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## Exposure to Polychlorinated Biphenyls Changes Estrous Cyclicity in the Rat: Implications for Basal Ganglia Dopamine

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Polychlorinated biphenyls (PCBs) are persistent organic pollutants. Studies show that exposure to PCBs reduces brain levels of dopamine (DA) and DA neuronal number in exposed animals. PCBs also inhibit DA re-uptake by inhibiting the dopamine transporter (DAT) in synaptic vesicles. However, these studies have only been conducted in male animals. Hence, information on the effects of PCB exposure on basal ganglia dopamine (DA) function in females is lacking. Loss of basal ganglia DA neurons is a primary cause of Parkinson's disease (PD). Epidemiological data from PCB-exposed workers show that basal ganglia DAT density negatively correlates with PCB body burden in exposed women compared to exposed men. In another study, PD mortality increased only in highly exposed female capacitor workers. Interestingly, findings from both studies were obtained in post-menopausal women, suggesting that reductions in ovarian hormones may contribute to gender differences in PCB-induced alterations in DA function and PD mortality. We initially investigated PCB effects on ovarian hormones in rats and found that oral exposure to PCBs alters estrus cyclicity. Studies have shown that changes in cyclicity are associated with alterations in the circulating concentrations of estrogen (E2) and progesterone (P). E2 and P are neuroprotective: Female rat brain DAT density is modulated by the estrus cycle and E2 attenuates the effects of neurotoxins that specifically target basal ganglia DA neurons. To better understand the role of ovarian hormones in influencing PCB-induced DA neurotoxicity, we are currently measuring indices of dopamine function in PCB-exposed animals.