CIGARETTE SMOKE INDUCED NF-κB ACTIVATION IN HUMAN LYMPHOCYTES: THE EFFECT OF LOW AND HIGH EXPOSURE TO GAS PHASE CIGARETTE SMOKE

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Background: Cigarette smoking is linked to various human disorders. Active and passive smokers suffer from inflammatory diseases of lungs and airways. Smoking-dependent airway inflammation is related to the cytotoxic effects of cigarette smoke (CS) and chronic recruitment of neutrophils and macrophages. NF- κB is a key inflammatory, redox-sensitive transcription factor. Its role in CS-induced airway inflammation is unclear.
Aim: This study investigated CS-induced NF-κB activation in human lymphocytes and the possible involvement of oxidative insult in this activation.
Results: A method for accurate and reproducible exposure of lymphocytes to CS was developed. The intensity of CS exposure was linearly correlated with nitrite concentration originating from nitric oxide present in CS. Mild, but not high exposure to CS, induced NF-κB activation in lymphocytes through an increase in oxidative stress and a reduction in the intracellular glutathione levels.
Conclusions: Lymphocytes can be exposed in vitro to reproducible levels of CS by monitoring nitrite formation in the exposed media containing the cells. Mild, but not high CS exposure, induces increased oxidative stress and NF-κB activation in lymphocytes. These findings may have implications to active as well as to passive smokers, suffering from inflammatory diseases of lungs and airways.