Bilayers at the ILL

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Interaction between tethered bilayer membranes and misfolded proteins

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A central event in pathogenesis of Alzhaimer's diseases are thought to be intracellular and extracellular accumulation, aggregation and misfolding of low molecular mass peptides such as β-amyloid (Aβ1-42), tau protein (Tau) and s100A9 [1,2, 6]. Small size aggregates-oligomers were found to be extremely neurotoxic in vitro and in vivo with the ability to disrupt the major neuron membranes [3,4] and lead to synaptic dysfunction, mithochondrial dysfunction, neuronal apoptosis and brain damage [5].

In this work different sizes of soluble recombinant s100A9 aggregates were used to investigate their interaction with tethered phospholipid membranes (tBLM). The morphology and size of misfolded protein aggregates were monitored by dynamic light scattering (DLS) and atomic force microscopy (AFM). These protein aggregates exhibited the different membrane damaging properties as probed by the electrochemical impedance spectroscopy (EIS). The function and morphology of s100A9 aggregates were depending on different oligomerisation conditions: temperature and time. The interaction between s100A9 and tBLM was monitored by EIS time series measurements. The observed lag phase of this interaction were significantly decreased at s100A9 aggregates concentration level. Membrane composition was found to be one of the important factors affecting the interaction of the s100A9 oligomers to phospholipid membranes.

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