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Commentary
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Maternal Thyroid Dysfunctions and Developing Visual Abnormalities

Ahmed RG*

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

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***Corresponding author:** Ahmed RG, Department of Zoology, Beni-Suef University, Beni-Suef, Egypt, Tel: 002-010-91471828; Email: ahmedragab08@gmail.com

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Abbreviations: HPTA: Hypothalamic-Pituitary-Thyroid Axis; THs: Thyroid Hormones; T3: 3,5,3'-triiodothyronine; T4: Thyroxine; TRα and TRβ: Thyroid receptors; PND: Post Natal Day; INL: Inner Nuclear Layer; GCL: Ganglion Cell Layer

Commentary

The activity of the maternal hypothalamic-pituitary-thyroid axis (HPTA) is warranted for the normal development [1-60], especially the morphogenesis and angiogenesis of the retina and its gene expression [61-68]. Regulation the development of the retina by 3,5,3'-triiodothyronine (T3) and thyroxine (T4) is depending on the expression of the thyroid receptors (TRα and TRβ) [69-74]. During the prenatal period, Thyroid Hormones (THs) regulate the standard development of the retina and eye [75-78]. During the postnatal period, THs control the differentiation and growth of spectral cone types [79-82]. At the postnatal day (PND) 10, the progress in the levels of THs in the dorsal retina is concomitant with the terminal maturation of the cones [83-87]. Also, expression of TRβ2 is required for the cones [88-90] to stimulate the expression of Mopsin and to inhibit the expression of S opsinduring the development [91,92] and adulthood [93]. More importantly, the signaling expression of TRβ2 is vital for the development particularly during the embryonic day 15 to PND 5 [94].

On the other hand, several studies reported that the maternal thyroid dysfunction (hypothyroidism) during the gestation and lactation periods can cause numerous defects as the following [95,96]:

a. Reduce the size of eye;

b. Delay the development of retina;

c. Decrease the cell densities of the inner nuclear layer (INL) and ganglion cell layer (GCL, ganglion cells and displaced amacrine cells); and

d. Abnormalities in the morphology of photoreceptors and inner retina.

In patients with severe Graves' orbitopathy, several authors noticed the abnormalities in the visual acuity [97].

Given these considerations, the current observations proposed that the maternal THs play significant actions in the developing retina, cones, and eye during the prenatal and postnatal periods. The maternal thyroid disorders may directly or indirectly disrupt the morphogenesis and angiogenesis of the retina and its gene expression. This disruption may delay the normal development of the primary visual system and cause retinopathy and neonatal blindness. Additional researchers are needed to allow us to determine the T3 signaling at the level of individual retinal Cells, in particular, the inner cells during the prenatal and postnatal periods.

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