The interplay of apoplastic and symplastic transport in sugar loading and phloem transport

Françoise Vilaine, Rozenn Le Hir, Beate Hoffmann, Catherine Bellini, Sylvie Dinant

The interplay of apoplastic and symplastic transport in sugar loading and phloem transport

Françoise Vilaine[1], Rozenn Le Hir[1], Beate Hoffmann[1], Catherine Bellini[1,2], Sylvie Dinant [1]

[1] *Université Paris-Saclay, INRAE, AgroParisTech, Institut Jean-Pierre Bourgin (IJPB), 78000 Versailles, France*

[2] *Umeå Plant Science Centre, Department of Plant Physiology, Umeå University, 90183 Umeå, Sweden*

In source leaves, sucrose is transported from the mesophyll cells to the phloem cells of the minor veins before it enters the sieve elements for long-distance transport. In these tissues, sucrose is first effluxed to the apoplasm by sugar facilitators of the SWEET family before being transported back to the cytosol of the companion cells or the sieve elements by sugar transporters of the SUC/SUT family. In Arabidopsis thaliana, sucrose transport requires SWEET11/SWEET12 for efflux to the apoplasm of the phloem parenchyma cells and SUC2 for uptake into the companion cells (CC) or the sieve elements (SE). In some species, such as Arabidopsis thaliana, the uptake of sucrose from the apoplasm takes place in the CC. Thus, *suc2* mutants are impaired in sugar phloem transport, leading to adnormal accumulation of sugars in source organs. Then the entry of sucrose into the SE occurs by diffusion from the companion cells to the sieve elements through plasmodesmata, leading to an additional step in regulating sugar loading in the phloem. The NHL26 protein, a member of the NDR1/NHL family, is located in plasmodesmata and in the endoplasmic reticulum at the interface between SE and CC. The overexpression of *NHL26* impairs sugar loading, likely by an alteration of PD permeability at the SE/CC interface. This data suggests that both apoplasmic and symplasmic pathways are involved in regulating sugar loading in Arabidopsis. However, the relative contribution of these pathways and consequences of their regulation on plant response to the environment remains unclear.

To get more insight into the role of these pathways, we used a reverse genetic approach, and we created transgenic lines in which we specifically altered symplasmic or apoplasmic steps of sugar loading, either in the minor veins or alongside the transport phloem. We observed that ectopic deregulation of *SUC2* or *NHL26* had various consequences on plant growth, the transition from vegetative to reproductive stage, sugar transport and homeostasis, and seed filling. The expression of several genes involved in sugar transport and sugar metabolism was also impaired, although blocking apoplasmic and symplasmic pathways did not impact them the same way. These data raise new questions on the interplay between sugar transport, central metabolism, and phloem transport and how the balance between symplasmic and apoplasmic sugar loading can enhance plant tolerance to biotic and abiotic stresses plants.