Transition from vegetative to reproductive phase
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During the past two years, significant progress has been made towards understanding the molecular basis of how multiple pathways regulating the floral transition are integrated. The transcriptional regulation of several genes, the floral meristem identity gene *LEAFY* and the 'flowering-time' genes *FLOWERING LOCUS T* and *SUPPRESSOR OF OVEREXPRESSION OF* CO *1* (also known as *AGAMOUS-LIKE 20*), is a point at which multiple pathways that promote flowering are integrated.

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Abbreviations

AGL20	*AGAMOUS-LIKE20*
AP1	*APETALA1*
bp	basepair
CAL	*CAULIFLOWER*
CO	*CONSTANS*
FLC	*FLOWERING LOCUS C*
FRI	*FRIGIDA*
FT	*FLOWERING LOCUS T*
FUL	*FRUITFUL*
GA	gibberellin
ga1–3	*gibberellin-requiring 1–3*
Hd-1	*Heading-date 1*
LD	long-day
LFY	*LEAFY*
QTL	quantitative trait locus
SAM	shoot apical meristem
SD	short-day
Se1	*Photoperiod sensitivity1*
soc1	*suppression of overexpression of* CO *1*
SVP	*SHORT VEGETATIVE PHASE*
TFL1	*TERMINAL FLOWER1*

Introduction
During their post-embryonic development, higher plants progress through a series of more or less distinct growth phases, each characterized by the identity of the lateral primordia that are produced by the shoot apical meristem (SAM) [1]. In *Arabidopsis*, the SAM produces leaf primordia that subtend secondary shoot meristems during the vegetative phase, of which juvenile and adult phases are discerned by the shape of the leaves and the distribution of trichomes on the leaf surface [2–5]. During the early reproductive phase, cauline leaf primordia that subtend axillary inflorescence meristems are produced. Determinate floral primordia that will develop into a bractless flower are produced in the late reproductive phase (Figure 1). The transition from vegetative phase to reproductive phase, that is, the floral transition, is the most dramatic phase change in plant development. This transition is regulated by a complex genetic network that monitors the developmental state of the plants

as well as environmental conditions such as light and temperature [6,7]. Genetic analyses of 'flowering-time' mutants have identified about 80 genes placed in multiple genetic pathways that control the floral transition. The photoperiod pathway and the vernalization promotion pathway mediate signals from the environment. The autonomous pathway probably monitors endogenous cues from the developmental state. Genes involved in gibberellin (GA) biosynthesis and GA signal transduction have been suggested to form a distinct promotion pathway ([6,7]; Figure 1).

A number of genetic models have proposed that signals from multiple promotion pathways converge on a central floral repressor [6,8,9], possibly encoded by the *EMBRYONIC FLOWER* genes [8,10]. The inactivation of the floral repressor, in turn, has been suggested to lead to the activation of the floral meristem identity genes, such as *LEAFY* (*LFY*) or *APETALA1* (*AP1*), which specify the floral fate of nascent lateral primordia produced by the SAM. An alternative possibility is that different promotion pathways are directly integrated at the promoters of the floral meristem identity genes, such as *LFY* and a class of 'flowering-time' genes that act in parallel with *LFY*. In this short review, I summarize the latest advances in identifying genes that are involved in integrating the multiple pathways that regulate the floral transition in *Arabidopsis*. Recent reviews describing the regulation of the floral transition by individual pathways are available elsewhere [11–13].
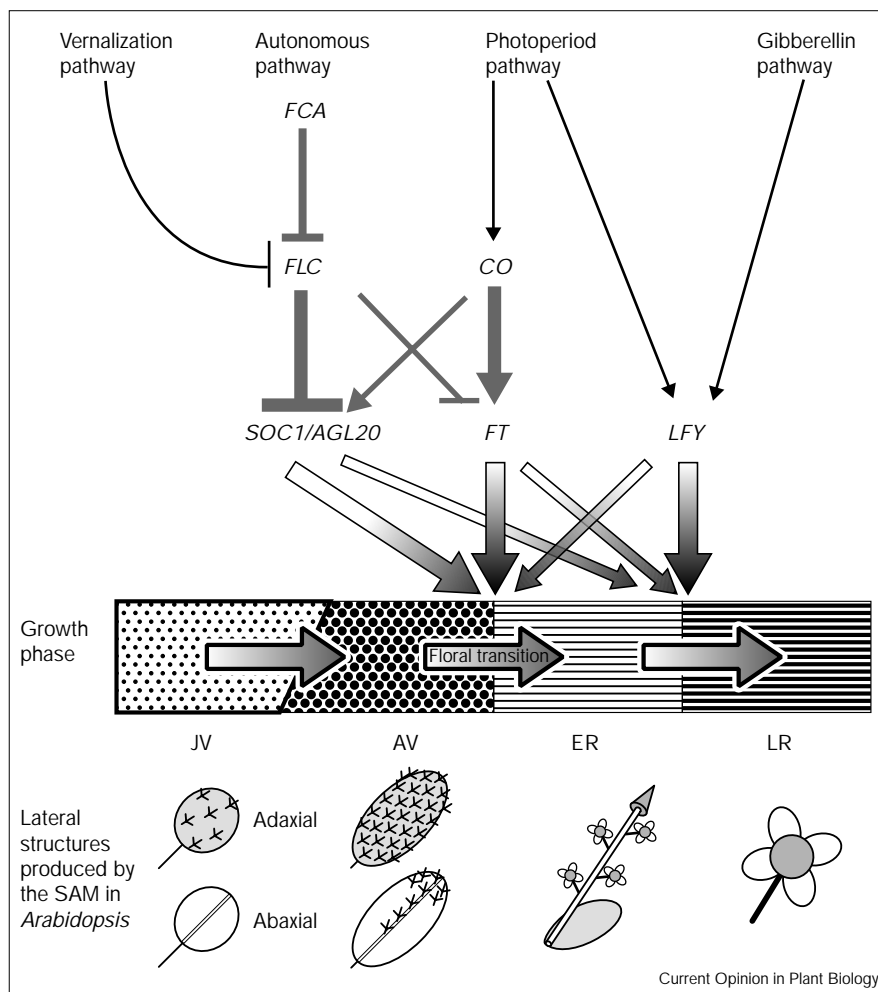
Integration at the promoter of the floral meristem identity gene *LEAFY*
In addition to its central role in the transition from early reproductive to late reproductive phase [14–16], *LFY* plays a role in promoting the floral transition [17,18]. During the vegetative phase, *LFY* is expressed in leaf primordia and is regulated by photoperiod [18]. In long-day (LD) conditions, *Arabidopsis* plants make the floral transition soon after germination, and this is paralleled by rapid upregulation of *LFY* expression. In contrast, the floral transition is delayed by several weeks and *LFY* expression increases only gradually in short-day (SD) conditions.

Levels of *LFY* expression during the floral transition are greatly reduced in mutants that are defective in genes of the photoperiod pathway, such as *CONSTANS* (*CO*) and *GIGANTEA* [19]. Rapid upregulation of *LFY* promoter activity in LD conditions is not observed in these mutants. Activation of *CO*, a key regulator in the photoperiod pathway [20], on the other hand, triggers a rapid increase in *LFY* expression [21] (*LFY* is not, however, a direct target of CO; see below). In SD conditions, the *ga1–3* (*gibberellin-requiring 1–3*) mutation, which severely reduces endogenous GA levels, abolishes the upregulation of *LFY* and prevents the floral transition; the exogenous application of

Figure 1

Progression through growth phases during post-embryonic development in higher plants. In *Arabidopsis*, four phases – the juvenile vegetative phase (JV), the adult vegetative phase (AV), the early reproductive phase (ER), and the late reproductive phase (LR) – are discerned by the lateral structures produced by the SAM [2–5]. (Adaxial and abaxial surfaces of a leaf are shown to illustrate the difference in trichome distribution between the JV and the AV.) The most dramatic change is the transition from the vegetative phase (dotted regions) to the reproductive phase (striped regions), called the floral transition. In *Arabidopsis*, the floral transition is regulated by multiple genetic pathways [6,7]. The vernalization pathway and the autonomous pathway promote the floral transition by reducing the levels of the floral repressor *FLC* [12,13]. The photoperiod pathway mediates signals from LD photoperiods and acts via a transcription factor, CO [11,12]. The gibberellin (GA) pathway has an essential role in SD conditions [7,23]. These four promotion pathways are integrated at the transcriptional regulation of two 'flowering-time' genes, *FT* [27••–29••] and *SOC1/AGL20* [29••,31••], and a floral meristem identity gene, *LFY* [24••]. *FT* and *SOC1/AGL20* are direct targets of CO [29••]. In contrast, *LFY* is not a direct target of CO, but is indirectly regulated by CO [21,29••] (not shown). The floral repressor *FLC* is likely to negatively regulate *FT* and *SOC1/AGL20* [29••,31••]. The balance between CO and FLC activity may determine the levels of *FT* and *SOC1/AGL20* expression. However, the relative regulatory contributions of CO and FLC in the control of *FT* and *SOC1/AGL20* expression may differ (as indicated by thin or thick arrows and T-bars) [29••,31••]. Regulation of *LFY* by the photoperiod and GA pathways is mediated by different *cis* elements on the *LFY* promoter [24••]. *LFY* is also regulated by the autonomous pathway [19,25,26•] (not shown). FT, SOC1/AGL20, and LFY differ in their relative contributions to the floral transition and to the ER→LR transition. *FT* and *SOC1/AGL20* have a major role in the former, whereas *LFY* has a major role in the latter [18,24••,27••–29••,31••]. A positive role of *FT* in the ER→LR transition is supported by the observation that *ft-1* mutation increased the number of cauline leaves in SD conditions [38]. *SOC1/AGL20*, *FT*, and *LFY* may also have roles in the JV→AV transition (not shown). However, it has been reported that overexpression of *LFY* had no effect on the JV→AV transition in both LD and SD conditions [17,53], and that *ft-1* mutation had little effect on JV→AV transition in LD conditions [4].

GA restores both [22,23]. In summary, *LFY* expression during the vegetative phase is regulated by both photoperiod and GA. Consistent with this, *LFY* mRNA levels remain low and the floral transition is abolished in *co* ; *ga1* double mutants, in which both the photoperiod pathway and the GA pathway are blocked [20,24••].

A distal and a proximal regulatory region of the *LFY* promoter were identified by deletion analysis [24••]. A sequence containing both of these regulatory regions, named GOF9, can mimic the full-length (2290-base-pair [bp]) promoter. Both the full-length promoter and the GOF9 promoter exhibited a rapid increase in transcriptional activity in LD conditions, and a gradual increase in SD conditions, which was accelerated by GA treatment.

Comparison of the sequence of the *Arabidopsis LFY* promoter with that of a cottonwood homolog revealed a conserved 8-bp motif (i.e. CAACTGTC) in the proximal region, which conforms to the consensus for the binding site of a MYB transcription factor of the plant R2R3 family (a family of MYB proteins that contain two repeats, R2 and R3). A GOF9 promoter with a mutated 8-bp motif (i.e. GOF9m) had no transcriptional activity and did not respond to GA in SD conditions. However, the rapid upregulation of GOF9m promoter activity in LD conditions was not affected. Combination of the GOF9m promoter with a *LFY* cDNA created a *LFY* allele that is active only in LD conditions. Transgenic *lfy-12* plants carrying this GOF9m::*LFY* construct showed LD-specific complementation of the mutant phenotype, whereas the

phenotype of transgenic *lfy-12* carrying the GOF9::*LFY* construct was rescued irrespective of photoperiod. These results clearly indicate that the photoperiod pathway, which mediates LD signals, and the GA pathway act on different *cis* elements in the *LFY* promoter. It will be interesting to know whether the 8-bp motif is also involved in the small but significant response to GA observed in LD conditions [22]. So far, the *cis* elements involved in the rapid upregulation of *LFY* expression in LD conditions have not been identified. Identification of such *cis* elements and the protein factors binding to them will further deepen our understanding of the molecular mechanism by which pathways are integrated at the *LFY* promoter.

Do signals from different pathways have interactive effects on the *LFY* promoter? Such interaction is not unlikely as it has already been reported that *ga1-3* delays the rapid upregulation of *LFY*::*GUS* upon transfer of plants from SD to LD conditions, suggesting a role for GA in the regulation of *LFY* by photoperiod [22]. Expression of *LFY* is also promoted by genes of the autonomous pathway, such as *FCA* and *LUMINIDEPENDENS* [19,25,26•]. Three promotion pathways therefore converge at the regulation of *LFY* expression (Figure 1).

Integration at the regulation of two 'flowering-time' genes, *FLOWERING LOCUS T* and *SUPPRESSOR OF OVEREXPRESSION OF* CO *1*

Overexpression of *LFY* alone cannot cause plants to make the floral transition without passing through the vegetative phase or in a photoperiod-independent manner, suggesting that some factors that are regulated by both the age of the plant and photoperiod determine the plant's competence to respond to *LFY* activity [17]. *FLOWERING LOCUS T* (*FT*) is a good candidate for such a factor, as *ft-1* mutation does not affect *LFY* expression but strongly suppresses the phenotype of plants overexpressing *LFY* [19]. Recently, *FT* was cloned and found to be a homolog of *TERMINAL FLOWER1* (*TFL1*) [27••,28••] (see below for relation with *TFL1*). It was demonstrated that simultaneous overexpression of *LFY* and *FT* almost eliminates the vegetative phase, such that the whole shoot is converted to a single terminal flower with one or two bracts [27••,28••].

Expression of *FT* is regulated by the photoperiod pathway via *CO* [27••,28••]. *FT* expression is rapidly upregulated in LD conditions but *co* mutations reduce and delay this upregulation. Conversely, induction of *CO* activity results in the immediate upregulation of *FT* expression [27••]. A recent study demonstrated that the induction of *FT* expression by CO protein does not require protein synthesis, suggesting that *FT* is a direct target of CO [29••]. The epistatic relations among the relevant transgenic and mutant plants are consistent with this explanation. Overexpression of *FT* caused precocious flowering independently of photoperiod and *CO* [27••,28••]. In contrast, *FT* function is required for overexpressed *CO* to cause precocious flowering [30••]. *FT* was still expressed, however,

in SD photoperiods or in *co* mutant backgrounds, suggesting that the photoperiod pathway is not the only pathway responsible for induction of *FT* expression [27••,28••]. As the *fca-1* mutation reduced the level of *FT* expression ([29••]; JH Ahn, D Weigel cited in [31••]), the autonomous pathway may play a role in the promotion of *FT* expression, possibly by reducing the levels of the floral repressor *FLOWERING LOCUS C* (*FLC*) [32•–34•], which may repress *FT* expression [29••].

A MADS-box gene, *AGAMOUS-LIKE 20* (*AGL20*), was among the other genes identified as direct targets of CO [29••]. A loss-of-function mutant of *AGL20* (named *suppressor of overexpression of* CO *1* [*soc1*]) has been identified as a suppressor of the early-flowering phenotype of transgenic plants that overexpress *CO* [30••]. This evidence supports the notion that *CO* requires the *AGL20* function (*AGL20* was renamed *SOC1* in [29••], and is referred to *SOC1*/*AGL20* hereafter) to promote the floral transition. Importantly, *SOC1*/*AGL20* was also identified by the activation T-DNA tagging approach as a suppressor of an extreme late-flowering phenotype conferred by the functional alleles of *FRIGIDA* (*FRI*) and *FLC* [31••]. *FRI* [35,36,37•] and *FLC* are key genes of the autonomous and vernalization pathways [12,13]. By detailed expression analysis, it has been clearly shown that *SOC1*/*AGL20* is regulated not only by the photoperiod pathway via *CO*, but also by the autonomous pathway and the vernalization pathway, which are integrated at the regulation of *FLC* [31••].

LFY, *FT* and *SOC1*/*AGL20* are genes that integrate signals from the multiple genetic pathways (Figure 1). It has been shown that *FT* and *LFY* are, for the most part, regulated independently of each other and act in parallel pathways [18,28••]. *SOC1*/*AGL20*, on the other hand, seems to interact with *LFY* and *FT*. The formation of solitary flowers in the axil of cauline leaves of plants that overexpress *SOC1*/*AGL20* when *FRI*/*FLC* activity is removed by mutation suggests that *SOC1*/*AGL20* can activate *LFY* and *AP1*, once *FLC* activity is removed. This means that *LFY* acts, at least in part, downstream of *SOC1*/*AGL20* [31••]. In contrast, *SOC1*/*AGL20* expression is reduced to a similar level in *ft* and *co* mutants, suggesting a role for *FT* in *SOC1*/*AGL20* regulation [31••]. These results indicate that there is substantial cross-regulation among genes that integrate multiple promotion pathways. Interestingly, *soc1*/*agl20* mutants are late flowering but, in sharp contrast to the photoperiod-insensitive *co* and *ft* mutants [38], are sensitive to photoperiod [30••,31••]. This observation indicates that, although the photoperiod pathway (via CO) and the autonomous and vernalization pathways (which converge at *FLC*) are integrated at the regulation of *FT* and *SOC1*/*AGL20*, these pathways differ in their relative contributions to the regulation of these two genes [29••] (Figure 1).

Redundancy and antagonism

It seems that redundant or antagonistic roles of homologous genes are an emerging theme in the current genetic

regulatory model of the floral transition. The floral promoter *SOC1/AGL20*, which integrates three promotion pathways, and the floral repressor *FLC*, on which the autonomous and vernalization pathways converge, are closely related genes within the MADS-box gene family (which may include 80 members [39], see a phylogenetic tree in [31••]). That *FLC* negatively regulates *SOC1/AGL20* [29••,31••] explains, in part, how these two genes play opposite roles. Another pair of homologous genes with antagonistic roles are *FT* and *TFL1* [27••,28••,40], although they are not the closest homologs among a small gene family of six members in *Arabidopsis* (see a phylogenetic tree in [27••]). The mechanisms underlying the antagonistic roles of *FT* and *TFL1* are yet to be elucidated. A growing list of homologous genes that are known to have redundant or antagonistic roles in the regulation of the floral transition includes further members of the MADS-box gene family. *FRUITFUL* (*FUL*) plays a role in promoting the floral transition, as well as a role in floral-fate specification redundantly with *AP1* and *CAULIFLOWER* (*CAL*) [41••]. It has been suggested that two MADS-box genes, *SOC1/AGL20* and *AGL24*, play a redundant role with *FUL* in promoting the floral transition [41••]. As discussed above, the elucidated role of *SOC1/AGL20* is consistent with this hypothesis [29••,31••], although redundancy with *FUL* remains to be tested. The role of *AGL24* in promoting the floral transition is still to be investigated. Interestingly, however, a MADS-box gene *SHORT VEGETATIVE PHASE* (*SVP*), which belongs to the same group as *AGL24*, has been identified as a dosage-dependent floral repressor like *FLC* [42•]. It should also be noted that the recently identified tomato *JOINTLESS* gene [43], which prevents inflorescence meristems from reverting to the vegetative state [44], is the closest homolog of *SVP* ('f14m13.6' in a phylogenetic tree in [43] is, indeed, SVP). *AGL24* and *SVP* may represent another pair of related genes with opposite roles in the regulation of the floral transition.

Conclusions and perspectives

In our current view, the multiple genetic pathways that promote the floral transition are directly integrated at the transcriptional regulation of the floral meristem identity gene *LFY* and the 'flowering-time' genes, *FT* and *SOC1/AGL20* (Figure 1). There may be extensive cross-talk among the pathways and integrating genes. An interesting recent finding is that the length of the circadian period is affected in null mutants of the floral repressor *FLC* [45•]. This may indicate the presence of unexpected cross-talk between pathways.

Whether the genetic regulatory model of the floral transition proposed for *Arabidopsis* is applicable to other plants, especially to SD plants, is obviously an important question to be addressed in the next few years. Until recently, the only homologs of the *Arabidopsis* 'flowering-time' genes to have been cloned and analyzed in detail were a *CO* homolog in *Brassica* [46] and a *SOC1/AGL20* homolog in *Sinapis* [47]. Both *Brassica* and *Sinapis* are LD plants that are closely related to *Arabidopsis*. Recently, a major quantitative trait locus (QTL) that is responsible for the photoperiod

sensitivity of rice (an SD plant) was cloned [48••]. This QTL, *Heading-date 1* (*Hd-1*), also known as *Photoperiod sensitivity1* (*Se1*), encodes a *CO* homolog. It was suggested that *Hd1/Se1* promotes the floral transition in SD conditions and inhibits it in LD conditions. This is in contrast to *Arabidopsis CO*, which has low levels of expression in non-inductive SD conditions but does not seem to have an inhibitory role [20]. Consistent with its role in both inductive SD and non-inductive LD conditions, the expression of *Hd1/Se1* was not regulated by photoperiod. Recent success in the molecular identification of *Hd1/Se1* and *Se5* (another photoperiod-sensitivity locus, which turned out to be a *HY1* homolog involved in phytochrome chromophore biosynthesis [49•]) and the ongoing rice genome project make rice the most promising system in which to study the genetic control of flowering in SD plants. The availability of rice homologs of *FT* [27••] and *LFY* [50], as well as wealth of photoperiod sensitivity QTLs from rice [51] (some of which have interesting genetic interactions with *Hd1/Se1* [52]), will enable us to compare the mechanisms that regulate the floral transition in rice (as a representative SD plant) with those of *Arabidopsis* (a representative LD plant).

Update

Recently, the molecular nature of dominant *fwa* mutations has been elucidated [54••]. Dominant *fwa* mutations are interesting in that they share with *ft* a similar phenotype [38], similar genetic interactions with *lfy* and *ap1* [55], and similar suppressive effects on the *CO*-overexpression phenotype [30••]. *FWA* (in dominant *fwa* mutants) has been suggested to interfere with the action of *FT* [27••,28••,30••]. Soppe *et al.* [54••] demonstrated that hypomethylation of two direct repeats in the 5' region of the *FWA* gene, which encodes a homeodomain protein with similarity to ANTHOCYANINLESS2, caused ectopic expression of the gene in dominant epigenetic *fwa* mutants. In wild-type plants, *FWA* expression was detected only in siliques (from shortly after fertilization until seed maturation) and germinating seeds, but not in seedlings of any age. In accordance with this expression, loss-of-function mutants of *FWA* did not show any effects on the floral transition. These findings make interpretation of the role of *FWA* in the floral transition difficult [54••]. The FWA product may prevent germinating plants from precocious floral transition and may promote establishment of the vegetative phase.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

 • of special interest
 •• of outstanding interest

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24. Blázquez MA, Weigel D: **Integration of floral inductive signals in**
•   ***Arabidopsis*.** *Nature* 2000, **404**:889-892.
This paper provides the first clear evidence of where and how the multiple pathways that promote flowering are integrated. The authors report that GA activates *LFY* expression through *cis* elements in the promoter that are different from those involved in the daylength response. This suggests that the *LFY* promoter integrates signals from the photoperiod pathway and GA pathway through different regulatory elements.

25. Page T, MacKnight R, Yang C-H, Dean C: **Genetic interactions of the *Arabidopsis* flowering time gene *FCA*, with genes regulating floral initiation.** *Plant J* 1999, **17**:231-239.

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•   **flowering-time gene *LUMINIDEPENDENS* is expressed primarily in regions of cell proliferation and encodes a nuclear protein that regulates *LEAFY* expression.** *Plant J* 1999, **18**:195-203.
This paper describes an analysis of the expression pattern and subcellular localization of *LUMINIDEPENDENS*. This gene, which encodes a nuclear protein with a homeodomain-like region, is expressed in the meristematic regions, which include cells expressing *LFY*. Mutation in *LUMINIDEPENDENS* alone does not seem to affect *LFY* expression, at least in reproductive SAMs, after the floral transition. In the *ap1 ; cal* double mutant background, however, loss of *LUMINIDEPENDENS* function eliminates the residual expression of *LFY* in the reproductive SAM, suggesting that *LUMINIDEPENDENS* has a redundant role with *AP1* and *CAL* in promoting *LFY* expression.

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•• **genes with antagonistic roles in mediating flowering signals.** *Science* 1999, **286**:1960-1962.
This paper and [28••] report the molecular characterization of the flowering-time gene *FT*. Expression analysis and epistasis studies with mutants and transgenic plants show that *FT* acts, in part, downstream of *CO* and with *LFY* to promote the floral transition. *FT* determines the competence of the plant to respond to *LFY* in specifying the floral fate of meristem. This is demonstrated by transgenic plants that overexpress both *LFY* and *FT* in which the whole shoot is converted to a single flower with one or two bracts. *FT* belongs to a small gene family in *Arabidopsis* that includes *TFL1*. On the basis of mutant and transgenic phenotypes and of genetic interactions, the authors propose that the two homologous genes *FT* and *TFL1* have antagonistic roles in the phase transition: *FT* promotes and *TFL1* inhibits the transition.

28. Kardailsky I, Shukla VK, Ahn JH, Dagenais N, Christensen SK,
•• Nguyen JT, Chory J, Harrison MJ, Weigel D: **Activation tagging of the floral inducer *FT*.** *Science* 1999, **286**:1962-1965.
The authors of this paper and [31••] demonstrate the power of the activation T-DNA tagging approach in identifying key regulators of the floral transition. The constitutive overexpression of *FT* is shown to result in photoperiod- and *CO*-independent precocious flowering (as it is in [27••]), thereby confirming that *FT* is partially downstream of *CO*. Expression and transgenic studies confirm that *FT* and *LFY* act in parallel pathways and that *FT* determines the competence of the plants to respond to *LFY*. *FT* and its homolog *TFL1* encode proteins with homology to mammalian proteins that have been reported to be precursors of a hippocampal neuropeptide and, more recently, to be membrane-bound Raf1 kinase inhibitor proteins.

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Direct regulatory target genes of the CO protein are identified using an inducible system, which is based on a protein fusion of CO to the ligand binding-domain of the glucocorticoid receptor [21], in combination with a translational inhibitor (i.e. cycloheximide). The authors show that *FT* and *AGL20* are among four direct target genes of CO and that *AGL20* is identical to *SOC1* [30••]. This is in agreement with the finding that *FT* and *SOC1* (*AGL20* is renamed *SOC1* in this paper) are required for *CO* to promote the floral transition [30••]. Interestingly, induction of *LFY* requires protein synthesis, suggesting that *LFY* is not an immediate early target of CO. Levels of *FT* and *SOC1/AGL20* expression are reduced in *fca-1* mutants, in which expression of the floral repressor *FLC* is at a higher level than in the wild type [32•–34•]. The authors propose that a balance between *CO* and *FLC* activity may determine the levels of *FT* and *SOC1/AGL20* expression. Other direct targets of CO are genes involved in proline and ethylene biosynthesis, suggesting that the pathways that promote the floral transition and bolting diverge downstream of *CO*.

30. Onouchi H, Igeño I, Périlleux C, Graves K, Coupland G: **Mutagenesis**
•• **of plants overexpressing *CONSTANS* demonstrates novel interactions among *Arabidopsis* flowering-time genes.** *Plant Cell* 2000, **12**:885-900.
The work described in this paper beautifully corroborates that described in [29••]. The authors identify four mutants that suppress the precocious-flowering phenotype of transgenic plants that overexpress *CO*. Two of these mutants are alleles of *ft* and a third has a mutation that defines a novel locus named *SOC1*. The fourth mutant is an allele of *fwa*. The characterization of *soc1* and the effects of *fwa* alleles on the phenotype of transgenic plants that overexpress *CO* are described in detail.

31. Lee H, Suh S-S, Park E, Cho E, Ahn JH, Kim S-G, Lee JS, Kwon YM,
•• Lee I: **The AGAMOUS-LIKE 20 MADS domain protein integrates floral inductive pathways in** *Arabidopsis*. *Genes Dev* 2000, **14**:2366-2376.
As in [28••], the power of the activation T-DNA tagging approach is clearly demonstrated. The authors identify *AGL20* (renamed *SOC1* in [29••]) as a dominant suppressor of an extreme late-flowering phenotype conferred by functional alleles of *FRI* and *FLC*. They clearly demonstrate that *SOC1/AGL20* is regulated not only by the autonomous and vernalization pathways but also by the photoperiod pathway. Further, they suggest that *SOC1/AGL20* is an important integrator of these three pathways.

32. Sheldon CC, Burn JE, Perez PP, Metzger J, Edwards JA, Peacock WJ,
• Dennis ES: **The *FLF* MADS box gene: a repressor of flowering in** *Arabidopsis* **regulated by vernalization and methylation.** *Plant Cell* 1999, **11**:445-458.
The authors report the identification of a novel MADS-box gene, named *FLOWERING LOCUS F*, which acts as a repressor of floral transition. *FLOWERING LOCUS F* is regulated by genes in the autonomous pathway, by vernalization, and by demethylation. The authors propose that *FLOWERING LOCUS F* may regulate the action of GA at the shoot apical meristem, providing a further mechanism by which pathways are integrated. The cloning of *FLC* [33•] later revealed that *FLOWERING LOCUS F* is identical to *FLC*.

33. Michaels SD, Amasino RM: ***FLOWERING LOCUS C* encodes a novel**
• **MADS-box domain protein that acts as a repressor of flowering.** *Plant Cell* 1999, **11**:949-956.
The *FLC* gene is cloned and turns out to be identical with *FLOWERING LOCUS F* [32•]. The authors propose that the levels of *FLC* activity act through a rheostat-like mechanism to control the floral transition.

34. Sheldon CC, Rouse DT, Finnegan J, Peacock WJ, Dennis ES: **The**
• **molecular basis of vernalization: the central role of *FLOWERING LOCUS C* (*FLC*).** *Proc Natl Acad Sci USA* 2000, **97**:3753-3758.
The central role of *FLC* in vernalization is demonstrated in this paper. Interestingly, evidence suggesting a role for the *FD* gene, which has been placed in the photoperiod pathway [6,7], in the regulation of *FLC* expression is also reported.

35. Lee I, Bleeker A, Amasino RM: **Analysis of naturally occurring late flowering in** *Arabidopsis thaliana*. *Mol Gen Genet* 1993, **237**:171-176.

36. Clarke JH, Dean C: **Mapping *FRI*, a locus controlling flowering time and vernalization response in** *Arabidopsis thaliana*. *Mol Gen Genet* 1994, **222**:81-89.

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• **Molecular analysis of *FRIGIDA*, a major determinant of natural variation in** *Arabidopsis* **flowering time.** *Science* 2000, **290**:344-347.
The authors of this paper report the cloning, using a map-based approach, of the *FRI* locus, a major determinant of natural variation in vernalization requirement [35,36]. The detailed molecular analysis of naturally-occurring alleles of *FRI* is described. *FRI* encodes a novel protein with two predicted coiled-coil domains. *FRI* is a positive regulator of *FLC* expression [32•–34•].

38. Koornneef M, Hanhart CJ, van der Veen JH: **A genetic and physiological analysis of late flowering mutants in** *Arabidopsis thaliana*. *Mol Gen Genet* 1991, **229**:57-66.

39. Riechmann JL, Ratcliffe OJ: **A genomic perspective on plant transcription factors.** *Curr Opin Plant Biol* 2000, **3**:423-434.

40. Ratcliffe OJ, Amaya I, Vincent CA, Rothstein S, Carpenter R, Coen ES, Bradley DJ: **A common mechanism controls the life cycle and architecture of plants.** *Development* 1998, **125**:1609-1615.

41. Ferrándiz C, Gu Q, Martienssen R, Yanofsky MF: **Redundant**
•• **regulation of meristem identity and plant architecture by *FRUITFULL*, *APETALA1*, and *CAULIFLOWER*.** *Development* 2000, **127**:725-734.
A detailed analysis of the genetic interaction among three closely related MADS-box genes (*FUL*, *AP1* and *CAL*) and other meristem identity genes (*LFY* and *TFL1*) is presented. In addition to its role in carpel and fruit development, *FUL* acts as a regulator of the floral transition and regulates *LFY* and *TFL1* expression redundantly with *AP1* and *CAL*.

42. Hartmann U, Höhmann S, Nettesheim K, Wisman E, Saedler H,
• Huijser P: **Molecular cloning of *SVP*: a negative regulator of the floral transition in** *Arabidopsis*. *Plant J* 2000, **21**:351-360.
A novel MADS-box gene, *SVP*, that is involved in the regulation of the floral transition is identified. The semi-dominant early-flowering phenotype of the

loss-of-function mutants of *SVP* leads the authors suggest that *SVP*, like *FLC*, is a dosage-dependent repressor of the floral transition.

43. Mao L, Begum D, Chuang H-W, Budiman MA, Szymkowiak EJ, Irish EE, Wing RA: ***JOINTLESS* is a MADS-box gene controlling tomato flower abscission zone development.** *Nature* 2000, **406**:910-913.

44. Szymkowiak EJ, Irish EE: **Interactions between *jointless* and wild-type tomato tissues during development of the pedicel abscission zone and the inflorescence meristem.** *Plant Cell* 1999, **11**:159-175.

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• Koornneef M, Millar AJ: **Natural allelic variation identifies new genes in the** *Arabidopsis* **circadian system.** *Plant J* 1999, **20**:67-77.
The authors of this paper report a QTL analysis of the period length of the circadian clock. Interestingly, they show that three null alleles of *flc* shorten the period length and suggest that allelic difference at *FLC* can account for one of the QTLs.

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•• Baba T, Yamamoto K, Umehara Y, Nagamura Y, Sasaki T: ***Hd1*, a major photoperiod sensitivity QTL in rice, is closely related to the** *Arabidopsis* **flowering time gene** *CONSTANS*. *Plant Cell* 2000, **12**:in press.
The authors of this paper report the map-based cloning of a major photoperiod sensitivity QTL, *Hd1*, in rice. *Hd1* is a homolog of *CO*, which is a key regulator in the photoperiod pathway of *Arabidopsis*. Interestingly, the expression of *Hd1* does not seem to be regulated by photoperiod, and *Hd1* seems to be active in both inductive SD and non-inductive LD photoperiods.

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• **Phytochromes confer the photoperiodic control of flowering in rice (a short-day plant).** *Plant J* 2000, **22**:391-399.
A photoperiod sensitivity locus *Se5* is shown to encode a heme oxygenase that is involved in phytochrome chromophore biosynthesis. Mutations in the *Se5* locus completely abolish the photoperiodic response of floral transition, suggesting an essential role of phytochrome in this response in rice.

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The molecular nature of dominant *fwa* mutations is elucidated. The hypomethylation of two direct repeats in the 5′ region of the *FWA* gene, which encodes a homeodomain protein with similarity to ANTHOCYANIN-LESS2, caused ectopic expression of the gene in dominant epigenetic *fwa* mutants. In wild-type plants, *FWA* expression was detected only in siliques (from shortly after fertilization until seed maturation) and germinating seeds, but not in seedlings of any age. In accordance with this expression, loss-of-function mutants of *FWA* did not show any effects on the floral transition. The FWA product may prevent germinating plants from precocious floral transition and may promote establishment of the vegetative phase.

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