Hypothalamic and Liver Gene Expression in Neonates Perinatally-Exposed to OPFR

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As of recently, many concerns have arisen regarding the use of flame retardants, specifically PBDEs. PBDEs, as well as other flame retardants are known to be Endocrine disrupting chemicals (EDC). Due to the widespread health affects they cause, PBDEs are being phased out and replaced with another flame retardant known as organophosphate flame retardants (OPFR). OPFR concentrations have been found to be detectable in human urine samples and breast milk. The effects that maternal exposure of OPFR have on offspring metabolism is unknown. This study will provide evidence to the effects that OPFR has on hypothalamic and liver gene expression in the neonate of a wild-type mouse. Breeding pairs were established using virgin females and assigned to either oil or an OPFR mixture (triphenyl phosphate, tricresyl phosphate, and tris (1,3-dichloro-2-propyl)) at 1 mg/kg each from gestational day 7 to postnatal day 14. Total litter weight was taken at birth (day 0) and basal hypothalamic and liver tissue was collected from one female and one male along with individual body weights. On postnatal day 14, the same tissue was collected from a second female and male pup from each litter along with individual body weight. RNA was extracted and is currently being processed for measurement of hypothalamic genes (Bdnf, Pomc, Cart, Npy, Agrp, Kiss1, Pdyn, Tac2, Esr1, Pparg) and liver genes (G6pc, Fas, Dgat2, Pepck, Foxo1, Esr1, Pparg, Ppara) using quantitative real-time PCR. We predict that certain hypothalamic genes (Pomc, Agrp, Kiss1, and Pparg) and hepatic genes (G6pc, Ppara, and Pparg) will be up-regulated whereas other hypothalamic (Bdnf, Cart, Npy, and Esr1) and hepatic genes (Pepck and Esr1) will be down regulated and collectively in a sex-dependent manner. Supported by NIH R25ES020721, P30ES005022, and R21ES027119.