

SOS2 PROMOTES SALT TOLERANCE IN PART BY INTERACTING WITH THE TONOPLAST V-ATPASE AND UPREGULATING ITS TRANSPORT ACTIVITY

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To cope with salt stress conditions, plants have evolved strategies to maintain low Na⁺ concentrations in the cytoplasm. These strategies include the activation during salt stress conditions of membrane localized transporters such as SOS1 and NHX1 that extrude Na⁺ ions out of the cells or sequester them in the vacuole.

Previous studies have shown that the activity of both SOS1 and NHX1 is dependent of SOS2.

The driving force for SOS1 and NHX1, as well as many other transport activities, is the proton force motif (PMF) generated by H⁺ pumping ATPases, such as the plasma membrane H⁺-ATPase, and the tonoplast H⁺-pyrophosphatase and H⁺-ATPase. The Vacuolar H⁺-ATPase (V-ATPase, VHA) is the major proton pump that establishes and maintains an electrochemical proton-gradient across the tonoplast, thus providing the driving force for the secondary active transport of metabolites and ions.

The V-ATPase is a complex multi-subunit enzyme, composed of a V₁ peripheral stalk that binds and hydrolyses ATP and a V₀ membrane sector that provides the pathway for the entry of the protons into the vacuolar lumen. In the genome of *Arabidopsis thaliana*, genes encoding for at least 12 V-ATPase subunits have been identified.

In this study, we used Tandem Affinity Purification (TAP) tagging to isolate proteins interacting *in vivo* with the protein kinase SOS2. Using this strategy, we found that SOS2 interacts with subunits forming the cytoplasmic sector of the V-ATPase and that this interaction is enhanced under salt stress conditions. Parallel experiments using the Yeast-Two-Hybrid system showed that SOS2 interacts with at least 2 of the 3 VHA-B subunit isoforms present in *Arabidopsis*. Furthermore, we show that the V-ATPase activity is altered in both *sos2-2* and *sos3-1* mutants. The results suggest that, under salt stress conditions, SOS2 interacts with cytoplasmic VHA-B subunits thus stimulating H⁺ transport into the vacuole and providing an increased driving force for the compartmentation of Na⁺ ions in the vacuole.