting that LFY, AP1 and SEP3 may be in some common transcriptional complexes.

As mentioned above, LFY functions to suppress bract formation. However, it is unclear what genes it targets to perform this function. At least four other genes are known to have roles in bract suppression: *BLADE ON PETIOLE1 (BOP1)*, *BOP2*, *PUCHI* and *UNUSUAL FLORAL ORGANS (UFO)*. *BOP1* and *BOP2* encode proteins containing ankyrin repeats and BTB/POZ domains and are thought to function in protein-protein interactions. They belong to the NONEXPRESSOR OF PR GENES1 (NPR1) family of proteins and are partially redundant with one another. Genetically, they act together with *LFY* to inhibit the growth of bracts (Norberg *et al.*, 2005). BOP1 and BOP2 have been demonstrated to interact with the TGA transcription factor PERIANTHIA (PAN), although it is unclear if PAN is involved in bract suppression (Hepworth *et al.*,

2005). BOP1 and BOP2 inhibit bract growth, at least in part, by repression of expression of the *JAGGED (JAG)* and *JAGGED-LIKE (JGL)* genes, which encode C2H2 transcription factors. To date, neither *JAG* nor *JGL* has been demonstrated to be targets of LFY. BOP1 and BOP2 have also been shown to promote expression of *LFY* and *AP1* (Karim *et al*., 2009; Xu *et al*., 2010). The AP2 family transcription factor PUCHI has overlapping functions with BOP1/2 in bract suppression and also in promoting *LFY* and *AP1* expression (Karim *et al*., 2009), suggesting that upregulation of these floral meristem identity genes is essential for the inhibition of bract growth. Interestingly, *PUCHI* has been identified as a putative direct target of LFY, although this has not been confirmed (Moyroud *et al*., 2011). Finally, the F-box encoding gene *UFO* has also been shown to work jointly with LFY in floral meristem identity and suppression of bracts (Hepworth *et al*., 2006). UFO has been shown to be a LFY co-factor in the regulation of floral organ identity genes (Table 2; Lee *et al*., 1997; Chae *et al*., 2008).

Regulation of floral homeotic genes by LFY

After initiating the meristem identity switch, LFY has a second role in flower development through transcriptional activation of all

TABLE 1

SUMMARY OF LFY TARGET GENES

Locus ID	Gene Name	Type of protein	Regulation by LFY	References
At1g16070	*AtTLP8/ LMI5*	TUBBY family transcription factor	Activated	(William *et al*., 2004; Winter *et al*., 2011)
At1g19850	*MP/ ARF5/ IAA24*	ARF family transcription factor	ND[g]	(Moyroud *et al*., 2011)
At1g24260[a,b]	*SEP3/ AGL9*	MADS box transcription factor	Activated	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At1g25560[a,b]	*TEM1/ EDF1*	RAV family transcription factor	Activated[h]	
At1g26310[a,e]	*CAL/ AGL10*	MADS box transcription factor	Activated	(Wagner *et al*., 2004; William *et al*., 2004)
At1g30040[a]	*GA2OX2*	Gibberellin 2-oxidase	Activated	(Wagner *et al*., 2004; Moyroud *et al*., 2011)
At1g31140	*GOA/ AGL63*	MADS box transcription factor	ND	(Moyroud *et al*., 2011)
At1g59870	*PEN3/ PDR8*	ATP binding cassette transporter	ND	(Winter *et al*., 2011)
At1g59940	*ARR3*	Type A response regulator	ND	(Moyroud *et al*., 2011)
At1g65620	*AS2*	Transcriptional repressor characterized by cysteine repeats and a leucine zipper	Activated	(Yamaguchi *et al*., in press)
At1g69120[a,b,d,i]	*AP1*	MADS box transcription factor	Activated	(Wagner *et al*., 1999; Moyroud *et al*., 2011; Winter *et al*., 2011)
At1g80340	*GA30X2*	Gibberellin 3 β-hydroxylase	ND	(Moyroud *et al*., 2011)
At2g01420[a,b]	*PIN4*	Auxin efflux carrier	Repressed[h]	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At2g03710[a,b]	*SEP4/ AGL3*	MADS box transcription factor	Activated	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At2g28610[a,b]	*PRS/ WOX3*	WUSCHEL-like homeodomain transcription	ND	(Moyroud *et al*., 2011)
At2g33860[a,b]	*ETT/ ARF3*	ARF family transcription factor	Activated	(Winter *et al*., 2011; Wagner *et al*., 2004)
At2g34650[a]	*PID/ ABR*	Serine/ threonine kinase	ND	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At2g45190[b]	*FIL/ AFO/ YAB1*	YABBY transcription factor	ND	(Winter *et al*., 2011; Moyroud *et al*., 2011)
At2g45660[a,b]	*SOC1/ AGL20*	MADS box transcription factor	ND	(Moyroud *et al*., 2011)
At3g47340	*ASN1/ DIN6*	Glutamine-dependent asparagine synthase	Activated	(Wagner *et al*., 2004; William *et al*., 2004)
At3g54340[a,b,f]	*AP3*	MADS box transcription factor	Activated	(Lamb *et al*., 2002; Winter *et al*., 2011)
At3g58070[b]	*GIS*	C2H2 transcription factor	ND	(Moyroud *et al*., 2011)
At3g61250[a,b]	*LMI2/MYB17*	R2R3 MYB transcription factor	Activated	(William *et al*., 2004; Winter *et al*., 2011)
At3g63010[a,b]	*GID1B*	Gibberellin receptor	ND	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At3g63530	*BB*	E3 ubiquitin ligase	ND	(Moyroud *et al*., 2011)
At4g18960[a,c]	*AG*	MADS box transcription factor	Activated	(Busch *et al*., 1999; Moyroud *et al*., 2011; Winter *et al*., 2011)
At4g35900[a,b]	*FD/ bZIP14*	bZIP transcription factor	ND	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At4g36260	*STY2/ SRS2*	RING finger-like zinc finger transcription factor	ND	(Winter *et al*., 2011; Moyroud *et al*., 2011)
At4g37750	*ANT/ CKC1/ DRG*	AP2/ERF-type transcription factor	ND	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At5g03790[a,b]	*LMI1/ATHB51*	Homeodomain leucine zipper class I transcription factor	Activated	(William *et al*., 2004) Winter *et al*., 2011)
At5g03840[b]	*TFL1*	Phosphatidylethanolamine- binding protein belonging to CETS gene family	Repressed	(Winter *et al*., 2011; Moyroud *et al*., 2011)
At5g10510[b]	*AIL6/ PLT3*	AP2-domain transcription factor	ND	(Winter *et al*., 2011; Moyroud *et al*., 2011)
At5g11320	*YUC4*	Flavin monooxygenase	ND	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At5g11530[b]	*EMF1*	Histone H3-K27 methylase	Repressed	(Winter *et al*., 2011)
At5g15230[a]	*GASA4*	Gibberellin-regulated protein with redox activity	Activated	(Wagner *et al*., 2004; Moyroud *et al*., 2011)
At5g20240[a]	*PI*	MADS box transcription factor	Activated	(Winter *et al*., 2011)
At5g28640[b]	*AN3/ GIF1*	Transcriptional coactivator	ND	(Moyroud *et al*., 2011; Winter *et al*., 2011)
At5g46330[a]	*FLS2*	Leucine-rich repeat serine/threonine protein kinase	Activated[h]	(Winter *et al*., 2011)
At5g49770	*LMI3*	Leucine-rich repeat serine/threonine protein kinase	Activated	(William *et al*., 2004)
At5g53950	*CUC2/ ANAC098*	NAC family transcription factor	Activated[h]	(Wagner *et al*., 2004; Winter *et al*., 2011)
At5g60630	*LMI4*	Expressed protein	Activated	(William *et al*., 2004)
At5g61850[a,b,i]	*LFY*	Plant specific transcription factor	Activated	(Winter *et al*., 2011; Moyroud *et al*., 2011)

LFY targets were defined in the following ways: the gene had to be identified in at least two independent publications or by at least two independent experimental techniques or both. [a]Also SEP3 targets (Kaufmann *et al*., 2009); [b]Also AP1 targets (Parcy *et al*., 1998; Ng and Yanofsky, 2001; Lamb *et al*., 2002; Kaufmann *et al*., 2010; Winter *et al*., 2011); [c]Also WUS target (Lenhard *et al*., 2001; Lohmann *et al*., 2001); [d]Also LMI2 target (Pastore *et al*., 2011); [e]Also LMI1 target (Saddic *et al*., 2006); [f]Also UFO target (Chae *et al*., 2008); [g]ND, no data; [h]Genevestigator (*lfy-12* vs. Col dataset; (Zimmermann *et al*., 2004; Zimmermann *et al*., 2005); [i]Also AGL24/SVP targets (Grandi *et al*., in press).

TABLE 2

SUMMARY OF LFY CO-FACTORS

Locus ID	Gene Name	Encoded protein	Experimental evidence	References
At1g24260	*SEP3*	MADS box transcription factor	GST pull-down	(Liu *et al.*, 2009)
At1g30950	*UFO*	F-box protein	EMSA; GST pull-down; Y2H; co-IP; co-ChIP	(Chae *et al.*, 2008)
At3g61250	*LMI2/AtMYB17*	R2R3 MYB transcription factor	GST pull-down; Y2H; BiFC	(Pastore *et al.*, 2011)

LFY co-factors were defined as proteins that have been demonstrated to physically interact with LFY. EMSA, electromobility shift assay; Y2H, yeast two-hybrid; co-IP, co-immunoprecipitation; co-ChIP, co-chromatin immunoprecipitation; BiFC, bimolecular fluorescence complementation.

four classes of the floral organ identity genes (Fig. 5 and Table 1; Weigel and Meyerowitz, 1994). SEP3 and AP1 also regulate many of these genes (Table 1) and may act in common regulatory complexes with LFY. As mentioned above, some LFY target genes function in GA signaling. In addition to its role in floral initiation, GA signaling also activates floral homeotic gene expression (Yu *et al.*, 2004b), suggesting that LFY may act through these targets to regulate these genes in addition to its direct effects on their transcription. LFY directly activates *AP1* (Wagner *et al.*, 1999), which in addition to its function in floral meristem identity also functions as an A class gene in *Arabidopsis* (Irish and Sussex, 1990). The other classical A class gene, *AP2*, is not directly regulated by LFY. LFY also activates the E class genes *SEP3* and *SEP4* (Table 1), necessary for specification of all four whorls of the flower (Pelaz *et al.*, 2000; Ditta *et al.*, 2004).

LFY plays an especially important role in activation of the B class genes, as reflected in the complete absence of petals and stamens in an *lfy* null mutant (Fig. 3). It directly activates the expression of the B class genes *APETALA3 (AP3)* and *PISTILLATA (PI;* Fig. 5 and Table 1; Lamb *et al.*, 2002; Winter *et al.*, 2011). AP1 also regulates these genes (Ng and Yanofsky, 2001; Kaufmann *et al.*, 2010). However, LFY requires co-factors to activate B class genes, as it has been shown to be unable to do so on its own unless fused to a strong transcriptional activation domain (Parcy *et al.*, 1998). The F-box encoding gene *UFO* was originally identified for its roles in establishing the whorled phyllotaxy within the flower, floral determinacy and activating *AP3* and *PI* (Ingram *et al.*, 1995; Levin and Meyerowitz, 1995; Wilkinson and Haughn, 1995). At stages 2 and 3 of flower development, *UFO* is expressed in a domain that includes the presumptive petal and stamen primordia (Ingram *et al.*, 1995) and *UFO* activity is necessary for organ identity at these stages (Laufs *et al.*, 2003). Subsequent work showed that UFO activity is dependent on LFY and that both LFY and UFO are necessary for expression of *AP3* outside of the flower; this data lead to the proposal that UFO acts as a LFY co-factor to activate B class gene expression (Lee *et al.*, 1997). UFO is an F-box protein (Ingram *et al.*, 1995; Samach *et al.*, 1999). F-box proteins form part of SCF ubiquitin ligase complexes that polyubiquitinate proteins, targeting them for destruction via the 26S proteasome (Sullivan *et al.*, 2003; Wang *et al.*, 2003; Ni *et al.*, 2004). UFO has been shown to interact with components of SCF complexes and these complex members function in flower development (Samach *et al.*, 1999; Zhao *et al.*, 1999; Wang *et al.*, 2003; Ni *et al.*, 2004) and regulate B class gene expression (Zhao *et al.*, 2001). A model was proposed whereby UFO functioned in an SCF complex to mediate ubiquitination of a negative regulator of *AP3* expression (Samach *et al.*, 1999). Subsequently, UFO was shown to physically interact with LFY both *in vitro* and *in vivo* (Chae *et al.*, 2008), leading to the current model that UFO is involved in modifying LFY in order to enhance its transcriptional activity (Chae *et al.*, 2008). Similar activities for F-box proteins have been reported previously in yeast and mammals (Muratani and Tansey, 2003). UFO orthologs in petunia and rice [DOUBLE TOP (Souer *et al.*, 2008) and ABERRANT PANICLE ORGANIZATION1 (APO1; Ikeda-Kawakatsu *et al.*, 2012), respectively] have been shown to physically interact with their respective LFY orthologs (ABERRANT LEAF AND FLOWER and RFL/APO2, respectively), suggesting that the dependence of LFY on F-box regulation for some of its activity is conserved across angiosperms.

LFY directly activates the class C gene *AG* (Busch *et al.*, 1999). However, LFY is not absolutely required for its expression as *lfy* mutants have detectable amounts of *AG* (Weigel and Meyerowitz, 1993) and still make abnormal carpels (Fig. 3). UFO has been shown activate *AG* transcription in cooperation with LFY (Wilkinson and Haughn, 1995; Souer *et al.*, 2008). *AG* is necessary not only for stamen and carpel identity, but also for floral determinacy. *WUSCHEL (WUS)* is one of the key genes involved in maintaining stem cell populations of shoot meristems

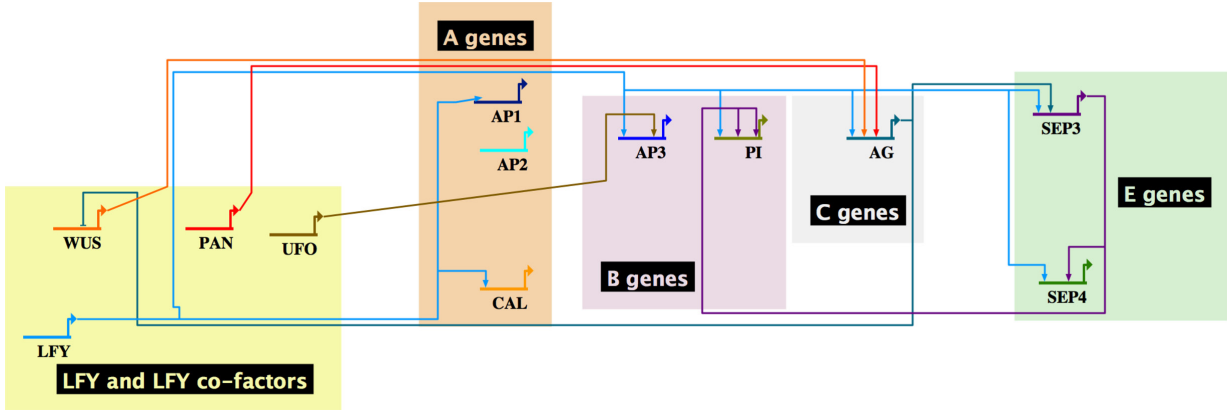

Fig. 5. Floral organ identity is controlled by multiple feedback loops. *The network was visualized using the BioTapestry program (Longabaugh* et al.*, 2009). LFY controls A, B, C and E class floral homeotic gene expression. Regulatory interations between floral homeotic genes are not shown to emphasize LFY's regulatory role.*

and encodes a homeobox transcription factor (Laux *et al.*, 1996; Mayer *et al.*, 1998). In stage 6 flowers the termination of *WUS* expression causes floral meristem termination (Lenhard *et al.*, 2001; Lohmann *et al.*, 2001). In addition, *WUS* functions as a co-factor of LFY in regulating expression of *AG* in the inner whorls (Fig. 5). LFY and WUS bind very closely together on *AG* cis-elements but do not physically interact with each other (Busch *et al.*, 1999; Lenhard *et al.*, 2001; Lohmann *et al.*, 2001; Hong *et al.*, 2003). Both LFY and WUS have to be present on the promoter to activate transcription (Lohmann *et al.*, 2001). *WUS* is expressed only in the center of the meristem (Mayer *et al.*, 1998), giving spatial specificity to the activation of *AG*. In turn, AG functions to repress *WUS* expression, therefore terminating floral meristem division. To date, only one other common target gene of LFY and WUS has been identified, although its regulation by these genes has not been confirmed. Both transcription factors are putative regulators of *HAP3B* (Busch *et al.*, 2010; Winter *et al.*, 2011). HAP3B is a CCAAT-binding transcription factor that has been implicated in regulation of flowering time (Cai *et al.*, 2007; Chen *et al.*, 2007). Its role in the floral meristem has not been investigated. Another activator of *AG* transcription, the TGA transcription factor PAN, may function with LFY (Fig. 5; Das *et al.*, 2009; Maier *et al.*, 2009). LFY and PAN both bind to binding sites in the *AG* second intron to promote *AG* expression and a *lfy* mutation enhances the floral defects of *pan* mutations, suggesting PAN may act as a co-factor of LFY, although it is unknown if they act in the same complexes (Das *et al.*, 2009; Maier *et al.*, 2009). Other transcription factors are likely to function in regulation of floral homeotic gene expression. For example, two BELL1-like homeodomain transcription factors, PENNYWISE (PNY) and POUND-FOOLISH (PNF), have been shown to act in parallel to LFY, UFO and WUS to regulate *AP3* and *AG* (Yu *et al.*, 2009).

In addition to these transcription factors, LFY interacts with epigenetic factors to regulate its homeotic gene targets. *SPLAYED* (*SYD*) interacts with *LFY* to regulate B class gene expression. SYD is a member of the Snf2p ATPase family of chromatin remodeling factors. It was identified though a genetic screen done to isolate enhancers of weak *lfy* mutant phenotypes (Wagner and Meyerowitz, 2002). It acts in the LFY-dependent activation of *AP3* and *PI* expression (Wagner and Meyerowitz, 2002), as well as other LFY-related functions as well as other developmental processes (Kwon *et al.*, 2005; Kwon *et al.*, 2006; Bezhani *et al.*, 2007) and stress responses (Walley *et al.*, 2008). A recent genome level study of LFY target genes identified potential LFY co-factor motifs from a *de novo* bioinformatic motif analysis (Winter *et al.*, 2011). Two such motifs, GA-repeat hexamers and octamers, were proposed to help recruit stage specific LFY co-factors. This type of repeats is often found in Polycomb Responsive Elements (PREs). The first plant polycomb protein indentified, CURLY LEAF (CLF), was found because of its function in repressing *AG*, *PI* and *AP3* expression in leaves (Goodrich *et al.*, 1997) and *AG* in the inflorescence stem and the first two whorls of the flower (Goodrich *et al.*, 1997; Schubert *et al.*, 2006). EMRYONIC FLOWER 2 (EMF2) and FERTILIZATION INDEPENDENT ENDOSPERM (FIE), which are also PcG proteins, repress *AP3*, *PI* and *AG* during vegetative growth (Chen *et al.*, 1997; Kinoshita *et al.*, 2001; Yoshida *et al.*, 2001). *TFL2*, also known as *LIKE-HETEROCHROMATIN PROTEIN 1* (*LHP1*), is expressed in proliferating cells of meristems, including in the FM, and binds to chromatin marked with H3K27me3 (Turck *et al.*, 2007; Zhang *et al.*, 2007). TFL2 directly associates with regulatory regions of *AP3*, *PI*, *AG* and *SEP3* and represses their expression during vegetative growth (Kotake *et al.*, 2003; Zhang *et al.*, 2007). A plant specific factor, EMF1 (Aubert *et al.*, 2001), also acts, together with EMF2-containing PcG complexes, to repress *AG*, *AP3* and *PI* expression during vegetative development (Calonje *et al.*, 2008; Kim *et al.*, 2010); interestingly, *EMF1* is a direct target of LFY (Table 1), which represses its expression, suggesting LFY might derepress floral homeotic gene expression as well as directly activating it. Clearly, LFY must cooperate and interact with a range of epigenetic enzymes and markers in order to correctly regulate its target genes.

LFY and floral reversion

Once the transition to flower formation has occurred, it is important to maintain this status to prevent floral reversion and lead to successful reproduction. Floral reversion is the reinitiation of shoot growth from a partially formed flower and was first described in 1880 (Buckhout, 1880). As discussed above, one mechanism *Arabidopsis* uses to prevent reversion is positive feedback loops among the floral meristem identity genes (Fig. 2). In *Arabidopsis*, floral reversion has been observed in floral meristem identity mutants including *lfy* and *ap1* under the noninductive short day photoperiod (Huala and Sussex, 1992; Bowman *et al.*, 1993; Okamuro *et al.*, 1996; Liu *et al.*, 2007). This phenotype is partially due to the inappropriate expression of three genes with roles in flowering time regulation, *AGL24*, *SVP* and *SUPPRESSOR OF CONSTANS1 (SOC1);* (Yu *et al.*, 2004a; Liu *et al.*, 2007), which all encode MADS box transcription factors. Overexpression of *AGL24* promotes the transformation of FMs into IMs, overexpression of *SOC1* further enhances this and overexpression of *SVP* converts FMs into vegetative shoots (Yu *et al.*, 2004a; Liu *et al.*, 2007). Loss of function mutants in these three genes individually or in combination reduces reversion defects seen in *ap1* mutants (Liu *et al.*, 2007). It has been shown that AP1 binds directly to promoters of these genes and represses their expression (Fig. 2; Liu *et al.*, 2007; Gregis *et al.*, 2008). LFY also may directly regulate *SOC1* (Table 1; Moyroud *et al.*, 2011) and acts indirectly through AP1 to repress the other genes (Yu *et al.*, 2004a; Gregis *et al.*, 2008). SEP genes also function to prevent floral reversion. Chromatin immunoprecipitation has shown that SEP3 directly binds to the promoters of both *AGL24* and *SVP* (Gregis *et al.*, 2008). *sep* mutants have *AGL24* and *SVP* expressed in the FM beyond their normal time of expression. This data suggests that SEPs act as repressors of *AGL24* and *SVP* to maintain FM identity. Another putative target of LFY activation (Table 1), shared with both AP1 and SEP3, is the *TEM1/EDF1* gene, encoding a RAV family transcription factor with a novel transcriptional repression domain (Ikeda and Ohme-Takagi, 2009). TEM1 represses the flowering time gene *FT*, contributing to floral transition (Castillejo and Pelaz, 2008). Clearly, an important function of floral meristem identity genes is to repress, directly or indirectly, expression of flowering time genes in later stage flowers to maintain floral fate.

LFY and floral organ differentiation

LFY expression persists into floral development stages during which organ differentiation is beginning, suggesting it could func-

tion to regulate genes involved in organ morphogenesis (Weigel *et al*., 1992). This is further supported by the defects seen in the carpels made in strong *lfy* loss of function mutants (Fig. 3). In *lfy* mutant flower-like structures, the carpels are partially unfused, revealing the ovules, and the stigma and style are reduced. In support for a role of LFY in pistil differentiation, several of its putative direct target genes have been demonstrated to be necessary for this developmental process. These include ETT, necessary for proper apical-basal patterning of the carpels in response to auxin (Sessions and Zambryski, 1995; Sessions *et al*., 1997; Sessions, 1997; Nemhauser *et al*., 2000). Another putative LFY target, *STY2*, encoding a member of the SHI family of ring finger proteins, is redundantly necessary with other family members for the growth of the marginal tissues of the gynoecium (Kuusk *et al*., 2006). The CUP-SHAPED COTYLEDON2 (CUC2) NAC transcription factor is redundantly necessary (with CUC1) for fusion of the septa of the carpels (Ishida *et al*., 2000). Finally, *GOA/AGL63*, encoding a paralog of the B-sister MADS box transcription factor found only in Brassicaceae, is necessary for regulation of fruit growth (Erdmann *et al*., 2010; Prasad *et al*., 2010). *ETT* and *CUC2* also have earlier roles in floral development and more work will be needed to determine if their regulation by LFY is important for earlier floral development events, pistil morphogenesis or both. LFY target genes also function in other morphogenetic events within the flower. For example, GA is important for stamen filament growth (Peng, 2009) and its signaling is also regulated by LFY. Likely LFY directly controls genes involved in morphogenesis of most, if not all, floral organs.

Conclusions

LFY is necessary for the formation of flowers across the angiosperms and its orthologs in gymnosperms are also implicated in regulation of reproductive development. LFY functions to control multiple aspects of floral development, including organ number and identity, organ arrangement and floral meristem termination. It does this by acting in multiprotein transcriptional complexes to regulate gene expression. Studies in *Arabidopsis* are providing insight into reproductive development that can be applied to other plants. *LFY* expression promotes early flowering in a number of commercially important crops, including rice (*Oryza sativa*) and poplar (*Populus trichocarpa*) (Kyozuka *et al*., 1998; Rottmann *et al*., 2000). Therefore unraveling the molecular mechanisms by which LFY performs its functions will provide a basis for the development of new strategies to increase agronomical values such as increased yield by manipulating LFY in economically important crops.

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