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Maternal Thyroid Hormones and Neonatal Appetite

Ahmed R.G

Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt

Corresponding Author:** ***Ahmed R.G, *Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt.* ***Email:*** *ahmedragab08@gmail.com*

COMMENTARY

Fundamental actions of maternal thyroid hormones (THs) during the gestation are required for a normal fetal and neonatal development and growth (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-u & 2018a,b; Ahmed et al., 2010, 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; Moog et al., 2017) in particular the energy, food intake, thermogenesis, body weight, metabolism of inorganic ions and nutrient, and metabolic rate and energy (Wolf et al., 1996;Ogilvy-Stuart, 2002; Manji et al., 2006; A min et al., 2011; Ahmed, 2013; Männistö et al., 2013). In addition, the balance in the hypothalamic-pituitary-thyroid axis (HPTA) has necessary roles for the critical developmental and functional periods(regulation of appetite and energy expenditure) (Ahmed et al., 2008; Ahmed and El-Gareib, 2017).The signaling of this axis [hypothalamic thyrotropin-releasing hormone (TRH), thyroid-stimulating hormone (TSH), 3,5,3'-triiodothyronine(T3) and thyroxine (T4)] can regulate the food intake process (Lin et al., 1983; Ishii et al., 2003 & 2008; Kong et al., 2004). More importantly, regulation of the appetite might be depending on the local actions of THs in the central nervous system (CNS) (Amin et al., 2011).In addition, the activity of deiodinase 3 (D3) can regulate the food intake process and energy homeostasis through mediation the levels of THs (Bianco et al., 2002; Barrett et al., 2007). Activation of 5'-adenosinemonophosphate-activated protein kina se (AMPK; sensor of cellular energy home ostasis) in the arcuate nucleus (ARC) simulates the process of food intake (Minokoshi et al., 2008). Expression of G protein-coupled trace amine-associated receptor 1 (TAAR1) in the hypothalamus can regulate the activation of energy homeostasis (Dhillo et al., 2009).On the other hand, thyroid disorders in clinical reports have several disturbances actions on the body weight and appetite (Amin et al., 2011). In hypothyroidism, the levels of basal energy expenditure were decreased (Wolf et al., 1996), and the levels of weight gain were increased (Iossa et al., 1996; Manji et al., 2006). In opposite, the levels of basal energy expenditure were increased and the levels of weight gain were decreased during the hyperthyroidism (Alton and O'Malley, 1985; Pijl et al., 2001; Klieverik e al., 2009). Also, food deprivation may increase the risk of hypothyroidism; decrease the release of T3 and T4 in rodents (Légràdi et al., 1997) and human (Chan et al., 2003).

From the previous considerations, the fetal and neonatal development (appetite, food intake, metabolic rate and body weight) may depend on the activities of maternal HPTA. As well, the dysfunctions in the activities of HPTA may disturb the appetite and food intake, and delay the fetal and neonatal development. However, the physiological and developmental relevance of these defects remains unidentified. Enhancement our understanding of the action of the HPTA and THs in appetite and food intake may recognize novel goals about the anti-obesity agents. Additional investigations are essential to explore these interactions during the gestation and lactation periods. Further work is warranted to recognize the roles of maternal thyroid receptors (TR; α and β) on the neonatal appetite, food intake and metabolic rate.

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