The Impact of Maternal Diet on Flame Retardant Related Gene Expression in Neonatal Mice

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The prevalence of the obesity epidemic and the ubiquity of endocrine disrupting chemicals (EDC) produces a unique problem for pregnant individuals. This is because interactions of maternal obesity and EDC exposure on developmental programming in the offspring, at the hypothalamic level, may lead to obesity and other metabolic disorders. The specific aim of this project is to determine the effects of maternal obesity on the neonatal expression of Blood Brain Barrier (BBB) genes following organophosphate flame retardants (OPFR) exposure. We conducted our experiment by feeding 10 WT females a low-fat diet (10% fat, LFD) and 10 WT females a high-fat diet (45% fat, HFD) for 8 weeks prior to mating. Once mating occurred all females were dosed with OPFRs from GD7 to PND14. At PND0 and PND14, the mediobasal hypothalamus from 1 female and 1 male pup from each litter were collected for analysis of gene expression. RNA was extracted and samples were prepared for quantitative real-time PCR. Our results suggest that genes associated with the BBB were not affected by maternal diet in the PND0 and PND14 cohorts. However, expression of CLDN1 and OCLN were different between the sexes at PND14. Other genes involved in reproduction and energy balance were altered by maternal HFD in the neonates and juveniles. Ongoing studies are characterizing the permeability of the BBB in littermates and characterizing the metabolic and behavioral effects in adult littermates. These results help us understand how maternal obesity interacts with OPFR exposure on the development of fetus and neonatal brains. Future direction will examine the hypothalamic and hepatic transcriptome in offspring from lean and obese dams with or without OPFR exposure. Supported by the SOT Intern and MARC Programs and EMSOP.

