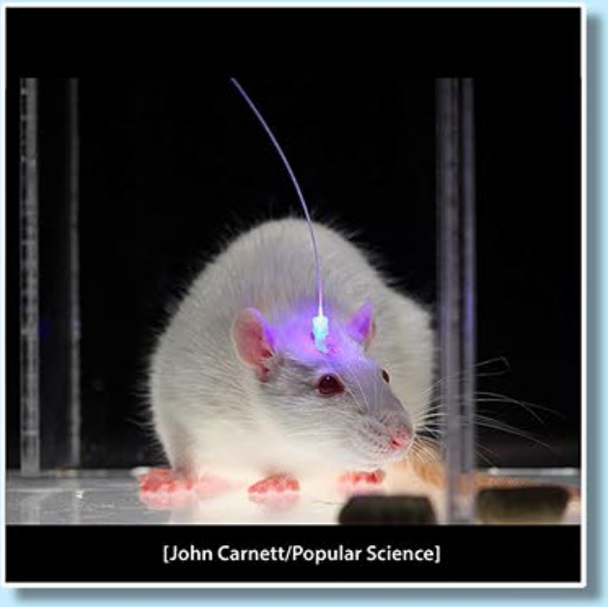

Rat receiving optogenetic stimulation

HANNAH JAGGERS

Faculty Research Mentor: Dr. Donita Robinson
Bowles Center for Alcohol Studies

Activation of Orbito-accumbens Projections Does Not Reliably Reverse Sign-tracking Behavior in Adolescent Alcohol Exposed Rats

America's youth consume more than 90% of their alcohol by binge drinking, which could lead to long-term behavioral deficits and alter the developing brain. In animal models, adolescent intermittent ethanol (AIE) has been shown to affect reward learning, where adult rats exposed to AIE exhibited reduced goal-tracking and enhanced sign-tracking behavior, which is thought to be less flexible than goal-tracking and can act as an indicator for addiction vulnerability. Adult rats exposed to AIE had significantly less resting-state functional connectivity between the orbitofrontal cortex (OFC) and the nucleus accumbens (NAcc) compared to control rats. We hypothesized that AIE-exposed rats would exhibit inflexible sign-tracking that could be shifted to goal-tracking through stimulation of orbito-accumbens projections. To determine how activation of the orbito-accumbens pathway affects behavior and behavioral flexibility in both AIE-exposed and control rats, *in vivo* optogenetic techniques were utilized to selectively activate OFC neurons projecting to the NAcc. Sign- and goal-tracking, as well as flexibility of these behaviors, were assessed using a PCA paradigm. We found that AIE-exposed animals displayed enhanced sign-tracking and reduced goal-tracking as well as reduced flexibility on reward manipulation days, but that activation of the orbito-accumbens projections did not produce a reliable shift in behavior from sign- to goal-tracking. This finding indicates that the orbito-accumbens pathway is not exclusively responsible for altering reward learning in AIE-exposed rats. Future studies should examine how projections from the OFC to other regions in the brain might be altered by adolescent binge drinking and how these alterations might affect susceptibility to AUDs in adulthood.