SWACSM Abstract

Modeling the Oxidative Metabolic Breakdown of Ethanol and Its Effects on the Cardiovascular System

JESSICA FLORES FARFAN, ALLAN KNOX, & GRADY HANRAHAN

Analytical Chemistry Laboratory; Integrative Human Physiology Laboratory; California Lutheran University; Thousand Oaks, CA

Category: Undergraduate

Advisor / Mentor: Hanrahan, Grady (ghanraha@callutheran.edu)

ABSTRACT

Chronic ethanol consumption contributes to the global prevalence's of cardiovascular disease by mechanisms involving an inflammatory response and consequent lipid peroxidation. Cytochrome p450 is a part of the microsomal ethanol oxidizing system (MEOS) and can utilize iron complexes and the reductant NADPH to catalyze the breakdown of acute/chronic ethanol consumption. However, efficacy of MEOS to prevent cardiovascular burden induced by ethanol consumption is not clear. **PURPOSE:** To demonstrate the response of the cardiovascular system in response to the oxidative metabolic breakdown of ethanol via MEOS. **METHODS:** An extensive literature search provided data to develop a mechanistic model of the metabolism of chronic and low volume ethanol consumption. Artificial neural networks were utilized to construct a colormap of correlation coefficients between ethanol consumption and markers of inflammation and lipid peroxidation. **RESULTS:** The model showed that 3.5 standard drinks (50g of alcohol) were sufficient to increased levels of malondialdehyde and C-Reactive Protein. **CONCLUSION**: These data indicate that ethanol consumption to a level equal to or above 3.5 standard drinks is sufficient to induce cardiovascular stress through increased reactive oxygen species and consequent inflammation, lipid peroxidation, and oxidative stress. The results also provide the foundation for targeted preventative or therapeutic interventions to enhance MEOS that may reduce the cardiovascular burden of alcohol consumption.