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"Shape of Cell"—An Auxin and Cell Wall Duet

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ABSTRACT

Understanding the mechanisms underlying cell shape acquisition is of fundamental importance in plant science, as this process ultimately defines the structure and function of plant organs. Plants produce cells of diverse shapes and sizes, including pavement cells and stomata of leaves, elongated epidermal cells of the hypocotyl, and cells with outgrowths such as root hairs, and so forth. Plant cells experience mechanical forces of variable magnitude during their development and interaction with neighboring cells and the surrounding environment. From the time of cytokinesis, they are encaged in a complex cell wall matrix, which offers mechanical support and enables directional growth and a differential rate of expansion towards adjacent cells via its mechanochemical heterogeneity. The phytohormone auxin is well characterized for its role in cell expansion and cell elasticity. The interaction between dynamic auxin redistribution and the mechanical properties of the cell wall within tissues drives the development of specific cell shapes. Here, we focus on the regulatory feedback loop involving auxin activity, its influence on cell wall chemistry and mechanical properties, and the coordination of cell shape formation. Integrating insights from molecular and cell biology, biophysics, and computational modeling, we explore the mechanistic link between auxin signaling and cell wall dynamics in shaping plant cells.

1 | Introduction

Cells are the basic building blocks of life, yet they exhibit remarkable diversity in shape (Liu et al. 2021; Luciano et al. 2022). Unlike animals, plant cells are immobile as they are encased in cell walls; thus, cell growth and shape formation depend entirely on controlled expansion and division rather than movement. A newly formed cell, created after cell division, typically has a simple, cube-like shape with straight borders and minimal curvature. This initial shape contrasts significantly with the specialized forms seen in mature plant organs. For young cells to develop into their final shapes, they must grow in size while also adjusting their proportions. This requires differential growth across the cell surface, with certain regions expanding more than others (Wang et al. 2022). This concept of differential cell growth was originally proposed by Paul Green, based on his work on the growing cells of *Nitella*, a green alga, which displayed distinct cell expansion behaviors, such as the isotropic expansion of apical cells and the anisotropic, longitudinal expansion of internodal cells (Green 1965). In plants, variations in cell shape across different plant organs are achieved by a fine-tuned interplay of genetic variations under environmental pressures (Cook et al. 2008; Wang et al. 2022). This highlights that cell shape acquisition is not just a passive feature but an active participant in the ongoing process of plant development (Wang et al. 2022). Consequently, cell shape plays a fundamental role in plant survival, regulating how they grow, interact with their environment, and adapt to different stresses (Mathur 2004).

Plant cell growth can be understood as the yielding of the cell wall, a complex network of polysaccharides outside the plasma membrane, to the turgor pressure exerted by the cell's vacuole. Since this internal pressure is uniformly distributed, the direction of growth is determined by the anisotropic flexibility of the cell wall, which allows for targeted expansion in specific directions (Green 1962; Baskin 2005; Braidwood et al. 2014; Ali et al. 2023). The plant cell wall is composed of cellulose, hemicelluloses, and pectins (Cosgrove 2024). Cellulose microfibrils are cable-like structures that resist the stretching forces, and their orientation determines the direction of cell expansion. Hemicellulose provides additional structural integrity by crosslinking the cellulose microfibrils. Pectin composition determines the wall's flexibility and porosity (Cosgrove 2024). The composition of the cell wall and its properties vary depending on the plant type, tissue, and developmental stage (Sarkar et al. 2009). Unlike the secondary cell wall, which is more rigid, the primary cell wall remains relatively thin and adaptable, enabling young or growing cells to expand and develop as needed. In the last decade, mechanical stress has emerged as one of the key drivers of cell shape change (Schiffhauer and Robinson 2017; Sapala et al. 2018; Coen and Cosgrove 2023).

Phytohormones play diverse roles in regulating cell growth and shape modulation by affecting cytoskeletal dynamics, cell wall properties, pH, and turgor pressure (Braidwood et al. 2014; Majda and Robert 2018; Durand-Smet et al. 2020; Vernoux et al. 2021; Wang et al. 2025). Auxin, brassinosteroids, cytokinins, and gibberellins promote cell elongation, while abscisic acid and ethylene often restrict growth under stress (Chaiwanon et al. 2016). Auxin participates in the establishment of polarity within cells and tissues by polarizing transporters and forming auxin gradients, driving asymmetric growth and organ patterning (Zhang et al. 2023). Recently, it has been shown that the apoplastic pH actively regulates the hypocotyl's growth response to varying levels of auxin and light. The cell wall is a critical location for converging light and auxin signaling activities in organ growth elongation (Wang et al. 2025). Though many phytohormones have been shown to affect cell growth under variable conditions, the functional role of auxin has been the best characterized in terms of cell shape modulation (Vernoux et al. 2021).

In this review, we will first discuss the components of the plant cell wall, followed by an exploration of how auxin signaling contributes to differential cell growth and cell shape acquisition in various *Arabidopsis thaliana* cell types, including meristematic cells, leaf pavement cells, guard cells, root epidermal cells with root hairs, and hypocotyl cells (Figure 1). Additionally, we will discuss the use of modeling studies across these cell types to provide a comprehensive understanding of cell morphogenesis.

2 | The Role of Cell Wall in Shaping Plant Cell Morphology: Mechanisms and Models Beyond Cell Elongation

2.1 | Molecular Biochemistry of Primary Cell Wall

The primary cell wall constitutes three dominant structural polysaccharides, including cellulose microfibrils, hemicelluloses, and pectins, which have different structural, conformational, and physical properties (Cosgrove 2024; Delmer et al. 2024). The cell wall also possesses small amounts of proteins and structural glycoproteins, which lack enzymatic activities (Marzol et al. 2018). Understanding how these elements interact together and form complex matrices is key to grasping the dynamic and structural roles of the cell wall during cell shape acquisition.

Cellulose microfibrils are chemically described as a linear chain of β(1,4)-linked D-glucose units and are considered the primary load-bearing components of the cell wall (Delmer et al. 2024). In plants, cellulose microfibrils are approximately 1–10 μm in length and about 3 nm in width, consisting of 18 tightly packed glucan chains that form a crystalline structure (Nixon et al. 2016; Pedersen et al. 2023; Cosgrove 2024). The physical properties and stability of cellulose microfibrils arise from extensive hydrogen bonds between the glucan chains and strong noncovalent interactions between parallel glucan chains (Wohlert et al. 2022; Jarvis 2023). Crystalline cellulose microfibrils are synthesized by a multimeric cellulose synthase complex (CSC) embedded in the plasma membrane (Purushotham et al. 2020; Wilson et al. 2021). Cortical microtubules guide the movement of the CSC at the plasma membrane surface to direct the incorporation of cellulose microfibrils into the expanding cell wall. The preexisting cellulose microfibrils also participate in the orientation of new cellulose microfibrils within the cell wall (Paredez et al. 2006; Gutierrez et al. 2009).

Hemicelluloses are complex structural polysaccharides that include xyloglucans, xylans, mannans, and mixed-linkage glucans. Hemicelluloses play a crucial role in the structure and function of primary and secondary cell walls (Pauly and Keegstra 2016). Xyloglucans are the predominant hemicelluloses in the primary cell wall of plants, accounting for 10%–20% of the total dry cell wall weight (Schultink et al. 2014). Xyloglucans are chemically defined by a backbone of β(1,4)-linked glucosyl residues, with α-linked xylose residues attached to the 6-position of glucose units in a regular, repeating pattern. The xylose residues are further appended with galactose, which may also be linked to fucose residues, forming branches of one, two, or three glycosyl residues (Scheller and Ulvskov 2010; Schultink et al. 2014). Xyloglucans are synthesized by various glycosyl transferases in the Golgi apparatus and are then transported to the cell surface by secretory vesicles (Hoffmann et al. 2021). Xyloglucans interpose between cellulose microfibrils to influence their local self-assembly in the primary cell wall (Cosgrove 2022).

Pectins are the most complex and major polysaccharides, comprising 30%–60% of the primary cell wall. Chemically, pectins are defined as galacturonic acid-rich polysaccharides, classified into three major groups: homogalacturonan, rhamnogalacturonan-I, and substituted galacturonans, which are further subcategorized as rhamnogalacturonan-II, xylogalacturonan, and apiogalacturonan (Caffall and Mohnen 2009; Delmer et al. 2024). Homogalacturonans are homopolymers of galacturonic acid (GalA) linked by α-D-(1,4) bonds, making up 65% of the total pectins in the cell wall. They are synthesized in a methyl-esterified state at the C-6 carboxyl residue in the Golgi apparatus and then secreted to the growing cell wall (Sterling et al., 2001; Hoffmann et al. 2021). The methyl ester is subsequently de-esterified in the wall (*in muro*) by PECTIN METHYLESTERASEs (PMEs), resulting in charge

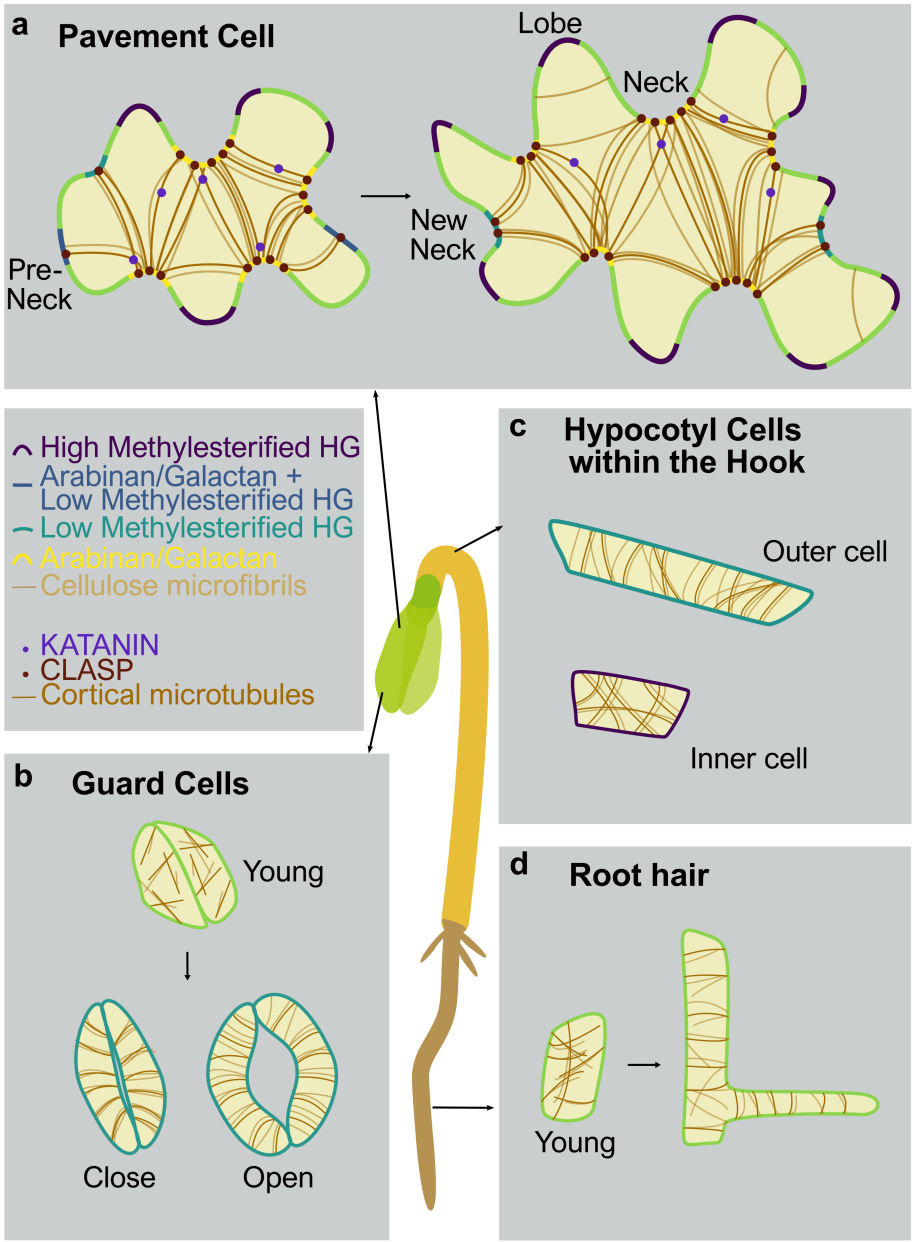

FIGURE 1 | Epidermal cells acquire different cell shapes to enable their functions and maintain tissue integrity in plants. Meristematic cells differentiate and expand into diverse mature cell types, including jigsaw puzzle-shaped pavement cells (a), round stomatal guard cells (b), elongated epidermal cells in hypocotyls and the apical hook (c), and root epidermal cells with root hairs (d). The spatial distribution and arrangement of cell wall polysaccharides, such as cellulose microfibrils, xyloglucans, and pectins (e.g., homogalacturonan represent as HG), and the abundance of methylester groups on these components in differentiating cells regulate anisotropy during cell growth, enabling the acquisition of specific cell shapes. In addition, proteins like KATANIN and CLASP reorient dynamically the cortical microtubules. These cortical microtubules rearrangement is in response to mechanical cues, either self-generated (as in b, c) or from neighboring cells (a, c), leading to the resulting cell shape.

repulsion between the $-COO^-$ residues, which increases the hydration and thickness of the cell wall (Wang et al. 2020). This finding supports the earlier hypothesis that pectins are the predominant factors influencing primary cell wall thickness (Jarvis 1992). The rhamnogalacturonan-I polysaccharides consist of a repeating backbone of galacturonic acid and rhamnose (Rha) disaccharides [—α-(1,4)-D-GalA-α-(1,2)-L-Rha—], with the Rha residues in the backbone variably linked to single or complex side chains of galactose, arabinose, and other sugars (Lau et al. 1985). The nature and complexity of these side chains are highly diverse, depending on the tissue type and developmental stage (Kaczmarska et al. 2022). Rhamnogalacturonans-I have been suggested to play a role in cell wall adhesion, but there is little evidence supporting their role in mechanical cell wall extension and the orientation of cellulose microfibrils in the growing cell wall (Yang et al. 2020; Saez-Aguayo and Largo-Gosens 2022; Saffer et al. 2023). Rhamnogalacturonans-II are complex pectic polysaccharides with a homogalactan backbone substituted with various oligosaccharide side chains, comprising about 10% of the primary cell wall (Cosgrove 2024). The apiosyl residues of rhamnogalacturonan-II monomers interact with borate to

form diester linkages, which contribute to reducing cell wall porosity and increasing its stiffness (Kobayashi et al. 1996; O'Neill et al. 1996; Dumont et al. 2014; Shi et al. 2017; Begum et al. 2023).

2.2 | Cellulose Microfibril Orientation Coordinates Anisotropic Cell Expansion

Cellulose microfibrils play a crucial role in controlling the cell wall's anisotropic expansion. The flexibility of cellulose is not entirely determined by the arrangement of glucan chains; rather, it depends on microfibril arrangement, hydrogen bonding, and interactions with other wall components. Instead, the glucan chain primarily reinforces linear arrangement and provides tensile strength (Zhang et al. 2021). Epidermal pavement cells in *Arabidopsis* leaves exhibit a complex, interlocking jigsaw puzzle shape, with multiple lobes and necks that interdigitate with neighboring cells, constituting an interesting system to study complex cell shape acquisition (Figure 1a). Mutations in the *CELLULOSE SYNTHESIS* genes *CESA1* and *CESA3* have been shown to cause abnormal interdigitations of pavement cells (Burn et al. 2002). A point mutation in the central cytosolic domain of CESA1, termed *anisotropy1* (*any1*), decreased cellulose crystallinity and stiffness of the cell wall, subsequently reducing the growth anisotropy of pavement cells (Fujita et al. 2013). Moreover, the exogenous application of cellulase on *Arabidopsis* cotyledons shifts the interdigitation of pavement cells to an elongated cell shape, indicating the crucial role of cellulose in regulating cell shapes (Higaki et al. 2017).

Cellulose is deposited perpendicular to the direction of cell expansion, and the reorientation of cellulose microfibrils imparts directional stiffness to the cell wall (Anderson et al. 2010). Cellulose microfibrils can shift from a transverse to a longitudinal orientation, offering resistance to turgor pressure along a specific axis and enabling anisotropic cell expansion (Suslov and Verbelen, 2006; Anderson et al. 2010). During pavement cell shape acquisition, the high tensile strength of cellulose microfibrils along their longitudinal axis regulates the initiation and further expansion of lobe growth along the cell contours (Figure 1a). Moreover, an enrichment of cellulose microfibrils perpendicular to the tangent at the tips of the lobes controls their expansion (Sampathkumar et al. 2014; Altartouri et al. 2019). The *any1* mutant shows normal lobe initiation but restricted lobe expansion, suggesting that high cellulose microfibril crystallinity is more critical for lobe expansion than for lobe initiation (Altartouri et al. 2019). Dynamic rearrangement of cellulose microfibrils is not limited to lobe expansion but also plays a role in cellular responses to mechanical and physiological stimuli, such as stomatal movements. The transition from a relatively uniform cellulose distribution in the open state to a more fibrillar pattern in the closed state (Figure 1b) reflects a reorganization that aligns with changes in turgor pressure in the guard cells (Rui and Anderson 2016).

Cellulose has traditionally been considered the least flexible and least extensible component of the plant cell wall during its expansion. However, recent studies demonstrate that cellulose microfibrils in the primary cell wall can stretch elastically up to 1% in response to relative biological tensile forces (Zhang et al. 2021). To explore this further, mesoscale coarse-grained molecular dynamics (CGMD) simulations have been used, offering valuable multiscale insights into cell wall mechanics. These simulations reveal that matrix polysaccharides contribute significantly to the wall's elastic stretchability, primarily due to their uncoiling properties. Moreover, the arrangement of cellulose microfibrils in the periclinal walls of epidermal cells, studied in *Arabidopsis* stems, maize coleoptiles, and onion scales, shows a distinct alignment pattern. Vibrational Sum Frequency Generation (VSFG) imaging results indicate that cellulose microfibrils are highly aligned at cell edges, while they display isotropic alignment in the flat regions, suggesting that anisotropic stress distribution plays a role in this organization (Lee et al. 2023). Additionally, the mechanism of cellulose microfibrils reorientation during cell wall extension was investigated under high strain in onion epidermal cells. Atomic force microscopy revealed that cellulose microfibrils reoriented and packed tightly under high strain, enabling the cell wall to stretch without compromising its load-bearing capacity (Yu et al. 2024). During extensive stretching, the longitudinal cellulose microfibrils straighten and become more ordered, while transverse cellulose microfibrils bend and twist (Yu et al. 2024). These studies collectively enhance our understanding of the dynamic behavior of cellulose and its role in the mechanical properties of plant cell walls.

2.3 | The Role of Xyloglucans in Cell Wall Biomechanics and Expansion

Xyloglucans mechanically tether cellulose microfibrils (Figure 2), influencing their local organization and serving as a binding site for cell wall-loosening enzymes, thereby regulating cell wall expansion (Zheng et al. 2018). Xyloglucan–cellulose junctions are seen as "biomechanical hotspots" that control cell wall loosening and expansibility (Park and Cosgrove 2012a). Interestingly, xyloglucan-specific endoglucanases have been shown to restrict cell wall expansion, whereas endoglucanases that target both cellulose and xyloglucans promote cell wall expansion, as observed in *Arabidopsis* and cucumber hypocotyls (Park and Cosgrove 2012a).

Mutations in the xyloglucan biosynthesis enzymes α-1,6-xylosyltransferases (XXT1 and XXT2) cause significant structural changes and a reduction in xyloglucans content to less than 5%. These mutations result in markedly altered cell wall mechanical properties, including reduced stiffness and increased extensibility compared to the wild type (Cavalier et al. 2008; Park and Cosgrove 2012b; Sowinski et al. 2022). Additionally, *xxt1/xxt2* double mutants display pronounced defects in hypocotyl cell elongation and apical hook formation in *Arabidopsis*, emphasizing the role of xyloglucans in regulating cell wall mechanical properties to facilitate cell elongation (Aryal et al. 2020; Sowinski et al. 2022). Interestingly, the absence of xyloglucans in the *xxt1/xxt2* double mutant affects the stability of microtubules, as well as the synthesis and organization of cellulose microfibrils. This highlights a clear connection between cellulose biosynthesis, the cytoskeleton, and xyloglucan content (Xiao et al. 2016).

The β-(1 → 2)-galactosyltransferase MURUS3 (MUR3) modifies the xyloglucan backbone by attaching a β-D-galactose to

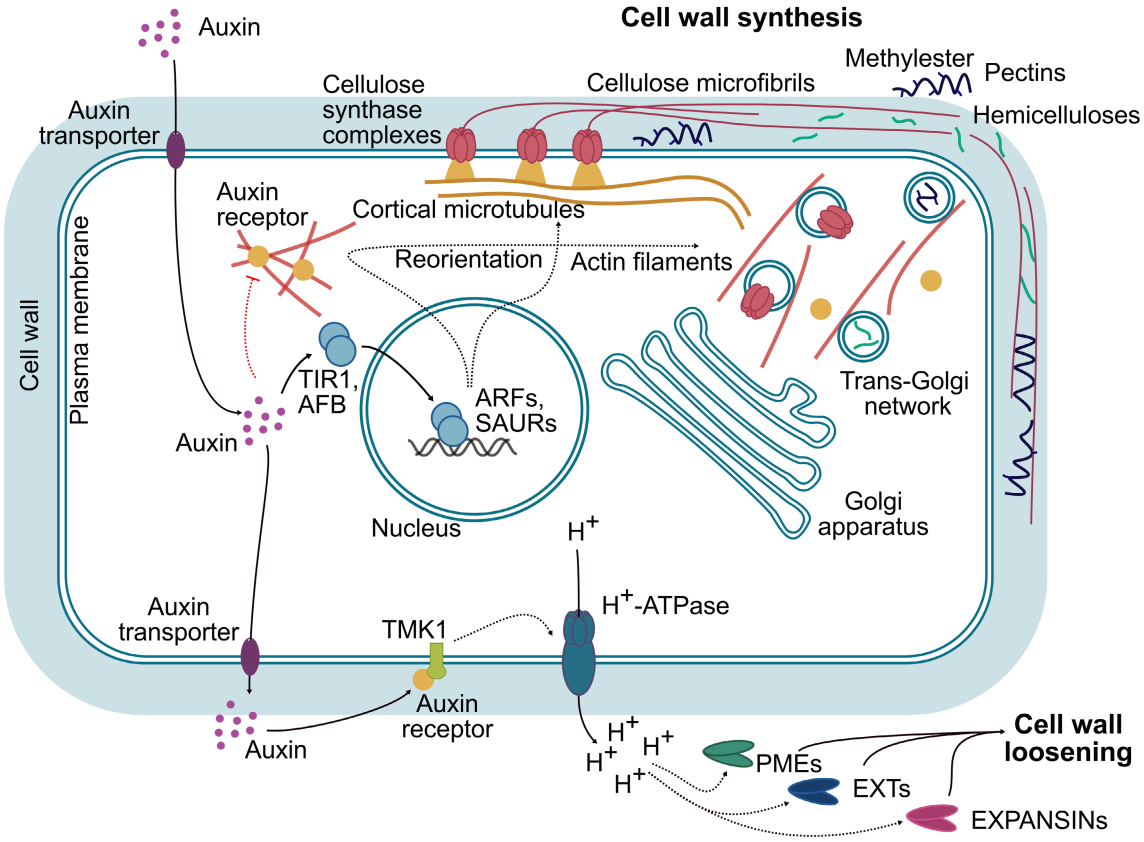

FIGURE 2 | Auxin controls cell expansion by regulating cell wall biosynthesis and cell remodeling. Auxin promotes cell expansion by acidifying the cell wall and activating wall synthesis and loosening enzymes. Auxin efflux (PINs) and influx (AUX1) transporters establish concentration gradients in growing tissue. Auxin enters cells via influx transporters and activates the TRANSPORT INHIBITOR RESPONSE 1/AUXIN SIGNALING F-BOX PROTEINS-AUXIN/INDOLE ACETIC ACID (TIR1/AFB-Aux/IAA) nuclear signaling cascade, which regulates auxin-responsive genes, including AUXIN RESPONSE FACTORs (ARFs) and SMALL AUXIN UP RNAs (SAURs). Auxin activates the H^+-ATPase proton pump through TRANSMEMBRANE KINASE 1 (TMK1), acidifying the cell wall and triggering loosening enzymes including PECTIN METHYLESTERASEs (PMEs), EXPANSINs, and XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASEs (EXTs). Cellulose microfibrils are synthesized by the plasma membrane-bound cellulose synthase complex (CSC), with cortical microtubules guiding the exocytosis of this complex toward expanding cell edges. Auxin coordinates the reorientation of cortical microtubules and actin filaments to regulate the trafficking of cell wall polysaccharides to ensure proper cell wall expansion and specific cell shape acquisition.

the O-2 position of the third xylose residue. This side-chain modification is essential for maintaining xyloglucan solubility within the Golgi apparatus (Kong et al. 2015). Mutations in the *MUR3* gene result in reduced hypocotyl cell elongation and decreased tensile strength (Madson et al. 2003; Sowinski et al. 2022). Furthermore, xyloglucans have been shown to be essential for normal post-Golgi vesicle secretion during cell expansion. The lack of xyloglucan side-chain modification in *mur3-7* results in xyloglucan precipitation, disrupting proper trafficking and causing polysaccharide-rich vesicle accumulation in the cytoplasm (Hoffmann and McFarlane 2024). Additionally, xyloglucan galactosylation is crucial for maintaining the integrity and homeostasis of the cell wall (Xiang et al. 2023). The loss of galactosylation in xyloglucan in *mur3-7* not only reconfigures the cell wall, disrupting normal cell wall synthesis but also destabilizes the actin cytoskeleton and the endomembrane system in expanding cells (Xiang et al. 2023).

The β-glucan synthase enzyme, encoded by *CELLULOSE SYNTHASE LIKE C* (*CSLC*), synthesizes the β-1,4 glucan backbone of xyloglucans (Kim et al. 2020). The *cslc* mutation reduces xyloglucan levels and results in a softer and weaker cell wall compared to the wild type (Daher et al. 2024). The deficiency of xyloglucans impairs cellulose biosynthesis and disrupts pectin content, ultimately leading to cell wall reconfiguration (Xiao et al. 2016; Xiang et al. 2023; Daher et al. 2024). In conclusion, this clearly reveals an existing feedback mechanism linking hemicellulose biosynthesis with cellulose and pectins in the cell wall, maintaining cell wall integrity and facilitating cell expansion.

2.4 | Pectin Dynamics Drives Mechanical Heterogeneity for Anisotropic Cell Expansion

Pectin polysaccharides play a crucial role in regulating cell wall mechanics and anisotropic cell expansion. Demethylesterification of pectins influences spatial variations in cell wall stiffness, while their methylesterification contributes to localized softening of the cell wall (Peaucelle et al. 2015; Majda et al. 2017; Haas et al. 2020; Liu et al. 2021). Atomic force microscopy analysis reveals

that demethylesterified pectins increase cell wall elasticity, enabling reversible changes in cell shape in response to mechanical stress (Peaucelle et al. 2011). It was later shown that demethylesterification of homogalacturonan initiates a shift from isotropic to anisotropic expansion in hypocotyl epidermal cells. This shift reorients microtubules from a random to a parallel transverse arrangement, which directs transverse cellulose microfibrils to support hypocotyl epidermal expansion along the established growth axis (Peaucelle et al. 2015). Additionally, alterations in the pectin polysaccharide rhamnogalacturonan-I in the *RHAMNOSE BIOSYNTHESIS 1* (*rhm1*) mutant disrupt cell wall expansion independently of microtubule orientation (Saffer et al. 2017). Although the de-esterification of homogalacturonan by PME softens the cell wall in onion epidermis, this process alone is insufficient to induce wall loosening and the extensibility required for expansion-mediated cell wall growth (Zhang et al. 2019; Wang et al. 2020).

In epidermal pavement cells of *Arabidopsis* cotyledons, demethylesterified homogalacturonan accumulates at the neck regions of periclinal walls (Altartouri et al. 2019; Figure 1a). This accumulation occurs before the formation of periclinal microtubule arrays during early cell wall curvature, indicating that pectins, rather than cellulose, might be contributing to increased cell wall stiffness at the early lobing stage (Altartouri et al. 2019). The use of a combination of super-resolution microscopy (3D-dSTORM) and cryo-scanning electron microscopy revealed the presence of organized pectin nanofilament structures in the anticlinal wall (Haas et al. 2020). These nanofilaments, which are absent in periclinal walls where homogalacturonan forms a mesh, suggest distinct mechanisms for lobe formation in anticlinal and periclinal walls. The nanofilaments in anticlinal walls are arranged in parallel arrays and are thicker on the neck side of lobe-neck junctions, where they are proposed to be low or demethylesterified, compared to the lobe side or straight wall regions, which are thought to be highly methylesterified (Figure 1a). This indicates a (de)methylesterification-based mechanism for reversible lateral stretching of anticlinal walls. The local enrichment of demethylesterified homogalacturonans, along with (1,4)-β-D-galactan and (1,5)-α-L-arabinan, accumulates in the central region of straight anticlinal cell walls in young *Arabidopsis* leaf pavement cells before lobes begin to form (Majda et al. 2017). Atomic force microscopy results revealed that this region is mechanically softer than the cell wall ends, suggesting that local softening of the cell wall may occur before lobe formation starts (Majda et al. 2017). Similarly, pectin modifications play critical roles in other specialized cell types. For instance, it was demonstrated that highly methyl-esterified homogalacturonans are excluded from guard cells while being abundant at the junctions between guard cells (Figure 1b) and their neighboring epidermal cells (Amsbury et al. 2016). In contrast, in root hairs, pectin modifications have been linked to growth regulation. The increased PME activity was shown to lead to a slowdown in root hair elongation (Schoenaers et al. 2018). These PMEs de-esterify homogalacturonans, enabling interactions with calcium ions (Ca^{2+}) to form egg-box structures that create a stiff, cross-linked matrix. Although this theory aligns with observed stiffness patterns, direct empirical evidence to fully support the mechanism remains scarce, emphasizing the need for further research. A parallel can be drawn with pollen tube growth, as highly methyl-esterified homogalacturonans are exocytotically secreted at the growing tip, enabling localized wall stiffness essential for their distinctive tip growth (Bosch and Hepler 2005). Alterations in rhamnogalacturonan-II dimerization disrupt cell wall integrity. Recent reports indicate that the *MUR1* gene mutant, defective in guanosine 5′-diphosphate (GDP)–fucose, impairs rhamnogalacturonan-II dimerization, leading to defects in apical hook development in *Arabidopsis* (Jewaria et al. 2025).

In conclusion, the complex chemistry of cellulose, hemicelluloses, and pectins modulates the biomechanical properties of the cell wall, requiring further research to better understand the lobing mechanism in *Arabidopsis* pavement cells. Also, our present understanding is based on experiments conducted on fixed tissue, but future advancements in live sample imaging techniques could provide a clearer understanding of the lobe initiation process.

2.5 | Cytoskeleton Machinery Regulates the Trafficking of Wall Polysaccharides and Cell Wall Mechanics

The cytoskeleton plays a crucial role in determining cell shape by directing the orientation of cell expansion (Fu et al. 2002; Landrein and Hamant 2013). Cortical microtubules, situated just beneath the plasma membrane, reorient themselves in response to mechanical forces and regulate directional cell expansion, playing a key role in shaping the cell (Kost and Chua 2002; Smith 2003; Landrein and Hamant 2013). More precisely, microtubules have been shown to orient themselves parallel to the direction of maximum principal tensile stress and avoid aligning with regions experiencing maximum compressive stress (Hejnowicz et al. 2000; Hamant et al. 2008). On the other hand, cell shape–derived mechanical stimuli direct the reorientation and alignment of microtubules along the axis of maximum stress intensity, reinforcing the cell wall (Sampathkumar et al. 2014). In *Arabidopsis* roots, mechanical forces during cell expansion stabilize microtubule bundles by driving their reorientation perpendicular to the direction of expansion (Hoermayer et al. 2024).

The microtubule-associated protein CYTOPLASMIC LINKER ASSOCIATED PROTEIN (CLASP) restricts cell edge-induced microtubule depolymerization, promoting the accumulation of microtubules around sharp edges and facilitating their orientation to span adjacent cell faces in *Arabidopsis* roots (Ambrose et al. 2011). The self-organization of microtubules is thought to be regulated by the microtubule-severing protein KATANIN, enabling cells to respond to mechanical stress between adjacent cells in *Arabidopsis* (Uyttewaal et al. 2012). The *katanin1* (*ktn1*) mutant exhibits defective supracellular microtubule dynamics, leading to random microtubule orientation in the shoot apical meristem, impaired anisotropic growth, and a diminished ability of meristematic cells to respond to mechanical stress. KATANIN is essential for maintaining a cell's ability to respond to mechanical stress from neighboring cells, supporting heterogeneous growth (Uyttewaal

et al. 2012). KATANIN and CLASP together mediate the reorientation of microtubules in response to mechanical stimuli during early pavement cell development (Figure 1a), as shown by the isotropic growth and altered cell wall deposition in the *ktn1–2 clasp-1* double mutant (Eng et al. 2021). During anisotropic cell expansion or elongation, cellulose microfibrils rearrange perpendicular to the direction of cell growth, promoting expansion (Sampathkumar et al. 2014; Altartouri et al. 2019). Microtubules guide the trafficking of the CSC to the plasma membrane, as evidenced by the effects of microtubule disruptions caused by drugs like oryzalin and colchicine, which alter cellulose microfibril organization and deposition orientation (Baskin 2005; Paredez et al. 2006, 2008). Cortical microtubules interact with CESA-interacting protein 1 (CSI1), which binds to the POM–POM2 (POM2) protein and facilitates the trafficking of the CESA complex from the trans-Golgi network to cortical microtubules and the plasma membrane (Bringmann et al. 2012; Li et al. 2012; Liu et al. 2023). This interaction influences microtubule organization under mechanical stress (Schneider et al. 2022) and stabilizes microtubules along the anisotropic stress axis to reinforce specific cell wall patterns, such as those in pavement cell lobe formation. Disrupting microtubule–CESA complex tethering, however, modulates microtubule orientation, enabling adaptive responses to mechanical stimuli.

The secretion and delivery of non-cellulosic polysaccharides, such as hemicelluloses and pectins, to the cell wall are mediated by the actin cytoskeleton, ensuring efficient transport and deposition of Golgi-derived materials (Breuer et al. 2017; Chebli et al. 2021). The orientation of the actin cytoskeleton is believed to predict Golgi-mediated trafficking in growing cells. Actin dynamics and remodeling are altered in response to cell wall disruption, whether caused by mechanical or pharmacological means, in the epidermal pavement cells of *Arabidopsis* leaves (Tolmie et al. 2017) and root cells exposed to cell wall-degrading enzymes (Wojtaszek et al. 2007). Mutations in the *SUPPRESSOR OF ACTIN 1* (*SAC1*) gene disrupt actin filament organization, resulting in reduced cell wall thickness and impaired cell elongation (Zhong et al. 2005). Beyond facilitating vesicle transport, the actin cytoskeleton coordinates the direction of cell growth and expansion by specifying exocytosis sites. In pollen tube cells, actin filaments align parallel to the growth axis, ensuring targeted delivery of cell wall vesicles to the growing tip (Bove et al. 2008; Cárdenas et al. 2008; Bou Daher and Geitmann 2011). Dynamic actin polymerization and depolymerization cycles at the apical tip reorient actin filaments toward the rapidly expanding region of the pollen tube (Qu et al. 2017), supporting its rapid, directional growth. Moreover, such intricate interplay between actin dynamics and vesicle trafficking enables cells to adapt to mechanical stimuli and ultimately acquire specific shapes in plants.

2.6 | Apoplastic pH-Mediated Post-Transcriptional Regulation of Cell Wall Remodeling

Regular remodeling of primary cell walls is crucial for the acquisition of plant cell shape, as it regulates cell wall extensibility and growth. Cell wall-loosening enzymes activate at specific pH levels, which coordinate the remodeling process (Cosgrove 2016; Hocq et al. 2017a). The typical apoplastic pH, the space between the cell wall and adjacent cells, ranges from 4.5 to 7, depending on tissue type and species (Yu et al. 2000). This pH is regulated by the plasma membrane proton pump (H^+-ATPase; Figure 2), which controls cell wall loosening and remodeling in growing plant cells (Spartz et al. 2014; Li et al. 2022). The cell wall rapidly extends within seconds at low pH and quickly ceases at neutral pH in *Avena sativa* L. coleoptiles and *Cucumis sativus* L. hypocotyl sections (Cleland et al. 1987). A recent study showed that lowering the apoplastic pH below 4.4 inhibits hypocotyl elongation in etiolated *Arabidopsis* seedlings within 30 min (Wang et al. 2025). This suggests that cell wall growth is highly sensitive to pH changes in the apoplastic space.

Among the cell wall-loosening enzymes, α-expansins play a key role by catalyzing the disruption of non-covalent bonds between cellulose and hemicellulose at low pH (4.5–6), thus facilitating cell wall loosening (McQueen-Mason et al. 1992; Cosgrove 2000). Additionally, the ectopic upregulation or exogenous application of α-expansins stimulates cell growth (Fleming et al. 1997; Link and Cosgrove 1998). Furthermore, XYLOGLUCAN ENDOTRANSGLYCOSYLASE/HYDROLASES (XTHs) and endoglucanases modify cell wall properties by cleaving or ligating xyloglucans and hydrolyzing glycosidic bonds, which promote cell expansion through the incorporation of new cell wall materials (Rose et al. 2002; Hayashi and Kaida, 2011; Park and Cosgrove 2015). XTH1 exhibits catalytic activity across a broad pH range (4.5–7.5) in *Selaginella kraussiana* (Van Sandt et al. 2006). The ectopic expression of maize *XTH1* enhances xyloglucan endohydrolase activity and alters cell wall structure and properties in *Arabidopsis* (Genovesi et al. 2008). However, XTH enzymes have not been shown to directly stimulate cell wall expansion (Kaewthai et al. 2013; Niraula et al. 2021).

PMEs and PECTIN ACETYLESTERASEs (PAEs), key cell wall remodeling enzymes, precisely regulate the degree of methylesterification and acetylation, controlling the distribution of pectins, especially homogalacturonans, during cell elongation (Pelloux et al. 2007; Hocq et al. 2017a). A mutation in the cell wall-modifying enzyme PME3 impairs root growth in *Arabidopsis* (Hewezi et al. 2008). The *Arabidopsis AtPME2* regulates hypocotyl elongation and shows higher catalytic activity at neutral pH (pH 8) compared to acidic pH (pH 5). In the *AtPME2* knockout mutant, hypocotyl elongation reduces under dark growth conditions due to increased cell wall stiffness in the apical region of the hypocotyl (Hocq et al. 2024). The fine-tuning of PME activity on homogalacturonans is regulated by proteinaceous inhibitors, known as PME INHIBITORs (PMEIs), along with pH and cation concentrations (Sénéchal et al. 2014). PMEIs interact with PMEs across a broad pH range from 3.5 to 10, with PME activity being more strongly inhibited at acidic pH than at neutral or alkaline pH (Sénéchal et al. 2015; Hocq, et al. 2017a; Hocq et al. 2017b). The interaction of PMEI7 with PME3 at acidic pH finely regulates the degree of homogalacturonan methylesterification in the elongated hypocotyl of *Arabidopsis* (Sénéchal et al. 2015). A recent report demonstrated that purified PMEI3 inhibits cell expansion in *Arabidopsis* roots by regulating PME activity at acidic pH and the de-methylesterification of

homogalacturonans on the root surface (Xu et al., 2022). Low pH reduces free PME activity and promotes the interaction between PMEs and PMEIs, limiting their specificity toward homogalacturonan and inhibiting its degradation (Sénéchal et al. 2015). Taken together, this pH feedback loop is crucial for fine-tuning PME activity and regulating pectin methylesterification during cell wall remodeling.

3 | Auxin Signaling Pathways and Their Multifaceted Roles in Shaping Plant Cells

3.1 | From Quick Responses to Steady-State Action: The Dual Facet of Auxin Response

Plants display a high degree of control of cell size and cell division pattern during their embryonic development (Jürgens et al. 1994). However, environmental factors play a dominant role in shaping the otherwise patterned process of postembryonic development (Celenza et al. 1995). Since plants grow in one place and their physiochemical environment often varies, they have evolved unique molecular actors that help them react quickly to the changes in their microenvironmental conditions. Auxin is one such molecular actor that plays a crucial role in regulating the rate of cell division, cell growth, and acquisition of differentiated cell shape (Leyser 2018; Jobert et al. 2023). Auxin exhibits its multifunctional nature across a range of concentrations, driving diverse developmental outcomes, such as in the epidermal layer of *Arabidopsis*, for example, where cell shape varies dramatically across the tissue. At low concentrations, auxin primarily promotes cell elongation, supporting the growth of specific plant tissues, while at higher concentrations, auxin inhibits cell elongation and promotes cell division, thus playing a key role in cell morphogenesis (Bhalerao and Bennett 2003; Tang et al. 2024).

The TRANSPORT INHIBITOR RESPONSE 1/AUXIN SIGNALING F-BOX PROTEINs (TIR1/AFBs)-AUXIN/INDOLE ACETIC ACID (Aux/IAA)-Auxin Response Factor (ARF) pathway is a well-established canonical nuclear auxin signaling pathway (Figure 2), regulating transcription to control auxin-related developmental processes (Gray et al. 2001; Tiwari et al. 2003; Dharmasiri et al. 2005; Kepinski and Leyser 2004, 2005; Weijers et al. 2005; Cohen and Strader 2024). Auxin is perceived by the TIR1/AFB1-5 proteins, which function as receptors and are part of the SCF ubiquitin ligase complex. In the absence of auxin, Aux/IAA proteins act as transcriptional repressors by binding to ARFs, blocking the activation of auxin-responsive genes. Increases in levels of the endogenous auxin indole-3-acetic acid (IAA) facilitate the interaction between TIR1/AFB1-5 and Aux/IAA proteins, promoting the ubiquitination and thus degradation of Aux/IAAs via the 26S proteasome pathway (Das et al. 2021; Zhang et al. 2024). The ARFs are now free to either activate or repress the transcription of downstream target genes by binding to the auxin-responsive elements (AuxREs) present in promoter regions (Chapman and Estelle 2009; Salehin et al. 2015; Liu et al. 2024). Though the nuclear auxin signaling pathway explains many of auxin's effects on plant growth, some processes occur too quickly to rely on TIR1/AFB1-5–dependent transcriptional regulation. These include rapid root growth inhibition, apoplast alkalinization, membrane depolarization, Ca^{2+} influxes, and cytoplasmic streaming (Shih et al. 2015; Fendrych et al. 2018; Li et al. 2021; Serre et al. 2021; Friml 2022). Such rapid responses also point towards the presence of a faster auxin response system that operates independently of transcription (Zhang et al. 2024). A recent report revealed that the TIR1/AFB receptor produces cAMP, which acts as a second messenger to mediate the downstream transcriptional responses. cAMP-mediated auxin signaling bypasses auxin-induced Aux/IAA degradation and directly activates ARF-mediated transcriptional auxin signaling (Chen et al. 2025).

Recently, functional roles of some molecular players like AFB1 and RAF-like protein kinases involved in rapid auxin response have been uncovered, thus unmasking an additional layer of auxin's mode of action (Prigge et al. 2020; Dubey et al. 2023; Kuhn et al. 2024). Therefore, auxin acts through two distinct modes to spatiotemporally regulate cell growth: a rapid, non-transcriptional response and a slower, transcriptional pathway-dependent response. Rapid auxin response is based on the acid growth theory, introduced in the early 1970s (Rayle and Cleland 1970, 1992; Hager et al. 1971). According to this theory, auxin activates H^+-ATPase proton pumps in the plasma membrane, which releases protons (H^+) into the cell wall space, or apoplast. This acidifies the apoplast, creating an environment where cell wall-loosening enzymes can function optimally, allowing the cell to expand rapidly (Figure 2). Two mechanisms have been identified through which auxin activates these H^+-ATPase pumps. In the first one, auxin promotes the expression of SMALL AUXIN UP RNA 19 (SAUR19) through the canonical nuclear auxin signaling pathway, which then activates H^+-ATPases by phosphorylating their C-terminal autoinhibitory domain (Takahashi et al. 2012; Spartz et al. 2014). This phosphorylation stimulates apoplastic acidification, a critical step for rapid cell expansion. In a second, faster response, occurring in minutes, extracellular auxin is sensed by the receptors AUXIN-BINDING PROTEIN 1 (ABP1)/ABP-LIKE1 and 2 (ABL1/2) along with TRANS-MEMBRANE KINASE 1/4 (TMK1/4) (Figure 2). This signaling activates H^+-ATPase through a cascade involving TMK-mediated phosphorylation of AHA1, another H^+-ATPase variant, leading to cell wall acidification and rapid growth in shoot tissues (Lin et al. 2021; Friml et al. 2022; Yu et al. 2023). The rapid nature of this pathway underscores auxin's ability to facilitate immediate responses in growth, adapting swiftly to environmental signals.

3.2 | Auxin-Mediated Regulation of Cell Wall Chemistry in Cell Shaping

The critical role auxin plays in regulating cell elongation and local stiffness of the cell wall, which in turn determines cell shape (Sassi et al. 2014; Jonsson et al. 2021), is mediated by the local accumulation and establishment of concentration gradients (Grones and Friml 2015). Auxin is actively transported against its concentration gradient to establish and maintain auxin maxima. These distribution patterns are formed through cell-to-cell transport, mediated by auxin influx and efflux transporters. Auxin efflux is facilitated by the PIN-FORMED

(PIN) transporters, while influx is governed by AUXIN TRANSPORTER PROTEIN 1 (AUX1) and the LIKE AUX1 (LAX) family. The dynamic orientation of PIN and AUX1/LAX transporters within cells and tissues enables directed auxin flow, establishing the concentration gradient necessary for regulating cell expansion (Gao et al. 2008). Interestingly, a local auxin concentration gradient is crucial for lobe initiation in the young pavement cells of the leaf epidermis in *Arabidopsis* (Grones et al. 2020). The dynamic distribution of auxin efflux and influx transporters, including PIN3, PIN7, AUX1, and LAX1, coordinates the establishment of fluctuating auxin gradients within developing spiral stomatal complexes, triggering lobing in young pavement cells (Grones et al. 2020). Alterations in cell wall composition also modulate auxin flux, suggesting a crosstalk between cell wall integrity and auxin transport (Feraru et al. 2011; Aryal et al. 2020). Interestingly, growth-induced mechanical strain has been shown to upregulate *PIN1* expression and auxin accumulation in the shoot apex of tomato plants (Nakayama et al. 2012). The sensitivity and dynamic distribution of PIN1 are regulated by mechanical stimuli (Nakayama et al. 2012), forming a robust positive feedback loop.

The asymmetrical distribution of auxin leads to differential cell growth, thus shaping organs. For example, localized auxin maxima increase the methylesterification of homogalacturonans on the inner side of the hypocotyl apex relative to the outer side (Figure 1d), locally enhancing cell wall stiffness and inhibiting cell elongation, driving hypocotyl bending to form the apical hook (Jonsson et al. 2021). In turn, the methylesterification of homogalacturonans supports the asymmetrical auxin response on the inner side of the hypocotyl by regulating the activity of polar auxin transporters (Jonsson et al. 2021). The absence of xyloglucans in the *xxt1xxt2* double mutant disrupts cell wall mechanics and impairs polar auxin transport (Aryal et al. 2020). The interaction between cell wall and ARF2-mediated transcriptional regulation of auxin transporters, such as PINs and AUX1, plays a crucial role in establishing local auxin response maxima, which inhibit cell elongation on the inner side of the hypocotyl during apical hook development (Aryal et al. 2020). Auxin response factors ARF7 and ARF19 function as downstream targets in the signaling pathway that transduces mechanochemical changes in the cell wall. Recent reports indicate that disruptions in rhamnogalacturonan-II dimerization attenuate *ARF7/ARF19*, leading to apical hook defects in the *mur1* mutant (Jewaria et al. 2025). Auxin participates in the regulation of the cell wall composition, particularly pectin dynamics, by recruiting the *Catharanthus roseus* RECEPTOR-LIKE KINASE 1-LIKE (CrRLK1L) kinase ERULUS (ERU) through direct interaction with ARF7 and ARF19, thereby controlling root hair tip growth in *Arabidopsis* (Schoenaers et al. 2018). The *eru* mutant shows increased accumulation of demethylesterified homogalacturonans, enhanced PME activity, and altered oscillations of pectin Ca^{2+}-binding sites in growing root hairs (Schoenaers et al. 2018).

According to the acid growth theory, auxin initiates the acidification of the apoplast by activating plasma membrane-localized H^+-ATPase pumps, and the acidic apoplast pH then activates cell wall-loosening enzymes, facilitating turgor-driven cell expansion (Rayle and Cleland 1970; Barbez et al. 2017; Dang et al. 2020). H^+-ATPase activity is regulated through phosphorylation, mediated by SMALL AUXIN UP-RNA (SAUR) proteins, which inhibit the negative regulator type 2C phosphatases (PP2Cs), thereby promoting cell expansion (Spartz et al. 2014). In barley coleoptiles, auxin-induced cell elongation involves the activation of exo- and endo-β-glucanases, which disrupt hemicelluloses within the cell wall (Kotake et al. 2000). Additionally, auxin regulates the expression of cell wall-modifying enzymes, such as EXPANSIN14 (EXP14) and EXP7, by recruiting the auxin response factor LATERAL ORGAN BOUNDARIES DOMAIN 18 (LDB18), which triggers cell wall loosening in *Arabidopsis* root pericycle cells (Lee and Kim 2013; Lee et al. 2013). Furthermore, the activity of the cell wall remodeling protein EXPANSIN A1 (EXPA1) in *Arabidopsis* roots is governed by IAA14- and IAA3-dependent auxin signaling (Ramakrishna et al. 2019). Recent findings show that overexpression of EXPA1 increases cell wall stiffness by altering cell wall remodeling. EXPA1 overexpression upregulates the transcripts of cell wall-associated genes, including *XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASES* (*XTHs*) and several *EXPs*, which collectively modulate the biomechanical properties of the cell wall (Samalova et al., 2024).

3.3 | Auxin-Cytoskeleton Connection and Regulation of Cell Expansion

The anisotropic expansion of cells is coordinated by the orientation of microtubules, which direct the alignment of cellulose microfibrils in the growing cell wall (Cosgrove 2005). Auxin facilitates the reorientation of cortical microtubules, arranging them perpendicular to the growth axis to mediate anisotropic cell expansion. Auxin triggers microtubule reorientation in *Arabidopsis* by activating RHO OF PLANTS 6 (ROP6) GTPase and its effector protein, ROP-interactive CRIB motif-containing protein 1 (RIC1), which interacts with KATANIN to activate its microtubule-severing activity (Lin et al. 2013; Xu et al. 2014). Auxin signal potentially plays a role in microtubule reorientation and clathrin-mediated endocytosis by interacting with ABP1 (Robert et al. 2010; Chen et al. 2014). Surprisingly, while the *abp1* knockout mutant exhibits normal plant development and auxin signaling, the *ABP1* gain-of-function shows impaired auxin effects on PIN polar distribution in *Arabidopsis* (Gao et al. 2015; Gelová et al. 2021). Additionally, auxin triggers the ROP2 signaling cascade via its effector protein RIC4 to regulate the orientation of cortical actin microfilaments and the asymmetric distribution of PIN1, mediating interdigitation in the pavement cells of *Arabidopsis* (Fu et al. 2002, 2005; Nagawa et al. 2012). Auxin reduces cell wall stiffness and directly regulates the reorientation of microtubules to promote isotropic growth in primordia emerging from the shoot apical meristem of *Arabidopsis*. It facilitates this isotropic growth by potentially disrupting the ordered orientation of microtubules through the suppression of the ABP1 and KATANIN1 protein networks in this organ (Sassi et al. 2014). Mechanical stress influences the alignment of PIN1 and the orientation of microtubules, coordinating directional growth at both cellular and tissue levels (Heisler et al. 2010; Feraru et al. 2011). In the *Arabidopsis* shoot apex, growth-induced stress organizes PIN1 and microtubules along stress directions, although their alignment functions independently in the epidermis (Heisler et al. 2010). Similarly, in root tissues, mechanical perturbations, such as those caused by the cellulose biosynthesis inhibitor isoxaben, disrupt the polar

distribution of PIN1 and the reorientation of cortical microtubules (Feraru et al. 2011). Auxin triggers rapid reorganization of the actin cytoskeleton by recruiting AUX1 (Arieti and Staiger 2020). In the *aux1* mutant, actin reorganization was resistant to exogenous IAA (auxin transported via AUX1) but exhibited a partial response to the membrane-permeable auxin 1-naphthylacetic acid (NAA, synthetic auxin), suggesting that AUX1, along with a cytoplasmic auxin receptor, amplifies the signaling pathway contributing to actin rearrangement (Dindas et al. 2018; Arieti and Staiger 2020). Auxin regulates directional cell expansion and cell shape by modulating actin dynamics in *Arabidopsis* (Vaddepalli et al. 2021). In the *iaa12/bdl* mutant, impaired auxin responses were found to disrupt microtubule dynamics and lead to the loss of a dense F-actin network (Vaddepalli et al. 2021).

4 | Modeling Cell Shape Acquisition: Integrating Mechanochemical Properties

Computational modeling in cell biology, especially in the context of plant morphogenesis, serves to connect theoretical frameworks with the dynamic processes involved in cellular growth. Through computational morphodynamics, researchers can challenge their understanding of complex interactions that define plant tissue structure. These models expose how factors such as mechanical stress, microtubule orientation, and hormonal signals collectively drive key processes like turgor pressure, wall extensibility, elasticity, and yield thresholds, fundamental elements of cellular shape and expansion. The base principle is that during cell extension, mechanical stress partially controls microtubule orientation, which in turn controls cellulose deposition and, therefore, cell wall anisotropy. At the same time, and separately, mechanical stress controls auxin transport direction (Nakayama et al. 2012), and as auxin regulates cell expansion, this also feeds into anisotropic growth. Such growth creates tissue shapes, and the combination of turgor pressure and tissue shape creates the mechanical stress tensor field (Hamant et al. 2008). Therefore, these coupled models allow simulation of auxin transport and show how phytohormone-driven growth patterns link with cellular strain and stress responses.

Historically, mechanochemical properties governing cell shape acquisition and auxin transport models have been developed separately. Early models tackle auxin's role in organized but static cell networks (Heisler et al. 2010; Sampathkumar et al. 2014). On the other hand, several computational studies have provided crucial insights into the mechanochemical properties governing cell shape acquisition. Biomechanical feedback, where tissue morphology influences stress distribution, subsequently guiding microtubule alignment, is critical for apical morphogenesis (Hamant et al. 2008). This feedback loop underscores how physical constraints, coupled with cellular structure, guide morphogenetic patterns. Such single-cell models often deal with heterogeneous mechanochemical properties of cell walls and localization and orientation of microfibrils, which control how the cell wall loosens under turgor pressure and drives water influx, leading to irreversible cell expansion. Pavement cell models demonstrate how heterogeneous cell wall mechanical properties or microtubule alignment with the location and directions of maximal tensile stress generate the lobing pattern (Sampathkumar et al. 2014; Majda et al. 2017; Sapala et al. 2018). It was further proposed that lobed shapes in pavement cells minimize mechanical stress, thereby preventing high structural bulging under turgor pressure (Sapala et al. 2018). Similarly, other studies have investigated the application of these stiffness anisotropy principles in various specialized cell types (Dumais et al. 2004; Fayant et al. 2010; Yanagisawa et al. 2015; Bou Daher et al. 2018; Yi and Anderson 2023; Ramos et al. 2024). These models highlight the role of cellular elasticity and anisotropy in shaping plant cells, showing how cellular-level mechanochemical feedback informs tissue-scale morphology. In more integrated frameworks, researchers are beginning to incorporate auxin signaling within mechanical models, linking hormonal regulation with cell wall mechanics. An attempt to expand simple cell arrays was made earlier (Grieneisen et al. 2007). More recent advancements have simulated auxin dynamics within growing, anatomically accurate cell networks, such as in the tomato shoot apex (Nakayama et al. 2012) and in *Arabidopsis* root (Marconi et al. 2021; Ramos et al. 2024). These models have been used to explore tissue mechanics and polar auxin transport in coordinating meristematic tissue growth and establishing cell polarity during early organ development.

Despite their utility, computational models have inherent limitations. One major challenge is the simplification of biological complexity; models often require assumptions that exclude certain cellular or molecular details to remain computationally tractable. Many models operate under assumptions that may not fully account for the dynamic nature of cell wall remodeling, feedback loops between auxin distribution and mechanical stress, or interactions with environmental factors. Additionally, while existing frameworks can simulate general trends in cell and tissue development, they often lack the resolution needed to predict the highly dynamic and heterogeneous nature of cell expansion in vivo. Future improvements should aim to integrate multi-scale modeling approaches. Furthermore, advances in computational power and machine learning could enhance model accuracy, refining our understanding of how mechanochemical feedback loops drive plant morphogenesis. However, the primary purpose of such models is not to replicate every biological detail but rather to provide a framework for hypothesis testing and exploring mechanistic principles.

5 | Conclusion

While there is a clear understanding of how auxin-mediated molecular signaling influences the dynamic remodeling of the cell wall, the precise mechanisms governing cell shaping in plants remain partially elusive. Many candidates involved in the acquisition of cell shape have been identified; however, the exact processes underpinning the shaping of different epidermal cells, for example, are not fully understood. Recent advancements have significantly improved our understanding of how auxin regulates cell expansion, including its role in local accumulation of auxin, cell wall polysaccharide trafficking, cytoskeleton reorientation, and feedback mechanisms that ensure coordinated cell expansion in different tissues. Computational modeling has further enhanced our comprehension by connecting experimental results with physics-based models of cell wall biomechanics and auxin distribution. Nevertheless, many questions remain

unanswered, particularly regarding the mechanisms by which auxin-mediated wall remodeling coordinates three-dimensional cell shaping. The interplay between inner cell layers and their signaling pathways in regulating epidermal cell shaping also needs further investigation.

Studies on the trafficking and orientation of cell wall polysaccharides, including cellulose, hemicelluloses, and pectins in leaf pavement cells and hypocotyl epidermal cells, have provided unprecedented insights into anisotropic cell expansion. These findings help explain how cell shapes are regulated. However, further research is needed to elucidate the rearrangement of cell wall polysaccharides in connection with the reorientation of auxin transporters and their signaling, which is essential for understanding three-dimensional cell-shaping mechanisms.

Future technical advancements in cell and molecular biology, such as live-cell imaging, vibrational spectroscopy, and super-resolution imaging, will illuminate the mechanisms of three-dimensional cell shaping in different cell types. These advances will also help clarify how inner cell layers might regulate cell shaping in the epidermal layer and how this maintains tissue integrity under environmental and mechanical stresses. Visual biomechanical mapping of cell walls and molecular signaling networks could provide critical insights into how mechanical signals are transmitted from neighboring cells to control cell shaping and tissue integrity. These insights could have applications in both basic and agricultural sciences.

6 | Outstanding Questions

1. How do the dynamic expression of different genes, such as auxin response factors, transcription factors, and cell wall synthesis genes, at the tissue level modulate the mechanical forces of neighboring cells to maintain cell morphogenesis?
2. Which auxin signaling pathway specifically regulates the mechanics of cell walls to achieve a particular cell shape?
3. How do distinct patterns of pectin methylesterification contribute to the mechanical and functional differentiation of specialized cell types, such as guard cells, root hairs, or pavement cells, and what mechanisms guide their spatial specificity during development?
4. How can computational models dynamically combine auxin transport, microtubule orientation, and cell wall mechanics in anatomically accurate, growing cell networks to estimate emergent tissue or single-cell structure and functions?
5. How can computational models effectively integrate multi-scale spatiotemporal dynamics while ensuring experimental validation in the face of incomplete data and unknown signaling pathways?
6. Choosing between continuum models (e.g., treating cellular tissues as materials, such as in the Finite Element Method) and discrete models (e.g., representing individual cells in a Position-Based Dynamics framework) impacts the applicability and accuracy of the simulation. Which approach best suits the integrated modeling framework to handle auxin fluxes and influence cell expansion?

Author Contributions

All authors contributed to the study conception and design. The review was conceptually designed by V.K., who also coordinated the overall structure. S.Y. and A.H. contributed substantially to writing the original draft and reviewing the manuscript. A.H. prepared the figures. S.R. supervised the project and contributed to the critical review of the manuscript. All authors read and approved the final manuscript.

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Data Availability Statement

This review article does not include any original data. All information discussed is derived from previously published sources, which are appropriately cited within the text. As such, no new datasets were generated or analyzed for this study.

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