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Short Communication

Maternal thyroid dysfunction and neonatal cardiac disorders

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Communication

The normal levels of thyroid hormones (THs; thyroxine, T4 & 3,5,3′-triiodo-L-thyronine, T3) are necessary for the normal development [1-48], particularly the fetal and neonatal cardiac growth and development [49]. The actions of THs are facilitated genomically by thyroid receptors (TRs, α and β) and non-genomically at the plasma membrane, in the cytoplasm and in cellular organelles [4,49-55], by stimulation of Na+, K+, Ca2+ and glucose transport, activation of protein kinase C (PKC), protein kinase A (PKA) and mitogen activated and protein kinase (ERK/MAPK) [4]. In addition, the transport of T4 and T3 in and out of cells is controlled by several classes of transmembrane TH-transporters (THTs) [56], including members of the organic anion transporter family (OATP), L-type amino acid transporters (LATs), Na+/Taurocholate cotransporting polypeptide (NTCP), and monocarboxylate transporters (MCTs) [4,49,57,58]. Adding additional complexity, the metabolism of T4 and T3 is regulated by 3 selenoenzyme iodothyronine deiodinases (Ds: D1, D2 and D3) [59-61]. On the other hand, the congenital hypothyroidism can cause the following [49,62-64], (1) congenital heart diseases; (2) diastolic hypertension; (3) reduced cardiac output, stroke volume and a narrow pulse pressure; (4) dilatation and overt heart failure; (5) elevation in the systemic vascular resistance [65-68]. Similarly, the chronic hyperthyroidism can cause the following [49,64]: (1) cardiac hypertrophy; (2) increase in the cardiomyocyte (CM) length rather than width; (3) noticeable diminution in systemic vascular resistance; (4) elevation in the cardiac contractility; (5) systolic hypertension; (6) increase in the cardiac output, venous volume return, blood volume and pulse pressure; and (7) reduction in the systemic vascular resistance [49,69]. T3-therapy can induce DNA synthesis and cardiomyocyte proliferation, and improve the cardiac contractility; though, this action is as still unidentified [49,70-74].

On the basis of these data, it can be reported that the T3 may stimulate the cardiac contractility and stimulate the hemodynamic changes (blood pressure, blood volume and heart rate) during the prenatal and postnatal periods. Additional studies are necessary to delineate the likely relations with human health. Future examinations are necessary to explore the multidirectional actions of TH-therapy in the maternal and neonatal cardiovascular diseases.

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