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Commentary

Maternal Hyperthyroidism and Developing Brain Dysfunction

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The collaboration between the activities of thyroid hormones (THs) and growth factors is critical for the development in particular the developing brain [1-51]. During the neocortical development, thyroxine (T4) deiodinated by the deiodinase 2 (D2) might be the single source of 3,5,3'-triodothyronine (T3) [52]. In addition, the latter authors reported that the normal activity of D2 in the cerebral cortex during the late gestation is significant to stimulate the expression of T3-dependent genes.

On the other hand, maternal hyperthyroidism can suppress the activity of neonatal hypothalamic-pituitary axis [53,54]. Similar observations are reported by Demet., *et al*. [55], Simon., *et al*. [56], Ahmed., *et al*. [54], Yu., *et al*. [57], and Stohn., *et al*. [58] who stated that the functional disturbances in the hypothalamic-pituitary-thyroid axis (HPTA) due to the hyperthyroidism can cause psychiatric diseases (mood disorders) such as anxiety and depression. In addition, the expression of neuronal cytoskeletal proteins was compromised in the late fetal brain development, indicative the augmentation the neuronal differentiation [54,59]. Collectively, several investigators reported that the gestational hyperthyroidism can alter the developing brain and cause the following [1,15,54,60-63]: (1) irreversible impairment; (2) physical retardation for the central nervous system (CNS); (3) abnormalities in the neural morphogenesis; (4) disorganization in the developing brain; and (5) disturbance in the neurobehavioral activities. It can be concluded from these data that the maternal hyperthyroidism can impair the genesis of the carbohydrates, delay the development and activity of the nerve cells, and the neurobehavioral response.

From the previous considerations, the balance in the maternal HPTA displays remarkable actions in the developing brain. In addition, the maternal thyroid dysfunctions (hyperthyroidism) may decline the developmental and functional brain. The sustained defects of the maternal hyperthyroidism can disrupt the neural connections, nerve impulses, synaptogenesis, and myelination, and cause the neurocognitive disorders. Preserved the normal activities of THs during the development and adulthood may be needed to get normal neurocognitive behaviors. Thus, the treatment of the maternal hyperthyroidism before the gestation should be decisive for the endocrine and neural enhancement. This can recover the balance in the thyroid-brain axis during the prenatal and postnatal periods. Nevertheless, other studies are wanted to determine whether the severity of maternal hyperthyroidism may cause persistent conflicts in the development and activity of fetal/neonatal brain. Also, additional works are needed to test the effect of chronic hyperthyroidism during the gestation on the differentiation of neurons and astrocytes. These points require more investigation.

Conflict of Interest

The author declares that no competing financial interests exist.

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