Thyroid hormones (THs) including thyroxine (T4) and 3,5,3′-triiodothyronine (T3) are dynamic for the vertebrate development (Elbaky et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-o; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014, 2015a,b; Ahmed and Ahmed, 2012; Ahmed and Incerpi, 2013; Van Herck et al., 2013; Ahmed and ElGareib, 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), particularly the developing Leydig, germ (steroidogenesis) and Sertoli cells, blood-testis barrier, gap junction and testicular function (van Haaster et al., 1993; Cooke et al., 1994; De Franca et al., 1995; Gareib et al., 1998; Ahmed et al., 2006; Schneider et al., 2009; Shibutani et al., 2009; Auharek and De Franca, 2010; Gao et al., 2014; Rijnjtes et al., 2017). In addition, the testicular cells can express the thyroid transporters, thyroid hormone receptors (TRs; α1, α2, α3, and β1, β2, β3) and deiodinases (Ds; D1, D2 and D3), and can keep the steady levels of THs in the seminiferous epithelium for the spermatogenesis (Buzzard et al., 2000; Jannini et al., 2000; O’Shea and Williams, 2002; Mendis-Handagama and Siril Ariyaratne, 2005; Hernandez et al., 2006; Schneider et al., 2006; Meeker et al., 2007; Wajner et al., 2007; Weng et al., 2007; Krassas et al., 2008 & 2010; Wagner et al., 2009; Pelletier, 2011; Zamoner et al., 2011; Tarulli et al., 2012; Gao et al., 2009 & 2014; Mayerl et al., 2014). More importantly, the coordination between THs, follicle-stimulating hormone (FSH), and luteinizing hormone (LH) might regulate the testicular development (Leydig and Sertoli cell) and function (Gao et al., 2014; Picut et al., 2015; Rijnjtes et al., 2017).

On the other hand, hypothyroidism can disrupt the testicular development and function as the following (Cooke and Meisami, 1991; Cooke et al., 1991; van Haaster et al., 1992; De Franca et al., 1995; Mendis-Handagama et al., 1998; Chaio et al., 2000; Cristovao et al., 2002; Dermott, 2004; Mendis-Handagama et al., 2007; Shibutani et al., 2009; Auharek and De Franca, 2010; Gao et al., 2014; Rijnjtes et al., 2017):

- Stimulate the proliferation of Sertoli cell;
- Delay the differentiation of Sertoli cell;
- Inhibit the blood–testis barrier;
- Increase the neonatal testis weight;
- Hypertrophy of fetal Leydig cells;
- Arrest the formation of neonatal Leydig cell progenitor;
- Inhibit the development of germ cells;
- Reduce the level of plasma testosterone (Jahan et al., 2012);
- Decrease the activity of 17β-hydroxy steroid dehydrogenase (17β-HSD) and post c-AMP pathways;
- Delay the puberty and fertility;
- Delay the spermatogenesis; and
- Increase the apoptotsis of spermatocytes.

On the basis of these investigations, it can be inferred that the steady activities of the maternal THs may noteworthy for the developing testis. Also, the maternal hypothyroidism may disturb the testicular development and function during the postnatal period. Hypothyroidism may act a negative role on the neonatal thyroid-reproductive axis. However, the mechanism of this disorder needs more examinations. Further examinations are crucial to clarify the variations in developing blood-testis barrier, dynamics of gap junctions and Sertoli-germ cell axis due to the effect of goitrogen-induced hypothyroidism model.
CONFLICT OF INTEREST: The author declares that no competing financial interests exist.

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