Fetal Alcohol Syndrome Affects in Retinal Cell Gene Expression

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Alcohol exposure during fetal development has many adverse effects, producing birth defects known as fetal alcohol spectrum disorder (FASD). Retinal development is consistently affected by ethanol exposure in human patients. Zebrafish are an excellent model to study FASD, due to their similar developmental pathways to humans. Previous studies show that ethanol exposure caused retinal cell differentiation defects leading to photoreceptor defects similar to those seen in human FASD patients. This research aims to understand the gene expression changes that occur in retinal cells due to ethanol exposure. Zebrafish embryos exposed to ethanol [100, 150 mM], from 2-24 hours post-fertilization (hpf), were grown in regular medium until 72 hpf. Eyes from ethanol treated and control zebrafish embryos were dissected and total RNA was isolated. The RNA was then purified and reverse transcribed into cDNA. Quantitative PCR was then used to analyze the cDNA using gene specific primers to determine relative expression levels of various genes present in the retinal developmental pathway. We examined specific signaling pathways including, Wnt, Notch, pro-neural gene targets, and other specific markers expressed by retinal precursor cell populations that comprise the differentiation pathways. This research provides insight into gene expression changes during retinal development that affects specific cell types after alcohol exposure. Our goal is to understand the genesis of FASD birth defects caused by ethanol exposure, and this research will possibly identify ethanol targets and therapeutic strategies to prevent or reverse the damage.

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