Linking Continued Exposure to E-Cigarette Vapor Constituents with Chronic Obstructive Pulmonary Disease

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E-cigarette usage is growing in popularity, however the correlation between frequent e-cigarette use and ensuing respiratory disease remains unexplored. To that end, a correlation between exposure to e-cigarette compounds and COPD was sought. Human bronchial epithelial cells (HBEs) were exposed to practical concentrations of e-cigarette liquid, nicotine, diacetyl, ethanol, & phosphate-buffered saline; an LDH cytotoxicity assay (indicative of cell/tissue damage) measured the toxicity of e-cigarette liquid, nicotine, & diacetyl. E-cigarette liquid caused a 32% increase in LDH, while diacetyl caused a 26% increase. Concurrently, the mRNA produced by cells was a cDNA template, which was analyzed for augmentation of genes MUC5AC & MUC5B. Increased expression is indicative of increased mucin production, which is directly linked to COPD. Increased MUC5AC gene expression was found for diacetyl (1.3x), e-cigarette liquid (2.2x) & nicotine (2.3x). Diacetyl caused 1.2x increase in MUC5B gene expression. A Western Immunoblot of proteins within e-cigarette-treated HBEs highlights a 54% increase of MUC5AC protein coded for by the MUC5AC gene, further supporting increased mucin production & increased COPD risk. Collectively, these increases highlight COPD risk for e-cigarette users. Next, genetically-modified D.melanogaster BDSC-52262 were exposed to e-cigarettes & tobacco cigarettes. For each, fruit flies experienced decreased locomotor activity, lifespans, & disrupted sleep cycles. Likewise, fly triglyceride concentration was lower for both cigarettes. While LDH/MUC5AC/MUC5B results highlight the causation of COPD from e-cigarette use, decrease in locomotor activity & triglycerides for both cigarettes further demonstrates their similarities in disrupting crucial metabolic pathways.

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