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(G*) (POS-53) Protective Effect of Trehalose Sugar Against Amyloid β Toxicity in Model Lipid Membranes

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The amyloid-β peptide (Aβ1-42) is regarded as a major pathogenic factor in Alzheimer's disease—a debilitating neurodegenerative disorder that causes memory loss and neuronal damage in elderly patients. Aβ forms toxic oligomers capable of binding to neuronal membranes and inducing damage through their insertion and subsequent formation of ion channels or pores in these membranes (Drolle et al, J. Drug Metabolism Research, 2014).
Trehalose, a disaccharide, has been shown to protect plant cellular membranes in extreme conditions and modify protein misfolding processes, including those seen in Aβ. We hypothesize that trehalose can protect the neuronal membrane from amyloid toxicity. In this work we studied the protective effect of trehalose against Aβ-induced damage in model lipid membranes (DPPC/POPC/Cholesterol in mass ratio of 4:4:2), which are used to mimic neuronal membranes. We used Black Lipid Membrane (BLM) technique to detect the changes in membrane permeability by measuring ion currents through suspended model lipid bilayers upon addition of Aβ and trehalose. Atomic Force Microscopy (AFM) was used to visualize the 3D surface topography of model lipid membranes as well as the changes under the incubation of Aβ and trehalose by measuring the atomic force between sample surface and scanning probes. Our results demonstrate that Aβ binds to membranes and leads to ionic current leakage across membrane due to channel incorporation and membrane damage. The presence of trehalose reduces the ion current caused by Aβ peptides' destructive insertion to lipid membranes. This may indicate potential protective effects of trehalose against Aβ toxicity in model membranes. AFM images are in a good agreement with BLM data and support the protective hypothesis of trehalose in model membranes.

Author: XU, Yue

Co-authors: FILICE, Carina; Prof. LEONENKO, Zoya

Presenter: XU, Yue

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