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Maternal Hypothyroidism and Neonatal Obesity

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COMMENTARY

A normal transportation of thyroid hormones (THs) from pregnant women to their fetuses/neonates is required for the normal 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 energy homeostasis, appetite, basal metabolic rate (BMR) and metabolic mechanisms (Laurberg et al., 2012; Kahr et al., 2016; Knight et al., 2016).On the other hand, several studies have shown a relationship between the subclinical hypothyroidism during gestation and an increase in the risk of gestational diabetes (Cleary-Goldman et al., 2008; Karakosta et al., 2012; Tudela et al., 2012; Chen et al., 2014; Toulis et al., 2014; Knight et al., 2016). In addition, mild maternal thyroid insufficiency (isolated hypothyroxinaemia; reduction in the level of free thyroxine (FT4) and an increase in the free triiodothyronine (FT3) to FT4 ratios) with poor obstetric outcomes during gestation can cause several adverse metabolic defects such as insulin resistance, glycaemia, obesity, and lipid profile disorders (hyper-lipidaemia) (De Pergola et al., 2007; Roos et al., 2007; Tian et al., 2007; Cleary-Goldman et al., 2008; Garduno-Garcia et al., 2010; Bassols et al., 2011; Dennedy et al., 2012; Laurberg et al., 2012; Vrijkotte et al., 2012; Mehran et al., 2014; Roef et al., 2014). More interestingly, the association between the maternal hypo thyroxinaemia and obesity was observed in iodine-deficient pregnant women (Gowachirapant et al., 2014). It was supposed that obesity-induced the activities of peripheral deiodinases(Ds) increasing the energy expenditure, the conversion of FT4 to FT3 and eventually the FT3 to FT4 ratio (Haddow et al., 2015). Also, Kahr et al. (2016) reported the maternal and childhood obesity was increased with the variations in the levels of both maternal and neonatal THs. More importantly, ameliorating the function of the thyroid gland might be by improvement the lifestyle and diet (Reinehr and Andler, 2002; Reinehr et al., 2006; Laurberg et al., 2012), gastric bypass surgery (Fazylov et al., 2008) or gastric banding (Dall'Asta et al., 2010). Also, the complex interaction between the hypothalamic-pituitary-thyroid axis (HPTA) and adipocyte cytokines should be monitored to control the progress of the obesity (Feldt-Rasmussen, 2007; Boelen et al., 2008; Obregon, 2008; Endo et al., 2012; Sainsbury and Zhang, 2012).

The normal functions of the maternal thyroid gland during pregnancy may be vital for the body composition and the neonatal development. In addition, any disruption in the levels of THs during the gestation may cause obesity and suppress the neonatal development. Thus, women have to avoid the hypothyroxinemia and any excess in the body weight gain. Maintaining normoglycaemia during pregnancy may play an important role in a healthy life for the newborns. However, additional studies are essential to replicate these observations and to explore the harmful effects of maternofetal thyroid dysfunction (hypothyroidism or isolated hypothyroxinaemia) and obesity (adverse metabolic parameters) on long-term growth and neonatal development. Furthermore, the connection between the molecular and epidemiological studies is required. This argument is still ambiguous because of the difficulties of the direct observation of thyroid dysfunction on obesity.

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