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Plant cell wall integrity maintenance as an essential component of biotic stress response mechanisms

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Plant cell walls provide structural support during development and represent together with the cuticle the first line of defense against biotic and abiotic stress. In recent years, evidence has accumulated that a dedicated plant cell wall integrity (CWI) maintenance mechanism exists. This mechanism monitors and maintains functional integrity of the cell wall during different biological processes. The available data suggest that it may represent a component of the stress response mechanisms underlying biotic and abiotic stress responses, which has not been identified previously as a distinct mechanism. Here I will review the available evidence regarding the mode of action of the CWI maintenance mechanism and discuss its role in the context of biotic plant stress response mechanisms.

Keywords: plant cell wall integrity, biotic stress, plant pathogen

INTRODUCTION

Plant cells need to maintain the functional integrity of their walls during cell morphogenesis and exposure to biotic/abiotic stress. The available evidence suggests that a dedicated plant cell wall integrity (CWI) maintenance mechanism exists (Wolf et al., 2011). While our understanding of the mechanisms regulating stress responses and morphogenesis has increased significantly, our knowledge regarding the processes maintaining CWI is still limited. In the last years several reviews have been published on the plant CWI maintenance mechanism illustrating the increased interest in this area (Humphrey et al., 2007; Hematy et al., 2009; Ringli, 2010; Seifert and Blaukopf, 2010). A recently published review focuses on CWI maintenance during plant cell wall morphogenesis (Wolf et al., 2011). Similarities between the yeast and plant CWI maintenance mechanisms have also been reviewed (Hamann and Denness, 2011). Therefore, the available knowledge regarding CWI maintenance during plant development and in yeast will be covered here only briefly to provide a conceptual framework regarding cellular processes involved and to illustrate the degree of functional conservation between species. This review will focus on recent developments regarding the role of CWI maintenance during biotic stress responses. It will discuss how CWI maintenance could have a previously unrecognized role in the perception of and response to biotic stress.

SIMILARITIES AND DIFFERENCES BETWEEN YEAST AND PLANT CELL WALL INTEGRITY MAINTENANCE MECHANISMS

While both plant and yeast cells are enveloped by cell walls, certain important differences exist that affect the biological role and function of the plant CWI maintenance mechanism. Yeast unlike plant cells, do not have to deal with biotic stress. In addition, plant cell walls are structurally and chemically more complex than the yeast cell wall. This means that in plant cells the sheer number of cell wall-related signaling events during development and plant–environment interaction could disguise the activity of a dedicated plant CWI maintenance mechanism.

The yeast CWI monitoring and maintenance network is quite complex, providing an indication of the possible complexity of the plant CWI maintenance network. By combining inputs from a turgor pressure sensor (SLN1), mechano-perception (MID1/CCH1), and dedicated cell wall damage (CWD) sensors (WSC1, 2, 3, MID2, MTL1) the yeast CWI maintenance network generates signals that permit highly specific responses to any challenge that impairs the functional integrity of the yeast cell wall. The available phenotypic and genetic data also implicate turgor pressure, mechano-perception, and CWD detection in plant CWI maintenance (Hamann et al., 2009; Denness et al., 2011). Interestingly, *ARABIDOPSIS HISTIDINE KINASE1* (*AHK1*) and *CYTOKININ RECEPTOR1/ARABIDOPSIS HISTIDINE KINASE4* (*CRE1*/*AHK4*) can at least partially rescue a yeast strain with a loss of function allele in *SLN1* (Urao et al., 1999; Inoue et al., 2001). In addition, expression of the *Arabidopsis thaliana MID1 COMPLEMENTING ACTIVITY 1* and *2* (*MCA1*, *2*) genes rescues a *MID1*-deficient yeast strain suggesting that they could function as stretch-activated calcium channels (Nakagawa et al., 2007; Yamanaka et al., 2010). Up to now no functional homologues for the yeast CWD sensors have been identified in plants. In yeast, the signals generated by the sensors are relayed to the response genes via different signaling cascades involving CALCINEURIN (MID1/CCH1); RHO1/MPK1 (WSC1, MID2), and YPD1 (SLN1) (Levin, 2005). Transcription factors mediating the response are SKN7, RLM1, and SWI4/6 (Levin, 2005). The response can involve activation of genes required for cell wall biosynthetic processes, remodeling of the cytoskeleton, and cell cycle progression. The available data suggest both organizational and functional

similarities between the yeast and plant CWI maintenance mechanism while also highlighting how signals from mechanical and chemical sensors regulate jointly the CWD response.

COMPOSITION AND STRUCTURE OF THE PLANT CELL WALL

The plant cell wall is comparable to an exoskeleton surrounding the plant cell and providing both structural support and protection from biotic as well as abiotic stresses. It consists of cellulose microfibrils, pectin, hemicelluloses, proteins, and in certain cases lignin (Somerville et al., 2004). Plant cell walls are divided into primary (laid down during cell elongation/differentiation) and secondary (formed after cell morphogenesis is concluded) walls. In parallel, dependent on the presence of certain polysaccharides type I and type II cell walls are distinguished (Popper et al., 2011). Cellulose microfibrils are the main load bearing elements, which are cross-linked to hemicelluloses and (*in vitro*) to pectin (Dick-Perez et al., 2011). Hemicelluloses and pectin also form direct links creating a matrix in which the microfibrils are embedded like the steel mesh in a concrete wall. Pectic polysaccharides like homogalacturonan (HG) are connected by calcium bridges between dimethyl-esterified parts of the molecules or through borate ester linkages in the case of rhamnogalacturonan II (RG II). They represent important cell wall components during morphogenesis facilitating cell expansion and plant–pathogen interaction (Hahn et al., 1981; Bellincampi et al., 1996). In the latter situation they are targeted by pathogen-derived cell wall degrading enzymes (polygalacturonases), which generate oligogalacturonides (OGAs) from HG (Hahn et al., 1981; Kars et al., 2005). Biologically active OGAs consist of chains of 9–15 galacturonic acid (GalA) monomers and can function as signaling molecules (see below).

During secondary cell wall formation monolignols (precursors for lignin) are secreted into the cell wall space and randomly cross-linked (Vanholme et al., 2010). The cross-linking is dependent on the availability of reactive oxygen species (ROS) generated by laccases and peroxidases. This process reinforces the wall against pathogen infection, waterproofs it, and increases structural integrity (Tronchet et al., 2010; Vanholme et al., 2010). Reduction of cellulose biosynthesis during primary cell wall formation through genetic or chemical means leads to lignin production (Ellis et al., 2002; Hamann et al., 2009). This highlights the ability of the cell to adapt to changes in cell wall composition and provides evidence for the existence of a CWI maintenance mechanism.

THE PLANT CWI MAINTENANCE MECHANISM AS A COMPONENT OF THE BIOTIC STRESS RESPONSE

Plant cell walls are capable of adjusting their composition and structure in response to pathogen infection (Dong et al., 2008). A mutation in (*CELLULOSE SYNTHASEA3*, *CESA3*) a subunit of the cellulose synthase complex leads to ectopic production of lignin, i.e., replacement of a missing load bearing cell wall component by another one (Cano-Delgado et al., 2000). Follow up studies found that inhibition of cellulose biosynthesis during primary cell wall formation either through mutations like *constitutive expression of VSP1* (*cev1*) and *ectopic lignification1* (*eli1*) or inhibitors such as isoxaben results in transcriptional activation of stress response mechanisms; ectopic production of ethylene (ET), salicylic acid (SA), jasmonic acid (JA), callose, and ROS as well as changes in cell wall composition and structure (Ellis et al., 2002; Cano-Delgado et al., 2003; Manfield et al., 2004; Hamann et al., 2009). The experiments also showed that the response to CWD consists of early and late stages reminiscent of the response to pathogen infection (Denness et al., 2011). During the early stage, ROS- and calcium-based signaling cascades are required for initiating the response to CWD (Denness et al., 2011). Interestingly, 1-aminocyclopropane-1-carboxylic acid (ACC, an ET precursor) and not ET itself seems to be acting as signaling substance during the early response to isoxaben with inhibition of cell expansion being an active process and not simply an automatic consequence of cellulose biosynthesis inhibition (Tsang et al., 2011). During the late stage, responses to CWD-like lignin deposition are initiated and the extent of lignin formation is apparently modulated by a negative feedback loop formed by ROS and JA (Denness et al., 2011). A combination of genetic and phenotypic analysis has implicated the NADPH oxidases *RESPIRATORY BURST OXIDASE HOMOLOGD* and *F* (*RBOHD*, *F*), the serine/threonine kinase *OXIDATIVE SIGNAL INDUCIBLE1* (*OXI1*), *MCA1*, the receptor-like kinase (RLK) *THESEUS1* (*THE1*) as well as the JA biosynthesis genes *ALLENE OXIDE SYNTHASE* (*AOS*) and *JASMONIC ACID RESISTANT1* (*JAR1*) in the signaling mechanism mediating the response to CWD in *Arabidopsis* seedlings. Interestingly, the *cev1* mutation that affects cellulose biosynthesis during primary cell wall formation also causes enhanced resistance to infection by different powdery mildews (*Erysiphe orontii*, *E. cichoracearum*, and *Oidium lycopersicum*; Ellis and Turner, 2001).

A screen for mutants causing resistance to powdery mildew infection provides further evidence of the close relationship between plant cell walls and pathogen resistance (Vogel and Somerville, 2000). Three of the powdery mildew resistance (*PMR*) mutants that have been identified on the molecular level affect genes involved in cell wall biosynthetic processes. *PMR4* encodes a callose synthase, *PMR5* a gene of unknown function required for pectin production and *PMR6* a pectate lyase (Vogel et al., 2002, 2004; Nishimura et al., 2003). *pmr4* resistance seems to be mediated via hyper-activation of SA signaling, whereas *pmr5* and *6* resistance phenotypes are independent of JA, SA, and ET signaling. Mutations in *IRREGULARXYLEM1* (*IRX1*/*CESA8*), *3* (*IRX3*/*CESA7*), and *5* (*IRX5*/*CESA4*) impair cellulose biosynthesis during secondary cell wall formation and cause enhanced resistance to the soil borne bacterium *Ralstonia solanacearum* and the necrotrophic fungus *Plectosphaerella cucumerina* (Hernandez-Blanco et al., 2007). Mutations affecting cellulose biosynthesis during primary cell wall formation (*cesa1, 3, 6*) or other components of the secondary cell wall (*pmr5*, *pmr6*) did not cause enhanced resistance to the same pathogens. Genetic analysis showed that the enhanced resistance in *cesa4, 7, 8* is independent of JA, SA, and ET-based signaling mechanisms. Results from expression profiling experiments and genetic analysis using different abscisic acid (ABA) mutants (*ABA insensitive 1-1*; *2-1*; *ABA1-6*) suggest that ABA is mediating developmental and pathogen resistance phenotypes caused by the *irx* mutants. However, it remains to be determined if the ABA involvement is direct or a secondary effect due to water stress caused by problems with xylem cell wall

formation in the mutants. To summarize, these results suggest distinct resistance signaling cascades are induced by defects in primary and secondary cell wall formation as well as for different secondary cell wall components. They also highlight the direct impact of changes in cell wall composition/structure on the response to pathogen infection.

SIGNALING MECHANISMS AND SENSORS IMPLICATED IN PLANT CWD PERCEPTION

The mode of action of the plant cell wall maintenance mechanism is not well understood. Based on the knowledge from yeast, chemical and physical signals could act as indicators for the functional integrity of the plant cell wall either individually or jointly. By combining these different types of signal the plant cell would receive precise information regarding the state of its cell wall and the exact type of CWI impairment occurring. Physical signals could be generated by stretching of the plasma membrane due to a weakened cell wall that cannot resist the high turgor pressure levels within a plant cell or a plasma membrane that is displaced relatively to the cell wall. These events could be detected by stretch-activated or mechanosensitive channel proteins that lead to calcium influx into the cytoplasm, indicating CWD. Sensor candidates could be encoded by members of the mechanosensitive channels of small conductance (MscS)-like (*MSL*) gene family like *MSL 9* and *10* affect mechano-perception in protoplasts derived from *Arabidopsis* root cells (Haswell et al., 2008). Another candidate of interest is the putative stretch-activated calcium channel MCA1 is required for CWD-induced lignin deposition. Interestingly, all isoxaben-induced CWD phenotypes can be suppressed by provision of osmotic support suggesting that changes in turgor pressure due to a weakened cell wall could result in signal generation via turgor pressure sensors (Hamann et al., 2009; Denness et al., 2011). While AHK1 and 4/CRE1 can function as osmosensors in yeast and have been implicated in abiotic stress responses, no clear evidence exists implicating them in CWD perception in plants (Urao et al., 1999; Inoue et al., 2001; Tran et al., 2007). In addition, AHK4/CRE1 has been shown to function as a cytokinin receptor (Inoue et al., 2001). Therefore, the question that needs to be resolved at this point is if turgor pressure is a passive element in the process (generating cell wall fragments due to a weakened cell wall) or an active component that is being monitored and provides input into the process.

The plant cell wall contains a large number of components that could generate chemical signals (ligands) indicative of CWD or general danger signals. The term damage associated molecular patterns (DAMPs) has been coined to describe such ligands and the number of possible DAMPs originating in plant cell walls is rather large (Zipfel, 2009). Here I will focus on the best-characterized group of signals, which are probably OGAs. They can be generated through degradation of HG by pathogen-derived enzymes (Kars et al., 2005; Ferrari et al., 2008). OGAs have been shown to induce gene expression changes, stomatal closure, production of ET, and ROS as well as cell wall reinforcement (Denoux et al., 2008; Ferrari et al., 2008). A hybrid kinase consisting of the extra cellular domain of *WALL-ASSOCIATED KINASE1* (*WAK1*) and the intracellular domain of elongation factor Tu receptor (EFR) kinase can bind OGAs and activate defense responses (Brutus et al., 2010). *WAK1* belongs to a family of five WAK genes encoding plasma membrane-localized Ser/Thr kinases that have been implicated in response to pathogen infection and regulation of cell elongation (Kohorn et al., 2011). The effects of the chimeric WAK1 kinase on pathogen resistance suggest that OGAs and WAKs represent an *in vivo* ligand–receptor pair. Results from the analysis of a dominant active WAK2 allele suggest the CWD signals perceived by WAKs could be relayed to downstream response genes through *MAPKINASE6* (*MPK6*) (Kohorn et al., 2009, 2011). Interestingly, WAK2 has also been implicated in regulation of invertase activity and turgor pressure during cell elongation (Kohorn et al., 2006). However, there is currently no confirmation that WAKs are actively involved in CWI maintenance.

In *Arabidopsis*, more than 600 RLKs have been identified and a large number of them have been implicated in developmental and stress response processes (Shiu and Bleecker, 2001). I will focus here on several kinases that have been implicated in CWD perception and/or pathogen response. Most of the RLKs implicated in CWI maintenance [THE1, HERCULES1 (HERK1), FERONIA (FER)] belong to the *Catharanthus roseus* RLK1 (CrRLK1)-like protein family, which has 17 members in *Arabidopsis*. *THE1* was isolated as a suppressor of the cellulose-deficient *cesa6 procuste* (*prc*) mutant, which exhibits a hypocotyl elongation defect (Hematy et al., 2007). Although *the1* suppresses the elongation defect, the cellulose deficiency is not reduced. Subsequently it has been shown that THE1 is required for cellulose biosynthesis inhibition-induced ROS production and lignification in the root elongation zone (Denness et al., 2011). THE1, HERK1, and FER have been implicated in brassinosteroid-induced cell elongation (Guo et al., 2009; Deslauriers and Larsen, 2010). Both FER and NORTIA/MILDEW RESISTANCE LOCUS O 7 (*MLO7*; a seven-transmembrane domain protein involved in powdery mildew resistance) are required for successful fertilization and resistance to infection by *Golovinomyces* (syn. *Erysiphe*) *orontii* (Kessler et al., 2010). Interestingly, ROPGEF (guanine-exchange factors) proteins have been identified as targets of FER activity (Duan et al., 2010). ROPGEFs are required for the activation of Rho GTPases, which in turn activate NADPH oxidases like RBOHD/F. These results suggest the same molecular components could mediate cell–cell interaction during development and plant–pathogen interaction.

Heterotrimeric G-proteins (Gα, Gβ, Gγ) form a highly conserved signaling complex that has been implicated in signal transduction during development and stress responses in mammals, yeast, and plants (Digby et al., 2006; Temple and Jones, 2007). In *Arabidopsis*, five genes GPA1 (Gα), AGB1 (Gβ), AGG1, 2, 3 (Gγ1, 2, 3) encode the subunits of the complex (Thung et al., 2011). Recently, it has been reported that mutations in *AGG1*, *2*, and *AGB1* apparently cause enhanced susceptibility to infection with *P. cucumerina* (Delgado-Cerezo et al., 2011). A combination of metabolomic and microarray-based expression profiling studies of the mutants established that the pathogen phenotype is independent of SA, JA, ABA, and ET signaling cascades. Interestingly, a large number of cell wall biosynthetic/modifying genes are mis-regulated in *agb1* and *agg12* plants. Analysis of cell wall composition/structure in these plants found reduced xylose contents in the mutants compared to wildtype.

To summarize, the available evidence supports the notion that the plant cell wall is an integral component contributing to pathogen response mechanisms and illustrates the influence of cell wall defects on infection. Specific signaling cascades seem to mediate the response to particular cell wall defects, which in turn affect the response to necrotrophic or biotrophic pathogens. More importantly the data presented above allow correlation between certain types of cell wall defects, and not only signaling cascades but also resistance phenotypes. Mutations in *CESA4*, *7*, *8*, *AGG1*, *2*, and *AGB1* affect resistance to necrotrophs and are independent of phytohormone-based signaling cascades. *pmr5*, *6* plants exhibit resistance to biotrophs and also do not rely on phytohormone-based signaling cascades. *pmr4* affects resistance to biotrophs and resistance depends on the integrity of the SA signaling cascade. *cesa3* plants show enhanced JA, ET biosynthesis, and resistance to biotrophs.

The available data allow different explanations for the specific effects on pathogen resistance observed. The cell wall composition/structure changes could prevent pathogen colonization simply because the infection machinery of the pathogen is too specialized to breach the chemically modified cell wall. However, no functional pathogen response mechanisms should be required for this possibility. Another option is that the cell wall mutants cause defects similar to those occurring during infection by particular pathogens, i.e., simulate infection by necrotrophs or biotrophs. This would cause early/constant activation of the CWI maintenance/defense mechanism, which "primes" plant immunity thus making successful infection more difficult. The latter would explain both the specificity of the responses observed and dependence on particular signaling mechanisms. Therefore, studies focusing on the effects of particular cell wall defects on pathogen resistance and the mode of action of the CWI maintenance mechanism could facilitate research into biotic stress response. The reason being, that by removing the potentially multiple effects of the pathogen during infection, they reduce the complexity of the interaction and should therefore allow novel insights into the mechanisms responsible for detection of infection and/or physical damage.

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