

Plasticity in the PFC Following 5-7-9 Postnatal Alcohol Exposure Using Exercise as an Intervention

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Abstract

Fetal alcohol spectrum disorder (FASD) is an umbrella term used to describe the wide range of deficits caused by exposure to alcohol in utero. Lack of restraint from conducting a behavior or a psychological process, is often impaired in these individuals (inhibition). One region that is attributed to this lack of inhibition is the medial prefrontal cortex. GABAergic parvalbumin interneurons (PV+) play a role in the inhibition surrounding neurons in the medial prefrontal cortex. Therefore, we hypothesize that alcohol exposed individuals will have less PV+ cells compared to their control counterparts. This study used C57/6J male adolescent mice. These subjects were divided into two groups, alcohol exposure (20% ethanol solution at 5 g/kg) or saline. Behavioral testing occurred between PD72-79. Subjects were tested on the Passive Avoidance task and Rotarod. On 85 PD mice were anesthetized, perfused using saline, and the brains processed using Parvalbumin antibody. Preliminary results show a decrease in PV+ cells in alcohol exposed subjects compared to control groups. Further, alcohol exposed subjects exhibited an increased latency to learn the passive avoidance task. Both the number of PV+ cells and the level of inhibitory control were decreased with prenatal alcohol exposure infer the differences in inhibition between the groups; can be seen by quantity of the PV+ cells in the medial prefrontal cortex. These results show the long term impact prenatal alcohol exposure have on the functioning and anatomy of the medial prefrontal cortex.