The making of a flower: control of floral meristem identity in *Arabidopsis*

Mark S. Pidkowich, Jennifer E. Klenz and George W. Haughn

During the reproductive phase of a plant, shoot meristems follow one of two developmental programs to produce either flowers or vegetative shoots. The decision as to which meristems give rise to flowers, and when they do so, determines the general morphology of an inflorescence. Molecular and genetic research in *Arabidopsis* and other model species has identified several genes that control the identity that a meristem will adopt. These meristem identity genes are activated in response to developmental and environmental cues, and can be assigned to three basic categories: those required either to initiate or maintain the floral program in some meristems and those required to maintain the vegetative program in others.

A plant's reproductive success is dependent on its ability to develop flowers. During vegetative plant growth the shoot apical meristem (SAM) establishes the shoot architecture by giving rise to a reiterative series of primordia that develop as leaves and/or lateral vegetative shoots (branches) with their own apical meristem. Following the switch to reproductive growth, most or all of the apical meristems give rise to flowers. Development of the floral shoot differs from that of the vegetative shoot in several dramatic ways.

- The flower has several types of specialized floral organs of which the number, arrangement and morphology are species specific.
- Initiation and development of lateral shoots are typically suppressed in the floral shoot.
- Unlike most vegetative shoots, floral shoots are determinate, and cease growing after the last reproductive organs have been initiated.

Clearly, floral development requires the coordination of a complex set of events.

In the past decade considerable progress has been made towards understanding the mechanisms by which a plant determines at what time, and in which meristems, the floral program is activated. Central to this progress has been the identification and cloning of the genes that are required to determine the fate of meristems in several species, including *Arabidopsis*, *Antirrhinum* and pea. In this review we focus on *Arabidopsis*, and highlight the concepts and approaches used to define the function and regulation of such meristem identity genes. It appears that the general rules governing floral development in *Arabidopsis* are applicable to all dicots. Additional information on the topic is available in several recent reviews[1,2].

Arabidopsis shoot morphology

Following germination, the *Arabidopsis* SAM generates leaf primordia in a spiral phyllotaxy to form a basal rosette with each leaf having an undeveloped lateral meristem in its axil. Transition to the reproductive phase, sometimes referred to as 'floral evocation' is facultative and promoted by long days (16 h photoperiod) or vernalization. Initially both the primary meristem and preformed secondary meristems give rise to shoots with vegetative characteristics – each grows indeterminately giving rise to lateral shoots in a spiral. However, following floral evocation all secondary meristems initiated adopt a floral identity, resulting in an inflorescence consisting of a spiral of flowers arranged on the primary shoot (Fig. 1). Although the morphology of the primary shoot differs before and after the reproductive transition, for simplicity we will refer to all shoots that are not flowers as vegetative shoots.

The floral initiation process

How does a plant switch from making vegetative shoots to making flowers? The discovery of loss-of-function mutants, in which flowers are replaced by structures intermediate between floral and vegetative shoots, revealed the existence of master regulators that control the entire floral initiation process (FLIP). To date, five *Arabidopsis* FLIP regulatory genes have been identified by mutation and cloned: *LEAFY* (*LFY*), *APETALA1* (*AP1*), *CAULIFLOWER* (*CAL*), *APETALA2* (*AP2*) and *UNUSUAL FLORAL ORGANS* (*UFO*)[3–17]. Single and double mutant phenotypes, gene sequence analysis and expression, and transgenic studies have provided information about how these genes function and interact to promote the floral program. Two of the FLIP genes, *LFY* and *AP1*, are considered to play a primary role in initiating the floral program. Loss-of-function mutations in either gene results in strong floral to vegetative homeotic transformation phenotypes. Plants with loss-of-function mutations in both genes fail to produce shoots with floral characteristics (Figs 1 and 2)[4–6,8,9]. Ectopic expression of either gene during the vegetative phase results in precocious flower formation, indicating that they can activate the floral program[18,19]. Both genes encode putative transcription factors and are strongly expressed in floral primordia[5,7]. Thus *LFY* and *AP1* are master regulators that mark primordial meristematic cells for a floral fate.

A third key gene, *CAL*, was identified owing to a recessive *cal* mutation that dramatically enhances the *ap1* floral mutant phenotype[8]. In *ap1* plants, floral shoots fail to develop sepals and petals (replaced by a few nodes, each bearing a leaf with an associated lateral shoot), but the terminal portion of the shoot consists of normal reproductive organs (Fig. 2). In contrast, *cal ap1* double-mutant floral meristems give rise to an indeterminate spiral of lateral meristems, each of which initiate third-order meristems and so on, such that the floral shoot develops as a mass of apical meristems with no differentiated organs (Fig. 2)[8]. This phenotype is equivalent to the morphology of the *Brassica oleracea* cauliflower varieties. Mutations in *CAL* alone result in no obvious morphological abnormalities, indicating that *CAL* is functionally redundant to *AP1*, a hypothesis also supported by the high similarity in sequence and expression patterns shared between the two genes[10].

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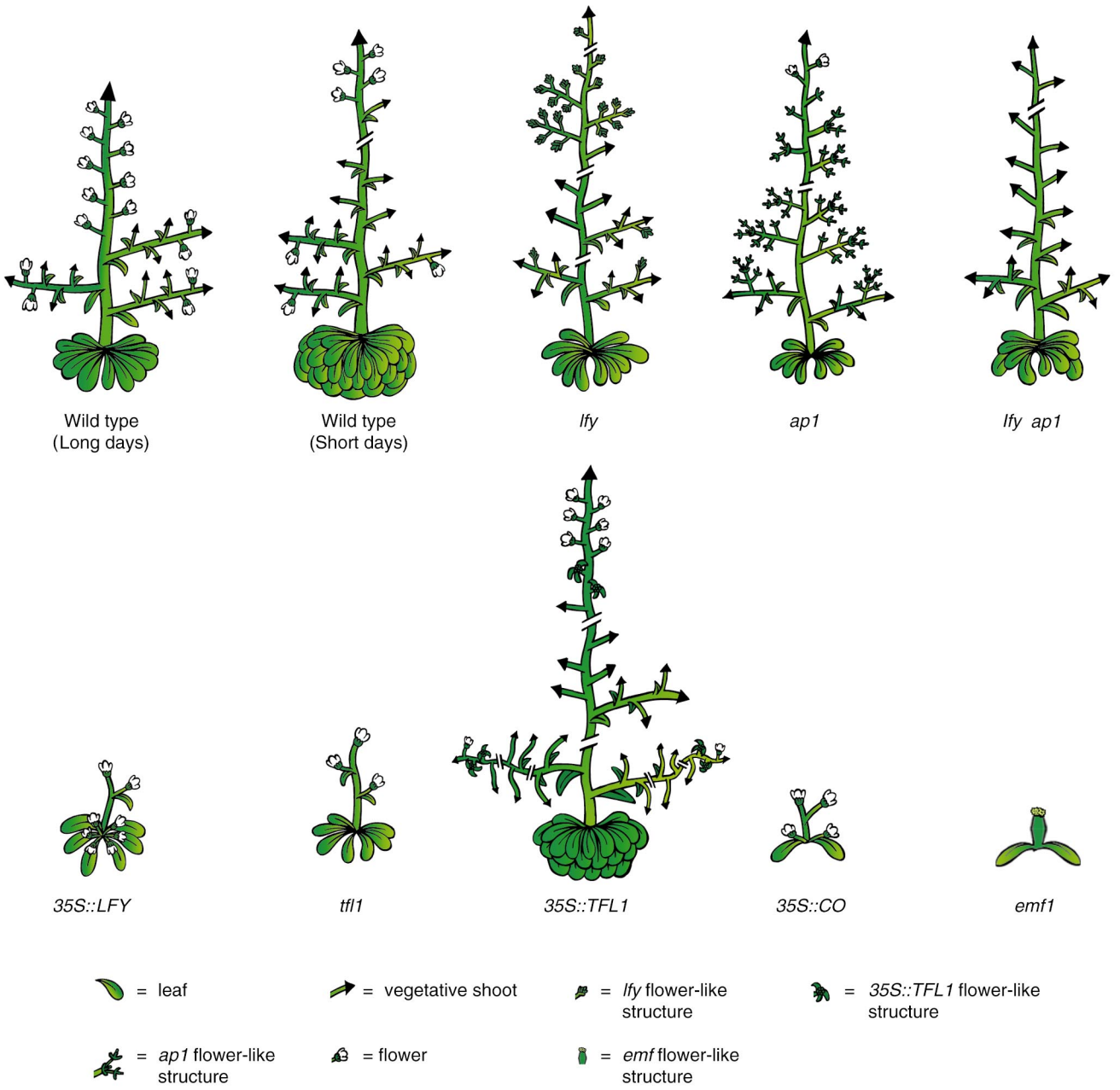

Fig. 1. Stylized illustrations of *Arabidopsis* shoot morphology in wild type and various mutants. The wild-type shoot grows indeterminately, producing lateral branches and then flowers. Flowering is delayed in short days relative to long days. Loss-of-function mutations at certain loci [*leafy* (*lfy*), *apetala1* (*ap1*)] affect the floral initiation process resulting in vegetative shoots and/or shoots intermediate between vegetative shoots and flowers replacing the shoots that develop as flowers in the wild type. Basal-most shoots are more vegetative, while the apical-most shoots are more floral. Double mutant combinations between *lfy* and *ap1* result in almost no flower-like structures being produced. In contrast, loss-of-function mutations at other loci [*terminal flower 1* (*tfl1*), *embryonic flower 1* (*emf1*)] cause vegetative shoots to develop as flowers, including a terminal flower. These vegetative to floral homeotic transformations, and vice versa, can be mimicked by the ectopic expression of genes that promote [fusion of the *35S* promoter to the *LFY* coding sequence (*35S::LFY*), *35S::CONSTANS* (*CO*)] or inhibit (*35S::TFL1*) the floral program. For example, ectopic expression of *TFL1* under the control of the *35S* promoter results in the replacement of flowers by vegetative shoots, which is the opposite of what occurs when *TFL1* function is lost. A severe *emf1* phenotype, which can amount to little more than a solitary gynoecium rising above the cotyledons[49], is depicted disproportionately large for the purposes of this review.

How do the FLIP genes function to promote the floral program? Much of the information available to date is summarized in Fig. 3. At least three lines of evidence from mutant phenotypic analysis and expression studies indicate that an important function of *LFY*, and *AP1* and *CAL* function (*AP1*/*CAL*) is to up-regulate each other in the floral primordium.

- Individual *lfy* and *ap1* loss-of-function mutants produce lateral shoots intermediate in morphology between a vegetative shoot and a flower[3–6,8,9]. The first such lateral shoots to develop (rosette proximal) are more vegetative in morphology, whereas each successive shoot (rosette distal) is more floral-like than the previous one (Fig. 1). Because *lfy ap1* double mutants never

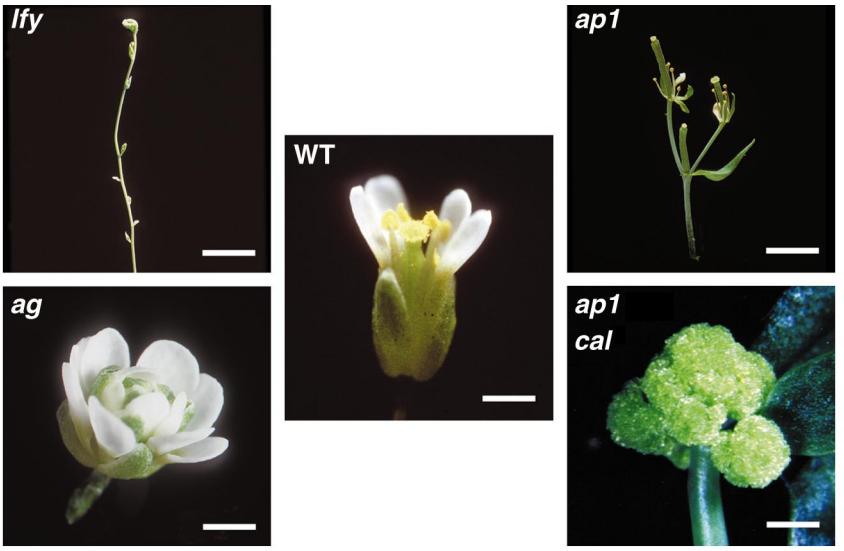

Fig. 2. Wild type (WT) and mutant *Arabidopsis* flowers. Loss-of-function mutations in *LEAFY* (*LFY*), *APETALA1* (*AP1*), *CAULIFLOWER* (*CAL*) and *AG* (*AGAMOUS*) compromise the capacity of a meristem to develop as a flower. *lfy* mutations cause indeterminate leafy branches to develop instead of flowers. Mutations in *ap1* result in flowers where sepals and petals are replaced by leaves with associated lateral shoots, whereas the reproductive organs develop normally. Mutations in *ag* result in indeterminate flowers that produce only sepals and petals. In short days, these flowers can revert to vegetative shoots (not shown). Double mutant combinations of *ap1 cal* cause flowers to be replaced by a cluster of undifferentiated meristems which resembles a cauliflower. Scale bars: *lfy*, 4.0 mm; *ap1*, 3.0 mm; WT, 0.8 mm; *ag*, 0.8 mm; *ap1 cal*, 0.5 mm.

form flower-like shoots[4,5,9] (Fig. 1) we conclude that *lfy* single mutants develop flowers due to the presence of *AP1*, and *ap1* single mutants form flowers due to the presence of *LFY*. The gradual transition from vegetative shoots to floral shoots within a single *lfy* or *ap1* mutant inflorescence suggests that both genes are required for the sharp transition observed in wild-type inflorescences.

- *LFY* is expressed more weakly in a *cal ap1* double mutant whereas *AP1* expression is delayed in a *lfy* mutant[7,19].
- Recent evidence strongly suggests that LFY can bind to a regulatory element of the *AP1* promoter to activate *AP1* transcription directly[20].

This cooperativity in FLIP gene upregulation probably ensures that the independently activated FLIP genes are expressed at high enough levels in the same floral primordium to ensure a complete switch to the floral program. This is especially important given that *LFY* and *AP1*/*CAL* functions are required to promote different aspects of the floral program.

The differences in *lfy*, *ap1* and *ap1 cal* loss-of-function mutant phenotypes demonstrate that *LFY* and *AP1*/*CAL* promote distinct aspects of the floral program (Fig. 3). Because loss-of-function mutations in one FLIP gene lead to a decrease in expression of other FLIP genes, it is difficult to ascertain the exact role of each FLIP gene in the floral program. Nevertheless, *LFY* appears to suppress leaf development and, within the flower, inhibits internode elongation while promoting Class B (petal and stamen) and Class C (stamen and carpel) organ identity (Fig. 3)[5,21]. In contrast *AP1*/*CAL* appears to suppress branching

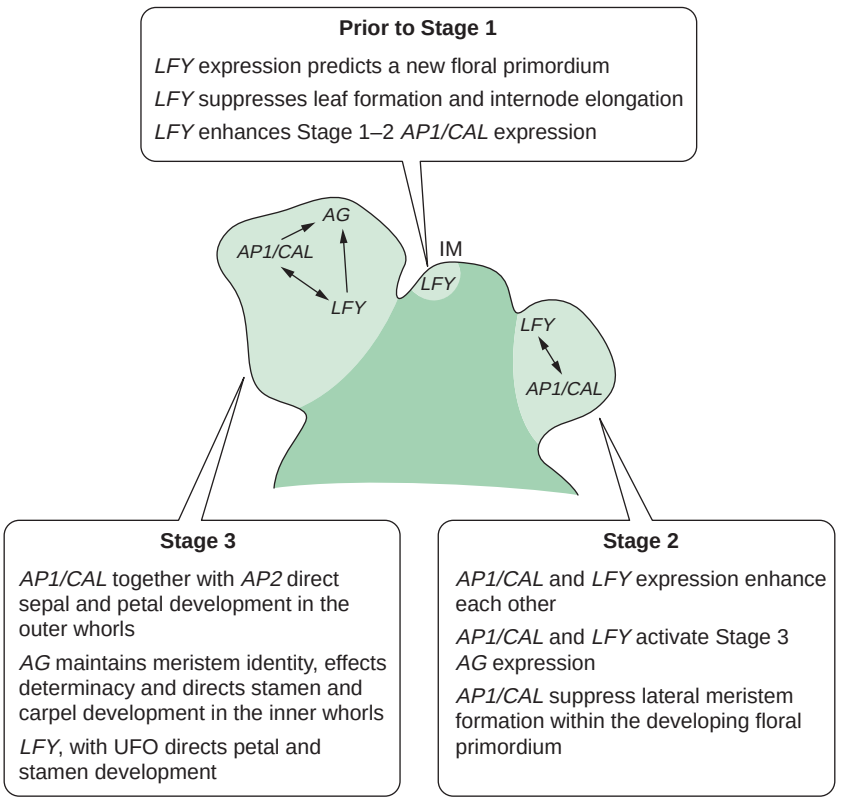

Fig. 3. Functional relationship between *LEAFY* (*LFY*), *APETALA1* (*AP1*), *CAULIFLOWER* (*CAL*) and *AGAMOUS* (*AG*). *LFY* is expressed before other floral initiation process (FLIP) genes. Early in floral meristem identification other FLIP genes are activated independent of *LFY*, but their expression is relatively weak. One role of *LFY* is to enhance the activation of *AP1*, *CAL* and *AG* at this stage of development. In turn *AP1* and *CAL* enhance expression of *LFY*. Once expressed, the FLIP genes are required to direct different aspects of floral development. For example, *LFY* is required for petal and stamen development and plays a role in the activation of *AG*, while *AP1* is required for sepal and petal development and indirectly for stamen and carpel development by activating the organ identity gene, *AG*. *AG* maintains meristem identity in the center of the floral primordium as well as promoting stamen and carpel development. (IM, inflorescence meristem; *UFO*, *UNUSUAL FLORAL ORGANS*.)

and promote Class A (sepal and petal) and Class C organ identity (Fig. 3)[8]. Both *LFY* and *AP1*/*CAL* probably promote Class C organ identity by activating transcription of the floral organ identity gene *AGAMOUS* (*AG*)[20,22].

Two other genes, *AP2* and *UFO*, have been implicated in the FLIP. Unlike *lfy* and *ap1*, the *ap2* or *ufo* mutants on their own have only subtle defects in meristem identity[9,11,13,15,16,23]. However *ap2* or *ufo* loss-of-function mutations enhance the meristem identity defects caused by *ap1* mutations[6,8,9,15,16,23]. What roles do these two genes play in conferring meristem identity? *UFO* appears to control only a subset of the functions regulated by *LFY* and requires *LFY* activity for its function (but not its expression)[20,24]. Therefore *UFO* could lie downstream of *LFY* in the regulatory hierarchy and control only a component of *LFY* function. Indeed it has been suggested that *UFO* and *LFY* are coactivators of Class B organ identity[24], with the *UFO* expression pattern[17,24] providing positional information[20]. Unlike the other FLIP genes that appear to encode transcription factors, *UFO* encodes an F-box protein: a class of proteins that target other proteins for destruction via the ubiquitin pathway, and are in many cases associated with the control of cell division[25,26]. Hence, identification of UFO protein targets could lead to a better understanding of *UFO* function.

AP2 could play a more substantial role in FLIP than is suggested by the *ap2* mutant phenotype. In *ap2* mutant flowers the *AG* gene is expressed ectopically in the outer (sepal and petal) whorls[27]: *AG* can itself promote floral meristem identity. Therefore in the *ap2* mutant, *AG* could partly compensate for the loss of *AP2* FLIP activity, thereby masking any meristem identity defects. Indeed, an *ap2* allele which gives rise to relatively weak ectopic expression of *AG* in the perianth has a greater effect on meristem identity than other *ap2* alleles[4,8,9]. However, genes other than *AG* would have to be involved in compensating for *ap2* mutations as no dramatic inflorescence defects have been reported in *ap2 ag* double mutants.

Maintaining the floral program

Some plant species, such as *Impatiens balsamina*, produce flowers that revert to vegetative shoots upon placement in non-inductive conditions[28]. However, in many species, including *Arabidopsis*, the floral program once initiated continues until completion, suggesting that mechanisms must exist to maintain the floral state. Recent studies on the *Arabidopsis AG* gene have identified just such a mechanism.

AG, which encodes a MADS domain transcription factor[29], is best known for its role in specifying reproductive organ identity and maintaining determinate growth within the floral shoot. In agreement with these functions, *AG* is transcribed later than FLIP genes and is limited to the center of the floral primordium. Loss of *AG* function results in homeotic conversions of reproductive organs to sepals and petals and indeterminate growth of the flower when the plant is grown in long (>16 h) photoperiods (Fig. 2)[30]. Significantly, the indeterminate *ag* floral meristems revert to flower-bearing vegetative meristems when grown under short days or in combination with *ap1*, indicating that *ag* flowers are not irreversibly committed to developing as flowers[8,22,31]. Furthermore, ectopic expression of *AG* is sufficient to transform vegetative meristems into flower-like shoots, indicating that *AG* can promote the floral program in a manner similar to a FLIP gene. Hence, *AG* represents a distinct class of meristem identity gene, which is required to maintain the floral program (Fig. 3).

Maintaining the vegetative program

Like many other angiosperm species, not all *Arabidopsis* shoot meristems develop as flowers following floral evocation. The primary meristem, and usually the lateral meristems initiated in the leaf axils, maintains an indeterminate vegetative program that gives rise to lateral shoots (Fig. 1). Thus, during the reproductive phase the plant is able to control which meristems will develop as flowers and which will not, thereby determining the architecture of the inflorescence. The study of one *Arabidopsis* gene, *TERMINAL FLOWER 1* (*TFL1*)[32,33], and counterparts from parallel systems[34,35] has provided insight into how the plant accomplishes this feat. Recessive mutations in *TFL1* result in the conversion of all apical meristems into floral meristems upon floral evocation (Fig. 1)[32,33], implicating this gene as a major player in maintaining the vegetative identity in inflorescence meristems. Consistent with such a function, *TFL1* is initially transcribed at low levels in the shoot apex before phase transition, and up-regulated in those meristems that will follow a vegetative program upon floral evocation[36]. The putative TFL1 protein has sequence similarity to phosphatidylethanolamine-binding proteins in animals, which can bind to membrane protein complexes[36,37]. Therefore, the hypothesis that *TFL1* is involved in transducing a signal generated by a floral program repressor in the SAM is attractive (Fig. 4).

The exact method of *TFL1* action in regulating FLIP is not clear. Recent work, however, demonstrates that ectopic *TFL1* expression neither eliminates *LFY* or *AP1* expression nor abolishes the floral program, but appears to distend the phase transition such that lateral shoots become progressively more flower-like towards

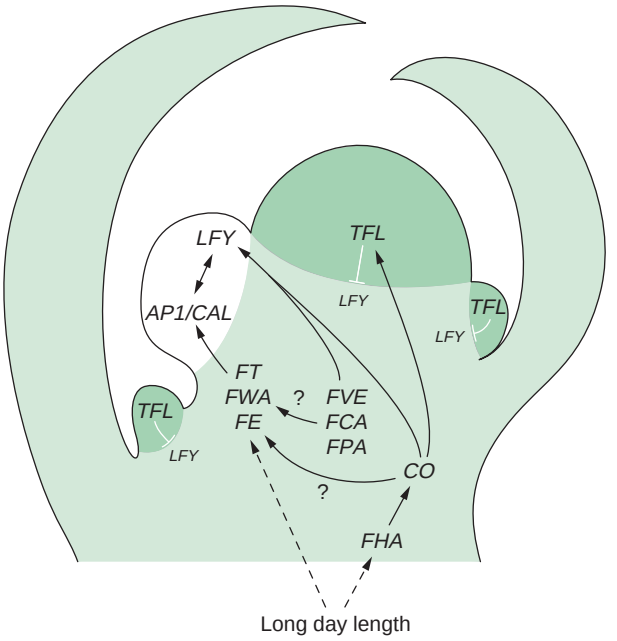

Fig. 4. Interactions between floral initiation process (FLIP) genes and flowering-time genes. Arrows depict positive interactions and bars depict negative interactions. Flowering is stimulated constitutively or by long day length (broken arrows), and both pathways have components that function to promote primarily *LEAFY* (*LFY*) or *APETALA1* and *CAULIFLOWER* function (*AP1*/*CAL*). In response to long days, *FHA* (*CRY2*) promotes *CONSTANS* (*CO*) which in turn promotes *LFY*. Meanwhile, *FE*, *FWA* and *FT* promote *AP1*/*CAL* in a parallel pathway (white primordium). *CO* also serves to promote *TERMINAL FLOWER 1* (*TFL1*), which inhibits FLIP in the vegetative meristems (dark green primordia). *FVE*, *FCA* and *FPA* (similar to *CO* function) work in the constitutive pathway to promote *LFY*, although they might also enhance *AP1* and other genes through *FE*, *FWA* and *FT*.

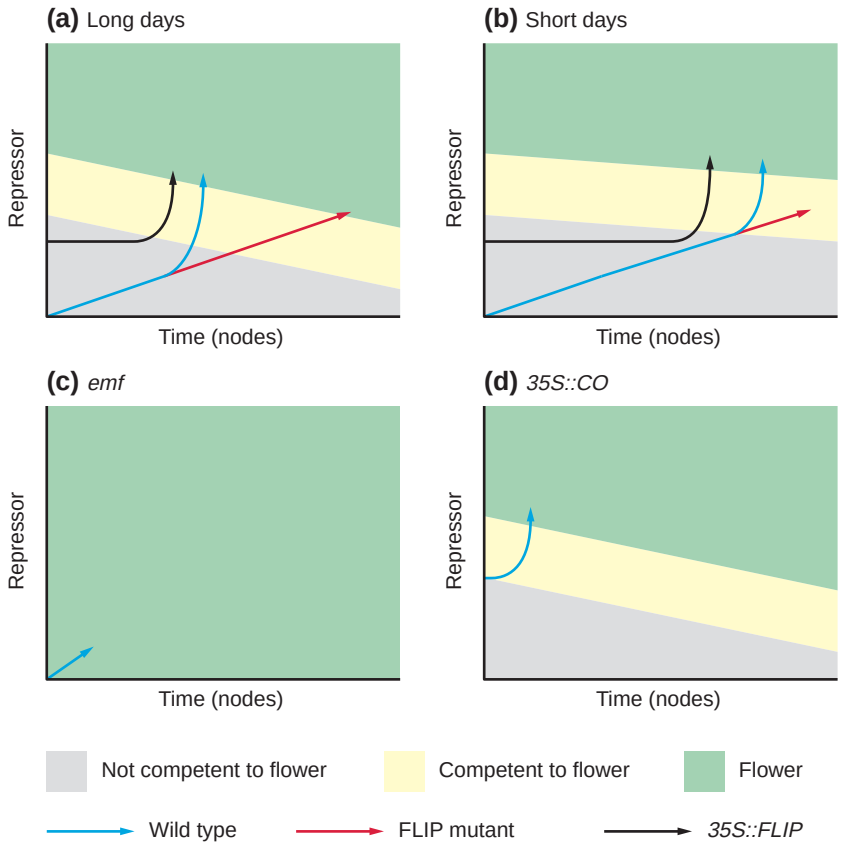

Fig. 5. A model illustrating the relationship between flowering-time genes, floral initiation process (FLIP) genes and competence to flower (modified from previous models[9,48]). (a) In long days, meristems are not initially competent to undergo FLIP (gray area). The competence of a meristem to develop as a flower (yellow area) depends on the activity of a floral repressor. Repressor levels decrease gradually with each node produced, with a concomitant increase in competence. In response, wild-type FLIP gene activity (blue arrow) increases gradually with each node, and is up-regulated before the transition to flowering[48]. At a certain balance between repressor levels and FLIP gene activity (yellow–gray interface), the floral program is activated rapidly and completely to effect a sharp transition to flowering (green). In FLIP mutants, FLIP gene activity (red arrow) cannot be sharply unregulated because cooperative interactions between FLIP genes are lacking. The result is a gradual phase transition from leafy shoots to flowers. In transgenic plants where FLIP genes are constitutively expressed [e.g. fusion of the *35S* promoter to the *LFY* coding sequence (*35S::LFY*)], high levels of FLIP gene activity (black arrow) can promote flower development at nodes that would develop as vegetative shoots in wild type. (b) In short days, the FLIP occurs in a manner similar to long days (a), but the repressor activity declines more slowly, resulting in slower FLIP gene activation and a delay in the time to flowering. (c) With *embryonic flower* (*emf*) mutations, the repressor is eliminated and meristems are immediately competent to undergo the FLIP. (d) In *35S::CO*, FLIP gene expression could be quickly promoted to high levels, resulting in a dramatic acceleration of flowering time. Alternatively, ectopic *CONSTANS* (*CO*) expression could be causing a rapid depletion of repressor, resulting in a situation similar to (c). The *35S::CO* phenotype could, of course, also result from a combination of both possibilities.

the apex (Fig. 1)[38]. Perhaps *TFL1* interferes with the ability of *LFY* and *AP1* to enhance each other's expression. This hypothesis predicts that fusion of the *35S* promoter to the *LFY* coding sequence (*35S::LFY*) and/or *35S::AP1* would be epistatic to *35S::TFL1*. How the plant decides which meristems should express *TFL1* is an important unanswered question, and could depend upon whether a meristem is formed before or after the onset of the reproductive phase.

At least one other *Arabidopsis* gene, *TERMINAL FLOWER 2*, is involved in maintaining the vegetative meristem and could provide further insight into how this is accomplished[39].

Activation of flower initiation

In *Arabidopsis*, flowering is facultative and promoted by long days (Fig. 1). Therefore, the plant must be able to respond to a variety of internal and external cues to activate FLIP under appropriate conditions. Genetic studies of late-flowering mutants have identified different pathways responsible for perceiving and mediating these cues[40]. Mutations at the loci *CONSTANS* (*CO*), *GIGANTEA* (*GI*), *FT*, *FWA*, *FE*, *FD* and *FHA* cause flowering time to be delayed in long days while remaining similar to the wild type in short days, suggesting that these genes mediate a primarily long-day stimulus to flower. Hence they have been placed in a day-length dependent pathway. Mutations at loci *FPA*, *FVE*, *FCA* and *FY* flower later than wild type in long or short days and, therefore, have been proposed to function within a constitutive pathway that promotes flowering regardless of environmental conditions. It is probable that these genes work cooperatively to effect the transition to reproductive growth by activating FLIP genes and/or modulating the capacity of meristems to respond to the FLIP genes. Hence, recent studies have tried to classify these flowering-time genes further in terms of their interactions with *LFY* and/or *AP1* (Refs 41,42).

Among the flowering-time genes, *CO* has been studied extensively. The regulation of *CO* transcription appears to be important for the control of flowering time because *CO* transcripts are more abundant in long days relative to short days and sharply up-regulated upon the transition to flowering[43]. Furthermore, additional copies of *CO* accelerate flowering time under both long and short days. Hence, it has been suggested that *CO* acts as a biological meter that measures exposure to light. Indeed, *CO* is positively regulated by CRYPTOCHROME 2, a blue-ultraviolet A light receptor that is the gene product of *FHA*[44].

The predicted CO protein resembles a zinc-finger transcription factor and, therefore, could be directly regulating the transcription of FLIP genes. Consistent with this hypothesis, *LFY* transcript levels rise rapidly with *CO* induction[45]. Furthermore, *35S::LFY* significantly reduces the late-flowering defect of *co* mutants[42]. *CO* clearly does more than activate *LFY*, however, because plants ectopically expressing *CO* flower significantly earlier than plants ectopically expressing *LFY*[45]. Recessive mutations in *CO* enhance the phenotype of *lfy* mutants, which suggests that *CO* could activate additional FLIP genes (Fig. 4)[43] such as *AP1*. However, upon *CO* induction *AP1* transcript accumulation is significantly delayed relative to *LFY*, indicating that *CO* activates *AP1* indirectly at most[45]. Hence it has been proposed that *CO* also increases the capacity of meristems to respond to *LFY* activity.

Interestingly, *CO* also rapidly induces *TFL1*, a FLIP antagonist (Fig. 4)[45]. This is easily rationalized, because mechanisms to maintain indeterminacy in the apical meristem must be up-regulated concomitant with phase transition to maximize flower production. However, *35S::CO* promotes ectopic expression of *LFY* in the apical meristem and the corresponding development of a terminal flower, even in the presence of *TFL1* expression. Obviously, there is still much to understand about how the antagonistic functions of *LFY* and *TFL1* are spatially and temporally regulated in response to *CO*.

The genes *FWA*, *FT* and *FE* also fall into the day-length-dependent class of flowering promoters, but differ from *CO* in their interactions with FLIP genes. The *fwa lfy* and *ft lfy* double mutants have a severe late-flowering phenotype and do not produce any flower-like structures, which is reminiscent of *lfy ap1* double mutants[41]. In addition, *35S::LFY* cannot correct the flowering time defects resulting from *ft*, *fwa* or *fe* mutations[42]. Taken together, these results indicate that *FWA*, *FT* and *FE* play roles that are partially redundant, yet independent of *LFY* in regulating FLIP. The genes *FT*, *FWA* and *FE* could be involved in the activation of *AP1* and *CAL*. Indeed, the phenotypes of *ft ap1* and *fwa ap1* double mutants resemble *cal ap1* double mutants, suggesting that *FT* and *FWA* are involved in the activation of *CAL*. Recent models have placed *FT*, *FWA* or *FE* in alternate orders with *CO* in the long day pathway based on the phenotypes of different double mutants[41,46]. We have interpreted the extreme vegetative phenotypes of *ft lfy* and *fwa lfy* double mutants, relative to the less extreme *co lfy* phenotype, as implicating that *FT* and *FWA* are closer to the FLIP, and thus downstream of *CO* (Fig. 4), perhaps mediating *CO* in regulating the capacity of the meristem to respond to *LFY*. This would predict that *FT* and *FWA* are necessary for the extreme acceleration of flowering time caused by *35S::CO*.

Another approach, which has been used to identify genes involved in promoting the FLIP, has been to isolate genes that encode factors that bind to the regulatory elements of FLIP gene promoters. One of the genes affecting flowering time, *SPL3*, was identified in this manner[47]. The putative SPL3 protein contains a *SQUAMOSA* promoter binding protein (SBP) DNA-binding domain and can bind to a conserved element of the *AP1* promoter. Moreover, ectopic expression of *SPL3* results in early flowering. However, *AP1* does not appear to be required for the *SPL3*-induced early flowering phenotype, indicating that *SPL3* could be regulating other FLIP genes. Furthermore, *SPL3* does not correspond to any known flowering-time loci which has been defined by mutation, suggesting that there could be considerable functional redundancy among genes regulating FLIP (Fig. 4).

Although they have been placed in the constitutive pathway, *FVE*, *FPA* and *FCA* strongly resemble *CO* in the way in which they regulate FLIP. Double mutant combinations of *fve* and *fpa* with *lfy* and *ap1* all develop flower-like shoots only after producing a much larger number of vegetative shoots than either of the mutant parents[41], indicating that *FVE* and *FPA* are involved in both *LFY* and *AP1* activation. Expression of *35S::LFY* can rescue the flowering time defects resulting from *fca* and *fve* mutations, suggesting that *FCA* and *FVE* primarily activate *LFY* (Ref. 42). *AP1* activation could be direct, or occur through *LFY*, *FWA* or *FT*.

The phenotypic similarity of double mutant combinations of *fve* (*fpa*/*fca*) and *co* with *lfy* and *35S::LFY* indicate that these genes could function in a similar or redundant manner to promote FLIP. Likewise, some of the remaining flowering-time genes might occupy similar roles, but function in separate pathways. Examination of the constitutive and long-day pathway mutants in double mutant combinations with *lfy*, *ap1* and other FLIP genes should further illuminate the precise function of each flowering time gene within the separate pathways. The cloning of more genes and subsequent transgenic studies will also provide answers to important questions. For example, which flowering time genes are necessary for the early-flowering phenotype of *35S::CO*?

Competence to respond to meristem identity genes

If the expression of a single meristem identity gene was sufficient to confer floral identity to a meristem, embryonic expression of this gene should abolish the vegetative phase and produce a flower immediately upon germination. It is interesting therefore, that although ectopic expression of *LFY*, *AP1* or *AG* results in an early flowering phenotype, none is sufficient in itself to immediately transform the SAM into a floral meristem. Rather, it appears that meristems must develop some degree of competence to respond to flower-promoting stimuli[18,45].The nature of this competence factor(s) remains unclear, and could be as simple as simultaneous high-level expression of at least two meristem identity genes. This hypothesis could be tested easily by determining whether or not transgenic plants, in which both *LFY* and *AP1* are expressed embryonically, lack a vegetative phase. Competence, more likely, entails the derepression and activation of a complex suite of genes that affect not only the expression of FLIP genes, but also their capacity to function.

The gradual transition from inflorescence shoots to floral shoots in FLIP mutant inflorescences indicates that activation of the floral program is inductive, and becomes stronger with each node produced. With this in mind, models, in which competence is regulated by a gradual increase of a floral activator or a decline of a floral repressor, can be proposed (Fig. 5)[9,48]. For simplicity we portray the regulator as a floral repressor. The repressor activity, which is present at high levels in the seedling, declines with each node produced until a threshold level is reached at which FLIP gene activity can successfully activate the floral program. The effects of day length and interesting mutations, which affect the time and place of flowering, can also be interpreted using this model (Fig. 5). Strong genetic candidates for floral repressors in *Arabidopsis* are *EMBRYONIC FLOWER 1* and *2* (*EMF1*, *2*) because plants with *emf* mutations bypass the vegetative rosette phase and proceed to terminate in a precocious flower immediately upon germination (Fig. 1)[49]. According to this hypothesis, ectopic expression of *EMF* might result in a plant that never becomes competent to flower and produces only early rosette leaves. Thus, the cloning of *EMF1* and *EMF2* is eagerly anticipated.

Prospects

Significant progress has been made in establishing the genetic basis of floral initiation in *Arabidopsis* and other model systems. Key genes have been identified and much is known about their regulation and early function. One of the pressing goals in the near future will be to identify more of the direct targets of the FLIP genes. In addition, much remains to be learned of the late functions of each FLIP gene in flower development. Although work in *Arabidopsis* has provided the latest paradigm for floral meristem identification, the contributions from studies in other model systems are invaluable. Subtle differences in gene regulation between species illuminate the functional capacities of genes in a variety of circumstances. Further work in parallel systems will help to clarify the function of the meristem identity genes, and continue to distinguish between those aspects of meristem identity that are universal from those that are species specific.

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Mark Pidkowich, Jennifer Klenz and George Haughn* are at the Dept of Botany and Biotechnology Laboratory, University of British Columbia, 6270 University Blvd, Vancouver, BC, Canada V6T 1Z4.

*Author for correspondence (tel +1 604 822 9089; fax +1 604 822 6089; e-mail haughn@unixg.ubc.ca).