

Chapter

The Molecular Orchestrators: Transcriptional Regulation in Flowering Time and Development

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Abstract

The transition from vegetative to reproductive growth is a pivotal developmental switch in flowering plants, orchestrated by complex transcriptional regulatory networks. This chapter comprehensively reviews the transcriptional regulatory networks that govern floral induction, the core transcription factor (TF) families that govern floral induction, including MADS-box proteins, the key floral integrators FLOWERING LOCUS T (FT) and SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1 (SOC1), and the major repressor FLOWERING LOCUS C (FLC). These TFs integrate endogenous and environmental cues such as photoperiod, vernalization, ambient temperature, gibberellin (GA), and age. MADS-box proteins assemble into higher-order complexes that specify floral organ identity through DNA looping, while floral integrators converge at the shoot apex to activate meristem identity genes (AP1, LFY, FUL). Vernalization triggers Polycomb-mediated epigenetic silencing of FLC, a process requiring VEL protein condensates and operating via a digital, cell-autonomous switching mechanism. In *perennial* plants, duplicated FT genes have acquired divergent roles in flowering and dormancy regulation. The miR156-SPL-miR172 cascade mediates age-dependent flowering: high miR156 levels in young plants repress SPL transcription factors; as miR156 declines with age, accumulated SPLs activate miR172, which downregulates AP2-like repressors (TOE1, TOE2, SMZ, SNZ), thereby de-repressing FT. Post-translational modifications dynamically modulate TF activity, adding another layer of control. Gene duplication, neofunctionalization, and the recruitment of conserved modules into alternative developmental pathways illustrate how evolutionary tinkering generates flowering-time diversity across angiosperms. The chapter concludes by examining the ecological dimensions of these mechanisms, highlighting their significance for plant adaptation and crop improvement. Collectively, this review underscores how the integration of multiple signals through these transcriptional networks ensures reproductive success and offers targets for crop breeding.

Keywords: flowering time, transcription factors, MADS-box, photoperiod pathway, vernalization, epigenetic regulation, regulatory networks, miRNA, chromatin remodeling

1. Introduction

Flowering, a critical developmental phase in angiosperms, marking the transition from vegetative to reproductive growth [1]. This process directly influences offspring reproductive capacity, population viability, and ecological adaptability [2, 3]. In crop and horticulture species, flowering time significantly impacts yield and quality, rendering the regulation of adaptive flowering an important agronomic trait of considerable interest to breeders [4–7].

Flowering time is modulated by a combination of endogenous and exogenous factors, including temperature, light, humidity, stress, carbohydrate levels, mineral nutrition status, plant growth regulators, and genetic factors [8]. Natural selection has refined the underlying regulatory mechanisms, resulting in sophisticated molecular circuits that align flowering with favorable conditions [9]. Premature flowering exposes reproductive organs to environmental stresses such as frost or drought, while delayed flowering may prevent seed maturation before seasonal changes [10–12].

Flowering is regulated by a complex network of endogenous and environmental signaling pathways that collectively ensure reproductive success. Autonomously promotes flowering through constitutive repression of floral inhibitors, particularly FLOWERING LOCUS C (FLC) [13]. Gibberellin (GA) pathway mediates hormonal signals by destabilizing DELLA repressor proteins [14]. Moreover, age pathway operates through the gradual decline of microRNA156 (miR156) and accumulation of SQUAMOSA PROMOTER BINDING PROTEIN-LIKE (SPL) transcription factors (TFs) [15].

Environmental pathways enable adaptive responses to external conditions. The Photoperiod Pathway senses day length through circadian-regulated accumulation of CONSTANS (CO) protein, which activates transcription of FLOWERING LOCUS T (FT) encoding the mobile florigen signal [16]. The Vernalization Pathway mediates response to prolonged winter cold by epigenetically silencing FLC via Polycomb-group proteins [17, 18]. The Ambient Temperature Pathway modulates flowering through thermosensitive mechanisms including stabilization of PHYTOCHROME INTERACTING FACTORS (PIFs) and regulation of histone variant H2A.Z occupancy [19, 20].

Crucially, these pathways do not function independently but form an extensively interconnected regulatory network. Vernalization, for instance, removes FLC-mediated repression, thereby enabling photoperiodic induction. Gibberellin signaling can promote flowering under non-inductive photoperiods when the CO-FT module is inactive. The age pathway, operating through miR156 and SPL transcription factors, modulates the sensitivity of meristems to these various inductive signals.

Post-translational modifications (PTMs), including phosphorylation, ubiquitination, SUMOylation, and acetylation, are important for regulating protein stability and function. Recent biochemical evidence suggests that the functioning of FLC is controlled at both the transcriptional and post-translational levels, indicating that PTMs play important roles in regulating FLC function. This chapter elaborates how these transcriptional and post-translational mechanisms together orchestrate flowering time control. It begins with the core TF families involved, including MADS-box proteins, flowering integrators, and pathway-specific repressors. Subsequent sections address the regulatory networks formed through pathway cross-talk and signal integration, and explore how PTMs dynamically modulate TF activity, stability, and interactions in response to endogenous and environmental cues.

2. Core transcription factor families in flowering

The transition from vegetative to reproductive development depends on a sophisticated regulatory network in which TFs integrate endogenous and environmental signals. These proteins coordinate the expression of target genes in specific spatial and temporal patterns, directing the shoot apical meristem to adopt a floral meristem identity. This section reviews the major transcription factor families that regulate floral induction and meristem identity, focusing on their molecular functions, regulatory interactions, and roles within the broader flowering network. The major transcription factor families involved in flowering control are summarized in **Table 1**.

Family	Gene	Function	Upstream signals	Downstream targets	Key references
MADS-box	AP1	Floral meristem identity, sepal/petal development	FT-FD complex, LFY	AP3, PI, AG	[21–23]
MADS-box	FLC	Floral repressor, vernalization integrator	Vernalization, autonomous pathway	FT, SOC1	[24–28]
MADS-box	SOC1	Floral integrator, multiple pathway convergence	CO (photoperiod), GA, relief of FLC repression	LFY, AP1	[29–33]
MADS-box	SEP1-4	Floral organ identity co-factor (E-function)	Developmental signals	Class A, B, C MADS-box proteins	[34–36]
PEBP	FT	Mobile florigen, floral integrator	CO (photoperiod), PIF4 (temperature)	AP1, SOC1	[37–43]
B-box zinc finger	CO	Photoperiodic switch, day-length measurement	Circadian clock (GI, FKF1, CDFs), photoreceptors	FT, TSF	[44–51]
bZIP	FD	Floral activation at shoot apex	FT (via 14-3-3)	AP1	[41–43]
bHLH	PIF4	Thermosensor, temperature-responsive activator	Elevated temperature	FT	[52]
SBP-box	SPLs	Age pathway, developmental timing	miR156 (declining with age)	miR172, SOC1, LFY	[15, 53]
AP2/ERF	TEM1/2	Floral repressor, auxin integration	Auxin signaling	FT	[54, 55]
PHD finger	VIN3	Vernalization initiator, PRC2 recruitment	Prolonged cold	FLC (chromatin)	[27, 56, 57]
VEL family	VRN5, VIN3	Polycomb silencing maintenance, condensate formation	Cold exposure	FLC (chromatin)	[57, 58]
B3 domain	VAL1	Silencing nucleation at FLC	Vernalization signals	FLC (chromatin)	[59]

Table 1.
Core transcription factor families in flowering time regulation.

2.1 MADS-box TFs

2.1.1 APETALA1, FRUITFULL, and SEPALLATA clades

The MADS-box family is among the most thoroughly studied transcription factor families in plants. These proteins share a conserved DNA-binding domain originally identified in MCM1, AGAMOUS, DEFICIENS, and SRF, and function as key regulators of diverse developmental processes. Their roles in floral organ specification and meristem identity have received particular attention (**Table 1**) [60, 61].

Among the MADS-box proteins, the APETALA1 (AP1)/FRUITFULL (FUL) and SEPALLATA (SEP) clades play fundamental roles in specifying floral meristem and organ identity. AP1 functions as both a floral meristem identity gene and a regulator of sepal and petal development. In *Arabidopsis thaliana*, AP1 expression is activated in response to floral inductive signals and serves to specify the fate of floral primordia while simultaneously repressing inflorescence meristem identity (**Table 1**) [21, 22]. Loss-of-function *ap1* mutants exhibit partial conversion of flowers into inflorescence shoots, highlighting the critical role of AP1 in establishing floral meristem identity.

FUL, a closely related MADS-box gene, exhibits partially redundant functions with AP1 and contributes to inflorescence architecture and fruit development. While *ful* single mutants display relatively subtle flowering phenotypes, *ap1 ful* double mutants show enhanced flowering delay and more severe defects in floral meristem specification, indicating functional overlap between these two regulators (**Table 1**) [23]. FUL also plays specific roles in cauline leaf development and valve differentiation during fruit development, demonstrating the pleiotropic functions characteristic of MADS-box proteins.

SEP1-4 proteins serve as essential co-factors that interact with other MADS-domain proteins to form higher-order transcriptional complexes. Originally identified through the severe floral phenotype of *sep1 sep2 sep3* triple mutants, which exhibit conversion of all floral organs into sepal-like structures, the SEP proteins are now recognized as required partners for the proper function of class A, B, and C MADS-box proteins (**Table 1**) [34, 35]. SEP3, in particular, acts as a central hub in the MADS-box protein interaction network, promoting formation of multimeric complexes with enhanced DNA-binding specificity and affinity (**Table 1**) [36].

2.1.2 The ABC(E) model

In the ABC model, a flower is composed of four concentric whorls bearing different kind of floral organs – sepals, petals, stamens, and carpels – sequentially arranged from the outer to the inner part of the flower. This floral structure is typical of core eudicots. The ABC model, initially based on floral mutant phenotypes in *Arabidopsis* and *Antirrhinum*, postulates that three different gene classes, or functions, specify the identity of these organs in the four floral whorls. In the outermost whorl, the A-function specifies sepal identity, while the A- and B-function genes together control petal identity in the second whorl. In the third whorl, the B- and C-function genes are required for stamen development, while the

C-function by itself specifies carpel identity in the inner whorl and terminates floral meristem activity. In addition, the A- and C-functions antagonize each other, enforcing the proper domains of activity. A later addition to the model are the E-function genes, which are required for all the other functions, since they encode proteins necessary for the formation of the higher order complexes in which most of the ABC-function proteins are active [62, 63]. Interestingly, except for one of the A-function genes, *APETALA2* (*AP2*), all the ABCE-functions are encoded by one or more type II MADS-box genes.

Class A genes, including *AP1* and *AP2*, specify sepal identity in the first floral whorl and contribute to petal development in combination with class B genes. Class B genes, represented by *APETALA3* (*AP3*) and *PISTILLATA* (*PI*), function together to specify petal and stamen identity in the second and third whorls, respectively. Class C genes, primarily *AGAMOUS* (*AG*), determine stamen and carpel identity in the third and fourth whorls while simultaneously repressing class A function in these inner whorls [64, 65]. Class E genes, the *SEP* clade, are required for the activities of all floral organ identity genes, functioning as obligatory co-factors that enable the formation of functional tetrameric complexes [34].

Several members have been shown to form dimers and consequently two functional regions within the MADS-box have been defined. The N-terminal half is the major determinant of DNA-binding specificity whilst the C-terminal half is necessary for dimerisation. This organisation allows the potential formation of numerous proteins, with subtly different DNA-binding specificities, from a limited number of genes by heterodimerisation between different MADS-box proteins. The majority of MADS-box proteins bind similar sites based on the consensus sequence $CC(A/T)_6GG$ although each protein apparently possesses a distinct binding specificity [66, 67]. This higher-order complex formation enables synergistic transcriptional activation and provides a mechanistic basis for the combinatorial specification of floral organ identity. Moreover, several MADS-box proteins specifically recruit other transcription factors into multi-component regulatory complexes.

Recent advances have highlighted the dynamic nature of these protein-protein interactions and their integration with hormonal and environmental signals. For instance, the *AP1-FUL* clade exhibits context-dependent interactions with additional co-factors, including *LEAFY* and its transcriptional co-regulators, thereby linking floral meristem identity specification with downstream organ development [68]. Moreover, chromatin immunoprecipitation coupled with high-throughput sequencing (ChIP-seq) has revealed extensive genome-wide binding patterns for MADS-box proteins, uncovering thousands of potential target genes and revealing complex regulatory hierarchies underlying floral patterning [69, 70].

2.2 Flowering integrators and pathway-specific regulators

MADS-box proteins such as *SOC1* and *FLC* act downstream of photoperiod, vernalization, and GA pathways to regulate flowering. Transcription factors including *CO*, *FT*, and *LFY* function as floral integrators, relaying inputs from these pathways to meristem identity genes. Together, they converge diverse regulatory signals to control flowering time and floral development.

2.2.1 *FT* and *SOC1*

FT and SOC1 function as primary floral integrators that regulate photoperiod, vernalization, GA, autonomous, and age pathways (**Table 1**) [37, 38]. FT, a member of the phosphatidylethanolamine-binding protein (PEBP) family, functions as the long-sought florigen. This mobile signal is synthesized in leaves and translocated via the phloem to the shoot apical meristem, where it initiates the floral transition (**Table 1**) [39, 40]. The systemic movement of FT protein through the phloem establishes a long-distance communication system that coordinates flowering responses across the plant body.

At the shoot apical meristem, FT interacts with the bZIP TF FD through a 14-3-3 protein-mediated mechanism, forming a transcriptional activation complex that directly induces expression of downstream floral meristem identity genes including *AP1* and *SOC1* (**Table 1**) [41–43]. This FT-FD module exhibits remarkable evolutionary conservation, with FT homologs identified as flowering regulators in diverse angiosperm species ranging from rice to poplar [71, 72].

SOC1, a MADS-box TF, integrates signals from multiple flowering pathways at the transcriptional level. SOC1 expression is directly activated by CO under long-day conditions, repressed by FLC in response to vernalization, and induced by GA signaling through the destabilization of DELLA repressors (**Table 1**) [29–31]. The SOC1 protein, in turn, directly activates floral meristem identity genes including *LFY* and *AP1*, positioning it as a central hub that translates diverse regulatory inputs into a coherent developmental output (**Table 1**) [32, 33].

2.2.2 *CO*

CO encodes a B-box zinc finger TF, and it plays a central role in photoperiodic flowering regulation [44]. *CO* expression is subject to complex circadian and light-mediated control at both transcriptional and post-translational levels, enabling precise measurement of day length. The circadian clock regulates *CO* transcription such that mRNA accumulation peaks approximately 12 hours after dawn, but this peak occurs during daylight only under long-day conditions (**Table 1**) [45, 46].

Post-translational mechanisms provide an additional regulatory layer. CO protein stability is directly modulated by light quality through the action of photoreceptors including phytochrome A (phyA), cryptochromes (CRY1 and CRY2), and the E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1). Under long-day conditions, light stabilizes CO protein during the afternoon, enabling accumulation to levels sufficient for FT activation. Under short days, CO protein peaks during darkness and is rapidly degraded by COP1-mediated ubiquitination (**Table 1**) [47, 48]. This dual regulation ensures that FT is activated only under inductive photoperiods, establishing the CO-FT module as a conserved mechanism linking photoperiod perception to floral induction across diverse angiosperm species [49].

Beyond FT regulation, CO directly targets a suite of additional genes involved in flowering time control and stress responses, expanding its regulatory influence beyond the canonical photoperiod pathway. Genome-wide binding studies have identified hundreds of CO-bound loci, including genes involved in hormone signaling, circadian clock regulation, and metabolic processes, suggesting broader roles for CO in coordinating developmental transitions with environmental conditions (**Table 1**) [50, 51].

2.2.3 *LEAFY* (*LFY*)

LFY functions as a master regulator of floral meristem identity, integrating upstream signals from both the photoperiod and GA pathways [73, 74]. *LFY* expression is directly activated by *SOC1* and by SPL TFs from the age pathway, positioning it downstream of multiple flowering integrators. Conversely, *LFY* also participates in positive feedback loops that reinforce flower development, including direct activation of *AP1* and indirect activation of *SOC1* through repression of the flowering repressor TERMINAL FLOWER 1 (*TFL1*) (**Table 1**) [75, 76].

At the molecular level, *LFY* functions as a pioneer TF capable of binding condensed chromatin and facilitating the recruitment of additional TFs to target loci. *LFY* directly activates the expression of floral homeotic genes, including *AP1*, *AP3*, and *AG*, by binding to specific *cis*-regulatory elements enriched in the promoters of these genes [77, 78]. Through direct activation of floral organ identity genes, *LFY* links floral induction to downstream organ development.

The DNA-binding specificity of *LFY* is modulated by interaction with co-factors including the TF UNUSUAL FLORAL ORGANS (*UFO*), which acts as a transcriptional co-activator to facilitate *LFY*-mediated activation of class B genes [79]. Through this co-factor interaction, *LFY* achieves whorl-specific transcriptional activity required for proper floral organ specification.

2.3 Transcriptional repressors and the fine-tuning of floral transition

While activators of flowering have received considerable attention, transcriptional repressors play equally important roles in preventing precocious flowering and ensuring that the floral transition occurs only under favorable conditions. These repressors integrate environmental and endogenous signals to fine-tune flowering timing and provide developmental plasticity.

2.3.1 *FLC* and its epigenetic regulation in vernalization

FLC, a MADS-box TF, functions as a potent repressor of flowering that integrates vernalization and autonomous pathway signals. *FLC* directly represses the transcription levels of floral integrators *FT* and *SOC1* by binding to specific CArG-box motifs in their regulatory regions, thereby preventing premature flowering under non-inductive conditions [24, 25]. In winter-annual ecotypes of *Arabidopsis* and many temperate cereals, high *FLC* expression establishes a strong block to flowering that can only be overcome by prolonged cold exposure.

The molecular mechanisms underlying *FLC* repression operate at multiple levels. The autonomous pathway maintains basal *FLC* repression through FLOWERING LOCUS D (*FLD*)-mediated histone deacetylation and FCA-mediated alternative splicing (**Figure 1**) [80, 81]. However, the most dramatic regulation occurs through vernalization, which aligns flowering with spring by epigenetically silencing *FLC* in response to winter cold.

A prerequisite for vernalization-induced silencing is transcriptional downregulation of *FLC*. This correlates with decreased levels of H3K36me3 and H3K4me3, histone marks deposited during FRIGIDA (*FRI*)-dependent *FLC* activation (**Figure 1**) [82, 83]. Cold exposure rapidly promotes the formation of *FRI* nuclear condensates that do not colocalize with the active *FLC* locus, reducing *FRI*

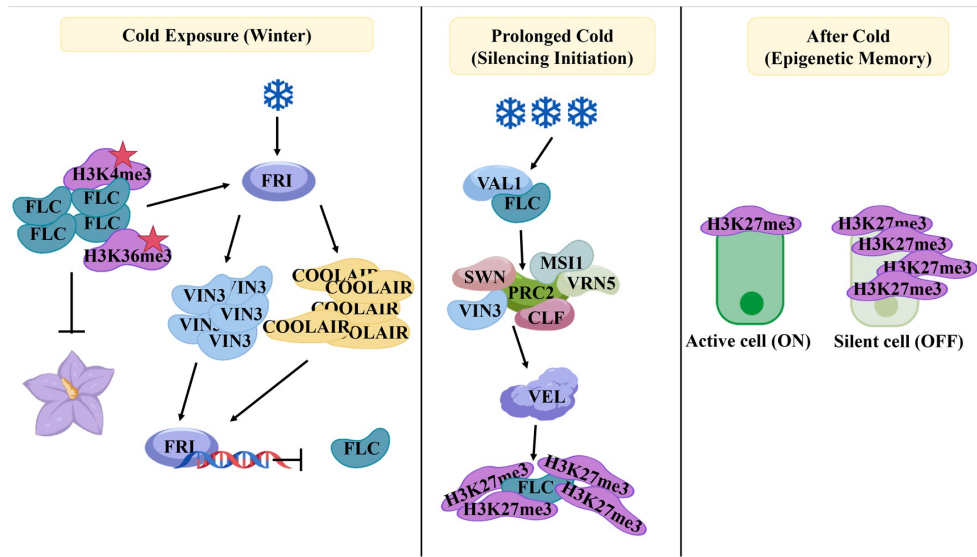


Figure 1.

Vernalization-mediated epigenetic silencing of FLC. Three-step molecular model of FLC repression triggered by prolonged cold. Step 1: cold exposure induces FRI condensate formation, VIN3 expression, and COOLAIR accumulation, leading to FLC transcriptional downregulation. Step 2: VAL1 recruits PRC2 complex to the nucleation region; VEL proteins form condensates to facilitate h3k27me3 spreading. Step 3: after cold, FLC is stably silenced by h3k27me3 in a digital, cell-autonomous, and mitotically stable manner.

occupancy at the FLC promoter and contributing to transcriptional repression. Warm temperature spikes can reverse this process, buffering FLC shutdown to prevent premature flowering. The accumulation of FRI condensates in the cold is influenced by specific co-transcriptional regulators and by cold-induced expression of a specific isoform of the antisense RNA COOLAIR.

Transcriptional downregulation sets the stage for Polycomb-mediated epigenetic silencing. During prolonged cold, the PHD finger protein VERNALIZATION INSENSITIVE 3 (VIN3) is induced and associates with Polycomb Repressive Complex 2 (PRC2), facilitating the deposition of repressive H3K27me3 marks across the FLC locus (**Figure 1, Table 1**) [26–28]. This Polycomb-mediated silencing is mitotically stable, maintaining winter memory through subsequent cell divisions after cold exposure ends. Longer cold leads to progressively higher H3K27me3 accumulation and more stable repression, allowing plants to integrate winter duration and flower only after vernalization requirements are fully met (**Figure 1, Table 1**) [82, 83].

The FLC system thus represents a paradigm for environmentally triggered epigenetic regulation, revealing how plants integrate seasonal cues with developmental programs.

2.3.2 TEMPRANILLO (TEM) and SMALL AUXIN UP RNA (SAUR) families in growth-reproduction balance

Several TF families function in parallel with FLC to coordinate vegetative growth and flowering. The TEMPRANILLO (TEM) genes, for example, encode AP2/ERF-family repressors that directly downregulate FT (**Table 1**) [54]. TEM1 and TEM2

bind *cis*-elements in the FT promoter and recruit transcriptional co-repressors, maintaining low FT expression during vegetative development.

TEM proteins also integrate auxin signaling with flowering time control, linking the balance between growth and reproduction. TEM1 directly regulates genes involved in auxin biosynthesis and response, and conversely, auxin signaling modulates TEM expression, establishing a feedback loop that coordinates developmental transitions with hormone status (**Table 1**) [55]. This integration ensures that flowering occurs only when plants have achieved sufficient vegetative development and resource accumulation.

SMALL AUXIN UP RNA (SAUR) family, best known for regulating auxin transport and cell expansion, also influence flowering time. Several SAUR family members show flowering phenotypes when overexpressed or mutated, suggesting roles in meristem maturation or hormone crosstalk [84]. SAUR-mediated auxin signaling may affect flowering via meristem maturation, photoperiod response, or GA pathway interactions, highlighting the complexity of hormonal crosstalk in flowering control.

Flowering time reflects the balance between repressors and activators. Repressors like FLC and TEM prevent precocious flowering, while activators like CO and FT promote the floral transition when environmental and developmental conditions are favorable. This antagonism allows the flowering network to integrate diverse signals and respond appropriately.

3. Regulatory networks and the integration of signaling pathways in flower development

The TFs described above do not act alone. They function within an interconnected network that integrates endogenous and environmental signals, allowing plants to align flowering with internal development and external conditions. This section examines the major pathways that converge on flowering time, including photoperiod, vernalization, gibberellin, age, and ambient temperature, focusing on their molecular mechanisms, cross-talk, and integration (**Figure 2**).

3.1 Photoperiodic pathway

Plants perceive seasonal changes in day length primarily through the photoperiod pathway, which adjusts flowering time accordingly. In *A. thaliana*, a facultative long-day species, flowering accelerates under long days via the CO-FT module [85, 86]. The circadian clock controls CO transcription through GIGANTEA (GI), FLAVIN-BINDING KELCH REPEAT F-BOX 1 (FKF1), and CYCLING DOF FACTORS (CDFs), restricting CO expression to specific times of day [85]. CO protein stability is further modulated by light quality through photoreceptors including phyA and cryptochromes, which antagonize COP1-mediated degradation [87]. FT, encoding the mobile florigen signal, is then activated specifically under inductive long days [88, 89]. Additional regulation of the CO-FT module comes from several factors. The chromatin-remodeling factor PICKLE (PKL) physically interacts with CO to facilitate its access to FT chromatin, mediating the diurnal switch between active and inactive states at dusk and night, respectively. Furthermore, the

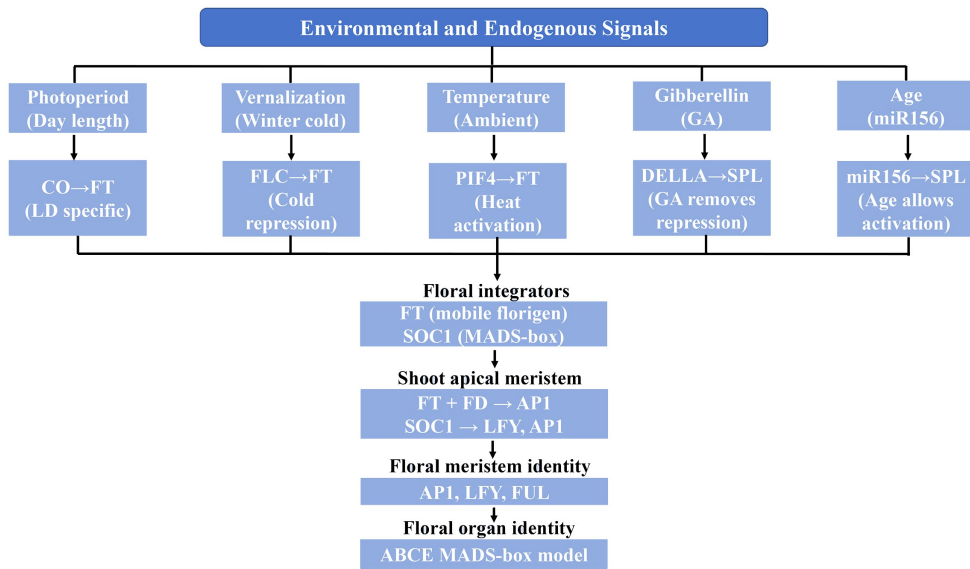


Figure 2.

Integration of multiple signaling pathways governing flowering time. Environmental and endogenous signals (photoperiod, vernalization, ambient temperature, gibberellin, and age) converge on floral integrators FT and SOC1. These integrators activate floral meristem identity genes (AP1, LFY, FUL) at the shoot apical meristem, which in turn specify floral organ identity through the ABCE mads-box model.

BBX28 and BBX29 proteins promote flowering under long-day conditions at low ambient temperature (16 °C) by interacting with CO to enhance its transcriptional activation of FT and its paralog TWIN SISTER OF FT (TSF). Consistent with this, *bbx28 bbx29* double mutants exhibit delayed flowering and reduced FT/TSF expression under these conditions [90].

3.1.1 Functional diversification through gene duplication

The CO-FT module varies considerably across angiosperm lineages, shaped by life history and ecological context [91, 92]. In the perennial tree *Populus trichocarpa*, for instance, gene duplication and subfunctionalization have produced two FT paralogs with divergent roles. *PtFT1* promotes flowering. Its overexpression induces precocious flowering within weeks (compared to the typical 8-20 year juvenile phase), and its expression is specifically induced by cold treatment in reproductively mature trees, corresponding to winter inflorescence development [92]. In contrast, *PtFT2* is expressed only under warm, long-day conditions and regulates the developmental switch between vegetative growth and dormancy. When heat-shock drives *PtFT2* expression, plants fail to set buds under normally inductive short days and instead continue growing [92]. Natural variation across latitudinal clines in European aspen shows that day-length thresholds for growth cessation shift with latitude, correlating with altered *PtFT2* expression and its putative regulator *PtCO2* [91]. Notably, *PtFT1* shows no diurnal variation and is primarily temperature-regulated, while *PtFT2* exhibits strong photoperiodic regulation, demonstrating subfunctionalization of duplicated FT genes [92]. The FD homolog *PtFD1* integrates these FT signals to coordinate both reproductive and vegetative phase transitions,

being transiently upregulated in apical buds during early short-day exposure; overexpression of *PtFD1* disrupts short-day-induced growth cessation and bud formation while also causing precocious flowering under long days [93]. Furthermore, the AP1 ortholog LAP1 acts downstream of the CO-FT module to regulate seasonal growth cessation through an AINTEGUMENTA-like TF rather than flowering-related targets, indicating that functional divergence downstream of AP1/LAP1 enables the same photoperiodic signal to control distinct developmental programs in different species [94].

3.1.2 Lineage-specific innovations in monocots

In rice (*Oryza sativa* L.), a facultative short-day plant, flowering time is controlled by both the evolutionarily conserved CO-FT module (Hd1-Hd3a) and a unique Ehd1-dependent pathway. Early heading date 4 (Ehd4) functions as a novel positive regulator of Ehd1, encoding a CCCH-type zinc finger protein localized to the nucleus that exhibits transcriptional activation activity [95]. The *ehd4* mutant displays a never-flowering phenotype under natural long-day conditions, indicating that Ehd4 is essential for floral induction under non-inductive photoperiods. *Ehd4* is predominantly expressed in young leaves with a diurnal pattern similar to *Ehd1* under both short-day and long-day conditions, and it promotes flowering by up-regulating *Ehd1* expression, which in turn activates the florigen genes *Hd3a* and *RFT1*. Notably, Ehd4 acts independently of other known Ehd1 regulators including Hd1, Ghd7, and OsMADS50, and is highly conserved within the *Oryza* genus but absent in other plant lineages, representing an *Oryza*-genus-specific innovation that integrates with the ancestral CO-FT pathway to confer photoperiodic flowering control [95].

In maize bamboo (*Dendrocalamus latiflorus*), a conserved CO-FT regulatory module exists wherein DICO1 regulates *DIFT1* expression, and *DIFT1* functions as a florigen candidate that promotes flowering by interacting with DIFD and upregulating downstream AP1, while DITFL1 antagonizes *DIFT1* function through competitive interaction with DIFD [96].

3.1.3 Recruitment for alternative developmental programs

The CO-FT module has been co-opted to regulate diverse photoperiodic responses beyond flowering. In potato (*Solanum tuberosum*), this module regulates photoperiodic tuberization rather than flowering, wherein StCO represses tuberization under non-inductive photoperiods by modulating expression of the mobile signals StBEL5 and StFT/StSP6A, which promote tuber formation [97]. In the perennial plant *Arabis alpina*, the CO-FT module regulates flowering in a shoot-specific manner. AaCO activates *AaFT/TSFL* transcription in leaves to promote flowering under long days, but while both genes are required for inflorescence development on the primary shoot, AaCO is dispensable for axillary branch flowering whereas *AaFT/TSFL* is essential, revealing that subfunctionalization of this module contributes to the complex polycarpic perennial life history [98].

3.1.4 Evolutionary dynamics in polyploid and domesticated species

In allotetraploid cotton (*Gossypium spp.*), eight group I *CONSTANS-like* (*COL*) genes exhibit diurnal expression patterns and have undergone asymmetric

evolutionary pressures between the A and D subgenomes following polyploidization, with *COL1*, *COL2*, and *COL8* showing evidence of domestication-associated selection, indicating that the conserved CO-FT module has been shaped by polyploidy and domestication to regulate photoperiodic flowering [99]. Similarly, in *Brassica napus*, quantitative trait loci (QTL) mapping and genome-wide association studies have identified FT, CO, and FLC as key loci associated with ecotypic differentiation and flowering time variation across multiple environments, confirming that the CO-FT module mediates photoperiodic flowering by integrating environmental signals at the shoot apical meristem, wherein FT acts as a floral pathway integrator together with SOC1 to initiate flowering [100].

3.1.5 Integration of diverse environmental cues

Beyond photoperiod, the CO-FT module functions as a central hub integrating diverse environmental signals. Transcriptomic analyses of *Arabidopsis* plants grown on the Chinese space station revealed that this module integrates spaceflight-associated microgravity signals with transcriptional networks involving ERF, bZIP, bHLH, and BES1 TFs to modulate adaptive growth responses, demonstrating the broad signal-integration capacity of the CO-FT regulatory hub [101].

Collectively, these comparative studies reveal that while the CO-FT module serves as a core regulatory hub across angiosperms, lineage-specific gene duplications, subfunctionalization, neofunctionalization, and recruitment for alternative developmental programs have generated remarkable diversity in how photoperiodic signals are transduced to control flowering time, dormancy transitions, tuberization, and inflorescence architecture.

3.2 Vernalization pathway

Vernalization refers to the promotion of flowering by prolonged exposure to winter cold, a mechanism that aligns reproductive development with favorable spring conditions. In *A. thaliana* and many temperate cereals, vernalization acts through epigenetic silencing of the potent floral repressor FLC [102, 103]. FLC encodes a MADS-box TF that directly represses the floral integrators FT and SOC1, and in winter-annual ecotypes, high *FLC* expression establishes a strong block to flowering that can only be overcome by cold exposure. The vernalization pathway has emerged as a paradigm for understanding environmentally triggered epigenetic regulation in plants, with recent advances revealing multiple interconnected layers of control operating at transcriptional, post-transcriptional, and chromatin organizational levels.

3.2.1 Nucleation and establishment of polycomb silencing

During vernalization, the PHD finger protein VIN3 is progressively induced and associates with PRC2 (**Table 1**) [56]. This complex catalyzes H3K27me₃, a repressive chromatin mark, across the FLC locus. The initiation of this silencing process requires sequence-specific targeting of transcriptional repressors to an intragenic nucleation region within FLC. The transcriptional repressor VAL1 binds to this nucleation region *in vivo*, promotes histone deacetylation, and interacts with the conserved ASAP complex, demonstrating that sequence-specific targeting recruits

the machinery necessary for establishing Polycomb silencing at FLC (**Table 1**) [59]. The importance of this nucleation site is underscored by the finding that a single point mutation within this region prevents Polycomb-mediated epigenetic silencing by blocking PHD-PRC2 nucleation [59].

Recent studies have revealed that VEL proteins (VERNALIZATION LIKE) play distinct accessory roles in Polycomb silencing, facilitating the spread and maintenance of H3K27me3 during and after cold exposure [57]. The VEL proteins VRN5 and VIN3 exhibit distinct molecular functions. VRN5 adopts a compact conformation that enables intimate association with multiple PRC2 components and is required for both H3K27me2 and H3K27me3 deposition, whereas VIN3 interacts with the transcriptional repressor VAL1 and is specifically required for H3K27me3 but not H3K27me2 [57]. Vernalization-mediated epigenetic silencing further requires the ability of VEL proteins to form biomolecular condensates, as they contain a four-helix bundle domain that undergoes concentration-dependent head-to-tail polymerization; mutations disrupting this polymerization prevent Polycomb silencing at FLC, demonstrating that VEL-mediated condensate assembly facilitates the nucleation and propagation of the silenced state (**Table 1**) [58].

3.2.2 Integration of transcriptional and chromatin regulatory layers

A computational model integrating co-transcriptional RNA processing, histone demethylation, and PRC2 function at FLC reveals that the RNA-binding protein FCA mediates proximal polyadenylation and termination, which recruits the histone demethylase FLD to remove H3K4me1 [104]. This H3K4me1 removal reduces RNA polymerase II processivity, reinforcing early termination and repressing productive transcription. The resulting transcription-coupled repression determines the level of transcriptional antagonism to PRC2 action, thereby dictating the timescale for establishment of PRC2-mediated H3K27me3 silencing and the rate of the ON-to-OFF epigenetic switch at FLC [104].

3.2.3 Temperature-dependent regulation of transcriptional activators

The flowering regulator FRIGIDA (FRI) undergoes temperature-dependent phase separation to control *FLC* expression, providing a mechanistic link between ambient temperature sensing and epigenetic regulation. In warm conditions, FRI remains soluble and associates with transcriptional co-activators to activate *FLC*, while cold exposure stabilizes FRI and promotes its sequestration into nuclear condensates, thereby removing it from the *FLC* promoter and facilitating transcriptional shutdown [105].

3.2.4 Nuclear organization and physical clustering

Vernalization involves cold-induced epigenetic silencing of FLC through Polycomb-mediated mechanisms accompanied by specific changes in nuclear organization, including disruption of an intragenic gene loop and physical clustering of FLC loci [106]. These nuclear reorganization events occur at distinct temporal phases, with locus clustering tightly correlated with the accumulation of Polycomb complexes and H3K27me3 at a localized intragenic nucleation site during cold exposure, suggesting that changes in nuclear architecture are mechanistically linked

to the cell-autonomous bistable switching that underlies quantitative vernalization response [106]. Live-cell imaging of FLC-lacO alleles has confirmed that cold exposure induces physical clustering of FLC loci, and this clustering depends on Polycomb factors required for establishing silencing (such as VIN3) but not on LIKE HETEROCHROMATIN PROTEIN 1 (LHP1), which maintains silencing, suggesting that physical clustering of Polycomb target loci may represent a conserved feature of epigenetic switching mechanisms during vernalization [107].

3.2.5 Digital switching and quantitative memory

The Polycomb-mediated silencing established during cold exposure is mitotically stable, persisting through subsequent cell divisions after temperatures rise. This epigenetic memory ensures that *FLC* remains repressed throughout the remainder of the plant's life cycle, enabling flowering to occur in the spring following winter [103]. Quantitative analyses have revealed that vernalization operates through a digital paradigm. Longer cold exposure leads to an increasing proportion of cells switching *FLC* to the OFF state, and this all-or-nothing cellular memory is maintained through subsequent cell divisions [103]. Recent single-cell analyses have confirmed that *FLC* expression exhibits bimodal distribution during vernalization, with cells existing in either fully ON or fully OFF states [108].

3.2.6 Natural variation in silencing stability

Natural variation in epigenetic silencing of *FLC* in *Arabidopsis* accessions involves an additional long-term perpetuated phase downstream of Polycomb-mediated H3K27me3 spreading. In the Lov-1 accession, noncoding SNPs in the nucleation region cause unstable silencing with frequent digital *FLC* reactivation in individual cells upon return to warm, with reactivation probability decreasing after longer cold due to reduced DNA replication post-flowering [109].

3.2.7 Epigenetic resetting across generations

The vernalized state must be reset during each generation to maintain the requirement for winter cold. This resetting occurs during embryogenesis and involves active removal of H3K27me3 marks from FLC chromatin [56]. The DNA polymerase α subunit INCURVATA2 (ICU2) and the histone chaperone CAF-1 complex contribute to the proper resetting of epigenetic marks during reproductive development [110]. The precise coordination between silencing and resetting ensures that vernalization requirements are faithfully transmitted across generations while preventing inappropriate inheritance of the vernalized state. Although originally identified as vernalization regulators, VIN3 and VRN5 co-associate with numerous H3K27me3-marked targets genome-wide, indicating broad relevance for PRC2-mediated silencing beyond FLC [57].

Together, sequence-specific nucleation, VEL-mediated condensate assembly, transcription-coupled repression, nuclear reorganization, digital epigenetic switching, and germline resetting constitute a sophisticated regulatory framework that enables plants to quantitatively perceive winter duration, establish mitotically stable epigenetic memory, and align flowering with favorable spring conditions.

3.3 GA pathway

GAs are tetracyclic diterpenoid hormones that regulate diverse developmental processes, including seed germination, stem elongation, and flowering. The GA pathway is particularly critical for flowering under non-inductive photoperiods, such as short days in many rosette plants [14].

DELLA proteins function as master repressors of GA signaling. Named for the conserved N-terminal DELLA motif, these nuclear-localized TFs suppress GA-responsive growth and development. In Arabidopsis, five DELLA proteins, including GA INSENSITIVE (GAI), REPRESSOR OF GA1-3 (RGA), RGA-LIKE1 (RGL1), RGL2, and RGL3, exhibit partially redundant functions, with RGA and RGL1 playing particularly prominent roles in flowering regulation [14].

GA signaling is initiated by binding of bioactive GAs to the GIBBERELLIN INSENSITIVE DWARF1 (GID1) receptor, which undergoes a conformational change that promotes interaction with DELLA proteins [14]. This GA-GID1-DELLA complex recruits F-box proteins of the SCF E3 ubiquitin ligase complex, leading to DELLA polyubiquitination and subsequent degradation by the 26S proteasome. This degradation relieves DELLA-mediated repression, permitting activation of downstream genes. Studies in lettuce have demonstrated that the DELLA protein LsRGL1 negatively regulates bolting and flowering by modulating both GA and auxin biosynthesis, indicating that DELLA functions as a hub connecting multiple hormone pathways [111].

DELLA proteins directly interact with several key flowering regulators. DELLAs bind to and inhibit the activity of SPL TFs, which activate floral integrators including SOC1 and LFY [14]. DELLAs also interact with PIFs, integrating GA signaling with light and temperature pathways. Additionally, DELLAs can sequester the floral activator FD, preventing its interaction with FT at the shoot apex. Through these multiple mechanisms, GA signaling modulates flowering time in response to developmental and environmental cues [14].

3.4 Autonomous pathway

Autonomous pathway promotes flowering through constitutive repression of FLC, independent of environmental signals. This pathway ensures that flowering eventually occurs even in the absence of inductive cues, acting as a developmental timer or fail-safe mechanism [56, 112, 113].

Several autonomous pathway genes encode proteins involved in RNA processing. FCA and FY function in alternative polyadenylation, regulating 3' end formation of FLC antisense transcripts. FPA, another RNA-binding protein, acts redundantly with FCA to promote FLC repression through effects on transcription termination and chromatin state. Recent studies have revealed that the Cleavage and Polyadenylation Specificity Factor (CPSF) phosphatase module, including APRF1 (yeast Swd2/human WDR82), TOPP4 (PP1 phosphatase), and LD (Like Heterochromatin Protein 1), links transcription termination to chromatin silencing at the FLC locus [114]. This module physically associates with chromatin modifiers, generating a transcription-coupled silencing mechanism that ensures robust FLC repression.

Additional autonomous pathway components modulate *FLC* expression through chromatin-based mechanisms. FVE (also known as AtMSI4) encodes

a retinoblastoma-associated protein that interacts with histone deacetylase complexes to promote repressive chromatin modifications at FLC [115]. FLD encodes a lysine-specific demethylase-like protein that reduces histone H3 lysine 4 (H3K4) methylation, an activating mark, at the FLC locus. Recent advances have demonstrated that FLD associates with LD and other factors to form a complex that specifically removes H3K4me1 in the central region of FLC, contributing to transcriptional silencing [114].

Autonomous pathway and vernalization pathway converge on FLC repression through complementary mechanisms. While vernalization establishes stable Polycomb-mediated silencing, autonomous pathway maintains basal repression levels and facilitates the rapid downregulation of *FLC* upon cold exposure [115]. Autonomous pathway also prevents inappropriate reactivation of *FLC* after vernalization, contributing to the stable maintenance of the silenced state. This coordination ensures robust FLC regulation across diverse environmental conditions.

3.5 Age pathway

Age pathway functions as an endogenous timing mechanism that ensures plants initiate flowering only after attaining appropriate developmental maturity, thereby integrating internal physiological status with environmental signals. This pathway is primarily orchestrated by the miR156-SPL-miR172 regulatory cascade (Figure 3).

During early vegetative development, high concentrations of miR156 maintain the juvenile phase by repressing the expression of SPL TFs. As plants undergo progressive maturation, miR156 levels gradually decline, permitting the accumulation of SPL proteins, which in turn activate downstream flowering-related genes including miR172. In *A. thaliana*, miR156 targets multiple SPL family members

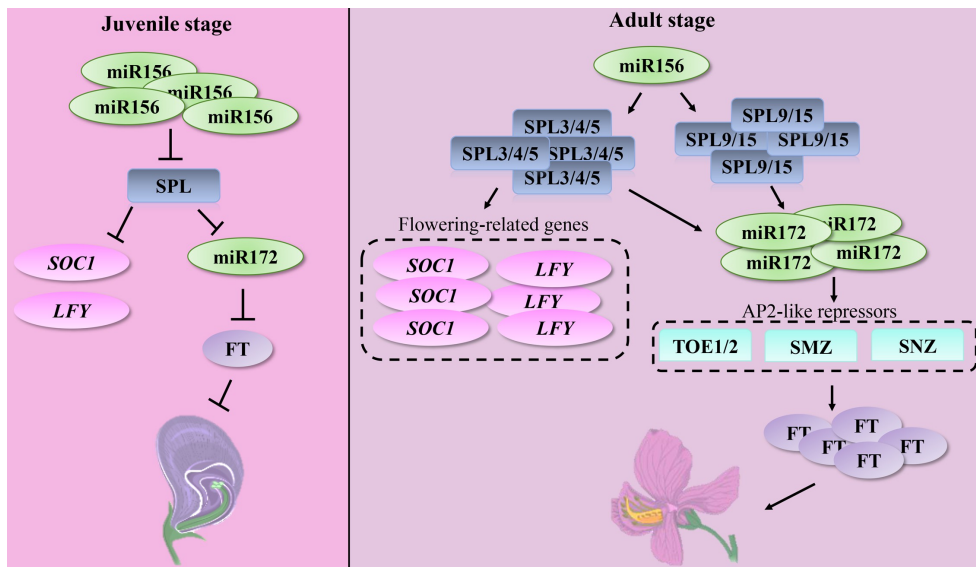


Figure 3.

The miR156-SPL-miR172 cascade in age-dependent flowering. During juvenile development, high miR156 represses SPL transcription factors. As plants mature, miR156 levels decline, allowing SPL proteins to accumulate and activate miR172. MiR172 represses ap2-like floral repressors (TOE1, TOE2, SMZ, SNZ), thereby de-repressing FT and promoting the floral transition.

with distinct functions. SPL2, SPL9, SPL10, SPL11, SPL13, and SPL15 are primarily associated with flowering time regulation, while SPL3, SPL4, and SPL5 specifically promote floral meristem differentiation (**Table 1, Figure 3**) [53]. Among these, SPL9 and SPL15 directly activate miR172 transcription by binding to its promoter region, establishing a regulatory link between the two microRNA modules.

miR172 functions downstream of the miR156-SPL module to promote flowering by repressing APETALA2 (AP2)-like TF genes. Specifically, miR172 targets *TARGET OF EARLY ACTIVATION TAGGED 1* (*TOE1*), *TOE2*, *SCHNARCHZAPFEN* (*SNZ*), and *SCHLAFMUTZE* (*SMZ*), which act as floral repressors. Degradation of these AP2-like transcripts relieves their repression of *FT*, thereby facilitating floral transition (**Table 1, Figure 3**) [116].

Notably, miR156 and miR172 exhibit temporally opposing expression patterns during shoot maturation. miR156 declines with age while miR172 concomitantly increases. Genetic analyses have established that miR156 functions upstream of miR172, as *SPL9* and *SPL15*, direct targets of miR156, mediate miR172 transcriptional activation (**Table 1, Figure 3**) [53]. This hierarchical regulatory cascade ensures that flowering occurs only after sufficient vegetative growth has been achieved, thereby optimizing reproductive success by aligning floral transition with developmental competence.

3.6 Thermosensory pathway

Ambient temperature regulates flowering time through the thermosensory pathway, which integrates temperature signals with endogenous developmental cues. The central floral integrator *FT* serves as the core output of this pathway, with its expression dynamically modulated by temperature fluctuations. In *A. thaliana*, an increase in ambient temperature from 23 °C to 27 °C promotes *FT* expression and accelerates flowering [117]. The thermosensitive TF PHYTOCHROME INTERACTING FACTOR 4 (PIF4) accumulates at elevated temperatures and directly activates *FT* transcription by binding to its promoter (**Table 1**) [52].

FT expression is also modulated by the MADS-box TFs FLOWERING LOCUS M (FLM) and SHORT VEGETATIVE PHASE (SVP). FLM undergoes temperature-dependent alternative splicing, generating two major isoforms. FLM- β predominates at 16 °C, while FLM- δ becomes the predominant isoform at 27 °C [118]. At low temperatures, the FLM- β -SVP complex represses *FT* transcription by binding to *FT* promoter motifs. Upon warming, the formation of the SVP-FLM- δ complex alleviates this transcriptional repression, thereby promoting flowering [118].

In addition to transcriptional regulation, microRNAs (miRNAs) contribute to thermosensory flowering control. Several miRNAs, including miR156, miR172, miR163, miR169, and miR399, are involved in temperature-mediated flowering regulation [119]. Among these, miR156 plays a predominant role. Elevated temperatures downregulate *miR156* expression, leading to increased accumulation of its target SPL TFs. The miR156-SPL regulatory module thus integrates temperature signals into the flowering pathway by modulating *FT* expression [120].

Collectively, PIF4-mediated transcriptional activation, FLM-SVP alternative splicing, and miRNA-mediated post-transcriptional regulation enable plants to fine-tune flowering time in response to ambient temperature fluctuations.

4. Ecological considerations in flowering time regulation

Flowering time is not merely a developmental decision; it is a critical ecological trait that determines plant fitness in natural and agricultural settings. The molecular networks described in this chapter – centered on TFs such as FLC, CO, FT, and SOC1 – enable plants to perceive and respond to seasonal cues, thereby aligning reproduction with favorable environmental conditions.

In natural populations, variation in flowering time contributes to local adaptation. For example, latitudinal clines in *Arabidopsis thaliana* are often associated with allelic variation at FLC and FRIGIDA, which modulate vernalization requirements and photoperiod sensitivity [91, 102]. Such variation allows populations to track climatic gradients and optimize reproductive success across different geographic regions.

Flowering phenology also influences species interactions. Shifts in flowering time can disrupt synchrony with pollinators, alter competitive dynamics among co-flowering species, or affect seed dispersal timing. In fragmented landscapes or under climate change, mismatches between flowering time and pollinator emergence can have cascading effects on plant reproductive success and population persistence [10, 11].

From an evolutionary perspective, flowering time variation can drive reproductive isolation. Divergent selection on flowering time genes may lead to allochronic speciation, where populations flowering at different times become genetically isolated. This process has been documented in several plant groups, highlighting the role of flowering time regulation in diversification [3].

In the context of global climate change, understanding the molecular basis of flowering time becomes increasingly urgent. Rising temperatures are shifting phenology worldwide, and the capacity of plant populations to track these changes depends on the standing genetic variation within flowering time networks. Species or populations with limited adaptive potential may face increased risk of phenological mismatch, reduced reproductive success, and population decline. For crops, breeding for climate-resilient flowering time is essential to maintain yield stability under variable environmental conditions [4, 5, 11].

Thus, the molecular orchestrators of flowering time are also ecological orchestrators. Their study bridges fundamental developmental biology with pressing challenges in biodiversity conservation and agricultural adaptation.

5. Conclusions and perspectives

The regulation of flowering time represents one of the most intricate and highly coordinated developmental decisions in the life cycle of flowering plants. As detailed throughout this chapter, this process is governed by a multilayered network of TFs that interpret and integrate diverse endogenous and environmental signals. From the MADS-box proteins that specify floral organ identity to the pathway-specific integrators such as CO, FT, and SOC1, and the repressors like FLC and TEM that prevent precocious flowering, each regulator contributes to a dynamic and robust system that ensures reproductive success under fluctuating conditions.

A recurring theme in flowering time control is the importance of PTMs, which provide rapid and reversible modulation of TF activity, stability, and subcellular

localization. Phosphorylation, ubiquitination, SUMOylation, and acetylation fine-tune the behavior of key regulators in response to light, temperature, and hormonal cues, enabling precise temporal control over floral transition. The discovery of PTM-mediated condensate formation, as seen in VEL proteins during vernalization, further highlights the emerging role of biomolecular phase separation in epigenetic memory and transcriptional regulation.

Looking forward, several frontiers promise to deepen our understanding of flowering time control. First, the application of single-cell genomics and spatial transcriptomics will enable researchers to dissect cellular heterogeneity and lineage-specific regulatory dynamics within the shoot apical meristem during floral transition. Second, advances in chromatin profiling and live-imaging technologies are poised to reveal the real-time dynamics of epigenetic silencing and nuclear organization, particularly at loci such as FLC. Third, comparative studies across diverse angiosperm lineages, ranging from perennials like poplar to crops like rice and Brassica, will continue to uncover lineage-specific innovations and conserved regulatory principles, informing both evolutionary biology and breeding strategies.

Finally, the integration of systems biology and synthetic biology approaches offers transformative potential for agriculture. By modeling flowering networks as predictive frameworks, it may become possible to engineer flowering time in crops to optimize yield under changing climatic conditions. The targeted manipulation of key TFs, their PTMs, or their interacting partners could enable precise control over flowering time, stress adaptation, and reproductive architecture.

In summary, the study of flowering time regulation has evolved from descriptive genetics to a mechanistically rich and systems-level discipline. The continued unraveling of its molecular orchestrators will not only illuminate fundamental principles of developmental biology but also empower innovative strategies for crop improvement and climate resilience.

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Conflict of Interest

The authors declare no conflict of interest.

Abbreviations

AG	AGAMOUS
AP1	APETALA1
AP2	APETALA2
AP3	APETALA3
ChIP-seq	chromatin immunoprecipitation coupled with high-throughput sequencing

CPSF	Cleavage and Polyadenylation Specificity Factor
CO	CONSTANS
COL	CONSTANS-like
COP1	CONSTITUTIVE PHOTOMORPHOGENIC 1
CDFs	CYCLING DOF FACTORs
Ehd4	Early heading date 4
FKF1	FLAVIN-BINDING KELCH REPEAT F-BOX 1
FLC	FLOWERING LOCUS C
FLD	FLOWERING LOCUS D
FLM	FLOWERING LOCUS M
FRI	FRIGIDA
FT	FLOWERING LOCUS T
FUL	FRUITFULL
GA	Gibberellin
GAI	GA INSENSITIVE
GID1	GIBBERELLIN INSENSITIVE DWARF1
GI	GIGANTEA
H3K4	histone H3 lysine 4
H3K27me3	H3 lysine 27 trimethylation
ICU2	INCURVATA2
LFY	LEAFY
LHP1	LIKE HETEROCHROMATIN PROTEIN 1
miR156	microRNA156
miRNAs	microRNAs
PKL	PICKLE
PI	PISTILLATA
PEBP	phosphatidylethanolamine-binding protein
phyA	phytochrome A
PIF4	PHYTOCHROME INTERACTING FACTOR 4
PIFs	PHYTOCHROME INTERACTING FACTORS
PRC2	Polycomb Repressive Complex 2
PTMs	post-translational modifications
QTL	quantitative trait loci
RGA	REPRESSOR OF GA1-3
RGL1	RGA-LIKE1
SNZ	SCHNARCHZAPFEN
SMZ	SCHLAFMUTZE
SEP	SEPALLATA
SVP	SHORT VEGETATIVE PHASE
SAUR	SMALL AUXIN UP RNA
SOC1	SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1
SPL	SQUAMOSA PROMOTER BINDING PROTEIN-LIKE
TOE1	TARGET OF EARLY ACTIVATION TAGGED 1
TEM	TEMPRANILLO
TFL1	TERMINAL FLOWER 1
TFs	transcription factors
TSF	TWIN SISTER OF FT
UFO	UNUSUAL FLORAL ORGANS
VIN3	VERNALIZATION INSENSITIVE 3

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References

- [1] Hong L, Niu F, Lin Y, Wang S, Chen L, Jiang L. MYB117 is a negative regulator of flowering time in *Arabidopsis*. *Plant Signaling and Behavior*. 2021;**16**(5):1901448. DOI: 10.1080/15592324.2021.1901448
- [2] Kawano S, Masuda J. The productive and reproductive biology of flowering plants: VII. resource allocation and reproductive capacity in wild populations of *Heloniopsis orientalis* (Thunb.) C. Tanaka (Liliaceae). *Oecologia*. 1980;**45**(3):307–317. DOI: 10.1007/BF00540197
- [3] Wang Y, Xu C, Sun J, Dong L, Li M, Liu Y, Wang J, Zhang X, Li D, Sun J, Zhang Y, Shan J, Li W, Zhao L. GmRAV confers ecological adaptation through photoperiod control of flowering time and maturity in soybean. *Plant Physiology*. 2021;**187**(1):361–377. DOI: 10.1093/plphys/kiab255
- [4] Michaels SD. Flowering time regulation produces much fruit. *Current Opinion in Plant Biology*. 2009;**12**(1):75–80. DOI: 10.1016/j.pbi.2008.09.005
- [5] Wang F, Liu S, Li H, Fang C, Fang S, Wang J, Li S, Liu H, Du H, Wang L, Pei X, Su B, Sun Z, Li Q, Dong L, Cheng Q, Zhao X, Liu B, Lu S, Kong F, Lin X. Artificial selection of two antagonistic E3 ubiquitin ligases finetunes soybean photoperiod adaptation and grain yield. *Proceedings of the National Academy of Sciences of the United States of America*. 2024;**121**(45):e2321473121. DOI: 10.1073/pnas.2321473121
- [6] Fang Y, Cui X, Mei Z, Zhang C, Guo L. Gibberellin and cytokinin are coordinately involved in regulating kiwifruit flowering. *Gene*. 2025;**972**:149817. DOI: 10.1016/j.gene.2025.149817
- [7] Ma M, Guo L, Jian C, Wu L, Zhang T, Li M, Wu B, Zhang Y, Peng H, Zhang K, Liu X, Li G, Jing R, Zhao H. Knockout of TaOTUB1 homoeologs in wheat promotes increased grain yield and earlier flowering. *The Plant Journal*. 2025;**123**(3):e70376. DOI: 10.1111/tpj.70376
- [8] Agustí M, Reig C, Martínez-Fuentes A, Mesejo C. Advances in Citrus flowering: A review. *Frontiers in Plant Science*. 2022;**13**:868831. DOI: 10.3389/fpls.2022.868831
- [9] Vollrath P, Chawla HS, Schiessl SV, Gabur I, Lee H, Snowdon RJ, Obermeier C. A novel deletion in FLOWERING LOCUS T modulates flowering time in winter oilseed rape. *Theoretical and Applied Genetics*. 2021;**134**(4):1217–1231. DOI: 10.1007/s00122-021-03768-4
- [10] Fournier-Level A, Taylor MA, Paril JF, Martínez-Berdeja A, Stitzer MC, Cooper MD, Roe JL, Wilczek AM, Schmitt J. Adaptive significance of flowering time variation across natural seasonal environments in *Arabidopsis thaliana*. *The New Phytologist*. 2022;**234**(2):719–734. DOI: 10.1111/nph.17999
- [11] Maple R, Zhu P, Hepworth J, Wang JW, Dean C. Flowering time: From physiology, through genetics to mechanism. *Plant Physiology*. 2024;**195**(1):190–212. DOI: 10.1093/plphys/kiac109

- [12] Roelfs KU, Känel A, Twyman RM, Prüfer D, Schulze Gronover C. Epigenetic variation in early and late flowering plants of the rubber-producing Russian dandelion *Taraxacum koksaghyz* provides insights into the regulation of flowering time. *Scientific Reports*. 2024;**14**(1):4283. DOI: 10.1038/s41598-024-54862-8
- [13] Cheng JZ, Zhou YP, Lv TX, Xie CP, Tian CE. Research progress on the autonomous flowering time pathway in *Arabidopsis*. *Physiology and Molecular Biology of Plants*. 2017;**23**(3):477–485. DOI: 10.1007/s12298-017-0458-3
- [14] Bao S, Hua C, Shen L, Yu H. New insights into gibberellin signaling in regulating flowering in *Arabidopsis*. *Journal of Integrative Plant Biology*. 2020;**62**(1):118–131. DOI: 10.1111/jipb.12892
- [15] Wang JW, Czech B, Weigel D. miR156-regulated SPL transcription factors define an endogenous flowering pathway in *Arabidopsis thaliana*. *Cell*. 2009;**138**(4):738–749. DOI: 10.1016/j.cell.2009.06.014
- [16] Takagi H, Hempton AK, Imaizumi T. Photoperiodic flowering in *Arabidopsis*: Multilayered regulatory mechanisms of CONSTANS and the florigen FLOWERING LOCUS T. *Plant Communications*. 2023;**4**(3):100552. DOI: 10.1016/j.xplc.2023.100552
- [17] He Y. Chromatin regulation of flowering. *Trends in Plant Science*. 2012;**17**(9):556–562. DOI: 10.1016/j.tplants.2012.05.001
- [18] Berry S, Dean C. Environmental perception and epigenetic memory: Mechanistic insight through FLC. *The Plant Journal*. 2015;**83**(1):133–148. DOI: 10.1111/tpj.12869
- [19] Kumar SV, Wigge PA. H2A.Z-containing nucleosomes mediate the thermosensory response in *Arabidopsis*. *Cell*. 2010;**140**(1):136–147. DOI: 10.1016/j.cell.2009.11.006
- [20] Jarillo JA, Piñeiro M. H2A.Z mediates different aspects of chromatin function and modulates flowering responses in *Arabidopsis*. *The Plant Journal*. 2015;**83**(1):96–109. DOI: 10.1111/tpj.12873
- [21] Mandel MA, Gustafson-Brown C, Savidge B, Yanofsky MF. Molecular characterization of the *Arabidopsis* floral homeotic gene APETALA1. *Nature*. 1992;**360**(6401):273–277. DOI: 10.1038/360273a0
- [22] Irish VF, Sussex IM. Function of the *apetala-1* gene during *Arabidopsis* floral development. *The Plant Cell*. 1990;**2**(8):741–753. DOI: 10.1105/tpc.2.8.741
- [23] Ferrándiz C, Gu Q, Martienssen R, Yanofsky MF. Redundant regulation of meristem identity and plant architecture by FRUITFULL, APETALA1 and CAULIFLOWER. *Development*. 2000;**127**(4):725–734. DOI: 10.1242/dev.127.4.725
- [24] Helliwell CA, Wood CC, Robertson M, James Peacock W, Dennis ES. The *Arabidopsis* FLC protein interacts directly in vivo with SOC1 and FT chromatin and is part of a high-molecular-weight protein complex. *The Plant Journal*. 2006;**46**(2):183–192. DOI: 10.1111/j.1365-313X.2006.02686.x
- [25] Deng W, Ying H, Helliwell CA, Taylor JM, Peacock WJ, Dennis ES. FLOWERING LOCUS C (FLC) regulates development pathways throughout the life cycle of *Arabidopsis*.

- Proceedings of the National Academy of Sciences of the United States of America. 2011;**108**(16):6680–6685. DOI: 10.1073/pnas.1103175108
- [26] Bastow R, Mylne JS, Lister C, Lippman Z, Martienssen RA, Dean C. Vernalization requires epigenetic silencing of FLC by histone methylation. *Nature*. 2004;**427**(6970):164–167. DOI: 10.1038/nature02269
- [27] Sung S, Amasino RM. Vernalization in *Arabidopsis thaliana* is mediated by the PHD finger protein VIN3. *Nature*. 2004;**427**(6970):159–164. DOI: 10.1038/nature02195
- [28] De Lucia F, Crevillen P, Jones AM, Greb T, Dean C. A PHD-polycomb repressive complex 2 triggers the epigenetic silencing of FLC during vernalization. *Proceedings of the National Academy of Sciences of the United States of America*. 2008;**105**(44):16831–16836. DOI: 10.1073/pnas.0808687105
- [29] Lee H, Suh SS, Park E, Cho E, Ahn JH, Kim SG, Lee JS, Kwon YM, Lee I. The AGAMOUS-LIKE 20 MADS domain protein integrates floral inductive pathways in *Arabidopsis*. *Genes and Development*. 2000;**14**(18):2366–2376. DOI: 10.1101/gad.813600
- [30] Hepworth SR, Valverde F, Ravenscroft D, Mouradov A, Coupland G. Antagonistic regulation of flowering-time gene SOC1 by CONSTANS and FLC via separate promoter motifs. *The EMBO Journal*. 2002;**21**(16):4327–4337. DOI: 10.1093/emboj/cdf432
- [31] Moon J, Suh SS, Lee H, Choi KR, Hong CB, Paek NC, Kim SG, Lee I. The SOC1 MADS-box gene integrates vernalization and gibberellin signals for flowering in *Arabidopsis*. *The Plant Journal*. 2003;**35**(5):613–623. DOI: 10.1046/j.1365-3113x.2003.01833.x
- [32] Liu C, Chen H, Er HL, Soo HM, Kumar PP, Han JH, Liou YC, Yu H. Direct interaction of AGL24 and SOC1 integrates flowering signals in *Arabidopsis*. *Development*. 2008;**135**(8):1481–1491. DOI: 10.1242/dev.020255
- [33] Immink RG, Posé D, Ferrario S, Ott F, Kaufmann K, Valentim FL, de Folter S, van der Wal F, van Dijk AD, Schmid M, Angenent GC. Characterization of SOC1's central role in flowering by the identification of its upstream and downstream regulators. *Plant Physiology*. 2012;**160**(1):433–449. DOI: 10.1104/pp.112.202614
- [34] Pelaz S, Ditta GS, Baumann E, Wisman E, Yanofsky MF. B and C floral organ identity functions require SEPALLATA MADS-box genes. *Nature*. 2000;**405**(6783):200–203. DOI: 10.1038/35012103
- [35] Ditta G, Pinyopich A, Robles P, Pelaz S, Yanofsky MF. The SEP4 gene of *Arabidopsis thaliana* functions in floral organ and meristem identity. *Current Biology*. 2004;**14**(21):1935–1940. DOI: 10.1016/j.cub.2004.10.028
- [36] Honma T, Goto K. Complexes of MADS-box proteins are sufficient to convert leaves into floral organs. *Nature*. 2001;**409**(6819):525–529. DOI: 10.1038/35054083
- [37] Mouradov A, Cremer F, Coupland G. Control of flowering time: Interacting pathways as a basis for

- diversity. *The Plant Cell*. 2002;**14** (Suppl(Suppl)):S111–S130. DOI: 10.1105/tpc.001362
- [38] Parcy F. Flowering: A time for integration. *The International Journal of Developmental Biology*. 2005;**49** (5-6):585–593. DOI: 10.1387/ijdb.041930fp
- [39] Corbesier L, Vincent C, Jang S, Fornara F, Fan Q, Searle I, Giakountis A, Farrona S, Gissot L, Turnbull C, Coupland G. FT protein movement contributes to long-distance signaling in floral induction of Arabidopsis. *Science*. 2007;**316**(5827):1030–1033. DOI: 10.1126/science.1141752
- [40] Jaeger KE, Wigge PA. FT protein acts as a long-range signal in Arabidopsis. *Current Biology*. 2007;**17** (12):1050–1054. DOI: 10.1016/j.cub.2007.05.008
- [41] Abe M, Kobayashi Y, Yamamoto S, Daimon Y, Yamaguchi A, Ikeda Y, Ichinoki H, Notaguchi M, Goto K, Araki T. FD, a bZIP protein mediating signals from the floral pathway integrator FT at the shoot apex. *Science*. 2005;**309**(5737):1052–1056. DOI: 10.1126/science.1115983
- [42] Wigge PA, Kim MC, Jaeger KE, Busch W, Schmid M, Lohmann JU, Weigel D. Integration of spatial and temporal information during floral induction in Arabidopsis. *Science*. 2005;**309**(5737):1056–1059. DOI: 10.1126/science.1114358
- [43] Taoka K, Ohki I, Tsuji H, Furuita K, Hayashi K, Yanase T, Yamaguchi M, Nakashima C, Purwestri YA, Tamaki S, Ogaki Y, Shimada C, Nakagawa A, Kojima C, Shimamoto K. 14-3-3 proteins act as intracellular receptors for rice Hd3a florigen. *Nature*. 2011;**476**(7360):332–335. DOI: 10.1038/nature10272
- [44] Putterill J, Robson F, Lee K, Simon R, Coupland G. The CONSTANS gene of Arabidopsis promotes flowering and encodes a protein showing similarities to zinc finger transcription factors. *Cell*. 1995;**80**(6):847–857. DOI: 10.1016/0092-8674(95)90288-0
- [45] Suárez-López P, Wheatley K, Robson F, Onouchi H, Valverde F, Coupland G. CONSTANS mediates between the circadian clock and the control of flowering in Arabidopsis. *Nature*. 2001;**410**(6832):1116–1120. DOI: 10.1038/35074138
- [46] Yanovsky MJ, Kay SA. Molecular basis of seasonal time measurement in Arabidopsis. *Nature*. 2002;**419** (6904):308–312. DOI: 10.1038/nature00996
- [47] Valverde F, Mouradov A, Soppe W, Ravenscroft D, Samach A, Coupland G. Photoreceptor regulation of CONSTANS protein in photoperiodic flowering. *Science*. 2004;**303** (5660):1003–1006. DOI: 10.1126/science.1091761
- [48] Jang S, Marchal V, Panigrahi KC, Wenkel S, Soppe W, Deng XW, Valverde F, Coupland G. Arabidopsis COP1 shapes the temporal pattern of CO accumulation conferring a photoperiodic flowering response. *The EMBO Journal*. 2008;**27**(8):1277–1288. DOI: 10.1038/emboj.2008.68
- [49] Andrés F, Coupland G. The genetic basis of flowering responses to seasonal cues. *Nature Reviews Genetics*. 2012;**13** (9):627–639. DOI: 10.1038/nrg3291
- [50] Adrian J, Farrona S, Reimer JJ, Albani MC, Coupland G, Turck F.

- cis-Regulatory elements and chromatin state coordinately control temporal and spatial expression of FLOWERING LOCUS T in Arabidopsis. *The Plant Cell*. 2010;**22**(5):1425–1440. DOI: 10.1105/tpc.110.074682
- [51] Gnesutta N, Kumimoto RW, Swain S, Chiara M, Siriwardana C, Horner DS, Holt 3rd BF, Mantovani R. CONSTANS imparts DNA sequence specificity to the histone fold NF-YB/NF-YC dimer. *The Plant Cell*. 2017;**29**(6):1516–1532. DOI: 10.1105/tpc.16.00864
- [52] Kumar SV, Lucyshyn D, Jaeger KE, Alós E, Alvey E, Harberd NP, Wigge PA. Transcription factor PIF4 controls the thermosensory activation of flowering. *Nature*. 2012;**484**(7393):242–245. DOI: 10.1038/nature10928
- [53] Wu G, Park MY, Conway SR, Wang JW, Weigel D, Poethig RS. The sequential action of miR156 and miR172 regulates developmental timing in Arabidopsis. *Cell*. 2009;**138**(4):750–759. DOI: 10.1016/j.cell.2009.06.031
- [54] Castillejo C, Pelaz S. The balance between CONSTANS and TEMPRANILLO activities determines FT expression to trigger flowering. *Current Biology*. 2008;**18**(17):1338–1343. DOI: 10.1016/j.cub.2008.07.075
- [55] Osnato M, Castillejo C, Matías-Hernández L, Pelaz S. TEMPRANILLO genes link photoperiod and gibberellin pathways to control flowering in Arabidopsis. *Nature Communications*. 2012;**3**:808. DOI: 10.1038/ncomms1810
- [56] He Y, Chen T, Zeng X. Genetic and epigenetic understanding of the seasonal timing of flowering. *Plant Communications*. 2019;**1**(1):100008. DOI: 10.1016/j.xplc.2019.100008
- [57] Franco-Echevarría E, Nielsen M, Schulten A, Cheema J, Morgan TE, Bienz M, Dean C. Distinct accessory roles of Arabidopsis VEL proteins in Polycomb silencing. *Genes and Development*. 2023;**37**(17-18):801–817. DOI: 10.1101/gad.350814.123
- [58] Fiedler M, Franco-Echevarría E, Schulten A, Nielsen M, Rutherford TJ, Yeates A, Ahsan B, Dean C, Bienz M. Head-to-tail polymerization by VEL proteins underpins cold-induced Polycomb silencing in flowering control. *Cell Reports*. 2022;**41**(6):111607. DOI: 10.1016/j.celrep.2022.111607
- [59] Qüesta JI, Song J, Geraldo N, An H, Dean C. Arabidopsis transcriptional repressor VAL1 triggers Polycomb silencing at FLC during vernalization. *Science*. 2016;**353**(6298):485–488. DOI: 10.1126/science.aaf7354
- [60] Becker A, Theissen G. The major clades of MADS-box genes and their role in the development and evolution of flowering plants. *Molecular Phylogenetics and Evolution*. 2003;**29**(3):464–489. DOI: 10.1016/s1055-7903(03)00207-0
- [61] Smaczniak C, Immink RG, Angenent GC, Kaufmann K. Developmental and evolutionary diversity of plant MADS-domain factors: Insights from recent studies. *Development*. 2012;**139**(17):3081–3098. DOI: 10.1242/dev.074674
- [62] Coen ES, Meyerowitz EM. The war of the whorls: Genetic interactions controlling flower development. *Nature*. 1991;**353**(6339):31–37. DOI: 10.1038/353031a0
- [63] Theissen G. Development of floral organ identity: Stories from the MADS

- house. *Current Opinion in Plant Biology*. 2001;**4**(1):75–85. DOI: 10.1016/S1369-5266(00)00139-4
- [64] Dennis L, Peacock J. Genes directing flower development in *Arabidopsis*. *The Plant Cell*. 2019;**31**(6):1192–1193. DOI: 10.1105/tpc.19.00276
- [65] Yanofsky MF, Ma H, Bowman JL, Drews GN, Feldmann KA, Meyerowitz EM. The protein encoded by the *Arabidopsis* homeotic gene *agamous* resembles transcription factors. *Nature*. 1990;**346**(6279):35–39. DOI: 10.1038/346035a0
- [66] Theissen G, Saedler H. Plant biology. Floral quartets. *Nature*. 2001;**409**(6819):469–471. DOI: 10.1038/35054172
- [67] Melzer R, Verelst W, Theissen G. The class E floral homeotic protein *SEPALLATA3* is sufficient to loop DNA in ‘floral quartet’-like complexes in vitro. *Nucleic Acids Research*. 2009;**37**(1):144–157. DOI: 10.1093/nar/gkn900
- [68] Winter CM, Austin RS, Blanvillain-Baufumé S, Reback MA, Monniaux M, Wu MF, Sang Y, Yamaguchi A, Yamaguchi N, Parker JE, Parcy F, Jensen ST, Li H, Wagner D. *LEAFY* target genes reveal floral regulatory logic, cis motifs, and a link to biotic stimulus response. *Developmental Cell*. 2011;**20**(4):430–443. DOI: 10.1016/j.devcel.2011.03.019
- [69] Kaufmann K, Muiño JM, Jauregui R, Airoidi CA, Smaczniak C, Krajewski P, Angenent GC. Target genes of the MADS transcription factor *SEPALLATA3*: Integration of developmental and hormonal pathways in the *Arabidopsis* flower. *PLoS Biology*. 2009;**7**(4):e1000090. DOI: 10.1371/journal.pbio.1000090
- [70] Wuest SE, O’Maoileidigh DS, Rae L, Kwasniewska K, Raganelli A, Hanczaryk K, Lohan AJ, Loftus B, Graciet E, Wellmer F. Molecular basis for the specification of floral organs by *APETALA3* and *PISTILLATA*. *Proceedings of the National Academy of Sciences of the United States of America*. 2012;**109**(33):13452–13457. DOI: 10.1073/pnas.1207075109
- [71] Kojima S, Takahashi Y, Kobayashi Y, Monna L, Sasaki T, Araki T, Yano M. *Hd3a*, a rice ortholog of the *Arabidopsis* *FT* gene, promotes transition to flowering downstream of *Hd1* under short-day conditions. *Plant and Cell Physiology*. 2002;**43**(10):1096–1105. DOI: 10.1093/pcp/pcf156
- [72] Hsu CY, Liu Y, Luthe DS, Yuceer C. *Poplar* *FT2* shortens the juvenile phase and promotes seasonal flowering. *The Plant Cell*. 2006;**18**(8):1846–1861. DOI: 10.1105/tpc.106.041038
- [73] Weigel D, Alvarez J, Smyth DR, Yanofsky MF, Meyerowitz EM. *LEAFY* controls floral meristem identity in *Arabidopsis*. *Cell*. 1992;**69**(5):843–859. DOI: 10.1016/0092-8674(92)90295-n
- [74] Blazquez MA, Green R, Nilsson O, Sussman MR, Weigel D. Gibberellins promote flowering of *arabidopsis* by activating the *LEAFY* promoter. *The Plant Cell*. 1998;**10**(5):791–800. DOI: 10.1105/tpc.10.5.791
- [75] Liljegren SJ, Gustafson-Brown C, Pinyopich A, Ditta GS, Yanofsky MF. Interactions among *APETALA1*, *LEAFY*, and *TERMINAL FLOWER1* specify meristem fate. *The Plant Cell*. 1999;**11**(6):1007–1018. DOI: 10.1105/tpc.11.6.1007

- [76] Liu C, Teo ZW, Bi Y, Song S, Xi W, Yang X, Yin Z, Yu H. A conserved genetic pathway determines inflorescence architecture in Arabidopsis and rice. *Developmental Cell*. 2013;**24**(6):612–622. DOI: 10.1016/j.devcel.2013.02.013
- [77] Parcy F, Nilsson O, Busch MA, Lee I, Weigel D. A genetic framework for floral patterning. *Nature*. 1998;**395** (6702):561–566. DOI: 10.1038/26903
- [78] Moyroud E, Minguet EG, Ott F, Yant L, Posé D, Monniaux M, Blanchet S, Bastien O, Thévenon E, Weigel D, Schmid M, Parcy F. Prediction of regulatory interactions from genome sequences using a biophysical model for the Arabidopsis LEAFY transcription factor. *The Plant Cell*. 2011;**23**(4):1293–1306. DOI: 10.1105/tpc.111.083329
- [79] Chae E, Tan QK, Hill TA, Irish VF. An Arabidopsis F-box protein acts as a transcriptional co-factor to regulate floral development. *Development*. 2008;**135**(7):1235–1245. DOI: 10.1242/dev.015842
- [80] Liu F, Marquardt S, Lister C, Swiezewski S, Dean C. Targeted 3' processing of antisense transcripts triggers Arabidopsis FLC chromatin silencing. *Science*. 2010;**327**(5961):94–97. DOI: 10.1126/science.1180278
- [81] Marquardt S, Raitskin O, Wu Z, Liu F, Sun Q, Dean C. Functional consequences of splicing of the antisense transcript COOLAIR on FLC transcription. *Molecular Cell*. 2014;**54** (1):156–165. DOI: 10.1016/j.molcel.2014.03.026
- [82] Angel A, Song J, Dean C, Howard M. A Polycomb-based switch underlying quantitative epigenetic memory. *Nature*. 2011;**476**(7358):105–108. DOI: 10.1038/nature10241
- [83] Coustham V, Li P, Strange A, Lister C, Song J, Dean C. Quantitative modulation of polycomb silencing underlies natural variation in vernalization. *Science*. 2012;**337** (6094):584–587. DOI: 10.1126/science.1221881
- [84] Stamm P, Kumar PP. Auxin and gibberellin responsive Arabidopsis SMALL AUXIN UP RNA36 regulates hypocotyl elongation in the light. *Plant Cell Reports*. 2013;**32**(6):759–769. DOI: 10.1007/s00299-013-1406-5
- [85] Song YH, Shim JS, Kinmonth-Schultz HA, Imaizumi T. Photoperiodic flowering: Time measurement mechanisms in leaves. *Annual Review of Plant Biology*. 2015;**66**:441–464. DOI: 10.1146/annurev-arplant-043014-115555
- [86] Shim JS, Kubota A, Imaizumi T. Circadian clock and photoperiodic flowering in Arabidopsis: CONSTANS is a hub for signal integration. *Plant Physiology*. 2017;**173**(1):5–15. DOI: 10.1104/pp.16.01327
- [87] Lian H, Xu P, He S, Wu J, Pan J, Wang W, Xu F, Wang S, Pan J, Huang J, Yang HQ. Photoexcited CRYPTOCHROME 1 interacts directly with G-Protein β subunit AGB1 to regulate the DNA-binding activity of HY5 and photomorphogenesis in Arabidopsis. *Molecular Plant*. 2018;**11** (10):1248–1263. DOI: 10.1016/j.molp.2018.08.004
- [88] Goraloglia GS, Liu TK, Zhao L, Panipinto PM, Groover ED, Bains YS, Imaizumi T. CYCLING DOF FACTOR 1 represses transcription through the TOPLESS co-repressor to control

- photoperiodic flowering in Arabidopsis. *The Plant Journal*. 2017;**92**(2):244–262. DOI: 10.1111/tpj.13649
- [89] Zhang B, Feng M, Zhang J, Song Z. Involvement of CONSTANS-like proteins in plant flowering and abiotic stress response. *International Journal of Molecular Sciences*. 2023;**24**(23):16585. DOI: 10.3390/ijms242316585
- [90] Wang MJ, Ding L, Liu XH, Liu JX. Two B-box domain proteins, BBX28 and BBX29, regulate flowering time at low ambient temperature in Arabidopsis. *Plant Molecular Biology*. 2021;**106**(1-2):21–32. DOI: 10.1007/s11103-021-01123-1
- [91] Böhlenius H, Huang T, Charbonnel-Campaa L, Brunner AM, Jansson S, Strauss SH, Nilsson O. CO/FT regulatory module controls timing of flowering and seasonal growth cessation in trees. *Science*. 2006;**312**(5776):1040–1043. DOI: 10.1126/science.1126038
- [92] Hsu CY, Adams JP, Kim H, No K, Ma C, Strauss SH, Drnevich J, Vandervelde L, Ellis JD, Rice BM, Wickett N, Gunter LE, Tuskan GA, Brunner AM, Page GP, Barakat A, Carlson JE, DePamphilis CW, Luthe DS, Yuceer C. FLOWERING LOCUS T duplication coordinates reproductive and vegetative growth in perennial poplar. *Proceedings of the National Academy of Sciences of the United States of America*. 2011;**108**(26):10756–10761. DOI: 10.1073/pnas.1104713108
- [93] Parmentier-Line CM, Coleman GD. Constitutive expression of the Poplar FD-like basic leucine zipper transcription factor alters growth and bud development. *Plant Biotechnology Journal*. 2016;**14**(1):260–270. DOI: 10.1111/pbi.12380
- [94] Azeez A, Miskolczi P, Tylewicz S, Bhalerao RP. A tree ortholog of APETALA1 mediates photoperiodic control of seasonal growth. *Current Biology*. 2014;**24**(7):717–724. DOI: 10.1016/j.cub.2014.02.037
- [95] Gao H, Zheng XM, Fei G, Chen J, Jin M, Ren Y, Wu W, Zhou K, Sheng P, Zhou F, Jiang L, Wang J, Zhang X, Guo X, Wang JL, Cheng Z, Wu C, Wang H, Wan JM. Ehd4 encodes a novel and Oryza-genus-specific regulator of photoperiodic flowering in rice. *PLoS Genetics*. 2013;**9**(2):e1003281. DOI: 10.1371/journal.pgen.1003281
- [96] Fan H, Zhuo R, Wang H, Xu J, Jin K, Huang B, Qiao G. A comprehensive analysis of the floral transition in ma bamboo (*Dendrocalamus latiflorus*) reveals the roles of DFTs involved in flowering. *Tree Physiology*. 2022;**42**(9):1899–1911. DOI: 10.1093/treephys/tpac035
- [97] González-Schain ND, Díaz-Mendoza M, Zurczak M, Suárez-López P. Potato CONSTANS is involved in photoperiodic tuberization in a graft-transmissible manner. *The Plant Journal*. 2012;**70**(4):678–690. DOI: 10.1111/j.1365-313X.2012.04909.x
- [98] Sashidhar N, Coupland G. In perennial *Arabis alpina*, CONSTANS and FLOWERING LOCUS T have common and distinct effects on flowering and inflorescence architecture. *The New Phytologist*. 2026;**250**:970–987. DOI: 10.1111/nph.70994
- [99] Zhang R, Ding J, Liu C, Cai C, Zhou B, Zhang T, Guo W. Molecular evolution and phylogenetic analysis of eight COL superfamily genes in group I related to photoperiodic regulation of flowering time in wild and domesticated

- cotton (*Gossypium*) species. *PloS One*. 2015;**10**(2):e0118669. DOI: 10.1371/journal.pone.0118669
- [100] Zhu H, Liu J, Kuang L, Meng J, Zhu J, Wang Y, Jiang J. Genetic and molecular regulation of flowering time in *Brassica napus* L. *Plant Science*. 2026;**364**:112963. DOI: 10.1016/j.plantsci.2025.112963
- [101] Xie J, Zhu J, Wang L, Jiao Y, Dou Y, Zhao Z, Luo B, Ha M, Zheng H. Potential regulatory modules to integrate microgravity signals into flowering pathways in *Arabidopsis thaliana* grown in space. *NPJ Microgravity*. 2025;**11**(1):87. DOI: 10.1038/s41526-025-00539-z
- [102] He Y, Michaels SD, Amasino RM. Regulation of flowering time by histone acetylation in *Arabidopsis*. *Science*. 2003;**302**(5651):1751–1754. DOI: 10.1126/science.1091109
- [103] Menon G, Schulten A, Dean C, Howard M. Digital paradigm for Polycomb epigenetic switching and memory. *Current Opinion in Plant Biology*. 2021;**61**:102012. DOI: 10.1016/j.pbi.2021.102012
- [104] Menon G, Mateo-Bonmati E, Reeck S, Maple R, Wu Z, Ietswaart R, Dean C, Howard M. Proximal termination generates a transcriptional state that determines the rate of establishment of Polycomb silencing. *Molecular Cell*. 2024;**84**(12):2255–2271.e9. DOI: 10.1016/j.molcel.2024.05.014
- [105] Zhu P, Lister C, Dean C. Cold-induced *Arabidopsis* FRIGIDA nuclear condensates for FLC repression. *Nature*. 2021;**599**(7886):657–661. DOI: 10.1038/s41586-021-04062-5
- [106] Zhu D, Rosa S, Dean C. Nuclear organization changes and the epigenetic silencing of FLC during vernalization. *Journal of Molecular Biology*. 2015;**427**(3):659–669. DOI: 10.1016/j.jmb.2014.08.025
- [107] Rosa S, De Lucia F, Mylne JS, Zhu D, Ohmido N, Pendle A, Kato N, Shaw P, Dean C. Physical clustering of FLC alleles during Polycomb-mediated epigenetic silencing in vernalization. *Genes and Development*. 2013;**27**(17):1845–1850. DOI: 10.1101/gad.221713.113
- [108] Antoniou-Kourounioli RL, Meschichi A, Reeck S, Berry S, Menon G, Zhao Y, Fozard J, Holmes T, Zhao L, Wang H, Hartley M, Dean C, Rosa S, Howard M. Integrating analog and digital modes of gene expression at *Arabidopsis* FLC. *eLife*. 2023;**12**(e79743). DOI: 10.7554/eLife.79743
- [109] Qüesta JI, Antoniou-Kourounioli RL, Rosa S, Li P, Duncan S, Whittaker C, Howard M, Dean C. Noncoding SNPs influence a distinct phase of Polycomb silencing to destabilize long-term epigenetic memory at *Arabidopsis* FLC. *Genes and Development*. 2020;**34**(5-6):446–461. DOI: 10.1101/gad.333245.119
- [110] Hyun Y, Yun H, Park K, Ohr H, Lee O, Kim DH, Sung S, Choi Y. The catalytic subunit of *Arabidopsis* DNA polymerase α ensures stable maintenance of histone modification. *Development*. 2013;**140**(1):156–166. DOI: 10.1242/dev.084624
- [111] Wang S, Luo C, Sun L, Ning K, Chen Z, Yang J, Wang Y, Wang Q. LsRGL1 controls the bolting and flowering times of lettuce by modulating the gibberellin pathway.

Plant Science. 2022;**316**:111175. DOI: 10.1016/j.plantsci.2021.111175

e106. DOI: 10.1371/journal.pgen.0020106

[112] Simpson GG. The autonomous pathway: Epigenetic and post-transcriptional gene regulation in the control of Arabidopsis flowering time. *Current Opinion in Plant Biology*. 2004;**7**(5):570–574. DOI: 10.1016/j.pbi.2004.07.002

[118] Posé D, Verhage L, Ott F, Yant L, Mathieu J, Angenent GC, Immink RG, Schmid M. Temperature-dependent regulation of flowering by antagonistic FLM variants. *Nature*. 2013;**503** (7476):414–417. DOI: 10.1038/nature12633

[113] Kyung J, Jeon M, Lee I. Recent advances in the chromatin-based mechanism of FLOWERING LOCUS C repression through autonomous pathway genes. *Frontiers in Plant Science*. 2022;**13**:964931. DOI: 10.3389/fpls.2022.964931

[119] Lee H, Yoo SJ, Lee JH, Kim W, Yoo SK, Fitzgerald H, Carrington JC, Ahn JH. Genetic framework for flowering-time regulation by ambient temperature-responsive miRNAs in Arabidopsis. *Nucleic Acids Research*. 2010;**38**(9):3081–3093. DOI: 10.1093/nar/gkp1240

[114] Mateo-Bonmatí E, Montez M, Maple R, Fiedler M, Fang X, Saalbach G, Passmore LA, Dean C. A CPF-like phosphatase module links transcription termination to chromatin silencing. *Molecular Cell*. 2024;**84**(12):2272–2286. e7. DOI: 10.1016/j.molcel.2024.05.016

[120] Kim JJ, Lee JH, Kim W, Jung HS, Huijser P, Ahn JH. The microRNA156-SQUAMOSA PROMOTER BINDING PROTEIN-LIKE3 module regulates ambient temperature-responsive flowering via FLOWERING LOCUS T in Arabidopsis. *Plant Physiology*. 2012;**159** (1):461–478. DOI: 10.1104/pp.111.192369

[115] Li Y, Zhang D, Wang J, Yang M, Yin Z, Zhu K, Liang Y, Tan X. Epigenetic regulation of floral transition. *Plants*. 2025;**14**(22):3471. DOI: 10.3390/plants14223471

[116] Sang Q, Vayssières A, Ó'Maoiléidigh DS, Yang X, Vincent C, Bertran Garcia de Olalla E, Cerise M, Franzen R, Coupland G. MicroRNA172 controls inflorescence meristem size through regulation of APETALA2 in Arabidopsis. *The New Phytologist*. 2022;**235**(1):356–371. DOI: 10.1111/nph.18111

[117] Balasubramanian S, Sureshkumar S, Lempe J, Weigel D. Potent induction of Arabidopsis thaliana flowering by elevated growth temperature. *PLoS Genetics*. 2006;**2**(7):