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MINI REVIEW

Making Two out of One: Kinesin Motors Driving Plant Cell Division

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ABSTRACT

The Kinesin superfamily of microtubule dependent motors is present in all eukaryotes. Not all of the subfamilies are represented in all kingdoms, and the ones that are do not always show conserved functions. Tight control of the cytoskeleton is essential for proper progression and completion of mitosis and cytokinesis, and key functions are carried out by kinesin motor proteins of various families. In this context, we take a closer look at plant kinesins involved in spindle formation and chromosome congression. Additionally, plant kinesins have been implicated in the deposition of cell plate material in the plane of cell division during cytokinesis in the angiosperm *Arabidopsis thaliana* and the moss *Physcomitrium patens*. In light of these recent discoveries, this mini-review aims to give an update on kinesins involved in plant cell division with brief reference to well-studied counterparts in other organisms.

1 | Introduction

Kinesins are molecular motors that can move along microtubules (MTs) while hydrolyzing ATP, serving elementary functions in protein and organelle trafficking, cytoskeleton organization, mechanical integrity of cells, and cell division (Miki et al. 2005; Li et al. 2012; Hirokawa and Tanaka 2015; Sun et al. 2024). In humans, compromised functionality of kinesins often causes severe diseases (Hirokawa and Tanaka 2015). The kinesin superfamily has been classed into 14 subfamilies based on sequence similarity of their motor domains (Lawrence et al. 2004; Miki et al. 2005). This conserved catalytic core of kinesins contains the MT binding and ATP hydrolyzing domain and can be located at the amino (N)-terminus, the central region, or at the carboxy (C)-terminus of the protein (Miki et al. 2005). Additional characteristic features of kinesin motor proteins are the neck, the stalk, and the highly variable tail regions. The neck contributes to the regulation of activity as well as directionality of motility with family-specific features. The stalk and tail domains are mainly responsible for cargo binding, dimerization, or interactions with other proteins (Miki et al. 2005, 2014). Plant genomes do not encode all of the kinesin subfamilies but display extended numbers of isoforms for some of them (Nebenführ and Dixit 2018). Particularly, kinesin families 3 and 11 are absent from land plants (Richardson et al. 2006). For a recent kinesin family tree, see Yin et al., in this special issue (Yin et al. 2026) and (Chen et al. 2024). The functions of most kinesins in embryophytes differ from those in their animal and fungi counterparts, which might be explained in part by the absence of canonical MT organizing centrosomes from vascular plants and the lack of the minus-end directed motor protein dynein (Li et al. 2012). In addition, myosin II, which in conjunction with actin facilitates constriction of the cleavage furrow in animals, is not present in the plant kingdom (Mühlhausen and Kollmar 2013; Aguilar-Cuenca et al. 2014).

Besides the mitotic spindle, that segregates chromosomes and is common to eukaryotes, plants evolved unique, plant-specific

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cytoskeletal arrays such as the preprophase band (PPB) and phragmoplast, which aid in the establishment of a division plane and insertion of the cell plate (Smertenko et al. 2017, 2018). The underlying mechanisms are tightly linked with plant-specific kinesin functions (Livanos and Müller 2019; Liu and Lee 2022).

In contrast to cell division in metazoans where the daughter cells are divided by constriction of the cleavage furrow, cell division in land plants proceeds from the inside of the cell outward (Lloyd and Chan 2006; Livanos and Müller 2019). This is achieved by initiation of the cell plate in the center of the cell and its lateral expansion, until it reaches the parental plasma membrane at the cell plate fusion site. Subsequently, the cell plate separating the new daughter cells matures into the cell wall (Jurgens 2005; Lloyd and Chan 2006; Livanos and Müller 2019). Responsible for cell plate biosynthesis is the cytokinetic phragmoplast, a bipolar MT array consisting of two sets of mostly antiparallel MTs that interdigitate with some of their plus ends at the so-called midzone, where they are embedded in the cell plate assembly matrix (Smertenko et al. 2018; Livanos and Müller 2019). Phragmoplast MTs depolymerize on the phragmoplast inside, the so-called lagging zone, persist in the transition zone, and nucleate and polymerize at the leading zone to extend outwards, driving the centrifugal expansion (Smertenko et al. 2018). The second plant-specific structure involved in cell division is the PPB, a cortical ring consisting of MTs, actin filaments, and associated proteins, that arises at the transition from G2 phase to M phase (Smertenko et al. 2017; Livanos and Müller 2019). It is the earliest landmark of the cortical division zone (CDZ), the area of plasma membrane and adjacent cell wall that marks the cell plate fusion site. Thus, even after PPB disassembly during prometaphase, the CDZ remains polarized until the end of cytokinesis (Livanos and Müller 2019; Dahiya and Bürstenbinder 2023). In this review, we provide an update on kinesin functions during plant cell division (Table 1).

TABLE 1 | Overview of recently reported localizations and functions of kinesins in cell division.

Process/family	Subcellular localizations	Functions	References
Spindle assembly			
Kin-14D1/MDKIN2	Nucleus, interpolar microtubules	MT convergence into spindle pole	Guo et al. (2024)
Kin-14D2	Kinetochore pairs, K-fibers		Guo et al. (2024)
Kin-14D3	Kinetochore pairs, K-fibers		Guo et al. (2024)
Kin-14A1/ATK1	Nucleus, Spindle MTs, Phragmoplast MTs	MT sorting/MT nucleation	Hotta et al. (2022)
Kin-14A2/ATK5	PPB, Spindle MTs, Phragmoplast MTs	MT sorting	Hotta et al. (2022)
Spindle bipolarity			
Kin-5C/AtKRP125b		Antiparallel MT sliding	Strauß et al. (2021)
Kin-12E	Perinuclear, Spindle midzone, phragmoplast midzone	MT sliding, poleward flux	Herrmann et al. (2020)
Chromosome congression			
Kin-7O/KAK1/POS3	Kinetochore pairs	Chromosome congression, kinetochore-MT attachment	Tang et al. (2024) Chen et al. (2025)
Phragmoplast organization/cell plate formation			
*Pp*Kin-12II	Phragmoplast midzone	Trafficking of cell plate material	Yamada et al. (2025)
Kin-12A/PAKRP1	Phragmoplast midzone leading zone	Phragmoplast organization, trafficking	Elliott et al. (2024)
Kin-12B/PAKRP1L	Phragmoplast midzone leading zone	Phragmoplast organization trafficking	Elliott et al. (2024)
Kin-12F	Phragmoplast midzone transition zone	Phragmoplast lateral expansion	Elliott et al. (2024)
Division plane maintenance			
Kin-12C/POK1	Cortical division zone	Phragmoplast guidance	Lipka et al. (2014)
Kin-12D/POK2	Cortical division zone, phragmoplast midzone leading zone	Phragmoplast guidance Midzone leading zone	Herrmann et al. (2018)

1.1 | Kinesins Facilitate Spindle Formation

The mitotic spindle is a microtubule-based micro-machine whose job is the segregation of chromosomes without mistakes (McIntosh et al. 2012). The assembly of the mitotic spindle is one of the crucial functions of kinesins in all eukaryotes. Despite mechanistic differences between animals and plants, the process of bipolar spindle formation and function involves well-conserved factors in most land plants (Liu and Lee 2022). As kinesin families are expanded in many plant species (Yin et al. 2026), spindle formation is remarkably robust, additionally hampering efforts to identify essential players.

In late prophase, before PPB disassembly, the acentrosomal spindle is initiated at the nuclear envelope through MT reorganization and nucleation (Masoud et al. 2013). The prophase spindle emerges from dynamic MTs that initially encapsulate the nucleus and are progressively sorted to converge into two diffuse poles along an axis perpendicular to the PPB (Figure 1a) (Ambrose and Cyr 2008). After nuclear envelope collapse, MT bundles connect the spindle poles with the kinetochores, forming the kinetochore-MT bundles (kinetochore fibers, Figure 1a) (Smirnova and Bajer 1998), while the interpolar MTs create antiparallel overlapping regions in the spindle midzone. The highly dynamic, laterally expanding kinetochore fibers align chromosomes during prometaphase and metaphase and finally arrange them at the metaphase plate (congression). Within the spindle midzone, the antiparallel interpolar MTs engage with each other; the majority of MTs orient their plus ends towards the chromosomes and their minus ends towards the poles (Figure 1a) (Euteneuer et al. 1982). Subsequent elongation of interpolar MTs and simultaneous shrinkage of the opposing kinetochore fibers facilitate the poleward segregation of chromosomes during anaphase (Figure 1a).

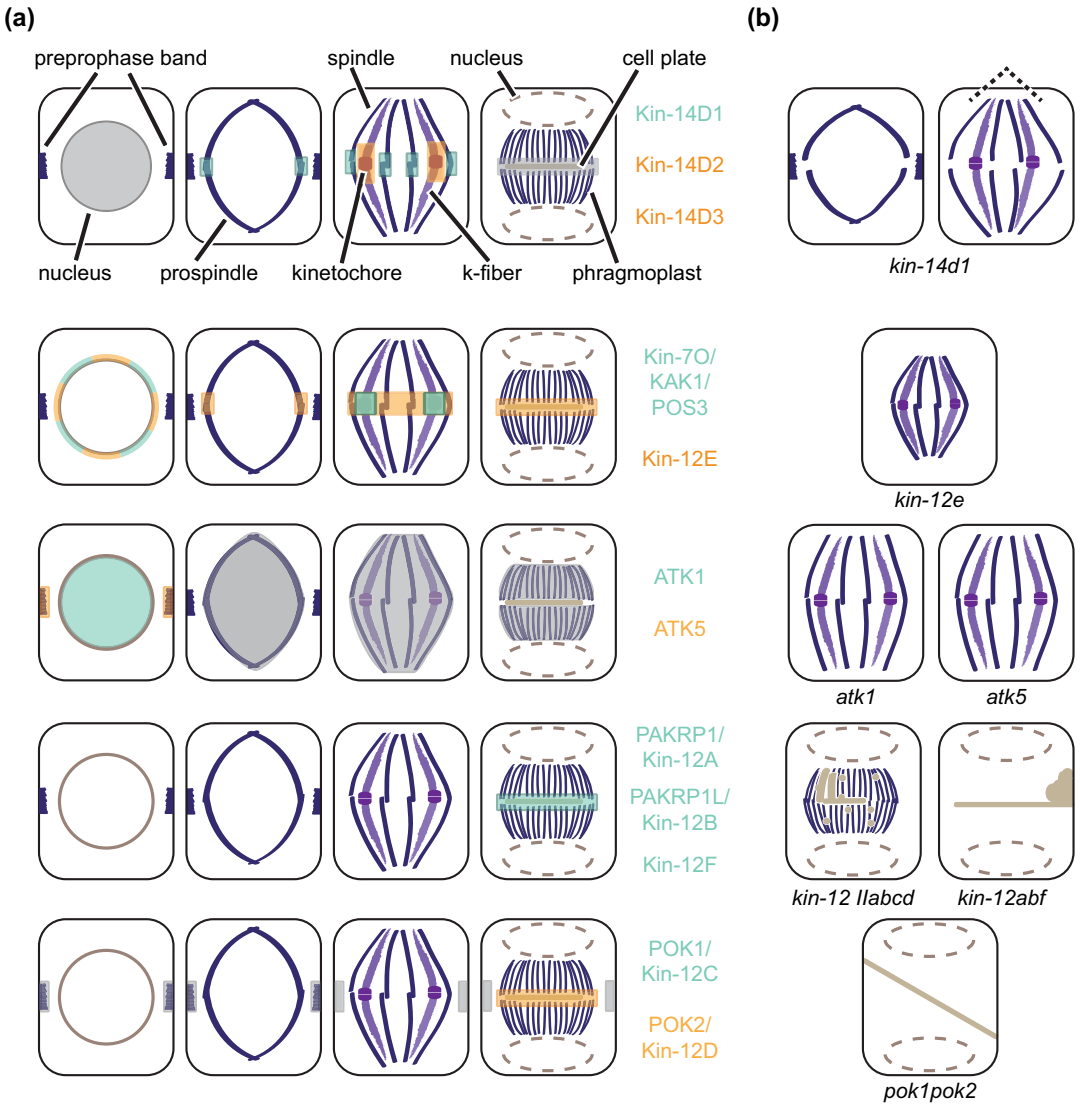


FIGURE 1 | Localization pattern and mutant phenotypes of kinesins during cell division. (a) The localization of different kinesins is indicated by the color-coded boxes. Grey boxes indicate shared localization. (b) Mutant phenotypes. The *kin-14d1* mutant displays widened spindle pole angles (dashed line), likely due to compromised interpolar microtubule connections. The *kin-12e* mutant spindles are smaller. *atk1* and *atk5* show spindles with broadened poles resulting in barrel shape. In *Physcomitrium patens kin-12 IIabcd* mutant, cell plate formation is disturbed and ectopic cell plate material (beige) accumulates along phragmoplast microtubules. The *Arabidopsis thaliana* class II kinesin-12 triple knockout *kin-12abf* contains gaps in cell walls and clustered deposition of cell wall material. The *pok1pok2* double mutant exhibits oblique cell walls.

Recently, Kinesin-14D1 has been implicated in spindle morphogenesis during prophase (Guo et al. 2024). When Kin-14D1 is missing, formation of the prophase spindle is delayed upon low dose treatment with the MT disrupting drug oryzalin. MTs of the opposite poles are not properly joined in the center of the prospindle, leading to splaying MT bundles and increased pole angles (Figure 1b). The authors suggest that in the treated *kinesin-14d1* mutant during meta- and anaphase the kinetochore fibers are not integrated into an organized spindle due to the lack of Kin-14D1 dependent interpolar MTs. Collapsed spindles likely caused the observed loss of viable root growth (Guo et al. 2024). Kinesin-14D1 resides in the nucleus, and upon nuclear envelope breakdown, and consistent with the cellular phenotypes, it associates with MTs at the spindle midzone but remains absent from centromeres and kinetochore fibers (Figures 1 and 2a). Its function relies on the plus end directed motility of the Kinesin-14D1 motor domain and its malectin homology domain. The latter distinguishes Kinesin-14D1 and Kinesin-14D2 from other kinesins, including closely related Kinesin-14D3. The malectin domain is not the only distinguishing characteristic of Kinesin-14D1, as all members of the kinesin-14 family exhibit minus end directed movement and only a few members show processive movement (Jonsson et al. 2015; She and Yang 2017; Gicking et al. 2018). Therefore, the plus end directed motility displayed by Kinesin-14D1 is an unexpected feature. The Kinesin-14 class D members share the central position of the motor domain preceded by the typical Kinesin-14-type neck linker and followed by C-terminal domains containing coiled-coils. However, localization patterns of Kinesin-14D1 differ from Kinesin-14D2 and Kinesin-14D3, which are prominent on kinetochore pairs and along kinetochore fibers. Guo et al. suggested that by crosslinking interpolar MTs, Kinesin-14D1 plays a critical role in organizing the kinetochore fiber complexes into a convergent bipolar spindle array. Previously, *Kinesin-14D1/MDKIN2* were implicated in defects during seed and pollen development (Galindo-Trigo et al. 2020). These phenotypes could not be corroborated by Guo et al., who in their study employed the same and independent *kinesin14d1* alleles. Differences in growth conditions might contribute to the disparate observations.

Arabidopsis thaliana (*A. thaliana*) has 21 Kinesin-14 family members divided into six classes, A through F. Besides class D members, Kinesin-14A1/ATK1 and Kinesin-14A2/ATK5 are also MT minus end directed and involved in spindle morphogenesis (Chen et al. 2002; Hotta et al. 2022). The involvement of ATK1 in the spindle has been implicated for some time (Chen et al. 2002). The *atk1-1* mutant shows disturbed chromosome segregation, causing frequent failures in male meiosis and reduced fertility. The *atk1-1* mutant spindles exhibit broadened poles and misaligned kinetochore fibers, a phenotype shared with the *Zea mays* ortholog mutation *divergent spindle 1* (Chen et al. 2002; Duffy et al. 2024). The synthetic lethality observed in *atk1-1; atk5* double mutants implies redundant function, but the two Kinesin-14A proteins localize differently during prophase (Figure 1). While ATK5 localizes to the PPB, ATK1 resides in the nucleus. However, both kinesins associate with spindle MTs upon nuclear envelope breakdown and later decorate the phragmoplast (Figure 1). The localization of ATK1 and ATK5 on the spindle is not interdependent, although they coordinately associate with each other in vivo. This association is most obvious on ATK1 dependent, highly focused/converged spindle poles upon hyperosmotic stress induction (Hotta et al. 2022). ATK1/

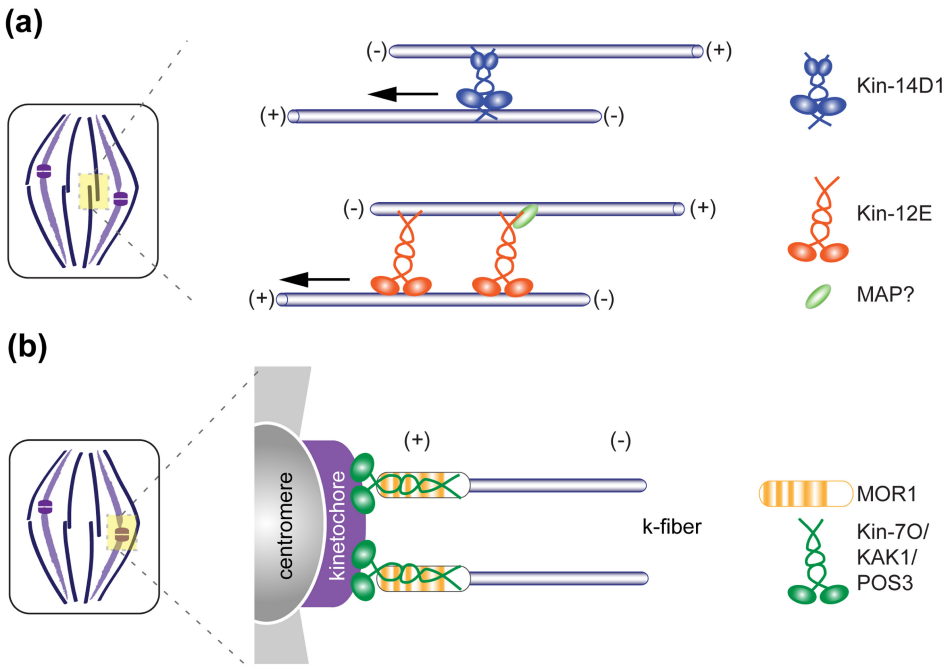


FIGURE 2 | Spindle assembly and chromosome congression. (a) Kinesin-14D1 is implicated in sliding of interpolar microtubules in prophase and metaphase, reliant on their microtubule plus-end directed motility and malectin-binding domain. Kinesin-12E likely shows plus end directed motility and facilitates sliding via a non-motor microtubule binding site or putative interacting microtubule associated protein (MAP). (b) Microtubule-Kinetochore attachment during chromosome congression is mediated by interaction of Kinesin-7O/KAK1/POS3 carboxy-terminal with the MOR1 microtubule polymerase carboxy-terminal, downstream of TOG domains (yellow bars). (+) and (–) indicate microtubule plus- and minus- ends. Arrows indicate direction of motility.

ATK5 likely oligomerize to organize poleward MT convergence by association with, and potentially sliding of, interpolar MTs. ATK5 tracks MT plus ends and modifies their stability via MT minus end directed motility, leading to MT length reduction as seen in in vitro studies of kinesin-14 (Ambrose et al. 2005; Ogren et al. 2022). Furthermore, ATK1, but not ATK5, is involved in the association of the MT nucleation factor augmin with spindle MTs (Hotta et al. 2022), whose phospho-regulation in turn is required for spindle morphology (Romeiro Motta et al. 2024). Thus, Kinesin-14 members likely facilitate MT sorting early on during prophase and may be required for MT nucleation at spindle poles (Motta and Schnittger 2021).

The key players of bipolar mitotic spindle assembly and morphology in animals are Kinesin-5 and Kinesin-12 family members. Establishment of a bipolar spindle is essential for chromosome segregation and stability. Genetic or pharmacological inhibition of animal Kinesin-5 Eg5, a MT plus-end directed motor that forms bipolar homotetramers, results in the collapse of the bipolar spindle into a monopolar spindle (Mann and Wadsworth 2019). Eg5 generates outward directed forces by crosslinking and sliding antiparallel MTs to separate spindle poles. In *A. thaliana*, AtKRP125c, one of the four Kinesin-5 family members, occupies all microtubular arrays throughout the cell cycle. Its temperature-sensitive mutant displays cytokinetic defects and compromised spindle bipolarity due to destabilization of the antiparallel MTs in the spindle midzone (Asada et al. 1997; Bannigan et al. 2007). Recent investigations into the plus-end directed processive motility and MT sliding of AtKRP125b in vitro further support canonical functions of plant Kinesin-5 (Strauß et al. 2021). In *Physcomitrium patens* (*P. patens*), Kinesin-5 localizes to prophase MTs, the spindle, and the phragmoplast, but surprisingly seems to be depleted from their midzones. Nevertheless, RNAi suppression of Kinesin-5 resulted in lagging chromosomes during anaphase and cytokinetic failure (Miki et al. 2014). At large, kinesin-5 function appears conserved in plants.

Different studies reported that human Kinesin-12 Kif15 formed homodimers (Brouwers et al. 2017; Reinemann et al. 2017) or homotetramers (Drechsler et al. 2014; Drechsler and McAinsh 2016; Mann et al. 2017) and bundled, crosslinked, and slid antiparallel MTs (Salazar and Ohi 2024), acting redundantly with Kinesin-5 Eg5. In *A. thaliana*, six kinesin-12 family members are divided into two classes (Müller and Livanos 2019). Of these, only one, the class I Kin-12E, has a function in spindle assembly. During prophase, it accumulates perinuclear, and after nuclear envelope breakdown, it decorates spindle MTs and steadily enriches at the spindle midzone (Figures 1 and 2a) (Herrmann et al. 2020). After spindle disassembly, Kin-12E accumulates at the phragmoplast midzone, where it might act redundantly with other kinesin-12 family members (Herrmann et al. 2020). *kin12e* mutants display a delay in chromosome congression, spindle assembly, and a reduction of spindle size. Additionally, the MT-kinetochore attachments or k-fiber mechanical properties are less stable since the distance between paired kinetochores decreased in these mutants (Herrmann et al. 2020). Similarly, Kif15 binds and crosslinks k-fiber MTs, fortifying and repairing them, thereby promoting k-fiber cohesion (Begley et al. 2021). Kin-12E not only stabilizes MT-kinetochore attachments, but it also modulates MT dynamics during metaphase, which is indicated by lower poleward flux in *kin12e* mutants and reduced MT dynamic instability upon overexpression of the truncated Kin-12E motor domain (Herrmann et al. 2020).

The Kin-12E^{T181N} motor-dead mutant, impaired in ATP hydrolysis, loses the spindle and phragmoplast midzone localization, suggests plus-end directed motility, but instead decorates the entire lattice of interphase and mitotic MT arrays (Herrmann et al. 2020). Quite expected, the motor-dead Kin-12E^{T181N} does not rescue the mutant phenotype. The involvement of Kin12E in spindle size control and kinetochore fiber dynamics points to a role, conserved in the kinesin-12 family, counteracting inward-directed forces within the spindle. Similarly, Kif15 supports spindle extension, as its depletion leads to shorter spindles, while simultaneously counteracting inward-directed forces by acting as a mechanical ratchet (Vanneste et al. 2009) (McHugh et al. 2018). However, Kif15's role in spindle assembly and maintenance of bipolarity is essential only in the absence of Eg5 (Tanenbaum et al. 2009; Vanneste et al. 2009). Thus, Eg5 is the primary driver of poleward flux in metazoans, but Kif15 and other plus-end directed kinesins contribute. Given the resemblance between loss of plant and animal kinesin-12 regarding spindle dynamics and spindle length control, as well as the highly conserved function of Kif15 and Xklp2, its homolog in *Xenopus*, it stands to reason that also in plants a kinesin-5 family member can compensate for the lack of Kin12E, explaining the relatively mild mutant phenotype (Mitchison 2005; Buster et al. 2007; Herrmann et al. 2020).

Creating higher order mutants of kinesin-5, such as KRP125c and Kin12E, or other kinesins involved in spindle formation, possibly also with the use of inducible RNAi to circumvent lethality, might help resolve the intricacies of this process. Additionally, it might be interesting to examine whether the combined mutation of MT plus-end directed Kin-14D1 and Kin12E influences spindle formation and forces within.

1.2 | Control of Chromosome Congression in the Spindle

Spindle formation is closely coordinated with chromosome congression (Maiato et al. 2017). Successful chromosome segregation during mitosis and meiosis relies on amphitelic attachment of kinetochores to MTs and alignment at the spindle midzone, monitored by the spindle assembly checkpoint (SAC) (Risteski et al. 2021; Liu and Lee 2022; Deng et al. 2024). In metazoans the kinesin-7 family member centromere protein E (CENP-E) is a key player in chromosome congression and is involved in SAC signaling (Craske and Welburn 2020). The MT plus-end directed CENP-E localizes to unattached kinetochores and aligns monotelic chromosomes towards the spindle midzone. In *A. thaliana* Kinesin-7O shows sequence homology to the CENP-E amino-terminal, and may fulfill a similar role (Tang et al. 2024; Chen et al. 2025). In two new studies, Kinesin-7O was characterized and named Kinetochore-associated Kinesin1 (KAK1) because of its continuous association with kinetochores (Tang et al. 2024), and Propyzamid Oversensitive 3 (POS3), respectively (Chen et al. 2025). Both studies report kinetochore localization throughout mitosis (Figure 2b) (Tang et al. 2024;

Chen et al. 2025), in contrast to animal CENP-E, which disappears from kinetochores after metaphase. After nuclear envelope breakdown KAK1/POS3 appeared as paired dots that associated with chromosomes within the spindle. *kak1* and *pos3-1* mutant seedlings showed growth delay upon oryzalin or propyzamide treatment respectively, leading to defects in chromosome alignment and delayed onset of anaphase (Tang et al. 2024; Chen et al. 2025). In contrast to animal CENP-E, plant SAC is still intact in the absence of KAK1/POS3, suggesting the involvement of additional players in plant chromosome congression and SAC signaling. Surprisingly, POS3 kinetochore localization depended on the MT polymerase MOR1, likely via direct binding of POS3 carboxy-terminal stalk with the region downstream of MOR1 TOG domains (Figures 2b and 3) (Chen et al. 2025). Animal CENP-E displays a kinetochore binding site in its carboxy-terminal, which is not present in plant kinesin-7. As CENP-E in metazoans works in concert with Kinesin-4, Kinesin-10 and Kinesin-13 in addition to dyneins (Craske and Welburn 2020), their plant orthologs and plant kinesin-14 might be involved in plant chromosome congression. Since Kin-12E also shows a delay in chromosome congression, it remains to be discovered whether these proteins cooperate during chromosome congression.

1.3 | The Phragmoplast and Kinesins

The initially disk-shaped bipolar phragmoplast is essential for cell plate biosynthesis. During phragmoplast expansion, MTs transition from the "leading zone" on the outside, through a "transition zone," to the "lagging zone" in the phragmoplast center (Smertenko et al. 2018). At the leading zone, new MTs polymerize, whereas at the lagging zone MT depolymerization occurs. These zones associate with developmental stages of the cell plate assembly, that is, with vesicle delivery to the phragmoplast midzone, and with fusion and tubule-vesicular network as the disk phragmoplast transforms into a ring (Smertenko et al. 2017). How phragmoplast MT expansion and cell plate assembly are coordinated is poorly understood. The key kinesin players in phragmoplast assembly, expansion, and guidance described to date are members of the kinesin-12 family (Lee et al. 2015; Müller and Livanos 2019). They are known to interact with numerous MT associated proteins and localize predominantly to the midzone of the cytokinetic apparatus (i.e., the phragmoplast) and to the CDZ (Müller and Livanos 2019). Recent reports shed new light on the role of kinesin-12 in phragmoplast expansion and cell plate assembly.

The members of the plant kinesin-12 family fall into distinct class I and class II (Shen et al. 2012; Miki et al. 2014; Müller and Livanos 2019). In *P. patens*, the kinesin-12 family is enlarged with 16 members in class I and four in class II, compared to three members each in *A. thaliana*. The pair of *A. thaliana* kinesin-12II, Phragmoplast Associated Kinesin Related Protein 1 (PAKRP1/Kin12A) and PAKRP1 like (PAKRP1L/Kin12B) were the first characterized members of plant kinesin-12 (Lee and Liu 2000; Pan et al. 2004). They are essential for bipolar phragmoplast organization during microspore development (Lee et al. 2007). In their absence in pollen grains, postmeiotic cytokinesis frequently failed as a consequence of misorganized phragmoplasts, and plants produced very few seeds (Lee et al. 2007). Consistently, class II kinesin-12 members localize

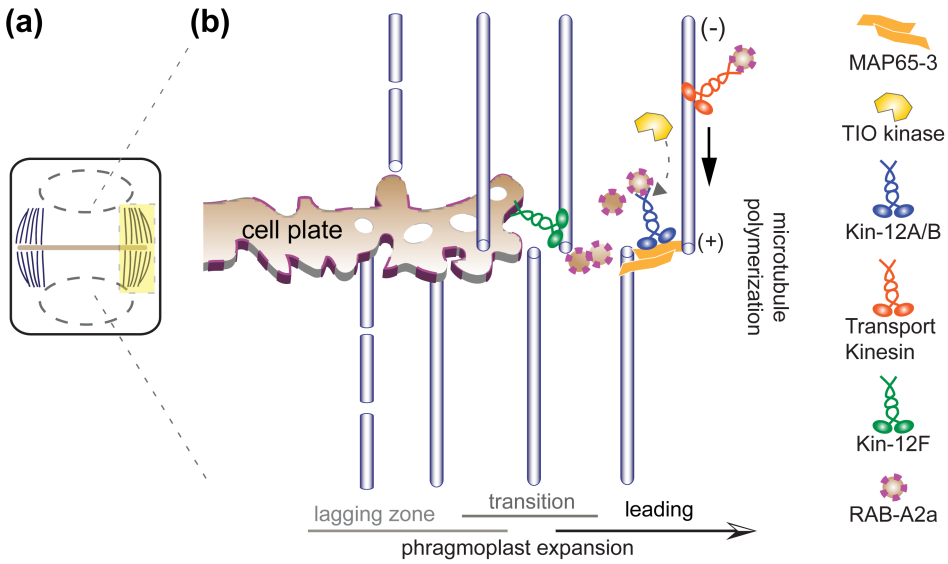


FIGURE 3 | Coordination of microtubule dynamics and vesicle transport during phragmoplast-assisted cell plate biosynthesis. (a) Graphical representation of a cytokinetic cell with ring phragmoplast. (b) Close-up of the phragmoplast region (shaded yellow in (a)). The expansion of the phragmoplast proceeds from the central position towards the parental wall (direction of arrow). Microtubules depolymerize in the lagging zone and are stabilized by MAP65-3 crosslinkers in the leading zone. Rab-A2a-bearing vesicles are transported to the phragmoplast midzone via microtubule plus-end directed kinesins of unknown identity; a role of Kin-12A/B cannot be excluded. Kin-12A/B are stabilized in the leading zone by MAP65-3, whereas Kin-12F resides in the transition zone independently of MAP65-3. Kin-12A/B and Kin-12F interact with Rab-A2a and provide platforms to support the formation of the tubulo-vesicular cell plate. RAB-A2a and Kin-12A&B determine the correct localization of TIO kinase in the midzone, linking stable phragmoplast polymerization to the presence of the membrane in this region. The association between RAB-A2a and the tail regions of Kin-12A/B promotes the subsequent binding of TIO-kinase (grey dashed arrow).

to the phragmoplast midzone in diverse plant species, that is, in *A. thaliana*, *Oryza sativa*, and *P. patens* (Guo et al. 2009; Miki et al. 2014).

Although electron tomography provided evidence that kinesins attach to vesicles along phragmoplast MTs during endosperm cellularization and pollen meiosis, respective kinesin candidates for vesicle trafficking remained elusive for decades (Otegui et al. 2001; Otegui and Staehelin 2004). The *P. patens* Armadillo-repeat containing kinesin moves organelles anterogradely during interphase, but a role in cell division has not been assigned (Yoshida et al. 2023). However, lately *P. patens* Class II kinesin-12 (kinesin12-II) has been implicated in the transport of cell plate material along phragmoplast MTs (Yamada et al. 2025). Surprisingly, in *kinesin-12 IIabcd* mutants, phragmoplast organization was intact, but expansion was delayed due to slower MT disassembly. Furthermore, the mutation manifested in the absence of a dark phragmoplast midzone in plants expressing GFP-tagged MT reporters, which indicates midzone instability. Treatment with brefeldin A, a drug impeding vesicle transport from the ER to the Golgi, mimicked the phenotype of the mutant (Yamada et al. 2025), suggesting that midzone instability originates from impaired membrane remodeling. The ultrastructural analysis of cytokinetic cells in the mutant revealed abnormal, nonuniform cross walls with protrusions. The latter corresponded to ectopic cell plate material deposited outside of the phragmoplast midzone, as observed in *kinesin-12 IIabcd* mutants by live cell imaging (Yamada et al. 2025). Dispersal of cytokinetic vesicles along phragmoplast MTs in the presence of non-motile kinesin12 IIc^{Rigour} further corroborates the proposed role of Kinesin-12 II in vesicle trafficking in *P. patens* (Yamada et al. 2025).

Somewhat related, Elliott et al. (2024) reported that class II kinesin-12s in *A. thaliana* act in a regulatory module with RAB-A2a and Two in One (TIO) kinase, coupling phragmoplast dynamics and vesicle trafficking rates. In somatic cells of the mutant *kin12a/b*, the authors observed that the phragmoplast expansion rate was reduced by 30%. The cell type-specific knockdown of the phragmoplast midzone localized class II Kin-12F in the *kin12a/b* mutant caused further enhancement of cytokinetic defects such as incomplete cross walls, multinucleate cells, and misaligned division planes. Additionally, large amorphous structures accumulated that contained GTPase RAB-A2a, likely representing mistargeted TGN/EE material (Elliott et al. 2024). Thus, also in *A. thaliana* class II kinesin-12 may be involved in vesicle targeting to the phragmoplast midzone, considering that Kin-12A partially rescued the *P. patens kinesin-12 IIabcd* mutant phenotype, and likely involving interaction with the GTPase RAB-A2a (Elliott et al. 2024; Yamada et al. 2025).

Although all three class II Kin-12 of *A. thaliana* interact with RAB-A2a, only Kin-12A/B interacted with TIO kinase, that localized to the midzone of the phragmoplast leading zone and was also implicated in male meiosis (Oh et al. 2012; Elliott et al. 2024). Besides, the localization at the phragmoplast midzone differed between Kin-12A/B and Kin-12F (Elliott et al. 2024). Kin-12A and Kin-12B enriched in the midzone of the phragmoplast leading zone dependent on the antiparallel MT cross-linkers MAP65-3, the plant homolog of the PRC1/Ase1 family. In contrast, Kin-12F located to the transition zone independently of MAP65-3 (Ho et al. 2011; Elliott et al. 2024). Kin-12A/B and TIO kinase mutually affected their localization at the phragmoplast leading zone (Oh et al. 2012). However, in the presence of inactive RAB-A2a, TIO kinase ectopically populated the entire cell plate, suggesting its distribution at the phragmoplast leading zone is also controlled by RAB-A2a in addition to Kin-12A/B (Elliott et al. 2024). In the proposed model, the delivery of RAB-A2a at the phragmoplast leading zone depends at least partially on Kin-12A/B and promotes TIO and Kin-12A/B binding. The abundance of active RAB-A2a maintains TIO at the leading zone and scales signaling for phragmoplast remodeling (Elliott et al. 2024). This is an attractive model explaining the coordination of phragmoplast expansion and vesicle trafficking. It will be interesting to see how it integrates with other players previously implicated in the coordination of membrane trafficking and MT turnover such as PI4Kβ kinase and phosphoinositide phosphatase SAC9 (Lin et al. 2019; Lebecq et al. 2023). SAC9 phosphatase reduces the phosphoinositide $PI(4,5)P_2$ levels at the leading zone, while PI4Kβ kinases maintain high PI4P levels and promote MT dynamics via signaling that leads to MAP65-3 inactivation.

Consistent with the role of kinesin-12 in cytokinesis, a study using bright yellow-2 tobacco cell culture identified class II kinesin-12 phospho-peptides with high abundance during cell division (Kimata et al. 2023). Also, a novel phosphorylation inhibitor PP2 blocked the localization of Kin-12A/B at the phragmoplast midzone and interfered with phragmoplast formation (Kimata et al. 2023), suggesting phospho-regulation, potentially via TIO kinase, contributes to class II Kin-12 localization/function in the phragmoplast.

Besides class II kinesin-12s, class I Kin-12D/Phragmoplast Orienting Kinesin 2 (POK2) and the aforementioned Kin-12E localize to the phragmoplast midzone in *A. thaliana* (Herrmann et al. 2018, 2020). Nothing is yet known about the role of Kin-12E in the phragmoplast. Similarly to class II Kin12A/B, POK2 is recruited to the phragmoplast midzone in a MAP65-3 dependent manner, likely via direct interaction (Herrmann et al. 2018). Coherently with its localization at the midzone, the phragmoplast expansion rate is reduced *in pok2* single mutants, albeit without effects on cell plate formation, suggesting extensive functional redundancies, likely with other kinesin-12 members (Herrmann et al. 2018; Müller and Livanos 2019). Kin-12D/POK2, together with its paralog Kin-12C/POK1, has essential redundant functions in division plane maintenance and phragmoplast guidance, severely affecting cell and organ shape and growth upon mutation (Müller et al. 2006; Lipka et al. 2014; Herrmann et al. 2018). POKs emerge at the PPB during prophase in a MT dependent manner and remain at the division site until the end of cytokinesis, acting as scaffolds for several division site resident proteins (Livanos and Müller 2019). Myosin XI was recently added to the list of POK-dependents, further corroborating a yet poorly understood role of actin in plant cell division. Upon recruitment to the PPB, myosin XI, POKs, and other motors form so-called cytoskeleton-associated motor assemblies (CAMPs) that reorganize into discrete foci in a Myosin XI and F-actin dependent manner late in mitosis (Huang et al. 2024). Moreover, division planes are misoriented in oryzalin hypersensitive *myoXI-4KO* mutants, and phragmoplasts are misguided in the Myosin XI maize mutant *opaque1*, underpinning the

importance of myosin and kinesin mediated microtubule—actin interplay in plant cell division.

How POKs tether to the division site is of particular interest due to their scaffolding function for most of their currently known interactors (Livanos and Müller 2019; Huang et al. 2024). Kin-12D/POK2 seem to associate with MTs of the PPB through a non-motor MT binding site in its carboxy-terminus and persist at the plasma membrane via at least two putative lipid binding sites (putative amphipathic helix and polybasic domain) (Livanos et al. 2025). Interestingly, deletion of the phospholipid and MT binding region does not abolish POK2 midzone association (Livanos et al. 2025).

Remarkably, the mislocalization of MAP65-3, observed upon the loss of phosphoinositide phosphatase SAC9, coincides with the branching of the cell plate at a specific distance from the parental wall in late cytokinesis. Interestingly, the attachment site of this cell plate branch becomes decorated with Kin-12C/POK1, in addition to the site predetermined by the PPB (Lebecq et al. 2023). This observation suggests late recruitment of Kin-12C/POK1, most likely from a pool of access Kin-12 at the phragmoplast, as previously reported in *tan1 air9* double mutants (Mills et al. 2022) and as observed when POK2 division site recruitment is compromised (Livanos et al. 2025). Considering the phospholipid binding properties of Kin12-D/POK2, late recruitment of class I Kin-12C/D might be mediated by direct binding to phospholipids at the plasma membrane (Livanos et al. 2025).

2 | Concluding Remarks

It is evident that many of the kinesin functions in plants have been modified to accommodate the unique cytoskeletal organization of plants, their reduced molecular motor repertoire, and their specific lifestyle (Lee et al. 2015). The larger size of many plant kinesin families combined with extensive functional redundancy has hampered the study of conserved and non-conserved kinesin functions in many plant kinesin families. Therefore, the phenotypes of the mutants are often subtle or require specific conditions to be observed. However, inducible reverse genetic approaches and the analysis of higher order mutants could prompt new opportunities.

The kinesin-12 family of *A. thaliana* has been fully characterized. While most members of the Kin-12 family have acquired plant-specific functions in phragmoplast organization, vesicle transport, and division plane establishment, class I Kin-12E appears to have some functional similarity to its opistokont orthologs in spindle assembly and spindle size regulation (Lee and Liu 2000; Pan et al. 2004; Müller et al. 2006; Lee et al. 2007; Ho et al. 2011; Lipka et al. 2014; Herrmann et al. 2018, 2020; Elliott et al. 2024). Kinesin-7O/KAK1/POS3 serves as another example for modified function as it might act similarly to CENP-E, although it seems to use a plant-specific mechanism in MT kinetochore binding, likely involving MOR1 MT polymerase. Furthermore, kinesin-7O/KAK1/POS3 is not involved in SAC signaling in plants.

It remains to be investigated to what extent the molecular mechanisms and functions of kinesins are actually conserved in the individual members of the plant family (Tang et al. 2024). Therefore, cross-species complementation experiments of plant mutants with basal plant kinesins from families with a smaller number of members (Yin et al. 2026) might be extremely useful for comparison of cross-species complementation with kinesins from basal opisthokonts.

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Choy Kriechbaum and **Sabine Müller:** conceptualization, drafting and figure preparation.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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