

Growth Control and Cell Wall Signaling in Plants

Sebastian Wolf,* Kian Hématy,* and Herman Höfte

Institut Jean-Pierre Bourgin, UMR 1318 INRA/AgroParisTech, 78026 Versailles Cedex, France; email: sebastian.wolf@versailles.inra.fr, kian.hematy@versailles.inra.fr, herman.hofte@versailles.inra.fr

Annu. Rev. Plant Biol. 2012. 63:381–407

First published online as a Review in Advance on January 3, 2012

The *Annual Review of Plant Biology* is online at plant.annualreviews.org

This article's doi:
10.1146/annurev-arplant-042811-105449

Copyright © 2012 by Annual Reviews.
All rights reserved

1543-5008/12/0602-0381\$20.00

*Both authors contributed equally.

Keywords

cell wall integrity signaling, pathogen resistance, receptor kinases, mechanosensing

Abstract

Plant cell walls have the remarkable property of combining extreme tensile strength with extensibility. The maintenance of such an exoskeleton creates nontrivial challenges for the plant cell: How can it control cell wall assembly and remodeling during growth while maintaining mechanical integrity? How can it deal with cell wall damage inflicted by herbivores, pathogens, or abiotic stresses? These processes likely require mechanisms to keep the cell informed about the status of the cell wall. In yeast, a cell wall integrity (CWI) signaling pathway has been described in great detail; in plants, the existence of CWI signaling has been demonstrated, but little is known about the signaling pathways involved. In this review, we first describe cell wall–related processes that may require or can be targets of CWI signaling and then discuss our current understanding of CWI signaling pathways and future prospects in this emerging field of plant biology.

Contents

INTRODUCTION	382
PLANT CELL WALL ARCHITECTURE:	
BELT AND BRACES	382
CELL WALLS AND GROWTH	
CONTROL	384
Cell Wall Synthesis and Cell Expansion	384
Pectin and Growth Control	388
Xyloglucan and Growth Control	390
CELL WALL SIGNALING	
IN PLANTS	391
Wall-Derived Signaling Molecules ..	391
Signal Perception at the Cell Surface	392
Wall-Associated Kinases	393
The CrRLK1-Like Family of Cell Wall Signaling Receptors	393
Downstream Signaling	396

INTRODUCTION

One of the most striking features of plant architecture is the use of a hydrostatic skeleton to generate the large surfaces required for efficient photosynthesis at low metabolic cost. To this end, plant cells are large and pressurized owing to two essential attributes: a large central vacuole, allowing the accumulation of water and solutes, and a strong cell wall. The wall is made primarily of carbohydrates (an abundant building material for a photosynthetic organism) with only a small amount of protein.

Cell walls are highly heterogeneous and complex structures, which in growing cells have the remarkable property of combining extreme tensile strength with extensibility. Once the final cell size is reached, a secondary cell wall is deposited in many cell types. The latter provides strength to cells and tissues even after the cell content has disappeared, and it can be made impermeable—for instance, in water-conducting vessels—through the incorporation of lignin. In some storage cells, the secondary walls contain easily degradable polysaccharides

that are used as a carbon reserve (156). Finally, cell wall polymers also mediate cell–cell adhesion and, as discussed below, can have a role in signaling.

PLANT CELL WALL ARCHITECTURE: BELT AND BRACES

Plant cell walls show a large diversity in composition in different species, in different cell types, and even in different subcellular cell wall domains and over time during cellular differentiation (49, 69, 136, 149). Despite this diversity, all cell walls are composite materials with a similar building plan based on stiff and tensionally strong cellulose microfibrils, which are cross-linked to a matrix consisting of hemicellulose and/or pectin, structural proteins, and, in certain cell types, lignin (41). In what follows, we focus on the primary dicot walls (so-called type I walls) because most of our knowledge of cell wall integrity (CWI) signaling concerns this type of wall.

The growth of walled cells depends on the force balance between the extensibility of the wall and the mechanical force exerted by the turgor pressure of the cells. In principle, growth can be controlled by changing either parameter; in most cases, however, growth changes reflect changes in wall mechanics. As outlined below, the wall extensibility is an emergent property of a complex network of covalent and noncovalent interactions between cell wall polymers. To understand growth regulation, it is fundamental to understand how the strength of these interactions can be modulated and kept in check.

Excellent recent reviews have appeared on the composition and synthesis (22, 27, 156, 164), growth (41), and evolution (140) of cell wall polymers. We therefore do not recapitulate the structure and synthesis of every polysaccharide here, but instead focus on the architectural features that are relevant for CWI control.

The primary cell wall is a robust structure consisting of partially redundant but interdependent polysaccharide and protein networks. Cellulose microfibrils, with their extreme

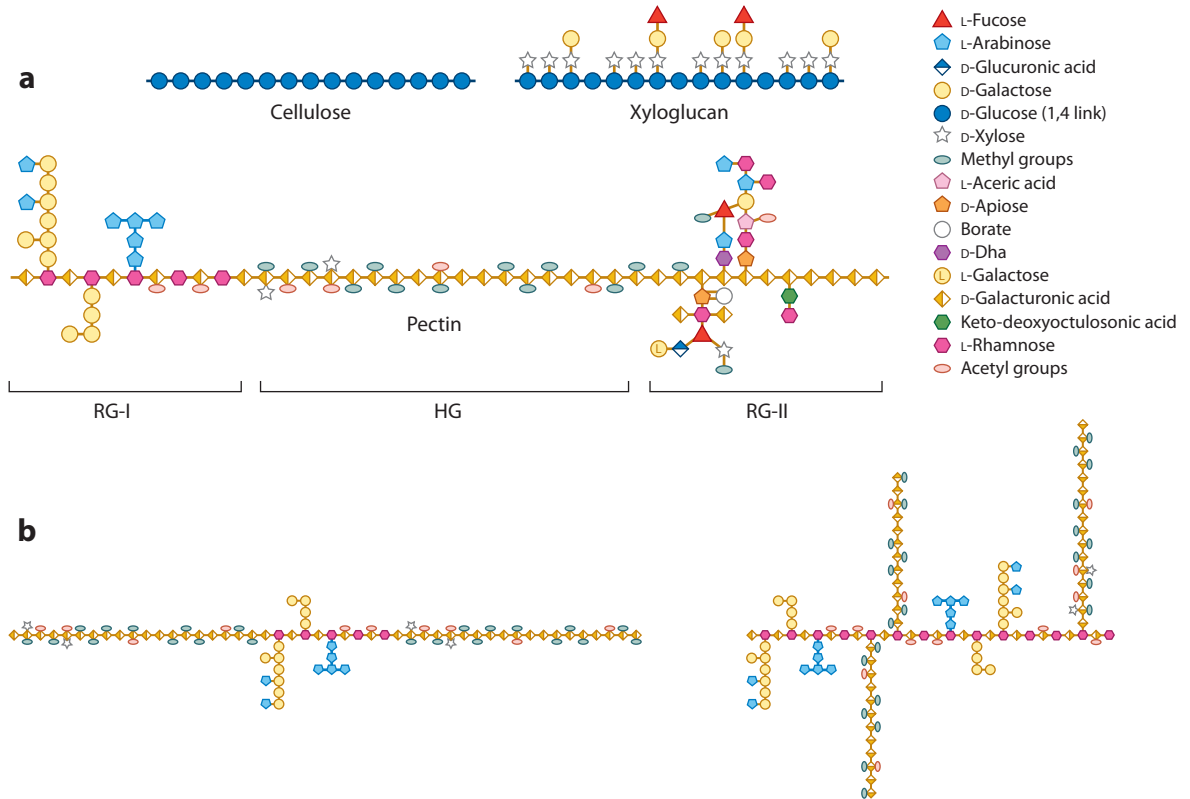


Figure 1

Structure of some cell wall components. (a) Schematic representation of cellulose, xyloglucan, and the pectic rhamnogalacturonan I (RG-I), homogalacturonan (HG), and RG-II. Adapted from Reference 22 with permission. (b) Alternative models for pectin domain organization. A linear, contiguous arrangement of HG interspersed with RG-I is shown on the left, whereas on the right, HG is drawn as side chains linked to Rha residues of the RG-I scaffold. A combination of both models may also be possible. Adapted from Reference 152 with permission.

tensile strength, are the main load-bearing elements of the cell wall. In most cases, microfibrils are highly oriented, creating mechanical anisotropy in the wall, which in turn determines the growth direction of the cell. Matrix polysaccharides not only cross-link microfibrils but also prevent the self-association of nascent microfibrils into larger aggregates. In type I walls, xyloglucan (XG) is the main hemicellulose (**Figure 1**). XG not only strongly binds to the cellulose surface but also may be trapped inside the microfibril (133) either during synthesis or through a covalent link via a transglycosylation reaction (89). Depending on the chain length, XG chains can cross-link microfibrils and reinforce the cell wall (168, 188).

How XG chain length (and hence the degree of cross-linking) is regulated is discussed below. Interestingly, this is not the only load-bearing network. Indeed, *Arabidopsis xxt1/xtt2* mutants, which entirely lack XG, have only subtle growth phenotypes and walls with mechanical properties comparable to those of the wild type (34). Pectin can also interact in vitro with cellulose through arabinan or galactan side chains of rhamnogalacturonan I (RG-I) (**Figure 1**) (203). Pectin molecules can be cross-linked to each other either through Ca^{2+} bonds between demethyl-esterified portions of homogalacturonan (HG) or through borate-diester bonds between two RG-II molecules (147). Recent 3-D solid-state nuclear magnetic

resonance data further confirmed in situ interactions between cellulose and pectin and showed that pectin and XG mechanically behave as a single entity (54), perhaps through covalent pectin-XG links (139).

In addition to the polysaccharide network, structural proteins also play an important role in cell wall architecture, and typically constitute approximately 10% of the wall of growing cells. Among the structural proteins, extensins are defined as extracellular, basic, hydroxyproline (Hyp)-rich structural glycoproteins with alternating hydrophilic ($X\text{-Hyp}_n$) and hydrophobic motifs that frequently carry tyrosine residues as potential cross-linking sites (105). *Arabidopsis* has 20 highly similar extensin genes (30). Mutant analysis shows an essential role for certain extensins in primary cell wall assembly. For instance, AtEXT3 is present in cell plates and mature walls, and loss-of-function mutants for the corresponding gene are embryo lethal with incomplete cell plates. The amphiphilic extensins can self-assemble into networks in vitro, as shown by atomic force microscopy (30). Interestingly, the positively charged extensins can create an interpenetrating network with negatively charged pectin, and thus constitute a scaffold for the assembly of the new cell plate. Ingenious in vitro studies (175) showed that extensin promotes the dehydration and compaction of the pectin and suggested that besides charge, the extensin conformation is also critical for this strong interaction. This conformation depends on proline hydroxylation and the presence of *O*-linked arabinosyl side chains on Hyp (179). Interestingly, pectin demethyl-esterification increases the negative charge density of pectin and thus can also promote extensin-pectin interaction and cell wall compaction (156).

CELL WALLS AND GROWTH CONTROL

Cell Wall Synthesis and Cell Expansion

The study of the growth regulation of various cell types suggests that turgor-driven cell

expansion is the result of a delicate balance between wall relaxation and wall stiffening linked by a mechanosensing feedback loop (**Figure 2d**). As shown below, this can be observed clearly by analyzing the oscillatory growth of tip-growing root hairs and pollen tubes, but it is likely that a similar regulatory loop controls the growth of diffusely growing cells (122). In principle, one can distinguish the following events: (a) hydration of newly deposited cell wall material and wall relaxation (**Figure 2a**); (b) turgor-driven deformation of the cell wall; (c) mechanosensing, followed by the release of apoplastic reactive oxygen species (ROS), cross-linking, partial dehydration, and stiffening of the wall; and (d) the secretion of new wall material. In what follows, these events are discussed separately.

Wall hydration. Rapid auxin-induced growth acceleration [lag time of 8–10 min (39)] is mediated at least in part by the activation of the plasma membrane proton ATPase (P-ATPase), causing the hyperpolarization of the plasma membrane and the acidification of the apoplast (39). Interestingly, this in turn causes the rapid hydration and swelling of the cell wall as observed in *Arabidopsis* hypocotyls (26). A more pronounced swelling (up to 40%) was observed upon addition of the brassinosteroid hormone brassinolide (BL). BL binds the receptor kinase BRI, which in turn activates the P-ATPase, presumably through a direct BL-promoted protein-protein interaction and requiring the BRI kinase activity (**Figure 2a**) (26). The mechanism of the cell wall swelling is not well understood, but it requires both acidification and plasma membrane hyperpolarization. One actor in this process may be the wall-loosening protein expansin, which has the ability to promote hydration of isolated walls with an acidic pH optimum (169, 195). The contribution of the plasma membrane hyperpolarization to the cell swelling in vivo is not clear, but it might influence the delivery of new matrix material to the cell wall.

The regulation of the hydration status of the wall also may involve other enzyme

activities. For instance, β -galactosidase activity encoded by the *MUM2* gene is required for normal hydration of seed mucilage in *Arabidopsis*. In the mutant *mum2*, normal amounts of mucilage are produced, which, however, fails to hydrate (111). Interestingly, the mutant mucilage has not only higher galactose content but also increased HG-methyl-esterification (184, 187). This suggests that the β -galactosidase activity is required for pectin methyl-esterase (PME, EC 3.1.1.11; see below) activity. The physicochemical basis of the effect of the removal of galactose residues and/or the PME activity on the hydration of the mucilage remains to be determined.

Wall extension. Increased hydration promotes the extensibility of the wall, as shown on isolated sunflower coleoptile walls (64). This is presumably the result of the increased spacing between the cellulose microfibrils in the hydrated wall, which should facilitate their rearrangement. For polylamellated walls, this rearrangement involves the rotation of the microfibrils (110), which was recently observed in living *Arabidopsis* root cells (1). Expansins are also thought to promote microfibril rearrangement through the rupturing of hydrogen bonds between cellulose and XGs (41). As discussed below, the control of XG length may also contribute to promoting wall rearrangement (168, 177).

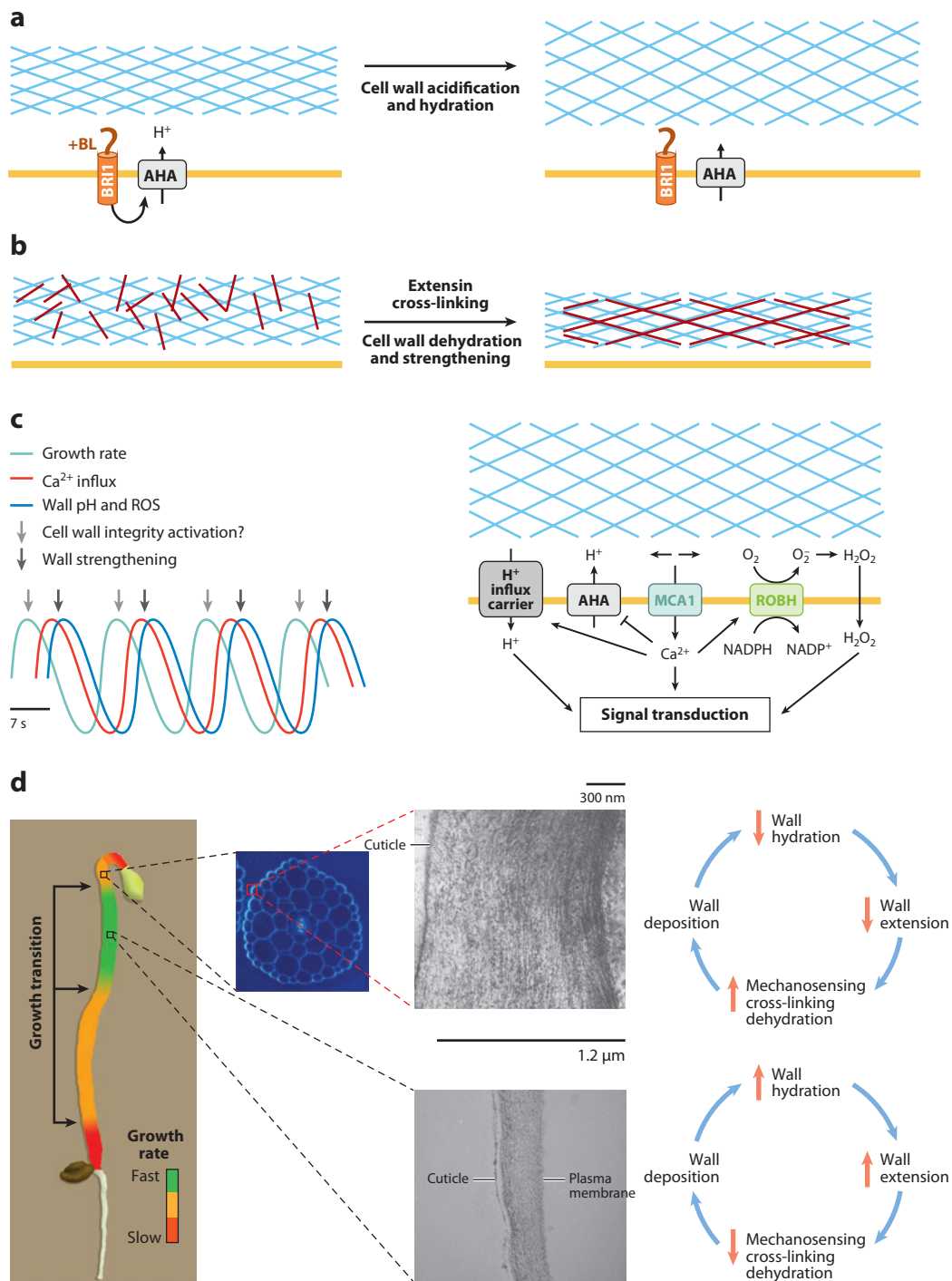
Mechanosensing and wall cross-linking.

Work on tip-growing root hairs and pollen tubes has shown that growth rate, cytosolic calcium levels, and cytosolic and apoplastic pH oscillate with the same period (**Figure 2c**) (120). The phase of the oscillations and the results of inhibitor studies suggest the involvement of a mechanosensing feedback loop (121): Wall relaxation and cell expansion are expected to stretch the plasma membrane and to open stretch-activated calcium channels. The resulting increase in cytosolic calcium slows down growth in at least two ways: First, it inhibits the P-ATPase and opens H^+ channels, leading to the alkalization of the apoplast, which in turn may lead to the inhibition of expansin

activity, the activation of PMEs, and pectin cross-linking (see below); second, it activates the plasma membrane NADPH-oxidase. This enzyme releases superoxide into the cell wall, which is rapidly converted into H_2O_2 spontaneously or catalyzed by apoplastic superoxide dismutases. H_2O_2 and peroxidases can cause the cross-linking of cell wall components, which may be extensins (105), feruloylated pectin (129), or monolignols (176), depending on the cell type and environment (**Figure 2c**). This leads to partial wall dehydration and slowing down of growth (**Figure 2b**) (137). Besides stretch-activated calcium channels, other mechanosensors may also play a role in growth control (see below).

Wall deposition. The accumulation of new wall material at the tip of growing pollen tubes also oscillates, and it peaks before the growth rate, indicating that cell wall deposition initiates a new growth cycle (117). The composition of the secreted material—which, besides polysaccharides, can include wall-modifying proteins, structural proteins, and phenolic compounds—depends on the cellular and environmental context. Cellulose is synthesized by large cellulose synthase complexes (CSCs) that migrate in the plasma membrane, propelled by the polymerization of the glucan chains. This can be observed spectacularly in living cells by using green fluorescent protein (GFP)-labeled CSCs (131).

Cortical microtubules play a key role in the organization of the cellulose deposition. They target the insertion of the CSCs at specific positions in the plasma membrane (42, 76), guide their trajectories, may control the CSC velocity (36), and may determine the density of the CSCs in the plasma membrane (42). Paradoxically, in the absence of cortical microtubules—for instance, upon treatment with the microtubule inhibitor oryzalin—the CSCs are still inserted into the plasma membrane and continue their trajectories (110). The difference is that insertion patterns are random and no re-orientation of the trajectories is possible. The cortical microtubules therefore orchestrate the



plasticity of the cellulose deposition patterns required for the generation of complex cell shapes in higher plants.

In contrast to cellulose, matrix polysaccharides are synthesized in the Golgi apparatus and transported via vesicles to the cell surface. (For excellent recent reviews and detailed discussions of the synthesis of matrix polysaccharides, see 22, 27, 156, 157.) It should be noted here that XGs are delivered to the cell surface as precursor molecules of unknown size, which can be modified in the apoplast by glycoside hydrolases and incorporated into the polymer by XG endo-transglycosylases (XETs) (156). Given the large molecular weights of certain pectin molecules (145), it is conceivable that similar apoplastic assembly steps occur, but so far no pectin-transglycosylase activity has been demonstrated (27, 102). The high turgor pressure of plant cells (0.5–1.5 MPa) appresses the plasma membrane to the cell wall. This high pressure also forces newly secreted wall material through pores into the cell wall. For instance,

the cell wall of the green alga *Chara* has a pore size of approximately 4.6 nm. Interestingly, depending on the turgor pressure, molecules with a larger diameter can be forced through these pores, probably by partially unfolding or deforming the polymer (141, 143). The insertion of new wall material into pores (intussusception) of a stretched wall should lead to a larger plastic deformation of the inner wall layers as opposed to the outer layer. This is indeed suggested by the buckling of inner layers in plasmolyzed cells observed in etiolated sunflower hypocotyls (82). Finally, the secretion of wall material can be restricted to highly specific locations within the cell—for instance, during the formation of specific cellular outgrowths (70) or pathogen attack (83).

The four events of the regulatory loop discussed above can be controlled differently in different cellular and environmental contexts. This is strikingly illustrated in the dark-grown *Arabidopsis* hypocotyl, where cells show two distinct postmitotic growth phases: a slow growth

Figure 2

Cell walls and growth control. (a) Growth promotion through wall hydration. In vivo cell wall swelling (at least in part due to hydration) has been observed within minutes after brassinolide or auxin treatment of *Arabidopsis* root or hypocotyl cells. Brassinolide-induced wall swelling requires the activation of the receptor BRI1, which in turn associates with and activates the P-type proton ATPase (AHA1). The combined apoplastic acidification and plasma membrane hyperpolarization causes the cell wall swelling (26). Hydration increases the extensibility of the wall, presumably by enlarging the distance between cellulose microfibrils (64). (b) Growth inhibition through extensin cross-linking. The hydroxyproline-rich cell wall protein extensin (red bars) can form networks through intermolecular tyrosine cross-links. The amphiphilic extensin binds to pectin through electrostatic interactions, leading to the dehydration of the matrix. This may be part of the mechanism for the decrease in cell wall extensibility and growth deceleration (137, 175). (c) Negative control of growth through a mechanosensing feedback loop. The growth rate of pollen tubes or root hairs oscillates with the same period as cytosolic Ca^{2+} concentration, intracellular and apoplastic pH, and extracellular reactive oxygen species (ROS) levels (121, 167). The phase shift of the respective oscillations and the results of pharmacological experiments lead to the following model: Wall extension causes plasma membrane stretching and Ca^{2+} influx, presumably through a stretch-activated Ca^{2+} channel related to MCA1. The increased cytosolic Ca^{2+} concentration, in turn, slows down growth by negatively regulating AHA1 and enhancing H^{+} influx. The resulting wall alkalization might inhibit the activity of expansins and activate wall-consolidating enzymes such as pectin methyltransferase. In addition, increased cytosolic Ca^{2+} levels cause ROS production through the activation of NADPH-oxidase. This leads to wall strengthening through cross-linking of, for instance, extensins. Panel adapted from Reference 167 with permission. (d) A model for a growth-controlling regulatory loop. This model summarizes evidence obtained from the different systems mentioned above. Growth acceleration is initiated by apoplastic acidification and plasma membrane hyperpolarization, causing the hydration and the increase in extensibility of the cell wall. Turgor-driven cell wall extension takes place, which in turn leads to the activation of the mechanosensing feedback loop, cell wall cross-linking, and dehydration. New cell wall material is deposited and inserted into the wall, and a new cycle is initiated. Plant growth regulators and environmental factors can impinge on different steps of this cycle. This is exemplified by *Arabidopsis* dark-grown hypocotyl development. Hypocotyl cells first show a phase of slow growth during which a thick, polylamellated wall is deposited. During this phase, wall hydration levels may be low and mechanosensing may be high. Transition to rapid growth may be initiated by increased wall hydration and loosening and reduced mechanosensing. After the growth acceleration, wall extension outpaces wall synthesis, resulting in the thin walls associated with elongated cells. Panel images adapted from References 65 and 146 with permission.

phase during which a thick multilamellated cell wall is deposited, followed by an abrupt growth acceleration (fivefold increase in strain rate), after which wall extension outpaces wall synthesis, leading to extensive thinning of the wall (**Figure 2d**) (49, 146). One can speculate that in slowly growing cells, the walls maintain a low extensibility through limited hydration and increased mechanosensing, whereas the growth acceleration may be initiated by the hydration of the multilamellated wall, the secretion of additional wall-loosening agents, and the lowering of the feedback control. Brassinosteroid- and auxin-induced wall swelling was indeed observed in this organ (26), as was the upregulation of transcripts encoding wall-loosening proteins prior to and during the growth acceleration (136). Finally, a lowering of the feedback control was also suggested by the lack of growth inhibition by the cellulose inhibitor isoxaben once the growth had accelerated (136, 146). Abiotic stresses and/or pathogen attack impinge on this scheme by promoting ROS production, extensin cross-linking (64), and (mostly irreversible) growth inhibition.

Pectin and Growth Control

A prominent example of how chemical modification of a component of the cell wall can influence its extensibility is the *in muro* demethyl-esterification of HG by PME (CAZY CE8). It is generally accepted that the amount and distribution of methyl groups impact the pectin matrix's rheological properties, adhesive capacities, and resistance against degradation (190). However, the precise nature and properties of individual HG epitopes remain to be elucidated (38, 189). One well-characterized epitope, at least *in vitro*, exhibits Ca²⁺-mediated cross-linking of blocks (>9) of demethyl-esterified HG, forming so-called egg-box pectin with characteristic gelling properties (25). The capacity of PMEs to produce multiple distinct epitopes is potentially modulated and refined by small interacting proteins termed PME inhibitors, the pH of the apoplast, and the methyl-esterification status of the neighboring GalA

residues (28, 29, 33, 47, 91, 96). Furthermore, regulated secretion of PME and/or the presence of alternative trafficking routes for polysaccharides might influence the biogenesis of pectin methyl-esterification epitopes (43, 108, 150, 171, 191). In addition to its effect on the properties of the pectin matrix, PME activity is also a prerequisite for pectin turnover, because the HG-degrading activities of polygalacturonases and presumably pectate lyases (114) are dependent on certain patterns of methyl-esterification (68, 182, 183). Immunological and spectroscopic detection of highly methyl- and demethyl-esterified HG epitopes seem to suggest that the former is associated with extensible walls in growing parts of the cell, whereas the latter is associated with nonextensible walls (50, 90, 132, 136). A well-documented example is on display in the pollen tube, where a highly methyl-esterified epitope is restricted to the growing tip region, whereas a less methyl-esterified epitope is confined to the shank. Notably, a sharp border zone is located at the position where wall stiffness (measured by microindentation) increases and where cell wall stress is predicted to peak by computational modeling (12, 66, 199). However, in more complex tissues, the correlation might be much less clear (135). Although there seems to be a general agreement that the amount of *in muro* demethyl-esterification correlates with the stiffness conferred on the wall by HG, little is known about the persistence and half-lives of the different epitopes that are observed *in situ* through immunolabeling. Therefore, a more rigid HG epitope could actually be a short-lived intermediate toward degradation and hence softening of the wall. This was illustrated by recent work on the *Arabidopsis* shoot apical meristem, in which the initiation of organ primordia depends on HG demethyl-esterification. Through the use of atomic force microscopy, a causal relationship could be established between HG demethyl-esterification and cell wall softening, not stiffening (134). A major challenge for future research will be the development of cell-biological tools for real-time measurements of the turnover of pectic

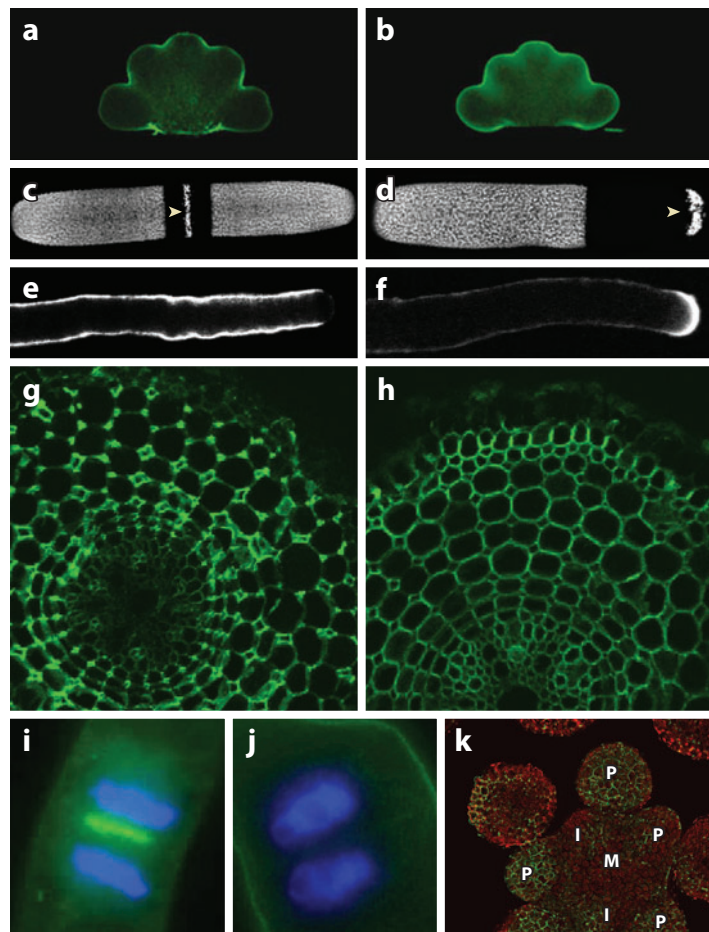
epitopes. The identification of pectin-binding domains (32, 46, 166, 181), which could be used as genetically encoded sensors, or the use of click chemistry for *in vivo* labeling of pectin (107) might help to overcome this bottleneck.

Judging from the presence or absence of different pectic polysaccharides in present-day plant taxa, HG is the evolutionarily most ancient pectin, whereas the more complex RG-I and RG-II seem to be later additions (116, 139, 165). HG can be readily detected in cell walls of charophycean green algae (CGAs), the group that presumably harbors the extinct

predecessor of land plants (56, 57, 60, 141). Interestingly, immunolabeling of CGA cell walls indicated the presence of HG epitopes with different degrees of methyl-esterification, very similar to what is observed in higher plants (**Figure 3**). Specifically, an immunolabel for highly methyl-esterified (and thus presumably softer) pectin was enriched in actively growing parts of the algal cells, mimicking the distribution described above for pollen tubes (13, 56, 57, 60, 132). Therefore, wall consolidation by regulated pectin demethyl-esterification seems to be a relatively old mechanism. This

Figure 3

Evolution of pectic epitopes. The functional module regulating pectin methyl-esterification status is present in both charophycean green algae and higher plants, where its use has expanded. Panels show immunostaining of pectin with JIM5 (panels *a*, *e*, *g*, and *i*) and 2F4 (panel *k*), both labeling an epitope with a low degree of methyl-esterification, and immunostaining of pectin with JIM7 (panels *b*, *f*, *h*, and *j*), which labels an epitope with a high degree of methyl-esterification. (*a,b*) Pectin immunolabeling of the charophycean green alga *Microsterias denticulate* with (*a*) JIM5 and (*b*) JIM7. Adapted from Reference 60. (*c,d*) *Penium margaritaceum* pulse-labeled with JIM5 and JIM7 (*arrowheads*) (*c*) before and (*d*) after cell separation. Algae were first labeled with JIM5, then recultured and labeled with JIM7. Only sites of active pectin secretion are strongly labeled by JIM7. Adapted from Reference 56. (*e,f*) Pectin immunolabeling of tobacco pollen tubes with (*e*) JIM5 and (*f*) JIM7, which label the shank and growing tip, respectively (14). Thus, in pollen tubes, as in *Penium* (*c* and *d*), the JIM7 epitope is associated with the growing apex. Adapted from Reference 14. (*g,h*) Pectin immunolabeling of root cross sections of rice with (*g*) JIM5 and (*h*) JIM7. JIM5 labels only the cell corners, whereas JIM7 labels the walls more homogeneously. Adapted from Reference 194. (*i,j*) Pectin immunolabeling of dividing tobacco BY-2 cells with (*i*) JIM5 and (*j*) JIM7. JIM5 labels pectin in the forming cell plate, whereas JIM7 does not. Adapted from Reference 53. (*k*) Demethyl-esterification of pectin precedes primordia formation in *Arabidopsis* shoot apical meristems, as demonstrated by labeling with the 2F4 antibody. Abbreviations: M, meristem dome; I, incipient primordium; P, primordium. Adapted from Reference 135. All images used with permission.



preexisting system of pectin demethyl-esterification has apparently been adopted by embryophytes for the control of development in the much more intricate context of a multicellular organism. For example, a profound impact of regulated HG methyl-esterification has been demonstrated for cellular adhesion (59, 103, 124, 170, 186), fruit ripening (19, 138), hypocotyl development (50, 136), pollen maturation (68), and, as mentioned above, phylloclastic patterning in the meristem (134, 135).

Taken together, the functional module controlling HG methyl-esterification status seems to have expanded from a relatively simple wall consolidation mechanism, as present in CGAs and pollen tubes, to an array of more complex functions in multicellular organisms. This is reflected, possibly, by the emerging interlacing of pectin with other cell wall polymers (see above) and the multitude of PME isoforms that differ in pI, pH optimum, salt dependence, and other traits. As a result, in higher plants, PME may promote at least the following processes: (a) wall stiffening by favoring the formation of egg boxes and electrostatic interactions with extensin (175), (b) wall loosening through promotion of wall hydration, (c) wall degradation through initiation of pectin turnover, and (d) wall signaling by favoring the generation of active oligogalacturonides (OGAs), small breakdown products of HG (discussed below).

Interestingly, the presence of HG with different methyl-esterification degrees coincides with the first appearance of the rosette-type CSCs in the CGAs (140, 165). Furthermore, evidence is accumulating for direct interaction between cellulose and pectin (54, 201–203) as well as for a role of pectin in cellulose deposition (196). Reconstruction of pectin-cellulose networks by bacterial cellulose synthesis into a preexisting pectin framework demonstrated not only a requirement for a certain degree of methyl-esterification but also pectin-induced alterations in the extensibility and stiffness of the microfibril network, which persisted even after the removal of the matrix polysaccharides from the network (35).

Consistent with these data, target analysis of cobtorin, a compound that disrupts microfibril alignment, identified HG as a key regulator of cellulose microfibril deposition (196).

Xyloglucan and Growth Control

XG strongly binds to the cellulose surface (79) and, depending on the length of the backbone and the nature of the side chains, can form cross-links between microfibrils (79, 188). These parameters are regulated by the interplay between sugar hydrolases and XETs in the apoplast. Recent findings suggest an unexpected role for phosphorylation in the regulation of enzyme activity in the cell wall. Phosphoproteomics on cell wall extracts from tobacco cell cultures showed that three cell wall enzymes are tyrosine-phosphorylated, two of which— α -xylosidase and β -glucosidase—are involved in XG and XG/cellulose metabolism, respectively (93). The cell wall-located purple acid phosphatase (PAP) dephosphorylates the two hydrolases *in vitro* and *in vivo*, which causes the loss of their activity. As expected, XG- and cello-oligosaccharides accumulated in a PAP-overexpression tobacco line (92, 93). Interestingly, PAP overexpression also promotes β -glucan (cellulose and callose) synthesis at the plasma membrane (92). The authors speculated that secretion of PAP indirectly stimulates β -glucan synthesis through the inhibition of XG and cello-oligosaccharide turnover. More work is needed, however, to prove such a causal relationship. These findings raise new important questions: What is the mechanism for tyrosine phosphorylation of cell wall proteins? How is the secretion of PAP regulated? Do XG- and/or cello-oligosaccharides promote glucan synthesis, and what is the biological relevance for such a regulation? Another consequence of the accumulation of these oligosaccharides may be the promotion of cell elongation. Indeed, Takeda et al. (168) showed in pea stem segments that the incorporation by XET of short XG fragments into XG promoted cell elongation, presumably through the loosening of the cell wall.

CELL WALL SIGNALING IN PLANTS

As outlined above, regulation of the cell wall's physical properties is pivotal for plant growth control during development. Moreover, the wall is constantly subjected to various extrinsic chemical and biophysical cues and constitutes a primary target of plant pathogens. Thus, the existence of a cell wall signaling pathway that reports on the status of the wall has long been inferred. Recently, evidence has begun to mount in support of this hypothesis. For example, cell wall biosynthesis and cell wall remodeling genes are differentially regulated in various cell wall-related mutants (reviewed in 158) and in response to such conditions as inhibition of cellulose synthesis (113), interference with arabinogalactan proteins (AGPs) (74), and exposure to pectin fragments (20, 123).

In addition, interference with CWI results in a variety of compensatory reactions, which have been studied extensively using genetic or pharmacological inhibition of cellulose synthesis. Under these conditions, a canonical set of responses seems to emerge—namely, ROS production, ectopic deposition of lignin (31, 48), altered pectin methyl-esterification status (21, 87, 113), elevated callose deposition (52), and an increase in jasmonic acid and ethylene production with an associated resistance to pathogens (61, 62). Further evidence for the existence of CWI signaling comes from the intriguing observation that stunted growth and reduced cell elongation associated with attenuation of cell wall biosynthesis are responses mediated by signaling components, rather than caused directly by a physically weakened cell wall (85, 173). Therefore, the reduced growth of many cell wall mutants is, at least to some extent, rather a secondary effect and can be considered part of the list of responses to cell wall damage outlined above.

Another context in which CWI signaling may play a role is the control of the cellulose-matrix ratio. In light of the two different subcellular locations of the synthesis, it is surprising that the cellulose-matrix ratio is so reproducible

for a given tissue at a given developmental stage. What controls this homeostasis is not known. An obvious metabolic connection is constituted by the equilibration of nucleotide sugar precursors for the different polysaccharides. The substrate for cellulose synthesis, UDP-Glc, is converted into other nucleotide sugars that are precursors for XG, pectin, and other polysaccharides (157), and in principle, polysaccharide ratios may be controlled by regulating the nucleotide sugar interconversion enzyme activities. Controlling the cellulose-matrix ratio in this way would require the presence of a cytoplasmic UDP-Glc pool shared between the Golgi and the plasma membrane biosynthetic processes. It should be noted, however, that the free cytosolic UDP-Glc pool is very small compared with that of sucrose [for instance, 30–35 versus 1,000 nmol mg⁻¹ chlorophyll in spinach leaves (71)], probably too small to support high cellulose-synthesis rates. An alternative possibility is that abundant cytosolic sucrose is used by plasma membrane-located sucrose synthases that channel UDP-Glc directly into the cellulose synthase (see Reference 40 for evidence for a role of sucrose synthase in cellulose synthesis and Reference 6 for an opposing view). This suggests that another mechanism exists to coordinate matrix delivery with cellulose synthesis. In this context it is interesting to note that pectin may actually undergo continuous secretion and internalization in growing cells (5, 53), which might facilitate the control of the cellulose-pectin ratio.

Despite the overwhelming evidence for cell wall signaling, only relatively few components involved in the respective pathways have been identified (see below).

Wall-Derived Signaling Molecules

It is a long-standing observation that specific cellular responses can be elicited by certain wall fragments that are thus considered signaling molecules (2, 3). For example, OGAs—short breakdown products of HG with a chain length between 9 and 15 GalA residues—can cause changes in gene expression, stomatal closure,

ethylene production, cell wall reinforcement, and ROS production. The potential functions of OGAs in defense have drawn by far the most attention, because pectin is a prominent target for many microbial cell wall-degrading enzymes during pathogenic attack (77, 123, 128, 130, 147, 163). However, a role during normal plant development also seems likely (8, 18). The signaling function of OGAs is dependent on HG demethyl-esterification and hence PME activity, as only certain HG epitopes are prone to degradation (68, 182, 183) and a certain amount of demethyl-esterification is essential for OGA activity (130).

As discussed above, Ca^{2+} -mediated HG cross-linking is associated in vitro with pectin gelation; however, egg-box formation is also potentially important for the biological activity of OGAs (25). The egg-box conformation is prone to perturbation by excess monovalent or divalent cations and the presence of polycations such as polyamines and chito-oligosaccharides, the latter of which are short deacetylated breakdown products of chitin (24, 25). Although speculative at this point, it is worth noting that these dynamic interactions, which are able to modulate plant responses (24), could be perceived by a cell wall surveillance system and in this way provide a sensing system for salt stress or pathogenic attack (P. Van Cutsem, personal communication).

In addition to a structural role, XG oligosaccharides may also have a role in signaling. XG nonasaccharides (XG₉s) have been shown to inhibit auxin-stimulated elongation of pea stem segments at nanomolar concentrations (197). XG₉ shows a bell-shaped dose-response curve owing to the XET-mediated growth-stimulatory effect of XG₉ at higher concentrations mentioned above. The specificity of the XG₉ effect was shown by the total absence of activity of XG₁₀ (XG₉ + galactose) and XG₈ (XG₉ – fucose), the latter showing the requirement of fucosylation for activity. The highly specific activity suggested the existence of an XG₉ receptor that may play a role in the feedback regulation of auxin-induced growth. However, the discovery of the *Arabidopsis* XG fucosyl

transferase mutant *mur2*, which produces XG lacking fucose and which does not show an observable phenotype (178), suggested that XG signaling does not play a significant role in vivo. Alternatively, XG signaling capacity may be species dependent (pea versus *Arabidopsis*), or other fucosyltransferase family members [there are 10 family members in *Arabidopsis* (154)] may allow the production of small amounts of fucosylated XG at critical developmental stages.

Signal Perception at the Cell Surface

Plasma membrane-localized kinase proteins with a distinguished extracellular domain appear to be primary candidates for CWI receptors. Among the over 600 receptor-like kinases (RLKs) in *Arabidopsis* (161), several proteins have been implicated in cell wall-related signaling. However, it is unclear to what extent structural cell wall components are actual ligands of these proteins (85, 193). For example, an interesting group of potential cell wall receptors are L-type lectin RLKs (17), of which one member has been shown to bind RGD (arginine–glycine–aspartic acid) tripeptides and has been implicated in plasma membrane–cell wall attachment (72). Although lectin domains are known for carbohydrate binding (9), no carbohydrate-binding partner for the lectin-like extracellular domain has been proposed.

In contrast, several RLKs have been implicated in cell wall binding, but no clear connection to CWI signaling has been established. Interestingly, one member of the proline-rich extensin-like receptor kinase (PERK) family involved in abscisic acid signaling has been found to be pectin associated (4). Other RLKs bind carbohydrates, such as those of the PR5-like receptor kinase/thaumatin family and the LysM family, but seem to have a specific role in defense signaling (94, 112, 118, 144, 160, 172, 185). Together, these findings demonstrate at the very least the multitude of carbohydrate-binding extracellular domains among RLKs.

Besides RLKs, stretch-activated ion channels are promising candidates for CWI sensors, because any weakening of the cell wall is

expected to influence membrane properties. For example, MCA1 partially complements the yeast *mid1* mutants and hence encodes a putative calcium channel likely involved in mechanoperception (**Figures 2c** and **4**) (125). In addition, plant homologs of the bacterial mechanosensitive channels of small conductance (MscS-like proteins) were shown to be required for mechanosensitive ion channel activity (80). Various other proteins (such as AGPs) may be implicated in cell wall signaling, linking the cell wall with the plasma membrane and potentially with the cytoskeleton (153, 162) and leucine-rich repeat (LRR) extensins, which could be cell wall cross-linked signaling molecules (7, 55). An intriguing recent discovery is the finding that AtFH1, a protein homologous to animal formin with an extracellular extensin-like domain, can interact with the cell wall and is localized in plasma membrane patches that align with actin microfilaments. Thus, AtFH1 could provide a direct link between the cell wall and the cytoskeleton, analogous to the integrin mechanosensors in mammalian cells (**Figure 4**) (115). In addition, a protein with sequence similarity to integrins has been recently identified and is involved in plasma membrane–cell wall adhesion (98).

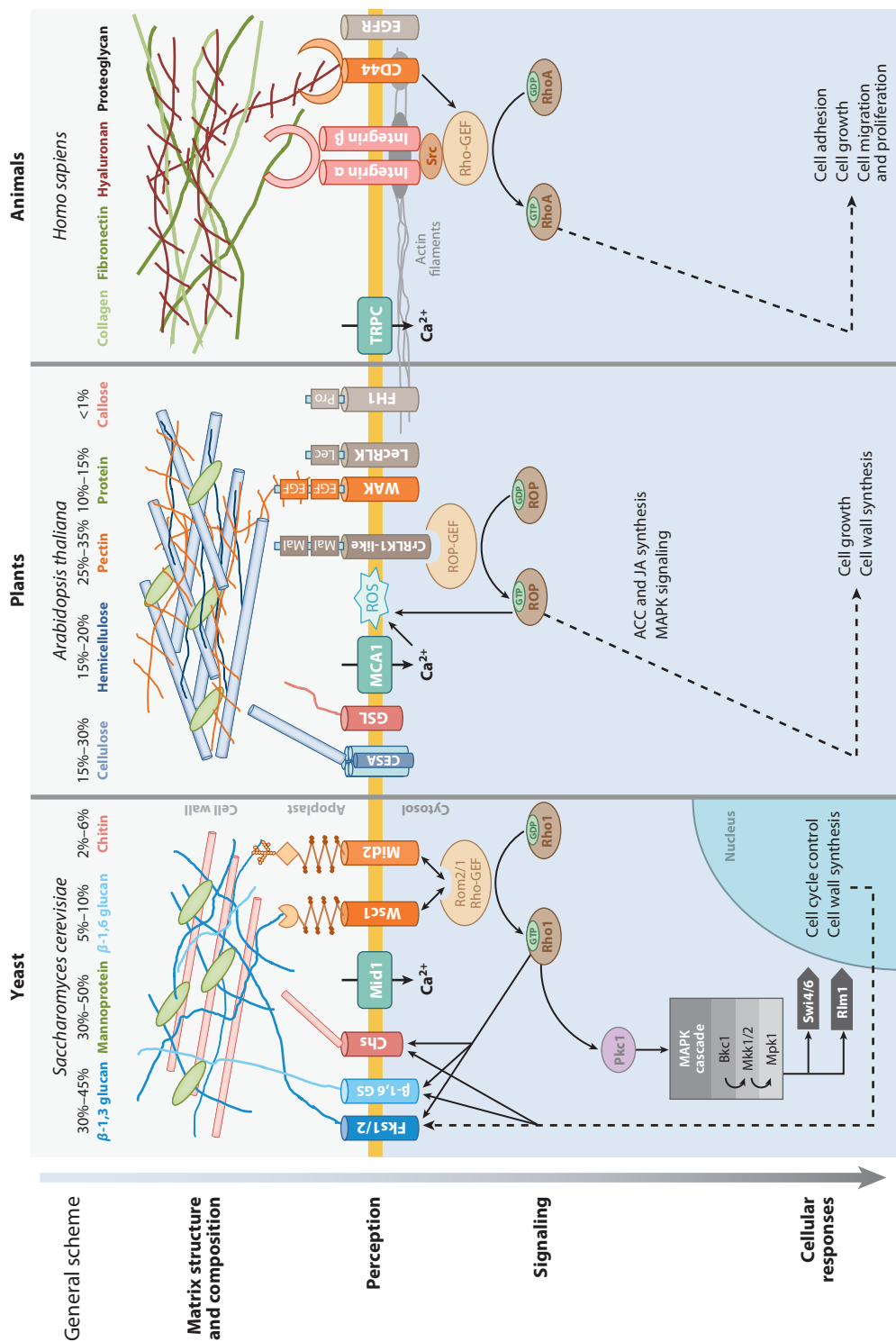
Recent findings show that the interaction of plasma membrane proteins with the cell wall may be more common than previously thought. One interesting case is the auxin efflux carrier PIN1 (67). The polar localization of this protein within the cell is responsible for the establishment of auxin gradients that control patterning and growth. FRAP (fluorescence recovery after photobleaching) experiments on GFP-labeled versions of PIN1 reveal a surprisingly low lateral mobility in the plasma membrane. This is most likely the result of an interaction with the cell wall, because cellulose-deficient mutants or very low concentrations of the cellulose-synthesis inhibitor isoxaben perturb the polar localization of GFP-PIN1. It will be interesting to see with which cell wall components PIN1 interacts and whether changes in the cell wall during development have an impact on auxin signaling in this way.

Wall-Associated Kinases

One of the best-studied potential CWI receptors belongs to the family of wall-associated kinases (WAKs). The *Arabidopsis* genome harbors five WAK genes arranged in a gene cluster and several WAK-like genes. WAKs are characterized by an intracellular serine–threonine kinase domain and an extracellular domain that contains epidermal growth factor–like repeats (81, 99). The WAK1 extracellular domain binds tightly to pectins (181), apparently with a pronounced preference for the Ca²⁺-mediated egg-box conformation (45, 46). Recently, using chimeric receptors between WAK1 and EFR, the receptor for the pathogen-associated molecular pattern EF-Tu (200), Brutus et al. (20) provided evidence that WAK1 is the receptor for OGAs in *Arabidopsis*. Despite the interaction of the WAK1 extracellular domain with OGAs, it remains unclear whether WAK1 is also involved in CWI signaling. The WAK1 extracellular domain can be sedimented in vitro with Ca²⁺-cross-linked HG, and therefore interaction is certainly not limited to short fragments (45). This, together with the above-mentioned possibility that biotic and abiotic stress could be perceived through conformational changes of egg-box pectin, places WAK1 among the most interesting candidates for true CWI receptors. Consistent with this, WAKs and WAK-like proteins have been implicated in cell elongation, salt tolerance, and the coordination of solute concentrations with growth (81, 88, 100, 101, 104, 181).

The CrRLK1-Like Family of Cell Wall Signaling Receptors

Members of the *Catharanthus roseus* RLK1 (CrRLK1)–like protein family have recently emerged as interesting candidates for CWI sensors. This plant-specific family of RLKs contains 17 members in *Arabidopsis* (84). *THESEUS1* (*THE1*) was identified in a screen for the suppression of the elongation defect of the cellulose-deficient mutant *cesa6/procuste1* (*prc1*) (85). Mutations in *THE1* attenuate the growth



inhibition and ectopic lignification of several cellulose-deficient mutants without rescuing the cellulose deficiency. *THE1* is also required for the oxidative burst induced by the cellulose-synthesis inhibitor isoxaben in the root (48). Among the genes upregulated in *prc1*, a subset depend on *THE1* signaling. Some of those encode ROS-detoxifying enzymes, extensins, and a peroxidase, suggesting a role in cell wall cross-linking. Other genes encode enzymes involved in the synthesis of glucosinolates and other defense proteins, indicating a role in pathogen defense. Together, these data suggest a role for *THE1* in transducing the responses to cell wall perturbations. The responses include ROS production, leading to the inhibition of cell growth, presumably through cell wall cross-linking. The responses may be triggered during pathogen attack and perhaps during normal development.

Other *Arabidopsis* members of the CrRLK1-like family have been implicated in control of cell growth in different biological contexts (10, 37, 84). Most notably, *FERONIA* (*FER*),

together with *THE1* and *HERCULES1* receptor kinase (*HERK1*), is required for normal brassinosteroid-induced cell elongation in both light and dark growth conditions (51, 75). *FER* is broadly expressed during plant development and is necessary for normal cell elongation of various cell types, including root hairs (58, 75). In the ovule, *FER* is specifically expressed in the synergid cells, where it is polarly localized at the filiform apparatus to detect pollen tube arrival and trigger discharge of its sperm cells (63). Interestingly, *ANXUR1/2* (*ANX1/2*), the two closest paralogs of *FER*, are expressed in pollen and are necessary for pollen tube growth because *anx1anx2* mutant pollen tubes burst prematurely before reaching the ovule (11, 119). Most recently, *FER* has been shown to control the polar localization of *AtMLO7/NORTIA*, another protein required for pollen tube recognition by the synergid (95). *AtMLO7* belongs to a 15-member protein family initially identified by *MLO1*, an integral membrane protein with seven membrane-spanning domains required for powdery mildew susceptibility in

Figure 4

Schematic illustration of the differences and similarities found in cell wall (CW) integrity pathways across kingdoms. Yeasts (*Saccharomyces cerevisiae*) have a layered cell wall mostly composed of noncrystalline β -glucans and highly mannosylated proteins on the outer face and chitin on the inner face (97). Higher-plant cell walls instead have a crystalline backbone made of cellulose microfibrils interconnected by hemicelluloses and embedded in a pectic matrix with few structural proteins (198). In mammals, the extracellular matrix (ECM) is organized around a protein scaffold made of collagen and fibronectin. It also contains proteoglycans from the group of glycosaminoglycans and a matrix of the polysaccharide hyaluronic acid. The ECM/CW integrity signaling pathway can be activated by several stresses, including mechanical forces (e.g., changes in the osmolarity of the environment), changes in the structure or composition of the ECM/CW during the cell cycle and cell expansion, and (a)biotic stresses. Perturbation of ECM/CW integrity is perceived by several kinds of sensors located at the plasma membrane; mechanical stress-induced membrane deformation can be sensed by stretch-activated calcium channels (namely, *Mid1*, *MCA1*, and *TRPC* in *Saccharomyces cerevisiae*, *Arabidopsis thaliana*, and *Homo sapiens*, respectively) but also by a variety of receptors that can probe different components of the ECM/CW. In *Saccharomyces*, the main sensors are *Mid2* and *Wsc1*, which are both anchored in the membrane and exhibit an extracellular domain composed of a highly mannose-rich region (STR domain) conferring nanospring properties on the sensors (109, 148). In animal cells, integrins transduce mechanical signals by linking the extracellular fibronectin scaffold to the intracellular cytoskeleton to sense ECM remodeling and control cell proliferation and migration (106). Hyaluronan is bound and internalized by the receptor *CD44* (15, 16). In *Arabidopsis*, several receptor-like kinase families comprise candidates for a CW integrity sensor, such as the CrRLK1-like family (including *THE1* and *FER*) and the pectin-bound wall-associated kinases (WAKs). Despite the variability in cell wall composition and sensor types between the three systems, some common mechanisms seem to be in place, such as mechanoreception through stretch-activated ion channels, downstream signaling through Rho-GTPases, and a common set of responses leading to ECM/CW repair and cell fate control. The signaling pathway downstream from the sensors is best characterized in yeast, where activation of Rho is relayed by *Plc1* to a mitogen-activated protein kinase (MAPK) cascade leading to nucleus translocation of the transcription factors *Rml1* and *Swi*, which control CW synthesis and cell cycle progression, respectively. A faster CW repair can be induced by Rho through posttranslational regulation of glucan synthases (e.g., *Fks1*). Other abbreviations: ACC, 1-aminocyclopropane-1-carboxylic acid; JA, jasmonate; ROS, reactive oxygen species.

barley (23). Interestingly, *fer* homozygous mutants are also resistant to powdery mildew (95), possibly as a result of the mislocalization of an MLO7-like protein.

The only direct downstream targets of a CrRLK1-like family member reported so far are ROPGEF proteins (58). ROPGEFs are guanine-exchange factors required for activation of the plant Rho GTPases (RAC/ROPs) into their GTP-bound state (73, 126, 192). FER is part of a dynamic complex containing a ROPGEF and a RAC/ROP. GTP-bound RAC/ROPs activate NADPH-oxidases, which produce ROS required for root hair formation.

Ligands for the CrRLK1-like extracellular domains are not known. All family members contain one or two copies of a domain with sequence similarity to the carbohydrate-binding malectin domain (Mal) from *Xenopus laevis* (10), which binds Glc₂-N-glycan in the endoplasmic reticulum (155). This suggests that CrRLK1-like proteins also recognize carbohydrate-containing ligands.

In conclusion, the CrRLK1-like family appears to be part of a regulatory module controlling ROS production, presumably triggered by a carbohydrate-containing ligand and involving, at least in one case, the RAC/ROP-mediated activation of NADPH-oxidase. This module has been recruited to control growth in different developmental contexts: pollen tube elongation (ANX1/2), synergid-induced pollen tube growth arrest and bursting (FER), root hair elongation (FER), fungal pathogen response (FER), control of leaf expansion (THE1, HERK, FER), and inhibition of cell expansion upon wall damage (THE1) (**Figure 4**).

Downstream Signaling

Several lines of evidence point to the involvement of ROS and the phytohormones ethylene, jasmonate, and salicylic acid in signaling events downstream of cell wall damage perception. Moreover, numerous studies have demonstrated convergence or overlap with defense signaling pathways (86, 127, 180). For example, the *cev1* (*cesa3*) mutant displays enhanced

ethylene and jasmonate signaling and shows increased resistance toward various pathogens (62), whereas treatment with isoxaben induces jasmonate and salicylic acid synthesis (78).

Interestingly, recent studies have demonstrated an involvement of 1-aminocyclopropane-1-carboxylic acid (ACC), the direct precursor of ethylene, in regulating cell wall function and the response to cell wall damage (44, 173, 193). Simultaneous mutation of two LRR-type RLKs, FEI1 and FEI2, leads to a conditional cell wall-related phenotype: *fei1fei2* mutant cells show anisotropic growth, ectopic lignin deposition, and hypersensitivity to isoxaben under high-sucrose or high-salt conditions, reminiscent of the AGP mutant *sos5* and cellulose-deficient mutants such as *prc1* and *cob-1* (65, 151, 159). In contrast to combination with *prc1* and *cob*, *fei1fei2* showed nonadditive effects when combined with *sos5* in a triple mutant, which suggests a role for the three genes in the same pathway.

Interestingly, interference with ACC function through the application of either a biosynthesis inhibitor (aminoethoxyvinylglycine or aminoxyacetic acid) or of a nonfunctional structural analogue (aminoisobutyric acid) completely rescued the *fei1fei2* conditional phenotype. Importantly, genetic or pharmacological interference with ethylene perception could not mimic this effect. These data, together with the observations that the FEIs can interact with ACC synthase and that the FEI kinase domain is dispensable for mutant complementation, suggest that the two RLKs are involved in recruiting a signaling platform that uses ACC rather than ethylene as a signaling molecule. Consistent with such a role for ACC in contrast to ethylene, mutants completely insensitive to ethylene are viable and seem relatively healthy, whereas simultaneous knockdown of multiple ACC synthase isoforms results in embryonic lethality (174).

Evidence for the involvement of ACC in signaling downstream of cell wall damage perception came from the cell length analysis in the root elongation zone in response to ACC and isoxaben (44, 173). It was shown that ACC

and isoxaben rapidly inhibit cell elongation (lag time <1 h) and that this inhibition is reversed by interfering with ACC synthesis, but not with ethylene perception. Remarkably, cell length could be completely reverted to wild-type levels despite the reduced cellulose content in the wall. As a result, the cells showed excessive bulging consistent with a fragilized cell wall (173). The short-term inhibition of cell elongation caused by integrity defects fosters the notion that reduced elongation after cell wall damage is mediated by signaling and is not a mere biophysical effect of the weakened cell wall (85). In addition to ACC, H₂O₂ production and auxin biosynthesis (but not auxin transport) were necessary for the short-term

inhibition of cell elongation in response to isoxaben. Conversely, the long-term response additionally relied on both ethylene perception and auxin transport, suggesting a cell-autonomous short-term response involving ACC and a long-term non-cell-autonomous response involving ethylene (173). It has to be noted, though, that cellulose synthesis is downstream of the proposed FEI signaling pathway, whereas isoxaben is the agent used for cell wall perturbation in the study of Tsang et al., thus placing cellulose biosynthesis upstream of the signaling events (173, 193). Therefore, ACC seems to function both in the control of cell wall synthesis and in the response to cell wall damage. Further work is needed to clarify this conundrum.

SUMMARY POINTS

1. The plant cell wall is a dynamic composite structure, consisting of distinct but interdependent networks that change during cellular development and in response to biotic or abiotic stresses.
2. Plant cells monitor the status of their cell walls to coordinate physicochemical and cellular processes involved in cell growth control.
3. Plants cells possess various types of sensors at the plasma membrane (and possibly in the cell wall) that can probe mechanical deformations or changes in cell wall structure or composition.
4. As for yeast and animals, early CWI signaling involves activation of Rho-like GTPases.
5. Plant cells respond to cell wall perturbation by a rapid growth inhibition coupled with production of ROS, ACC, and jasmonate and by reinforcing their cell walls through deposition of lignin and cross-linking of cell polymers.

FUTURE ISSUES

1. Researchers should work to establish a detailed understanding of cell wall architecture and critical interactions between polymers, including predictive models for the control of cell expansion.
2. Further development of real-time *in vivo* imaging tools is needed for the quantification of growth rate and parameters critical for the control of wall extensibility: ROS, pH, Ca²⁺, cell wall deposition rate, polymer composition and orientation, pectin-methyl-esterification status, and mechanical properties.
3. Actors involved in CWI signaling need identification using, for instance, forward genetic screens in cell wall-damaging conditions or analysis of protein complexes.

4. Cell wall-associated and free ligands for CrRLK1-like, lectin RLK, and other putative cell wall sensors are also still in need of identification.
5. The structural basis of the receptor-ligand interactions and the mechanism of CWI sensing need to be elucidated.
6. The CWI signaling networks require reconstruction, including the roles of ROPGEF, ROP, ROS, ACC, mitogen-activated protein kinase (MAPK), and jasmonate.
7. The role of regulated secretion and endocytosis in the regulation of cell wall deposition and remodeling needs dissection.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

We apologize to all colleagues whose work could not be cited owing to space constraints. We gratefully acknowledge support from the Agence Nationale de la Recherche [grants BLAN08-1_332764 (“Wallintegrity”), ANR-09-BLANC-0007-01 (“Growthpec”), and ANR-10-BLAN-1516 (“Mechastem”)], and the EU FP7 project “RENEWALL.” S.W. is the recipient of a fellowship from the German Research Foundation (Deutsche Forschungsgemeinschaft).

LITERATURE CITED

1. Anderson CT, Carroll A, Akhmetova L, Somerville C. 2009. Real-time imaging of cellulose reorientation during cell wall expansion in *Arabidopsis* roots. *Plant Physiol.* 152:787–96
2. Ayers AR, Valent B, Ebel J, Albersheim P. 1976. Host-pathogen interactions: XI. Composition and structure of wall-released elicitor fractions. *Plant Physiol.* 57:766–74
3. Aziz A, Gauthier A, Bezier A, Poinssot B, Joubert JM, et al. 2007. Elicitor and resistance-inducing activities of beta-1,4 cellooligosaccharides in grapevine, comparison with beta-1,3 glucans and alpha-1,4 oligogalacturonides. *J. Exp. Bot.* 58:1463–72
4. Bai L, Zhang G, Zhou Y, Zhang Z, Wang W, et al. 2009. Plasma membrane-associated proline-rich extensin-like receptor kinase 4, a novel regulator of Ca signalling, is required for abscisic acid responses in *Arabidopsis thaliana*. *Plant J.* 60:314–27
5. Baluska F, Hlavacka A, Samaj J, Palme K, Robinson DG, et al. 2002. F-actin-dependent endocytosis of cell wall pectins in meristematic root cells: insights from brefeldin A-induced compartments. *Plant Physiol.* 130:422–31
6. Barratt DH, Derbyshire P, Findlay K, Pike M, Wellner N, et al. 2009. Normal growth of *Arabidopsis* requires cytosolic invertase but not sucrose synthase. *Proc. Natl. Acad. Sci. USA* 106:13124–29
7. Baumberger N, Ringli C, Keller B. 2001. The chimeric leucine-rich repeat/extensin cell wall protein LRX1 is required for root hair morphogenesis in *Arabidopsis thaliana*. *Genes Dev.* 15:1128–39
8. Bellincampi D, Cardarelli M, Zaghi D, Serino G, Salvi G, et al. 1996. Oligogalacturonides prevent rhizogenesis in rolB-transformed tobacco explants by inhibiting auxin-induced expression of the rolB gene. *Plant Cell* 8:477–87
9. Beneteau J, Renard D, Marche L, Douville E, Lavenant L, et al. 2010. Binding properties of the N-acetylglucosamine and high-mannose N-glycan PP2-A1 phloem lectin in *Arabidopsis*. *Plant Physiol.* 153:1345–61
10. Boisson-Dernier A, Kessler SA, Grossniklaus U. 2011. The walls have ears: the role of plant CrRLK1Ls in sensing and transducing extracellular signals. *J. Exp. Bot.* 62:1581–91

11. Boisson-Dernier A, Roy S, Kritsas K, Grobei MA, Jaciubek M, et al. 2009. Disruption of the pollen-expressed *FERONIA* homologs *ANXUR1* and *ANXUR2* triggers pollen tube discharge. *Development* 136:3279–88
12. Bolduc JE, Lewis LJ, Aubin CE, Geitmann A. 2006. Finite-element analysis of geometrical factors in micro-indentation of pollen tubes. *Biomech. Model Mechanobiol.* 5:227–36
13. Bosch M, Cheung AY, Hepler PK. 2005. Pectin methylesterase, a regulator of pollen tube growth. *Plant Physiol.* 138:1334–46
14. Bosch M, Hepler PK. 2005. Pectin methylesterases and pectin dynamics in pollen tubes. *Plant Cell* 17:3219–26
15. Bourguignon LY. 2001. CD44-mediated oncogenic signaling and cytoskeleton activation during mammary tumor progression. *J. Mammary Gland. Biol. Neoplasia* 6:287–97
16. Bourguignon LY. 2008. Hyaluronan-mediated CD44 activation of RhoGTPase signaling and cytoskeleton function promotes tumor progression. *Semin. Cancer Biol.* 18:251–59
17. Bouwmeester K, Govers F. 2009. *Arabidopsis* L-type lectin receptor kinases: phylogeny, classification, and expression profiles. *J. Exp. Bot.* 60:4383–96
18. Branca C, Lorenzo GD, Cervone F. 1988. Competitive inhibition of the auxin-induced elongation by α -D-oligogalacturonides in pea stem segments. *Physiol. Plant.* 72:499–504
19. Brummell DA, Harpster MH. 2001. Cell wall metabolism in fruit softening and quality and its manipulation in transgenic plants. *Plant Mol. Biol.* 47:311–40
20. Brutus A, Sicilia F, Macone A, Cervone F, De Lorenzo G. 2010. A domain swap approach reveals a role of the plant wall-associated kinase 1 (WAK1) as a receptor of oligogalacturonides. *Proc. Natl. Acad. Sci. USA* 107:9452–57
21. Burton RA, Gibeat DM, Bacic A, Findlay K, Roberts K, et al. 2000. Virus-induced silencing of a plant cellulose synthase gene. *Plant Cell* 12:691–705
22. Burton RA, Gidley MJ, Fincher GB. 2010. Heterogeneity in the chemistry, structure and function of plant cell walls. *Nat. Chem. Biol.* 6:724–32
23. Buschges R, Hollricher K, Panstruga R, Simons G, Wolter M, et al. 1997. The barley *Mlo* gene: a novel control element of plant pathogen resistance. *Cell* 88:695–705
24. Cabrera JC, Boland A, Cambier P, Frettinger P, Van Cutsem P. 2010. Chitosan oligosaccharides modulate the supramolecular conformation and the biological activity of oligogalacturonides in *Arabidopsis*. *Glycobiology* 20:775–86
25. Cabrera JC, Boland A, Messiaen J, Cambier P, Van Cutsem P. 2008. Egg box conformation of oligogalacturonides: the time-dependent stabilization of the elicitor-active conformation increases its biological activity. *Glycobiology* 18:473–82
26. Caesar K, Elgass K, Chen Z, Huppenberger P, Witthoft J, et al. 2011. A fast brassinolide-regulated response pathway in the plasma membrane of *Arabidopsis thaliana*. *Plant J.* 66:528–40
27. Caffall KH, Mohnen D. 2009. The structure, function, and biosynthesis of plant cell wall pectic polysaccharides. *Carbohydr. Res.* 344:1879–900
28. Cameron RG, Luzio GA, Goodner K, Williams MAK. 2008. Demethylation of a model homogalacturonan with a salt-independent pectin methylesterase from citrus: I. Effect of pH on demethylated block size, block number and enzyme mode of action. *Carbohydr. Polym.* 71:287–99
29. Cameron RG, Luzio GA, Vasu P, Savary BJ, Williams MA. 2011. Enzymatic modification of a model homogalacturonan with the thermally tolerant pectin methylesterase from Citrus: 1. Nanostructural characterization, enzyme mode of action, and effect of pH. *J. Agric. Food Chem.* 59:2717–24
30. Cannon MC, Terneus K, Hall Q, Tan L, Wang Y, et al. 2008. Self-assembly of the plant cell wall requires an extensin scaffold. *Proc. Natl. Acad. Sci. USA* 105:2226–31
31. Cano-Delgado A, Penfield S, Smith C, Catley M, Bevan M. 2003. Reduced cellulose synthesis invokes lignification and defense responses in *Arabidopsis thaliana*. *Plant J.* 34:351–62
32. Carpin S, Crevecoeur M, de Meyer M, Simon P, Greppin H, Penel C. 2001. Identification of a Ca^{2+} -pectate binding site on an apoplastic peroxidase. *Plant Cell* 13:511–20
33. Catoire L, Pierron M, Morvan C, du Penhoat CH, Goldberg R. 1998. Investigation of the action patterns of pectinmethylesterase isoforms through kinetic analyses and NMR spectroscopy: implications in cell wall expansion. *J. Biol. Chem.* 273:33150–56

34. Cavalier DM, Lerouxel O, Neumetzler L, Yamauchi K, Reinecke A, et al. 2008. Disrupting two *Arabidopsis thaliana* xylosyltransferase genes results in plants deficient in xyloglucan, a major primary cell wall component. *Plant Cell* 20:1519–37
35. Chanliaud E, Gidley MJ. 1999. *In vitro* synthesis and properties of pectin/*Acetobacter xylinus* cellulose composites. *Plant J.* 20:25–35
36. Chen S, Ehrhardt DW, Somerville CR. 2010. Mutations of cellulose synthase (CESA1) phosphorylation sites modulate anisotropic cell expansion and bidirectional mobility of cellulose synthase. *Proc. Natl. Acad. Sci. USA* 107:17188–93
37. Cheung AY, Wu HM. 2011. THESEUS 1, FERONIA and relatives: a family of cell wall-sensing receptor kinases? *Curr. Opin. Plant Biol.* 14:632–41
38. Clausen MH, Willats WG, Knox JP. 2003. Synthetic methyl hexagalacturonate hapten inhibitors of anti-homogalacturonan monoclonal antibodies LM7, JIM5 and JIM7. *Carbohydr. Res.* 338:1797–800
39. Cleland R. 2004. Auxin and cell elongation. In *Plant Hormones, Biosynthesis, Signal Transduction, Action!*, ed. PJ Davis, pp. 204–20. Dordrecht: Kluwer Acad. 3rd ed.
40. Coleman HD, Yan J, Mansfield SD. 2009. Sucrose synthase affects carbon partitioning to increase cellulose production and altered cell wall ultrastructure. *Proc. Natl. Acad. Sci. USA* 106:13118–23
41. Cosgrove DJ. 2005. Growth of the plant cell wall. *Nat. Rev. Mol. Cell Biol.* 6:850–61
42. Crowell EF, Bischoff V, Desprez T, Rolland A, Stierhof YD, et al. 2009. Pausing of Golgi bodies on microtubules regulates secretion of cellulose synthase complexes in *Arabidopsis*. *Plant Cell* 21:1141–54
43. De Caroli M, Lenucci MS, Di Sansebastiano GP, Dalessandro G, De Lorenzo G, Piro G. 2011. Protein trafficking to the cell wall occurs through mechanisms distinguishable from default sorting in tobacco. *Plant J.* 65:295–308
44. De Cnodder T, Vissenberg K, Van Der Straeten D, Verbelen JP. 2005. Regulation of cell length in the *Arabidopsis thaliana* root by the ethylene precursor 1-aminocyclopropane-1-carboxylic acid: a matter of apoplastic reactions. *New Phytol.* 168:541–50
45. Decreux A, Messiaen J. 2005. Wall-associated kinase WAK1 interacts with cell wall pectins in a calcium-induced conformation. *Plant Cell Physiol.* 46:268–78
46. Decreux A, Thomas A, Spies B, Brasseur R, Van Cutsem P, Messiaen J. 2006. In vitro characterization of the homogalacturonan-binding domain of the wall-associated kinase WAK1 using site-directed mutagenesis. *Phytochemistry* 67:1068–79
47. Denes JM, Baron A, Renard CM, Pean C, Drilleau JF. 2000. Different action patterns for apple pectin methylesterase at pH 7.0 and 4.5. *Carbohydr. Res.* 327:385–93
48. Denness L, McKenna JF, Segonzac C, Wormit A, Madhou P, et al. 2011. Cell wall damage-induced lignin biosynthesis is regulated by a ROS- and jasmonic acid dependent process in *Arabidopsis*. *Plant Physiol.* 156:1364–74
49. Derbyshire P, Findlay K, McCann MC, Roberts K. 2007. Cell elongation in *Arabidopsis* hypocotyls involves dynamic changes in cell wall thickness. *J. Exp. Bot.* 58:2079–89
50. Derbyshire P, McCann MC, Roberts K. 2007. Restricted cell elongation in *Arabidopsis* hypocotyls is associated with a reduced average pectin esterification level. *BMC Plant Biol.* 7:31
51. Deslauriers SD, Larsen PB. 2010. FERONIA is a key modulator of brassinosteroid and ethylene responsiveness in *Arabidopsis* hypocotyls. *Mol. Plant* 3:626–40
52. Desprez T, Vernhettes S, Fagard M, Refregier G, Desnos T, et al. 2002. Resistance against herbicide isoxaben and cellulose deficiency caused by distinct mutations in same cellulose synthase isoform CESA6. *Plant Physiol.* 128:482–90
53. Dhonukshe P, Baluska F, Schlicht M, Hlavacka A, Samaj J, et al. 2006. Endocytosis of cell surface material mediates cell plate formation during plant cytokinesis. *Dev. Cell* 10:137–50
54. Dick-Perez M, Zhang Y, Hayes J, Salazar A, Zabolina OA, Hong M. 2011. Structure and interactions of plant cell-wall polysaccharides by two- and three-dimensional magic-angle-spinning solid-state NMR. *Biochemistry* 50:989–1000
55. Diet A, Link B, Seifert GJ, Schellenberg B, Wagner U, et al. 2006. The *Arabidopsis* root hair cell wall formation mutant *hrx1* is suppressed by mutations in the *RHMI* gene encoding a UDP-L-rhamnose synthase. *Plant Cell* 18:1630–41

56. Domozych DS, Lambiasse L, Kiemle SN, Gretz MR. 2009. Cell-wall development and bipolar growth in the desmid *Penium margaritaceum* (Zygnematophyceae, Streptophyta): asymmetry in a symmetric world. *J. Phycol.* 45:879–93
57. Domozych DS, Serfis A, Kiemle SN, Gretz MR. 2007. The structure and biochemistry of charophycean cell walls: I. Pectins of *Penium margaritaceum*. *Protoplasma* 230:99–115
58. Duan Q, Kita D, Li C, Cheung AY, Wu HM. 2010. FERONIA receptor-like kinase regulates RHO GTPase signaling of root hair development. *Proc. Natl. Acad. Sci. USA* 107:17821–26
59. Durand C, Vire-Gibouin M, Follet-Gueye ML, Duponchel L, Moreau M, et al. 2009. The organization pattern of root border-like cells of *Arabidopsis* is dependent on cell wall homogalacturonan. *Plant Physiol.* 150:1411–21
60. Eder M, Lutz-Meindl U. 2008. Pectin-like carbohydrates in the green alga *Micrasterias* characterized by cytochemical analysis and energy filtering TEM. *J. Microsc.* 231:201–14
61. Ellis C, Karafyllidis I, Turner JG. 2002. Constitutive activation of jasmonate signaling in an *Arabidopsis* mutant correlates with enhanced resistance to *Erysiphe cichoracearum*, *Pseudomonas syringae*, and *Myzus persicae*. *Mol. Plant Microbe Interact.* 15:1025–30
62. Ellis C, Karafyllidis I, Wasternack C, Turner JG. 2002. The *Arabidopsis* mutant *cev1* links cell wall signaling to jasmonate and ethylene responses. *Plant Cell* 14:1557–66
63. Escobar-Restrepo JM, Huck N, Kessler S, Gagliardini V, Gheyselinck J, et al. 2007. The FERONIA receptor-like kinase mediates male-female interactions during pollen tube reception. *Science* 317:656–60
64. Evered C, Majeবাদia B, Thompson DS. 2007. Cell wall water content has a direct effect on extensibility in growing hypocotyls of sunflower (*Helianthus annuus* L.). *J. Exp. Bot.* 58:3361–71
65. Fagard M, Desnos T, Desprez T, Goubet F, Refregier G, et al. 2000. *PROCUSTE1* encodes a cellulose synthase required for normal cell elongation specifically in roots and dark-grown hypocotyls of *Arabidopsis*. *Plant Cell* 12:2409–24
66. Fayant P, Girlanda O, Chebli Y, Aubin CE, Villemure I, Geitmann A. 2010. Finite element model of polar growth in pollen tubes. *Plant Cell* 22:2579–93
67. Feraru E, Feraru MI, Kleine-Vehn J, Martiniere A, Mouille G, et al. 2011. PIN polarity maintenance by the cell wall in *Arabidopsis*. *Curr. Biol.* 21:338–43
68. Francis KE, Lam SY, Copenhaver GP. 2006. Separation of *Arabidopsis* pollen tetrads is regulated by *QUARTE1*, a pectin methylesterase gene. *Plant Physiol.* 142:1004–13
69. Freshour G, Clay RP, Fuller MS, Albersheim P, Darvill AG, Hahn MG. 1996. Developmental and tissue-specific structural alterations of the cell-wall polysaccharides of *Arabidopsis thaliana* roots. *Plant Physiol.* 110:1413–29
70. Fu Y, Gu Y, Zheng Z, Wasteneys G, Yang Z. 2005. *Arabidopsis* interdigitating cell growth requires two antagonistic pathways with opposing action on cell morphogenesis. *Cell* 120:687–700
71. Gerhardt R, Stitt M, Heldt HW. 1987. Subcellular metabolite levels in spinach leaves: Regulation of sucrose synthesis during diurnal alterations in photosynthetic partitioning. *Plant Physiol.* 83:399–407
72. Gouget A, Senchou V, Govers F, Sanson A, Barre A, et al. 2006. Lectin receptor kinases participate in protein-protein interactions to mediate plasma membrane-cell wall adhesions in *Arabidopsis*. *Plant Physiol.* 140:81–90
73. Gu Y, Li S, Lord EM, Yang Z. 2006. Members of a novel class of *Arabidopsis* Rho guanine nucleotide exchange factors control Rho GTPase-dependent polar growth. *Plant Cell* 18:366–81
74. Guan Y, Nothnagel EA. 2004. Binding of arabinogalactan proteins by Yariv phenylglycoside triggers wound-like responses in *Arabidopsis* cell cultures. *Plant Physiol.* 135:1346–66
75. Guo H, Li L, Ye H, Yu X, Algreen A, Yin Y. 2009. Three related receptor-like kinases are required for optimal cell elongation in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 106:7648–53
76. Gutierrez R, Lindeboom JJ, Paredez AR, Emons AM, Ehrhardt DW. 2009. *Arabidopsis* cortical microtubules position cellulose synthase delivery to the plasma membrane and interact with cellulose synthase trafficking compartments. *Nat. Cell Biol.* 11:797–806
77. Hahn MG, Darvill AG, Albersheim P. 1981. Host-pathogen interactions: XIX. The endogenous elicitor, a fragment of a plant cell wall polysaccharide that elicits phytoalexin accumulation in soybeans. *Plant Physiol.* 68:1161–69

78. Hamann T, Bennett M, Mansfield J, Somerville C. 2009. Identification of cell-wall stress as a hexose-dependent and osmosensitive regulator of plant responses. *Plant J.* 57:1015–26
79. Hanus J, Mazeau K. 2006. The xyloglucan-cellulose assembly at the atomic scale. *Biopolymers* 82:59–73
80. Haswell ES, Peyronnet R, Barbier-Brygoo H, Meyerowitz EM, Frachisse JM. 2008. Two MscS homologs provide mechanosensitive channel activities in the *Arabidopsis* root. *Curr. Biol.* 18:730–34
81. He ZH, Fujiki M, Kohorn BD. 1996. A cell wall-associated, receptor-like protein kinase. *J. Biol. Chem.* 271:19789–93
82. Hejnowicz Z, Borowska-Wykret D. 2005. Buckling of inner cell wall layers after manipulations to reduce tensile stress: observations and interpretations for stress transmission. *Planta* 220:465–73
83. Hématy K, Cherk C, Somerville S. 2009. Host-pathogen warfare at the plant cell wall. *Curr. Opin. Plant Biol.* 12:406–13
84. Hématy K, Höfte H. 2008. Novel receptor kinases involved in growth regulation. *Curr. Opin. Plant Biol.* 11:321–28
85. Hématy K, Sado PE, Van Tuinen A, Rochange S, Desnos T, et al. 2007. A receptor-like kinase mediates the response of *Arabidopsis* cells to the inhibition of cellulose synthesis. *Curr. Biol.* 17:922–31
86. Hernandez-Blanco C, Feng DX, Hu J, Sanchez-Vallet A, Deslandes L, et al. 2007. Impairment of cellulose synthases required for *Arabidopsis* secondary cell wall formation enhances disease resistance. *Plant Cell* 19:890–903
87. His I, Driouich A, Nicol F, Jauneau A, Höfte H. 2001. Altered pectin composition in primary cell walls of *korrigan*, a dwarf mutant of *Arabidopsis* deficient in a membrane-bound endo-1,4-beta-glucanase. *Planta* 212:348–58
88. Hou X, Tong H, Selby J, Dewitt J, Peng X, He ZH. 2005. Involvement of a cell wall-associated kinase, WAKL4, in *Arabidopsis* mineral responses. *Plant Physiol.* 139:1704–16
89. Hrmova M, Farkas V, Lahnstein J, Fincher GB. 2007. A barley xyloglucan xyloglucosyl transferase covalently links xyloglucan, cellulosic substrates, and (1,3;1,4)-beta-D-glucans. *J. Biol. Chem.* 282:12951–62
90. Jiang L, Yang SL, Xie LF, Pua CS, Zhang XQ, et al. 2005. *VANGUARD1* encodes a pectin methylesterase that enhances pollen tube growth in the *Arabidopsis* style and transmitting tract. *Plant Cell* 17:584–96
91. Jolie RP, Duvetter T, Van Loey AM, Hendrickx ME. 2010. Pectin methylesterase and its proteinaceous inhibitor: a review. *Carbohydr. Res.* 345:2583–95
92. Kaida R, Satoh Y, Bulone V, Yamada Y, Kaku T, et al. 2009. Activation of beta-glucan synthases by wall-bound purple acid phosphatase in tobacco cells. *Plant Physiol.* 150:1822–30
93. Kaida R, Serada S, Norioka N, Norioka S, Neumetzler L, et al. 2010. Potential role for purple acid phosphatase in the dephosphorylation of wall proteins in tobacco cells. *Plant Physiol.* 153:603–10
94. Kaku H, Nishizawa Y, Ishii-Minami N, Akimoto-Tomiyama C, Dohmae N, et al. 2006. Plant cells recognize chitin fragments for defense signaling through a plasma membrane receptor. *Proc. Natl. Acad. Sci. USA* 103:11086–91
95. Kessler SA, Shimosato-Asano H, Keinath NF, Wuest SE, Ingram G, et al. 2010. Conserved molecular components for pollen tube reception and fungal invasion. *Science* 330:968–71
96. Kim Y, Teng Q, Wicker L. 2005. Action pattern of Valencia orange PME de-esterification of high methoxyl pectin and characterization of modified pectins. *Carbohydr. Res.* 340:2620–29
97. Klis FM, Boorsma A, De Groot PW. 2006. Cell wall construction in *Saccharomyces cerevisiae*. *Yeast* 23:185–202
98. Knepper C, Savory EA, Day B. 2011. *Arabidopsis* NDR1 is an integrin-like protein with a role in fluid loss and plasma membrane-cell wall adhesion. *Plant Physiol.* 156:286–300
99. Kohorn BD. 2001. WAKs; cell wall associated kinases. *Curr. Opin. Cell Biol.* 13:529–33
100. Kohorn BD, Johansen S, Shishido A, Todorova T, Martinez R, et al. 2009. Pectin activation of MAP kinase and gene expression is WAK2 dependent. *Plant J.* 60:974–82
101. Kohorn BD, Kobayashi M, Johansen S, Riese J, Huang LF, et al. 2006. An *Arabidopsis* cell wall-associated kinase required for invertase activity and cell growth. *Plant J.* 46:307–16
102. Kosik O, Auburn RP, Russell S, Stratilova E, Garajova S, et al. 2009. Polysaccharide microarrays for high-throughput screening of transglycosylase activities in plant extracts. *Glycoconj. J.* 27:79–87

103. Krupkova E, Immerzeel P, Pauly M, Schmullig T. 2007. The *TUMOROUS SHOOT DEVELOPMENT2* gene of *Arabidopsis* encoding a putative methyltransferase is required for cell adhesion and co-ordinated plant development. *Plant J.* 50:735–50
104. Lally D, Ingmire P, Tong HY, He ZH. 2001. Antisense expression of a cell wall-associated protein kinase, WAK4, inhibits cell elongation and alters morphology. *Plant Cell* 13:1317–31
105. Lampart DT, Kieliszewski MJ, Chen Y, Cannon MC. 2011. Role of the extensin superfamily in primary cell wall architecture. *Plant Physiol.* 156:11–19
106. Larsen M, Artym VV, Green JA, Yamada KM. 2006. The matrix reorganized: extracellular matrix remodeling and integrin signaling. *Curr. Opin. Cell Biol.* 18:463–71
107. Laughlin ST, Baskin JM, Amacher SL, Bertozzi CR. 2008. In vivo imaging of membrane-associated glycans in developing zebrafish. *Science* 320:664–67
108. Leucci MR, Di Sansebastiano GP, Gigante M, Dalessandro G, Piro G. 2007. Secretion marker proteins and cell-wall polysaccharides move through different secretory pathways. *Planta* 225:1001–17
109. Levin DE. 2005. Cell wall integrity signaling in *Saccharomyces cerevisiae*. *Microbiol. Mol. Biol. Rev.* 69:262–91
110. Lloyd C. 2011. Dynamic microtubules and the texture of plant cell walls. *Int. Rev. Cell Mol. Biol.* 287:287–329
111. Macquet A, Ralet MC, Loudet O, Kronenberger J, Mouille G, et al. 2007. A naturally occurring mutation in an *Arabidopsis* accession affects a beta-D-galactosidase that increases the hydrophilic potential of rhamnogalacturonan I in seed mucilage. *Plant Cell* 19:3990–4006
112. Madsen EB, Madsen LH, Radutoiu S, Olbryt M, Rakwalska M, et al. 2003. A receptor kinase gene of the LysM type is involved in legume perception of rhizobial signals. *Nature* 425:637–40
113. Manfield IW, Orfila C, McCartney L, Harholt J, Bernal AJ, et al. 2004. Novel cell wall architecture of isoxaben-habituated *Arabidopsis* suspension-cultured cells: global transcript profiling and cellular analysis. *Plant J.* 40:260–75
114. Marin-Rodriguez MC, Orchard J, Seymour GB. 2002. Pectate lyases, cell wall degradation and fruit softening. *J. Exp. Bot.* 53:2115–19
115. Martiniere A, Gayral P, Hawes C, Runions J. 2011. Building bridges: formin1 of *Arabidopsis* forms a connection between the cell wall and the actin cytoskeleton. *Plant J.* 66:354–65
116. Matsunaga T, Ishii T, Matsumoto S, Higuchi M, Darvill A, et al. 2004. Occurrence of the primary cell wall polysaccharide rhamnogalacturonan II in pteridophytes, lycophytes, and bryophytes: implications for the evolution of vascular plants. *Plant Physiol.* 134:339–51
117. McKenna ST, Kunkel JG, Bosch M, Rounds CM, Vidali L, et al. 2009. Exocytosis precedes and predicts the increase in growth in oscillating pollen tubes. *Plant Cell* 21:3026–40
118. Miya A, Albert P, Shinya T, Desaki Y, Ichimura K, et al. 2007. CERK1, a LysM receptor kinase, is essential for chitin elicitor signaling in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* 104:19613–18
119. Miyazaki S, Murata T, Sakurai-Ozato N, Kubo M, Demura T, et al. 2009. *ANXUR1* and 2, sister genes to *FERONIA/SIRENE*, are male factors for coordinated fertilization. *Curr. Biol.* 19:1327–31
120. Monshausen GB, Bibikova TN, Weisenseel MH, Gilroy S. 2009. Ca²⁺ regulates reactive oxygen species production and pH during mechanosensing in *Arabidopsis* roots. *Plant Cell* 21:2341–56
121. Monshausen GB, Gilroy S. 2009. Feeling green: mechanosensing in plants. *Trends Cell Biol.* 19:228–35
122. Monshausen GB, Miller ND, Murphy AS, Gilroy S. 2011. Dynamics of auxin-dependent Ca²⁺ and pH signaling in root growth revealed by integrating high-resolution imaging with automated computer vision-based analysis. *Plant J.* 65:309–18
123. Moscaticello R, Mariani P, Sanders D, Maathuis FJ. 2006. Transcriptional analysis of calcium-dependent and calcium-independent signalling pathways induced by oligogalacturonides. *J. Exp. Bot.* 57:2847–65
124. Mouille G, Ralet MC, Cavelier C, Eland C, Effroy D, et al. 2007. Homogalacturonan synthesis in *Arabidopsis thaliana* requires a Golgi-localized protein with a putative methyltransferase domain. *Plant J.* 50:605–14
125. Nakagawa Y, Katagiri T, Shinozaki K, Qi Z, Tatsumi H, et al. 2007. *Arabidopsis* plasma membrane protein crucial for Ca²⁺ influx and touch sensing in roots. *Proc. Natl. Acad. Sci. USA* 104:3639–44
126. Nibau C, Wu HM, Cheung AY. 2006. RAC/ROP GTPases: ‘hubs’ for signal integration and diversification in plants. *Trends Plant Sci.* 11:309–15

127. Nishimura MT, Stein M, Hou BH, Vogel JP, Edwards H, Somerville SC. 2003. Loss of a callose synthase results in salicylic acid-dependent disease resistance. *Science* 301:969–72
128. Nothnagel EA, McNeil M, Albersheim P, Dell A. 1983. Host-pathogen interactions: XXII. A galacturonic acid oligosaccharide from plant cell walls elicits phytoalexins. *Plant Physiol.* 71:916–26
129. Oosterveld A, Beldman G, Voragen AG. 2000. Oxidative cross-linking of pectic polysaccharides from sugar beet pulp. *Carbohydr. Res.* 328:199–207
130. Osorio S, Castillejo C, Quesada MA, Medina-Escobar N, Brownsey GJ, et al. 2008. Partial demethylation of oligogalacturonides by pectin methyl esterase 1 is required for eliciting defence responses in wild strawberry (*Fragaria vesca*). *Plant J.* 54:43–55
131. Paredes AR, Somerville CR, Ehrhardt DW. 2006. Visualization of cellulose synthase demonstrates functional association with microtubules. *Science* 312:1491–95
132. Parre E, Geitmann A. 2005. Pectin and the role of the physical properties of the cell wall in pollen tube growth of *Solanum tuberosum*. *Planta* 220:582–92
133. Pauly M, Albersheim P, Darvill A, York WS. 1999. Molecular domains of the cellulose/xyloglucan network in the cell walls of higher plants. *Plant J.* 20:629–39
134. Peaucelle A, Braybrook SA, Le Guillou L, Bron E, Kuhlemeier C, Höfte H. 2011. Pectin-induced changes in cell wall mechanics underlie organ initiation in *Arabidopsis*. *Curr. Biol.* 21:1720–26
135. Peaucelle A, Louvet R, Johansen JN, Höfte H, Laufs P, et al. 2008. *Arabidopsis* phyllotaxis is controlled by the methyl-esterification status of cell-wall pectins. *Curr. Biol.* 18:1943–48
136. Pelletier S, Van Orden J, Wolf S, Vissenberg K, Delacourt J, et al. 2010. A role for pectin demethylesterification in a developmentally regulated growth acceleration in dark-grown *Arabidopsis* hypocotyls. *New Phytol.* 188:726–39
137. Pereira CS, Ribeiro JM, Vatulescu AD, Findlay K, Macdougall AJ, Jackson PA. 2011. Extensin network formation in *Vitis vinifera* callus cells is an essential and causal event in rapid and H₂O₂-induced reduction in primary cell wall hydration. *BMC Plant Biol.* 11:106
138. Phan TD, Bo W, West G, Lycett GW, Tucker GA. 2007. Silencing of the major salt-dependent isoform of pectinesterase in tomato alters fruit softening. *Plant Physiol.* 144:1960–67
139. Popper ZA, Fry SC. 2008. Xyloglucan-pectin linkages are formed intra-protoplasmically, contribute to wall-assembly, and remain stable in the cell wall. *Planta* 227:781–94
140. Popper ZA, Michel G, Herve C, Domozych DS, Willats WG, et al. 2011. Evolution and diversity of plant cell walls: from algae to flowering plants. *Annu. Rev. Plant Biol.* 62:567–90
141. Proseus TE, Boyer JS. 2005. Turgor pressure moves polysaccharides into growing cell walls of *Chara corallina*. *Ann. Bot.* 95:967–79
142. Proseus TE, Boyer JS. 2006. Calcium pectate chemistry controls growth rate of *Chara corallina*. *J. Exp. Bot.* 57:3989–4002
143. Proseus TE, Boyer JS. 2006. Periplasm turgor pressure controls wall deposition and assembly in growing *Chara corallina* cells. *Ann. Bot.* 98:93–105
144. Radutoiu S, Madsen LH, Madsen EB, Felle HH, Umehara Y, et al. 2003. Plant recognition of symbiotic bacteria requires two LysM receptor-like kinases. *Nature* 425:585–92
145. Ralet MC, Crepeau MJ, Lefebvre J, Mouille G, Höfte H, Thibault JF. 2008. Reduced number of homogalacturonan domains in pectins of an *Arabidopsis* mutant enhances the flexibility of the polymer. *Biomacromolecules* 9:1454–60
146. Refregier G, Pelletier S, Jaillard D, Höfte H. 2004. Interaction between wall deposition and cell elongation in dark-grown hypocotyl cells in *Arabidopsis*. *Plant Physiol.* 135:959–68
147. Ridley BL, O'Neill MA, Mohnen D. 2001. Pectins: structure, biosynthesis, and oligogalacturonide-related signaling. *Phytochemistry* 57:929–67
148. Rodicio R, Heinisch JJ. 2010. Together we are strong—cell wall integrity sensors in yeasts. *Yeast* 27:531–40
149. Roppolo D, De Rybel B, Tendon VD, Pfister A, Allassimone J, et al. 2011. A novel protein family mediates Casparian strip formation in the endodermis. *Nature* 473:380–83
150. Rose JK, Lee SJ. 2010. Straying off the highway: trafficking of secreted plant proteins and complexity in the plant cell wall proteome. *Plant Physiol.* 153:433–36

151. Roudier F, Fernandez AG, Fujita M, Himmelspach R, Borner GH, et al. 2005. COBRA, an *Arabidopsis* extracellular glycosyl-phosphatidyl inositol-anchored protein, specifically controls highly anisotropic expansion through its involvement in cellulose microfibril orientation. *Plant Cell* 17:1749–63
152. Round AN, Rigby NM, MacDougall AJ, Morris VJ. 2010. A new view of pectin structure revealed by acid hydrolysis and atomic force microscopy. *Carbohydr. Res.* 345:487–97
153. Sardar HS, Yang J, Showalter AM. 2006. Molecular interactions of arabinogalactan proteins with cortical microtubules and F-actin in Bright Yellow-2 tobacco cultured cells. *Plant Physiol.* 142:1469–79
154. Sarria R, Wagner TA, O'Neill MA, Faik A, Wilkerson CG, et al. 2001. Characterization of a family of *Arabidopsis* genes related to xyloglucan fucosyltransferase1. *Plant Physiol.* 127:1595–606
155. Schallus T, Jaeckh C, Feher K, Palma AS, Liu Y, et al. 2008. Malectin: a novel carbohydrate-binding protein of the endoplasmic reticulum and a candidate player in the early steps of protein N-glycosylation. *Mol. Biol. Cell* 19:3404–14
156. Scheller HV, Ulvskov P. 2010. Hemicelluloses. *Annu. Rev. Plant Biol.* 61:263–89
157. Seifert GJ. 2004. Nucleotide sugar interconversions and cell wall biosynthesis: how to bring the inside to the outside. *Curr. Opin. Plant Biol.* 7:277–84
158. Seifert GJ, Blaukopf C. 2010. Irritable walls: the plant extracellular matrix and signaling. *Plant Physiol.* 153:467–78
159. Shi H, Kim Y, Guo Y, Stevenson B, Zhu JK. 2003. The *Arabidopsis* *SOS5* locus encodes a putative cell surface adhesion protein and is required for normal cell expansion. *Plant Cell* 15:19–32
160. Shimizu T, Nakano T, Takamizawa D, Desaki Y, Ishii-Minami N, et al. 2010. Two LysM receptor molecules, CEBiP and OsCERK1, cooperatively regulate chitin elicitor signaling in rice. *Plant J.* 64:204–14
161. Shiu SH, Bleecker AB. 2001. Receptor-like kinases from *Arabidopsis* form a monophyletic gene family related to animal receptor kinases. *Proc. Natl. Acad. Sci. USA* 98:10763–68
162. Showalter AM. 2001. Arabinogalactan-proteins: structure, expression and function. *Cell. Mol. Life Sci.* 58:1399–417
163. Simpson SD, Ashford DA, Harvey DJ, Bowles DJ. 1998. Short chain oligogalacturonides induce ethylene production and expression of the gene encoding aminocyclopropane 1-carboxylic acid oxidase in tomato plants. *Glycobiology* 8:579–83
164. Somerville C. 2006. Cellulose synthesis in higher plants. *Annu. Rev. Cell Dev. Biol.* 22:53–78
165. Sorensen I, Domozych D, Willats WG. 2011. How have plant cell walls evolved? *Plant Physiol.* 153:366–72
166. Spadoni S, Zabolina O, Di Matteo A, Mikkelsen JD, Cervone F, et al. 2006. Polygalacturonase-inhibiting protein interacts with pectin through a binding site formed by four clustered residues of arginine and lysine. *Plant Physiol.* 141:557–64
167. Swanson S, Gilroy S. 2010. ROS in plant development. *Physiol. Plant.* 138:384–92
168. Takeda T, Furuta Y, Awano T, Mizuno K, Mitsuishi Y, Hayashi T. 2002. Suppression and acceleration of cell elongation by integration of xyloglucans in pea stem segments. *Proc. Natl. Acad. Sci. USA* 99:9055–60
169. Thompson DS. 2008. Space and time in the plant cell wall: relationships between cell type, cell wall rheology and cell function. *Ann. Bot.* 101:203–11
170. Tieman DM, Handa AK. 1994. Reduction in pectin methylesterase activity modifies tissue integrity and cation levels in ripening tomato (*Lycopersicon esculentum* Mill.) fruits. *Plant Physiol.* 106:429–36
171. Toyooka K, Goto Y, Asatsuma S, Koizumi M, Mitsui T, Matsuoka K. 2009. A mobile secretory vesicle cluster involved in mass transport from the Golgi to the plant cell exterior. *Plant Cell* 21:1212–29
172. Trudel J, Grenier J, Potvin C, Asselin A. 1998. Several thaumatin-like proteins bind to beta-1,3-glucans. *Plant Physiol.* 118:1431–38
173. Tsang DL, Edmond C, Harrington JL, Nuhse TS. 2011. Cell wall integrity controls root elongation via a general 1-aminocyclopropane-1-carboxylic acid-dependent, ethylene-independent pathway. *Plant Physiol.* 156:596–604
174. Tsuchisaka A, Yu G, Jin H, Alonso JM, Ecker JR, et al. 2009. A combinatorial interplay among the 1-aminocyclopropane-1-carboxylate isoforms regulates ethylene biosynthesis in *Arabidopsis thaliana*. *Genetics* 183:979–1003

175. Valentin R, Cerclier C, Geneix N, Aguié-Beghin V, Gaillard C, et al. 2010. Elaboration of extensin-pectin thin film model of primary plant cell wall. *Langmuir* 26:9891–98
176. Vanholme R, Demedts B, Morreel K, Ralph J, Boerjan W. 2010. Lignin biosynthesis and structure. *Plant Physiol.* 153:895–905
177. Van Sandt VS, Suslov D, Verbelen JP, Vissenberg K. 2007. Xyloglucan endotransglucosylase activity loosens a plant cell wall. *Ann. Bot.* 100:1467–73
178. Vanzin GF, Madson M, Carpita NC, Raikhel NV, Keegstra K, Reiter WD. 2002. The *mur2* mutant of *Arabidopsis thaliana* lacks fucosylated xyloglucan because of a lesion in fucosyltransferase AtFUT1. *Proc. Natl. Acad. Sci. USA* 99:3340–45
179. Velasquez SM, Ricardi MM, Dorosz JG, Fernandez PV, Nadra AD, et al. 2011. O-glycosylated cell wall proteins are essential in root hair growth. *Science* 332:1401–3
180. Vogel JP, Raab TK, Somerville CR, Somerville SC. 2004. Mutations in *PMR5* result in powdery mildew resistance and altered cell wall composition. *Plant J.* 40:968–78
181. Wagner TA, Kohorn BD. 2001. Wall-associated kinases are expressed throughout plant development and are required for cell expansion. *Plant Cell* 13:303–18
182. Wakabayashi K, Chun J-P, Huber DJ. 2000. Extensive solubilization and depolymerization of cell wall polysaccharides during avocado (*Persea americana*) ripening involves concerted action of polygalacturonase and pectinmethylesterase. *Physiol. Plant.* 108:345–52
183. Wakabayashi K, Hoson T, Huber DJ. 2003. Methyl de-esterification as a major factor regulating the extent of pectin depolymerization during fruit ripening: a comparison of the action of avocado (*Persea americana*) and tomato (*Lycopersicon esculentum*) polygalacturonases. *J. Plant Physiol.* 160:667–73
184. Walker M, Tehseen M, Doblin MS, Pettolino FA, Wilson SM, et al. 2011. The transcriptional regulator *LEUNIG_HOMOLOG* regulates mucilage release from the *Arabidopsis* testa. *Plant Physiol.* 156:46–60
185. Wang X, Zafian P, Choudhary M, Lawton M. 1996. The PR5K receptor protein kinase from *Arabidopsis thaliana* is structurally related to a family of plant defense proteins. *Proc. Natl. Acad. Sci. USA* 93:2598–602
186. Wen F, Zhu Y, Hawes MC. 1999. Effect of pectin methylesterase gene expression on pea root development. *Plant Cell* 11:1129–40
187. Western TL, Burn J, Tan WL, Skinner DJ, Martin-McCaffrey L, et al. 2001. Isolation and characterization of mutants defective in seed coat mucilage secretory cell development in *Arabidopsis*. *Plant Physiol.* 127:998–1011
188. Whitney SE, Wilson E, Webster J, Bacic A, Reid JS, Gidley MJ. 2006. Effects of structural variation in xyloglucan polymers on interactions with bacterial cellulose. *Am. J. Bot.* 93:1402–14
189. Willats WG, Orfila C, Limberg G, Buchholt HC, van Alebeek GJ, et al. 2001. Modulation of the degree and pattern of methyl-esterification of pectic homogalacturonan in plant cell walls: implications for pectin methyl esterase action, matrix properties, and cell adhesion. *J. Biol. Chem.* 276:19404–13
190. Wolf S, Mouille G, Pelloux J. 2009. Homogalacturonan methyl-esterification and plant development. *Mol. Plant* 2:851–60
191. Wolf S, Rausch T, Greiner S. 2009. The N-terminal pro region mediates retention of unprocessed type-I PME in the Golgi apparatus. *Plant J.* 58:361–75
192. Wu HM, Hazak O, Cheung AY, Yalovsky S. 2011. RAC/ROP GTPases and auxin signaling. *Plant Cell* 23:1208–18
193. Xu SL, Rahman A, Baskin TI, Kieber JJ. 2008. Two leucine-rich repeat receptor kinases mediate signaling, linking cell wall biosynthesis and ACC synthase in *Arabidopsis*. *Plant Cell* 20:3065–79
194. Yang JL, Li YY, Zhang YJ, Zhang SS, Wu YR, et al. 2008. Cell wall polysaccharides are specifically involved in the exclusion of aluminum from the rice root apex. *Plant Physiol.* 146:602–11
195. Yennawar NH, Li LC, Dudzinski DM, Tabuchi A, Cosgrove DJ. 2006. Crystal structure and activities of EXPB1 (*Zea m 1*), a beta-expansin and group-1 pollen allergen from maize. *Proc. Natl. Acad. Sci. USA* 103:14664–71
196. Yoneda A, Ito T, Higaki T, Kutsuna N, Saito T, et al. 2010. Cobtorin target analysis reveals that pectin functions in the deposition of cellulose microfibrils in parallel with cortical microtubules. *Plant J.* 64:657–67
197. York WS, Darvill AG, Albersheim P. 1984. Inhibition of 2,4-dichlorophenoxyacetic acid-stimulated elongation of pea stem segments by a xyloglucan oligosaccharide. *Plant Physiol.* 75:295–97

198. Zablackis E, Huang J, Muller B, Darvill AG, Albersheim P. 1995. Characterization of the cell-wall polysaccharides of *Arabidopsis thaliana* leaves. *Plant Physiol.* 107:1129–38
199. Zerzour R, Kroeger J, Geitmann A. 2009. Polar growth in pollen tubes is associated with spatially confined dynamic changes in cell mechanical properties. *Dev. Biol.* 334:437–46
200. Zipfel C, Kunze G, Chinchilla D, Caniard A, Jones JD, et al. 2006. Perception of the bacterial PAMP EF-Tu by the receptor EFR restricts *Agrobacterium*-mediated transformation. *Cell* 125:749–60
201. Zykwinska A, Gaillard C, Buleon A, Pontoire B, Garnier C, et al. 2007. Assessment of in vitro binding of isolated pectic domains to cellulose by adsorption isotherms, electron microscopy, and X-ray diffraction methods. *Biomacromolecules* 8:223–32
202. Zykwinska A, Thibault JF, Ralet MC. 2007. Organization of pectic arabinan and galactan side chains in association with cellulose microfibrils in primary cell walls and related models envisaged. *J. Exp. Bot.* 58:1795–802
203. Zykwinska AW, Ralet MC, Garnier CD, Thibault JF. 2005. Evidence for in vitro binding of pectin side chains to cellulose. *Plant Physiol.* 139:397–407



Contents

There Ought to Be an Equation for That <i>Joseph A. Berry</i>	1
Photorespiration and the Evolution of C ₄ Photosynthesis <i>Rowan F. Sage, Tammy L. Sage, and Ferit Kocacinar</i>	19
The Evolution of Flavin-Binding Photoreceptors: An Ancient Chromophore Serving Trendy Blue-Light Sensors <i>Aba Losi and Wolfgang Gärtner</i>	49
The Shikimate Pathway and Aromatic Amino Acid Biosynthesis in Plants <i>Hiroshi Maeda and Natalia Dudareva</i>	73
Regulation of Seed Germination and Seedling Growth by Chemical Signals from Burning Vegetation <i>David C. Nelson, Gavin R. Flematti, Emilio L. Gbisalberti, Kingsley W. Dixon, and Steven M. Smith</i>	107
Iron Uptake, Translocation, and Regulation in Higher Plants <i>Takanori Kobayashi and Naoko K. Nishizawa</i>	131
Plant Nitrogen Assimilation and Use Efficiency <i>Guobua Xu, Xiaorong Fan, and Anthony J. Miller</i>	153
Vacuolar Transporters in Their Physiological Context <i>Enrico Martinoia, Stefan Meyer, Alexis De Angeli, and Réka Nagy</i>	183
Autophagy: Pathways for Self-Eating in Plant Cells <i>Yimo Liu and Diane C. Bassham</i>	215
Plasmodesmata Paradigm Shift: Regulation from Without Versus Within <i>Tessa M. Burch-Smith and Patricia C. Zambryski</i>	239
Small Molecules Present Large Opportunities in Plant Biology <i>Glenn R. Hicks and Natasha V. Raikhel</i>	261
Genome-Enabled Insights into Legume Biology <i>Nevin D. Young and Arvind K. Bharti</i>	283

Synthetic Chromosome Platforms in Plants <i>Robert T. Gaeta, Rick E. Masonbrink, Lakshminarasimhan Krishnaswamy, Changzeng Zhao, and James A. Birchler</i>	307
Epigenetic Mechanisms Underlying Genomic Imprinting in Plants <i>Claudia Köhler, Philip Wolff, and Charles Spillane</i>	331
Cytokinin Signaling Networks <i>Ildoo Hwang, Jen Sheen, and Bruno Müller</i>	353
Growth Control and Cell Wall Signaling in Plants <i>Sebastian Wolf, Kian Hématy, and Herman Höfte</i>	381
Phosphoinositide Signaling <i>Wendy F. Boss and Yang Ju Im</i>	409
Plant Defense Against Herbivores: Chemical Aspects <i>Axel Mithöfer and Wilhelm Boland</i>	431
Plant Innate Immunity: Perception of Conserved Microbial Signatures <i>Benjamin Schwessinger and Pamela C. Ronald</i>	451
Early Embryogenesis in Flowering Plants: Setting Up the Basic Body Pattern <i>Steffen Lau, Daniel Slane, Ole Herud, Jixiang Kong, and Gerd Jürgens</i>	483
Seed Germination and Vigor <i>Loïc Rajjou, Manuel Duval, Karine Gallardo, Julie Catusse, Julia Bally, Claudette Job, and Dominique Job</i>	507
A New Development: Evolving Concepts in Leaf Ontogeny <i>Brad T. Townsley and Neelima R. Sinha</i>	535
Control of <i>Arabidopsis</i> Root Development <i>Jalean J. Petricka, Cara M. Winter, and Philip N. Benfey</i>	563
Mechanisms of Stomatal Development <i>Lynn Jo Pillitteri and Keiko U. Torii</i>	591
Plant Stem Cell Niches <i>Ernst Aichinger, Noortje Kornet, Thomas Friedrich, and Thomas Laux</i>	615
The Effects of Tropospheric Ozone on Net Primary Productivity and Implications for Climate Change <i>Elizabeth A. Ainsworth, Craig R. Yendrek, Stephen Sitch, William J. Collins, and Lisa D. Emberson</i>	637
Quantitative Imaging with Fluorescent Biosensors <i>Sakiko Okumoto, Alexander Jones, and Wolf B. Frommer</i>	663