TIFR Centre for Interdisciplinary Sciences
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Seminar

Protein aggregation and toxicity

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Protein aggregation is a hallmark of aging. The deposition of proteins in the form of amyloid fibrils is a characteristic feature of multiple age-related degenerative conditions. Within the crowded environment of the cell, these amyloid fibrils interact with numerous other metastable proteins that result in amorphous aggregation of those interactors. During 'normal' aging, even in absence of the amyloid fibrils, several proteins misfold and form amorphous aggregates due to gradual collapse of protein homeostasis mechanism, however; the kinetics is slow. Collectively, all these proteins lose their function leading to proteotoxicity.

In our lab, we are studying the impact of protein aggregation on cellular metabolism. We are inducing aggregation of α-synuclein and FlucDM-EGFP in cell culture and analysing the rest of the proteome in dose-dependent presence of these aggregation-prone proteins. α-Synuclein is a well-characterized amyloid forming protein and FlucDM is a variant of Firefly luciferase that forms amorphous aggregates with slight perturbation of protein homeostasis machinery. We have successfully created these aggregation-models and initiated some large-scale quantitative proteomics experiments to investigate the stepwise propagation of proteotoxicity in these models. Initial results from these experiments will be discussed.

Monday, May 8th 2017

4:00 PM (Tea/Coffee at 3:45 PM)

Seminar Hall, TCIS