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Developmental Thyroid Diseases and Monoaminergic Dysfunction

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Dear Editor,

Thyroid hormones (THs) regulate the pre- and post-natal development, particular brain development [1-25]. Also, THs can regulate the development of monoaminergic [norepinephrine (NE), epinephrine (E), dopamine (DA) and serotonin (5-HT)] system [14,26]. These monoamines were elevated during the postnatal period in different brain regions [27,28]. These elevations were reported in rat [29], mice [30], guinea pig [31] and chick [32]. In addition, this development might also reflex the elevation of sympathetic activity with the age progress.

THs defects (hypothyroidism) can impair this system during development [33]. Thus, these maternal impairments may be attributed to altered their synthesis and metabolism. These resulting in a fetal/neonatal mal-development and pathophysiological state. However, there was decreased in the level of DA and increased in the levels of NE and 5-HT [34] or decreased in the levels of NE and 5-HT in the hypothyroid rats [35]. Also, hypothyroidism can decrease the activities of β-adrenergic post-synaptic receptors, initiating a diminution in the noradrenergic neurotransmission [36]. In contrast to these results, Singh et al. [37,38] found that in rat, the content of NE and DA did not change after thyroidectomy while other authors [39,40] revealed that hypothyroidism may increase the CA contents in the brain. This argument reflects the complex structure of the brain and the secondary effects of THs [41].

On the other hand, there was decreased in the content of NE and increased in the contents of 5-HT and DA in the hyperthyroid young rats [42]. These changes may be due to disturbance in the synthesis, turnover and release of these amines through the neurons impairment or may attributed to an alteration pattern of their synthesis and/or degradative enzymes or changes in the sensitivity of their receptors [43]. Jacoby et al. reported that there was acceleration in the accumulation of 5-HT and catecholamines in hyperthyroid rats. Also, the elevation in monoamine levels, in hyperthyroid state, may be attributed to stimulated their synthesis and receptors [44-46]. Collectively, I 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. Future studies should be focused on identifying the genomic actions of THs disorders across the developmental time and brain region.

CONFLICT OF INTEREST

The author declares that no competing financial interests exist.

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