Bridging the divide between cytokinesis and cell expansion

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Two of the most fundamental processes in plant development are cytokinesis, by which new cells are formed, and cell expansion, by which existing cells grow and establish their functional morphology. In this review we summarize recent progress in understanding the pathways necessary for cytokinesis and cell expansion, including the role of the cytoskeleton, cell wall biogenesis, and membrane trafficking. Here, we focus on genes and lipids that are involved in both cytokinesis and cell expansion and bridge the divide between these two processes. In addition, we discuss our understanding of and controversies surrounding the role of endocytosis in both of these processes.

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Introduction

The *de novo* creation of plasma membrane and cell wall at the end of mitosis in plant cells requires a dynamic cytoskeletal array called the phragmoplast that directs vast movement of material, including lipids, proteins, and cell wall components, to and from the division plane to assemble the cytokinetic organelle known as the cell plate. The stages of somatic cell plate development include (1) creation of the phragmoplast from mitotic spindle remnants; (2) trafficking of vesicles to the division plane and their fusion to generate a tubular–vesicular network; (3) continued fusion of membrane tubules and their transformation into membrane sheets upon the deposition of callose, followed by deposition and organization of cellulose and other cell wall components; (4) recycling of excess membrane and other material from the cell plate; and (5) fusion with the parental cell wall. These events are accompanied by the reorganization of other endomembrane systems including the ER. Although morphologically very different, there are many parallels between cell plate development and the process of cell expansion, which involves the addition of membrane to an existing plasma membrane and reorganization of the cell wall. It has become clear that these two processes have many similarities at a mechanistic level, with many of the same pathways and often even the same proteins being involved in both (reviewed in reference [1]). Here, we review recent progress in understanding cell expansion and cell plate formation, and the many new links between these two processes (Table 1).

The role of the plant cytoskeleton in cytokinesis and cell expansion

The dynamic organization of the plant cell actin and microtubule (MT) cytoskeletons is essential for the formation of the cell plate and plasma membrane dynamics. Cytoskeletal structure is largely regulated by cytoskeletal-interacting proteins known as actin-binding proteins (ABPs) or microtubule-associated proteins (MAPs). These binding proteins regulate the assembly and disassembly of these polymers, determining length, stability, and organization. Some of these interacting proteins have been shown to have crucial roles in cytokinesis and cell expansion.

Microtubules

MICROTUBULE ORGANIZATION1 (MOR1) is a member of the highly conserved MAP215/DIS1 family of MAPs [2], which promote tubulin polymerization *in vitro* and regulate MT length by promoting dynamic instability. Arabidopsis *mor1* mutants are severely stunted with short, radially swollen organs, hallmarks of defective polar growth. Temperature-sensitive *mor1* mutants display defects in cortical MT arrays after 1.5 h at the restrictive temperature of 29 °C [2]. After 24–48 h at 30 °C, spindles and phragmoplasts are misaligned, discontinuous, branched, crooked, and contain short and aberrantly organized MTs, resulting in the formation of incomplete cell walls (cell wall stubs) and internal cell wall inclusions, indicative of defects in cell plate biogenesis [3•,4•]. The malformed *mor1* spindles and phragmoplasts persist longer than those observed in wild-type cells, delaying progression of cell division, and many of the cells enter M phase without ever forming a preprophase band (PPB), while other cells contained a mislocalized or underdeveloped PPB-like structure [4•]. The *gemini pollen1* (*gem1*) mutant, which displays altered cell division symmetry and ectopic cell plate growth in haploid gametophyte development resulting in a lack of proper microspore cell polarity establishment and subsequent aberrant cell fate [5–7], is an

allele of *mor1*, which produces truncated MOR1 protein [8].

Immunolocalization of MOR1/GEM1 revealed that MOR1 colocalizes with cortical MTs in interphase cells and with PPBs, spindle MTs, and phragmoplasts [8]. An antibody against the N-terminus of MOR1 [4•] revealed that MOR1 localizes along the entire length of MTs at all stages of the cell cycle, even in *mor1-1* cells after exposure to 29 °C for 24–48 h. These data agree with previous evidence of strong colocalization of tubulin with MAP200, the tobacco homolog of MOR1, along PPBs, spindles, and phragmoplasts in BY2 cells [9]. The defects observed in *mor1* morphology and the coincident mis-organization of MTs, as well as colocalization of MOR1 with cytokinesis-specific MT structures, underline the importance of MT organization to proper cell expansion and cytokinesis.

Actin

Several ABPs have been recently shown to play roles in cell expansion, particularly polar cell growth [10–14], and to regulate cell plate guidance, mutants of which cause misaligned crosswalls between daughter cells [15–17]. However, only one class of ABPs, the formins, has been implicated to be directly involved in cell plate formation. Formins act as positive regulators of actin polymerization by promoting actin monomer release from the G-actin-sequestering protein profilin and subsequent nucleation of unbranched actin filaments (reviewed in reference [18]). The Arabidopsis formins AFH1 and AtFH8 play a role in polar cell expansion of pollen tubes and root hairs, respectively, while AtFH5 plays a role in the timing and rate of cytokinesis [19–22].

Overexpression of AFH1 in pollen tubes reveals that proper regulation of AFH1 activity at the surface of an actively growing cell is required to maintain tip-focused polar cell expansion. In wild-type pollen tubes and root hairs, actin cables are seen in the shank of the pollen tube and terminate in the subapical region, while a fine actin meshwork is present at the tip. Overexpression of the formin homology 1 (FH1) and formin homology 2 (FH2) domains of AFH1 results in actin cable formation throughout the length of the pollen tube. Broadening of the pollen tube, loss of tip-focused growth, and severe membrane deformation at the tip are also observed in the overexpression mutants [19].

Table 1

Selected mutants with abnormal cell expansion and cytokinesis defects

Gene	Mutation	Cell expansion defects	Cytokinesis defects	References
Microtubules				
GEM1/MOR1	TS	Short, radially swollen organs; abarrently organized MTs	Misaligned phragmoplasts; incomplete cell walls	[2,3•,4•]
	Truncation	N/A	Ectopic cell plate growth	[5,6,8]
Actin				
AFH1	OE	Swollen pollen tube tips	N/A	[19]
AtFH8	OE	Swollen root hair tips	N/A	[22]
	DN	Growth arrest of root hairs	N/A	[20]
AtFH5	KO	ND	Slower/delayed cytokinesis	[21]
Cell wall				
VGD1	KO	Slow growth; pollen tubes rupture	ND	[26]
Membrane trafficking				
DRP1A	KO	Non-polar stigmatic papillae growth	ND	[50]
DRP1A/DRP1E	KO	Unexpanded embryonic cells	Cell plate stubs in embryo	[51]
DRP1C	KO	Hydrated pollen PM invaginations	N/A	[51]
TPLATE	KO	Hydrated pollen PM invaginations	N/A	[52•]
	RNAi	Organ swelling	Fusion w/parental PM	[52•]
PIK4β1	KO	Abnormal root hair morphology	Occasional cell wall stubs	[56], Kang and Nielsen, personal communication
Other/unknown				
elch	KO	Clustered trichomes	Multinucleate cells; cell wall stubs	[43••]
sfh1	KO	Shorter, branched root hairs	N/A	[58]
srd		Shorter organs; swollen root	Supernumerary root cortex cells	[63]
scd1	TS	Shorter organs	Multinucleate stomata and pavement cells w/cell wall stubs	[64]
	KO	Shorter organs; less lobed epidermal pavement cells; trichomes rupture	Multinucleate stomata and pavement cells w/cell wall stubs; branched root hairs	[64]
korrigan	TS	Shorter, swollen organs	N/A	[65]

Abbreviations: TS, temperature sensitive; OE, overexpression; DN, dominant negative; KO, knockout; RNAi, RNA-mediated interference; PM, plasma membrane; ND, none detected.

Overexpression of AtFH8 results in similar changes in root hair morphogenesis. In weak AtFH8 overexpression lines, actin cables extend to the extreme tip of root hairs, resulting in short, wavy, or swollen root hairs. In lines with higher expression, actin organizes into an irregular mesh resulting in root hairs with branched tips or bulb-shaped bases, or multiple hairs originating from the same cell [22]. By contrast, Arabidopsis plants expressing an inducible dominant-negative AtFH8 lacking its FH2 domain display growth arrest of root hairs upon induction [20].

The Arabidopsis formin, AtFH5, functions in cytokinesis. A constitutively expressed GFP fusion to AtFH5 is localized at the division zone in dividing Arabidopsis root cells and exhibits strong fluorescence as the cell plate expands and gradually disappears after the cell plate fuses with the parent cell wall. Insertional T-DNA *atfh5* knockout lines show delays in cellularization and a slowing of cell plate formation [21]. The combined evidence of a role for AtFH5 in cytokinesis, and AFH1 and AtFH8 in polar cell expansion shows the essential role of actin regulation by formins in both processes.

Cell wall biogenesis during cytokinesis and cell expansion

The processes of cytokinesis and cell expansion both require the addition of new cell wall materials to impart mechanical strength to and dictate the shape of the underlying cellular membrane. The plant primary cell wall is a complex and heavily cross-linked polysaccharide made up of crystalline cellulose microfibrils within a matrix of hemicelluloses and pectins [23]. Regulated pectin cross-linking functions to allow cellulose microfibril separation during cell growth, and subsequently, to affix cells in place once growth has ceased [23]. Pectins are synthesized as neutral methylesters and secreted into the cell wall where they must be de-esterified by an enzyme called pectin methylesterase (PME), which exposes the acidic carboxyl groups making them available for calcium or boron cross-linking.

Recent studies have demonstrated that pectins are important to both cytokinesis and cell expansion. In lily and tobacco, exogenously applied PME was found to induce cell wall thickening and inhibition of growth at the apical tip of pollen tubes. A tobacco pollen-specific PME, NtPPME1, expressed as a C-terminal GFP fusion to the prepro-NtPPME1, and methylesterified pectins, are detected at the apical tip of pollen tubes, while de-esterified pectins and cell wall thickening are detected along the flanks [24]. Likewise, the apical tip of growing *Solanum chacoense* pollen tubes has the greatest concentration of methylesterified pectin with an increase in de-esterified pectin along the axis of the pollen tube. This apical–distal gradient correlates with an increase in rigidity and a decrease in visco-elasticity along the length of the pollen tube [25].

Pollen of the Arabidopsis *vanguard1* (*vgd1*) PME knockout mutant produces unstable pollen tubes that burst when germinated *in vitro*. During fertilization *vgd1* pollen tubes grow more slowly down the style and do not reach the terminal length of wild-type pollen tubes when germinated on the stigma [26]. This is also observed in knockout mutants of another pollen-specific PME, AtPPME1. Supporting the importance of PME activity at the cell wall of the growing pollen tube, functional YFP-AtPPME1 protein fusions show fluorescence at the tip and along the periphery of the pollen tube, as well as in internal structures believed to be Golgi and/or ER [27], and a C-terminal GFP VGD1 fusion protein localizes to the cell wall region in plasmolyzed pollen tubes [26].

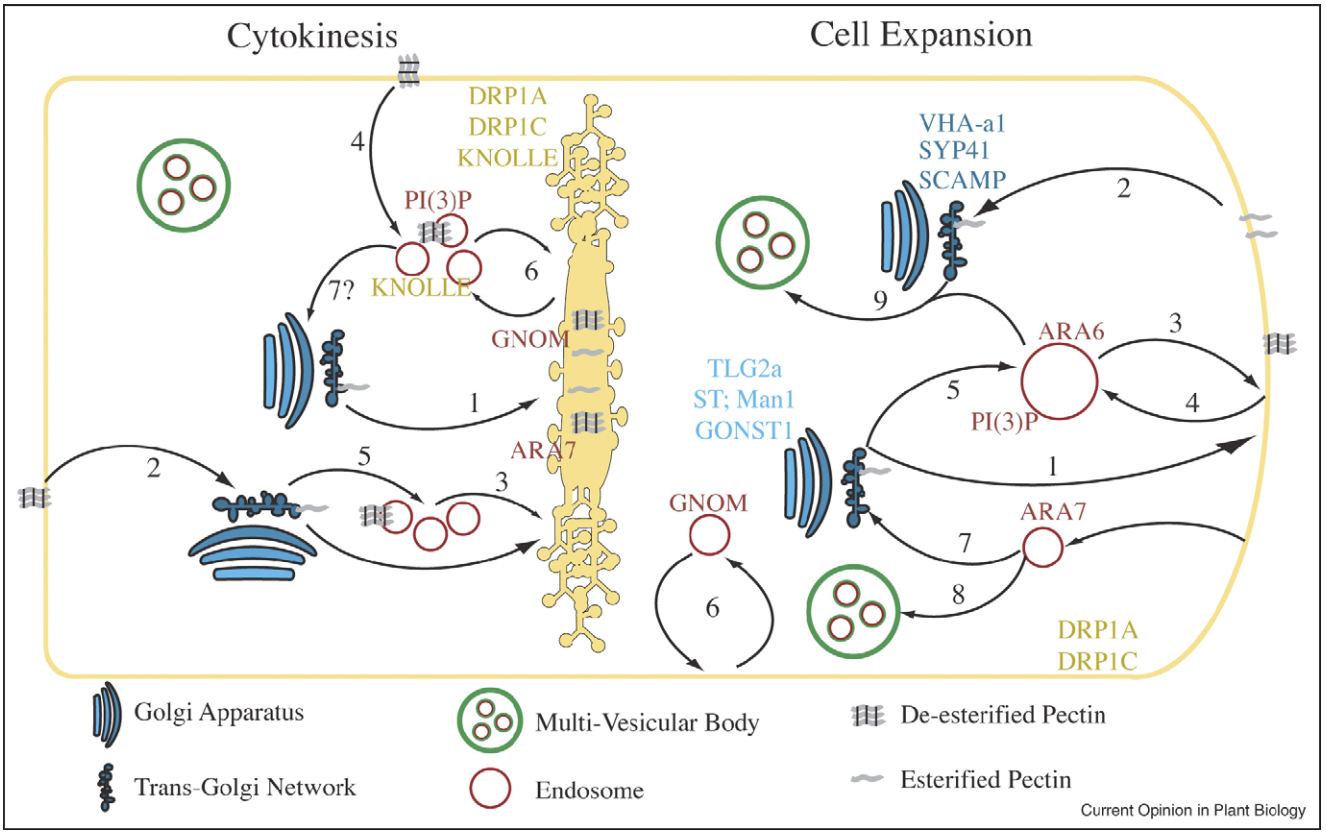
Pectins are also abundant in the forming cell plate, although PMEs that function at the cell plate remain to be identified. In dividing and expanding maize root cells [28•], as well as in dividing tobacco BY2 suspension-cultured cells [29••], immuno-electron microscopic analysis has shown the accumulation of arabinan and homogalacturonan and homogalacturanan and rhamnogalacturonan pectins, respectively, in the cell plate. Interestingly, antibodies reactive to de-esterified pectin label not only the mature cell wall but also the cell plate, even in its early stages, as well as multivesicular bodies/endosomes. By contrast, antibodies to methylesterified pectins, which strongly label the Golgi, show only weak reactivity at the cell plate. This has led to the suggestion, as illustrated in Figure 1, that the majority of pectins in the forming cell plates are derived from a mature cell wall via endocytosis, as discussed below [28•,29••].

Membrane trafficking during cytokinesis and cell expansion

The conventional theory of secretory activity in cell plate construction and cell expansion is that Golgi-derived vesicles are delivered toward the site of secretion via the cytoskeleton and fuse with other vesicles or membrane tubules with the aid of the exocyst complex and other fusion factors. Exocyst-like structures were observed in tomographic reconstruction of the cell plate [30]. Several exocyst mutants have growth defects in polar cell types [31,32], and although no cytokinetic defects have been reported in those mutants, it is hypothesized that the identification of additional exocyst mutants will uncover cell plate defects.

Morphological analysis over the past decade has suggested that the primary trafficking to the cell plate is via the Golgi biosynthetic pathway. However, several different groups have assembled evidence suggesting that endocytic trafficking is directed to the cell plate in dividing cells. Proposed membrane-trafficking pathways during interphase and cytokinesis are illustrated in Figure 1.

Figure 1



Schematic of membrane-trafficking pathways implicated in cell expansion and cell plate development. The biosynthetic secretion pathway of *trans*-Golgi network (TGN) to plasma membrane **(1)** contributes to anisotropic cell expansion and has been thought to be the main pathway of membrane delivery to the cell plate on the basis of EM studies. Although uncharacterized in plants, a Golgi to endosome secretion pathway **(5)** cannot be ruled out. Recent studies suggest that the TGN may also function as an endocytic organelle **(2)** in interphase. Endocytosis via the endosomes **(4)** or the TGN **(2)** has recently been introduced as another source for cell plate membrane on the basis of plasma membrane protein localization and cell wall component recycling. Because endocytosed cell wall components and the plasma membrane auxin carrier PIN1 have been visualized at the cell plate, it is possible that recycling functions proposed for ARA6 positive **(3)** and GNOM positive **(6)** endosomes during cell expansion may also function during cell plate development. Endosome to Golgi retrograde trafficking has been proposed **(7)** via the retromer complex (of which GRIP is a component), but it is not clear if this also occurs during cytokinesis. During interphase, much of the endocytic cargo is eventually targeted to the multivesicular body **(8)** for subsequent delivery to, and destruction in, the vacuole. In light of the recent evidence for an endocytic role in cell plate development, the regulation of this pathway may prove vital for proper cell division. See text for further details and references.

The lipophilic endocytic tracer FM4-64 is incorporated into developing cell plates [29••,33•,34], suggesting that endocytosed material is delivered to initiating and expanding cell plates. Questions remain however, regarding the mechanism of FM4-64 transport considering the vast interconnectivity of the plant endomembrane system. More convincing than the trafficking of FM4-64 is the observation that pectins and xyloglucans that previously resided in cell walls based on their cross-linking composition have been detected at the cell plate [28•]. It is hypothesized that endosomes, and not Golgi, are the source of the cross-linked cell wall components [28•,29••]. It is unclear, however, whether Golgi-derived pectins can be cross-linked in a developing cell plate or other membrane compartments. Dhonukshe *et al.* [29••] estimated that endocytic traffic doubles in dividing cells and found that the same plasma membrane proteins that were shown to be endocytosed by clathrin-dependent endocytosis [35] are also present at very young, developing cell plates [29••]. In addition, endosomal markers (GFP-ARA7 and GNOM-myc) and not Golgi markers (ST-YFP and TLG2a) are significantly found at the division zone [29••]. A note of caution should be added as most of these GFP-tagged markers were constitutively or heterologously expressed and their localization may not truly represent native localization. The large amounts of membrane trafficking to the cell plate may cause many overexpressed proteins to become mis-localized to the cell plate.

On the basis of these data, the authors suggest that endosomes organize around the cell plate, confirming

what was observed with the endosomal-resident lipid PI(3)P, and that cell plate formation is initiated by fusion of endosomes at the division plane. However, endosomes have not been detected in electron tomographs of the early stages of cell plate formation in somatic, endosperm, or pollen cytokinesis [30,36–38]. Instead, a cloud of vesicles of the typical diameter of post-Golgi anterograde traffic is seen [30,36,37,39]. Multivesicular bodies (MVBs), a late compartment in the endocytic pathway, were detected around the cell plate by electron microscopy in later stages of its development, but not until the maturation phase when there is substantial membrane recycling, at which point the total number of MVBs in the cell quadruples [38,39]. By contrast, the number of Golgi stacks doubles before G2 phase and are concentrated around the cell plate in cytokinesis [38]. Interestingly, there is also a change in vacuole morphology and a reduction of vacuole surface area by half in the late stages of cytokinesis [38]. Nonetheless, the data presented by Dhonukshe *et al.* [29••] do support the notion that endocytic membrane and cargo can become integrated into the cell plate. In this case, one would suspect that the regulatory mechanisms that sort to the degradation and recycling pathways may be superseded or shut down during cytokinesis. A next step might be to look for cytokinesis-specific signals that could mediate this redirection.

A clue may come from MVBs, now thought to be equivalent to prevacuolar compartments (PVCs) [40]. Endocytic proteins targeted for degradation or newly synthesized vacuolar-resident proteins are ubiquitinated and recognized by an ESCRT-I complex, after which the vesicle fuses with MVBs with help from the ESCRT-II and ESCRT-III complexes (reviewed in references [41,42]). A mutant in an Arabidopsis homolog of a subunit of the ESCRT-I complex that binds ubiquitin, *elch*, exhibits multinucleated cells with incomplete cell walls, albeit at very low frequency, a hallmark of cytokinetic defects [43••]. Presumably ELCH recognizes a protein targeted for degradation. In light of the idea that endocytic cargo can reach the cell plate (Figure 1), it is possible that a protein targeted for degradation is mis-targeted to the cell plate, causing the cytokinetic defects in the *elch* mutant. Epistatic interactions with a weak allele of tubulin-folding cofactor A (*kiesel*) suggest that ELCH may act through microtubules. *elc|kis-T1* double mutants have smaller cells and an increased number of multinucleated cells than the single mutants alone, suggesting the genes may operate in the same pathway. It will be interesting to determine the targets of ELCH during both interphase and cytokinesis.

Another issue that complicates the debate between exocytic and endocytic pathways during cytokinesis and cell expansion is the recent controversy over the nature of the *trans*-Golgi network (TGN) in plants. Two recent reports concerning the localization of TGN-resident proteins and endocytic cargo have suggested that the TGN may function as an early endosome. The V-ATPase, VHA-a1 [33•], and TGN-localized SNARE protein SYP41 [44] do not colocalize with Golgi or PVC but do aggregate when cells are treated with Brefeldin A, a characteristic of Golgi membranes [45]. Interestingly, endocytic trafficking was blocked when the activity of the VHA isoforms were inhibited [33•]. Subsequently, a rice homolog of the endocytosis-mediating protein, Secretory CArrier Membrane Proteins, SCAMP1, that was also found to reside in VHA-a1 and SYP41 positive structures had a localization separate from that of the *cis*-Golgi-resident protein GONST1-YFP, and *trans*-Golgi-resident protein Mannosidase1-GFP markers [46•]. This suggests that the TGN in plants may be able to dissociate from the rest of the Golgi. In a time course assay, FM4-64 labeled the VHA-a1 positive TGN structures earlier than the ARA6 or ARA7 positive endosomes, suggesting that the TGN may also act as an early endosome [33•]. If the TGN is involved in both anterograde and endocytic trafficking, as the above studies suggest, the observations of PM and endocytic-resident proteins appearing early at the cell plate [29••] are not surprising, as the TGN is at the intersection of these pathways. The crucial step is to fully define the nature and function of the TGN and to determine if the localization of VHA-a1, SYP41, and SCAMP is truly at the TGN and not in an uncharacterized intermediate recycling compartment between the TGN and early endosomes.

Membrane recycling and phosphoinositides in cytokinesis and cell expansion

Endocytosis plays an essential role in cytokinesis and cell expansion regardless of whether or not the PM is a source of new membrane material for the growing cell plate because of the need for large-scale membrane recycling during the maturation of the newly formed membrane. An estimated 70% and 80% of membrane delivered to the cell plate [36] and growing pollen tubes [47], respectively, is recycled. Much of the machinery in membrane recycling – and endocytosis in plants in general – has not been determined in plants, but clathrin has been detected both at the cell plate [39] and in pollen tubes [48], and studies of cell expansion mutants have begun to implicate additional proteins [49–51]. Membrane recycling and endocytosis also require lipid determinants, as does exocytosis, and recent work focusing on phosphorylated phosphoinositides (PIPs) has begun to elucidate their role in cytokinesis and cell expansion as well.

Several mutants with altered membrane morphology characterized by large plasma membrane invaginations, such as *tplate* [52•] and the dynamins, *drp1A* [50] and *drp1C* [51], fail to undergo proper expansion in developing pollen (*tplate* and *drp1C*) or stigmatic papillae (*drp1A*). Although the roles these proteins play in endocytosis or other membrane dynamics have not been elucidated, the mutants' defects in plasma membrane morphology

suggest an absolute requirement for endocytosis in cell expansion. DRP1A has also been shown to interact with VAN3, a small GTPase-activating protein involved in post-Golgi trafficking, and to cooperate with VAN3 in vascular patterning [53].

GFP fusion proteins of DRP1A and TPLATE localize not only to regions of active cell expansion but also to the cell plate [49,52•]. DRP1A is thought to function during the early stages of cytokinesis and localizes to the growing edge of the cell plate, whereas TPLATE is involved in positioning and fusion of the maturing cell plate with the parental plasma membrane, and localizes to both the cell plate and the site of fusion [30,49,52•].

Additional evidence that membrane recycling plays a crucial role in cell expansion is the localization of PI(3)P, which has been studied throughout the cell cycle using a YFP-2xFYVE reporter [54]. It is found primarily in rapidly moving vesicles dispersed throughout the cytoplasm, most of which colocalize with the endosomal/prevacuolar marker mRFP-AtRABF2b. These PI(3)P-labeled vesicles are enriched at the tips of growing root hairs, consistent with the reported localization of other endosomal markers [55], and are also clustered around the growing edges of the forming cell plate, although the cell plate itself is not PI(3)P enriched. If these PI(3)P-labeled vesicles at the division plane are indeed endosomes, this is further support for the involvement of endocytic structures in cytokinesis, whether purely in recycling or possibly as a direct source of material for the newly forming membrane.

Other phosphoinositides play a crucial role in cell expansion, although their roles remain to be determined. PI(4)P, when visualized either by antibodies or an eYFP-hFAPP1 reporter, is also found at the tip and, to a lesser extent, the sides of growing root hairs (Nielsen, personal communication). PI-4 kinase β1 (PI4Kβ1) has been shown to be an effector of the small GTPase RABA4b and resides with it on a compartment at the tips of root hairs that appears by immuno-electron microscopy to be TGN [56,57••]. PIK4β1 mutants have aberrant root hair morphology, and show a disorganized TGN at the root hair tip, suggesting a role for PI(4)P in exocytic vesicle trafficking during polar cell expansion [57••]. These mutants also have mild defects in cytokinesis (Kang and Nielsen, personal communication) suggesting that PI(4)P may also be involved in membrane trafficking at the forming cell plate, although the localization of PI(4)P during cytokinesis has not been studied.

$PI(4,5)P_2$ also localizes strongly to the tip of growing root hairs, and in a spiral pattern along the flanks [58]. Proper PI(4,5)P localization is essential for proper root hair growth, and its disruption leads to disorganization of the actin cytoskeleton [58,59] that is thought to be important for both endocytosis and exocytosis at the growing tip. A direct role for $PI(4,5)P_2$ in endocytosis, exocytosis, or cytokinesis, such as it plays in animal systems [60,61], has not yet been demonstrated.

One protein that regulates $PI(4,5)P_2$ localization at the tip of growing root hairs is a SEC14-type PI-transfer protein, AtSFH1. Root hairs from a *sfh1* null mutant have reduced tip-localization of $PI(4,5)P_2$ and are shorter, multiply branched, and deformed [58]. Recently, another SEC14-type protein, PATELLIN1, was shown to localize to the maturing cell plate [62]. PATELLIN1 fractionates as a peripheral membrane protein and binds PI(5)P and $PI(4,5)P_2$ *in vitro*, although its effects on cellular PI levels or distribution are unknown. The localization of PATELLIN1 to maturing sections of the forming cell plate suggests it as a possible link between $PI(4,5)P_2$ or PI(5)P and membrane recycling during cell plate maturation, although $PI(4,5)P_2$ has not been visualized directly during cytokinesis.

The Arabidopsis genome encodes ~30 additional SEC14 domain-containing proteins, most of which are uncharacterized, but which probably also play a role in PI regulation. The further study of these and other lipid-interacting protein families, along with the use of new and existing reporters to characterize PI distributions in all parts of the cell throughout the cell cycle and especially during cytokinesis, should shed further light on the mechanisms of membrane recycling and dynamics during cell expansion and cell plate maturation and probably highlight further similarities between these two processes.

Conclusion

Over the past few years, research from many laboratories has continued to support the notion that cell expansion and cell plate development utilize similar pathways, and we expect future studies to provide even more detail about both the protein and lipid determinants of these pathways. Things to look for in the coming years include the *short root and dwarfism* mutant, a currently unmapped mutant with cell expansion and cytokinesis defects [63], as well as further work on the known expansion and cytokinesis mutants *stomatal cytokinesis defective 1* (*scd1*) [64] and *korrigan* [65]. Also, while ROP GTPases are receiving extensive attention in their roles in polar cell expansion in root hairs and epidermal pavement cells (reviewed in reference [66]), there has not been any recent study of ROPs in cytokinesis, even though ROP4 had previously been reported to localize to the cell plate in BY2 cells [67]. As live cell imaging, large-scale protein interaction studies, and genetic technologies become ever more sophisticated, the roles of these and other proteins will be understood in greater detail and the similarities and differences between cytokinesis and cell expansion will be even more clearly defined.

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