Maternofetal Thyroid Hormones and Risk of Diabetes

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The regulation between the thyroid hormones (THs) (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-h; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b; Ahmed and Ahmed, 2012; 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; End endijk et al., 2017; Gigena et al., 2017) and insulin (Ahmed, 2013, 2016a,b) is required during the early development. In addition, several investigations support the view that THs, glucose homeostasis (Fain and Bahouth, 1998; Candelotti et al., 2015), insulin sensitivity (Scherer, 2006), insulin growth factors (IGFs), and adipocytokines (Akin et al., 2009; Ahmed, 2013 & 2015b) might have an important role in perinatal development (Ahmed et al., 2008, 2010 & 2012; Ozard et al., 2012). In particular, the levels of maternal blood glucose and insulin are influenced by the thyroid functions and thyroid-binding globulin (TBG) (Fister et al., 2009; Lazarus, 2011; Yang et al., 2016).

On the other hand, there is a connection between the thyroid disorders/diseases and gestational diabetes mellitus (GDM) (Karakosta et al., 2012; Tudela et al., 2012; Toulis et al., 2014; Maleki and Tavosi, 2015; Oguz et al., 2015; Fatima et al., 2016). Also, the decrease in the levels of free thyroxine (FT4) during the gestation, particularly in the 2nd and 3rd trimesters (Olivieri et al., 2000; Cleary-Goldman et al., 2008; Velkoska Nakova et al., 2010; Oguz et al., 2015; Haddow et al., 2015 & 2016) but not in the 1st trimester (Agarwal et al., 2006; Cleary-Goldman et al., 2008; Oguz et al., 2015; Haddow et al., 2015 & 2016) might increase the possibility of developing GDM (Yang et al., 2016), and obesity (Laurberg et al., 2012). These observations might be compensated by augmented the availability of placental triiodothyronine (T3)/ thyroxine (T4) via elevation the activity of maternofetal THs-transporters and/or diminution the activity of maternofetal THs metabolism by deiodinases (Guzman-Gutiérrez et al., 2014). On the other hand, increase the body weight during the severe hypothyroidism might be attributed to expansion the water compartment and accumulating the water - binding glycosaminoglycans in skin and several tissues causing myxoedematous (Laurberg et al., 2012). An alternative explanation for these findings is ascribed to increase the activity of peripheral deiodinase and the caloric intake (characterized by high weight) (Ashoor et al., 2010; Mannisto et al., 2011; Bestwick et al., 2014; Haddow et al., 2015 & 2016). Thus, it seems that over-nutrition might be disposed to elevate the activities of deiodinases (endogenous glucose production) and ectopic fats (insulin resistance) (Shulman, 2014; Haddow et al., 2016). On the basis of these data, it can be decided that the maternal thyroid dysfunction (hypothyroidism) may distort the communication between the maternofetal thyroid-insulin axis. In the future, additional studies are necessary to clarify the association between the maternofetal thyroid-pituitary functions [FT4, free triiodothyronine (FT3) and thyroid stimulating hormone (TSH)] and thyroid sensitivity, and their receptors during the prenatal and postnatal periods. Also, further studies are necessary to explore the connection between THs replacement therapy and obesity.

CONFLICT OF INTEREST

The author declares that no competing financial interests exist.
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REFERENCES


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