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Getting physical: invasive growth events during plant development

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Plant cells are enclosed in cell walls that weld them together, meaning that cells rarely change neighbours. Nonetheless, invasive growth events play critical roles in plant development and are often key hubs for the integration of environmental and/or developmental signalling. Here we review cellular processes involved in three such events: lateral root emergence, pollen tube growth through stigma and style tissues, and embryo expansion through the endosperm (Figures 1–3). We consider processes such as regulation of water fluxes and cell turgor (driving growth), cell wall modifications (e.g. cell separation) and cell death (for creating space) within these three contexts with the aim of identifying key mechanisms implicated in providing a chemical and biophysical environments permitting invasive growth events.

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Introduction: a balance of forces

During invasive growth, one tissue (the invader) must overcome the physical resistance imposed by the 'invaded' tissue. Whether invasion occurs depends on the balance between the biophysical properties of the two tissues involved. The 'invading' force is generated by growth, whilst the biophysical properties (rigidity, cell adhesion) of 'invaded' tissues are often significantly modified (a process generally known as 'accommodation') in order to facilitate breaching. The main driving force of growth is hydrostatic pressure (turgor) developed within tissues, which drives the deformation of surrounding cell walls. Turgor likely also significantly impacts the biophysical properties (rigidity) of 'invaded' tissues, however tissue breaching is most often associated with cell wall modifications.

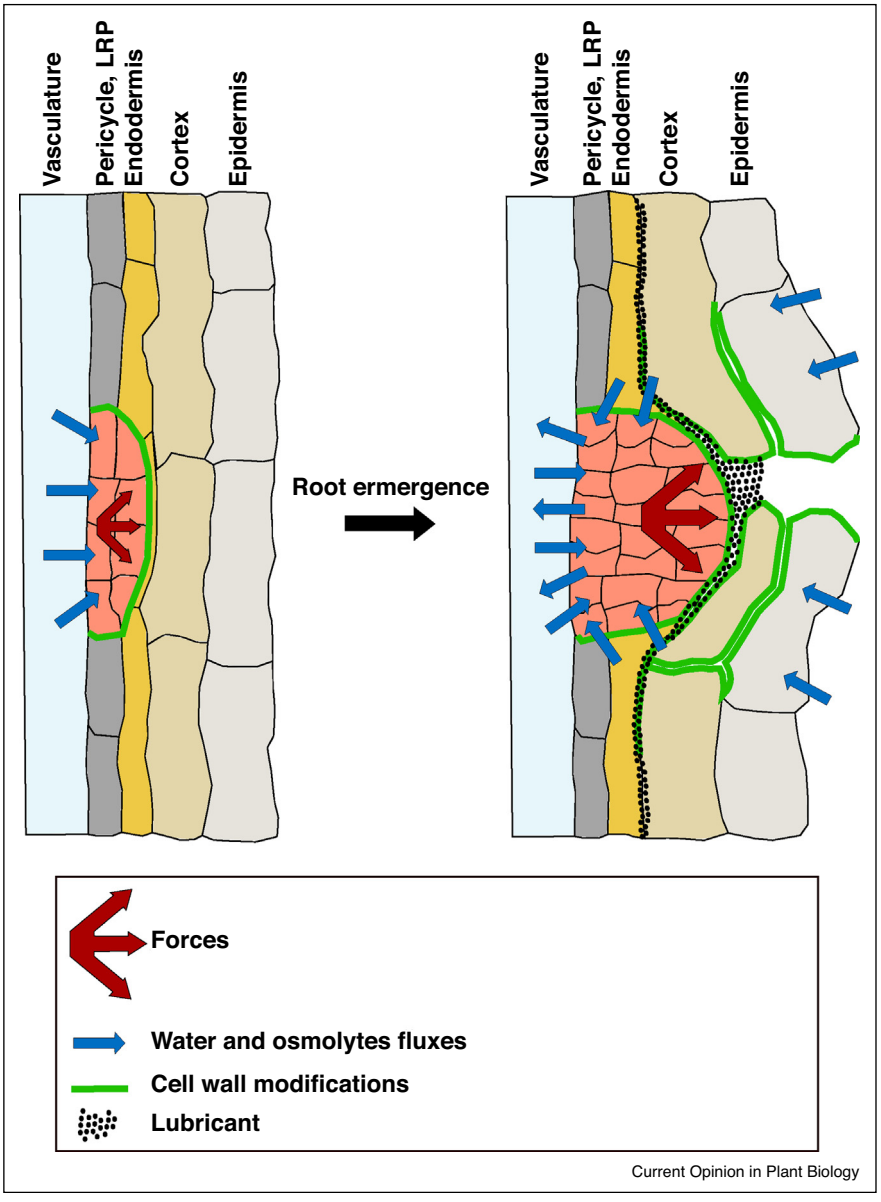
The force within: turgor drives invasive growth

Turgor is generated by the passive movement of water down gradients in water potential generated by the accumulation of osmolytes (of both organic and inorganic origin) within cells [1]. The regulation of turgor, particularly during growth, is influenced by both osmolyte accumulation, and cell and tissue-level hydraulic permeability. Global osmolyte measurements at the single cell level have not been made in any of the invasive growth systems studied. Although it is tempting to speculate that active transfer of osmolytes from the invaded to the invading tissue may be of critical importance, the mechanisms involved, and the distinction between uptake for metabolism versus turgor generation remain uninvestigated. Hydraulic permeability is dependent on the activity of water-permeable membrane-localised pores (aquaporins) [2].

Symplastic channels (plasmodesmata) may also affect conductivity but their role is poorly understood and likely highly tissue dependent [1]. Although an active regulation of plasmodesmatal aperture was reported to influence lateral root primordium (LRP) outgrowth [3], the mechanism responsible is unclear. Both the embryo and the pollen tube are symplastically isolated from surrounding tissues [4,5], and the role of plasmodesmata will therefore not be further considered here.

LRP outgrowth is dependent upon auxin localisation and signalling [6,7]. A role for auxin-mediated regulation of the expression of plasma-membrane localised aquaporins (PIPs) in LRP outgrowth has also been described [8]. More recently tonoplast-localised aquaporins (TIPs) have also been shown to facilitate LRP outgrowth [9•]. In both cases gene expression patterns are complex, and although mathematical modelling was used to predict water fluxes affected by altered PIP expression (Figure 1), and root hydraulic conductivity was shown to be affected by auxin, no measurement of turgor or water fluxes either in, or around LRP have to date been published in Arabidopsis. This is regrettable, particularly as models suggest that transfer of water from cells overlying the emerging LRP to the LRP itself could contribute to spatial accommodation that has been reported during LRP initiation and expansion [10] (Figure 1). A recent study in maize has linked hydro-patterning (positioning of lateral roots towards available water) to growth-induced gradients of water potential within the growth zone of the root [11•], again linking potential changes in turgor to LRP initiation and emergence.

Figure 1



Lateral root primordium emergence. Lateral root formation (cells in red) starts with the division of a small subset of pericycle cells (in dark grey). Anticlinal and periclinal divisions of these cells give rise to a dome-shaped lateral root primordium (LRP) which emerges through three cell layers: the endodermis (in yellow), the cortex (in brown) and the epidermis (in grey). In addition to the formation of new cells by division, LRPs grow by cell expansion driven by turgor pressure (red arrows) due to water movements (blue arrows) across cell membranes. Water movements are facilitated by aquaporins: plasma membrane intrinsic protein (PIPs) and tonoplast intrinsic protein (TIPs) whose complex gene expression patterns are regulated by auxin. In a model proposed by Peret *et al*., PIPs promote water transfer from overlying cells into the primordium and from the primordium into the stele. In a recent study, Reinhardt *et al*. proposed that TIPs promote an influx of water from the stele and surrounding tissues to the base of LRP meaning that in addition to plasma membrane permeability, the regulation of tonoplast water permeability plays an important role in early lateral root growth and development. LRP emergence also depends on the expression of cell wall modifying enzymes, and ROS production in the overlaying cells (bright green lines) to induce the separation of cortex and epidermis cells and facilitate primordium emergence. These enzymes do not seem to act on the LRP. The gap created between LRP and surrounding cells is filled with suberin-like lubricating compounds (black dots), which appear to protect LRP and enhance lateral root emergence.

Alterations in turgor are also associated with the ability of embryos to grow through endosperm tissue during seed development. After fertilization the endosperm expands rapidly, driving seed growth. Indentation-based measurements combined with physical models, validated by pressure probe studies, show a decrease in endosperm turgor pressure from around 0.15 MPa at early stages to around 0.05 MPa at endosperm cellularisation [12••]. In