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Maternal Thyroid Function and Placental Hemodynamics

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LETTER TO EDITOR

The concentrations of thyroid hormones (THs) are necessary for the normal development(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) in particular the placentation (Barber et al., 2005; Krassas et al., 2010; Loubiere et al., 2010; Aghajanova et al., 2011; Patel et al., 2011; Barjaktarovic et al., 2017). On account of the normal placentation (interstitial invasion of fetal trophoblast cells into maternal decidua and endovascular trophoblast (EVT) invasion into maternal spiral arteries, Cartwright et al., 2010) is vital for maintaining the gestation and for the optimal fetal development; supply nutrients, exchange gases and eliminate the metabolic waste products, any disorders in the placental functions can cause several pregnancy complications such as the preeclampsia, premature delivery, and fetal growth restriction (Carter, 2012; Guttmacher et al., 2014; Fowden et al., 2015). These conditions may impair the fetal blood supply, deteriorate the placental hemodynamics and cause maternal and perinatal mortality and morbidity (Kovo et al., 2011; Bilano et al., 2014; Odibo et al., 2014; Vinnars et al., 2015; Barjaktarovic et al., 2017). THs can regulate the trophoblast proliferation, motility and EVT invasion by initiating the secretion of several growth factors (epidermal growth factor (EGF), and vascular endothelial growth factor-A (VEGF-A)), cytokines (tumor necrosis factor alpha (TNF-α), and interleukin 10 (IL-10)), and angiogenesis of maternofetal placental vessels such as angiopoietin 2 (Ang-2) and angiogenin (Matsuo et al., 1993; Oki et al., 2004; Barber et al., 2005; Vasilopoulou et al., 2014). These processes can be mediated by the normal expression of thyroid receptors (TRs; α and β) and the normal activities of thyroid transporters (THTs) in the trophoblast cells (Barber et al., 2005; Loubiere et al., 2010; Aghajanova et al., 2011; Patel et al., 2011). On the other hand, impair the normal placentation in early pregnancy (Cartwright et al., 2010) due to hyperthyroidism could cause fetal growth restriction (Medici et al., 2013; Haddow et al., 2014) and preeclampsia (Aggarawal et al., 2014; Medici et al., 2014). In addition, abnormal placentation in early pregnancy (Cartwright et al., 2010) due to hypothyroidism could cause premature delivery (Korevaar et al., 2013; Sheehan et al., 2015). From the clinical data, there are association between the thyroid disorders and placental dysfunction, and pregnancy complications (Korevaar et al., 2013; Medici et al., 2013, 2014; Odibo et al., 2014).

From the aforementioned clarifications and the current opinion, it can be inferred that the early normal activities of the maternal thyroid gland may be a regulator of the normal placentation. In addition, maternal thyroid dysfunctions (hyperthyroidism or hypothyroidism) can impair the placental growth factors, cytokines, and blood supply. The placental dysfunction may increase the risk of preeclampsia, fetal growth restriction and mortality. Thus, treatment of thyroid disorders before or during the gestation may decrease the risk of pregnancy complications. Additional investigations are necessary not only to examine the biological and molecular mechanisms between the thyroid dysfunction and placental disorders, preeclampsia and premature delivery but also to compare the data with the clinical studies.

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