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Maternal Hypothyroidism and Developing Hyperhomocysteinemia

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BRIEF REPORT

A regular transfer of thyroid hormones (THs) from the placenta to fetuses/neonates is needed for the development(El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v, 2018a-l; Ahmed and Ahmed, 2012; Ahmed et al., 2008; 2010; 2012; 2013a,b, 2014, 2015a,b, 2018a,b; Ahmed and Incerpi, 2013;Van Hercket 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), particularly the levels of homocysteine and the concentrations of lipid profile (Catargi et al., 2009; Saleh, 2015).In addition, the number of studies has displayed relations between the hypothyroidism and increased the levels of homocysteine (hyperhomocysteinemia), lipid abnormalities, anda dynamic atherogenic state(Catargi et al., 2009; Yang et al., 2015).There are associations between the clinical hypothyroidism (Colleran et al., 2005) orovert hypothyroidism (Sengül et al., 2004) with the hyperhomocysteinemia. This abnormal state can increase the risk of atherosclerotic heart diseases (cardiovascular diseases) such as coronary artery disease (CAD) (Sengül et al., 2004; Catargi et al., 2009; Bamashmooset al., 2013; Morris et al., 2013). More importantly, Yang et al. (2015) reported that hyperho-mocysteinemia-induced with hypothyroidism or subclinical hypothyroidism can increase the homeostatic index of insulin resistance (HOMA-IR).IR can perturb the insulin pathway, glucose metabolism, adipokine production, and lipogenesis in several organs such as the adipose tissue, muscles and liver (Schachter et al., 2003; Wang et al., 2005; Li et al., 2008; Yilmaz et al., 2008; Yang et al., 2015). Several studies have demonstrated that IR can cause dysfunction in the vascular endothelium, initiate atherosclerosis and increase the risk of CAD (Bornfeldt and Tabas, 2011; Karrowni et al., 2013; He et al., 2010; Li et al., 2013). Hyperhomocysteinemia can increase the cardiovascular diseases by several mechanisms as the following (Ji et al., 2004; Wang et al., 2005; Edirimanne et al., 2007; Cui et al., 2008; Chiang et al., 2011; Wang et al., 2012; Deyneli et al., 2014; Yang et al., 2015): **(1)** oxidative stress; **(2)** platelet aggregation; **(3)** endoplasmic reticulum stress; **(4)** endothelial dysfunction; **(5)** smooth muscle cell proliferation; and **(6)** augmenting the responsiveness of monocytes to inflammatory stimuli. However, the underlying effects of hypothyroidism and hyperhomocysteinemia on the IR remain indistinguishable.

From the above investigations, it is worth mentioning that the present report throws up some vital observations. The regular levels of maternal THs during the pregnancy may regulate the levels of homocysteine and the developing fetuses and neonates. In addition, the disorders in the activities of THs (hypothyroidism) during the gestation may cause hyperhomocysteinemia and increase the risk of atherosclerosis, strokes and heart attacks. This disruption may delay the fetal and neonatal development. In general, this report suggested that pregnant dams should be encouraged to evade the hypothyroxinaemia and hyperhom-ocysteinemia by following the profile of thyroid gland and the levels of homocysteine during the gestation. More information is wanted to evaluate these investigations and to follow the destructive effects of maternofetal thyroid disorders and hyperhomocysteinemiaon the IR and fetal and neonatal development. In addition, the connection between the developmental and biochemical studies is required.

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