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PERSPECTIVE

A genetic access to plant cell axis formation

Thomas Berleth[1] · Christian S. Hardtke[2] · Jim Mattson[3] · Gerhard K. H. Przemeck[4,5]

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Abstract

The formation of a functional body pattern by plant cells became the subject of large-scale genetic approaches in the early 1990s. Various concepts, which either excluded or included plant hormones, were debated for several years before the focus shifted to partly self-regulatory mechanisms that could involve polar auxin flow in both cellular and organismal axis formation. A publication by Przemeck et al. contributed to this shift by explaining the embryo patterning function of the gene *MONOPTEROS (MP)* as part of a more general role in promoting cellular axis formation. Directional auxin flow was considered as a mechanism for aligning cell axialization along the apical-basal body axis and the *MP* gene was later found to encode a member of the Auxin Response Factor (*ARF*) family. The subsequent molecular elucidation of the signal transduction and transport mechanisms for auxin and other hormones made it possible to track hormone signalling much more precisely and, in many cases, to integrate hormone signals into models of plant pattern formation.

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✉ Thomas Berleth
thomas.berleth@utoronto.ca

1 Department of Cell and Systems Biology, University of Toronto, 25 Willcocks Street, Toronto, ON M5S 3B2, Canada

2 Department of Plant Molecular Biology, University of Lausanne, 1015 Lausanne, Switzerland

3 Biology Department, Simon Fraser University, 8888 University Drive, Burnaby, BC V5A 1G3, Canada

4 Institute of Experimental Genetics, Helmholtz Zentrum München, German Research Center for Environmental Health, Neuherberg, Germany

5 German Center for Diabetes Research (DZD), Neuherberg, Germany

Skepticism and enthusiasm

We all cherish, sometimes even teach, how science is characterized by an attitude of modest skepticism. How knowledge remains uncertain and provisional, subject to constant review with the aim of falsifying and replacing hypotheses with ever better ones (Popper 1963). Certainly, relatively speaking, debates in the natural sciences have arguably been the most productive dialogue in the history of human civilization, but this is not to say that science is free from self-deceiving enthusiasm, hypes and fads. Whether individual researchers cling too tightly to their favorite concepts, or an entire community is exuberantly excited by a new technology, emotions and prejudices can overshadow evidence at least for some time (Caulfield 2018). As a common feature, impressive scientific breakthroughs can trigger waves of inflated expectations, much like new technology developments, for which a bias toward short term overestimation has been described (Strawn 2021).

A certain overdose of enthusiasm may have gripped developmental geneticists in the 1990s, because the genetic dissection of *Drosophila* pattern formation in the preceding decade had felt like a moon mission for developmental genetics. In the 1980s, cloning a gene based on a well-studied *Drosophila* mutant could take years, and determining the sequence or expression pattern of a single gene could keep a graduate student busy for months. There were no "omics" technologies of any kind available, no comprehensive cDNA libraries, no fluorescent reporters and only few genomic phage or yeast artificial chromosome (*YAC*) libraries. Yet, it was within this technological wasteland that a combination of strategically planned forward-genetic screens, together with countless painstaking map-based gene cloning attempts and supported by some fortunate cell biological characteristics of the *Drosophila* embryo, enabled the rapid generation of a fairly comprehensive picture of a transcription-factor driven cascade of regulatory interactions. Identifying the actual proteins that subdivide two polar axes of an animal embryo in less than a decade had appeared unimaginable before it was actually attempted. That such a lofty goal could

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be achieved despite woefully inadequate technology does indeed bear some resemblance to the moon missions of the 1960s.

The pioneering days of Arabidopsis forward genetics

Under the encouraging influence of the *Drosophila* example, 'The awesome power of genetics' was a term often heard when *Arabidopsis* was branded as the plant genetic model system in the 1980 s (Meyerowitz 1989). Not only the strategies and technologies, also researchers moved from animal systems to the now-preferred model of plant developmental genetics. The enthusiasm was further fueled, when new sophisticated mutant screens identified long-sought, but until then purely hypothetical, developmental regulators. A screen for ethylene-resistant mutants, for example, identified a family of membrane-bound ethylene receptors along with numerous downstream components of an ethylene signaling pathway (Ecker 1995; Guzman and Ecker 1990). Large-scale screens for mutants with altered patterns of floral whorls led to the discovery of a combinatorial code of MADS box transcription factors that specifies whorl identities based on a vegetative leaf ground state (Bowman et al. 1989; Yanofsky et al. 1990). Combinatorial algorithms of homeotic gene functions had already been described and molecularly investigated in *Drosophila* and other animals (Akam 1989). The fact that similar combinatorial codes, but based on a different transcription factor family, were used in plants, illustrated the independent acquisition of regulatory strategies in the two kingdoms (Weigel and Meyerowitz 1994). And each of these surprisingly rapid successes spurred new mutant screening initiatives and improved funding opportunities for *Arabidopsis* forward genetics (Koornneef and Meinke 2010; Rabinowicz and Byrne 2001).

Against this background, a genetic screen for dissecting the embryonic patterning process, was approached by the Juergens group in Munich (Germany) in the early 1990s. Objectives, strategy and underlying reasoning of this screen were explained in a separate publication in 1991 (Jurgens et al. 1991). Firstly and in analogy with searches for embryo pattern mutants in *Drosophila*, M2 screens in small families were used to recover recessive lethal mutations via their heterozygous siblings. Secondly, as it was suspected that numerous mundane cell functions would lead to embryo lethality (Meinke and Sussex 1979), screening at the seedling stage was used to selectively identify pattern regulators with no role in basic cell biology. However, once a putative pattern mutant was further investigated, it was crucial that its pattern abnormalities were recognizable as early as the heart-stage, when wild type embryos already display the arrangement of all major pattern elements. Thirdly, a body pattern alteration should meet sharply defined criteria like the mis-positioning, duplication, polarity distortion or deletion of predefined pattern elements (Jurgens et al. 1991). Finally, average allele frequencies were determined as a standard for the probability for having identified at least one allele in any gene detectable by the screen criteria in order to document the saturation of the screen (Jurgens et al. 1991; Jurgens et al. 1994; Mayer et al. 1991).

The results of this saturating screen could be measured against the predefined criteria. Recessive mutations in a number of genes caused defined pattern alterations at the seedling stage (Fig. 1a) (Mayer et al. 1991), among them twelve allelic mutations in a gene called '*MONOPTEROS*' *(MP)*. Fulfilling the criteria of an apical-basal pattern deletion mutant, *mp* loss-of-function seedlings produced cotyledons and even a shoot meristem with normal external morphology, while the hypocotyl and radicle were invariably absent (Fig. 1b) (Berleth and Jurgens 1993; Mayer et al. 1991). Moreover, deviations from wildtype development were already detectable early in embryogenesis (Fig. 1c). Oddly, however, *mp* mutants exhibited variable numbers of cotyledons, often only one (the name stands for 'single wing' in Greek), and the cotyledon vasculature was incomplete. But these were variable, not fully penetrant traits that did not question the categorization as an apical-basal pattern deletion.

Plants are not green flies

Even in retrospect, strategy and conclusions of the 1991 embryonic pattern mutant screen were probably the most straightforward and promising approach to the task at this time. Had technology in those days allowed for the rapid identification of the newly discovered genes' products, there would have been a simple straight line from the screening rationale to the gene products and their cellular functions. Alas, the slow pace of gene cloning in the mid-1990s provided ample time and opportunity for introducing and debating grand schemes. Soon pattern-deletion mutants were compared to *Drosophila* gap-gene functions, which was bold as the latter represented transcriptional regulators within a hierarchy subdividing the anterior-posterior axis (Fig. 1d-e). While nobody intended to treat plants like green flies, there was fascination with the idea that genetics could reveal unorthodox analogies between the kingdoms. Or at least there was the expectation that sharply defined mutant phenotypes might provide direct clues to gene products that convey positional information, as it had been the case in *Drosophila*. Besides pertinent mutations, body pattern distortions could to some degree be induced by growth regulator-related treatments (Okada et al. 1991; Schiavone and Cooke 1987), but the use of plant hormones by plant

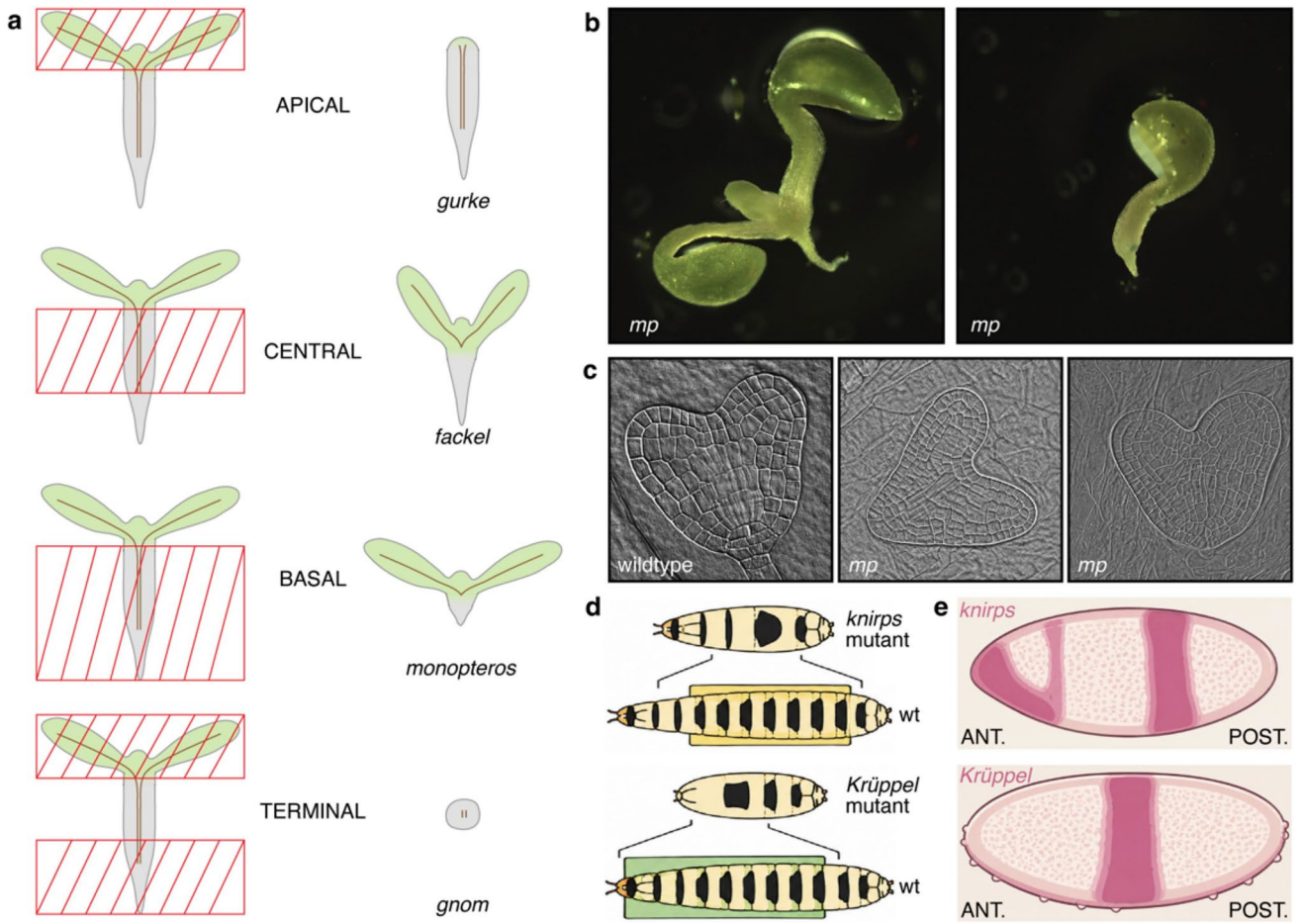
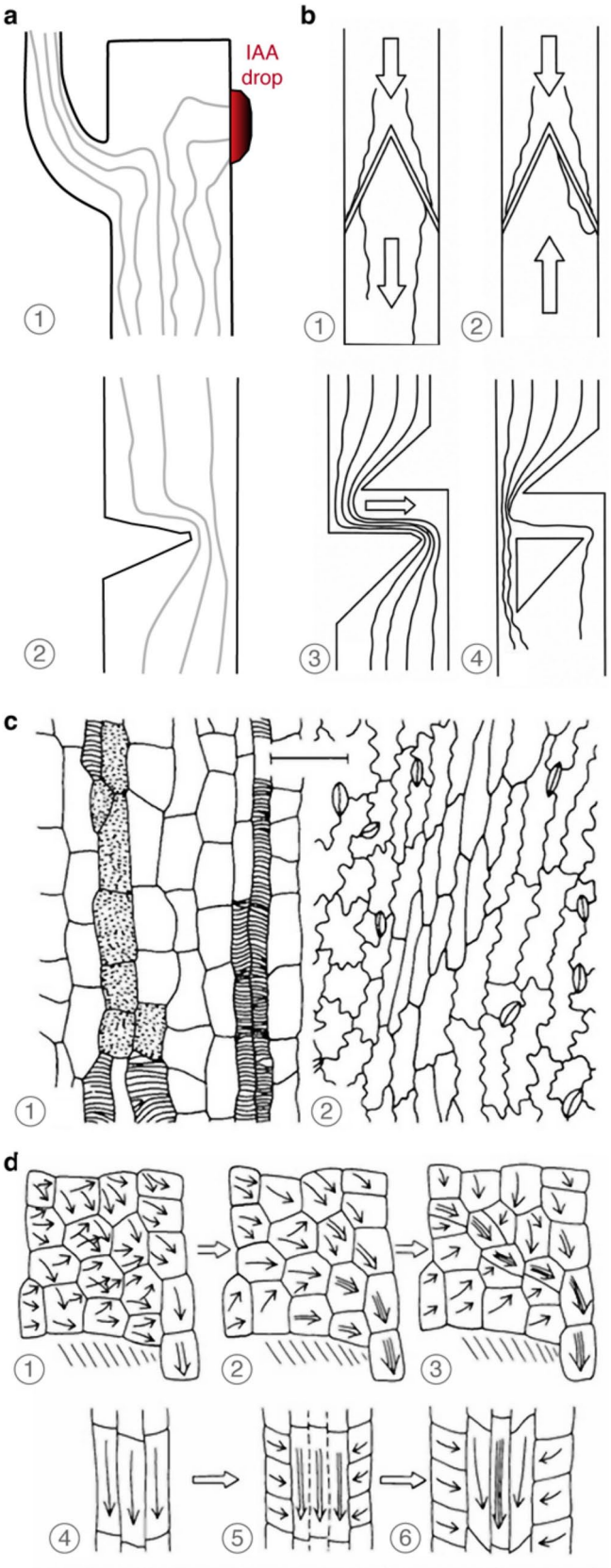


Fig. 1 Deletions of pattern elements along the apical-basal axis as published in 1991.**a** Schematic representations of pattern deletion phenotype classes, each represented by a complementation group identified in a forward-genetic screen. Redrawn and modified from Mayer et al. 1991. **b** Homozygous *mp* mutants invariantly lack hypocotyl and primary root, but can form one or two (sometimes fused) cotyledons and a shoot meristem. **c** Wildtype and *monopteros* (*mp*) mutant embryos of different allelic strength at early heart stage, documenting the absence of region-specific cell behavior in the basal part of the embryo. **d-e** *Drosophila* "gap-type" deletion mutants (here, *knirps* and *Krüppel*) lack pattern elements along the anteroposterior body axis **d**. The genes defined by these mutants are expressed in the corresponding regions at very early embryonic stages **e**. The transcription factors encoded by these genes transmit positional information as part of a hierarchical interactive regulatory cascade that subdivides the embryo into the correct number of segments along the anteroposterior axis.

developmental researchers was hard to accept for many geneticists. For them, low-molecular weight components whose origin, concentration and decay were difficult to trace were probably the least attractive candidates for conveying instructive signals in patterning processes. This may have fueled the favored view that interactions within a defined set of protein regulators, each identifiable by a mutant, could be exclusively responsible for all decisions defining a plant's body pattern. To do justice to audacious speculations in the early 1990s, it should be noted that this was also the time of startling surprises derived from genetic discoveries, which had repeatedly defied traditional expectations. Homeobox-containing transcription factors, derived from *Drosophila*, were implicated in both protostome and deuterostome development (Krumlauf 1992), and the genetic cues that determine eye positions in animal body plans had turned out far more universal than expected (Gehring 1996). Not least, the above-mentioned algorithmic similarities between homeotic functions in animals and plants, even documented convergent evolution of regulatory mechanisms across kingdoms (Meyerowitz 1997, 1999). In short, there is nothing wrong with speculating outside the box, as long as one fully acknowledges the body of knowledge inside the box.

An axial concept

At least in the early 1990s, genetic and experimental approaches to plant development were still largely separate universes, despite the extensive knowledge already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◂**Fig. 2** Coordinated cell-axialization events and their interpretation as published in 1991. **a** 1. After removal of the original shoot meristem from a stem segment, a new side shoot (left) triggers the formation of continuous vascular strands (grey) oriented towards the root pole. Similar strands are formed, when a droplet of a transportable auxin, like indole-acetic acid (*IAA*), is applied (right). 2. The self-organizing properties of the system are visualized by new, continuous vascular strands, which form in response to an obstacle (like a wedge-shaped incision) in the original shoot-root connection. **b** Newly oriented axial cell differentiation under the influence of overall shoot-root polarity. 1. Grafted tissues with identical apical-basal polarity are connected by new vascular strands along a nearly straight path from shoot to root pole. 2. By contrast, this path is turned around when the root pole of the lower graft is pointed towards the shoot pole of the upper graft. 3. New vascular strands will connect shoot and root pole even at right angle to the original path, if this is the only direction left by incisions, but will predominantly form along the most direct path, if this is possible (4.). **c** Examples of coordinated cell axialization in and beyond vascular tissues. 1. Vessel elements (helical reinforcements or borded pits) form with each cell's axis being sufficiently aligned with those of other cells in the strand to ultimately form a continuous tube. 2. Cells adjoining minor veins in a pea leaf epidermis elongate along the axis of the corresponding vein. **d** Axializing (1.−3.) and canalizing (4.−6.) consequences of a self-reinforcing signal flow. 1.−3. Cells exposed to a new shoot-root polarity, like the ones creating a nearly horizontal channel in (c) 3., gradually align along a news axis of signal flow, whereby cells with higher signal conductance become even better conductors (multiple arrows). 4.−6. The increasing conductance differences between cell files can lead to distinct tissue identities, thereby generating a radial pattern perpendicular to the axis of signal flow. Illustrations redrawn and modified from Sachs 1991.

available from experimental intervention studies. Models from classic experiments by pioneers like Ian Sussex, Taylor Steeves, Mary and Robert Snow, Katherine Esau and Tsvi Sachs (and many more) often had guiding roles and became well embedded in the emerging molecular pictures during the following decades (Hajny et al. 2022; Kuhlemeier and Timmermans 2016; Scarpella 2024). A synthesis of the two approaches was to be expected, since - apart from the use of experimental interventions instead of genetic inputs - the intellectual framework was often very similar: deriving mechanistic models from organismal responses to targeted interventions. The work of Tsvi Sachs and colleagues is particularly interesting, because on several topics they had used highly sophisticated combinations of experimental manipulations to interrogate the developmental algorithms, which even instilled mathematical modelling already in the 1980s (Goldsmith et al. 1981; Mitchison 1981). This can be illustrated in the work on the generation of new vascular connections and on cell polarization and axialization responses in general (Sachs 1981, 1991, 2000). Sachs and coworkers did indeed use hormone (auxin) applications, but in an extremely controlled and thoughtful manner. As a central conclusion, he postulated a signal substance transported in an apical-basal direction, with a cell biological feedback increasing cell conductance in response to the signal flow (Fig. 2a-b). This can be seen as a mechanism generating and stabilizing a polar axis as a basic element in plant patterning at the cellular as well as the organismal level ("axialization"; Fig. 2c) (Gersani and Sachs 1984; Sachs 2000), but is better known as the 'canalization hypothesis', because of another consequence of the model: if signal conductance amplifies itself, this would also increase the conductance difference among parallel cell files and could ultimately select those with highest conductance as vascular cells (Fig. 2d). Hence the model encompasses the acquisition of apical-basal polarity, the axial alignment of cells between these poles and ultimately radial (inside-out) patterning by selecting vascular cell files. How strictly formal Tsvi Sachs' conclusions were, can be seen in the fact that he often wrote about a polarly transported signal substance rather than auxin, although this substance could not be experimentally separated from auxin.

For any geneticist studying Tsvi Sachs' work, an obvious question was: 'what would a loss-of-function mutant in this axis-acquisition feedback loop look like?', especially if the affected gene was crucial from the zygote-stage onward? The highly reproducible abnormal embryo development of *mp* mutants was striking, not only by what went wrong, but also by what remained unaffected (Fig. 3a). There were no discernible defects in general cell functions: neither abnormal cell numbers, nor abnormal mitotic features, nor abnormal cell walls, shapes etc., were observed. The suspensor and the epidermis also formed normally, as did the ground tissue layers in the cotyledons. Just the divisions of inner embryonic cells remained randomly oriented, where those normally form perfectly parallel files of elongated cells, all aligned along the apical-basal axis (Fig. 3b). If *mp* was interpreted as an axialization mutant, this was a non-penetrant trait, as seedlings were neither apolar nor completely non-axial. Cotyledons formed invariantly from apical cells, and within those cotyledons even a vascular strand in the position of the midvein was formed (Fig. 3c).

The post-embryonic phenotype of mp mutants

The publication in focus here ("Studies on the role of the gene *MONOPTEROS* in vascular development and plant cell axialization") primarily dealt with the post-embryo development of *mp* mutants (Przemeck et al. 1996) and marked a paradigm shift away from an apical-basal pattern deletion model and towards an axis formation function for the *MP* gene. This shift was not easy to communicate in presentations as well as in debates with rejecting journal editors. The idea of a completely penetrant apical-basal deletion mutant was clearly more appealing, embedded in a league of other mutants and conceptual publications. The alternative, axial interpretation, was less attractive to most audiences in at least five respects: 1st Cell 'axialization' was merely reduced

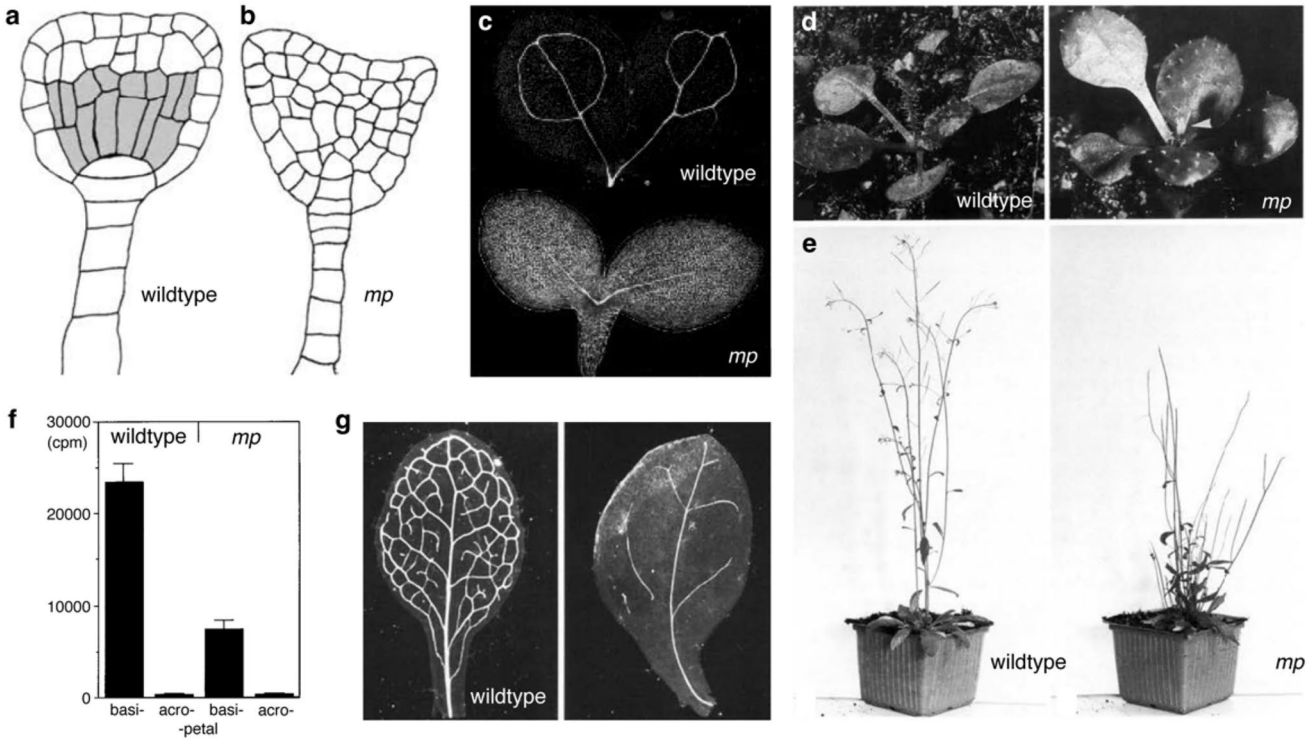

Fig. 3 Signature features of impaired axialization reported in Przemeck et al. 1996. **a** In wildtype *Arabidopsis* embryos the progenitor cells of major seedling organs and tissues can already be identified in an early heart-stage embryo. **b** In *mp* mutant embryos the parallel, sub-epidermal cell files in the bottom part of the embryo (grey) are selectively absent (see text for details). This abnormality could be interpreted either as a lack of positional information for being located in the basal half of the embryo (pattern deletion), or, alternatively, as a general inability of mutant cells to align along a common axis (axialization defect). **c** At the seedling stage, the *mp* mutant not only completely lacks the vascular tissue in the area of the hypocotyl and radicle, but the vascular tissue is also reduced to the midvein in the cotyledons. **d-e** No gross morphological abnormalities are discernible in *mp* rosettes (**d**), but inflorescences (**e**) are largely flowerless and instead comprised of pin-shaped inflorescence stalks. **f** Basipetal versus acropetal transport of labelled IAA in wildtype and *mp* mutant inflorescence stems. Similar reduced auxin transport in flowerless inflorescence stems had been observed in wild type plants exposed to auxin transport inhibitors and in *pin1* mutant stems. **g** Wildtype rosette leaves exhibit a hierarchically ordered venation system. The midvein forms the first order, from which the veins of the second order branch off, and higher-order veins (third and fourth order) form irregularly in between. Mutant rosette leaves always develop midveins, but a reduced number of second-order veins. The development of higher-order veins may be greatly delayed or completely absent. All panels are from the original Przemeck et al. 1996 publication, except (**c**), which was reproduced from Berleth & Juergens 1993.

in mutants, not completely absent like the basal part of the embryo; 2nd journals anticipated controversy, as the conclusions impinged on the interpretation of other mutants; 3rd the term 'axialization' was not clear to most readers. While it seemed understandable to those familiar with studies on the role of plant hormones in development, this branch of research was traditionally far removed from genetics; 4th discussing models implicating auxin, no matter how cautiously and indirectly, without triggering reviewer requests for auxin-specific experiments, felt a lot like walking a tightrope; 5th not only geneticists, also plant physiologists were very hesitant to attribute patterning functions to a plant hormone like auxin. In this community, the counterarguments focused on the perplexing diversity of auxin functions and on the abundance of auxin in certain plant tissues. Those criticisms explain the overly cautious wording to avoid further rejections, as there was indeed no direct evidence for a role of *MP* in auxin transport or auxin perception.

The post-embryonic phenotype of *mp* mutants not only had signature features fitting in an axialization-canalization concept, it also violated many expectations derived from pattern-deletion interpretation. Mutants were not incapable of forming a basal pole *sensu strictu*, instead could grow large, branched root systems once an adventitious root meristem had been formed. Mutants could also grow morphologically normal shoots (Fig. 3d), except that at reproductive stages, they formed pin-shaped inflorescence axes, instead of fertile flowers (Fig. 3e). This feature was conspicuously reminiscent of Arabidopsis plants treated with chemical inhibitors of auxin transport or of *pin-formed 1* (*pin1*) mutants, which had been reported to display an impaired auxin transport capacity (Okada et al. 1991). Measurements confirmed that

also in *mp* mutants, apical-basal auxin transport was reduced by 66% relative to wild type (Fig. 3f). Furthermore, unlike in plants with inhibited auxin transport, *mp* mutants showed a reduced capacity to form vascular tissue, and this in a peculiar pattern. What was selectively missing in venations of all kinds of leaves were the peripheral and higher-order veins, while the mid vein or other early veins were formed (Fig. 3g). If 'canalization' is viewed as the drainage of a ubiquitously produced signal substance, the midvein would be least affected by a reduced cellular responsiveness to the signal, whereas minor, later formed veins would be delayed or even absent (Fig. 3g). Finally, even the visible vascular bundles appeared thinner and less aligned, possibly as a result of their delayed formation.

Conceptual consequences of an axializing MP function

The axialization interpretation in the study was admittedly conceptual, speculative and without experimental link to auxin. The only measurement directly related to auxin was the reduced transport of labelled IAA in *mp* mutant stem segments, but this could just as easily been interpreted as merely the consequence of less auxin-conductive immature vascular tissue. Consequently, the extremely cautious discussion section focussed on diagnostic features of morphological, anatomical and physiological nature, which were individually interpreted in the light of a signal-flow feedback model. It turned out that the defects in *mp* were in even better agreement with the defects expected under conditions of reduced signal-flow feedback than the phenotypes of plants with pharmacologically obstructed auxin-transport or of the widely accepted auxin transport mutant *pin1* (Liu et al. 1993; Okada et al. 1991). Nevertheless, as conclusions were nearly exclusively based on anatomical evidence, the boldest statements in the discussion were that in the light of "widely accepted feed-back models" it is suggested that "MP function contributes to axialization in plant development, possibly by mediating the canalization of auxin flux".

As cautious as this speculation was, it was consequential. If the basal deletion in the *mp* seedling pattern was merely a consequence of a generally weak axialization response, which could also be detected in all later stages, then the core element of zygotic embryo patterning, axis formation, would be just another expression of an innate axialization capacity of plant cells. (Dettmer and Friml 2011). Zygotic embryogenesis would likely employ additional controls to accelerate the process and to enhance its reproducibility in a small contained system, but the core pattern coordinates would be set by auto-regulatory regeneration properties typical of plant cells. The further development of this view was accompanied by a very friendly exchange between us (and other genetically inspired groups) and Tsvi Sachs. On our side this contact began with a rather timid letter in an air-mail envelope sent to the esteemed senior scientist Professor Sachs in 1996, which, to our pleasant surprise, was promptly answered with questions and suggestions. Tsvi treated us, the molecular novices in plant biology, with patience, respect and curiosity, and taught us by his example how to firmly adhere to reproducible evidence, even if this kept models at a formalistic level. The very pleasant and productive exchange fortunately continued into the exciting molecular era after 1998 and lasted until Tsvi's far too early passing in 2007.

From 1998, model testing using molecular tools

Because of the acceptance of the manuscript by *Planta,* the axialization interpretation could be presented already in 1996, more than a year prior to the cloning of the *MP* gene. At this time, bridging the gap between the genetic and experimental contributions in this field was still the exception. Perhaps because of this early bridge function, the paper was well received. Those positive responses, in turn, helped to maintain funding for our continued map-based cloning efforts, which were entirely based on very soft money. Sometime in 1997, a cDNA sequence, tentatively identified as MP, showed transcription factor motives and translational fusions localized the product to the nucleus. Three publications in the following year, 1998, revealed the identity of the MP (Hardtke and Berleth 1998) and of the PIN1 gene products (Galweiler et al. 1998) as well as the mode of action of an entire transcription factor family, the AUXIN RESPONSE FACTORs (ARFs) (Guilfoyle et al. 1998). Together with the cloning of numerous other components of the auxin signal transduction and auxin transport mechanisms, this development quickly generated a coherent and testable molecular picture of the role of auxin in embryos and beyond. The MP transcription factor represented the most functionally characterized ARF (ARF5) in this context, and PIN1 was the best characterized member of a membrane protein family with a presumed, and subsequently confirmed function in facilitating the efflux of IAA from plant cells. The polar cellular localization of PIN proteins was striking, with the highest concentration on the side of presumed auxin efflux in organismal models (Galweiler et al. 1998). Thus, PIN proteins served as markers for auxin flux, and promoter-reporter constructs derived from ARF binding sites served as markers for auxin response (Ulmasov et al. 1997).

In the early 2000 s, the acceptance of PIN proteins as auxin flux direction markers, of auxin response elements as indicators of auxin concentration in combination with numerous newly available genetic inputs to manipulate either auxin transport, perception, synthesis or degradation,

spurred the publication of new auxin-based models, for example, for the establishment of root and shoot meristems, leaf initiation sites, leaf veins and several other patterning processes (Overvoorde et al. 2010; Pernisova and Vernoux 2021; Scarpella 2024; Vanneste et al. 2025; Xiong and Jiao 2019; Reinhardt et al. 2003). Over time, of course, enthusiasm triggered by the abundance of available tools and data may have once again displaced skepticism as auxin models grew like weed - followed by sobering discoveries of inconsistencies (Friml 2022). However, this tidal movement may just reflect how biology works. As a product of evolution, biological regulation cannot be expected to ever follow a simple reductionistic grand scheme. Rather, it is to be expected that multiple levels of regulation keep operating in parallel. The regulatory system's fitness is then not determined solely by its capacity to generate a certain body pattern, but by its regulatory robustness, self-regulatory adaptability and evolvability (Davidson 2006; Kirschner 2015). Any attempt to interpret such a system with reductionistic mindset, will inevitably go through phases of euphoric plausibility, oscillating with phases of inconsistent findings.

From this perspective, the significance of auxin flow in the context of other controls will not be resolved anytime soon. Should there be any lasting merit of the Przemeck et al. 1996 paper, it may lie in its attempts to integrate knowledge from (then) separate areas of plant development research, to point in the direction of signal-flow mechanisms, and to place self-regulatory axis formation, rather than zygotic embryogenesis, at the centre of the plant pattern formation topic. A further lesson for all young PIs might be to not delay the publication of conceptual thoughts in an open-minded journal. Publications are superior to seminars and other presentations for introducing a concept in the context of all underlying evidence, because readers have more time and opportunity to appreciate new, unconventional concepts; plus there is the possibility that a publication can be re-reviewed in a 'perspective' review 30 years later.

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Ethical approval Not applicable

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