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Abstract Title

Prenatal Alcohol Exposure (PAE) Disrupts Hepatic Lipid Metabolism and Elevates Inflammation in Rats

Background

Prenatal alcohol exposure (PAE) is a major public health concern due to its detrimental effects on fetal development and its association with fetal alcohol spectrum disorders (FASD). The liver, as a central organ for nutrient and alcohol metabolism, may play a pivotal role in mediating the effects of PAE.

Objective

This study aimed to investigate the impact of alcohol consumption during pregnancy on maternal and fetal liver lipid metabolism.

Methods

Pregnant Sprague Dawley rats were assigned to one of three groups: standard chow (n=11), chow with 20% ethanol (v/v, EtOH) (n=11), and chow pair-fed (n=9). On gestational day 20 (GD 20), maternal and fetal liver and plasma samples were collected for analysis of lipid metabolism and inflammatory markers.

Results

EtOH consumption increased triacylglycerol (TAG) levels in maternal livers without affecting fetal livers compared to the chow and pair-fed group. The EtOH group showed a reduction in total phospholipids particularly docosahexaenoic acid (DHA), in both maternal and fetal livers ($p < 0.05$). Plasma levels of polyunsaturated fatty acids (PUFAs), including DHA, were also decreased in both dams and fetuses. Furthermore, inflammatory markers, TNF- α , KC/GRO, IL-6, and IL-10, were elevated in the EtOH group.

Conclusion

These findings demonstrate that PAE disrupts hepatic lipid metabolism and induces inflammation in both maternal and fetal livers, highlighting potential mechanisms underlying FASD and alcohol-related hepatic dysfunction. Further studies are warranted to investigate key proteins involved in hepatic DHA metabolism.

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