**IN BRIEF**

**Sterols Modulate Cell-to-Cell Connectivity at Plasmodesmata**

Plasmodesmata (PD) connect neighboring plant cells and establish cytoplasmic and membrane continuity throughout most of the plant. Since callose at the neck region of PD regulates plasmodesmatal permeability (De Storme and Geelen, 2014), much research has focused on identifying proteins that affect callose turnover and stability at PD. For instance, the PD callose binding proteins (PDCBs), a family of glycosylphosphatidylinositol (GPI)-linked proteins, were localized to the neck region of PD and shown to regulate callose deposition and cell-to-cell transport (Simpson et al., 2009). Lipids are also thought to affect the structure and function of PD (Delage and Zurzolo, 2013), but little is known about the lipid constituents of PD.

Grison et al. (2015) analyzed the lipid composition of membranes lining primary PD using highly purified PD-derived membranes from *Arabidopsis thaliana* suspension cells. A comparative mass spectrometry-based analysis showed that the lipid composition of PD membranes differed from that of the surrounding plasma membrane, with PD membranes being enriched in sterols and complex sphingolipids with very long saturated fatty acid tails. The authors then examined whether modulating the sterol composition of cells would interfere with the targeting of two GPI-anchored proteins, PDCB1 and PdBG2 (β1-3 glucanase, a callose degrading enzyme), to young primary PD. Using drugs that inhibit sterol biosynthesis (fenpropimorph and lovastatin), the authors showed that the GPI-anchored proteins were mislocalized in the root epidermal cells of YFP-PDCB1 or mCitrine-PdBG2 transgenic seedlings when the sterol composition was altered (see figure). Furthermore, these drug treatments altered the intercellular trafficking of GFP in the roots of transgenic plants expressing GFP under the control of a phloem-specific promoter (ProSUC2:GFP). Finally, the authors used immunofluorescence microscopy to show that sterol biosynthesis disruption caused a significant increase in callose deposition in the phloem tissue. Interestingly, callose deposition was either slightly decreased or unchanged in nonvascular tissues, indicating that callose accumulation is differentially regulated in different cell types.

In sum, these results suggest that sterols play a role in targeting GPI-anchored proteins to primary PD and in modulating the permeability of PD. This work represents a breakthrough in our understanding of PD function and sets the stage for studies investigating how specialized lipid domains are established and maintained within PD and how PD lipid profiles are altered in response to various environmental and cellular conditions.

**REFERENCES**


