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Expression of sPLA2 isoforms in cigarette smoke condensate induced molecular changes in human macrophage like THP-1 cells

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Environmental carcinogens such as cigarette smoke contribute to chronic inflammation which involves the up-regulation of various groups of phospholipase A2 (PLA2) that predispose to different forms of cancer induction. In this study, for the first time we evaluated the role of cigarette smoke condensate (CSC) induced molecular changes such as cell viability, apoptosis, ROS production, cellular integrity and secretory PLA2 expression in human macrophage like THP-1 cells using MTT assay, Annexin-PI staining, DHE staining, FDA-EtBr staining and RT-PCR respectively at varying concentrations of CSC for 24 hours. Our results revealed a significant decrease in the cell viability in response to increasing CSC concentrations along with a significant mRNA expression of sPLA2 groups: IB, IID, III. In conclusion, CSC contributes towards the oxidative stress in macrophages thereby inducing sPLA2s expression which may be responsible for the regulation of inflammatory cascade leading to cancer development.

Biography

Balwinder Kaur after completing her M.Sc in Biochemistry is pursuing her Ph.D in the field of cancer biology from Postgraduate Graduate Institute of Medical Education and Research under the guidance of Dr. C. M. Pathak, Professor and Head, department of Biophysics. Her present research work includes the study of various phospholipase A2 isoforms in cigarette smoke condensate induced molecular changes in human macrophage like THP-1 cells. Earlier she has worked in a state funded research project related to 14Carbon-urea breath test in adult patients and has published two research papers in reputed international journals. She is actively engaged in teaching and research activities.

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