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Review

Cytoskeletal regulation of primary plant cell wall assembly

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SUMMARY

The plant cell wall is an extracellular matrix that envelopes cells, gives them structure and shape, constitutes the interface with symbionts, and defends plants against external biotic and abiotic stress factors. The assembly of this matrix is regulated and mediated by the cytoskeleton. Cytoskeletal elements define where new cell wall material is added and how fibrillar macromolecules are oriented in the wall. Inversely, the cytoskeleton is also key in the perception of mechanical cues generated by structural changes in the cell wall as well as the mediation of intracellular responses. We review the delivery processes of the cell wall precursors that are required for the cell wall assembly process and the structural continuity between the inside and the outside of the cell. We provide an overview of the different morphogenetic processes for which cell wall assembly is a crucial element and elaborate on relevant feedback mechanisms.

Introduction

Plant cells are enveloped in an extracellular matrix, the cell wall, a material that is largely composed of a hydrated polysaccharide network containing proteins and ions[1,2]. A similar material, the middle lamella, serves as a mortar that glues plant cells together into stable tissue architectures[3]. The plant cell wall is deposited outside the plasma membrane in a layered microstructure that distinguishes two principal features. The primary cell wall is present in virtually all plant cell types: its thickness varies between ~100 and >1,000 nm and it is the only layer present in cells forming primary tissues and meristems — the stem cell niches giving rise to organ growth. A secondary cell wall is present in certain cell types characterizing secondary tissues that feature stiffer structures such as fibers, tracheids, or vessel elements. Its thickness can be up to several micrometers and it is deposited between the primary wall and the plasma membrane. Whereas the primary wall is flexible allowing cells to grow and change shape, deposition of the secondary cell wall serves to stiffen the cell wall and typically occurs only once cell growth has ceased.

Assembly of plant cell wall material occurs at the cell surface, but the exact mechanism depends on the type of macromolecule. Some polysaccharides are synthesized directly at the plasma membrane and extruded into the apoplastic space located immediately outside of the plasma membrane, whereas others are delivered to the cell surface in the form of precursors synthesized in the endomembrane system, notably the Golgi. Upon surface deposition, cell wall polysaccharides and their linkages are further modified by enzymatic and non-enzymatic proteins, as well as other factors such as pH, ions, and hydration status. These modifications are controlled with subcellular precision to regulate cellular activities such as cell growth and division[4]. Cell wall assembly can be initiated *de novo*, during cell division, or new polymers can be added to existing wall. The latter occurs not only during normal cell growth and differentiation, but also in response to both pathogenic and symbiotic microbes. In all these situations, the spatiotemporal regulation of the assembly process is crucial and relies on a functional cytoskeleton. This creates an intimate relationship between the cell's internal machinery and the microstructure of the extracellular matrix.

The connectedness between the cytoskeletal network and cell wall architecture is manifested in the similarity of phenotypes caused by mutations affecting either cytoskeletal functioning or cell wall synthesis. Importantly, the relationship is reciprocal and signaling occurs in both directions. The cytoskeleton not only regulates cell wall assembly, but also perceives changes in the cell wall, forming regulatory feedback loops that govern growth, development, and responses to environmental cues. In this review, we provide an overview of the ways in which the cytoskeleton mediates and regulates the delivery of cell wall precursors to the cell surface. We focus on the assembly of the primary plant cell wall during growth and morphogenesis. For the construction of the secondary wall or for assembly of wall material during symbiosis and defense we refer to excellent reviews on lignin deposition[5], cellulose deposition in the xylem[6], plant–microbe interactions[7], and the interface between arbuscular mycorrhizae and root tissues[8–10].

Cytoskeletal coordination of cell wall assembly

The different delivery pathways of cell wall polysaccharides and proteins to the cell surface involve the action of the cytoskeletal elements in a variety of ways (Figure 1).

Vesicular transport

Golgi-synthesized polysaccharides such as hemicelluloses and pectins, polysaccharide-modifying proteins, and the synthases

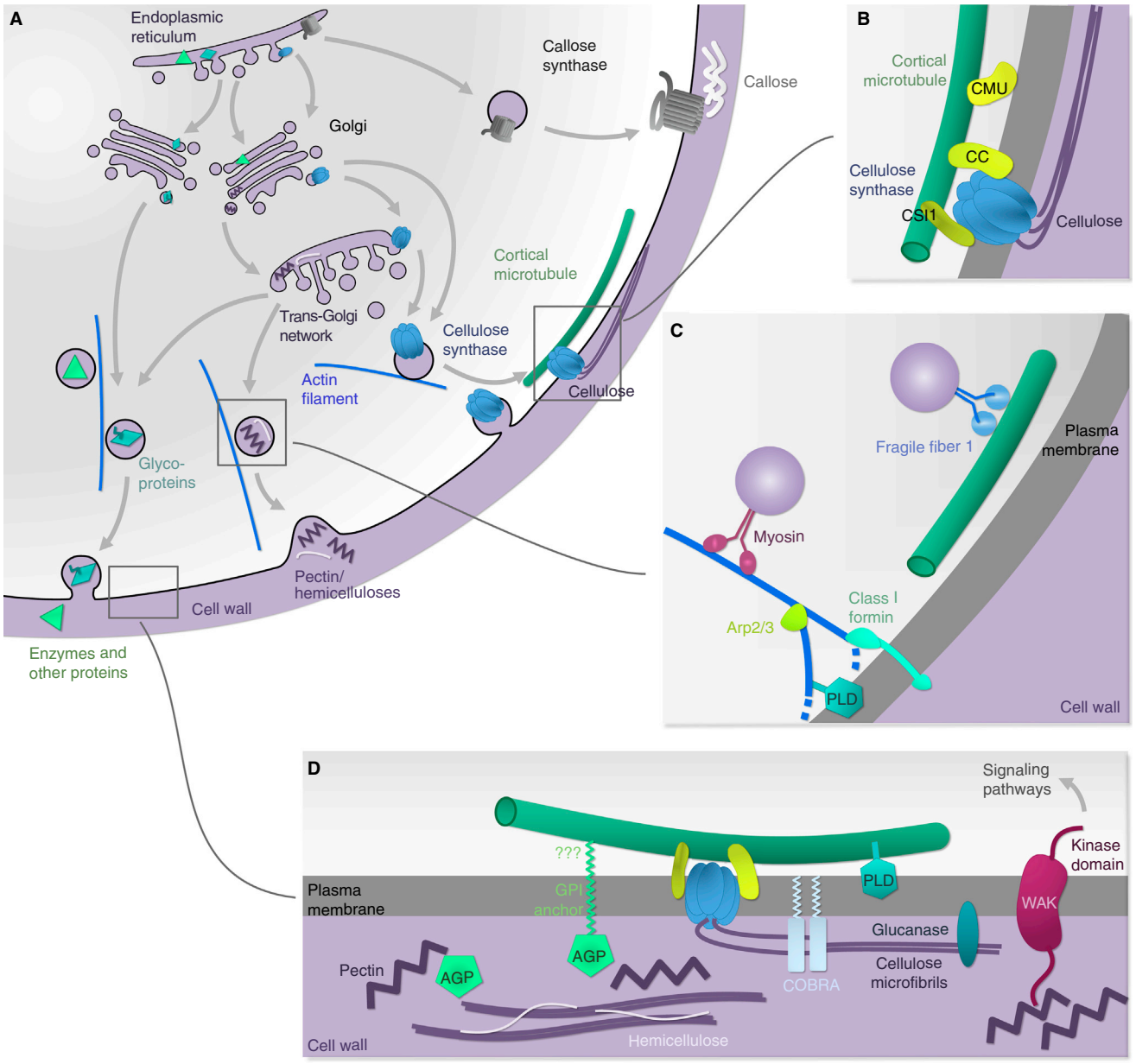

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Figure 1. Delivery pathways of polysaccharides and proteins to the plant cell surface and elements of the cell wall–membrane–cytoskeleton continuum.

(A) Cellulose and callose are synthesized directly at the cell surface by enzymes delivered to the plasma membrane through vesicular transport from the endoplasmic reticulum or Golgi and inserted by exocytosis. Cellulose synthase complexes move in the plasma membrane propelled by their synthetic activities; spatial control is provided by cortical microtubules. The interaction between these cellulose-synthase complexes and microtubules involves multiple proteins including 'cellulose synthase interacting 1' (CSI1), 'companion of cellulose synthase' (CC) and 'cellulose synthase-microtubule uncoupling' (CMU) proteins, as shown in (B). Pectins and hemicelluloses are typically synthesized in the Golgi and delivered by exocytosis, often passing through the trans-Golgi network. Enzymes and other proteins modulating cell wall polysaccharides are delivered by exocytosis. The schematic in (C) highlights how most vesicular transport in plants is mediated by the actin cytoskeleton and involves myosin; some vesicles travel on microtubules mediated by motor proteins in the kinesin family. In (D), selected protein links between cell wall glucans, plasma membrane and the cytoskeleton are highlighted. Cell wall-associated kinases (WAKs) are anchored in the plasma membrane and have an extracellular domain that reaches into the cell wall, specifically linking pectins or oligogalacturonides, to trigger distinct cellular responses through the activation of different MAP kinase cascades via their cytoplasmic kinase domain. Arabinogalactan proteins (AGP) bind to hemicellulose and pectins and can be either anchored in the membrane via their GPI anchor or secreted into the cell wall. Both forms provide adhesive or positional cues. AGP are also suspected to be linked to cortical microtubules, but the details of the linking mechanism remain unclear. Proteins like COBRA and enzymes like glucanases are located in the plasma membrane and link to cellulose. They play a role in modifying the cellulosic network by either facilitating the interactions between glucan chains or cleaving the cellulose fibrils. Phospholipase D (PLD) is located in the plasma membrane and binds to both actin (shown in (C)) and microtubules (shown in (D)); they are prime candidates for the transmission of extracellular signals (D).

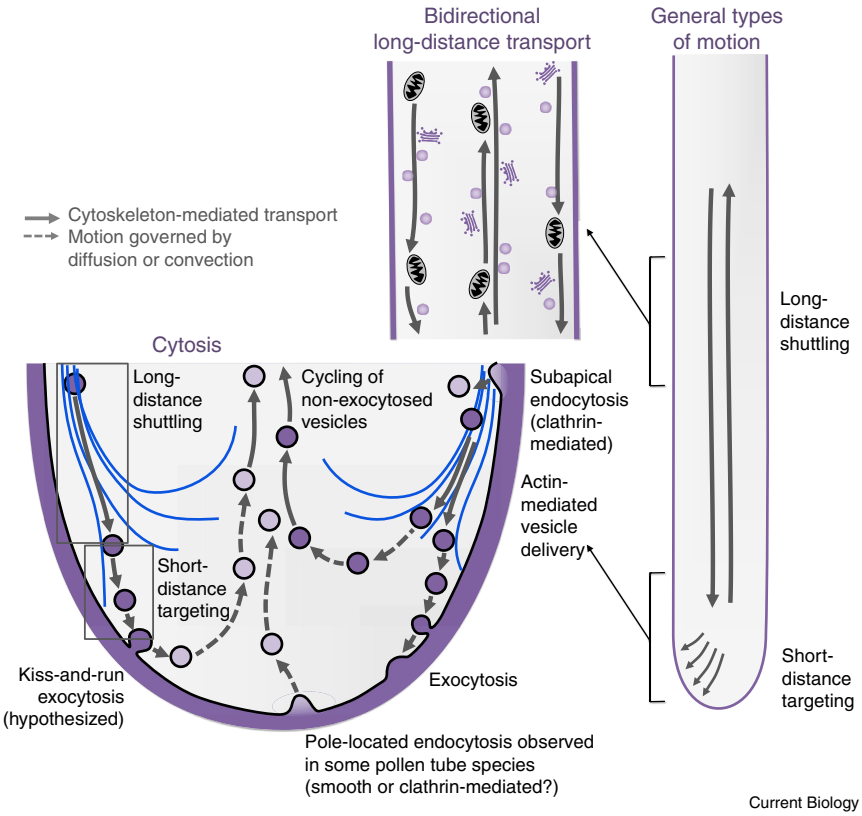

Figure 2. Types of intracellular transport.
Long-distance organelle transport in plant cells relies mostly on actin-myosin-based mechanisms and occurs typically on bundles of actin filaments. Some organelles may be shuttled via a kinesin-microtubule-based mechanism. Targeting vesicles to a precisely defined site at the cell envelope is carried out on highly dynamic individual actin filaments. Some vesicle transport to the site of exocytosis may also be accomplished by convection. The drawing illustrates these patterns using the example of an angiosperm pollen tube.

that produce cellulose (cellulose synthase A, CESA) and callose directly at the cell surface are delivered to the plasma membrane by vesicular transport followed by exocytosis. Various vesicular pathways exist between the endoplasmic reticulum or Golgi and the cell surface: conventional secretion typically involves the trans-Golgi network[11], but can bypass it[12], whereas unconventional pathways can be based on the exocyst-positive organelle, multi-vesicular bodies, or 'small cytosolic CESA compartments'[13,14]. Both actin and microtubules are implicated in cargo vesicle delivery, but to different extents depending on cell type. Actin-based transport is propelled by the motor-protein myosin XI[15,16], as shown in a variety of plant systems. Acto-myosin mediated transport occurs also in fungal systems (to which we will allude occasionally in this review) even though their cell wall biochemistry differs from plants, as do the respective roles of actin and microtubules. Efficient cargo delivery can involve a combination of rapid mass transport over long distances and slower but delicately controlled guidance of vesicles to the exact sites of exocytosis (Figure 2). Long-distance vesicle motion typically occurs along thicker actin bundles, whereas final targeting is mediated by finer bundles or highly dynamic individual actin filaments[17,18]. The drag forces generated by actively transported vesicles and organelles entrains the cytosol, causing a general motion that is visible as cytoplasmic streaming, which mixes the cytoplasm[19–22]. The resulting convection has also been suggested to deliver vesicles to their target exocytosis site[23] (Figure 3). Although acto-myosin-mediated vesicle transport has been described and quantified for many cell types, the mechanisms underlying the specificity of the vesicular transport in plants have remained largely unknown. Haraguchi *et al.*[24] proposed an affinity-selective transport model in which specific vesicles are selected for transport based on their affinity to a specific actin–myosin complex. Different vesicles might also travel on different types of actin arrays[25]. Whether different polysaccharides, proteins, and enzymes are systematically segregated into different cargo vesicles or travel sometimes or always in the same vesicles remains elusive.

Plasma membrane-located synthesis

In the plasma membrane, polysaccharide synthesis is performed by enzymes moving in the planar space of the membrane (Figure 1A,B). These enzymes recruit precursors supplied in the cytosol, assemble them into polysaccharides and immediately extrude the elongating polymers into the apoplastic space. Cellulose and callose are produced through this processive, on-site mechanism. Cellulose consists of repeating glucose residues linked by β(1,4)-bonds, and the linear polymer chains typically coalesce to form crystals in the shape of rigid microfibrils. Callose is also a linear homopolysaccharide composed of glucose residues but they are linked through β(1,3)-bonds and do not crystallize. The synthesizing enzymes for both polymers are thought to be activated only upon their insertion into the plasma membrane, but in some fast-growing cell types the activation and initiation of membrane-located polysaccharide synthesis may begin in the cargo vesicles[26].

Cellulose is synthesized by protein clusters formed from multiple CESAs. Each of these cellulose-synthase complexes appears to be comprised of 18 CESA proteins[27,28]. Cellulose-synthase complexes deposit cellulose microfibrils just outside the plasma membrane onto the inner face of the cell wall. The process is similar for callose, which is produced by callose synthases or glucan synthase-like proteins[29–31]. Whereas cellulose is produced in all plant tissues, callose is only synthesized in specialized cells, during certain developmental phases, or in response to certain external stimuli. Glucan synthase-like genes are therefore expressed in a more pronounced, tissue-specific, and temporally changing manner than CESA genes. The motion of cellulose-synthase complexes in the plasma membrane is known to be propelled by the processive synthesis process and guided by microtubules (as detailed below). Whether the same applies to callose synthases is poorly understood, although there is evidence for their association with microtubules[32].

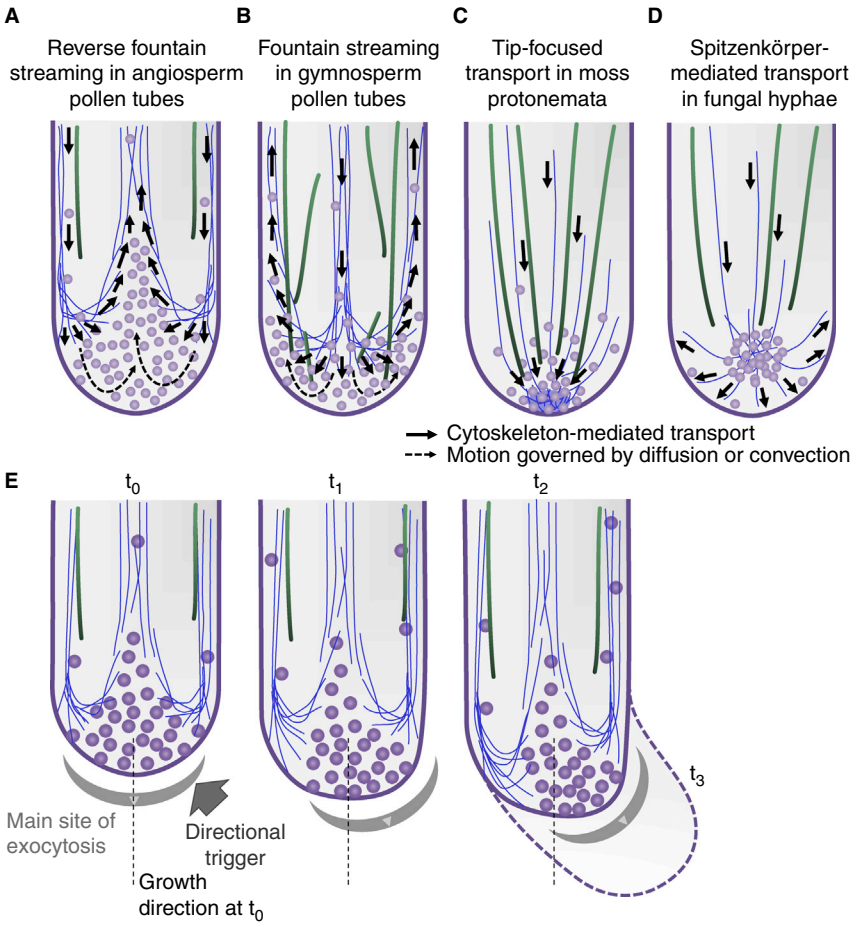

Figure 3. Motion of vesicles and arrangement of cytoskeletal arrays in tip-growing cells.
(A) Angiosperm pollen tubes display inverse fountain-like cytoplasmic streaming of vesicles (light purple spheres) largely choreographed by actin filaments (thin blue lines). Microtubules (thick green lines) are absent in the growing tip. (B) In gymnosperm pollen tubes, organelles move in a fountain pattern. Microtubules are enriched in the tip. (C) Moss protonemata are characterized by an apical actin cluster. (D) Fungal hyphae typically have a Spitzenkörper from where vesicles are dispatched to the apical cell surface. (E) In a straight-growing angiosperm pollen tube, the actin cytoskeleton polymerizes near the apex to extend the apical array in lockstep with the growth of the cell envelope. Upon receiving a directional cue, the apical actin array polymerizes asymmetrically, resulting in asymmetric delivery of vesicles and a reorientation of the direction of cellular expansion.

The cell wall–membrane–cytoskeleton continuum

Due to their respective locations inside and outside the plasma membrane, the interaction between cytoskeleton and cell wall inevitably involves and crosses the plasma membrane. The structural continuum of these three cellular components involves multiple players allowing information to be transmitted in both inward and outward directions. This tight coupling is evident from mutations or pharmacological interference that act on one side of the plasma membrane and effect changes at the opposite side. For example, the *Arabidopsis thaliana act2act7* actin double mutant shows decreased cellulose content and non-uniform cell wall thickness[33]. Inversely, treating mature root epidermal cells with isoxaben, a herbicide inhibiting cellulose biosynthesis in higher plants, affects actin filament dynamics[33,34]. How direct or indirect these effects are remains to be investigated. The players involved in the continuum traversing the plasma membrane are either transmembrane proteins or act near the plasma membrane on the symplastic (inside the plasma membrane) or apoplastic side (Figure 1B–D).

Spatial heterogeneity in the interactions between cell wall, plasma membrane, and cytoskeleton

The nature of the interactions between cell wall, plasma membrane, and cytoskeleton varies significantly across the cell surface. Examples are the formation of Hechtian strands — where the plasma membrane stays attached to the cell wall at certain locations but easily detaches in other regions — and the highly polar distribution of cell wall assembly in tip-growing cells[26]. Precise spatial domain identity is also required to control cell behavior at cell edges[35] or selected cell faces[36]. Targeted delivery ensures the local enrichment of a given molecular player, but what controls and confines the spatial distribution and lateral movement of plasma-membrane-located proteins once inserted at the target site at the cell surface? Plasma-membrane-located proteins are known to diffuse within the plane of the membrane. This diffusion is influenced by interactions with other proteins, with the cytoskeleton or with lipid nanodomains[37,38]. Using fluorescence recovery after photobleaching (FRAP) and total internal reflection fluorescence (TIRF) microscopy on *Arabidopsis* seedlings and *Nicotiana tabacum* leaf protoplasts, Martinière *et al.*[39] showed that protein diffusion in the plasma membrane is also constrained by the presence of the cell wall. Consistent with this, plasmolysis or enzymatic removal of the cell wall renders largely immobile proteins mobile, even those with larger extracellular domains. This was demonstrated in *Arabidopsis* root-epidermal cells, where plasmolysis resulted in the loss of polarity in the distribution of PIN proteins[40]. Tight connections between the cell wall and the plasma membrane, therefore, limit the lateral diffusion of proteins and contribute to the establishment and regulation of distinct regions in the plasma membrane, analogous to tight junctions in animal cells[41].

In rapidly growing cells, the maintenance of plasma-membrane polarity seems particularly challenging because vigorous exocytosis constantly modifies the cell surface. In pollen tubes, polarity maintenance involves the activity of plant Rho GTPases (ROP proteins). ROPs are inserted in the pollen-tube apical plasma membrane and then sequestered back into the cytosol in the sub-apical region by Rho GDP-dissociation inhibitors (RhoGDIs), thus confining plasma membrane-located ROPs to the growing region[42,43]. ROP activity is regulated by the action

of activating (RhoGEF, RhoGAPs) and inhibiting (RhoGDIs) proteins. Downstream effectors of ROP signaling include AtRIC1, which severs actin filaments at the apical membrane[42,44], as well as AtRIC4 and AtRIC3, which mediate actin assembly and disassembly by stimulating an influx of Ca^{2+} into the cytoplasm[45,46]. The interplay between these agents is crucial, and mutations in any of these result in abnormal ROP distribution at the apical membrane, leading to perturbed apical vesicle exocytosis, which in turn alters the spatial distribution of cell wall components and the cell growth pattern[47].

Cell wall assembly during cell growth and morphogenesis

It seems intuitive that plant cell growth must require the assembly of additional cell wall surface. However, the causality between material supply and cell expansion is complex. A first step involves the loosening of the existing cell wall resulting in the relaxation of wall stress. This is achieved by enzymatically modifying the bonds between existing cell wall polymers or through the action of wall-loosening agents such as expansins[2,48]. This relaxation of the wall stress reduces cell turgor, inducing water uptake by osmosis, which in turn stretches the cell wall and restores the turgor. Without the supply of additional wall material, continuous stretching would lead to wall thinning and eventually rupture. To sustain the expansion process, new material must be supplied to the expanding surface. The principal role of delivery of new cell wall polysaccharides during cell growth is, therefore, the maintenance of the integrity and the thickness of the expanding wall; it is not the physical driver of the expansion.

This concept has recently been challenged by Haas *et al.*[49], who suggested that cell growth could also be initiated by local swelling of the cell wall, independently of turgor. This was based on combining chemically induced swelling of the cell wall[50,51] with the notion of homogalacturonan 'pectin nanofilaments'[52,53]. The authors interpreted patches of immunofluorescence signal in the anticlinal walls of pavement cells observed using direct stochastic optical reconstruction microscopy (dSTORM) to correspond to vertically oriented pectin nanofilaments. They proposed that these expand both in diameter and spacing upon de-esterification, leading to cell growth and shape formation. Oddly, these nanofilaments were not observed in the periclinal walls of the same cells despite the physical continuity between anticlinal and periclinal walls and although the latter are generally considered to be crucial in pavement-cell morphogenesis[54–56]. Alternative explanations for the patchy pattern of pectin label in the anticlinal walls, such as nano-wrinkles or locally heterogeneous distribution because of the presence of well-documented vertical cellulose microfibrils[57,58], were not further explored and, therefore, true cause–effect relationships remain to be explored. The lack of consensus around the fundamental underpinnings of plant cell growth points to the need to address basic questions with regards to cell wall biochemistry and cell differentiation[55,56].

Rather than swelling, the widely accepted concept behind the formation of complex cell shapes relies on spatio-temporal regulation of turgor-driven cell-expansion events. This entails control over the sites not only where expansion is facilitated through cell wall loosening but also where it is prevented. The latter requires strategic stiffening of the wall. Combined, the spatially coordinated softening and stiffening of the cell wall is responsible for the generation of the kaleidoscopic diversity of plant cell shapes ranging from the longitudinal cells found in stems and roots to the intricate jigsaw puzzle-shaped cells in the leaf epidermis of many plant species[59]. The local modification of cell wall properties determines whether cell-surface expansion is uniformly distributed over the cell envelope or whether it occurs at specific locations creating a non-uniform distribution of the expansion pattern[60] (Figure 4). It also regulates whether the expansion of a given unit element in the cell envelope occurs equally in all directions (isotropically) or preferentially along one axis (anisotropically)[61]. This cell wall-focused concept of plant cell growth is crucial since the driving force for cell expansion, the turgor, is virtually uniform within the cell volume and, as pressure is a scalar, it is non-directional.

Morphogenesis through non-uniform expansion

One way to locally modulate cell wall expansion is through pectin chemistry. Pectins are exocytosed to the apoplastic space in a highly methylesterified form. The chemical configuration can then be modified post exocytosis through de-esterification, Ca^{2+}-mediated gelation, or acetylation[62,63]. These molecular changes alter the rheology and deformability of the pectin material and can be controlled at subcellular scale to generate spatially heterogeneous mechanical properties of the cell wall that underlie growth patterns at both subcellular and organ levels[57,64–66].

In no other cell type is the non-uniform cell-expansion pattern as dramatic as in tip-growing cells. These unidimensionally elongating, cylindrical cells include pollen tubes, root hairs, fibers, and tip-growing moss cells such as protonemata. In the growing tip of the pollen tubes, pectin is highly methylesterified, whereas in the non-growing flank regions it becomes de-esterified and thus stiffer because of gelation by calcium ions[67]. The continuous addition of softer, methyl-esterified pectin to the polar growth site and the gradual gelation ensure the cylindrical geometry of the cell shape[26,64]. In cells with more complex shapes (Figure 4), such as those forming the jigsaw puzzle-like leaf epidermis, similar principles are employed and this is reflected in a heterogeneous distribution of pectin at growing and non-growing regions[57,68].

In most tip-growing cells, including those of fungal and oomycete hyphae, the delivery of cell wall material is executed by exquisitely choreographed cytoplasmic arrays of actin filaments[69,70]. Pharmacological interference with the actin cytoskeleton immediately depolarizes or halts tip growth in pollen tubes, fungal hyphae, and moss protonemata[71–73], and myosin XI-deficient mutant moss lines display stunted protonema growth with small spherical cells[74]. The abundance of actin filaments in the apical zone of the pollen tube also correlates temporally with changes in the growth rate observed in tubes displaying oscillatory growth[75], a phenomenon that is abolished upon pharmacological slowdown of actin polymerization[76]. Even slight manipulation of actin dynamics compromises the pollen tube's ability to overcome mechanical obstacles, and fungal hyphae to form branches[71,72]. These observations underline the crucial role of the actin network in maintaining tip growth by ensuring the targeted delivery of cell wall material. Interestingly, the architecture of the actin arrays varies significantly between different tip-growing cell types, although all result in a perfectly cylindrical

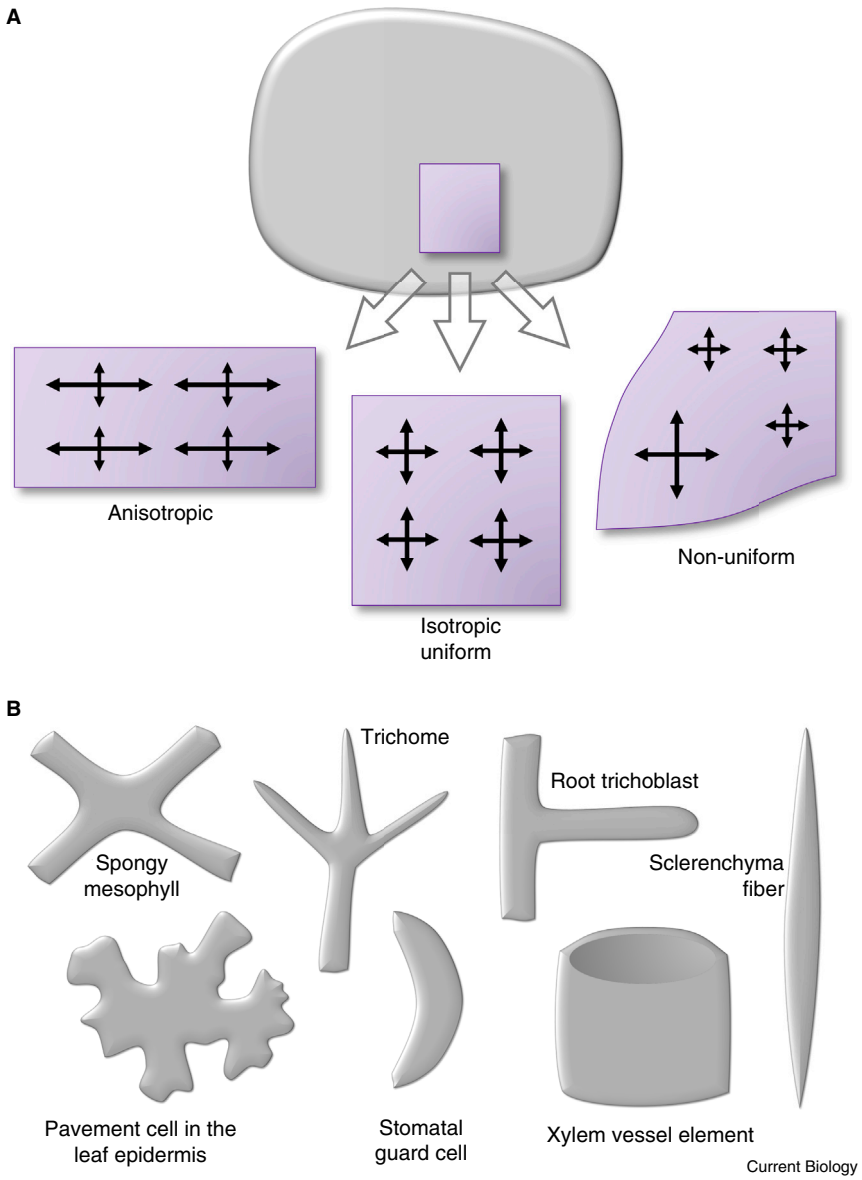

Figure 4. Expansion during plant-cell morphogenesis.
(A) The cellular envelope can expand uniformly with identical rates over its entire surface or non-uniformly with locally differing rates. The expansion of a given surface section can be isotropic (equal in all directions) or anisotropic. (B) When combined, these expansion modes can create any shape within the kaleidoscopic collection of plant-cell types.

cell shape[77] (Figure 3A–D). Importantly, the locations and mechanisms of actin polymerization differ. Kroeger *et al.*[18] hypothesized that actin filaments located in the shank of the angiosperm pollen tube extend from the shank into the tip region with their polymerizing tips pointing toward the apical vesicle cone, but Qu *et al.*[78] have suggested that actin nucleates at the apical plasma membrane instead. As the angiosperm pollen tube features two distinct actin arrays, one that aligns with the central axis and guides retrograde organelle transport and another one consisting of peripherally arranged cables mediating anterograde transport, these concepts are not mutually exclusive. However, the complexity of the transport logistics opens interesting questions regarding the choreography between polymerization of cytoskeletal elements, expansion of the cell surface, and the increase of the cell lumen. Other questions pertain to the coordination between exocytosis and endocytosis. In tip-growing cells, vesicle-mediated delivery of cell wall precursors adds a large excess of membrane material to the cell envelope. Because of the need to quickly reincorporate this membrane material, endocytosis is highly active near the growth site and a kiss-and-run mechanism has been proposed[79], but evidence remains elusive (Figure 2).

The actin cytoskeleton not only ensures the supply of vesicles carrying cell wall precursors, but also regulates the precise direction of the growth process by defining the exact location of exocytosis. In both pollen tubes and fungal hyphae, the actin cytoskeleton assumes an asymmetric configuration prior to a visible reorientation of growth[71,79–81] (Figure 3E). In fungal hyphae, this is readily detected since the Spitzenkörper[82], a prominent cytoplasmic structure comprising vesicles and cytoskeletal elements in the apical region of hyphae (Figure 3D), assumes a lateral position that initiates a change in growth direction[83]. Similarly, a spherical apical actin accumulation marks the direction of tip growth in moss protonemata[84] (Figure 3C). The ability to change growth direction allows tip-growing cells to respond to directional cues — an essential skill that enables them to expand the size of the entire organism (hyphal mycelium, protonemata developing into moss gametophyte), find nutrients (hyphae) or reach targets (pollen tubes).

The role of the microtubule cytoskeleton in tip growth is less clear and varies by cell type. In angiosperm pollen tubes, microtubules do not reach into the apical growth zone, and their depolymerization does not prevent polar cell elongation although it compromises the control of growth directionality[71]. In fungal hyphae, microtubules are prominent in the Spitzenkörper[85] and contribute to vesicular transport[83]. Using micropatterned microfluidics devices, Held *et al.*[86] demonstrated that fungal hyphae rely on the presence of a functional Spitzenkörper–microtubule complex to efficiently navigate physical obstacles. In moss protonemata, microtubules converge just below the apical actin cluster and seem to participate in directional steering (Figure 3C). Microtubule-stabilizing drugs perturb the dynamics of these actin clusters and result in defects in cell expansion,

thus suggesting a close association of microtubules and actin in steering polarized growth[84]. Unlike pollen tubes, cell polarity in root hairs is strongly dependent on microtubules[87]. Depolymerization of endoplasmic microtubules in *Medicago* root hairs abolishes the polar distribution of cytoplasm and interferes with the carefully controlled distancing between the nucleus and the growing tip, leading to a reduction of growth rate[88]. In *Arabidopsis* root hairs, pharmacological interference with microtubules causes a wavy pattern, consistent with their role in steering growth through the manipulation of the apical cell wall[89,90].

Anisotropic growth through directional cell wall reinforcement

The molecular network of the plant cell wall can be arranged to confer anisotropic material behavior. This occurs when the main stress-bearing components are oriented to display a directional bias. This concept is employed, for example, to generate the cylindrical cells in the shoots and roots. By reinforcing these cells along the hoop orientation, turgor-driven yielding of the cell wall is directed into the perpendicular axis generating cylinders[91,92]. The anisotropic stiffening is achieved by directionally biased deposition of cellulose microfibrils[2,60,93].

Because spatial control of microfibril deposition is exerted by cortical microtubules, interference with microtubule dynamics affects cell wall microstructure and, consequently, cell shape. A typical phenotype resulting from pharmacological or genetic interference with microtubule dynamics is a radial swelling in cell types that normally form narrow cylinders. Such aberrant cell swelling is observed, for example, in mutants with defects in katanin-like proteins, which are essential for normal patterning of cortical microtubules during the first steps of cell elongation[94,95]. Organ elongation, polarity, and patterning are affected in different *Arabidopsis* mutants in which cortical microtubule arrangement is altered: the *fra2* mutation affecting the AtKTN1 gene shows an increase in cell width and root diameter[96], mutations of ANGUSTIFOLIA (a member of the CtBP gene family) and ROTUNDIFOLIA3 (cytochrome P450) genes alter leaf shape and their polarity-dependent elongation pattern, and tubulin mutants (*tua* and *tub* mutants) show severe anatomical and morphological phenotypes, such as cytokinesis defects, disconnected vasculature, deformed trichomes and root hairs, isotropic cell expansion and twisted growth of elongating organs[97,98]. The spatial organization of cortical microtubules is thus key in determining cell and organ shape by way of controlling how cellulose microfibrils are deposited in the cell wall[99].

Cell plate formation

Cytokinesis in plants is not only fundamental for cell proliferation but also a crucial regulator of tissue and organ architecture. Since plant cells are cemented by the middle lamella, rendering them immobile within the tissue, the orientation of cell division becomes pivotal in tissue morphogenesis and differentiation. In plants, cytokinesis involves *de novo* assembly of a cell wall in the center of the dividing cell (Figure 5). The newly developing wall precursor — the cell plate — eventually fuses to the parental wall to divide the daughter cells, but its formation is initiated at a location in the cell lumen where no existing template exists onto which new cell wall material could be 'plastered'. Instead, a cytoskeletal scaffold establishes the target site for the initiation of the new wall through vesicle fusion. The phragmoplast forms after the anaphase[99–101] and is composed of bipolar arrays of actin filaments, microtubules and the cell plate assembly matrix — an amorphous scaffold consisting of proteins and vesicles. Golgi-derived vesicles containing cell plate precursors and proteins move along the polarized phragmoplast microtubules toward their interdigitated plus ends[102]. The vesicles initially fuse to each other and then to the growing reticulate structure to deliver their content to the developing tubulovesicular network. The actin filaments form two opposing sets that are oriented parallel to the microtubules, connecting the phragmoplast to the parental cell cortex and restraining the accumulation and fusion of vesicles to the midzone of the cell plate[103,104]. Using tobacco BY-2 cell culture lines and *Arabidopsis* root epidermal cells, van Oostende-Triplet *et al.*[105] showed that cell-plate development entails three distinct phases: an initial plate assembly phase is followed by primary and secondary centrifugal growth stages. These stages are characterized by different growth rates of the cell plate and involve different cytoskeletal elements. Pharmacological inhibition of cytoskeleton polymerization revealed that microtubules but not actin are required in the early rapid phases of the plate expansion. Although initially not required for vesicle delivery, the actin network confines the vesicle delivery area[105]. The transition to the secondary centrifugal phase requires restructuring of the phragmoplast cytoskeletal elements. Centrally located microtubules and cell plate assembly matrix are dismantled, resulting in a ring-shaped phragmoplast that subsequently widens, focusing vesicle trafficking to the periphery of the expanding cell plate[106]. Pharmacological interference with actin polymerization during the secondary centrifugal growth stage reduces the expansion rate and prevents the cell plate from fusing with the parental plasma membrane[105,107].

Fine regulation of deposition of cell wall polysaccharides by the cytoskeleton is critical for cytokinesis. At the early stages of the cell plate development, callose is synthesized directly at specific locations of the phragmoplast. Although present in small quantities and replaced with cellulose at later stages, timely deposition of callose is essential to cell plate formation[108,109]. Callose synthase mutants of *Arabidopsis* and tobacco BY-2 cells show defects in cell plate formation and insertion, resulting in multinucleated cells with cell wall stubs consistent with their role in early cell plate development[99,108,110–112]. At later stages, cellulose dominates and treatment of cultured cell lines of BY-2 and *Arabidopsis* with dichlobenil (2,6-dichlorobenzonitrile), a specific inhibitor of cellulose synthesis, arrests the maturation of the cell plate[113,114]. Delivery of CESAs occurs through phragmoplast-associated vesicles regulated by microtubule-associated proteins[115,116]. Despite the multitude of details established for plant cytokinesis, many questions remain. For example, what are the processes that ensure the formation of a perfectly flat, circular cell plate rather than a spherical structure? How do the cytoskeletal arrays coordinate the transitions between the cell plate expansion phases? Are different cargoes delivered in the same or in different types of vesicles? These are pieces of the puzzle that will be crucial to our understanding of cytokinesis and plant development at the cellular scale.

Mechanoregulation

Although the cytoskeleton is key in the mechano-chemical machinery delivering wall building and modifying materials, inversely,

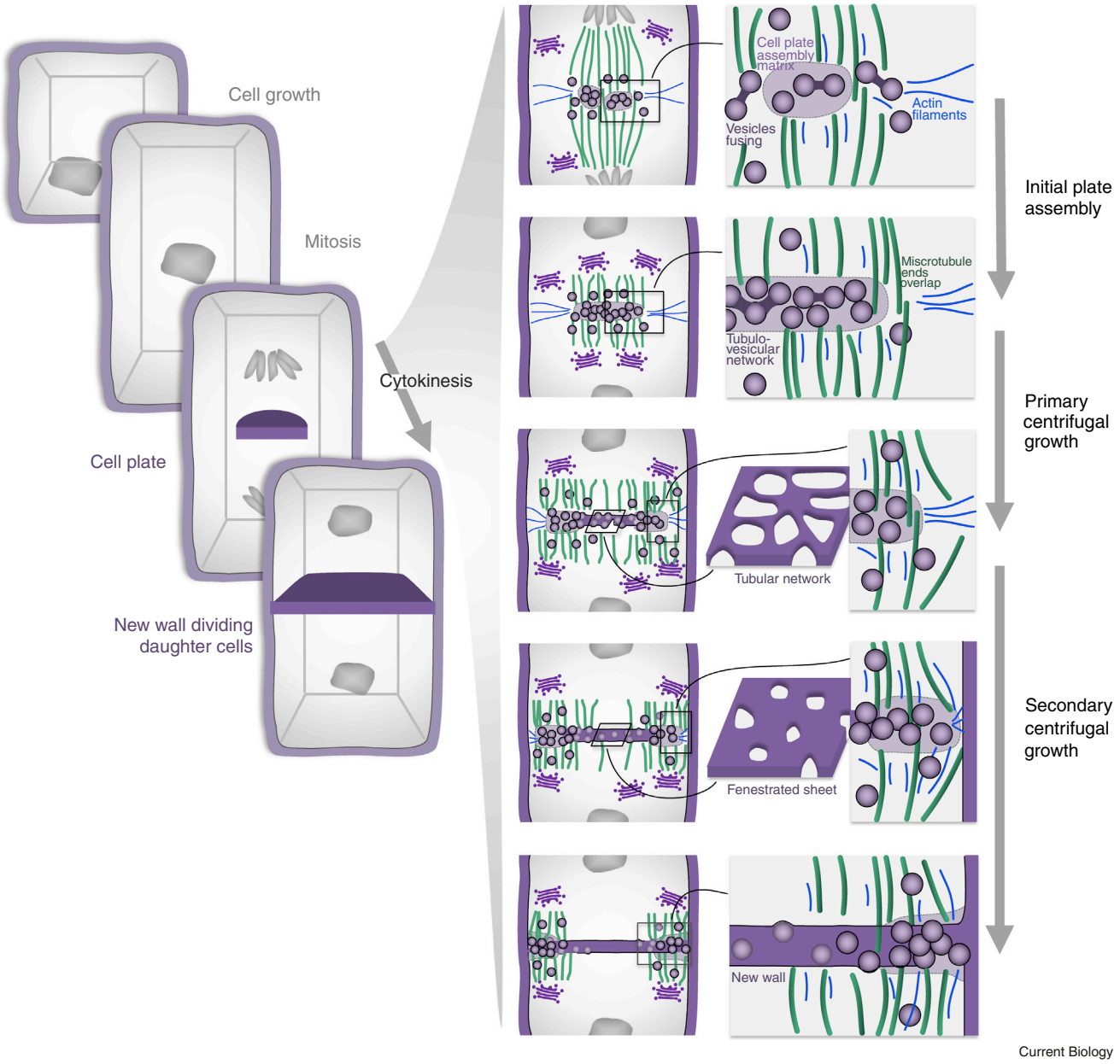

Figure 5. Cytokinesis in plants.
Initiation of a new cell wall separating daughter cells occurs through the cytoskeleton-mediated transport of vesicles carrying cell wall precursors and synthesizing enzymes to a central location within the cell lumen. Vesicles fuse to build a tubulovesicular network. The disk-shaped cell plate expands radially through addition of material to its periphery, while the central region matures, passing through the stages of a tubular network and fenestrated sheet, followed by a continuous planar structure. Eventually, the periphery of the cell plate connects with the parental plasma membrane and cell wall. The process is controlled by cytoskeletal arrays responsible for cargo vesicle delivery and for the positioning of the plate within the cell lumen as well as the spatially correct attachment to the parental wall. Distinct phases in the process differ in their susceptibility to pharmacological interference with cytoskeletal dynamics.

it also perceives changes in the cell wall, thus forming regulatory feedback loops. By controlling cell division and growth, these feedback loops are the basis of cell functioning, the robust control of organ size and shape, integrity maintenance and defense mechanisms[56,117,118]. Local and global scale mechanical signals and changes in the cell geometry are closely related cues that can transmit through the continuum. The longstanding questions are, which mechanical signals do the different cytoskeletal elements actually perceive — stresses or strains? And how are these signals perceived at the molecular level[119–121]? Due to their high bending rigidity, microtubules tend to self-align cortically[122], facilitating coupling with the plasma membrane and cell wall. Comparing directions of growth and maximal stress in different plant cell types, microtubules are suggested to align with stress rather than perpendicular to strain. Yet, important details such as how mechanical stress rearranges microtubules remain largely unknown[122]. Cylindrically growing cells such as those of shoot and root are often used to investigate stress-driven microtubule

organization and cell wall assembly. Elongation growth of cylindrical cells occurs primarily along a single axis, which simplifies the spatial coordinate system needed to analyze and to fully describe the spatial distribution of growth and mechanical stress. The correlation of these parameters with structural features such as the alignment of microtubules and orientation of cellulose microfibrils thus becomes more straightforward compared to more geometrically complex cell shapes. Typically, the orientation of microtubules in elongating cylindrical cells is in hoop direction. However, in some situations, microtubules assume longitudinal orientations[19,123,124], a reorientation that often appears to coincide with growth deceleration[125,126]. Although this temporal correlation might suggest that the deposition of cellulose microfibrils guided by these longitudinal microtubule arrays may terminate cell expansion by stiffening the wall in the direction of growth, no conclusive evidence for a causal relationship is available. The sensitivity of microtubules to geometrical and mechanical cues is also implicated in the orientation of cytokinesis[127]. Here, both geometry and mechanical signals are integrated to determine the location and orientation of the cell plate such that cell, tissue, and organ growth occur in directions that produce specific organ shapes. Changes in the mechanical stress field, for example by wounding or ablation of neighboring cells, result in a reorientation of the microtubule arrays and, consequently, the reorientation of the cell-division planes[127–129]. The reverse mechanical coupling between cell wall and microtubules becomes evident when interfering with cellulose synthesis, or when defects in xyloglucan and arabinogalactans result in altered microtubule organization[130–133]. These extracellular events are transmitted through the coupling between the cell wall and the microtubules. In addition to mechanical cues, microtubules are suggested to reorient in response to the application of hormones such as ethylene[134], brassinosteroid[135] and auxin[136,137]. These relationships can be complex and overlapping. For instance, the effect of auxin on microtubule reorientation is suggested to be indirect and mediated through auxin-induced changes in cell wall mechanics[138]. Inversely, microtubule organization is correlated with PIN1 polarity[139].

The involvement of the actin cytoskeleton on the perception side of plant cell mechanoregulation is not well understood. Actin and microtubules interact[140,141], involving various proteins such as formins[142,143], and the behavior of their combined network might be dominated by their interactions rather than their individual behaviors[144]. Direct interaction between actin and microtubules requires that both be present in the same subcellular region. In interphase cells the interactions would therefore be confined to the extreme cell cortex as microtubules rarely cross the interphase cell lumen. The interaction between the cytoskeletal arrays mutually influences their mechanical behavior. For example, lateral support from the neighboring actin filaments and, in animal cells, intermediate filaments can substantially reinforce microtubules against buckling[145]. In vitro and mammalian-cell studies suggest that mechanical forces result in conformational changes in actin filaments and their binding proteins[146]. Using in vitro actin assays, Risca *et al.*[147] demonstrated that force-induced curvature of the filaments affects their branching. This suggests a way in which cortical actin filaments may perceive mechanical forces transmitted through the continuum and respond by altering their dynamics which, in turn, affects cell wall assembly processes. Altered actin dynamics may also reorganize microtubules, affecting the cell wall assembly indirectly. By forcing mammalian cells to assume specific shapes using 3D microniches, Bao *et al.*[148] showed that the encapsulation geometry alone can organize subcellular units including actin filaments. For instance, actin filaments could be seen to orient longitudinally in capsules, a phenomenon that was reproduced using isolated plant protoplasts[123], indicating a tendency of both microtubules and actin filaments to orient longitudinally when only under geometrical control[123,149]. Based on the behavior of protoplasts, Durand-Smet *et al.*[123] suggested that actin organization relies on microtubules, but not vice versa, corroborating findings made in intact plant tissues[141]. On the other hand, actin filaments are suggested to influence the delivery rate, lifetime, and trajectory of CESA on the plasma membrane, thereby potentially collaborating with microtubules in the regulation of cell wall anisotropy. Actin is also suggested to organize microtubules during the early stages of cytokinesis[150]. Therefore, whether actin filaments or microtubules lead the organization in response to mechanical cues may be situation dependent. Interestingly, Branco *et al.*[151] reported that the actin cytoskeleton aggregates beneath the location of experimentally induced nanoindentation in epidermal cells of *Arabidopsis* hypocotyls. The resulting actin response occurred at forces and in time frames considerably smaller than those required for microtubules[152,153]. Furthermore, similar to microtubules, changes in cellulose synthesis and the cell wall also affect actin organization[34,154]. These findings raise the possibility of an actin-based sensory mechanism in plants that, similar to microtubules, perceives mechanical cues, for example due to pathogen invasion[155], and responds by ensuring immediate vesicle supply of relevant material such as callose synthases to the sites of infection[156]. Vesicles can be coupled with, and move along, actin filaments; alternatively, their motion through the cytoplasmic space may result from hydrodynamic flow generated by the active cytoskeleton-mediated transport of bigger organelles[18,157].

Sustained cell growth, functional cell–cell adhesion, and the response to biotic and abiotic stressors require the maintenance of the cell wall. Surveillance of cell wall integrity is under mechano-chemical control and involves signals, sensors, and pathways to perceive and respond to changes in the continuum and turgor[158]. Mechanical signals regarding the cell wall's integrity can arise in the form of stresses or strains that cause tension, compression, or shear in and between the wall-polymer network, the cell membrane, and the cytoskeleton. Turgor is involved in the adherence between the elements of the continuum. Detachment of the membrane from the cell wall due to turgor drop may act as a mechanical signal, as it can alter the mechanical state of the wall and the membrane, giving rise to localized stresses at limited adhesion sites such as Hechtian strands. Thus, changes in turgor can be both a response and a signal. Mechanical signals generated in the cell wall network can be transmitted to the membrane via membrane-linked proteins such as COBRA which is linked to cellulose microfibrils[159]. Proteins with transmembrane domains, such as cell wall-associated kinases[160] and FERONIA[161], that preferentially bind pectins may directly transmit cell wall mechanical signals to the cytoskeleton. Information about cell wall integrity can also be transmitted to the microtubules through their association with cellulose-synthase

complexes and proteins such as CSI1/POM2[162], leading to the recruitment of microtubule-based machinery for mechanoregulation of the cell wall in response to biotic and abiotic stress factors[163–167]. Given the crosstalk between the cytoskeletal arrays[144], it is reasonable to hypothesize that mechanical signals can be transmitted through the same route to the actin network. Because of direct mechanical coupling with the cell wall, substantial changes in the cell wall microrheology can also be transmitted to the membrane, thus activating mechanosensitive ion channels[168] without relying on cell wall-binding protein linkages.

It is known that plant cells are able to detect the presence of residues from chemical damage, such as cellobiose or oligogalacturonides resulting from fungal damage to cell wall polysaccharides, and respond by rearranging the cytoskeleton, altering cell wall synthesis, and/or depositing callose or lignin[169,170]. We wonder, therefore, whether the newly generated surfaces and exposed polymer fragments due to mechanical damage might similarly initiate an immune response. Our understanding of cell wall-integrity-related proteins acting as specific links between the cell wall polymers, membrane, and the cytoskeleton is surprisingly limited[158,171,172]. We speculate that mechanical signals are likely to be sensed through multiple pathways due to crosslinking and interactions between the constituents of the continuum. This adds another level of complexity to efforts aimed at unraveling the details of mechanosensation. Alterations in the synthesis or chemistry of a certain wall polymer can give rise to changes in wall nano-rheological properties and redistribution of the mechanical stress in the network which, in turn, can be picked up by any sensory protein linking other polymers. Disruption of SOS5, a membrane-linked protein, is suggested to affect both pectin organization and cellulose synthesis in seed coat mucilage[173,174]. Therefore, it can be envisaged that changes in pectin rheology, for example through de-esterification and calcium crosslinking, may lead to mechanical changes in the cell wall, eliciting a response in the form of microtubule and cellulose reorganization[48,57]. Compensatory mechanisms in response to genetic or pharmacological alterations of cell wall polysaccharides or stress factors likely work through the same routes to restore the multiscale mechanical homeostasis of the continuum. This mechanistic view may explain the response of cells to wounding, acclimation to stress factors, and the observed plasticity challenging the targeted cell wall modifications[175–177]. Time scale is likely a key parameter in the maintenance of cell wall integrity, and mechanoregulation not only governs the viscous mechanics of the cell wall but also is inherent to molecular processes regulating cell function. In this way, mechanoregulation of cell wall integrity and assembly accommodates both long-term and acute events, for example growth and response to wounding, and also adds to the intricate interplay between heterogeneous cell wall mechanics, unidentified mechanical signals, elusive linking proteins and complex cytoskeletal dynamics. These speculations may point to pieces of an intricate machinery in which cytoskeletal elements work in tandem, integrating hormonal fields, geometrical control, and mechanical rules to regulate cell wall synthesis, to maintain its integrity and to generate robust organ shapes[178]. These hypothetical concepts also point to the need for models that incorporate and yield information such as non-uniform strains and stresses forming in the cell wall polymer network and functional interactions between and among the cytoskeletal and cell wall components.

Concluding remarks

The cytoskeleton is at the core of plant cell development by mediating delivery of cell wall precursors, providing the scaffold for cell-plate formation, and by occupying a central role in feedback loops required for regulation of cell division, growth, immune response, and integrity. However, our understanding is surprisingly limited when it comes to the mechanistic details of these processes. For example, the specificity of the cytoskeletal cargo delivery system as to the type of vesicles and the mechanisms enabling exact spatial targeting remains poorly understood. The mechanoregulation of plant cells poses challenges and opportunities for interdisciplinary research. Cell biological approaches must be used to inform and are also guided by mechanical models to account for the heterogeneous mechanical properties of the cell wall, the detailed mapping of structural proteins and mechanosensitive ion channels acting as mechanical sensors throughout the continuum. Similarly, characterizing the interactions between microtubules and actin will be a key step in understanding how the cytoskeletal array as a whole senses stress, and how it orchestrates cell wall assembly and integrity. A better understanding of how the spatiotemporal regulation of cytokinesis and cell expansion integrates hormonal and mechanical stress fields with cytoskeletal dynamics puts the cytoskeleton at the center stage of plant developmental research.

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AUTHOR CONTRIBUTIONS

All authors contributed to the writing; A.G. and Y.C. prepared the figures; A.J.B., Y.C. and A.G. reviewed the final manuscript.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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