Plant Physiol. (1993) 102: 1–6

Update on Cell Walls

How Do Plant Cell Walls Extend?[1]

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Plant cells are constrained by a tough, yet flexible, polymeric wall that determines cell shape, permits high turgor pressures to develop, and confers important mechanical advantages. However, these walls present a special problem to growing cells, which must expand and deform this "exoskeleton" to enlarge and, yet, at the same time keep the wall strong enough to withstand the large mechanical stresses that arise from cell turgor pressure. These stresses may exceed 10^8 N m^{-2} (1000 atm) because the expansive forces generated by turgor are focused on the thin cell wall. Because of this mechanical situation, plant cells cannot simply deposit more material to the wall to make it extend. Rather, the polymeric network that confines the cell must shear (slip) to create new surface area while still maintaining sufficient structural integrity to resist large tensile forces. This seems like a perfect recipe for disaster: local wall expansion would cause local wall thinning and, consequently, further weakening and expansion, leading to an aneurysm or a blowout of the wall. The fact that this rarely happens implies a built-in braking action in the mechanism of wall surface expansion.

Current models of the walls of vascular plants show three interwoven polymeric networks: a network of cellulose microfibrils linked together by matrix polysaccharides, a gelled network of pectins ionically linked by calcium bridges, and a network of structural proteins covalently cross-linked to one another and perhaps to other elements in the wall matrix (Talbott and Ray, 1992a; Carpita and Gibeaut, 1993). In muro, these networks probably interact with one another in many ways and may not be as separable as implied in this description. At least one of these networks must bear the mechanical stresses in the wall, yet surprisingly little is known about the distribution of stresses among these wall components in growing cells. Biophysical and biochemical analyses of cell walls point to the matrix as being most significant for governing the growth properties of walls. Recent work on cells with modified walls has shown that walls can be modified to a remarkable degree and still maintain structural integrity (Shedletzky et al., 1992). Evidently, plant cells can adapt to a wide range of wall structures and still function well enough to survive and grow slowly in cultures. Such adaptability and developmental plasticity suggest that plant cells possess more than one mechanism for extending their walls.

This article briefly summarizes recent work that identifies the biophysical and biochemical processes that give rise to the extension of plant cell walls. I begin with the biophysical notion of stress relaxation of the wall and follow with recent studies of wall enzymes thought to catalyze wall extension and relaxation. Readers should refer to detailed reviews for more comprehensive discussion of earlier literature (Taiz, 1984; Carpita and Gibeaut, 1993; Cosgrove, 1993).

STRESS RELAXATION LEADS TO WATER UPTAKE AND WALL EXPANSION

Because the wall surrounds the protoplast, the wall cannot expand unless the protoplast increases in volume, and the protoplast cannot enlarge without expansion of the wall. This may sound like the proverbial chicken-and-egg problem (which came first?), but a closer scrutiny resolves this conundrum and provides deeper insight into the biophysical nature of plant cell enlargement.

As a cell absorbs water, the wall extends passively, and polymers in the load-bearing network(s) are distended. In nongrowing cells, wall stress increases as the polymers are stretched like springs. Elastic energy is stored in the strained bonds of these polymers (and also in the increased order of the polymers), and this elastic energy does work on the cell protoplast by compressing it, thereby increasing its turgor pressure and water potential. When the cell water potential increases to the point where it matches that of the external water, net water uptake ceases. In growing cells, this equilibrium is never quite reached because the wall "relaxes," which means that the load-bearing network breaks, slips, or is cut, and the distended polymers assume a more relaxed condition. Elastic energy of the wall is lost as heat, and a turgor reduction inevitably accompanies the reduced wall stress. Note, however, that this relaxation by itself does not entail a physical expansion of the wall or a change in cell volume.[2] Turgor decreases because the wall simply stops compressing the protoplast. Expansion follows secondarily, as the cell absorbs water in response to the reduced water potential created by the reduction of turgor pressure. This process is illustrated in a stepwise fashion in Figure 1. In reality, both relaxation and water uptake occur simultaneously in a cell growing at a steady rate so that wall stress and turgor remain steady.

The validity of this view of cell enlargement has been tested by studies of wall relaxation in growing cells. To

[1] Supported by the National Science Foundation and the U.S. Department of Energy.

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[2] This is true to a very good approximation because water is nearly incompressible. A 1-bar reduction in turgor pressure should be accompanied by an expansion of only 1 part in 10^5.

Abbreviation: XET, xyloglucan endotransglycosylase.

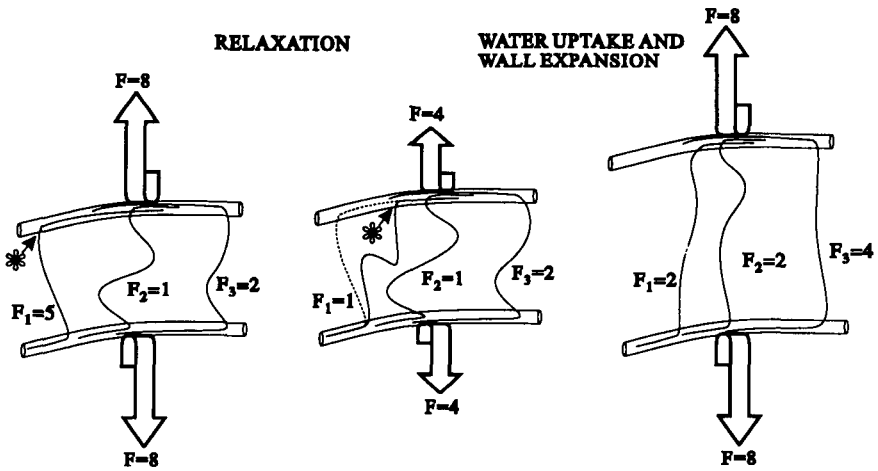

Figure 1. Model of wall stress relaxation as the underlying basis for wall expansion and water uptake by growing cells. Portions of two cellulose microfibrils are shown tethered together by three xyloglucans that are under tensile stress due to turgor. The total force on the wall in the axial direction (F = 8) is made up of the individual forces carried by each tether. Stress relaxation is shown here as resulting from a disruption of the hydrogen bonding between the xyloglucan and the surface of the microfibril (asterisk with arrow). Other means of stress relaxation are also possible, such as xyloglucan scission, with or without transfer to another polymer. In any of these cases, the polymer relaxes into a state of reduced tension (middle diagram). Note that the force on the wall has decreased because one of the tethers has come partially unglued, but the wall has not expanded. Surface expansion occurs secondarily as the cell takes up water and stretches the wall, resulting in a restoration of wall stress (right diagram). This figure is intended to illustrate the biophysical nature of wall relaxation; the biochemical basis is still not determined for any plant cell—it might be due to slippage as illustrated here or hydrolysis of matrix polymers, transglycosylation, or some other novel mechanism. Also, this diagram separates wall extension into a causal sequence for purposes of discussion. In fact, during steady growth both relaxation and extension occur simultaneously and in a balanced fashion, so that wall stress does not jog up and down but remains at a steady value determined by how readily water is taken up by the growing cell.

measure relaxation, cell size is held constant without interfering with the biochemical processes that give rise to relaxation. This condition may be met most easily by excising the growing tissue from the rest of the plant and holding it in a humid chamber to inhibit evaporative water loss. Without an external water source (usually supplied via the xylem or the phloem), the cells do not enlarge. However, such cells lose turgor pressure as wall relaxation proceeds. This turgor loss has been measured directly with the cell pressure probe and indirectly with pressure chamber or psychrometric methods (Cosgrove, 1987). It is also possible to measure wall relaxation in intact plants by sealing the growing tissue into a pressure chamber and applying the minimum air pressure required to block cell extension. This is called the pressure-block method. As the walls relax, greater pressure must be applied to block the cells from taking up water and extending.

These relaxation methods confirmed experimentally that cell enlargement is initiated by stress relaxation of the wall. It is also possible that cell enlargement could be initiated by an influx of solutes, which would decrease the cell water potential and cause water uptake. However, in such a case cell turgor and wall stress would not decrease in a relaxation experiment but would stay constant or even increase. We and others found that turgor pressure in growing tissues decays quickly at first and then more slowly as the wall stress relaxes (see review by Cosgrove, 1993). This result does not necessarily imply that the biochemical processes causing relaxation are inhibited as wall stress decreases; instead, each biochemical reaction could diminish wall stress progressively less because the stress borne by each polymer, on average, is progressively reduced during wall relaxation. It may also be that the biochemical reactions underlying relaxation require the wall polymers to be in a strained condition, but this possibility has not yet been tested adequately.

Relaxation behavior consonant with these theoretical expectations was found in the growing stems of pea mutants (dwarfs) that were deficient in GA biosynthesis or were unresponsive to applied GA (Behringer et al., 1990). Plants with wild-type (tall) growth showed fast and large relaxations, whereas dwarf lines exhibited slower and smaller total relaxations. Nongrowing tissues do not exhibit wall relaxation (Cosgrove, 1987). The relaxation properties of plant tissues have been found in numerous studies to correspond quite closely to their growth properties. For assessment of the wall growth properties of plants, I believe that relaxation assays of living tissues have proved superior to other methods (Cosgrove, 1993).

Despite recent advances in measuring wall relaxation, the underlying biochemistry of this crucial process remains, in my view, largely speculative. The literature concerning cell wall biochemistry does not lack potential candidates for this process, but it does lack adequate tests that these candidates can indeed catalyze the type of wall relaxation that initiates and maintains cell enlargement. At the heart of this issue are questions about the meaning of "wall loosening" and about

the relation between wall relaxation and wall viscoelastic properties.

IS WALL STRESS RELAXATION A SIMPLE VISCOELASTIC PROCESS?

The polymeric nature of the plant cell wall confers on it certain viscoelastic properties. Viscoelasticity refers to the mechanical properties of materials that exhibit viscous and retarded elastic deformations in response to stress. Wall viscoelasticity is usually measured by applying a force to a specimen and measuring the resulting extension or by extending the wall and measuring the resulting force (Cosgrove, 1993). Viscoelasticity does not include deformations that are mediated strictly by biochemical reactions within the material. An example of a biochemically mediated extension is the sliding of actin along myosin fibrils, where ATP hydrolysis generates the mechanical force for the viscoelastic motion of the polymers and controls the rate of such sliding. In plant walls, the mechanical force needed for viscoelastic extension originates from cell turgor, and wall extension undoubtedly entails a passive viscoelastic slippage of wall polymers. However, it does not necessarily follow that the critical relaxation is controlled by wall viscoelasticity. When relaxation is initiated by biochemical cleavage of a load-bearing cross-link between two polymers, the extension is termed a chemorheological process.

Although the distinction between a viscoelastic extension and a chemorheological extension might seem a fine one, it is important because many of the physical tests for "wall extensibility" and wall loosening actually measure wall viscoelasticity (see review by Cosgrove, 1993). If cell wall expansion were mediated by a chemorheological process, there might be little correlation between the viscoelastic properties of the wall and its growth behavior. For example, a transglycosylase might cleave a load-bearing glucan and rejoin one of the free ends to another glucan. This would permit a type of chemorheological extension in which there was no net change in the number of wall cross-links after extension and, thus, no change in wall viscoelasticity. Several studies have documented examples where wall viscoelasticity seemed unrelated to growth behavior or wall relaxation behavior (see review by Cosgrove, 1993). There are also studies that show a correlation between wall viscoelasticity and growth, but the significance of the altered wall viscoelasticity for the alteration in growth is difficult to assess. Despite frequent assertions that growth depends on wall viscoelasticity, I think the facts argue otherwise in many cases. A correct statement would be that growth depends on wall relaxation processes that may or may not be viscoelastic in nature.

DO WALL-LOOSENING ENZYMES CATALYZE WALL RELAXATION?

A favored hypothesis, still largely unproven, is that wall-loosening enzymes modify the wall to allow turgor-driven extension. The term wall loosening deserves some comment because it has been used by authors to mean various things. In one sense it denotes a mechanical weakening of the wall as measured by viscoelastic (mechanical) assays. A recent example of such usage is that by Hoson and Masuda (1992), in which mechanical weakening of isolated walls was detected with a tensile tester. A second meaning is more biochemical and denotes any cleavage of wall structural polymers. The inference is that such action weakens the wall mechanically or induces wall relaxation. On this basis, XET has been termed a wall-loosening enzyme (Fry et al., 1992; Nishitani and Tominaga, 1992), despite lack of evidence that it either alters wall viscoelasticity or causes wall stress relaxation. Finally, wall loosening is used in the broadest sense to denote any action that causes wall relaxation and extension, regardless of its mechanical and biochemical basis (Taiz, 1984; Rayle and Cleland, 1992; Cosgrove, 1993). Wall loosening in this sense could occur without viscoelastic weakening of the wall or hydrolysis of wall polymers.

If growth were a simple matter of breaking up the wall matrix to reduce its viscosity and thereby permit viscous polymer flow, these three meanings of wall loosening would be consistent with one another. Numerous results make this simple view doubtful. Hoson and Masuda (1992) found that polysaccharide synthesis inhibitors reduced growth of rice coleoptiles without significant effects on wall mechanical properties (measured by tensile tester). Because the inhibitors slowed wall expansion, they must have slowed wall relaxation and inhibited wall loosening (in the growth sense) but without a detectable change in wall mechanical properties.

An important issue is the relationship between wall synthesis and wall expansion. Polymer deposition, without wall relaxation, is insufficient to cause surface expansion in cells that have significant turgor. Wall synthesis without relaxation would only cause wall thickening without inducing the water uptake needed for wall extension and cell volume enlargement. Wall synthesis without growth occurs during secondary wall formation of maturing cells. For synthesis to induce wall relaxation so that the cell could absorb water, the new polymers would have to disrupt bonding in the load-bearing networks of the wall, either by direct chemical displacement of bonds or by the agency of an enzyme. It seems likely that newly synthesized polymers can be bound to the wall in such a way that they eventually become part of the load-bearing network (Taiz, 1984; Edelmann and Fry, 1992). However, at this time there is no good evidence that wall deposition, per se, can induce wall relaxation in any plant system (for further discussion of this point, see Taiz, 1984). Thus, most attention has been given to enzymes known to cleave matrix polymers.

For many years, glucanases have been thought of as wall-loosening enzymes because of evidence of breakdown of wall matrix polysaccharides and of changes in wall viscoelasticity after auxin treatment and during normal development (see review by Carpita and Gibeaut, 1993). This idea has been further supported in recent years by studies in which selective reagents were used to interfere with wall glycanase activity. Treatment of corn coleoptile segments with polyclonal antibodies against cell wall glycanases interfered with auxin-induced growth, wall autolysis, and changes in wall viscoelasticity (Inouhe and Nevins, 1991). In a similar vein, antibodies and lectins that recognize xyloglucans in azuku bean (Hoson and Masuda, 1991) or (1→3, 1→4)-β-D-glucans in maize coleoptiles (Hoson et al., 1992) interfered with auxin-induced growth in excised sections. These results were

interpreted to mean that auxin-induced growth requires hydrolytic breakdown of matrix polysaccharides that bind cellulose microfibrils.

In several recent papers the authors have drawn attention to XET (also called endoxyloglucan transferase and xyloglucan recombinase by Nishitani and Tominaga, 1992) as a wall-loosening enzyme. Smith and Fry (1991) found that xyloglucan chains in vivo could be cleaved and transferred to other xyloglucans in the wall. Fry et al. (1992) obtained cell-free extracts containing this activity from a wide range of species (bryophytes, monocots, and dicots) and determined that the enzymic activity was highly specific for xyloglucan. Nishitani and Tominaga (1992) isolated what appears to be the identical enzyme from *Vigna* epicotyls: it is a glycoprotein of 33 kD and requires xyloglucan as both acceptor and donor. Fanutti et al. (1993) recently discovered XET activity by an enzyme previously identified as an endo-(1→4)-β-D-glucanase from the cotyledons of germinating nasturtium seeds (which solubilize a large stock of storage xyloglucan during germination).

When a cDNA clone encoding this enzyme was isolated and sequenced (de Silva et al., 1993), it proved to lack sequence similarity with other known endo-(1→4)-β-D-glucanases ("cellulases"), but it shared 52% sequence similarity at the amino acid level with *meri-5*. *meri-5* is a gene of unknown function that is expressed in the shoot apical meristem and other tissues of *Arabidopsis* (Medford et al., 1991). de Silva et al. (1993) suggested that *meri-5* may be an XET involved in cell expansion; however, *meri-5* is not expressed in rapidly expanding leaves or in the elongation zone of the stem (Medford et al., 1991). de Silva et al. (1993) identified a 33.5-kD precursor of XET with an N-terminal signal sequence and a mature, unglycosylated protein of 31 kD. By immunolocalization, this protein appeared to be concentrated in the walls of the germinating cotyledon (J. de Silva, personal communication). The sequence and the enzymic properties of the nasturtium enzyme are similar to those of the *Vigna* enzyme (K. Nishitani, personal communication). The nasturtium enzyme apparently acts as a hydrolase when substrate concentration is low and it acts as an endotransglycosylase at higher xyloglucan concentrations (Fanutti et al., 1993), whereas this hydrolytic activity is apparently lacking in the enzymes obtained from growing tissues (Fry et al., 1992; Nishitani and Tominaga, 1992). This difference may relate to the functions of the enzymes in their native tissues.

The notion that XET activity causes wall extension is attractive as a biochemical theory but is still speculative in terms of physiological and biophysical evidence. Fry et al. (1992) reported that XET activity was highest in the third internode of 7-d-old etiolated pea seedlings. They took these data as a positive correlation with growth (not measured). However, I interpret their data as circumstantial evidence against a direct role in cell elongation because the activity peaks at a point on the epicotyl where growth rate should be trailing off. Moreover, XET activity is still quite high in the region below the elongation zone of the epicotyl. Talbott and Ray (1992b) observed large changes in xyloglucan size when pea segments were treated with auxin or were kept under conditions that would induce stress relaxation. It is plausible that these size changes were the result of XET activity. Unfortunately, the effects of such XET activity on either wall viscoelasticity or wall relaxation properties were not tested. McQueen-Mason et al. (1993) applied a crude extract containing high XET activity to isolated cucumber walls under tension and found that it failed to cause wall extension, a result at odds with the putative role of XET as a wall-loosening enzyme. It could be that the enzyme could not access the load-bearing bonds of the wall under these reconstitution conditions and, therefore, had no effect. However, other proteins of sizes similar to XET, but without XET activity, were able to induce wall extension under these conditions (McQueen-Mason et al., 1993). Perhaps XET serves other functions in vivo, such as anchoring of newly deposited xyloglucan into the wall (Edelmann and Fry, 1992) or elongation (or shortening) of xyloglucan chains or control of wall porosity.

Studies of extension, or "creep," of isolated walls from growing tissues afford an opportunity to study wall extension under conditions that avoid the complexities of living cells (e.g. wall synthesis, wall acidification, turgor changes, and so on). The discovery that isolated walls extend under acidic conditions is one piece of evidence in support of the acid-growth hypothesis, which proposes that low pH activates undefined wall-loosening processes (Rayle and Cleland, 1992). Acid-induced wall extension appears to require the activity of wall proteins (Cosgrove, 1989). Although some wall hydrolases exhibit a pH dependence compatible with the acid-growth hypothesis, there are no reports that wall hydrolases or transferases can induce extension of isolated walls. I think this is a crucial test of the thesis that an enzyme possesses wall-loosening activity. It is important to note that walls may be weakened, in the viscoelastic sense, by enzymic or chemical treatments without enabling the wall to undergo sustained extension (Cosgrove, 1989, 1993).

Recently, McQueen-Mason et al. (1992) reported that they could reconstitute wall extension activity in cucumber hypocotyl walls by application of crude protein fractions extracted from the walls of growing hypocotyls. This extractable activity was present in growing tissues but was lacking in non-growing tissues, a result that suggests a developmental significance to this activity. Fractionation of the extracts revealed two active proteins of 29 and 30 kD. Each protein by itself was competent to induce extension in heat-inactivated walls, and these proteins were effective when tested on walls from various dicots and monocots. The evidence suggests that these proteins are responsible for the acid-growth responses of isolated walls. We have named these proteins expansins (extensin would have been a perfect name but it is already used to describe a group of wall structural glycoproteins that probably are not involved in wall extension). Expansins appear to be the first endogenous wall proteins identified with the demonstrated ability to induce extension in isolated walls.

Biochemical characterization of expansins indicates that they are responsible for the acid-induced extension of isolated walls and perhaps of intact tissues, but their biochemical mode of action is still uncertain. They lack detectable glycanase activity and XET activity (McQueen-Mason et al., 1992, 1993). These and related results lead me to suggest that it is premature to ascribe wall-loosening functions to wall-degrading enzymes such as glucanases and wall-modifying

enzymes such as XET without evidence that they can cause either wall relaxation or wall expansion in vitro. As a case in point, wall degradation is thought to contribute to fruit softening, but it evidently does not lead to substantial wall expansion or cell enlargement.

SUMMARY AND PROSPECTUS

Growing plant cells rearrange the load-bearing network in their walls to reduce wall stress and cell turgor pressure, thereby enabling the cell to take up water and extend the wall. Physical and chemical evidence points to matrix polymers as the site of these wall rearrangements. Although viscoelastic slippage of wall polymers inevitably occurs during wall extension, wall viscoelasticity per se does not appear to control wall extension in many cases. Instead, wall-loosening processes seem to be important, perhaps in competition with wall-stiffening processes. Recent evidence supports a role for wall glycanases and endotransglycosylases in wall-loosening action, but a crucial piece of evidence in favor of these ideas is lacking, namely, that their activity can result in extension of isolated walls. Recent progress in reconstituting extension activity in isolated wall specimens offers a promising approach for testing the activity of putative wall-loosening and wall-stiffening enzymes.

Rapid advances have been made in recent years in the molecular description of the plant cell wall, e.g. through studies of biochemical composition and structure, cytolocalization, and analysis of genes coding for wall structural proteins and enzymes that synthesize or metabolize wall polysaccharides. Despite these steps forward, many basic questions about the functional significance of the various wall components have been given only tentative or purely speculative answers. It is clear that wall composition is modified during development, that many wall components are metabolically active, and that certain wall components can quickly change their pattern of cross-linking. A fascinating example is the rapid oxidative cross-linking of proteins in the wall upon treatment with elicitors (Bradley et al., 1992). This response is mediated by a flush of hydrogen peroxide into the wall when treated with elicitor. The significance of this cross-linking for the wall's physical, chemical, and growth properties remains unanswered, but the results of this study reinforce a view of the wall as a dynamic structure. More definitive assignments of functions will come from interdisciplinary approaches, in which plants with altered wall structure are assayed for their ability to grow, undergo wall relaxation, change form, influence developmental events, participate in biochemical responses, and respond to physical, chemical, and biological assaults. On the horizon is the development of genetic mutants with altered wall structure, created by antisense removal of specific genes, by insertion of specific genes involved in wall structure, by selection of cell cultures grown in the presence of wall biosynthetic inhibitors (Shedletzky et al., 1992), or by mutagenesis and screening for mutants deficient in specific wall components (Reiter et al., 1992). These mutants will add a potent avenue to the study of the mechanisms by which cell walls extend during growth.

Received November 2, 1992; accepted January 22, 1993.
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