World Academy of Science, Engineering and Technology
International Journal of Medical and Health Sciences
Vol:13, No:01, 2019

Curative Role of Bromoenol Lactone, an Inhibitor of Phospholipase A2 Enzyme, during Cigarette Smoke Condensate Induced Anomalies in Lung Epithelium

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Abstract : Background: It is well known that cigarette smoke is one of the causative factors in various lung diseases especially cancer. Carcinogens and oxidant molecules present in cigarette smoke not only damage the cellular constituents (lipids, proteins, DNA) but may also regulate the molecular pathways involved in inflammation and cancer. Continuous oxidative stress caused by the constituents of cigarette smoke leads to higher PhospholipaseA_2 (PLA_2) activity, resulting in elevated levels of secondary metabolites whose role is well defined in cancer. To reduce the burden of chronic inflammation as well as oxidative stress, and higher levels of secondary metabolites, we checked the curative potential of PLA_2 inhibitor Bromoenol Lactone (BEL) during continuous exposure of cigarette smoke condensate (CSC). Aim: To check the therapeutic potential of Bromoenol Lactone (BEL), an inhibitor of PhospholipaseA_2s, in pathways of CSC-induced changes in type I and type II alveolar epithelial cells. Methods: Effect of BEL on CSC-induced PLA2 activity were checked using colorimetric assay, cellular toxicity using cell viability assay, membrane integrity using fluorescein di-acetate (FDA) uptake assay, reactive oxygen species (ROS) levels and apoptosis markers through flow cytometry, and cellular regulation using MAPKinases levels, in lung epithelium. Results: BEL significantly mimicked CSC-induced PLA_2 activity, ROS levels, apoptosis, and kinases level whereas improved cellular viability and membrane integrity. Conclusions: Current observations revealed that BEL may be a potential therapeutic agent during Cigarette smoke-induced anomalies in lung epithelium.

Keywords : cigarette smoke condensate, phospholipase A_2, oxidative stress, alveolar epithelium, bromoenol lactone
Conference Title : ICLC 2019 : International Conference on Lung Cancer
Conference Location : Sydney, Australia
Conference Dates : January 30-31, 2019

Open Science Index, Medical and Health Sciences Vol:13, No:01, 2019 publications.waset.org/abstracts/100096.pdf