Research Abstract
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Polychlorinated biphenyls (PCBs) remain a significant risk to human health, and a primary target of concern is the developing brain. Research on PCB developmental neurotoxicity (DNT) has focused almost exclusively on the higher chlorinated (HC)-PCBs; in contrast, our understanding of the potential for lower-chlorinated (LC)-PCBs to interfere with neurodevelopment is extremely limited. This is a troubling data gap in light of recent reports that the LC-PCBs 11 and 28 comprise >70% of the PCBs in the serum of pregnant women at increased risk for having a child with a neurodevelopmental disorder. Preliminary data suggest that PCB 11 enhances dendritic arborization in primary neuron-glia co-cultures at concentrations relevant to the human gestational environment. But whether PCB 11 alters neurodevelopment and whether the neurotoxic effects of PCB 11 are autonomous to neurons or require glial interactions are not known. To address these questions, I will test the hypothesis that PCB 11 alters CREB-dependent neurodevelopmental processes, specifically dendritic arborization and apoptosis, via neuron-glia interactions. The findings from this work will inform risk assessments of LC-PCBs, and may provide mechanistic insight on factors that increase vulnerability (e.g., individual differences in innate immunity) as well targets for preventing/mitigating adverse neurodevelopmental outcomes in exposed susceptible populations.