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Postembryonic control of root meristem growth and development

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Organ development in multicellular organisms is dependent on the proper balance between cell proliferation and differentiation. In the Arabidopsis root apical meristem, meristem growth is the result of cell divisions in the proximal meristem and cell differentiation in the elongation and differentiation zones. Hormones, transcription factors and small peptides underpin the molecular mechanisms governing these processes. Computer modeling has aided our understanding of the dynamic interactions involved in stem cell maintenance and meristem activity. Here we review recent advances in our understanding of postembryonic root stem cell maintenance and control of meristem size.

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Introduction

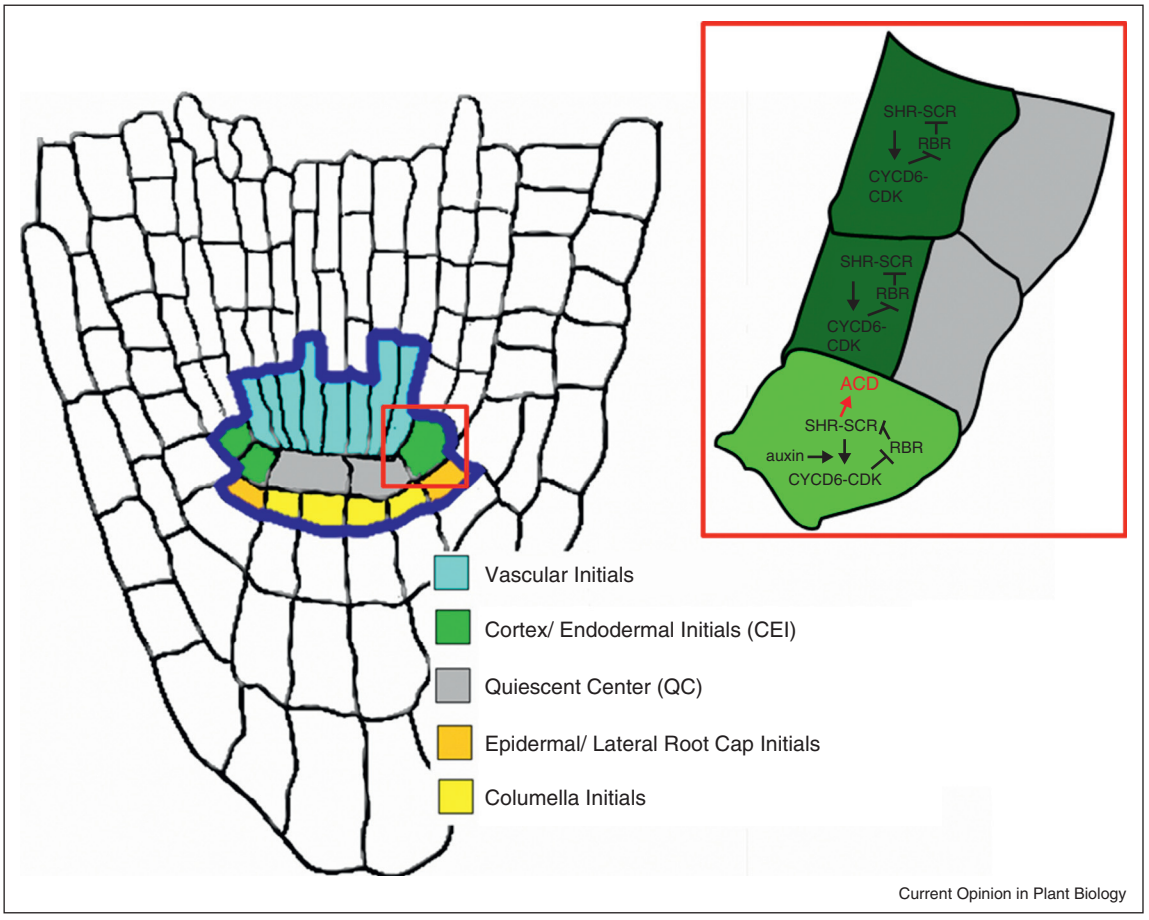
Development in multicellular organisms requires production of many specialized cell types and sophisticated mechanisms of coordination among them. Stem cells are the source of all cell types; the balance between stem cell self-renewal and differentiation of their progeny regulates organ growth. The root apical meristem (RAM) is established during embryogenesis and all postembryonic root development is derived from this reservoir of stem cells. In the RAM, four sets of stem cells — vascular, cortex/endodermal, epidermal/lateral root cap and columella initials — surround the quiescent center (QC) (Figure 1). The QC cells, which divide infrequently, are required for specification of the stem cell niche and maintenance of the undifferentiated state of stem cell initials [1]. Stem cells continuously undergo asymmetric divisions to produce daughter cells that are displaced from the stem cell niche and start to differentiate [2]. Because plant cells do not move and stem cells divide in a stereotypical manner, the root is organized into cell layers where entire cell lineages are spatially restricted. This spatial restriction results in clear developmental zones along the longitudinal axis of the root. Cell division occurs in the proximal meristem (Figure 2). As cells age they expand and begin to differentiate in the elongation and differentiation zone. The region in which cells exit the meristem and begin to elongate is termed the transition zone (Figure 2). Here we review the molecular programs known to be involved in specification and maintenance of postembryonic root stem cell niches and meristem growth (for a *comprehensive review* on embryonic stem cell initiation see [3–5]).

Root stem cell niche

Root stem cell specification and maintenance is the result of convergent phytohormone, transcription factor, and peptide signaling pathways. Among these, the hormone auxin signals through the auxin inducible PLETHORA (PLT1-4) transcription factors [6]. Auxin accumulates within the distal stem cell region via both local biosynthesis [7,8] and polar transport from the shoot [9], where it forms and auxin response maximum that is required for QC function [9,10]. Auxin is directed by transporters such as the PINFORMED (PIN) family proteins, leads to a graded expression of PLT genes [11–13]. High PLT concentrations in the root tip specify and maintain the stem cell niche. In contrast, low PLT levels correlate with stem cell differentiation [13]. The highest PLT levels in the QC appear to be further controlled by root growth factors (RGF) both transcriptionally and posttranscriptionally [14]. The current model proposes that auxin induces the expression of the posttranslational modifying enzyme, TYROSYLPROTEIN SULFOTRANSFERASE (TPST), which sulfates RGF peptides and, in turn, up-regulate PLT genes. Sulfated RGF1 in combination with other two tyrosine-sulphated peptides, phytosulfokine (PSK) and plant peptide containing sulfated tyrosine (PSY1), are sufficient to maintain QC cells, and thus root growth [15,16]. Taken together, these results indicate that stem cell maintenance relies on the crosstalk between auxin and a peptide signaling pathway.

A parallel mechanism for root stem cell maintenance involves the GRAS family transcription factors SHORTROOT (SHR), SCARECROW (SCR), and some of their target genes and interacting proteins [17–20]. *SHR* is transcribed in the stele and moves one cell layer into the endodermis, cortex/endodermal initial (CEI), and QC where it firstly, activates the transcription of *SCR* and finally, regulates the asymmetric division of the CEI and maintains QC identity [21,22]. Conversely, SCR sustains QC functions and stem cell identity through cell-autonomous activity [17]. In addition, the SHR/SCR pathway mediates stem cell fate decisions by modulating

Figure 1



Schematic of a longitudinal section of the *Arabidopsis* root tip with each stem cell type differentially colored. Inset: asymmetric cell division of CEI. The combinatorial action of auxin, SHR and SCR restrict ACD in space and time by activating CYCD6. Auxin acts downstream of SHR and SCR in augmenting CYCD6 expression. CYCD6 together with a heterodimeric partner cyclin-dependent kinase (CDK), through phosphorylation, inhibits the activity of RBR. RBR represses CEI division by forming a ternary complex with SHR and SCR.

the expression of cell cycle genes involved in asymmetric divisions [20] (Figure 1). *SHR* and *SCR* activity within the QC, CEI, and endodermis is affected by their downstream genes *MAGPIE* (*MGP*), and *JACKDAW* (*JKD*) [19]. Notably *SHR*, *SCR*, and *JKD* are required for QC identity, whereas *MGP*, *SHR*, *SCR*, and *JKD* influence CEI fate. Accordingly, phenotypic analysis of loss of function alleles for each of these genes shows loss of QC identity and radial patterning defects [19]. Instead, it has been shown that SCR sustains stem cell activity by repressing cytokinin dependent cell differentiation, insinuating another hormone in the establishment and maintenance of the stem cell niche. Specifically, SCR maintains the activities of both QC and stem cells by directly suppressing the expression of the cytokinin response regulator *ARABIDOPSIS RESPONSE REGULATOR 1* (*ARR1*) in the QC [17,23••]. Furthermore, brassinosteroids (BR) alter the expression of regulators of the stem cell niche [24,25] and ethylene regulates QC divisions [26]. Thus, additional layers of regulation for stem cell maintenance are mediated by the other plant hormones.

Because stem cells that lose contact with their neighboring QC differentiate, a short-range QC-derived signal that maintains stem cell identity has been proposed [1]. Although the signal remains elusive, a WUSCHEL-RELATED HOMEOBOX 5 (WOX5) and/or its target(s) are considered candidates [27]. In addition to the combinatorial action of the two sets of key transcriptional regulators that we described above — *PLT*, *SHR* and *SCR* — WOX5 was also shown to be essential for QC specification. However, *wox5* mutants affect only columella stem cells (COL) that are distal to the QC, so maintenance of stem cell types proximal to the QC is likely accomplished by some factors partially redundant to WOX5. The molecular mechanisms have been elucidated and involve, together with WOX5, the peptide