

Students' Annual Webinar

Lipids and apolipoprotein E alter the pathway of aggregation of A β 42

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Amyloid aggregation is involved in several neurodegenerative diseases. In Alzheimer's disease (AD) amyloid- β plaques are found to be one of the hallmarks of the disease. In this context it is important to understand the underlying mechanism of amyloid aggregation. There are several microscopic pathways that are involved in the growth of the amyloids e.g., primary nucleation, elongation and secondary nucleation etc. Here in this work, we attempt to investigate the effects of lipid (DMPC) and apolipoprotein E on the various pathways of aggregation of amyloid- β 42 (A β 42) using real time TIRF microscopy. We found that DMPC leads A β 42 to form lipid-peptide condensates. At early phase of growth, the condensates exhibit liquid -like properties, but at later phase turn into solid -like aggregates. Additionally, we observed that Apolipoprotein E, particularly apoE4 promotes formation of the lipid- A β 42 condensates, possibly by inhibiting elongation of the regular amyloid fibrils.

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4:00 PM