

Comprehensive Seminar

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

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Ca²⁺ is a highly versatile intracellular signalling molecule. Cells maintain and regulate levels of Ca²⁺ by controlling the opening and closing of several channels. CRAC (Calcium Release-Activated Ca²⁺) channels are ubiquitously present and activate upon depletion of ER Ca²⁺ stores hence the phenomenon is termed Store-Operated Calcium Entry (SOCE). Orai1/CRACM1 (CRAC Modulator 1) a PM-resident protein and STIM1 (Stromal Interaction Molecule 1) an ER-membrane protein are the key players in the formation of the channel. CRAC channels form the most important pathway for Ca²⁺ influx, especially in non-excitabile cells. A genome-wide screening was performed and α -SNAP (Soluble NSF attachment protein) was found to regulate SOCE by interacting with STIM1 and Orai1 proteins. Stx11, a target membrane SNAP (t-SNARE) came up during a recent targeted screen to determine other genes that might have a function in regulating SOCE. Stx11 mutation is known to cause Familial Hemophagocytic Lymphohistiocytosis type 4 (FHL-4) which is characterised by defective NK (Natural Killer) and CTL (Cytotoxic T Lymphocytes) degranulation and cytotoxicity. Reduced Ca²⁺ influx by CRAC channels also causes similar defects in these cells. I am interested in investigating how Stx11 interacts with the channel and regulates SOCE.

Monday, Apr 15th 2024

14:00 Hrs (Tea / Coffee 13:45 Hrs)

Seminar Hall, TIFR-H