Published in final edited form as:
Annu Rev Plant Biol. 2012 June 2; 63: 563–590. doi:10.1146/annurev-arplant-042811-105501.

Control of *Arabidopsis* Root Development

Jalean J. Petricka[*], **Cara M. Winter**[*], and **Philip N. Benfey**
Department of Biology and Duke Center for Systems Biology, Duke University, Durham, North Carolina 27708

Jalean J. Petricka: jalean.petricka@gmail.com; Cara M. Winter: cara.winter@duke.edu; Philip N. Benfey: philip.benfey@duke.edu

Abstract

The *Arabidopsis* root has been the subject of intense research over the past decades. This research has led to significantly improved understanding of the molecular mechanisms underlying root development. Key insights into the specification of individual cell types, cell patterning, growth and differentiation, branching of the primary root, and responses of the root to the environment have been achieved. Transcription factors and plant hormones play key regulatory roles. Recently, mechanisms involving protein movement and the oscillation of gene expression have also been uncovered. Root gene regulatory networks controlling root development have been reconstructed from genome-wide profiling experiments, revealing novel molecular connections and models. Future refinement of these models will lead to a more complete description of the complex molecular interactions that give rise to a simple growing root.

Keywords

embryogenesis; radial patterning; developmental zones; hormone; lateral root; gene regulatory network

INTRODUCTION

Roots are plant organs that typically lie below the surface of the soil, where they grow and respond to a variety of environmental barriers and insults. Roots not only provide structural support to the aerial portion of the plant but also acquire nutrients and water vital to plant growth. Thus, overall plant survival depends on appropriate root development, growth, and function. Study of root development, however, is challenging because roots are inaccessible and observation often requires invasive measures. Additionally, the root systems of many plant species are highly complex, making characterization of basic developmental mechanisms intractable.

The study of root development has been greatly advanced through the use of the model organism *Arabidopsis thaliana*. Roots of *Arabidopsis* have a very simple cellular organization and can be easily grown in nonsoil media, which facilitates analysis. Knowledge of *Arabidopsis* root development is derived from work over the past 25 years, which began with classical genetic experiments and has been accelerated by the use of

[*]These authors contributed equally to this work.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

modern molecular biology and genomics techniques. We now have a picture of the molecular state of individual cell types, at different developmental stages, and in response to various stimuli, at a level of detail that is unavailable for any other organ. This in-depth characterization has propelled the root to the forefront of the field of plant developmental biology.

Here we provide an overview of our present understanding of how *Arabidopsis* root development is controlled at the molecular level, from initiation in the embryo to elaboration in the adult plant. We highlight recent studies of the root apical meristem (RAM), a region of the root tip consisting of undifferentiated cells that give rise to the different cells of the root. The pathways and mechanisms regulating patterning of the tissues and developmental zones of the root, the production of lateral roots (LRs), and root responses to environmental stimuli are discussed. We suggest future prospects and give an overview of how genome-scale data are being used to model and probe the gene regulatory networks controlling root development and response.

EMBRYOGENESIS: THE MAKING OF A ROOT

Apical-basal polarity of the embryo is established early in embryogenesis as a shoot meristem positioned between embryonic leaves (cotyledons) at the top (apical) end and an embryonic root and root meristem at the bottom (basal) end (93). In *Arabidopsis* embryogenesis (Figure 1) the zygote first divides asymmetrically to produce a smaller apical cell that divides vertically to produce the proembryo, and a larger basal cell that divides horizontally to produce the suspensor connecting the embryo to maternal tissue. The uppermost suspensor cell is later specified to become the founder cell of the root meristem, called the hypophysis. The hypophysis divides asymmetrically to generate an upper lens-shaped cell that subsequently becomes the quiescent center (QC) and a lower basal cell from which the columella stem cells and columella are derived. The proembryo concurrently contributes stem cells for the root vascular, ground, and epidermal tissues (129). Thus, root formation during embryogenesis involves coordination between the two embryo poles.

Embryonic *WOX* Expression

A few factors have been shown to be involved in the making of an embryonic root. *WUSCHEL-related homeobox* (*WOX*) genes, encoding transcription factors (TFs), are expressed in dynamic and partially overlapping patterns during embryogenesis from the single-cell zygote through the eight-cell embryo stage (Figure 1*a*) (64). *WOX* expression coincides with cell fate decisions during embryogenesis, suggesting that WOX TF-mediated gene regulation underlies these decisions. However, mutations in these WOX TFs do not result in strong embryonic phenotypes (64), indicating that additional factors function with WOX in embryonic patterning. Further experiments will likely identify these factors and determine the exact role of *WOX* pathways in cell fate decisions during embryogenesis and their involvement with the plant hormone auxin (64).

Auxin Regulates Embryonic Root Formation

Many factors that control embryonic root formation implicate the involvement of auxin. For example, genetic perturbation of auxin transport [*pinformed1,3,4,7* (*pin1*,3,4,7) (50); *gnom* (82, 95, 141, 171)], perception [*transport inhibitor response1* (*tir1*); *auxin signaling f-box1,2,3* (*afb1*,2,3) (40)], or synthesis [*yucca1,4,10,11* (31); *tryptophan aminotransferase of Arabidopsis1* (*taa1*); *tryptophan aminotransferase related1,2* (*tar1*,2) (143)] prevents embryonic root formation. Further, the hypophysis is not specified and roots are not formed in mutants lacking *MONOPTEROS* (*MP*)/*AUXIN RESPONSE FACTOR5* (*ARF5*), which is one of the 23 *ARF* genes encoding auxin-induced TFs (68). ARF activity is inhibited by

auxin (Aux)/indole-3-acetic acid (IAA) proteins such as BODEN-LOS (BDL)/INDOLE-3-ACETIC ACID INDUCIBLE12 (IAA12). Dominant mutations in BDL/IAA12 render it insensitive to auxin-dependent degradation and lead to root phenotypes similar to *mp*/*arf5* (66, 67, 164).

Intriguingly, even though MP/ARF5 and BDL/IAA12 interact and are involved in hypophysis specification, these proteins are not expressed in the hypophysis (66, 165). Instead, they are expressed in provascular cells adjacent to the hypophysis, where auxin-induced degradation of BDL/IAA12 releases MP/ARF5 from cotranscriptional repression by BDL/IAA12 and TOPLESS (147, 165). This derepression of MP/ARF5 allows upregulation in the provascular cells of *PIN1*, an auxin transporter thought to direct auxin flow basally into the hypophysis. In turn, PIN1-mediated accumulation of auxin in the hypophysis is proposed to affect hypophysis specification through the action of additional auxin-responsive Aux/IAA and ARF pairs expressed in the hypophysis (165). Potential candidate Aux/IAAs and ARFs expressed in the embryo involved in hypophysis specification are emerging (117, 118, 164).

Recently, microarrays of MP revealed *TARGET OF MP* (*TMO*) genes that mediate signaling from the proembryo to the hypophysis (132). Four *TMO* genes (*TMO3*, -5, *-6*, and *-7*) are coexpressed with MP in the proembryo provascular cells adjacent to the hypophysis. Two of these genes (*TMO5* and *TMO7*) encode basic helix-loop-helix TFs and are functional, direct MP targets as their promoters are directly bound by MP. Further, *TMO5* or *TMO7* expression in *mp* mutants partially rescues the *mp* rootless phenotype, which suggests that these genes are important downstream targets of MP. Strikingly, when Schlereth et al. (132) evaluated TMO5 and TMO7 expression patterns, they found that TMO7 protein, and not TMO5, was expressed in the hypophysis and affected hypophysis divisions when knocked down. Further experiments showed that TMO7 protein moves from the cytoplasm and nucleus of provascular cells to the nucleus of the hypophysis. This TMO7 movement provided a mechanism by which MP exerts its non-cell-autonomous effects on the hypophysis (132, 165).

Chemical inhibition of auxin transport also interferes with specification of the hypophysis and root formation, consistent with a role for PIN transporters in root formation (50, 63). Taken together with the localization of PINs and auxin responses during embryogenesis, this observation led to a proposed model describing how auxin accumulation and transport establish not only the apical-basal axis but also the hypophysis (Figure 1) (50). In the two-cell stage, PIN7 in the basal cell facilitates auxin transport to the apical cell to generate an auxin response maximum. Until the globular stage of embryogenesis, PIN7 is apically localized in the suspensor cells of the basal cell lineage, and this localization maintains the auxin maximum in the proembryo (Figure 1*a*). During the early globular stage, an auxin response maximum in the presumptive hypophysis then arises from concurrent localization of PIN1 to the basal membranes of the inner cells of the proembryo and redistribution of PIN7 to the basal membrane of suspensor cells (Figure 1*b*) (50). Thus, provascular localization of PIN1 combined with a reversal in PIN7 polarity might specify the hypophysis during embryogenesis and lead to root formation.

Other Hormones Regulate Embryonic Root Formation

Despite its key role, auxin is insufficient to form a root. This is because auxin response as detected by synthetic reporters is not specific to the precursor cell of the hypophysis and is insufficient to specify the hypophysis (165). Cytokinin response, visualized by a synthetic reporter, is detected specifically in the hypophysis and its apical daughter cell (Figure 1*b*) (103). This finding suggests that cytokinin regulates the hypophysis and subsequent production of the embryonic root. Interestingly, auxin response viewed by a synthetic

reporter has an inverse profile to that of cytokinin response. Auxin response is found in the basal daughter cell produced from the asymmetric hypophyseal cell division (Figure 1*b*). Further, auxin induces two repressors of cytokinin response, ARABIDOPSIS RESPONSE REGULATOR7 (ARR7) and ARR15, in this basal hypophyseal cell (103). Thus, the antagonistic action of auxin and cytokinin regulates the establishment of the root meristem in the specific spatiotemporal context of the hypophyseal cell division.

In addition to cytokinin and auxin, other hormones/regulators might impact root formation during embryogenesis. Intriguingly, the direct targets of MP, *TMO5* and *TMO7*, were previously identified and implicated in response and/or signaling of cytokinin or gibberellin and brassinosteroids, respectively (85, 119, 132, 162). This finding suggests that other hormones and growth regulators act in hypophysis specification and that crosstalk between them is important for root formation. Indeed, crosstalk between brassinosteroids and auxin is implicated in this process. Mutants have been isolated that affect signaling of both of these factors and also exhibit defects in hypophysis specification, such as *brassinosteroid insensitive 1-ems-suppressor interacting myc-like protein* and *brevis radix* (29, 101, 126). Future studies should identify additional factors involved in embryonic events that lead to hypophyseal cell specification and division and determine how these factors are integrated with hormonal signals to orchestrate root formation.

PATTERNING THE ROOT APICAL MERISTEM

The RAM established during embryogenesis provides new cells for the growing root; it contains a set of initial cells (stem cells) that surround the QC, a group of less mitotically active cells. Together these form the stem cell niche, similar in concept to that found in animals (Figure 2*a*) (127, 139). Stem cells on the shootward and lateral sides of the QC produce the vascular, endodermal, cortex, epidermal, and lateral root cap (LRC) cells, whereas stem cells on the rootward face of the QC produce the columella root cap. Stem cell daughters generate single-cell files that extend along the longitudinal root axis and form distinct tissue layers (Figure 2*a*). Counterintuitively, although a mature tissue type has a distinct cell lineage and is derived from a single initial cell, cell position rather than lineage determines cell identity (79, 154).

Stem Cell Niche

The QC is essential for specification of the stem cell niche and maintenance of the undifferentiated state of stem cell initials (155). QC identity is specified by two parallel pathways: the PLETHORA (PLT) pathway and the SHORT ROOT (SHR)/SCARECROW (SCR) pathway (2, 55, 123). Loss-of-function alleles of these genes result in loss of QC identity and premature termination of root growth. The *PLT* genes encode AP2-domain transcription factors (55), whereas *SHR* and *SCR* encode members of the GRAS [GIBBERELLIN INSENSITIVE (GAI), REPRESSOR OF GA1–3 (RGA), SCR] family of transcription factors (2, 123). The highest levels of PLT proteins are found in the niche, where they promote cell division (55). SHR moves from the stele into the adjacent cell layers to activate *SCR* transcription (35, 72, 86). *SCR* expression in the QC maintains QC and stem cell identity through a cell-autonomous mechanism (123). Expression patterns of different enhancer-trap QC markers are altered in the *plt* as opposed to the *shr* and *scr* mutant backgrounds, indicating that these genes regulate the stem cell niche through parallel mechanisms (2).

QC cells appear to produce a short-range signal maintaining stem cell identity because stem cells in direct contact with a laserablated QC cell differentiate (155). This signal might be WOX5 or its direct target(s), as *WOX5* is expressed in the QC and is required non-cell-autonomously to prevent stem cell differentiation (125). WOX5 acts downstream of SHR

and SCR but not the PLT proteins (125). Restriction of *WOX5* expression to the QC regulates the size of the stem cell pool by a mechanism similar to that of the shoot (22, 61, 81, 104, 120): The CLAVATA3/EMBRYO SURROUNDING REGION (CLE) peptide CLE40 acts through the receptor-like kinase ARABIDOPSIS CRINKLY4 (ACR4) to exclude *WOX5* expression outside of the QC (140). Other regulators of stem cell identity include small peptides called root meristem growth factors, which promote postembryonic maintenance of the stem cell niche through posttranscriptional regulation of the PLT proteins (94, 180), and JACKDAW (JKD), a C2H2 zinc finger TF necessary for *SCR* expression in the QC (166). *JKD* expression itself is dependent on SCR, suggesting a complex feedback between these two regulators.

Stele

Plant vasculature includes xylem and phloem vessels that transport vital water and nutrients (xylem) and photosynthates (phloem) to and from the shoot. In *Arabidopsis* roots, the vasculature is organized into a central cylinder or stele, which contains xylem and phloem interspersed with undifferentiated procambial cells and a surrounding pericycle layer (Figure 2*b*). These tissues are derived from the set of pericycle/vascular initials proximal to the QC (43, 109, 129). Vasculature grows longitudinally during primary growth and radially during secondary growth (for a recent review of secondary growth, see 178).

Whereas the root's outer tissues exhibit radial symmetry, the stele is bilaterally symmetric. A transverse section through a mature *Arabidopsis* root (Figure 2*b*) reveals a central axis of xylem cells with protoxylem at the poles and metaxylem in the center. Two phloem bundles are found at the poles of the axis perpendicular to the xylem axis. *LONESOME HIGHWAY* (*LHW*) is a key regulator of this bilateral symmetry (108). In *lhw* mutants, only single xylem and phloem poles are formed.

Phloem consists of sieve elements and companion cells that arise from asymmetric divisions of phloem/procambial initial cells (7, 18, 91). The MYB TF ALTERED PHLOEM DEVELOPMENT (APL) promotes phloem development and also represses xylem identity. In *apl* mutants, phloem identity is lost, and cells with xylem characteristics appear at the phloem poles instead (18).

The two types of xylem, protoxylem and metaxylem, differ in structure and function. Protoxylem cells have spiral cell wall thickenings, are smaller, differentiate early, and are usually destroyed as the plant matures, whereas metaxylem cells have pitted cell wall thickenings, differentiate later, are larger, and form the water-conducting vessels of the plant. Correct patterning of xylem depends on positional information provided by SHR because in *shr* and *scr* mutants, metaxylem differentiates ectopically in place of protoxylem. Carlsbecker et al. (27) showed that after SHR moves from the stele into the endodermis, its activation of *SCR* allows SHR/SCR activation of microRNA165/166 (miR165/166) expression. In turn, miR165/166 moves back into the stele to repress class III homeodomain-leucine zipper (HD-ZIP III) TF expression at the stele periphery. The dosage of HD-ZIP expression specifies the type of xylem: High levels promote metaxylem formation, whereas low levels promote protoxylem formation. Loss of function of all five HD-ZIP TFs results in complete loss of xylem cells, indicating that these genes are essential for xylem identity (27). *VASCULAR RELATED NAC-DOMAIN PROTEIN6* (*VND6*) and *VND7* are also important for xylem specification. Ectopic expression of *VND6* induces ectopic metaxylem formation, whereas ectopic expression of *VND7* induces ectopic protoxylem (83). Further examination of direct targets of these genes (110, 174) and genes enriched in xylem precursor cells and differentiating xylem cells (19) will likely provide future insights into the mechanisms that underlie the development and differentiation of xylem tissues.

Cytokinin also plays an essential role in vascular patterning. Mutation of the CRE cytokinin receptor gene *WOODEN LEG* [*WOL*/*CRE1*/*ARABIDOPSIS HISTIDINE KINASE4* (*AHK4*)], either alone or in combination with the cytokinin receptors *AHK2* and *AHK3*, results in many fewer procambial cells, and all cells differentiate as protoxylem (26, 128). Consistently, an inhibitor of cytokinin activity, ARABIDOPSIS HISTIDINE PHOSPHOTRANSFER PROTEIN 6 (AHP6) promotes protoxylem identity (90). Thus, cytokinin signaling is essential for specification of phloem and metaxylem cell types.

Ground Tissue

Cortex and endodermal cell layers are derived from the cortex/endodermal initial (CEI) cells, which divide anticlinally to regenerate themselves and produce the CEI daughter (CEID) cells. The CEID cells then divide periclinally to generate the endodermal and cortex precursors (Figure 2*c*). The TFs SHR and SCR, which act as key regulators of this process, were first isolated based on their short-root mutant phenotypes (9, 128). Both *shr* and *scr* mutants have a single ground tissue layer, indicating that SHR and SCR control asymmetric division of the CEID cell (41, 72). In the *shr* mutant, this single layer of ground tissue displays only cortex features, whereas in the *scr* mutant it has characteristics of both endodermis and cortex (9, 41). These data suggest that SHR plays an additional role in endodermal specification. Further SHR and SCR characterization led to a model of endodermal cell specification whereby SHR protein moves from the stele into the endodermis and directly upregulates *SCR* and *JKD* expression, which in turn leads to restriction of SHR movement to a single cell layer of endodermis (35, 106, 166). SHR and SCR then act in a complex to regulate downstream targets promoting CEID division and endodermal cell specification (35, 86). Recent work has demonstrated a direct link between organ patterning and division of the CEID cell via SHR/SCR direct upregulation of the cell cycle regulator gene *CYCLIN D6* (*CYCD6*) (137).

Epidermis, Lateral Root Cap, and Columella

The LRC and columella form a protective cell layer that continually sloughs off as the root tip explores new territory. Along with the epidermis, these tissues constitute the exterior surface of the root. Columella cells function in gravity sensing, whereas epidermal root hairs play an important role in water and nutrient uptake. The columella originates from division of the columella initials located rootward from the QC, whereas the epidermis and LRC are derived from the epidermis/LRC initials found lateral to the QC. The epidermis/LRC initials divide periclinally to produce a daughter cell that will become a new layer of the LRC and anticlinally to produce a daughter cell that will differentiate into epidermal tissue (Figure 2*d*). The NAC domain TFs FEZ and SOMBRERO (SMB) are important for the periclinal divisions of the columella and epidermis/LRC initial cells; *fez* mutants have reduced columella and LRC layers, whereas *sombrero* mutants have increased cell layers (169). *FEZ* is expressed in the initial cells, where it autonomously promotes cell division and nonautonomously promotes *SMB* expression in the daughter cells. SMB in turn represses *FEZ* expression in the daughter cells, thereby restricting divisions to the initial cells (169). Maturation of the root cap is determined by *SMB*, *BEARSKIN1* (*BRN1*), and *BRN2* (12). A key feature of the root cap is the regulated detachment of cells into the soil. In *smb* mutants the LRC cells fail to detach, whereas in *brn1 brn2* double mutants the columella root cap cells fail to detach (12).

Future studies will continue to shed light on how cell identity is specified in the RAM. Important questions include how asymmetric cell division is controlled and how this process gives rise to cell lineages with different fates. Given that positional information plays a key role in cell identity, understanding signaling pathways in the root will also contribute significantly to our understanding of root patterning.

DEVELOPMENTAL ZONES OF THE ROOT

New cells produced by the stem cell initials in the RAM progress through three distinct developmental phases on their way to maturity (Figure 3*a*). In the meristematic zone (MZ), they divide multiple times to generate a pool of cells that will elongate and differentiate. In the elongation zone (EZ), cells lose their ability to divide and increase in length by many times their width. Finally, in the differentiation zone (DZ), cells acquire their specialized characteristics and functions.

Cell Proliferation and Division in the Meristematic Zone

Mitotic competence in the meristem decreases with increasing distance from the stem cell niche, until cells finally exit the cell cycle at the boundary, or transition zone (TZ), between the MZ and EZ. The position of this boundary determines the size of the meristem, which is directly related to the rate of root growth (8). Hormones play a key role in delineating the position of the TZ (discussed below), but ultimately regulation occurs at the level of the cell cycle. In animals, exit from the mitotic cell cycle into endoreduplication is a key step toward differentiation and is largely controlled by repression of mitotic cyclin-dependent kinase activity by the ubiquitin ligase-containing anaphase-promoting complex/cyclosome (APC/C) (136, 175). A similar process appears to occur in *Arabidopsis. HOBBIT*/*CELL DIVISION CYCLE 27 HOMOLOG B* (*CDC27B*) is a homolog of a core component of the APC/C complex, and *hobbit*/*cdc27b* mutations cause cell division defects in the RAM (16, 115, 135). The *CELL CYCLE SWITCH PROTEIN 52A* (*CCS52A*) genes are *Arabidopsis* homologs of genes that control APC/C activity. The CCS52A1 isoform promotes differentiation in the TZ, whereas CCS52A2 promotes quiescence in the QC (159). Finally, *HIGH PLOIDY2* (*HPY2*), a small ubiquitin-like modifier (SUMO) E3 ligase, prevents endocycle onset in meristematic cells by promoting the expression of mitotic cell cycle regulators (76). The *PLT* genes may act through *HPY2* to promote the mitotic cell cycle because RAM size is not enlarged in steroid-induced PLT2-GR *hpy2* plants compared with controls. Additionally, *HPY2* expression in meristematic cells requires the *PLT* genes (76). These results suggest that HPY2 is an effector of auxin control of cell division in the meristem. Recent evidence indicates that the balance of reactive oxygen species also controls the transition from proliferation to differentiation. *UPBEAT1*, a basic helix-loop-helix TF expressed at the TZ, directly regulates peroxidases that alter the distribution of reactive oxygen species. Cell proliferation requires high levels of $O_2^{\cdot-}$, whereas high H_2O_2 induces differentiation (150).

Cellular Expansion in the Elongation Zone

Expansion of root cells in the EZ contributes to primary root growth and is largely regulated by plant hormones (discussed below). Cells transitioning from the mitotic cycle in the MZ to a period of elongation undergo cellular changes in preparation for rapid unidirectional growth, including development of a central vacuole, cytoskeletal reorganization, and increases in cell width (161). Rapid cell expansion is associated with vacuole expansion through water uptake (33). Consistently, the tonoplast aquaporin GAMMA TONOPLAST INTRINSIC PROTEIN (GAMMA-TIP) is specifically expressed in the MZ where cells begin rapid elongation (88), and plant cells defective in vacuole expansion do not elongate (130, 134).

Changes in cell walls are also important for cell expansion (34, 36). The transverse orientation of cortical microtubules (like rings around a barrel) is thought to control the polar expansion of cells by providing a track for cellulose synthase for new cell wall deposition. This idea is supported by mutants such as *botero1* (*bot1*) and *fragile fiber* (*fra2*), which exhibit defects in cell expansion associated with misoriented cortical microtubules

and cellulose microfibrils (13, 23). However, other cell expansion mutants, such as *radially swollen4* (*rsw4*) and *rsw7*, have normal microtubule and cellulose microfibril arrays, indicating that oriented cellulose microfibrils are insufficient for polar expansion of root cells (71, 168). Expansion defects in mutants of genes involved in cellulose synthesis, such as *rsw1–3* and *procuste*, highlight the importance of cell wall assembly and modification in root cell elongation (3, 47). The *COBRA* (*COB*) gene also regulates polar expansion of root cells and encodes a member of a family of glycosylphosphatidylinositol (GPI)–anchored proteins located at the interface between the plasma membrane and the cell wall (20, 131). COB is specifically localized to the longitudinal sides of expanding cells, where it influences unidirectional growth in the EZ (131). Finally, *roothairless* mutants are defective in the endocycle and have short roots with small cells, suggesting that an increase in DNA content through the cell cycle is necessary for cell expansion (144).

Features of the Differentiation Zone: Casparian Strip and Root Hair Formation

After cells reach their final size, they enter the DZ, where they acquire specialized characteristics. Relatively little is known about the molecular mechanisms underlying the switch to a differentiating state. Two notable features of the DZ include the Casparian strip and root hairs.

The Casparian strip consists of lignin and suberin deposits between the transverse walls of endodermal cells (Figure 3*b*) that form an impermeable barrier preventing apoplastic (through spaces between cells) entry of water and solutes into the central vascular cylinder. Thus, molecules must traverse the cytoplasm of endodermal cells via transporters to enter vascular tissues. The *CASP* genes (*CASP1–5*) are preferentially enriched in the endodermis and encode plasma membrane–localized proteins (14, 121). Time-lapse imaging of green fluorescent protein (GFP)–tagged CASPs revealed that the proteins first localize to the perimeter of the plasma membrane and then slowly aggregate to the domain of Casparian strip formation (CSD). When expressed in other cell layers, CASP proteins mainly localize to the plasma membrane but not to the CSD, indicating that other endodermal-specific factors are necessary for proper CASP localization. The CASP proteins are also instructive for the Casparian strip because *CASP* expression precedes CSD formation and the Casparian strip in *casp1 casp3* mutants is a patchy noncontiguous structure. Interestingly, the CASP proteins are localized to the EZ and DZ, indicating that differentiation begins prior to the morphological appearance of differentiated structures (121).

Another key feature of the DZ is the development of root hairs. Epidermal cells produced in the RAM become hair cells or nonhair cells based on their position relative to the underlying cortical cells. An epidermal cell in contact with two cortex cells will develop as a hair cell, whereas one adjacent to a single cortex cell will become a nonhair cell (Figure 3*c*). Many studies have revealed a cadre of components involved in the specification and differentiation of hair and nonhair cells (reviewed in detail in 149). Within nonhair cells, a complex involving TRANSPARENT TESTA GLABRA1 (TTG1), GLABRA3 (GL3), ENHANCER OF GLABRA3 (EGL3), and WEREWOLF (WER) activates the expression of GL2 and CAPRICE (CPC). Activation of GL2 results in nonhair cell fate. In a form of lateral inhibition, CPC then travels into the neighboring presumptive hair cell, where it competes with WER for binding to the TTG1/GL3/EGL3 complex. Repression of *WER* by SCRAMBLED (SCM) in the presumptive hair cells tips the balance, allowing CPC to outcompete WER, resulting in loss of activation of *GL2* and consequent hair cell specification. SCM is a leucine-rich-repeat receptor-like kinase that was predicted to repress *WER* specifically in hair cells through transduction of a signal coming from the overlying cortical cell junction. Recent evidence suggests that this signal is produced downstream of JKD in cortical cells. The greater contact surface between hair cells and the two overlying

cortex cells likely results in greater signal and specific activation of SCM in hair cells (Figure 3*c*) (70).

In the DZ, root hairs emerge at the base of root hair cells. Root hair bulging is associated with cellular changes (reviewed in detail in 149), including the influx of Ca^{2+} and K^{+} ions, phospholipid signaling, rearrangement of the cytoskeleton, acidification of the cell wall, and reassembly and synthesis of new cell wall material. Auxin accumulates at high concentrations at the root hair tip, sustaining tip growth. Notably, differences in size between presumptive hair and nonhair cells are evident even in the MZ (45). These data suggest that differentiation of epidermal cell types, similar to differentiation of the Casparian strip in endodermal cells, begins long before the final appearance of root hairs in the DZ. Future studies will begin to unravel the molecular events that lead from specification of cell types in the root tip to fully differentiated cells. It will be interesting to determine whether there is a general differentiation signal or whether this process is controlled independently in the different cell types.

HORMONAL CONTROL OF ROOT GROWTH

The major plant hormones—abscisic acid (ABA), brassinosteroids, cytokinin, ethylene, gibberellic acid (GA), and IAA (auxin)—are key regulators of the cell division (in the MZ) and elongation (in the EZ) that contribute to primary root growth. In the past few decades, their biosynthesis, catabolism, perception, and transport have been elucidated. Protein degradation of repressors is a common mechanism in the response and/or signaling of most of these regulators, except for ABA and cytokinin, which act through phosphorylation of TFs by kinases. These hormonal pathways and molecular mechanisms were characterized through mutant analyses that also uncovered mutants common to multiple hormonal responses, pointing to hormonal crosstalk in root growth (10, 172).

Auxin

From the earliest studies, hormonal responses of plants have been both intriguing and mysterious. For example, in 1936, Thimann (148) noted the enigmatic ability of applied auxin to both stimulate root initiation and inhibit root elongation. Indeed, auxin, ABA, and brassinosteroids act to promote or inhibit root growth depending on the concentration applied to *Arabidopsis* roots (6, 32, 38, 46, 48, 105, 160, 176). Recent reports have shown that the auxin biosynthesis genes are expressed specifically in the root stem cell niche, where they increase auxin levels (19, 142, 143). However, auxin is thought to accumulate mainly in the primary root tip by polar transport from the shoot, largely via the action of directional transporters such as the PINs (17, 56, 60). Each of the PINs localizes to different cell types, and study of *pin* mutants in conjunction with the synthetic reporter of auxin response, DR5:GUS, revealed that both rootward auxin transport and lateral auxin redistribution are crucial for root meristem maintenance (17, 60).

These data suggested the existence of an auxin reflux loop that generates an auxin gradient in the root. In the current model, auxin is first transported by PINs from the shoot rootward through the root vasculature and then laterally redistributed in the root cap to the external root cell types; auxin is then transported back toward the shoot via PINs in the external layers. To maintain an auxin maximum in the root stem cell niche, auxin reflux is proposed to occur at the transition from the root MZ to EZ by auxin transport from the external root layers back to, and downward into, the root vascular auxin stream by PIN1, PIN3, and PIN7. Root responses to the auxin gradient are then mediated, at least in part, by the activation of auxin response factors, such as the TFs MP and NONPHOTOTROPIC HYPOCOTYL4 (NPH4) (2). These TFs, in turn, are thought to activate an expression gradient of PLT proteins that activate *PIN* expression and act in a dosage-dependent fashion: At locations of

high PLT concentrations the root stem cell niche is formed and maintained, at sites of moderate PLT concentrations cell proliferation occurs in the root meristem, and at regions of low PLT concentrations root cells differentiate (2, 55). The auxin and PLT gradients thus provide a plausible explanation for how auxin can regulate stem cell maintenance as well as root cellular proliferation, elongation, and differentiation via its localization and concentration.

Auxin-Cytokinin Antagonism

Although auxin has a central role in the control of root growth, other hormones are also required for proper establishment and maintenance of cellular proliferation and elongation in the root. Root meristem size is enlarged in multiple mutants of the ATP/ADP isopentenyltransferases (IPTs) that catalyze the rate-limiting step of cytokinin biosynthesis and in the cytokinin receptor AHK3 and its suggested downstream target ARR1. In contrast, meristem size is reduced by exogenous cytokinin application, indicating that cytokinin inhibits cell division (37, 96). However, the rate of cell division in the MZ is unaffected. Further, expression of cytokinin oxidase-dehydrogenase gene 1 specifically in the TZ between dividing and elongating root cells drastically reduces endogenous cytokinins in that region and phenocopies triple *ipt* mutants. Together, these data suggest that cytokinins reduce the number of dividing cells and inhibit root growth by promoting cellular differentiation in the TZ (37).

A subsequent microarray study of roots overexpressing *ARR1* revealed that *SHORT HYPOCOTYL2* (*SHY2*)/*IAA3*—a member of the IAA protein family that heterodimerizes with ARFs, preventing the activation of auxin responses—was an ARR1 direct target, pointing to a mechanism for the long-hypothesized antagonism between auxin and cytokinin in root growth (37, 38). Further experiments showed that SHY2/IAA3 upregulates cytokinin biosynthesis via IPT5. These experiments also showed that SHY2/IAA3 activation by cytokinin results in downregulation and a restricted expression domain of the auxin transporters *PIN1*, *PIN3*, and *PIN7*. Thus, an antagonistic auxin-cytokinin network functions at the border between the MZ and EZ through induction of SHY2/IAA3 by the AHK3 cytokinin receptor and ARR1; in turn, SHY2/IAA3 interferes with auxin transport by PINs. Auxin-induced expression of cytokinin biosynthesis by IPT5 is simultaneously activated by SHY2/IAA3 to generate a negative-feedback loop (38). This antagonistic auxin-cytokinin network provides the molecular basis for the opposite effects observed in root meristem size (enlarged and reduced, respectively) resulting from exogenous cytokinin or auxin treatment (37, 38). In summary, auxin promotes cell division, whereas cytokinin promotes differentiation.

Gibberellic Acid as an Integrator of Auxin-Cytokinin Antagonism

Interestingly, GA might be the integrator of auxin-cytokinin hormonal antagonism. GA has been described as altering both cell proliferation and elongation in the endodermis to control root growth (1, 151, 152). Downstream of auxin, GA promotes cell division in the MZ, and mutations in GA biosynthesis or signaling genes, like auxin mutations, result in roots that exhibit reduced meristem size (1, 52, 151, 152). Further supporting the positive relationship between auxin and GA is the fact that auxin induces the degradation of DELLA proteins that repress GA signaling involved in the promotion of root elongation and the expression of GA biosynthesis genes in the root MZ and TZ (49). GA has also been reported to repress the root growth inhibition mediated by cytokinin (59, 100). Taken together, these findings of GA interactions with auxin and cytokinin suggest that GA plays an important role in the auxin-cytokinin antagonism regulating root growth.

Other Hormones

Adding another layer of complexity, brassinosteroids also control root growth by affecting cell division in the MZ (58, 62). Although this is similar to the effect of GA on root growth, only brassinosteroids seem to alter the expression of regulators of the stem cell niche, such as *SCR* and *WOX5*, for the maintenance of stem cell identity and organization (58, 62). Similar to brassinosteroids, ABA and ethylene also regulate QC divisions; however, unlike brassinosteroids, regulators of the stem cell niche are expressed in divided QC cells, suggesting that the identity of these cells is maintained (112). These studies also suggest that the effects of brassinosteroids and ethylene on root growth by the induction and regulation of QC divisions are partially independent of auxin, whereas ABA-induced QC divisions might result from alterations in auxin responses modulated by MP (58, 112, 177). Future work might address whether and how ABA, brassinosteroid, and ethylene signaling are integrated with cytokinin, GA, and auxin to affect QC divisions and/or root growth. These future studies will also likely focus on the integration of developmental regulators at the cellular level because brassinosteroids, GA, and cytokinin have been shown to control growth of the entire root organ by activity in a specific cell layer (epidermis, endodermis, and vasculature, respectively) (1, 37, 151, 152).

BRANCHING TO PRODUCE LATERAL ROOTS

Primary Root Branching

After the pattern of the primary root has been established and the individual cell types have begun to differentiate, further growth results from branching of the primary root to produce an indeterminate and dynamic root system. The root system is largely composed of LRs and the primary root. LRs form in the DZ from mature pericycle cells positioned adjacent to the xylem poles, called the xylem pole pericycle (Figure 4). A subset of these cells, called founder cells, are stimulated to divide to form a lateral root primordium (LRP) (15). LRs initiate with these first anticlinal cell divisions of the founder cells during the first stage of LRP development. Including this first stage, there are seven stages that typify LRP development (Figure 4) (92). During the last stages, the LRP begins to resemble the primary root tip. Subsequent emergence of the LR through the parent root epidermis is thought to occur primarily via cell expansion. After emergence, the apical meristem of the LR is activated and begins growing. To generate the *Arabidopsis* root system, LRs arise iteratively along the primary root such that the newest LRPs are positioned rootward from the older LRPs and LRs (44). Further elaboration of the root system occurs through iterative branching of the LRs. It remains an open question how cellular-level processes of LR formation are coordinated to generate the overall architecture of the root system.

The Role of Auxin in Lateral Root Initiation

The plant hormone auxin has emerged as an important player in LR production. An early study exposing pea roots to exogenous auxin found that auxin treatment induces LR formation (148). Later studies in *Arabidopsis* showed that auxin treatment can induce LRPs along the length of the xylem pole pericycle cells (73, 84). LR initiation that is mediated by auxin involves cell cycle activation in founder cells through the control of cyclin pathways (73, 124, 157). Indeed, mutations in genes involved in auxin biosynthesis, response, signaling, and/or transport have defects in the density and/or development of LRs (28, 40). Perhaps the best-known of these genes is *SOLITARY-ROOT* (*SLR*)/*IAA14*. Gain-of-function *iaa14* mutants lack LRs owing to a block in auxin-induced pericycle divisions (53, 54). Recent work has revealed factors functioning downstream of SLR/IAA14 that include *NPH4*/*ARF7*, *ARF19*, and their downstream transcriptional targets (53, 75, 111, 170).

Collectively, these studies have led to a model whereby when auxin is present, Aux/IAA transcriptional repressors, such as SLR/IAA14, interact directly with the auxin receptor TIR1 and are targeted for degradation. The degradation of Aux/IAA repressors would allow ARFs, such as ARF7 and ARF19, to activate transcription of auxin-responsive genes, which is predicted to lead to LR initiation (53, 111). Future work elucidating the mechanisms controlling LR initiation will likely focus on refining this model and on potential crosstalk between auxin and other hormones (113).

Iterative Production of Lateral Roots

The question of how the iterative production of LRs along the main root axis is controlled is another central focus of recent research. Auxin has been hypothesized to regulate LR production prior to LR initiation by priming subsets of pericycle cells to become founder cells, forming an LR prebranch site. In accordance with this idea, stronger, periodic staining of the synthetic auxin-responsive reporter DR5:GUS (122) was found in the basal meristem, near the TZ (39). This spatiotemporal pattern of staining was correlated with LR initiation, leading the authors to hypothesize that auxin in the basal meristem regulates the positioning of LRs along the primary root (39).

However, a more recent study found that auxin is not sufficient to specify prebranch sites along the primary root. Time-lapse imaging of the DR5 promoter element coupled to the luciferase coding region (DR5:LUC) allowed in vivo characterization of the reporter over time. Observations of DR5:LUC revealed oscillations of expression that occurred with a periodicity of approximately six hours in a region broader than the basal meristem, called the oscillation zone. The appearance of an expression site always led to the production of LRPs (98). To determine whether these oscillations were related to changes in auxin levels, the authors fused the promoters of *IAA7*, *SLR*/*IAA14*, and *IAA19* to the luciferase reporter and tested for response to exogenous auxin applied in the zone where DR5:LUC was found to oscillate (98). The p*IAA19*:LUC reporter had a similar expression and dose response to exogenous auxin as DR5:LUC. However, p*IAA19*:LUC did not exhibit oscillatory behavior, and exogenous auxin applied with or without auxin transport inhibitors could not induce prebranch sites. This suggested that auxin was unlikely to be sufficient to generate the periodic pulses of DR5:LUC expression (98). This conclusion leads to a number of interesting questions about LR production (see sidebar, Questions About Lateral Root Production).

One prominent question remains: If auxin is not sufficient to generate prebranch sites, then what regulates the production of these sites? Clues to the answer might be found among the endogenous genes that show oscillatory expression in the same region as DR5:LUC (98). For instance, the SHATTERPROOF TFs were found to oscillate and, when mutated, affected the number of prebranch sites (98). Future work will determine the upstream regulators and downstream targets of these TFs and their potential regulation of prebranch formation. Although temperature, sucrose concentrations, and day length do not appear to alter the number of prebranch sites along the main root, it is possible that other environmental factors perturb the oscillations associated with prebranch site formation and LR emergence. Candidates include gravity (98), strigolactones (78), other hormones, and a host of nutrients that are absent or excessively concentrated in soil known to perturb LR root branching and development (113). Alternatively, the number of prebranch sites may remain constant while these environmental factors alter the subsequent development and emergence of LRPs.

ROOT RESPONSES TO STIMULI

Root development does not adhere strictly to a predefined genetic program, but instead is highly responsive to changing environmental conditions. Signals such as gravity, light, water, and touch stimulate changes in the direction or rate of growth as well as in the number of LRs. Abiotic stressors such as salt, drought, heat, or cold as well as nutrient deprivation and toxicity (potassium, phosphorus, nitrogen, iron, sulfur) also cause a host of phenotypic and molecular responses. In many cases, it is not yet clear which of these responses act to mitigate the effects of the stress and which are symptoms of the stress. Although extensive research has been performed on the signal transduction pathways that constitute the initial response of plants to various environmental stimuli, little is known about how these stimuli affect development (74, 153). Root responses to gravity and salt stress are relatively well characterized and will be considered here as examples.

Response to Gravity

The phenomenon of gravitropism has been a focus of research since the nineteenth century (for reviews, see 30, 99). The classic starchstatolith hypothesis posits that gravity is perceived primarily in the columella root cap cells by specialized cells called statocytes. Statocytes contain starch-filled plastids (amyloplasts) that are denser than the cytoplasm. Reorientation of roots causes sedimentation of amyloplasts to the bottoms of cells, which triggers the redistribution of auxin, resulting in greater growth on the upper side of the root relative to the lower side. This growth differential results in a characteristic curvature of the root tip, which realigns the growth and gravity vectors.

QUESTIONS ABOUT LATERAL ROOT PRODUCTION

- What regulates the gene expression oscillations that generate prebranch sites?
- How early are oscillations detected? Do they occur during embryogenesis?
- What is the specific role of phytohormones in the positioning, initiation, development, and outgrowth of lateral roots?
- How do different cell types and populations generate and act on information from oscillations?
- What triggers lateral root initiation, which begins with asymmetric divisions of founder cells?
- When, where, and how are founder cells specified?
- Is there a relationship between the processes and timing of root and shoot branching?

Recent studies provide molecular evidence for this model. The auxin efflux carrier PIN3 is initially distributed throughout the plasma membrane of statocytes, but is redistributed to the lower side of statocytes upon gravistimulation (51). This redistribution requires endomembrane-localized ALTERED RESPONSE TO GRAVITY1 (ARG1) and ARG1-LIKE2 (ARL2) (69) through an unknown mechanism. PIN3 relocalization directs auxin laterally, where it flows through the auxin influx and efflux carriers AUX1 and PIN2 in the LRC and is transported to the epidermal cells. The resulting asymmetric accumulation of auxin on the lower side of the root inhibits cell elongation (102, 146). This response may be essentially a modulation of the auxin reflux loop that acts to precisely position the transition from cell division to elongation under normal developmental conditions (see Hormonal Control of Root Growth, above). Auxin is normally required to maintain cell division in the

meristem, and reduction of shootward auxin transport through the LRC and epidermal cells results in a decrease in meristem size (17). Thus, it is possible that the redistribution of auxin toward the lower side of the root maintains these cells in a more meristematic state, keeping them in a cell division pattern, whereas cells on the upper side of the root progress more quickly to elongation (11).

Response to High Salinity

High soil salinity is a very different type of environmental stimulus than gravity. The plant's first response to salinity is to maintain low levels of cytosolic Na^+ in the root through compartmentalization into vacuoles, export into the soil, or selective ion uptake (reviewed in 181). When this response fails, Na^+ accumulates in the cytosol, resulting in toxicity. In addition to this ionic stress, high salinity also imposes an osmotic stress similar to that imposed by drought. Salt stress affects the root in all developmental zones. Cell division is reduced in the MZ and cell expansion is attenuated in the EZ, resulting in reduced overall growth (167). Cells also expand radially in the EZ (25), and root hair outgrowth is suppressed in the DZ (65). Salt stress additionally results in agravitropic growth (145) as well as reduced LR number under high-salt conditions and enhanced LR number under moderate-salt conditions (163, 182).

The effects of salt stress on gravitropism and LR development appear to be mediated by changes in auxin distribution (163, 182). Mild salt stress promotes the accumulation of auxin in developing LRPs, preventing their developmental arrest and resulting in greater numbers of LRs (182). This auxin accumulation appears to be mediated by the SOS (salt overly sensitive) signaling pathway (179). The agravitropism exhibited by salt-stressed roots is due to repression of transcription and localization of PIN2, resulting in reduced shootward auxin reflux in the LRC and epidermal cells (145).

Transcriptional profiling at the level of individual cell types has advanced our understanding of how roots respond to salt stress. Dinneny et al. (42) profiled six different cell types for changes in expression in response to salt exposure. Of the 3,862 total genes differentially expressed in any cell layer, the majority were significantly changed in expression in only one cell type. This key finding indicates that individual cell types in the root respond differently to salt stress, likely executing key cell type–specific functions that together constitute the whole root response. Although most gene expression changes were specific to individual cell types, not all cell types responded with equal sensitivity. In particular, cells in the cortex, epidermis, and stele exhibited the greatest numbers of differentially expressed genes, indicating a primary role for these tissues in mediating the salt stress response. Notably, expression of *COB*, *RSW3*, and *KOBITO1*—three genes required to repress radial cell expansion during normal development (24, 114, 131)—was reduced in the cortex and epidermis, suggesting a mechanism for the salt-induced radial expansion of these tissues. In further support of this hypothesis, the authors also demonstrated that a hypomorphic allele of *COB* enhances the salt-induced radial swelling (42). Further mining of these data sets will likely provide key future insights into how roots respond to salt stress.

Root responses to environmental stimuli such as gravity and high salt involve both a perception/signaling component and a final translation of those signals into a developmental response. The developmental response involves changes in the root system architecture through modifications of the rate and direction of primary root growth and the positioning and outgrowth of LRs. Different mechanisms must be in place to perceive the wide array of environmental stimuli. However, it is likely that these signals converge to directly modify basal developmental pathways. In the case of gravity and salt, both of these stimuli appear to converge on the auxin pathway to modulate development. One open question is how environmental stimuli affect LR development. In the case of salt stress, it appears to be LR

outgrowth that is affected. It is unknown whether environmental signals can also regulate LR initiation through modulation of prebranch site selection. Future studies will continue to explore the complex responses of roots to environmental stimuli and the mechanisms by which these signals affect development.

ROOT GENE REGULATORY NETWORKS

Root development and response to the environment are thought to be controlled by gene regulatory networks. Recent genome-wide studies describing the *Arabidopsis* transcriptome, metabolome, and proteome as well as genome-wide TF binding have facilitated the reconstruction of gene regulatory networks and the investigation of pathways functioning in them (for a review, see 97). Useful deliverables of genome-wide studies include expression maps that provide valuable information about the expression profiles of many individual molecular components, the relationships between them, and global expression trends or patterns. The next challenging step will be to reconstruct gene regulatory networks from these maps and integrate maps of different data types (e.g., transcriptome and proteome) so that outcomes of network perturbances might be reliably predicted (116). Such an understanding of gene regulatory networks in the *Arabidopsis* root promises to inform studies aimed at predictions and manipulations of traits of agronomic importance in crop species.

Expression Maps

An expression map provides a multifaceted representation of the spatial, temporal, or environmental-responsive expression of many genes, metabolites, or proteins in an organism or cell type. The transcriptomes and proteomes of whole seedlings or roots have been extensively profiled for a variety of developmental stages, hormonal treatments, and environmental conditions (5, 57, 80, 133, 173). Laser-capture microdissection followed by microarray analysis has also been used to isolate and profile RNA from the basal domains of globular-stage embryos as well as the root pole of heart- and torpedo-stage embryos (138). These studies have produced maps that allow global comparison of expression patterns in the root with those of other organs or conditions. However, much of the cell- and tissue-level information is diluted or missing from these root-organ maps. Fluorescence-activated cell sorting of GFP-marked cell populations has allowed isolation and transcriptional profiling at cell type resolution in the root (14, 19, 42, 77, 107). Maps generated by these studies have revealed expression patterns in individual cell types and in response to environmental perturbations, as well as profiles of genes uncharacterized by whole-organ studies (133).

Coexpression of Genes

Transcriptional expression maps have been heavily exploited to infer relationships between genes and gene function based on coexpression of genes. For this purpose, the transcriptional profiles of genes are computationally clustered. Clustered genes are prime suspects for being coregulated or functioning in the same pathway or process. Coexpression analyses of whole-root or seedling data have suggested interesting global phenomena: Genes that are physically close on a given chromosome are more likely to be coexpressed (14, 133), the initial transcriptional response to abiotic stress involves an adjustment in energy balance (80), and hormonal responses are temporally regulated and involve crosstalk (57). Cell type–level coexpression analyses in the root have revealed expression domains of genes that are involved in hormonal processes (14), spatiotemporal gene expression signatures that imply the existence of previously unknown cellular functions (19), and genes that respond to different abiotic stresses in discrete cell types (42, 77). Thus, coexpression analyses using data from gene expression maps provide valuable candidate functions for unknown genes

and insight into how genes function together in space and time and in response to the environment.

Reconstruction of Gene Regulatory Networks

Transcriptional expression maps have also been used to reconstruct gene regulatory networks. In *Arabidopsis*, gene regulatory networks have been reconstructed from data that include whole roots (89, 156) or from root cell type–specific data (19, 21). These reconstructed regulatory networks have led to novel predictions and transcriptional modules, some of which were demonstrated experimentally. Interesting transcriptional modules have been uncovered from mining these gene regulatory networks, including those governing stress and energy signaling (4). Expression data are more powerful in identifying regulatory networks when combined with TF binding information (116). In the root, this has been used to identify modules involved in asymmetric cell division (137), iron homeostasis (87), abiotic stress (77), and the balance between root cell proliferation and differentiation by reactive oxygen species (150). These studies demonstrate the power of gene regulatory networks to reveal novel players and mechanisms functioning in root development and response.

Despite the advances in understanding gene regulatory networks in the root, much of the molecular complexity inherent to them remains to be untangled. For example, information about TF binding, metabolites, small RNAs, proteins, and protein-protein interactions have not yet been acquired at the cell type level in roots. Future work will focus on obtaining and integrating these with gene expression maps to elucidate, improve, and refine the molecular details of root regulatory networks.

CONCLUDING REMARKS

Development of the *Arabidopsis* root is a dynamic process that involves a complex interplay between transcriptional regulators and plant hormones. In particular, auxin plays a role in nearly every aspect of root development. The auxin pathway provides a common point of convergence that may allow the integration of many different environmental and endogenous cues, leading to a coordinated morphogenic response. Understanding the *Arabidopsis* root as a system will require the integration of many different types of data to understand how RNA, proteins, and metabolites interact to produce a developmental outcome. The modeling of gene regulatory networks is one way researchers are addressing and will continue to address this complexity. Advances from these efforts will strengthen the accuracy of predictions used for manipulation of crop species for desirable phenotypic traits.

Acknowledgments

This work was supported by NIH grants (1P50-GM081883, R01-GM043778), the NSF Arabidopsis 2010 program, a grant from DARPA, and an NIH Ruth L. Kirschstein NRSA award (F32GM086976) to J.J.P.

Glossary

RAM	root apical meristem
LR	lateral root
Founder cells	xylem pole pericycle cells that divide to form a lateral root primordium
QC	quiescent center
TF	transcription factor

Stem cell niche	the microenvironment that maintains the stem cells; includes the quiescent center and stem cells
LRC	lateral root cap
Protoxylem	small cells that differentiate early and are usually destroyed as the plant matures
Metaxylem	large cells that form the water-conducting vessels of the plant
CEI	cortex/ endodermal initial
CEID	cortex/ endodermal initial daughter
MZ	meristematic zone
EZ	elongation zone
DZ	differentiation zone
TZ	transition zone
CSD	Casparian strip domain
ABA	abscisic acid
GA	gibberellic acid
Xylem pole pericycle	pericycle cells adjacent to the xylem poles, some of which will give rise to lateral roots
LRP	lateral root primordium
Prebranch site	a group of cells in the primary root that statically express DR5: LUC and are competent to form a lateral root

LITERATURE CITED

1. Achard P, Gusti A, Cheminant S, Alioua M, Dhondt S, et al. Gibberellin signaling controls cell proliferation rate in *Arabidopsis*. Curr Biol. 2009; 19:1188–93. [PubMed: 19576768]
2. Aida M, Beis D, Heidstra R, Willemsen V, Blilou I, et al. The *PLETHORA* genes mediate patterning of the *Arabidopsis* root stem cell niche. Cell. 2004; 119:109–20. [PubMed: 15454085]
3. Arioli T, Peng L, Betzner AS, Burn J, Wittke W, et al. Molecular analysis of cellulose biosynthesis in *Arabidopsis*. Science. 1998; 279:717–20. [PubMed: 9445479]
4. Baena-González E, Rolland F, Thevelein JM, Sheen J. A central integrator of transcription networks in plant stress and energy signalling. Nature. 2007; 448:938–42. [PubMed: 17671505]
5. Baerenfaller K, Grossmann J, Grobei MA, Hull R, Hirsch-Hoffmann M, et al. Genome-scale proteomics reveals *Arabidopsis thaliana* gene models and proteome dynamics. Science. 2008; 320:938–41. [PubMed: 18436743]
6. Barrero JM, Piqueras P, Gonzalez-Guzman M, Serrano R, Rodriguez PL, et al. A mutational analysis of the *ABA1* gene of *Arabidopsis thaliana* highlights the involvement of ABA in vegetative development. J Exp Bot. 2005; 56:2071–83. [PubMed: 15983017]
7. Baum SF, Dubrovsky JG, Rost TL. Apical organization and maturation of the cortex and vascular cylinder in *Arabidopsis thaliana* (Brassicaceae) roots. Am J Bot. 2002; 89:908–20. [PubMed: 21665690]
8. Beemster GTS, Baskin TI. Analysis of cell division and elongation underlying the developmental acceleration of root growth in *Arabidopsis thaliana*. Plant Physiol. 1998; 116:1515–26. [PubMed: 9536070]
9. Benfey PN, Linstead PJ, Roberts K, Schiefelbein JW, Hauser MT, Aeschbacher RA. Root development in *Arabidopsis*: four mutants with dramatically altered root morphogenesis. Development. 1993; 119:57–70. [PubMed: 8275864]

10. Benkova E, Hejatko J. Hormone interactions at the root apical meristem. Plant Mol Biol. 2009; 69:383–96. [PubMed: 18807199]
11. Bennett T, Scheres B. Root development—two meristems for the price of one? Curr Top Dev Biol. 2010; 91:67–102. [PubMed: 20705179]
12. Bennett T, van den Toorn A, Sanchez-Perez GF, Campilho A, Willemsen V, et al. SOMBRERO, BEARSKIN1, and BEARSKIN2 regulate root cap maturation in *Arabidopsis*. Plant Cell. 2010; 22:640–54. [PubMed: 20197506]
13. Bichet A, Desnos T, Turner S, Grandjean O, Hofte H. *BOTERO1* is required for normal orientation of cortical microtubules and anisotropic cell expansion in *Arabidopsis*. Plant J. 2001; 25:137–48. [PubMed: 11169190]
14. Birnbaum K, Shasha DE, Wang JY, Jung JW, Lambert GM, et al. A gene expression map of the *Arabidopsis* root. Science. 2003; 302:1956–60. [PubMed: 14671301]
15. Blakely LM, Evans TA. Cell dynamics studies on the pericycle of radish seedling roots. Plant Sci Lett. 1979; 14:79–83.
16. Blilou I, Frugier F, Folmer S, Serralbo O, Willemsen V, et al. The *Arabidopsis HOBBIT* gene encodes a CDC27 homolog that links the plant cell cycle to progression of cell differentiation. Genes Dev. 2002; 16:2566–75. [PubMed: 12368267]
17. Blilou I, Xu J, Wildwater M, Willemsen V, Paponov I, et al. The PIN auxin efflux facilitator network controls growth and patterning in *Arabidopsis* roots. Nature. 2005; 433:39–44. [PubMed: 15635403]
18. Bonke M, Thitamadee S, Mahonen AP, Hauser M-T, Helariutta Y. APL regulates vascular tissue identity in *Arabidopsis*. Nature. 2003; 426:181–86. [PubMed: 14614507]
19. Brady SM, Orlando DA, Lee JY, Wang JY, Koch J, et al. A high-resolution root spatiotemporal map reveals dominant expression patterns. Science. 2007; 318:801–6. [PubMed: 17975066]
20. Brady SM, Song S, Dhugga KS, Rafalski JA, Benfey PN. Combining expression and comparative evolutionary analysis. The *COBRA* gene family. Plant Physiol. 2007; 143:172–87. [PubMed: 17098858]
21. Brady SM, Zhang L, Megraw M, Martinez NJ, Jiang E, et al. A stele-enriched gene regulatory network in the Arabidopsis root. Mol Syst Biol. 2011; 7:459. [PubMed: 21245844]
22. Brand U, Fletcher JC, Hobe M, Meyerowitz EM, Simon R. Dependence of stem cell fate in *Arabidopsis* on a feedback loop regulated by *CLV3* activity. Science. 2000; 289:617–19. [PubMed: 10915624]
23. Burk DH, Liu B, Zhong R, Morrison WH, Ye Z-H. A katanin-like protein regulates normal cell wall biosynthesis and cell elongation. Plant Cell. 2001; 13:807–28. [PubMed: 11283338]
24. Burn JE, Hurley UA, Birch RJ, Arioli T, Cork A, Williamson RE. The cellulose-deficient *Arabidopsis* mutant *rsw3* is defective in a gene encoding a putative glucosidase II, an enzyme processing N-glycans during ER quality control. Plant J. 2002; 32:949–60. [PubMed: 12492837]
25. Burssens S, Himanen K, van de Cotte B, Beeckman T, Van Montagu M, et al. Expression of cell cycle regulatory genes and morphological alterations in response to salt stress in *Arabidopsis thaliana*. Planta. 2000; 211:632–40. [PubMed: 11089675]
26. Cano-Delgado AI, Metzlaff K, Bevan MW. The *eli1* mutation reveals a link between cell expansion and secondary cell wall formation in *Arabidopsis thaliana*. Development. 2000; 127:3395–405. [PubMed: 10887094]
27. Carlsbecker A, Lee JY, Roberts CJ, Dettmer J, Lehesranta S, et al. Cell signalling by microRNA165/6 directs gene dose-dependent root cell fate. Nature. 2010; 465:316–21. [PubMed: 20410882]
28. Casimiro I, Beeckman T, Graham N, Bhalerao R, Zhang HM, et al. Dissecting *Arabidopsis* lateral root development. Trends Plant Sci. 2003; 8:165–71. [PubMed: 12711228]
29. Chandler JW, Cole M, Flier A, Werr W. BIM1, a bHLH protein involved in brassinosteroid signalling, controls *Arabidopsis* embryonic patterning via interaction with DORNROSCHEN and DORNROSCHEN-LIKE. Plant Mol Biol. 2009; 69:57–68. [PubMed: 18830673]
30. Chen R, Rosen E, Masson PH. Gravitropism in higher plants. Plant Physiol. 1999; 120:343–50. [PubMed: 11541950]

31. Cheng YF, Dai XH, Zhao YD. Auxin synthesized by the YUCCA flavin monooxygenases is essential for embryogenesis and leaf formation in *Arabidopsis*. Plant Cell. 2007; 19:2430–39. [PubMed: 17704214]
32. Clouse SD, Langford M, McMorris TC. A brassinosteroid-insensitive mutant in *Arabidopsis thaliana* exhibits multiple defects in growth and development. Plant Physiol. 1996; 111:671–78. [PubMed: 8754677]
33. Cosgrove DJ. How do plant cell walls extend? Plant Physiol. 1993; 102:1–6. [PubMed: 11536544]
34. Cosgrove DJ. Expansive growth of plant cell walls. Plant Physiol Biochem. 2000; 38:109–24. [PubMed: 11543185]
35. Cui H, Levesque MP, Vernoux T, Jung JW, Paquette AJ, et al. An evolutionarily conserved mechanism delimiting SHR movement defines a single layer of endodermis in plants. Science. 2007; 316:421–25. [PubMed: 17446396]
36. Darley CP, Forrester AM, McQueen-Mason SJ. The molecular basis of plant cell wall extension. Plant Mol Biol. 2001; 47:179–95. [PubMed: 11554471]
37. Dello Ioio R, Linhares FS, Scacchi E, Casamitjana-Martinez E, Heidstra R, et al. Cytokinins determine *Arabidopsis* root-meristem size by controlling cell differentiation. Curr Biol. 2007; 17:678–82. [PubMed: 17363254]
38. Dello Ioio R, Nakamura K, Moubayidin L, Perilli S, Taniguchi M, et al. A genetic framework for the control of cell division and differentiation in the root meristem. Science. 2008; 322:1380–84. [PubMed: 19039136]
39. De Smet I, Tetsumura T, De Rybel B, Frey NF, Laplaze L, et al. Auxin-dependent regulation of lateral root positioning in the basal meristem of *Arabidopsis*. Development. 2007; 134:681–90. [PubMed: 17215297]
40. Dharmasiri N, Dharmasiri S, Estelle M. The F-box protein TIR1 is an auxin receptor. Nature. 2005; 435:441–45. [PubMed: 15917797]
41. Di Laurenzio L, Wysocka-Diller J, Malamy JE, Pysh L, Helariutta Y, et al. The *SCARECROW* gene regulates an asymmetric cell division that is essential for generating the radial organization of the Arabidopsis root. Cell. 1996; 86:423–33. [PubMed: 8756724]
42. Dinneny JR, Long TA, Wang JY, Jung JW, Mace D, et al. Cell identity mediates the response of *Arabidopsis* roots to abiotic stress. Science. 2008; 320:942–45. [PubMed: 18436742]
43. Dolan L, Janmaat K, Willemsen V, Linstead P, Poethig S, et al. Cellular organisation of the *Arabidopsis thaliana* root. Development. 1993; 119:71–84. [PubMed: 8275865]
44. Dubrovsky JG, Gambetta GA, Hernández-Barrera A, Shishkova S, González I. Lateral root initiation in *Arabidopsis*: developmental window, spatial patterning, density and predictability. Ann Bot. 2006; 97:903–15. [PubMed: 16390845]
45. Duckett CM, Grierson C, Linstead P, Schneider K, Lawson E, et al. Clonal relationships and cell patterning in the root epidermis of *Arabidopsis*. Development. 1994; 120:2465–74.
46. Ephritikhine G, Fellner M, Vannini C, Lapous D, Barbier-Brygoo H. The *sax1* dwarf mutant of *Arabidopsis thaliana* shows altered sensitivity of growth responses to abscisic acid, auxin, gibberellins and ethylene and is partially rescued by exogenous brassinosteroid. Plant J. 1999; 18:303–14. [PubMed: 10377995]
47. Fagard M, Desnos T, Desprez T, Goubet F, Refregier G, et al. *PROCUSTE1* encodes a cellulose synthase required for normal cell elongation specifically in roots and dark-grown hypocotyls of Arabidopsis. Plant Cell. 2000; 12:2409–24. [PubMed: 11148287]
48. Finkelstein RR, Gampala SSL, Rock CD. Abscisic acid signaling in seeds and seedlings. Plant Cell. 2002; 14:S15–45. [PubMed: 12045268]
49. Frigerio M, Alabadi D, Perez-Gomez J, Garcia-Carcel L, Phillips AL, et al. Transcriptional regulation of gibberellin metabolism genes by auxin signaling in *Arabidopsis*. Plant Physiol. 2006; 142:553–63. [PubMed: 16905669]
50. Friml J, Vieten A, Sauer M, Weijers D, Schwarz H, et al. Efflux-dependent auxin gradients establish the apical-basal axis of *Arabidopsis*. Nature. 2003; 426:147–53. [PubMed: 14614497]
51. Friml J, Wisniewska J, Benkova E, Mendgen K, Palme K. Lateral relocation of auxin efflux regulator PIN3 mediates tropism in *Arabidopsis*. Nature. 2002; 415:806–09. [PubMed: 11845211]

52. Fu XD, Harberd NP. Auxin promotes *Arabidopsis* root growth by modulating gibberellin response. Nature. 2003; 421:740–43. [PubMed: 12610625]
53. Fukaki H, Nakao Y, Okushima Y, Theologis A, Tasaka M. Tissue-specific expression of stabilized SOLITARY-ROOT/IAA14 alters lateral root development in Arabidopsis. Plant J. 2005; 44:382–95. [PubMed: 16236149]
54. Fukaki H, Tameda S, Masuda H, Tasaka M. Lateral root formation is blocked by a gain-of-function mutation in the *SOLITARY-ROOT/IAA14* gene of *Arabidopsis*. Plant J. 2002; 29:153–68. [PubMed: 11862947]
55. Galinha C, Hofhuis H, Luijten M, Willemsen V, Blilou I, et al. PLETHORA proteins as dose-dependent master regulators of *Arabidopsis* root development. Nature. 2007; 449:1053–57. [PubMed: 17960244]
56. Galweiler L, Guan C, Muller A, Wisman E, Mendgen K, et al. Regulation of polar auxin transport by AtPIN1 in *Arabidopsis* vascular tissue. Science. 1998; 282:2226–30. [PubMed: 9856939]
57. Goda H, Sasaki E, Akiyama K, Maruyama-Nakashita A, Nakabayashi K, et al. The AtGenExpress hormone and chemical treatment data set: experimental design, data evaluation, model data analysis and data access. Plant J. 2008; 55:526–42. [PubMed: 18419781]
58. Gonzalez-Garcia MP, Vilarrasa-Blasi J, Zhiponova M, Divol F, Mora-Garcia S, et al. Brassinosteroids control meristem size by promoting cell cycle progression in *Arabidopsis* roots. Development. 2011; 138:849–59. [PubMed: 21270057]
59. Greenboim-Wainberg Y, Maymon I, Borochov R, Alvarez J, Olszewski N, et al. Cross talk between gibberellin and cytokinin: The Arabidopsis GA response inhibitor SPINDLY plays a positive role in cytokinin signaling. Plant Cell. 2005; 17:92–102. [PubMed: 15608330]
60. Grieneisen VA, Xu J, Marée AFM, Hogeweg P, Scheres B. Auxin transport is sufficient to generate a maximum and gradient guiding root growth. Nature. 2007; 449:1008–13. [PubMed: 17960234]
61. Guo Y, Han L, Hymes M, Denver R, Clark SE. CLAVATA2 forms a distinct CLE-binding receptor complex regulating Arabidopsis stem cell specification. Plant J. 2010; 63:889–900. [PubMed: 20626648]
62. Hacham Y, Holland N, Butterfield C, Ubeda-Tomas S, Bennett MJ, et al. Brassinosteroid perception in the epidermis controls root meristem size. Development. 2011; 138:839–48. [PubMed: 21270053]
63. Hadfi K, Speth V, Neuhaus G. Auxin-induced developmental patterns in *Brassica juncea* embryos. Development. 1998; 125:879–87. [PubMed: 9449670]
64. Haecker A, Groß-Hardt R, Geiges B, Sarkar A, Breuninger H, et al. Expression dynamics of *WOX* genes mark cell fate decisions during early embryonic patterning in *Arabidopsis thaliana*. Development. 2004; 131:657–68. [PubMed: 14711878]
65. Halperin SJ, Gilroy S, Lynch JP. Sodium chloride reduces growth and cytosolic calcium, but does not affect cytosolic pH, in root hairs of *Arabidopsis thaliana* L. J Exp Bot. 2003; 54:1269–80. [PubMed: 12654878]
66. Hamann T, Benkova E, Baurle I, Kientz M, Jurgens G. The *Arabidopsis BODENLOS* gene encodes an auxin response protein inhibiting MONOPTEROS-mediated embryo patterning. Genes Dev. 2002; 16:1610–15. [PubMed: 12101120]
67. Hamann T, Mayer U, Jurgens G. The auxin-insensitive *bodenlos* mutation affects primary root formation and apical-basal patterning in the *Arabidopsis* embryo. Development. 1999; 126:1387–95. [PubMed: 10068632]
68. Hardtke CS, Berleth T. The *Arabidopsis* gene *MONOPTEROS* encodes a transcription factor mediating embryo axis formation and vascular development. EMBO J. 1998; 17:1405–11. [PubMed: 9482737]
69. Harrison BR, Masson PH. ARL2, ARG1 and PIN3 define a gravity signal transduction pathway in root statocytes. Plant J. 2008; 53:380–92. [PubMed: 18047472]
70. Hassan H, Scheres B, Blilou I. JACKDAW controls epidermal patterning in the *Arabidopsis* root meristem through a non-cell-autonomous mechanism. Development. 2010; 137:1523–29. [PubMed: 20356954]

71. Hauser MT, Morikami A, Benfey PN. Conditional root expansion mutants of *Arabidopsis*. Development. 1995; 121:1237–52. [PubMed: 7743935]
72. Helariutta Y, Fukaki H, Wysocka-Diller J, Nakajima K, Jung J, et al. The *SHORT-ROOT* gene controls radial patterning of the *Arabidopsis* root through radial signaling. Cell. 2000; 101:555–67. [PubMed: 10850497]
73. Himanen K, Boucheron E, Vanneste S, de Almeida Engler J, Inzé D, et al. Auxin-mediated cell cycle activation during early lateral root initiation. Plant Cell. 2002; 14:2339–51. [PubMed: 12368490]
74. Hirayama T, Shinozaki K. Research on plant abiotic stress responses in the post-genome era: past, present and future. Plant J. 2010; 61:1041–52. [PubMed: 20409277]
75. Hirota A, Kato T, Fukaki H, Aida M, Tasaka M. The auxin-regulated AP2/EREBP gene *PUCHI* is required for morphogenesis in the early lateral root primordium of *Arabidopsis*. Plant Cell. 2007; 19:2156–68. [PubMed: 17630277]
76. Ishida T, Fujiwara S, Miura K, Stacey N, Yoshimura M, et al. SUMO E3 ligase HIGH PLOIDY2 regulates endocycle onset and meristem maintenance in *Arabidopsis*. Plant Cell. 2009; 21:2284–97. [PubMed: 19666737]
77. Iyer-Pascuzzi AS, Jackson T, Cui H, Petricka JJ, Busch W, et al. Cell identity regulators link development and stress responses in the *Arabidopsis* root. Dev Cell. 2011; 21:770–82. [PubMed: 22014526]
78. Kapulnik Y, Delaux P-M, Resnick N, Mayzlish-Gati E, Wininger S, et al. Strigolactones affect lateral root formation and root-hair elongation in *Arabidopsis*. Planta. 2010; 233:209–16. [PubMed: 21080198]
79. Kidner C, Sundaresan V, Roberts K, Dolan L. Clonal analysis of the *Arabidopsis* root confirms that position, not lineage, determines cell fate. Planta. 2000; 211:191–99. [PubMed: 10945213]
80. Kilian J, Whitehead D, Horak J, Wanke D, Weinl S, et al. The AtGenExpress global stress expression data set: protocols, evaluation and model data analysis of UV-B light, drought and cold stress responses. Plant J. 2007; 50:347–63. [PubMed: 17376166]
81. Kinoshita A, Betsuyaku S, Osakabe Y, Mizuno S, Nagawa S, et al. RPK2 is an essential receptor-like kinase that transmits the CLV3 signal in *Arabidopsis*. Development. 2010; 137:3911–20. [PubMed: 20978082]
82. Kleine-Vehn J, Huang F, Naramoto S, Zhang J, Michniewicz M, et al. PIN auxin efflux carrier polarity is regulated by PINOID kinase-mediated recruitment into GNOM-independent trafficking in Arabidopsis. Plant Cell. 2009; 21:3839–49. [PubMed: 20040538]
83. Kubo M, Udagawa M, Nishikubo N, Horiguchi G, Yamaguchi M, et al. Transcription switches for protoxylem and metaxylem vessel formation. Genes Dev. 2005; 19:1855–60. [PubMed: 16103214]
84. Laskowski MJ, Williams ME, Nusbaum HC, Sussex IM. Formation of lateral root meristems is a two-stage process. Development. 1995; 121:3303–10. [PubMed: 7588064]
85. Lee S, Yang KY, Kim YM, Park SY, Kim SY, Soh MS. Overexpression of *PRE1* and its homologous genes activates gibberellin-dependent responses in *Arabidopsis thaliana*. Plant Cell Physiol. 2006; 47:591–600. [PubMed: 16527868]
86. Levesque MP, Vernoux T, Busch W, Cui H, Wang JY, et al. Whole-genome analysis of the SHORT-ROOT developmental pathway in *Arabidopsis*. PLoS Biol. 2006; 4:e143. [PubMed: 16640459]
87. Long TA, Tsukagoshi H, Busch W, Lahner B, Salt DE, Benfey PN. The bHLH transcription factor POPEYE regulates response to iron deficiency in *Arabidopsis* roots. Plant Cell. 2010; 22:2219–36. [PubMed: 20675571]
88. Ludevid D, Hofte H, Himelblau E, Chrispeels MJ. The expression pattern of the tonoplast intrinsic protein γ-TIP in *Arabidopsis thaliana* is correlated with cell enlargement. Plant Physiol. 1992; 100:1633–39. [PubMed: 16653178]
89. Ma S, Bohnert HJ. Integration of *Arabidopsis thaliana* stress-related transcript profiles, promoter structures, and cell-specific expression. Genome Biol. 2007; 8:R49. [PubMed: 17408486]

90. Mähönen AP, Bishopp A, Higuchi M, Nieminen KM, Kinoshita K, et al. Cytokinin signaling and its inhibitor AHP6 regulate cell fate during vascular development. Science. 2006; 311:94–98. [PubMed: 16400151]
91. Mähönen AP, Bonke M, Kauppinen L, Riikonen M, Benfey PN, Helariutta Y. A novel two-component hybrid molecule regulates vascular morphogenesis of the *Arabidopsis* root. Genes Dev. 2000; 14:2938–43. [PubMed: 11114883]
92. Malamy JE, Benfey PN. Organization and cell differentiation in lateral roots of *Arabidopsis thaliana*. Development. 1997; 124:33–44. [PubMed: 9006065]
93. Mansfield SG, Briarty LG. Early embryogenesis in *Arabidopsis thaliana*. II. The developing embryo. Can J Bot. 1991; 69:461–76.
94. Matsuzaki Y, Ogawa-Ohnishi M, Mori A, Matsubayashi Y. Secreted peptide signals required for maintenance of root stem cell niche in *Arabidopsis*. Science. 2010; 329:1065–67. [PubMed: 20798316]
95. Mayer U, Buttner G, Jurgens G. Apical-basal pattern formation in the *Arabidopsis* embryo—studies on the role of the *gnom* gene. Development. 1993; 117:149–62.
96. Miyawaki K, Matsumoto-Kitano M, Kakimoto T. Expression of cytokinin biosynthetic isopentenyltransferase genes in *Arabidopsis*: tissue specificity and regulation by auxin, cytokinin, and nitrate. Plant J. 2004; 37:128–38. [PubMed: 14675438]
97. Moreno-Risueno MA, Busch W, Benfey PN. Omics meet networks—using systems approaches to infer regulatory networks in plants. Curr Opin Plant Biol. 2010; 13:126–31. [PubMed: 20036612]
98. Moreno-Risueno MA, Van Norman JM, Moreno A, Zhang J, Ahnert SE, Benfey PN. Oscillating gene expression determines competence for periodic *Arabidopsis* root branching. Science. 2010; 329:1306–11. [PubMed: 20829477]
99. Morita MT. Directional gravity sensing in gravitropism. Annu Rev Plant Biol. 2010; 61:705–20. [PubMed: 19152486]
100. Moubayidin L, Perilli S, Dello Ioio R, Di Mambro R, Costantino P, Sabatini S. The rate of cell differentiation controls the *Arabidopsis* root meristem growth phase. Curr Biol. 2010; 20:1138–43. [PubMed: 20605455]
101. Mouchel CF, Osmont KS, Hardtke CS. *BRX* mediates feedback between brassinosteroid levels and auxin signalling in root growth. Nature. 2006; 443:458–61. [PubMed: 17006513]
102. Müller A, Guan C, Galweiler L, Tanzler P, Huijser P, et al. *AtPIN2* defines a locus of *Arabidopsis* for root gravitropism control. EMBO J. 1998; 17:6903–11. [PubMed: 9843496]
103. Müller B, Sheen J. Cytokinin and auxin interaction in root stem-cell specification during early embryogenesis. Nature. 2008; 453:1094–97. [PubMed: 18463635]
104. Müller R, Bleckmann A, Simon R. The receptor kinase CORYNE of *Arabidopsis* transmits the stem cell–limiting signal CLAVATA3 independently of CLAVATA1. Plant Cell. 2008; 20:934–46. [PubMed: 18381924]
105. Mussig C, Shin GH, Altmann T. Brassinosteroids promote root growth in *Arabidopsis*. Plant Physiol. 2003; 133:1261–71. [PubMed: 14526105]
106. Nakajima K, Sena G, Nawy T, Benfey PN. Intercellular movement of the putative transcription factor SHR in root patterning. Nature. 2001; 413:307–11. [PubMed: 11565032]
107. Nawy T, Lee JY, Colinas J, Wang JY, Thongrod SC, et al. Transcriptional profile of the Arabidopsis root quiescent center. Plant Cell. 2005; 17:1908–25. [PubMed: 15937229]
108. Ohashi-Ito K, Bergmann DC. Regulation of the *Arabidopsis* root vascular initial population by *LONESOME HIGHWAY*. Development. 2007; 134:2959–68. [PubMed: 17626058]
109. Ohashi-Ito K, Fukuda H. Transcriptional regulation of vascular cell fates. Curr Opin Plant Biol. 2010; 13:670–76. [PubMed: 20869293]
110. Ohashi-Ito K, Oda Y, Fukuda H. *Arabidopsis* VASCULAR-RELATED NAC-DOMAIN6 directly regulates the genes that govern programmed cell death and secondary wall formation during xylem differentiation. Plant Cell. 2010; 22:3461–73. [PubMed: 20952636]
111. Okushima Y, Fukaki H, Onoda M, Theologis A, Tasaka M. ARF7 and ARF19 regulate lateral root formation via direct activation of *LBD*/*ASL* genes in *Arabidopsis*. Plant Cell. 2007; 19:118–30. [PubMed: 17259263]

112. Ortega-Martinez O, Pernas M, Carol RJ, Dolan L. Ethylene modulates stem cell division in the *Arabidopsis thaliana* root. Science. 2007; 317:507–10. [PubMed: 17656722]
113. Osmont KS, Sibout R, Hardtke CS. Hidden branches: developments in root system architecture. Annu Rev Plant Biol. 2007; 58:93–113. [PubMed: 17177637]
114. Pagant S, Bichet A, Sugimoto K, Lerouxel O, Desprez T, et al. *KOBITO1* encodes a novel plasma membrane protein necessary for normal synthesis of cellulose during cell expansion in Arabidopsis. Plant Cell. 2002; 14:2001–13. [PubMed: 12215501]
115. Pérez-Pérez JM, Serralbo O, Vanstraelen M, González C, Criqui M-C, et al. Specialization of CDC27 function in the *Arabidopsis thaliana* anaphase-promoting complex (APC/C). Plant J. 2008; 53:78–89. [PubMed: 17944809]
116. Petricka JJ, Benfey PN. Reconstructing regulatory network transitions. Trends Cell Biol. 2011; 21:442–51. [PubMed: 21632251]
117. Ploense SE, Wu MF, Nagpal P, Reed JW. A gain-of-function mutation in *IAA18* alters *Arabidopsis* embryonic apical patterning. Development. 2009; 136:1509–17. [PubMed: 19363152]
118. Rademacher EH, Möller B, Lokerse AS, Llavata-Peris CI, van den Berg W, Weijers D. A cellular expression map of the Arabidopsis *AUXIN RESPONSE FACTOR* gene family. Plant J. 2011; 68:597–606. [PubMed: 21831209]
119. Rashotte AM, Mason MG, Hutchison CE, Ferreira FJ, Schaller GE, Kieber JJ. A subset of *Arabidopsis* AP2 transcription factors mediates cytokinin responses in concert with a two-component pathway. Proc Natl Acad Sci USA. 2006; 103:11081–85. [PubMed: 16832061]
120. Rojo E, Sharma VK, Kovaleva V, Raikhel NV, Fletcher JC. CLV3 is localized to the extracellular space, where it activates the Arabidopsis CLAVATA stem cell signaling pathway. Plant Cell. 2002; 14:969–77. [PubMed: 12034890]
121. Roppolo D, De Rybel B, Tendon VD, Pfister A, Alassimone J, et al. A novel protein family mediates Casparian strip formation in the endodermis. Nature. 2011; 473:380–83. [PubMed: 21593871]
122. Sabatini S, Beis D, Wolkenfelt H, Murfett J, Guilfoyle T, et al. An auxin-dependent distal organizer of pattern and polarity in the *Arabidopsis* root. Cell. 1999; 99:463–72. [PubMed: 10589675]
123. Sabatini S, Heidstra R, Wildwater M, Scheres B. SCARECROW is involved in positioning the stem cell niche in the *Arabidopsis* root meristem. Genes Dev. 2003; 17:354–58. [PubMed: 12569126]
124. Sanz L, Dewitte W, Forzani C, Patell F, Nieuwland J, et al. The *Arabidopsis* D-type cyclin CYCD2;1 and the inhibitor ICK2/KRP2 modulate auxin-induced lateral root formation. Plant Cell. 2011; 23:641–60. [PubMed: 21357490]
125. Sarkar AK, Luijten M, Miyashima S, Lenhard M, Hashimoto T, et al. Conserved factors regulate signalling in *Arabidopsis thaliana* shoot and root stem cell organizers. Nature. 2007; 446:811–14. [PubMed: 17429400]
126. Scacchi E, Osmont KS, Beuchat J, Salinas P, Navarrete-Gomez M, et al. Dynamic, auxin-responsive plasma membrane-to-nucleus movement of *Arabidopsis* BRX. Development. 2009; 136:2059–67. [PubMed: 19465596]
127. Scheres B. Stem-cell niches: nursery rhymes across kingdoms. Nat Rev Mol Cell Biol. 2007; 8:345–54. [PubMed: 17450175]
128. Scheres B, Di Laurenzio L, Willemsen V, Hauser MT, Janmaat K, et al. Mutations affecting the radial organisation of the *Arabidopsis* root display specific defects throughout the embryonic axis. Development. 1995; 121:53–62.
129. Scheres B, Wolkenfelt H, Willemsen V, Terlouw M, Lawson E, et al. Embryonic origin of the *Arabidopsis* primary root and root meristem initials. Development. 1994; 120:2475–87.
130. Schiefelbein JW, Somerville C. Genetic control of root hair development in *Arabidopsis thaliana*. Plant Cell. 1990; 2:235–43. [PubMed: 12354956]
131. Schindelman G, Morikami A, Jung J, Baskin TI, Carpita NC, et al. COBRA encodes a putative GPI-anchored protein, which is polarly localized and necessary for oriented cell expansion in *Arabidopsis*. Genes Dev. 2001; 15:1115–27. [PubMed: 11331607]

132. Schlereth A, Möller B, Liu W, Kientz M, Flipse J, et al. MONOPTEROS controls embryonic root initiation by regulating a mobile transcription factor. Nature. 2010; 464:913–16. [PubMed: 20220754]
133. Schmid M, Davison TS, Henz SR, Pape UJ, Demar M, et al. A gene expression map of *Arabidopsis thaliana* development. Nat Genet. 2005; 37:501–06. [PubMed: 15806101]
134. Schumacher K, Vafeados D, McCarthy M, Sze H, Wilkins T, Chory J. The *Arabidopsis det3* mutant reveals a central role for the vacuolar H^+-ATPase in plant growth and development. Genes Dev. 1999; 13:3259–70. [PubMed: 10617574]
135. Serralbo O, Pérez-Pérez JM, Heidstra R, Scheres B. Non-cell-autonomous rescue of anaphase-promoting complex function revealed by mosaic analysis of *HOBBIT*, an *Arabidopsis CDC27* homolog. Proc Natl Acad Sci USA. 2006; 103:13250–55. [PubMed: 16938844]
136. Sigrist SJ, Lehner CF. Drosophila *fizzy-related* down-regulates mitotic cyclins and is required for cell proliferation arrest and entry into endocycles. Cell. 1997; 90:671–81. [PubMed: 9288747]
137. Sozzani R, Cui H, Moreno-Risueno MA, Busch W, Van Norman JM, et al. Spatiotemporal regulation of cell-cycle genes by SHORTROOT links patterning and growth. Nature. 2010; 466:128–32. [PubMed: 20596025]
138. Spencer MWB, Casson SA, Lindsey K. Transcriptional profiling of the Arabidopsis embryo. Plant Physiol. 2006; 143:924–40. [PubMed: 17189330]
139. Spradling A, Drummond-Barbosa D, Kai T. Stem cells find their niche. Nature. 2001; 414:98–104. [PubMed: 11689954]
140. Stahl Y, Wink RH, Ingram GC, Simon R. A signaling module controlling the stem cell niche in *Arabidopsis* root meristems. Curr Biol. 2009; 19:909–14. [PubMed: 19398337]
141. Steinmann T, Geldner N, Grebe M, Mangold S, Jackson CL, et al. Coordinated polar localization of auxin efflux carrier PIN1 by GNOM ARF GEF. Science. 1999; 286:316–18. [PubMed: 10514379]
142. Stepanova AN, Hoyt JM, Hamilton AA, Alonso JM. A link between ethylene and auxin uncovered by the characterization of two root-specific ethylene-insensitive mutants in Arabidopsis. Plant Cell. 2005; 17:2230–42. [PubMed: 15980261]
143. Stepanova AN, Robertson-Hoyt J, Yun J, Benavente LM, Xie DY, et al. TAA1-mediated auxin biosynthesis is essential for hormone crosstalk and plant development. Cell. 2008; 133:177–91. [PubMed: 18394997]
144. Sugimoto-Shirasu K, Stacey NJ, Corsar J, Roberts K, McCann MC. DNA topoisomerase VI is essential for endoreduplication in *Arabidopsis*. Curr Biol. 2002; 12:1782–86. [PubMed: 12401175]
145. Sun F, Zhang W, Hu H, Li B, Wang Y, et al. Salt modulates gravity signaling pathway to regulate growth direction of primary roots in Arabidopsis. Plant Physiol. 2008; 146:178–88. [PubMed: 18024552]
146. Swarup R, Kramer EM, Perry P, Knox K, Leyser HMO, et al. Root gravitropism requires lateral root cap and epidermal cells for transport and response to a mobile auxin signal. Nat Cell Biol. 2005; 7:1057–65. [PubMed: 16244669]
147. Szemenyei H, Hannon M, Long JA. TOPLESS mediates auxin-dependent transcriptional repression during *Arabidopsis* embryogenesis. Science. 2008; 319:1384–86. [PubMed: 18258861]
148. Thimann KV. Auxins and the growth of roots. Am J Bot. 1936; 23:561–69.
149. Tominaga-Wada R, Ishida T, Wada T. New insights into the mechanism of development of *Arabidopsis* root hairs and trichomes. Int Rev Cell Mol Biol. 2011; 286:67–106. [PubMed: 21199780]
150. Tsukagoshi H, Busch W, Benfey PN. Transcriptional regulation of ROS controls transition from proliferation to differentiation in the root. Cell. 2010; 143:606–16. [PubMed: 21074051]
151. Ubeda-Tomás S, Federici F, Casimiro I, Beemster GTS, Bhalerao R, et al. Gibberellin signaling in the endodermis controls *Arabidopsis* root meristem size. Curr Biol. 2009; 19:1194–99. [PubMed: 19576770]

152. Ubeda-Tomás S, Swarup R, Coates J, Swarup K, Laplaze L, et al. Root growth in *Arabidopsis* requires gibberellin/DELLA signalling in the endodermis. Nat Cell Biol. 2008; 10:625–28. [PubMed: 18425113]
153. Urano K, Kurihara Y, Seki M, Shinozaki K. "Omics" analyses of regulatory networks in plant abiotic stress responses. Curr Opin Plant Biol. 2010; 13:132–38. [PubMed: 20080055]
154. van den Berg C, Willemsen V, Hage W, Weisbeek P, Scheres B. Cell fate in the *Arabidopsis* root meristem determined by directional signalling. Nature. 1995; 378:62–65. [PubMed: 7477287]
155. van den Berg C, Willemsen V, Hendriks G, Weisbeek P, Scheres B. Short-range control of cell differentiation in the *Arabidopsis* root meristem. Nature. 1997; 390:287–89. [PubMed: 9384380]
156. Vandepoele K, Quimbaya M, Casneuf T, De Veylder L, Van de Peer Y. Unraveling transcriptional control in Arabidopsis using cis-regulatory elements and coexpression networks. Plant Physiol. 2009; 150:535–46. [PubMed: 19357200]
157. Vanneste S, De Rybel B, Beemster GTS, Ljung K, De Smet I, et al. Cell cycle progression in the pericycle is not sufficient for SOLITARY ROOT/IAA14-mediated lateral root initiation in *Arabidopsis thaliana*. Plant Cell. 2005; 17:3035–50. [PubMed: 16243906]
158. Van Norman J, Breakfield NW, Benfey PN. Intercellular communication during plant development. Plant Cell. 2011; 23:855–64. [PubMed: 21386031]
159. Vanstraelen M, Baloban M, Da Ines O, Cultrone A, Lammens T, et al. APC/C^{CCS52A} complexes control meristem maintenance in the *Arabidopsis* root. Proc Natl Acad Sci USA. 2009; 106:11806–11. [PubMed: 19553203]
160. Vartanian N, Marcotte L, Giraudat J. Drought rhizogenesis in *Arabidopsis thaliana*: differential responses of hormonal mutants. Plant Physiol. 1994; 104:761–67. [PubMed: 12232124]
161. Verbelen JP, De Cnodder T, Le J, Vissenberg K, Baluska F. The root apex of *Arabidopsis thaliana* consists of four distinct zones of growth activities: meristematic zone, transition zone, fast elongation zone and growth terminating zone. Plant Signal Behav. 2006; 1:296–304. [PubMed: 19517000]
162. Wang H, Zhu Y, Fujioka S, Asami T, Li J. Regulation of *Arabidopsis* brassinosteroid signaling by atypical basic helix-loop-helix proteins. Plant Cell. 2009; 21:3781–91. [PubMed: 20023194]
163. Wang Y, Li K, Li X. Auxin redistribution modulates plastic development of root system architecture under salt stress in *Arabidopsis thaliana*. J Plant Physiol. 2009; 166:1637–45. [PubMed: 19457582]
164. Weijers D, Benkova E, Jager KE, Schlereth A, Hamann T, et al. Developmental specificity of auxin response by pairs of ARF and Aux/IAA transcriptional regulators. EMBO J. 2005; 24:1874–85. [PubMed: 15889151]
165. Weijers D, Schlereth A, Ehrismann JS, Schwank G, Kientz M, Jürgens G. Auxin triggers transient local signaling for cell specification in *Arabidopsis* embryogenesis. Dev Cell. 2006; 10:265–70. [PubMed: 16459305]
166. Welch D, Hassan H, Blilou I, Immink R, Heidstra R, Scheres B. *Arabidopsis* JACKDAW and MAGPIE zinc finger proteins delimit asymmetric cell division and stabilize tissue boundaries by restricting SHORT-ROOT action. Genes Dev. 2007; 21:2196–204. [PubMed: 17785527]
167. West G, Inzé D, Beemster GTS. Cell cycle modulation in the response of the primary root of Arabidopsis to salt stress. Plant Physiol. 2004; 135:1050–58. [PubMed: 15181207]
168. Wiedemeier AMD, Judy-March JE, Hocart CH, Wasteneys GO, Williamson RE, Baskin TI. Mutant alleles of *Arabidopsis RADIALLY SWOLLEN 4* and *7* reduce growth anisotropy without altering the transverse orientation of cortical microtubules or cellulose microfibrils. Development. 2002; 129:4821–30. [PubMed: 12361973]
169. Willemsen V, Bauch M, Bennett T, Campilho A, Wolkenfelt H, et al. The NAC domain transcription factors FEZ and SOMBRERO control the orientation of cell division plane in *Arabidopsis* root stem cells. Dev Cell. 2008; 15:913–22. [PubMed: 19081078]
170. Wilmoth JC, Wang S, Tiwari SB, Joshi AD, Hagen G, et al. NPH4/ARF7 and ARF19 promote leaf expansion and auxin-induced lateral root formation. Plant J. 2005; 43:118–30. [PubMed: 15960621]

171. Wolters H, Anders N, Geldner N, Gavidia R, Jurgens G. Coordination of apical and basal embryo development revealed by tissue-specific GNOM functions. Development. 2010; 138:117–26. [PubMed: 21138974]

172. Wolters H, Jürgens G. Survival of the flexible: hormonal growth control and adaptation in plant development. Nat Rev Genet. 2009; 10:305–17. [PubMed: 19360022]

173. Yamada K, Lim J, Dale JM, Chen H, Shinn P, et al. Empirical analysis of transcriptional activity in the *Arabidopsis* genome. Science. 2003; 302:842–46. [PubMed: 14593172]

174. Yamaguchi M, Mitsuda N, Ohtani M, Ohme-Takagi M, Kato K, Demura T. VASCULAR-RELATED NAC-DOMAIN 7 directly regulates the expression of a broad range of genes for xylem vessel formation. Plant J. 2011; 66:579–90. [PubMed: 21284754]

175. Yamaguchi S, Murakami H, Okayama H. A WD repeat protein controls the cell cycle and differentiation by negatively regulating Cdc2/B-type cyclin complexes. Mol Biol Cell. 1997; 8:2475–86. [PubMed: 9398669]

176. Zeevaart JAD, Creelman RA. Metabolism and physiology of abscisic acid. Annu Rev Plant Physiol Plant Mol Biol. 1988; 39:439–73.

177. Zhang H, Han W, De Smet I, Talboys P, Loya R, et al. ABA promotes quiescence of the quiescent centre and suppresses stem cell differentiation in the Arabidopsis primary root meristem. Plant J. 2010; 64:764–74. [PubMed: 21105924]

178. Zhang J, Elo A, Helariutta Y. *Arabidopsis* as a model for wood formation. Curr Opin Biotechnol. 2011; 22:293–99. [PubMed: 21144727]

179. Zhao Y, Wang T, Zhang W, Li X. SOS3 mediates lateral root development under low salt stress through regulation of auxin redistribution and maxima in Arabidopsis. New Phytol. 2011; 189:1122–34. [PubMed: 21087263]

180. Zhou W, Wei L, Xu J, Zhai Q, Jiang H, et al. *Arabidopsis* tyrosylprotein sulfotransferase acts in the Auxin/PLETHORA pathway in regulating postembryonic maintenance of the root stem cell niche. Plant Cell. 2010; 22:3692–709. [PubMed: 21045165]

181. Zhu J-K. Salt and drought stress signal transduction in plants. Annu Rev Plant Biol. 2002; 53:247–73. [PubMed: 12221975]

182. Zolla G, Heimer YM, Barak S. Mild salinity stimulates a stress-induced morphogenic response in *Arabidopsis thaliana* roots. J Exp Bot. 2010; 61:211–24. [PubMed: 19783843]

SUMMARY POINTS

1. Formation of the embryonic root involves hormones and transcriptional regulation that includes a non-cell-autonomous mechanism of hypophyseal cell specification.
2. Hormonal crosstalk impacts RAM size. In particular, antagonism between auxin and cytokinin is important in control of the RAM.
3. Studies of root branching indicate that auxin also plays a role in LR initiation. Oscillations of transcriptional expression correspond to the formation of prebranch sites along the primary root that precedes LR initiation.
4. The auxin pathway may integrate endogenous and exogenous cues to coordinate a developmental response.
5. Root gene regulatory networks have been inferred from coexpression analysis that uses data generated from genome-wide profiling experiments. New predictions about gene interactions and functions have emerged that might be used for manipulation in crop species.

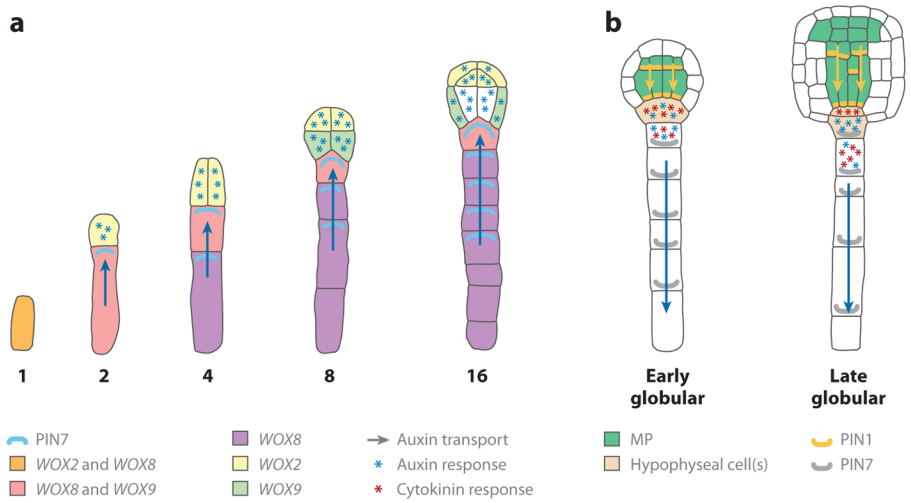

Figure 1.

The making of an embryonic root: a schematic of early embryogenesis. (*a*) The 1–16-cell embryonic stages. *WOX* genes are expressed in differential and overlapping patterns that might regulate cell fate decisions, including the specification of apical-basal polarity at the 2-cell stage by *WOX2* in the apical cell (*yellow*) and *WOX8* and *WOX9* in the basal cell (*pink*). Auxin also establishes this polarity as the PIN7 transporter (*light blue*) directs auxin apically from the basal and suspensor cells into the apical portion of the embryo. The blue arrows denote the direction of auxin flow in these early stages; the blue stars indicate the cells where auxin response maxima are observed. (*b*) The early and late globular stages. During these stages the precursor cell to embryonic root formation, the hypophyseal cell (*peach*), is specified and then divides asymmetrically to produce different root cell lineages. Expression of the transcription factor MP (*green*) results in upregulation of *TMO7* RNA in the proembryo. In turn, some of the TMO7 proteins move into the presumptive hypophyseal cell to specify hypophyseal cell identity. Auxin also establishes this identity as it is transported into the hypophyseal cell by PIN1 carriers (*yellow*) of the proembryo and out of this cell by basally localized PIN7 carriers (*gray*) in the suspensor cells. As a result of transport, a maximum of auxin response is seen in the hypophyseal cell and the uppermost suspensor cell of the early globular stage (*blue stars*). Cytokinin response (*red stars*) is observed in the same cells as auxin response at this stage. However, after hypophyseal division, cytokinin and auxin responses are found in the apical and basal daughter cells, respectively, of the late-globular-stage embryo. This inverse expression profile is thought to be important for subsequent specification of embryonic root tissues. Yellow and blue arrows indicate the direction of auxin transport.

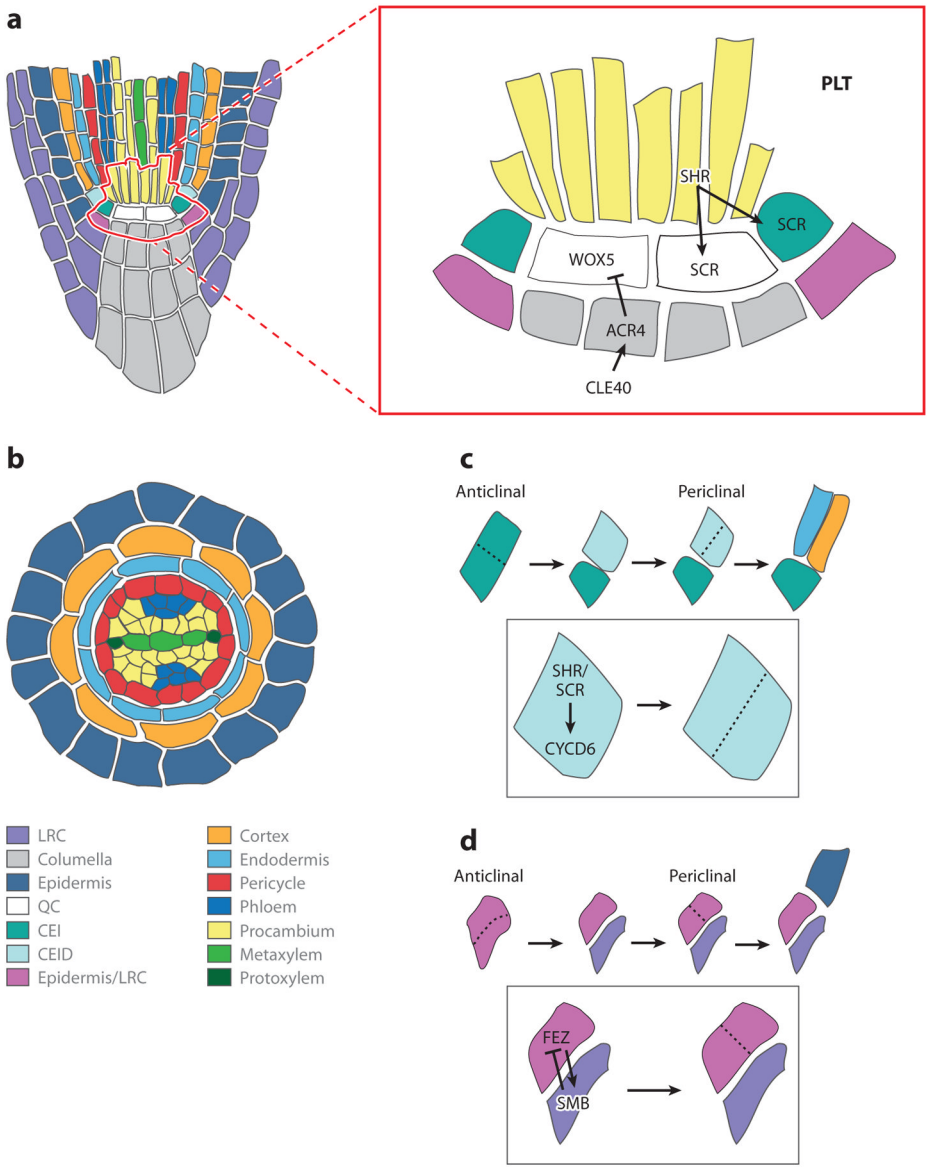

Figure 2.

Patterning the root apical meristem. (*a*) Organization of the root apical meristem. Different cell types are arranged in cell files along the length of the root. The magnified region shows the stem cell niche and regulatory interactions that maintain it. SHR expressed in the stele moves into the quiescent center (QC) and cortex/endodermal initial (CEI) cells to maintain QC and stem cell identity; WOX5 maintains identity of the surrounding stem cells. *WOX5* expression is confined to the QC through repression by ACR4, triggered by the CLE40 signal originating from differentiating columella cells. *PLT* expression throughout the niche also maintains QC and stem cell identity. (*b*) Organization of cell types within the stele. The diagram shows a cross section of the root tip. The pattern of cell types in the stele is bilaterally symmetric: A central axis of xylem is flanked by two phloem bundles. (*c*) Divisions of the CEI. The CEI first divides anticlinally to give rise to the CEI daughter (CEID) cell. The CEID then divides periclinally to give rise to the cortex and endodermal cell lineages. Activation of *CYCD6* by SHR and SCR plays an important role in this asymmetric cell division. (*d*) Divisions of the epidermis/lateral root cap (LRC) initial. The

epidermis/ LRC initial divides first periclinally to produce an LRC cell and then anticlinally to produce an epidermal cell. FEZ, expressed in the initial, represses *SMB* in the LRC daughter cell. SMB then represses *FEZ*, restricting it to the initial cells. Figure adapted from Reference 158.

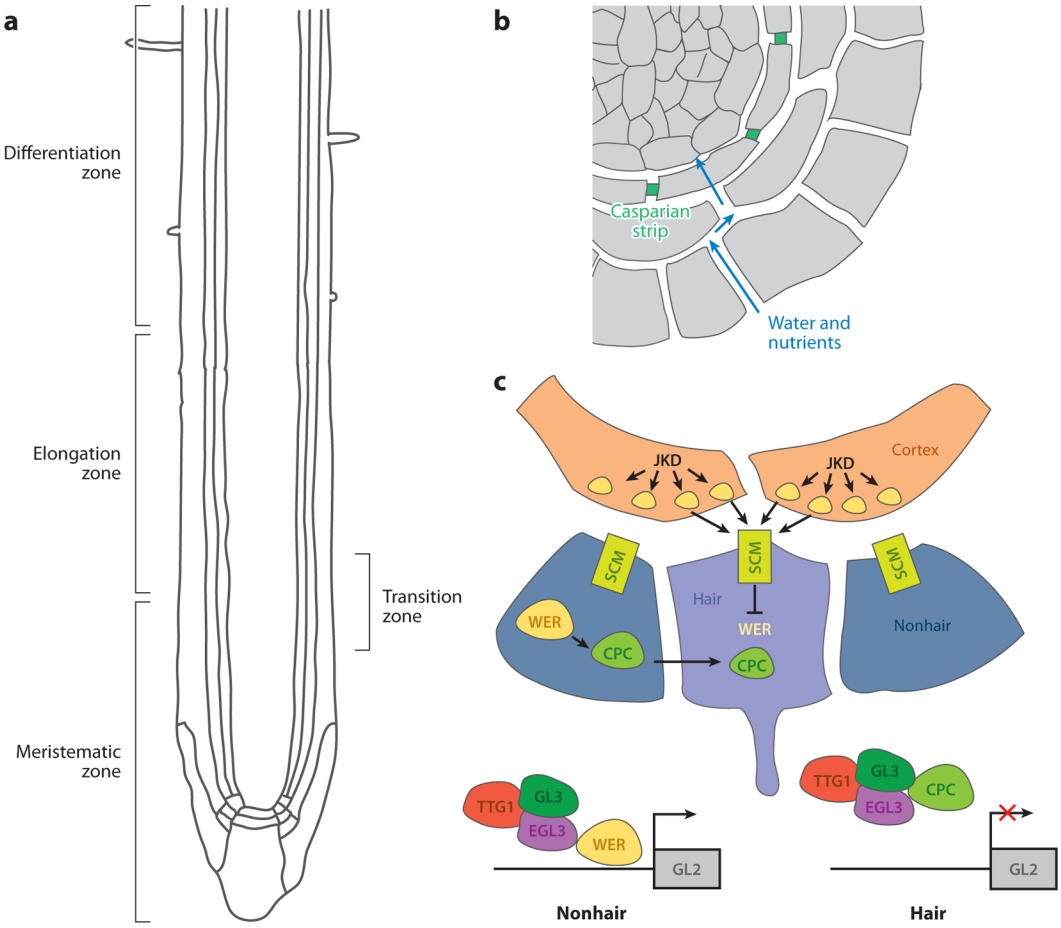

Figure 3.

Developmental zones of the *Arabidopsis* root. (*a*) Distinct developmental zones, shown in a longitudinal section through the primary root. Cell division occurs in the meristematic zone, cell expansion and elongation occur in the elongation zone, and cell differentiation (indicated by the formation of root hairs) occurs in the differentiation zone. The zone of transition between the meristematic and elongation zones is also indicated. (*b*) Cross section of the root tip showing the Casparian strip, which forms an impermeable barrier between endodermal cells such that water and nutrients must pass through endodermal cells en route to the vasculature. (*c*) Specification of root hairs. WER binds to a complex of other factors and activates *GL2* and *CPC* in nonhair cells. CPC then moves to presumptive hair cells, where it competes with WER for binding to the complex. Signaling through SCM represses *WER*, which tips the balance in favor of CPC. The CPC-containing complex cannot upregulate *GL2* expression, allowing specification of hair cell fate. The greater surface area between hair cells and overlying cortex cells may allow greater transmission of a JKD-dependent signal, which preferentially activates SCM in presumptive hair cells.

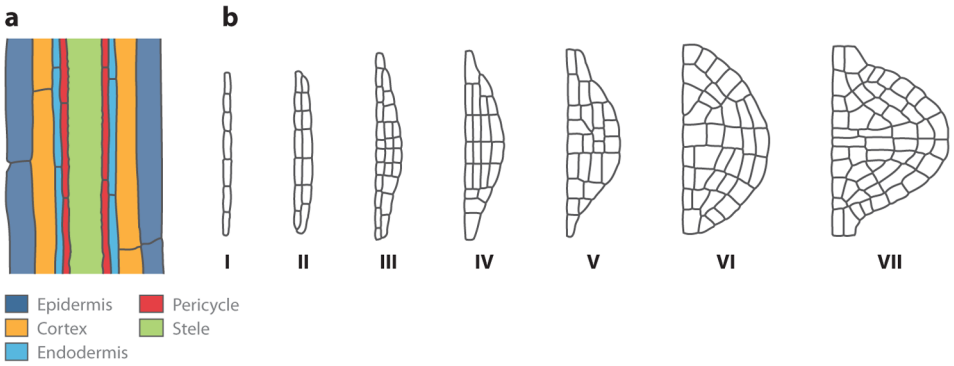

Figure 4.

Lateral root development. (*a*) Schematic of the differentiation zone, showing a slice through the longitudinal axis of the primary root in the differentiation zone. Lateral roots are initiated in the pericycle cell layer (*red*) when anticlinal cell divisions occur in a subset of cells in this layer. (*b*) Stages of lateral root development. In stage I, small pericycle cells are seen that result from the anticlinal divisions in this layer. During stage II, the cells of stage I divide periclinally to form inner and outer layers. By stage III, the dome shape of the lateral root primordium (LRP) is apparent owing to the periclinal divisions of the outer layer and absence of these divisions in the more peripheral cells. The three-layered LRP of stage III becomes a four-layered LRP as a result of periclinal divisions during stage IV. In stage V, the cells of all layers undergo anticlinal divisions to generate an LRP that begins to push through the cortex layer of the primary root. In stage VI, the LRP starts to resemble a mature root tip, with epidermal, cortex, and endodermal cell layers from the outside to the inside of the LRP, respectively. The innermost tissue, the stele, becomes distinguishable and the LRP continues to undergo anticlinal cell divisions as it enlarges during stage VII. At the end of this stage, the LRP is about to emerge from the epidermis of the parent primary root as a lateral root.