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Regulation of plant lateral-organ growth by modulating cell number and size

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Leaves and floral organs grow to distinct, species-specific sizes and shapes. Research over the last few years has increased our understanding of how genetic pathways modulate cell proliferation and cell expansion to determine these sizes and shapes. In particular, the timing of proliferation arrest is an important point of control for organ size, and work on the regulators involved is showing how this control is achieved mechanistically and integrates environmental information. We are also beginning to understand how growth differs in different organs to produce their characteristic shapes, and how growth is integrated between different tissues that make up plant organs. Lastly, components of the general machinery in eukaryotic cells have been identified as having important roles in growth control.

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Introduction

The final size and shape of plant organs must be regulated in response to the developmental stage and the environment to optimally exploit the plant's surroundings — whether leaves to catch the sunlight, or floral organs to attract pollinators. Nevertheless, however favourable or stressful the environment, genetic control maintains a restrictive bracket on the sizes to which any organ will finally grow, and genetically different individuals show reproducible differences in the size of lateral organs.

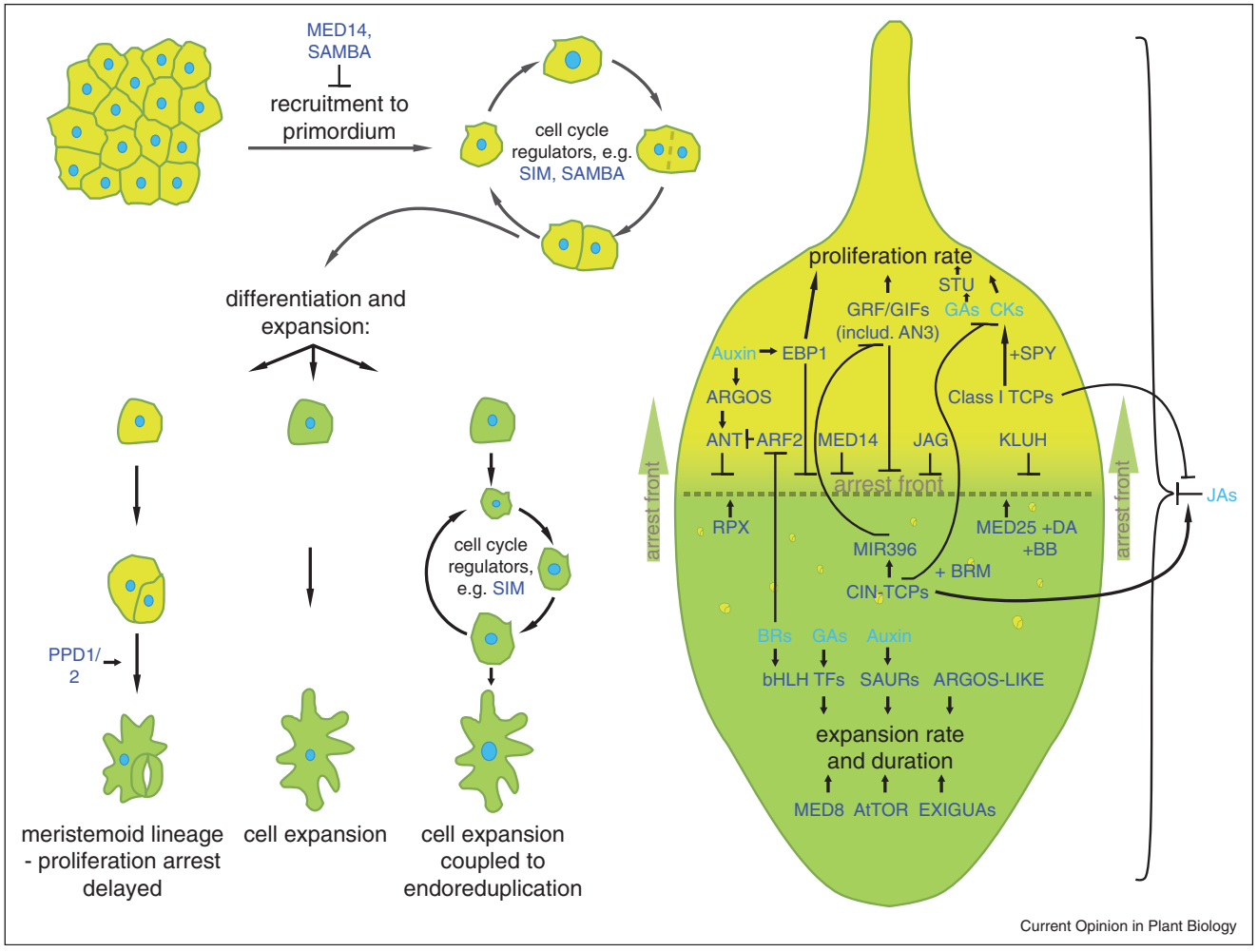
The growth of lateral organs from meristematic primordium to final size can be viewed as a stepwise process, starting at founder cell recruitment to the primordium, followed by two phases of growth: firstly cell proliferation (cell growth coupled to division), then cell expansion (cell growth without division) [1]. Regulation of growth can therefore act firstly on the number of cells recruited to the primordium, secondly on the rate of proliferation, thirdly on the timing of proliferation arrest and finally on the rate and duration of expansion [2]. Several genetic factors are known to act at each of these potential control points [3–5]. As many of the factors involved in organ size have been extensively reviewed elsewhere, these have been summarised in Figures 1 and 2. In this review we will focus on new additions from the last two years to our understanding of growth and size control.

Temporal and spatial control of proliferation

While there are only a few cases where differences in final organ size appear to be due to differential recruitment of founder cells to the primordium [6,7] or to result from changes in the rate of cell proliferation [8], the timing of proliferation arrest is affected in a large number of mutants with altered organ sizes. Thus, this step seems to represent a major control point, even though the paucity of examples for control at the level of recruitment or proliferation rate may in part also reflect technical difficulties in clearly demonstrating such effects. Proliferation in leaves does not arrest simultaneously throughout the primordia, but first at the tip, while more basal cells (as well as stomatal and procambial cells in the distal regions) continue proliferating (Figure 1) [1]. The dynamics of the 'arrest front' that thus seems to move towards the primordium base have been characterised in more detail by two recent studies in *Arabidopsis* [9,10••]. The boundary between proliferating and non-proliferating regions appears rapidly, then remains at a rather constant distance from the leaf base for some days, before rapidly disappearing as all pavement cells stop proliferating. Parallel transcriptional analysis suggests a role for chloroplast retrograde signalling in promoting the onset of cell expansion, thereby coupling photosynthetic ability to the appearance of the arrest front [10••].

In addition to basic regulators of the cell cycle that limit proliferation in lateral organs, several transcription factor families contribute to the control of proliferation arrest (Figure 1). The TEOSINTE-BRANCHED1/CYCLOIDEA/PCF (TCP) family is split into two classes that antagonistically control growth in a spatially restricted and often redundant manner. The class I TCPs studied so far promote proliferation and cell growth in a context-dependent fashion [11,12], whereas the class II (also known as CIN-type TCPs) promote differentiation and the progression of the cell-cycle arrest front [13]. CIN-TCPs upregulate the expression of microRNA *MIR396*, which targets members of the proliferation-promoting *GROWTH-REGULATING FACTOR* (*GRF*) family of

Figure 1



Principal genetic factors controlling growth by proliferation and expansion. Factors identified in *Arabidopsis* are summarised, with cellular events shown on the left, and whole leaf processes illustrated on the right. Cells are first recruited to the primordium and then go through several rounds of the cell cycle under tight genetic regulation (yellow cells/leaf area: for examples, see [34]). Exit into expansion (green cells/leaf area) is regulated by several factors, such as cell position relative to the arrest front, which moves from the leaf apex to its base and is itself regulated by a variety of genetic pathways. Cells committed to the stomatal lineage (left) or vascular lineage (not shown) may continue to divide. Expansion is often coupled to endoreduplication, which as a modified version of the cell cycle is subject to some of the same controls. For pathways not discussed in the text, the reader is referred to more detailed reviews [2–5,34]. Writing key: black, processes; dark blue, genes; light blue, phytohormones. Bars, repression/inhibition; pointed arrows, promotion. *Abbreviations*: *ANT*, *AINTEGUMENTA*; *ARF2*, *AUXIN RESPONSE FACTOR2*; *AtTOR*, *A. thaliana TARGET-OF-RAPAMYCIN*; *BRM*, *BRAHMA*; *EBP1*, *ErbB-3 EPIDERMAL GROWTH FACTOR BINDING PROTEIN1*; *GIF, GRF-INTERACTING FACTOR*; *PPD*, *PEAPOD1/2*; *SAUR*, *SMALL AUXIN UP RNA*; *SPY*, *SPINDLY*; TF, transcription factor. Other abbreviations are found in the text.

transcription factors [14]. The overexpression of *MIR396* restricts proliferation in favour of endoreduplication, and when combined with mutants in *ASYMMETRIC LEAVES1* and *ASYMMETRIC LEAVES2* — themselves downregulated by CIN-TCPs — abolishes extension of the leaf lamina. Thus, *GRF*-promoted proliferation appears to be required for lamina outgrowth [15–17].

Lamina outgrowth occurs where cells with adaxial (towards the shoot) and abaxial (away from the shoot) identities are juxtaposed [18]. The WUSCHEL-LIKE HOMEOBOX 1 (WOX1) and WOX3 transcription factors are expressed at the boundary region between these two leaf domains. They are required downstream of adaxial/abaxial polarity establishment to stimulate cell proliferation towards the leaf margins that drives lamina outgrowth, presumably via effects on auxin/cytokinin homeostasis and signalling by other growth-promoting factors [19–22]. Abaxial factors appear to ensure the formation of a flat lamina by excluding WOX1 expression from the abaxial leaf domain. While this function is carried out by WOX1 and WOX3 orthologues in a largely