Letter to Editor

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Hypothyroidism and Brain Development

Ahmed R.G

Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt

Corresponding author: Ahmed RG, Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt, Tel/Fax: 002-010-91471828; E-mail: ahmedragab08@gmail.com

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Thyroid hormones (THs) are central for the development of all biological systems Abalovich et al., El-bakry et al., Ahmed et al., Ahmed and Incerpi, Van Herck **et al.,** Deepti et al., El-Ghareeb et al., Ahmed and El-Gareib and Caty et al. [1-4]. A moderate iodine deficiency causes maternal hypothyroxinemia, congenital hypothyroidism, preterm birth and mental retardation. Hypothyroidism during the development caused the following. Reduced the myelination of neuronal axons altered the dendritic structure cortical pyramidal cell [5-8]. Thompson and Potter reduced the Purkinje cell dendritic arborizations, the parallel fiber outgrowth and migration of the granule cells, and the number of granule cells diminished the synaptogenesis altered the molecular, morphological and functional actions of hippocampus and delayed the hypothalamic-pituitary development. Generally, THs deficiency during the early developmental period might delay the neuronal differentiation, decrease the neuronal connectivity, impair the motor skills and visual processing, and cause severe and permanent brain damage or mental retardation. Thus, these adverse effects may facilitate directly by a loss of the maternal THs contribution to the fetus or indirectly by the metabolic impairment of gestation, or both [9-12].

Conflict of Interest

The author declares that no competing financial interests exist.

References

1. Abalovich M, Llesuy S, Gutierrez S, Repetto M (2003) Peripheral parameters of oxidative stress in Graves' disease: the effects of methimazole and 131 iodine treatments. Clin Endocrinol 59: 321-327.
2. El-bakry AM, El-Ghareeb AW, Ahmed RG (2010) Comparative study of the effects of experimentally-induced hypothyroidism and hyperthyroidism in some brain regions in albino rats. Int J Dev Neurosci 28: 371-389.
3. Ahmed RG (2011) Perinatal 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin exposure alters developmental neuroendocrine system. Food Chem Toxi 49: 1276-1284.
4. Ahmed OM, Ahmed RG (2012) Hypothyroidism. In A New Look At Hypothyroidism. Springer (Ed.), ISBN: 978-953-51-0020-1, In Tech Open Access Publisher, Chapter 1, 1-20.
5. Ahmed OM, Ahmed RG, El-Gareib AW, El-Bakry AM, Abd El-Tawaba SM (2012) Effects of experimentally induced maternal hypothyroidism and hyperthyroidism on the development of rat offspring: II-The developmental pattern of neurons in relation to oxidative stress and antioxidant defense system. Int J Dev Neurosci 30: 517-537.
6. Ahmed RG (2013) Early weaning PCB 95 exposure alters the neonatal endocrine system: thyroid adipokine dysfunction. J Endocrinol 219: 205-215.
7. Ahmed RG (2014) Editorial: Do PCBs modify the thyroid-adipokine axis during development? Annals Thyroid Res 1: 11-12.
8. Ahmed RG (2015) Chapter 1: Hypothyroidism and brain development. In Advances in Hypothyroidism Treatment. Avid Science Borsigstr. 9, 10115 Berlin, Berlin, Germany. Avid Science Publications level 6, Melange Towers, Wing a, Hitec City, Hyderabad, Telangana, India. 1-40.
9. Ahmed RG (2015) Hypothyroidism and brain developmental players. Thyroid Research J 8: 1-12.
10. Ahmed RG (2015) Editorials and Commentary: Maternofetal thyroid action and brain development. J adv biol 7: 1207-1213.
11. Ahmed RG (2016) Gestational dexamethasone alters fetal neuroendocrine axis. Toxicology Letters 258: 46-54.
12. Ahmed RG (2016) Neonatal polychlorinated biphenyls-induced endocrine dysfunction. Ann Thyroid. Res 2: 34-35.