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DHA supplementation during prenatal ethanol exposure alters the expression of fetal rat liver genes involved in oxidative stress regulation

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Background:

Prenatal ethanol (EtOH) is known to have adverse effects on fetal brain development. The liver is the first organ to receive enriched blood after placental transport; therefore, it could be also damaged by EtOH. Docosahexaenoic acid (DHA) is a known nutrient upregulating the glutathione antioxidant mechanisms.

Objective:

We assessed the effects of maternal DHA on fatty acid metabolism and gene expression of key enzymes of the glutathione antioxidant system in the fetal liver after prenatal EtOH exposure.

Methods:

Sprague-Dawley rat dams (n=4 dams/group) were fed with EtOH during the first 10 days of pregnancy (3g/kg, twice a day) vs. dextrose (isocaloric to EtOH), while being fed a control or DHA-supplemented diet (1.4%, w/w fatty acids). Fetal livers (n=6/group) were collected at gestational day 20 and the free fatty acid (FFA) and phospholipid (PL) fattyacid compositions, as well as the glutathione reductase (GR) and glutathione peroxidase-1 (Gpx1) gene expressions were assessed.

Results:

Fetal liver size was ~8% bigger in response to prenatal EtOH ($P<0.05$).Maternal EtOH exposure minimally affected the fetal liver fatty acid composition. Significant increases in DHA were observed when maternal DHA was provided (1.7 and 1.5 times more in PL; 3.7 and 2.4 times more in FFA) in comparison to the control and EtOH groups, respectively. GR and Gpx1 expressions significantly increased (3-fold) and decreased (2-fold), respectively, after EtOH in comparison to all other groups ($P<0.0001$). Dietary DHA normalized both GR and GPx1 expression to control levels ($P<0.0001$).

Conclusion:

Conclusion: This study is the first to show the effects of maternal DHA supplementation on genes involved in fetal liver oxidative stress removal afterprenatal EtOH exposure. This may be associated with reduced oxidative stress in fetal brain found in previous studies, ultimately mitigating the signs and symptoms of fetal alcohol spectrum disorders in the offspring.