
Departmental Seminar

Seminar Title : Berberine-Induced Apoptosis in Glioblastoma Cell Lines: Involvement of ROS-Mediated Mitochondrial Disruption Through Epigenetic Reprogramming of Nrf2/HO-1 Signaling Dynamics

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Venue : LS Seminar Hall

Date and Time : 18 Nov 2024 (16:00 hrs)

Abstract : Glioblastoma multiforme (GBM) is one of the most prevalent, aggressive, and lethal forms of primary astrocytomas, with a poor prognosis. The current standard treatment includes surgical resection of the tumor, followed by radiotherapy in combination with chemotherapy, typically using temozolomide. In recent years, phytochemicals have garnered significant attention due to their diverse therapeutic properties. In this study, we explored the potential of berberine, a naturally occurring isoquinoline alkaloid, to induce reactive oxygen species (ROS) in glioblastoma cells. Our results demonstrate that berberine significantly reduces cell viability, colony formation, and migratory capacity, while increasing ROS production and disrupting mitochondrial membrane potential. To our knowledge, this study represents the first comprehensive investigation into the cytotoxic effects of berberine on LN229 and U87MG glioblastoma cells. Our findings elucidate the involvement of ROS-mediated, mitochondria-associated apoptosis, along with the disruption of the Nrf2-mediated antioxidant response. Specifically, berberine downregulates KDM6B, leading to an increase in H3K27me3 levels on the Nrf2 promoter, thereby suppressing Nrf2 expression and inhibiting downstream signaling pathways. This research offers valuable insights into the potential therapeutic mechanisms of berberine in targeting GBM. **KEYWORDS:** Berberine, Brain tumor targeting, ROS Production, Mitochondria Membrane disruption, Epigenetic regulation, Cell cycle arrest.