

# Thyroid Hormones are Essential for Normal Life

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**Received date:** November 03, 2024; **Accepted date:** November 11, 2024; **Published date:** December 28, 2024

**Citation:** Siniša Franjić, Thyroid Hormones are Essential for Normal Life, *Endocrinology and Dysfunctions*, vol 1(1). DOI: 10.9567/ISSN.2024/WSJ.80

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## Abstract

The thyroid gland is an organ located in the front of the neck just below the Adam's apple. The thyroid gland is butterfly-shaped and consists of two lobes located on either side of the trachea. A normal thyroid gland cannot be felt by pressing a finger on the neck, nor can it be seen externally. The function of the thyroid gland is to produce hormones whose task is to regulate the body's metabolism so that it controls the work of the heart, muscles and digestion, brain development and bone maintenance. The proper functioning of the thyroid depends to a large extent on a good supply of iodine from the diet. Thyroid hormone-producing cells are specialized in extracting and absorbing iodine from the blood and incorporating it into thyroid hormones.

**Key Words:** Thyroid Gland, Thyroid Hormones, TSH, Homeostasis, Health

## Introduction

Thyroid hormones are imperative regulators of different physiological forms, such as cellular development and digestion system [1]. The thyroid organ display in the front neck is dependable for the blend and emission of thyroid hormones: thyroxine (T4) and triiodothyronine (T3).

Thyroid hormones influence the work of all organs [2]. Without them, there is no ordinary improvement or working of the central apprehensive framework. They are vital for keeping up body temperature and keeping up the ordinary action of the respiratory center, controlling vitality use and oxygen utilization. They act on the heart by expanding the contractility of the heart muscle and speeding up the work of the heart, and moreover increment intestinal motility, influence bone remodeling, sugar homeostasis and blood fat. The thyroid organ produces its hormones in typical sums unless one of the thyroid disorders has created. It can at that point have either intemperate or lacking generation called hyperthyroidism or hypothyroidism. The indications are inverse: on the one hand, overproduction causes weight misfortune, palpitations, affectability to warm and sweating, quickened digestion system and shuddering. On the other hand, inadequately generation leads to weight pick up, affectability to cold, obstruction, weakness and sleepiness.

### Types

T4 is the overwhelming thyroid hormone emitted by the thyroid organ, bookkeeping for around 90% of endogenous add up to thyroid hormone generation [1]. It ought to be famous that T4 is, in reality, a prohormone with generally low organic action

compared to T3. Basically, T4 comprises four iodine molecules bound to a tyrosine-derived spine. Most T4 is bound to carrier proteins, such as thyroxine-binding globulin (TBG), transthyretin, and albumin in circulation, with as it were a little division (around 0.03%) display as free thyroxine (FT4). Free T4 is the unbound shape that can enter target cells and apply different intracellular effects.

On the other hand, T3, the naturally dynamic thyroid hormone, accounts for nearly 10% of the

total endogenous thyroid hormone generation. T3 has around three to five times more prominent strength than T4 and is created through fringe deiodination.

T3 circulates bound to carrier proteins (primarily TBG and albumin), with a little division (approximately 0.3%) existing as free triiodothyronine (FT3). Free T3 applies its impacts by official to its cognate intracellular thyroid hormone receptors (TRs).

Reverse T3 (rT3) is an dormant metabolite of T4, created by the activity of sort 3 deiodinase,

which specifically evacuates an iodine particle from the internal ring of T4. In spite of the fact that rT3 offers auxiliary closeness with T3, it does not tie to thyroid receptors TRs with tall liking and, in this manner, does not display any critical organic action. rT3 is a by-product of thyroid hormone inactivation and is clinically valuable as an vital marker of the debilitated euthyroid syndrome.

### Carrier Proteins

Thyroid hormones, particularly T4 and T3 are bound to carrier

proteins in the common circulation [1]. These carrier proteins ship thyroid hormones all through the body, in this way controlling their accessibility to target tissues. In fact, more than 95% of thyroid hormones in serum are bound to three major carrier proteins, taking off a little, unbound division. Thyroid-binding globulin (TBG) is the essential and most plenteous carrier protein, which ties around 70% of T4 and 80% of T3.

Transthyretin, or thyroxine-binding prealbumin (TBPA), ties approximately 10–15% of T4 and a irrelevant extent of T3. Egg whites, the most copious protein in human serum, has the most reduced liking for thyroid hormone among all carrier proteins. Albumin ties approximately 15–20% of T4 and 5–10% of T3. In spite of the fact that albumin has a moderately lower liking for thyroid hormones than TBG and transthyretin, its tall concentration in the circulation system implies it plays a noteworthy part in the transport of thyroid hormones.

Only a minor division of T4 (approximately 0.03%) and T3 (around 0.3%) remains unbound or “free” in the serum [10]. The free division of add up to thyroid hormones in circulation is promptly accessible for cell take-up, where it applies its intracellular impacts. In differentiate, protein-bound thyroid hormones are considered a promptly available store of thyroid hormones that are organically inert.

### Delodination

Deiodination is the most critical pathway that metabolizes thyroid hormones and directs T3 bioavailability in human tissues [1]. The thyroid organ produces as it were a little sum of T3, whereas most of T3 (generally 80%) in fringe tissues is created by enzymatic deiodination by means of external ring deiodination (ORD) of T4. Essentially, the metabolite rT3 is created through internal ring deiodination (IRD) of T4. Consequently, the digestion system of thyroid hormones by deiodinase enzymes comes about in either their actuation or inactivation.

**Formation**Levothyroxine (LT4) is a engineered planning of endogenous thyroid hormone T4, utilized for

the treatment of hypothyroidism, independent of the basic cause [1]. In arrange to apply its physiological impacts, levothyroxine is changed over by different deiodinases into the naturally dynamic hormone T3. As a result, T3 interatomic with atomic TRs to eventually balance quality transcription.

Levothyroxine is changed over to the dynamic hormone T3 by the activity of deiodinase proteins, fundamentally type 1 (D1) and type 2 (D2) deiodinases. For illustration, sort I deiodinase (D1), which is found overwhelmingly in hepatic and renal tissues, is mindful for the change of the prohormone T4 to its naturally dynamic shape, T3. Moreover, D1 changes over T4 to rT3, its inert metabolite.

Type 2 deiodinase (D2) is display basically in the brain, pituitary organ, brown adipose tissue (BAT), skeletal muscle, and other fringe tissues. D2 essentially changes over T4 to T3 for intracellular utilize, in this way playing a basic part in the neighborhood concentration of dynamic thyroid hormone. It is worth noticing that negative input control of the hypothalamic-pituitary-thyroid (HPT) pivot depends on T3 produced in the pituitary organ itself.

Once shaped, T3 enters the core of target cells and ties to atomic TRs, which are individuals of the atomic receptor superfamily.

TRs frame heterodimers with retinoid X receptors (RXR) and tie to particular DNA groupings called thyroid hormone response elements (TREs) found in the promoter locales of target qualities. The thyroid hormone receptor that is as of now bound as a heterodimer with RXR (retinoid X receptors) ties to the particular TH reaction component arrangements (TRE) found in the promoter locales of T3 target qualities in the core and controls the expression of these qualities in a ligand-dependent way. On the opposite, unliganded TRs interface with TREs in T3 target qualities and control transcriptional restraint. In the nonappearance of T3, corepressor proteins are enlisted into the RXR-TR heterodimer and hinder the expression of the target quality. Diverse sorts of mRNAs are delivered as a result of translation; these mRNAs at that point move from the core into the cytosol, where they experience interpretation to create proteins.

Transcriptional direction interceded by T3-bound TRs impacts different physiological forms, counting basal metabolic rate (BMR), protein blend, carbohydrate and lipid digestion system, bone development and advancement, and anxious framework development. The net impact of levothyroxine treatment is reestablishing typical thyroid hormone levels and lightening hypothyroid symptoms.

### TSH

The inability to normalize serum TSH (Thyroid Stimulating Hormone) with verbal levothyroxine is a regularly experienced clinical issue [3]. Most people require a measurements of 1.6–1.8 mcg/kg of verbal levothyroxine day by day, but a few stay hypothyroid in spite of heightening measurements. The most common reason for incapability of thyroid hormone substitution is nonadherence to treatment (“pseudomalabsorption”). The to begin with step in the clinical evaluation is to guarantee reliable every day levothyroxine ingestion, partitioned from nourishment admissions. Every so often, drug store fill records may require to be gotten to find out compliance.

After prohibition of adherence issues, sedate intuitive ought to be surveyed, as concomitant solutions can impede assimilation and influence digestion system of thyroid hormone. Multivitamin and dietary supplements may contain calcium or press salts, which sequester levothyroxine by shaping complexes that are ineffectively ingested. Since typical gastric corrosive discharge is vital for full thyroid hormone retention, dosage alterations are regularly required when proton pump inhibitors and stomach settling agents are begun. Anti-epileptic drugs, such as carbamazepine, phenytoin, and phenobarbital, can increment the hepatic digestion system of thyroid hormone. Drugs that diminish thyroid hormone amalgamation (amiodarone, iodine, lithium) may result in a higher dosage prerequisite in people who as of now have constrained save. Solutions that increment serum thyroxine-binding globulin (TBG), such as estrogen or tamoxifen, may result in a rise in TSH and signal the require for an expanded measurements. A more broad list and discourse can be found in the American Thyroid Affiliation Rules for Treatment of Hypothyroidism.

Other conditions can accelerate compounding of known hypothyroidism in a already well-controlled person. In this case, the understanding displayed with dynamic edema and was rapidly distinguished to have nephrotic-range proteinuria and

hypoalbuminemia. Early ponders of nephrotic disorder have illustrated urinary misfortunes of TBG and other proteins that tie thyroid hormone (albumin and transthyretin), coming about in moo add up to T4 concentrations. Much appreciated to input alterations affected by a ordinarily working hypothalamic-pituitary-thyroid pivot, most patients with proteinuria will stay euthyroid, but in patients with constrained thyroid save, or subordinate on verbal levothyroxine, the urinary misfortunes may trigger new-onset hypothyroidism or compound preexisting hypothyroidism. In pediatric ponders, the every day urinary protein excretion showed up to relate emphatically with the degree of urinary misfortune of TBG, FT4, and FT3 and relate contrarily with comparing serum levels.

More commonly, an undiscovered clutter of the stomach related tract is the guilty party of an incapable levothyroxine dosage. Atrophic gastritis, which can result in achlorhydria, can affect levothyroxine prerequisites. Since levothyroxine retention happens basically in the jejunum and ileum, a number of conditions (inflammatory bowel disease, celiac disease, intestinal surgery) can result in malabsorption. Bowel edema from congestive heart failure, hepatic cirrhosis, or nephrotic disorder can too contribute, the last mentioned of which was likely important to this case think about. Work-up of these etiologies may require extra consultative administrations or subspecialty input. If there is a tall degree of doubt of nonadherence, a levothyroxine retention test can be considered to run the show out compliance issues earlier to starting an extensive evaluation for a natural cause.

### Homeostasis

The thyroid gland produces two hormones, thyroxine (T4), which is feebly naturally active, and triiodothyronine (T3), which is 3–5 times more powerful than T4. T4 and T3 are discharged into the circulation at a proportion of 14–20:1, and T4 is changed over into the more dynamic T3 inside cells T4 has a much longer half-life than T3, to a great extent due to being unequivocally bound to plasma thyroid hormone-binding proteins, so acts as a “pool” of thyroid hormone to offer assistance keep up homeostasis [4]. As the larger part of circulating T4 and T3 is protein-bound, and hence not organically dynamic, free T4 (FT4) and free T3 (FT3) are measured in clinical hone. The generation of T4 and T3 is kept up inside tight limits to keep up typical working of the body tissues. This homeostasis is kept up through the hypothalamic-pituitary-thyroid hub. The hypothalamus produces thyrotropinreleasing hormone (TRH) in reaction to moo circulating levels of T4 and T3. TRH fortifies the pituitary to create thyroid fortifying hormone (TSH), which in turn fortifies the thyroid organ to create more T4 and T3. TRH is not routinely measured in clinical hone. As the levels of T4 and T3 rise, they apply negative criticism on both the hypothalamus and pituitary, the generation of TRH and TSH weaken, and the thyroid diminishes the generation of T4 and T3.

The tremendous majority of patients with thyroid hormone brokenness will have a issue emerging in the thyroid organ itself, so the hypothalamus and pituitary can be expected to be responding fittingly to the winning thyroid hormone levels. Subsequently, in most circumstances, a TSH level inside typical limits proposes that the levels of T4 and T3 are ordinary. For this reason, a few research facilities will degree as it were TSH as a screening test, or maybe than TSH, FT3, and FT4. If the thyroid gland over-secretes T4 and T3 (e.g. in Graves’ disease), the

hypothalamus and pituitary distinguish these tall levels and suitably diminish generation of TRH and TSH. Alternately, if the thyroid organ falls flat to make sufficient T4 and T3 (e.g. in immune system hypothyroidism), these moo levels are identified by the hypothalamus and pituitary, TRH discharge increments suitably and hence the pituitary increments TSH emission appropriately.

Rarely, the essential deformity is exterior of the thyroid gland and the TSH level may be misleading. These conditions are uncommon but ought to be considered where the patient’s side effects and signs do not fit with the TSH result, or if there is clinical doubt of a condition influencing the pituitary or hypothalamus. Central hypothyroidism is as a rule due to a issue with the pituitary gland that anticipates it from reacting to TRH, T4 and T3, and from emitting satisfactory sums of TSH. Causes incorporate compression of the organ by a tumor, circulatory trouble, surgery, radiotherapy, immune system hypophysitis (essential or medicate related), and disease. In this circumstance, T4 and T3 levels drop and the understanding creates indications of hypothyroidism, but will have either a low TSH or TSH at the lower conclusion of the typical extend that is unseemly for the circulating low levels of T4 and T3. The pituitary gland is included in the control of numerous other hormonal tomahawks in expansion to the thyroid pivot, so patients with pituitary harm may well have indications of other hormonal shortages counting hypoadrenalism and hypogonadism. Exceptionally once in a while, the pituitary may create an adenoma that independently secretes TSH coming about in central hyperthyroidism. These adenomas are called TSH-omas. Brokenness of the hypothalamus is exceptionally uncommon and is ordinarily clear some time recently side effects and signs of thyroid brokenness develop.

### Function

Although the thyroid gland can carry out all the steps of hormone biosynthesis, capacity, and discharge in the nonappearance of any outside signals, independent work is distant as well drowsy to meet real needs for thyroid hormone [5]. The central controller of thyroid work is the thyroid-stimulating hormone (TSH), which is discharged by thyrotropes in the pituitary gland. It may be reviewed that TSH comprises of two glycosylated peptide subunits counting the same  $\alpha$ -subunit that is also found in FSH (follicle-stimulating hormone), and LH (luteinizing hormone). The  $\beta$ -subunit is the portion of the hormone that confers thyroidspecific invigorating action, but free  $\beta$ -subunits are inert, and fortify the thyroid as it were when connected to  $\alpha$ -subunits in a complex three-dimensional configuration.

Thyroid-stimulating hormone ties to a single lesson of heptahelical G-protein-coupled receptors in the basolateral surface layers of thyroid follicular cells. The TSH receptor is the item of a single quality, but it is comprised of two subunits held together by a disulfide bond. It shows up that after the atom has been legitimately collapsed and its disulfide bonds shaped, a circle of almost 50 amino acids is extracted proteolytically from the extracellular parcel of the receptor. The  $\alpha$ -subunit incorporates approximately 300 buildups at the amino end and contains most of the TSH official surfaces. The  $\beta$ -subunit contains the seven membrane-spanning alpha helices and the brief carboxyl terminal tail in the cytoplasm. Decrease of the disulfide bond may lead to discharge of the  $\alpha$ -subunit into the extracellular liquid, and may have critical suggestions for the improvement of antibodies to the TSH receptor and thyroid disease.

Binding of TSH to the receptor comes about in enactment of both adenylyl cyclase through G $\alpha$ s and phospholipase C through G $\alpha$ q and leads to increments in both the cyclic AMP (Adenosine monophosphate) and diacylglycerol/IP3 moment delivery person pathways. Enactment of the cyclic AMP pathway shows up to be the more vital transduction component, as all the known effects of TSH can be copied by cyclic AMP. Since TSH increments cyclic AMP generation at much lower concentrations than are required to increment phospholipid turnover, it is likely that IP3 and DAG (diacylglycerol) are repetitive go betweens that strengthen the impacts of cyclic AMP at times of seriously incitement, but it is moreover conceivable that these moment flag-bearers flag a few interesting reactions. Expanded turnover of phospholipid is related with discharge of arachidonic corrosive and the resulting expanded generation of prostaglandins that moreover takes after TSH incitement of the thyroid.

In expansion to controlling all viewpoints of hormone biosynthesis and discharge, TSH increments blood stream to the thyroid. With delayed incitement TSH moreover increments the tallness of the follicular epithelium (hypertrophy), and can fortify division of follicular cells (hyperplasia).

Stimulation of thyroid follicular cells by TSH is a great illustration of a pleiotropic effect of a hormone in which there are different isolated but complementary activities that summate to deliver an in general reaction. Each step of hormone biosynthesis, capacity, and emission shows up to be straightforwardly invigorated by a cyclic AMP-dependent handle that is quickened freely of the going before or taking after steps in the pathway. In this way indeed when expanded iodide transport is blocked with a sedate that specifically affects the iodide pump, TSH by the by quickens the remaining steps in the manufactured and secretory prepare. Essentially, when iodination of tyrosine is blocked by a sedate particular for the organification handle, TSH still invigorates iodide transport and thyroglobulin synthesis.

Most of the reactions to TSH depend upon actuation of protein kinase A and the resultant phosphorylation of proteins counting translation components such as CREB (cyclic AMP reaction component authoritative protein). TSH increments expression of qualities for the sodium iodide symporter, thyroglobulin, thyroid oxidase, and thyroid peroxidase. These effects are applied through agreeable intuitive of TSH actuated atomic proteins with thyroidspecific translation variables whose expression is too improved by TSH. TSH shows up to increment blood stream by actuating the quality for the inducible shape of nitric oxide synthase, which increments generation of the strong vasodilator, nitric oxide, and by actuating expression of paracrine components that advance capillary development (angiogenesis). Absolutely how TSH increments thyroid development is not caught on, but it is clear that blend and emission of a assortment of neighborhood development components is induced.

### Thyrotoxicosis

The term thyrotoxicosis applies to a clinical condition coming about from expanded thyroid hormone concentration and activity [6]. When the clinical condition is analyzed by appropriate laboratory tests, the etiology ought to be decided. A tall radioiodine take-up of thyroid will as a rule show either a exceptionally common condition called Graves' hyperthyroidism or a exceptionally exceptional TSH-secreting pituitary adenoma. A exceptionally moo take-up or no take-up will demonstrate

dangerous thyroiditis, iodine-induced hyperthyroidism, or exceptionally uncommon cases of extra-thyroidal thyroid hormone generation or exogenous thyroid hormone admissions. Ordinary radioactive thyroid take-up can happen in mellow Graves' hyperthyroidism or in multinodular poisonous goiter and poisonous adenoma. Administration ought to be issue arranged and ought to depend on the etiology. Antithyroid solutions, surgery, or radioactive iodine treatment can be utilized for tall take-up sorts, and symptomatic treatment can be utilized for damaging thyroiditis. Iodine-induced hyperthyroidism will react to antithyroid solutions and disposal of exogenous iodine.

When the etiology is related to expanded rate of thyroid hormone union, the term hyperthyroidism is pertinent. Thyrotoxicosis can also result from a damaging handle in the thyroid coming about in unregulated abundance discharge of put away thyroid hormones without expanded generation. The thyrotoxicosis disorder may also be due to exogenous source either iatrogenic or factitious. Hyperthyroidism is considered subclinical if TSH is low with ordinary FT4 and FreeT3. In this case hypothalamus-pituitary hub faculties the overabundance, and the negative input component comes about in smothered or unusually low thyrotrophic hormone (TSH). Hence it can be contended that this is a biochemical definition or maybe than a clinical one. Subclinical hyperthyroidism may be symptomatic or asymptomatic but in either case may have antagonistic effects

Thyrotoxicosis is a disorder with numerous differing etiologies. When clinical symptomatology along with biochemical discoveries sets up abundance thyroid hormone impact, demonstrative measures ought to be coordinated at finding the particular etiology, since administration and treatment will depend on the etiology. Graves' hyperthyroidism is the most common cause of hyperthyroidism in the United States. Poisonous multinodular goiter and poisonous adenomas are the another common causes. Nodular poisonous goiter is more common in more seasoned people and in geographic ranges with verifiable iodine insufficiency. Unseemly overabundance thyroxine (T4) treatment or T4 suppressive treatments for follicular cell-derived thyroid cancer are moreover common causes of subclinical hyperthyroidism.

### Hypothyroidism

Hypothyroidism is the clinical state which comes about from either insufficient generation of thyroid hormone or disabled activity of thyroid hormone at the tissue level [7]. It is characterized by the research facility parameters of a moo free thyroxine (FT4), related with an raised thyroid-stimulating hormone (TSH) in essential hypothyroidism or, less commonly, a low to low-normal TSH in central hypothyroidism.

Primary hypothyroidism, where the imperfection is at the level of the thyroid gland itself, accounts for over 95% of cases of unmistakable hypothyroidism. The remaining 5% are caused by auxiliary or tertiary hypothyroidism (deformity at the level of the pituitary gland or the hypothalamus) or thyroid hormone resistance.

Subclinical hypothyroidism (SCH), characterized as an rise in TSH but with a comparing typical FT4 level, expect that there is an intaglio hypothalamic-pituitary-thyroid pivot and an nonappearance of intercurrent ailment. The values ought to moreover be reproducible over a 4–6-week period.

Given unclear symptomatology that covers with other endocrine and non-endocrine disorders, hypothyroidism is commonly tried in clinical hone. It in this way gets to be imperative to separate obvious hypothyroidism that thyroid hormone substitution, from non-thyroidal cause of such indications as weariness, weight pick up, or disabled cognitive function.

## Conclusion

Thyroid hormones are essential for normal life. They have numerous functions, the most important of which are regulating the metabolism (exchange of substances and processes) of all organs and cells, stimulating the growth, development and differentiation of cells, influencing the work of the heart and the production of energy for muscle work, speeding up digestive processes and improving the function of the intestines, acting on fat metabolism, stimulate mental functions and bone growth, influence the maintenance of pregnancy, normal growth and development of the child and numerous other functions. In the case of various diseases manifested by reduced thyroid function, hormonal treatment is used. The thyroid gland can be completely healthy, but there is a disturbance in the level of its hormones. Conditions that can lead to this are: severe illness, stress, medications, liver and kidney diseases, malnutrition, etc. Thyroid hormones are called thyroxine (T4) and triiodothyronine (T3). Both of these hormones are secreted from the thyroid gland into the blood and primarily affect the body's metabolism by increasing heat generation, oxygen utilization, stimulating glucose absorption and synthesis, fat breakdown, and protein formation. They stimulate heart activity and bone growth and maturation.

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