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Hyperthyroidism and Renal Disorders

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COMMENTARY

The normal levels of thyroid hormones (THs) during the gestation (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v & 2018a-s; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b& 2018a,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) are crucial for the renal homeostasis and development(Iglesias and Diez, 2009; Mariani and Berns, 2012; Dousdampanis et al., 2014; Iglesias et al., 2016).As well, THs adjust the transportation during the proximal convoluted tubule (Basu and Mohapatra, 2012).

Hyperthyroidism can alter the hemodynamic processessuch as increase the sensitivity to β-adrenergic stimulus, the release of renin, the angiogenesis, the nitric oxide (NO) levels and the filtration pressure (Danzi and Klein, 2003; Fazio et al., 2004; Iglesias et al., 2005; Kahaly and Dillmann, 2005; Prisant et al., 2006; Vargas et al., 2012; Rodriguez-Gomez et al., 2013; Koch and Chrousos, 2016). In addition, Hyperthyroidism can alter the glomerular processes (Conger et al., 1989; Graves et al., 1994; den Hollander et al., 2005; Vargas et al., 2006; Syme, 2007; Iglesias et al., 2016) as the following: **(1)** increases the glomerular filtration rate (GFR) and the levels of cystatin C; **(2)** accelerates the chronic kidney disease (CKD); **(3)** Intraglomerular hypertension and hyperfiltration; and **(4)** decreases the serum creatinine level. On the other hand, Hyperthyroidism can vary the tubular processes (Wijkhuisen et al., 1995; Baum et al., 1998; Kumar and Prasad, 2002; Wang et al., 2007; Pothiwala and Levine, 2010) as the following: **(1)** decreases the ability to concentrate urine;**(2)** increases the activity of Na-H exchanger and Na+-K+-ATPase;**(3)** elevates the reabsorption capacity and tubular secretory;**(4)** increases the calcium reabsorption; and **(5)** elevates the reabsorption of sodium and chloride. As well, in hyperthyroid rats, the elevation in the renal and cardiac capillarity and in the vascularization of the mesenteric bed was observed by Rodriguez-Gomez et al. (2013) and Iglesias et al. (2016). On the other hand, the hyper-functioning of thyroid gland can **(1)** decrease the vascular resistance by increasing the vasodilation and reducing the vasoconstriction(Fazio et al., 2004; Kahaly and Dillmann, 2005; Iglesias et al., 2016); **(2)** increase the systolic blood pressure by decreasing the systemic vascular resistance, and elevating the heart rate and cardiac output (Woeber, 1992; Danzi and Klein, 2003; Iglesias et al., 2005; Prisant et al., 2006; Koch and Chrousos, 2016); and **(3)** increase the level of NO causing a hyperdynamic circulation (Iglesias et al., 2016). However, standardization the function of thyroid by therapy of thyrotoxicosis might normalize the renal function (den Hollander et al., 2005). On the basis of these data, it can be inferred that the thyroid dysfunction (hyperthyroidism) may disturb the renal development and function. Further investigations are desired to understand the probable connections between the thyroid disorders (hyperthyroidism) and kidney during the gestation and lactation to avoid unnecessary treatments.

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