Developmental exposure to PBDEs disregulates neuroendocrine and physiological systems relevant to homeostasis and stress

Polybrominated diphenyl ethers (PBDEs) and the structurally similar chemicals polychlorinated biphenyls (PCBs) disrupt the thyroid and reproductive systems with consequences for development, onset of puberty and reproductive function. We have shown that PCBs and PBDEs also disrupt the secretion of vasopressin (VP) from the hypothalamus during hyperosmotic activation. Since peripheral and central vasopressinergic axes are critical for osmotic and cardiovascular regulation, we examined whether perinatal PBDE exposure could impact these functions during physiological activation. Perinatal exposure to the commercial PBDE mixture, DE-71 (1.7, 30.6 mg/kg/day; oral gavage to dam; GD 6–PD 21) resulted in cardiovascular responses measured at 14–18 months of age. Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR), measured using tail cuff sphygmomanometry, were normalized to pretreatment values (baseline). Hyperosmotic treatment increased SBP in PBDE exposed rats only by 36.1% (low dose) and 64.7% (high dose), at 3h post-treatment. No treatment effects were measured for diastolic BP and HR. Hyperosmotic treatment increased mean plasma osmolality values by 45 min after injection which then stabilized at 3 h relative to normosmotic controls. In contrast, mean plasma osmolality in PBDE-exposed group (high dose) was elevated at 3hr relative to 45min post hyperosmotic injection. Our findings suggest that perinatal exposure to PBDEs can significantly impair cardiovascular function, increasing cardiovascular reactivity to physiological stress. Moreover, PBDE exposure disregulates osmoregulatory responses to hyperosmolality. These effects are seen in late adulthood, indicating that developmental exposure to PBDEs has permanent effects on the function of physiological systems relevant to homeostasis and stress.

Biography

Margarita C. Curras-Collazo completed her Ph.D in Medical Physiology from The Ohio State University and postdoctoral studies in neuropharmacology at the University of North Carolina, Chapel Hill and at Emory University. She is an Associate Professor of Neuroscience in the Department of Cell Biology & Neuroscience at the University of California, Riverside. Research in the Curras-Collazo lab focuses on transcellular and biochemical mechanisms underlying neurosecretion in the neuroendocrine hypothalamus as well as the neurotoxicological and endocrine disruptive effects of environmental pollutants such as brominated flame retardants. She has published more than 35 papers in reputed journals and has served as an ad hoc reviewer for the National Science Foundation, American Heart Association, and Department of Defense as well as international agencies.

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