TITLE: Effects of ethanol exposure on oligodendrocyte development in the brain

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ABSTRACT: **Background:** Prenatal Alcohol Exposure (PAE) is an event when an expecting mother consumes alcohol, there are no safe times in the pregnancy to consume alcohol nor is there a safe quantity to consume during pregnancy. PAE will lead to children to develop Fetal alcohol spectrum disorders (FASD) which are a group of conditions that can rang from intellectual disabilities or physical deformities. Research has shown that glial cells are affected by PAE with the result of irreversible damage to the brain. Glial cells such as OPCs are responsible for the myelination of axons to conduct correct action potential. In this project, we propose that pups exposed to ethanol at the third trimester will experience reduced mature oligodendrocyte. **Methods:** Our project used mice that were either air (control) exposure or ethanol (experimental) exposure. At day P0, pups were injected with Tamoxifen to induce Cre and label OPCs that were present at the time of experimental exposure (air vs. ethanol). Pups experimental exposure time (P4-P8) was determined to a lined with gliogenesis which is the beginning production of OPCs. Mice were exposed to ethanol by a vapor chamber for 4-5 hour during exposure time. Pups were either perfused on day P8 or kept alive till P30 to study long term effects. Immunohistochemistry was used to study PAE effects on OPCs in the cortex and corpus callosum. Oligodendrocyte markers OLIG2, MBP and CC1 were used to determine the effects of PAE, Cleaved Caspase 3 (CC3) which is a cell death marker was also used. **Conclusions:** Pups that experienced PAE showed less myelination and which might result from low mature oligodendrocytes (CC1) when comparing to air exposed pups.

FUNDING: NIH