



Knowing when to grow: signals regulating bud dormancy

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Dormancy regulation in vegetative buds is a complex process necessary for plant survival, development and architecture. Our understanding of and ability to manipulate these processes are crucial for increasing the yield and availability of much of the world's food. In many cases, release of dormancy results in increased cell division and changes in developmental programs. Much can be learned about dormancy regulation by identifying interactions of signals in these crucial processes. Internal signals such as hormones and sugar, and external signals such as light act through specific, overlapping signal transduction pathways to regulate endo-, eco- and paradormancy. Epigenetic-like regulation of endodormancy suggests a possible role for chromatin remodeling similar to that known for the vernalization responses during flowering.

Understanding the mechanisms controlling plant growth and dormancy is crucial to solving many problems in agriculture. Such studies affect the reproduction of both valuable and troublesome perennial plants, and the storage and distribution of staple crops such as potato (*Solanum tuberosum*), cassava (*Manihot esculenta*) and onions (*Allium cepa*). Recent successes using genomics-based approaches to understand hormone action and control of crucial physiological processes such as cell division have opened up new avenues for research on growth and dormancy in plants. In light of these findings, it seems timely to review the current state of knowledge on the regulation of dormancy in vegetative buds.

Plant growth and development typically occur in the context of a sessile existence. Consequently, plants have evolved elaborate mechanisms for surviving unfavorable growing conditions experienced in nature. Production of vegetative buds provides plants with a safety net for regrowth or reproduction if environmental conditions result in the death of actively growing or metabolizing tissues. However, uncontrolled growth of vegetative buds during favorable growing conditions would have disastrous effects on plant architecture, reproduction and survival. Thus, plants have also evolved finely orchestrated signaling mechanisms to inhibit vegetative bud growth and development.

Regrowth of new plant tissue from shoots was initially studied more than 100 years ago and it was noted that signals from the apical shoot inhibited growth of proximal

buds. More recent studies have identified conditions under which bud growth is inhibited by signals generated inside the vegetative bud or as the direct result of unfavorable conditions for growth. Lang *et al.* [1] categorized these seemingly separate growth inhibited or dormant states as: (i) paradormancy, the inhibition of growth by distal organs; (ii) endodormancy, the inhibition of growth by internal bud signals; and (iii) ecodormancy, the inhibition of growth by temporary unfavorable environmental conditions (Figure 1). Although these types of dormancy are usually thought of as occurring separately, any given bud might be simultaneously controlled by any, or all of the signals regulating these aspects of dormancy.

Studies of the regulation of bud dormancy have resulted in some of the most fundamental discoveries in plant science and, given continued interest in this subject, will probably continue to do so. Previous research on vegetative bud dormancy has led to the identification of many classic plant hormones and the environmental signals that control the production and perception of these hormones. Related studies of plant growth and shoot development have identified many genes involved in meristem initiation and organ formation, and many genes and signals controlling cell division in plants. The emerging interplay between bud dormancy status (bud growth and development) and cell division suggests that these two fundamental processes are probably regulated by common signaling pathways.

Regulation of growth

Following the breaking of dormancy, vegetative bud growth is associated with the action of specific hormones and is often accompanied by increased cell division. Changes in cell-cycle-specific gene expression occur during release of axillary buds of pea (*Pisum sativum*) [2] and potato [3], and adventitious buds of leafy spurge (*Euphorbia esula*) [4] and Jerusalem artichoke (*Helianthus tuberosus*) [5] from dormancy.

Cell division is represented by a continuous cycle of 'phases' (Figure 2). Cells in G1 phase expand and prepare for DNA replication, which occurs during S phase. After DNA replication, cells enter G2 phase and continue to expand and prepare for mitosis, which occurs during M phase. In plants, non-dividing cells can arrest at the G1–S-phase transition, just before DNA replication, or at the G2–M-phase transition, just before mitosis [6]. In most cases, cells in vegetative buds and shoots appear to be arrested in G1 phase, before the S phase of the cell cycle [7].

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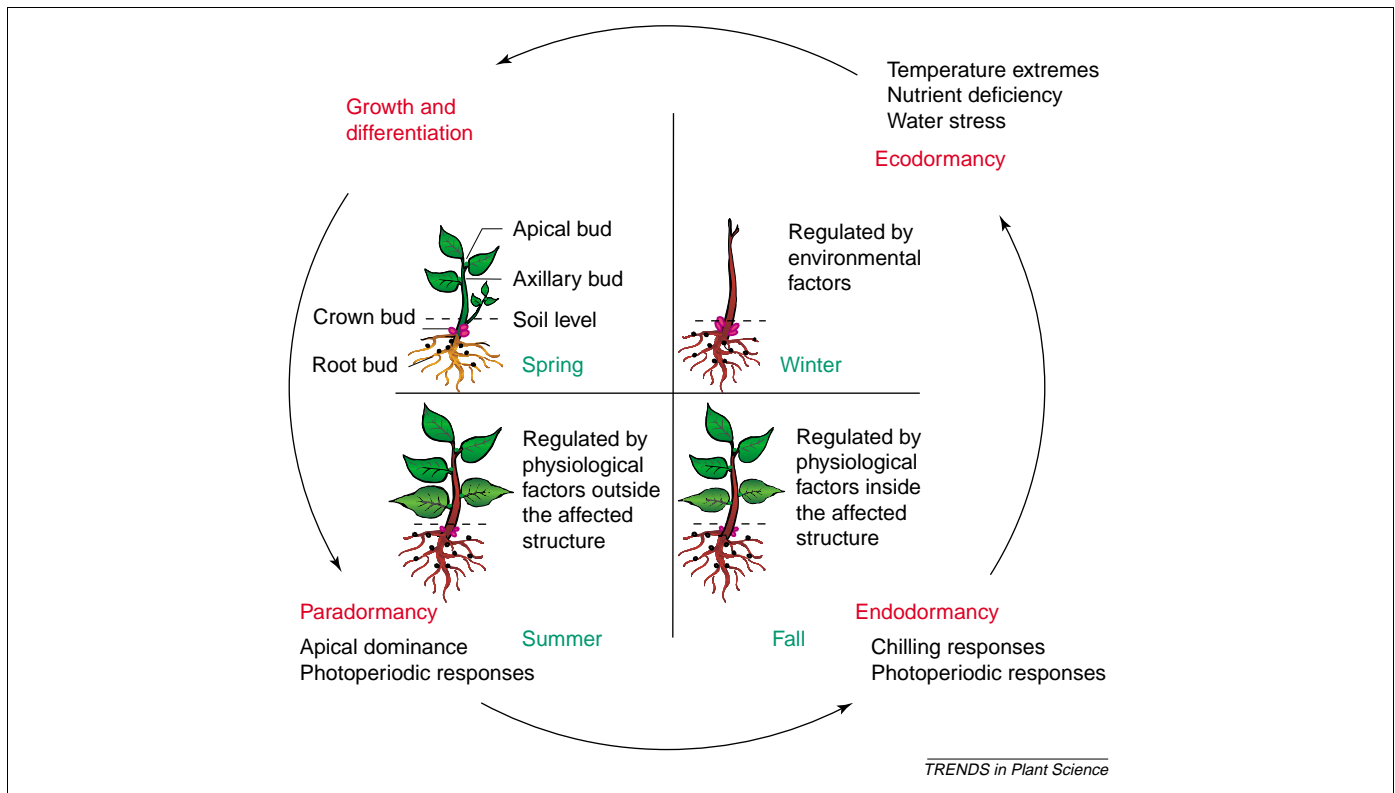


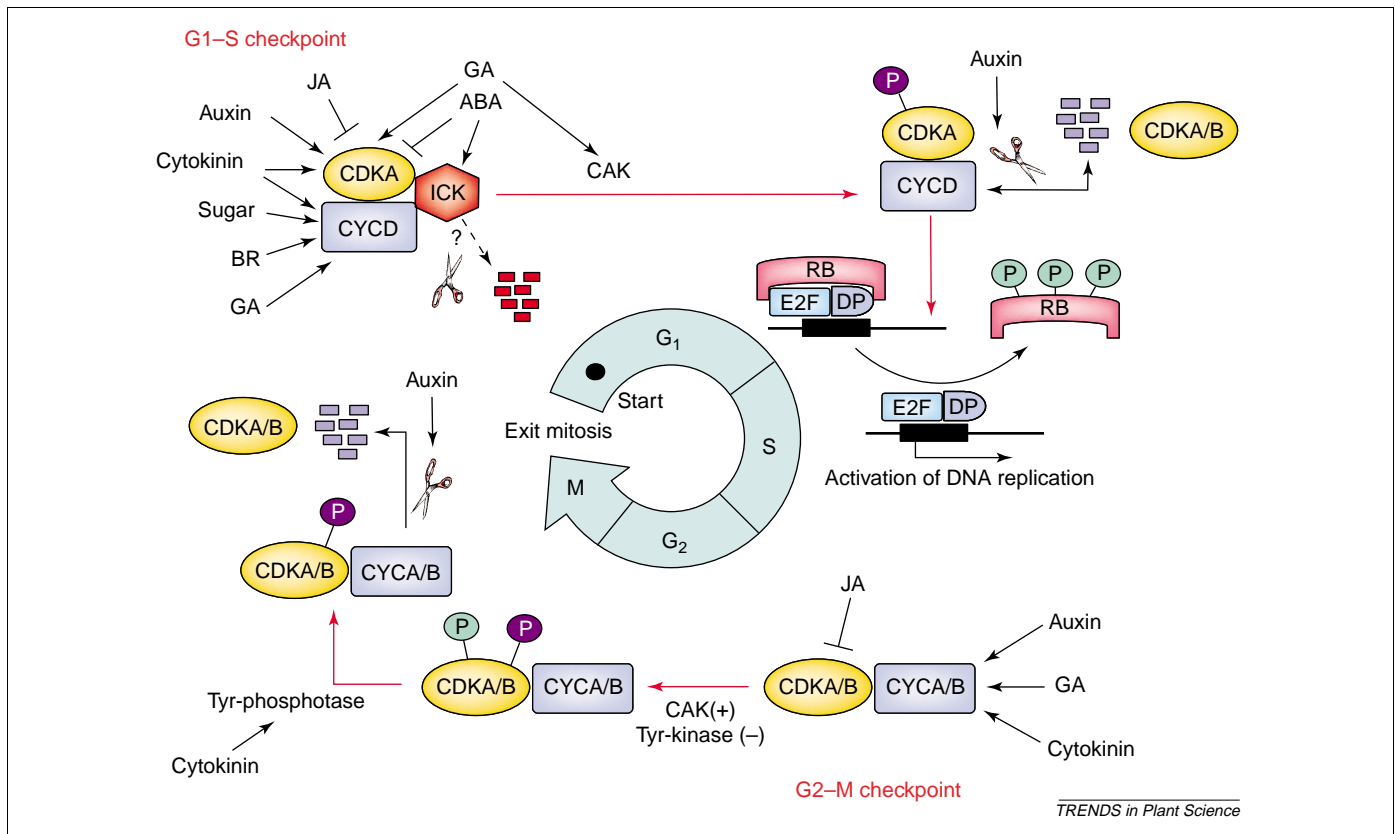
Figure 1. Diagram of signals and typical seasons corresponding to the different types of dormancy. Dormancy is generally defined as the temporary suspension of visible growth of any plant structure (e.g. buds containing meristems). Lang *et al.* [1] further categorized dormancy into paradormancy, endodormancy and ecodormancy. This figure illustrates the cycle of three types of dormancy and the general signals and seasons that are associated with dormancy of perennial weeds, woody plants (shrubs and trees) and shoot buds of tubers (potato and yam).

Dormancy breaking results in upregulation of genes that act at the G1–S-phase transition, such as D-type cyclins (*CYCD*) and histones [2,4,5]. In addition, several post-translational modifications to key enzymes involved in cell cycle regulation occur shortly after dormancy break in several plant systems [3,8]. Consequently, the effort to understand the regulation of cell cycle genes is proving fruitful in understanding the molecular basis of growth arrest imposed by dormancy.

The G1–S-phase transition is well studied in plants and its induction is regulated by perception of various growth-inducing signals such as growth factors or plant hormones. Generally, these signals are initially transduced through post-translational modification of a series of proteins resulting in the transcription of *CYCD* [9]. In *Arabidopsis*, ten different *CYCD* genes have been identified [10]. Several classes of *CYCD* are expressed in various plant tissues and in response to different stimuli, including cytokinin, brassinosteroids, gibberellic acid (GA) and sugar [11–14]. In at least one plant system, protein synthesis was not required for induction of *CYCD* upon stimulation with cytokinin or sugar but was required for induction by brassinosteroids [11,12]. The hormone GA also induces the expression of S-phase-specific markers such as histone genes [4,15]. In underground buds of leafy spurge and germinating seeds of *Arabidopsis*, GA increases the expression of genes involved in the G1–S-phase transition but does not induce genes involved in the G2–M-phase transition during dormancy breaking [4,13].

Once *CYCD* is produced, it binds to cyclin-dependent kinases (CDKs) and this complex phosphorylates the retinoblastoma protein (RB) [16]. However, before CDKs can phosphorylate RB, the CDKs themselves must be activated by a CDK-activating kinase (CAK) [17] (Figure 2). Thus, the G1–S-phase transition is under the control of a phosphorylation signaling cascade. Upon hyperphosphorylation, RB releases bound transcription factors, including E2F, that induce genes needed for DNA synthesis. Additionally, there is evidence that RB–E2F complexes play a role in the chromatin remodeling required for appropriate expression of cell cycle-regulated genes [18]. Both plants and animals have functional orthologs and paralogs of all these genes [19], suggesting similar regulatory pathways.

Cell division proceeds from S phase through to G2–M phase, when additional signals can block the cell cycle before mitosis. Initiation of the G2–M-phase transition requires induction of the B-type cyclins (*CYCB*) and the *CDKB* gene. The plant hormones auxin, cytokinin and GA have all been implicated in *CYCB* and *CDKB* expression and/or stability [20]. *CYCB* interacts with *CDKB* to initiate phosphorylation and activation of proteins and expression of genes required for cytokinesis [19]. Another potential block in M-phase progression occurs via a phosphorylation of tyrosine 15 within CDK that inhibits activity [21]. Phosphorylation of tyrosine 15 is mediated by the tyrosine kinase WEE1 [22]. Cytokinin is required for dephosphorylation of this tyrosine residue. In animals and fungi, CDC25 is a phosphatase that performs this function



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Figure 2. Model for G1-S and G2-M transitions in plants based on combined models by Gutierrez, Anderson *et al.* and Stals and Inzé [7,60,61], and on recent results obtained from plants and animals. Activation of G1 progression involves the expression of D-type cyclins (CYCD) and their catalytic subunit, cyclin-dependent kinase (CDKA), dissociation of CDK inhibitory protein (ICK1) from CDKA-CYCD complex, and phosphorylation of the Thr160 residue (P highlighted in purple) of CDKA. *CYCD* and *CDKA* are upregulated by various growth regulators including auxin, cytokinin, brassinosteroids (BR), sugar and gibberellic acid (GA). *ICK1* is induced by abscisic acid (ABA). Phosphorylation of CDKA is the activity of CDK-activating kinase (CAK), which is induced by GA [14]. Active CDKA-CYCD complex hyperphosphorylates retinoblastoma protein (RB), which inhibits its binding to transcription factors (E2F) and the docking protein (DP), thus initiating chromatin remodeling, transcription activation, DNA replication and S-phase transition. The SCF (SKP1-Cullin-F-box-protein) complex mediates ubiquitination and proteolysis of *ICK1* [62] and *CYCD* that is necessary to trigger the G1-S-phase transition in yeast (and possibly plants, denoted by '?'). Initiation of the G2-M-phase transition requires induction of the A- and B-type cyclins (*CYCA/B*) and the activity of A- and B-type CDKs (*CDKA/B*). The plant hormones auxin, cytokinin and GA have all been implicated in *CYCA/B* and *CDKA/B* expression and/or stability. At G2, the Thr160 (P highlighted in purple) of CDKs is positively phosphorylated (+) by CAK, and the Thr14 or Tyr15 (P highlighted in green) of CDKs are negatively phosphorylated (-) by a tyrosine kinase in the CDKA/B-CYCA/B complex. A cytokinin-regulated tyrosine phosphatase (*CDC25*) removes the inhibitory phosphate and allows the G2-M-phase transition to occur. Commitment to mitosis requires ubiquitin-dependent proteolysis of B-type cyclins. The anaphase-promoting complex (APC) regulates ubiquitination and proteolysis of *CYCA/B*. Auxin appears to be involved in the degradation of cyclins. Jasmonic acid (JA) inhibits CDK activity in both the G1-S-phase and the G2-M-phase transitions.

and expression of the mammalian ortholog is sufficient for cell cycle progression in cytokinin-starved plant cells blocked at G2-M phase [20]. Homologs of *Wee1* have been recently cloned from plants [23,24]. H.J. Rogers *et al.* (unpublished)* recently reported that a functional *Cdc25*-like gene has been cloned from *Arabidopsis*.

Cell differentiation does not occur until RB is reactivated [25]. Likewise, in *Arabidopsis*, a decrease in CAK activity leads to a decrease in CDK activity and differentiation of root initial cells, followed by cessation of cell division [26], indicating that CAK plays a role in both cell division and differentiation. Once the cycle(s) of cell division are complete, RB becomes hypophosphorylated and the cells expand and differentiate into the types required for normal development. Consequently, regulation of cellular developmental processes will also be tied tightly to signals regulating dormancy. Although there is substantial information about the regulation of growth

and development, only recently has there been information tying these processes directly to signals regulating dormancy in adventitious or axillary buds.

Paradormancy

Paradormancy is the best studied of the three types of dormancy and is sometimes referred to as apical dominance or correlative inhibition. This process allows the plant to devote resources to reproduction and to control plant architecture, maximizing light harvesting while allowing for regeneration should the individual shoots become damaged. Many studies have implicated basipetally transported auxin as the primary signal regulating paradormancy. However, it should be realized that grafting studies have implicated other signals than auxin, produced in the roots and stem, as having a profound effect on shoot outgrowth [27,28]. The role of auxin in growth inhibition is complicated slightly by the apparent production of auxin in growing buds and by plant growth's requirement for auxin. However, auxin produced in distal meristems appears to affect the buds differently than does auxin produced inside the buds once they have broken

* H.J. Rogers *et al.* (2003) Controlling cell size through cell cycle regulators in *Arabidopsis*. Abstract #132, 14th International Conference on *Arabidopsis* Research, University of Wisconsin, Madison, WI, USA. (<http://www.union.wisc.edu/conferenceservices/arabidopsis/Arab2003%20abstracts%20draft.pdf>).

dormancy [29]. The signaling mechanisms regulating the growth inhibitory effects of auxin on the buds are beginning to be deciphered. Several genes involved in the auxin-regulated growth inhibition have been identified in pea and *Arabidopsis*. The *ramosus* mutants of pea (*rms1–rms6*) and the *max1–max5* mutants of *Arabidopsis* are drawing the most interest (see [30] and Ottoline Leyser in this issue of *Trends in Plant Science* [31]).

In several systems, auxin signaling alters cell cycle regulation directly or through cross talk with other plant hormones. Auxin inhibits the production or sensing of cytokinin, a plant hormone that is required to induce both *CYCD3* and *CDKB* expression [20]. Other plant hormones that act in correlative inhibition and plant growth in general include GA and abscisic acid (ABA). GA promotes growth, whereas ABA inhibits growth. Studies have indicated that M-phase progression in rice, through induction of *CYCA*, *CYCB* and *CDKB*, requires GA signaling [15]. GA also induces S-phase progression but not full induction of growth and development in paradormant underground buds of leafy spurge [4]. ABA induces expression of the p27^{CIP/KIP} ortholog *ICK1* (an inhibitor of CDK action at the G1–S-phase transition) [32]. The antagonistic actions of GA and ABA provide a possible mechanism for the observed progression of S-phase in leafy spurge buds.

Auxin signaling functions, in part, through targeted degradation of specific proteins and regulation of cytokinin production in the stem segments adjacent to the axillary buds [33,34]. Interestingly, RGA, a protein involved in blocking GA signaling in roots of *Arabidopsis*, is degraded by an auxin-regulated ubiquitin–proteasome pathway [35]. It has also been suggested that auxin might regulate ABA levels via expression of a P450 mono-oxygenase [34]. However, whether degradation of ABA or GA signaling in buds is directly regulated by an auxin controlled proteasome complex remains to be shown.

In addition to auxin and other hormones, sugar plays a complex role in paradormancy. It is required for expression of *CYCD3* in *Arabidopsis* [10,16]. However, sugar also acts synergistically with ABA signaling [36] and might therefore enhance expression of *ICK1*. In leafy spurge, sugar from photosynthesizing leaves independently inhibits growth of paradormant underground buds [37]. Similarly, sugar also interacts with ABA and GA in potato tuber formation [38]. Further research will be needed to determine the action of sugar in these two seemingly conflicting roles in growth control.

Endodormancy

Endodormancy is the result of physiological changes internal to the bud that prevent untimely growth during seasonal transitions, when environmental conditions often fluctuate between those permissive or inhibitory to growth. Endodormancy provides an important mechanism for protecting vegetative buds by ensuring that meristems will not resume growth until the stable return of permissive conditions. In contrast to paradormancy, the molecular aspects of endodormancy are poorly understood. The molecular biology of endodormancy has been studied in poplar (*Populus deltoids*), grape (*Vitis vinifera*) and potato. In poplar and grape, external environmental cues induce

changes in axillary or terminal buds on branches and inhibit growth even after permissive environmental conditions return and they are released from known sources of correlative inhibition. In potato, developmental cues establish conditions that control bud growth from tubers. Although the nature of these changes is completely unknown, the signals that induce or break endodormancy are reasonably well characterized.

Light and temperature both play a significant role in the induction and breaking of endodormancy, with light playing the dominant role in most woody perennials. In deciduous trees, shortening day length induces a developmental change in terminal buds that results in a leaf primordium forming scales instead of leaf buds [39]. In trees such as birch (*Betula papyrifera*) and poplar (*Populus tremuloides*), additional changes take place that induce cold hardiness, cessation of cell division and induction of dormancy in the terminal meristems [40]. Extended chilling or freezing are often required to break the endodormant state, but chemicals such as hydrogen cyanamide (HC) break endodormancy as well [41]. Although the mechanisms for dormancy release by HC are unknown, there is evidence that it requires the action of an *SNF*-like protein kinase [42].

Some signals mediating the induction of endodormancy are being characterized, although the mechanisms through which the signals are mediated remain to be elucidated. Phytochrome has long been known to be a key regulator of light responses in plants. Quantitative trait locus (QTL) analysis of progeny between poplar ecotypes with differing dormancy induction thresholds identified two major QTLs, one of which mapped to a region of the chromosome that contains a phytochrome-encoding gene [43]. Senescence in plants is often associated with, or occurs simultaneously with, induction of endodormancy. Ethylene and ABA play roles in the induction of plant senescence. In fact, senescence is implicated in the induction of endodormancy in several plant systems [36]. Ethylene directly induces endodormancy in potato microtubers [44]. Additionally, the role of ABA in endodormancy is well established in several systems, and instances of phytochrome acting synergistically with ethylene and ABA are known [45,46]. A better understanding of the molecular mechanisms through which these signaling pathways operate is developing but, with the exception of ABA action, no solid connection has been characterized between the signaling pathways for these stimuli and dormancy.

More recently, there has been an effort to characterize molecular changes directly regulated during onset and breaking of dormancy. The expression of several dormancy responsive genes in the buds of deciduous trees and other plants has been characterized. Perhaps the most interesting studies have focused on genes coding for *KNOTTED*-like homeodomain proteins. Certain members of this family, along with members of the *CLAVATA* group and *WUSCHEL*, play a crucial role in the proliferation of undifferentiated cells of shoot meristems and are required for maintenance of growth [47]. The expression of *KNAP2* (a *KNOTTED*-like gene) is upregulated during dormancy onset but downregulated during breaking of dormancy in apple [48]. In a related result, overexpression of a

KNOTTED-like gene in potato reduced the level of GA in the whole plant [49]. These findings are counterintuitive considering that *KNOTTED*-like genes are often associated with increased meristem growth. More consistent with the hypothesis that *KNOTTED*-like genes should be positively associated with growing rather than dormant meristems are studies in *Arabidopsis* indicating that *KNAT2* responds positively to cytokinin and antagonistically to ethylene signaling [50]. Also, mutations in the *PASTICCINO* genes, which negatively regulate cell division by coupling hormonal control of cell proliferation and development, can partially complement the meristemless phenotype associated with mutations in certain *KNOTTED* genes [51]. It should be realized that *KNOTTED*-like genes are a small gene family. Thus, one explanation for these conflicting results is that different members of the *KNOTTED*-like homeodomain gene family might be expressed differently.

In addition to genes that are expressed differently, some proteins are relocated or modified during the induction and release of endodormancy. During dormancy onset in silver birch (*Betula pubescens*), the plasmodesmata are blocked by 1,3- β -D-glucan. They remained so until chilling caused movement of small spherosome-like vacuoles containing 1,3- β -D-glucanase that aligned with the plasmodesmata before releasing 1,3- β -D-glucanase. Dyes and fluorescein-tagged GA confirmed that the reopening of the plasmodesmata followed chilling [52]. Additionally, various isoforms of pectin methylesterase show different expression patterns during bud dormancy and release in hybrid aspen [53], and differences in the temperature-dependent K_m of plasmalemma ATPase activity were noted in vegetative buds of peach [54].

Along with changes in gene expression, there is also evidence for more general epigenetic changes associated with endodormancy induction and release. Changes in DNA methylation have been observed during the induction and breaking of endodormancy in potato buds [55]. Increased DNA methylation following induction of dormancy suggests that chromatin remodeling might play a role in regulating bud dormancy. Interestingly, the previously mentioned SNF1-like protein kinase, activated in grape by HC, is similar to a known component of a DNA modifying protein complex SWI–SNF from yeast and animals [56]. Other components of this complex interact with RB–E2F in both plants and animals [18]. Notably, flowering is also regulated by light and often requires extended chilling or freezing temperatures for induction of flowering. Recent advances in the molecular mechanisms regulating the temperature and light regulation of flowering have defined several proteins involved in chromatin remodeling (Box 1). One such protein, FAS1, is involved in chromosomal remodeling and the flowering response, and is regulated in a cell-cycle-dependent manner in cell cultures of *Arabidopsis* [57]. Given the overlap in the physical signals regulating flowering and endodormancy, it might prove fruitful to search for common signaling pathways as well.

Ecodormancy

Ecodormancy is imposed by external environmental factors such as cold or drought stress, which induce critical

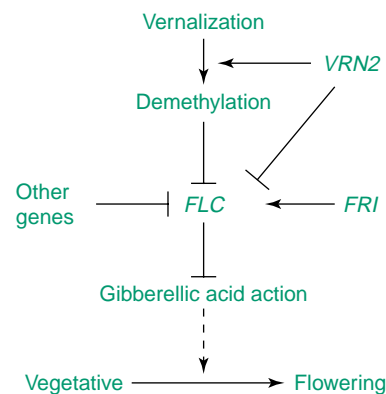
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Box 1. Flowering and dormancy: two processes regulated by similar signals with epigenetic-like characteristics

Flowering in some plants such as *Arabidopsis* requires an extended period of cool temperature in the seedling stage (i.e. vernalization) to induce timely flowering. In the same way, some endodormant buds require a period of cool temperatures for the transition to a non-dormant state capable of shoot growth. Likewise, both processes are affected by day length. Late-flowering ecotypes of *Arabidopsis* contain dominant alleles of *FLOWERING LOCUS C* (*FLC*) and *FRIGIDA* (*FRI*) that suppress flowering (Figure 1). The effect of these genes is suppressed by vernalization and some other factors. The *FLC* gene encodes a MADS domain protein (one of a large family of transcription factors) that acts as a repressor of flowering and *FLC* mRNA levels are controlled by *FRI* and other proteins in the autonomous flowering pathway. *FLC* suppresses flowering by a rheostat mechanism in which the level of *FLC* activity is proportional to the lateness of flowering. *VRN2* encodes a nuclear protein required for maintenance of *FLC* repression when temperatures return to normal levels. The *VRN2* gene shares similarity with the *Drosophila* gene *polycomb* (*PcG*), a component of a chromatin remodeling complex that is responsible for epigenetic-like regulation of body-part development.

Chromatin remodeling indicates modifications in chromatin structure and organization. In recent years, it has been shown to play a key role in eukaryotic gene expression. Multiprotein complexes regulate the structure of chromatin and such complexes ensure stable inheritance or cellular memory of 'on' and 'off' states of gene expression through many cycles of cell division. Components involved in transcriptional control of many developmental regulators that provide this type of cellular memory include proteins of the *PcG* and trithorax (*TrxG*) groups. In *Drosophila*, these genes play a role in regulating homeotic genes, with *PcG* proteins involved in maintaining an inactive state, whereas *TrxG* proteins maintain an active state. Two multimeric complexes in *Drosophila* that contain *PcG* proteins also contain histone deacetylase, indicating a role for histone deacetylation in *PcG*-mediated repression. Some of these complex component proteins contain domains that confer histone methyltransferase, which might play a role in potato bud dormancy through gene silencing.

Given the similarity in signals regulating dormancy and flowering, investigations should be conducted to determine whether genes or mechanisms active in vernalization have a similar role in modifying the dormancy status of vegetative buds.



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Figure 1.

signals that prevent bud growth. These extended periods of environmentally unfavorable growing conditions generally are required to signal the breaking of endodormancy, while at the same time imposing ecodormancy. Although

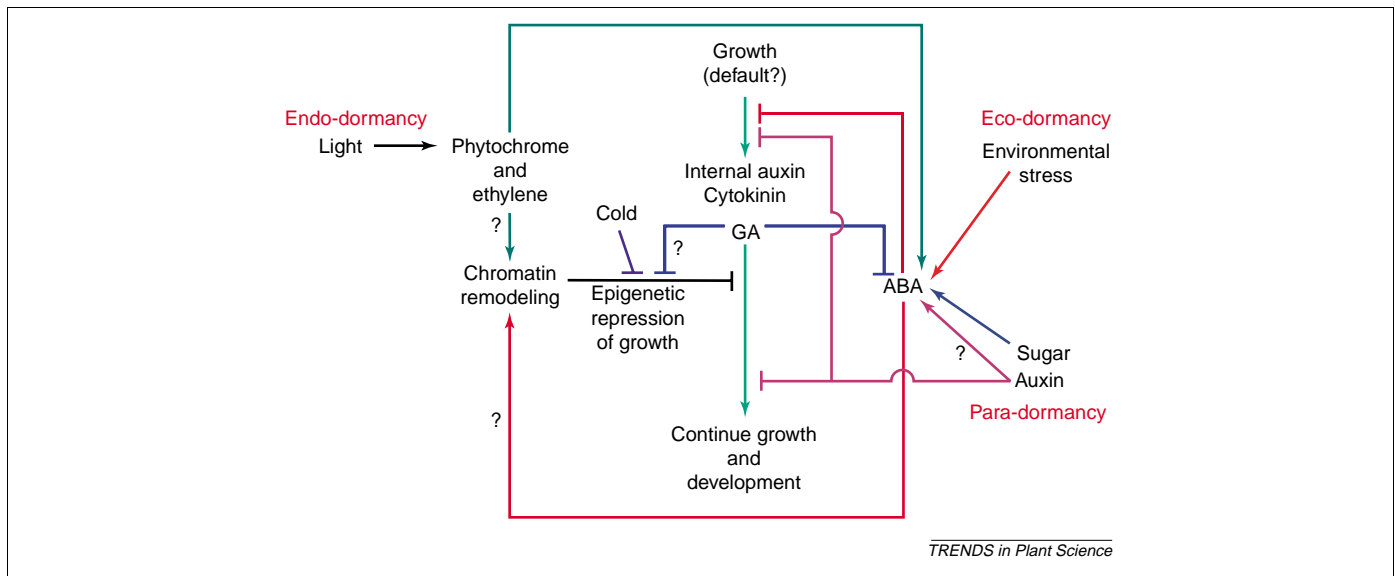


Figure 3. Pathways regulating dormancy in plants. Growth inhibition in paradormancy is primarily controlled by auxin and sugar, via abscisic acid (ABA) inhibition and gibberellic acid (GA) and cytokinin signaling. ABA is the primary signal regulating ecodormancy. Endodormancy is primarily regulated by phytochrome and/or ethylene, and might act via a chromatin remodeling epigenetic-like mechanism and/or ABA-mediated growth arrest. Cross talk of signal transduction pathways between ethylene/phytochrome and ABA plays a role in dormancy. Question marks represent likely pathways that have not yet been shown to exist.

the molecular mechanisms controlling ecodormancy have not been fully characterized, there is sufficient information on the physiological signal transduction processes regulating responses to cold and drought to provide one possible mechanism for growth inhibition. The plant hormone ABA has long been recognized as a key signal induced during both cold and drought stress [58]. Given the role that this hormone probably plays in regulating *ICK1*, it is easy to envision a model in which cold- or drought-induced ABA accumulation blocks further growth and development by turning on *ICK1* to prevent cell division in buds (Figure 2).

Conclusion and future directions

The picture emerging from this work points to a complex set of overlapping hormonal signals that are responsive to various environmental and physiological conditions (Figure 3). These signals interact to regulate specific phases of cell division and development. Ethylene and phytochrome play a significant role in endodormancy, and auxin primarily plays a significant role in paradormancy. ABA, GA and cytokinin appear to play significant but antagonistic roles in all three types of dormancy. It is noteworthy that ABA, GA, auxin and cytokinin all have defined roles in regulating specific components of the cell cycle regulatory machinery. Precise identification of the signaling events through which dormancy-regulated signals interact with components of the cell cycle machinery should illuminate the mechanisms involved in the induction and breaking of dormancy. However, it should be realized that there are cases in which growth occurs in seeds without full developmental induction of meristem function and development [59]. Also, there are the conflicting results with expression of *KNOTTED*-like homeotic genes in response to dormancy regulating signals. Combined, these observations point to a potential link between the cessation of cell division and developmental processes in plants. Continued studies of cell cycle

regulation in relation to dormancy and linking plant development with induction and breaking of dormancy provides obvious directions for future research.

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