Annals of Thyroid Research

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Letter to the Editor

Neonatal Polychlorinated Biphenyls-Induced Endocrine Dysfunction

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Received: January 15, 2016; **Accepted:** January 18, 2016; **Published:** January 27, 2016

Letter to the Editor

The World Health Organization (available at www.who.int/quantifying_ehimpacts/publications/preventing disease/en/) estimated that each year there are more than 13 million deaths caused by environmental causes. Exposure to Endocrine Disrupting Chemicals (EDCs) during in-utero and/or neonatal development can cause long-term health outcomes [1-4]. Polychlorinated Biphenyls (PCBs) are a chemical used primarily in the manufacture of polycarbonate plastic, epoxy resins and as a non-polymer additive to other plastics [5-8]. Because of PCBs extensive use in the manufacture of consumer goods and products, including polycarbonate food containers and utensils, dental sealants, protective coatings, some flame retardants, and water supply pipes, there is a widespread and well-documented human exposure to PCBs [9-15]. Several evidences have demonstrated that human and wildlife populations are exposed to levels of PCBs which cause adverse reproductive and developmental effects in a number of different wildlife species and laboratory animal models [16-19]. Also, PCBs are now widely detected in human urine and blood. Public health concerns have been fueled by findings that PCBs exposure can influence brain development [20-25]. However, there are major uncertainties surrounding the spectrum of PCBs mechanisms of action, the tissue-specific impacts of exposures, and the critical windows of susceptibility during which target tissues can be sensitive to PCBs exposures.

The study of Thyroid Hormone (TH) disorders provides a template for relating TH-mediated effects on the brain to these compounds [26,27]. Alterations in thyroid structure and function may be important in the pathogenesis of certain metabolic disorders associated with PCB intoxication [28-30]. Several conclusions can be drawn from the recent Publications. The first is that the developmental exposure to PCB 95 in the early weaning period seems to alter TH synthesis and secretion, either by acting directly on the thyroid gland or by acting on the pituitary or hypothalamic control of Thyrotropin (TSH) or Growth Hormone (GH)/Insulin Growth Factor 1 (IGF1) secretion [3]. The second is that the administration appears to induce hypothyroidism via thyroid dysgenesis and dyshormonogenesis. These drastic effects may play a significant role in thyroid diseases [3,4]. The third is that PCB 95 seems to play the role of a stress-responsive factor in the neonatal endocrine system [3]. The fourth is that hypothyroidism caused by PCB 95 seems to alter the development of the adipokine axis, fat metabolism, and in general postnatal development. The final conclusion is that the administration of PCB 95 seems to lead to thyroid adipokine dysfunction [3,4]. These changes may be either directly or indirectly related to TH action [3,25,26]. I hypothesized that if the endocrine system is disrupted earlier in development, then it is more likely to persist into adulthood. This may cause a persistent hypothyroidism and disrupt the hypothalamic neural circuitry and estrogenic activity. More interestingly, the toxicity of PCBs is dependent on compound congeners, dose, exposure duration, developmental period, and the species involved. I also observed the importance of further studies on implications at later life stages following the hormonal alteration occurring in the developing infants. Further investigations are required to elucidate the potential associations with human health.

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Citation: Ahmed RG. Neonatal Polychlorinated Biphenyls-Induced Endocrine Dysfunction. Annals Thyroid Res. 2016; 2(1): 34-35.

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Annals Thyroid Res - Volume 2 Issue 1 - 2016
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