Plant Physiol. (1996) 111: 959–964

Update on Development

Roots Redefined: Anatomical and Genetic Analysis of Root Development

Ben Scheres*, Heather I. McKhann, and Claudia van den Berg

Department of Molecular Cell Biology, Utrecht University, Padualaan 8, 3584 CH Utrecht, The Netherlands

The postembryonic development of plants is fueled by apical meristems, which are the local production sites of new cells that form a pattern of different cell types within an organ. The regularity of this pattern in the root yielded ideas on its formation from the meristem well before critical studies on the shoot apex were performed (e.g. Hanstein, 1870). The model plant *Arabidopsis thaliana*, which allows genetic dissection of root development, is a paragon of this regularity. In this *Update*, we review recent studies on root anatomy and genetics that are allowing us to refine, and perhaps redefine, our understanding of organ development. We will focus on two pivotal aspects of root development: pattern formation and cell proliferation. Important work on other aspects of Arabidopsis roots, such as cell elongation/morphogenesis, tropism, and cell size and shape, has been covered in recent reviews (Benfey and Schiefelbein, 1994; Dolan and Roberts, 1995) and will not be discussed here.

THE ARABIDOPSIS ROOT HAS A REGULAR CELLULAR ORGANIZATION

Arabidopsis roots are particularly consistent in their cellular organization (Fig. 1A) (Dolan et al., 1993). They contain four main tissues: an outer layer of epidermal cells, a middle layer of parenchymal and endodermal cortex cells, an innermost region of pericycle and vascular cells, and the distally located root cap. The four tissues arise from files of cells that originate from dividing cells at the root apical meristem. The cells at the end points of these files are defined as the meristem initial cells. With each division they add one cell to the plant body, while the initial cell retains its position within the meristem. The epidermis/lateral root cap layers, the cortical parenchyma/endodermis layers, and the pericycle/vascular bundle arise in this way from two rings and one plate of initials, respectively (Fig. 1A). The columella root cap arises from a distal plate of initial cells (Fig. 1A). All initial cells together are defined as the promeristem, the minimal group of cells that is capable of making all tissues by ordered divisions (Clowes, 1954). In Arabidopsis, the initial cells surround and contact a small group of cells that are mostly mitotically inactive, the quiescent center.

* Corresponding author; e-mail b.scheres@cc.ruu.nl; fax 31–30–251–3655.

ONTOGENY OF PRIMARY AND LATERAL ROOTS IN ARABIDOPSIS

The primary root is laid down at the basal end of the embryo. Secondary roots are formed postembryonically in a different developmental context (Fig. 1, B and C). Lateral roots, for example, arise from pericycle cells within the primary root that recommence divisions. Nevertheless, the resulting cellular organization of both root types is virtually identical. To highlight differences and similarities in ontogeny, we will apply the terms "founder cells," "primordium," and "apical meristem" in the following way: The term "founder cells" is used to define the minimal set of preexisting cells that exclusively contribute to a complete organ. We use "primordium" to refer to precursor cells of an organ from the stage that they form a separate cell group until the cessation of division of a subset of terminally differentiating cells. When terminally differentiating cells have ceased to divide, the developing organ is no longer at the primordium stage, and we use "apical meristem" for the cells that remain mitotically active at the apex.[1]

Primary Roots

During embryogenesis, the root primordium is formed within a mass of dividing cells. In Arabidopsis, the embryonic root primordium becomes a distinct cell group at the heart stage of embryogenesis (Scheres et al., 1994). Part of this primordium is derived from daughters of the apical cell produced at the first zygotic division. Another part derives from the hypophyseal cell, a daughter of the basal cell formed in the first zygotic division (Fig. 1B). A few individual daughter cells of the uppermost tier of the late heart-stage root primordium, at the boundary between root and hypocotyl, may become incorporated into the hypocotyl (Scheres et al., 1994; cf. Fig. 1B). Since cells at the organ boundary are not committed to root fate, we argue against using the term "founder cells" for the earliest distinguishable primary root primordium. We include this upper tier in the root primordium because the cells at this

[1] To clarify the difference between the root meristem and the shoot apical meristem, the former can be called a first-order meristem (producing differentiated cell types directly) and the latter a second-order meristem (producing organ primordia as appendages; these primordia will produce first-order meristems).

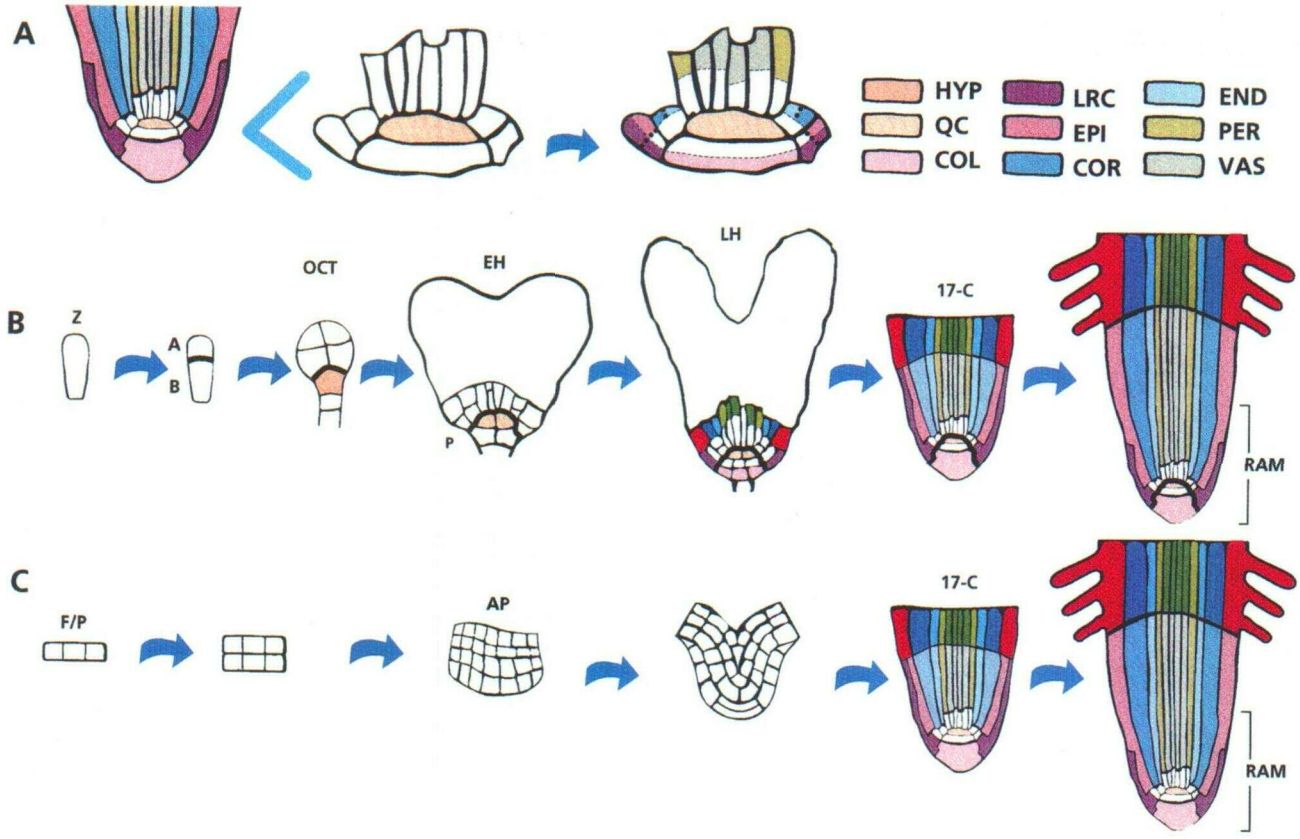

Figure 1. Organization and ontogeny of the Arabidopsis root. A, Root apical meristem organization. Promeristem is in white; the middle and right illustrations depict how initial cells maintain the promeristem and add cells to the various tissues. Dashed line, First division; dotted lines, second division. B, Primary root ontogeny. From left to right, Zygote, two-cell, octant, early-heart, late-heart, and mature ("17-cell") stages of embryogenesis, and seedling root. Intense coloring, Upper tier of the primordium, which terminally differentiates upon germination and produces a few uppermost daughters that acquire hypocotyl fate. Z, Zygote; A, apical cell; B, basal cell; Oct, octant-stage embryo; P, earliest separated root primordium; EH, early-heart stage; LH, late-heart stage; 17-C, primordium with 17 cortex/epidermis cells per file, RAM, root apical meristem. C, Schematic representation of lateral root ontogeny. F/P, Founder cells, earliest separated primordium; AP, autonomous primordium; HYP, hypophyseal cell; QC, quiescent center; COL, columella root cap; LRC, lateral root cap; EPI, epidermis; COR, cortex parenchyma; END, endodermis; PER, pericycle; VAS, vascular tissue.

position act predominantly as precursors for the root, with only a few descendants ending up in the hypocotyl.

Cells within the upper tier of the late heart-stage root primordium perform a small number of embryonic divisions and undergo terminal differentiation upon germination. The other tiers contain the incipient meristem initial cells. Stereotyped divisions of the initial cells and their derivatives lead to the production of the part of the mature embryonic root that is enclosed by the incipient root cap (Fig. 1B). Only this part of the root recommences division upon germination and forms the root apical meristem. The meristem continues to produce cells that elongate and differentiate within the growing root (Fig. 1B).

Lateral Roots

Lateral root formation is initiated from within a previously nondividing tissue. Hence, the onset of cell division and separation of the root primordium coincide during lateral root formation. Laskowski et al. (1995) estimated that the Arabidopsis lateral root initiates from about 11 founder cells within the pericycle. Radial expansion and periclinal divisions enlarge the root primordium to several cell layers (Fig. 1C). Root primordia of at least three to five layers can develop autonomously upon excision (Laskowski et al., 1995) (Fig. 1C, AP). This important experiment defines a stage of root development by functional criteria, whereas the criteria mentioned so far are based on anatomy. Autonomous primordia were termed "meristems," but to avoid confusion with the more general use of this term we propose to name this stage the "autonomous primordium" stage. The autonomous primordium is similar in cell number to the first distinguishable root primordium at the early heart stage (cf. Fig. 1, B and C).

The three- to five-cell-layer lateral root primordium undergoes further cell proliferation until its first (oldest) derivatives cease to divide and differentiate terminally, leav-

Table I. *Root patterning and cell division mutants of A. thaliana*
P, Pattern; A/B, apical basal; R, radial; M, morphogenesis; D, division.

Genes	Class	Stage of First Mutant Defect	Primary Root Phenotype		Secondary Root Phenotype	Reference
			Embryonic	Postembryonic		
monopteros (mp)	P; A/B	Octant	Aberrant axialization of lower tier; aberrant hypophysis	Lack of root and hypocotyl; vascularization defects	Normal	Berleth and Jürgens, 1993
hobbit (hbt) *bombadil (bbl)* *orc* *gremlin (grm)*	P; A/B	Globular, heart	Aberrant hypophysis	Misspecification hypophysis-derived cells; no meristem activity	Like primary root	Scheres et al., 1996
shortroot (shr)	P; R	Heart	Tissue layer missing	No endodermis	Like primary root	Benfey et al., 1993; Scheres et al., 1995
scarecrow (scr) *wooden leg (wol)*	P/M; R	Heart	Tissue layer/cells missing	No cortex parenchyma/ vascular cells	Like primary root (*wol*) intermediate	
transparent testa glabra (ttg), glabra (gl2), constitutive triple response (ctrl)	P	Postembryonic root (?)		Excessive hair-bearing epidermis cells	Like primary root	Dolan et al., 1994; Galway et al., 1994; Masucci et al., 1996
root meristemless (rml1,2)	D; auxin independent	"17-cell-" root primordium		No cell division after germination	No cell division at primary root primordium size	Cheng et al., 1995
aberrant lateral root formation (alf3)	D; auxin dependent	1- to 2-mm lateral root primordium		Arrested growth by 8 d after germination	Multiple aborted lateral root primordia	Celenza et al., 1995
superroot (sur1 = alf1 = hls3 = rty)	D; auxin dependent	Lateral root founder cells		Reduced elongation; elevated auxin levels	Multiple secondary root primordia	Boerjan et al., 1995; Celenza et al., 1995
alf4	D/P	Lateral root founder cells			No lateral root primordia	Celenza et al., 1995

ing the remaining dividing cells as the new apical meristem. Reliable anatomical similarities between lateral root primordia of five to eight layers and primary root primordia have not yet been reported. Only when the lateral root primordium grows beyond eight cell layers do the anatomical and histological pattern elements similar to those of the primary root appear.

PATTERN FORMATION AND POSITIONAL INFORMATION

Within the root primordium, cells acquire distinct identities in a specific spatial arrangement. When does this process of pattern formation take place, and how is the apical meristem involved? When the primary root primordium is first recognized as a separate cell group, distinct cellular features can be observed: the procambium cells are elongated and characteristic divisions in the hypophyseal cell are manifest. Although the distinct cell shapes in the root primordium imply that a pattern is laid down at that stage, it should be noted that cell shape is not necessarily linked to cell type. Cell types are also specified correctly in mutant backgrounds with irregular cell shapes (Torres-Ruiz and Jürgens, 1994; Traas et al., 1995). Nevertheless, the anatomical features hint that pattern formation is occurring early, before the cells that make up the root form a separate group.

The strict lineage relationships of cells within the Arabidopsis primary root may mislead one to assume that all cells of a given tissue layer, up to the meristem initial cells, have a fixed fate after early pattern formation. The importance and continued existence of positional information within the Arabidopsis root meristem is demonstrated by laser ablation experiments (van den Berg et al., 1995). For example, when the quiescent center was ablated, pericycle and provascular cells were displaced to the distal tip of the root and they subsequently acquired root cap characteristics. Upon ablation of cortical initials, pericycle initial cells expanded, divided into the resulting cavity, and subsequently formed cortical parenchyma and endodermis. These and related experiments show that positional information is important in the determination of cell fate. Where does this information come from? Ablation of daughter cells of cortical initial cells indicates that the initial needs contact with daughter cortical cells to give rise to two different cell types (van den Berg et al., 1995). This implies that cortical initial cells, and perhaps all initial cells, depend on positional information from more mature daughters within the same cell layer. In conclusion, meristem initial cells that need to adopt different fates cannot rely exclusively on intrinsic information. Whether positional information is continuously generated in more mature regions of the root, or if it is perpetuated from cells that have been instructed in a single early embryonic patterning event is currently unknown.

GENES DEFINE ROOT DEVELOPMENT

A description of root development based on the wild-type embryo and seedling can form a context in which to raise meaningful questions. However, root mutants are necessary to understand the mechanisms that underlie development. Genes define root structure and function from the plant's point of view. Table I summarizes mutants

affecting cell division and pattern formation in the root primordium/meristem. The table contains genes defined by multiple recessive alleles, which allow fairly reliable predictions on gene function, as well as loci defined by single recessive alleles, which may give an incomplete picture of gene function. The identification of single-allele loci, as well as loci defined by more then 10 alleles in the same mutant screen (e.g. Scheres et al., 1996) and the anatomical linkage between the root pattern and early embryonic events, suggest that null mutants in a number of single-allele loci display embryo-lethal phenotypes.

Genes Involved in Pattern Formation

The *MONOPTEROS* (*MP*) gene is identified by numerous alleles. Strong *mp* alleles lead to a seedling that lacks both root and hypocotyl (Berleth and Jürgens, 1993). *mp* mutants first display aberrant elongation and division patterns in the basal embryo region at or before the octant stage of embryogenesis, in line with the fact that the mutant seedlings display a large basal deletion. The embryonic phenotype suggests that the *MP* gene product might be required for inductive signaling in the basal region of the embryo. In *mp* mutants, normal secondary roots can be formed, so they are not impaired in root formation per se. Apparently, *MP* gene activity sets the stage for root primordium formation only during embryogenesis.

Hypophyseal cell mutants have a later and more locally restricted embryonic phenotype. In mutant embryos, the cells in the position of the hypophyseal cell perform aberrant divisions. The best-analyzed mutants are those in the *HOBBIT* (*HBT*) gene, which is defined by more than 10 alleles. In *hbt* seedlings, cells derived from the hypophyseal region are misspecified, e.g. strong mutants form no columella root cap (Scheres et al., 1996). It is noteworthy that no active root meristem is established in any of the hypophyseal cell mutants. Correct specification of the hypophyseal cell region appears to be a necessary prerequisite for the establishment of an apical meristem. Secondary roots from these mutants establish no apical meristem and, as in the primary root, cell specification within the cap region is aberrant. Therefore, the corresponding genes also appear to be necessary in secondary roots, although an equivalent to the hypophyseal cell cannot be identified anatomically.

In addition to the apparently important hypophyseal cell derivatives, the root meristem initial cells can be identified at the earliest cellular separation of the embryonic primordium. Do "initial-specific" genes exist that directly specify the promeristem? Or, alternatively, are initial cells different from their immediate progeny only because they reside at a tissue boundary? The laser ablation studies provide no evidence that meristem initial cells are sources of patterning information or that they possess unique developmental potential. Some tissue-specific markers are expressed in initial cells, also indicating that initial cells are not different from their immediate progeny (van den Berg et al., 1995). However, the characteristic organization of all initial cells surrounding the quiescent center suggests some function. Future analysis of the expression domain of hypophyseal cell genes, or the identification of promeristem-specific marker lines, may shed light on this anticipated function.

A number of radial mutants lack particular tissues in both the root and the hypocotyl, highlighting the fact that the two organs share genes important for their radial organization (Benfey et al., 1993; Scheres et al., 1995). All mutants display aberrations in the corresponding cell layers within the embryo axis. The *wooden leg* (*wol*), *scarecrow* (*scr*), and *shortroot* (*shr*) genes simultaneously reduce the number of cells and the number of cell types. In double mutants with the *fass* mutant, which contains additional embryonic cell layers, the missing cell types in *scr* and *wol* appeared to be restored. The missing endodermis in *shr* could not be restored (Scheres et al., 1995). Therefore, radial pattern formation may be influenced by two classes of genes: one directly involved in pattern formation (e.g. *shr*) and a less-recognized class that acts on the resulting pattern via regulation of morphogenesis (i.e. cell division). This may turn out to be a common theme in plants. For example, the floral gene *SUPERMAN* also appears to act on pattern formation via regulation of cell proliferation (Sakai et al., 1995). The radial mutants exhibit their characteristic defects in secondary roots, as do the hypophyseal group mutants, implying that the corresponding genes are necessary for primordium formation in different developmental contexts.

The trichome patterning mutants *glabrous* (*gl2*) and *transparent testa glabra* (*ttg*) and the ethylene response mutant *constitutive triple response* (*ctr1*) have been found to display an altered distribution of hair and nonhair files in the root epidermis (Dolan et al., 1994; Galway et al., 1994; Masucci et al., 1996). The relation of trichome patterning and ethylene perception to root epidermal patterning is amenable to molecular studies, because the *CTR1* and *GL2* genes have been cloned (Kieber et al., 1993; Rerie et al., 1994). No embryonic markers are available yet for hair cells, so the cloned genes will have to reveal whether hair cell patterning is an exclusively postembryonic phase of root development.

Two major conclusions can be drawn from the mutants with defects in root-pattern formation. First, the phenotypic defects of the majority of patterning mutants are first visible at or before the cellular separation of the embryonic root primordium. This indicates that patterning information is already laid down when the embryonic root primordium can be first recognized, as was suggested from the morphological divergence of cell types. Second, pattern formation within the secondary root primordium involves to a large extent the same genes that are used in the embryo. Lateral roots display morphological differentiation characteristics relatively late (at the 8- to 10-layer stage), which may indicate that the final cellular pattern can be laid down only after initial cell proliferation has created a critical primordium size.

Genes Involved in Cell Proliferation of Primary and Secondary Roots

The *root meristemless* (*rml1* and *rml2*) mutants undergo normal embryogenesis based on anatomical criteria but

lack cell proliferation in the root postembryonically. As a result, these mutants fail to establish a root apical meristem (Cheng et al., 1995). It is interesting that in two independent *rml1* alleles, the largest lateral root primordia have a size comparable to that of the mature embryo root primordium: about 17 epidermal and cortical cells in a single file. The *RML1* gene may define a threshold point for root apical meristem establishment in primary roots (at the mature embryonic root size) as well as in secondary roots. This stage corresponds to the anatomically defined transition from the root primordium to the apical meristem stage, which may also be defined genetically.

Another mutant affecting the maintenance of cell proliferation is *aberrant lateral root formation* (*alf3*) (Celenza et al., 1995). In contrast to the *rml* mutants, *alf3* is considered a lateral root mutant, since it forms multiple, arrested lateral root primordia on a growing primary root. However, *alf3* mutant plants are also defective in primary root development by 8 d after germination. Whether stronger *alf3* alleles exist that completely block cell proliferation in the primary root remains to be seen. The *alf3* phenotype can be rescued by indole or IAA (Celenza et al., 1995). In contrast, the *rml* mutants cannot be rescued by any tested plant growth regulator (Cheng et al., 1995). Arrested *alf3* lateral root primordia are variable in size and therefore do not sharply define a developmental transition (J. Celenza, personal communication).

Genes Involved in Secondary Root Formation

Mutants with defects only in secondary roots are also known. The *superroot* (*sur1*) (= *alf1*, *hls3*, and *rty*) mutants overproliferate lateral root primordia and contain elevated levels of free and conjugated IAA (Boerjan et al., 1995; Celenza et al., 1995, and refs. therein). These mutants demonstrate genetically the long-known fact that auxin stimulates lateral root initiation. On the other hand, the *alf4* mutant lacks detectable lateral and adventitious root primordia (Celenza et al., 1995). If *alf4* is a null mutant and not a weak allele of a gene also involved in primary root development, the *ALF4* gene may be involved in the spatial definition of the lateral root founder cells as a patterning gene. Alternatively, the *ALF4* gene could be a specific cell-cycle gene responding to a result of pattern formation.

In conclusion, primary and lateral root primordium formation appear to have different genetic requirements. This correlates with the obvious differences in primary and secondary root primordium formation. In the embryo, the root is specified as an element of the complete apical-basal embryonic pattern, depending on the early activity of patterning genes such as *MP*. In lateral roots, pericycle cells need to dedifferentiate and divide, perhaps specifically controlled by *ALF4*.

CONCLUSIONS AND PROSPECTS: FROM CELL FAITH TO CELL FATE

Different stages of Arabidopsis primary and lateral root development can now be defined with some precision. However, regardless of how much faith one has in anatomical definitions, they should not be taken as more than a means of communication prior to subsequent genetic analysis. Mutants in patterning and cell proliferation within the root have now been identified. Most patterning mutants affect embryogenesis and secondary root formation. Apical/basal patterning genes such as *MP* and *HBT* act prior to the separation of the root primordium. Mutants affecting the radial organization affect the complete embryonic axis, and gene activity is thus not restricted to the root primordium. On the other hand, some mutants affect local cell proliferation and define stages that we can also recognize anatomically.

The emerging picture is one of embryonic genes that bring about the cellular organization of the root, and later-acting cell proliferation genes that establish a meristem that continuously perceives positional information. Therefore, in terms of pattern formation, the root appears to define the meristem, and not vice versa. However, it is too early to design genetic models explaining how genes make a root. The single-allele status of many loci hampers phenotypic interpretation and indicates that more, perhaps early embryonic, genes are yet to be discovered.

Can we now concentrate on Arabidopsis and forget about the classical plant species from which roots have been studied in detail? Of course, the answer is no for at least two reasons. First, some roots greatly facilitate biochemical and histological studies. For example, recent experiments in maize on the quiescent center, a group of cells whose function is still a mystery, have provided clues to how such an area of restrained growth may be set up within the root apex (Kerk and Feldman, 1995). Second, orthologs of any gene implicated in Arabidopsis root formation need to be studied in other well-described root systems to judge how generally these genes are used. In this respect, the notion that the Arabidopsis root meristem cells rely on positional cues for pattern formation is important. It suggests that what we will learn about the acquisition of cell fate within the exceptionally rigidly organized Arabidopsis root meristem may hold true for root meristems in general.

ACKNOWLEDGMENTS

We thank Dick Smit for preparing the figure; John Celenza for sharing unpublished results; and Philip Benfey, Thomas Berleth, and Peter Weisbeek for useful suggestions on the manuscript.

Received February 21, 1996; accepted March 19, 1996.
Copyright Clearance Center: 0032–0889/96/111/0959/06.

LITERATURE CITED

Benfey P, Linstead P, Roberts K, Schiefelbein J, Hauser M-T, Aeschbacher R (1993) Root development in *Arabidopsis*: four mutants with dramatically altered root morphogenesis. Development **119:** 57–70

Benfey P, Schiefelbein J (1994) Getting to the root of plant development: the genetics of *Arabidopsis* root formation. Trends Genet **10:** 84–88

Berleth T, Jürgens G (1993) The role of the *monopteros* gene in organising the basal body region of the *Arabidopsis* embryo. Development **118:** 575–587

Boerjan W, Cervera M-T, Delarue M, Beeckman T, Dewitte W, Bellini C, Caboche M, Van Onckelen H, Van Montagu M, Inzé D (1995) *superroot*, a recessive mutation in *Arabidopsis*, confers auxin overproduction. Plant Cell **7:** 1405–1419

Celenza J Jr, Grisafi P, Fink G (1995) A pathway for lateral root formation in *Arabidopsis thaliana*. Genes Dev **9:** 2131–2142

Cheng J-C, Seeley K, Sung ZR (1995) *RML1* and *RML2*, *Arabidopsis* genes required for cell proliferation at the root tip. Plant Physiol **107:** 365–376

Clowes FAL (1954) The promeristem and the minimal constructional centre in grass root apices. New Phytol **52:** 48–57

Dolan L, Duckett C, Grierson C, Linstead P, Schneider K, Lawson E, Dean C, Poethig S, Roberts K (1994) Clonal relationships and cell patterning in the root epidermis of *Arabidopsis*. Development **120:** 2465–2474

Dolan L, Janmaat K, Willemsen V, Linstead P, Poethig S, Roberts K, Scheres B (1993) Cellular organisation of the *Arabidopsis thaliana* root. Development **119:** 71–84

Dolan L, Roberts K (1995) Plant development: pulled up by the roots. Curr Opin Genet Devel **5:** 432–438

Galway M, Masucci J, Lloyd A, Walbot V, Davis R, Schiefelbein J (1994) The *TTG* gene is required to specify epidermal cell fate and cell patterning in the *Arabidopsis* root. Dev Biol **166:** 740–754

Hanstein J (1870) Die Entwicklung des Keimes der Monocotylen und der Dikotylen. Bot Abhandl (Bonn) **1:** 1–112

Kerk N, Feldman L (1995) A biochemical model for the initiation and maintenance of the quiescent center: implications for organization of root meristems. Development **121:** 2825–2833

Kieber J, Rothenberg M, Roman G, Feldmann K, Ecker JR (1993) *CTR1*, negative regulator of the ethylene response pathway in *Arabidopsis*, encodes a member of the Raf family of protein kinases. Cell **72:** 427–441

Laskowski M, Williams M, Nusbaum C, Sussex IM (1995) Formation of lateral root meristems is a two-stage process. Development **121:** 3303–3310

Masucci J, Rerie W, Foreman D, Zhang M, Galway M, Marks D, Schiefelbein J (1996) The homeobox gene *GLABRA 2* is required for position-dependent cell differentiation in the root epidermis of *Arabidopsis thaliana*. Development **122:** 1253–1260

Rerie W, Feldmann K, Marks MD (1994) The *GLABRA2* gene encodes a homeodomain protein required for normal trichome development in *Arabidopsis*. Genes Dev **8:** 1388–1399

Sakai H, Medrano L, Meyerowitz EM (1995) Role of *SUPERMAN* in maintaining *Arabidopsis* floral whorl boundaries. Nature **378:** 199–203

Scheres B, Di Laurenzio L, Willemsen V, Hauser M-T, Janmaat K, Weisbeek P, Benfey PN (1995) Mutations affecting the radial organisation of the *Arabidopsis* root display specific defects throughout the embryonic axis. Development **121:** 53–62

Scheres B, McKhann H, van den Berg C, Willemsen V, Wolkenfelt H, de Vrieze G, Weisbeek P (1996) Experimental and genetic analysis of root development in *Arabidopsis thaliana*. *In* Plant Roots: From Cells to Systems. Kluwer, Dordrecht, The Netherlands (in press)

Scheres B, Wolkenfelt H, Willemsen V, Terlouw M, Lawson E, Dean C, Weisbeek P (1994) Embryonic origin of the *Arabidopsis* primary root and root meristem initials. Development **120:** 2475–2487

Torres-Ruiz RA, Jürgens G (1994) Mutations in the *FASS* gene uncouple pattern formation and morphogenesis in *Arabidopsis* development. Development **120:** 2967–2978

Traas J, Bellini C, Nacry P, Kronenberger J, Bouchez D, Caboche M (1995) Normal differentiation patterns in plants lacking microtubular preprophase bands. Nature **375:** 676–677

van den Berg C, Willemsen V, Hage W, Weisbeek P, Scheres B (1995) Cell fate in the *Arabidopsis* root meristem determined by directional signalling. Nature **378:** 62–65