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► **To cite this version:**

Camila Goldy, Marie-Cécile Caillaud. Maintaining asymmetry in cell division. *Science*, 2023, 381 (6653), pp.27-28. 10.1126/science.adi6664 . hal-04277375

HAL Id: hal-04277375

<https://hal.science/hal-04277375>

Submitted on 15 Nov 2023

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PLANT BIOLOGY

Maintaining asymmetry in cell division

Establishing asymmetric division through microtubule dynamics establishes cell fate

By Camila Goldy and Marie-Cécile Caillaud

In multicellular organisms, cell division contributes to the generation of specialized cells. In walled organisms such as plants, the orientation of the division plane by which two daughter cells separate is of special importance because cells are embedded in a matrix and cannot relocate. Plants have evolved a specific structure called the preprophase band (PPB), which is composed of microtubules and defines the future cell division zone before mitosis. In the context of asymmetric cell division, a polar domain is established at the plasma membrane to allow the differentiation of one of the daughter cells while maintaining a stem cell population. How plant cells establish the polar domain remains enigmatic. On page XXX of this issue, Muroyama *et al.* (1) reveal that a polarized destabilization of microtubules demarcates a region of the plant cell as incompatible with microtubule PPB establishment, thereby orienting asymmetric cell division and the development of stomata (pores for gas exchange).

During leaf development, stomata are produced in the surface cell layer by iterative asymmetric cell divisions. This process involves the conversion of a subset of protodermal cells into meristemoid mother cells that divide asymmetrically to produce two daughter cells of unequal volumes. The small daughter cell becomes a meristemoid, which ultimately produces guard cells surrounding the stomatal pore while the larger daughter cell (the stomatal lineage ground cell, SLGC) further divides asymmetrically to eventually differentiate into puzzle-shape epidermal cells (pavement cells) while maintaining a controlled amount of meristemoids (2). During this process, a plasma membrane-associated polarity crescent defined by BREAKING OF ASYMMETRY IN THE STOMATAL LINEAGE (BASL) and BREVIS RADIX family (BRXF) proteins is formed and maintained throughout asymmetric cell division (3). The orientation of the subsequent asymmetric cell division, defined by the BASL-BRXF polarity crescent, is tightly controlled to enforce the “one-cell spacing rule,” which ensures that stomata do not directly touch, thus optimizing gas exchange during photosynthesis while minimizing water loss (4).

To address the long-standing question of how the orientation of asymmetric divisions is determined and maintained, Muroyama *et al.* used stomatal lineage formation in *Arabidopsis thaliana* as a model. They showed that the cortical division zone (a landmark for the site of future division) and the microtubule PPB cannot form within the BASL-BRXF polarity crescent. This result agrees with earlier reports showing that the localization of BASL-BRXF is polarized prior to PPB formation when cortical division zone components are initially recruited to the cortex (5–7). Within asymmetrically dividing cells in the leaf epidermis, the default state for cell division orientation appears to be the shortest wall (requiring the least energy consumption). However, when the shortest path conflicts with the one-cell spacing rule, these rules need to be broken. The authors reported that the polarized BASL-BRXF domain is required to override default division patterns during formative asymmetric divisions and this is altered in *basl* mutants. Reciprocally, the establishment of the microtubule PPB is required for stomatal patterning. In a mutant that no longer forms the microtubule PPB, the division plane often bisects the BASL-BRXF polarity crescent, leading to aberrant cell fate in the resulting daughter cells.

How does the BASL-BRXF polar domain influence PPB positioning? Using time-lapse imaging in planta, the authors demonstrated that the BASL-BRXF polar domain locally depletes cortical microtubules from the plasma membrane. There, the BASL-BRXF polar domain induces microtubule plus-end depolymerization rather than physically preventing microtubule polymerization into the domain per se. Based on these results, the authors concluded that polarization-mediated destabilization of the microtubule plus ends renders a region of the plasma membrane incompatible with microtubule PPB formation, thereby orienting cell division.

As in plant cells, division orientation in animal cells often prescribes daughter cell polarity by determining if a cell inherits a cortical polar domain. For division plane specification in animal cells, the orientation of the nucleus and/or mitotic spindle emerge from physical constraints that are sensed by microtubules, leading to bisection of the spindle axis in cytokinesis. To bias geometrical rules to break symmetry, polar domains typically recruit or activate cytoskeleton-associated proteins at

the cortex, yielding net forces that reorient the mitotic structures such as the astral microtubules which extend from the spindle poles (8). In the context of asymmetrical division in plant cells, the study by Muroyama *et al.* suggests that the orientation of the asymmetric division is not obtained through an effect of astral microtubules because plants do not have them, but rather through effects on cortical microtubules transitioning toward the formation of a PPB before mitosis. Moreover, in plant asymmetric cell division, the polar domain does not specify a position for cytokinesis but acts as a forbidden landmark where microtubules cannot align, preventing the division plane from colliding with it.

The proposed mechanism by which polarity proteins could specify the division plane by creating a microtubule-depleted zone raises the question of how the BASL-BRXF complex is recruited to the plasma membrane and how does it exclude microtubules? Based on preliminary genetic evidence (9), BASL and BRXF are mutually required for the polarization of each other. In the current model, BRX proteins reversibly associate with the plasma membrane via palmitoylation at their N-termini (9). BASL is recruited through an interaction between BASL and the BRX domains that define the BRXF proteins. The nature of the upstream signal that defines the location of BASL-BRXF polarization remains to be discovered.

It is likely that the scaffold proteins BASL-BRXF recruit a microtubule-associated protein to affect microtubule dynamics, rather than directly mediating plus-end depolymerization. The critical microtubule-associated proteins might not be cell division-specific, because the same microtubule depletion is observed post-division in SLGCs for as long as the polar domain persists. But how can the component responsible for the direct effect on microtubules be identified? The establishment of asymmetry in animal cells often involves both biochemical and mechanical processes (8). Because microtubules were proposed to be tension sensors (10), a better understanding of the mechanical constraints and subsequent signaling might be one of the aspects to consider.

During pavement cell differentiation, specific small guanosine triphosphatases (GTPases) called Rho of Plants (ROPs) act as master regulators of cell polarity, mediating the recruitment of microtubule-associated

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1 proteins in the polar domain to shape cyto-
2 skeleton distribution. Upstream, ROPs are
3 regulated by several stimuli including the re-
4 ceptor-like kinase FERONIA (FER) pathway,
5 which participates in sensing and/or transduc-
6 ing mechanical signals (11–13). Investigating
7 the crosstalk between ROP signaling and
8 symmetry breaking in the context of asym-
9 metric cell division might be key in the future.
10 Overall, this work highlights the singular
11 mechanism used by plants to control the in-
12 heritance of fate regulators by connecting po-
13 larity with microtubule interactions.

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38 ACKNOWLEDGMENTS

39 The authors are grateful to Y. Jaillais and G. Ingram
40 for their input on the manuscript. M.C.C. is sup-
41 ported by French National Research Agency
42 ANR-20-CE13-0026-02. C.G. is funded by an
43 EMBO fellowship (ATLF 646-2021).

44 10.1126/science.adi6664