



## Maternal Hypothyroidism-Developing Dyslipidemia: What is the Connection?

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### COMMENTARY

Maternal thyroid hormones (THs) are essential for the developing newborns (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v, 2018a-s; Ahmed and Ahmed, 2012; Ahmed et al., 2013a,b, 2014, 2015a,b, 2018a,b; 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), especially the thermogenesis, lipogenesis, and lipid contents (fatty acids composition and phospholipids in the cell membrane) (Prasad & Kumar, 2005; Catargi et al., 2009; Zhu and Chang, 2010; Saleh, 2015).

On the other hand, several investigations have revealed the link between the hypothyroidism and dyslipidemia (Stone, 1994; Beyer et al., 1998; Tsimihodimos et al., 1999; Celiket al., 2012; Benetti-Pinto et al., 2013; Raza and Mahmood, 2013). In subclinical hypothyroidism, numerous studies have reported that dyslipidemia might be accompanied by the following: (1) elevations in the levels of triglycerides (TGs) (Milionis et al., 2005; Toruner et al., 2008); (2) reductions in the levels of high-density lipoprotein cholesterol (HDL-C) (Erdem et al., 2008); (3) elevations in the levels of low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) (Tanis et al., 1996; Danese et al., 2000; Luboshitzky et al., 2002; Jung et al., 2003; Monzani et al., 2004; Walsh et al., 2005; Erem, 2006; Duman et al., 2007); (4) decrease the ratio of cholesterol/TGs (Brenta et al., 2007); (5) increase the levels of apolipoprotein B (ApoB) (Efstathiadou et al., 2001); and (6) increase the levels of lipoprotein(a) [Lp(a)] and apolipoprotein(a) [apo(a)] (Kung et al., 1995; Efstathiadou et al., 2001; Caraccio et al., 2002; Milionis et al., 2003; Iqbal et al., 2006). More importantly, the above disruptions can increase the risk of the cardiovascular diseases (Raza and Mahmood, 2013). In fact, Hueston and Pearson (2004) observed that the subclinical hypothyroidism did not have any effect on the lipid profile if we adjust the race, sex, age and use of lipid-lowering drugs. In addition, Knudsen et al (2005) recorded that any change in the activities of the thyroid gland can be harmful to the body mass index (BMI) and cause obesity. On the other hand, the presence of insulin resistance can reinforce the effect of hypothyroidism on the lipid profile (Bakker et al., 2001; Chubb et al., 2005).

On the basis of these observations, I hypothesized the following: (1) THs, perhaps of maternal origin, play vital roles in the homeostasis of the fetal lipid profile; (2) the maternal thyroid disorders (hypothyroidism) may perturb the lipid homeostasis (contents, metabolism and distributions) in liver and adipose tissues during the gestation and lactation; (3) the maternal hypothyroidism may cause fetal/neonatal dyslipidemia; and (4) the maternal hypothyroidism may delay the fetal/neonatal development. Thus, adjusting the levels of maternal, fetal and neonatal THs and thyroid-stimulating hormone (TSH) can easily prevent this disturbance during the gestation and lactation. Future studies are required to understand the connection between the genomic and non-genomic actions of THs during the abnormal conditions (hypothyroidism or hyperthyroidism during the gestation and lactation) and fetal dyslipidemia. This view will assist to prevent not only the thyroid dysfunction during the critical developmental periods but also the cardiovascular disease and obesity (predominant diseases in our life). Thus, the association between the molecular and developmental investigations is warranted.

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**Citation:** R.G. Ahmed, " Maternal Hypothyroidism-Developing Dyslipidemia: What is the Connection?" *ARC Journal of Pharmaceutical Sciences (AJPS)*, vol. 4, no. 1, p. 1-6, 2018. <http://dx.doi.org/10.20431/2455-1538.0401001>

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