THE INTERPLAY OF APOPLASTIC AND SYMPLASTIC TRANSPORT IN SUGAR LOADING AND PHLOEM TRANSPORT
Françoise Vilaine, Rozenn Le Hir, Beate Hoffmann, Catherine Bellini, Sylvie Dinant

To cite this version:
Françoise Vilaine, Rozenn Le Hir, Beate Hoffmann, Catherine Bellini, Sylvie Dinant. THE INTERPLAY OF APOPLASTIC AND SYMPLASTIC TRANSPORT IN SUGAR LOADING AND PHLOEM TRANSPORT. 13ème Congrès international de la SFBV, Aug 2022, Montpellier, France. hal-03762693

HAL Id: hal-03762693
https://hal.inrae.fr/hal-03762693
Submitted on 28 Aug 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers. L’archive ouverte pluridisciplinaire HAL, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d’enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.
In source leaves, before it enters sieve elements for long-distance transport, sucrose is transported from mesophyll cells to phloem cells of the minor veins. Sucrose is first effluxed from mesophyll cells to the apoplasm by sugar transport facilitators, then imported into the cytosol of the companion cells (CC) or the sieve elements (SE) by sugar transporters of the SUC/SUT family. In *Arabidopsis thaliana*, the uptake of sucrose from the apoplasm in the CC is mediated by SUC2. The suc2 mutants are impaired in phloem transport, which leads to an abnormal accumulation of sugars in source organs. Subsequently, the entry of sucrose into the SE occurs by diffusion from CC to SE through plasmodesmata. The NHL26 protein, a member of the NDR1/NHL family, is located in the plasmodesmata (PD) and the endoplasmic reticulum at the interface between SE and CC. Overexpression of NHL26 impairs sugar loading, likely by an alteration of PD permeability at the SE/CC interface. This suggests that both apoplastic and symplastic pathways are involved in sugar loading. But the respective contribution of each pathway remains unclear.

We used a reverse genetic approach and created transgenic lines in which we specifically altered symplastic or apoplastic steps of sugar loading, either in minor veins or alongside the transport phloem. We observed that ectopic deregulation of SUC2 or NHL26 had various consequences on plant growth, transition from vegetative to reproductive stage, sugar transport and homeostasis, and seed filling. The expression of several genes involved in sugar transport and metabolism was impaired, although blocking apoplastic and symplastic pathways did not always have the same effect. These data raise new questions about the interplay between sugar transport, central metabolism, and phloem transport and about the fine tuning between symplastic and apoplastic sugar loading especially in plant tolerance to biotic or abiotic stresses.