Maternal Hyperthyroidism and Neonatal Testicular Dysfunction

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HYPOTHESIS

The functions of hypothalamic-pituitary-thyroid axis (HPTA) are warranted for the developing newborns (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v, 2018a-r; Ahmed and Ahmed, 2012; Ahmed et al., 2008; 2010; 2012; 2013a,b, 2014, 2015a,b, 2018a,b; 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), specifically the developing testes (Gao et al., 2014; Rijntjes et al., 2017; Ahmed, 2018a). During the different developmental periods, the 3,5,3’-triiodothyronine (T3) and its receptors and transporters can regulate the proliferation of the Sertoli and Leydig cells, steroidogenesis, and testicular development and maturation (Buzzard et al., 2000; Krassas and Perros, 2003; Maran, 2003; Haider, 2004; Holsberger et al., 2005; Mendis-Handagama et al., 2007, and Rijntjes et al. (2008) who demonstrated that daily administration of T3 has been revealed the following: (1) reduction in the number of Sertoli cells; (2) premature cessation for the proliferation of Sertoli cells; and (3) progress in the development of Leydig cell progenitors and in the cell population of adult-type Leydig cell. On the other hand, the treatment of thyrotoxicosis might improve the density and motility of the sperm, but no change was observed in the morphology of the sperm (Krassas et al., 2002) or the erectile complaints (Krassas et al., 2008).

In the light of the above considerations, my data hypothesize several views. The adequate functioning of the maternal HPTA plays a significant role in the development of the neonatal testis (spermatogenesis, sperm mobility and eventually fertility). In addition, the maternal hyperthyroidism may directly or indirectly delay the morphogenesis and functional development of the neonatal testis. This may perturb the structure of the sperm, decrease the quality, and amount of the semen, and cause infertility. Thus, the management of the maternal hyperthyroidism can be necessary to prevent these syndromes. This would afford improvement in maximizing the fertility consequence. However, it remains to be shown whether the severity of the hyperthyroidism during the gestation may cause more persistent perturbations in the neonatal testes. The molecular, biochemical and developmental mechanisms are required to explore these abnormal conditions.

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