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# Hyperthyroidism and Developmental Dysfunction

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## Letter to Editor

Thyroid hormones (THs) are essential for normal growth and development [1-30]. Hyperthyroidism causes a thyrotoxicosis [31], membrane lipid alterations [32], TSH reduction [4], neuropsychiatric and neurologic syndromes and myopathy [4,33]. In neonatal hyperthyroidism, cardiac symptoms, and abnormal neurological behaviors were observed by several authors [4,34-37]. Fetal thyrotoxicosis is the result of thyroid-stimulating antibody transfer to the fetus in the setting of maternal Grave's disease [6,38]. A fetal hydrops, demise, intrauterine growth restriction, and goiter were reported by Zimmerman et al. [39] and Ahmed et al. [4]. In maternal hyperthyroidism, the placenta lacks to optimize the maternaofetal transfer of THs [6].

My group reported the following (1) The hyperthyroid rat newborns showed larger and less thyroid follicles with flattened cell lining epithelium, decreased thyroid gland size, hyperemic blood vessels, dispersing oedema and degenerative changes along the first 4 postnatal weeks [27]; (2) The maternofetal thyrotoxicosis by L-thyroxine (L-T4) alters the developing hypothalamic-pituitary-thyroid axis (HPTA), growth hormone (GH)/insulin-like growth factor-1 (IGF1) and cytokines/adipokines axes [10-12]; (3) Maternal hyperthyroidism disturbs the levels of iodothyronine 5'-monodeiodinase (5'-DI), monoamines [norepinephrine (NE), epinephrine (E), dopamine (DA) and serotonin (5-HT)],  $\gamma$ -aminobutyric acid (GABA), acetylcholinesterase (AChE), and ATPase-enzymes (Na<sup>+</sup>, K<sup>+</sup>-ATPase, Ca<sup>2+</sup>-ATPase and Mg<sup>2+</sup>-ATPase) in brain of neonatal rats [1]; (4) Hyperthyroidism disrupts the neurogenesis, interneuronal connections, neuronal integration and functional alterations [4] and this may be attributed to prooxidant and antioxidant disorders [6,7]; (5) Maternal hyperthyroidism degenerates the pyramidal and polymorphic cells was noted with loss of the axons or lateral dendrites of pyramidal cells [1]; (6) Maternal hyperthyroidism degenerates the stellate, basket, Purkinje, Golgi and granule cells of neonatal cerebellum [4]; and (7) Maternal hyperthyroidism increases the growth of neonatal skeletal system [27]. Thus, the administrations of L-T4 to mothers may cause several injurious anomalies in the development of their newborns and may lead to a pathophysiological state [6]. Age may represent a factor determining the severity and reversibility of the effects of hyper

functioning of thyroid gland in the growth of various rat organs. These findings fit well with our previous review [4]. However, whether the adverse effects of maternal hyperthyroidism on fetal development are mediated directly by loss of the maternal hormones contribution to the fetus, indirectly by metabolic impairment of gestation, or both. Further studies need to be done to emphasize this concept. Collectively, my work recommended that the importance of maintaining normal maternal thyroid functions during pregnancy or lactation periods is required to prevent the appearance of any embryonic or fetal disorders.

## Conflict of Interest

The author declares that no competing financial interests exist.

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