TIFR Centre for Interdisciplinary Sciences
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Internal Seminar

To unravel the inhibitory mechanism of chaperones in amyloid protein aggregation

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Neurodegenerative diseases are well characterized by the misfolding and aggregation of toxic proteins referred as amyloids. Examples of such conformational disorders are Alzheimer, Parkinson, and the glutamine-encoding expansion diseases. The oligomers form fibrils and interfere with neuronal signaling which in turn leads to dementia and neuronal dysfunction. Proteins with different sequences can form amyloids of 4-12 nm size with rich β sheet fibres. Efforts are being made to understand the mechanism of amyloid aggregation using several biophysical techniques which in turn can be exploited to design the therapeutics. Several proteins interact with the amyloid fibres and are known to inhibit or promote fibril formation. One such class of proteins is chaperones which prevent aggregation events by blocking intermolecular interactions. However, the mechanism explaining the interaction is still to be understood.

My present talk is aimed to discuss the mutagenesis, expression and purification of amyloid proteins such as amyloid-β, α-synuclein and apolipoprotein E using recombinant DNA technology and bacterial expression system. An in house expression facility would enable us to modify and express the proteins of interest in large scale. In later part of my talk, I will describe my project which has been initiated to study the interaction of chaperones and amyloid proteins. Our preliminary studies with E. coli hsp70 showed inhibitory effects on amyloid aggregation.

Wednesday, Jan 6th 2016

2:00 PM (Tea/Coffee at 1:45 PM)

Seminar Hall, TCIS