National Institute of Technology Rourkela

Departmental Seminar

Seminar Title	: PAX9 reactivation promotes autophagy-mediated cellular differentiation by controlling stemness via AKT-GSK3-β signaling in oral cancer cells
Speaker	: Rakesh Kumar Kar (Roll No : 522ls2003)
Supervisor	: Santosh Kumar #2787
Venue	: LS Seminar Hall
Date and Time	: 19 Nov 2024 (11:30 AM)
Abstract	: Paired box 9 (PAX9) is a member of the paired box family involved in cancer cell growth, proliferation, and differentiation. PAX9 downregulation has been established in oral cancer associated with cancer growth and progression. To investigate the role of PAX9 in stemness and differentiation, we initially separated CD44+ and CD44- from oral cancer cells by MACS and confirmed by flow cytometry and western blot analysis. We found that PAX9 decreased expression in CD44+ cells compared to CD44- cells, suggesting higher stemness and lower differentiation activity in CD44+ cells. PAX9 was overexpressed in CD44+ cells, and its role in inhibiting stemness was analyzed through sphere-forming potential and western blot. Furthermore, differentiation-related markers were monitored through western blot in PAX9 overexpressed CD44+ cells. We also reported lower expression of Nanog, SOX2, β-Catenin, p-GSK3-β, and p-AKT in PAX9 overexpressed CD44+cells, which shows decreased stemness capability in PAX9 overexpressed CD44+cells. PAX9 overexpression inhibited AKT/GSK3-β/BMP2 signalling in CD44+ cells, confirming the inverse relation between PAX9 and stemness in oral cancer stem cells. In addition, we identify a PAX9-activating compound from Bacopa monnieri, Bacopaside-II (BS-II) inhibited stemness through autophagy activation of PAX9 in CD44+ cells.