XIV REUNIÓN DE BIOLOGÍA MOLECULAR DE PLANTAS

SALAMANCA, 4-6 de Julio 2018

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Session: Abiotic stress
Communication (Oral or Poster): Oral

Arabidopsis Synaptotagmins 1 and 3 are involved in lipid homeostasis at ER-PM contact sites under cold stress.

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As sessile organisms, plants must cope with abiotic stress such as soil salinity, drought, and extreme temperatures. This stress signal can activate a phospholipase C (PLC), which hydrolyses PIP_2 to generate IP_3 and diacylglycerol (DAG). 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 SYT3 are proteins located in these ER-PM contact sites that are essential for freezing tolerance. Additionally, like mammalian Extended Synaptotagmins, membrane tethering is mediated by C2-domains which interact with acidic phospholipids (enhanced by Ca^{2+}). Our experiments of depletion of PM PI(4)P triggers loss of SYT1 and SYT3 at ER-PM CS. Moreover, our analysis in SYT1 and SYT3 proteins predicted a SMP domain like the recently crystalized E-SYT2 which exhibits a hydrophobic groove capable of harbouring phospholipids, suggesting that SYT1 and SYT3 mediate lipid exchange between the ER and the PM. This idea is supported by the over-accumulation of saturated DAG found in SYT1 after a high-resolution lipidome analysis. Additionally, we have identified DGK2 (diacylglycerol

kinase 2) as an interactor of SYT1. In summary, our recent studies suggest that SYT1 and SYT3 are ER-PM tether components responsible for the elimination of excess DAG from the PM after its acute generation by PLC in cold conditions.

The authors acknowledge the support by the Plan Propio from University of Malaga, Campus de Excelencia Internacional de Andalucía and by the Redes of Excelencia (BIO2014-56153-REDT) and BIO2017-82609-R & BIO2014-55380-R of the Ministerio de Economía, Industria y Competitividad.