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Role of cigarette smoke compounds in the pathogenesis of COPD

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Statement of the Problem: Cigarette smoke (CS) has always been considered as a risk factor for chronic obstructive pulmonary diseases (COPD). Among the various defense mechanisms against CS-induced oxidative stress, the glutathione S-transferase (GST) family of enzymes is quite well known. The prime responsibility of these enzymes is to neutralize the xenobiotic compounds as well as the ROS generated during various metabolic reactions. Thus it is important to study the effect of individual cigarette smoke compounds on cellular GSH levels and GST activity, if any.

Methodology & Theoretical Orientation: In this study, we have examined the effect of ten cigarette smoke compounds (nicotine, benzo[a]pyrene, naphthalene, formaldehyde, ammonia, acrylic acid, toluene, benzene, m-xylene, and hexamine) on glutathione S transferase (GST) activity, a Phase II metabolic enzyme and their possible role in inflammatory pathophysiology leading to COPD.

Findings: Lower Glutathione (GSH) levels and GST activity and higher CRP, TNF-á, and IL-6 levels were observed in COPD patients compared to age and gender-matched controls. Using human recombinant GST and plasma as well as erythrocytes collected from normal subjects, this study demonstrates that out of the ten compounds, nicotine (5 mg mL^{-1}), benzo[a]-pyrene (10 ng mL^{-1}), naphthalene (250 ìg mL^{-1}), and formaldehyde (5 pg mL^{-1}) caused a significant decrease in recombinant, plasma, and erythrocyte GST activity. Further cell culture studies show that exposure to nicotine, benzo[a]pyrene, naphthalene, and formaldehyde caused a significant decrease in GSH levels and GST activity and its protein expression and an increase in intracellular ROS production in THP-1 monocytes. Interestingly, treatment with benzo[a]pyrene and naphthalene significantly up regulated the phosphorylation of the p65 subunit of NF-êB and increased the secretion of TNF-á and CRP compared to control. This study suggests the potential role of benzo[a]pyrene and naphthalene in the activation of inflammatory signaling pathway leading to cigarette smoke-induced COPD.

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