Sucrose-Induced Receptor Kinase 1 is modulated by an interacting kinase with short extracellular domain

Abstract

Sucrose as a product of photosynthesis is the major carbohydrate translocated from photosynthetic leaves to growing non-photosynthetic organs such as roots and seeds. These growing tissues, besides carbohydrate supply, require uptake of water through aquaporins to enhance cell expansion during growth. Previous work revealed Sucrose Induced Receptor Kinase, SIRK1, to control aquaporin activity via phosphorylation in response to external sucrose stimulation. Here, we present the regulatory role of AT3G02880 (QSK1), a receptor kinase with a short external domain, in modulation of SIRK1 activity. Our results suggest that SIRK1 autophosphorylates at Ser-744 after sucrose treatment. Autophosphorylated SIRK1 then interacts with and transphosphorylates QSK1 and QSK2. Upon interaction with QSK1, SIRK1 phosphorylates aquaporins at their regulatory C-terminal phosphorylation sites. Consequently, in root protoplast swelling assays, the qsk1qsk2 mutant showed reduced water influx rates under iso-osmotic sucrose stimulation, confirming an involvement in the same signaling pathway as the receptor kinase SIRK1. Large-scale phosphoproteomics comparing single mutant sirk1, qsk1, and double mutant sirk1 qsk1 revealed that aquaporins were regulated by phosphorylation depending on an activated receptor kinase complex of SIRK1, as well as QSK1. QSK1 thereby acts as a coreceptor stabilizing and enhancing SIRK1 activity and recruiting substrate proteins, such as aquaporins.