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Maternal Thyroid-Adrenal Dysfunction and Fetal-Neonatal Depression

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COMMENTARY

The coordination between the thyroid hormones (THs) (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., 2008; 2010; 2012; 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; Endendijk et al., 2017; Gigena et al., 2017) and glucocorticoids (Ahmed, 2016b) is necessary for the maternal-fetal communications, the intrauterine/ extrauterine homeostasis, and the initiation of parturition (Fisher , 1997; Mesiano and Jaffe, 1997; Ng, 2000; Ishimoto and Jaffe, 2011; Hillman et al., 2012; Chung, 2014). After labor, the fetal hormones of thyroid [thyroxine (T4) and triiodothyronine (T3)] and adrenal cortex (cortisol) quickly respond to avoid the hypocalcemia, hypoglycemia, and hypothermia due to the absence of the placental supply of energy and nutrients (Chung, 2014). The cortisol surge (increase the level of cortisol with the progress of the gestation) augments the conversion of T4 to T3 (Kronenberg et al., 2008).

On the other hand, the endocrine anomalies may increase the risk of morbidity and several developmental disorders (Watterberg, 2004; La Gamma et al., 2009). Dysfunction in the hypothalamus-pituitary-thyroid axis (HPTA) and the hypothalamic-pituitary-adrenal axis (HPAA) can cause the major depressive disorders (Jackson, 1998; Kirkegaard and Faber, 1998; Brouwer et al., 2005). In depression, the dysfunction in HPTA can be attributed to **(1)** deficiency in the concentrations of serotonin and/or norepinephrine (Kirkegaard and Faber, 1998); and **(2)** elevation in the level of cortisol (hypercortisolism) (Brouwer et al., 2005). In major depression, the stimulation of HPAA can be explained by **(1)** the glucocorticoid resistance (Brouwer et al., 2005); and **(2)** hypothalamic overdrive of corticotrophin-releasing hormone (CRH) (Holsboer and Barden, 1996). In disagreement with these data, some authors did not find association between the major depression and the disorders in the HPTA (Haggerty et al., 1987; Sullivan et al., 1997; Engum et al., 2002) or HPAA (Rubin et al., 1987; Oldehinkel et al., 2001; Young et al., 2001). The inconsistency in these associations may depend on the status of the patient (in/outpatient), unipolar/bipolar depression, sex type, antidepressant medications (short- or long-term) (Dilsaver and Greden, 1985; Kraus et al., 1987; Baumgartner et al., 1988; Barden et al., 1995; McCowen et al., 1997; Bauer et al., 2002; Schule et al., 2003; Brouwer et al., 2005), and small sample sizes (Kirkegaard et al., 1990; Michelson et al., 1996; Coiro et al., 1998; Young et al., 2001; Brouwer et al., 2005). On the basis of these data, it can be decided that any disorders in the communications between the HPTA and HPAA during the gestation may cause a depression in both mothers and fetuses/neonates. Thus, additional studies are necessary to understand the potential associations between the fetal/perinatal adrenal-thyroid disorders and depression. Future examinations are wanted to discover whether the effect of maternal thyroid hormone replacement therapy on the developmental

thyroid-adrenal axis play a role in modifying the signaling pathways to enhance the atypical, melancholic or severe depression during the perinatal period.

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