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"The characterization of rodent diet influences on ethanol consumption in mice"

The gut-brain axis is a distinct, yet uncharacterized tract of the nervous system that provides direct communication between the myenteric and the central nervous systems. The gut-brain axis is implicated in numerous underlying pathological phenomena, such as depression, Parkinson's disease, and autoimmune disorders. Signaling along the gut-brain axis is primarily mediated by the Vagus nerve, which projects to the Nucleus Tractus Solitarius (NTS). From the NTS, projections link to higher order brain structures, namely reward regions, such as the paraventricular nucleus of the hypothalamus, Locus Coeruleus and the Prefrontal cortex.

Alcohol Use Disorder is a complex and widespread disease with limited pharmacotherapeutic options. Emerging evidence indicates that the gut microbiome influences alcohol intake. Chronic alcohol use leads to gut dysbiosis which is correlated with psychological symptoms such as depression and increased alcohol craving. Diet is a potent regulator of the gut microbiome. In this study, we investigated the role of various rodent diet formulations on alcohol consumption and preference in C57BL/6J mice. This study builds on accidental preliminary findings implicating a strong link between standard rodent diets and alcohol drinking. In this study, we sought to confirm these preliminary findings and extend them by determining whether diet influences on alcohol consumption were a) reversible b) resistant to quinine adulteration of alcohol and c) secondary to alterations in taste preference. The overarching hypothesis is that rodent diet alters the gut microbiome which in turn alters signaling across the gut-brain axis to influence alcohol intake.

The rodent diets analyzed were LabDiet 5001 (LD 5001), LabDiet 5053 (LD 5053), and Teklad (TK). These diets vary immensely in composition and nutritional density. Voluntary alcohol consumption was measured using an intermittent access two bottle choice protocol, providing mice with 15% ethanol and water for 24h per session. Sucrose, saccharin, and quinine consumption were performed using the continuous access protocol providing mice access every day for 24-48h per session. Stool samples were collected at various timepoints to analyze metabolites and bacterial colonies inhabiting the gut microbiome.

Alcohol consumption and preference were increased in mice that were fed LD 5001 or 5053 compared to TK. The effects of diet on alcohol intake were reversible suggesting that the increase in alcohol intake was not due to novelty preference. There were no significant differences in body weights or sucrose, saccharin, and quinine preference between mice that were fed the different diets. Further, alcohol consumption in mice fed LD 5001 was quinine-resistant compared to mice fed TK. Future studies will determine if gut microbiome and metabolite production was altered by the diets. In summary, our results rule out confounding factors like taste and novelty preference and provide preliminary evidence for rodent diet formulations influencing the motivation to consume alcohol.