

# Cell wall integrity

## Targeted post-synthetic modifications to reveal its role in plant growth and defense against pathogens

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The plant cell wall, a dynamic network of polysaccharides and glycoproteins of significant compositional and structural complexity, functions in plant growth, development and stress responses. In recent years, the existence of plant cell wall integrity (CWI) maintenance mechanisms has been demonstrated, but little is known about the signaling pathways involved, or their components. Examination of key mutants has shed light on the relationships between cell wall remodeling and plant cell responses, indicating a central role for the regulatory network that monitors and controls cell wall performance and integrity. In this review, we present a short overview of cell wall composition and discuss post-synthetic cell wall modification as a valuable approach for studying CWI perception and signaling pathways.

### Plant Cell Wall Composition and Functions

The plant cell wall is a unique, complex molecular network of different polysaccharides with a composition and structure that differs between plant species and can be modified during plant development and in response to stress.<sup>1</sup> The flexible and expandable primary cell wall consists of cellulosic microfibrils (1,4- $\beta$ -D-glucan), hemicellulosic polysaccharides such as  $\beta$ -(1-3,1-4)-glucan, xyloglucan (XG), xylans, mannans and pectic polysaccharides such as homogalacturonan (HG), xylogalacturonan (XGA) and rhamnogalacturonans I and II (RGI and RGI).<sup>2,3</sup> In addition, the primary wall contains different types of glycoproteins.<sup>4</sup> In contrast to the primary cell wall, the secondary cell wall contains strengthening cross-linked racemic lignin macromolecules and lower amounts of pectins and xyloglucan.<sup>5</sup> Most plant cell wall polymers, except cellulose and some glucans, can be substituted with acetyl groups<sup>6</sup> and HG and glucuronoxylan can also be methyl-esterified.<sup>7,8</sup>

The modern view of the plant cell wall is of a dynamic extracellular complex that responds to external or internal cellular signals and forms a continuum with the plasma membrane and cytoskeleton.<sup>9</sup> In addition to well established cell wall functions in maintaining and determining cell shape,<sup>10,11</sup> resisting internal turgor pressure,<sup>12</sup> controlling and directing cell and plant growth,<sup>13</sup> contributing to plant morphology<sup>14</sup> and regulating diffusion through the apoplast, the cell wall also has recently revealed signaling functions. In particular, mechanisms involved in cell to cell communication,<sup>15</sup> cell wall integrity (CWI) maintenance,<sup>16-18</sup> as well as perception and signaling in plant development and defense,<sup>19-23</sup> have attracted significant interest.

### Expression of Cell Wall Enzymes or Their Inhibitors Cause Post-Synthetic Cell Wall Modifications that Affect Plant Growth and Development

Plant cell wall polysaccharide metabolism is regulated by the balance between biosynthesis and degradation; a shift in this balance can lead to dramatic changes in cell wall structure, function and, consequently, in plant growth.<sup>16</sup> More than 2,000 genes are predicted to be involved in these processes.<sup>24,25</sup> Although significant progress has been made in functional characterization of these genes, using forward and reverse genetics,<sup>26-30</sup> these studies are far from complete.

CWI can be affected either by manipulation of biosynthetic pathways or through post-synthetic modification of cell wall structure or composition. When biosynthesis of polysaccharides is disrupted by knocking down or mutating synthetic enzymes, plants react to this modification by activating compensatory mechanisms leading to the alteration of other cell wall components. For example, some mutations in cellulose synthase encoding genes (CesA) lead to increased lignin production<sup>31,32</sup> or cause embryo-lethality.<sup>33-35</sup> Mutations in pectin-related genes cause a

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dwarf phenotype and affect cellular adhesion.<sup>36</sup> Reduction of fucose in arabinogalactan, a cell wall specific protein, affected root cell elongation.<sup>37</sup>

Plants themselves express a broad variety of cell wall modifying enzymes (CWMEs) in the apoplast to maintain proper wall plasticity and flexibility and to assist in wall remodeling and degradation during growth and development.<sup>38</sup> In addition, CWMEs produced by microorganisms during host invasion cause cell wall modifications that can initiate plant defense responses. Thus, heterologous expression of CWMEs can lead to a broad range of effects that could potentially mimic the plant's responses to endogenous or external CWMEs.

For example, targeted modification of pectin structure and integrity by the overexpression of pectin modifying enzymes can provide insight into the role of different pectic polysaccharides in plant growth. Expression of rhamnogalacturonan lyase (eRGL) from *Aspergillus aculeatus* in potato plants resulted in a degradation of the RGI backbone and a decrease in both galactan and arabinan side-chains, leading to wrinkled tubers.<sup>39</sup> The specific removal of RGI arabinan side-chains resulting from the expression in the apoplast of an endo-1,5- $\alpha$ -arabinanase of *A. aculeatus* caused the transgenic potato plants to have severe phenotypic changes including the inability to produce tubers.<sup>40</sup> By contrast, the overexpression of a fungal (*A. aculeatus*) endo-galactanase in potato caused a reduction of galactan side-chains of RGI with a compensatory increase of uronic acids, without affecting tuber development.<sup>41</sup> Although RGI galactan and arabinan side-chains have an, as yet unclear, role in planta, the RGI backbone must play an important role in the integrity and function of the cell wall, since its degradation by expression of hydrolases results in morphological changes.

Overexpression of an endopolygalacturonase (AnPGII) of *Aspergillus niger* in tobacco caused a significant reduction of pectic homogalacturonan partially compensated by an increase in rhamnose, galactose and arabinose in RGI; the resulting transgenic tobacco lines are drastically dwarfed,<sup>42</sup> indicating homogalacturonan integrity is a critical factor in plant growth.

Acetyl content depends on the plant organ and developmental stage<sup>43</sup> and acetylation of plant cell wall polysaccharides also plays an important role in plant development. De-acetylation of pectins resulting from overexpression of mung bean acetyltransferase in potato increased the mechanical strength of tubers due to a decrease in pectin-related cell wall elasticity. In these plants, the altered pectin acetylation can affect the interaction between pectin and cellulose chains.<sup>44</sup> The overexpression of pectin acetyltransferase 1 (PtPAE1) from cottonwood (*Populus trichocarpa*) in tobacco resulted in a reduction in acetyl groups of pectin but not in xylan and affected cell expansion and plant organ growth direction as well as altered pollen germination and, consequently, plant reproduction.<sup>45</sup> In addition, expression of genes involved in cell wall acetylation was shown to be associated with secondary wall thickening.<sup>46</sup>

Methylation of cell wall polysaccharides can be involved in growth regulation and polysaccharide accessibility to glycosyl hydrolases.<sup>47,48</sup> *Arabidopsis* plants overexpressing the endogenous PME3 exhibited a dramatic increase in PME activity and

significantly longer roots; a *pme3* knockout mutant showed the opposite phenotype.<sup>49</sup> In aspen trees (*Populus tremula* ssp. *tremuloides*), upregulation of PtPME1 inhibited the apical elongation of wood fibers.<sup>50</sup> In addition to transcriptional control, PME activity is regulated by endogenous inhibitor proteins (PMEIs), which were discovered in kiwi fruit and subsequently identified in *Arabidopsis*, pepper, broccoli, wheat and tomato.<sup>51-59</sup> PMEIs function in apical meristem development, cell growth acceleration and pollen tube growth.<sup>47,53,54,60</sup> Also, overexpression of AtPMEI in *Arabidopsis* significantly increases the level of pectin methylation and improves leaf and root growth.<sup>61,62</sup> Methylation of xylan alters lignin structure but does not affect plant growth and wall degradability by cellulases.<sup>8,63</sup>

### Post-Synthetic Modifications of the Cell Wall Affect CWI and Plant Disease Resistance

Pathogenesis involves plant cell wall alteration by CWMEs targeted to wall polysaccharides. It is well established that the plant susceptibility to pathogens depends on the cell wall composition and structure, which determine its recalcitrance to degradation by CWMEs produced by pathogens.<sup>64</sup> Moreover, plants have evolved the ability to sense cell wall damage and trigger defense responses leading to an improved resistance to disease.<sup>65</sup> Therefore, expression of endogenous or microbial CWMEs and their inhibitors in planta represents a useful tool to improve plant resistance to pathogens and to investigate the role of altered structure and integrity of wall polysaccharides in plant-pathogen interactions.

The manipulation of cell wall biosynthetic pathways or post-synthetic modifications of cell wall structure or composition can affect the defense against pathogens. For example, some *Arabidopsis* mutants in cell wall biosynthetic genes are affected in resistance to pathogens.<sup>66-68</sup> Cel2, a putative  $\beta$ -1,4-endoglucanase, which targets cellulose or hemicellulose, makes plants more susceptible to *Botrytis cinerea* when overexpressed in ripening inhibited tomato mutants, suggesting a role for cellulose/hemicellulose integrity in plant susceptibility to pathogens.<sup>69</sup> Cellodextrins are the end products of cellulose degradation in plant cell walls. Cellodextrins with a degree of polymerization <sup>3</sup> 7 induce defense responses in grapevine,<sup>70</sup> consistent with results indicating that cellulase from *Trichoderma viride* can release cellodextrins which induce plant defenses.<sup>71</sup>

Depolymerization of xyloglucan has been proposed to play an important role during both cell wall expansion and pathogen invasion.<sup>72-74</sup> Endo- $\beta$ -1,4-xylanases are key enzymes in the degradation of xylans and a number of endoxylanases are produced by microbial pathogens to break through the plant cell wall. Rice plants expressing a thermostable exogenous xylanase gene (ATX) exhibited upregulation of SOD, CAT and xylanase inhibitor (RIXI) genes, which could play roles in plant defense.<sup>75</sup>

Acetylation of cell wall polysaccharides affects plant responses to invading pathogens. For example, the *Arabidopsis* reduced wall acetylation 2 (*rwa2*) mutant exhibited reduced cell wall acetylation and increased resistance to the necrotrophic fungal pathogen *Botrytis cinerea*.<sup>76</sup> *Arabidopsis* and *Brachypodium* plants

expressing a fungal *Aspergillus nidulans* acetyltransferase (AnAXE) had reduced cell wall acetylation and increased resistance to *B. cinerea* and *Bipolaris sorokiniana*.<sup>19</sup> Interestingly, the resistance to pathogens in dicot and monocot AnAXE-expressing plants was mediated by the activation of different defense responses indicating that different plants perceive reduced cell wall acetylation, but exploit different defense signaling pathways to respond to necrotrophic pathogens.<sup>19</sup> For protection against invading pathogens, plants have evolved a surveillance system based on proteinaceous inhibitors of microbial CWMEs. To counteract hemicellulose degradation by microbial hemicellulases, plants produce inhibitors such as the *Triticum aestivum* xylanase inhibitor (TAXI), xylanase inhibitor protein (XIP) and xyloglucan endoglucanase inhibiting protein (XEGIP).<sup>58,77</sup> These endogenous inhibitors, induced during pathogen infection, play a role in the inhibition of enzymes produced by microorganisms.<sup>58</sup> The transient overexpression of pepper CaXEGIP1 in *Arabidopsis*, pepper and *Nicotiana benthamiana* leaves, triggered pathogen-independent, spontaneous cell death.<sup>78</sup> Transient overexpression of CaXEGIP1 in *Arabidopsis* enhanced resistance to biotrophic downy mildew pathogen *Hyaloperonospora arabidopsidis*. Comparative histochemical and proteomic analyses revealed that CaXEGIP1 overexpression induced defense-related genes as well as cell wall thickening and darkening. Together, these results suggest that pathogen-inducible CaXEGIP1 positively regulates cell death-mediated defense responses and resistance to pathogens in plants.<sup>78</sup>

Pectin is one of the first structures to be altered during pathogen invasion and accumulating evidence indicates that post-synthetic alteration of pectic polysaccharides affects plant resistance to pathogen.<sup>79,80</sup> Pectin degradation requires the combined action of several pectinases, of which the most extensively studied in terms of pathogen attack are the polygalacturonases (PGs). The expression of an attenuated version of endopolygalacturonase II from *Aspergillus niger* (AnPGII) in tobacco and *Arabidopsis* caused a reduction of galacturonic acid content due to HG breakdown and improved plant resistance to the fungal and bacterial pathogens *B. cinerea* and *Pseudomonas syringae*.<sup>42,81</sup> It was proposed that *Arabidopsis* and tobacco overexpressing microbial AnPGII accumulate oligogalacturonide (OG) fragments, which serve as damage associated molecular patterns (DAMPs) acting in signaling the presence of cell wall damage caused by pathogens and leading to induction of disease resistance. Consistent with this hypothesis, PG-expressing plants showed enhanced constitutive defense responses, such as accumulation of UV-fluorescent metabolites, H<sub>2</sub>O<sub>2</sub>,  $\beta$ -1,3-glucanase and peroxidase and expression of defense-related genes.<sup>81</sup> The size of OGs (usually between 10 and 15 glycosides) and their degree of methyl- and acetylation are important features for their ability to trigger plant defense responses. The de-esterification of OGs by overexpression of a fruit-specific PME in wild strawberry was required to elicit defense-related gene expression and induce plant resistance to *B. cinerea*.<sup>82,83</sup> Consistent with this, chemical methylesterification of OGs greatly diminishes their ability to elicit biological responses.<sup>84,85</sup> Treatment with OGs can protect wheat, a grass species with low pectin contents, against *Blumeria graminis*.

Also, partially acetylated OGs are more efficient elicitors and induce additional defense responses, such as the increased accumulation of phenolic autofluorescent compounds in papillae and a decreased formation of haustoria during fungal pathogen infection.<sup>86</sup> The degree of methylesterification and acetylation of homogalacturonan is different in various plant species and is regulated during development. Therefore, PGs expressed in different plants could release OGs with different degrees of methylesterification and acetylation capable of eliciting diverse specific biological responses.<sup>87</sup>

The loss of cell wall integrity can result in the activation of cell wall strengthening mechanisms. Potato tubers expressing an *Erwinia carotovora* pectate lyase exhibited increased resistance to *Erwinia* soft rot and increased phenol oxidase activity. These responses may be induced by active unesterified OG fragments, indicating a possible role of phenol biosynthesis and oxidation in the reinforcement of the cell wall against pathogens.<sup>88</sup>

Esterification of pectic polysaccharides can also affect their susceptibility to degradation by microbial enzymes.<sup>89,90</sup> Genetic and molecular evidence indicates a critical role of pectin methylesterification and acetylation in plant defense against pathogens. The expression of inhibitors of pectin methylesterase (PMEI), affecting the activity of endogenous PMEs, has been used as a strategy to increase the level of methylesterification in the cell wall. In *Arabidopsis*, the overexpression of AtPMEI-1 or AtPMEI-2 causes increased post-synthetic pectin methylesterification and decreased susceptibility to *B. cinerea* and *Pectobacterium carotovorum*.<sup>62,91</sup> This strategy was effective also in wheat, where the ectopic expression of AcPMEI from kiwi reduced the susceptibility to *Fusarium graminearum* and *B. sorokiniana*. Interestingly, PMEI-expressing plants did not show induction of defense responses and their reduced susceptibility was due to the higher resistance of pectins to microbial hydrolases and to the reduced pathogen growth on highly methylated cell walls. The overexpression of different PMEI isoforms can affect plant-pathogen interactions through different mechanisms. For example, overexpression of pepper CaPMEI1 in *Arabidopsis* improved plant resistance to *P. syringae* possibly due to the antimicrobial activity of the protein and the activation of the salicylic acid pathway, although a possible contribution of altered cell wall esterification has not been excluded.<sup>52</sup> Moreover, CaPMEI overexpressing plants exhibited higher drought and oxidative stress tolerance.<sup>52</sup>

Pectin acetylation can also affect plant resistance against invading pathogens. Recent work demonstrated that reduction of pectin acetylation increased *Arabidopsis* resistance to microbial pathogens.<sup>19,76</sup> *Arabidopsis* plants expressing rhamnogalacturonan acetyltransferase from *A. nidulans* (AnRAE) exhibited reduced pectin and xyloglucan acetylation and increased resistance to *B. cinerea* mediated by the activation of defense responses such as callose deposition, H<sub>2</sub>O<sub>2</sub> accumulation and activation of defense-related genes.<sup>19</sup> Since responses observed in these transgenic plants were similar to those usually induced by OGs, it was proposed that increased susceptibility of deacetylated pectin to endogenous and microbial PGs favors the accumulation of active OGs eliciting constitutive and pathogen-induced defense responses. Polygalacturonase-inhibiting proteins (PGIPs)

produced by plants are the best-characterized inhibitors of microbial pectic enzymes. PGIPs play a double function in protecting plants: they hinder pectin degradation by inhibiting microbial PG activities and also favor the accumulation of elicitor-active OGs.<sup>80,92,93</sup> The expression of PGIPs reduced susceptibility to *B. cinerea* in *Arabidopsis*, tomato and grape and induced higher resistance to *F. graminearum* in wheat.<sup>21,94-96</sup>

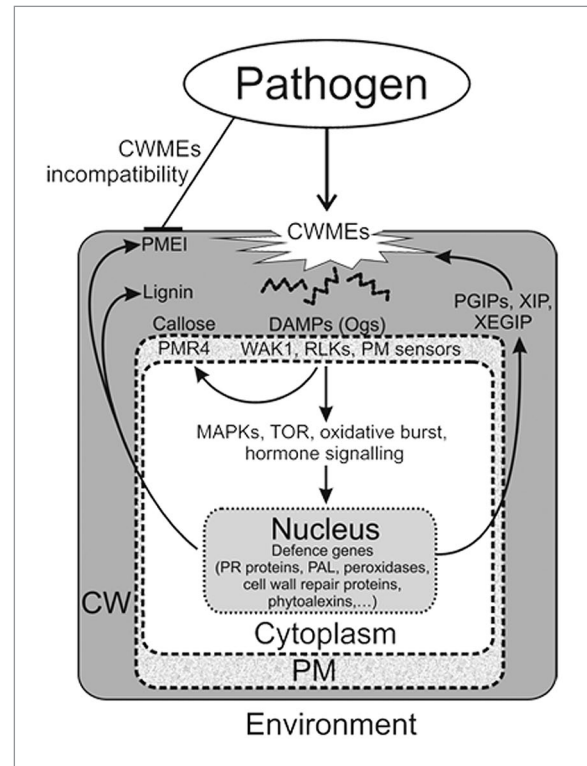
Pectin lyases are produced by pathogenic bacteria and fungi to catalyze the  $\beta$ -elimination cleavage of methylesterified homogalacturonan. A cell wall pectin lyase inhibitor protein that inhibits a pectin lyase from *Rhizoctonia solani* was identified in sugar beet.<sup>97</sup> The level of expression of the inhibitor protein correlated with plant resistance to the pathogen but details on the inhibitor structure, its spatial and temporal regulation and its occurrence in plants were not reported.

A common plant response to fungal attack is the deposition of callose, a (1,3)- $\beta$ -glucan polymer, at the sites of pathogen penetration. The role of callose in plant defense is believed to be dependent on the infection strategy of the pathogen. Higher callose accumulation contributes to resistance against necrotrophic fungal pathogens, but the role of callose in resistance against biotrophic pathogens is controversial.<sup>98,99</sup> However, emerging evidence support the active role of callose synthesis in plant resistance to biotrophic microbial pathogens. Transgenic *Arabidopsis* plants expressing POWDERY MILDEW RESISTANT 4 (PMR4), which encodes a stress-induced callose synthase, showed a higher callose synthase activity and increased early callose deposition at the sites of pathogen penetration, resulting in prevention of haustoria formation and complete resistance to penetration by powdery mildews *Golovinomyces cichoracearum* and *Blumeria graminis*.<sup>100</sup> PMR4 overexpressing leaf tissues showed alterations in cell wall composition, decreases in fucose and galactose and an increase in glucose, which are not directly related to callose synthesis. The localized callose synthesis was proposed to be required for an early structural reinforcement of the cell wall supporting complete resistance to the fungal pathogens.

### Cell Wall Integrity Perception

The modification of cell wall composition affects plant development and responses to pathogens by two possible mechanisms: through changes in mechanistic properties and through initiation of signaling pathways. For example, changes of sugar content in the plant cell can be sensed through hexokinases, which modify responsive gene expression.<sup>101-103</sup> The concept of biologically active oligosaccharides, termed oligosaccharins, was first proposed by Albersheim and colleagues,<sup>3</sup> and numerous studies have demonstrated a diversity of oligosaccharin structure and functionality, as described in several comprehensive reviews.<sup>104-106</sup>

The plant cell wall contains a large number of constituents that could potentially release signals recognizable by specific receptors. During pathogen attack, Damage Associated Molecular Patterns (DAMPs) have been described to function as such signals and the number of possible DAMPs is most likely large.<sup>107</sup> Similarly, remodeling of cell walls in response to abiotic stresses such as drought and cold is also believed to



**Figure 1.** Cell wall integrity signaling components mediate cellular responses upon pathogen attack. Pathogen invasion can be limited by a cell wall structure and composition that is resistant to degradation by cell wall modifying enzymes (CWMEs). Pathogens can be detected by the presence of cell wall fragments (i.e., oligogalacturonides; OGs) generated by CWMEs; these fragments can function as DAMPs and are perceived by Wall Associated Kinases (WAKs), Receptor Like Kinases (RLKs) and Plasma Membrane (PM) sensors. Downstream signaling pathways mediate defense responses (MAPKs, TOR, oxidative burst, hormone mediated pathways of Pathogen Related (PR), CW repair and peroxidases genes, PAL, phytoalexins activation and/or cell wall structural rearrangements such as cell wall repair (PMR4-mediated callose deposition) or cell wall reinforcement (lignin) and de novo synthesis of defensive compounds such as defense proteins including CWME inhibitors (PGIPs, XIP, XEGIP, PMEI).

be a source of oligosaccharins released during these processes, thus mediating transduction of signals between cell wall and cytoplasm.<sup>108,109</sup>

Changes in cell wall mechanical properties can also be involved in stress responses; this was strongly evidenced by demonstration of the relationship between cytoskeleton orientation and cellulose deposition.<sup>110</sup> During water stress, microtubules are eliminated by proteolytic degradation (Wang et al., 2011) followed by cellulose microfibril re-organization and activation of phospholipase-D dependent signaling during osmotic adaptation.<sup>111-113</sup>

Release of DAMPs and CWI modifications can be perceived by membrane receptors, communicated by trans-membrane channels and can trigger internal cell signaling cascades to activate appropriate response genes. Thus, the large family of receptor like kinases (RLKs), arabinogalactan proteins, the Mitogen-Activated Protein Kinase (MAPK) pathway and the Target of Rapamycin (TOR) pathway have been suggested



to be potential components of CWI perception mechanisms (Fig. 1).<sup>9,17,24</sup> The MAPK pathway activates or suppresses gene expression via phosphorylation of appropriate transcription factors or receptors.<sup>24,114</sup> Oligogalacturonides (OGs) released from pectins by microbial polygalacturonase elicit signals involved in plant innate immunity.<sup>115</sup> OGs serve as DAMPs and are perceived by Wall Associated Kinase-like proteins, which have high affinity to pectin and may act as pectin integrity sensors.<sup>18,116-118</sup> RLKs belonging to the *Catharanthus roseus* (CrRLK)-like protein family were implicated in CWI signaling mechanisms. Among these are THESEUS1 (THE1), HERCULES1 and FERONIA, which have been implicated in brassinosteroid-induced cell elongation.<sup>119,120</sup> The THE1 plasma-membrane receptor-like kinase has been proposed to mediate the response of plant cells to perturbations in cellulose content and may act as a cell-wall-integrity sensor.<sup>32</sup> THE1 is also required for induction of ROS production and ectopic lignification in *Arabidopsis* roots in response to inhibition of cellulose biosynthesis.<sup>28</sup> Some RLKs have a lectin-like extracellular domain. Lectin receptor kinases may bind cell wall polysaccharides by mediating structural continuity between cell wall and membrane and sensing changes in integrity of the cell wall after pathogen attack.<sup>32,121</sup>

Recently, Wolf et al. proposed that brassinosteroid feedback signaling is part of a compensatory response that is activated to protect the plant against the loss of CWI caused by imbalanced pectin modifications.<sup>24</sup> Another example is G-proteins that form highly conserved signaling complexes and participate in signal transduction during development and stress responses.<sup>122,123</sup> TOR kinase is essential for *Arabidopsis* development and TOR pathways are involved in regulating cell wall development.<sup>124-127</sup> In addition, arabinogalactan proteins, COBRA and extensin-like receptor kinases are examples of GPI-containing proteins that are attached to the plasma membrane and implicated in connecting intracellular and extracellular space, thus affecting cell wall development.<sup>17</sup> These transmembrane receptor proteins are good candidates for transducing signals from the extracellular space to the cytoplasm. The high diversity of cell wall structural components and large numbers of potential receptors and transducers suggest the significant complexity of plant CWI maintenance and signaling mechanisms, many of which have yet to be uncovered.

## Outlook

The plant cell wall is a dynamic and highly complex structure, consisting of interdependent networks that constantly change during development and in response to environmental cues. Plant cells monitor the status of their cell walls with various types of sensors and receptors at the plasma membrane, some of which may interact with cell wall components, to coordinate mechanical deformations or changes in cell wall structure and cellular

responses. Although a number of plant responses to cell wall damage or modification have been described and the existence of CWI maintenance mechanisms in plants is indubitable, detailed transduction pathways involved in CWI signaling via a signal and receptor, leading to a specific output, are yet to be revealed. Most evidence about CWI signaling in plants comes from mutant studies where biosynthesis of various cell wall components was compromised, causing wall structural damage or rearrangement. Post-synthetic modification of cell wall components via their partial remodeling by overexpressed cell wall modifying enzymes/inhibitors is another powerful approach to investigate CWI signaling mechanisms. Fine structural remodeling of particular polysaccharides via side chain cleavage, de-esterification or partial depolymerization leads to the initiation of specific signaling pathways related to these particular modifications. This approach can assist in revealing putative components of signaling pathways initiated in response to such highly specific cell wall modifications and thus allow us to dissect otherwise highly complex cell responses that occur during plant development or defense responses. Because of plant broad adaptability and cell wall complexity, we expect that the near future holds significant discoveries of many new receptors, signal transduction components and transcriptional regulators involved in cell wall-sensing mechanisms and post-synthetic wall modifications.

Post-synthetic cell wall modifications will also find wide application in crop biotechnology and in bioenergy biomass feedstock improvement. Remodeling of particular cell wall components directed toward increasing digestibility can improve biomass saccharification during biofuel production, reducing costs. However, such modifications can also have different effects on plant growth and stress tolerance. Modifications that compromise cell wall mechanical properties can reduce plant resistance to biotic stresses, but other modifications can have the opposite effect by initiating defense mechanisms. Therefore, such modifications require an informed approach to be effective for feedstock improvement. Further detailed studies of post-synthetic modification by overexpression of cell wall modifying enzymes and their effects on plant responses will significantly contribute to improvement of crop productivity and sustainability.

## Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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