

Seminar

Mitochondrial cristae shape cellular metabolism and disease

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Mitochondrial cristae are highly organised membrane structures that are essential for mitochondrial function, yet how their architecture influences cellular physiology and human disease remains incompletely understood. The MICOS complex is a central regulator of cristae organisation, and our work has uncovered key mechanisms governing its function and assembly, demonstrating that MIC13 and SLP2 cooperate to stabilise MICOS components and maintain crista junction architecture.

To understand how disruption of these mechanisms contributes to disease, we established an iPSC model carrying patient-specific MIC13 mutations causing mitochondrial hepato-encephalopathy, a severe and currently untreatable multisystem mitochondrial disorder affecting organs including the liver and brain. Differentiation of these iPSCs into hepatocyte-like cells faithfully recapitulated mitochondrial cristae defects and provided a human model to investigate disease mechanisms. Using integrated imaging, biochemical, and multi-omics approaches, we demonstrate that loss of MIC13-dependent cristae organisation drives metabolic dysfunction, cellular stress responses, and early fibrotic changes, revealing how mitochondrial architecture contributes to tissue-specific disease progression. Together, our findings establish mitochondrial cristae as dynamic regulators of cellular metabolism and disease, revealing how genetic defects reshape cellular function and drive disease progression.

Thursday, Aug 13th 2026

11:30 Hrs (Tea / Coffee 11:15 Hrs)

Seminar Hall, TIFRH