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Effects of Adolescent Alcohol Exposure on Glutamatergic Transmissions in Male and Female Mice

Adolescence is a time of physical and mental change and is commonly when alcohol use is initiated. In addition, the manner of alcohol consumption among adolescents is typically in a heavy binge-like manner. This adolescent alcohol exposure has been linked to increased likelihood to develop an alcohol use disorder (AUD) in adulthood. A primary driver of continued alcohol use and relapse is negative affect behaviors such as anxiety, depression, and the interaction with stress. A brain region of particular interest in these behaviors is the bed of the nucleus stria terminalis (BNST) which serves as an integration center for information received from cortical and hippocampal regions and projects to a number of hypothalamic, amygdala, and other downstream brain regions. The BNST has far reaching effects within the brain, from influencing negative affect and stress-induced relapse to alcohol use. Furthermore, the changes to the BNST during adolescent alcohol exposure persist into adulthood as the BNST maintains its plasticity. The current project investigates the long-term consequences of adolescent alcohol use within this BNST circuitry. Specifically, changes in glutamatergic signaling will be analyzed, as it serves as a mechanism for long-term changes in plasticity within the BNST circuitry.

To model adolescent alcohol exposure, male and female adolescent mice were exposed to intermittent bouts of alcohol vapor. Brains of mice were evaluated either immediately following alcohol exposure during withdrawal or allowed to mature to adolescence and then stressed. These brains were then analyzed using electrophysiological techniques. Electrophysiology in the BNST measured the number of excitatory neuronal events, with the frequency indicating the number of events (e.g. vesicle release) occurring at the presynaptic terminal and amplitude measuring the response of the post-synaptic neuron. The electrophysiology data will show the change in pre and post-synaptic events in the context of corticotropin releasing hormone (CRH) under stress. Future studies will examine activation of BNST circuity using immunohistochemistry to examine c-fos activation.