Determination of Symmetric and Asymmetric Division Planes in Plant Cells

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Annu. Rev. Plant Biol. 2011. 62:387–409

First published online as a Review in Advance on March 8, 2011

The *Annual Review of Plant Biology* is online at plant.annualreviews.org

This article's doi: 10.1146/annurev-arplant-042110-103802

1543-5008/11/0602-0387$20.00

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Keywords

cytokinesis, asymmetric cell division, preprophase band, phragmoplast, cell plate, cell polarity

Abstract

The cellular organization of plant tissues is determined by patterns of cell division and growth coupled with cellular differentiation. Cells proliferate mainly via symmetric division, whereas asymmetric divisions are associated with initiation of new developmental patterns and cell types. Division planes in both symmetrically and asymmetrically dividing cells are established through the action of a cortical preprophase band (PPB) of cytoskeletal filaments, which is disassembled upon transition to metaphase, leaving behind a cortical division site (CDS) to which the cytokinetic phragmoplast is later guided to position the cell plate. Recent progress has been made in understanding PPB formation and function as well as the nature and function of the CDS. In asymmetrically dividing cells, division plane establishment is governed by cell polarity. Recent work is beginning to shed light on polarization mechanisms in asymmetrically dividing cells, with receptor-like proteins and potential downstream effectors emerging as important players in this process.

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INTRODUCTION

Unlike animal cells, plant cells are embedded in a matrix of wall material and do not migrate. Therefore, the cellular organization of plant tissues is determined by patterns of cell elongation and division coupled with cellular differentiation. Symmetric divisions usually occur in planes that can be predicted by simple, geometrical rules related to mother cell shape and/or the major axis of elongation of the mother cell and lead to simple proliferation. In contrast, asymmetric divisions are associated with initiation of new developmental patterns, cell layers, or cell types. They are oriented in planes that would not be predicted by mother cell shape and generate daughters with distinct sizes, shapes, and/or developmental fates. For example, asymmetric cell divisions in root meristems follow a stereotyped pattern that establishes distinct cell layers with different identities and functions (1). Thus, understanding mechanisms governing the orientation of both symmetric and asymmetric cell division is crucial for gaining insight into plant development.

Cell plate: a nascent cell wall formed by fusion of vesicles and tubules at the midline of the phragmoplast that eventually becomes attached to the mother cell wall to complete cytokinesis

Phragmoplast: the cytokinetic apparatus of plant cells, composed of short microtubules and actin filaments that guide vesicles to the phragmoplast midline where they fuse to form the cell plate

Plant cells divide by forming a new cell wall (cell plate) between daughter nuclei after mitosis (**Figure 1**). Cell plates are formed via vesicle fusion at the midline of a cytokinetic structure called the phragmoplast, which assembles during telophase (113, 143). The phragmoplast is composed of two opposing disks of short microtubules and actin filaments and expands, along with the cell plate, centrifugally toward the cell periphery as cytokinesis proceeds. The cell plate then attaches to the mother cell wall to complete cytokinesis. In contrast, animal and fungal cells divide by creating an acto-myosin ring that constricts centripetally to pinch the daughter cells apart, although the completion of cytokinesis requires vesicle delivery and fusion as in plant cells (128). Cell plate formation and phragmoplast dynamics have been the subject of recent reviews, (e.g., 59, 65, 118, 138). This review instead focuses on the problem of how plant cells orient their divisions.

In keeping with their distinct mode of cytokinesis, the mechanisms by which division planes are determined in plants appear to be different from those in fungi and animals. In symmetrically dividing animal cells, the position of the spindle primarily determines the final division plane (109), whereas in fungi, division planes are determined before mitosis, e.g., by the position of the prophase nucleus in fission yeast and the previous bud site in budding yeast (96). Like fungi, plant cells establish their division planes prior to mitosis. Plant division planes are established via a cortical array of cytoskeletal filaments called a preprophase band (PPB) (**Figure 1**). The PPB forms during G2 and persists throughout prophase but is disassembled upon nuclear envelope breakdown as the spindle forms (29). One function of the PPB is to establish a specialized cortical domain that we refer to here as the cortical division site (CDS), to which the expanding phragmoplast/cell plate is actively guided during cytokinesis. In this review, we discuss recent progress in understanding division plane selection, PPB formation and function, and the molecular features and function of the

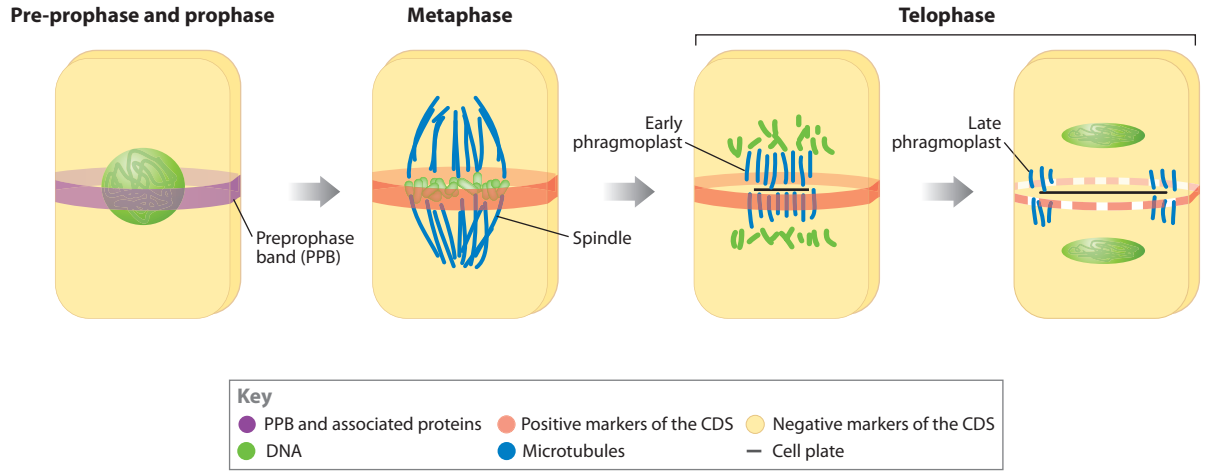

Figure 1

Symmetric, transverse division in a plant cell. Microtubule arrays (PPB, spindle, phragmoplast) are illustrated in blue with positive CDS markers (TAN, RAN-GAP1, and DCD1/ADD1) in red; the combination of blue and red renders the PPB in purple. For simplicity, actin filaments are not shown, but negative markers of the CDS (cortical F-actin and KCA1) are represented in yellow. Abbreviations: CDS, cortical division site; PPB, preprophase band. Modified from Reference 110.

CDS. Most of what is known about division plane control in plants is based on studies of symmetrically dividing cells, and these are discussed first. Later we consider the special case of asymmetric divisions, where division planes are governed by the polarity of the mother cell.

SYMMETRIC CELL DIVISION

Most symmetrically dividing cells orient their divisions according to simple geometrical rules: The division plane corresponds to the shortest possible path that halves the volume of the cell (34) and is perpendicular to the major axis of cell expansion if the cell is elongated (58). A model that considers the tension on cytoplasmic strands as the PPB forms has been proposed to explain how cells follow these rules (78). The premitotic nucleus becomes centered within the division plane via a microtubule-dependent mechanism as the PPB forms (147). Displacement of the premitotic nucleus resulted in a corresponding change in the position of the PPB (15, 92), indicating that the nucleus also plays a role in instructing the cell where to form a PPB.

PPBs and Division Plane Determination

Before embarking on our discussion of PPBs, it should be noted that they are not a universal feature of plant cell division. Some dividing plant cells lack PPBs, e.g., starchy endosperm (112) and meiocytes (98). Moreover, vegetative cells (chloronema and caulonema) in the mosses *Physcomitrella patens* (32) and *Funaria hygrometrica* (116) lack PPBs, although PPBs are observed in the leafy gametophytes (31, 117, 126). Intriguingly, moss caulonema and chloronema cells display well-ordered division planes in spite of the absence of PPBs, suggesting that PPB-independent mechanisms control division planes in these cells. Interestingly, their division planes are nevertheless altered by the microtubule-disrupting drug colchicine (117).

However, in cells that normally make PPBs, lack of a PPB due to mutation (6, 17, 68, 126, 135, 146), experimental treatments that block PPB formation (87, 110, 141), or natural variation in the formation or morphology of the PPB (20, 47, 150) lead to misoriented cell division planes. These observations together with the universal correlation between PPB position

Preprophase band (PPB): a band of microtubules and actin filaments that forms in the future division plane during G2, which persists throughout prophase and is disassembled upon transition to metaphase

Cortical division site (CDS): a zone of the cell cortex during mitosis and cytokinesis located at the former preprophase band site; the site where the cell plate will become attached to the mother cell wall

and the subsequent division plane strongly argue that the PPB plays a major role in determination of the division plane in somatic plant cells.

PPBs were originally discovered as microtubule structures (105) but also have an actin component. The actin PPB is wider than the microtubule PPB in many plant cells, and its formation depends on microtubules (99, 136). Conversely, formation of the microtubule PPB does not appear to depend on F-actin (99), although the narrowing of the microtubule PPB that occurs as prophase progresses is actin-dependent (88). However, drug treatments to depolymerize microtubules prior to PPB narrowing (followed by drug washout to allow cells to proceed through mitosis and cytokinesis) did not interfere with correct cell plate orientation (80). Moreover, wide PPBs observed in *clasp* mutants (described below) do not cause division plane defects (4, 72). These observations suggest that narrowing is not essential for PPB function. The function of the actin component of the PPB remains unclear, although experiments involving timed application of actin-depolymerizing drugs point to an important role for actin in division plane establishment prior to mitosis (61, 114).

PPB Formation and Disassembly

Several proteins with multiple roles in microtubule dynamics or organization are required for normal PPB formation. One of these is MOR1/GEM1, a homolog of XMAP215/TMP200, a regulator of microtubule dynamics (14, 69, 71). MOR1/GEM1 colocalizes with PPBs and other microtubule arrays (54, 68, 149), and it promotes PPB formation and normal division plane orientation (68). Another regulator of microtubule dynamics implicated in PPB formation is CLASP, which localizes to PPBs along with other microtubule arrays (4, 72). Although *clasp* mutant PPBs are wide and disorganized, *clasp* mutants have no division plane defect, presumably because the PPB defects are minor.

Phosphorylation/dephosphorylation of unknown target proteins appears to be critical for PPB formation and disassembly. A PP2A phosphatase B″ regulatory subunit encoded by *FASS/TON2* in *Arabidopsis thaliana* (17) and two closely related paralogs, *dcd1* and *add1*, in maize (146) are required for PPB formation, as well as for proper organization of interphase microtubule arrays (83, 134). Conversely, phosphorylation has been associated with destabilization of PPB microtubules. Cyclin-dependent kinases (CDK) and associated proteins have been shown to localize to PPBs along with other mitotic microtubule arrays (12, 24, 89). Microinjection of CDK into *Tradescantia* stamen hairs caused rapid depolymerization of the PPB, suggesting that CDK/cyclin-dependent phosphorylation promotes the disassembly of PPB microtubules at prometaphase (63).

RSA-1, the *Caenorhabditis elegans* homolog of FASS/DCD1/ADD1, localizes to the centrosome, a structure with no counterpart in plant cells, which serves as a site of microtubule nucleation in animal cells, where it interacts with proteins that mediate microtubule outgrowth and stability (115). Another parallel between centrosomes and PPBs has recently emerged from studies of the *A. thaliana ton1* mutant, which, like *fass*, lacks PPBs and has severely misoriented cell divisions (135). The *ton1* phenotype is caused by mutations in two closely linked and highly similar genes, *TON1a* and *TON1b*. The TON1 proteins are related to centrosomal proteins, interact with centrin (another centrosomal protein), and colocalize with PPBs (6). TON1 function is conserved in the moss *P. patens*. In the leafy gametophytic cells that make PPBs, *ton1* mutants have drastic division plane defects. In vegetative cells lacking PPBs, the *ton1* mutation has no impact on division planes but somehow impairs gravitropic response (126). Together, these findings suggest that centrosomal proteins are functionally conserved in plants but spatially distributed to form a cortical band that supports PPB formation. It will be interesting to know whether TON1 and/or other PPB-associated proteins discussed earlier are direct targets of

dephosphorylation by a FASS/DCD1/ADD1-dependent PP2A phosphatase.

A variety of other proteins colocalize with PPBs along with other microtubule arrays, but their roles in PPB formation or function are not yet well defined. These include microtubule-interacting proteins, e.g., MAP65s (139); cytoskeletal motor proteins, e.g., kinesin-like calmodulin binding protein KCBP (13); a variety of cell cycle regulators (12 and references therein); and kinases, e.g., RUNKEL (75). Recently, the MAP kinase MPK6 (90) and the anaphase promoting complex (APC) activator CCS52A2 (12) were shown to colocalize with PPBs and other structures in dividing cells. Both *mpk6* and *ccs525A2* mutants appear to have a division plane defect in roots (90, 140), which may reflect roles for these proteins in PPB formation or function, but additional work is needed to clarify their roles in division plane control.

Spindles and Division Plane Determination

Classic experiments demonstrated that spindles do not determine the division planes of plant cells. If the spindle (or early phragmoplast) is displaced from the division plane by centrifugation, the expanding phragmoplast/cell plate can subsequently track back to the CDS (48, 97). However, the ability of a displaced phragmoplast to track back to the CDS works only over relatively small distances in the cell (41, 80, 97), so formation of a spindle whose axis bisects and is perpendicular to the division plane, and maintenance of this spindle orientation/position throughout mitosis, facilitates later insertion of the cell plate at the CDS.

With this thought in mind, it is interesting that recent work has established a role for the PPB in the formation and initial orientation of the spindle. At prometaphase, spindle assembly begins prior to PPB breakdown, with the spindle axis perpendicular to the plane of the PPB. In cultured cells lacking PPBs, spindle formation was delayed and the spindles that formed were flattened or multipolar. Later, by an unknown mechanism, these deformed spindles transitioned into proper bipolar spindles, but mitosis was delayed (20). In cultured cells that formed two PPBs, multipolar spindle formation also occurred early in metaphase and was later corrected (150). How the PPB promotes the timely formation of a bipolar spindle remains to be elucidated, but a recent study has suggested an important role for "bridge" microtubules connecting the PPB to the prophase nucleus (3).

Spindle: a bipolar array of microtubules that separates daughter chromosomes during mitosis

A variety of studies have revealed a cage of actin filament bundles that surrounds the spindle and connects it to the cell periphery, which maintains spindle position during mitosis (67, 79, 142). Recent work has also suggested a role for a member of the *A. thaliana* EB1 family, EB1c, in maintenance of spindle position and morphology. In a wide variety of eukaryotic species, EB1 proteins bind to the highly dynamic "plus" ends of microtubules, mediating their interactions with other proteins (19, 120). Unlike EB1a and EB1b, *A. thaliana* EB1c has not been shown to associate with microtubule plus ends, although it does colocalize broadly with mitotic microtubule arrays and, surprisingly, is also localized to the nucleus during interphase (11, 27, 73, 139). Spindles of *A. thaliana eb1c* mutants are occasionally misoriented or collapsed, and these defects are greatly enhanced by treatment with low doses of the microtubule-depolymerizing drug oryzalin. These spindle defects are only partially corrected during cytokinesis, often resulting in misoriented cell divisions (73). Thus, EB1c may mediate an interaction between spindle microtubules and the surrounding actin cage that helps to maintain proper spindle morphology and position during mitosis. Alternatively, EB1c could affect spindle morphology and orientation by altering spindle microtubule dynamics.

Cortical Division Site

The observation that expanding phragmoplasts can track back to the former location of the PPB (commonly referred to as the CDS) has led to

the long-standing idea that the PPB functions to modify the cortex in a manner that persists after the PPB itself is gone. What is the nature of the CDS, and how might the PPB function to create it? Several proteins have been discovered that either mark the CDS or are excluded from it (**Figure 1**) and therefore have the potential to contribute to the function of the CDS in guidance of the expanding phragmoplast during cytokinesis.

Negative markers of the CDS. The first "negative" marker of the CDS to be identified was cortical F-actin. The actin PPB disassembles along with the microtubule PPB at the transition from prophase to metaphase, but cortical actin persists elsewhere, creating an actin depleted zone (ADZ) at the division site in diverse plant species and cell types (22, 100, 114). When actin-depolymerizing drugs were applied early in mitosis and then washed out, division planes were frequently misoriented, but application only during cytokinesis had very little effect on division planes (61, 114). These observations suggest that the ADZ or actin PPB plays an as yet unknown role in division plane establishment prior to cytokinesis.

Another negative marker of the CDS is the kinesin-like protein KCA1, a plasma membrane– and cell plate–localized protein. KCA1 is excluded from the PPB zone during preprophase/prophase in a microtubule-dependent manner. After PPB disassembly, exclusion of KCA1 from the division site is maintained until the cell plate inserts into the mother cell wall (141). A surprising twist in the KCA1 story has come with the recent finding that KCA1 and the closely-related kinesin-like protein KCA2 are essential for actin-mediated chloroplast movement and attachment to the plasma membrane, and that KCA1 binds actin but not microtubules in vitro in spite of its homology to kinesins (130). Notably, the latter finding is consistent with the colocalization of KCA1 and cortical F-actin in mitotic cells. Treatment with actin-depolymerizing drugs did not alter KCA1 localization (141), but KCA1 may somehow direct the pattern of cortical F-actin accumulation in dividing cells. Alternatively, KCA1 may mediate an actin-dependent process important for cell division, e.g., chloroplast inheritance. Further work is needed to clarify the relationship (if any) between KCA1 and KCA2 function in actin-based chloroplast movement and their exclusion from the CDS.

Vesicle trafficking at the CDS. Recent observations have suggested a possible role for endocytosis in division plane establishment, particularly in negative marking of the CDS. An elevated frequency of endocytic (mostly clathrin-coated) vesicles was observed adjacent to the cortex near the PPB relative to other areas (66). Endocytosis in the PPB region was also observed via uptake of the dye FM4-64 in tobacco BY-2 cells, and FM4-64 uptake was reduced when microtubules were depolymerized with oryzalin (26). These observations suggest that PPB-associated microtubules direct localized, selective removal of certain molecules from the cell surface via endocytosis, potentially explaining microtubule-dependent depletion of F-actin and/or KCA1 from the CDS.

Secretion has also been postulated to play a role in establishment or maintenance of the CDS based on observations of vesicles near the PPB (e.g., 33, 42, 43, 49) and a local accumulation of Golgi stacks in the cortical cytoplasm near the CDS, referred to as a Golgi belt, which forms either during preprophase/prophase (26, 28) or later in mitosis (95). However, treatment of dividing BY-2 cells with the Golgi-disrupting compound Brefeldin A, followed by subsequent drug washout to permit cytokinesis, did not alter phragmoplast expansion to the CDS, suggesting that Golgi-derived secretion is not necessary for division plane orientation (28). Rather than participating in division plane establishment or maintenance, the Golgi belt could play a role in achieving equal distribution of Golgi between daughter cells.

Vesicle trafficking has also been implicated in cell plate attachment at the CDS via the discovery of TPLATE, which has homology to coat proteins involved in vesicle formation.

During cytokinesis, TPLATE localizes at the cell plate and also appears at the CDS upon contact with the cell plate (137). Functional studies demonstrated that TPLATE promotes cell plate attachment, potentially via localized vesicle trafficking at the junction between the cell plate and the CDS (137).

Positive markers of the CDS. The first protein identified as a positive marker of the CDS throughout mitosis and cytokinesis was TANGLED (TAN), originally discovered in maize where loss of this protein causes a high frequency of misoriented division planes (122). TAN is a highly basic protein that binds microtubules in vitro (121). It is not required for PPB formation or orientation but rather for phragmoplast guidance to the CDS (23, 144). TAN colocalizes with PPBs and remains localized at the CDS as cells proceed through metaphase and cytokinesis (144). These observations support the conclusion that TAN functions at the CDS to help guide the expanding phragmoplast during cytokinesis. Intriguingly, recent data have shown that TAN is not statically maintained at the CDS during mitosis and cytokinesis. Instead, it is recruited to the division site by different mechanisms during prophase versus telophase. Thus, the CDS is actively modified throughout division rather than being set up by the PPB and subsequently maintained by a passive retention mechanism. The significance of early versus late recruitment of TAN to the division site is presently unknown, but both have been shown to depend on the formation of the PPB (110).

The only other protein presently known to localize at the CDS during mitosis and cytokinesis is RAN-GAP1, a protein that stimulates the GTPase activity of RAN, a regulator of nucleocytoplasmic transport and several aspects of mitosis (21). Like TAN, RAN-GAP1 colocalizes with the PPB and then remains at the CDS during mitosis and cytokinesis. Unlike TAN, it also localizes to the spindle midzone, the kinetochores, the cell plate, and the nuclear envelope. Knockdown of RAN-GAP function by RNAi leads to misoriented division planes and other defects in cytokinesis (148). Thus, RAN-GAP is required for both cytokinesis and division plane orientation, a role consistent with its localization at the CDS and cell plate. How it serves these functions remains to be determined.

Two closely related kinesins, POK1 and POK2, are required in combination for division plane orientation in *A. thaliana* (91). *pok1/pok2* double mutant cells have defects in PPB placement and phragmoplast guidance to the division site (91). Interestingly, POK1 interacts in yeast with RAN-GAP1 and with the domain of TAN that directs its localization to the CDS during cytokinesis (110, 148). Moreover, POKs are required for localization of both TAN and RAN-GAP1 to the CDS during cytokinesis (144, 148). Thus, POKs may deliver TAN and RAN-GAP1 as cargo to the CDS during cytokinesis along microtubules that link the phragmoplast to the cortex (20, 26). Alternatively, the POKs may interact with microtubules to colocalize with the PPB and function later at the CDS during cytokinesis to recruit TAN and RAN-GAP1. Localization of POKs will help clarify their role in division plane orientation.

A functionally redundant pair of PP2A phosphatase B″ subunits in maize, DCD1 and ADD1, represent a third positive marker of the CDS, although a transient one. As discussed earlier, these proteins, like their *A. thaliana* homolog FASS, are required for PPB formation (17, 146). In *Z. mays*, DCD1 and ADD1 proteins colocalize with the PPB, remain at the CDS until metaphase, and subsequently disappear from the cortex, suggesting that they have another function at the division site besides promoting PPB formation (146). One role may be the maintenance of TAN and RAN-GAP1 at the division site after PPB disassembly, as both require FASS for CDS localization in *A. thaliana* (144, 148). Supporting this possibility, cells treated with the PP2A inhibitor okadaic acid lose cortical TAN localization during prophase and metaphase (110). Identification of the targets of DCD1/ADD1 and FASS should help to clarify their role(s) at the division plane after PPB disassembly.

In addition to its role in division plane orientation, there has long been evidence that some component(s) of the CDS is required for cell plate maturation. For example, if the cell plate attaches to the cortex outside of the CDS, the new wall fails to mature normally (48, 86). AIR9, an essential microtubule-associated protein, has a localization pattern that fulfills many expectations for a CDS-associated cell plate maturation factor. AIR9 colocalizes with the PPB via an N-terminal microtubule binding domain, disappears from cortex, then relocalizes to the CDS via a separate domain of the protein when the cell plate makes contact (but only if contact is made at the CDS, not elsewhere in the cortex; 16). Subsequently, AIR9 becomes distributed across the maturing cell plate. Cell plates that fuse somewhere other than the CDS and do not acquire AIR9 fail to mature properly as indicated by persistence of callose (16).

Future Directions

Much still needs to be learned about how division planes are controlled in symmetrically dividing plant cells. For instance, although several factors needed for PPB formation have now been identified, very little is known about how cell shape and nuclear position are communicated to the cortex to determine the site of PPB formation. Although some components of the CDS have now been identified, very little is known about their functions or interrelationships, and many more components surely await discovery. Furthermore, the question of how the expanding phragmoplast and cell plate interact with the CDS to position the division plane there have barely begun to be addressed at a mechanistic level.

ASYMMETRIC CELL DIVISIONS

Asymmetric cell division generates cellular diversity, contributing to cell patterning and stem cell maintenance during plant development. Daughter cells of different sizes, shapes, and/or fates are formed when a cell divides in an asymmetric manner. To achieve a physically asymmetric division, the mother cell establishes an axis of polarity prior to mitosis involving nuclear migration to an asymmetric location along with reorganization of other cell components and then orients the division plane in relation to this axis. According to a classic paradigm, developmental fates of the daughter cells are specified by one of two mechanisms (60). In intrinsically asymmetric divisions, mother cell polarity directs a nonuniform distribution of cell fate determinants, which are inherited unequally by the daughters. In this case, proper placement of the division plane in relation to the axis of cell polarity ensures unequal inheritance of fate determinants. In extrinsically asymmetric divisions, daughter cells have initially equivalent potential but are exposed to different positional cues that cause them to develop differently. Here, properly oriented division ensures that daughters will be born in distinct environments, leading them down divergent developmental paths.

The majority of work in the field of plant asymmetric cell division has been concerned with mechanisms specifying the fates of daughter cells. Indeed, much exciting work has emerged in this area in recent years and has been recently reviewed (1, 85). However, our objective in this part of the review is to focus specifically on the question of how asymmetric division planes are oriented, considering where possible how the literature on symmetric division can inform our thinking about this problem.

Extensive studies of asymmetric division in diverse animal model systems have revealed that a variety of upstream polarizing cues can be translated into polarization of premitotic cells via a conserved set of polarity regulators that includes the PAR proteins originally discovered in *C. elegans* (46). Anterior and posterior cortical domains are occupied by different combinations of polarity proteins, which then interact with astral microtubules to orient the spindle and thus orient the division plane (82). Perhaps not surprisingly, given the different mechanisms used by plant cells to determine their division planes, homologs of PARs and

many other conserved regulators of polarity in animal cells have not been identified in the fully sequenced genomes of several plants, suggesting that plants use different mechanisms to generate polarity for asymmetric cell division. Nonetheless, a similar logic may be at work.

Asymmetrically dividing cells do not follow the rules already outlined that relate the division plane to mother cell shape in symmetrically dividing cells. Further unlike symmetrically dividing cells, actin governs the migration of the premitotic nucleus into the future division plane (70, 88, 101) and the placement of the PPB (88) in asymmetrically dividing cells. Moreover, displacement of the premitotic nucleus in asymmetrically dividing cells does not lead to repositioning of the PPB as it does in symmetrically dividing cells (41, 104), and even without intentional displacement, the premitotic nucleus can sometimes be observed outside the plane of the PPB (101). These observations support the view that in asymmetrically dividing cells, actin-dependent cell polarity overrides cell shape and the premitotic nucleus as the primary determinants of PPB position, and it also directs nuclear migration into the future division plane.

The fragmented nature of our understanding of how asymmetric divisions are oriented in plants precludes presentation of a unified view, so instead we organize our discussion around different types of asymmetric cell divisions that give us small glimpses into mechanisms of division plane control.

The First Zygotic Division

In angiosperms, the first division of the zygote is highly asymmetric. Once fertilized, the zygote elongates and divides asymmetrically, with the smaller apical cell generating most of the embryo and the larger basal cell giving rise mainly to the extraembryonic suspensor. Genetic studies in *A. thaliana* have identified a variety of factors required for proper specification of apical and basal daughters and their derivatives, including components of a MAPK cascade; components of the auxin synthesis, transport, and response machinery; and WUSCHEL-RELATED HOMEOBOX (WOX) transcription factors (25). However, none of these are clearly implicated in orientation of the first division plane, although a zygotic elongation defect in MAPK cascade mutants leads to a less dramatic size difference in the daughters of the first division. A striking defect in the plane of the first zygotic division is observed in *gnom* mutants, where the zygote elongates normally but then divides almost symmetrically (81). GNOM is a positive regulator of ARF GTPases that promote vesicle formation in the secretory pathway and is well known for its role in polarized trafficking of certain PIN auxin efflux carriers (45, 129). Defects in embryonic patterning seen in *gnom* mutants are thought to be due to loss of PIN polarity and consequent alterations in auxin distribution after the first zygotic division. It is not known how GNOM helps to polarize the first zygotic division, but its molecular identity clearly indicates a role for vesicle formation/trafficking (111).

The inaccessibility of plant zygotes for observation and experimental (other than genetic) manipulation has made it difficult to study factors governing the first asymmetric division plane. Further complicating studies of this division is the fact that the zygote is formed by fertilization of the embryo sac, a polarized, multicellular structure, so the polarity of the zygote is related to that of the embryo sac. In contrast, unpolarized eggs of the fucoid algae *Fucus distichus* and *Silvetia* (formerly *Pelvetia*) *compressa* can be fertilized in vitro to generate a synchronous population of zygotes that become polarized and divide asymmetrically within 24 hours (**Figure 2**). These algae have been the subject of extensive investigation regarding the establishment of polarity and orientation of the first, asymmetric division plane perpendicular to the axis of polarity (reviewed, 10). Unlike land plants, fucoid algae do not make PPBs and have centrosomes acquired from the sperm. The centrosomes become associated with the nuclear surface, where they nucleate astral microtubules that mediate a rotation of the premitotic nucleus, partially aligning the

Figure 2

Polarization and asymmetric division in zygotes of fucoid algae (*Fucus distichus, Silvetia compressa*). Polarization is initially established at the site of sperm entry and marked by formation of a localized cortical F-actin patch. The polar axis is reoriented by light but becomes fixed prior to germination. The nucleus rotates into alignment with the axis of polarity and the zygote then divides asymmetrically in a plane determined by the positions of telophase nuclei. All illustrated F-actin except that associated with the cell plate appears to be nucleated by the Arp2/3 complex. Modified with permission from Reference 8.

centrosome axis with the axis of zygotic polarity (2). After mitosis, astral microtubules nucleated at the spindle poles mediate an interaction between the telophase nuclei and the cortex that completes the alignment with the polarity axis and determines the first division plane (9), much as in an animal cell. Cytokinesis is achieved via fusion of vesicles to form a cell plate without plasma membrane contraction, but this process does not involve a phragmoplast and instead utilizes a plate-like array of actin in the division plane (**Figure 2**). Thus, the manner in which zygotic polarity leads to asymmetric placement of the division plane is clearly very different in fucoid algae compared to land plants, but the mechanisms used to generate polarity in the zygote may be more similar to those used by plant cells. In any case, the studies in fucoid algae are interesting in their own right.

As in *C. elegans*, the initial polarity cue in fucoid zygotes is sperm entry (50), but in natural settings, this initial polarity is usually overridden by directional environmental cues such as gravity and light. The first known marker of polarity is a cortical actin patch that forms at the site of sperm entry (52) (**Figure 2**). The polarity axis remains labile for several hours and can be reoriented during this time by changes in the direction of light or gravity. Interestingly, auxin and polar auxin transport are implicated in reorientation of the polarity axis by light or gravity during this labile phase, suggesting that directional environmental cues may be translated into an intracellular auxin gradient or a nonuniform pattern of auxin efflux that reorients zygotic polarity (131). Further elaboration of the polar axis involves formation of rhizoid pole–focused H^+ and Ca^+ gradients (74, 108), microtubule-dependent polarization of the endoplasmic reticulum toward the rhizoid pole (103), and vesicle trafficking focused at the rhizoid pole (53), which appears to be facilitated by a cone-shaped network of Arp2/3 complex–nucleated, branched actin filaments (51) (**Figure 2**). Fixation of the axis of polarity just prior to germination requires the actin cytoskeleton and the cell wall, suggesting that cortical polarity factors that are initially

deposited nonuniformly via polarized vesicle trafficking become tethered to the cell wall via actin-dependent, transmembrane linkages (reviewed, 38). Insights gained from these studies provide abundant food for thought about mechanisms governing polarization of plant cells preparing for asymmetric division.

Hypophysis and Procambial Divisions in the Embryo

In the *A. thaliana* embryo, the uppermost cell formed through a series of transverse divisions within the suspensor is called the hypophysis (**Figure 3**). The hypophysis divides asymmetrically; the smaller apical daughter gives rise to the quiescent center (QC) of the root, and the larger basal daughter gives rise to the root columella. In *A. thaliana* mutants lacking the PP2C phosphatases POLTERGEIST (POL) and POLTERGEIST-LIKE1 (PLL1), this division becomes nearly symmetric and the daughters adopt neither QC nor columella identity (123). *pol/pll* double mutants exhibit a similar loss of both division and fate asymmetry in a division occurring in the procambium at the same embryonic stage (123) (**Figure 3**). These division defects are associated with complete loss of the root meristem and vasculature. Recently, POL and PLL1 have been shown to localize to the plasma membrane via dual acylation (both palmitoylation and myristoylation), and this localization is required for their function (39). Although POL and PLL1 show some nonuniformity in their distribution in the root, they were not shown to be asymmetrically localized in the cells whose divisions are affected in the mutants. In vitro, POL and PLL1 bind to various membrane phospholipids, and their enzymatic activity is stimulated by some of these phospholipids, particularly PI(4)P. Interestingly, members of the lipid kinase family that generates PI(4)P are implicated in polarized membrane trafficking in tip-growing plant cells (107, 132), suggesting a common function for PI(4)P in polarization of cell division and cell growth.

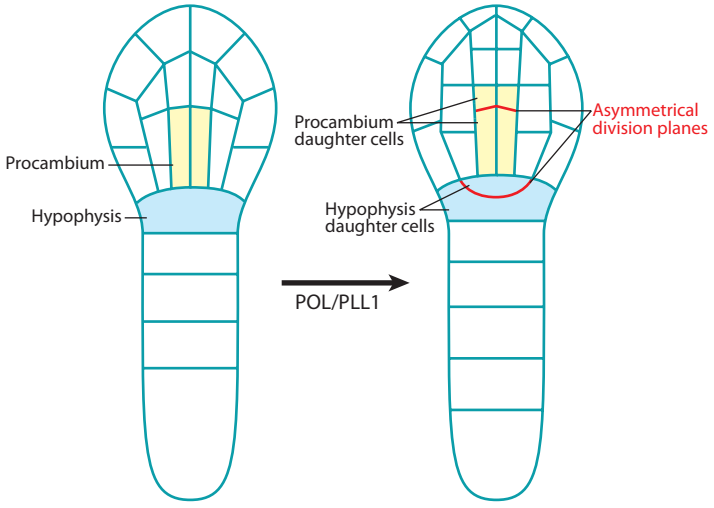

Figure 3

Cellular organization of globular stage *Arabidopsis thaliana* embryos. Hypophysis and procambial cells are indicated, with their asymmetric division planes shown in red. These division planes become nearly symmetric in *pol/pll1* double mutants.

These studies have led to a model in which an external cue (proposed to be a member of the CLE family of CLAVATA3-related, small secreted peptides) interacts with an unknown receptor on one side of the responding cell, bringing about the asymmetric downregulation of POLL/PLL1 activity on that side, either by reducing PI(4)P levels or somehow antagonizing the activating effect of PI(4)P. This model is based in part on the role that POL and PLL1 also play in the shoot, acting downstream of CLAVATA receptors and their putative CLAVATA3 ligand to maintain the shoot meristem (124, 151). Asymmetric inactivation of POLL/PLL1 was proposed to lead to inheritance of active POLL/PLL1 by only one daughter, resulting in distinct cell fates (39), but this model does not obviously explain why the division plane itself is affected in *pol/pll1* mutants. Therefore, an alternative model could be that asymmetric activity of POLL/PLL1 is critical for premitotic polarization, thus driving both asymmetric placement of the division plane and the polarized distribution of other molecules (e.g., transcription factors) that act

as fate determinants and are unequally inherited by daughter cells.

Anticlinal: a division plane perpendicular to the surface of the organ; adds cells to an existing cell layer or file

Periclinal: a division plane parallel to the surface of the organ; creates a new cell layer or file

Root Stem Cells and Initials

In the *A. thaliana* root meristem, asymmetric divisions in three different stem cell populations generate the distinct cell lineages making up the root (1) (**Figure 4**). The cortex/endodermal stem cell undergoes an anticlinal division to generate another stem cell and a cortex/endodermal initial, which divides asymmetrically in the opposite (periclinal) orientation to form cortex and endodermal cells (together referred to as ground tissue). Similarly, columella stem cells divide asymmetrically to produce another stem cell and a columella cell. In contrast, epidermal/lateral root cap stem cells divide asymmetrically in alternating planes: Anticlinal divisions generate epidermal cells, whereas periclinal divisions generate lateral root cap cells (**Figure 4**).

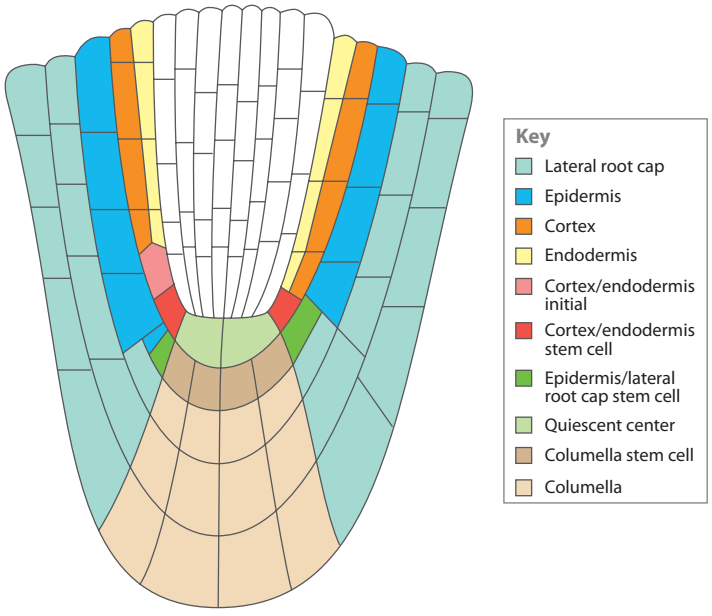

Figure 4

Cellular organization of the *A. thaliana* root meristem with color coding identifying the cell types discussed in the text. Asymmetric, anticlinal divisions of cortex/endodermis stem cells give rise to cortex/endodermis initials, which divide asymmetrically and periclinally to give rise to cortex and endodermis. Columella stem cells divide asymmetrically to give rise to columella. Epidermis/lateral root cap stem cells divide asymmetrically in alternating planes: Anticlinal divisions generate epidermis, whereas periclinal divisions generate root cap.

A variety of transcription factors have been shown to regulate these asymmetric divisions, specifying cell fates as well as the occurrence or orientation of these divisions. SCARECROW (SCR) and SHORT ROOT (SHR) (GRAS family transcription factors) specify endodermal cell fate through a fascinating process involving cell–cell movement of SHR and its sequestration in the endodermal lineage by SCR (1). SCR and SHR also promote the asymmetric divisions of cortex/endodermal initials, as these divisions fail to occur in *scr* and *shr* mutants. SCHIZORIZA (SCZ), a nuclear protein with homology to heat shock transcription factors, specifies cortex/endodermal stem cells (102) and influences the fates of various cells in the root meristem (93, 133). SCZ is also implicated in regulation of division planes by the observation that ground tissue cells in *scz* mutants often divide periclinally (a division plane normally observed only in cortex/endodermis initials), producing ectopic ground tissue layers (93). Finally, FEZ and SOMBRERO transcription factors of the NAC domain family regulate the asymmetric divisions of both columella and epidermal/lateral root cap stem cells (145). Of particular interest for this review, FEZ appears to act in the epidermal/lateral root cap stem cell to specify periclinal division planes, based on the observation that its ectopic expression in endodermal cells causes them to divide periclinally (145). These are fascinating observations, but it remains largely mysterious how transcription factors can promote the occurrence of asymmetric divisions (e.g., SCR and SHR) or control their orientations (e.g., SCZ and FEZ). The influence of these transcription factors on cell division may be at least in part a consequence of their effects on cell fate. However, the recent identification of a D-type cyclin as a direct transcriptional target of SHR points to a direct role for SHR in stimulation of cell division in cortex-endodermal initials (125). Similarly, root patterning transcription factors could influence cell division

orientations by regulating the expression of genes encoding cell polarity proteins.

Vascular Development

The vascular meristem provides an interesting system for the study of polarized cell divisions. A band of procambial cells separates the xylem and phloem. Vascular initials in the procambium divide periclinally in a polarized fashion, generating xylem cells on one side of the procambium and phloem cells on the other. PHLOEM INTERCALATED WITH XYLEM (PXY) is a receptor-like kinase implicated in orienting cell divisions in the procambium (37). *pxy* mutants display phloem interspersed with or adjacent to xylem, instead of being separated by the procambium as in wild-type, apparently due to misoriented procambial divisions. The small peptide CLE41/44 is expressed throughout the phloem, adjacent to the procambium where PXY is expressed, and was identified as a ligand for PXY (57). Both overexpression and mislocalization of CLE41 result in disorganization of the usually precisely oriented cell divisions in the procambium (35). These observations suggest that asymmetric activation of PXY on the surface of procambial cells by phloem-derived CLE41/44 orients procambial divisions. In contrast, separate studies of this receptor–ligand pair have led to a different interpretation of its function in vascular development. CLE41/44 was originally identified as tracheary element differentiation inhibitory factor (TDIF), a suppressor of xylem differentiation in cultured cells (64). PXY was isolated as a receptor interacting with TDIF and named TDR (TDIF receptor) (57). The model arising from these studies is that phloem-derived TDIF/CLE41/44 is perceived by TDR/PXY at the surface of procambial cells, promoting their division and suppressing their differentiation into xylem. According to this model, procambial cells farthest from the phloem receive a weaker TDIF signal and thus differentiate into xylem cells (56). It will be interesting to see how these two models are reconciled with further work on this system.

Stomatal Development

Maize. Stomatal divisions in grasses and other monocots have long been subjects for investigation of asymmetric cell division in plants. The stomatal complexes of grasses are formed through an invariant series of oriented cell divisions (127) (**Figure 5*a***). The guard mother cell (GMC) is formed via an asymmetric, transverse division, and its lateral neighbors, the subsidiary mother cells (SMCs), then divide asymmetrically to form subsidiary cells flanking the GMC. Finally, the GMC divides symmetrically to generate a guard cell pair. Prior to the asymmetric division of the SMC, it becomes polarized toward the GMC, forming a dense patch of cortical F-actin at the GMC contact site, relocating its nucleus to that site, and forming an asymmetric PPB predicting the asymmetric division plane (40).

A receptor-like protein, PANGLOSS1 (PAN1), has been shown to play a role in orienting the asymmetric division of the SMC (18, 44). PAN1 accumulates in a polarized manner at the GMC contact site in premitotic SMCs prior to the appearance of other polarity markers (**Figure 5*a***). *pan1* mutants display defects in premitotic SMC polarization (delocalization and loss of actin patches, and lack of nuclear polarization), leading to aberrantly oriented SMC divisions. PAN1 may perceive a polarizing cue from the GMC or may be recruited to the GMC contact site in response to an upstream sensor of GMC position, helping somehow to translate a GMC-derived cue into SMC polarization. However, not all SMCs are affected in *pan1* mutants, suggesting PAN1 acts redundantly with other proteins to perform its role. To date, a ligand for PAN1 has not been identified.

Phospholipids in the plasma membrane are thought to contribute to cell polarity by providing a local docking site for proteins or substrates for the localized generation of signaling molecules (36, 84). Treatment with inhibitors of phosphatidic acid (PA) synthesis has implicated this phospholipid in SMC polarization (5). In particular, PA is implicated in the formation of cortical actin patches, nuclear

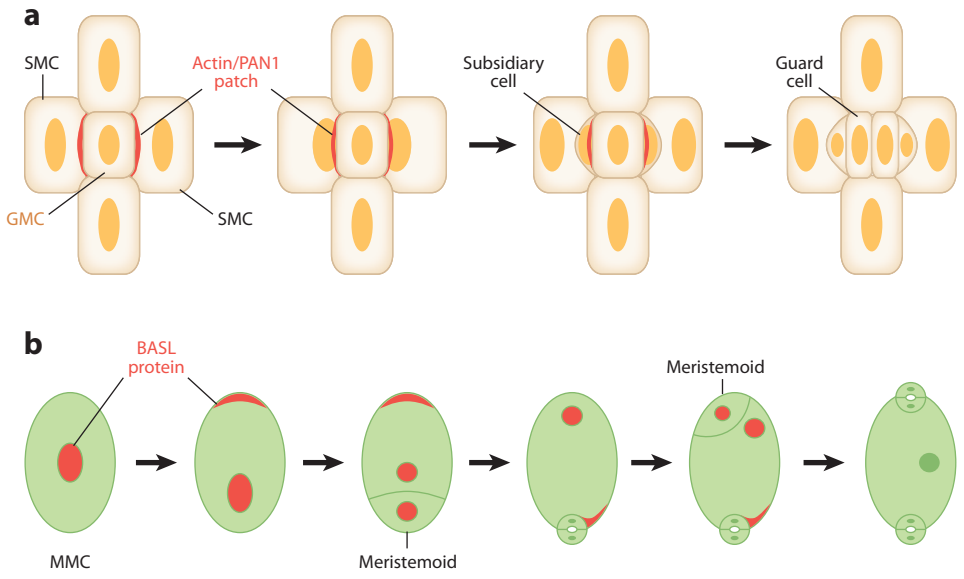

Figure 5

Polarization and asymmetric division of stomatal lineage cells in maize and *A. thaliana*. (*a*) In maize, guard mother cells (GMCs) are generated by a transverse, asymmetric division not illustrated. PAN1 and actin patches (*red*) form within the adjacent subsidiary mother cells (SMCs) at the site of contact with GMCs prior to migration of the nucleus (*yellow*) to this site. Subsequent asymmetric division of the SMC generates a small, triangular-shaped subsidiary cell. Finally, symmetric division of the GMC generates a guard cell pair. (*b*) In *A. thaliana*, a meristemoid mother cell (MMC) becomes polarized, localizing BASL protein (*red*) in the nucleus and at the cell surface at the pole opposite the site where asymmetric division later occurs to generate a meristemoid. The sister cell relocalizes BASL in preparation for a second asymmetric division generating another meristemoid opposite the first one. Both meristemoids divide symmetrically to generate a guard cell pair separated by one nonstomatal cell.

polarization, and completion of the division. These observations are consistent with the conclusion of an earlier study that PA promotes F-actin polymerization by inhibiting actin capping protein (62). The proposed role for PA in SMC polarization is also consistent with a more recent study demonstrating that PA participates in a positive feedback loop in tobacco pollen tubes in which PA activates F-actin polymerization and actin, in turn, activates phospholipase D, further increasing PA levels (106). PA may also stimulate SMC polarization via activation of a MAPK cascade, as demonstrated in soybean cells responding to wounding (77). Indeed, a MAPK cascade in *A. thaliana* has been implicated in polarization of stomatal lineage divisions, as discussed in the next paragraph. It will be interesting to determine whether localized PA synthesis at GMC contact sites in SMCs is stimulated as a consequence of PAN1 function.

A. thaliana. In *A. thaliana*, asymmetric division of a meristemoid mother cell generates a small meristemoid and a large sister cell (the stomatal lineage ground cell, or SLGC) (**Figure 5*b***). The meristemoid can divide up to three times in an asymmetric manner to generate additional meristemoids, and the SLGC can also undergo additional meristemoid-forming divisions. Each meristemoid eventually becomes a GMC that divides symmetrically to form a guard cell pair (**Figure 5*b***). The one-cell spacing rule states that there is at least one nonstomatal cell between neighboring stomatal complexes; this is achieved by orienting meristemoid-forming asymmetric

divisions away from existing guard cells or their precursors (7).

TOO MANY MOUTHS (TMM) is a leucine-rich repeat (LRR) receptor-like protein, which is expressed in meristemoids and cells capable of dividing to produce them (94). Analysis of *tmm* mutants has demonstrated that TMM inhibits meristemoid-forming divisions and also orients these divisions to achieve the one-cell spacing rule (94). ERECTA family LRR receptor-like kinases (ERLs) have recently been implicated as potential coreceptors for TMM, which lacks an intracellular kinase domain so would be incapable of functioning in signal transduction on its own (119). A potential ligand for TMM+ERLs, EPIDERMAL PATTERNING FACTOR1 (EPF1), has been identified as a regulator of the one-cell spacing rule (55). The EPF1 peptide is produced by stomatal precursors, potentially serving as a spatial cue to signal TMM+ERLs to orient meristemoid-forming divisions away from these precursors. Signaling via TMM+ERLs is thought to be transduced via a MAPK cascade (reviewed, 1). Mechanistic insights have been achieved into how this pathway negatively regulates a transcription factor that promotes the formation of meristemoids (76), but how this pathway controls meristemoid division polarity remains to be elucidated. However, recent colocalization of MPK6 with PPBs, phragmoplasts, and cell plates suggests that the MAP kinases acting downstream of TMM/ERLs could play direct roles in positioning these mitotic structures (90). Notably, the effects of the TMM/ERL pathway on asymmetric division are different from those of PAN1 in that the TMM/ERL pathway is not required for polarization per se but determines the direction of polarization.

BREAKING OF ASYMMETRY IN THE STOMATAL LINEAGE (BASL) is a novel, plant-specific protein that promotes division polarity within the stomatal lineage of *A. thaliana* (30). In *basl* mutants, asymmetric stomatal lineage divisions display reduced physical and cell fate asymmetry. As illustrated in **Figure 5*b***, BASL is localized in nuclei and at the cell surface in a polarized crescent on one side of meristemoid mother cells, distal to the prospective site of asymmetric division. Subsequently, BASL is unequally inherited by the daughter cells: In the meristemoid, it is found only in the nucleus, whereas the larger daughter cell localizes BASL in both the nucleus and the crescent. Ectopic expression experiments suggested a role for cell surface–localized BASL in promoting localized cell expansion, so unequal inheritance of BASL may increase the size asymmetry between daughters after division. BASL appears to act downstream of TMM and EPF1, forming crescents positioned as expected for the misoriented divisions that occur in *tmm* and *epf1* mutants (i.e., opposite to the prospective division site). Identification of BASL-interacting proteins may yield insight into how it achieves its intriguing localization pattern and how it participates in orienting asymmetric cell division.

The polar localization of PAN1 in maize and BASL in *A. thaliana* and the roles demonstrated for both proteins in polarization of asymmetric division during stomatal development raises the question of whether they are involved in the same pathway. A key difference in the polar localization of the two proteins is that although PAN1 is restricted to the site of asymmetric division, BASL is localized opposite this site. Although this may be reminiscent of the way different PAR complexes in animal cells partition to opposite cell poles, the fact that mutants lacking the closest relatives of PAN1 in *A. thaliana* do not display any obvious disruptions to asymmetric divisions in the stomatal lineage, and that these PAN1-like proteins do not localize asymmetrically in stomatal lineage cells (M. Rodriguez, L. Clark, and L.G. Smith, unpublished) suggests that PAN1-like proteins do not promote division asymmetry during stomatal development in *A. thaliana*.

Future Directions

Although exciting advances have been made in understanding orientation of asymmetric divisions in plants, it is too early to tie these advances together into a unifying model. It is

exciting to see an emerging theme involving receptor-like proteins implicated in perception or amplification of positional cues to polarize cells for asymmetric division and some hints as to what might lie downstream of these receptors, but in no case has a complete pathway from receptor-ligand interaction to cell polarization been mapped out. With the exception of POL and PLL1, no division-polarizing or -orienting factor has yet been shown to operate in more than one cell type. A key question that has yet to be addressed in any system is how premitotic cell polarity specifies the location of the PPB or premitotic nucleus. Thus, this field is wide open for breakthroughs to be made, and we look forward to witnessing them in the years to come.

SUMMARY POINTS

1. In most plant cells, the division plane is established prior to mitosis via a preprophase band (PPB) of microtubules and actin filaments, which forms in G2, persists throughout prophase, and disassembles upon entry into metaphase.
2. In symmetrically dividing cells, the location of the PPB is governed by mother cell shape and the position of the premitotic nucleus. In asymmetrically dividing cells, actin-dependent cell polarity appears to override these influences to direct the asymmetric placement of the PPB and the premitotic nucleus.
3. A large number of PPB-associated proteins have been identified. Those known to promote PPB formation include regulators of microtubule dynamics, PP2A phosphatase regulatory subunits, and proteins related to those found in animal centrosomes.
4. The PPB appears to function in two ways to orient division planes. First, it promotes the timely formation of a bipolar mitotic spindle oriented perpendicular to the plane of the PPB. Second, it supports the formation of a cortical division site (CDS) that is left behind upon PPB disassembly. The CDS guides phragmoplast expansion and cell plate attachment.
5. The CDS is marked negatively throughout mitosis and cytokinesis by local exclusion of F-actin and a kinesin-like protein KCA1 and positively by TANGLED and RAN-GAP1. How these markers may participate in guidance of the phragmoplast and attachment of the cell plate is not known.
6. In fucoid algae, polarization of the zygote requires actin and can be oriented by environmental cues in an auxin-dependent manner. Polarity is amplified by polarized vesicle trafficking and ion gradients and becomes fixed via a mechanism requiring actin and the cell wall. Microtubule-mediated interaction between the dividing nucleus and cortex orients the asymmetric division plane relative to the zygotic axis of polarity.
7. A variety of transcription factors act in the *A. thaliana* root meristem to control the occurrence and orientation of asymmetric divisions, but how these transcription factors influence cell division planes is unknown.
8. A pair of plasma membrane–localized PP2C phosphatases is required to polarize certain asymmetric divisions in the *A. thaliana* embryo. Asymmetric activity of these phosphatases has been proposed to result from a receptor-ligand interaction occurring on one face of the mother cell.

9. Receptor-like proteins are implicated in the polarization of cell divisions in the procambium of *A. thaliana* and in stomatal lineage cells in both maize and *A. thaliana*. A novel protein, BASL, acts downstream of receptor signaling to polarize stomatal divisions in *A. thaliana* by an unknown mechanism.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

The authors' research in the subject area of this review has been funded most recently by NSF grants IOB-0544226 and IOS-0843704, and by USDA NRICGP grant 2006-35304-17342. CGR also gratefully acknowledges the American Cancer Society for Postdoctoral Fellowship Grant #PF-08-280-01. We thank Sherryl Bisgrove (Simon Fraser University) for the drawings used in **Figure 2**.

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Annual Review of Plant Biology

Volume 62, 2011

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