IN VITRO EVALUATION OF ALUMINUM CHLOROHYDRATE INHALATION TOXICITY: OXIDATIVE STRESS AND EPIGENETIC CHANGES

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Consumer products, such as cosmetics in aerosol formulations, might be sources of inhalation exposure to chemicals in humans. Aluminum chlorohydrate (ACH) is an important aerosol component frequently used as the active ingredient in antiperspirants; however, few studies have addressed its effects on the human respiratory tract. Research has shown that inhalation exposure to various xenobiotics (*e.g.*, tobacco smoke) can induce reactive oxygen species (ROS) and modulate epigenetic pathways in airway cells, which might be associated with respiratory disorders, such as lung cancer and asthma. Therefore, we evaluated the inhalation toxicity of ACH regarding oxidative stress and epigenetic changes using A549 cells as an *in vitro* model of human alveolar cells. Cells were exposed to three non-cytotoxic concentrations of ACH (0.25, 0.5 and 1 mg/mL) for 24 hours. Intracellular ROS were measured using H_2DCFDA probe (flow cytometry). To assess the epigenetic changes, the global DNA methylation pattern [5-methylcytosine (5-mC) and 5-hydroxymethylcytosine (5-hmC)] and the histone modification associated with DNA damage [phospho-histone H2AX (γ-H2AX)] were evaluated by immunostaining for flow cytometry. Our results showed that ACH induced ROS production with a two-fold increase in median fluorescence intensity (MFI). However, it could not alter DNA methylation pattern, or induce DNA damage associated with γ-H2AX in A549 cells. The findings of this study suggest that ACH might be safe for the human respiratory tract regarding epigenetic changes, but it may induce oxidative stress on alveolar cells. Therefore, further research is needed to ensure the inhalation safety of ACH.