Dysfunction of Maternal Thyroid Hormones and Psychiatric Symptoms

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Commentary

The steady in the functions of gestational thyroid hormones (THs) displays major actions in the developing brain in particular the development and differentiation of neurons, oligodendrocytes, astrocytes and microglial cells (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-u & 2018a,b; Ahmed et al., 2010, 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; Moog et al., 2017).

On the other hand, in the developing or mature central nervous system (CNS), any deviations in the activities of THs (hypothyroidism or hyperthyroidism) can impair the development and function of the neurons and microglial cells (an immune cell population in the CNS) (Duntas and Maillis, 2013; Noda, 2015). These impairments might increase the risk of mental/psychiatric disorders (neurological and behavioral defects) including depression, schizophrenia, spasticity, anxiety, bipolar disorder, and ataxia (Thompson and Potter, 2000; Noda, 2015). In addition, these changes are irreversible and permanent (Di Liegro, 2008; Henrichs et al., 2010; Noda, 2015). More importantly, there are several psychiatric symptoms during the hypothyroidism such as anergia, psychosis, mania, apathy, mood instability, hypersomnia, dementia (Osterweil et al., 1992; Goh et al., 2014), attentional abnormalities and delaying the psychomotor (Awad, 2000). These symptoms with the cognitive impairment might be increased with the age progress (Mallett et al., 1995; Dugbartey, 1998). On the other hand, there are other psychiatric symptoms during the hyperthyroidism such as depression and anxiety (Demet et al., 2002), abnormalities in the appetite and sleep, emotional lability, exaggerated sensitivity to noise, distractible overactivity, and impatience or irritability (Awad, 2000). Additionally, in the elderly, Alzheimer’s disease (AD), dementia, and cognitive defects were observed in the subclinical hyperthyroidism (Kalmijn et al., 2000; van Osch et al., 2004; Wijsman et al., 2013). From the previous results, it can be inferred that the hyperthyroidism or hypothyroidism may elevate the possibility of neurodegeneration and cognitive defects. These changes can be attributed to the effect of thyroid disorders (hyperthyroidism or hypothyroidism) on the immune system. Hypothyroidism can cause several abnormalities as the following (Klecha et al., 2000 & 2006; De Vito et al., 2011): (1) increase the levels of reactive oxygen species (ROS); (2) augmentation of phagocytosis; (3) elevate the activities of the proinflammatory markers such as interleukin-1β and macrophage inflammatory protein-1α; (4) decrease the activities of the antioxidant enzymes; (5) reduce the lymphocyte proliferation parameters; (6) diminish the cell migration and the production of antibody; and (7) decrease the immune response. As well, hyperthyroidism can reduce the activities of the proinflammatory markers such as monocytes and macrophages (Klecha et al., 2008; De Vito et al., 2012). According these reports, it is probable that indirect roles of THs via glial cells are significant for the activity of neuronal cells and their defects may at least in part, cause several psychiatric symptoms (Noda, 2015).
Based on the above suggestions, the regulation in the maternal hypothalamus-pituitary-thyroid axis (HPTA) displays vital actions during the development of CNS. In addition, the dysregulations in the activities of maternal THs (hypothyroidism or hyperthyroidism) may interrupt the development of CNS and cause persistent neural disorders, mental retardation, and several psychiatric symptoms. Additional studies are required to elucidate the interactions between, thyroid dysfunctions, microglia, and neuropsychological disorders.

**References**


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