Plant Developmental Biology

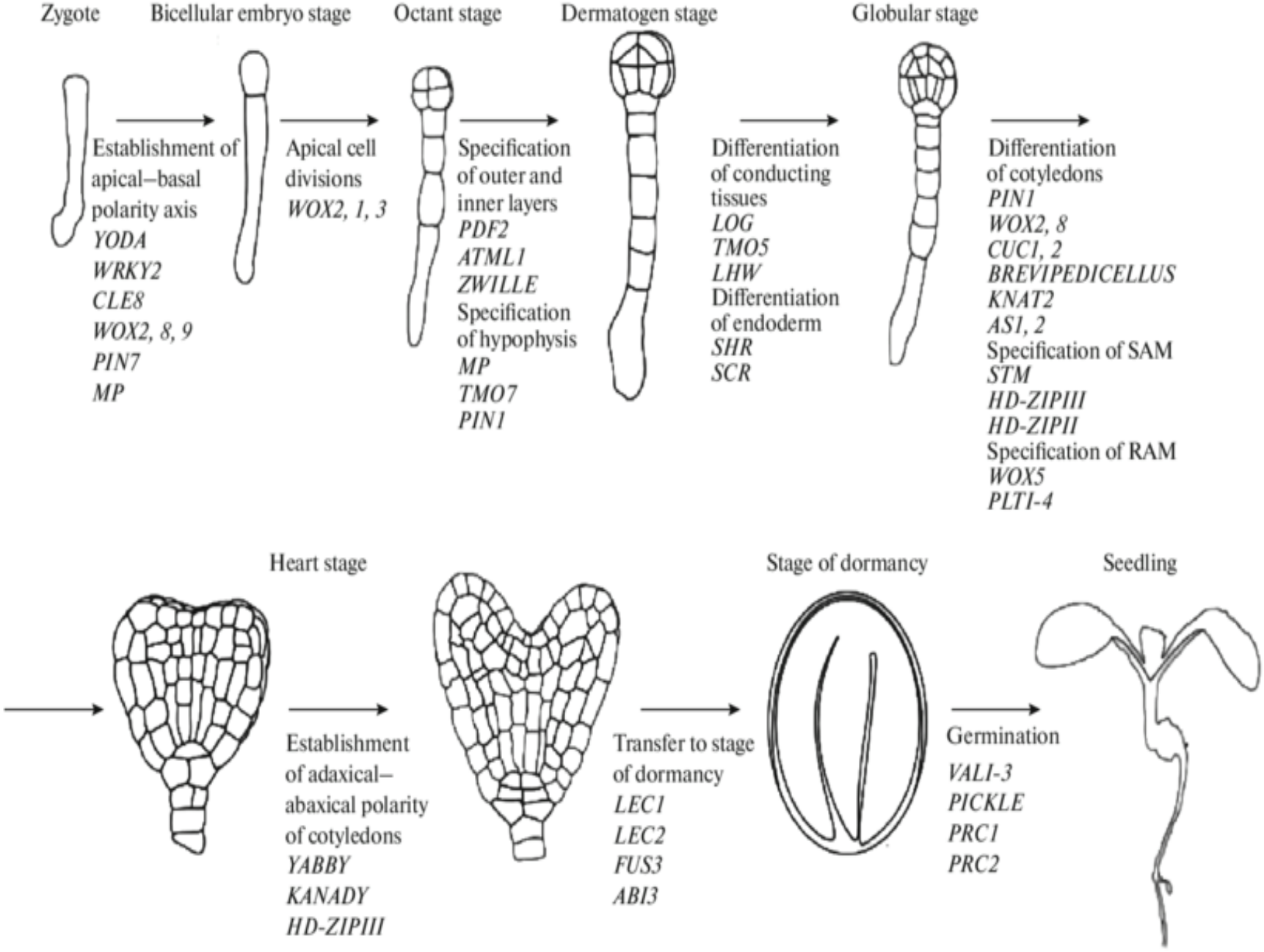

Zygote
Bicellular embryo stage
Octant stage
Dermatogen stage
Globular stage
Establishment of apical–basal polarity axis
YODA
WRKY2
CLE8
WOX2, 8, 9
PIN7
MP
Apical cell divisions
WOX2, 1, 3
Specification of outer and inner layers
PDF2
ATML1
ZWILLE
Specification of hypophysis
MP
TMO7
PIN1
Differentiation of conducting tissues
LOG
TMO5
LHW
Differentiation of endoderm
SHR
SCR
Differentiation of cotyledons
PIN1
WOX2, 8
CUC1, 2
BREVIPEDICELLUS
KNAT2
AS1, 2
Specification of SAM
STM
HD-ZIPIII
HD-ZIPII
Specification of RAM
WOX5
PLTI-4
Heart stage
Stage of dormancy
Seedling
Establishment of adaxical–abaxical polarity of cotyledons
YABBY
KANADY
HD-ZIPIII
Transfer to stage of dormancy
LEC1
LEC2
FUS3
ABI3
Germination
VALI-3
PICKLE
PRC1
PRC2

Table 1 | Genes involved in the hormonal control of seed development in Arabidopsis and maize.

Gene name	Acronym	Biological function of the encoded protein	Seed compartment expression	Species*
YUCCA 1 *YUCCA 4* *YUCCA 10* *YUCCA 11*	*YUC1* *YUC4* *YUC10* *YUC11*	Key enzymes of tryptophan-dependent auxin biosynthesis. Function in seed development and morphogenesis	Embryo, Endosperm, Seed coat	A
PINFORMED 1 *PINFORMED 3* *PINFORMED 4* *PINFORMED 7*	*PIN1* *PIN3* *PIN4* *PIN7*	Auxin efflux carriers involved in early embryo development. Establish the apical-basal auxin gradient	Embryo	A
Transport Inhibitor Response 1	*TIR1*	Part of the SCF E3-ubiquitin ligase complex. Functions as auxin receptor and is responsible for auxin signal transduction	Embryo, Endosperm	A
AUXIN SIGNALING F-BOX 1 *AUXIN SIGNALING F-BOX 2* *AUXIN SIGNALING F-BOX 3*	*AFB1* *AFB2* *AFB3*	F-box proteins that form a complex with TIR1. Involved in regulation of auxin response	Seed coat (*AFB2* and *AFB3*) and Embryo	A
MONOPTEROS/ AUXIN RESPONSE FACTOR 5	*MP/ARF5*	Transcriptional activator. Regulates embryo development	Embryo	A
BODENLOSS/ Auxin/INDOLACETICACID 12	*BDL/ Aux/1AA12*	Transcriptional repressor. Interacts with MP preventing it from activating its targets	Embryo	A

ETTIN/AUXIN RESPONSE FACTOR 3	*ETT/ARF3*	Controls the integument development	Seed coat	A
ABERRANT TESTA SHAPE	*ATS*	Forms a complex with ETT. Involvement in integument formation	Seed coat	A
MEGAINTEGUMENTA/AUXIN RESPONSE FACTOR 2	*MNT/ARF2*	Regulates seed size. Interacts with BIN2. Growth repressor	Seed coat, Embryo	A
ZmYUCCA 1	*ZmYUC1*	Involved in auxin biosynthesis in maize endosperm. Controls seed size	Endosperm	M
Sparse inflorescence1	*Spi1*	A YUCCA ortholog in maize. Role in maize inflorescence development	Embryo	M
Vanishing tassel 2	*Vt2*	A TAA ortholog in maize. Role in vegetative and reproductive development	Embryo	M
Orange pericarp 1 *Orange pericarp 2*	*orp1* *orp2*	Both *orp1* and *orp2* encode the *beta* subunit of tryptophan synthase. Required for seedling development	Embryo Endosperm	M
Miniature 1	*Mn1*	Cell wall invertase. Role in nutrient allocation and crosstalk with auxin	Endosperm	M
ZmPINFORMED 1 *ZmPINFORMED 2* *ZmPINFORMED 5* *ZmPINFORMED 8* *ZmPINFORMED 10*	*ZmPIN1* *ZmPIN2* *ZmPIN5* *ZmPIN8* *ZmPIN10*	Auxin efflux carriers involved in polar transport during embryogenesis and endosperm formation	Embryo Endosperm	M
SEMAPHORE1	*SEM1*	Regulator of *knox* gene expression. Required for proper kernel development	Embryo Endosperm	M
ABERRANT PHYLLOTAXY 1	*ABPH1*	Cytokinin-inducible type A response regulator. Negative regulator of SAM size and positive regulator of *PIN1* expression	Embryo	M

Gene name	Acronym	Biological function of the encoded protein	Seed compartment expression	Species*
Histidine phosphotransfer proteins	*AHPs*	Cytokinin signal transducers. Regulate seed size	Endosperm, seed coat	A
Histidine Kinase	*AHK*	Cytokinin receptor. Regulates seed size	Seed coat	A
Response Regulators	*ARRs*	Targets of the AHPs. Together with cytokinin response proteins regulate endosperm development	Endosperm	A
SHRINK/CYP72C1	*SHK1*	Decreases brassinosteroids levels. Regulates cell division and seed size	Embryo Endosperm, Seed coat	A
CITOKININ OXYDASE 1 *CITOKININ OXYDASE 2* *CITOKININ OXYDASE 3*	*CKX1 CKX2* *CKX3*	Regulate seed size and weight	Endosperm	A
DWARF 5	*DWF5*	Endoplasmic reticulum transmembrane protein involved in brassinosteroids signaling	Embryo, endosperm, seed coat	A
ZmHistidine kinase	*ZmHK1* *ZmHK1a2* *ZmHK2* *ZmHK3b* *ZmHK2a2*	Cytokinin receptor-like genes. Control seed size	Embryo	M

DE-ETIOLATED 2	*DET2*	Gene of brassinosteroids biosynthesis. Controls embryo development, seed size and embryo cell number	Embryo, Endosperm	A
BRASSINOSTEROIDS INSENSITIVE 1 *BRASSINOSTEROIDS INSENSITIVE 2*	*BRI1* *BIN2*	Protein kinase, regulate brassinosteroids and phosphorilatesARF2. Growth Repressor	Seed coat, Endosperm	A
BRASSINAZOLE-RESISTANT 1	*BZR1*	Positive brassinosteroid-signaling protein. Phosphorylated by BIN2	Endosperm	A
ZmStarch synthase I	*ZmSSI*	Starch synthase induced by ABA	Endosperm	M

Only the genes mentioned in the text are showed. "A, Arabidopsis; M, Maize.

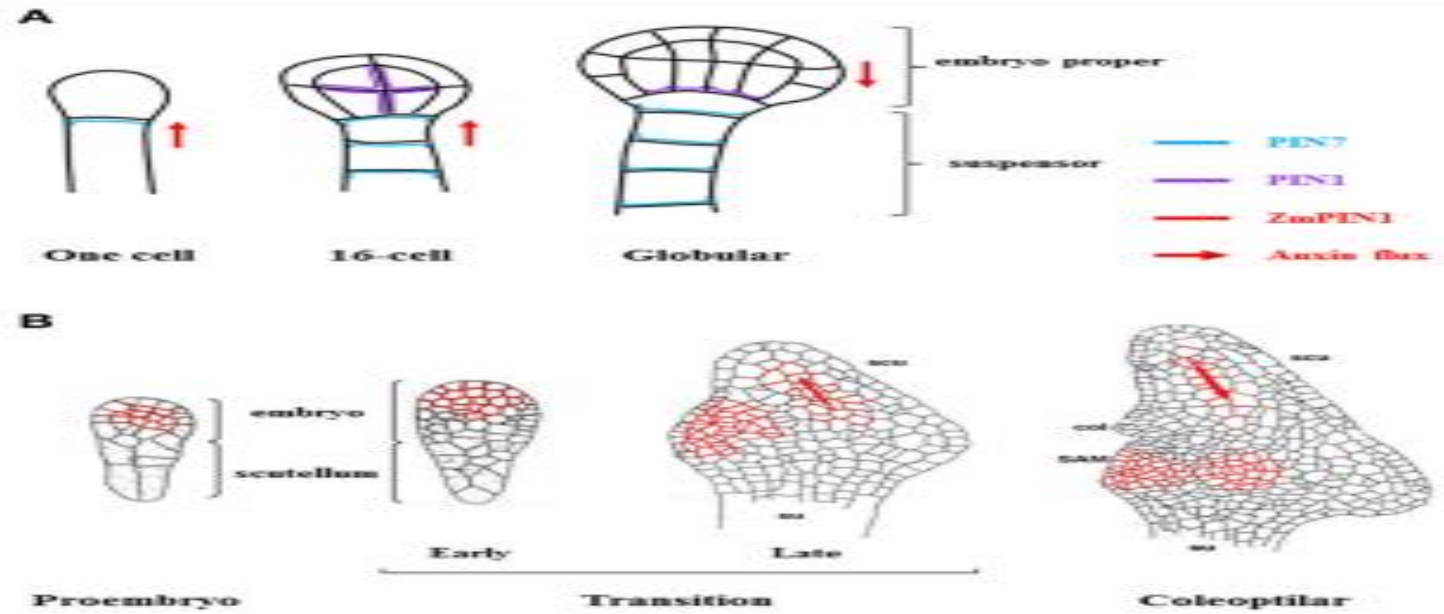


FIGURE 2 | Auxin transport during the embryogenesis development of Arabidopsis and maize. (A) Schematic representation of auxin transport during embryo development in Arabidopsis. In early embryo (one-cell stage to 16-cell stage in the figure), PIN7 (blue) is expressed in the suspensor cells localizing to the apical membranes, mediating auxin transport toward the proembryo. During the octant (not shown) and 16-cell stage, all proembryo cells express PIN1 (purple), which is evenly distributed along the inner cell membranes and not polarly localized. Later, during the transition to the globular stage, the subcellular localization of PIN1 becomes polar, facing the basal membranes. Simultaneously and similarly, the polarity of PIN7 localization is reversed, now localized at the basal membrane of suspensor cells. The localization of PIN1 and PIN7 in the basal membranes establishes an apical-basal flux of auxins that will be maintained throughout the life cycle of the plant (adapted from Friml et al., 2003; Weijers and Jurgens, 2005; Nawy et al., 2008). **(B)** Model for the *Zm*PIN1-mediated auxin transport during early stages of maize embryogenesis. Medial longitudinal sections of maize embryos at proembryo, transition and coleoptilar stages are shown. The *Zm*PIN1 protein (red) localizes in embryo plasma membranes. After the first division of the zygote, several cell divisions in different planes lead to the formation of the small embryo and the larger suspensor (proembryo stage). At this stage, auxins accumulate in the endosperm above the embryo but not in the embryo itself (not shown), and *Zm*PIN1 localizes at the cell boundaries of the undifferentiated proembryo core, without any polarity. Later, adaxial/abaxial polarity is established by the outgrowth of the scutellum at the abaxial side of the embryo (early transition stage). *Zm*PIN1 is polarly localized in the apical anticlinal membranes, marking the provascular cells of the differentiating scutellum, indicating an auxin flux toward the tip of the single maize cotyledon (late transition stage). At the coleoptilar stage there is the switch from apical to basal gradient of *Zm*PIN1, followed by a change of the auxin flux (adapted from Forestan et al., 2010; Chen et al., 2014). col, coleoptile; SAM, shoot apical meristem; scu, scutellum; su, suspensor. In both A and B the red arrows indicate the auxin efflux mediated by PINs.

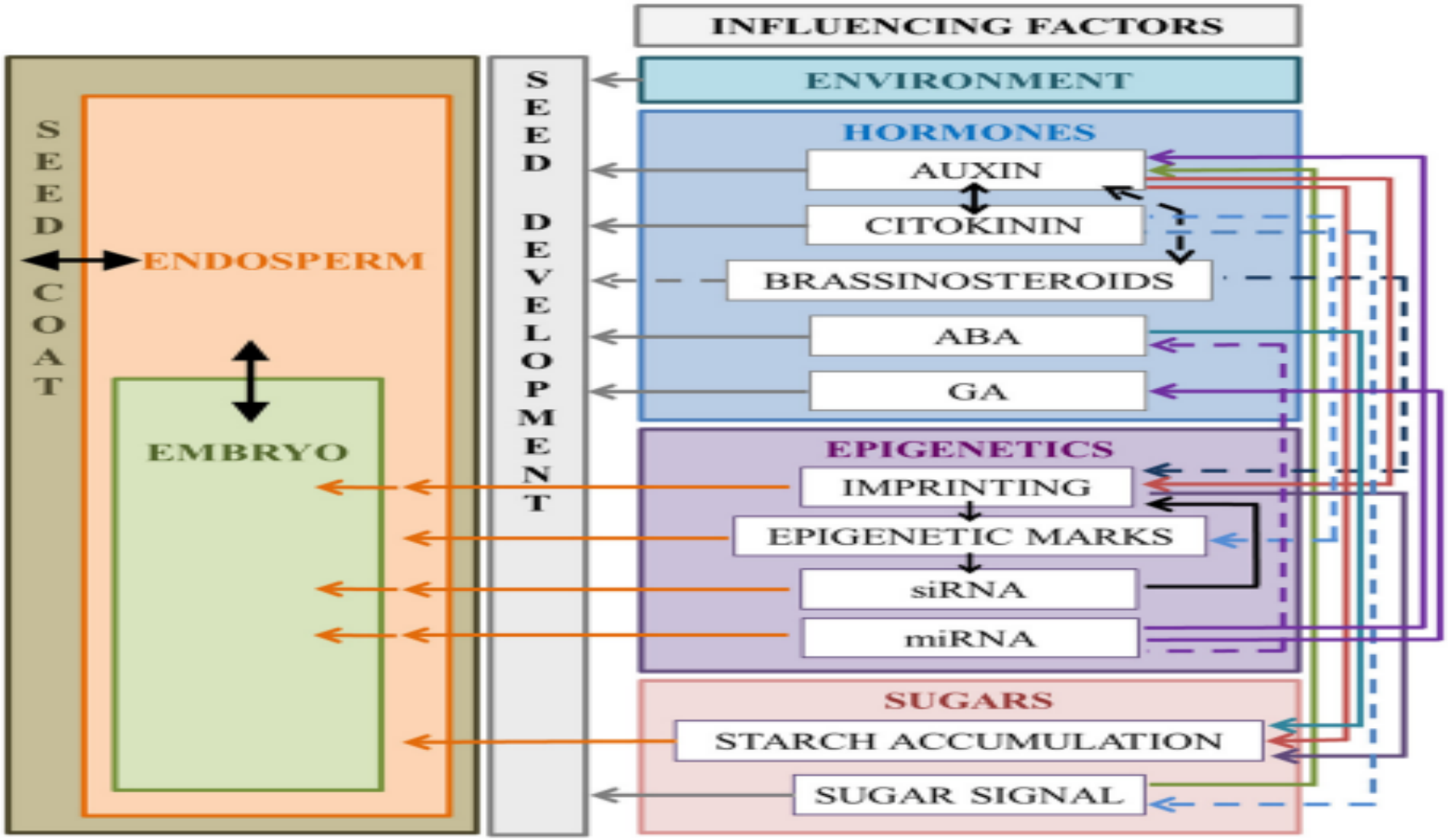
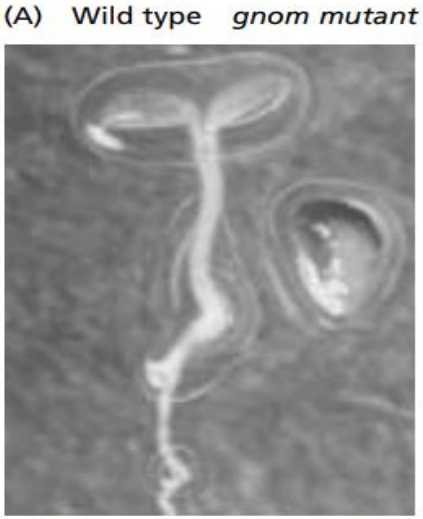


FIGURE 3 | Schematic representation of the factors affecting seed development in Arabidopsis and maize. Communication among seed compartments can involve one or more of the factors displayed on the right. The figure shows only the elements cited in this review. Single-headed arrows indicate regulation, double-headed arrows indicate reciprocal influence or regulation, and dashed arrows indicate an effect demonstrated only in one of the two species. Full-headed arrows indicate the communication among seed compartments.

Embryogenesis Requires Specific Gene Expression Analysis of Arabidopsis mutants that either fail to establish axial polarity or develop abnormally during embryogenesis has led to the identification of genes whose expression participates in tissue patterning during embryogenesis.

The GNOM gene: Axial patterning. Seedlings homozygous for mutations in the GNOM gene lack both roots and cotyledons. Defects in gnom embryos first appear during the initial division of the zygote, and they persist throughout embryogenesis. In the most extreme mutants, gnom embryos are spherical and lack axial polarity entirely. We can conclude that GNOM gene expression is required for the establishment of axial polarity

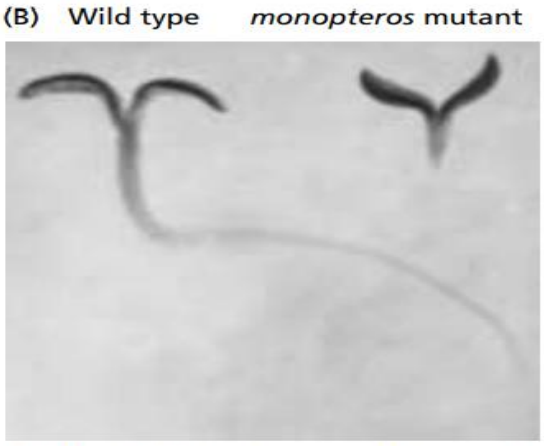


GNOM genes control apical–basal polarity

Genes whose functions are essential for Arabidopsis embryogenesis have been identified by the selection of mutants in which a stage of embryogenesis is blocked, such as gnom and monopteros. The development of mutant seedlings is contrasted here with that of the wild type at the same stage of development. (A) The GNOM gene helps establish apical–basal polarity. A plant homozygous for gnom is shown on the right.

The MONOPTEROS gene

- Primary root and vascular tissue.
- Mutations in the MONOPTEROS (MP) gene result in seedlings that lack both a hypocotyl and a root, although they do produce an apical region.
- The apical structures in the mp mutant embryos are not structurally normal, however, and the tissues of the cotyledons are disorganized.
- Embryos of mp mutants first show abnormalities at the octant stage, and they do not form a procambium in the lower part of the globular embryo, the part that should give rise to the hypocotyl and root.
- Later some vascular tissue does form in the cotyledons, but the strands are improperly connected.



MONOPTEROS genes control formation of the primary root

The MONOPTEROS gene is necessary for basal patterning and formation of the primary root. Plants homozygous for the monopteros mutation have a hypocotyl, a normal shoot apical meristem, and cotyledons, but they lack the primary root.
(A from Willemsen et al. 1998; B from Berleth and Jürgens 1993.)

- Although the mp mutant embryos lack a primary root when they germinate, they will form adventitious roots as the seedlings grow into adult plants.
- The vascular tissues in all organs of these mutant plants are poorly developed, with frequent discontinuities.
- Thus the MP gene is required for the formation of the embryonic primary root, but not for root formation in the adult plant.
- The MP gene is important for the formation of vascular tissue in postembryonic development.

The SHORT ROOT and SCARECROW genes

Ground tissue development: Genes have been identified that function in the establishment of the radial tissue pattern in the root and hypocotyl during embryogenesis.

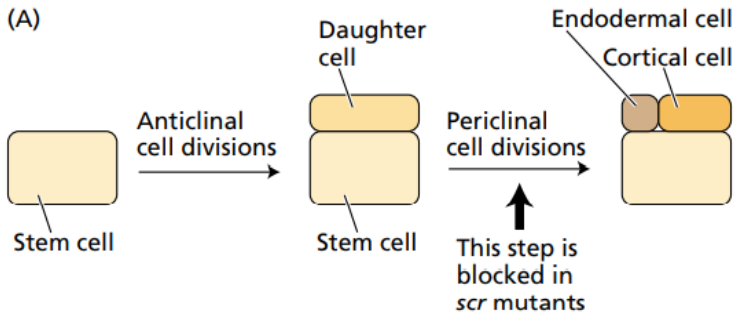
These genes also are required for maintenance of the radial pattern during postembryonic development.

To identify these genes, investigators isolated Arabidopsis mutants that caused roots to grow slowly.

Analysis of these mutants identified several that have defects in the radial tissue pattern.

Two of the affected genes, SHORT ROOT (SHR) and SCARECROW (SCR), are necessary for tissue differentiation and cell differentiation not only in the embryo, but also in both primary and secondary roots and in the hypocotyl.

Mutants of SHR and SCR both produce roots with a single-celled layer of ground tissue.

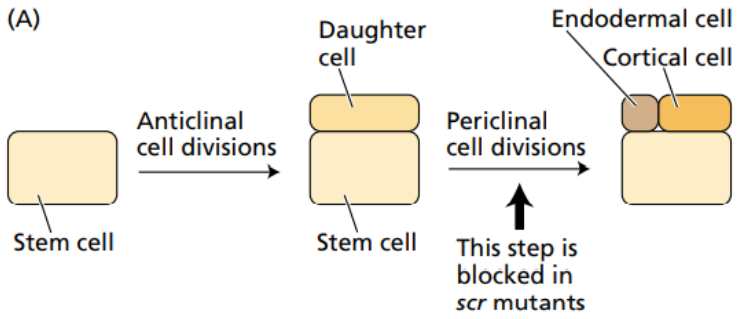


Mutations in the Arabidopsis gene SCARECROW (SCR) alter the pattern of tissues in the root. (A) The cell divisions forming the endodermis and cortex. The endodermal cells and cortical cells are derived from the same initial cells as a result of two asymmetric cell divisions. The cortical–endodermal stem cell (uncommitted cell) expands and then divides anticlinally, reproducing itself and a daughter cell. The daughter cell then divides periclinally to produce a small cell that develops endodermal characteristics and a larger cell that becomes a cortical cell. The second asymmetric division does not occur in scr mutants, and the daughter cell formed as a result of the anticlinal division of the initial has characteristics of both cortical and endodermal cells.

- Cells making up the single-celled layer of ground tissue have a mixed identity and show characteristics of both endodermal and cortical cells in plants with the scr mutation.

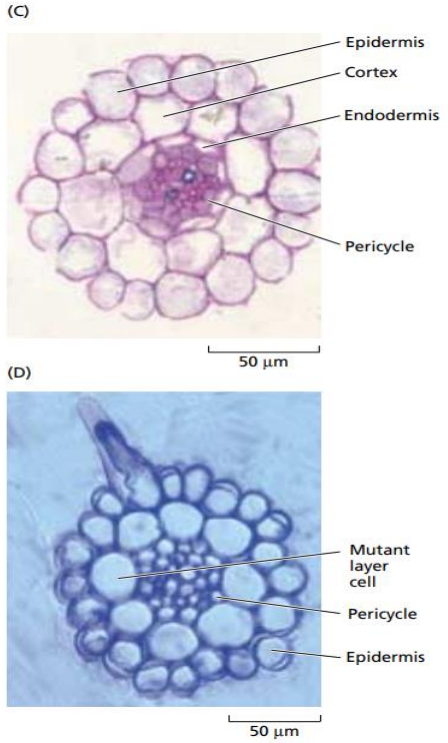
- These scr mutants also lack the cell layer called the starch sheath, a structure that is involved in the growth response to gravity.

- Roots of plants with the shr mutation also have a single layer of ground tissue, but it has only cortical cell characteristics and lacks endodermal characteristics.



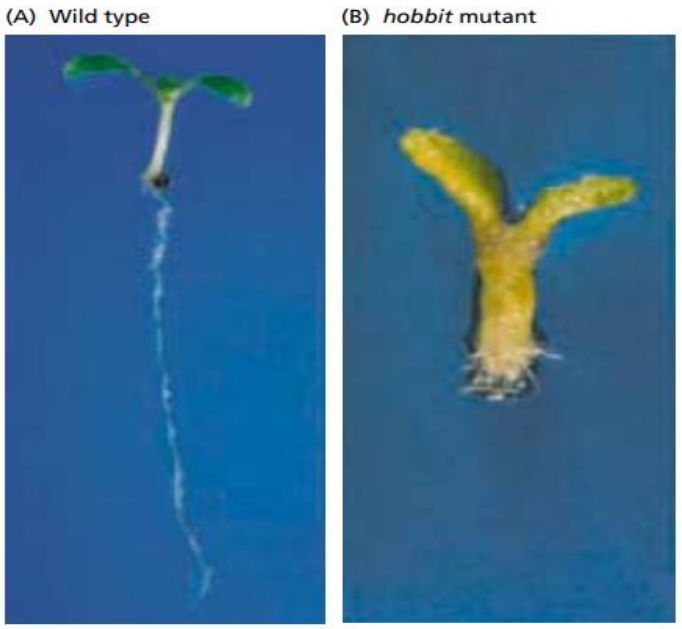
(B) The growth of a 12-day-old wild-type seedling (left) is compared with that of two 12-day-old seedlings homozygous for a mutation in the SCARECROW (SCR) gene (middle and right).

(C) Cross section of the primary root of a wild-type seedling. (D) Cross section of the primary root of a seedling homozygous for the scr mutant. (From Di Laurenzio et al. 1996; photos © Cell Press, courtesy of P. Benfey.)



The HOBBIT gene: The root meristem.

- **The primary root and shoot meristems are established during embryogenesis.**
- **Because in most cases they do not become active at this time, the term promeristem may be more appropriate to describe these structures.**
- **A promeristem may be defined as an embryonic structure that will become a meristem upon germination.**
- **A molecular marker for the root promeristem has not yet been identified, but it appears to be determined early in embryogenesis. Root cap stem cells (the cells that divide to produce the root cap) are formed from the hypophysis at the heart stage of embryogenesis, indicating that the root promeristem is established at least by this stage of embryogenesis.**
- **The expression of the HOBBIT gene may be an early marker of root meristem identity.**



The HOBBIT (HBT) gene is important for the development of a functional root apical meristem. (A) Wildtype Arabidopsis seedling; (B) hobbit mutant seedling.

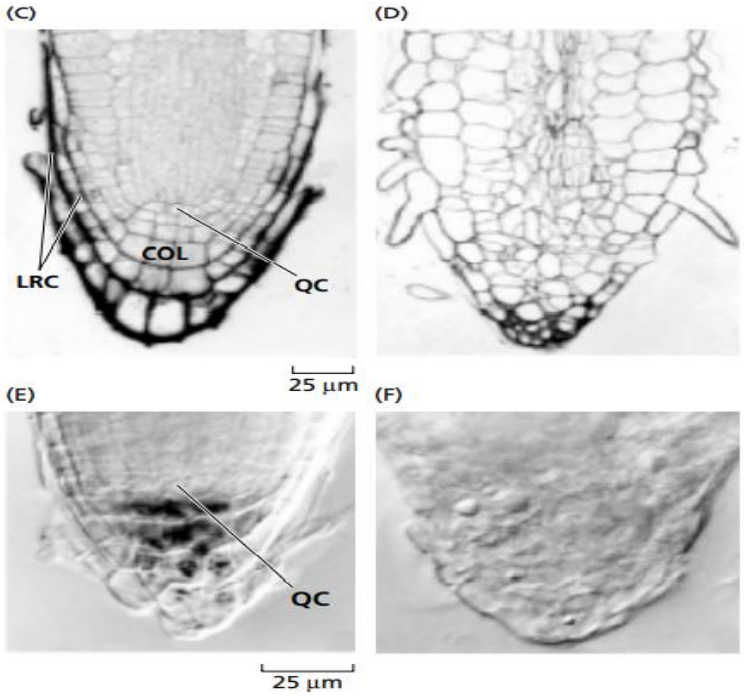
- Mutants of the HOBBIT (HBT) gene are defective in the formation of a functional embryonic root, as are plants with mp mutants.

- However, these two mutations act in very different ways.
- The hbt mutants begin to show abnormalities at the two- or four-cell stage, before the formation of the globular embryo.

- The primary defect in hbt mutants is in the hypophyseal precursor, which divides vertically instead of horizontally.

- As a result, the hypophysis does not form, and the root meristem that subsequently forms lacks a quiescent center and the columella.

- Embryos of hbt mutants appear to have a root meristem, but it does not function when the seedlings germinate.
- Furthermore, plants grown from hbt mutant embryos are unable to form lateral roots.



(C) root tip of wild type showing quiescent center (QC), columella (COL) and lateral root cap (LRC); (D) root tip of hobbit mutant; (E) quiescent center and columella of wild-type; (F) absence of quiescent center and columella in hobbit. The seedlings in A and B are both shown 7 days after germination (4× magnification). Staining with iodine reveals starch grains in the columella cells of the root cap in the wild type (E). No starch grains are present in the hbt mutant root tip (F). (From Willemsen et al. 1998.

The SHOOTMERISTEMLESS gene: The shoot promeristem.

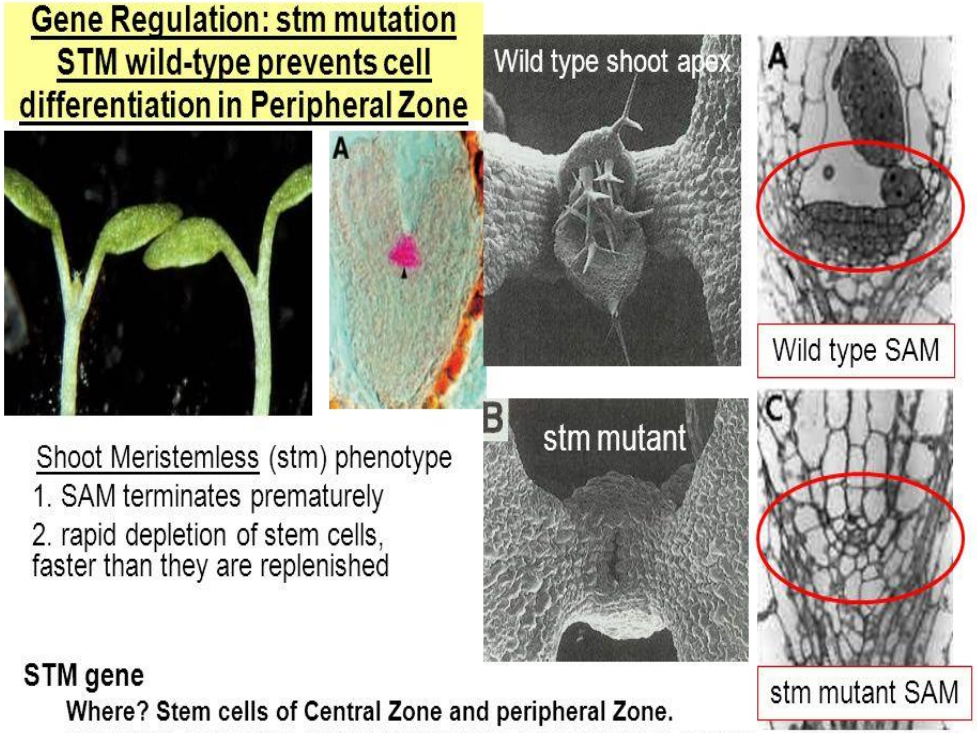
The shoot promeristem can be recognized morphologically by the torpedo stage of embryogenesis in Arabidopsis.

Oriented cell divisions of some of the cells between the cotyledons result in a layered appearance of this region that is characteristic of the shoot apical meristem.

However, the progenitors of these cells probably acquired the molecular identity of the shoot apical meristem cells much earlier, during the globular stage.

The SHOOTMERISTEMLESS (STM) gene is expressed specifically in the cells that will become the shoot apical meristem, and its expression in these cells is required for the formation of the shoot promeristem.

Arabidopsis plants homozygous for a mutated, loss-of-function STM gene do not form a shoot apical meristem, and instead all the cells in this region differentiate.



- The product of the wild-type STM gene appears to suppress cell differentiation, ensuring that the meristem cells remain undifferentiated.
- STM mRNA can first be detected in one or two cells at the apical end of the midglobular embryo.
- By the heart stage, STM expression is confined to a few cells between the cotyledons.
- Because STM acts as a marker for these cells, the shoot apical meristem must be specified long before it can be recognized morphologically.
- The STM gene is necessary not only for the formation of the embryonic shoot apical meristem, but also for the maintenance of shoot apical meristem identity in the adult plant.
- The role of the nucleus in controlling development was first demonstrated in the giant algal unicell, Acetabularia.