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Review Article

A Comprehensive Review of Thyroid Disease

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ABSTRACT

Hypothyroidism and hyperthyroidism are two common thyroid disorders that have a major influence on world health. The thyroid, a butterfly-shaped gland in the neck, produces the thyroid hormones thyroxine (T4) and triiodothyronine (T3), which control metabolism. These hormones affect growth, energy production, and other body processes. Whether hypo- or hyper-active, thyroid dysfunction impacts metabolic processes and can result in a variety of clinical symptoms and illnesses, such as goiter, autoimmune disorders, and benign and malignant thyroid nodules. The synthesis, storage, and release of thyroid hormones—which are regulated by the hypothalamic-pituitary-thyroid axis—are the main topics of this review, which also discusses the architecture, function, and pathology of the thyroid. Thyroid-stimulating hormone (TSH), which is secreted by the anterior pituitary and causes the thyroid gland to produce more T3 and T4, regulates the thyroid. The body's equilibrium depends on these hormones being released into the bloodstream. Iodide absorption, oxidation, iodination, coupling, and storage in the thyroid follicles are all steps in the manufacture of thyroid hormones. Iodine deficiency is the primary cause of thyroid disorders worldwide, however genetic, environmental, and autoimmune factors also play a role. Both hyperthyroidism and hypothyroidism are prevalent, with differing prevalence rates, in nations with sufficient iodine. Whereas hyperthyroidism results in symptoms like anxiety, heat sensitivity, and weight loss, hypothyroidism can bring despair, exhaustion, and weight gain. TSH, T3, T4, and thyroid antibodies are frequently measured as part of thyroid screening. Currently, beta-blockers, radioactive iodine therapy, antithyroid medications, and surgery are used to treat thyroid diseases. Since many thyroid disorders are linked to iodine deficiency, prevention is restricted to treating it. The body's equilibrium depends on these hormones being released into the bloodstream. Iodide absorption, oxidation, iodination, coupling, and storage in the thyroid follicles are all steps in the manufacture of thyroid hormones.

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due to hereditary or autoimmune causes. The potential therapeutic benefits of plants like *chamomile*,*bugleweed*, and *ashwagandha* on thyroid health are also being investigated. To improve the quality of life for people with thyroid problems around the world, evidence-based, context-specific guidelines for diagnosis, therapy, and care are crucial.

INTRODUCTION

A medical problem that prevents your thyroid from producing the proper amount of hormones is commonly referred to as thyroid illness. Individuals of various ages may be affected. The thyroid is a little gland beneath the skin that resembles a butterfly and is situated at the front of your neck. As a component of your endocrine system, it produces and releases thyroid hormones, such as triiodothyronine (T3) and thyroxine (T4), which regulate a number of vital bodily processes. The primary function of your thyroid is to regulate your metabolic rate, or how quickly you burn food. This is the mechanism via which your body converts the food you eat into energy. Your body's cells all require energy to function. Your entire body may be affected by an underactive thyroid. (1) In order for growth and metabolism to occur normally, thyroid hormones (TH) are essential. Thyroid conditions can significantly affect people's well-being, quality of life (QoL), and the nation's healthcare system.Thus, current, evidence-based guidelines for the diagnosis, treatment, and management of these illnesses that are tailored to the local context are urgently needed. Benign or malignant thyroid nodules, as well as hypo- or hyperfunction of the gland, can be symptoms of thyroid diseases. This clinical practice guideline focuses on thyrotoxicosis and hypothyroidism. [2]

Literature Review:

The thyroid gland is often found below and anterior to the larynx, and it is composed of two lobes joined by a thin isthmus. Every lobe is separated into lobules, which have 20–40 follicles distributed uniformly inside each. Cuboidal to low columnar epithelial cells lining the follicles produce thyroglobulin, the active thyroid hormone synthesized from iodinated precursor protein. The homogenous suspension known as colloid, which contains thyroglobulin, is kept in the lumen of follicles. Thyrotrophs in the anterior pituitary release TSH, also known as thyrotropin, into the bloodstream in response to trophic substances from the hypothalamus. Thyroid follicular epithelial cells have receptors for TSH, and when TSH attaches to one of these cells, a stimulatory G-protein is activated. This signaling pathway then causes the manufacture and release of thyroid hormone. (3)

Anatomy of thyroid :

The gland that resembles a butterfly, the thyroid, is situated in front of the neck, directly above the trachea. An adult human weighs between 15 and 20 grams of it. The isthmus, which passes through the second and third tracheal rings to connect the two lobes of the thyroid gland, is located in the middle of the upper trachea. In its anatomical position, the thyroid gland wraps around the cricoid cartilage and tracheal rings, posterior to the sternothyroid and sternohyoid muscles. Usually related to the spinal levels C5-T1, it is situated inferior to the laryngeal thyroid cartilage. The lateral suspensory ligament, also known as Berry's ligament, is a consolidation of connective tissue that connects the thyroid to the trachea. Each thyroid lobe is connected to the other via this ligament. Pre tracheal fascia encloses the visceral compartment of the neck, which contains the thyroid gland as well as the pharynx, esophagus, and trachea. (5) The hypothalamic-pituitary-thyroid axis is a self- regulatory circuit that is made up of the thyroid, anterior pituitary, and hypothalamus. Thyroxine, also known as tetraiodothyronine (T4), and triiodothyronine (T3) are the two primary hormones that the thyroid gland produces. To preserve appropriate feedback mechanisms and homeostasis, thyroid- stimulating

hormone (TSH) from the anterior pituitary gland, thyroid-releasing hormone (TRH) from the hypothalamus, and T4 cooperate synchronously. The symptoms of hypothyroidism, which is brought on by an underactive thyroid gland, usually include bradycardia, constipation, exhaustion, and weight gain. In contrast, weight loss, heat intolerance, diarrhea, fine tremor, and muscle weakness are symptoms of hyperthyroidism, which is brought on by increased thyroid gland function. The hypothalamus is where thyroid hormone regulation begins. Thyrotropin-releasing hormone (TRH) is released from the hypothalamus into the anterior pituitary gland through the hypothalamic- hypophyseal portal system. Thyroid-stimulating hormone (TSH) is released by thyrotropin cells in the anterior pituitary in response to stimulation by TRH. The cell bodies in the hypothalamic periventricular nucleus (PVN) produce the peptide hormone known as TRH. Before TRH reaches the anterior pituitary, it can concentrate in the hypophyseal portal circulation, which is where these cell bodies project their neurosecretory neurons. (4)

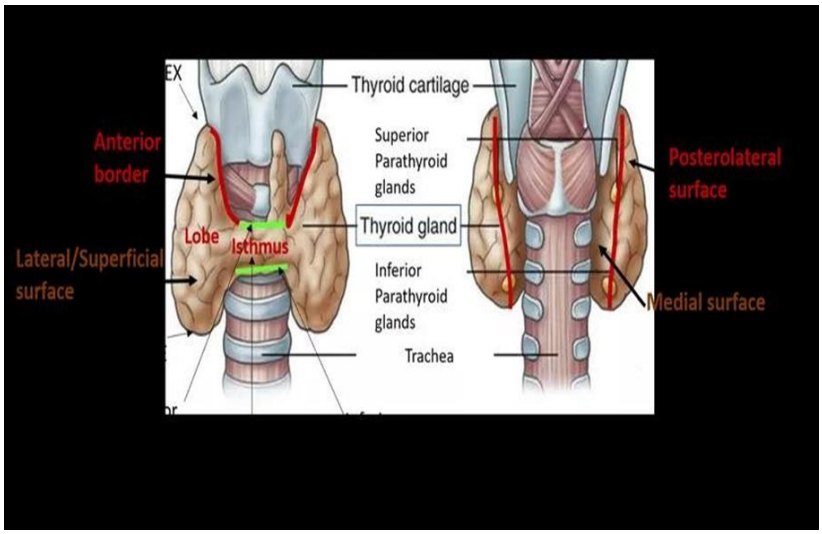


Fig [1] Anatomy Of Thyroid Gland (5)

Synthesis, storage & seceration of Thyroid Hormone :

The thyroglobulin molecule, a glycoprotein produced by thyroid cells with a molecular weight of MW 660 KDa and 10% sugar, is where the thyroid hormones are produced and stored in the thyroid follicles. The following procedures are involved in the production, storage, and release of T4 and T3, as shown in Fig. (2).

(1) The gland receives the inorganic iodide.

(2) Under the influence of peroxidase and H2O2, intrathyroidal iodide is converted to iodine.

(3) Monoiodotyrosine and diiodotyrosine are created when iodine and tyrosine are linked together in thyroglobulin.

(4) Thyroxine (t4) and triiodothyronine (t3) are produced by the enzymatic coupling of iodotyrosines.

(5) Thyroglobulin stores the iodothyronines, t4 and t3, until they are released into the bloodstream; and

(6) Iodide is recycled once unused iodotyrosines are deiodinated.(6)

Iodide Uptake.

About one-fifth of the 30–50 mg of I2 that the body contains overall from food and water is found in the thyroid. Although blood iodide concentrations are low (0.2–0.4 μg/dl), thyroid cells have an active transport mechanism (Na^+: I^- symporter or NIS) to concentrate this anion; TSH stimulates this trapping to surpass a gradient of

more than 100 times. The thyroid gland's I2 level controls the uptake process in some way, with big stores limiting it and meager stores activating it. The process that concentrates iodide is not exclusive to the thyroid; it is also present in the epidermis, salivary glands, gastric mucosa, gut, mammary glands, and placenta. However, TSH does not increase its uptake in these organs.

Oxidation & Iodination

With the aid of H2O2, iodide trapped by follicular cells is transported across the apical membrane by a different transporter known as "pendrin," and the membrane-bound thyroid peroxidase enzyme oxidizes it to iodinium (I+) ions, hypoiodous acid (HOI), or enzyme-linked hypoiodate (EOI). These forms of iodine combine avidly with thyroglobulin's tyrosil residues to form monoiodotyrosine (MIT) and diiodotyrosine (DIT) while the residues are still attached to the thyroglobulin chains.

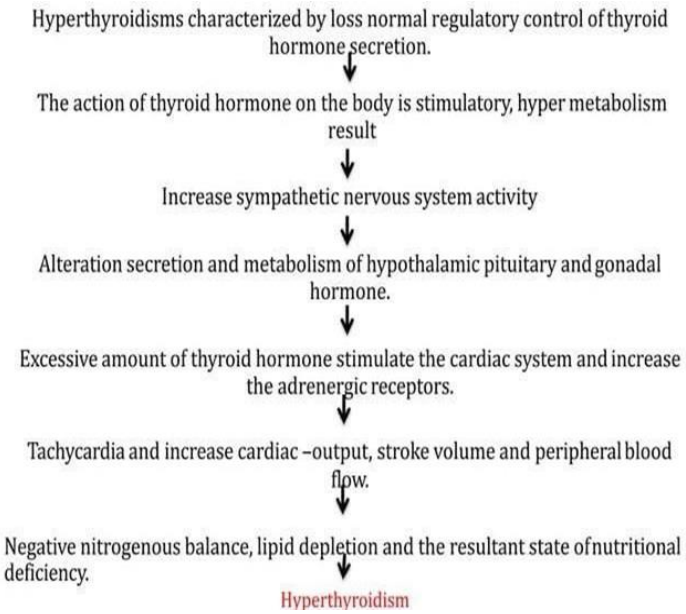
Storage & Release

Transported to the inside of the follicles, thyroglobulin with iodinated tyrosil and thyronil residues is kept as thyroid colloid until endocytosis returns it to the cells and lysosomal proteases break it down. While the MIT and DIT residues are de-iodinated and the iodide released is repurposed, the T4 and T3 that are so liberated are secreted into circulation. The quiscent gland has follicles enlarged with colloid and cells that are flat or cubical, but the TSH-stimulated gland has columnar cells and colloid that almost vanishes. TSH stimulates the uptake of colloid and proteolysis. Every day, the average human thyroid secretes 10–30 µg of T3 and 60–90 µg of T4.

Peripheral conversion of T4 to T3

T4 is converted to T3 by peripheral organs, particularly the liver and kidney. The majority of the T3 in plasma comes from the liver, and around one-third of the T4 released by the thyroid passes through this transformation**.** With the exception of the brain and pituitary, which absorb T4 and transform it into T3 inside their own cells, target tissues absorb T3 from the bloodstream for their metabolic needs. The peripheral produces nearly equal amounts of 3, 5, 3' triiodothyronine (normal T3: active) and 3, 3′, 5′ triiodothyronine (reverse T3: inactive) not carbimazole(7).

Pathophysiology of thyroid



Flow chart (1) of Pathophysiology of Thyroid (7)

Global situation

- Prevalence: Thyroid illness is thought to affect 200 million people globally.
- Gender: The prevalence of thyroid disease is 8:1 higher in women than in men.
- Risk factors: The most frequent cause of thyroid problems globally is iodine insufficiency.
- Cancer: The most prevalent endocrine cancer, thyroid cancer, is becoming more prevalent worldwide.
- Hyperthyroidism and hypothyroidism prevalence: In regions with adequate iodine, overt hyperthyroidism is found in 0.2–1.3% of people, while hypothyroidism is seen in 1-2.0% of people. It is estimated that up to 50% of the population has tiny nodules. Occult papillary carcinoma: This type Of cancer affects 3.5% of individuals. Fifteen percent of individuals have palpablegoiters.
- Abnormal level of thyroid-stimulating hormone: 10% of individuals have an abnormal level of this hormone. (8)

Types of thyroid disease:

Hpyothyrodism:

When the thyroid gland produces insufficient thyroid hormone to meet the body's needs, the condition known as hypothyroidism develops. Thyroid hormone influences almost every organ in the body and controls metabolism, or how the body consumes energy. Numerous bodily processes slow down when thyroid hormone levels are low. Hypothyroidism affects about4.6% of American saged 12 and up.(9) Congenital hypothyroidism is arguably the most significant of the different types of hypothyroidism because it necessitates an early diagnosis, which is typically followed by suitable treatment that can stop brain damage from developing. When compared to the global average, research from Mumbai has indicated that congenital hypothyroidism is prevalent in India, affecting 1 out of every 2640 newborns.1 in 3800 topics is the value. In the nation, the diagnosis of congenital hypothyroidism is frequently delayed. The absence of facilities or screening programs to thoroughly screen and test neonates for this illness, as well as a lack of knowledge about the sickness, are the causes of this delay. Children may also develop hypothyroidism. 79% of the 800 children with thyroid illness in a Mumbai clinic-based study had hypothyroidism. Thyroiditis, thyroid dysgenesis, and dyshormonogenesis were common causes of hypothyroidism in the sekids.(1) Recent research has examined the prevalence of hypothyroidism among Indian adults. Hypothyroidism was seen in 3.9% of the 971 adult participants in this Cochin population-based investigation. The frequency of hypothyroidism that is subclinical very high, with a value of 9.4% in this investigation. Compared to men, who had a frequency of 6.2%, women had a greater prevalence of 11.4%. As people aged, the prevalence of subclinical hypothyroidism rose. Anti-TPO antibody levels were positive in almost 53% of individuals with subclinical hypothyroidism. Cluster sampling was employed in this population- based investigation. In this investigation, 954 participants from the same group sampled had their urinary iodine status examined; the median value was 211µg/l (1).

Symptoms and causes

Thyroid dysfunction symptoms are sensitive yet nonspecific. Therefore, only a small percentage of individuals who complain of obesity, constipation, or cold intolerance are hypothyroid, even though weight gain, constipation, and cold intolerance are commonly associated with hypothyroidism. However, the likelihood of such a diagnosis increases with each new complaint, especially if there are physical signs of hypothyroidism. Laboratory results showing, for instance, a low total T4 and an increased TSH, indicating primary thyroid dysfunction, provide additional confirmation. In a similar vein, hyperthyroidism

symptoms are sensitive on an individual basis but specific only when combined.

1. **Symptoms of hypothyroidism include:**

- A slower heart rate than usual.
- Experiencing tiredness.
- Weight gain that is not explained.
- Having a chilly sensitivity.
- Dry skin and coarse, dry hair.
- A depressed state of mind.
- Prolonged menstruation (menorrhagia)

2. **Symptoms of hyperthyroidism include:**

- Tachycardia, or a faster-than-normal heart rate.
- Having trouble sleeping.
- Inexplicable loss of weight.
- Sensitivity to warmth. Sweaty or clammy skin.
- Having a worried, agitated, or anxious feeling.
- Amenorrhea, or irregular menstrual cycles or no periods.[1]

Thyroid disease-related enzymes or proteins:

TPO, or thyroid peroxidase It is a protein present in the thyroid gland and a crucial enzyme in the production of thyroid hormones. The different impacts of the TPO enzyme are as follows:

- **Autoimmunity and TPO**

Immunodominant region A (IDR-A) and B are two conformational characteristics on the molecule's surface that are targeted by the bulk of TPOAbs found in the sera of patients with AITD, including HT and GD. utilizing a panel of mouse monoclonal antibodies (mAbs) and patient autoantibodies, the first epitopic map of the TPO molecule surface was created. These findings were later validated by research utilizing recombinant human TPO-specific Fab fragments.

- **Expression of TPO in tissues outside the thyroid**

Although it is also found in various extra-thyroidal tissues, TPO is a crucial enzyme for the manufacture of thyroid hormone in thyrocytes. Normal human skin, tumoral and peri-tumoral human breast tissues, human kidney (case study), human thymus, and human and rat heart have all been found to have TPO mRNA and/or protein. Furthermore, human fibrocytes isolated from the blood of both normal and Graves' disease (GD) patients showed TPO mRNA expression.

- **TPO expression in tissues other than the thyroid**

TPO is an essential enzyme for thyrocytes to produce thyroid hormone, while it is also present in other extra-thyroidal tissues. It has been discovered that TPO mRNA and/or protein are present in normal human skin, tumoral and peri-tumoral human breast tissues, human kidney (case study), human thymus, and human and rat heart. In addition, TPO mRNA expression was detected in human fibrocytes that were isolated from the blood of both normal and Graves' disease (GD) patients.

- **TPO in cancer**

TPO's antigenicity remains unchanged despite a substantial drop in TPO expression in thyroid carcinoma (TC) tissues. TPO expression in breast cancer was found to exhibit similar characteristics. TPO immunostaining in TC cells occurs in the cytoplasmic periphery and is coarse. Malignant cells also lack the perinuclear ring-shaped staining of TPO that is characteristic of normal thyrocytes. Differences in the choices, as previously mentioned Conclusions.(10)

ELISA Capture

A pool of 20 sera from patients with AITD and anti-P14 antibody to TPO was examined using capture ELISA as reported, with a few minor adjustments, to evaluate the binding of rhFabs. Briefly, mAb A4 was applied at a concentration of 30 lg/mL in carbonate-bicarbonate buffer containing 0.1% sodium azide to the wells of polystyrene high binding microtiter plates (Costar), and the plates were then incubated at 4 C for the entire night. Wells were cleaned three times with TBS containing 0.1% Tween 20 (TBST) following each incubation. Total cell proteins isolated from CHO cells were added at a rate of 10 lg/mL in TBST containing 2 mg/mL BSA (TBST-

BSA) following blocking by incubation with TBS containing 2 mg/mL BSA. The next day, an OD at 450 was obtained by diluting anti-TPO-specific antibodies in TBST-BSA 1.0 to 2.0 nm, and then incubated for one hour at ambient temperature. After one hour of room temperature incubation with either an HRP-conjugated goat anti-human IgG Fab- specific (Sigma-Aldrich), an HRP-conjugated rabbit anti-human IgG (Sigma-Aldrich), or a H conjugated goat anti-rabbit IgG (Dako), the bound Fab fragments or rabbit antibodies were found. Tetramethylbenzidine (TMB) at 0.1 mg/mL in citrate buffer (pH 4.0) was used to develop the plates. 100 lL of 1 M sulfuric acid was used to stop the reaction, and a 450 n microtiter reader was used to determine the optical density.(11).

Screening method

"The application of a test to detect a potential disease or condition in a person who has no known signs or symptoms of that condition at the time the test is done" is the definition of screening methods. Accordingly, thyroid function test screening can detect patients with modest, nonspecific symptoms like heat or cold intolerance, as well as those who are asymptomatic. The clinician may be highly suspicious that the patient has thyroid illness if numerous symptoms and indicators appear at the same time. Patients who report one or two symptoms, however, might not be any more likely than those who do not to have abnormal thyroid function tests. In pregnant women and elderly individuals, such Trying to differentiate between "asymptomatic" patients and those who have symptoms that might or might not be connected to thyroid status is pointless because symptoms are so prevalent.(12).

1. **TSH:**

One of the best tests for evaluating thyroid function is this one. Three generations of assay tests have emerged in recent years. The diagnosis of primary hypothyroidism (low thyroid hormones owing to thyroid gland illness) can be aided by the ability of all three-generation assays to detect increased levels of TSH.immunological detection of the second generation.0.1 mU/Land are useful for hyperthyroidism screening.With detection limits of 0.01 m U/L, third-generation chemiluminescent tests are better able to differentiate between hypothyroidism and hyperthyroidism. TSH levels range between 0.4 to 4.2 mu*U/L at normal levels.

2. **Using immunometric methods** :

Such as chemiluminescence or radioimmunoassay (RIA), serum T3 and T4 levels are estimated. Normal T3 levels range from 70 to 204 ng/dl. Normal T4 levels range from 6 to 11 µg/dl. Only the free thyroid hormone is the active type; the other 99.9% of thyroid hormones are linked to proteins. As a result, methods for measuring free T_{y} and free T hormones have been established.The typical range for free T levels is 210–440 pg/dl. Free T_1 levels typically range from 0.8 to 2.7 ng/dl.

3. **Thyroglobulin**

Together with T y 'T_{4} and TSH levels, thyroid function testing can predict thyroglobulin levels. The thyroid gland's follicular cells synthesize it, and either benign or malignant thyroid cancers raise its levels in the blood.

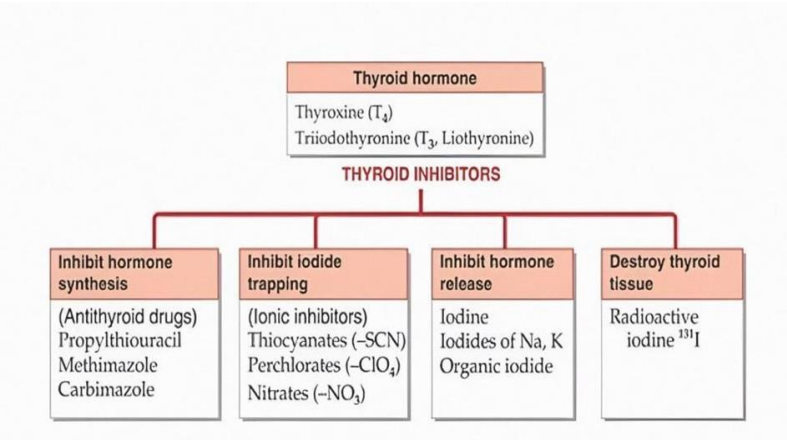
4. **Autoimmune markers**:

TSH receptor antibody tests (TRAB, TSH-Ab) can identify both stimulatory and TSH receptor-blocking antibodies. More than 70% of patients with Graves' illness will test positive for both stimulating and blocking antibodies. The hallmark of chronic lymphocytic thyroiditis, also known as Hashimoto's thyroiditis, is the generation of antibodies, which causes the gland to produce less thyroid hormone. Over 80% of these patients have the thyroid peroxidase (TPO) antibodies. Antibodies generated against thyro globuline are measured in another test.

5. **Aspiration Biopsy with Fine Needles:**

This aids in the evaluation of thyroid nodules. In addition to helping patients with benign nodules avoid needless surgery, it can aid in the early detection and treatment of thyroid cancers. For lesions that are not palpable, ultrasound imaging can help with biopsy.(13)

Antithyroid Activity



Classification Of Thyriod Inhibitors

Fig. (2)Classification of Antithyroid Drugs

Handling and Therapy

1. Antithyroid meds: propylthiouracil and methimazole: these drugs prevent your thyroid from producing hormones.

2. Radioiodine (radioactive iodine) therapy: This procedure harms your thyroid's cells, which stops it from producing large amounts of thyroid hormone. (radioactive iodine) therapy: This procedure harms your thyroid's cells, which stops it from producing large amounts of thyroid hormone.

3. Beta-blockers: These drugs help control certain symptoms, such as an elevated heart rate, but they have no effect on your thyroid.

4. Surgery: For a more long-lasting kind of care.(1)

Avoidance

In most cases, thyroid disorders cannot be avoided. This is due to the fact that the majority of thyroid disease instances are brought on by autoimmune diseases and/or heredity, neither of which can be prevented. Thyroid issues associated with either an excess or a deficit of iodine are the two conditions that you might be able to avoid. If you're worried about getting enough iodine in your diet, consult your doctor.(1)

Plants Used to Treat Thyroid Conditions

Plants have been used to improve thyroid function in many ways, particularly for conditions like hypothyroidism and hyperthyroidism. Some plants and herbs that are commonly discussed are as follows:

1. Mountaincoffee,
2. chamomile,
3. sage,
4. bugleweed
5. ashwagandha.

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