Contribution ID: **52** Type: **Poster**

Effect of Amyloid-β(1-42)-Monomer and protofibrils on dynamics of brain phospholipid liposomes and the Aggregation Kinetics Amyloid-β(1-42)

Thursday, 22 June 2023 10:00 (15 minutes)

The cognitive dysfunctionality stems from Amyloid Aβ (1-42), a neurotoxic peptide which is primarily responsible for Alzheimer's disease and is predominantly found in the extracellular spaces in the form of senile plaques. Aβ42 has a strong propensity to interact with the phospholipid membrane, sphingomyelin, ganglioside GM1, and cholesterol. The plasma membrane is the first biological building block encountered by the Aβ42. Hence, the impact of Aβ42 on the structure and dynamics of the brain phospholipid membrane is important to understand Aβ42 pathogenesis. Furthermore, the aggregation kinetics of Aβ42 in presence of brain phospholipids is of prime interest due neurotoxic behaviour of the Aβ42. We have prepared 100 nm unilamellar vesicles from the brain phospholipids which are mainly composed of phospholipids and sphingomyelin extracted from the porcine brain tissues. Moreover, we have prepared different aggregation states of Aβ42-monomer (M) and proto-fibrils (pf). The unilamellar vesicles were characterized by CryoTEM and Dynamic light scattering and the bilayer thickness is characterized using small angle x-ray scattering. We have investigated the aggregation kinetics of Aβ42 with and without liposomes using Cryo transmission electron microscopy. We have investigated the effect of Aβ42 monomer and protofibrils on the dynamics of unilamellar vesicles using quasielastic neutron scattering. CryoTEM study shows a higher aggregation of amyloid Aβ42 in presence of brain phospholipids and the onset of fibrillation. This study shows Aβ42 brain phospholipids association and the impact of Aβ42 on the dynamics of unilamellar vesicles extracted from brain tissues on the picosecond time scale and Aβ42 fibrillation kinetics.

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Session Classification: Parallel 5