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Maternal Hypothyroidism and Neonatal Depression: Current Perspective

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COMMENTARY

The maternal thyroid hormones (THs) facilitate significant actions on central nervous system (CNS) during the prenatal and postnatal development and whole life(El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-u & 2018a-c; Ahmed et al., 2010, 2013a,b, 2014, 2015a,b &2018a,b; Ahmed and Incerpi, 2013;Van Hercket al., 2013; Ahmed andEl-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).On the other hand, postpartum thyroid disorders(hypothyroidism) are associated with the mood disorder and postpartum depression (PPD),and vice versa (Harris, 1999; Sylvén et al., 2013; Le Donne et al., 2017; Zhou et al., 2017). Several investigators reported that postpartum mood dysfunctions (PPD or severe psychosis) are the main feature of maternal psychiatric morbidity (Miller, 2002; Fergerson et al., 2002; Bloch et al., 2003; Dennis, 2004; Henshaw et al., 2004; Le Donne et al., 2012; Mento et al., 2017).During the perinatal and adulthood periods, TH deficiency (hypothyroidism) can cause the following (Miller et al., 2007; Samuels et al., 2007): **(1)** impair the neurotransmission activity in different brain regions particularly the hippocampus (learning and memory); **(2)**delay the cellular proliferation and migration (Leonard and Farwell, 1997; Farwell et al., 2006; Ahmed et al., 2008); **(3)**neurological and behavioral deficits; **(4)** irreversible mental retardation; **(5)** irreversible motor dysfunctions; and **(6)** impair the memory and learning. As well, hypothyroidism can prompt the psychological distress, depressive symptoms, and cognitive dysfunction (Constant et al., 2005; Bould et al., 2011; Mowla et al., 2011). The severity of depression and depressive episodes can be associated with the reduction in the levels of 3,5,3'-triiodothyronine (T3) (Saxena et al., 2000; Stipcevićet al, 2008) and reduction or elevation in the level of thyroxine (T4) (Kirkegaard and Faber, 1998; Saxena et al., 2000). In addition, a reduction in the levels of thyroid-stimulating hormone (TSH) and an elevation in the levels of T4 could cause depression in young men (Forman-Hoffman and Philibert, 2006). However, in women, an elevation in the levels of T4 only could cause some depression syndrome (Forman-Hoffman and Philibert, 2006). There are several mechanisms can be ascribed these disturbances as the following: **(1)** THs can cause catecholamine (norepinephrine) deficit via stimulation the post-synaptic β-adrenergic receptors in the different brain regions (cerebral cortex and cerebellum) (Atterwill et al., 1984; Ahmed et al., 2008); and **(2)** THs can reduce the impulse rate of neurons and the monoamine neurotransmission like serotonin (5-HT) (Heal and Smith, 1988; Ahmed et al., 2008; Belmaker and Agam 2008). These defects may reverse with adequate therapy such as the serotoninergic and adrenergic antidepressants (Łojko and Rybakowski, 2007; MacQueen, 2009; Brühl et al., 2011).

From the previousdata, the maternal hypothyroidism and depressive symptoms are very important social health problems. In addition, the maternal hypothyroidism may cause several developmental defects and growth retardation in the fetal and neonatal central nervous system (CNS). This retardation may induce several mood disorders in particular the depression and cognitive and behavioral dysfunctions. More importantly, the neonatal depression due to the maternal hypothyroidism may cause noteworthy social and economic problems. Thus, additional research is desired not only to determine the interactions between the activities of maternal thyroid gland and developing brain in depressive disturbances but also to evaluate the genetic variations in deiodinases

and neuroimaging. This strategy can assist in the treatment of depressive disorders during the development.

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