

# **Understanding Hypothyroidism**

## **Western and Ayurvedic Perspective**

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October 01, 2016

## **Introduction**

Millions of Americans suffer from fatigue, weight gain, depression, and cognitive impairment. Many believe that they have no choice but to accept and live with these seemingly “age-related” declines in quality of life. At the same time hypothyroidism has been increasingly linked to symptoms of fatigue, lack of energy and general malaise even when the blood values of thyroid hormone are seemingly within the normal range. More and more health care providers are now paying more attention in an attempt to understand what is referred to as sub clinical hypothyroidism.

Underactive thyroid or sub clinical hypothyroidism is often overlooked or misdiagnosed and can be the underlying cause of these symptoms. Patients and their doctors often disregard these common signs of thyroid hormone deficiency, mistaking them for normal aging. (1)

Hypothyroidism is a common endocrine disorder resulting from deficiency of thyroid hormone. The most common cause is considered an autoimmune condition called Hashimoto’s Thyroiditis, an autoimmune thyroid disease. This is the most common cause of hypothyroidism, worldwide; inadequate iodine intake or iodine deficiency remains the foremost cause. (2)

## **Anatomy and Physiology of the Thyroid Gland**

The thyroid is a small gland, immediately below the larynx (or the Adam’s apple), under the skin, measuring about 2 inches (5 centimeters) across, in the neck, anterior to trachea. The two halves (lobes) of the gland are connected in the middle (called the isthmus), giving the thyroid gland the shape of a bow tie as illustrated in figure 1. Normally, the thyroid gland cannot be seen and can barely be felt. If it becomes enlarged, doctors can feel it easily, and a prominent bulge (goiter) may appear below or to the sides of the Adam’s apple. (3)

The thyroid gland secretes thyroid hormones, which control the speed at which the body’s chemical functions proceed what is known as metabolic rate. Thyroid hormones influence the metabolic rate in the following ways:

- By stimulating almost every tissue in the body to synthesis proteins (4)
- By increasing the amount of intracellular enzymes and oxygenation (4)
- By increasing the size and number of the mitochondria in most cells of the body(4)
- By increasing the active transport of ions through cell membrane(4)

It is clear that we know that the thyroid hormones affect many vital function of the body at the cellular level influencing many of the physiological functions such as: the heart rate, the rate at which calories are burned, skin maintenance, growth, heat production, fertility, and digestion. “Yet a specific metabolic mechanism that leads to all of these effects has been elusive. But at present the most likely basic function of the thyroid hormones is their capability to activate the DNA transcription process in the cell nucleus with the resulting formation of many new cellular proteins, many of which are metabolic enzymes.” (4)

### Anatomy of the Thyroid and Parathyroid Glands

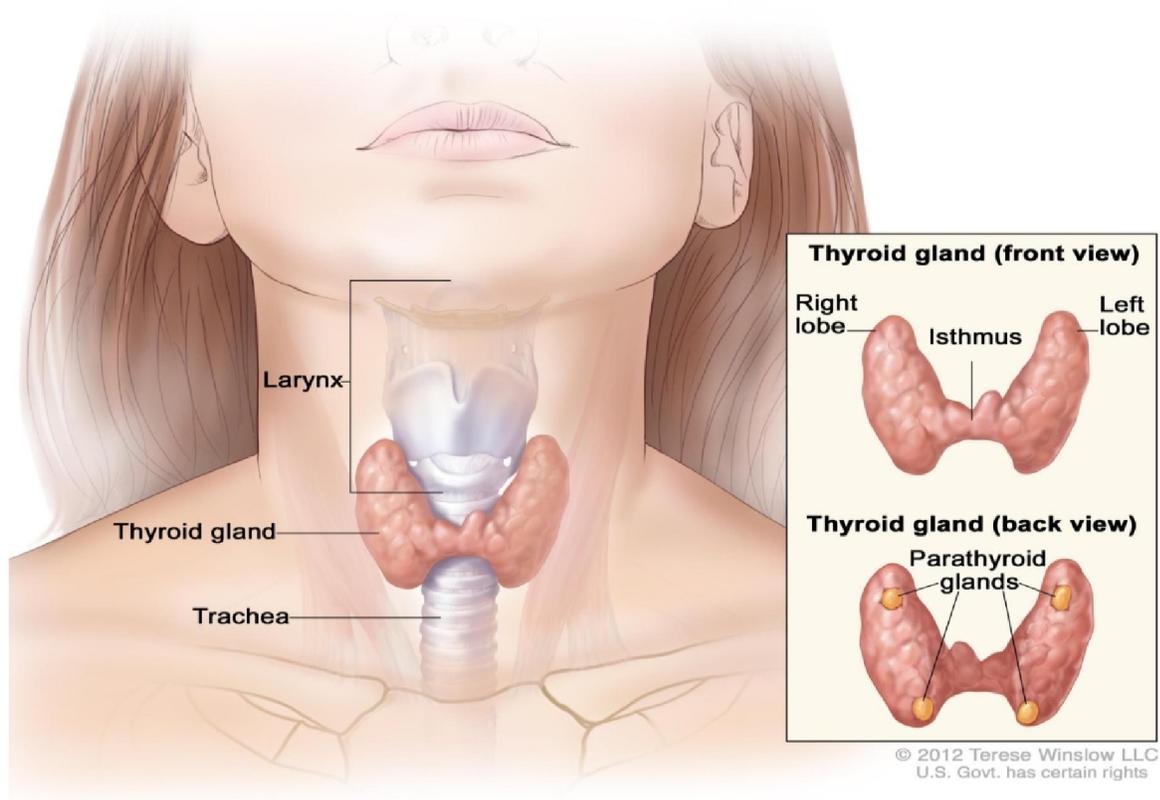


Figure 1

The thyroid gland is composed of large number of follicles lined with epithelioid cells the main constituent of which is a large glycoprotein called Thyroglobulin, which contains the thyroid hormone as part of its molecule. (4) Thyroid gland secretions are the 2 thyroid hormones Triiodothyronine or T3 and Thyroxine or T4 as well as Calcitonin. “The production of T4 and T3 in the thyroid gland is regulated by the hypothalamus and pituitary gland through a complex feedback mechanism. To ensure stable levels of thyroid hormone levels, the hypothalamus monitors circulating thyroid hormone levels and responds to low levels by releasing thyrotropin-releasing hormone (TRH). This TRH then stimulates the pituitary to release thyroid stimulating hormone TSH.” (5) When thyroid hormone levels increase, production of TSH decreases, which in turn slows the release of new hormone from the thyroid gland as illustrated in figure 2 below;

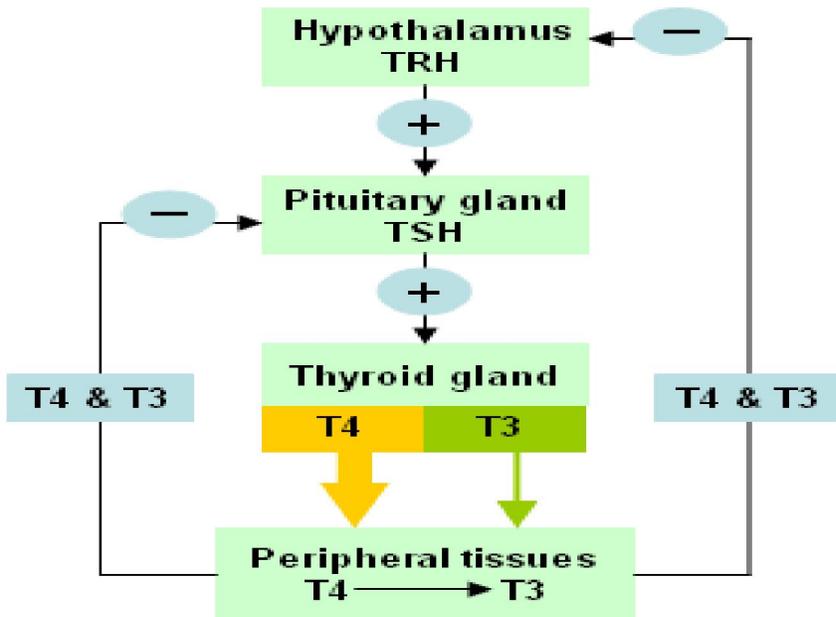


Figure 2

The most abundant form of Thyroid hormone is T4 thyroxine but not as potent as T3. On the other hand T3 is not as abundant in the blood stream however it is much more potent than T4 and persists for a much shorter time than thyroxine or T4.

## **Role of Iodine and L-Tyrosine in Thyroid Hormone Synthesis**

Iodine is an element which is found in water and food. Thyroid gland requires Iodine for the formation of the thyroid hormones. To form normal levels of Thyroxine approximately 50 mg of ingested iodine are required each year or 1 milligram per week to prevent iodine deficiency. The cell membranes of the thyroid follicles transport iodides into the cell via a transport mechanism called iodine pump. Each molecule of the thyroglobulin contains 140 tyrosine amino acids and these are the major substrates that combine with iodine to form the thyroid hormones. (6)

“The most important step in this process is the conversion of iodide ions to the oxides form of iodine that is capable of combining directly with the amino acid tyrosine. This is possible by utilization of an enzyme called Peroxidase and accompanying hydrogen peroxide which together provide a potent system capable of this oxidation process.” (6)

As illustrated in figure 3 on the next page, the iodized tyrosine molecules bind together to first form monoiodotyrosine and subsequently to diiodotyrosine. When 2 of the diiodotyrosines bind, as the result of this coupling process, T4 or Tyroxine is formed. T3 or triiodotyronine forms as a result of binding of a monoiodotyrosine with a diiodotyrosine. (6)

diiodotyrosine + diiodotyrosine → T4

diiodotyrosine + monoiodotyrosine → T3

### **Importance of Iodine:**

“A diet deficient in iodine can limit how much T4 the thyroid gland can produce and lead to hypothyroidism. Iodine (as the iodide) is relatively abundant in seawater and seafood is a rich dietary source. Fruits and vegetables also contain significant concentrations of iodine, although the amount depends on the soil and growing region. Areas of iodine deficiency tend to be inland, at high altitude and isolated and daily iodine intake may be as low as 25 µg. A lack of iodine in the diet leads to hypothyroidism”. (8)

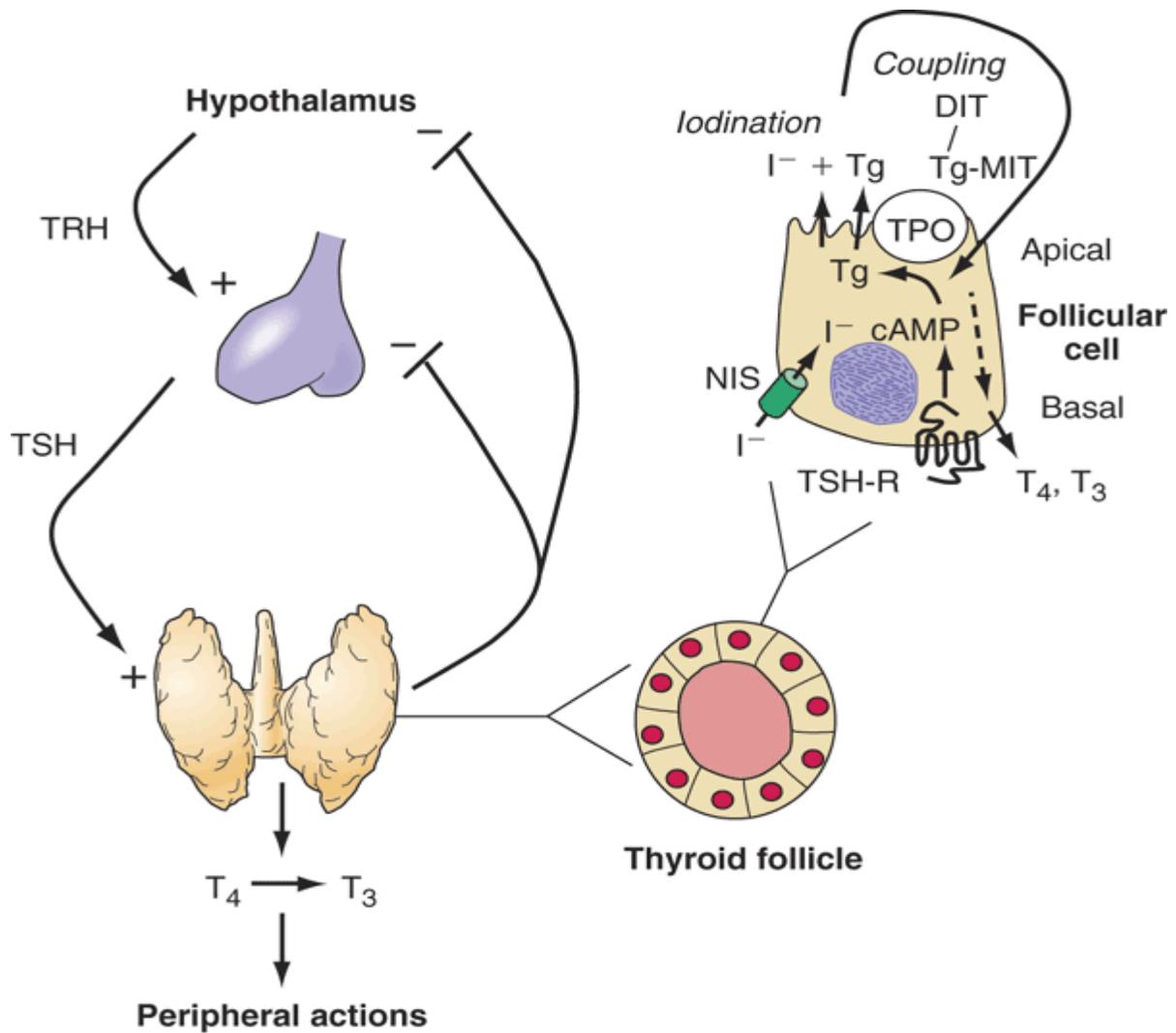


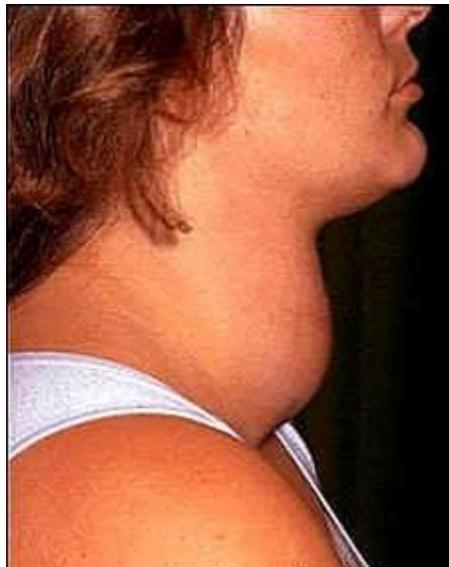
Figure 3

“The normal European daily dietary intake of iodine is about 150 µg, of which approximately 125 µg is taken up by the thyroid gland and used for hormone synthesis. The term cretinism, used to define the severe impairment of physical and neurological development resulting from iodine deficiency during fetal and post-natal development, derived from ‘cretein’, (Hypothyroidism in the unborn child, congenital hypothyroidism or cretinism) a term first used in the Swiss Alps. The word cretin entered the vernacular as a term of abuse indicating severe mental retardation”. Because of this, iodine deficiency remains one of the leading causes of mental retardation. (1)(8)

In 1994, almost 30% of the world population was at risk of iodine deficiency. The World Health Organization in 1990 made a commitment to eliminate iodine deficiency disorders by the year 2000. In 2007 the WHO estimated that over 30% of the world's population (2 billion people) has insufficient iodine intake as measured by urinary iodine excretion below 100 µg/L. (1), (10) This commitment by WHO is accomplished by a policy of supplementation of iodized salt and this has been proven effective as preventing iodine deficiency. (1),(10)

**Goiter; (Galaganda in Ayurveda)** which means a greatly enlarged thyroid gland is mainly due to iodine deficiency. The most common cause of Goiter formation is found in regions of dietary deficiency of iodine and is illustrated in figure 4. This is due to reduced synthesis of thyroid hormone and subsequent compensatory increased secretion of the pituitary hormone TSH, Thyroid stimulating Hormone as a result of feedback loop. Conversely increased iodine intake as well can induce goiter formation. This results from increased thyroid hormone synthesis and storage. (8)

Figure 4



## **Components of the Thyroid Hormones and their clinical significance**

### **T3, T4, Free T3, Free T4, rT3, DIT, MIT**

“Approximately 100 µg of thyroid hormones are secreted from the gland each day, mostly in the form of T4.” (8) About 90% of what we call thyroid hormone is in the form of T4 and only 10% makes up T3. Most of the circulating T3 and T4 are bound to proteins for transport rendering them inactive. The main bounding protein is thyroxine binding globulin or TBG which accounts for 80% of the bound thyroid hormone. The remaining 20% is bound to albumin and thyroxine binding prealbumin. Only about 0.05% serum T4 and 0.5% of T3 remains free and active. It is important to note that only Free T3 and Free T4 are available to act on the peripheral tissues. (11)

### **Role of Selenium in T3 Synthesis:**

Only about 15% of the circulating T3 hormone is produced by the thyroid gland but the majority is produced as a result of T4 undergoing conversion to the more active T3 at the peripheral tissue, mainly in the liver and but also kidney. This conversion is carried out by a selenium-dependent enzyme resulting in production of the biological active form of thyroid hormone; T3 which is 10 times more potent than T4 but less abundant. (1)(11) (8) “Various factors including nutrient deficiencies, drugs, and chemical toxicity may interfere with conversion of T4 to T3”. (7)(30)

“**Reverse T3 or rT3** is another form of inactive T3 as a result of T4 conversion by an enzyme in liver and kidney which does not have any hormonal potency; “instead it blocks the thyroid hormone receptors in the cell hindering action of regular active T3”. (9) T3 is also more potent than reverse T3 (rT3) that has little or no biological activity. (1)

“Very small quantities of other iodinated molecules, such as MIT (monoiodothyronine) and DIT (diiodothyronine) as well as thyroglobulin, are also measurable in the circulation. As this thyroglobulin originates from the normal secretory process, its measurement in the serum is used, for example, to detect endogenous thyroid secretion when patients are taking oral T4 replacement which is an important clinical use”. (8)

The thyroid gland needs iodine and Selenium and the amino acid L-tyrosine to make T4 and T3. A diet deficient in iodine and selenium can limit how much T4 thyroid gland can produce and how much is converted to T3, leading to hypothyroidism. (1)(12)

## **Physiological Effects of Thyroid Hormone**

- Stimulating effects on carbohydrate metabolism, by rapid uptake of glucose, enhanced glycolysis and gluconeogenesis, increased insulin secretion, increased rate of absorption from the GI tract. (4)
- Enhancing effects on fat metabolism, mobilization of lipids from the fat tissue, increasing fatty acid concentration in the plasma, accelerating fatty acid oxidation(4)
- Effects on body weight; increased hormone decreases body weight and decreased hormone increases the body weight.(4)
- Effects on the cardiovascular system; rapid utilization of oxygen causes vasodilatation and increased blood flow to all body parts.(4)
- Increase heart rate and cardiac output and increase pulse pressure.(4)
- Effects on respiration; increased metabolism and increased utilization of oxygen and the resultant carbon dioxide formation activate mechanisms of increased rate and depth of respiration. (4)
- Effects on GI tract; increased rate of secretion of digestive juices as well as motility of the GI tract. This may result in diarrhea and increased appetite and lack of the hormone can cause constipation. (4)
- Effects on Central Nervous System; in general thyroid hormone increases the rapidity of cerebration and on the other hand lack of thyroid hormone decreases this function. Hence a hypothyroid individual tends to have extreme nervousness and other psychoneurotic tendencies such as anxiety, extreme worry and or paranoia. Also in hyperthyroid individuals a muscle tremor may be present. (4)
- Effects on Sleep; due to its effects on CNS and musculature, hyperthyroid condition causes excitation of the nervous system and though the person often has a feeling of constant tiredness but they cannot sleep. On the other hand with hypothyroidism the person is somnolence and sleeps longer than average. (4)

The physiological effects of the thyroid hormones on the different systems and tissues of the body therefore have a direct correlation with the signs and symptoms of too little or too much of the hormone in hypothyroid and hyperthyroidism.

## **Hypothyroidism and Subclinical Hypothyroid**

Hypothyroidism is a condition in which the thyroid gland does not make enough thyroid hormones, characterized by a reduction in metabolic rate and it is more common in women. Pregnant women and women older than 60 as well as patients with type 1 diabetes or other autoimmune disease are at a higher risk. (18) Hashimoto's Thyroiditis is the most common cause of primary hypothyroidism in which body's immune system mistakenly attacks the thyroid tissue impairing the ability to make the hormone. (13) Secondary Hypothyroidism can be a result of Hypothalamus or Pituitary glands dysfunction or disease.

Subclinical hypothyroidism, an often under-diagnosed thyroid disorder, manifests as elevated TSH, normal T4 and normal T3 levels. (14) Individuals with subclinical hypothyroidism are at greater risk for developing overt hypothyroidism. (15) An August 2010 study reported that 8.3% of women with no history of thyroid disease suffer from subclinical hypothyroidism. (16) An article in the American Family Physician in 2005 estimated that about 20% of women over the age of 60 suffer from subclinical hypothyroidism. (17) "The new guidelines (2003) narrow the range for acceptable thyroid function and the AACE is now encouraging doctors to consider thyroid treatment for patients who test outside the target TSH level of 0.3 to 3.04, which is a far narrower range." (29)

The main symptoms of hypothyroidism are fatigue, weakness, increased sensitivity to cold, constipation, hoarseness, unexplained weight gain, dry skin, hair loss or coarse dry hair, muscle cramps, headache, muscle weakness, joint stiffness, elevated LDL cholesterol, depression, sleepless (if adrenals are involved) or too much sleep and memory loss. However, most symptoms take years to develop. The slower the metabolism gets, the more obvious the signs and symptoms will become. If hypothyroidism goes untreated, the signs and symptoms could become severe, such as a swollen thyroid gland (goiter), slow thought processes, or dementia and impaired fertility. (18)(31)

## Testing Thyroid Function

**Thyroid stimulating hormone (TSH);** TSH level is the most common test for screening thyroid dysfunction. “It is now recognized that the TSH measurement is a more sensitive test than T4 for detecting both hypo- and hyperthyroidism. (20) In 2008 many labs adopted the reference range for TSH, 0.45 to 4.50  $\mu$ IU/mL recommended by both the Endocrine Society and the American Medical Association. Although this range is an improvement over the previous 0.45-5.5 mIU/L, it is still considered too broad by many clinicians”. (20)(21)(22)

There is evidence that the standard blood TSH test reference range may cause many cases of hypothyroidism to be missed. According to a report in Lancet, various TSH levels that fall within normal range are associated with adverse health outcomes. (22)(1)

- TSH greater than 2.0: increased 20-year risk of hypothyroidism and increased risk of thyroid autoimmune disease
- TSH between 2.0 and 4.0: hypercholesterolemia and cholesterol levels decline in response to T4 therapy
- TSH greater than 4.0: greater risk of heart disease

In functional medicine many physicians are adopting a smaller range for the normal values of TSH, for example Life Extension suggests an optimal level of TSH between 1.0 and 2.0  $\mu$ IU/mL, as some studies have noted that a TSH above 2.0 may be associated with adverse cardiovascular risk factors. In addition, a TSH between 1.0 and 2.0  $\mu$ IU/mL has been associated with the lowest subsequent incidence of abnormal thyroid function. (1) (24)

**Tests for T4 and T3;** Thyroid hormones can be tested in both their free and protein-bound forms. Tests for the protein-bound forms and unbound form of T4 or T3 are generally referred to as Total T4 or Total T3 respectively; unbound forms are called Free T4 and Free T3. Each of these tests gives information about how the body is making, activating, and responding to thyroid hormone. Free T4 is a more accurate measurement because the bound level can be affected by protein levels, pregnancy, and hormone replacement therapy. Levels of free T3 and T4 will be below normal in clinical hypothyroidism. In subclinical hypothyroidism the TSH will be elevated while the thyroid hormone levels are still in the normal reference range (1)

The most common cause of low thyroid function is the autoimmune disease, Hashimoto's thyroiditis. A decreased level of total T4 may indicate a protein-depleted state if free T4 is normal. (25)

T3 is primarily used to diagnose hyperthyroidism. The conversion of T4 to T3 occurs in the liver and non-thyroid disorders can disrupt the production of T3. As a result, hypothyroidism is difficult to diagnose using T3. However, the conversion requires a selenium-dependent enzyme and its deficiency can cause hypothyroidism. The same limitations mentioned above for measuring total T4 exist with the measurement of total T3. As a result, free T3 provides a more accurate indicator of hyperthyroidism. (25)(26)

**Reverse T3 (rT3)** is an inactive form of T3 that can interfere with regular T3 activity by blocking the thyroid hormone receptors in cells. In some individuals although T4 and T3 levels may be normal, due to increased levels of rT3; the hormones are unable to penetrate the cells because cell receptors are blocked by rT3. This will result in classic symptoms of hypothyroidism despite adequate levels of thyroid hormone. (1)

**Antithyroglobulin antibody, AgAb;** The antithyroglobulin antibody test can be used to identify autoimmune thyroiditis such as Hashimoto's and other diseases involving the thyroid. This is usually conducted in conjunction with antithyroid peroxidase antibody test to increase the sensitivity of the test. (25) Thyroglobulin is a protein specific to the thyroid that is involved in thyroid hormone production, storage, and circulation. When there is a demand for TG production, it is released from the thyroid follicles.(25)Antibodies form against the TG when they are released into the bloodstream due to injury to the thyroid follicles from inflammation, quick growth, or a hemorrhage. Some diseases that may cause this include: Hashimoto's thyroiditis, nontoxic goiters, Graves disease, Thyroid cancer, myxedema.(25)

**Basal Body Temperature;** An alternative method for assessing thyroid status is widely used in addition to accurate thyroid function blood tests and that is the basal body temperature test. The temperature is taken when the body is at complete rest, immediately after waking and before beginning any activity. The normal basal temperature is 97.6-98.2 °F, and some alternative practitioners believe that a 5-day consecutive temperature reading below 97.6 °F is indicative of hypothyroidism. (1)(28)

**Additional testing;** “Sometimes biopsy or enzymatic studies are required to establish a definite diagnosis for thyroid dysfunction. Major abnormalities of the thyroid gland detected in physical exam can be further assessed by ultrasound or a procedure known as scintigraphy.” (1) Additional tests may include Thyroid-stimulating immunoglobulins (TSI) and Antithyroid peroxidase antibody (ATPOAb). (25)

## **Stress, Adrenal glands and Hypothyroidism**

**Adrenals;** Medical literature and research suggest a strong relationship between the thyroid, the adrenal glands and the sex hormones. (27) If hypothyroidism is suspected, an evaluation of the adrenal glands as well as the sex hormones is suggested. Elevated cortisol levels decrease TRH as well as TSH and thereby decrease T3 and T4 and blocking conversion of T4 to T3. So in cases of suspected adrenal involvement cortisol can be measured by a salivary adrenal stress test. (1)(28)

Stress is known to be a significant contributor to thyroid dysfunction. Whenever stress is experienced, adrenal glands produce cortisol. This is an evolutionary protective mechanism that originally developed as a response to physical threats. It creates the “fight or flight” response and once the physical threat is gone, cortisol levels go back to their normal levels.” Long terms effects of stress and subsequent continuous high levels of cortisol cause what is called; “Thyroid Resistance”. (31)

“Every cell in the body has receptors for both thyroid hormone and cortisol. Cortisol acts synergistically with thyroid hormone at the epigenetic level. Normal levels of cortisol (neither too much nor too little) need to be present bound to its receptors for optimal function of not only the thyroid gland itself but for every tissue in the body. Too much cortisol causes the tissues to no longer respond to the thyroid hormone signal. This is known as thyroid resistance, meaning that thyroid hormone levels can be normal, but tissues fail to respond as efficiently to the thyroid signal. It can cause TSH levels to be elevated while T4 and T3 are within the normal range.” (31)

Excess cortisol can also inhibit deiodinase, the enzyme responsible for the conversion of T4 into T3 in the peripheral tissues, thereby leading to hypothyroidism. (31)

“High levels of cortisol can also lead to an increased risk of Hashimotos thyroiditis by causing an abnormal ratio between T1 and T2 lymphocytes. A shift toward T1 may be associated with Hashimotos. (31) So when stress and cortisol is the cause of hypothyroid symptoms, TSH, T3 and T4 may be normal but the body will most likely be making rT3. In this case the individual is treated with T3 and this may relieve the symptoms. (28)

## **Ayurvedic Interpretation of Thyroid Gland**

Even though there are no direct references in Ayurvedic classical texts in terms of hyper or hypo production of the hormone by the thyroid gland (Avatu Granthi in Sanscrit), but there is a disease by the name Galaganda and Gandamaala, characterized by neck swelling that is known to be diseases of Thyroid gland. (33)(32) “The first description of neck swelling was mentioned in Atharva Veda by the name Apachi. Charaka mentioned the disease under 20 sleshma vikaras (34). Sushruta (renowned ancient Indian surgeon) in Shareera Sthana has mentioned that of the seven layers of the skin, the sixth layer Rohini is the seat of Galaganda (35). In Nidana Sthana he described Galaganda as two encapsulated small or big swellings in the anterior angle of the neck, which hang like scrotum (36), whereas Charaka mentioned Galaganda as a solitary swelling.” (37)

The climatic conditions, water supply, dietary conditions, etc., are mentioned as the main etiological factors. Sushruta stated that Himvatprabhava rivers might give rise to the occurrence of Galaganda (38). “Bhela described that Sleepda and Galaganda are more common in prachya desa (eastern part of India) of the country, and that persons consuming predominantly fish are liable to develop Galgaganda. Harita Samhitakara described the role of dushtambu (contaminated water) and krimi dosha (infection) in the precipitation of Galaganda.” (39). “Kashyapa Samhitakara added that any part of the country that is cold, damp, with densely grown long trees, water stagnation and heavy rains may be prone for the development of Galandana.” (40)(32) Today in maintaining region of Himalaya and Himachala pradesh endemic Goiter is more common. It is due to the less iodine content in water. (43)

From the description of Galgaganda it may seem reasonable to assume the condition refers to Goiter which is abnormal swelling in the thyroid gland with the most common etiology being Iodine deficiency. And based on the western interpretation of the Goiter, this is a condition in which the thyroid gland is over stimulated by over production of TSH in hypothyroidism. Conversely increased iodine intake as well can induce goiter formation, perhaps due to dietary habits and climatic conditions as mentioned above per the references to the classical Ayurvedic texts.

In Ayurveda Galganda is due to vitiation of the Kapha dosha mainly but also of vata and medas. (33). But it is mainly due to vitiation of the Kapha which then results in disturbances of medas dhatu which then forms the Galganda. (41)(42) “Rasadhatu plays a major role in pathogenesis as Rasaja Vikaras mentioned in Charak samhita are similar to the clinical features of hypothyroidism. Hormonal disturbances are the dysfunction of Agni. Rasadhatvagni-mandhyata leads to Rasa Vridhi and over production of Mala of Rasadhatu i.e. Mala Kapha Vridhi. Dhatvagnimandhya is also the major features of the disease and all these features contribute with the modern concept of metabolism i.e., decreased Basal Metabolic Rate.” (41)(43)

### **Ayurvedic Pathogenesis of Hypothyroidism**

Whether caused by an autoimmune disorder and most commonly Hashimoto’s or due to iodine deficiency, the condition usually begins as a vata-pitta imbalance followed by vata pushing kapha out of balance. Once clinical hypothyroidism is present kapha and its subdosha, Avalambaka are vitiated, accumulate in AnnavahaSrota and then relocate into the rasa dhatu of the thyroid glands RasavahaSrota being a hypervascular epithelia tissue. If left untreated, many dhatus can be affected. (33)

The original vata-pitta imbalance dries the tissues and drains the ojas and thus symptoms that are Kapha in nature will arise. On the more subtle levels, energetically, the flow of prana through the vishuddha chakra is diminished. (33)

## **Ayurvedic and Alternative Treatment of Hypothyroidism:**

- Eating goitrogenic foods such as rapeseed, cabbage, brussels sprouts, broccoli, cauliflower, kale, sweet potatoes, maize, lima beans, soya, pine and millet should be limited. Cooking is known to make the goitrogens elements less effective, but it would be wise not eat these foods raw. (33)
- Build ojas by resting and reducing sexual activity. If lethargic then chanting mantras and pranayama should be reduced or stops until energy is regained. (33)
- Reduce ama and increase Agni by utilizing a kitcheree mono diet combined with intake of dipanas and pachanas (33)
- A vata kapha pacifying diet by avoiding cold, dry and heavy foods increasing sour and pungent taste and reducing salty and astringent and avoiding sweet and bitter taste. (33)
- If overweight, use herbs; Lekhanas such as guggul & chitrak but use caution as pitta may be vitiated so use cooling Lekhanas and dipanas
- Manage constipation with herbal laxatives, dry skin with oil massage and sluggishness with nervine and circulatory stimulants and excessive menstrual bleeding with reproductive hemostats (33)
- Herbs; Bladderwrack, Kachanar, Kelp and Guggul, Maca, Ashwagandha, Coleus forskohlii and classical polyherbal Ayurvedic formula Kachnar guggulu. (33)(31)(44)(45)
- Iodine is recommended found in shellfish, seaweed, deep-water fish and certain vegetables, including: garlic, chard, spinach, and mustard and turnip greens, lima beans, sesame seeds.. The iodine content in food *does not* seem to aggravate a Hashimoto's thyroid in the same way as it does when administered in supplements, but it is still prudent not to consume these items in large quantities in case of hashimoto's.(30)(32)
- Selenium is recommended daily either through the diet or supplementation. (30)
- Yoga Sarvangasana (shoulder stand), Matsyasana (fish pose) and Halasna (plough pose), Surya Namaskara (Sun salutation), Pavanamuktasana (wind relieving pose) with emphasis on head and neck exercises, Supta Vajrasana (sleeping thunderbolt pose) and all backward bending asanas. (31)
- If ojas are good pranayama can include Nadi Shuddhanam, Jalandhar bhanda and Ujjayi Pranayaam (31)
- Desiccated Thyroid; Armour thyroid, Nature-throid, and Westhroid are prescription medications that contain desiccated porcine thyroid gland. (1)

Natural thyroid extracts have been used since 1892 and were approved by the Food and Drug Administration in 1939. Armour thyroid and most other natural glandular preparations are made to standards approved by the United States Pharmacopoeia.

- Other supplements; zinc, copper, L-tyrosine, Curcumin (1)

### **Abstract:**

“Thyroid problems are among the most common endocrine disorders presently seen worldwide. Hypothyroidism results when the thyroid gland fails to produce enough of the thyroid hormone, due to structural or functional impairment that significantly impairs its output of hormones, this leads to the hypo metabolic state of hypothyroidism. It is estimated to affect between 3.8-4.6% of the general population. The prevalence of primary hypothyroidism is 1:100, but increases to 5:100. The female-male ratio is approximately 6:1. There is no direct reference of thyroid in *Ayurvedic* classics, but *Galganda* and *Gandmala* have been frequently used in these classics. According to *Charaka* presentation of multiple *Granthi* around the neck is called *Gandmala* and single swelling on the *Parshava* of the neck is *Galganda*. So *Galganda* and *Gandmala* can be co-related with hypothyroidism. The incidence of hypothyroidism is increasing day by day, and there is increasing demand to treat the disease through the *Ayurvedic* system of medicine, as it is completely natural and safe. The root cause of hypothyroidism is disequilibrium of *tridosha*. In this article effort is made to review some *Ayurvedic* herbs for correction of imbalance in *tridosha* and flawed function of the thyroid gland. Keywords: Hypothyroidism, *Galganda*, *Gandamala*, *Ayurvedic* herbs.” (32)

“According to the American Association of Clinical Endocrinologists (AACE), doctors have typically been basing their diagnoses on the "normal" range for the TSH test. The typical normal levels (at most laboratories) have fallen in the 0.5 to 5.0 range. The new guidelines (2003) narrow the range for acceptable thyroid function and the AACE is now encouraging doctors to consider thyroid treatment for patients who test the target TSH level of 0.3 to 3.04, a far narrower range. AACE believes the new range will result in proper diagnosis for millions of Americans who suffer from a mild thyroid disorder but have gone untreated until now.

At a press conference, Hossein Gharib, MD, FACE, and president of AACE, said: "This means that there are more people with minor thyroid abnormalities than previously perceived. AACE estimates that the new guidelines actually double the number of people who have abnormal thyroid function, bringing the total to as many as 27 million, up from 13 million thought to have the condition under the old guidelines." (29)

"*Ayurveda* from its existence into the modern world is facing a major challenge. Endocrine disorders are difficult to understand in *Ayurveda*. Though certain disorders like Diabetes mellitus are well described in various *Ayurvedic* texts but as far as diseases of thyroid gland are concerned they are not well understood. In *Ayurveda*, we correlate the disorders caused by thyroid gland as *Galganda*, *Gandmaala* etc but the concept of hormone overproduction or under secretion is somewhere missing. Hence, here is an attempt to get the understanding of disease Hypothyroidism with *Galganda* through various *Ayurvedic* principles so as to set its treatment goals. Keywords: Endocrine Disorders, *Galganda*, *Gandmaala*, Hypothyroidism, Thyroid gland". (41)

"An extract of the plant *Coleus forskohlii* has been used for centuries in Ayurvedic medicine to treat various diseases such as hypothyroidism, heart disease, and respiratory disorders. Additionally, complex herbal mixtures containing this extract are gaining popularity in United States for their putative "fat-burning" properties. The active ingredient in *C. forskohlii* extract is the diterpene compound forskolin. Forskolin is a widely used biochemical tool that activates adenylyl cyclase, thereby increasing intracellular concentration of cAMP and thus activating the protein kinase A (PKA) signal transduction pathway. We show herein that both forskolin and its nonadenylyl cyclase-activating analog 1,9 dideoxyforskolin induce CYP3A gene expression in primary hepatocytes by functioning as agonists of the pregnane X receptor (PXR). We show that activation of PKA signaling potentiates PXR-mediated induction of CYP3A gene expression in cultured hepatocytes and increases the strength of PXR-coactivator protein-protein interaction in cell-based assays. Kinase assays show that PXR can serve as a substrate for catalytically active PKA in vitro. Our data provide important insights into the molecular mechanism of both the PKA-dependent and -independent effects of forskolin on the expression of drug-metabolizing enzymes in liver. Finally, our data suggest that herbal therapy with *C. forskohlii* extract should be approached cautiously due to the potential for herb-drug interactions in patients on combination therapy." (44)

“*Ayurveda* is one of the traditional medicinal systems of Indian. The philosophy behind *Ayurveda* is preventing unnecessary suffering and living a long healthy life. *Ayurveda* involves the use of natural elements to eliminate the root cause of the disease by restoring balance, at the same time create a healthy life-style to prevent the recurrence of imbalance. Herbal medicines have existed world-wide with long recorded history and they were used in ancient Chinese, Greek, Egyptian and Indian medicine for various therapies purposes. World Health Organization estimated that 80% of the world's inhabitants still rely mainly on traditional medicines for their health care. The subcontinent of India is well-known to be one of the major biodiversity centers with about 45,000 plant species. In India, about 15,000 medicinal plants have been recorded, in which the communities used 7,000-7,500 plants for curing different diseases. In *Ayurveda*, single or multiple herbs (polyherbal) are used for the treatment. The *Ayurvedic* literature *Sarangdhar Samhita* highlighted the concept of polyherbalism to achieve greater therapeutic efficacy. The active phytochemical constituents of individual plants are insufficient to achieve the desirable therapeutic effects. When combining the multiple herbs in a particular ratio, it will give a better therapeutic effect and reduce the toxicity. This review mainly focuses on important of the polyherbalism and its clinical significance. Keywords: *Ayurveda*, *panchamahabhutas*, polyherbal formulation” (45)

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