Research Abstract

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Air pollution is a known risk factor for lung cancer. Ozone is a major component of air pollution, which exhibits toxicity in distal lung regions affected by lung adenocarcinoma (LA). LA is the most common tumor type in nonsmokers, which originates from multiple cell types, including bronchoalveolar stem cells and type II alveolar (AT2) cells. The effects of ozone in AT2 cells are not well understood. Signaling lipids modulate integral processes altered in lung cancer, which could be disrupted through ozone-mediated reactive oxygen species (ROS), lipid oxidation, and inflammation. My hypothesis is chronic ozone exposure results in dysfunctional lipid transport and signaling in AT2 cells. I will use untargeted metabolomics and qPCR of gross dissected lung lobes and microdissected AT2 cells from ozone-exposed mice to determine global changes in metabolism and inflammation. Furthermore, I will implement targeted metabolomics, immunohistochemistry, and cell death assays to elucidate the role of ozone exposure on lipid signaling. The findings will provide novel insights into the toxic mechanisms of chronic ozone exposure mediated by metabolic factors. Such data will detail how ozone may contribute to increased risk of lung cancer following primary air carcinogen exposures and underscore its influence on human health.