5634

Effects of DHA & Choline Supplementation on Developmental Outcomes of the Typically Developing and Fetal Alcohol Exposed Rat Pup

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Objective: Fetal Alcohol Exposure (FAE) causes a range of neurodevelopmentally abnormal phenotypes. Effects on hippocampus and frontal lobe, areas that preferentially accrete docosahexaenoic acid (DHA), are seen consistently, possibly due to changes to the epigenome during gestation. DHA and choline play an integral role in neural development and subsequent cognitive function, but their effects are generally studied in isolation. Recent work in our lab has shown that DHA and choline act together in support of infant and toddler cognition. Thus, a nutritional intervention using a combination of DHA and choline may have promise for the amelioration of FAE. The primary goal of this study was to examine the effects of DHA and choline supplementation on development in typically-developing and alcohol-exposed pups. The secondary goal was to evaluate the feasibility of DHA and choline as a prenatal supplement for rat dams by designing a supplemented liquid-ethanol diet.

Methods: Rat dams were randomly assigned to four treatment groups, with EtOH and/or DHA & choline (S) (EtOH, EtOH-S, pair-fed, and pair-fed-S), and a fifth group added as ad libitum control. The experimental diet was administered from gestational day 5 to 20. Diet was isocaloric (~100 cal/day/rat) with 35% of daily calories supplied by either EtOH or maltose dextrin. Dams were supplemented daily with DHA (10.8 mg) and choline (155 mg). Food intake was measured for the EtOH-fed dams; weight-matched dams were fed this amount (pair-fed). On postnatal day 2 through 21, markers for development were assessed on bar-holding and negative geotaxis in pups (n=65). Days to first success (latency) on each test were used as a measure of development.

Results: We successfully developed a palatable DHA- and choline-supplemented liquid diet. PROC GLM (SAS Ver 9.4) was used to test group differences. FAE resulted in significantly longer latency to success on bar-holding in EtOH compared to pair-fed and control animals ($p<0.001$). There were no significant differences in latencies on either task between the EtOH-S group and pair-fed-S. The EtOH-S group had significantly shorter latency on both tasks than did the EtOH group (all $p<0.001$). Finally, there were no significant differences between the EtOH-S group and the ad libitum control ($p<0.10$ with the trend in favor of EtOH-S), and both performed significantly better than the EtOH group ($p<0.01$).

Conclusion: The mitigating effect of DHA and choline supplementation on FAE shows promise and warrants further investigation. The addition of an ad libitum supplemented group is needed to strengthen our conclusion. However, the data suggest a positive developmental impact of DHA & choline supplementation on the typically-developing and alcohol-exposed fetus.

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