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Permanently compromised nitric oxide signaling necessary for neuroendocrine function after early life exposure to PCBs

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Organohalogen pollutants such as polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) act as endocrine disrupting chemicals. We have also shown that these toxicants alter vasopressin neurosecretion from magnocellular neuroendocrine cells of the supraoptic nucleus of the rat hypothalamus (SON) and osmoregulatory capacity mediated by vasopressin. Because VP responses to hyperosmotic stimulation are regulated by nitric oxide (NO) signaling, we studied NO synthase (NOS) activity in the SON as potential target of PCB-induced disruption of neuroendocrine processes. To examine PCB-induced changes in NOS activity under normosmotic and hyperosmotic conditions, male Sprague-Dawley rats were exposed to the industrial PCB mixture, Aroclor 1254 (30 mg/kg/day) during gestation. NADPH-diaphorase (NADPH-d) activity assessed in SON sections at 3 ages: postnatal day 10, early adult (3-5 months) or late adult (14-16 months). Hyperosmotic treatment increased mean NADPH-d staining density of oil hyperosmotic controls by 19.9% in early adults and 58% in late adulthood vs normosmotic controls. *In utero* exposure to PCBs occluded hyperosmotic-induced upregulation of NADPH-d activity to control levels in early adults. In contrast, rats that received PCB exposure as early adults orally for 14 days displayed normal NOS responses to hyperosmotic stimulation. Therefore, our findings show that developmental but not adult exposure to PCBs significantly reduces NOS responses to hyperosmolality in neuroendocrine cells. Late adulthood hyperosmotic rats still displayed 28% reduced NOS activity and reduced osmoregulatory capacity during osmotically activated conditions produced by *in utero* exposure. Plasma osmolality values were 375.0 ± 9 vs 348.6 ± 8 mOsm/L for hyperosmotic late adult rats treated with A1254 vs oil controls, respectively. These findings suggest that developmental PCBs permanently compromise NOS signaling necessary for activated neuroendocrine responses with potential osmoregulatory consequences.

Biography

Margarita 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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