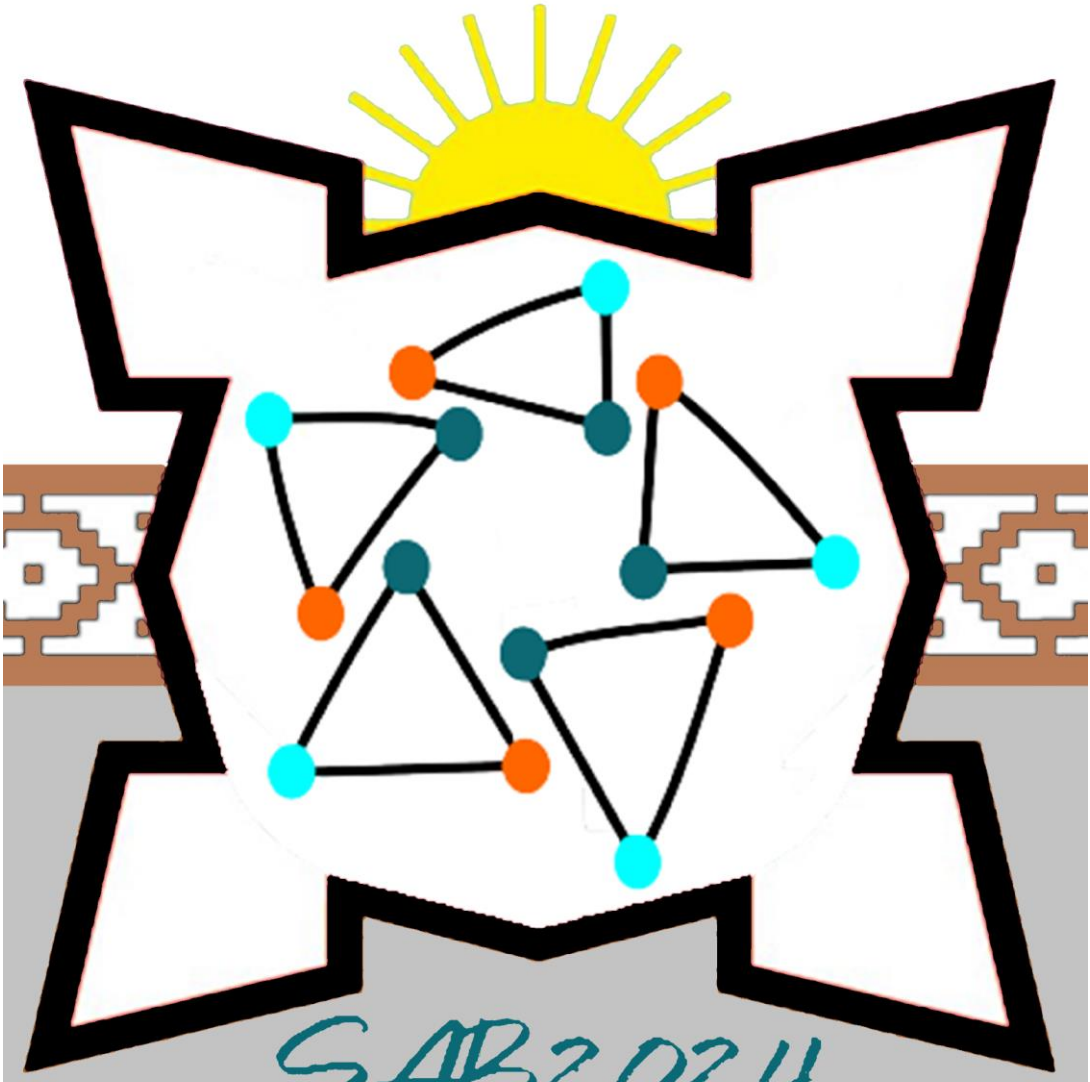
Sociedad Argentina de Biofísica

LII Reunión Anual

27 al 29 de noviembre



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Bahía Blanca, Argentina

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Bahía Blanca, Argentina

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Key Drivers of Beta-Amyloid Peptide Aggregation: Cholesterol Content and Membrane Organization

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Cholesterol (Chol) is an essential lipid molecule critical for both the development and proper function of the nervous system. The adult brain contains over 20% of the body's total Chol. In contrast to the distribution found in most mammalian cell membranes, approximately 85% of the Chol in synaptosomal membranes is located in the inner hemilayer, optimizing membrane rheology and supporting neuronal function. However, significant changes occur with aging, resulting in an increased proportion of Chol in the outer hemilayer. Conflicting hypotheses exist regarding how these changes affect β-amyloid (Aβ) aggregation, a hallmark of the Alzheimer's disease, and the precise mechanisms, particularly at low Aβ concentrations, remain unclear. To gain deeper insight into this process, lipid model membranes (large and giant unilamellar vesicles, LUVs and GUVs, respectively) composed of different lipids and Chol levels were employed. Several experimental techniques, including fluorescence spectroscopy, fluorescence microscopy, transmission electron microscopy, and dot blot were used to explore how the Chol membrane content and the resulting membrane biophysical status of the coexisting liquid-ordered (Lo) and liquid-disordered (Ld) phases impact Aβ aggregation. A direct correlation between higher Chol membrane levels and an increase in Aβ aggregation over time was observed. In the presence of Aβ, high Chol levels were correlated with a marked increase in the membrane transition temperature (Tt) over time. By maintaining the same Chol level but changing other lipids, it was possible to conclude that, although Chol is essential for the initial stages of Aβ oligomerization, the biophysical state of the Ld-Lo coexistence is ultimately responsible for Aβ fibrillation. These findings may offer critical insights into the early molecular events of Alzheimer's disease, potentially advancing the understanding of its pathogenesis and opening new avenues for therapeutic intervention.

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