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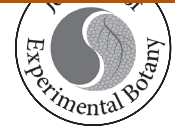

REVIEW PAPER

At the border: the plasma membrane–cell wall continuum

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Abstract

Plant cells rely on their cell walls for directed growth and environmental adaptation. Synthesis and remodelling of the cell walls are membrane-related processes. During cell growth and exposure to external stimuli, there is a constant exchange of lipids, proteins, and other cell wall components between the cytosol and the plasma membrane/apoplast. This exchange of material and the localization of cell wall proteins at certain spots in the plasma membrane seem to rely on a particular membrane composition. In addition, sensors at the plasma membrane detect changes in the cell wall architecture, and activate cytoplasmic signalling schemes and ultimately cell wall remodelling. The apoplastic polysaccharide matrix is, on the other hand, crucial for preventing proteins diffusing uncontrollably in the membrane. Therefore, the cell wall–plasma membrane link is essential for plant development and responses to external stimuli. This review focuses on the relationship between the cell wall and plasma membrane, and its importance for plant tissue organization.

Key words: Anchor proteins, cell wall, plasma membrane, signalling, vesicle trafficking.

Introduction

Cell walls are mechanistically strong and dynamic structures that encase plant cells. The cell walls protect the cells against environmental stresses and play key roles in directed plant growth and cell differentiation (Somerville *et al.*, 2004; Tsukaya, 2006; Ivakov and Persson, 2013), and are mainly composed of polysaccharides, proteins, and polyphenols (Boerjan *et al.*, 2003; Rose, 2003; Jamet *et al.*, 2006). All growing plant cells are surrounded by primary cell walls, which in dicot species comprise cellulose, hemicelluloses, pectins, and proteins, such as RGD (Arg-Gly-Asp tripeptide) recognizing proteins and glycosylphosphatidylinositol (GPI) anchored proteins (Canut *et al.*, 1998; Cosgrove, 2005; Gillmor *et al.*, 2005). Additional secondary cell wall layers are produced after cessation of cell expansion in some specialized cells. These walls typically provide mechanical support to the plant body through their ordered cellulose fibrils and lignin content (Boerjan *et al.*, 2003).

With the exception of cellulose, cell wall polysaccharides and proteins are synthesized in the endoplasmic reticulum and Golgi apparatus, and secreted to the apoplast via vesicle trafficking. Cellulose is synthesized at the plasma membrane by transmembrane cellulose synthase (CesA) complexes (CSCs), which are assembled in either the endoplasmic reticulum or Golgi, and subsequently delivered to the plasma membrane (Lerouxel *et al.*, 2006; Geisler *et al.*, 2008). Once at the plasma membrane, the CSCs synthesize unbranched glucan chains that assemble into para-crystalline cellulose microfibrils (Morgan *et al.*, 2013; Fig. 1). The microfibrils become fixed in the cell wall and the catalytic capacity of the CSCs has therefore been proposed to exert a force that moves the complex forward inside the plasma membrane during cellulose synthesis. This hypothesis is supported by recent structural estimations of cellulose synthase from *Rhodobacter* (Morgan *et al.*, 2013; Sethaphong *et al.*, 2013). Thus, the motility of the CSCs should reflect the rate of cellulose production and might be dependent on the composition of the plasma membrane. The CSCs have been shown to have an average lifetime

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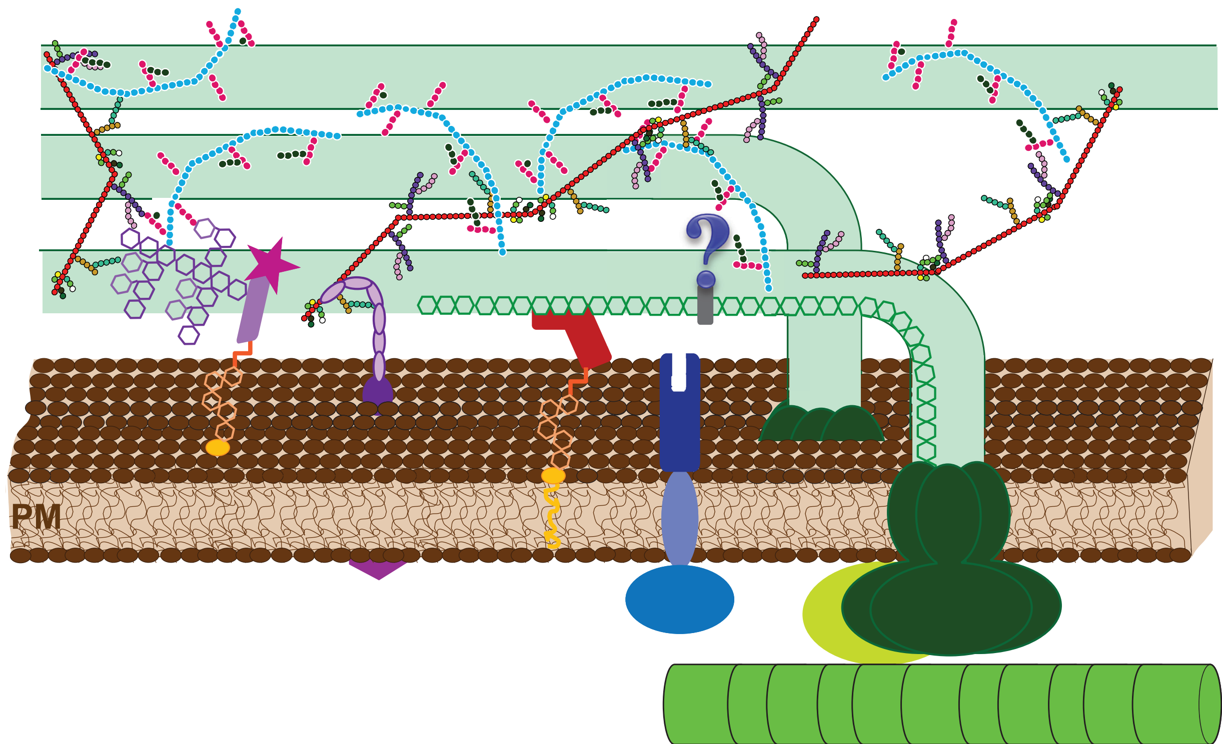

Fig. 1. The plasma membrane–cell wall anchors. Cell walls are mainly composed of cellulose microfibrils and matrix polysaccharides. Each cellulose microfibril is formed by glucan chains assembled and translocated by a CSC, which moves in the plasma membrane guided by cortical microtubules through CSI1. The cell wall and plasma membrane are physically linked by plasma membrane proteins, characterized by particular domains, which anchor apoplastic polysaccharides. Thus GPI is necessary for anchoring certain proteins to the plasma membrane, such as COBRA and fasciclins. Other proteins, including integrin-like and receptor like kinases, such as WAKs, are attached to the plasma membrane through transmembrane domains. The ability of these proteins to bind to the cell wall relies on the presence of cellulose-binding domains (in COBRA) or arabinogalactan moieties which covalently attach to matrix polysaccharides (as in the case of arabinogalactan proteins and FLAs). Integrin-like proteins can bind to the RGD domain of apoplastic proteins, which may bind cell wall polysaccharides. However, many questions are still open regarding these interactions, since no RGD-containing protein has been identified in plants.

at the plasma membrane of 10–20 min (Paredez, *et al.*, 2006; Gutierrez *et al.*, 2009; Sampathkumar *et al.*, 2013), suggesting that, after they have fulfilled their function, they are internalized via endocytosis. This process also removes lipids, other proteins, and probably cell wall constituents from the plasma membrane and apoplast. Hence, cell wall architecture and remodelling depends on efficient exchange of material between the cell's interior and the plasma membrane/apoplastic space (Drakakaki and Dandekar, 2013; Sánchez-Rodríguez and Persson, 2014).

While being synthesized and remodelled, the cell wall is physically anchored to the plasma membrane via the cellulose fibrils and plasma membrane proteins that interact with cell wall components (Baluška *et al.*, 2003). This anchoring is essential for proteins to retain their localization at the plasma membrane (e.g. PIN2 and other proteins: Feraru *et al*, 2011; Martinière and Runions, 2013), for cell wall synthesis and remodelling (e.g. during Casparian strip formation: Alassimone *et al.*, 2010; Roppolo and Geldner, 2012), and for signal transduction (Wolf *et al.*, 2012*a*).

Taken together, the cell wall–plasma membrane interactions comprise four main areas: the plasma membrane as a gating point for cell wall components; the plasma membrane as a plane for CSC movement; the plasma membrane–cell wall cross-links; and cell wall signalling via plasma membrane-located signalling transmitters. Considering the vast number of reviews dealing with the first, second and fourth of these areas (Wolf *et al.*, 2012*a*; Bashline *et al.*, 2014; McFarlane *et al.*, 2014; Sánchez-Rodríguez and Persson, 2014), we only briefly review and provide recent updates of these, and instead focus on cross-linking between the plasma membrane and cell wall in plant cells.

The plasma membrane: the 'customs' of the plant cell

Many biological components are made in one location but needed in another. This certainly holds true for the majority of cell wall components, which are synthesized inside the cell, but assembled at the apoplast. Therefore, efficient membrane trafficking is important for cell wall formation and remodelling (for recent reviews see Bashline *et al.*, 2014; McFarlane *et al.*, 2014; Sánchez-Rodríguez and Persson, 2014).

Soluble cell wall polysaccharides; i.e. pectins and hemicelluloses, have been found in post-Golgi vesicles together with enzymes involved in their synthesis (Young *et al.*, 2008; Driouich *et al.*, 2012; Atmodjo *et al.*, 2013). These observations support the hypothesis that soluble cell wall polysaccharides are synthesized inside the cells, and that they are translocated to the apoplast by secretion. During exocytosis, vesicles derived from the trans-Golgi network (TGN) traffic through the cytosol via actin filaments and bundles, and fuse to the plasma membrane, which leads to the release of their content into the existing cell wall (Drakakaki and Dandekar, 2013; Sánchez-Rodríguez and Persson, 2014). Recently, an ECH/YIP complex-dependent pathway was found to promote the formation of soluble polysaccharide-containing vesicles at the TGN (Gendre *et al.*, 2013). Accordingly, lesions in members of the complex led to alterations in cell elongation, seed coat mucilage deposition, and anther and pollen development (Gendre *et al.*, 2011; Fan *et al.*, 2014). After vesicle shuttling through the cytosol, the vesicles need to be targeted and tethered to the plasma membrane before membrane fusion. The SEC8 and EXO70A1 subunits of the tethering exocyst complex, and their interactor ROH1, have been reported to influence pectin deposition in the seed coat (Kulich *et al.*, 2010), indicating that the exocyst secretes soluble polysaccharides. Other cell wall components, e.g. lignin precursors, can be translocated from the cytosol to the apoplast by plasma membrane-localized ABC transporters (McFarlane *et al.*, 2010; Kang *et al.*, 2011; Alejandro *et al.*, 2012). In contrast, cellulose is synthesized at the plasma membrane. Therefore, efficient transport of the CSCs to and from the plasma membrane is important for cellulose synthesis (Sampathkumar *et al.*, 2013; McFarlane *et al.*, 2014).

Morphometric studies have estimated that around 75% of the total membrane incorporated into the plasma membrane of an expanding cell, or cell plate during cytokinesis, is recycled by endocytosis (Samuels and Bisalputra, 1990; Ketelaar *et al.*, 2008). Hence, a finely tuned balance of exo- and endocytosis appears to be important for proper cell wall production. This hypothesis is supported by recent data, which indicate that clathrin-mediated endocytosis (CME) is required for cell wall biogenesis, in particular for cellulose synthesis. Various CME proteins have been reported to be essential for plant cell division and cell elongation based on their localization and on cellulose deficiencies in corresponding mutants (Kang *et al.*, 2003; Van Damme *et al.*, 2006; Collings *et al.*, 2008; Frigerio, 2010; Xiong *et al.*, 2010; Bashline *et al.*, 2013; Kim *et al.*, 2013; Miart *et al.*, 2014). However, the mechanism by which the CME subunits internalize CSCs is still being elucidated. Interestingly, endocytosis of the GPI-anchored proteins, involved in cell adhesion, has been reported in animals (Lakhan *et al.*, 2009). Related plant cell wall protein might, therefore, also be internalized from the plasma membrane; however, this remains to be proven. In addition, although the data are inconclusive, internalization of pectins and hemicelluloses by CME is possible (Baluška *et al.*, 2002; Baluška *et al.*, 2005).

Movement of CSCs in the plasma membrane

Recent observations indicate that the CSCs are preferentially inserted into the plasma membrane adjacent to cortical microtubules, but how this is achieved is not yet known (Gutierrez *et al.*, 2009). It is plausible that the membrane composition is important for this, as it seems to play a key role in exo- and endocytosis (Bessueille *et al.*, 2009; Žárský *et al.*, 2009; Zhang *et al.*, 2014). Proteomic analysis of detergent-resistant membrane (DRM) fractions extracted from various plant species revealed that the plasma membrane domains contain many different plasma membrane proteins (Morel *et al.*, 2006; Lefebvre *et al.*, 2007; Bessueille *et al.*, 2009), including CesAs (Bessueille

et al., 2009). However, the CesA proteins were not identified in other proteomic studies of DRMs (Borner et al., 2005; Morel et al., 2006; Lefebvre et al., 2007), probably due to the different plant species and different extraction methods used. Indeed, the large size of the CSCs makes them difficult to isolate and identify by mass spectroscopy, even in enriched total membrane fractions, where they are abundant (Alexandersson et al., 2004). Therefore, although still controversial, it is hypothesized that the CSCs are delivered to DRM microdomains.

Lipid analysis revealed that DRMs are enriched in sterols and sphingolipids (Borner et al., 2005; Lefebvre et al., 2007). Sterols are one of the most important regulators of plasma membrane microdomain maintenance (Zauber et al., 2014). Perturbations in sterol biosynthesis, either via inhibitors or mutations, lead to reduced cellulose content (Schrick et al., 2004), indicating that sterols promote cellulose synthesis (reviewed in Schrick et al., 2012). However, sterols are also implicated in various metabolic pathways, such as the biosynthesis of sitosterol-β-glucoside (Peng et al., 2002) and the phytohormone brassinosteroid, which also contribute to cellulose synthesis (Xie et al., 2011; Wolf et al., 2012*b*). Therefore, the cellulose deficiencies observed in sterol-impaired plants could be a consequence of such metabolic pathways rather than membrane composition defects. More biochemical and cell biology data are needed to clarify the direct influence of plasma membrane sterols on cellulose synthesis.

The rather imposing size of the CSCs (the membrane-spanning region has a diameter of about 30 nm, and the cytosolic domain at least twice this diameter) triggers interesting questions about how the movement of the complex in the plasma membrane affects the membrane structure and local composition. Membrane elasticity was included as a key factor in a biophysical model of CSC movement in the plasma membrane (Diotallevi and Mulder, 2007). In addition, recent computational simulations showed that the plasma membrane has an important function in the initiation of glucan assembly (Haigler et al., 2014), maintaining a non-crystalline group of chains at the base of the fibril. However, experimental data on how the membrane composition affects CSC trafficking and cellulose synthesis are missing.

The CSC has been shown to be associated with other plasma membrane proteins to fulfil its function. KORRIGAN1 (KOR1), a membrane-localized endo-β-1,4-glucanase, interacts with the CSC, influences the synthesis of cellulose, and increases the amount of non-crystalline cellulose (Nicol et al., 1998; Lane et al., 2001; Takahashi et al. 2009; Lei et al., 2014; Vain et al., 2014). Cellulose Synthase Interactor 1 (CSI1) is necessary for the alignment of CSC trajectories and cortical microtubules, and might bind to plasma membrane phospholipids through a C2-domain at its C-terminus (Bringmann et al., 2012; Li et al., 2012; Mei et al., 2012). Considering their plasma membrane localization, both KOR1 and CSI1 might influence the CSC–membrane connexion.

The plasma membrane–cell wall continuum

In living plant cells, the plasma membrane and cell wall are in close contact, largely due to the immense turgor pressure (similar to that of a car tyre in growing cells) from within the cell that pushes the plasma membrane against the cell wall (Proseus and Boyer, 2005). Physical links between the plasma membrane and the cell wall are well established, as visualized by membranous structures from the plasma membrane that remain firmly anchored to the cell wall under stress generated by partial cell plasmolysis. These plasma membrane–cell wall connections are called Hechtian strands (Oparka, 1994). The Hechtian strands are mainly located at the plasmodesmata, but are also found in other plasma membrane regions (Pont-Lezica et al., 1993). Although Hecht observed these strands more than 100 years ago (Hecht, 1912), the components involved in their generation and maintenance are still largely unknown.

Cellulose microfibrils

Various data support the role of cellulose fibres in plasma membrane–cell wall adhesion (reviewed in Cvřcková, 2013; Martinière and Runions, 2013). Interestingly, cellulose has recently been shown to restrict plasma membrane protein diffusion, which may explain the slow diffusion of plant plasma membrane proteins compared to those in animal cells that lack cell walls (Owen et al., 2009). These observations were obtained either by plasmolysis or by inhibiting cellulose synthesis, during which plasma membrane protein diffusion rates significantly increased (Feraru et al., 2011; Martinière et al., 2011; Martinière et al., 2012). Interestingly, this effect relied on the extracellular domain of the tested proteins, which included AtFH1, GFP-GPI, and PINs (Feraru et al., 2011; Martinière et al., 2011; Martinière et al., 2012). These data support a model in which diffusion of proteins in the plasma membrane may be regulated by interactions between their extracellular domains and cellulose (Martinière et al., 2011; Martinière and Runions, 2013; Fig. 1). However, among the tested proteins above, only AtFH1 has an extracellular domain (the expansin-like domain) which may interact with cell wall polysaccharides. Therefore, the suggested interaction between cell wall and extracellular domains is not necessarily specific and the reduced lateral diffusion reported of the proteins in the presence of an intact cell wall can be explained by steric interference. Moreover, mutations in CesA subunits block the apical-basal localization of PINs in primary root cells. This suggests that the cell wall not only restricts the diffusion of plasma membrane proteins, but also supports specific localization of proteins (Feraru et al., 2011). This is in agreement with observations made with Casparian Strip Protein 1 (CASP1), which is involved in the formation of the Casparian strips in root endodermis. Indeed, CASP1 showed lateral diffusion at the lateral plasma membranes, but became exceptionally slow in the Casparian strip domain. In addition, the strong linkage of CASP1 to the extracellular matrix can only be disrupted with strong detergents. These data suggest that CASP1 might be physically linked to the cell wall via its extracellular domains, and may partially explain CASP1-specific localization (Alassimone et al., 2010; Roppolo and Geldner, 2012).

Under field emission scanning electron microscopy, cellulose fibre-like structures were observed at the ends of Hechtian strands in *Tradescantia virginiana* leaf epidermal cells. The same work showed that treatment of cells with cellulase resulted in a loss of the connecting fibres (Lang *et al.*, 2004). Therefore, cellulose microfibrils were proposed to provide a physical link between the cell wall and plasma membrane. However, more recent work showed that pre-treatment of etiolated hypocotyl cells with cellulose synthesis inhibitors did not block the formation of Hechtian strands (DeBolt *et al.*, 2007). As only qualitative measurements have been done it is difficult to conclude whether the cellulose fibres actively anchor the Hechtian strands to the cell walls, or if this is achieved through other components. Interestingly, certain groups of proteins have been suggested to participate in plasma membrane–cell wall adhesion in plants, including RGD-recognizing proteins (Canut *et al.*, 1998) and GPI-anchored proteins.

The RGD-binding proteins

RGD-dependent linkages provide a basis for cell adhesion in both plants and animals. In animals, extracellular adhesive glycoproteins typically contain RGD sequences that are recognized by plasma membrane receptors called integrins. The RGD–integrin interaction is essential for efficient cell adhesion (Alberts *et al.*, 2002). The presence of an RGD-dependent recognition mechanism and its role in cell adhesion in plants was confirmed by the application of synthetic RGD peptides to soybean suspension cultures. The exogenous RGD was here assumed to compete with the endogenous RGD-containing proteins, and this competition led to growth alterations, which were not observed when cell cultures were treated with RGE or DGR substituted proteins (Schindler *et al.*, 1989). Similar results were reported in onion and *Arabidopsis* cells, where the application of synthetic RGD peptides inhibited Hechtian strand formation (Canut *et al.*, 1998). Moreover, by immunoblotting extracts of soybean cells or protoplasts, Schindler *et al.* (1989) identified a putative plant RGD receptor that was recognized by antibodies raised against a human integrin. The RGD peptide was also found to promote the cell wall regeneration of *Nicotiana tabacum* cv. BY-2 protoplasts (Zaban *et al.*, 2013). These data indicate that plants, similarly to animals, have RGD-recognizing proteins which might help in anchoring the cell wall to the plasma membrane.

In animals, the recognition of RGD sequences by integrins activates inter- and intracellular signal transduction (Baluška *et al.*, 2003). Although plants do not have canonical integrin proteins, integrin-like proteins have been identified (Lü *et al.*, 2007*a*). One such example is AT14A, which contains a domain that shares high sequence similarity with animal integrins (Nagpal and Quatrano, 1999). Mutations in this protein led to a reduction in cell adhesion and an alteration in cell shape and cell wall thickness in *Arabidopsis* suspension cultures (Lü *et al.*, 2012). The Non-race-specific Disease Resistance 1 (NDR1) protein was also identified as an *Arabidopsis* integrin-like protein due to high structural similarity of its core domain to an animal integrin subunit (Knepper *et al.*, 2011*a*). NDR1 contains an apoplastic RGD-like recognition motif, named NGD, and a transmembrane domain which promotes plasma membrane localization. In addition, NDR1 contains a GPI anchor (Coppinger *et al.*, 2004), which is present in many other proteins involved in plasma membrane–cell wall connections as discussed below. The receptor kinase LecRK-I.9 is a lectin-like protein with an RGD-binding motif (Bouwmeester *et al.*, 2011). Application of the recombinant LecRK-I.9 RGD-binding motif to *Arabidopsis* hypocotyls reduced the plasma membrane–cell wall contact sites, possibly by competing with the interaction between the endogenous LecRK-I.9 and RGD-binding protein (Gouget *et al.*, 2006). Subsequently, an alteration in plasma membrane–cell wall linkage was also observed in mutants altered in the receptor LecRK-I.9 (Bouwmeester *et al.*, 2011). While the plant RGD-containing protein that is recognized by LecRK-I.9 has not been identified, the RGD-binding motif of the lectin-like protein responded to a *Phytophtora infestans* effector (Senchou *et al.*, 2004). Therefore, it is plausible that RGD peptide-mediated plasma membrane–cell wall adhesion also occurs in plants (Fig. 1).

The GPI-anchored proteins

GPI can be regarded as a surface anchor for apoplastic proteins (Fig. 1). These proteins are consequently named as GPI-anchored proteins (GAPs). The GPI anchors usually attach GAPs to microdomains in the plasma membrane (Peskan *et al.*, 2000; Bessueille *et al.*, 2009). Phospholipases (PLCs) can cleave the GPI anchor and release GAPs into the cell wall (Griffith and Ryan, 1999). The anchoring of the GAPs to the plasma membrane means that they can establish bonds to cell wall components, and may thus be regarded as promising candidates for plasma membrane–cell wall interactions. *Arabidopsis* has more than 200 putative GAPs, and 41 of these have been confirmed as targets for PLC-dependent release in callus or pollen tubes (Borner *et al.*, 2002; Borner *et al.*, 2003). The GPI-anchored proteins are essential for cell wall architecture, since mutations in GPI synthesis genes induced severe cell wall defects and growth aberrant phenotypes (Lalanne *et al.*, 2004; Gillmor *et al.*, 2005).

GAPs belong to different protein families. One of these families is represented by the *Arabidopsis* protein COBRA and its homologues found in different plants. COBRA and COBRA-like proteins have been reported to be important for anisotropic cell expansion and for the maintenance of cell wall integrity (Roudier *et al.*, 2005; Dai *et al.*, 2011; Cao *et al.*, 2012). The members of this family typically contain an N-terminal carbohydrate-binding module that can bind to crystallized cellulose *in vitro* (Liu *et al.*, 2013; Sorek *et al.*, 2014). This led the authors to hypothesize that COBRA modulates cellulose assembly, both in rice and *Arabidopsis*, by interacting with cellulose chains, which could affect microfibril crystallinity (Fig. 1). Interestingly, COBRA shows a localization pattern reminiscent of cortical microtubules in growing *Arabidopsis* cells (Roudier *et al.*, 2005). As CSCs track along cortical microtubules, these data support a role for COBRA in the assembly of cellulose fibres and their connection to the cytoskeleton. While the biochemical functions

of COBRA remain obscure, the COBRA-related proteins may thus be important factors in maintaining a plasma membrane–cell wall connection. Additional GAPs that regulate cell wall integrity exist, such as SKU5 (Sedbrook *et al.*, 2002), PMR5, and PMR6 (powdery mildew resistant gene 5 and 6: Vogel *et al.*, 2002; Vogel *et al.*, 2004), and LORELEI (Capron *et al.*, 2008; Tsukamoto *et al.*, 2010). However, the exact function of these proteins has not been clarified, and more studies are therefore required to explore their role as possible plasma membrane–cell wall anchors.

Arabinogalactan proteins

Arabinogalactan proteins (AGPs) are apoplastic proteins that are decorated with a glycan moiety of arabinose and galactose sugars, which accounts for more than 90% of the total mass and is suggested to be essential for AGPs function. In addition, the AGPs are typically anchored to the plasma membrane by GPI at their C-terminus (Seifert and Roberts, 2007). AGPs were shown to be covalently attached to cell wall matrix polysaccharides, i.e. to hemicellulose and pectins, by their arabinogalactan decorations (Tan *et al.*, 2013; Fig. 1). Recently, AGP31 was revealed to interact with pectins through its PAC (PRP–AGP-containing Cys) domain, which is not decorated with arabinogalactans (Hijazi *et al.*, 2014). These results indicate that the AGPs have diverse ways of interacting with different cell wall components. Many AGP mutants display defects in plant growth or cell expansion (Ellis *et al.*, 2010). For instance, a null mutation in the *Arabidopsis* classical lysine-rich AGP, AtAGP17, induced plant developmental defects during cell division and expansion (Yang *et al.*, 2007). Overexpression of its homologue, AtAGP18, resulted in bushy plants (Zhang *et al.*, 2011). In addition, AtAGP30 is involved in root regeneration and seed germination (van Hengel and Roberts, 2003). However, although the AGPs are good candidates as plasma membrane–cell wall anchors, it is unclear whether the growth-related defects seen in AGP mutants are consequences of altered plasma membrane–cell wall connections or cell wall integrity defects.

Fasciclin-like arabinogalactan proteins

Fasciclin-like arabinogalactan proteins (FLAs) are another class of GAPs. In *Arabidopsis*, there are 21 predicted FLAs falling into four groups (A to D), of which group A and C are GPI anchored (Johnson *et al.*, 2003). In addition, each FLA contains a fasciclin domain and one or two arabinogalactan domains (Johnson *et al.*, 2003; Fig. 1). The fasciclin domain is an extracellular module of about 140 amino acid residues with low sequence similarity, but with two highly conserved regions of ~10 amino acids each. The fasciclin domain is suggested to be an ancient cell adhesion domain common to a broad spectrum of organisms (Kawamoto *et al.*, 1998), and there are well studied cell adhesion proteins in, for example, fruitfly and human (Snow *et al.*, 1989; Elkins *et al.*, 1990; Goodman *et al.*, 1997). Based on their structure, the plant FLAs are also thought to be involved in plant plasma membrane–cell wall adhesion; however, this has not been widely explored. Interestingly, the *Arabidopsis fla4* mutant was reported to have increased plasma membrane–cell wall interface spacing, but there was not precise quantification (Shi *et al.*, 2003). Nevertheless, indirect data support a role for plant FLAs in cell wall integrity, especially during cellulose deposition. For example, FLA4 is necessary for proper cell expansion, and for cellulose and pectin synthesis in the seeds coat (Harpaz-Saad *et al.*, 2011; Griffiths *et al.*, 2014). In addition, many *fla* mutants, in both *Arabidopsis* and cotton, showed reduced cellulose content (Li *et al.*, 2010; MacMillan *et al.*, 2010; Johnson *et al.*, 2011; Huang *et al.*, 2013).

Plasma membrane receptors

Another group of plasma membrane proteins that has been suggested to be plasma membrane–cell wall anchors are receptor-like kinases (RLKs) with extracellular domains that can interact with cell wall components, such as the Wall-Associated Kinases (WAKs; Kohorn and Kohorn, 2012) and members of the *Catharanthus roseus* RLK (CrRLK) family (including FERONIA, THESEUS, HERK, and ANXUR: Cheung and Wu, 2011). The extracellular domain of WAKs has been shown to bind *in vitro* to native pectin of the cell wall (Decreux and Messiaen, 2005) and, therefore, might take part in the plasma membrane–cell wall connection by covalently binding to apoplastic pectins (Wagner and Kohorn, 2001; Wolf *et al.*, 2012*a*). Members of the CrRLK family contain extracellular malectin-like (ML) domains with high sequence similarity to the carbohydrate-binding protein, malectin, from *Xenopus laevis* (Lindner *et al.* 2012). Similarly, CrRLKs may interact with cell wall components through their extracellular ML domain. Indeed, a point mutation in the ML domain of THESEUS restored the growth deficiency of cellulose mutants (Hématy *et al.*, 2007). Both the WAK and CrRLK families are essential for correct cell wall growth and cell expansion (Hématy *et al.* 2007; Fujikura *et al.* 2014; Haruta *et al.*, 2014), and they have been previously reviewed by Cheung and Wu (2011) and Kohorn and Kohorn (2012). However, plasma membrane–cell wall linking defects in *wak* and *crrlk* mutants have not been identified.

Cell sensing through plasma membrane–cell wall anchors

The cell wall needs to adapt its structure during cell growth, and to respond to both internal and external stresses. Therefore, physical perturbations of the cell wall must be communicated to the cell via the plasma membrane. Wolf *et al.* (2012*a*) recently reviewed plasma membrane receptors that might transduce cell wall-related signals. We therefore only briefly discuss RLKs in cell wall sensing, and elaborate on the putative function of other plasma membrane–cell wall anchors as sensors of cell wall signals, and their influence on plant resistance and cell morphology.

Plasma membrane–cell wall junctions may function as barriers that protect the plant cell against pathogen attacks (Mellersh and Heath, 2001). In line with this hypothesis, pre-treatment of plants with synthetic RGD peptides, which blocked plasma membrane–cell wall adhesion, increased pathogen penetration, especially of biotrophic pathogens (Mellersh and Heath, 2001). Not surprisingly, one mechanism

of biotroph pathogenicity is the disruption of plasma membrane–cell wall adhesion. Thus, the *Phytophthora infestans* RXLR-dEER effector, IPI-O, is an RGD-containing protein whose overexpression in *Arabidopsis* reduced plasma membrane–cell wall interaction (Bouwmeester *et al.*, 2011). The IPI-O is recognized by the integrin-like LecRK-I.9, and competes with the endogenous RGD-containing proteins that are made in the plant. This competition led to the destabilization of the plasma membrane–cell wall connection, benefiting progression of the pathogen. Similarly, plants mutated in LecRK-I.9 are more susceptible to *P. infestans* than wild-type plants (Bouwmeester *et al.*, 2011). In addition, mutations in the integrin-like protein NDR1 also increased plant susceptibility to pathogens (Knepper *et al.*, 2011*b*). Besides their function in cell expansion, WAKs have also been involved in plant immune responses (Rosli *et al.*, 2013) by recognizing pectin fragments [oligogalacturonides (OGAs)] derived from pathogen digestion of the cell wall (Brutus *et al.* 2010; Kohorn *et al.*, 2014). Interestingly, WAKs seem to distinguish the pectin native polymers from the OGAs, differentiating expansion- from stress-related signals (Kohorn *et al.*, 2014).

Cell wall mechanical sensing is another process which probably depends on the plasma membrane–cell wall interaction. A recent study showed that the plasma membrane receptor FERONIA is important for the Ca^{2+}-dependent mechanical response pathway (Shih *et al.*, 2014). Complementary work has recently shown that FERONIA activation by the secreted peptide RALF (rapid alkalinization factor) inhibits plasma membrane proton activity, which induces extracellular alkalinization and therefore inhibits cell expansion (Haruta *et al.*, 2014). It would be interesting to know whether FERONIA senses the cell wall components directly during the mechanical stress or senses other signals induced by the mechanical stress (like RALF and Ca^{2+}), and subsequently alters cell growth.

The plasma membrane–cell wall connection might also be important for responses to other external stimuli, such as osmotic stress. One of the clearest outputs of osmotic stress is the production of abscisic acid (ABA) (Xiong *et al.*, 2001; Xiong *et al.*, 2002). Application of synthetic RGD peptides decreased accumulation of ABA after osmotic stress in both *Arabidopsis* and maize suspension cultures (Lü *et al.*, 2007*a*; Lü *et al.*, 2007*b*). Interestingly, a similar study using protoplasts revealed no impairment in ABA levels, corroborating the function of the plasma membrane–cell wall interaction in the osmotic stress response. Furthermore, the expression of some AGPs and FLAs was reported to respond to hormones: ATAGP30, FLA1, FLA2, and FLA8 to ABA; and ATAGP31 to jasmonic acid (van Hengel and Roberts, 2003; Liu and Mehdy, 2007). Additional studies indicate that FLAs participate in stress signalling pathways and might regulate cell wall biosynthesis. Thus, cell swelling phenotypes observed in *fla4*/*salt overly sensitive5* (*sos5*) mutants under elevated salt and high sucrose conditions could be rescued by the addition of external ABA (Seifert *et al.*, 2014) and ethylene biosynthesis inhibitor (Xu *et al.*, 2008), respectively. Moreover, FLA4/SOS5 was found to be genetically redundant with the FEI1/FEI2 receptor-like kinases, based on their non-additive triple mutant phenotype (Xu *et al.*, 2008). The double mutant *fei1 fei2* displays alterations in anisotropic cell expansion and cell wall synthesis, and can be rescued by inhibition of ethylene biosynthesis, but not by blocking the perception of ethylene (Xu *et al.*, 2008). These data suggest that cell wall synthesis might be regulated by plasma membrane–cell wall anchors through hormone-signalling pathways, but the mechanism underpinning these connections is unknown.

Conclusion and future perspectives

The interdependence of the cell wall and intracellular compartments is important for plant development and relies on adequate connections between the cell wall and plasma membrane. Important questions remain to be answered in the fields of secretion and internalization of cell wall material, and of the coordination between cell wall synthesis and degradation. One especially puzzling question relates to how the plasma membrane lipids and CSCs interact during cellulose synthesis. Indeed, the way in which plasma membrane lipids affect protein function *in vivo*, and how they change during vesicle trafficking, is largely unknown. Interestingly, cell wall architecture influences the activity of the CSCs. Moreover, the motility and localization of other plasma membrane proteins are also controlled by cell wall polymers, probably by physical connections which have not been fully elucidated. Therefore, these plasma membrane proteins and CSCs might act as plasma membrane–cell wall anchors with putative roles in cell adhesion.

Cellular adhesion is essential for maintaining the multicellular structure and for signal transduction in eukaryotes. It is plausible that the plasma membrane proteins involved in cell adhesion act as mechanosensors and as receptors of pathogen effectors (Baluška *et al.*, 2003). It is becoming increasingly clear that plants share a similar mechanism to that described in animals for perceiving apoplastic signals by plasma membrane–cell wall adhesion domains, and integrate them into cytoskeletal and hormonal responses to regulate cell expansion (Baluška *et al.*, 2003; Sampathkumar *et al.*, 2014). While plants have many putative plasma membrane–cell wall anchoring proteins, similar to those identified in cell adhesion in other eukayotes, much remains to be investigated. For example, which plant proteins contain RGD motifs? And do these RGD-containing proteins bind to cell wall polysaccharides? Moreover, little information is available about the downstream effects, and effectors, of changes in cell adhesion properties, e.g. impacts on the cytoskeleton and hormone responses. In addition, a complete characterization of plasma membrane–cell wall adhesion needs a multicellular perspective, i.e. including influences from neighbouring cells. We envision that we will get closer to such insights by the development and combination of proteomic, biochemical, biomechanical, and imaging tools. Thus, we expect that a deeper understanding of the plant plasma membrane–cell wall connection will increase our knowledge about how plant tissues are organized through cell adhesion and cell–cell communication

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