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"Inflammation and Vascular Dysfunction in a Chronic Plus Binge Alcohol and Vaping Mouse Model"

BACKGROUND: Recent epidemiological data suggest a connection between e-cigarette use and increased rates of risky alcohol consumption in U.S. adults. While both substances independently contribute to adverse cardiovascular outcomes—such as elevated blood pressure, myocardial damage, and vascular dysfunction—their combined impact when used concurrently is still poorly understood. This study investigates how chronic exposure to both nicotine vapor and binge alcohol consumption affects cardiovascular function. We hypothesize that co-exposure leads to cardiovascular dysfunction driven by inflammation and oxidative stress.

METHODS: Male mice were subjected to a 20-day dual exposure protocol involving both chronic plus binge alcohol consumption and nicotine vaping. Following a 5-day acclimation period on the Lieber-DeCarli liquid diet, animals were maintained on either a 5% ethanol-containing diet or an isocaloric control diet. Simultaneously, mice were exposed for 12 hours daily (2s hit/10 min) to one of three conditions: nicotine vapor, vegetable glycerin/propylene glycol (VG/PG) vapor, or ambient air. To simulate binge drinking, ethanol-fed mice received a 5 g/kg ethanol bolus by oral gavage on days 10 and 20. Vascular reactivity was evaluated through pin myography of the pulmonary artery and descending aorta. Additionally, real-time PCR was performed on left ventricular (LV) tissue to measure expression levels of pro- and anti-inflammatory markers such as IL-1β, IL-6, and IL-10 and oxidative stress markers such as glutathione peroxidase 1 and 4. Statistical analysis will be conducted using two-way ANOVA followed by Tukey's multiple comparisons test or Fisher's LSD, with significance defined as $p<0.05$.

RESULTS: Pin myography of the pulmonary artery showed increased reactivity to VG/PG, but not in the nicotine vapor group. Ethanol alone decreased reactiveness to the vasodilatory agent sodium nitroprusside. Conversly, in combination with VG/PG with and without nicotine, ethanol led to a decrease in SNP responsiveness. While there was not an increase in proinflammatory markers in the left ventricle, there was a two-fold increase in glutathione peroxidase 4 expression in the combined nicotine vapor and ethanol group; these results point to compensatory changes in the left ventricle to counteract increased lipid peroxidation. Together, these results suggest that nicotine vapor and ethanol interact in ways beyond synergism.