**Tata Institute of Fundamental Research**
Survey No. 36/P, Gopanpally Village, Serilingampally, Ranga Reddy Dist., Hyderabad – 500 046

# Seminar

## Investigating the Role of Synaptic Proteins in Store Operated Calcium Entry (SOCE)

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### TIFR, Hyderabad

$Ca^{2+}$ is a highly versatile intracellular signalling molecule. Depletion of Endoplasmic Reticulum (ER) $Ca^{2+}$ stores during cellular signalling activates Calcium Release-Activated $Ca^{2+}$(CRAC) channels on Plasma Membrane (PM). This phenomenon is termed as Store-Operated Calcium Entry (SOCE). CRAC channels create the most important pathway for $Ca^{2+}$ influx, especially in non-excitable cells, and serve a plethora of physiological functions ranging from gene expression, motility, and development to differentiation. Orai1 and Stim1 are considered key players in forming CRAC channels. Orai1 forms the channel pore and permits only $Ca^{2+}$ ions into the cytoplasm while Stim1 detects depletion of ER stores. An array of accessory proteins have been discovered that interact to regulate the channel. For instance, α-SNAP, a protein involved in recycling the SNARE complex post-vesicle fusion, was found to associate with and regulate the formation of calcium-selective CRAC channels. A limited-RNAi screen was performed to investigate the role of other SNARE proteins in CRAC channel regulation. From this screen, I further investigated the role of t-SNAREs namely SNAP23, SNAP25, SNAP29, and STX11 in SOCE by performing $Ca^{2+}$ assays and binding studies. I found that STX11 directly interacted with Orai1. I further performed domain dissection of this interaction and studied the effect of STX11 depletion on the translocation of calcium-dependent transcription factor NFAT to the nucleus.

***Friday, Feb 7th 2025***

***16:00 Hrs (Tea / Coffee 15:45 Hrs)***

***Seminar Hall, TIFRH***