Vascular Reactivity, Inflammation, and Oxidative Stress in a Chronic Plus Binge Alcohol and Vaping Mouse Model

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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, using a mouse model. We hypothesize that co-exposure leads to cardiovascular dysfunction via inflammation and oxidative stress. Statistical analysis performed using 2-way ANOVA.

Methods

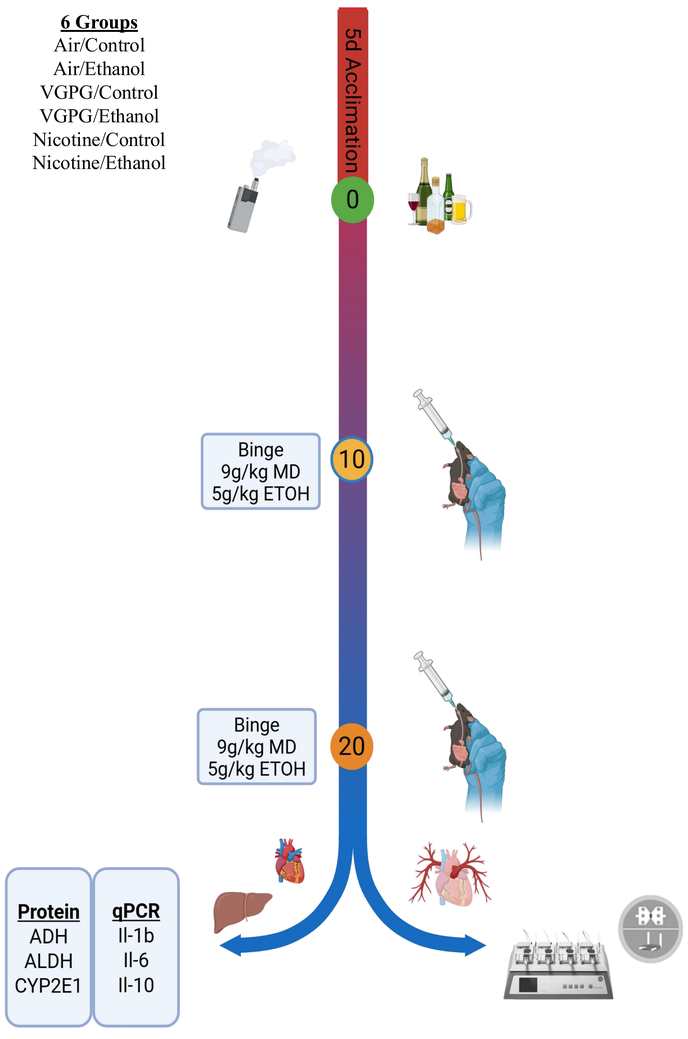

Figure 1. Experimental Timeline. MD = Maltose Dextrin

Pulmonary Artery Wire Myography

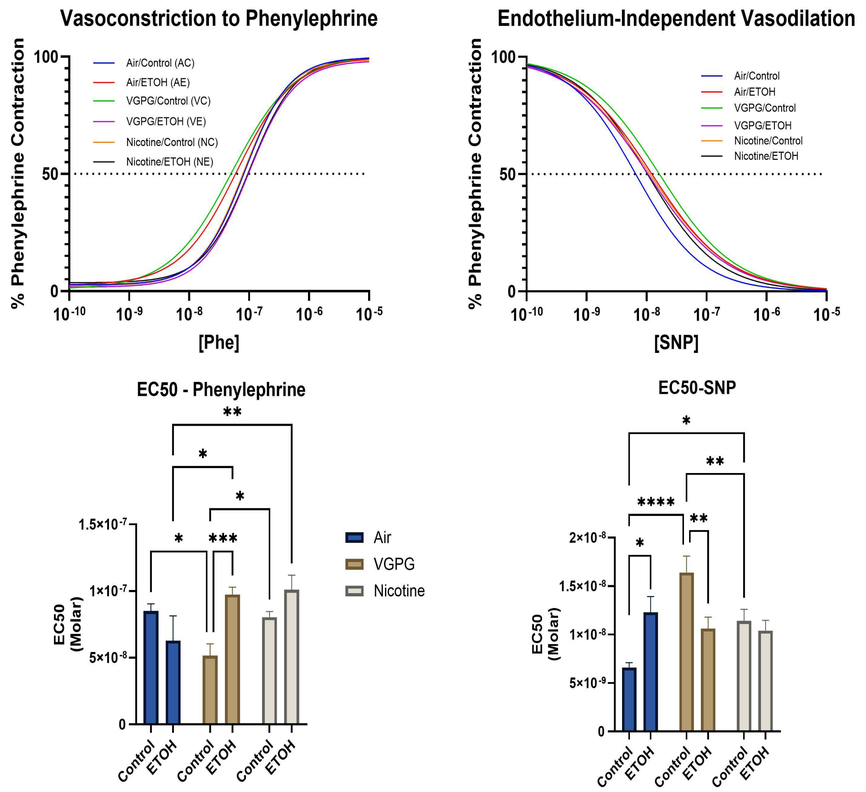

Figure 2. Vasoreactivity of Pulmonary Artery. Top panels show dose-response curves for vasoconstriction (left, Phenylephrine) and endothelium-independent vasodilation (right, SNP). Bottom panels show EC_{50} values derived from non-linear regression.

Liver ETOH Metabolizing Enzyme Expression

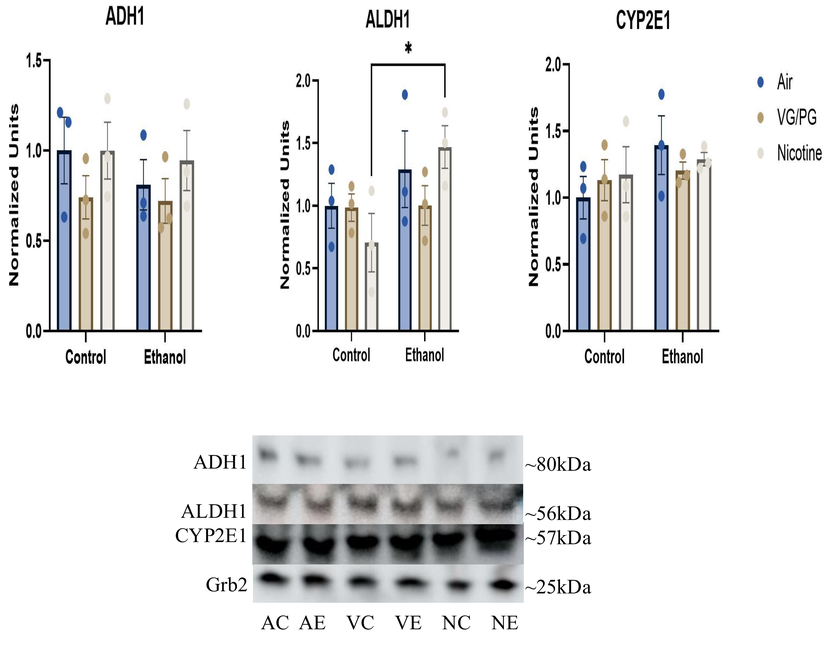

Figure 3. Protein expression of ADH1, ALDH1, CYP2E1, and Grb2 (Loading Control) in mouse liver following air, VG/PG, or nicotine vapor exposure with and without ethanol.

Oxidative Stress Markers

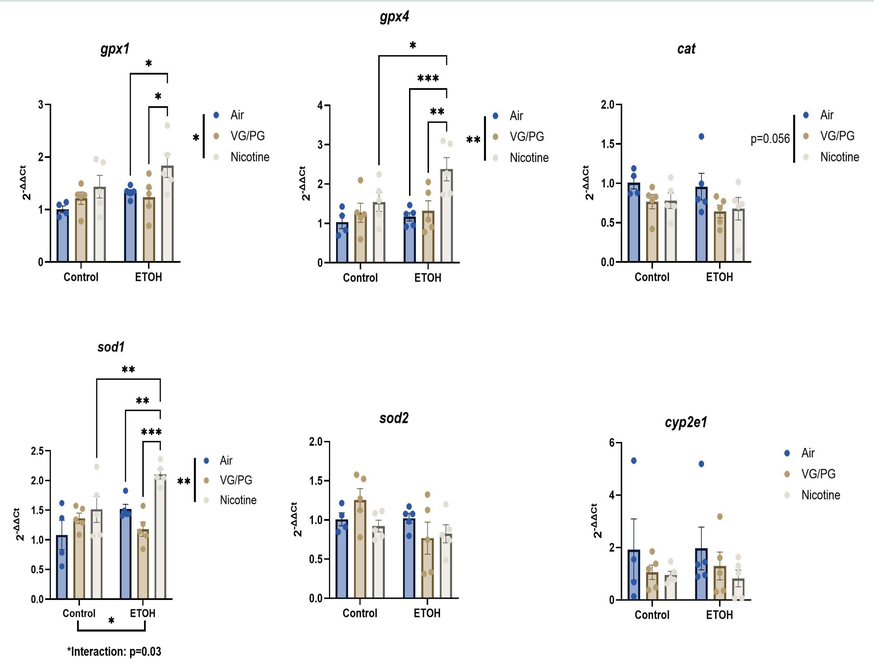

Figure 4. Oxidative and Metabolic Gene Expression in the Left Ventricle. Relative mRNA expression of *gpx1*, *gpx4*, *cat*, *sod1*, *sod2*, and *cyp2e1* in control and ETOH-treated mice after exposure to air, VG/PG, or nicotine vapor.

Inflammatory Markers

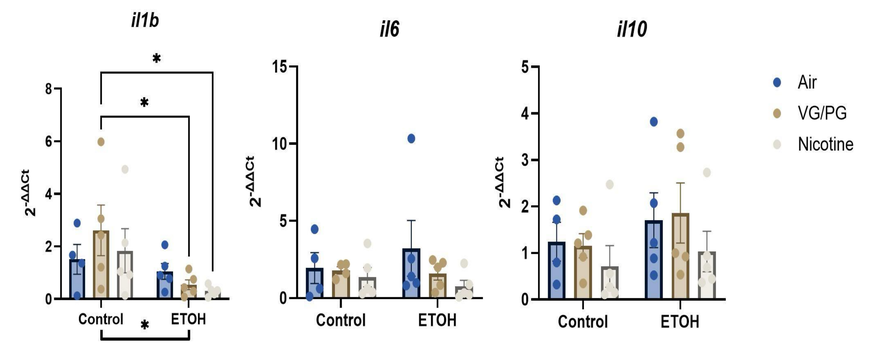

Figure 5. Relative mRNA expression of inflammatory cytokines IL1β, IL6, and IL10 in the left ventricle of control and ETOH-treated groups following exposure to air, VG/PG, or nicotine vapor.

Summary

- Chronic exposure to alcohol and nicotine vapor disrupted vascular reactivity and altered inflammatory cytokine expression (IL1β).
- Gene expression analysis revealed upregulation of oxidative stress markers (GPX1, GPX4, SOD1) with ETOH and nicotine dual use.
- Combined exposure of nicotine ethanol upregulated hepatic ALDH1.

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