Title: Gene expression differences following early life exposure to polychlorinated biphenyls in three genotypes of mice

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Polychlorinated biphenyls are persistent organic pollutants widely known to affect the developing brain. Our previous work revealed that allelic differences in the aryl hydrocarbon receptor and cytochrome P450 1A2 (CYP1A2) affect susceptibility to developmental PCB exposure, resulting in cognitive deficits and motor dysfunction. High-affinity *AhrbCyp1a2(-/-)* mice were most susceptible compared with poor-affinity *AhrdCyp1a2(-/-)* and wild type *AhrbCyp1a2(+/+)* mice. Our follow-up studies assessed biochemical, histological and gene expression changes to identify the brain regions and pathways affected. The greatest changes were seen in the cerebellum where a foliation defect was over-represented in *Cyp1a2(-/-)* mice. In contrast, we found no difference in tyrosine hydroxylase immuno-staining in the striatum. Tyrosine hydroxylase is the rate-limiting enzyme for dopamine production, and this pathway is disrupted in Parkinson’s disease. Gene expression patterns varied across the three genotypes, but there was clear evidence of AHR activation. Together, our data suggest that the AHR pathway plays a role in developmental PCB neurotoxicity, but we found little evidence that developmental exposure is a risk factor for Parkinson’s disease.