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Maternal Thyroid Hormones Trajectories and Neonatal Behavioral Disorders

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COMMENTARY

The normal adequate of the maternal thyroid hormones (THs) (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-t; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b; Ahmed and Ahmed, 2012; 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; Endendijk et al., 2017; Gigena et al., 2017) is necessary for the fetal/neonatal brain development (Henrichs et al., 2013). During the normal gestation, the level of thyrotropin (TSH) was increased and the level of free thyroxine (FT4) was correspondingly decreased (Stricker et al., 2007; Ahmed et al., 2008). These alterations are within the normal range and may be predictive of neonatal development (Pop et al., 1995, 1999 &2003; Ahmed et al., 2008). However, mothers with problems in the THs before and during pregnancy had neonates with the lowest mental and motor development scores (Pop et al., 1995, 1999 &2003; Endendijk et al., 2017). In addition, it seems that mild increases in TSH might delay the fetal brain development and consequent attention disorders or problems, specifically in boys (Glinoer et al., 1993; Endendijk et al., 2017).

Several studies reported that the disturbance in the thyroid functions during the gestation (maternal hypothyroxinemia; low FT4 and normal TSH levels) can cause neonatal cognitive disorders such as depression or anxiety (internalizing problems), and several cognitive problems such as aggression, attention problems or hyperactivity (externalizing problems) (Brownlie et al., 2004; Hagberg et al., 2010; Henrichs et al., 2010; Van Mil et al., 2012; Bornstein et al., 2013; Henrichs et al., 2013; Endendijk et al., 2017). In addition, these disorders can extend to delay the language pronunciation (Henrichs et al., 2010) and impair the executive functioning (Van Mil et al., 2012; Endendijk et al., 2017). In human, the maternal thyroid dysfunction (hypothyroxinemia, higher TSH and thyroid-peroxidase antibody (TPO) levels) can stimulate several externalizing problems in their infants (Ghassabian et al., 2011, 2012 & 2014). On the other hand, the connections between the maternal THs, neonatal cognitive development and child sex type are almost unidentified (Henrichs et al., 2013). Females are more susceptible to developing internalizing problems while males are reliably more vulnerable to developing externalizing problems (biological or environmental risk factors) (Zahn - Waxler et al., 2008). However, this link is weak and the fundamental mechanisms undistinguishable (Endendijk et al., 2017).

On the basis of these data, it can be concluded that the maternal thyroid dysfunctions (maternal hypothyroidism) during the gestation may cause several disorders in the neonatal cognitive and social behaviors. These effects might depend on the severity of thyroid disorders, sex type and developmental period. Additional studies are necessary to examine whether the individual variations in THs trajectories (FT4 and TSH levels) during the gestation will be a better indicator of neonatal cognitive behavioral disorders than maternal THs levels evaluated at one trimester of pregnancy. In addition, several studies are warranted to examine whether there are sex differences in the interactions between

the maternal THs trajectories and neonatal cognitive behavioral disorders.

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