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Plant cell walls as mechanical signaling hubs for morphogenesis

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SUMMARY

The instructive role of mechanical cues during morphogenesis is increasingly being recognized in all kingdoms. Patterns of mechanical stress depend on shape, growth and external factors. In plants, the cell wall integrates these three parameters to function as a hub for mechanical feedback. Plant cells are interconnected by cell walls that provide structural integrity and yet are flexible enough to act as both targets and transducers of mechanical cues. Such cues may act locally at the subcellular level or across entire tissues, requiring tight control of both cell-wall composition and cell–cell adhesion. Here we focus on how changes in cell-wall chemistry and mechanics act in communicating diverse cues to direct growth asymmetries required for plant morphogenesis. We explore the role of cellulose microfibrils, microtubule arrays and pectin methylesterification in the transduction of mechanical cues during morphogenesis. Plant hormones can affect the mechanochemical composition of the cell wall and, in turn, the cell wall can modulate hormone signaling pathways, as well as the tissue-level distribution of these hormones. This also leads us to revisit the position of biochemical growth factors, such as plant hormones, acting both upstream and downstream of mechanical signaling. Finally, while the structure of the cell wall is being elucidated with increasing precision, existing data clearly show that the integration of genetic, biochemical and theoretical studies will be essential for a better understanding of the role of the cell wall as a hub for the mechanical control of plant morphogenesis.

Introduction

The coordinated behavior of adjacent cells in developing multicellular organisms involves a wide variety of signals. Within tissues, cells are in direct physical contact with their neighbors, and mechanical signals prescribed by cell geometry and differential growth, as well as external cues, influence morphogenesis. In animal cells, many mechanosensing modules have been uncovered, such as the well-documented focal adhesion sites[1], and their implications in animal development have been extensively studied[2].

In contrast to animal cells, plant cells are attached to one another by a stiff wall. Hence, organ deformation in plants does not rely on cell contractility, but instead on the modulation of growth rate, growth anisotropy and growth direction[3]. A consequence of the cell–cell connections through shared walls is the build-up of mechanical stress, which can provide instructive intercellular signals. The intrinsic cause of mechanical stress in a plant cell is turgor pressure, which generates tensile stress in the cell wall[4]. Plant cells and tissues can therefore be viewed as pressure vessels. Consequently, the intensity and direction of mechanical stress can be biased by the shape of the cell or the tissue, which already provides a potential cue. At the tissue scale, growth conflicts due to differential growth rates between adjacent cells can further bias local stress patterns. Thus, local stress patterns result from a combination of turgor pressure, cell-wall properties, cell and tissue geometry, and growth conflicts.

Growth occurs through cell-wall remodeling and/or yielding to turgor pressure, while simultaneously maintaining the structural integrity of the cell[5]. Such coordination between stress, loosening and reinforcement points towards a continuous monitoring of mechanical status at the cellular and tissue levels. Several pathways are considered to serve this purpose, ranging from those involving proteins that sense cell-wall integrity[6] to pressure valves[7]. Additionally, mechanical forces can play wider instructive roles, but little is known about the underlying mechanisms and pathways in plants. Several reviews have highlighted the contribution of specific chemical modifications in cell walls and potential molecular sensors or receptors in mechanosensing[8,9]. However, such mechanical cues are unlikely to act independently of more established biochemical pathways. In particular, all known mechanosensors in animals are also well-established sensors of biochemical cues (for example, integrins). In this review, we explore the role of mechanical cues at plant cell walls and their possible interactions with known chemical cues, such as hormones, in morphogenesis.

Cell geometry and mechanical cues

The cell wall is a complex composite material, mainly composed of cellulose, hemicelluloses, pectins and structural proteins. As

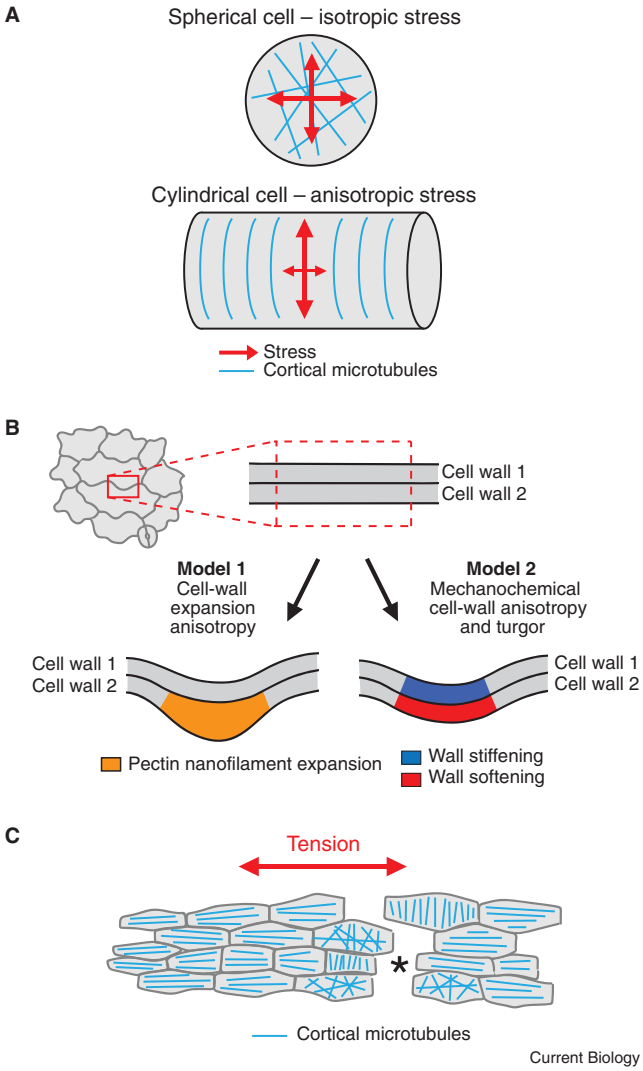

Figure 1. Mechanical cues guide cell shape and cell–cell coordination.

(A) In spherical pressurized cells or dome-shaped tissues, tensile stress is isotropic; this pattern correlates with cortical microtubule organization. In cylindrical elongating cells, the amplitude of tensile stress is related to cell diameter, but not cell length, and is maximal in the transverse direction; this pattern correlates with cortical microtubule organization. (B) Lobe formation in puzzle-shaped cells (such as leaf epidermal cells) is initiated by the establishment of local cell-wall heterogeneity between walls of neighboring cells. Current models suggest that such asymmetry is generated either by local cell-wall expansion via pectin nanofilament swelling in a turgor-independent manner (Model 1, proposed in[26]) or by establishment of local mechano-chemical asymmetries between cell walls (Model 2, proposed in[28–30]). Subsequent compression of the anticlinal cell wall and tensile stress in the outer cell wall reinforces cell lobing (not shown). (C) Tissue stress direction can be revealed by cell–cell adhesion defects; conversely, tensile stress propagation across cells requires proper cell–cell connectivity, as monitored by cortical microtubule behavior. The asterisk denotes a cell–cell adhesion defect.

well as providing structural support for plants, the primary cell wall of a growing cell is highly flexible and dynamically reorganized in response to diverse cues. Even when assuming a uniform cell-wall profile, cell geometry alone is sufficient to prescribe a mechanical stress pattern in the cell wall[10,11]. According to the Laplace–Young law, cell curvature and tension are directly related. Spherical cells experience isotropic tensile stress in their walls. In contrast, elongated cells are under anisotropic tensile stress, with diameter, but not length, determining mechanical stress amplitude[11] (Figure 1A). Remarkably, however, turgor-driven mechanical stress patterns negatively correlate with growth axes because expansion generally occurs in the direction of minimal tensile stress. For instance, epidermal cells in expanding hypocotyls and pollen tubes grow longitudinally, while largely maintaining a constant diameter (where stress is greater)[11,12]. This implies that shape-based stresses are compensated for by the mechanical properties of the cell wall. Accordingly, as proposed more than 50 years ago, the load-bearing cellulose microfibril network is usually deposited parallel to the direction of maximal tensile stress[13]. This reinforces the cell wall in that direction and the minimal direction of tensile stress becomes the maximal direction of cell expansion.

Cellulose microfibril deposition is guided by the cortical microtubule network, which is highly responsive to mechanical cues. Cortical microtubules preferentially orient parallel to the maximal tensile stress direction in numerous tissues, such as the shoot apical meristem (SAM), cotyledons and hypocotyls[14–17]. How these microtubules might sense tensile stress in the cell wall is unknown. Cortical microtubules of protoplasts (plant cells lacking a cell wall) confined in rectangular wells align with predicted maximal tension (along the transverse axis, following the Laplace–Young law) upon pressurization, then return to longitudinal orientation upon depressurization[18]. Thus, cortical microtubule responses to stress may not require a cell wall and microtubules themselves might possibly act directly as mechanosensors[19], echoing the proposed role of actin as a mechanosensor in animal cells[20]. Cortical microtubules also align with predicted maximal tensile stress in relation to cell geometry in epidermal pavement cells[16] and tissue geometry in SAMs[14]. Therefore, shape can influence tensile stress patterns at different scales, to which cortical microtubules can respond. Yet, cortical microtubule alignment with such stresses must involve an indirect, as yet unexplored, connection between these microtubules and the cell wall *in vivo*.

Effects of mechanical cues on cell-wall biochemistry

The alignment of cellulose microfibrils with maximal tensile stress direction shows that the mechanical anisotropy of cell walls can change in response to stress through biochemical modifications; this may also apply to the cell-wall matrix. Notably, in young hypocotyls, before anisotropic growth initiation, isodiametric epidermal cells exhibit asymmetric pectin methylesterification between longitudinal and transverse walls[12,21]. Intriguingly, Peaucelle *et al.*[21] and Bou Daher *et al.*[12] report opposing effects of pectin de-esterification. Whereas Peaucelle *et al.*[21] observed decreased stiffness and increased cell size, Bou Daher *et al.*[12] reported increased stiffness and shorter cells as a result of pectin methylesterase overexpression in the hypocotyl. The resolution of these contrasting results awaits a satisfactory explanation. However, it may be related to compensatory and feedback mechanisms that could be affected by the particular experimental context, by tissue-specific interactions with the main load-bearing polymer cellulose, or by non-identical experimental setups. These studies clearly highlight the complex nature of cell-wall interactions and

underscore the need for further analysis. Importantly, these findings[12,21] clearly indicate that an initial cell-wall biochemical asymmetry may underlie mechanical heterogeneity, directing subsequent reinforcement via cellulose deposition guided by cortical microtubules. Interestingly, cortical microtubules first appear as organized arrays during seed imbibition in *Arabidopsis thaliana* and align with the predicted maximal tensile stress direction[22]. This provides a scenario in which these microtubules would initially align with stress, after which pectin-based mechanical polarity would be established in the growing hypocotyl and then further stabilized by cortical microtubules and cellulose microfibrils.

In hypocotyls, biochemical and mechanical modifications of cell walls can influence the degree of growth anisotropy. As such, the promotion of growth isotropy has been associated with symmetry-breaking events. In particular, localized cell-wall loosening (via de-methylesterification of the cell-wall component homogalacturonan)[23] and cortical microtubule randomization[24] are associated with organ outgrowth from the SAM. It is unknown whether mechanical stress acts as an overarching directional signal to guide both matrix and cellulose microfibril modifications in hypocotyls and SAMs.

Although the load-bearing role of cellulose microfibrils was described through computational modeling approaches[25], the regulatory role of the cell-wall matrix should not be ignored. This includes its contribution to cell-wall texture (for example, spacing between wall components), wall stiffness (such as distribution and dynamics of mechanical hotspots) and wall chemistry (for example, water content and porosity). Pectins in particular are receiving increasing attention for their multiple and still-mysterious contributions. For instance, a mechanism has recently been proposed[26] by which enzymatic de-esterification of pectin nanofibrils causes anticlinal cell-wall swelling during lobe formation in leaf epidermal pavement cells, enabling cell-wall expansion independently of turgor pressure. Thus, the cell wall, rather than being a passive target, would participate in driving initial shape asymmetry independently of force input such as turgor, which itself lacks directionality (Figure 1B). This proposed mechanism for symmetry breaking is at odds with other studies regarding pectin structure and distribution, as well as the observed cell-wall growth in lobed regions, and does not entirely consider the influence of periclinal cell walls during lobe formation[27]. Moreover, these findings starkly contrast with the more generally accepted view that cell-wall mechanics and biochemistry yield to turgor. Formation and maintenance of jigsaw-puzzle-shaped pavement cells in the leaf epidermis can be explained by a response to turgor pressure. Firstly, compositional asymmetry along and across the anticlinal walls would mechanically prime the cell wall to yield anisotropically to turgor[28,29] (Figure 1B). Secondly, the resulting compression pattern in the anticlinal cell wall would generate stress hotspots, leading to local reinforcement via cellulose microfibril alignment and pectin modifications in anticlinal cell walls[30]. Thirdly, the resulting local growth restrictions would generate necks at the periclinal cell wall[31], thereby reinforcing stress hotspots through geometry, triggering local reinforcement of the cell wall and maintaining a jigsaw-puzzle shape in a 'lock-in' mechanism[16]. Intriguingly, the many mechanisms put forward earlier[26,28] independently yield initial asymmetry when modeled *in silico*, but are not necessarily mutually exclusive. Thus, numerous concurrent mechanisms may operate *in planta* in combination with cortical-microtubule-guided cellulose reinforcement to robustly control cell-shape generation and consolidation.

Cell walls provide short- and long-distance mechanical cues

In elongating tissues such as hypocotyls, anisotropic tissue growth, which itself results from anisotropic growth at the cellular level, provides additional directional mechanical cues that reinforce the effects of cellular growth patterns on tissue morphogenesis. In other words, tissue stress directs the anisotropic growth of individual cells. Typically, in a growing stem, tissue stress and cortical microtubule alignment in epidermal cells are transverse[17]. However, as shown in epidermal pavement cells, cell shape alone can prescribe a maximal tensile stress direction at a subcellular scale[16]. Stress intensity also depends on cell shape, with stress being positively related to cell width. So, in addition to the responses to tensile stress directions that allow cells to resist local stress hotspots, the formation of jigsaw-puzzle shapes may decrease the mechanical stress levels in cells by restricting their maximal diameter[11]. It follows that mechanical cues essentially act across cell and tissue scales, thereby raising questions about the integration and resolution of conflicting signals.

Cell-wall stiffness may act as an initial signal integration point. For instance, the cellulose biosynthesis inhibitor isoxaben can enhance the supracellular organization of microtubule arrays in the SAM, most likely because weaker cell walls additionally lead to increased cell-wall tension[32]. This response is also observed when cell-wall composition is modified via genetic means. For instance, cortical microtubule organization is perturbed in *A. thaliana* cellulose synthase mutants[33], and xyloglucan deficiency in *xxt1 xxt2* mutants accelerates microtubule depolymerization[34]. These findings may reflect changes in cell-wall properties (and thus stress levels), as well as putative defects in the mechanotransduction capacity of the cell wall, although this remains to be investigated.

More simply, the integration of different stress components may come down to a 'stress threshold'. The observation of consistent microtubule alignments, independent of cell geometry, following artificial modulation of tissue stress by laser ablation or tissue compression/stretching suggests that global stress patterns may override cell-based stress patterns in both leaves and hypocotyls[15,16]. It may be hypothesized that the levels of mechanical stress are crucial: if tissue stress is higher than cell-derived stress, tissue stress would also dominate as a signal. Relatively less is known, however, about the relative amplitudes of these stresses[35]. One way to assess stress amplitude would be to consider the interplay between mechanical stress and cell–cell adhesion, as discussed in the following section.

Intercellular mechanical signaling through cell–cell adhesion

For multicellular systems in which cells expand differentially, the morphogenetic transduction of information by cell walls across cell networks likely depends on the physical connectivity between the cells. This is corroborated by the observed

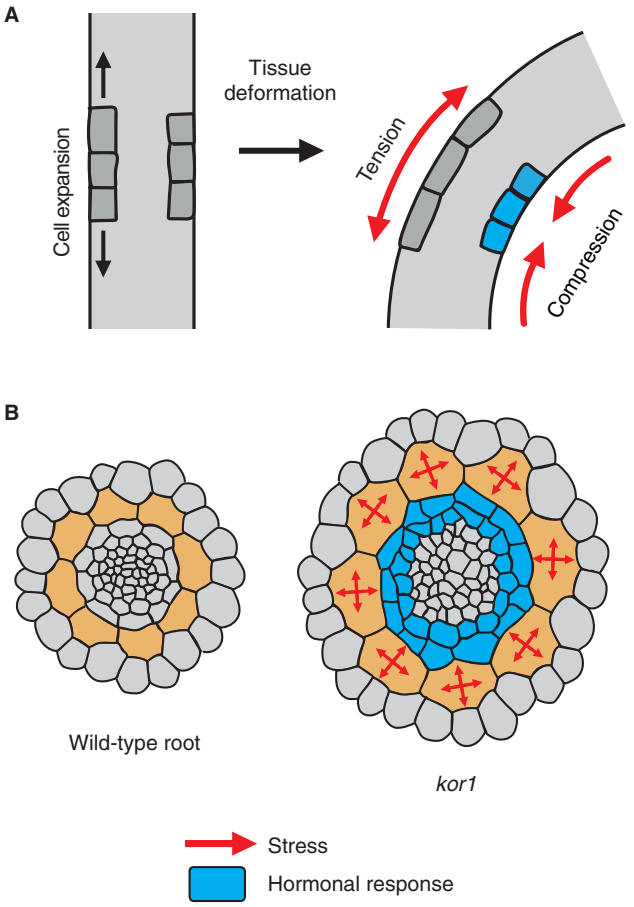

Figure 2. Tissue-based stresses influence hormonal responses.
Mechanical cues may propagate across organs to trigger hormonal responses for tissue-wide growth coordination. (A) As a result of growth asymmetry, tension and compression build up across a bending organ. In the apical hook of a seedling, compression on the inner side may trigger an auxin signaling response (depicted in blue), likely informing subsequent cellular growth decisions. (B) In the root, represented in a cross-sectional view, mechanical stress undergoes constant monitoring. When cortical cells (depicted in orange) swell, as in the cellulose synthase *kor1* mutant, inner root tissues experience compression, triggering a jasmonate hormone signaling response in the endodermal and pericycle cells (depicted in blue).

perturbation of the cell–cell coordination of microtubule organization derived from tensile stress patterns and of the tissue-level growth coordination in cell-adhesion mutants such as *qua2*[17] (Figure 1C). In *qua2* mutants, cell connectivity is severely affected as a result of defects in pectins, key components of the middle lamella that connects cells.

Interestingly, there are also distinct spatiotemporal patterns in homogalacturonan methylesterification distributions in the middle lamella. At sites with high predicted mechanical stress levels, such as tricellular junctions[36], calcium and blockwise extended continuous stretches of de-esterified homogalacturonan residues are initially highly enriched[37]. As intercellular spaces subsequently form at such junctions, for instance in cortical cells of stems, blockwise de-esterified homogalacturonan becomes concentrated at the corners of the intercellular spaces where adjacent cells are still in contact[37] and predicted stress levels are highest[36].

In contrast, high homogalacturonan methylesterification levels are maintained in flat sheets of the middle lamella between longitudinal walls of adjacent cells. Homogalacturonan methylesterification patterns in cell walls are known to influence cell-wall mechanical properties. Blockwise de-esterification, together with calcium-mediated crosslinking, is hypothesized to stiffen cell walls, while de-esterification alone is proposed to promote pectin degradation, triggering cell-wall softening[38]. However, whether and how mechanical stress influences the properties of the middle lamella is not well understood. Clearly, functional adhesion is vital for the propagation of mechanical cues between cells and within tissues, highlighting the importance of cell-wall chemistry and physical connectivity for mechanical communication in multicellular systems. However, understanding the specific contribution of the middle lamella to cellular mechanics and mechanotransduction is hampered by its partial continuity and overlapping composition with the cell wall. Intriguingly, *in vitro* studies show that increasing de-esterification by pectin methylesterase application, while softening the cell wall, simultaneously reduces acid-induced creep[39] (the formation of irreversible cell-wall extensions resulting from de-esterification activity). Such observations underline the need for more in-depth analyses of the network topology, chemistry and mechanics of cell-wall components to mechanistically link pectin modifications with mechanochemical control of growth processes.

Hormones as secondary messengers of mechanical cues

Plant hormones are often thought to provide the initial trigger of morphogenetic changes, starting with the first asymmetric cell division of zygotes to produce two unequally sized daughter cells[40]. The biochemical heterogeneity in leaf epidermal pavement cells before necks and lobes emerge suggests that biochemical cues can indeed precede mechanical signaling. Hormones are also known to act as supracellular coordinators that trigger regional increases in growth rates and directionality. Here we consider the possible feedback mechanism: once shape changes occur, the pattern of mechanical stress is also modified. Could this also channel hormone patterns and, if so, what would be the role of the cell wall in this signaling pathway?

The apical hook of germinated seedlings is an attractive model system to address these questions because their differential growth-based bending is predictable and well characterized at the molecular and mechanical levels. Asymmetric distribution of the hormone auxin is thought to underlie such processes via various mechanisms, such as H^+-ATPase-mediated apoplastic pH alteration[41], transcriptional control of cell-wall-biosynthesis and cell-wall-modifying genes[42], and cortical microtubule control[43]. While external cues such as light and gravity may trigger growth asymmetry, for instance via polar auxin transport, bending itself may elicit these responses[44]. Interestingly, during apical hook formation, hypocotyl bending appears to precede the establishment of an asymmetric auxin response[45], suggesting that the auxin machinery responds to an upstream cue. As bending progresses, cell-wall compositional and mechanical asymmetry becomes pronounced across the bending hypocotyl and seems to be concurrent with the establishment of an asymmetric auxin response that is strongly focused towards the inner, auxin-rich, slow-growing side, where cells are substantially

stiffer than cells on the outer side[46,47]. Overexpression of pectin methylesterase inhibitor enzymes, which enhance the stiffness of cells on the outer side, elicits a stronger auxin response on that side. Conversely, reduced stiffness on the inner side by xyloglucan reduction (as in *xxt1 xxt2* mutants) dampens the auxin maximum[46], indicating that auxin responds to the mechanical status of cells via a feedback loop involving cell-wall mechanics. It is unclear whether this mechanical input is mediated cell autonomously through cell- or cell-shape-based stress or non-cell autonomously by tissue-based stress. In particular, cell-wall perturbations also reduce the bending angle, likely changing the mechanical tension and compression landscape affecting cells across the bending organ.

The plant hormone ethylene is also implicated in mechanical control of growth. For example, ethylene signaling is required for penetration of hypocotyls through soil because ethylene-insensitive mutants are impaired in soil emergence. In contrast, the hookless phenotype of the *A. thaliana katanin* mutant, which is deficient in microtubule severing, can be rescued when grown in soil[48]. Since the *katanin* mutant is insensitive to ethylene treatment, ethylene may not act as a mediator of the mechanical cues from soil in this particular case. Although independent of ethylene, mechanical cues from soil nevertheless impact differential growth as suggested by analysis of the *katanin* mutant. Functional cortical microtubules are required for the establishment of an asymmetric auxin response, which is impaired in the *katanin* mutant, thereby resulting in a loss of growth repression on the inner side of the hook. Interestingly, the mechanical effect of soil — presumably mainly in direct contact with the outer side of the hook — on the *katanin* mutant is demonstrated by the restoration of auxin response asymmetry and growth repression on the inner side. This suggests that tissue-based mechanical stresses propagate across the organ and can be translated into morphological changes via hormone signaling pathways (Figure 2A).

Such mechanical control of auxin signaling is analogous to the effect of both local and global mechanical stress patterns on the plasma membrane retention and polarity of cellular auxin efflux carriers in roots, SAMs and cotyledons[14,49], essentially forming a feedback system that orchestrates tissue patterning. Similarly, in roots, hormone responses may be induced by non-cell-autonomous mechanical signals, given that cortical cell swelling in the *A. thaliana* cellulose synthase *kor1* mutant triggers a jasmonate hormone response specifically in endodermal and pericycle (but not epidermal) cells[50]. This is thought to arise from the compression of these internal cell layers, which are physically constrained, unlike epidermal cells at the root surface (Figure 2B).

Conclusions

To conclude, the interplay between mechanical and hormonal signals is more ubiquitous than initially anticipated. Mechanical forces may constitute ubiquitous and continuously changing primary sources of morphogenetic cues throughout plant development. These cues cell-autonomously and non-cell-autonomously guide the growth of tissues and the cells within them, in large part by triggering hormonal responses. Such hormonal action may translate the mechanical cues into growth responses by controlling morphogenetic processes, such as shifts in gene expression and cell-wall modifications, to consolidate morphogenetic behavior. In this scenario, the dialog between mechanical and chemical cues constitutes a critical regulator of morphogenesis.

The inner structure of plant cell walls and the interactions between the key cell-wall components are being elucidated with increasing precision and resolution. The multilamellar structure of the cell wall and the distinct rigidity and elasticity of its different layers need to be considered when explaining the role of the cell wall as a hub for mechanochemical control of morphogenesis. Equally important are the observations that the genetic or chemical perturbations that result in cell-wall softening, such as overexpression of pectin methylesterase or a deficiency in xyloglucan, are associated with reduced acid-induced creep. Thus, cell-wall softening may not necessarily lead to a corresponding increase in growth. Similarly, multiple *in silico* models explain growth asymmetry in the case of leaf epidermal pavement cells, indicating the complexity of cell-wall-mediated mechanical control of morphogenesis. The context of mechanochemical alteration of cell-wall changes also needs to be considered, as this may be relevant for assessing the impact of cell-wall-mediated signaling on morphogenesis. Additionally, it is essential to better connect *in vivo* and *in vitro* methods to assess dynamic changes in cell-wall mechanics and composition, and to integrate and test model predictions. Nevertheless, a platform has now been established for answering some of the key questions in mechanochemical control of plant morphogenesis, such as: how do cells sense stress direction and stress levels in cell walls? How do compositional, mechanical or topological changes in the cell wall act as signals for controlling downstream responses? How are mechanical cues translated into hormonal responses? How do microtubules respond to cell-wall stress? Addressing these questions will provide further important insights into the interplay between mechanical cues, the cell wall and hormonal responses in the control of plant morphogenesis.

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DECLARATION OF INTERESTS

The authors declare no competing interests.

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