

Signals and Mechanisms in the Control of Plant Growth

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Abstract Plant growth is mediated by three fundamental processes: cell growth, division, and expansion. The mechanistic analysis of their contributions are complicated by the observation that the balance of their contributions to organ growth are not hard-wired. Reduced cell proliferation, irrespective of whether this is caused by decreased cell growth or diminished cell division, can be, at least partially, compensated for by increased cell expansion. It is therefore argued that for a functional understanding of how gene regulatory networks control growth of the plant body, it is essential that all cellular parameters contributing to organ growth are quantified in concert. Plant growth behavior is exquisitely responsive to environmental change. Cell growth, division, and expansion, in aggregate, are promoted by nutrient availability and inhibited by abiotic stress. Recent studies that address how light intensity, CO₂ concentration, water activity, and temperature have complex effects on proliferation, cell expansion, and endoreplication that affect leaf organ growth are reviewed. Root growth rates and patterns are also very sensitive to mineral nutrient concentration and distribution. The mechanistic basis of plant organ growth still remains unknown; but such knowledge is critical for rational approaches to manipulate plant growth. Critical steps towards this goal are discussed.

1

Introduction and Background

Plants adapt exquisitely to their environment: physiology and metabolism change diurnally and in response to many environmental conditions, and reproductive development is generally sensitive to day length, temperature, or other proxies of seasonal change. The most fundamental adaptation to environmental change in plants is altered growth behavior, involving changes to root or shoot growth patterns, rates, or both.

Despite their fundamental importance for our understanding of plant growth, for rational approaches to sustainably enhance yields in agriculture and forestry and, ultimately, for human welfare, we still understand surprisingly little about the mechanisms that govern growth in plants. In this chapter, I will consider the signals and genetic mechanisms involved in controlling growth in aerial and underground organs.

1.1

Distinct Processes Contribute to Plant Growth

At the whole plant level, growth of the plant body proceeds by the linear extension of stems and branches, the production of leaf or floral organs, and the elongation and branching of roots, mediated by apical, axillary, and lateral meristems. Secondary growth, or radial thickening mediated by cambial cells, contributes to body size increase in many plants, but will not be considered further in this chapter. Primary stem or leaf and root organ growth, here defined simply as an increase in volume, proceeds in two stages, which I will call phase I, during which cells multiply in cycles of growth and division; and phase II, during which cells cease dividing but expand until differentiation is completed. High rates of proliferation are observed in meristems, in young leaf and floral primordia, but not in stem cells and the stem cell niche (Fig. 1).

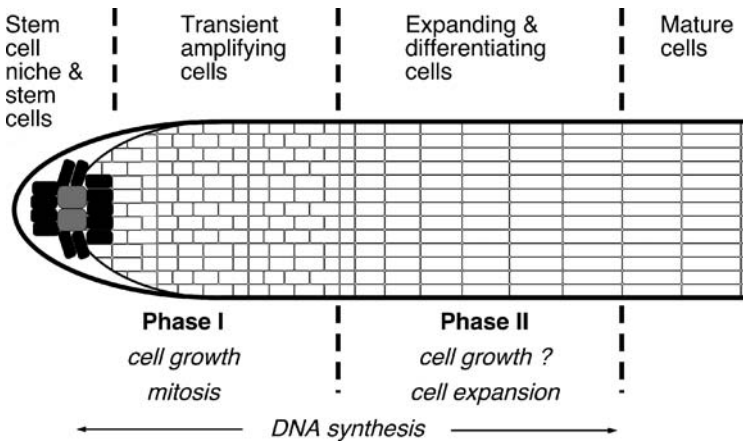


Fig. 1 Schematic representation of the root apical meristem illustrating the different zones of growth and the positional extent of various growth processes

1.2

Cell Growth

In phase I, cell growth alternates with division in mitotic cells. Cell growth is a prerequisite for division in meristems and organ primordia, and is driven by the increase of cell mass by synthesis of macromolecular cell constituents (Jorgensen and Tyers 2004). Ribosomes limit macromolecular synthesis and, therefore, their synthesis and its regulation is at the nexus of growth control. For example, yeast cells commit ~50% of their total transcription activity and a large fraction of their energy budget towards building ribosomes (Warner 1999) and quantitative studies reveal a strong positive correlation of ribosome synthesis with cell growth (Planta 1997; Warner 1999). There is good

evidence that impaired ribosome biosynthesis reduces plant growth (Van Lijsebettens et al. 1994; Weijers et al. 2001; Horvath and Bogre, this volume), but no detailed information is yet available on how well ribosome biosynthesis correlates with growth activity in plants. The expression of many components of the plant ribosome is regulated transcriptionally (McIntosh and Bonham-Smith 2006), but it is still poorly understood how ribosomal RNA and protein synthesis for ribosome production are coordinated mechanistically (for review, see McIntosh and Bonham-Smith 2006).

Cell growth is under control of the target of rapamycin (TOR) pathway, which couples nutritional cues to the regulation of ribosome biosynthesis, the rates of protein synthesis and proliferation. The TOR pathway interacts with the PI-3-kinase pathway, which mediates growth factor cues, and this interaction insures coordinate cellular growth responses (Arsham and Neufeld 2006; Jorgensen and Tyers 2004). The TOR pathway has been well characterized in animal and yeast systems, but much detail remains to be uncovered in plants: Orthologs of the TOR kinase, and of some additional components of the TOR signaling pathway have been identified in plants (Bogre et al. 2003; Menand et al. 2002; Wang et al. 2003), but their functional significance for plant cell growth control, specifically for coupling environmental change to growth responses, are only beginning to be examined in detail (Mahfouz et al. 2006). Likewise, plant homologs of PI-3-kinases and their effectors, the AGC kinases have been identified (Wang et al. 2003). At least one AGC kinase has been shown to be responsive to auxin and cytokinin growth regulator inputs (Anthony et al. 2004), and IRE (an AGC kinase) positively regulates root hair tip growth (Oyama et al. 2002). However, many gaps need to be filled until we understand the mechanisms of how growth regulator and nutrient inputs converge on cell growth control in plants.

1.3

Cell Division

In contrast, the mechanisms controlling cell division are much better understood than those regulating cell growth in plants (see Inze and De Veylder (2006) for an excellent recent review). Components of the plant cell cycle machinery (cyclins, cyclin-dependent kinases), orthologs of the retinoblastoma (Rb) gene, and E2F/DP-type transcription factors were identified based on their sequence homology (Vandepoele et al. 2002). Largely based on gain-of-function studies with transgenic plants over- or ectopically expressing cell cycle regulators and expression analysis, the following view is emerging: In association with CDKA (A-type cyclin-dependent kinase), D-type cyclins are involved in controlling the entry into the cell cycle (Menges et al. 2006, Riou-Khamlichi et al. 1999), whereas A- and B-type cyclins, in association with CDKA and CDKB play a major role in S-phase and entry into M-phase, respectively (Doerner et al. 1996; Weingartner et al. 2003). As in animal systems,

the E2F/DP and related genes, promote S-phase and DNA synthesis, but are also involved in controlling the switch between mitotic cell cycles and the endoreplication cycle. Likewise, CDK inhibitors function in post-translational control of cyclin-CDK complex activity. Anaphase promoting complex (APC) proteolytic activity at the metaphase-to-anaphase transition insures the irreversible directionality of cell cycle progression, as in other model systems.

Cell growth is coupled to cell division progression by mechanisms that monitor cell size. For example, in yeast, coupling of growth to cell cycle entry converges on the regulation of G1-type CLN3 cyclin abundance (Morgan 2007), although this view may be too simplified (Jorgensen and Tyers 2004). CLN3 abundance is regulated at the transcriptional, translational, and post-translational level (MacKay et al. 2001; Morgan 2007; Polymenis and Schmidt 1997). In aggregate, these mechanisms result in a steep stimulus-response coupling (ultrasensitive response) of CLN3 protein levels, and hence of CLN3-CDK complex activity, to the rate of mRNA translation by ribosomes, which reflects the activity of the TOR and other growth regulating pathways. Cell cycle entry in plants requires D-type cyclins. In Arabidopsis, cyclin D3;1 mediates the stimulatory effect of cytokinins on proliferation, while cyclin D2 abundance is responsive to sucrose levels (Riou-Khamlichi et al. 1999, 2000). Cyclin D3;1 is a labile protein (Planchais et al. 2004), as would be expected of a limiting regulator responsive to potentially rapidly changing environments. Moreover, cyclin D3;1 promotes the G1/S transition (Menges et al. 2006). Based on this limited information, it is therefore reasonable to predict that key aspects of the mechanisms that couple cell growth to cell division are conserved in all eukaryotes.

1.4

Cell Expansion

After cells pass through the domain with high rates of cell growth and division, they cease dividing and cell size rapidly becomes larger. This transition from phase I to II is visually distinct in root meristems, whereas in leaf organs this transition is morphologically less conspicuous. Cell expansion in phase II is not driven by macromolecular synthesis but is the result of turgor-driven water uptake and concomitant cell wall loosening. The generation of increased osmotic pressure requires the activities of three major proteins or protein complexes in the tonoplast membrane: The V-type H^+ ATPase, H^+ pyrophosphatase and aquaporins (see Maeshima 2001 for review). This is balanced by cell wall loosening that permits the cell to expand mostly in one direction, and which involves several activities including expansins, xyloglucan endotransglycolase/hydrolase (XET), endo-(1,4)- β -D-glucanase, and hydroxyl radicals (see Cosgrove 2005 for review). In quantitative terms, cell expansion contributes most to organ growth: during cell expansion, volume increases from 20- to 1000-fold. Thus, the extent of cell growth and division

during phase I define the potential for organ growth by producing the cellular building blocks; during phase II, this latent ability is fulfilled during cell expansion.

The phase I/II boundary marks a transition of the cellular mechanism that mediates organ growth: from growth by cell production to organ growth by cell expansion. However, not all processes associated with organ and plant growth change at this transition. DNA synthesis persists during this transition, but in the absence of division, it leads to endoreplication. Therefore, DNA replication can be considered as the process that frames the entire organ growth process. In *Arabidopsis*, endoreplication can result in ploidy levels of up to 64C (with 1C being a haploid genome equivalent), indicating that cells undertake up to five additional rounds of DNA synthesis without dividing. In *Arabidopsis* leaves, cellular DNA content is positively correlated with mature, fully expanded cell size (Melaragno et al. 1993), however, in roots no such correlation was found (Beemster et al. 2002). DNA synthesis, and with it endoreplication and cell expansion, is thought to cease when cells become fully differentiated and primary organ growth is completed.

Although expanding cells increase their size by a different mechanism than cells growing in the proliferative zone, they continue entering the DNA replication cycle as long as they undertake endoreplication cycles. The bulk of the volume increase in expanding cells is mediated by inflation of the vacuole, but it is likely that the cytoplasm must also increase in mass to insure that the necessary concentration of reactants is thermodynamically favorable. This raises the interesting, and as yet unresolved, question whether the onset of S-phase in endoreplicating cells is also coupled to proxies of cell growth such as the rate of mRNA translation.

2

Regulation of Growth

Much progress has been made in identifying and functionally characterizing components of the plant cell division apparatus (Inze and Veylder 2006), and the mechanisms involved in cell expansion are also beginning to be quite well understood (Carol and Dolan 2006; Cosgrove 2005; Tsukaya 2006). In contrast, cell growth control is mechanistically still less well understood. Based on the preceding analysis of plant growth processes, I propose the existence of two major growth control points in plants likely to be sensitive to developmental or environmental inputs. The first is suggested to co-regulate cell growth and the onset of the cell cycle; the second is the switch of growth mechanisms at the phase I/II boundary to suppress mitosis and activate cell expansion. The identification of components involved in these control points, the mechanisms by which they operate and how they are coupled to cues will be major milestones to improve our understanding of plant growth control.

Plant Growth Signaling

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