

## **DORMANCY AND THE CELL CYCLE**

Michael A. Campbell

School of Science  
Penn State Erie—The Behrend College  
Erie, Pennsylvania 16563

### **INTRODUCTION**

Dormancy can be viewed as a complex trait that encompasses physiological and developmental responses to environmental signals. There has been considerable research and emphasis in the past on the dormancy of seeds, which is to be expected in view of the significant impact to agricultural yields, but it has only been in the last few decades that significant research success has yielded information on the topic of bud dormancy. Thus, this review will focus on the recent results regarding bud dormancy and the interaction of dormancy to issues related to phytohormones and cell division.

### **TYPES OF DORMANCY AND THE CELL CYCLE**

Biologically, dormancy is an avoidance response to drought, cold, or shortening days. The dormant situation is a complex set of physiological states and conditions in which plants respond to a series of stresses such as drought and

over-wintering by entering a state of growth suspension. This state of growth suspension can be exhibited by degrees of dormancy. The classification and degrees of dormancy found in plant vegetative structures have been defined as endodormancy, paradormancy, and ecodormancy (Figure 1) (1). Ecodormancy is a reduced growth response to an external stimulus such as drought or cold. Removal of the stimulus results in a resumption of growth. Paradormancy is a reduced growth response induced by a biochemical signal that is transported to a target tissue. Removal of the signal results in a resumption of growth. A good example of paradormancy is apical dominance, where auxin transported from an apical shoot suppresses the growth of lateral buds. Endodormancy is due to an endogenous signal that results in growth suppression. Older references often used the term “deep dormancy” to describe this phenomenon. In some situations, time is all that is required for endodormancy to terminate (e.g., the potato) and in others there is a need for a cold treatment in order to break the dormant state (such as most flowering temperate trees and shrubs).

Defining a dormant state in a plant can be difficult because a tissue may progress from one dormant condition to another without any phenotypic change. For example, a meristem may enter a phase of reduced growth in late summer in response to day-length or drought. Thus, the meristem could be considered to be in a state of ecodormancy. This situation could be followed by a physiological shift and a transition into a state of endodormancy. As winter progresses the endodormant state may terminate but growth will not resume due to unfavorable conditions and the meristem is now again in an ecodormant state. This shift between dormant states suggests that control of the growth cycle is a complex interaction between endogenous and environmental factors, and there is no simple genetic solution to increasing yields by manipulating dormancy. This concept is supported by breeding experiments and quantitative trait analysis, which is demonstrated by the significant genetic complexity controlling the onset and breakage of dormancy in poplar (2).

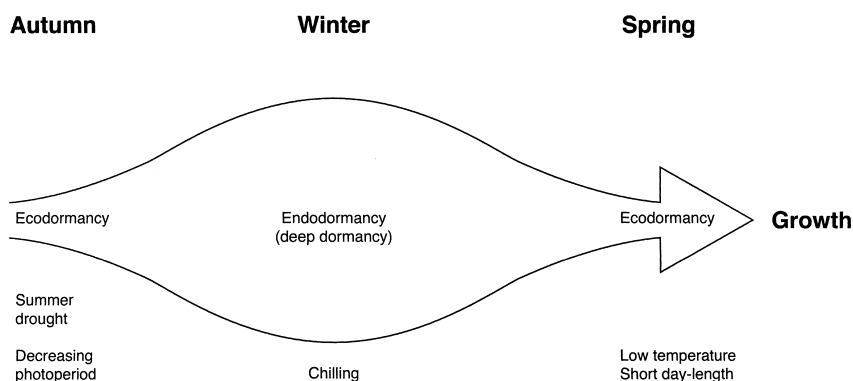


Figure 1. Diagram of dormancy states in a typical perennial bud. As the season progresses, dormancy types may shift between ecodormancy and endodormancy and back to ecodormancy. Redrawn from M. Lang et al. (1).

The variations and changes in the types of dormancy suggest that different biological and physiological mechanisms are involved with the changing of dormant states. As this pertains to cell cycle regulation, it is unclear whether different states of dormancy are associated with variations in cell cycle control. The cell cycle is regulated by a set of protein interactions between cell division kinases (CDKs), cyclins (CYCs), and inhibitors or regulators of the CDK/CYC complex [reviewed in (3)]. The activity of the CDK/CYC complex is associated with the establishment of restriction, or control, points throughout the cell cycle. These control points are positioned in the gap phases (G1 or G2) of the cell cycle (Figure 2). Thus, dormancy, which is characterized by very low or absent rates of cell division, must function by the established control points found in the G1 and G2 phases of the cell cycle (4). The arrest of cell division in endodormant ash buds (5), endodormant *Helianthus* buds (6), endodormant potato meristems (7), and paradormant axillary buds of pea (8) appear to be predominantly at the G1/S-phase of the cell cycle. This commonality of regulation, despite the specific type of dormancy, suggests that attempts to alter or control the dormant state in plants, with respect to the cell cycle state, would probably require manipulation of the factors or signal transduction mechanisms that interact with the G1/S restriction point. The interaction of various types of dormancy with the G1/S portion of the cell cycle demonstrates that artificial manipulation dormancy, by alteration of cell division or growth, will most likely involve genes or proteins that regulate the transition from the G1 to the S-phase of the cell cycle. At this point I do not suggest that direct alteration of genes regulating the G1/S restriction point is a practical solution to dormancy manipulation. Cell cycle control is

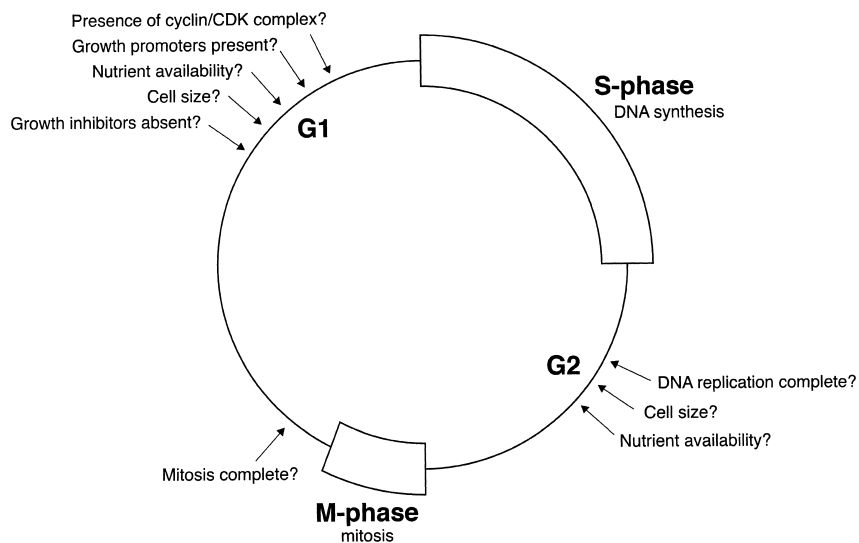


Figure 2. The major regulatory positions of plant cells.

fundamental to cell proliferation and survival. However, recent analysis of plant dormancy has narrowed the field to a number of potential targets that might impact dormancy onset and length without directly altering basic cell cycle machinery.

### THE PARADORMANT STATE

Paradormancy, specifically apical arrest of lateral buds, is regulated by auxin but it is not clear whether this response is directly or indirectly associated with that phytohormone [reviewed by Horvath et al., 2003 (9, 10)]. Shimizu-Sato and Mori (11) recently reviewed the topic of dormancy regulation in lateral meristems and they proposed a scheme for hormonal regulation of growth (Figure 3). In this model, auxin, produced in the apical regions of the plant, does not directly inhibit cell division in the lateral meristems, but does result in abscisic acid (ABA)-induced genes in the node region and the lateral meristem. The model is supported by the work of Gocal et al. (12), who demonstrated that auxin levels in dormant meristems are low and they increase after release of apical repression. This suggests that, inasmuch as high levels of auxin are not present in paradormant lateral meristems, growth inhibition must result from some other signal. The increase in auxin as lateral buds are released from apical repression may have some association with entry into the cell cycle, since it has been shown that tobacco BY2 cells express D-type cyclins in response to auxin (13). Thus, a potential working model for paradormancy may be lateral meristem arrest by nodal ABA signals. Entry into the cell cycle begins following removal of the auxin/ABA inhibition and production of a phytohormonal signal, such as cytokinin, that initiates cell division.

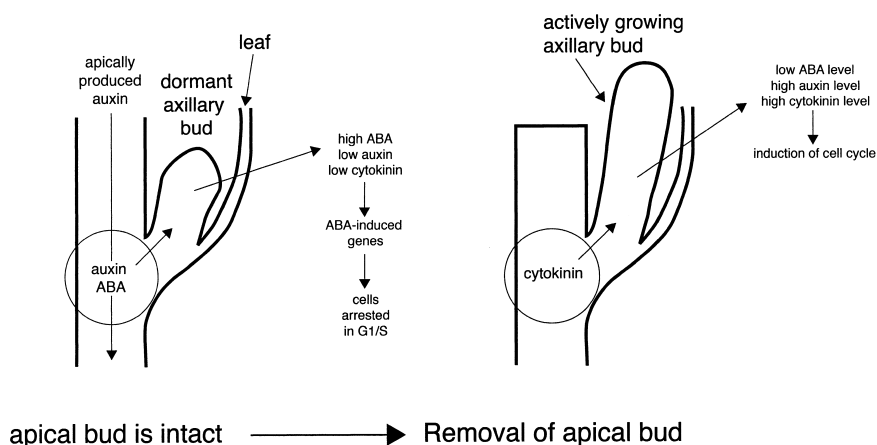


Figure 3. The mechanism of action in a paradormant axillary bud induced by apical dominance. Redrawn from Shimizu-Sato and Mori (11).

Transgenic plants that overproduce cytokinins exhibit a reduced level of lateral bud arrest (14, 15). Thus, it can be concluded that the interaction of cytokinins and auxins regulate lateral bud growth and paradormancy. These two hormones are probably functioning at two levels, an inhibitor process controlled by apically produced auxin/ABA and a growth-promoting process regulated by cytokinin.

## THE ENDODORMANT STATE

In many perennial woody plants the breakage of the endodormant state requires chilling. It has been found that chilling results in a rearrangement of symplastic connections between cells of the apical meristem by the formation of 1,3-beta-D-glucan blockages (16). Thus, chilling induced capacity for cell division may be a result of removal of symplastic blockage by 1,3-beta-D-glucanase resulting in increased intercellular communication. It is possible that this increased communication results in the transport of hormonal signals throughout the meristem. However, it has not been shown that alterations of intercellular connections directly change hormonal transport or metabolism in meristems.

There has been recent research focused on the subject of vernalization, which is a requirement of some species for a cold treatment for the induction of flower development. Some parallels exist between the process of vernalization and cold-induced breakage of endodormancy. Vernalization has been shown to be an epigenetic process that requires prolonged exposure to cold temperatures, resulting in a developmental shift from vegetative to floral development [(17), reviewed in (18)]. Thus, vernalization and the breakage of endodormancy both require a similar temporal exposure to cold. Little is known about the molecular mechanisms associated with the breakage of endodormancy, but significant research advances have been accomplished regarding the process of vernalization. In *Arabidopsis*, vernalization has been shown to be an epigenetic process where extended exposure to cold results in the repression of the gene *FLOWERING LOCUS C* (*FLC*) with the use of chromatin remodeling induced by the genes *VRN1*, *VRN2*, and *VIN3* (19).

Exposure to cold can induce cold acclimation, vernalization, and the breakage of endodormancy in some species. Is there any similarity in the cold-induced regulation of these three processes? Cold treatment also induces elevated ABA levels and a series of cold-regulated genes controlled by the transcription factors CBF1, CBF2, and CBF3 (20, 21), but prolonged cold treatment reduces ABA in over-wintering endodormant buds. Liu et al. (22) demonstrated that vernalization is not regulated by ABA or the cold-induced transcription factors. While the onset of endodormancy appears to require ABA, it has been shown that cold is not necessary to induce the endodormant state in grape (23). However, to break endodormancy in some species, cold is a requirement and, in other species, such as potato, the breakage of endodormancy only requires time. A commonality between potato and species that require cold might be the reduction of ABA levels in meristems, shifting endodormancy to an ecodormant state. Another possibility is that the control of endodormancy is similar to vernalization, where chromatin remodeling, controlled by

ABA, time, and/or cold treatment is a requirement. Support for this hypothesis can be found in the work of Law and Suttle (24), who showed that in potato tubers demethylation of CCGG regions of DNA has been linked to the breakage of endodormancy. What remains to be determined is what regions of the genome are remodeled. Additionally, it would be important to discern whether a similar mechanism of remodeling is occurring in species that require cold treatment for the breakage of endodormancy and potato, which only requires a temporal exposure for dormancy loss. Is it possible that termination of endodormancy follows a pattern similar to that of vernalization: cold or time results in chromatin remodeling in an area of the genome that contains genes that suppress growth? Meristems that enter endodormancy have usually undergone a significant developmental shift with leaves replaced by bracts or bud scales at nodal regions and such developmental changes can be associated with chromatin remodeling (25).

### **IS THERE A UNIFYING THEORY TO DORMANCY?**

Environmental stress, such as drought, induces the production of ABA, which results, with a complex set of responses, in growth arrest (ecodormancy). In paradormancy, in particular apical dominance, it has been demonstrated that there is an auxin-induced ABA response at nodal regions, which results in growth arrest in lateral meristems. Elevated ABA levels, at least in the potato system, induce endodormancy. Thus, a common theme in a number of plant systems, regardless of the type of dormancy, appears to involve an ABA response at some level.

However, the removal of ABA is not always sufficient to end the dormant state and initiate growth. Additional hormones, such as cytokinin and gibberellins, appear to be necessary for the resumption of cell division. Thus, dormancy, irrespective of the specific type, is controlled by a process of growth suppression and growth initiation. This might explain some of the genetic complexity found by breeders who are interested in the process of dormancy. The fact that both growth inhibitors and growth promoters regulate dormancy suggests that cell cycle control would follow a similar path; there would be cell cycle inhibitory mechanisms as well as cell cycle promotive mechanisms associated with dormancy. In potato endodormancy, growth arrest seems to occur upstream of the mechanisms of direct cell cycle control (7). This situation may be a result of endodormancy-inducing inhibitors of the cell cycle and that ABA has a central role in maintaining the endodormant state.

### **REGULATION OF THE G1 TO S TRANSITION OF THE PLANT CELL CYCLE**

The cdk/cyclin protein complex regulates cell cycle transitions and, because dormancy appears to be a G1/S arrest, it is necessary to elucidate the components of the CDK/CYC complex associated with that arrest. Currently, specific proteins associated with dormancy G1/S arrest have not been found. In *Arabidopsis*, there are at least four different CDKs and the activity of one class

of the A-type of CDKs increases during the G1 to S-phase transition of the cell cycle [reviewed in (3, 26, 27)]. Potato meristems do not change in the levels of transcript for p34cdc2 kinase as endodormancy terminates (7). Thus, a working hypothesis is that dormancy regulates the activity, not the transcript levels, of a class of the A-type CDKs. The activity of a CDK requires the presence of specific cyclins, a specific phosphorylation state, and the absence of active inhibitors. This complex arrangement for CDK activity suggests that dormancy repression of the cell cycle at the G1 to S transition may result with the regulation of a number of different targets including cyclin levels, kinase activity, phosphatase activity, and the manipulation of inhibitors.

Plant cells contain a diverse population of cyclins, including A, B, D, and H-types (3, 28). More cyclins await description in plants, particularly in perennial species, but among the classes of cyclins known, the ones associated with G1/S cell cycle regulation are of direct interest to dormancy studies. The D-type cyclins have been shown to be associated with G1 to S-phase transitions in yeast (29). In *Arabidopsis*, genomic analysis has revealed that there are 49 different cyclins, which can be assigned to nine different subgroups: *CYCA*, *CYCB*, *CYCC*, *CYCD*, *CYCH*, *CYCT*, *CYCL*, *CYCU*, and *SDS* (30). Although function has not been determined for each of the cyclin-like genes, experimental evidence strongly suggests that the *CYCD* and *CYCA* classes are associated with the G1/S transition of the cell cycle [(27, 31), reviewed in (32-34)]. Thus, the *CYCD* and *CYCA* class of cyclins may be directly regulated by dormancy in plant tissues. It should be noted that cells not undergoing a cell cycle might exhibit low levels of many different classes of cyclins but dormant tissues, arrested in the G1 position, would first need to express the *CYCD* and *CYCA* proteins for entry into the S-phase.

The activity of the CDK/CYC complex is regulated by additional cellular and biochemical mechanisms. A class of proteins classified as CDK inhibitors (CKIs) interacts with the CDK/CYC complex and prevents cell cycle progression [reviewed in (33, 34)]. These inhibitors are interesting targets for investigating the interaction of dormancy and the cell cycle. In mammalian systems, G1/S-specific CKIs are represented by p21Cip1, p27Kip1, and p57Kip2 (17, 35-37). De Veylder et al. (38) have examined the activity of five Kip-related proteins (KRPs) in *Arabidopsis thaliana*. Thus, in comparison to mammalian systems, plants appear to have a greater diversity in KRP-type CDK inhibitors. Does this suggest that plant systems utilize a greater diversity of cell division inhibitors for spatial or temporal regulation of cell division? The results of De Veylder et al. (38) demonstrated more of a structural relationship between the KRPs and regulation of cell division. In *Arabidopsis* there are at least seven KRPs that appear to have diverse functions temporally and spatially in the shoot apex (39), but it is not clear how KRPs are associated with meristem activity and the process of dormancy. An additional class of cell division inhibitors called ICK1 and ICK2 has been identified in plants (40, 41). ICK1 has been shown to be induced by ABA (40), suggesting a relationship between a phytohormone associated with the dormancy response and a protein preventing entry into the cell cycle. The direct connection between ABA-induced dormancy and cell cycle inhibitors has yet to be adequately demonstrated.

## THE PHYTOHORMONE CONNECTION

ABA has been shown to regulate response to drought, cold, salt stress, and seed dormancy through a complex set of fast and slow responses [reviewed in (42-45)]. The signal transduction mechanisms associated with ABA exposure in plants have recently been reviewed (42, 46), and currently there are about 50 genes associated with ABA responses in *Arabidopsis* (43), affecting more than 1,300 different transcripts (47). The interaction of the ABA signal transduction mechanism with genes or proteins that directly affect the dormancy response, which is a slow response, is not clear, and it has been difficult to separate ABA responses associated with stress and cold from those that are directly related to dormancy. ABA has been implicated with the onset and maintenance of endodormancy in potato (48), white birch (49), and lily (50). The interaction of ABA with the process of cell division is still not clear. Application or inhibition of ABA to meristematic tissues may alter the onset of endodormancy but it also results in developmental shifts resulting in the formation of bud scales in place of primordial leaf. Additionally, cross-talk between ABA and other hormones, particularly those that induce growth such as cytokinin, gibberellin, and auxin, complicates the experimental approaches necessary to elucidate specific responses. The role of ABA in seed dormancy and germination has progressed significantly (51), but due to a lack of a model system, ABA control of perennial meristem growth remains undefined. In *Arabidopsis*, an ABA application to germinating embryos results in reversible growth arrest. In tomato, ABA-deficient mutants exhibit an increase in cells arrested in G2/M, suggesting that ABA might regulate the G1/S restriction point. The ABA regulation at the G1/S restriction point may be due to cell cycle inhibitors such as ICK1, but there is some speculation that seedling dormancy might be a function of a p53-regulated process (52, 53). The idea that p53 might regulate cell division in seeds is based on the concept that seeds can be exposed to prolonged storage, resulting in environmentally induced DNA damage. This becomes an interesting issue in long-lived perennial species where lateral bud arrest (paradormancy) may occur on the order of hundreds or thousands of years and may result in significant DNA damage. However, the connection between ABA-induced stress and DNA damage has yet to be elucidated. ABA also results in reduced levels of metabolic activity, which might reflect a lack of nutrient mobilization. In animal systems it has been shown that serum starvation induces p53 activation and growth arrest by the ribosomal protein L11 (54).

In addition to the production of cell cycle inhibitors, ABA appears to interact with the phosphorylation cascade that is associated with the regulation of cell division [reviewed in (43, 46)]. ABA interacts with inositol polyphosphate 5-phosphatase (55), phospholipase C (55), cyclin-dependent kinase (56), protein phosphatase 2C (57, 58), and mitogen-activated protein kinase (59). Additionally, ABA is involved with the regulation of RNA metabolism including transcript abundance, RNA stability, transport and degradation [reviewed in (42)] and some of these transcripts may relate to cell cycle regulation.



Cytokinins have an important role in the breakage of dormancy (60-62). The resumption of cell division following dormancy is often associated with an increase in cytokinin levels. Within the last decade, significant progress has been made regarding the molecular and genetic mechanisms associated with cytokinin signal transduction [reviewed in (63)]. In endodormant tissues, such as potato, meristems change in their sensitivity to exogenous cytokinins; close to harvest, or deep dormancy, cytokinins have little effect on sprouting, but as tubers age the sensitivity to cytokinins increases (62). It has been suggested that endormant tubers do not respond to exogenous cytokinin because of the lack of a cytokinin receptor or inactivity of the signal transduction mechanism of cytokinin action (64). In *Arabidopsis*, it has been determined that receptors for cytokinins (AHK2, AHK3, CRE1/AHK4) are transmembrane histidine kinases (65-68). The expression of the cytokinin receptors appears to occur in all tissues of *Arabidopsis* (63) and the AHK receptors relay endogenous cytokinin signals resulting in shoot and root meristem growth (69). It is not known whether dormancy alters receptor levels. Interestingly, *Arabidopsis cre1* mutants, which have a decreased response to endogenous cytokinin, exhibit a slight increased sensitivity to ABA (65), suggesting a level of cross-talk between the cytokinin and ABA signal transduction systems. Exogenous cytokinin induces D-type cyclin expression and cell division (70), and cytokinin-induced cell division can be replaced by overexpression of D-type cyclins (71). This suggests that as meristems become active following dormancy they enter the cell cycle by cytokinin-induced expression of D-type cyclins. Thus, entry into the cell cycle may not be controlled directly by the dormancy process but by phytohormone production after tissues have exited the dormant state.

In addition to being associated with the breakage of seed dormancy, gibberellins (GAs) have been linked with dormancy release in tulips [reviewed in (72), potato (73), and lily bulbs (74, 75)]. However, the complexity of GA types in cells and tissues, the possible presence of inhibitors, and the difficulty assessing the specific dormant state makes it unclear whether GA is involved with the breakage of dormancy or is a postdormancy growth response. Results by Horvath et al. (76) suggest that the application of GA<sub>3</sub> to leafy spurge resulted in G1 to S-phase transition in adventitious buds (Figure 4).

GA has been shown to bind to a GCR1 receptor in *A. thaliana* seeds (77). Additionally, G-protein-type receptors are associated with GA perception (78). These types of binding by GA probably result in a signal transduction cascade that has yet to be defined in its entirety, but ultimately there must be some impact on the cell cycle machinery. In deepwater rice, GA application induces cell division (79, 80). The regulation of GA on rice cell division appears to be largely at the G2/M restriction point (81). One of the responses of rice to exogenous GA is to increase the levels of transcripts for mitotic cyclins and a specific class of cyclin-division kinase (82, 83). In the meristems of dicots, specifically tomato, GA induces the expression of transcripts for expansins, proteins that alter cell wall extensibility and cell expansion (84). The change in expansion expression may suggest another avenue for GA impact on cell cycle machinery, since cell size has been associated with cell cycle regulation in a number of eukaryotic organisms.

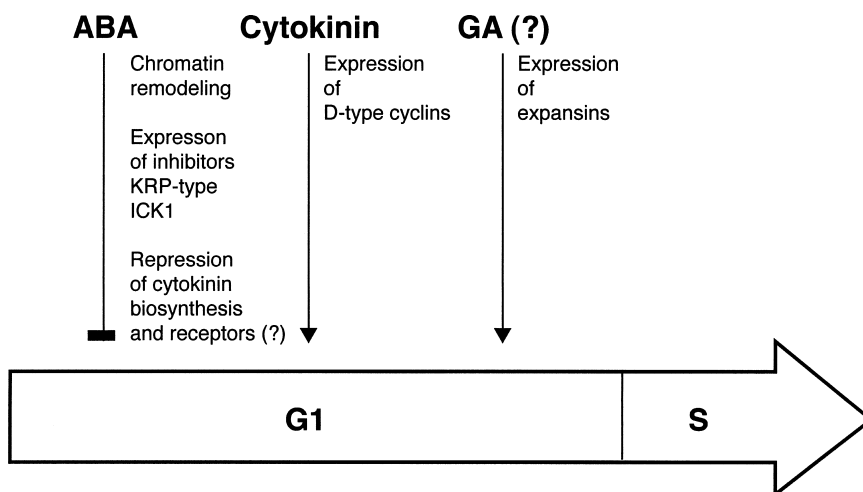


Figure 4. Possible regulatory steps in G1 by phytohormones that regulate dormancy.

## CONCLUSIONS

The localization of the Kip-related proteins to meristematic regions suggests that these proteins might be associated with dormancy regulation. Additionally, the localization of chromatin remodeling to specific genes and loci may reveal the important regulation mechanisms for the dormant state. Developmental mutants that fail to develop bud scales and shift meristem programs toward an over-wintering bud would be informative in identifying.

A substantial body of work has been accomplished in identifying genes and proteins associated with cell division and the cell cycle in plants. However, most of the recent research has focused on the annual *A. thaliana* as a model. In order to progress rapidly in the area of plant meristem dormancy, a model system has to be adopted. Many perennial species are slow growing, genetically complex, and have little background genetic research that can be used to support dormancy studies. Recent advances into the genetic structure of poplar and recent interest in some perennial relatives of *A. thaliana* may create opportunities for models systems.

## REFERENCES

- 1 Lang, G.E., Early, J.D., Martin, G.C. and Darnell, R.L. (1987) HortScience, 22(3), 371-377.
- 2 Chen, T.H.H., Davis, J., Frewen, B.E., Howe, G.T. and Bradshaw, H.D. (2000) in Dormancy in Plants: From Whole Plant Behavior to Cellular Control (J.-D. Viemont and J. Crabbe, eds.) CABI Publishing, New York, NY. pp. 319-330.
- 3 Dewitte, W. and Murray, J.A. (2003) Ann. Rev. Plant Physiol. Plant Mol. Biol. 54, 235-264.

- 4 Van't Hof, J. (1985) in *The Cell Division Cycle in Plants* (J.A. Bryant and D. Francis, eds.) Cambridge University Press, pp. 1-13.
- 5 Cottignies, A. (1979) *Planta* 147, 15-19.
- 6 Tepfer, S.S., Nougarede, A. and Rondet, P. (1981) *Canadian J. Bot.* 59, 1918-1940.
- 7 Campbell, M.A., Suttle, J.C. and Sell, T.W. (1996) *Physiol. Plant.* 98, 743-752.
- 8 Devitt, M.L. and Stafstrom, J.P. (1995) *Plant Mol. Biol.* 29 (2), 255-265.
- 9 Horvath, D.P., Anderson, J.V., Chao, W.S. and Foley, M.E. (2003) *Trends Plant Sci.* 8(11), 534-540.
- 10 Cline, M.G. (1994) *Physiol. Plant.* 90, 230-237.
- 11 Shimizu-Sato, S. and Mori, H. (2001) *Plant Physiol.* 127(4), 1405-1413.
- 12 Gocal, G.F.W., Pharis, R.P., Yeung, E.C. and Pearce, D. (1991) *Plant Physiol.* 95, 344-350.
- 13 Sorrell, D.A., Combettes, B., Chaubet-Gigot, N. and Murray, J.A. (1999) *Plant Physiol.* 119(1), 343-352.
- 14 Medford, H., Horgan, R., El-Sawi, Z. and Klee, H.J. (1989) *Plant Cell* 1(4), 403-413.
- 15 Romano, C.P., Hein, M.B. and Klee, H.J. (1991) *Genes Dev.* 5(3), 438-446.
- 16 Rinne, P.L.H., Kaikuranta, P.M. and Schoot, C. van der (2001) *Plant J.* 26(3), 249-264.
- 17 Lee, I. and Amasino, R.M. (1995) *Plant Physiol.* 108(1), 157-162.
- 18 Sung, S. and Amasino, R.M. (2004) *Curr. Opin. Plant Biol.* 7, 4-10.
- 19 Sung, S. and Amasino, R.M. (2004) *Nature* 427, 159-164.
- 20 Gilmour, S.J., Zarka, D.G., Stockinger, E.J., Salazar, M.P., Houghton, J.M. and Thomashow, M.F. (1998) *Plant J.* 16(4), 433-442.
- 21 Medina, J., Bagues, M., Perez-Alonso, T.J.M. and Salinas, J. (1999) *Plant Physiol.* 119(2), 463-470.
- 22 Liu, J., Gilmour, S.J., Thomashow, M.F. and van Nocker, S. (2002) *Physiol. Plant.* 114, 125-134.
- 23 Fennell, A. and Hoover, E. (1991) *J. Amer. Soc. Hort.* 116, 270-273.
- 24 Law, R.D. and Suttle, J.C. (2003) *Plant Mol. Biol.* 51(3), 437-447.
- 25 Wagner, D. (2003) *Curr. Opin. Plant Biol.* 6, 20-28.
- 26 Hemerly, A., Engler, de A.J., Bergounioux, C., Van Montagu, M., Engler, G. Inze, D. and Ferreira, P. (1995) *EMBO J.* 14, 3925-3936.
- 27 Magyar, Z., Meszaros, T., Miskolezi, P., Deak, M., Feher, A., Brown, S., Kondo, E., Athanasiadis, A., Pongor, S., Bilgin, M., Bako, I., Koncz, C. and Dudits, D. (1997) *Plant Cell* 9, 223-235.
- 28 Vandepoele, K., Raes, J., De Veylder, L., Rouze, P., Rombauts, S. and Inze, D. (2002) *Plant Cell* 14(4), 903-916.
- 29 Dahl, M., Meskiene, I., Bogre, L., Ha, D.T., Swoboda, I., Hubmann, R., Hirt, H. and Heberle-Bors, E., (1995) *Plant Cell* 7, 1847-1857.
- 30 Wang, G., Kong, H., Sun, Y., Zhang, X., Zhang, W., Altman, N., DePamphilis, C. and Ma, H. (2004) *Plant Physiol.* 135, 1084-1099.
- 31 Meijer, M. and Murray, J.A.H. (2000) *Plant Mol. Biol.* 43, 621-633.
- 32 Stals, H. and Inze, D. (2001) *Trends Plant Sci.* 6, 359-364.

- 33 Rossi, V. and Varotto, S. (2002) *Planta* 215, 345-356.
- 34 De Veylder, L., Joubes, J. and Inze, D. (2003) *Curr. Opin. Plant Biol.* 6, 536-543.
- 35 Toyoshima, H. and Hunter, T. (1994) *Cell* 78, 67-74.
- 36 Zhang, P., Wong, C., DePinho, R.A., Harper, J.W. and Elledge, S.J. (1998) *Genes Dev.* 12, 3162-3167.
- 37 Zhang, P., Wong, C., Liu, D., Finegold, M., Harper, J.W. and Elledge, S.J. (1999) *Genes Dev.* 13, 213-224.
- 38 De Veylder, L., Beeckman, T., Beenster, G.T.S., Krols, L., Terrasa, T., Landrieu, I., Van Der Schueren, E., Maes, S., Naudts, M. and Inze, D. (2001) *Plant Cell* 13, 1653-1667.
- 39 Ormenese, S., de Almeida Engler, J., De Groodt, R., De Veylder, L., Inze, D. and Jacqmard, A. (2004) *Ann. Bot. (London)* 93(5), 575-580.
- 40 Wang, H., Qi, Q., Schorr, P., Cutler, A.J., Crosby, W.L. and Fowke, L.C. (1998) *Plant J.* 15, 501-510.
- 41 Lui, H., Wang, H., DeLong, C., Fowke, L.C., Crosby, W.L. and Fobert, P.R. (2000) *Plant J.* 21, 379-385.
- 42 Kuhn, J.M. and Schroeder, J.L. (2003) *Curr. Opin. Plant Biol.* 6, 463-469.
- 43 Finkelstein, R.R., Gampala, S.S.L. and Rock, C.D. (2002) *Plant Cell* 14, 515-545.
- 44 Zhu, J., Gong, Z., Zhang, C., Song, C.P., Damsz, B., Inan, G., Koiwa, H., Zhu, J.K., Hasegawa, P.M. and Bressan, R.A. (2002) *Plant Cell* 14, 3009-3028.
- 45 Leung, J., Leung, J., Merlot, S., Gosti, F., Bertauche, N., Blatt, M.R. and Giraudat, J. (1998) *Symp. Soc. Exp. Biol.* 51, 65-71.
- 46 Himmelbach, A., Yang, Y. and Grill, E. (2003) *Curr. Opin. Plant Biol.* 6, 470-479.
- 47 Hoth, S., Morgante, M., Sanchez, J.P., Hanafey, M.K., Tingey, S.V. and Chua, N.H. (2002) *J. Cell Sci.* 115, 4891-4900.
- 48 Suttle, J.C. and Hultstrand, J.F. (1994) *Plant Physiol.* 105(3), 891-896.
- 49 Rinne, P., Tuominen, H. and Junttila, O. (1994) *Tree Physiol.* 14(6), 549-561.
- 50 Kim, K.S., Davelaar, E. and De Klerk, G.J. (1994) *Physiol. Plant.* 90, 59-64.
- 51 Koornneef, M., Bentsink, L. and Hilhorst, H. (2002) *Curr. Opin. Plant Biol.* 5(1), 33-36.
- 52 Whittle, C.A., Beardmore, T. and Johnston, M.O. (2001) *Trends Plant Sci.* 6(6), 248-251.
- 53 Korthout, H.A.A.J., Martien, P.M., Caspers, M.J., Kottenhagen, Q.H. and Wang, M. (2002) *FEBS Lett.* 526, 53-57.
- 54 Bhat, K.P., Itahana, K., Jin, A. and Zhang, Y. (2004) *EMBO J.* 23, 2402-2412.
- 55 Sanchez, J.P. and Chua, N.H. (2001) *Plant Cell* 13, 1143-1154.
- 56 Hemerly, A.S., Ferreira, P., de Almeida Engler, J., Van Montagu, M., Engler, G. and Inze, D. (1993) *Plant Cell* 5, 1711-1723.
- 57 Sheen, J. (1998) *Proc. Nat. Acad. Sci. U.S.A.* 95, 975-980.

- 58 Tahtiharju, S. and Palva, T. (2001) *Plant J.* 26, 461-470.
- 59 Lu, C., Han, M.-H., Guevara-Garcia, A. and Fedoroff, N.V. (2002) *Proc. Nat. Acad. Sci. U.S.A.* 99(24), 15812-15817.
- 60 Hemberg, T. (1970) *Physiol. Plant.* 23, 850-858.
- 61 Arias, O. and Crabbe, J. (1975) *Physiol. Veg.* 13, 69-81.
- 62 Suttle, J.C. (1998) *Physiol. Plant.* 103, 59-69.
- 63 Heyl, A. and Schmulling, T. (2003) *Curr. Opin. Plant Biol.* 6, 480-488.
- 64 Suttle, J.C. (2000) in *Dormancy in Plants from Whole Plant Behavior to Cellular Control* (Viemont, J.-D. and Crabbe, J., eds.) CABI Publishing, New York, NY. pp. 211-226.
- 65 Inoue, T., Higuchi, M., Hashimoto, Y., Seki, M., Kobayashi, M., Kato, T., Tabata, S., Shinozaki, K. and Kakimoto, T. (2001) *Nature* 409, 1060-1063.
- 66 Suzuki, T., Miwa, K., Ishikawa, K., Yamada, H., Aiba, H. and Mizuno, T. (2001) *Plant Cell Physiol.* 42, 107-113.
- 67 Ueguchi, C., Sato, S., Kato, T. and Tabata, T. (2001) *Plant Cell Physiol.* 42, 751-755.
- 68 Yamada, H., Suzuki, T., Terada, K., Takei, K., Ishikawa, K., Miwa, K. and Mizuno, T. (2001) *Plant Cell Physiol.* 42, 1017-1023.
- 69 Nishimura, C., Ohashi, Y., Sato, S., Kato, T., Tabata, S. and Ueguchi, C. (2004) *Plant Cell* 16, 1365-1377.
- 70 Soni, R., Carmichael, J.P., Shah, Z.H. and Murray, J.A. (1995) *Plant Cell* 7, 85-103.
- 71 Riou-Khamlichi, C., Menges, M., Healy, J.M. and Murray, J.A. (2000) *Mol. Cell. Biol.* 20(13), 4513-4521.
- 72 Saniewski, M., Kawa-Miszcza, L., Wegrzynowicz-Lesiak, E. and Okubo, H. (2000) in *Dormancy in Plants: from Whole Plant Behavior to Cellular Control* (Viemont, J.-D. and Crabbe, J., eds.) CABI Publishers: New York, NY. pp. 227-244.
- 73 Rappaport, L. and Wolf, N. (1969) *Symp. Soc. Exp. Biol.* 23, 219-40.
- 74 Ohkawa, K. (1979) *Sci. Hort.* 10, 255-260.
- 75 Nimi, Y., Endo, Y. and Arisaka, E. (1988) *J. Jap. Soc. Hort. Sci.* 57, 250-257.
- 76 Horvath, D.P., Chao, W.S. and Anderson, J.V. (2002) *Plant Physiol.* 128(4), 1439-1446.
- 77 Chen, J.-G., Pandey, S., Huang, J., Alonso, J.M., Ecker, J.R., Assmann, S.M. and Jones, A.M. (2004) *Plant Physiol.* 135, 907-915.
- 78 Iwasaki, Y., Fujisawa, Y. and Kato, H. (2003) *J. Plant Growth Regul.* 22, 126-133.
- 79 Metraux, M.C. and Kende, H. (1984) *Planta* 160, 73-77.
- 80 Raskin, I. and Kende, H. (1984) *Plant Physiol.* 76, 947-950.
- 81 Sauter, M. and Kende, H. (1992) *Planta* 190, 354-362.
- 82 Sauter, M. (1997) *Plant J.* 11, 181-190.
- 83 Fabian, T., Lorbiecke, R., Umeda, M. and Sauter, M. (2000) *Planta* 211, 376-383.
- 84 Vogler, H., Caderas, D., Mandel, T. and Kuhlemeier, C. (2003) *Plant Mol. Biol.* 53, 267-272.



<http://www.springer.com/978-0-387-25855-3>

Genetic Engineering  
Principles and Methods  
Setlow, J.K. (Ed.)  
2006, XXII, 262 p., Hardcover  
ISBN: 978-0-387-25855-3