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Current Opinion in Plant Biology

The growth of a stable stationary structure: coordinating cell behavior and patterning at the shoot apical meristem

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Plants are characterized by their ability to produce new organs post-embryonically throughout their entire life cycle. In particular development of all above-ground organs relies almost entirely on the function of the shoot apical meristem (SAM). The SAM performs a dual role by maintaining a pool of undifferentiated cells and simultaneously driving cell differentiation to initiate organogenesis. Both processes require strict coordination between individual cells which leads to formation of reproducible morphological and molecular patterns within SAM. The patterns are formed and maintained in large part due to spatio-temporal variation in signaling of plant hormones auxin and cytokinin resulting in tissue-specific transcriptional regulation. Integration of these mechanisms into computational models further identifies the key regulatory interactions involved in SAM function.

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Current Opinion in Plant Biology 2018, **41**:83–88

This review comes from a themed issue on **Growth and development**

Edited by **Gwyneth Ingram** and **Ari Pekka Mähönen**

http://dx.doi.org/10.1016/j.pbi.2017.09.011

Introduction

Multicellular organisms are characterized by the presence of recognizable patterns shaping their body structures. These patterns are produced from complex distributions of cell fates in space and time. In higher plants the shoot apical meristem (SAM) is a dynamic structure with undifferentiated stem cells in its center and differentiated organ primordia at its periphery (reviewed in [1,2]). Two opposite processes occur in the SAM: the stem cell pool is constantly maintained and renewed whereas some cells accelerate their growth and division rate and eventually differentiate to become part of the newly forming organs (the leaves and flowers). The balance between these two processes is strictly controlled over the life of the plant and the location and timing of new organ emergence appears to be tightly regulated. This regulation can be first seen from the organization of the SAM which is divided into functional zones with distinct cellular behaviors (division and expansion) and distinct cellular identities. In *Arabidopsis thaliana* the dome-shaped structure of the SAM is divided into the central, peripheral and rib zones. The central zone is found at the summit and contains undifferentiated stem cells. The laterally located peripheral zone is the site of organ primordia initiation. The rib zone, situated below central and peripheral zones, produces the internal tissues of the stem. The SAM can be further divided into individual cell layers. In the center of the SAM the top two layers (L1 and L2; collectively referred as tunica) are able to divide only in one direction (anticlinally) whereas the deeper layers (L3 and further; collectively referred as corpus) are able to divide in any direction. This organization is largely similar in other higher plants with some variations in the number of tunica layers. The molecular patterns associated with the functional zones of the SAM are established by tissue-specific expression of key regulatory genes as well as mobile signals such as proteins and hormones, which move between cells in the different SAM zones. In this review, we summarize recent findings on the mechanisms controlling and coordinating cell behavior and pattern formation at the shoot apical meristem.

Setting the geometry of the SAM: coordinating growth and cell division

Pattern formation at the SAM begins at the cellular level. The cells in the central zone where the stem cells are located divide slower than the cells in the peripheral zone [3,4]. The cells in the central zone of the SAM have overall similar cell size despite frequently observed asymmetric cell divisions [5•,6•]. A few recent studies have started to elucidate how the balance between cells division and cell expansion is maintained [5•,6•,7]. In particular, the cell divisions appear to be triggered by a combination of factors including reaching a critical size and adding a critical cell volume increment instead of a single decisive event [5•]. Moreover, the cell cycle length [6•] and the cell growth rate [5•,7] are adjusted to the variable initial cell size acquired after geometrically asymmetric cell division illustrating the presence of a compensatory mechanism that allows the meristem to maintain the

desired overall uniform structure. Indeed, the local variability of cell growth rates in the meristem plays a key role in setting the geometry of the meristem [8], highlighting the importance of cell behavior in generating a specific shape.

The occurrence of both geometrically symmetric and asymmetric cell divisions in the SAM questions not only the long-standing debate of how cells determine where to build a new cell wall but also whether this might play a role in the function of the SAM. A few recent publications discuss the rules behind positioning of new cell walls [9,10••]. The assumption that the cells divide along local minima of plane area [11] was challenged by providing evidence that the new division planes orient along the maxima of mechanical tensions in cell walls induced by local heterogeneous growth which can differ from the plane area minimum [10••].

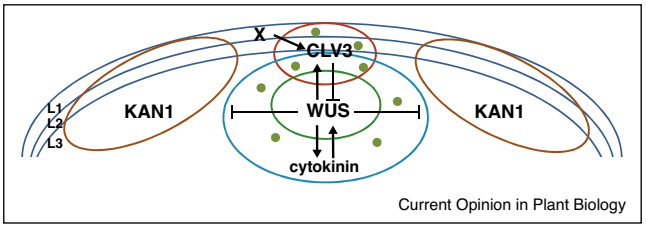
Several recent publications are further highlighting the importance of mechanical signals in the SAM. The division of the SAM into the central and peripheral zones indeed correlates with differences in mechanical properties: the central zone of the SAM is characterized by increased stiffness of the tissue compared to more peripheral regions [12–14] or organ primordia [15,16]. These mechanical properties appear to be genetically controlled [12] and caused by differences in auxin content [15]. However how the spatio-temporal distribution of the mechanical properties affects cell behavior and SAM function still remains to be clearly established.

Setting the organization of the SAM: the gene network controlling cell identities

The functional zones of the SAM are characterized by specific expression of master regulatory genes with CLV3 in the central zone [17], WUS in the organizing center [18] and KAN1 in the boundary domain [19•] amongst many others. Several recent publications have attempted to model SAM maintenance based on expression patterns and interactions of these regulatory genes [19•,20••,21,22,23••]. Computer simulations attempted to define the minimal regulatory networks required for functioning of the SAM (Figure 1). The models always include the well-described WUS-CLV3 feedback loop, which dynamically maintains the size of stem cell niche [24–27]. Repression of the differentiation-promoting genes such as KAN1 by WUS contributes to the entry into differentiation [19•,20••]. Furthermore, this modeling work emphasizes the importance of cytokinin signaling in SAM maintenance by showing that regulation of WUS expression by cytokinin [28,29] and activation of cytokinin signaling by WUS [30] are fundamental for correct positioning of WUS in the SAM [20••,21]. Recently an additional signaling network was identified which includes putative movement of a CLE peptide produced in organ primordia to the center of the SAM where it regulates stem cell activity [23••] thus providing an extra feedback regulation from developing organs on the stem cell niche and providing an interesting mechanisms for integrating stem cell maintenance and organogenesis.

The maintenance of the stem cell niche though WUS continues to be the subject of extensive research. WUS was shown to be a mobile protein which moves from the WUS expression domain into L1 and L2 layers of the SAM [25]. Lately, plasmodesmata were confirmed to mediate this movement [31]. Structural domains responsible for the spatial distribution and subcellular localization of WUS protein were identified with partially contradicting results in two independent studies [31,32]. Homodimerization appears to play a crucial role in restricting WUS protein movement [31,32], in control of CLV3 expression [33•] and in regulation of SAM growth [31,32]. Surprisingly, WUS was shown to activate CLV3 at low concentrations and repress at high concentrations, which accounts for the restriction of CLV3 expression to the upper layers of the central zone [33•]. Interestingly, misexpression of WUS reduced its protein stability leading to degradation [32] which sheds a new light on the previously described influence of WUS misexpression on SAM size and cell division rates in the peripheral zone [34,35]. These molecular details would need to be considered in future models as they modify significantly our understanding of the molecular regulations at play. The WUS-mediated control of the stem cell niche involves direct regulation of multiple genes [19•,36] but only WUS-CLV3 interaction has been characterized in details [24–27,33•]. A recent publication focuses on HEC1, a direct target of WUS, which influences stem cell activity and controls expression of a subset of WUS target genes in a manner antagonistic to WUS [37]. This illustrates the need for broadening the analysis of the different targets of WUS.

Figure 1



Minimal gene interaction network controlling SAM maintenance. CLV3 (red circle) and WUS (green circle) regulate each other expression though the movement of WUS protein (green dots). The expression of the cell differentiation-promoting genes such as KAN1 (brown circle) is limited to the periphery of the SAM through direct repression by WUS protein. WUS is activated by cytokinin signaling (light blue circle) and in turn activates cytokinin signaling by itself. CLV3 is activated by a hypothetical L1-derived signal (X).
Adapted from [19–21].