



## Maternal Hyperthyroidism and Developing Hematopoiesis Dysfunction

Ahmed R.G.

*Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt*

**\*Corresponding Author:** *Ahmed R.G., Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt*

### SHORT COMMUNICATION

Maternal thyroid hormones [THs; 3,5,3'-triiodothyronine (T3) and thyroxine (T4)] show central roles in the fetal and neonatal development during the gestation and lactation periods (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 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), in particular the hematopoietic cell proliferation and growth, the number of red blood cells (RBCs) and white blood cells (WBCs), and platelet counts (Grymula et al., 2007; Kawa et al., 2010; Pascual and Aranda, 2013; Kandir and Keskin, 2016). In the bone marrow, Kendrick et al. (2008) reported that THs can induce its cellular production. In human early hematopoietic cells, THs might play a significant action in the regulation of the growth (Gruber et al., 1999; Grymula et al., 2007; Kawa et al., 2010). More importantly, the genomic and non-genomic actions of THs might be regulated these mechanisms (Gruber et al., 1999; Milne et al., 1999; Dorshkind and Horseman, 2000; Porter and Mandel, 2000; Bauer et al., 2001; Omazic et al., 2001; Marongiu et al., 2004; Kawa et al., 2010; Barreiro Arcos et al., 2011; Franchini et al., 2011; Kandir and Keskin, 2016). In addition, Kawa et al. (2010) reported that any change in the gene expressions of thyroid receptors (TRs;  $\alpha$  and  $\beta$ ) might change the hematopoietic progenitors.

Previously, Fein and Rivlin (1975) and Corrocher et al. (1981) observed that hyperthyroidism can cause erythrocytosis, increase the number of mononuclear and eosinophil cells, and decrease the number of neutrophil cells. In several cases of hyperthyroidism, Axelrod and Bergman (1951) reported that hyperplasia in all myeloid lineages was noticed in the bone marrow. In addition, there is an association between the overt thyroid dysfunctions and hemorrhage or thrombosis (disruption the 1<sup>st</sup> and 2<sup>nd</sup> physiological hemostasis) (Squizzato et al., 2007). In hyperthyroid rats for 60 days, the number of RBCs did not change while the levels of mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), hematocrit (Hct), and hemoglobin (Hb) was increased (Zahediasl et al., 2010). On the contrary, administration of L-thyroxine (L-T4) to rats for 5 weeks decreased the levels of Hb and Hct, and the number of RBCs (Messarah et al., 2011). These conflicting observations can be attributed to the enhancement the levels of lipid peroxidation (LPO) due to elevated the rate of metabolism during the hyperthyroidism (Yücel et al., 2009; Messarah et al., 2011). These abnormal states may increase the hemolysis and osmotic fragility of erythrocytes (Yücel et al., 2009).

From the above collections, several suggestions may be proposed as the following; (1) the genomic and non-genomic actions of THs may regulate the developmental hematopoiesis system (cell proliferation and growth, and cell cycle), the RBCs, WBCs, and platelet counts; (2) Maternal hyperthyroidism may disrupt the genomic and non-genomic mechanisms of THs; and (3) Maternal hyperthyroidism may cause dysfunction in the developing hematopoietic system because of THs can induce the production of the hematopoietic cell in the bone marrow. Future works are desired to overview the defects of maternal hyperthyroidism on the physiological (RBCs, WBCs, and platelet counts, and thrombocytopoiesis) and developmental hematopoiesis (hematopoietic stem cell proliferation and growth, cell cycle and apoptosis) in experimental animals and humans. Specifically, the additional

studies are required to investigate the significant roles of THs in controlling the development of leukopoiesis, erythropoiesis, and hematopoietic lineages. This may benefit to increase our consideration of the interactions between THs and hematopoiesis during the development and suggest new therapeutic interferences in thyroid disorders. These arguments need more examination.

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**Citation:** R. Ahmed, "Maternal Hyperthyroidism and Developing Hematopoiesis Dysfunction", *International Journal of Research Studies in Biosciences (IJRSB)*, vol. 6, no. 3, pp. 1-5, 2018. <http://dx.doi.org/10.20431/2349-0365.0603001>

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