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Actinonin represses TRAP1-key molecule to counter resistance in non small cell lung cancer

Priyanca Ahlawat
Post Graduate Institute of Medical Education and Research, India

Statement of the problem: Lung cancer is the most widely prevalent type of cancer worldwide. Approximately 85% of such cases are of non-small cell lung carcinoma histology. The emergence of resistance against prescribed chemotherapy has posed additional trouble. Mitochondria play a critical role in the maintenance of cancer cell homeostasis. Researchers are now focusing on understanding the importance of this organelle in cancer progression and designing novel therapeutics against it. More than a decade ago, Actinonin, a peptidomimetic compound naturally produced by actinomycetes that inhibits human peptide deformylase was observed to have anti-cancerous properties. It has been shown to disrupt the mitochondrial permeability and compromise the cellular health via induction of apoptosis. On the other hand TRAP1, a cytoprotective mitochondrial chaperone has proven to be involved in poor prognosis of resistant cases. However, the effect of actinonin on the expression of TRAP1 has not been studied which might hold the key for apoptosis induction by actinonin.

Methodology & Theoretical Orientation: The IC_{50} of actinonin for non-small cell lung carcinoma cell line H520 was calculated using MTT assay after 24hours of actinonin's treatment. The IC_{50} dose was used to treat the cells and mRNA expression was analyzed for TRAP1, Caspase 8, HIF-1α, Vimentin, and N-Cadherin by real-time PCR. The apoptosis in H520 cells at 24hours of actinonin treatment was analyzed using AnnexinV/PI staining.

Findings: The mRNA expression at 24 hours exposure of H520 cells to actinonin, induced nearly four fold downregulation of TRAP1 expression and a two-fold decrease in Caspase 8 expression. AnnexinV/PI staining confirmed cells in early and late apoptosis with no necrosis upon 24 hours actinonin treatment.

Conclusion & Significance: This study supports that actinonin can be utilized against lung cancer cases and other cancer types in which *TRAP1* gene's higher expression causes resistance against prescribed chemotherapy.

Biography

Priyanca Ahlawat is a research student in the Post Graduate Institute of Medical Education and Research, Chandigarh, India. Her research interests includes molecular biology and mitochondrial biology of non small cell lung cancer and nanotechnology based delivery systems against cancer.

priyanca.91@gmail.com

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