Tata Institute of Fundamental Research
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Internal Seminar

Deciphering the mechanism of Bendless mediated mitochondrial size regulation

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Mitochondrial health is achieved by maintaining balance between mitochondrial fission and fusion. This is attained by coherent activity of several key mitochondrial dynamics proteins including pro-fusion proteins, such as mitofusins, pro-fission proteins like Dynamin related protein 1 (Drp1), and mitophagy related proteins like Pink1 and Parkin. Their dysregulation has radical effects on mitochondrial function, as happens in neurodegenerative disorders like Parkinson's disease. To identify the genes involved in the regulation of mitofusin, a collection of Drosophila mutants were screened for altered levels of Marf, the fly homolog of human mitofusins. Interestingly, mutation in lrpprc2, human homolog of which is linked to French Canadian Leigh syndrome (LSFC), leads to reduced Marf protein. However, the upregulated Marf mRNA indicates a post- transcriptional mechanism of its regulation in these mutants. Further, we found that this Marf degradation depends on the activity of Bendless (Ben), an E2 conjugase that specifically mediates K63 polyubiquitination. In my talk, I will discuss the work carried out towards elucidation of the mechanism of Ben mediated Marf regulation in lrpprc2 mutants.

Monday, Oct 22nd 2018

2:30 PM (Tea/Coffee at 2:00 PM)

Seminar Hall, TIFR-H