National Institute of Technology Rourkela

Departmental Seminar

Seminar Title	: Furamidine-induced autophagy and IL-23 signalling synergistically enhance anti-mycobacterial defense via the Ca^{2+}/pAMPK/SIRT1/FOXO3a and TYK2/STAT3 pathways in human macrophages
Speaker	: Salina Patel - 520ls2001
Supervisor	: 2787
Venue	: LS Seminar Hall
Date and Time	: 07 Aug 2025 (16:00 hrs)
Abstract	: Tuberculosis (TB), driven by Mycobacterium tuberculosis (M. tb), persists as a formidable global health burden. The present study delineates the anti-mycobacterial potential of Furamidine. Initially, Furamidine treatment compromised intracellular mycobacterial growth. Autophagy was explored as a host-defense mechanism, with LC3 conversion, autophagic markers, and MDC staining confirming Furamidine-induced autophagy, linked to elevated intracellular Ca2+ in dTHP-1 cells. Increased intracellular Ca2+ level enhances the expression of pAMPK and SIRT1. Upregulation of SIRT1 leads to FOXO3a activation upon Furamidine treatment. Furthermore, administering various inhibitors impacted intracellular mycobacteria clearance, suggesting that Furamidine triggered the Ca2+/pAMPK/SIRT1/FOXO3a pathway in dTHP-1 cells. Next, we examined the immunomodulatory effects of Furamidine and observed that its treatment enhances IL-23 production, which modulates the phosphorylation of downstream signalling molecules TYK2 and STAT3. Ongoing studies aim to elucidate the precise mechanistic interplay between IL-23-mediated intracellular mycobacterial clearance and its potential cross-talk with autophagy.