In somatic cell division, cytokinesis is the final step of the cell cycle and physically divides the mother cytoplasm into two daughter cells. In the meiotic cell division, however, pollen mother cells (PMCs) undergo two successive nuclear divisions without an intervening S-phase and consequently generate four haploid daughter nuclei out of one parental cell. In line with this, the physical separation of meiotic nuclei does not follow the conventional cytokinesis pathway, but instead is mediated by alternative processes, including polar-based phragmoplast outgrowth and RMA-mediated cell wall positioning. In this review, we outline the different cytological mechanisms of cell plate formation operating in different types of PMCs and additionally focus on some important features associated with meiotic cytokinesis, including cytoskeletal dynamics and callose deposition. We also provide an up-to-date overview of the main molecular actors involved in PMC wall formation and additionally highlight some recent advances on the effect of cold stress on meiotic cytokinesis in plants.

Cell Plate Formation in Plants

In mitotically dividing cells, separation of chromosomes (e.g., nuclear division) is followed by a physical separation of the two daughter nuclei by the establishment of a cell plate and/or cell wall (e.g., cell division). This process is generally termed cytokinesis and is considered an essential part of the mitotic cell cycle, more specifically as the final step of the mitotic M-phase. Indeed, loss of cell plate formation in most organisms typically leads to severe defects in cell proliferation and cell differentiation, mostly causing a premature abortion of the tissue or organ involved.1-3

Based on the different cell structure and morphology of various biological systems (e.g., rigid cell wall in plants does not occur in animal cells), the process of cytokinesis shows a large variability between the different kingdoms, with a strong difference between plants and other eukaryotic organisms.4,5 In animals and yeast, following nuclear division, cytokinesis is initiated at the periphery of the division plane with the specification of the cleavage plane and subsequent rearrangement of microtubule (MT) structures that form a structural basis for the formation of the actin-myosin-based contractile ring.6-9 This actomyosin ring contracts centripetally and mediates the ingestion of cleavage furrows, eventually forming a small intercellular bridge (1–1.5 μm) that contains a microtubular midbody. In the final step of cytokinesis, MT-mediated vesicle trafficking completes midzone assembly and forms an internuclear membrane, physically separating the two newly formed daughter cells.7,10-12

In contrast to the centripetal-directed furrow ingression observed in animals and yeast, cell plate formation in plant cells typically involves the establishment of a centrally initiated MT structure; the phragmoplast.13-16 The phragmoplast is composed of short, overlapping microtubules of opposite polarity formed at the center of the cell and centrifugally expands to mediate the deposition of membrane vesicles along a plane marked earlier by a cortical microtubule array; the preprophase band (PPB).17-19 Subsequent fusion of Golgi-derived vesicles and endosomes at the equatorial midzone of the phragmoplast generates a transient tubule-vesicular membrane, also called the cell plate, which grows out in a centrifugal direction to fuse with the parental plasma membrane at the cell periphery.18-20 In the final step, the intermediate cell plate matures and transforms into a rigid cell wall; a process which involves different actions, including the closure of plate fenestrae, incorporation of pectins and xyloglucans, removal of excess membrane and replacement of callose by cellulose.21-22

In the meiotic cell division, similar to the mitotic one, segregation of chromosomes (nuclear division) is always accompanied by the formation of a cell plate (cytokinesis). In plant male meiosis, however, two different types of cell plate formation are documented; namely successive and simultaneous cytokinesis. In the former mechanism each meiotic cell division is directly followed by a cytokinetic event. As such, a transitory dyad is generated after meiosis I and a tetrad is formed after meiosis II (Fig. 1). Inherent to this way of division, the tetrad figure in successive-type PMCs is typically constrained to a tetragonal (isobilateral) shape and sometimes to a T-shaped or linear shape,23,24 as for example observed in the Asparagales clade.25 In contrast, in the process of simultaneous cytokinesis, cell plate formation is uncoupled from chromosomal segregation and cytokinesis occurs when both meiotic divisions are finalized. So, in this mechanism, a double ‘perpendicular’ cell plate structure is generated at the end of meiosis II.26-27 Resulting tetrads are hereby much more variable in morphology (Fig. 1), displaying both tetragonal, rhomboidal and tetrahedral arrangements.25 In general, the successive-type of cytokinesis is typically observed in monocotyledonous PMCs, whereas the simultaneous-type is characteristic for dicotyledonous male meiocytes.28-30
Cytological Mechanisms of Male Meiotic Cell Plate Formation

From a mechanistic point of view, cell plate formation in successive-type male meiocytes follows a similar pattern as observed in “conventiona” cytokinesis. Indeed, by studying cytoskeletal rearrangements during meiotic telophase I in a number of monocotyledonous plant species, Shamina et al. (2007) demonstrated that the successive-type of meiotic cytokinesis is directed by a centrifugally expanding phragmoplast and that this mobile phragmoplast directly guides vesicle-mediated deposition of cell wall components to generate the cell plate. However, compared with normal “conventional” cell plate formation, meiotic cytokinesis displays some alterations, more specifically in the establishment and dynamics of the MT structures. First, the position of the meiotic cell plate is not predefined by a pre-prophase band, but instead is determined by the MI spindle structure, more specifically by the central spindle fibers. These MT fibers are installed at mid-prophase and, upon separation of homologous chromosomes, are not removed, but instead remain positioned at the meiocyte’s midzone. During MI cytokinesis these central spindle fibers form a phragmoplast-like MT structure that, supplemented with and spatially guided by polar MT bundles, surrounds the growing cell plate and mediates centrifugal cell wall expansion (Fig. 2). As such, Shamina et al. (2007) stated that the position of the MI cell plate depends on the spatial organization of the MI spindle apparatus and the localization of the MI nuclear poles. In support of this, the absence of a central bipolar spindle in maize de PMC (e.g., chaotic, monopolar and non-polar spindle figures) often results in a complete loss of meiotic cell plate formation, eventually yielding multinuclear monads that occasionally display randomly positioned cell wall fragments. Similarly, other defects in MI spindle formation, such as the unequal distribution of free MT bundles between the two MI half-spindles and alterations in anti-parallel spindle connection (e.g., the “disconnected” spindle phenotype in wide F1 hybrids) typically leads to MI cytokinetic abnormalities, including loss of cell wall establishment and the formation of short daughter membranes (e.g., incisions).

A second feature of successive PMC cytokinesis, not observed in conventional somatic cytokinesis, is the increased circumference (increased number of MTs) and progressive curvature of the phragmoplast MT fibers during centrifugal movement. This increased circumference and associated MT accumulation is not attributed to MT recycling within the phragmoplast, as observed in mitotic cells, but instead is caused by the polymerization of new MTs emanating from the polar regions of the telophase spindle structure. These newly formed MTs grow toward the equator, closely associating with the pre-existing central phragmoplast fibers, and connect with the opposite polar MTs to replicate the midzonal phragmoplast structure. This polar-based MT supply not only increases the cytoskeletal MT amount of the expanding phragmoplast, but also constitutes the basis for the C-curved...
shape of the phragmoplast edge fibers (Fig. 2). The antiparallel connection at the equator region combined with the progressive expansion of the polar MT fibers is hereby suggested to be the driving force inducing the enhanced curvature of the phragmoplast MT fibers.\(^{32}\)

Based on these unique features, the cytological mechanism underlying centrifugal movement of the phragmoplast in successive-type PMCs is thought to be differentially regulated as compared with mitotic cell division. In ‘conventional’ somatic cytokinesis, lateral phragmoplast expansion is mediated through dynamic MT fiber processing; e.g., by degrading centrally located MTs and emergence of new MTs at the external side (Fig. 2). This MT-driven lateral expansion is most likely guided by a pre-established cytoskeletal array, composed of actin fibers that connect the phragmoplast midzone with the cortical division plane.\(^{35}\) In meiotic cell division, however, no such actin structures are present\(^{36,37}\) and phragmoplast MTs do not show an outward polymerization-depolymerization process. Based on these findings, Shamina et al. (2007) speculated that centrifugal phragmoplast movement in successive-type PMCs is analogous to that of anaphase-B in animal cell division.\(^{32}\)

In contrast to the successive-type of cytokinesis, the simultaneous-type strongly differs from somatic cytokinesis as neither a preprophase band (PPB)\(^{33}\) nor a classical phragmoplast structure is formed.\(^{35,38-41}\) Moreover, in contrast to the centrifugally directed outgrowth of the cell plate in somatic and successive-type PMC cells, cell plates in these PMCs typically show an inward-oriented infurrowing of the callosic parental wall to partition the meiotic cytoplasm.\(^{30,31,39,42}\) This centripetal ingrowth process is mediated by and directed along phragmoplast-like MT structures, called radial microtubule arrays (RMAs), which physically demarcate the meiocyte’s nuclear cytoplasmic domains (NCDs) and define the position of the future cell wall.\(^{38,43}\) RMAs are formed by the interaction of actin filaments and microtubuli that emanate from the microtubule organizing centers (MTOCs) localized on the outer surface of the telophase II nuclei.\(^{41,44}\) Hence, subsequent formation of the meiotic cell plate is not fixed to pre-defined positions on the parental wall, but rather is imposed by the spatial

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**Figure 2.** Microtubule array formation and direction of cell wall formation in “conventional” cytokinesis and in male meiosis I and II of respectively successive and simultaneous type PMC cytokinesis. The newly formed cell plate and associated deposition of transient callose is presented in blue. Phragmoplast and RMA microtubule structures are indicated in green and polar MT bundles are shown in gray.
positioning of the nuclei at the end of MII.6,37,45,46 This is well demonstrated by several Arabidopsis meiotic mutants, in which post-meiotic cleavage plane formation is not constituted by a pre-defined conformation pattern, but instead always occurs in between the gametophytic nuclei formed,47 generating polyads instead of the normal tetrads. As such, the simultaneous-type of meiotic cytokinesis constitutes a special type of cell plate formation, in which the position of the cell plate is mediated by the location of the newly formed daughter nuclei and not through a predefined cytoskeletal imprinting (e.g., PPB). A similar mechanism of cell plate formation also occurs in the nuclear endosperm, more specifically at the moment of cellularization.35,48 Indeed, in nuclear endosperms, initial development is comprised of several successive cycles of nuclear division without cytokinesis, generating a syncytial group of nuclei that reposition to the cortical cytoplasm. Next, similar as in simultaneous-type PMCs, MT arrays radiate from MTOCs on the nuclear envelope and interdigitate at the equator regions, defining the cellular boundaries and NCDs of the syncytial nuclei.49 Cell plate formation mediated by these RMA structures then finally leads to the assembly of cell walls between both sister and non-sister nuclei (e.g., cellularization). Strikingly, in another syncytial plant organ, namely the female gametophyte (embryo sac with eight nuclei), ‘non-conventional’ RMA-mediated cell plate formation only occurs between non-sister nuclei, whereas a conventional cytokinesis event (spindle-derived interzonal MTs) occurs between sister chromatids.35,48

Thus, in a more general perspective, RMA-based cell plate formation in simultaneous-type PMCs is not a unique biological process, but instead can be considered as a general type of cytokinesis, specifically occurring in multinuclear coenocytic cells.

**Callose Deposition at the Meiotic Cell Plate**

A characteristic feature of meiotic cytokinesis is the abundant presence of callose. Although deposition of callose is also observed in mitotic cell division, more specifically as a transient intermediate in de novo cell plate formation,35,51 in meiotically dividing cells callose is placed both at the division site as well as at the outer cell wall.52 Outer PMC callose deposition initiates at prophase I and progressively continues during the meiotic cell cycle, resulting in a thick callosic cell wall at the end of MII.53 A similar callose deposition has also been observed in female meiocytes,54 suggesting that the callosic cell wall functions as a molecular filter to isolate developing PMCs and MMCs from the surrounding tissue, enabling the specific differentiation and programmed development of the enclosed meiocytes.55 In addition, meiotic deposition of callose is also essential for subsequent spore development, and more specifically for the establishment of a proper pollen cell wall.55 In male sporeogenesis, the outer callose wall of newly formed microspores constitutes a basic framework for the deposition of new cell wall components (e.g., sporopollenin) and, upon degradation, provides components essential for the synthesis of the outer pollen wall layer; e.g., the exine. In line with this, loss of meiotic callose deposition typically leads to an abortion of microspores and associated gametophytic sterility.56,58

Besides its functional role in microspore development, callose deposition at PMC cytokinesis also strongly influences pollen morphology and aperture pattern ontogeny.59,60 Albert et al. (2011) hereby found that PMC cytokinetic callose deposition is a two-step process, in which transient callosic cell plates are formed first, as an intermediate structure for cell wall formation, whereas callosic plugs are deposited later at specific places on the outer tetrad wall to specify the position and the pattern of the pollen apertures.61

**Molecular Regulation of PMC Cytokinesis**

In contrast to somatic cytokinesis, little is yet known about the molecular mechanism(s) underlying meiotic cell plate formation. Most cytokinesis-defective mutants (e.g., hyl1, knolle, kor, keule) are seedling-lethal, impeding the analysis of putative meiosis-specific functionalities.52,63 It is suggested that the construction of the cell wall in both mitotic and meiotic cells is dependent on or shares similar molecular mechanisms, however, both cytological and genetic studies in Arabidopsis have revealed some distinct alterations in molecular regulation. For example, immunocytological examination demonstrated that KNOLLE, a cytokinesis-specific syntaxin protein63 that normally localizes to the newly formed cell plate in mitotically dividing cells, is not required for male meiotic cell plate formation.64 As such, it was suggested that -at least partially- different cell plate forming mechanisms operate in both types of cell division.

Forward genetic screens in Arabidopsis have resulted in the isolation and characterization of several mutants showing clear defects in male meiotic cytokinesis; e.g., tes, stud, anq/mkk6 and mpk4.65-68 All these mutants show normal meiotic chromosome segregation but exhibit a complete failure of cytokinesis at the end of male meiosis and consequently generate large tetr-spores harboring four haploid nuclei.26 Molecular identification of tes and stud revealed that they are two allelic variants of the same gene, namely TES/STUD/AtNACK2. The corresponding protein has an N-terminal domain homologous to kinesin motor proteins and shares sequence similarity with a small number of plant kinesins, including the Arabidopsis HINKEL (HIK) protein and NACK1 and NACK2 from tobacco.66,69-71 Since all these proteins are involved in normal meiotic chromosome segregation, a cytokinesis-defective mutant of the same gene, namely TES/STUD/AtNACK2 was also isolated. As such, it was suggested that -at least partially- different cell plate forming mechanisms operate in both types of cell division.

Similar to HIK/AtNACK1, ANQ1/MKK6 and MPK4 are both components of the MAPK signaling cascade that regulates somatic cytokinesis in Arabidopsis. Indeed, using in vitro expression assays in yeast, Takahashi et al. (2010) demonstrated that cytokinesis is controlled by a pathway that consists of ANP MAPKKKs, that can be activated by HIK, and which in turn activate downstream ANQ1/MKK6 MAPK, with MPK4 being the presumed target of ANQ1/MKK652 (Fig. 3A). Consequently, functional loss of one of these proteins, such as MPK4 and ANQ1, causes severe defects in somatic cell plate formation and leads to dwarfish and stunted growth.73-75 In addition,
loss of MPK4 and ANQ1 in Arabidopsis also induces defects in meale meiotic cell plate formation, similar as observed in tes.67 As such, it is now generally conceived that TES, as a functional homolog of HIK, together with MPK4 and ANQ1/MKK6 constitute a similar MAPK signaling pathway that mediates post-meiotic RMA and cell plate formation in Arabidopsis74 (Fig. 3A).

The exact mechanism by which this MAPK signaling cascade initiates and regulates cell plate formation in PMCs is yet unknown. However, recent studies have demonstrated that the downstream MAPK actor MPK4 is able to phosphorylate three members of the microtubule-associated protein MAP65 family; e.g., MAP65-1, -2 and -3.68,76 Similarly, in the orthologous tobacco NACK-PQR pathway, the MPK4 ortholog NRK1/NTF6 was found to phosphorylate MAP65-1.77 MAP65s are MT-associated proteins that are involved in the dynamic organization and structural positioning of MTs at distinct sites during the cell cycle.79 More specifically, AtMAP65-1 promotes tubulin polymerization and microtubule nucleation79 and additionally drives microtubule cross-bridging between parallel or antiparallel aligned MT to induce the formation of large MT bundles, making them more resistant to cold and MT-destabilizing drugs.78,80-82 Similarly, MAP65-3 controls MT stabilization by selectively cross-linking antiparallel interdigitating microtubules (IMTs) toward their plus ends.83 For MAP65-2, less is known about the specific function in MT dynamics. GFP expression studies and genetic analysis in somatic cells demonstrated that both MAP65-1, -2 and -3 localize at the phragmoplast76,81,85 and are essential for phragmoplast formation and cell plate assembly.86 Although subcellular localization and functional assessment of these MAP65s has not been performed in developing PMCs, the strong homology with somatic cytokinesis suggests that MPK4 most likely controls meiotic cell plate formation through the phosphorylation of MAP65s. A more detailed mechanistic insight into MAPK-mediated cytokinesis comes from Sasabe et al. (2006) who found that phosphorylated NtMAP65-1 accumulates during the late M phase of the cell cycle (at the phragmoplast) and that phosphorylation of NtMAP65-1 by NRK1/NTF6 reduces its MT-bundling activity in vitro.77 Thus, in contrast to its presumed promotive function in MT bundling, it is now assumed that MAPK-mediated phosphorylation of MAP65-1 and other MAP65s enhances the destabilization and turnover of MTs at the equator of the phragmoplast, thereby mediating or facilitating the expansion of the phragmoplast at the end of the cell cycle (Fig. 3B).
Another protein involved in male meiotic RMA formation in Arabidopsis is SEPARASE (AESP). AESP is a caspase family protease that is required for the proteolytic cleavage of the cohesin complex at the metaphase-to-anaphase transition and the release of sister chromatid cohesion during meiosis and mitosis. Interestingly, besides a persisted chromatid cohesion and aberrant chromosome segregation, aep meiocytes (e.g., RDA1 and the conditional rsw4 mutant) also show clear alterations in telophase II RMA formation, including reduced MT extension, partial phragmoplast formation and complete loss of RMA establishment. In addition, aep PMCs often generate microspores with multiple nuclei, clearly indicating for a defect in meiotic cytokinesis. Since these alterations are not observed in other chromosome segregation-defective mutants (e.g., Arabidopsis syn1 and ask1 and maize dit), AESP is thought to play a pivotal role in PMC cytokinesis, and more specifically in the formation of the RMA. In addition, Yang et al (2011) speculated that AESP regulates the maintenance of PMC cell polarity, most presumably through the control of cyclin levels. However, a clear connection between AESP activity and meiotic RMA formation remains to be established.

Two proteins have been found to be specifically required for the deposition of callose at the developing cell plate in male meiotic cytokinesis: glucan synthase-like GSL1 and GSL5. Indeed, cytological analysis of double gsl1gsl1 gsl5+ and gsl1+ gsl5 gsl5 mutants not only revealed defects in pollen grain development, but additionally displayed loss of callose at newly formed PMC cell plates, without affecting the deposition of callose at the outer wall. Moreover, since the resulting microspores were occasionally larger and multinucleate, similar as observed in the cytokinesis-defective tes and mpk4, the deposition of callose at the male meiotic cell plate appears to be essential for normal PMC cytokinesis and haploid spore formation. Single gsl1 or gsl5 mutants, on the other hand, do not show any defect in meiotic callose deposition or microspore development. As such, Enns et al. (2005) concluded that GSL1 and GSL5 are both redundantly required for the deposition of callose at PMC cell plates and for the proper separation of the four daughter cytoplasmics.

Glucan synthase-like proteins, also termed callose synthases (CalSs) have been shown to interact with phragmoplastin, annexins, Rop1, sucrose synthase (SuSy) and UGT1 to form a functional complex that mediates callose deposition in a wide set of cell or organ types. Proteomic studies revealed that annexin-like proteins may modulate CalS activity whereas SuSy could be involved in providing primer substrate for callose synthesis. The Arabidopsis genome encodes 12 putative callose synthases (CalS1-12) and most have been found to mediate the deposition of callose in one or more specific organs or cell types; e.g., the cell plate, pollen tube, plasma membrane, plasmodesmata, etc. As such, GSL5 is not only required for the callosic cell wall in dividing PMCs but also mediates deposition of callose in developing pollen and wounded tissues (e.g., papillae). Moreover, GSL5 expression is highly induced in leaves subjected to wounding, pathogen infection and SA treatment.

Impact of Temperature Stress on PMC Cell Plate Formation

Recent publications have demonstrated that meiotic cell plate formation, both in successive- and simultaneous-type PMCs, is extremely sensitive to temperature stress, and more particularly to cold stress. Tang et al. (2011) for example found that cold-induced pollen lethality in a wheat thermo-sensitive genic male sterile (TGMS) line is caused by an abnormal separation of dyads during male meiosis I, indicating for a specific defect in male cytokinesis. Histological studies hereby revealed that low temperatures alter the formation of the MI phragmoplast, leading to severe defects in cell plate assembly and meiocyte development. A more in depth cytological examination demonstrated that cold stress specifically affects the typical Chinese lantern-shaped actin MI phragmoplast structure. In addition, transcriptome studies revealed that cold stress dramatically represses the expression of many structural cytoskeleton-associated genes, including profilin, ADF (actin-depolymerizing factor), myosin and formins. Hence, sensitivity of meiotic cytokinesis to low temperature stress appears to be mediated by transcriptomic alterations of genes that play key roles in the dynamic organization of the actin cytoskeleton. Similar to TGMS wheat, cold also induces defects in meiotic cell plate formation in the simultaneous-type PMCs of Arabidopsis. Cytological examination hereby revealed that short periods of low temperature stress specifically alter the formation the microtubular RMAs at telophase II and hence cause defects in meiotic cytokinesis and cell wall formation, yielding restituted meiocytes that contain bi- and multinucleate spores. However, in contrast to the TGMS wheat line, the Arabidopsis cold-induced multinucleate spores do not abort, but instead display nuclear fusion before pollen mitosis I (PMI), eventually generating diploid and polyploid pollen grains. Thus, in plants showing simultaneous type of meiotic cytokinesis, cold stress may lead to sexual polyploidization, yielding polyploid offspring with higher genetic plasticity and phenotypic adaptability to cope with adverse climate conditions. In support of this hypothesis, also in Brassica, a substantial increase in 2n male gamete formation was observed under low temperature conditions, but the mechanism by which these cold-induced gametes are formed is not yet known.

In contrast to temperature-affected MT figures in somatic cells, cold-affected RMA structures in Arabidopsis PMCs are not reassembled upon transfer to normal conditions, but instead persist and lead to defects in cell plate formation, similar as observed in the Arabidopsis mutants tes and mpk4. As such, one could presume that the cold-sensitivity of meiotic RMAs is not based upon physical or structural features, but instead is regulated on the molecular level, for example, through alterations in MAPK signaling. Initial studies hereby did not show any regulatory involvement of TES, ANQ, MPK4 or MKK2; a cold-induced kinase that positively regulates MPK4 activity. However, more in depth studies are needed to fully examine the molecular mechanism underlying the cold sensitivity of PMC cytokinesis.
Conclusions and Future Perspectives

The formation of the cell plate in plant male meiocytes is a unique biological process that shares both cytological and molecular features with somatic cytokinesis. The large variability in meiotic tetrad forms together with the different cytokinetic dynamics observed in successive- and simultaneous-type PMCs demonstrates that male meiotic cytokinesis has adopted many forms and may be regulated differently between different plant species and/or clades. Although during the last years some regulatory cues have been revealed (e.g., MAPK signaling cascade), many essential components are still unknown, withholding us from a complete insight into the molecular regulation of PMC cytokinesis. As such, future research needs to be focused on the identification and elucidation of both molecular and environmental factors underlying the specificity and variability in male meiotic cell plate formation in plants.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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