Common mechanisms regulate flowering and dormancy

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1. Introduction

In most temperate perennial plants, light and temperature regulate flowering and dormancy. Based on this simple observation, several groups have hypothesized that similar mechanisms regulate both processes [1–3]. Likewise, several excellent reviews have directly or indirectly discussed possible mechanisms that relate to this hypothesis [4–6]. This review will discuss recent findings on the regulation of bud dormancy and flowering that are beginning to provide mechanistic support of this hypothesis.

1.1. Brief overview of floral regulation

Due to its importance in plant reproduction, extensive research has been conducted which has identified many of the environ-
mental controls and genes involved in regulating flowering. Indeed there are numerous in-depth reviews on this process in both annual dicot and monocot plants [7–11], as well as in perennial trees [12,13]. Flowering occurs when meristems receive developmental and or environmental signals that cause the meristem to develop into flowers. These meristems may originally be predestined to flower upon growth, or they may initially be actively growing vegetative meristems that transition to floral meristems. In either case, induction of two key genes appear to initiate a cascade of events that alters the development of organ primordia within the meristem so that sepals, petals, pistils, and stamen are produced rather than leaves and maintenance of an undifferentiated core of cells at the center of the meristem. In the well-studied systems of rice (Oryza sativa), poplar (Populus spp.), Citrus ssp., and arabidopsis (Arabidopsis thaliana), very similar genes and signaling networks appear to regulate flowering although arguably most of the research has been done on the winter annual arabidopsis, and thus unless noted otherwise, generalizations will refer to floral regulation in this plant.

There are many genes and signals that regulate flowering, most of which converge on FLOWERING LOCUS T (FT) (Fig. 1). FT has been touted as an essential component of the graft transmissible florigen whose existence was long hypothesized [8]. FT is mostly expressed in mature leaf tissue in response to floral promoting environmental conditions; however there is evidence for its expression in young leaves in the shoot apices, and in dormant bud tissue [14,15]. Leaf expressed FT is known to be phloem-transmissible and is transported to the meristem where it initiates floral morphogenesis.

The genes that initiate the developmental cascade towards flowering are APETELA1 (AP1) and LEAFY (LFY) [16,17]. AP1 is directly induced by FT [18], and LFY is directly induced by SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1 (SOC1) [19]. In turn, both FT and SOC1 are positively regulated by CONSTANS (CO) [20], and both are negatively regulated by the MADS-box transcription factor FLOWERING LOCUS C (FLC) [21–23]. CO is in turn regulated by light through the various genes encoding components that make up the circadian clock and by PHYTOCHROME A (PHYA) [24].

Environmental and developmental signals that include aspects of chromatin remodeling and response to extended cold temperatures needed for vernalization (the process through which seeds and sometimes buds “remember” winter conditions and become competent to flower in the following spring) also regulate FT [25,26]. FT expression is also suppressed by another MADS-box transcription factor called SHORT VEGETATIVE PHASE (SVP) [27]. Like FLC, SVP binds to various regulatory sequences within the FT gene and inhibits its expression. However, SVP is primarily involved in ambient temperature regulation of FT [27] whereas FLC plays a more prominent role in the vernalization response.

With the exception of FLC, all plants appear to have functional homologues to these floral regulatory genes. In the two best characterized perennial model species, poplar and leafy spurge, there are homologues to genes related to FLC, specifically, MADS AFFECTING FLOWERING 2 (MAF2). It is also noteworthy that the FT gene family is expanded in perennials such as poplar [28]. However, there are differences such as altered responses of FT to CO in short day flowering rice relative to long day flowering
This suggests that although similar genes may be involved in flowering in long day and short day plants, the precise environmental regulation and timing of the interaction among these regulatory components may be different between species.

1.2. Brief overview of bud dormancy

Bud dormancy in temperate perennials is a well-studied phenomenon at the physiological level [1,4,30], but the molecular and genetic components of the signaling networks regulating dormancy are as yet poorly described (relative to flowering). Bud dormancy is complicated by the fact that buds may fail to grow due to a number of interacting developmental and physiological processes, and that buds of some perennials may be dormant at formation, while others may be actively growing and then transition to a dormant state. There also appears to be differences in dormancy status and responses depending on where the buds are located on the plant, and if the buds are floral or vegetative.

Precise definitions of dormancy processes such as those described by Lang can mitigate these complicating factors to some extent [31] (Fig. 2). In this system, dormancy states could be separated into paradormancy by which buds are prevented from growing due to signals produced in distal parts of the plant. In most cases, auxin and other signals regulate paradormancy. Paradormancy has also been described as correlative inhibition or apical dominance. There are several excellent reviews of the signals regulating this type of dormancy [32,33]. Buds may also be characterized as ecodormant. Harsh environmental conditions prevent bud growth during ecodormancy. For example, buds may cease growth during periods of drought, low temperatures, or in some cases short day lengths. However, ecodormant buds will grow immediately upon resumption of growth-conducive conditions. In the early autumn in most temperate climates, the buds of many perennial plants will become endodormant. Endodormant buds will have greatly delayed and often reduced growth rates relative to non-dormant buds when the plant is placed in growth-conducive conditions.

Short day lengths promote endodormancy in plants such as poplar while in others such as apples and leafy spurge, short periods of cold temperature induce endodormancy, while yet others including dogwood, response to cold and light is ecotype dependent [34–37]. It usually requires an extended cold or drought treatment, akin to vernalization, to break endodormancy and reinstate growth-competency to the buds. The similarities between the environmental signals regulating endodormancy induction and release and those regulating flowering and vernalization were the first clues that signaling mechanisms might be shared between these processes [2]. We are only beginning to identify genes and molecular signaling processes regulating endodormancy induction and release. So far, evidence exists for only two gene families in altering endodormancy. One set of genes include FT and a closely related gene named CENTRORADIALIS (CEN) for which direct or indirect over-expression in poplar was associated with failure of buds to enter endodormancy following dormancy-inducing short day conditions [15,38]. The other set of genes have been collectively named DORMANCY ASSOCIATED MADS-BOX (DAM) genes. DAM genes comprise a small gene family in poplar and deletion of a locus containing six DAM genes in peach produces trees which have terminal buds that are incapable of going into endodormancy under short day conditions [39,40]. DAM genes have also been cloned and expression analysis has linked them to endodormancy induction and release in peach, poplar, apricot, raspberry, and leafy spurge [14,34,41–43].

2. Environmental regulations of endodormancy and flowering

2.1. Circadian regulation of endodormancy and flowering, a likely connection

Because perception of day length appears to play a role in both flowering and endodormancy induction in some plants, the genes and processes responsible for perceiving and disseminating these day length signals likely control both these developmental processes. This system has been dubbed the “circadian clock,” and both light and temperature influence the timing and impact of circadian clock gene expression. Many of the circadian clock genes were discovered due to their impact on flower time regulation. Indeed, virtually every circadian clock regulator has been shown to influence flowering time in arabidopsis. The names of such key clock regulatory genes such as EARLY FLOWERING 3 (ELF3) and EARLY FLOWERING 4 (ELF4) underscore this connection. As there are numerous and excellent reviews on circadian regulation, and
many focus on the impact of circadian responses on flowering 
[24,44,45], it will not be covered here save to list the genes 
involved and denote their connection to flowering and dormancy 
processes (Supplemental Table 1). Far less is known about the 
precise role that the circadian clock plays in endodormancy; 
however numerous observations suggest a strong connection.

In chestnut, circadian responses appear to cease functioning 
during winter dormancy [46,47]. Interestingly, in chestnut, key 
circadian regulatory genes appear to be turned on constitutively in 
endodormant buds and in cold treated seedlings. A similar 
phenomenon was observed in the cycling of PHYA and PHYB in 
grape leaves experiencing dormancy-inducing day lengths [14,48]. 
In these experiments, PHYA and PHYB levels oscillated as would be 
expected during the normal growing season. However, when the 
plants were shifted to short day dormancy-inducing conditions, 
both PHYA and PHYB ceased oscillating and instead were 
constitutively expressed at elevated levels. Likewise, comparisons of 
transcriptiome profiles between various dormancy states in leafy 
spurge (Euphorbia esula) identified genes of several circadian 
regulators such as ELF4, GIANTEA (GI), FLAVIN-BINDING, KELCH 
REPEAT, F BOX 1 (FKF1) and CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) 
as significantly up-regulated during endo and ecodormancy [14]. 
Expression patterns of these genes, as shown in genevestigator in 
arabidopsis [49], indicate that all are either somewhat or strongly 
induced by cold temperatures. This observation suggests con-
servation in this signaling process.

These results appear to suggest that constitutive over-expres-
sion of circadian clock regulators are caused by endodormancy-
inducing conditions. However, it is also possible that the 
environmental factors that cause endodormancy might act 
through alterations in the expression of circadian response genes. 
To ascertain the importance of these observations experiments are 
needed that either maintains circadian cycling under dormancy-
inducing conditions or causes constitutive over-expression of 
these genes under normal growing conditions.

The observation that over-expression of PHYA leads to over-
expression of CO and prevents endodormancy appears to contra-
dict the hypothesis that over-expression of circadian response 
genes results in endodormancy. However, it is noteworthy that 
there are numerous CO-like genes in most perennials, and that in 
leafy spurge some appeared to be generally up-regulated in 
response to endodormancy induction and others were down-
regulated. A similar observation has been made in grape [50]. 
These observations indicate more complex interactions between 
circadian clock regulators and other environmental signals that 
control expression of transcriptional regulators such as CO and CO-
like genes and subsequently other downstream processes that 
might be related to endodormancy induction, and possibly vice 
versa.

2.2. Day length regulation of flowering and dormancy: the PHYA connection

Day length obviously helps entrain the regulators of the 
circadian clock, but day length also appears to more directly alter 
flowering time through the action of the red light photoreceptor 
PHYA. Alterations in PHYA expression disrupt the circadian clock 
under low light intensities [51] and inhibit the perception of 
flower-inducing long day conditions in arabidopsis [52]. The 
impact of PHYA on flowering is likely due, at least in part, because 
PHYA, along with GI directly regulates the expression of CO [53], 
and CO induces FT which leads to floral induction. Short day 
inhibits flowering in arabidopsis. SVP and FLC also regulate FT 
expression [27] and analyses of publicly available microarray 
results indicate that short day length also induce SVP in 
arabidopsis, however, no obvious PHYA regulation of SVP has 
been reported. Cold-regulated expression of FLC is well established 
and there is evidence that expression of many cold-regulated genes 
has been linked to PHYA regulation. This would be consistent with 
a link between light- and cold-regulated expression of these floral 
regulators (see below).

Short day lengths induce endodormancy induction in poplar,- 
certain grape and dogwood varieties (Svendsen [37], Olsen [55], 
Wake and Fennell [56]). Treatments that alter day length 
perception also alter dormancy responses in poplar and related 
trees [54,55]. In poplar, alterations in the expression of PHYA 
significantly alter the day length thresholds required for endo-
dormancy induction with over-expression of PHYA inhibiting 
dormancy induction under shorter day lengths, and under-
expression of PHYA resulting in enhanced dormancy induction 
under longer day lengths. Interestingly, over-expression of PHYA 
expression also prevented the reduction of gibberellic acid levels 
and cold hardening in response to short day length in hybrid aspen 
[55].

Over-expression of PHYA produces poplar trees that over-
express CO [38]. As observed in arabidopsis, over-expression of CO 
also led to over-expression of FT in poplar. An attempt to alter 
flowering time in poplar by directly over-expressing FT not only 
produced poplar that flowered earlier than wild type [57], but also 
resulted in poplar that did not respond to threshold short day 
lengths with endodormancy induction [38]. Although these studies 
could only relate over-expression of FT to inhibited growth 
cessation in response to short day, further studies associated 
over-expression of several FT orthologues and the related gene 
from poplar named CENTRORADIALIS-LIKE 1 (CENL1) with 
dormancy per se [15]. These later studies indicated that PHYA over-
expression, specifically in the rib meristem (RM) and upper rib 
zone (RZ) at the base of the meristem (but not in the shoot apical 
meristem), impacts bud dormancy and prevents short day-induced 
blockage of the plasmodesma. CENL1 is also strongly down-
regulated in the RM/RZ of wild type plants after 3 weeks in short 
day, but CENL1 is not down-regulated in PHYA over-expressing 
lines. Expression of another related gene, FT2, initially drops in 
these source leaves in response to short day in both wild type and 
PHYA over-expressing lines, but recovers only in the lines over-
expressing PHYA. Consequently, FT2 may also be regulated by short 
day through both a slower PHYA-dependent and a faster PHYA-
independent pathway. Reduction in FT-like genes preceding 
dormancy is not unique to poplar. FT-like genes are also 
down-regulated in underground crown buds of leafy spurge 
concomitantly with endodormancy induction [14].

Day length regulation of FT through the perception and action 
of PHYA is not the only mechanism by which dormancy is regulated. 
DAM genes are closely related to SVP, and thus it is conceivable that 
they might at least partially induce endodormancy induction by 
-binding to and inhibiting FT expression in perennials much like SVP 
does in arabidopsis. Experiments to test this hypothesis are still 
underway. In any case, DAM genes very likely play a role in 
dormancy induction. Transcriptome analysis of poplar during 
dormancy induction indicated that at least one DAM gene was 
up-regulated by short day conditions [34]. Indeed, an analysis of 
the promoter of several DAM genes from poplar and leafy spurge 
indicated that they contained EVENING elements which suggest 
they are regulated by components of the circadian response 
pathways [14].

2.3. Temperature regulation of flowering and dormancy

Cold temperatures effect many physiological processes, among 
them both flowering and dormancy induction. The later processes 
by which plants perceive cold to alter gene expression is 
moderately well understood. Many of the gene expression
differences caused by cold temperatures are dependent on the action of a MYC-like transcription factor known as INDUCER OF CBF EXPRESSION 1 (ICE1) [58]. Among the genes that are induced by ICE1 is another small family of API-like transcription factors named C-REPEAT BINDING FACTOR (CBF) which are also known as DEHYDRATION-RESPONSIVE ELEMENT-BINDING PROTEINS (DREB). The proteins encoded by these genes induce a large number of other cold-responsive genes [59].

The literature abounds with reports of altered flowering time in response to temperature. The cross-talk between light and temperature signaling may provide possible mechanisms for this phenomenon, particularly as they affect key floral regulators such as FLC, CO, and FT [56,60]. There are indications that light responses transmitted through phytochromes alter temperature responses [61–63]. There is also a report linking temperature responses directly to the action of PHYB in regulating flowering time through FT [62]. Although the authors of this study discount a role for CO or FLC in this response, FLC levels did increase in response to low temperatures in wild type plants and this might have accounted for the slight down regulation of FT in wild type lines. Likewise, only CO expression levels were measured, and much of the activity of CO is controlled at the post-translational level. Thus, temperature levels could have altered CO stability resulting in the decreased FT expression. It is equally possible that another FLc-like proteins might have altered FT expression. For example, misexpression of MAF2 expression using the genevestigator response viewer tool, shows it is up-regulated by cold acclimation, and it is not down-regulated by extended cold treatments as FLC is [64].

In a particularly interesting related study, cryptochrome2 (cry2) mutants significantly altered flowering time at low temperatures. In this case, the results suggested that the impact of cry2 was at least partially due to reduced PHYA level/activity at low temperature [65]. This would be consistent with reduced PHYA levels resulting in reduced FT expression during autumn. However, in leafy spurge [14] and in hybrid aspen [55], PHYA levels increased in buds transitioning into endodormancy.

FCA and FVE encode well-known thermo-responsive regulators of flowering as part of the autonomous pathway. These genes act to repress FLC expression in response to increased temperatures [66]. However, experiments that studied the impact of low temperatures on FT expression in fca and fve mutants indicated that these genes may also have an FLC-independent role in suppression of FT in low temperatures [65]. Indeed, SVP was shown to mediate the ambient temperature regulation of flowering that is controlled by FCA/FVE [27]. Also, this study did not look for any impact on MAF genes that might also be affected by FCA and FVE. The fact that MAF1 is likely responsible for reducing flowering time under elevated temperatures underscores this possibility [67].

In addition to temperature modifying light-regulated responses of known floral regulators and vice versa, there is evidence that some cold-responsive genes may directly play a role in flowering. Indeed, one member of the CBF gene family, DWARF AND DELAYED FLOWERING 1 (DDF1) was identified as a mutation that affected flowering time through repression of GA synthesis [68]. Light and circadian responses directly and indirectly impact expression of this several members of this gene family [63,69].

Not all perennial species use day lengths as the signal regulating endodormancy induction. Leafy spurge, apples, and some ecotypes of dogwood are known to primarily rely on cold temperature as the endodormancy-inducing signal [35–37]. Again the mechanisms regulating this process are not known. However, promoter analysis of a particular leafy spurge DAM gene indicated that it contained likely CBF binding sites suggesting it is induced by cold temperatures [14]. No CBF binding sites were found in the promoters of the poplar genes which are consistent with their limited induction by cold. Leafy spurge DAM genes are primarily cold-induced, with their greatest expression under long day conditions where cold temperatures are perceived several hours before and after illumination, suggesting some interaction with or gating through circadian response regulators. This gating effect would be consistent with CBF regulated gene expression since CBF is also most highly expressed during cold early light conditions [69]. Also, as was observed in DAM genes of leafy spurge, both FLC and MAF2 promoters contain putative CBF binding sites. It is also interesting to note that FLC is generally induced initially by cold temperatures [65], and like some DAM genes, is then repressed by extended exposure to cold temperatures suggesting similar mechanisms may regulate the expression of these two genes.

Both FT and CENL1 genes are repressed in leafy spurge in response to endodormancy inducing temperatures. Although, as noted above, the reduction in FT and CENL1 expression may well be due to negative regulation of these genes by cold-induced DAM genes. Although gene expression profiles of leafy spurge buds did not indicate differential regulation of FVE- or FCA-like genes at the 0.005 p-value cut-off used in the original study, both of these genes are down-regulated in leafy spurge buds during endodormancy inducing cold treatments at the 0.05 and 0.1 p-value levels, respectively. This would be consistent with the coordinate up-regulation of the possible MAF2 orthologue in leafy spurge during the same period. Work in arabidopsis suggest that these genes may regulate other processes associated with flowering besides control of FLC [65], and thus they may also play a role in regulating genes associated with endodormancy processes as well.

2.4. Extended cold temperatures impacts both flowering and endodormancy

The commonalities between signals regulating vernalization and endodormancy release were the primary observation that resulted in the hypothesis that these two processes might share mechanisms of action. Vernalization processes have been well studied in arabidopsis. Flowering competency is induced by extended cold treatments through epigenetic silencing of FLC. These modifications prevent expression of FLC which thus allows flowering to occur when plants are exposed to floral inducing light and temperature regimes. Key regulatory genes involved in epigenetic modification of FLC include VERNALIZATION INSENSITIVE 3 (VIN3), VERNALIZATION 1 (VRN1) VERNALIZATION 2 (VRN2), and LIKHE TETEROCHROMATIN PROTEIN 1 (LHP1). Combined, these genes alter the methylation and acetylation state of histones associated with the promoter and portions of the coding region of the FLC gene and prevent its expression under normal growing conditions [25]. Although VRN1 and VRN2 are not differentially expressed in response to vernalizing conditions, VIN3 is, suggesting it may play a key regulatory role in this process.

Chromatin remodeling does not only regulate flowering through vernalization processes and impacts other flowering genes besides FLC. Chromatin alterations also regulate FT to some extent. TERMINAL FLOWER 2, also known as HETEROCROMATIN PROTEIN 1 specifically silences FT in arabidopsis [70]. Likewise, the gene EARLY BOLTING IN SHORT DAYS (EBS) that is similar to a group of bromo/homeodomain containing zinc finger proteins associated with chromatin modifying complexes impacts FT expression [26]. Indeed, mutations in other chromatin modifying proteins such as various members of the SWI/SNF protein complexes, POLYCOMB group proteins, and other known chromatin modifying proteins impact floral timing and development (Supplemental Table 1) [71,72].

Extended cold also effects flowering potential of buds as well as seeds. In citrus, low temperature treatments both induce flowering [73], and initiates bud dormancy [74]. The effects on dormancy release and flowering could be separated in that non-dormant
buds could still be induced to flower by cold treatments [75]. Likewise, both low temperatures and drought can induce flowering in numerous tropical and sub-tropical species [76]. More recently, low temperature has been shown to directly impact FT expression in Satsuma mandarin (Citrus unshiu Marck.) [77]. Interestingly, the impact of cold treatment on FT expression was limited to mature trees that were floral competent. FT was not cold-induced in immature trees. These observations suggest that tropical species regulate dormancy, FT expression, and flowering, by temperature much as has been observed for leafy spurge.

As with vernalized seedlings or buds that have to “remember” that they had perceived extended cold temperatures and are now flowering competent, endormant buds need to “remember” that they are dormant, particularly during brief warm spells that are common in late fall and early winter. Because both vernalization and endormancy release occur concomitantly in many species, and epigenetic mechanisms are part of this process, dormancy regulation by epigenetic mechanisms is an attractive hypothesis. Although it has been pointed out that chromatin remodeling that occurs during establishment of floral competency is maintained through multiple rounds of cell division which is not necessarily the case in endormancy where one might expect limited cell division [4]. Some evidence for chromatin remodeling in endormancy comes from studies on methylation in potato bud dormancy. Histone modification accompanies release of potato buds from endormancy [78], and during bud set in Castanea sativa [79]. Likewise, several chromatin modifying proteins are differentially expressed during dormancy induction and release in leafy spurge, poplar (in both terminal and cambial meristems), grape, and potato [14,34,80–82]. The role that epigenetic factors play in dormancy induction or release needs to be proven as does the function of genes differentially expressed during endormancy transitions. However, the DAM1 gene of leafy spurge which, like FLC, is induced by cold and then is repressed by extended cold also undergo alterations in chromatin structure during the transition from endormancy to ecodormancy. Thus, the same mechanisms that inhibit FLC expression during extended cold may also turn off DAM gene expression as buds transition from being largely growth-incompetent during endormancy to growth-competent during ecodormancy.

3. Hormonal regulation of endormancy and flowering

GA is required for initiation of flowering under short day conditions [83], through regulation of SOC1 and LFY [84,85]. Indeed, some GA deficient mutants cannot flower at all under short day conditions. However, over-expression of FT in these mutants allows flowering suggesting that GA acts at least partially upstream of the induction of FT [86]. Abscisic acid (ABA) is often antagonistic to GA. Thus, it is not surprising that ABA has been shown to inhibit floral formation. Interestingly, signaling of both ABA and ethylene, another stress related hormone, alter flowering via DELLA proteins which are required for GA signaling [87]. Salicylic acid (SA) also is known to promote flowering via an interaction with the SMALL UBIQUITIN-RELATED MODIFIER (SUMO) E3 ligase and SAP AND MIZ1 (SIZ1) which results in altered chromatin structure of the FLC gene [88]. Auxin and cytokinin are required for flower formation, but these hormones appear to act downstream of FT and are generally considered to be required for the growth and development of the floral organs.

Auxin and cytokinin signaling have been well established in regulation of paradormancy, but their role in endormancy is not well understood. Although both auxin and cytokinin responsive genes are differentially expressed as plants transition from paradormancy into endormancy [14], there has been little evidence that auxin and cytokinin play a direct role in the process. In contrast to auxin and cytokinin, and in common with floral regulation, the role of ethylene, ABA, and GA in dormancy transitions has been fairly well established. In poplar, potato, and leafy spurge, there is evidence for a spike in ethylene signaling which precedes and may even be required for the induction of ABA that is generally observed at the onset of endormancy [14,34,89,90]. As buds transition from endormancy to ecodormancy, ABA levels are generally known to fall [14,91]. However, in poplar, ethylene appears to be required for bud formation, but not bud endormancy induction [90]. As for a possible role of SA in dormancy, the evidence is only circumstantial. SA treatment can induce an oxidative burst responses in plants [92], and oxidative stresses have been implicated in endormancy release [80,93]. No published results have indicated that SA plays a role in endormancy release.

4. Conclusion

It is not surprising that many circadian response genes are regulated differentially during dormancy and flowering transitions. After all, differential day lengths are a common trigger for both dormancy and flowering transitions. However, studies on PHYA over-expressing plants clearly indicate some light sensing proteins can directly impact dormancy and flowering outside the circadian regulatory pathway. More studies are needed to identify the mechanisms through which circadian regulated genes control dormancy induction. It is worth noting that circadian outputs affect many different plant processes, and that both light and temperature can impact circadian responses both directly, and through various feed back loops from output connections. Also, in many cases, clock regulatory proteins must be present at the same time to interact, and the precise temporal expression of many of the circadian clock genes greatly impacts the clock output. Understanding how day length and temperature modify expression of specific clock genes could provide insight into the mechanisms through which these environmental factors regulate dormancy. Additional studies should illuminate these mechanisms.

Based on what is known about the function of floral regulatory genes and about which of these genes play a role in dormancy induction, a model can be developed that could serve as a paradigm for further testing (Fig. 3). FT and related genes such as CEN1, clearly play a role in the initial seasonal growth cessation required for endormancy induction, and possibly endormancy per se. Indeed, these genes seem to play a role in growth that is separate from their role in flowering [94]. It seems likely that DAM proteins bind to regulatory regions of FT family genes and regulate their expression. DAM genes themselves appear to be responsive to dormancy-inducing environmental conditions in a species-specific manner. Thus, cold rather than light induces dormancy in leafy spurge and vice versa in poplar. DAM genes of leafy spurge are primarily cold responsive whereas in poplar they are primarily light responsive. More work is clearly needed at both ends of this signaling cascade. For example, more information is needed to determine the mechanisms through which dormancy-inducing environmental signals are perceived and transduced to the DAM genes, and the function of FT/CEN1 family genes in regulating growth in both vegetative and floral buds (Fig. 3).

Besides growth regulation, it is likely that other less well-characterized developmental and physiological processes might be shared between flowering and endormancy. There are several microarray analyses of flower induction in leafy spurge and poplar that are underway (Anderson and Brunner, personal communications). Once these are complete, between species comparisons of differentially-expressed gene lists between dormancy and flowering transitions should be done to look for other commonalities.
The analysis of the EVERGROWING (EVG) locus in peach and poplar suggests that perhaps there has been some duplication of the DAM genes, which might have lead to specialization of these genes for unique roles in dormancy regulation. Indeed, nearly all of the genes mentioned in this review are members of gene families. Work on understanding the evolution of these gene families and the specific factors controlling both their regulation and function is needed to determine what, if any, role individual family members have in dormancy regulation. Numerous genes involved in multiple physiological, developmental, and biochemical responses show conserved differential patterns of expression during endodormancy transitions based on comparisons of microarray results. Many of these same genes are also known to be differentially-regulated during floral transitions. Additional research is needed to determine what impact, if any, each of these putative floral/dormancy regulators have on the observed changes in gene expression.

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Appendix A. Supplementary data


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