Ethanol exposure increases susceptibility to *Listeria* infections in RAW264.7 macrophages, Caco-2 cells, and high alcohol preferring mice

Ryan Restrepo[1], and Yvonne Sun[1].
[1]University of Dayton Department of Biology

Overview

- Excessive alcohol consumption has long been an issue in the United States.
- *Listeria monocytogenes* (LM), a foodborne pathogen, was used as an experimental model to investigate the impact of alcohol consumption on opportunistic infections.
- LM can pass through the intestinal epithelial barrier and infiltrate immune macrophages tasked with preventing the spread of infection.
- LM produces the toxin Listeriolysin O (LLO) in order to proliferate in the host cell cytosol.
- Previous experiments have shown that alcohol consumption increases intestinal permeability for LM.
- Through the use of RAW264.7 macrophages, Caco-2 colonic epithelial cells, and High Alcohol Preferring mice, the effect of alcohol on immune cell function was studied.
- These experiments were conducted in order to establish a more complete picture of the effect of alcohol on human susceptibility to LM infection.

Methods

1. A gentamicin protection assay was used to determine the effect of ethanol pretreatment on the susceptibility of RAW264.7 macrophages and Caco-2 colonic epithelial cells to LM infections.

2. An oral infection experiment using High Alcohol Preferring (HAPII) mice was performed where mice were given ethanol (10%, v/v in water) for 3 weeks to assess animal susceptibility to LM infections.

Main Objectives

1. Determine the impact of ethanol pretreatment on Caco-2 and RAW264.7 cell infection susceptibility

2. Understand the effect of ethanol consumption on the immune capability of High Alcohol Preferring mice (HAP)

Results and Discussion

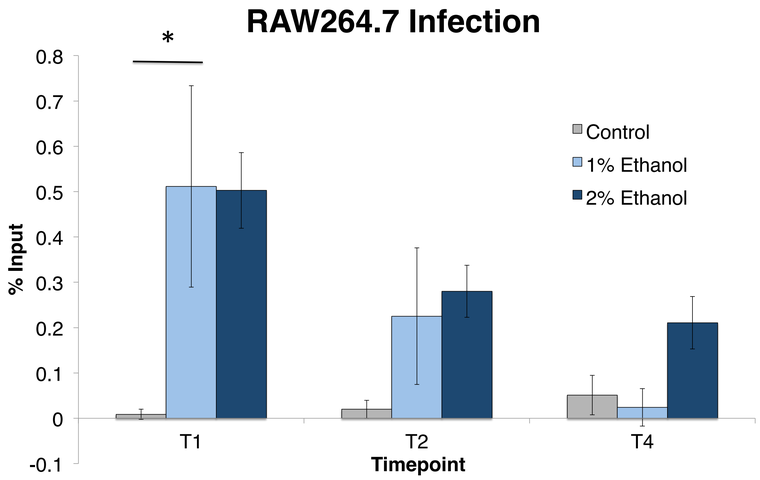

Figure 1: Intracellular LM in RAW264.7 cells with ethanol treatment prior to infections. Supplementation of ethanol in RAW264.7 macrophages significantly increases infiltration of LM during infection.

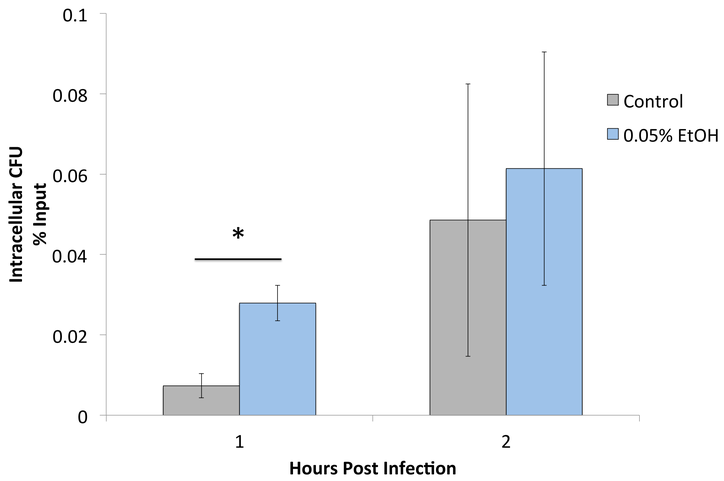

Figure 2: Intracellular LM in Caco-2 cells with ethanol treatment prior to infections. Without ethanol during infection, there was a significant increase in the number of intracellular LM in cells pretreated with ethanol at 1 hpi.

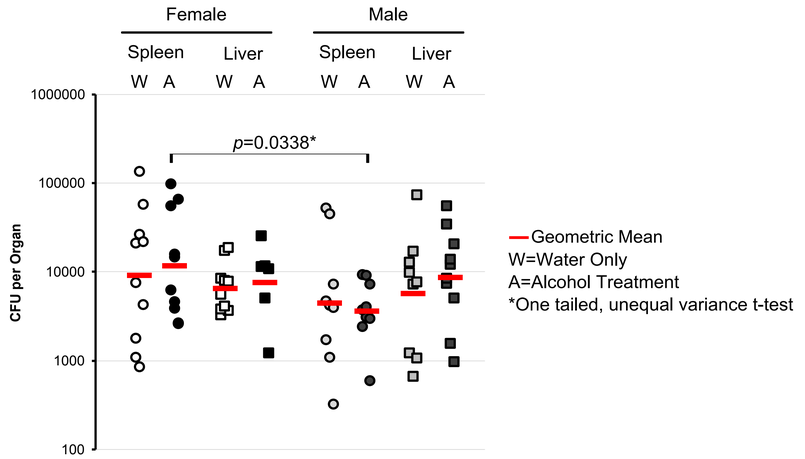

Figure 3: Oral infections in alcohol-treated high alcohol preferring (HAPII) mice. An oral infection experiment using High Alcohol Preferring (HAPII) mice was performed where mice were given ethanol (10%, v/v in water) for 3 weeks to assess animal susceptibility to LM infections.

Conclusions

1. Ethanol treatment increases LM infiltration of RAW264.7 macrophages and Caco-2 colonic epithelial cells.

2. HAPII mice are susceptible to LM oral infections. The ethanol treatment regimen did not significantly alter the susceptibility of individual animal to LM infections.

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Contact
Ryan Restrepo
University of Dayton
Email: restrepor1@udayton.edu
University of Dayton
ANAEROBIC MICROBIOLOGY LAB