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Commentary

Thyroid Diseases and Developmental Adenosinergic Imbalance - @

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COMMENTARY

Thyroid Hormones (THs) regulate the development of all biological systems, particularly CNS [1-30], neuronal excitability and the ionic gradient [6,8]. The fetal hypothyroidism diminished the levels of nucleotides, ATPases (Na⁺, K⁺-ATPase, Ca²⁺-ATPase and Mg²⁺-ATPase) activities, and total adenylate; however, the adenylate energy charges displayed a tendency to an increase in cerebrum and cerebellum at gestation days 15 and 20 [25]. The synthesis of ATP delays considerably during hypothyroidism state [31] and changed the hydrolysis of adenine nucleotide in synaptosomes from hippocampus and cerebral cortex of rats [32]. The alterations in fetal ATPases might disrupt the neurotransmitter roles in the CNS [6]. The neonatal hypothyroidism alters the activity of these enzymes [33] and impairs the metabolic process, ionic transportation and neuronal functions [34-36].

On the other hand, perinatal hyperthyroidism stimulates the activities of ATPases and causes a maldevelopment and pathophysiological state [3]. However, Bruno, et al. [37] reported that hyperthyroidism during development inhibits the hydrolysis of AMP, ADP and ATP in synaptosomes from hippocampus and cerebral cortex of rats. This inhibition can disrupt the brain excitability [6]. Generally, both hypo- or hyper-thyroidism disordered the activities of ATPase in the frontal cortex and the hippocampus of rat [38]. Thus, hyperthyroidism changes the hydrolysis of ATP to adenosine, while hypothyroidism appears to delay the production of adenosine [6,32,37]. Thus, any thyroid disorders might alter the activities of ATPases and hydrolysis of nucleotides during the development. This can inhibit the nerve transmission and delay the cognitive functions. Further studies are needed to explore the non-genomic mechanisms of THs and developmental adenosinergic axis.

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