Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Plant Biology

Endomembrane trafficking overarching cell plate formation

Joanna Boruc[1,2] and Daniel Van Damme[1,2]

By contrast to other eukaryotic kingdoms, plant cytokinesis is an inside-out process. A coordinated action of cytoskeletal transitions and endomembrane trafficking events builds a novel membrane compartment, the cell plate. Deposition of cell wall polymers transforms the lumen of this membrane compartment into a new cross wall, physically separating the daughter cells. The characterization of tethering complexes acting at discrete phases during cell plate formation and upstream of vesicle fusion events, the presence of modulators directing secretion and recycling during cytokinesis, as well as the identification and temporal recruitment of the endocytic machinery, provides a starting point to dissect the transitions in endomembrane trafficking which shape this process. This review aims to integrate recent findings on endomembrane trafficking events which spatio-temporally act to construct the cell plate.

Addresses
[1] Department of Plant Systems Biology, VIB, B-9052 Ghent, Belgium
[2] Department of Plant Biotechnology and Bioinformatics, Ghent University, B-9052 Ghent, Belgium

Corresponding author: Van Damme, Daniel (daniel.vandamme@psb-vib.ugent.be)

Current Opinion in Plant Biology 2015, **28**:92–98

This review comes from a themed issue on **Cell biology**

Edited by **Hiroo Fukuda** and **Zhenbiao Yang**

For a complete overview see the Issue and the Editorial

Available online 24th October 2015

http://dx.doi.org/10.1016/j.pbi.2015.09.007

Introduction

Cytokinesis is the final phase of cell division which, via a multi-stage process, physically divides daughter nuclei. In plants, this involves both the biogenesis of a transient membrane compartment as well as its transition into a plasma membrane (PM) and a mature cross wall. This review positions recent findings regarding vesicle trafficking events throughout the different stages of cell plate formation (Figure 1).

Actively guided transport and fusion of vesicles at the equator of the cell builds the cell plate. Vesicle guidance occurs via the phragmoplast, which evolves out of the anaphase spindle and consists of two parallel-oriented microtubule arrays that initially centrally interdigitate at their plus ends and which are stabilized by microtubule-bundling proteins (Figure 1a) [1–4]. Vesicle transport along the phragmoplast microtubules is believed to occur via kinesin motors. Although AtPAKRP2 has been proposed as a candidate to fulfil this task in *Arabidopsis* [5], its function might, however, not be conserved within the plant kingdom, based on localization data and functional analysis in moss [6,7•]. Next to vesicle fusion, membrane recycling from the cell plate via clathrin-mediated endocytosis (CME) shapes it to its final composition [8], but membrane removal by CME is apparently not essential for cell plate expansion [9].

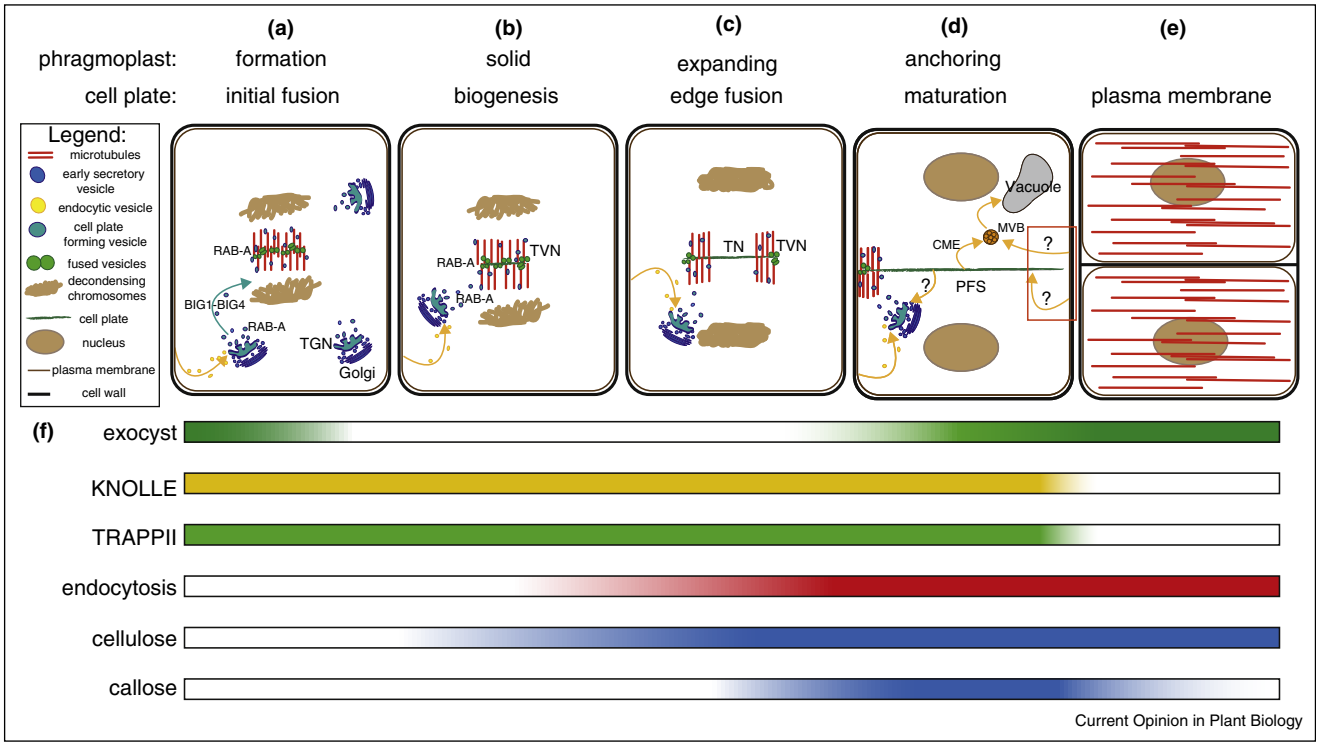
Sequential changes in polysaccharide composition are also hallmarks of cell plate progression (reviewed in [10]). Callose, as the predominant cell plate polysaccharide, has long been proposed to stabilize and aid the expansion of the cell plate tubules (Figure 1c,d) [11,12]. Indeed, mutants and compounds which interfere with callose biosynthesis lead to cytokinesis defects [13–18]. As the defects observed in the absence of callose, however, largely concern anchoring deficiencies following cell plate expansion, callose probably predominantly functions during the transformation of the cell plate into a cross wall, in agreement with its late appearance [17].

The hypothesis that cellulose is synthesized only during consolidation of the cell plate [11] was recently challenged. Primary cell wall cellulose synthase (CESA) subunits accumulated together with early cytokinesis markers and were active before the expansion stage (Figure 1b,c) [19•]. CESA intensities decreasing at the center of the cell plate during the expansion phase intuitively fit best with cellulose deposition occurring immediately after the recruitment of the CESA complexes to the cell plate. The apparent discrepancy regarding the timing of cellulose deposition and the functional relevance of its early deposition with respect to callose, as well as with cellulose synthesis during cross wall formation, leaves room for further studies.

Initiating cell plate formation

The inability of plant cells to constrict their PM necessitates de novo cell plate biogenesis from spatially restricted vesicle fusion events, which are tightly coordinated with cell cycle progression. Both the temporal and spatial restrictions are established by the transition of the anaphase spindle into the phragmoplast, which ensures the spatio-temporal accumulation of cell

Figure 1



Vesicle trafficking during consecutive stages of cell plate formation. **(a)** Cell plate initiation stage when TGN-derived late secretory vesicles migrate along the phragmoplast towards the cell equator. In this phase BIG1-BIG4 ARF GEFs are required for trafficking of both endocytosed (yellow arrow) and newly synthesized proteins to the forming cell plate (blue arrow representing targeting of cell plate-forming vesicles). Both EXOCYST and TRAPPII-dependent tethering aid initial vesicle fusion. **(b)** Continued vesicle fusion facilitated by TRAPPII, RAB-A GTPases and dynamin-based restriction leads to the formation of a tubulovesicular network (TVN). **(c)** The early cell plate starts to expand by microtubule depolymerization at the center and polymerization at the periphery, directing vesicle fusion at the edges to drive cell plate expansion. Microtubule depolymerization at the center also coincides with maturation of the central part of the cell plate, the TVN cell plate at the center undergoes a morphological change and forms a tubular network (TN). **(d)** Further membrane remodeling including removal of excess material at the center via clathrin-mediated endocytosis (CME) transforms the TN to a planar fenestrated sheet (PFS). The orange rectangle at the right side of the cell indicates a spot where phragmoplast microtubules were removed for clarity. This is the cortical division zone where CME is likely to cause membrane remodeling before anchoring and recycling and/or degradation of proteins diffusing from the cell plate upon anchoring (yellow arrows with question marks). **(e)** Upon transformation of the cell plate into a cross wall, daughter cells are separated. (F) Visual representation of the timing of the action of tethering complexes (EXOCYST and TRAPPII, green), SNARE-dependent vesicle fusion (KNOLLE, yellow), endocytosis (CME, red) and polysaccharide deposition (cellulose and callose, blue) throughout the cell plate formation stages. By contrast to TRAPPII-dependent tethering, KNOLLE-syntaxin-dependent fusion and callose deposition, EXOCYST-dependent secretion, CME and cellulose deposition continue following cytokinesis.

plate-forming vesicles. Initial vesicle coalescence and dynamin-mediated restriction creates a dumbbell-shaped tubulo-vesicular membrane network (TVN), the early cell plate (Figure 1a,b).

SNARE-dependent fusion of cell plate-forming vesicles is preceded by vesicle tethering. SNARE function during cytokinesis, the interplay between the cytokinesis-specific syntaxin KNOLLE [20], the Sec1/Munc18 protein KEULE which stabilizes its open conformation [21] and the other SNARE proteins driving vesicle fusion in complex with KNOLLE, was reviewed recently [22]. Tethering complexes such as EXOCYST [23•] and TRAPPII [24,25,26••,27] function upstream of SNARE-dependent vesicle fusion and provide spatial specificity in concert with RAB-A GTPases [25,28,29]. The EXOCYST tethering complex likely participates in the initiation of cell plate formation due to (1) the prominent association of several subunits during the earliest phases of cell plate formation [23•,26••,30], (2) the fact that EXOCYST-shaped complexes were observed in electron tomograms connecting unfused vesicles [12] and (3) the formation of aberrantly fused initial cell plate membranes in the exo70A1 mutant [23•]. The exact role of the exocyst complex in cell plate initiation remains however unclear as these initial defects are somehow overcome. By contrast to the EXOCYST, the TRAPPII complex labels the cell plate from the onset throughout cytokinesis and is required for its biogenesis [26••] (Figure 1f).