

Comprehensive Seminar

Mic19 – A redox-dependent regulator of mitochondrial cristae morphology?

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The mitochondrial contact site and cristae organising system (MICOS) complex is present at cristae junctions (CJs) in the IM and is integral to the maintenance of the morphology of the cristae and consequently, mitochondria. Defects in cristae structure are both a cause and a consequence of a plethora of mitochondria-associated diseases. Mic19 is a peripheral membrane protein component of the Mic60 subcomplex of MICOS. It also interacts with Sam50 in the outer membrane (OM) to form the mitochondrial intermembrane space bridging (MIB) complex, which is thought to maintain contact sites between the OM and IM. Depletion of Mic19 in cells causes mitochondrial fragmentation, loss of cristae junctions and reduced respiratory capacity.

The structure, interaction dynamics, physiological functions and metabolic cues that regulate Mic19 are either unknown or poorly elucidated. A deeper understanding of Mic19 and its interactors through structural and biophysical characterisation *in vitro* and exploration of its physiological functions *in vivo* would deepen our understanding of basic mitochondrial biology.

Monday, Jul 10th 2023

10:00 AM (Tea / Coffee 09.45 AM)

Auditorium, TIFR-H