

Seminar

Decoding intracellular mechanisms of innate stress sensing and inflammatory cell death

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Immune cell homeostasis and survival are constantly challenged by stress stimuli or pathogenic infections. When exposed to these stressors, each immune cell interprets these insults and assembles distinct cytosolic oligomeric structures to decide its fate. These cytosolic oligomeric structures allow stressed cells to either survive and withstand changes in homeostatic flux or to die by executing programmed cell death. These cytosolic structures also facilitate inflammatory responses and tissue repair to ultimately restore organismal homeostasis. Investigating the fundamental mechanisms of how stress stimuli are sensed and how cell fate decisions are made to restore homeostasis or cause cell death and inflammation is critical for understanding how biological systems deal with stressors and infections. This enables us to therapeutically manage stress-inducing conditions. In this presentation, I will describe my recent work relating to: i) how an immune cell (the macrophage) makes contrasting cell fate choices (survival vs inflammatory cell death) by orchestrating nucleation of distinct cytosolic oligomeric structures; ii) how these cytosolic structures affect immune responses following stress stimuli; and iii) a new mechanism of viral ribonucleoprotein (vRNP) sensing and how this activates multiple programmed cell death pathways and inflammation.

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11:30 AM (Tea/Coffee at 11:00 AM)

Seminar Hall, TIFR-H