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# **Functions and Development of Thyroid Hormone**

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### Commentary

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# DESCRIPTION

Thyroid refers to a bodily condition where the hormone is functioning properly. There are different thyroid disorders such as hyperthyroidism, hypothyroidism, thyroiditis, goitre, thyroid nodules, and thyroid cancer. The autoimmune condition Graves' disease is the most prevalent cause of hyperthyroidism, which is characterized by increased thyroid hormone production. Iodine deficiency is the most common cause of hypothyroidism, which is defined by an inadequate release of thyroid hormones.

At 3–4 weeks of pregnancy, the thyroid gland begins to form at the base of the tongue in the floor of the pharynx. From that, it descends in front of the pharyngeal gut and gradually migrates to the base of the neck over the coming few weeks. The thyroglossal duct, a tiny canal that connects the thyroid to the tongue during migration, remains in place. The thyroglossal duct degenerates around the end of the fifth week, and the detached thyroid migrates to its final position over the next two weeks.

#### Development

The thyroid gland develops as an epithelial proliferation in the floor of the pharynx at the base of the tongue between the tuberculum impar and the copula linguae during 3–4 weeks of gestation. The hypopharyngeal prominence quickly engulfs the copula, which is later marked by the foramen cecum. The thyroid then passes *via* the thyroglossal duct and descends in front of the pharyngeal gut as a bilobed diverticulum. It moves toward the base of the neck throughout the coming weeks, passing in front of the hyoid bone. The thyroglossal duct, a tiny canal that connects the thyroid to the tongue during migration, remains in place.

Thyrotropin-Releasing Hormone (TRH) and Thyroid-Stimulating Hormone (TSH) are first released by the hypothalamus and pituitary of the growing embryo (TSH). First TSH measurements are made at 11 weeks. Thyroxine (T<sub>4</sub>) production reaches a clinically significant and self-sufficient level by 18–20 weeks. Till 30 weeks, the foetal

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triiodothyronine  $(T_3)$  level is low (less than 15 ng/dL), then it rises to 50 ng/dL at term. To prevent neurodevelopmental problems brought on by maternal hypothyroidism, the foetus must be self-sufficient in thyroid hormones. lodine needs to be present in appropriate amounts for proper neurodevelopment.

# **Functions**

- The production of the iodine-containing thyroid hormones triiodothyronine (T<sub>3</sub>), thyroxine or tetraiodothyronine (T<sub>4</sub>), as well as the peptide hormone calcitonin, is the thyroid's primary function. The thyroid hormones are produced from iodine and tyrosine. Iodine atoms constitute four of the molecules that form T<sub>4</sub> and three of the molecules that form T<sub>3</sub>, respectively. The effects of thyroid hormones on the human body are very different.
- Metabolic thyroid hormones affect practically all bodily tissues, including the cardiovascular system, and raise basal metabolic rate.
- Cardiovascular hormones increase heart rate frequently. They speed up breathing, oxygen intake, and oxygen consumption, as well as mitochondrial activity.
- Hormone production thyroglobulin is used to make the thyroid hormones. This protein is part of the colloid in the follicular lumen and is made in the rough endoplasmic reticulum of follicular cells before being transported there. Tyrosine, which is present in thyroglobulin in units of 123 that interacts with iodine in the follicular lumen.
- Thyroid-Stimulating Hormone (TSH), which is secreted by the anterior pituitary gland, is principally responsible for controlling the production of thyroxine and triiodothyronine. Thyrotropin Releasing Hormone (TRH), which is pulsatilely produced from the hypothalamus, in turn stimulates TSH release. TSH production is inhibited when thyroid hormone levels are high due to the thyroid hormones' negative feedback on the thyrotropes TSH and TRH.
- The hormone calcitonin, which aids in controlling blood calcium levels, is additionally produced by the thyroid gland. High blood calcium levels cause parafollicular cells to generate calcitonin. By reducing the activity of osteoclasts, the cells that break down bone, calciumtonin reduces the release of calcium from bone. Osteoclasts and osteoblasts continuously produce and resorb bone; hence calcitonin effectively increases the transport of calcium into bone.