National Institute of Technology Rourkela

Registration Seminar

Seminar Title	: Investigating the role of MTP18 in mitochondrial fission-induced mtDNA stress and inflammation
Speaker	: Sushmita Patra (Rollno : 523ls1002)
Supervisor	: Sujit Kumar Bhutia
Venue	: LS Seminar Room
Date and Time	: 24 Jul 2025 (3.30PM)
Abstract	: Oral cancer prevalence in India, with 40% of all types of cancer, has set an alarm for the dire need of an effective therapeutic strategy. Although several advancements have been made in treatment approaches, the 5-year survival rate still stands at 50%. These low survival and high prevalence rates are attributed to aggressive disease progression and acquired chemotherapy resistance. Interestingly, recent evidence has reported a significant association of aberrant mitochondrial function with drug resistance and increased proliferation. Cancer cells modulate mitochondrial dynamics to neutralize chemotherapy stress. Excessive stress causes mtDNA to leak out and drive an inflammatory response. Our study is focused on exploring the underlying mechanism of cisplatin resistance acquired by oral cancer cells by modulating mitochondrial dynamics. We demonstrate that cisplatin treatment induces mitochondrial fission and inflammation activation in oral cancer cells. The inner mitochondrial protein MTP18 is involved in the execution of mitochondrial fission and has LIR motif that interacts with LC3 and promotes mitophagy. This study aims to explore the role of an inner mitochondrial membrane protein MTP18 in oral cancer survival and proliferation under therapeutic stress. MTP18 overexpression promotes mitochondrial fission and mitochondrial fission-mediated mtDNA-stress that induces inflammasome activation through cGAS-STING and NLRP3 pathways. Moreover, it has elucidated the underlying mechanism by how MTP18 protects cancer cells from apoptosis by activating autophagy. Inhibiting autophagy flux in MTP18 overexpression cells further increases type-I interferon and inflammatory signaling.