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Maternal Thyroid Hormones and Ageing Process

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SHORT COMMUNICATION

The regular levels of maternal thyroid hormones (THs) during the gestation show a fundamental action in the developing brain of fetuses, neonates (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v, 2018a-r; Ahmed and Ahmed, 2012; Ahmed et al., 2008; 2010; 2012; 2013a,b, 2014, 2015a,b, 2018a,b; Ahmed and Incerpi, 2013; Van Herck et al., 2013; Ahmed and El-Gareib, 2014, Incerpi et al., 2014; Candelotti et al., 2015; De Vito et al., 2015; El-Ghareeb et al., 2016; Ahmed and El-Gareib, 2017), youth, and adult (Hollowell et al., 2002; Gussekloo et al., 2004; Razvi et al., 2008; Cooper and Biondi, 2012; Gesing et al., 2012; Aggarwal and Razvi, 2013; Gesing, 2015; Pasqualetti et al., 2016).

On the other hand, the prevalence of subclinical hypothyroidism [the levels of free thyroxine (FT4) and free triiodothyronine (FT3) is normal but the level of thyroid-stimulating hormone (TSH) is increased)] is more frequently in the elderly than in the neonates (Gesing, 2015). These disturbances can increase the risk of longevity (Cooper and Biondi, 2012; Kahapola-Arachchige et al., 2012; Gesing, 2015; Hennessey et al., 2015). In patients <65 years, there are direct associations between the subclinical hypothyroidism and cardiovascular disorders (both ischemic heart disease and stroke) (Rodondi et al., 2010; Pasqualetti et al., 2013 & 2016). In mild thyroid dysfunctions with the ageing process, several neurological diseases such as cognitive defects and dementia can be developed and progressed (Kaddurah-daouk et al., 2010; Ge et al., 2012; Tognini et al., 2014; Pasqualetti et al., 2015). More importantly, data from several animal models proposed that a strong adverse link between the level of THs and lifespan/longevity (Buffenstein et al., 2001; Bowers et al., 2013). The distortion in the levels of THs in the brain (basal forebrain cholinergic neurons) due to some modifications in the activities of deiodinases could cause Alzheimer disease (AD; abnormal deposition or aggregations of beta-amyloid precursor protein (APP) in the brain) (Mafrica and Fodale, 2008). This might be related to the clinical appearance of dementia (Davis et al., 2004). On the other hand, in mice brain and neuronal cell lines, the treatment by T3 can inhibit the expression of APP gene at promoter region (histone H3 acetylation and methylation) (Belakavadi et al., 2011).

Based on the above evidences, the equilibrium in the maternal hypothalamus-pituitary-thyroid axis (HPTA) shows significant roles during the development and adulthood periods. The previous observations raise the possibility that the frequency of thyroid disorders may upsurge with age. In addition, the dysregulations in the maternal THs may delay the development of neural connections, and neurobehavioral responses. These sustained problems may confirm the presence of ageing disorders in the future. However, these alterations may depend on the nature of population, sex, age, nutrition, and ethnicity. Thus, serum T4, T3 and TSH testing should be done in both mothers and their neonates during the early suckling period so that any thyroid dysfunctions can be identified early and treated to avoid the ageing disorders in adult. In addition, supplementary studies are desired to identify whether the problems of maternal thyroid dysfunctions may increase the risk of persistent conflicts and ageing defects. These arguments require more examinations and special attentions in elderly persons.

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