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Plant dormancy in the perennial context

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A key feature of the perennial life style in plants is the ability to cease meristem activity and to establish a dormant state in which the meristem is rendered insensitive to growth-promoting signals for some time before it is released and can resume growth. The seasonal cycling between growth and dormancy has received little attention despite its importance for perennial behaviour. In this review, we reconsider seasonal cycles of growth and dormancy in view of a new definition of dormancy as a state within the meristem, together with recent exciting developments in the study of perennials, particularly the identification of common signalling intermediates between flowering time and growth cessation in trees.

Seasonal dormancy is one characteristic of perenniality

Plant growth largely depends on where and when meristem(s) generate new appendages. Meristem activity and its vegetative, inflorescence or floral identities are integrated into diverse life strategies that exist among plants. The timing of flowering and the determinacy of the meristem(s) are important hallmarks in the difference between annual, biannual and perennial life styles. Perennial plants, such as trees, distinguish themselves from other plants in their ability to suspend and resume growth recurrently in response to environmental, and often seasonal, conditions. In principle, perenniality requires at least one indeterminate meristem that continues vegetative growth the following season. The concept of meristem indeterminacy is widely recognized [1,2], but the fact that this meristem must simultaneously attain the capacity to survive the non-favourable season is largely overlooked. Among the various facets of survival, dormancy is pivotal for perennials.

Dormancy is essentially a condition of the meristem

In tree research, dormancy is most frequently referred to as 'absence of visible growth in any plant structure containing a meristem' [3]. Gregory Lang [3] distinguished three types of dormancy: ecodormancy, provoked by limitations in environmental factors; paradormancy, where the growth inhibition arises from another part of the plant; and endodormancy, where the inhibition resides in the dormant structure itself [3]. This pragmatic definition seems a bit unfortunate for several reasons. First, growth within meristems is not readily 'visible' because it is either 'hidden' within organs or minimal. Second, and more importantly, the mere absence of growth is an ambiguous term because dormancy constitutes an inability to resume growth. The definition of dormancy is better dealt with for seed dormancy as the 'failure of a viable intact seed to complete germination under favourable conditions' [4,5]. An additional complication is that growth consists of both cell division and cell elongation and these can occur independently in time and space. For instance, the production of new xylem cells through cell division ceases long before the 'last' xylem cell completes cell elongation during dormancy induction in the vascular cambium of trees. At bud flush, cell elongation of preformed leaves inside the buds precedes new cell divisions. Likewise, cell division is not essential during early stages of germination (imbibition up to radical emergence [6]).

For horticultural and ecological studies, the description of dormancy in the whole structure (bud, cambium and seed) suffices, but is inadequate for unravelling the molecular components that govern transitions into and out of dormancy, particularly at the cellular level. For this purpose, we define dormancy as the inability to initiate growth from meristems (and other organs and cells with the capacity to resume growth) under favourable conditions. Importantly, the capacity to resume growth, be it by elongation or by cell division, distinguishes the meristem and derived organs that are yet to complete their development from surrounding organs that undergo little, if any, growth following release from dormancy (bud scales and seed coat). First, this definition accommodates dormancy residing in the meristems, either cambial and shoot apical or embryonic, but not coat-imposed [5] or physical dormancy [7]. The seed-coat-imposed restriction to germination relies on regulatory circuits that are different from those in the embryo [5,7]. Second, the inability to initiate growth clearly distinguishes between endodormancy in trees and the subsequent stage, when growth capacity is restored in late winter but growth itself is not yet initiated. This essentially different state, often termed ecodormancy [3], is not covered by our definition. Third, the proposed definition includes axillary buds that are dormant because of correlative inhibition or apical dominance; they fail to grow unless the inhibiting organ is removed from the plant.

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An important aspect of dormancy is its quantitative nature: the intensity can vary with time and conditions, for example, individual tree buds have different chilling requirements for dormancy release. Last, but not least, irrespective of the inability to initiate growth during dormancy, active physiological and molecular changes take place in the morphologically unchanging dormant structures [8–10].

We will limit the, by no means exhaustive, discussion to recent breakthroughs with respect to dormancy regulation in trees that primarily use photoperiodic signals for growth cessation and dormancy. We hope to establish new cornerstones for the processes involved in the recurrent activity–dormancy cycling in perennials. Because excellent reviews cover physiological, hormonal and the few molecular aspects of dormancy in woody plants [9,11,12], our focus will be primarily on regulatory aspects of dormancy.

Growth cessation: enabling dormancy establishment

The first step towards establishing dormancy is growth cessation, which is provoked by various informative and, over the years, stable environmental cues, such as photoperiod, cold or drought. For >60 years, photoperiod has been known to govern growth cessation of many trees in temperate climates, including the model tree poplar (*Populus* sp.) [13,14] (Figure 1). Leaves perceive photoperiod and emit a signal to the apex, where inactivity is installed [15,16]. This stimulus, present in short-day-exposed leaves, can be transferred by leaf extracts to actively growing seedlings and via grafts [17,18]. Until recently, except for phytochrome, the components of the signal transduction chain acting downstream of the critical day length were unknown [19,20].

A breakthrough was achieved with the identification of poplar FT and CONSTANS (CO) as mediators of short-day signals for growth cessation [21]. When *FT1* and *CO* homologues of poplar (*Populus trichocarpa*) were overexpressed in transgenic aspen (*Populus tremula* × *P. tremuloides*), growth did not stop upon exposure to short days [21]. Furthermore, in poplar ecotypes originating from various latitudes and differing in their critical daylength for growth cessation, the phase of *CO* expression relative to the light period was decisive for its target *FT1* to be expressed and growth to continue. The down-regulation of FT by interference RNA (RNAi) led to growth cessation and bud set, independently of day length [21]. This fact and the absence of *FT1* expression after only three short days argue for an association of *FT1* with growth maintenance rather than for the induction of dormancy. Prior to these results in poplar, the CO/FT module had been invoked in

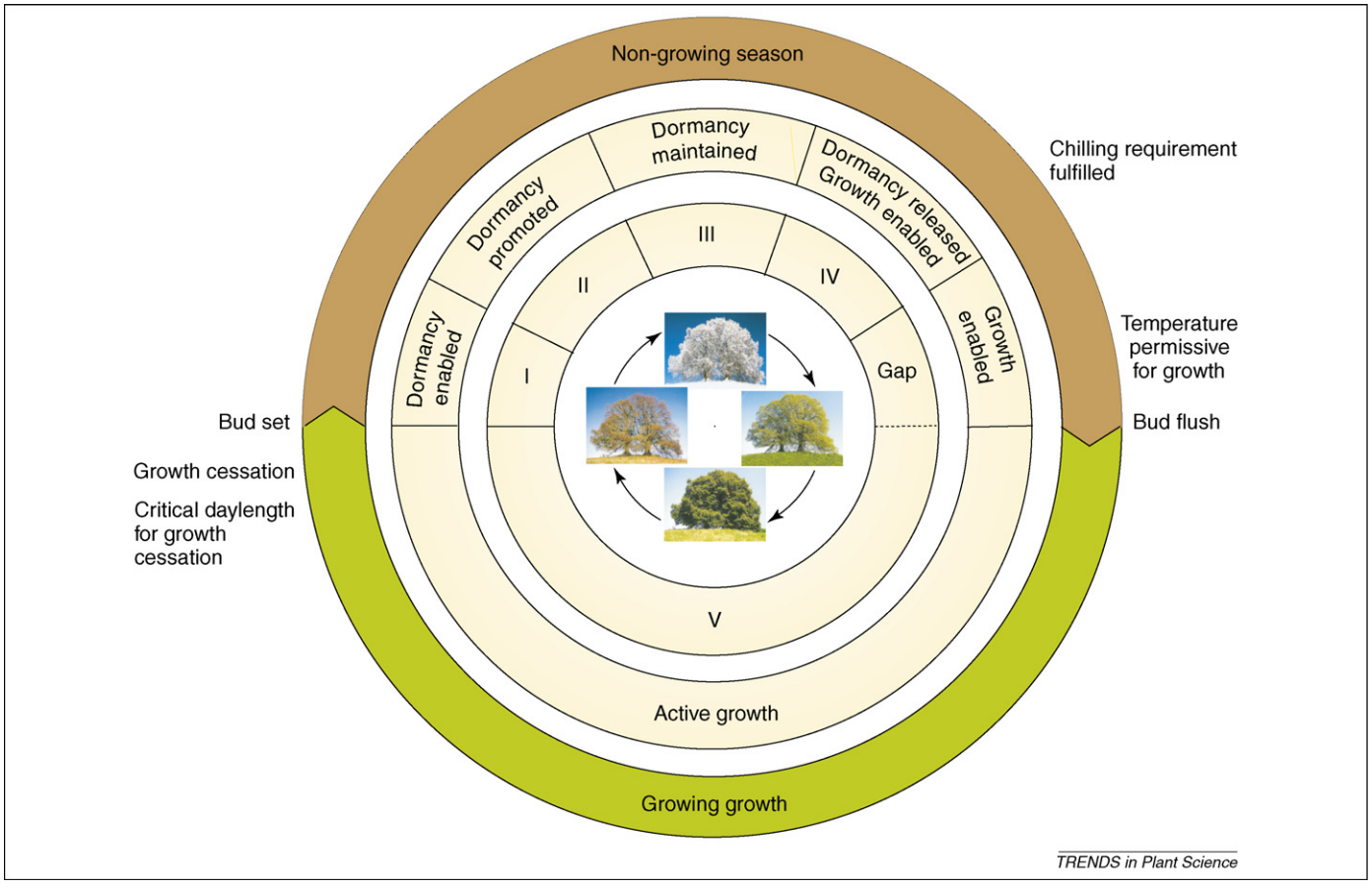

Figure 1. Transitions in seasonal growth–dormancy cycling in *Populus* sp. Poplars synchronize the onset of the dormant period mainly with changes in day length that are sensed by phytochromes. Bud flush and bud set delimit the growing season. Prolonged exposure to chilling temperatures will release plants from dormancy. Growth resumes once the temperature passes a critical threshold. Absence of growth before and after endodormancy is caused by different environmental factors. The inner circles depict the growth–dormancy status and the corresponding meristem stages: I, cessation of cell division; II, establishment of dormancy or loss of responsiveness to growth-promoting signals; III, maintenance of dormancy; IV, release from dormancy state or cell cycle machinery regaining responsiveness to growth-promoting signals; and V, resumption of cell division. 'Gap' between stage IV and V denotes the phase where growth does not occur because of purely environmental restraints. Additional morphological events are covered in detail elsewhere [29,74,75].

floral meristem transition. The regulon underlies photoperiodic flowering in long- and short-day plants and the tuberization response in short-day-requiring wild potatoes (*Solanum tuberosum*) [22,23]. In tomato (*Lycopersicon esculentum*), the FT orthologue SINGLE-FLOWER TRUSS (SFT) induces flowering in day-neutral tomato and tobacco (*Nicotiana tabacum*). Furthermore, SFT-dependent graft-transmissible signals can substitute for long- and short-day stimuli in other plants [24]. Similarly, transcript abundance of poplar FT1 and FT2, a second FT orthologue, was high during flower initiation in long days and low during growth cessation [21,25]. Thus, FT is apparently a versatile regulator that has been recruited for environmental control of meristem transitions, such as floral transition and growth cessation. *MOTHER OF FT AND TFL 1* (*MFT*), another FT family member with an FT-like function in *Arabidopsis thaliana* [26], is also expressed during dormancy initiation in vegetative and floral buds of poplar. However, its overexpression or down-regulation had no pronounced effects on the annual growth cycle (R. Mohamed, PhD thesis, Oregon State University, 2006). Interestingly, a poplar orthologue of *TERMINAL FLOWER 1* (*TFL1*), another member of the FT family with an FT-opposing effect on flowering in *Arabidopsis* [27], is expressed in post-dormant buds up to bud flush and re-appears in newly formed axillary buds in June (R. Mohamed, PhD thesis, Oregon State University, 2006). Overexpression of *TFL1* in poplar seems to postpone vegetative bud flush under field conditions (R. Mohamed, PhD thesis, Oregon State University, 2006). If FT function is associated with growth in poplar, TFL1 might counteract FT at growth resumption in one possible scenario.

Now that the difference in critical day length for growth cessation can be correlated with the phase of *CO* expression [21], the coincidence model would suggest the involvement of circadian clock regulators in growth cessation. When the expression of *LONG HYPOCOTYL* and *TIMING OF CAB EXPRESSION*, which make up the central oscillator of the clock, was studied in dormant chestnut (*Castanea sativa*), circadian rhythms were disrupted by changes in temperature that neither caused growth cessation nor dormancy [28]. Warm temperature restored the correct circadian rhythms of these two genes in chestnut stems and yet the stems remained completely endodormant. Thus, further experimentation is needed to demonstrate a role for circadian clock regulators in dormancy regulation.

Dormancy establishment: rendering the meristem insensitive to growth-promoting signals

Once growth has ended, the dormant state becomes progressively established and results in the complete inability of the meristem cells to respond to growth-promoting signals (Figure 1). Currently, we know little about the changes that occur after growth cessation and before dormancy establishment, in part because this phase is masked by the concurrent bud formation. The bud is required for successful survival but not for dormancy. Poplar (*Populus tremula* × *P. alba*) overexpressing *ABSCISIC ACID INSENSITIVE 3* (*ABI3*) and birch (*Betula pendula*) with a dominant-negative version of *ETHYLENE TRIPLE RESPONSE 1* (*ETR1*) do not form buds and yet become dormant, indicating that bud formation is independent of dormancy [29,30]. In poplar cambium, where dormancy establishment is not obscured by morphogenetic changes, post-transcriptional alterations in the components of the cell-cycle machinery can be unequivocally linked to the cell-cycle arrest at dormancy induction. Prior to the transition to endodormancy, E2F phosphorylation is elevated, whereas retinoblastoma phosphorylation decreases after the transition to endodormancy [31].

Given that dormancy induction requires a period of consecutive short days, factors involved in growth cessation could also act in the subsequent establishment of endodormancy. Although not suitable for a direct role, because its expression terminates after three short days, down-regulation of FT could trigger subsequent events leading to dormancy. It is striking that the expression of the poplar homologues of *FCA* is up-regulated when apical dormancy is triggered in poplar (A. Rohde, unpublished). The induction of *FCA*-like genes during apical dormancy is particularly interesting in the context of the recent finding that FCA can bind abscisic acid (ABA) [32]. Although the influence of ABA on dormancy is controversial [11], ABA might affect aspects of dormancy acquisition. ABA peaks in poplar apical buds after growth cessation and before bud set [29]. FCA might be involved in setting the scene for dormancy. In *Arabidopsis*, *FCA* down-regulates *FLC* [33], but ABA binding prevents the formation of active FCA–FY complexes that are required for *FLC* repression and the progression to flowering [32]. Furthermore, ABSCISIC ACID HYPERSENSITIVE 1 (ABH1) in combination with FRIGIDA can enhance FLC levels in *Arabidopsis* [34]. Thus, in both cases, during the initial stages of dormancy establishment, ABA could act via ABH1 or FCA to install and/or maintain high levels of *FLC* during dormancy. Interestingly, the naturally occurring *evergrowing* mutant of peach (*Prunus persica*), which fails to cease terminal growth and form buds, lacks a gene cluster of six MICK-type MADS box genes [35,36]. However, in the absence of established functional information, an FLC function to repress *FT* (or other targets) during poplar bud dormancy remains hypothetical.

Independent of a putative role for *FLC*-like genes, a role for chromatin remodelling in dormancy is attractive [12]. The tremendous alteration of meristem transcriptome during the transition from growth to dormancy in the vascular cambium of poplar [37] suggests that a global mechanism could underlie the massive reprogramming. Virtually nothing is known about the regulation of histone modifications during the activity–dormancy cycle in trees. The only example linking epigenetic modifications to dormancy involves DNA methylation and histone multi-acetylation in dormancy release of potato tubers [38,39]. An intriguing observation in this regard is the induction of a poplar homologue of *FERTILISATION-INDEPENDENT ENDOSPERM DEVELOPMENT* (*FIE*) as part of the complex interacting with histone deacetylases during the induction of dormancy in both the cambium and apical bud [37] (A. Rohde, unpublished).

Dormancy maintenance

Once dormancy is established, it is maintained by hitherto unknown mechanisms. From a purely mechanistic point

of view, meristem cells could be insulated from growth-promoting signals, such as gibberellins. Indeed, meristematic cells get symplasmically isolated upon transition to endodormancy by disconnection of the plasmodesmatal circuitry to neighbouring cells [40,41]. Whereas plugging the plasmodesmatal connections with callose will prevent gibberellin transport, auxin (and other regulatory molecules) might rely on different transport systems. Auxin is transported by specialized carriers, whose mRNAs in poplar cambium are detectable after endodormancy is established [37] (A. Karlberg and R. Bhalerao, unpublished). Furthermore, auxin levels do not change in cambial cells throughout the entire activity–dormancy cycle, but the responsiveness to auxin does [42,43]. The role of gibberellins, and the putative significance of their exclusion from the meristem, is unclear in growth re-initiation. At least in poplar cambium, gibberellin application even after chilling exposure does not induce cell division, in contrast to auxin (R. Bhalerao, unpublished).

Release from endodormancy: enabling growth

Dormancy release requires exposure to chilling temperatures (Figure 1). Back in 1960, Pierre Chouard [44] noted and critically assessed the similarities of release from dormancy and vernalization. Like vernalization, dormancy is released not by short-term but by long-term exposure to low temperatures, and chilling restores the ability to grow but does not promote growth. A key feature of vernalization in *Arabidopsis* is the mitotically stable repression of *FLC* after prolonged exposure to low temperatures [45]. This epigenetic repression of *FLC* involves a series of histone modifications that implicate the gene products of *VERNALIZATION1*, *VERNALIZATION2*, *VERNALIZATION INSENSITIVE 3*, and *LIKE HETEROCHROMATIN PROTEIN 1* [46–49]. Recently, *FLC*-like genes have been shown to be differentially regulated during the completion of the chilling requirement in vegetative buds of poplar [50].

Even in view of these obvious similarities, there are crucial differences between vernalization and release from dormancy. Vernalization is thought to occur effectively only in actively dividing cells [51,52]. In contrast to vernalization, endodormancy is released by exposure to low temperatures after termination of cell division. However, there are a few reports of growth occurring at a slow rate during dormancy [44,53]. While cells do not divide in dormant cambium [31,42], the lack of cell division in the apical meristem of buds has yet to be unequivocally demonstrated. Furthermore, *Arabidopsis* seeds require less chilling for dormancy release than for vernalization and no obvious dormancy phenotypes have been described in vernalization mutants. Thus, the measurement of cold accumulation might be shared between vernalization and dormancy, but memorization of experienced cold through a mitotically stable repression is not required for dormancy: the cells that cease and resume growth are identical meristem cells.

Dormancy resetting in a perennial context

An important issue in a perennial context is that once dormancy is released and growth resumes, resetting needs to occur. The state in which dormancy induction is possible together with the subsequent need for the chilling is re-established. If epigenetic mechanisms were involved in the induction of dormancy, epigenetic marks would have to be reset. In annual plants, resetting of an epigenetically fixed, vernalized state happens during meiosis and before seed dormancy [48]. Resetting in vegetative meristems has to occur through another, so far undescribed mechanism. A few studies have suggested an alternative devernalization mechanism that is triggered by accumulated heat in summer [1,44,54]. In summary, any simple analogy of vernalization and dormancy release is highly speculative at the moment.

Commonalities of seasonal dormancy with other types of dormancy

The ability to form buds and to undergo cycles of growth and dormancy has been central to the evolution of the perennial life strategy. After the evolution of branching, the subsequent acquisition of the bud structure enabled growth to be arrested in some, but not all, meristems. With this strategy, plants could adapt their branching to a greater variety of conditions, particularly seasonal environments. In this sense, bud dormancy evolved as a morphogenetic strategy and, secondarily, was adopted for adaptive purposes [55]. The bud appeared ~100 million–400 million years earlier than the seed. It was not until the rapid warming of the Earth's climate that annual life forms were favoured [55]. This evolutionary trajectory implies that endodormancy is derived from the evolutionarily older paradormancy and might still share molecular mechanisms today. Similarly, the most recent, independently developed seed dormancy, which synchronizes seed germination to periods when seedling establishment is likely to be successful, might also share commonalities with seasonal endodormancy. In both axillary buds and seeds, dormancy has been systematically characterized in model plants, particularly at the genetic and molecular levels [5,7,56–59]. Although extensively studied, comparatively little is known about the induction of dormancy in these systems compared with the release from dormancy (Figure 2). An understanding of these systems might nevertheless usefully guide the quest for regulators of seasonal endodormancy. Although the inducing mechanisms might not be shared directly, similar signalling circuits could be adopted, as has become apparent for regulators of the transition to flowering (Figure 2), and meristem inactivation might be controlled by common downstream factors.

For example, basipetally transported auxin from the apex and young leaves is the major inhibitor of axillary bud outgrowth, but does not act directly within the inhibited buds (Figure 2). Several novel components, such as MORE AXILLARY GROWTH (MAX), MAP KINASE KINASE 7 and RAMOSUS (RMS), have been implicated in relaying the auxin signal into the buds (Figure 2) [59–64]. During seed development, cell division in the embryo is arrested ~10 days after pollination [65]. Thereafter, the embryo matures and attains dormancy (Figure 2). To date, only three embryo-growth-arrest mutants are known (Figure 2) [65]. For dormancy release in seeds, several important

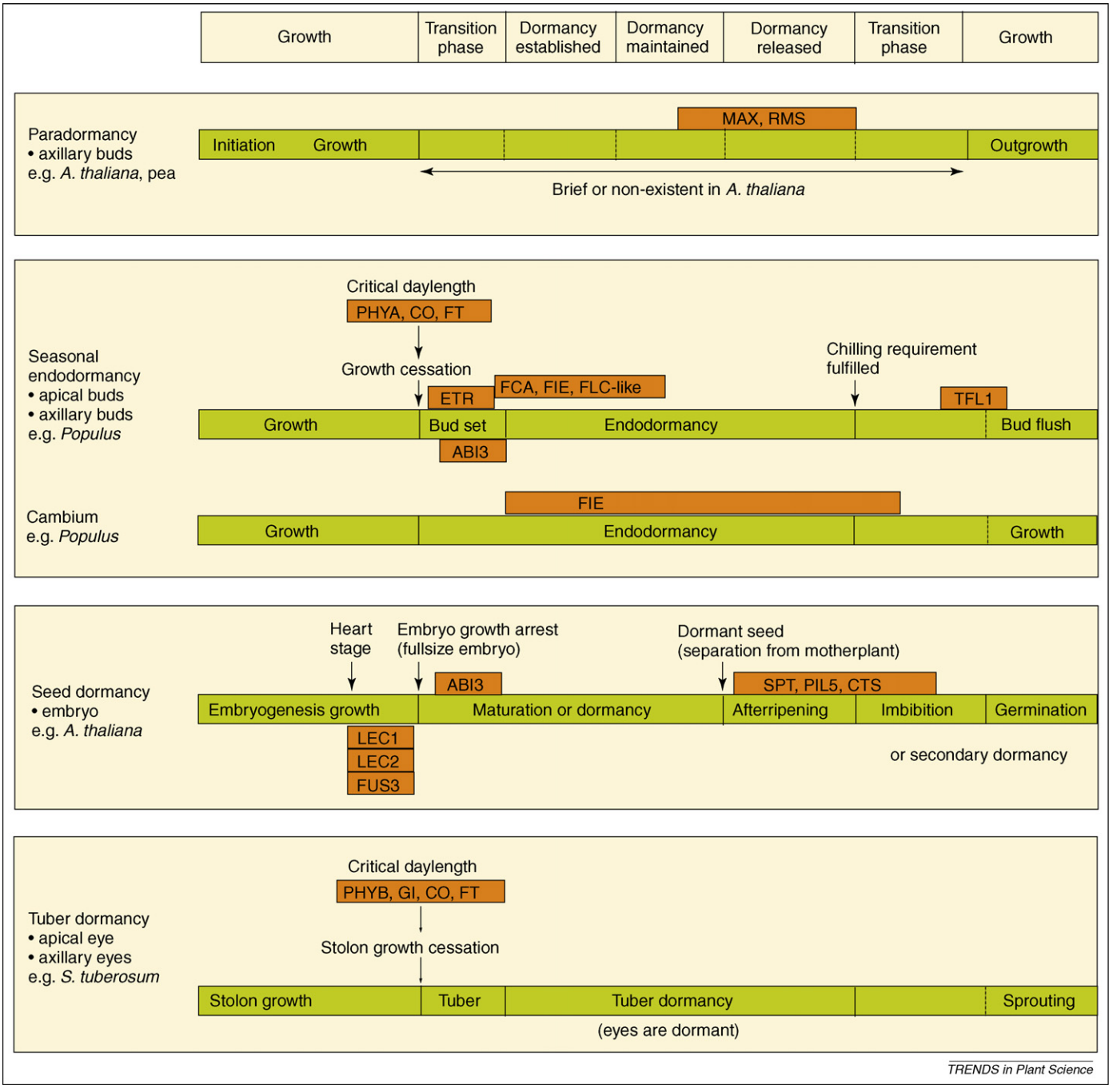

Figure 2. Seasonal dormancy compared with paradormancy, seed dormancy and tuber dormancy. For each of the processes, the description of the events is given together with the major regulators identified. Tuber dormancy represents a specific case where an otherwise annual plant generates a vegetative perennating organ. Secondary dormancy might be installed when conditions during imbibition are not favourable for germination and seedling establishment. Germination is considered to consist of elongation of the embryo axis and radicle protrusion.

regulators have been identified that together act on ABA–gibberellin homeostasis (Figure 2) [66–68]. Furthermore, ethylene interferes with ABA signalling, thereby promoting germination [69,70]. New regulators of either dormancy or dormancy release are expected from the cloning of the seven *DELAY OF GERMINATION* (*DOG*) loci, which was identified by using a quantitative trait locus (QTL) approach in a Landsberg *erecta* (L*er*) × Cape Verde Islands (Cvi) cross [71]. *DOG1*, the only cloned gene, is expressed specifically in the seed and is a member of a novel, plant-specific gene family, the function of which has still to be determined [72].

Together, these primarily hormone-related signalling cascades might act during seasonal endodormancy, and, so far, gibberellins and auxin at least are known to play a role in poplar cambium. One example of signalling mechanisms acting across different types of dormancy is the dominant-negative ETR1 transgenic birch, which not only has a shallower seasonal endodormancy, but also a reduced apical dominance [30]. Furthermore, in the past two years, many genes have been identified for their differential expression during the transition from active to dormant meristems through genomic approaches [10,37] (R. Bhalerao, unpublished; T. Ruttink and A. Rohde, unpublished).

In poplar, ~25% of the genes that were differentially expressed during the transition from growth to dormancy in apical buds were also detected in cambium with similar dynamics (T. Ruttink and A. Rohde, unpublished).

Perspectives

Unexpected discoveries have linked regulons of flowering time control with seasonal growth cessation, illustrating that regulatory hierarchies of developmental processes might be recruited into the control of seemingly unrelated processes, and opening new avenues to critically assess regulatory components for an involvement in seasonal endodormancy. Recruitment of similar transcription modules indicates that the comparative approach should help to define the commonalities and particularities of different types of dormancies, particularly in terms of gene expression (Figure 2). Alternative, unbiased approaches, such as the identification of the causative genes within dormancy-related QTL regions, should complement the comparative approach. The integration of gene expression information with genetic approaches, such as QTLs or association genetics, is currently undergoing a renaissance in poplar research thanks to the availability of the genome sequence [73]. These approaches should identify regulators that are important under natural conditions. Furthermore, dormancy provoked by other inducing signals, such as cold or drought (in synergy with photoperiodic signals or not), deserves comparison with photoperiodically induced dormancy. We are convinced that it is of vital interest for molecular research to comprehend dormancy as a condition in the meristem (and other growth-resuming organs and cells).

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