National Institute of Technology Rourkela

Registration Seminar

Seminar Title	: Role of mechanical signal transduction in regulating epigenetic modulations and gene expression in oral cancer
Speaker	: Jagdish Mishra (Rollno : 522ls2005)
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Venue	: LS Seminar Room
Date and Time	: 23 Jul 2024 (10:00AM)
Abstract	: Mechanical forces may be generated within a cell due to tissue stiffness, cytoskeletal reorganization and the changes (even subtle) in the cell&rsquos physical surroundings. These changes of forces impose a mechanical tension within the intracellular protein network (both cytosolic and nuclear). Mechanical tension could be released by a series of protein-protein interactions often facilitated by membrane lipids, lectins and sugar molecules and thus generate a type of signal to drive cellular processes, including cell differentiation, polarity, growth, adhesion, movement and survival. Mechanosensitive proteins in the cell&rsquos plasma membrane discern the physical forces and channel the information to the cell interior. FAK signaling is an integral component of this pathway. Mechanical signals are transmitted to the nucleus via FAK signaling through the connection of the cytoskeleton and nucleoskeleton. The nuclear transmission of force leads to the activation of chromatin modifiers and modulation of the epigenetic landscape, inducing chromatin reorganization and gene expression regulation. Hence, we tried to disrupt the mechanical signaling pathway in oral cancer cells by using MBCyD. MBCyD treatment downregulates FAK signaling, reduces EGFR expression and decreases EMT markers expression. Further, FAK inhibition reduces EGFR and mesenchymal marker expression. Also, FAK inhibition and MBCyD treatment downregulates the expression of epigenetic modifier ZRF1. From these observations, it can be inferred that lipid raft disruption by MBCyD treatment downregulates EGFR/FAK signaling axis and reduces epithelial-to-mesenchymal transition in oral cancer cells and also this signaling axis regulates the expression of the epigenetic activator ZRF1.