National Institute of Technology Rourkela

Departmental Seminar

Seminar Title	: Piperlongumine induces mitochondrial dysfunction via reactive oxygen species, resulting in apoptosis in human colon cancer cells
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Venue	: LS Seminar Hall
Date and Time	: 03 Jan 2025 (16:00 hrs)
Abstract	: Phytocompounds based research has revealed novel approaches for treating multiple cancers, including colorectal cancer, by mitigating chemoresistance and exhibiting minimal side effects compared to conventional chemotherapy. Piperlongumine (PIP) is a physiologically active alkaloid derived from the roots of the Piper longum plant. Piperlongumine demonstrates significant cytotoxic, anti-proliferative, and apoptotic effects in several malignancies. Nonetheless, the anti-cancer efficacy of piperlongumine and its cytotoxic mechanism in colorectal cancer remains unexplored. This study examined the anti-cancer efficacy of piperlongumine in human colon cancer cell lines HT-29 and SW-480. The cell viability data revealed the half-maximal inhibitory concentration at 3μM and 4 μM of PIP after 48 hr treatment in HT-29 and SW-480 cell line, respectively. PIP was found to inhibit the cell proliferation and migration as well as disrupting the cytoskeletal organisation of the cells. Phytocompounds are known to increase reactive oxygen species (ROS) that accelerates the oxidative stress leading to apoptosis. The confocal microscopy revealed that PIP elevates ROS levels in human colon cancer cells which was supported by the flow cytometry data. Elevated ROS levels led to depolarisation of the mitochondrial membrane resulting in release of cytochrome c into the cytosol from the mitochondria. PIP also damages the DNA which was confirmed by the comet assay. In addition, PIP induces cell cycle arrest at the G2/M stage of cell cycle in human colon cancer cells. Moreover, PIP bring changes in the cellular as well as nuclear morphology due to chromatin condensation and nuclear disintegration. Furthermore, release of cytochrome c into the cytosol activated the caspase dependent apoptotic pathway. Collectively, our results showed that PIP disrupts mitochondrial membrane and induces apoptosis due to elevated ROS production.