Division decisions and the spatial regulation of cytokinesis
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Cytokinesis in plant cells is accomplished when a membranous cell plate is guided to a pre-established division site. The orientation of the new wall establishes the starting position of a cell in a growing tissue, but the impact of this position on future development varies. Recently, proteins have been identified that participate in forming, stabilizing and guiding the cell plate to the correct division site. Mutations that affect cytokinesis with varying impacts on plant development are providing information about the mechanics of cytokinesis and also about how the division site is selected.

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Abbreviations
dcd             discordia
KCBP            kinesin-like calmodulin binding protein
KATAp           kinesin from Arabidopsis thaliana A peptide
MAPK            mitogen-activated protein kinase
MMK3            Medicago mitogen-activated protein kinase 3
MT              microtubule
NPK1            Nicotiana protein kinase 1
PP              phragmoplast
PPB             preprophase band
TKRP125         tobacco kinesin-related polypeptide of 125 kDa
wty1            warty1

Introduction
Plant cell division serves a dual function of increasing cell number and also positioning new cross walls during cytokinesis. Once the new wall is formed, cells acquire a final shape by differential cell expansion. Several important decisions are thus made during division and expansion, including when and how often division should occur, where a new cell wall should be located, and in what direction to expand. The outcome of any one of these decisions could be critical to the successful development of an organ because new daughter cells are joined from inception by cell walls. In reality, however, the position of new walls has a variable influence on the shape of an organ or function of a tissue. This is because the spatial regulation of cytokinesis, which determines where new walls will be positioned, affects development to different degrees [1]. Regardless of the impact of division pattern on development, successful cytokinesis is needed to maintain the integrity of the plant body. Understanding how and when cytokinesis occurs will help to evaluate the effect of different stages of development on its spatial regulation.

Cytokinesis is accomplished by progressive deposition of membranes and associated wall synthesizing compounds into a cell plate, which is first located centrally in the equatorial zone between recently formed daughter nuclei [2–5]. The growing cell plate then spreads out radially, as an expanding interlacing membranous network, until it joins with the parental walls at the periphery. A cytoskeletal structure called a phragmoplast (PP) not only guides vesicles to the equatorial zone but also directs the growing cell plate toward a specific site at or near the membrane of the parental cell wall — the cortical division site. This ill-defined site is first evident prior to mitosis when the preprophase band (PPB) appears transiently as a continuous band of cytoskeletal elements in the cell cortex. Remarkably, the position of the PPB forecasts the future site of attachment between the cell plate and the parental cell walls. The integrated behavior of these cytoskeletal structures thus accomplishes the feat of depositing a new wall at a specific position. What is the mechanism of cell plate formation? What determines where this new wall will form? How does cell position affect future development? This review presents recent progress in answering these questions and also evaluates potential versatility in the process, considering that some divisions have more impact on future development than others. Recent advances in understanding cell cycle regulation [6], mechanisms of mitosis [7] or cell expansion [8,9 (D Cosgrove, pp 73–78)] are relevant topics, but are not covered here. Excellent diagrams showing the detailed structure of the growing cell plate and PP are available elsewhere [2,4,5].

Forming the cell plate
Completion of cytokinesis requires the interaction of fusion secretory vesicles, dynamic microtubules (MTs), actin and numerous associated proteins (Figure 1), as confirmed by electron and immunofluorescence microscopy and by experimental manipulation of cytokinesis. Disruption of the Golgi complex with the inhibitor brefeldin A, for example, prevents completion of the cell plate, suggesting that new vesicles are Golgi-derived [10]. Vesicles will also not accumulate in the equatorial zone of cells treated with inhibitors that depolymerize MTs [11], as a result of either transport inhibition or altered organization of the Golgi complex [12]. On the other hand, taxol, which hyperstabilizes MTs by preventing their depolymerization, increases the number of vesicles in the equatorial zone initially, although cytokinesis is eventually arrested [13]. These results demonstrate that MTs must be present but not necessarily dynamic for successful vesicle transport, supporting the idea that vesicles migrate along MTs with the aid of motor proteins [14].

Vesicle transport
The exact mechanism of vesicle transport is still an open question. The bipolar MTs in the PP are arranged with their plus ends overlapping in the equatorial zone and
minus ends directed toward the daughter nuclei (Figure 1), suggesting vesicles might be transported using a plus-end directed motor protein [5]. A candidate kinesin-related protein isolated from tobacco PPs (TKRP125) has the expected domains for motor activity [15]. The protein localizes, however, along the entire length of MTs in the PP, rather than in the punctate patterns expected for vesicle association. TKRP125 could still interact with other as yet unidentified proteins associated with vesicles. In permeabilized cells, however, antibodies against TKRP125 actually inhibit plus-end MT translocation [15]. Together, these results suggest that TKRP125 might contribute to maintaining the structure of the MTs in the PP, rather than transporting vesicles to the cell plate.

Minus-end directed kinesin-like proteins have been found associated with the PP, including kinesin-like calmodulin binding protein (KCBP) and a kinesin from Arabidopsis thaliana A peptide (KatAp). KCBP mediates MT translocation in vitro [16] and its motor domain binds MTs [17*]. KCBP localizes to the MTs in the PP and also to the nuclear membrane early in telophase and within the equatorial zone [18*]. KatAp is initially present in the spindle mid-zone before the PP is organized and then in association with the PP itself [19]. The localization patterns for both proteins are consistent with a function in organizing the MTs in the PP, rather than in directing vesicle transport [18*,19], particularly as similar kinesins in other systems help to assemble and organize the mitotic spindle [20]. The translocation of MTs toward the minus end could propel vesicles toward the plus ends. Most evidence, however, points to an organizational function for these proteins, perhaps mediated by these minus-end directed motors.

F-actin could be a candidate for vesicle motility because it co-localizes with MTs in the PP, and is oriented as expected for a motility system [21]. Myosin may also be present in the PP [22]. Although disruption of actin filaments with cytochalasin does not affect vesicle accumulation, it does disorient the cell plate [11]. F-actin could facilitate vesicle transport through stabilizing the MTs, as discussed below but whether it plays a direct role in vesicle transport is not clear.

Organizing the phragmoplast
MT translocation might help to establish and then maintain the bipolar MT array of the PP [23], possibly mediated by the kinesin-related proteins discussed above. Differential MT depolymerization is also important in PP function [10]. Centrifugal growth of the cell plate is prevented when MTs are stabilized, mirroring an effect of preventing depolymerization on spindle function [7]. These studies point to a required balance between polymerization at the leading edge and depolymerization at the trailing edge of the PP. It is likely that similar organizational mechanisms are found for the PP as found for other MT structures, such as the mitotic spindle.

Vesicle accretion and fusion in the cell plate
Newly arriving vesicles coalesce into a membranous network as the cell plate extends out radially [4]. Clathrin-coated vesicles are observed in conjunction with the growing tubulo-vesicular network of the cell plate, providing evidence for fusion and membrane recycling as part of cell plate...
formation [4]. A protein that probably facilitates this process is phragmoplastin, a dynamin-related protein originally isolated from soybean [24] and also from Arabidopsis (called Arabidopsis dynamin-like protein 1 [ADL1]) [25]. In animal systems, dynamin is a GTPase that functions in endocytosis, particularly in membrane separation [26]. Both phragmoplastin and ADL1 localize to the growing cell plate [24,27]. Phragmoplastin is not dependent on MT organization, as it remains at the cell plate margins after the PP has dissociated near the end of cytokinesis. Taxol-stabilization of MTs does not eliminate phragmoplastin from its earliest position in the equatorial zone but does prevent its redistribution to the leading edge [24]. In vivo observations using green-fluorescent protein (GFP)-fusion proteins show that phragmoplastin associates with sites of high membrane activity [28], consistent with a dynamin-like role for the protein. Many essential protein functions are regulated by phosphorylation, including dynamin assembly and kinesin activity [29,30]. Recently, mitogen-activated protein kinases (MAPKs) were found in the cell plate. MAPKs are cell-cycle regulated proteins involved in organizing the cytoskeleton and are also abundant in diverse signaling pathways [31,32]. Two proteins similar to MAPKs, (p43Ntf6 from tobacco and Medicago mitogen-activated protein kinase 3 [MMK3] from alfalfa) are activated during late anaphase if functional MTs are present [33*,34*]. MTs are not required to maintain distribution of MMK3 in the cell plate once the PP is established [34*]. Another protein, Nicotiana protein kinase 1 (NPK1) is similar to upstream kinases in a MAPK chain, referred to as MAPK kinase kinases [35]. NPK1 is expressed in dividing cells [36*] and complements yeast mutations in homologs to NPK1 [35], but its localization patterns relative to the cell plate have not yet been reported. Pending these results, NPK1 in conjunction with p43Ntf6 and MMK3 could be candidate transducers in a phosphorylation cascade in the cell plate.

Proteins that participate in vesicle targeting are also found in the cell plate. One example is the syntaxin-related protein KNOLLE [27,37]. Syntaxins (also called t-SNARES) are located in target membranes and specifically interact with proteins in arriving vesicles, called v-SNARES [2,38]. Syntaxins in nonplant systems participate in vesicle fusion during cytokinesis [39]. Localization of KNOLLE to vesicles and to the equatorial zone suggests it might serve similar recognition and docking functions in the growing cell plate [27,37]. Mutations in KNOLLE cause abnormal cytokinesis consistent with this role: the cell plate is often incomplete, malformed or even absent in mutant cells [37]. In nonplant systems, interactions between target membrane and vesicle proteins are mediated by other soluble proteins, including Vaccinia virus complement control protein (VCP) and its homolog in yeast, Cdc48p [40,41]. An Arabidopsis homolog to Cdc48p (AtCDC48) is present in dividing plant cells, localizes to the PP area and is able to complement yeast cdc48 mutations [42].

A multi-step process of membrane targeting must be present in plant cytokinesis: target membranes must be first established before the arriving vesicles can be docked. Next, the cell plate membrane becomes a functional plasma membrane, which may occur after cytokinesis is complete. It is interesting that a multi-step process is described ultrastructurally [4], is demonstrated in inhibitor studies [28,43] and is also postulated on the basis of the distribution of typical plasma membrane proteins such as...
ATPase, which is absent in the forming cell plate [27]. It will be interesting to find out precisely where and when KNOLLE, for example, is localized in relation to other proteins potentially involved in targeting, such as AtCDC48.

**Guiding the cell plate to the division site**

**The nature of the ‘correct site’ at the parental cell wall**

To accomplish cytokinesis, the PP has to lead the forming cell plate to the right place at about the right time. This intriguing process involves interaction among dynamic MTs of the PP, actin filaments and the cortical division site near the parental cell. The nature of this site is still a mystery. Both MTs and actin filaments are present and are required to mark the correct site prior to mitosis when the transient PPB forms [2]. Disassembly of the PPB can be accelerated by injection of CDC2, suggesting cell cycle progression may participate in marking the site [44]. Several hypotheses could explain how the cortical division site is marked. One hypothesis is that marker proteins are phosphorylated by CDC2 [45]. Alternatively, a localization of ion pumps at the membrane site has been proposed [46].

Another intriguing idea is that the site could be specified by the absence, rather than the presence, of a marker protein. It is interesting that actin remains in the cortex throughout division except at the division site, a position referred to as the actin-depleted zone (Figure 1; [47]). This hypothesis suggests that the absence of scaffolding in the division site balances the PP against the extensive actin scaffolding elsewhere in the cell. Other than the actin-depleted zone, no persistent structural or biochemical changes have been detected in the cortical division site.

**Selecting the division site**

An explanation is still needed for how the site is chosen, even if specific markers are eventually identified. The division site could indeed be established earlier than just prior to mitosis. For example, the site for any single new cross wall could be influenced by the size and shape of the parent cell and neighboring cells [48,49]. Change in cell wall characteristics could be induced by local strains, caused by differential expansion among interconnected cells [48]. The new division site would then be selected in response to the changed physical parameters of the tissue. Another related view is that the division site is selected on the basis of geometrically correct positions with the cell [50]. Such physical explanations could signal a cell to divide in a particular location, but the biochemical responses elicited by the signal remain unknown.

Spatial signals of cytokinesis were studied recently in onion root cells [51*]. When division is temporarily disrupted by caffeine, the resulting binucleate cells will eventually adjust and begin dividing again [51*]. Enlarged binucleate cells generally produced a single PPB at a site predicted from the size and shape of the cell prior to the treatment. These cells would then continue to divide with altered spindle and PP alignments, frequently not related to the most recent PPB position. The results contradict other cases, however, in which the PPB is absolutely required for correct wall placement, such as when a mutant lacks the PPB [52] or when PPs adjust to PPB position after normal spindle rotations [53]. In the experimentally induced binucleate cells, PP position may be signaled by the nucleus or alternatively, by older signals remaining from a prior division. These observations help define the normal parameters of site selection but we clearly do not know all the molecules involved in selecting the division site.

A genetic approach may help explain how the division site is selected. The mutation *warty1* (*wty1*) is providing information about cell sizes that constrain normal cytokinesis [54*]. The *wty1* mutants have cytokinesis defects restricted to groups of cells, producing warts in the leaf blade (Figure 2). Cell sizes can be measured in developing warts near the base of the maize leaf primordium, because of the graduated balance between division and expansion (Figure 3). Epidermal cells in a developing wart initially appear normal with respect to size (Figure 3) and in distribution of cytoskeletal arrays (AW Sylvester, unpublished data). Mutant cells that begin to...
exceed a constant aspect ratio (length to width ratio) at a given position in the primordium will eventually be unable to complete cytokinesis (Figure 3). One explanation for these observations is that over-enlargement prevents normal cytokinesis, possibly by destroying spatial signals within the cell. In these cells, expansion may be prolonged due to an inability to enter mitosis, as wart cells also have endoreduplicated nuclei with condensed chromosomes (AW Sylvester, unpublished data). Apparently, PP are able to form in warts but appear to lack directional information. Ongoing analysis of the gene and its product will help clarify the role of WTY1 in site selection.

Stabilizing and guiding the phragmoplast
The same proteins may be involved in selecting and also guiding the PP to the division site. For example, actin patches were observed in the actin-depleted zone in dividing cells [47]. Actin filaments emanating from the PP could attach to these patches left behind when the PPB disappears [47,55]. The PP could thus be tethered to the division site by the same proteins that originally established the site. Injected profilin, which effectively binds monomeric actin, disrupts cytokinesis, potentially by abolishing an actin-based mechanical support to the forming cell plate [55]. Similarly, caffeine, which is thought to inhibit cytokinesis by disrupting necessary Ca2+ gradients, degrades actin filaments at the leading edge of the cell plate [43]. Consequently, the cell plate arrives at the wrong location, if at all, and is often incomplete. It is reasonable to conclude that one role of actin is in the guidance of the PP to the division site [55].

The PP and cell plate may also be stabilized internally by actin. In Clivia endosperm cells, actin filaments are initially interspersed among spindle MTs but soon begin to shorten and polymerize in the equatorial zone, eventually interdigitating among the MTs of the PP (Figure 1; [56•]). These shorter actin filaments are also associated with a vinculin-like protein [56•]. Vinculin is an actin-associated protein known to moderate actin–membrane connections [57]. It is possible, therefore, that actin and associated proteins, such as vinculin, could be binding the vesicular network into a stable structure, while also guiding it to the division site.

Analysis of the discordia (dcd) mutations dcd1 and dcd2 may lead us to the molecules that participate in PP guidance [58••]. The dcd mutations cause abnormal growth of cells that undergo asymmetric cell division in the leaf epidermis, such as the subsidiary cells in the stomatal complex. Disruption of cell shape is preceded by improperly positioned cell walls, caused by misguided PPs. Actin disruption by cytochalasin phenocopies the mutant defect and the mutant phenotype is also exacerbated by cytochalasin treatment. These results suggest that DCD may function in either residual marking of the division site and/or in successful guidance of the PP to the site. Even if actin proves to be a structural guide, other proteins may participate in the process. Clues are coming from another maize gene, tangled1 (tan1) [59,60••]. Mutant tan1 leaves have disorganized cells compared with the well-oriented cells in the normal maize leaf. Cell disorganization is attributed partly to the inability of mutant cells to divide in a particular longitudinal orientation (parallel to the leaf axis). Mutant tan1 cells also lack the normal distribution of longitudinally oriented PPB, demonstrating that the protein is directly or indirectly required for specifying the longitudinal division plane. Furthermore, TAN1 appears to be necessary for proper PP guidance: all PPs in the mutant are slightly disoriented compared with those in normal cells, including those that are presumed to give rise to transversely oriented cell walls. These results are particularly intriguing because they point to the potential for post-cytokinesis adjustments in wall position: cells that have slightly oblique PPs may expand differentially and very slightly, thereby adjusting the new cross wall to a more typical transverse orientation. Preliminary reports suggest the gene may encode a protein with hydrophobicity characteristics of a cell-wall protein [61]. Completed analysis of the gene and its protein will be very interesting, as will an evaluation of the localization of TAN1 relative to PP and PPBs.

Impact of division ‘decisions’ on the plant body
Division orientations of individual cells vary in their impact on the plant body. For example, morphogenesis of some leaves does not require carefully orchestrated cell divisions [1,59]. On the other hand, some roots have stereotyped division patterns that result in proper disposition of cell layers, required for the acquisition of cell identities [62]. The impact of cytokinesis defects depends on the organ, the developmental stage and the nature of the gene involved. Currently, the phenotypes of mutants that affect cytokinesis reflect this variability. Some of the genes identified are probably redundant, others are directly involved in the mechanics of cytokinesis and others may play a more indirect role in division and expansion. The remainder of this review will evaluate these mutants based on the severity of their phenotype.

Fatal divisions
Many mutants with defects in cytokinesis were identified in genetic screens of seedling-lethal embryo-defective mutants [63]. The cyd1 mutant of Arabidopsis has defective cross walls and excessive accumulation of callose, evident early in embryogenesis [64••]. Interfering with cellulose synthesis can phenocopy the mutant, suggesting the gene plays a role in wall production during cytokinesis [64••]. The well-characterized knolle mutation affects body organization of the Arabidopsis embryo and specifically alters the forming cell plate. As discussed earlier, KNOILLE must be one of the key players in successful cytokinesis, probably through a syntaxin-like role in vesicle targeting. The keule mutant of Arabidopsis also lacks normal cytokinesis [65] as does the cyd mutant of pea [66]. In these cases,
cross walls are initiated but not completed, mimicking the effect of caffeine on cytokinesis.

The *gnom*/*emb30* mutation appears to affect all aspects of cell division and expansion. Most *gnom* mutant cells are able to complete cytokinesis normally, but are disoriented from the first zygotic division [67]. The EMB30/GNOM protein shares a domain with a secretory protein in yeast called Sec7p [68]. Other regions of the protein are similar to a non-essential yeast protein Yec2p [67]. Although the precise role of the protein is not known, the pleiotropic effects of the mutation may be due to generalized defects in secretion or vesicle function, rather than just secretion during cell plate formation.

Differentiation of specific cell types sometimes requires a highly regulated pattern of division [1,69]. Mistakes frequently cannot be tolerated in these types of differentiation divisions. One example is in the required asymmetric division that yields polarized cells. Pollen grains will differentiate appropriately only if the first mitotic cross wall is deposited asymmetrically. Incomplete cytokinesis, as seen in *gem1* mutants, disrupts differentiation of the vegetative and generative cells [70•]. Similarly, asymmetric cell division precedes differentiation of the layered *Arabidopsis* root, which is prevented in *ser* mutants [71]. In this case, the division alteration is more likely due to a response to a change in positional information rather than a direct effect on cytokinesis. Even so, mutations such as *ser* may provide information about how these signals are read by cells, an important aspect of the spatial regulation of division [69].

**Near fatal divisions**

Drastic alterations in cell division may have only partial effects on development. For example, the *fass* mutants in *Arabidopsis* have defects in cytokinesis that result in abnormally shaped cells [72]. Organ morphogenesis is also severely altered, with plants acquiring a short stubby appearance, but tissues are distributed normally. These mutants also lack the normal PPB, suggesting the gene may be involved in selecting and/or marking the division site [52]. The *tan1*/*fass* mutants, among others, show that tight spatial regulation of cytokinesis is not necessary for basic tissue differentiation.

Division effects may vary based on whether a gene is required throughout development or in all cells. The *tos1* mutants, for example, show cytokinesis defects only in floral organs and ovules [73,74]. In floral meristems and sepals, cross walls are incomplete and nuclei endoreduplicated, similar to the cytokinetic defects in plants with *knolle*, *keule*, *cyl* and *wty1* mutations. Developmental arrest of ovules in *tos1* mutants appears to be due to abnormal and uncoordinated cell expansion, a good example of the same gene being required for division or expansion, depending on the organ [73]. Similarly, *wty1* mutants show altered cytokinesis, but only in leaf blade cells [54•]. If these are all null alleles, organ specificity of mutant defects suggests either redundant functions of the genes in other organs or that the specific controls of division may vary in different organs.

**Spatially adjustable divisions**

Several mutants have problems with cytokinesis but show quite normal development. Loss of spatial information by altered cytokinesis may be corrected in a growing organ by adjusting the direction or rate of cell expansion or increasing the rate of cell division. For example, *tan1* mutants have normally shaped leaves and organs, despite incorrectly placed cross walls. Cells in *tan1* mutants that are experimentally forced to expand more, by growing in dark or low-light conditions, show corrected orientations of cell walls (M Mitkovski, AW Sylvester, unpublished data). Cell adjustments are also seen in *wty1* mutants: improper growth of warts is balanced by neighboring cells, which divide more as the leaf is growing [54•]. This neighborly behavior of cells is consistent with the idea that the emerging organ shape prevails as a signal for the spatial regulation of cell division during morphogenesis of leaves. The idea requires that cells respond together and communicate locally, via regulated plasmodesmatal trafficking [75] or other means of intercellular communication. MTs and cellulose microfibrils, which together direct oriented cell expansion, are observed to be co-aligned in cell groups [49,60•,76], consistent with other observations that neighboring cells grow in coordinated patterns [49]. Mutants, such as *tan1* and *wty1*, provide information about the capacity of cells to adjust within a given framework. Such adjustments suggest that the selection of the division site itself may be flexibly regulated.

**Conclusions**

The steps of cytokinesis are integrated so that a growing cell plate is guided to a pre-established division site. Several proteins are known that probably contribute to the membranous construction of the cell plate, including phragmoplastin and KNOLLE. Potential phosphorylation requirements in the cell plate will be understood as more kinases are identified. Also, other proteins that might be involved specifically in vesicle targeting, such as CDC48, will provide information about the interactions between soluble and membrane-bound proteins. There are several phases in the construction and growth of the cell plate. In the future, it will be interesting to understand how these vesicle proteins may behave uniquely in the cell plate.

Two major cytoskeletal components are required for successful cytokinesis — MTs and actin. The relationship of the arrays to the forming cell plate has been clarified experimentally and by cytological observations. Kinesin-related proteins that participate in maintaining the structure and function of the PP are also being identified. Motorized transport along MTs is still the best hypothesis for how vesicles get to the cell plate but, as yet, proteins that mediate this process have not been definitively identified. Actin
is also likely to be responsible for unifying the cell plate and for providing internal as well as lateral support for the PP. Actin may well serve as a scaffolding upon which the PP is poised as it extends radially to the division site. The mechanism of guiding the PP to its division site is just beginning to unfold, as new mutants are found that influence this process. Mutations such as dcd1 and dcd2 are likely to provide key information about PP guidance.

One outcome of cytokinesis is that two daughter cells are initially positioned relative to neighbors and to the original parent cell. On the basis of mutant phenotypes, the impact of this division ‘decision’ on subsequent development depends on the developmental stage, the type of protein required, or on the extent of genetic redundancy. Some mutants, such as tan1 and wty1 can adjust for cytokinesis defects, presumably by expanding or dividing differently to compensate for the loss of spatially correct information. In other cases, a correct cell division orientation may be required prior to a critical differentiation step, so that a spatially defective cytokinesis cannot be tolerated, as in the gem1 mutant. Finally, seedling-lethal mutations, such as cyt1, knolle, keule, cyd may cause the loss of some essential function in cytokinesis itself. While the mechanics of cytokinesis is probably shared for different cells in different organs, it is possible that the means of selecting the division site may prove to be variable. In the long term, the question of how a division site is selected should be considered in the context of the entire growing tissue. The interconnections of plant cells suggest that any single division ‘decision’ influences neighboring cells biochemically and ultimately physically. Continued characterization of mutants, and molecular analysis of the genes involved, will help to clarify both the mechanics and the determinants of cytokinesis.

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References and recommended reading
Papers of particular interest, published within the annual period of review, have been highlighted as:
* of special interest
** of outstanding interest


52. Binucleate wheat cells are produced by caffeine treatment and subsequent patterns of phragmoplast arrangement are recorded in cells of different sizes. The authors conclude that PP could form without respect to the PPB in some cases, suggesting pre-existing spatial cues may dictate cell wall position under certain circumstances.


56. Cells with wty1 mutations undergo faulty cytokinesis once the cells have begun to expand-over. The results suggest that only one spatial cue for the division site may be appropriate length to width or volume ratio in the cell. In addition, the neighboring cells in the wty1 mutant, which can look normal, appear to adjust to the defect by dividing more.


59. The association of actin and an associated vinculin-like protein is described in endosperm cells of Ovulis. Short actin filaments are seen in the center of the PP and co-localize with vinculin. The authors suggest the proteins are important in maintaining and perhaps guiding the PP during cytokinesis.


62. These mutants show an interesting and informative phenotype. Asymmetric divisions that occur during differentiation of the epidermis are affected so that cytokinesis is disrupted and cells expand in abnormal orientations. Normally positioned PPB are present, predicting the correct site for the impending new wall. PP localize to the site, however, suggesting that they are not guided properly or that residual information marking the site is lost. Particularly intriguing is the fact that actin filament disruption can phenocopy the mutants. The results implicate the genes in a PP guidance process that may require actin or associated proteins.


65. Stunning views of the distribution of MTs and actin filaments in tan1 and non-mutant leaf primordia are presented. Detailed analysis of cytoskeletal arrays in mutants compared with non-mutants confirm that longitudinal divisions are specifically disrupted in the mutant. PPB do not occupy the expected longitudinally oriented position. PP formations are also slightly disoriented compared with normal.
Growth and development


64. Nickle TC, Meinke DW: A cytokinesis-defective mutant of Arabidopsis (cyt1) characterized by embryonic lethality, incomplete cell walls, and excessive callose accumulation. Plant J 1998, 15:321-332. The cyt1 mutant shows lethal defects in cytokinesis that result in abnormal cross-walls and excessive accumulation of callose – a component of the forming cell plate. Cellulose synthesis disruption phenocopies the mutant defect, suggesting the gene may be involved in regulating the transition from cytokinesis to cell wall synthesis.


70. Park SK, Howden R, Twel D: The Arabidopsis thaliana gametophytic mutation gemini pollen1 disrupts microspore polarity, division asymmetry and pollen cell fate. Development 1998, 125:3789-3799. The gem1 mutation is an example of a differentiation process that requires the correct spatial information of an asymmetric division. Cytokinesis is disrupted in the developing pollen grain so that essential asymmetries are not carried forward to permit specification of the vegetative and generative cells.


