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

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.

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

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.
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 innermost 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).

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, 1983; 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; Poutteau and Albertini, 2009; Poutteau 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 IDENTITITY1 (LM11; (Saddic et al., 2006) and the R2R3 class MYB transcription factor LATE MERISTEM IDENTITY2 (LM2/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 (CENTORADIALIS/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 (Manuel 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., 2000; 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, 2000; 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 axis 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; cal1-1 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, euAP1 (in which Arabidopsis AP1 and CAL fall) and euFUL (to which FUL belongs; Litt and Irish, 2003). The euFUL clade underwent a duplication event to generate two subclades: euFUL1, including FUL, and euFULII, 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 SEPA, 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., 1999; Yu et al., 2002; Michaels et al., 2003; Yu et al., 2004a; Lee et al., 2007; 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 well 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 ap1-1; 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
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 domestication 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., 1999). 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. Ify 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 PRL-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; Carlsebecker 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 hermaphrodite 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 (Percy 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 y-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/ANNNCCANTG/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 fly flowers 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). Ily 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 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-ERF 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.

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., 2006).
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

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### Table 1

**Summary of LFY Target Genes**

<table>
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<tr>
<th>Locus ID</th>
<th>Gene Name</th>
<th>Type of Protein</th>
<th>Regulation by LFY</th>
<th>References</th>
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<td>At1g16070</td>
<td>AtTLP8/ LMIS</td>
<td>TUBBY family transcription factor</td>
<td>Activated</td>
<td>(William et al., 2004; Winter et al., 2011)</td>
</tr>
<tr>
<td>At1g19850</td>
<td>MP1/ ARFS/I AA24</td>
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<td>(Moyroud et al., 2011)</td>
</tr>
<tr>
<td>At1g24260</td>
<td>SEP3/ AGL9</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>At1g25560</td>
<td>TEM1/ EDF1</td>
<td>RAV family transcription factor</td>
<td>Activated</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>At1g26310</td>
<td>CAL/ AGL10</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Wagner et al., 2004; William et al., 2004)</td>
</tr>
<tr>
<td>At1g30040</td>
<td>GA20X2</td>
<td>Gibberellin 2-oxidase</td>
<td>Activated</td>
<td>(Wagner et al., 2004; Moyroud et al., 2011)</td>
</tr>
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<td>At1g31140</td>
<td>GO4/ AGL63</td>
<td>MADS box transcription factor</td>
<td>ND</td>
<td>(Moyroud et al., 2011)</td>
</tr>
<tr>
<td>At1g59870</td>
<td>PEn3/ PDR8</td>
<td>ATP binding cassette transporter</td>
<td>ND</td>
<td>(Winter et al., 2011)</td>
</tr>
<tr>
<td>At1g59940</td>
<td>ARR3</td>
<td>Type A response regulator</td>
<td>Activated</td>
<td>(Moyroud et al., 2011)</td>
</tr>
<tr>
<td>At1g65620</td>
<td>AS2</td>
<td>Transcriptional repressor characterized by cysteine repeats and a leucine zipper</td>
<td>Activated</td>
<td>(Yamaguchi et al., in press)</td>
</tr>
<tr>
<td>At1g69120</td>
<td>AP1</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Wagner et al., 1999; Moyroud et al., 2011)</td>
</tr>
<tr>
<td>At1g80340</td>
<td>GA30X2</td>
<td>Gibberellin 3β-hydroxylase</td>
<td>ND</td>
<td>(Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A2g01420</td>
<td>PIN4</td>
<td>Auxin efflux carrier</td>
<td>Repressed</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A2g03710</td>
<td>SEP4/ AGL3</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A2g28610</td>
<td>PWS/ WOX3</td>
<td>WUSCHEL-like homeomain transcription factor</td>
<td>ND</td>
<td>(Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A2g33860</td>
<td>ETT/ ARF3</td>
<td>ARF family transcription factor</td>
<td>Activated</td>
<td>(Winter et al., 2011; Wagner et al., 2004)</td>
</tr>
<tr>
<td>A2g34650</td>
<td>PID/ ABR</td>
<td>Serine/threonine kinase</td>
<td>ND</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A2g45190</td>
<td>FIL/ AFO/ YAB1</td>
<td>YABBY transcription factor</td>
<td>ND</td>
<td>(Winter et al., 2011; Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A2g45660</td>
<td>SOC1/ AGL20</td>
<td>MADS box transcription factor</td>
<td>ND</td>
<td>(Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A3g47340</td>
<td>ASN1/ DIN6</td>
<td>Glutamine-dependent asparagine synthase</td>
<td>Activated</td>
<td>(Wagner et al., 2004; William et al., 2004)</td>
</tr>
<tr>
<td>A3g54340</td>
<td>AP3</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Lamb et al., 2002; Winter et al., 2011)</td>
</tr>
<tr>
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<td>GIS</td>
<td>C2H2 transcription factor</td>
<td>ND</td>
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<td>LMIS/ MYB17</td>
<td>R2R3 MYB transcription factor</td>
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</tr>
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<td>A3g63010</td>
<td>GID1B</td>
<td>Gibberelin receptor</td>
<td>ND</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A3g63530</td>
<td>BB</td>
<td>E3 ubiquitin ligase</td>
<td>ND</td>
<td>(Moyroud et al., 2011)</td>
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<tr>
<td>A4g18960</td>
<td>AG</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Busch et al., 1999; Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
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<td>A4g35900</td>
<td>FDI/ bZIP14</td>
<td>bZIP transcription factor</td>
<td>ND</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
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<tr>
<td>A4g36260</td>
<td>STY2/ SRS2</td>
<td>RING finger-like zinc finger transcription factor</td>
<td>ND</td>
<td>(Winter et al., 2011; Moyroud et al., 2011)</td>
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<tr>
<td>A4g37750</td>
<td>ANT/ CUC1/ ORG</td>
<td>AP2/ERF-type transcription factor</td>
<td>ND</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g03790</td>
<td>LMIS/ AATHBS1</td>
<td>Homeomain leucine zipper class I transcription factor</td>
<td>Activated</td>
<td>(William et al., 2004; Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g03840</td>
<td>TFL1</td>
<td>Phosphatidylethanolamine-binding protein belonging to CETS gene family</td>
<td>Repressed</td>
<td>(Winter et al., 2011; Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A5g10510</td>
<td>AIL6/ PLT3</td>
<td>AP2-domain transcription factor</td>
<td>ND</td>
<td>(Winter et al., 2011; Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A5g11320</td>
<td>YUC4</td>
<td>flavin monooxygenase</td>
<td>ND</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g11530</td>
<td>EMF1</td>
<td>Histone H3-K27 methylase</td>
<td>Repressed</td>
<td>(Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g15230</td>
<td>GASA4</td>
<td>Gibberellin-regulated protein with redox activity</td>
<td>Activated</td>
<td>(Wagner et al., 2004; Moyroud et al., 2011)</td>
</tr>
<tr>
<td>A5g20240</td>
<td>PI</td>
<td>MADS box transcription factor</td>
<td>Activated</td>
<td>(Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g28640</td>
<td>AN3/ GFI1</td>
<td>Transcriptional coactivator</td>
<td>Activated</td>
<td>(Moyroud et al., 2011; Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g46030</td>
<td>FLS2</td>
<td>Leucine-rich repeat serine/threonine protein kinase</td>
<td>Activated</td>
<td>(Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g49770</td>
<td>LM3</td>
<td>Leucine-rich repeat serine/threonine protein kinase</td>
<td>Activated</td>
<td>(Winter et al., 2004)</td>
</tr>
<tr>
<td>A5g53860</td>
<td>CUC2/ ANAC098</td>
<td>NAC family transcription factor</td>
<td>Activated</td>
<td>(Wagner et al., 2004; Winter et al., 2011)</td>
</tr>
<tr>
<td>A5g60090</td>
<td>LM4</td>
<td>expressed protein</td>
<td>Activated</td>
<td>(Wagner et al., 2004)</td>
</tr>
<tr>
<td>A5g16850</td>
<td>LFY</td>
<td>Plant specific transcription factor</td>
<td>Activated</td>
<td>(Winter et al., 2011; Moyroud et al., 2011)</td>
</tr>
</tbody>
</table>

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. *Also SEP3 targets (Kaufmann et al., 2009); Also AP1 targets (Parcy et al., 1998; Ng and Yanofsky, 2001; Lamb et al., 2002; Kaufmann et al., 2010; Winter et al., 2011); Also WUS target (Lenhard et al., 2001; Lohmann et al., 2001); Also LMI2 target (Pastore et al., 2011); Also LMI1 target (Saddic et al., 2006); Also UFO target (Chae et al., 2008); ND, no data; *Genevestigator (lfy-12 vs. Col dataset; Zimmermann et al., 2004; Zimmermann et al., 2005); Also AGL24/SVP targets (Grandi et al., in press).
At3g61250 LMI2/AtMYB17

Sibata et al., 2004). UFO has been used to examine the interaction between LFY and its co-factors, which were defined as proteins that have been demonstrated to physically interact 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 role 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 ABER-RANT 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 ify 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 and is necessary for the proper formation of floral meristems.
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 fly 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 factors. It was identified though a genetic screen done to isolate factors (Calonje 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/ED1 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 *fly* loss of function mutants (Fig. 3). In *fly* mutant flower-like structures, the carpels are partially fused, 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 Zambrayski, 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*) (Kyozuaka 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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**References**


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