

Pattern formation in plant embryogenesis: A reassessment



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Pattern formation in embryogenesis generates the basic body plan of flowering plants by establishing the diversity of position-dependent cell fates characteristic of the seedling. The concept of pattern formation, which was originally based on the analysis of Arabidopsis pattern mutants, is re-examined in the light of recent data that address the origin of the axis of polarity, the origin of the primary meristems as elements of the apical-basal pattern and the formation of the radial pattern of tissue layers. The available evidence from genetic, molecular and experimental approaches supports the notion that embryonic pattern formation occurs in steps involving communication between clonally unrelated cells.

Key words: embryo / *Arabidopsis* / pattern formation / cell fate

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STARTING FROM THE reproductive single-cell stage, plant embryogenesis establishes the multicellular body organisation of a juvenile form, the seedling, that bears little resemblance to the adult. It is only during post-embryonic development that the meristems of the shoot and the root give rise to most structures of the adult plant. Nevertheless, the primary meristems are embedded in the body organisation of the mature plant embryo or seedling, constituting the terminal elements of an apical–basal pattern along the main axis of polarity. The two meristems are separated from one another by a linear array of juvenile structures consisting of cotyledons (embryonic leaves), hypocotyl (embryonic stem) and radicle (embryonic root). Another, radial pattern is apparent as a concentric arrangement of cell layers in the stem–root axis of the seedling. These layers represent, from the

periphery to the centre, the main tissue types epidermis, ground tissue (cortex and endodermis) and vascular tissue (pericycle, xylem and phloem).

The developmental processes that establish a multicellular organisation are usually subsumed under the term 'pattern formation' which essentially means that cell fates are singled out in a position-dependent manner.¹ When applied to plant embryogenesis, pattern formation is still a concept in search for a molecular mechanism. To identify principles of pattern formation, a genetic approach was taken in *Arabidopsis*, and a concept of embryonic pattern formation was derived from the analysis of mutant phenotypes.^{2,3} Numerous studies, utilising experimental, genetic or molecular approaches, have since accumulated a body of relevant data.⁴

The aim of this review is to re-examine the concept of embryonic pattern formation by addressing the following questions. How is the axis of polarity of the embryo established? How does apical–basal patterning give rise to the primary meristems? How is the radial pattern of concentric tissue layers generated? Although our main focus is on principles of cell fate segregation we will also discuss possible mechanisms where appropriate.

Apical–basal pattern formation: origin of embryo polarity

The apical–basal axis of the *Arabidopsis* embryo can be traced back to an earlier embryo-suspensor axis. The extra-embryonic suspensor is attached to the basal pole of the embryo. Both the embryo and the suspensor originate from the fertilised egg cell, the zygote.

In many flowering-plant species, the zygote divides perpendicular to the future apical–basal axis of the embryo. Its daughter cells give rise, in a complementary fashion, to the embryo and the extra-embryonic suspensor although the boundary between their progenies does not coincide with the embryo-suspensor

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1084-9521/98/020187+07 \$25.00/0/sr970210

boundary in many species, including *Arabidopsis*. The *Arabidopsis* zygote divides asymmetrically to give a small apical cell and a large basal cell. The apical cell produces most of the embryo while the basal cell generates the very basal end of the embryo and the entire suspensor.^{5,6} The apical, but not the basal, cell starts to express a homeo box-containing gene named *ARABIDOPSIS THALIANA MERISTEM LAYER 1* (*ATML1*) and this gene continues to be expressed in all derivatives of the apical cell until the octant stage.⁷ Thus, the apical cell appears to be routed towards an embryogenic fate. By contrast, the fate of the basal cell seems to be more flexible. In normal development, the basal cell generates a file of 6–9 cells of which all but one differentiate into extra-embryonic suspensor cells. Only the uppermost derivative takes on an embryonic fate secondarily (see later), becoming the hypophysis which gives rise to the quiescent centre of the root meristem and to the central root cap initials. Suspensor cells can switch to embryogenic fate in *Arabidopsis* mutants, such as *sus* or *twi*, and give rise to secondary embryos.^{8,9} Embryogenesis may thus be viewed as a default pathway of development which is repressed in the extra-embryonic suspensor cells by signal(s) from the embryo.

If the asymmetric division of the *Arabidopsis* zygote segregates embryogenic and extra-embryonic cell fates, is this event conditioned by some intrinsic property of the egg cell or by the polar organisation of the surrounding maternal tissue of the ovule? (see Grossniklaus and Schneitz, this issue). Alternatively, does the segregation of cell fates among its daughter cells reflect a polarising process within the zygote itself? Maize zygotes formed by *in vitro* fusion of isolated egg and sperm cells acquire an axis of polarity before undergoing asymmetric division,¹⁰ arguing against an imprinting of polarity onto the reproductive cell by the surrounding maternal tissue. Egg cells show a morphological axis of polarity in some species but not in others. Furthermore, their polarity can be reversed upon fertilisation, suggesting that before fertilisation there is no stable intrinsic axis from which the axis of the embryo can be derived. It has been observed recently that isolated somatic cells of *Arabidopsis* can initiate embryogenesis, forming a polar outgrowth that results in an asymmetric division.¹¹ An even more striking case is presented by the *twi1* secondary embryo that has its axis of polarity in either tandem or reverse orientation relative to the developing primary embryo within the same seed.⁹ The embryogenic suspensor cell thus appears to establish its own axis of polarity upon initiation of

embryogenesis and in this regard resembles a zygote or an isolated somatic cell that initiates embryogenesis *in vitro*.

Mutations in the *Arabidopsis* *GNOM* (*GN*) gene cause variable apical–basal polarity, which has been correlated with reduced elongation and abnormal partitioning of the zygote.¹² The apical cell is enlarged at the expense of the basal cell. While the apical cell divides abnormally, resulting in a twin-octant embryo, the basal cell gives rise to a shorter than normal suspensor. Thus, the division of the *gn* zygote still segregates embryogenic from extra-embryogenic fate but this is not sufficient for fixing the apical–basal axis of the embryo, as evidenced by the variable expression pattern of an apical marker gene.¹³ More recently, a ‘root pole’ marker gene has been found to be preferentially albeit variably expressed in the basal part of *gn* (*emb30*) embryos although the root meristem is absent.¹⁴ The *GN* protein shows sequence similarity to a yeast guanine-nucleotide exchange factor,¹⁵ raising the possibility that targeted vesicle trafficking may be involved in axis stabilisation, which would be reminiscent of the process of axis fixation in the *Fucus* zygote.¹⁶ Since *GN* is a zygotically required gene function, its presumed requirement for axis fixation is consistent with the view that the apical–basal axis of embryo polarity originates from a post-fertilisation event.¹⁷

On balance, the available evidence suggests that the apical–basal axis of polarity of the flowering-plant embryo may originate from an intrinsic polarisation of the zygote, with the surrounding tissue possibly influencing the orientation of the axis. As a result of this initial event, the embryogenic potential is restricted to the daughter cell(s) at the future apical pole. Since the future basal pole of the embryo is adjacent to the suspensor, embryogenic and extra-embryonic cells may interact to establish the apical–basal axis of polarity of the embryo.

Origin of the primary meristems as elements of the apical–basal pattern

The reproducible cell division patterns in embryogenesis of *Arabidopsis* and other crucifers has helped to trace the origins of seedling structures back to cell groups in the early embryo.^{5,6,18,19} The analysis of mutant phenotypes in *Arabidopsis* has led to the idea that the axis of the embryo is initially partitioned into three main regions named apical, central and basal.³ The apical region gives rise to the shoot meristem

and most of the cotyledons, the central region also contributes to the cotyledons ('shoulder region'²⁰), but mainly produces hypocotyl, root and root meristem initials. Finally, the basal region generates the quiescent centre of the root meristem and the central root cap. The apical and central regions are polyclones originating, respectively, in the upper and lower tiers of the octant-stage embryo which themselves are derived from the apical daughter cell of the zygote. The basal region corresponds to the clonal descendants of the hypophysis, the uppermost derivative of the basal daughter cell of the zygote. The three regions display different patterns of cell division in the globular embryo: apical cells divide without preferential orientation, central cells divide perpendicular to the axis, thus generating cell files, and basal cells undergo a stereotyped sequence of divisions.⁶ These cell division patterns appear to reflect different development commitments, as evidenced by mutant phenotypes.⁴

Clonal analysis ruled out that cell ancestry plays a major role in generating the elements of the apical-basal pattern, thus confirming the histological observations.²⁰ Thus, cell fate appears to depend on relative position along the apical-basal axis. Much the same can be said about embryos of other plant species many of which display less reproducible cell division patterns than the *Arabidopsis* embryo. In the following, we will discuss the role of cell-cell communication in the origin of apical-basal pattern elements, focusing on the primary meristems of the shoot and the root in *Arabidopsis*. The root meristem represents a pattern element that is derived from two clonally distinct regions of the embryo, central and basal. By contrast, the shoot meristem originates within a single region, the apical region of the embryo.

The *Arabidopsis* root meristem becomes active in the heart-stage embryo, producing tiers of root tissues and layers of central root cap from the initials above and below the quiescent centre, respectively.²¹ Mutations in several *Arabidopsis* genes affect the formation of the root. Seedlings of *monopteros* (*mp*) lack hypocotyl, root and root meristem but are able to form a root upon wounding, suggesting that *MP* is required for an embryo-specific process of root formation.²² The *mp* octant-stage embryo has four rather than two tiers of embryonic cells and a normal-sized filament of extra-embryogenic cells.²² Subsequently, the embryonic cells divide without preferential orientation, suggesting that the central region is not established. Furthermore, the presumptive hypophysis

divides like an extra-embryonic suspensor cell rather than generating the basal region. Thus, the hypophysis is a potentially extra-embryonic cell that normally adopts an embryonic fate in response to signals from the embryonic cells of the octant stage. This interaction appears to require *MP* gene function although its primary role may be in 'cell axialisation' which results in the formation of cell files.²³ The abnormal organisation of central region cells in *mp* embryos could reduce or eliminate their ability to interact with the basally adjacent cell. Mutations in *HOBBIT* (*HBT*) and other 'hypophyseal cell group' genes affect the development of the basal, but not the central region, resulting in the lack of a functional root meristem, and do not make a root.²⁴ In contrast to *mp* seedlings, *hbt* seedlings initiate but do not develop adventitious roots from their hypocotyl because they are unable to form new root meristems.²⁴ These data suggest that *mp* embryos fail to signal to the uppermost derivative of the basal daughter cell of the zygote while this cell is unable to respond if the *HBT* gene is defective. The mutant phenotype of the *Arabidopsis* *BODENLOS* (*BDL*) gene has features of both *hbt* and *mp* phenotypes: *bdl* embryos and seedlings resemble *hbt* mutants phenotypically but *bdl*, like *mp* seedlings, form adventitious roots, suggesting that *BDL* may play a role in central region-hypophysis signaling (T. Hamann, GJ and UM, unpublished results).

The absence of a functional hypophysis appears to result in a failure to establish the root meristem initials which are the lowermost derivatives of the central region, suggesting that the hypophysis or its derivative, the quiescent centre, normally signals back to the central region. This interpretation is based on the observation that laser ablation of a quiescent centre cell at the seedling stage results in the differentiation of adjacent initial cells.²⁵ In conclusion, establishment of the root meristem may involve two successive induction events across the clonal boundary between the central and the basal region: initially the central region induces hypophyseal cell fate and later on, the hypophysis or its derivative, the quiescent centre, induces the adjacent cells of the central region to take on the cell fate of root meristem initials. It should also be noted that the root meristem initials do not have intrinsic patterning activity but their differentiating progeny receive that information from the mature tissues of the embryonic root²⁶ (see Dolan and Scheres, this issue).

The small shoot meristem of the *Arabidopsis* embryo originates within the apical region and is flanked

by cotyledons whose primordia become recognisable at the transition to the heart stage.

It has been proposed on the basis of comparative morphology that the cotyledons are derivatives of the shoot meristem which initially occupies the entire apical region of the globular embryo.²⁷ However, the available data from *Arabidopsis* support an alternative view: cotyledon and shoot meristem primordia originate by partitioning of the apical region of the embryo.

With the exception of *gurke*,²⁸ *Arabidopsis* mutants do not affect the entire apical region. For example, *fass* mutations result in an enlarged radial dimension of the hypocotyl-root axis which is correlated with the formation of supernumerary cotyledons, without affecting the initial organisation of the shoot meristem.²⁹ Conversely, mutations in genes involved in the development of the shoot meristem during both embryogenesis and postembryonic development, such as *CLAVATA1* (*CLV1*) and *WUSCHEL* (*WUS*), do not affect the development of the cotyledons,^{30,31} and mutations in the *ZWILLE* (*ZLL*) gene affect the embryonic shoot meristem, again leaving the cotyledons unaffected.^{32,33} The *ZLL* gene appears to be specifically required for establishing a self-maintaining shoot meristem late in embryogenesis at which time the cotyledon primordia are rapidly growing (see Laux and Mayer, this issue). In conclusion, it seems very likely that the cotyledons are established independently of the shoot meristem. It should also be noted that the cotyledons provide a reference for phyllotaxis, i.e. the positioning of leaf primordia made by the shoot meristem (see Laux and Mayer, this issue).

The morphological partitioning of the apical region into cotyledon and shoot meristem primordia is presaged by restricted GUS reporter gene expression patterns in enhancer trap lines (G. Martin *et al*, unpublished results). In addition, two well-characterised genes display nearly complementary expression patterns at the late-globular stage. A few cells in the centre of the apical region start to express the *SHOOT MERISTEMLESS* (*STM*) gene, which is required for maintaining the undifferentiated state of shoot meristem cells and expressed in the central zone of the postembryonic shoot meristem.^{34,35} Thus, those cells at the top of the late-globular embryo appear to represent the incipient shoot meristem. Two flanking cell groups express the *AINTEGUMENTA* (*ANT*) gene, which later on is expressed in the primordia of cotyledons, leaves and flowers,³⁶ suggesting that those cell groups are destined to initiate the cotyledon primordia. If the early

expression patterns of *STM* and *ANT* reflect position-dependent cell fate segregation, how are these patterns established? Although the activating signals are not known it is unlikely that the activation can be so precise that *STM* is expressed in only a few cells. It seems to be more plausible that some mechanism of lateral inhibition underlies the correct positioning of *STM* activation within the apical region. For example, all cells within the apical region could respond to an activating signal from the vascular tissue of the central region. Those cells closest to the source would turn on *STM* expression and send an inhibitory signal to the cells on either side (see Dolan *et al* this issue, for other examples of lateral inhibition).

Radial pattern formation: successive establishment of concentric tissue layers

Radial patterning is initiated with the formation of a surface layer of epidermal precursor cells overlying non-epidermal cells. This event takes place at different developmental times in different flowering-plant species. In *Arabidopsis*, it is already at the octant stage that the eight embryonic cells divide tangentially (periclinally) to give outer and inner daughter cells.^{5,6} The outer daughter cells form the primordium of the epidermis and subsequently divide anticlinally, thus maintaining the integrity of the layer in the growing embryo. The outer cells differ from the inner cells in the expression of specific genes. The *ATML1* gene, which was expressed in the embryonic cells of the octant stage, continues to be expressed in the outer, but not the inner, daughter cells,⁷ supporting the idea that the epidermis is a developmental ground state derived from the zygote.³⁷ Soon after their formation, the outer cells start to express the *LIPID TRANSFER PROTEIN* (*LTP*) gene.^{13,38} These two genes remain continuously expressed in dividing epidermal cells during both embryogenesis and postembryonic development.^{7,38} If cytokinesis is incomplete due to mutation in the *KNOLLE* (*KN*) gene,³⁹ the *LTP* gene is expressed across the globular embryo,¹³ suggesting that the proper spatial regulation of epidermis-specific gene expression requires physical separation of the primordium. However, internal cells of *kn* embryos later discontinue the expression of the *LTP* gene and differentiate vascular structures.¹³ This change of cell fate is reminiscent of the origin of periclinal chimeras in which an abnormally oriented cell division in the L1 (epidermis) layer produces an inner daughter cell that takes on a subepidermal cell

fate according to its new position. It is tempting to speculate that epidermal versus subepidermal fate is determined by the activity of the *ATML1* gene. Lateral root formation is the only developmental context in which the epidermis is formed *de novo* from another tissue, the pericycle, and it would be interesting to determine whether this event is accompanied by the activation of the *ATML1* gene.

Subsequent events of radial patterning are confined to the shoot–root axis of the developing embryo which in *Arabidopsis* derives from the central region of the apical–basal axis. The subepidermal cells of the globular embryo divide again periclinally to give outer ground-tissue and inner vascular primordia. Slightly later, the ground-tissue cells begin to express the *SCARECROW* (*SCR*) gene which is required for the periclinal division generating cortex and endodermis derivatives.^{40,41} The endodermal, but not the cortical, cells continue to express the *SCR* gene. Several enhancer trap lines express the GUS reporter gene in the vascular primordium of the globular embryo (G Martin *et al*, unpublished results). These examples of tissue-specific gene expression support the idea that radial patterning generates tissue-specific cell fates in a position-dependent manner, progressing from the periphery to the centre. However, additional marker genes need to be identified not only for visualising pattern formation in more detail but also for analysing the underlying regulatory mechanisms. Good candidates are the tissue-specific marker genes whose expression patterns have been analysed in the seedling root and in the developing lateral root primordium⁴² because radial patterning in the lateral root primordium appears to proceed essentially the same way as radial patterning in the developing embryo axis (see below).

Surprisingly few mutants with specific radial-pattern defects have been described in *Arabidopsis*. Interestingly, these mutants were originally isolated for their seedling–root phenotypes but subsequently found to display the same radial pattern defect in the hypocotyl as well as in the root.⁴⁰ The *wooden leg* (*wol*) mutant phenotype supports the view of centripetal patterning: fewer vascular cells are made of which all differentiate into xylem at the expense of phloem.⁴⁰ The *scr* defect is also due to a shortage of cells. In this case, the ground-tissue cells fail to divide to give cortex and endodermis, and the single layer of ground tissue differentiates features of both.^{40,41} These two phenotypes are suppressed by a *fass* (*fs*) mutation which results in supernumerary cell layers,⁴⁰ suggesting that the segregation of tissue-specific cell

fates requires a minimum number of cell layers. By contrast, the *short root* (*shr*) embryo fails to establish the endodermis, and this defect cannot be compensated for by *fs*-mediated extra cell divisions, suggesting that the *SHR* gene confers endodermal identity.⁴⁰ Thus, the generation of endodermis and cortex from the ground tissue involves an unequal division segregating cell fates. It should be noted that all these mutants display the same radial pattern defects in their lateral roots,⁴⁰ which supports the view that mechanisms of pattern formation that operate in the embryo are also used in other developmental contexts.

Conclusions

The formal principles of pattern formation in plant embryogenesis have been substantiated in the past several years, mainly through genetic and experimental analysis in *Arabidopsis*. It is now evident that the basic body plan of flowering plants is established to a remarkable extent during embryogenesis and that this organisation conditions the way the meristems operate during postembryonic development. What is still lacking, however, is an understanding of the mechanisms that generate the basic body plan. Although cell–cell communication, in a broad sense, plays a very important role in patterning, it is not known whether this involves signaling across the cell surface via leucine-rich repeat receptor serine-threonine kinases⁴³ or exchange of molecules through plasmodesmata.⁴⁴ Nor are the signals known. Although some of the events discussed appear to involve short-range signaling between adjacent cells, other events may require long-range signaling.⁴ It should also be noted that a few cells of the developing embryo undergo unequal division segregating cell fates, which may be necessary for initiating new directions of development.^{17,45}

Acknowledgements

We thank our colleagues Markus Grebe and Thorsten Hamann for critical reading of the manuscript.

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