

Letter to the Editor

Neonatal Polychlorinated Biphenyls-Induced Endocrine Dysfunction

Ahmed RG*

Zoology Department, Beni-Suef University, Egypt

***Corresponding author:** Ahmed RG, Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt

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 dysmorphogenesis. 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.

References

- Abdelouhab N, Langlois MF, Lavoie L, Corbin F, Pasquier JC, Takser L. Maternal and cord-blood thyroid hormone levels and exposure to polychlorinated diphenyl ethers and polychlorinated biphenyls during early pregnancy. *Am J Epidemiol.* 2013; 178: 701-713.
- Ahima RS, Flier JS. Adipose tissue as an endocrine organ. *Trends Endocrinol Metab.* 2000; 11: 327-332.
- Ahmed RG. Early weaning PCB 95 exposure alters the neonatal endocrine system: thyroid adipokine dysfunction. *J Endocrinol.* 2013; 219: 205-215.
- Ahmed RG. Do PCBs modify the thyroid-adipokine axis during development? *Annals Thyroid Res.* 2014; 1: 11-12.
- Anbalagan J, Kanagaraj P, Srinivasan N, Aruldas MM, Arunakaran J. Effect of polychlorinated biphenyl, Aroclor 1254 on rat epididymis. *Indian J Med Res.* 2003; 118: 236-242.
- Arsenescu V, Arsenescu RI, King V, Swanson H, Cassis LA. Polychlorinated biphenyl-77 induces adipocyte differentiation and proinflammatory adipokines and promotes obesity and atherosclerosis. *Environ Health Perspect.* 2008; 116: 761-768.
- Baker NA, Karounos M, English V, Fang J, Wei Y, Stromberg A, et al. Coplanar polychlorinated biphenyls impair glucose homeostasis in lean C57BL/6 mice and mitigate beneficial effects of weight loss on glucose homeostasis in obese mice. *Environ Health Perspect.* 2013; 121: 105-110.
- Boas M, Feldt-Rasmussen U, Main KM. Thyroid effects of endocrine disrupting chemicals. *Mol Cell Endocrinol.* 2012; 355: 240-248.
- Boas M, Feldt-Rasmussen U, Skakkebaek NE, Main KM. Environmental chemicals and thyroid function. *Eur J Endocrinol.* 2006; 154: 599-611.
- Bowers WJ, Nakai JS, Chu I, Wade MG, Moir D, Yagminas A, et al. Early developmental neurotoxicity of a PCB/organochlorine mixture in rodents after gestational and lactational exposure. *Toxicol Sci.* 2004; 77: 51-62.
- Crofton KM, Zoeller RT. Mode of action: neurotoxicity induced by thyroid hormone disruption during development--hearing loss resulting from exposure to PHAHs. *Crit Rev Toxicol.* 2005; 35: 757-769.
- El Majidi N, Bouchard M, Carrier G. Systematic analysis of the relationship between standardized biological levels of polychlorinated biphenyls and thyroid function in pregnant women and newborns. *Chemosphere.* 2014; 98: 1-17.
- Elabbas LE, Esteban J, Barber X, Hamscher G, Nau H, Bowers WJ, et al. In utero and lactational exposure to a mixture of environmental contaminants

- detected in Canadian Arctic human populations alters retinoid levels in rat offspring with low margins of exposure. *J Toxicol Environ Health A*. 2014; 77: 223-245.
14. Grimm FA, Lehmler H-J, He X, Robertson LW, Duffel MW. Sulfated metabolites of polychlorinated biphenyls are high-affinity ligands for the thyroid hormone transport protein transthyretin. *Environ Health Perspect*. 2013; 121: 657-662.
15. Gu JY, Qian CH, Tang W, Wu XH, Xu KF, Scherbaum WA, et al. Polychlorinated biphenyls affect thyroid function and induce autoimmunity in Sprague-Dawley rats. *Horm Metab Res*. 2009; 41: 471-474.
16. Hayley S, Mangano E, Crowe G, Li N, Bowers WJ. An *in vivo* animal study assessing long-term changes in hypothalamic cytokines following perinatal exposure to a chemical mixture based on Arctic maternal body burden. *Environ Health*. 2011; 10: 65.
17. Hisada A, Shimodaira K, Okai T, Watanabe K, Takemori H, Takasuga T, et al. Associations between levels of hydroxylated PCBs and PCBs in serum of pregnant women and blood thyroid hormone levels and body size of neonates. *Int J Hyg Environ Health*. 2014; 217: 546-553.
18. Kim S, Park J, Kim HJ, Lee JJ, Choi G, Choi S, et al. Association between Several Persistent Organic Pollutants and Thyroid Hormone Levels in Cord Blood Serum and Bloodspot of the Newborn Infants of Korea. *PLoS One*. 2015; 10: e0125213.
19. Kobayashi K, Miyagawa M, Wang RS, Suda M, Sekiguchi S, Honma T. Effects of in utero exposure to 2,2',4,4',5,5'-hexachlorobiphenyl on postnatal development and thyroid function in rat offspring. *Ind Health*. 2009; 47: 189-197.
20. Leijts MM, ten Tusscher GW, Olie K, van Teunenbroek T, van Aalderen WM, de Voogt P, et al. Thyroid hormone metabolism and environmental chemical exposure. *Environ Health*. 2012; 11: 10-16.
21. Lv QX, Wang W, Li XH, Yu L, Zhang Y, Tian Y. Polychlorinated biphenyls and polybrominated biphenyl ethers in adipose tissue and matched serum from an E-waste recycling area (Wenling, China). *Environ Pollut*. 2015; 199: 219-226.
22. Miller MD, Crofton KM, Rice DC, Zoeller RT. Thyroid-disrupting chemicals: interpreting upstream biomarkers of adverse outcomes. *Environ Health Perspect*. 2009; 117: 1033-1041.
23. Miller VM, Sanchez-Morrissey S, Brosch KO, Seegal RF. Developmental coexposure to polychlorinated biphenyls and polybrominated diphenyl ethers has additive effects on circulating thyroxine levels in rats. *Toxicol Sci*. 2012; 127: 76-83.
24. Su PH, Chen HY, Chen SJ, Chen JY, Liou SH, Wang SL. Thyroid and growth hormone concentrations in 8-year-old children exposed in utero to dioxins and polychlorinated biphenyls. *J Toxicol Sci*. 2015; 40: 309-319.
25. Tang-Péronard JL, Heitmann BL, Jensen TK, Vinggaard AM, Madsbad S, Steuerwald U, et al. Prenatal exposure to persistent organochlorine pollutants is associated with high insulin levels in 5-year-old girls. *Environ Res*. 2015; 142: 407-413.
26. Ahmed RG. Editorials and Commentary: Maternofetal thyroid action and brain development. *J of Advances in Biology*. 2015; 7: 1207-1213.
27. Ahmed RG. Hypothyroidism and brain developmental players. *Thyroid Res*. 2015; 8: 2.
28. Wadzinski TL, Geromini K, McKinley Brewer J, Bansal R, Abdelouahab N, Langlois MF, et al. Endocrine disruption in human placenta: expression of the dioxin-inducible enzyme, CYP1A1, is correlated with that of thyroid hormone-regulated genes. *J ClinEndocrinolMetab*. 2014; 99: 2735-2743.
29. Xu X, Chiung YM, Lu F, Qiu S, Ji M, Huo X. Associations of cadmium, bisphenol A and polychlorinated biphenyl co-exposure in utero with placental gene expression and neonatal outcomes. *Reprod Toxicol*. 2015; 52: 62-70.
30. Zoeller RT, Brown TR, Doan LL, Gore AC, Skakkebaek NE, Soto AM, et al. Endocrine-disrupting chemicals and public health protection: a statement of principles from The Endocrine Society. *Endocrinology*. 2012; 153: 4097-4110.