Diacylglycerol transport by Arabidopsis Synaptotagmin 1 at ER-plasma membrane contact sites under abiotic stress.

OBJECTIVES: The objective of our research is to decipher the molecular mechanisms of lipid transport and lipid signalling at ER-PM contact sites by synaptotagmins under abiotic stress conditions.

RESULTS:
Bulk lipid transport between membranes within cells involves vesicles, however membrane contact sites have recently been discovered as mediators of non-vesicular lipid transfer. ER-PM contact sites are conserved structures defined as regions of the endoplasmic reticulum (ER) that tightly associate with the plasma membrane (PM). Our recent data suggest that the constitutively expressed Arabidopsis Synaptotagmin 1 (SYT1) and the cold-induced homolog AtSYT3 are proteins located in these ER-PM contact sites that are essential for the tolerance various abiotic stresses. Arabidopsis SYTs proteins are integral membrane proteins that contain multiple Ca2+-binding C2 domains and a synaptotagmin-like mitochondrial lipid-binding protein (SMP) domain that contains a hydrophobic groove. In mammals, several SMP proteins are responsible for the inter-organelle transport of glycerophospholipids. Our experiments have demonstrated that there is a recruitment of AtSYT1 and AtSTYT3 to ER-PM contact sites under stress conditions and it requires phosphatidylinositol 4-phosphate, PI(4)P in the PM, in opposition to the recruitment of PI(4,5)P2 in mammals. Moreover, our recent high-resolution lipidome analysis suggest that saturated diacylglycerols (DAGs) are the lipids that AtSYT1 is transferring between the PM and ER. Additionally, we have identified AtDGK2 (diacylglycerol kinase 2) as a key interactor of AtSYT1. Generally, in response to a stress stimulus, a phospholipase C (PLC), hydrolyses PIP2 after the elevation of cytosolic Ca2+, generating DAGs which immediately can be converted to phosphatidic acid (PA) by DGKs.

CONCLUSIONS:
In summary, our recent studies suggest that in response to abiotic stress which result in the production of DAGs in the PM (e.g. hydrolysis of PM PI(4,5)P2 by phospholipase C) AtSYT1 and AtSYT3 contribute to the lipid homeostasis of the PM by reversing the accumulation of saturated DAGs in the PM and transferring them to the ER for metabolic recycling and by regulating the lipid signalling PA molecules generated from DAGs at the PM by AtDGK2.

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