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Departmental Seminar

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Seminar Title : Lipid rafts disruption by MBCD impairs EGFR/FAK axis and ZRF1-mediated EMT and stemness in oral cancer cells

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Venue : LS Seminar Hall

Date and Time : 24 Jan 2025 (11:00 AM)

Abstract : Lipid rafts play prominent role in modulating various cellular processes including cell proliferation, migration, invasion and stemness of oral cancer cells. This study investigates the impact of lipid raft disruption by methyl beta-cyclodextrin (MBCD) treatment on FAK signaling, EGFR expression, epithelial-to-mesenchymal transition (EMT) markers, and the regulation of the epigenetic activator ZRF1. Our results demonstrate that MBCD treatment leads to the downregulation of FAK signaling and reduces EGFR expression, accompanied by a decrease in mesenchymal markers such as N-cadherin and vimentin. Additionally, inhibition of FAK results in reduced EGFR and mesenchymal marker expression and downregulates ZRF1, indicating that the EGFR/FAK signaling axis influences ZRF1 regulation. Importantly, MBCD treatment and FAK inhibition also decrease sphere formation and reduce the expression of pluripotency markers Sox2 and Pax6, suggesting an impact on cancer cell stemness. Furthermore, siRNA-mediated knockdown of ZRF1 results in diminished Sox2 and Pax6 expression, reduced mesenchymal markers, and increased epithelial marker E-cadherin expression, highlighting ZRF1's role in maintaining mesenchymal and stemness characteristics. Collectively, these findings suggest that lipid raft disruption by MBCD suppresses the EGFR/FAK signaling pathway, leading to reduced EMT and stemness in oral cancer cells, with ZRF1 acting as a key downstream effector. This work underscores the therapeutic potential of targeting lipid rafts and the EGFR/FAK/ZRF1 axis in oral cancer treatment.