

Students' Annual Seminar

Biophysical characterization of the interactions between Apolipoprotein E and Amyloid- β peptide

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by extracellular deposition of amyloid β ($A\beta$) peptides in the brain. Apolipoprotein E (Apo E) is a 299 residues lipoprotein, that plays important role in regulating metabolism of lipids and cholesterol. There are three major isoforms of ApoE namely: ApoE2 (Cys 112, Cys 158), ApoE3 (Cys 112, Arg 158), and ApoE4 (Arg 112, Arg 158). While ApoE is an important functional protein, ApoE4 is a major risk factor for Alzheimer's disease. All three isoforms of ApoE are found to co-deposit in the senile plaques in AD. Recent in vitro studies suggest that WT-ApoE delays the aggregation of $A\beta$. Here I investigate which region(s) in the sequence of ApoE interact with $A\beta$. To address this we have prepared three different truncated parts of ApoE4, viz, the N-terminal, C-terminal and the Hinge Domain. Our results suggest that all the three domains of ApoE interact with $A\beta$ 42. Furthermore, we find that the effects of the individual domains are additive. Therefore, we hypothesize that structure of ApoE contains multiple independent interacting sites for $A\beta$ 42.

Friday, Mar 23rd 2018

04:30 PM (Tea/Coffee at 03:30 PM)

Seminar Hall, TIFR-H