International Journal of Pharmacology and Clinical Research (IJPCR)

IJPCR |Volume 5 | Issue 2 | Apr - Jun - 2021
www.ijpcr.net

Research article | Clinical research

ISSN: 2521-2206

Pugos nutrition for hypothyroidism

GovindShukla, Sandeep kunche, MonicaYadav, Anusha kandala, Uddhav L Kanade, Arun Kumar Junjipelly, C.J.Sampath Kumar

PUGOS Products Pvt. Ltd.
42, 2nd Floor, Leelavathi Mansion, 6th Cross, Margosa Main Road Malleshwaram Bangalore-56003, INDIA

Corresponding author: GovindShukla
Email id: lactonovaresearch44@gmail.com

ABSTRACT

The thyroid gland is located at the base of our neck, It produces two hormones, triiodothyronine (T3) and thyroxine (T4), which circulate through our bloodstream and control metabolic activity in every cell in the body.
Hypothyroidism or underactive thyroid occurs when the thyroid gland cannot produce enough thyroid hormones to meet the body's demands. This causes all bodily functions to slow down and we feel tired, sluggish, achy, and gain weight.
The most common cause of hypothyroidism was iodine deficiency. Iodine is required for the production of thyroid hormone. The most common cause of hypothyroidism is Hashimoto's disease, which is an autoimmune disorder in which the body makes antibodies that attack the thyroid gland. This impairs the production of thyroid hormone. People with Hashimoto's develop a lump on their thyroid called a goiter.
Hypothyroidism can also result from treatment of Graves' disease (hyperthyroidism) with radioactive iodine, which destroys the thyroid gland, leaving it unable to produce hormones, and from surgical removal of the thyroid gland due to thyroid cancer. A baby can be born without a thyroid gland (congenital hypothyroidism).Diseases of the hypothalamus or pituitary gland can also cause hypothyroidism. These glands are involved in the regulation of the thyroid gland and the amount of thyroid hormone that is released. The hypothalamus releases thyrotropin-releasing hormone (TRH), which signals your pituitary gland to make thyroid-stimulating hormone (TSH). The amount of TSH released depends on how much T3 and T4 are in our blood. The thyroid gland regulates its production of hormones based on the amount of TSH it receives. Hypothyroidism is easily treated today with thyroid hormones, Nutritional supplements, and various lifestyle approaches.

INTRODUCTION

The thyroid gland is located at the base of our neck, It produces two hormones, triiodothyronine (T3) and thyroxine (T4), which circulate through our bloodstream and control metabolic activity in every cell in the body.

Causes

Hypothyroidism or underactive thyroid occurs when the thyroid gland cannot produce enough thyroid hormones to meet the body's demands.
This causes all bodily functions to slow down and we feel tired, sluggish, achy, and gain weight.

The most common cause of hypothyroidism was iodine deficiency. Iodine is required for the production of thyroid hormone
The most common cause of hypothyroidism is Hashimoto's disease, which is an
autoimmune disorder in which the body makes antibodies that attack the thyroid
gland. This impairs the production of thyroid hormone. People with Hashimoto's
develop a lump on their thyroid called a goiter.
Hypothyroidism can also result from treatment of Graves' disease (hyperthyroidism) with radioactive iodine, which destroys the thyroid gland, leaving it unable to produce

hormones, and from surgical removal of the thyroid gland due to thyroid cancer.
A baby can be born without a thyroid gland (congenital hypothyroidism).
Diseases of the hypothalamus or pituitary gland can also cause hypothyroidism.
These glands are involved in the regulation of the thyroid gland and the amount
of thyroid hormone that is released. The hypothalamus releases thyrotropin-releasing hormone (TRH), which signals your pituitary gland to make thyroid-stimulating hormone (TSH). The amount of TSH released depends on how much T3 and T4 are in our blood. The thyroid gland regulates its production of hormones based on the amount of TSH it receives.
Hypothyroidism is easily treated today with thyroid hormones, Nutritional supplements, and various lifestyle approaches.

Sign & Symptoms of Hypothyroidism

• Depression, irritability, and anxiety
• Dry, brittle nails
• Dry eyes and droopy eyelids
• Dry, itchy skin; dry hair and hair loss (including eyebrow hair loss)
• Fatigue and sluggishness
• Headaches
• High cholesterol
• Hoarse voice
• Insomnia
• Joint aching
• Low libido
• Memory loss
• Menstrual irregularities
• Muscle swelling or cramps
• Slow heart rate
• Tingling or numbness in hands and feet
• Weight gain

Risk Factors effecting Hypothyroidism

• Age: It is most common after age 40
• Family history
• Gender: It is 10 times more common in women
• History of hyperthyroidism, Graves' disease, or thyroid cancer
• Hormone imbalance (high estrogen and cortisol)
• Iodine deficiency
• Medications (lithium, estrogen)
• Poor diet: Lack of iodine or selenium
• Pregnancy: The body produces antibodies that attack the thyroid gland, increasing the risk of miscarriage, premature delivery, pre-eclampsia, and damage to the fetus.
• Stress

Patho-physiology and Diagnosis of Thyroid Disease

The thyroid is a butterfly-shaped gland located in the front of the neck just above the trachea. It weighs approximately 15 to 20 grams in the adult human. The thyroid produces and releases into the circulation at least two potent hormones, thyroxine (T_4) and triiodothyronine (T_3), which influence basal metabolic processes and/or enhance oxygen consumption in nearly all body tissues. Thyroid hormones also influence linear growth, brain function including intelligence and memory, neural development, dentition, and bone development (Larsen, 2003).
The thyroid gland produces T_4 and T_3 utilizing iodide obtained either from dietary sources or from the metabolism of thyroid hormones and other iodinated compounds. About 100 µg of iodide is required on a daily basis to generate sufficient quantities of thyroid hormone. Dietary ingestion of iodide in the United States ranges between 200 and 500 µg/day and varies geographically; ingestion is higher in the western part of the United States than in the eastern states. The specialized thyroid epithelial cells of the thyroid gland are equipped with a Na/I symporter that helps concentrate iodide 30 to 40 times the level in plasma to ensure adequate amounts for the synthesis of thyroid hormone. The iodide trapped by the thyroid gland is subsequently oxidized to iodine by the enzyme thyroid peroxidase. The iodine then undergoes a series of organic reactions within the thyroid gland to produce tetraiodothyronine or thyroxine (T_4) and triiodothyronine (T_3). T_3 is also produced in other tissues such as the pituitary, liver, and kidney by the removal of an iodine molecule from T_4. T_4 is considered to be more of a pro-hormone, while T_3 is the most potent thyroid hormone produced. T_4 and $T_3$are both stored in the thyroglobulin protein of the thyroid gland and released into the circulation through the action of pituitary derived thyrotropin (thyroid stimulating hormone or TSH). A normal individual produces from the thyroid gland approximately 90 to 100 µg of T_4 and 30 to 35 µg of T_3 on a daily basis. An estimated 80 percent of the T_3 produced daily in humans is derived from peripheral metabolism (5′-monodeiodination) of T_4, with only about 20 percent secreted directly from the thyroid gland itself. On a weight basis, T_3 is about 3 to 5 times more potent as a thyroid hormone than T_4 and is believed to be the biologically active form of the hormone.
TSH, secreted by thyrotroph cells located in the anterior pituitary gland, regulates thyroid gland function and hormone synthesis and release. The pituitary secretion of TSH in turn is influenced by the releasing factor, thyrotropin-releasing hormone (TRH) produced in the hypothalamus. The secretion of both TSH and TRH is regulated by negative feedback from thyroid hormone, predominantly T_3, from the circulation and/or T_3 that is produced locally from intracellular conversion of T_4 to T_3. When circulating thyroid hormone levels are elevated, both the synthesis and secretion of serum TSH are blunted. In contrast, when circulating levels of T_4 and T_3 are low, serum TSH levels are increased in a compensatory fashion. The geometric mean level of serum TSH in normal individuals is approximately 1.5 µU/ml as recently reported in the NHANES III study (Hollowell et al., 2002). When hypothalamic pituitary function is intact, serum TSH levels are markedly suppressed (to <0.05 µU/ml) in patients with hyperthyroidism and elevated circulatory levels of serum thyroxine, while a marked increase in TSH (>5 µU/ml) occurs in patients with hypothyroidism and low blood levels of serum T_4. The mechanism through which TSH binds to and activates the thyroid gland is well understood. TSH binds to a specific membrane receptor located on the surface of the thyroid epithelial cell and activates the cell signaling mechanisms through the enzyme adenylate cyclase located in the plasma membrane. Activation of adenylate cyclase increases intracellular cyclic adenosine monophosphate (cAMP) levels, which in turn stimulate additional

intracellular signaling events that lead to thyroid hormone formation and secretion. (Figure 1)

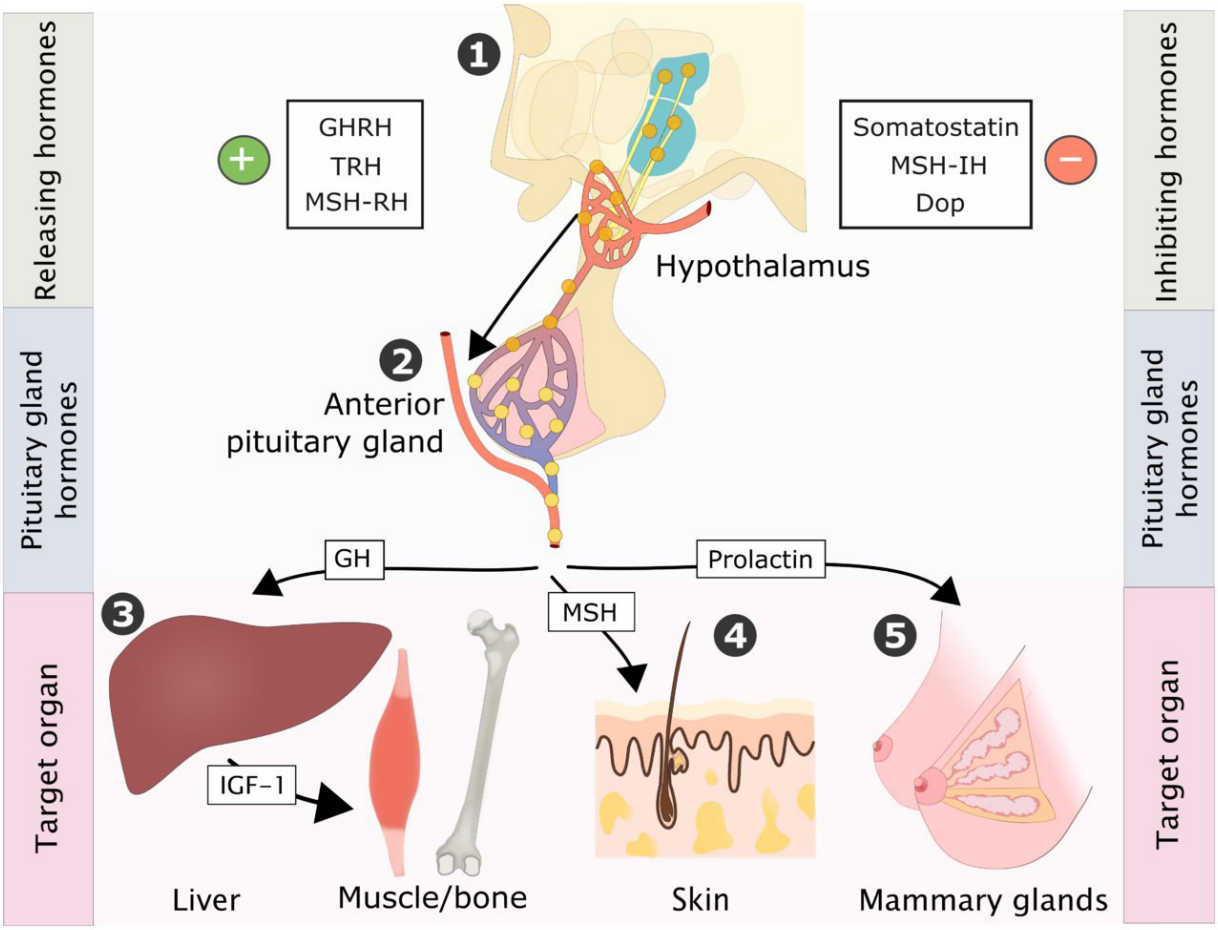

Figure 1

T_4 and T_3 circulate bound primarily to carrier proteins. T_4 binds strongly to thyroxine binding globulin (TBG, ~ 75 percent) and weakly to thyroxine binding prealbumin (TBPA, transthyretin, ~ 20 percent) and albumin (~5 percent). T_3 binds tightly to TBG and weakly to albumin, with little binding to TBPA. The geometric mean for serum T_4 in normal individuals is approximately 8 µg/dl, while the mean serum T_3 level is approximately 130 ng/dl. Under normal protein binding conditions, all but 0.03 percent of serum T_4 and 0.3 percent of serum T_3 is protein bound. Only a small amount of unbound (or free) T_4 (approximately 2 ng/dl) and T_3 (approximately 0.3 ng/dl) circulates in a free state, and it is this free concentration that is considered responsible for the biological effects of the thyroid hormones. There are physiologic situations associated with a change in the serum concentration of these thyroid-binding proteins—such as pregnancy, non-thyroidal illness, or ingestion of drugs—that affect the level and/or affinity of these binding proteins. Under these circumstances, the serum concentrations of total T_4 and total T_3 change in parallel to the changes that occur in the thyroid hormone binding proteins, but the serum concentrations of free T_4 and free T_3 remain normal and the individual remains euthyroid. In contrast, the serum concentration of free T_4 and free T_3 are raised in hyperthyroidism and decreased in hypothyroidism.

Thyroid function testing

At the present time, serum-based tests available by immunoassay for measuring the concentration of thyroid hormones in the circulation include total (TT_4 and TT_3) and free (FT_4 and FT_3) hormone. In addition, direct measurements of thyroid hormone binding plasma proteins, thyroxine binding globulin (TBG), transthyretin (TTR)/prealbumin (TBPA), and albumin are also available. However, the thyroid test measurement that has the greatest utility for evaluating patients suspected of thyroid disease is the third-generation thyroid stimulating hormone (TSH, thyrotropin) assay. Most third-generation TSH assays today that can reliably detect differences of 0.02 µU/ml or better (interassay imprecision <20 percent) can easily distinguish both hyper- and hypothyroidism from euthyroidism (normal thyroid function) and may differentiate the patient suffering from the "euthyroid sick syndrome" from true hyperthyroidism. Other methods in thyroid testing include the measurement of thyroid gland auto antibodies, including antithyroid peroxidase (TPOab), antithyroglobulin (Tgab), and antibodies against the TSH receptor (Trab). All of these thyroid test methods are routinely available on automated immunoassay instruments located in most hospital and reference laboratories with tight (<10 percent) method between run coefficients of variation.

Testing for diagnosis and management of thyroid dysfunction

The most sensitive test in an ambulatory population at risk for thyroid dysfunction is the serum TSH (Demers and Spencer, in press). Serum TSH assays today have sufficient sensitivity and specificity to identify individuals with all forms of thyroid dysfunction in the general population. However, among individuals with serious, acute illness, the serum TSH is less specific for thyroid disease because a serious illness alone can depress TSH secretion (to be discussed). TSH screening of the neonatal population to detect congenital hypothyroidism before it is clinically evident is mandated throughout the United States and in many other countries.

When an abnormal serum TSH value is obtained, the usual next step is to repeat the measurement of TSH and also measure a serum frees T_4. The latter can be performed in several ways and among non-hospitalized individuals, most methods give results that are inversely correlated with the serum TSH result. The most common cause of discordance between the TSH and free T_4 result occurs in patients with subclinical thyroid dysfunction with high or low serum TSH values and a normal serum free T_4 result.

Serum TSH measurements may yield misleading results for individuals with changing levels of thyroid hormones. For example, a serum TSH level may remain high for weeks in hypothyroid patients treated with T_4. Similarly, serum TSH levels may remain low for weeks after the serum T_4 level falls to normal in patients treated for hyperthyroidism.

Thyroid Function Testing in the Elderly (Figure 2)

Name of Marker	NORMAL Range	OPTIMAL Range
TSH	.35 to 5.0	1.8 to 3.0
Total T4 or TT4	6-12 ug/d	5.4-11.5 ug/d
FTI	4.6-10.9 mg/dl	1.2-4.9 mg/dl
Free T4 or FT4	0.7-1.53 ng/dL	1.0 – 1.53 ng/dL
Resin T3 Uptake	24 – 39 md/dl	28 – 38 md/dl
Free T3 or FT3	260 – 480 pg/mL	300 – 450 pg/mL
TBG	15 -30 ug/dl	18 -27 ug/dl
TPO Antibody	<15	<15

Figure 2

The prevalence of both low and high serum TSH levels (with normal serum free T_4 results) is increased in elderly subjects compared with younger people. With respect to high serum TSH values, the increase is thought to represent an increased prevalence of autoimmune thyroiditis, especially in women, as will be discussed. The higher prevalence of low serum TSH values may be due to thyroid nodular disease or unrecognized non-thyroid illness.

Diagnosis of Hypothyroidism

Hypothyroidism is a hypometabolic state that results from a deficiency in T_4 and T_3. Its major clinical manifestations are fatigue, lethargy, cold intolerance, slowed speech and intellectual function, slowed reflexes, hair loss, dry skin, weight gain, and constipation. It is more prevalent in women than men. The most common cause of hypothyroidism is disease of the thyroid itself, primary hypothyroidism.

The most common cause of primary hypothyroidism is chronic autoimmune thyroiditis (Hashimoto's disease), in which the thyroid is destroyed by antibodies or lymphocytes that attack the gland. Other causes are radioactive iodine and surgical therapy for hyperthyroidism or thyroid cancer, thyroid inflammatory disease, iodine deficiency, and several drugs that interfere with the synthesis or availability of thyroid hormone. Hypothyroidism may also occur rarely (<1 percent of cases) as a result of deficiency of TRH or impaired TSH secretion due to hypothalamic or pituitary disease, respectively. This is known as secondary or central hypothyroidism because of the negative feedback relationship between serum T_4 and T_3 levels and TSH secretion. As, people with primary hypothyroidism have high serum TSH levels. If an individual has a high serum TSH value, serum free T_4 should be measured. The concomitant finding of a high serum TSH concentration and a low free T_4 level confirms the diagnosis of primary hypothyroidism. People with a high serum TSH concentration and a normal or low-normal serum free T_4 level have, by definition, subclinical hypothyroidism. The diagnosis of secondary hypothyroidism is based on the findings of a low serum free T_4 level and a serum TSH level that is normal or low. People with secondary hypothyroidism are unlikely to be detected by a screening program based on measurements of serum TSH, but the condition is much less common than primary hypothyroidism.

Diagnosis of Hyperthyroidism

Hyperthyroidism is a hypermetabolic state that results from excess production of T_4 and T_3. Its major clinical manifestations are nervousness, anxiety, heart palpitations,

rapid pulse, fatigability, and tremor, and muscle weakness, weight loss with increased appetite, heat intolerance, frequent bowel movements, increased perspiration, and often thyroid gland enlargement (goiter). Most individuals with hyperthyroidism are women.

The most common cause of hyperthyroidism is Graves' disease, an autoimmune disease characterized by the production of antibodies that activate the TSH receptor, resulting in stimulation of T_4 and T_3 production and enlargement of the thyroid. Other caused of hyperthyroidism are a multinodular goiter, solitary thyroid adenoma, thyroiditis, iodide- or drug-induced hyperthyroidism, and, very rarely, a TSH secreting pituitary tumor.

The diagnosis of hyperthyroidism is based on the findings of a high serum free T_4 level and a low serum TSH concentration. Occasionally, people with hyperthyroidism have a normal serum free T_4 and high serum free T_3 concentrations. These patients have what is called T_3-hyperthyroidism. An increase in serum thyroid hormone binding protein will raise the serum total T_4 level but not free T_4 concentrations. In these patients the serum TSH remains normal. Patients with a low serum TSH concentration and normal serum free T_4 and free T_3 levels have, by definition, subclinical hyperthyroidism.

Effect of Medications on Thyroid Test Results

Several medications have in vivo or in vitro effects on thyroid function tests that can create misleading results. Medications, notably estrogens, that raise serum TBG levels result in an increase in serum total T_4, but no change in serum free T_4 levels and no change in serum TSH concentrations. High doses of glucocorticoids (adrenal hormones) can lower the serum T_3 concentration by inhibiting the peripheral conversion of T_4 to T_3 and lower serum T_4 (and T_3) by inhibiting TSH secretion. Iodide, contained in solutions used to sterilize the skin and in radiopaque contrast media used in coronary angiography and many other radiological procedures, can cause either hyper- or hypothyroidism, depending on whether the individual has a nodular goiter or some unsuspected thyroid injury. The iodide-containing drug amiodarone, given to patients with cardiac arrhythmias, can also cause either hypothyroidism or hyperthyroidism in appropriately susceptible individuals.

Other drugs have effects that alter thyroid function test results directly. For example, the anticoagulant heparin can raise serum free T_4 concentrations by stimulating release of free fatty acids from triglycerides in serum. Thyroid test methods that use fluorescence detection may be sensitive to the presence of fluorophore-containing drugs or diagnostics agents used in radiology.

Thyroid Function Testing and Nonthyroidal Illness

Many people who are seriously ill have abnormal thyroid test results but no other evidence of thyroid dysfunction. These abnormalities occur in people with both acute and chronic illnesses and tend to be greater in those with more serious illnesses. Thus the laboratory diagnosis of thyroid disease can be extremely difficult to make in very sick people, especially those who need to be hospitalized. The effects of illness include decreased peripheral conversion of T_4 to T_3, decreases in serum concentrations of thyroid hormone binding proteins, and decreases in TSH secretion. These changes are reversible and do not seem to cause clinical manifestations of thyroid deficiency. Among healthier individuals, a few may have small changes in thyroid test results as a result of unrecognized nonthyroidal illness rather than thyroid dysfunction.

Prescription Drugs

Hypothyroidism is often a chronic problem that requires lifelong treatment. Doctors typically prescribe synthetic thyroid hormone (T4), such as Eltroxin

Blood tests are done to check T3, T4, and TSH levels

Calcium and iron supplements may reduce the absorption of thyroid hormone, so

take these products six hours away from your thyroid medication.

Dietary Recommendations

Foods to include

• Essential fatty acids are important for proper thyroid function.

Eat more fish and flaxseed.

• Sea vegetables such as kelp, nori, dulse, and wakame contain iodine, which is used by the body to make thyroid hormone. Shellfish and saltwater fish also contain iodine.

Foods to avoid

• Soybean, Broccoli, Brussels sprouts, cabbage, cauliflower, collard greens, and kale contain goitrogens, which interfere with thyroid hormone synthesis.

• Tap water contains fluorine and chlorine, which can inhibit the body's ability to absorb iodine.

Lifestyle Suggestions

• Don't smoke, as smoking can worsen hypothyroidism.

• Get regular exercise. Physical activity stimulates the thyroid to secrete more hormone and makes the body more sensitive to any thyroid hormone that is circulating.

• Manage your stress levels. Stress triggers the release of cortisol, which can suppress thyroid function.

Recommended Supplements

Guggul: Increases production of thyroid hormone (T3). Dosage: 25 mg of guggulsterones (active component) three times daily.

Multivitamin/mineral complex

Many nutrients are required to produce thyroid hormone, such as vitamin C, E, A, and the B-vitamins. Selenium is required for the conversion from T4 to T3. Many people are deficient in selenium, which may hamper thyroid hormone levels, so a complete multivitamin can ensure that all essential nutrients requirements are met.

Ashwaganda

An herbal product that helps boosts thyroid function and also reduces stress. Dosage: 500 mg three times daily.

Tyrosine

An amino acid involved in the synthesis of thyroid hormone. Dosage: 500 mg twice daily on an empty stomach.

Recommended Pugos Supplements

Astashine/Astashine gold, liquimega, Optigision gold, Nutrese weight loss therapy.

REFERENCES

1. Baloch Z, Carayon P, Conte-Devolx B, Demers LM, Feldt-Rasmussen U, Henry JF, LiVosli VA, Niccoli-Sire P, John R, Ruf J, Smyth PP, Spencer CA, Stockigt JR, Guidelines Committee, National Academy of Clinical Biochemistry. Laboratory medicine practice guidelines. Laboratory support for the diagnosis and monitoring of thyroid disease. Laboratory medicine practice guidelines. Thyroid. 2003; 13(1):3-126. doi: 10.1089/105072503321086962, PMID 12625976.
2. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, Braverman LE. Serum TSH, T (4), and thyroid antibodies in the United States population (1988-1994): national Health and Nutrition Examination Survey (Nhanes III). J Clin Endocrinol Metab. 2002; 87(2):489-99. doi: 10.1210/jcem.87.2.8182, PMID 11836274.
3. Larsen PR, Davies TF, Schlumberger MJ, Hay ID. Thyroid physiology and diagnostic evaluation of patients with thyroid disorders. In: Larsen PR, editor; 2003; , Kronenberg HM, editor; , Melmed S, editor; , Polonsky K, editor. , eds. Williams. Textbook of endocrinology. 10th ed. Philadelphia: W B Saunders Company. p. 389-516.