

The Role of Receptor-Like Kinases in Regulating Cell Wall Function¹

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The plant cell wall is a rigid but highly dynamic structure that provides mechanical support, protection against pathogen attack, and determines the direction and extent of cell expansion (Humphrey et al., 2007). The dynamic nature of the plant cell wall allows growing cells to expand while providing the mechanical strength required to resist the forces of turgor pressure exerted on the cell (Cosgrove, 2000). The properties of the cell wall are modified during growth and development as well as in response to a wide variety of environmental stimuli. In order to maintain the integrity of the wall and to adjust its properties to accommodate the changing needs of the cell, plants respond to perturbations to the wall and environmental cues by remodeling matrix polysaccharides and by regulating the cell wall biosynthetic machinery. The components and mechanisms underlying such a signaling system remain largely unknown, but emerging evidence has implicated several receptor-like kinases as regulators of cell wall function.

Plant cell walls are composite structures composed primarily of cellulose and matrix polysaccharides such as hemicelluloses and pectins (Somerville et al., 2004). In addition to wall polymers, structural proteins provide a quantitatively small but important contribution to the wall. The major load-bearing components of the cell wall are the cellulose microfibrils, which, in longitudinally expanding cells, are deposited primarily in an orientation perpendicular to the axis of expansion, thus constricting radial expansion (Green, 1980; Taiz, 1984; Baskin, 2005). Consistent with a role in differential cell expansion, cellulose-deficient mutants and seedlings treated with inhibitors of cellulose synthesis display reduced or no growth anisotropy, and this is generally accompanied by cell and organ swelling (Somerville, 2006). The oriented deposition of cellulose is guided by underlying cortical microtubules; thus, cortical microtubules are thought to be key determinants of anisotropic growth (Baskin, 2001; Paredez et al., 2006; Lucas and Shaw, 2008).

In the primary cell wall, cellulose is synthesized at the plasma membrane by a hexameric protein complex called cellulose synthase (CESA). Each hexamer is composed of six CESA proteins that each synthesize a β -1,4-linked glucan chain. A combination of expression analyses, genetic studies, and coimmunoprecipitation experiments have defined roles for the various CESA isoforms in *Arabidopsis thaliana*. CESA1, CESA3, and CESA6 interact with each other to form a class of rosettes that function in primary cell wall biosynthesis (Desprez et al., 2007). CESA2, CESA5, and CESA9 also likely function in primary cell wall synthesis in a manner such that they are partially redundant with CESA6 at different stages of growth (Desprez et al., 2007; Persson et al., 2007). CESA4, CESA7, and CESA8 constitute a distinct subset of rosettes that function in secondary cell wall biosynthesis (Taylor et al., 2000, 2003). Single mutants in any one of the genes encoding these CESA proteins are deficient in cellulose biosynthesis, which suggests that a functional cellulose synthase complex requires contributions from three different CESA subunits (Desnos et al., 1996; Arioli et al., 1998; Taylor et al., 1999, 2000; Fagard et al., 2000; Caño-Delgado et al., 2003; Taylor, 2008).

The same structure and composition that lends strength and rigidity to the wall also serves to constrain cell expansion. While cell wall loosening is essential for expansion, this must be balanced with polymer synthesis and wall restrengthening to prevent the cell wall from rupturing. Such wall remodeling is facilitated by the activity of loosening and strengthening agents that modify cell wall polysaccharides. For example, wall loosening is accomplished through the activities of hydroxyl radicals, expansins, xyloglucan endoglucosylase/hydrolases, and endo-(1,4)- β -D-glucanases, whereas the extensins and peroxidases function in wall rigidification (Cosgrove, 2005; Humphrey et al., 2007). Coordinating wall loosening with wall-strengthening activities during cell expansion requires the ability of the cell to monitor changes in wall integrity and to signal back to regulate the machinery involved in the synthesis and modification of the cell wall components.

Such a cell wall signaling system has been well characterized in the yeast *Saccharomyces cerevisiae* (Levin, 2005). In this system, the cell wall integrity and stress response component (WSC) and mating induced death 2 (MID2) cell surface receptors func-

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tion as sensors of cell wall integrity. Both WSC and MID2 contain an extracellular domain rich in Ser/Thr residues, a single transmembrane domain, and a small C-terminal cytoplasmic domain that interacts with ROM1 and ROM2 (Philip and Levin, 2001). Upon activation, ROM1 and ROM2 stimulate the small GTP-binding protein Rho1, which in turn initiates a variety of processes, including changes in the synthesis of β -glucan, nucleation of actin filaments, secretory vesicle targeting, and activation of a mitogen-activated protein kinase (MAPK) cascade that leads to changes in gene expression related to cell wall biogenesis (Ozaki et al., 1996; Levin, 2005).

The hypothesis that plant cells have the ability to sense and respond to changes in wall integrity is supported by the observation that genetic or chemical perturbation of cellulose biosynthesis results in an ectopic deposition of lignin. Lignification, which increases the rigidity of the cell wall, normally occurs in the secondary cell walls of the vascular tissue and in response to pathogen attack. Ectopic lignin deposition has been observed in several cellulose-deficient mutants (Vance et al., 1980), including *rsw1* (*root swelling1*), *eli1* (*ectopic lignification*), and *prc1* (*procuste*), which disrupt *CESA1*, *CESA3*, and *CESA6*, respectively, as well as the *korrigan* and *fei* mutants (Nicol et al., 1998; Caño-Delgado et al., 2000, 2003; Fagard et al., 2000; Xu et al., 2008). Ectopic lignin deposition also occurs in seedlings treated with the cellulose synthesis inhibitors 2,6-dichlorobenzonitrile and isoxaben (Caño-Delgado et al., 2003). In addition to increased lignin deposition, disruption of cellulose synthesis also results in other changes, including changes in gene expression, activation of ethylene and jasmonic acid signaling pathways, and the inhibition of cell elongation (Caño-Delgado et al., 2003; Duva and Beaudoin, 2009). These changes in cellular function indicate that the cell not only senses changes in the wall but that there is a feedback system in place to maintain cell wall integrity.

Relatively little is known about the molecular components and signal transduction pathways involved in the regulation of plant cell wall function. Recent studies have implicated multiple receptor-like kinases (RLKs) in cell wall signaling. RLKs represent a large (approximately 600 in Arabidopsis), diverse family of proteins (Shiu and Bleeker, 2001) that physically link the cell wall to the cytoplasm, making them ideal candidates for cell wall sensors. RLKs are situated at the plasma membrane and contain an extracellular domain, a transmembrane domain, and an intracellular Ser/Thr kinase domain. They have been implicated in various signaling pathways, including meristem function, brassinosteroid perception, floral abscission, ovule development and embryogenesis, plant defense, and overall plant morphology (Becraft, 2002). This review highlights the role of RLKs in cell wall function.

THE WALL-ASSOCIATED KINASES

The wall-associated kinases (WAKs) are a set of RLKs that are tightly bound to the cell wall (He et al., 1996). There are five highly conserved WAK genes in Arabidopsis and an additional 26 WAK-like genes that encode proteins with divergent extracellular domains (Verica et al., 2003). The WAK proteins consist of an extracellular domain, a transmembrane domain, and a cytoplasmic Ser/Thr protein kinase domain. The extracellular domains of the WAKs are 40% to 60% identical to each other and contain two epidermal growth factor-like repeats. This domain binds tightly to pectin in a calcium-dependent fashion (Decreux and Messiaen, 2005). This association with pectin first occurs in an endomembrane compartment, most likely the Golgi (Kohorn et al., 2006a). The intracellular kinase domains of the WAKs are more highly conserved than their extracellular domains, which might reflect similar downstream targets; alternatively, this catalytic domain may be more evolutionarily constrained. All five WAKs are expressed widely throughout the plant in the expanding cells of leaves, stems, roots, and fruits, and their expression is differentially regulated by environmental and developmental cues such as wounding, pathogen infection, and aluminum (He et al., 1998; He et al., 1999; Wagner and Kohorn, 2001; Sivaguru et al., 2003).

WAKs are required for cell expansion during plant development. Disruption of WAK function using inducible expression of full-length WAK2 antisense RNA, which likely disrupts multiple WAKs, compromised leaf cell expansion (Lally et al., 2001; Wagner and Kohorn, 2001). Consistent with these results, root cell elongation is impaired in *wak2* loss-of-function mutants and in seedlings expressing WAK4 antisense RNA (Kohorn et al., 2006b). The growth of a *wak2* loss-of-function mutant was dependent on exogenous sugars, suggesting that the mutation may alter sugar metabolism (Lally et al., 2001; Kohorn et al., 2006b). This idea is supported by the finding that *wak2* mutant roots show reduced vacuolar invertase activity, which is critical for the generation of solutes required to maintain turgor pressure during cell expansion (Kohorn et al., 2006b). Furthermore, both the expression of *INV1*, which encodes an invertase enzyme, and MAPK3 activity are induced in Arabidopsis mesophyll protoplasts treated with pectin in a WAK2-dependent manner (Kohorn et al., 2009). Loss-of-function *mapk3* mutants, which are aphenotypic, enhanced the phenotypic effects of a WAK2 dominant negative transgene. Together, these results suggest that WAK2 and MAPK3 may be involved in a pathway that modulates the activity of vacuolar invertase by detecting pectin-based signals in the cell wall (Kohorn et al., 2009).

A combination of in vitro and in vivo studies have identified a WAK1 protein complex that includes a Gly-rich-extracellular protein (AtGRP-3) and a kinase-associated protein phosphatase (KAPP; Park et al.,

2001). In plants, Gly-rich proteins are considered structural components of the cell wall (Keller, 1993); thus, in addition to binding pectin, the extracellular domain of WAK1 likely also binds AtGRP-3. The KAPP protein binds to the cytoplasmic kinase domain of multiple receptor kinases in a phosphorylation-dependent manner (Braun et al., 1997; Shah et al., 2002), and in several cases, this interaction has been demonstrated to be functionally relevant. AtGRP-3 specifically interacts with WAK1; however, KAPP binds to the kinase domains of both WAK1 and WAK2 as well as to the kinase domains of other RLKs. The expression of *WAK1* and *AtGRP-3* was up-regulated by exogenously added AtGRP-3 protein, suggesting that they are regulated by a positive feedback loop (Park et al., 2001). Although the biological significance of the WAK1/AtGRP-3 interaction has not been determined, the specificity of this interaction, together with the distinct expression patterns of the various WAK genes, suggest the possibility that the WAKs may sense different signals from the wall.

THE *CATHARANTHUS ROSEUS* RLK1-LIKE FAMILY

The *Catharanthus roseus* RLK1-Like (CrRLK1L) family is named after its founding member, CrRLK1, which was identified from the plant *C. roseus* (Schulze-Muth et al., 1996). There are 17 members of the Arabidopsis CrRLK1L subfamily of RLKs, and four of these have been implicated in regulating cell wall function: FERONIA (FER), THESEUS1 (THE1), HERCULES1 (HERK1), and HERK2 (Hematy and Höfte, 2008; Guo et al., 2009a, 2009b).

The FER RLK was identified by its role in pollen tube function (Huck et al., 2003). FER-dependent signaling in the synergid cell appears to be required for pollen tube growth arrest and the release of sperm cells in the female gametophyte during fertilization (Huck et al., 2003; Escobar-Restrepo et al., 2007). In *fer* mutant ovules, pollen tubes fail to cease growth and to rupture upon reaching the micropylar entrance of the embryo sac; instead, they continue to grow within the embryo sac, thus failing to fertilize the ovule (Escobar-Restrepo et al., 2007). More recent studies have demonstrated that *fer* mutant seedlings display a pronounced decrease in hypocotyl elongation, petiole length, and overall shoot growth when compared with wild-type seedlings, suggesting that FER also regulates cell elongation in these contexts (Guo et al., 2009a).

The THE1 RLK was identified as a suppressor of the hypocotyl elongation defect of a loss-of-function mutation in the catalytic subunit cellulose synthase 6 (*cesA6^{prc1}*). *the1* was found to also suppress the hypocotyl growth inhibition of a subset of other mutants altered in cell wall function, including *cesA3^{elil}* and *cesA^{rsw1}*, and to suppress the ectopic lignin accumulation observed in these cellulose-deficient mutants. However, surprisingly, *the1* did not suppress the

defect in cellulose biosynthesis of the *cesA6^{prc1}* mutant. These results suggest that the inhibition of hypocotyl elongation and the ectopic lignin deposition in the *cesA6^{prc1}* mutant is an active response to cell wall defects that requires signaling through the THE1 receptor. Consistent with this, transcriptional profiling identified 36 genes that were altered by *cesA6^{prc1}* in a THE1-dependent manner. The THE1-dependent genes included two transcription factors, several proteins involved in protecting the cell against oxidative stress, potential pathogen defense proteins, and multiple genes encoding cell wall proteins (Hematy et al., 2007).

Single loss-of-function mutations in THE1 in an otherwise wild-type background did not result in any detectable change in plant growth and development (Hematy et al., 2007), suggesting that THE1 function is only revealed when the cell wall is perturbed. However, recent studies have shown that THE1 is genetically redundant with other members of the CrRLK1L gene family, as combining *the1* with *herk1* and/or *herk2* mutations, single mutants that are also aphenotypic, resulted in strong effects on cell expansion, including decreased petiole length and shoot growth (Guo et al., 2009a, 2009b), similar to the effects of the *fer* mutation. The overall decreased growth in the double *the1 herk1* mutants was found to be a consequence of reduced cell elongation, implicating these RLKs as important regulators of cell expansion within the cell wall.

Interestingly, six of the 17 CrRLK1L genes are regulated by brassinosteroid, including THE1, HERK1, HERK2, and FER (Guo et al., 2009a, 2009b). Furthermore, the *herk1 the1* mutations enhanced the dwarfed phenotype of the loss-of-function brassinosteroid receptor mutant, *bri1*, and partially suppressed the excessive cell elongation phenotype of a gain-of-function *bes1-D* mutant (Guo et al., 2009a). Expression profiling of the *the1 herk1* double mutant and the *fer* single mutant suggests that these receptors regulate overlapping sets of genes. Furthermore, 16% of the genes affected in these mutants are regulated by brassinosteroid. These data suggest that while THE1, HERK1, HERK2, and FER may act in a common pathway required for cell elongation, there is cross talk between this pathway and the pathway mediating brassinosteroid-regulated cell elongation.

The reduced cell expansion observed in the *the1/herk* multiple mutants at first seems at odds with the increased cell expansion brought about by the *the1* mutation in the *cesA6^{prc1}* background. One simple model to resolve this apparent discrepancy invokes a threshold mechanism: the reduction in THE1/HERK signaling resulting from single *the1* mutations is enough to disrupt the feedback system involved in perception of the altered cell wall function of the *cesA6^{prc1}* mutant, but it is not drastic enough to substantially alter basal cell wall synthesis. In contrast, further disruption of this class of receptors (i.e. the *the/herk* multiple mutants) decreases signaling below a threshold necessary for proper regulation of cell wall

synthesis even in basal conditions. The cell must maintain a delicate and dynamic balance between wall rigidity and extensibility during growth; thus, perturbing this proposed feedback system to different levels could shift this balance with distinct outcomes.

THE LEU-RICH REPEAT RLKS

The leucine-rich repeat (LRR) RLK family represents the largest group of RLKs encoded by higher plant genomes. The Arabidopsis LRR-RLK family is composed of 216 genes distributed among 13 different subfamilies (Shiu and Bleeker, 2001). In animals, LRR proteins are important signaling components of many developmental and host defense pathways. However, unlike in plants, animal LRR proteins do not contain a cytoplasmic protein kinase domain but instead transduce signals across the plasma membrane by activating coreceptors, a mechanism that may be conserved in plants.

Recently, two LRR-RLKs (FEI1 and FEI2) were demonstrated to play a role in the regulation of cell wall function. Although single *fei1* and *fei2* mutants showed no obvious phenotypes, double *fei1 fei2* mutants displayed conditional root anisotropic growth and ectopic lignin deposition. These phenotypes are characteristic of cellulose deficiency; indeed, *fei1 fei2* mutant roots displayed a significant decrease in the synthesis of cellulose and possibly other cell wall polymers when grown in nonpermissive conditions, suggesting that the FEI receptors regulate the synthesis of cell wall components (Xu et al., 2008). The *sos5* mutant, which was isolated as a mutant that displayed a swollen root tip in the presence of moderately high salt (Shi et al., 2003), was found to have a similar phenotype to *fei1 fei2*. Genetic analysis revealed that *SOS5* and the FEIs act through the same pathway to regulate cell wall function (Xu et al., 2008). *SOS5* encodes a cell surface glycosylphosphatidylinositol-anchored protein with fasciclin-like domains (Shi et al., 2003) and could act as, or may be involved in the production or presentation of, a FEI ligand.

Both the *fei1 fei2* and *sos5* mutants display swollen root phenotypes only when elevated levels of Suc or salt are present in the medium, which is also observed in several other root-swelling mutants, including *cesA6^{prc1}*, weak alleles of *cobra* (Xu et al., 2008), and *pom1* and *pom2* mutants (Hauser et al., 1995). This suggests that elevated levels of Suc or salt sensitize roots to perturbations in cell wall synthesis through an as yet unknown mechanism. It has been suggested that the Suc-dependent phenotype of *cobra* and several other root-swelling mutants could be linked to the relative rate of root growth, with defects occurring only under conditions of maximal growth rates (Hauser et al., 1995). However, *fei1 fei2* (and *sos5* and weak *cobra* alleles) also display swollen roots on medium containing moderately elevated levels of NaCl (Xu et al.,

2008), a condition that decreases the rate of root growth.

Further analysis indicated a role for 1-aminocyclopropane-1-carboxylate (ACC) synthase, which catalyzes the rate-limiting step in ethylene biosynthesis, in FEI function. Both FEI1 and FEI2 directly interact with ACC synthase, as shown by yeast two-hybrid assays. Furthermore, inhibition of ACC function using either α -aminoisobutyric acid (AIB; a structural analog of ACC) or aminooxy-acetic acid (inhibits ACC synthase) reverted the root-swelling phenotype of both the *fei1 fei2* and the *sos5* mutants. As aminooxy-acetic acid and AIB block ethylene biosynthesis by distinct mechanisms, it is unlikely that this phenotypic reversion of *fei1 fei2* is due to off-target effects of the inhibitors. Furthermore, this is not a general effect of AIB, as it did not revert the root-swelling phenotype of the *cobra* mutant (Xu et al., 2008). Surprisingly, inhibition of ethylene perception via mutations or chemical inhibitors had no appreciable effect on the root phenotype of *fei1 fei2* or *sos5-2* mutants (Xu et al., 2008). This suggests that either swelling in the absence of FEI depends on a hitherto undiscovered pathway for ethylene perception or that ACC itself is acting as a signaling molecule. Consistent with this hypothesis, recent data indicate that, in addition to acting as the immediate precursor to ethylene, ACC itself may also act as an essential regulator of plant growth and development (Tsuchisaka et al., 2009). Genetic disruption of all eight ACC synthase genes in Arabidopsis caused embryonic lethality, in contrast to mutations that eliminate ethylene perception, such as *etr1* and *ein2*, which have only relatively modest effects on plant development. The precise role of ACC in plant development in general and in the FEI pathway specifically, and how this potential signaling molecule is perceived, are important questions that need to be addressed.

CONCLUSION

While the identification of multiple RLKs that likely play a role in regulating cell wall function is an important beginning in our understanding of cell wall signaling, the field is only in its infancy and many questions remain unanswered. There are over 600 RLKs in Arabidopsis, and it is likely that additional RLKs play a role in regulating wall function. To further enhance our understanding of this signaling system, it is crucial to identify the immediate targets of the RLKs implicated in cell wall function. One potential target could be the cellulose synthase enzyme itself, as multiple phosphorylation sites have been identified, clustered primarily in the N-terminal domain of several CESA proteins (Nuhse et al., 2004; Brown et al., 2005; Persson et al., 2007), and phosphorylation of CESA7 has been linked to its degradation via a 26S proteasome-dependent pathway (Taylor, 2007). However, an intact kinase catalytic domain is not required for the function of FEI1/FEI2 (Xu et al.,

2008), raising the possibility that, at least for this class of RLKs, the targets may not be regulated solely by phosphorylation. How these RLKs interact with each other and with other signaling pathways to regulate cell wall function is unknown. Finally, while pectin has been identified as a possible ligand for the WAKs, there are no clear candidate ligands for the other RLKs. The near future will likely reveal answers to these and other questions, and perhaps an integrated model describing the mechanisms by which cell walls perceive and respond to signals will emerge.

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