3. REVIEW OF SCIENTIFIC LITERATURE

Aim to examine the consequence of *yoga* on thyroid dysfunction, and very few studies are available. One of the studies conducted in of Human Consciousness and *Yoga* Science department, Mangalore University, included seven (five female and two male) subjects. The purpose is to know the change in thyroid functions before and after practicing *yoga* exercises for the subjects are aged between 20 to 50 years. Before practicing *yoga*, the subjects underwent increased or decreased Weight, fatigue, hair loss, constipation, sluggishness, and improper hormone level. *Yoga* practice has reduced all the issues significantly. The *yoga* practices fetched good improvement in the thyroid disorder patients and variation in the T3, T4, and TSH can be seen after *yoga* practice. In the therapeutic view, psychosomatic diseases are aroused from the *cittavritti* (mind stuff). So, by decreasing the *cittavritti*, one can achieve good health. By practicing *yoga*, one can see the clear cut improvement in the entire subject domain's health. This was achieved by the regular *yoga* course (Sharma & Mahabala, 2016). Another case report conducted by Bhavanani and others confirmed that average thyroid level is maintained through *yoga* (Bhavanani et al., 2011). Another study conducted by Pajai and others, *yoga* role on the prevention of hypothyroidism concluded that *yoga* effectively brought successful results in subjects with hypothyroidism in controlling the symptoms. *Yoga* is a unique complementary therapy in treating hypothyroid disorder when it is combined with medical therapy (Pajai & Pajai, 2014). One pilot study on hypothyroidism conducted by Nilakanthan and others concluded that six months of *yoga* practice improved cholesterol level and serum TSH, further there is reduction in thyroxine requirement in female hypothyroidism patients. They conclude that further there is a need for randomized controlled studies to confirm the present findings (Nilakanthan et al., 2016). The available antithyroid medications in the market today are associated with severe harmful side effects, and they reduce thyroxine (T4) and triiodothyronine (T3) hormones discharge and they significantly damage the liver. These medicines also increase the vulnerability to infections, cause allergic reactions, and lead to premature death (Kiran & Neeta, 2017). In a previous report, the impact of six months of intensive *yoga* practice on serum TSH levels in hypothyroidism suffering women: a pilot study reported an increase in

TSH levels and beneficial hypothyroidism results (Bhavanani et al., 2011; Savitri et al., 2016). In a survey of twenty hypothyroidism women, one month of *yoga* practice resulted in a considerable modification in Quality of Life (Singh et al., 2011), and Bhavanani records a significant decrease in TSH levels in another case study (Bhavanani et al., 2011). The patient had hypothyroidism with a TSH of 36.6, according to one case report on hypothyroidism. The TSH level was lowered to 5.82 after the *yoga* therapy was completed. The patient did not get current medical care (Kumar et al., 2018). The effectiveness of *yoga* exercises on thyroid disorders has been relatively shown, based on improvements in the levels of these hormones in the bloodstream and there is reduction in the left ventricular ejection fraction and myocardial contractile due to *yoga* is observed in the study (Krishna et al., 2014). One of the studies described an increase in parasympathetic activity and a decrease in sympathetic activity due to practicing of *yoga* (Dvivedi et al., 2008; Vempati & Telles, 2016). *Yoga* can reduce subjects' anxiety with a comprehensive but brief *Yoga* based lifestyle intervention (Rocha et al., 2012). Wallace's study on thyroid disorder patients concluded that *yoga* relaxation techniques might produce psychosomatic harmonization and induce a sense of calmness. *Yoga* brings serenity due to hypo mental activity (Wallace et al., 1971) and showed a reduction in physical demand for thyroxin due to a decrease in metabolic activity (Rawal et al., 1994). In one of the scientific research, considerable changes in T4 levels were identified in hyperthyroidism patients. This observation was in line with previous findings. A study observed effective control on TSH due to *yogic* practices (Bhavanani, 2011; Savitri et al., 2016). One of the studies showed a significant increase in T3 level with a twelve week program of *yogic* practices (Maske & Barnwal, 2016). In a survey of *yoga* programs among female hyperthyroidism patients, practicing *yoga* has improved the endocrine activity and corrected metabolism and biological functions. The value of T4 decreased significantly due to the practice of *yoga*. The study also used the WHO Quality of Life scale to assess the quality of life of female hypothyroid patients, demonstrating the usefulness of *yoga* in controlling hypothyroid symptoms (Akhter, 2019; Shivaprasad et al., 2018).

3.1 HOW TO IMPROVE HYPOTHYROID CONDITION

3.1.1 Stress Coping Techniques – Stress coping techniques include *yoga*, spiritual prayers, and meditation. The adrenal adaptogens or adrenal glandular are also considered in tolerating stress and increasing energy levels. Diet suitable for hypothyroidism and hashimoto's, and the diet should increase energy level and aids in Weight loss. Exercise and routine movement are a combination of both high intensity and low intensity exercises. Initially, low intensity exercises such as walking are suggested, and high intensity exercises once the thyroid function is improved (Ciloglu et al., 2005). Finally, high quality, restful sleep getting 8 hours of deep sleep per night is essential to aid the hormones to function optimally (Tripathi, 2018)

3.1.2 Addressing Nutrient Deficiencies

Primarily the existing nutrient deficiencies should be addressed to improve thyroid function. Both nutrients and minerals are involved in the enzymatic process in the body. For example, iron is involved in the cellular thyroid function. Unfortunately, more than 50% of thyroid patients are spotted with B12 vitamin deficiency (Humberto et al., 1988). Low B12 results in low energy and reduced thyroid function (Saravanan et al., 2002).

3.1.3 Focusing on the Conversion of T4 and T3

Increasing the T4 to T3 conversion rate is another way that improves thyroid functioning. Increasing the T4 to T3 conversion reverses T3 levels, as results current dose of the thyroid hormone are increased. Increasing the conversion of T4 to T3 is accomplished easily with the intake of the supplement that improves the T4 to T3 conversions, such as zinc, guggulu extract, rosemary, and selenium. In addition, intake of probiotics that are high in quality reduces inflammation in the gut and increases absorption of nutrients (Koulouri et al., 2013).

3.1.4 Increase in Anti TPO Levels

Antibodies such as anti TPO indicate that the thyroid function is sub optimal and results in an inflammatory state. It alarms the emergency to undergo immediate treatment. The presence of anti TPO in the circulation, on the other hand, suggested an autoimmune disease affecting the thyroid gland. The existence of anti TPO antibodies suggested a weakened immune system, and these antibodies collectively see the thyroid gland as enemy tissue, attempting to kill it. This condition is called auto (yourself) and immune (immune system) (Fröhlich & Wahl, 2017). Low vitamin D increases intestinal permeability, gut

inflammation, poor diet, and high stress. Treatment possibilities of high anti TPO levels patients supplements, medications, hormones, diet, avoid endocrine disruptors (Fröhlich & Wahl, 2017).

3.2 TOP REASONS FOR HASHIMOTO'S FLARE-UPS

Extreme physiological or emotional stress for example, fear of death of loved ones, divorce, and other problems in school, office, and work, physical trauma or repetitive physical trauma, chronic nutrient deficiencies, viral infections such as Epstein-Barr virus (EBV) and Cytomegalovirus (CMV), the hormonal imbalance that affects the immune system functions, exposure to endocrine disrupting chemicals, exposure to heavy metals, nutritionally depleted diet high in industrial seed oil and inflammatory fats, increased intestinal permeability due to overgrowth syndrome and intestinal obesity are top reasons for Hashimoto's flare-ups. The reasons for autoimmunity and inflammation in the thyroid gland control multiple body functions, including metabolism—low metabolism results in Weight gain, low body temperature, and fatigue (Mullur et al., 2014). The thyroid influences the hormones, and low thyroid function leads to hormonal imbalance, such as low progesterone (Datta et al., 1998). Untreated autoimmune condition permanently damages the thyroid gland. The autoimmune thyroiditis symptoms do not match the common hypothyroidism. Anti TPO presence in the bloodstream indicated autoimmunity and inflammation, and this condition is entirely different from regular hypothyroidism. The treatment for this condition is entirely different. While treating the anti TPO antibodies, it is imperative to reduce inflammation and autoimmunity—untreated inflammation results in permanent damage to the thyroid gland. The presence of antibodies indicates several other problems in the body that need to be addressed immediately, including nutrient deficiencies (Homsy et al., 1986), gastrointestinal issues and hormonal imbalance (Cutolo et al., 2006). Active inflammation increases the risk of developing other autoimmune diseases. Treating these conditions improve thyroid function and reduce the symptoms without taking thyroid hormone. It is imperative to differentiate hypothyroidism and autoimmune thyroiditis because both conditions' treatment methods are entirely different. The presence of autoimmune thyroiditis indicates that organs in the body system are involved, and it is imperative to treat immediately for the best results. Medications such as levothyroxine or synthyroid should be suggested, and the patient's TSH level should be monitored to bring it to the average level. Thyroid hormone management is not simple, and several studies have shown that treatment based on TSH leads to low T3 levels (Peterson et al., 2016) that

reduces Quality of Life (Kalra & Khandelwal, 2011) and Weight gain because of low metabolism (Samuels et al., 2016).

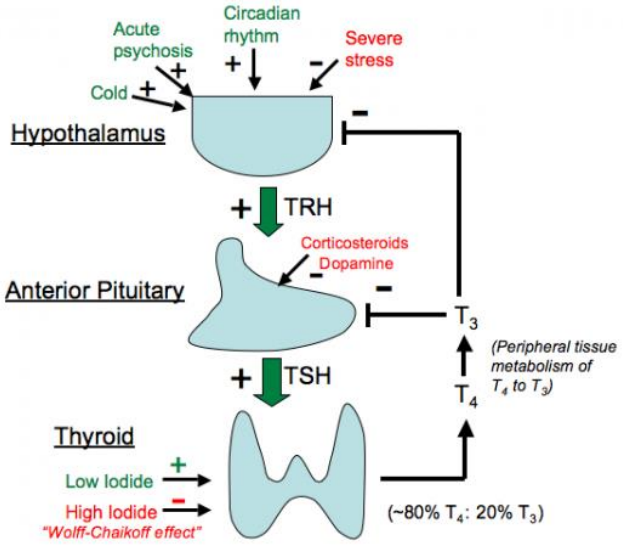

Figure 6: The thyroid gland & its regulation by the hypothalamic

Source: [Click Here]

Free T3 is a single most crucial thyroid hormone responsible for attaching to the surface and nucleus of the cells to perform the genetic changes to those cells. Though this is a simple process, it makes the thyroid increase the energy, manage Weight and regulate the cycle. An insufficient amount of Free T4 results in symptoms of hypothyroidism. Because Free T3 is a game maker for the thyroid (Marsili et al., 2011). Even a small change in the blood levels in hormone and serum levels leads to drastic function changes that result in symptoms—diagnosing hypothyroidism because the body's remarkable capacity to reserve serum thyroid level under condition is extreme stress. Combining these two factors makes it easy to conclude why to depend on one test TSH is a step to misdiagnose while treating hypothyroid patients. Here are some examples of lab tests that should be considered normal, but they are suboptimal in reality. The patient presented with Weight gain, slow metabolism rate, hair loss, and fatigue with hypothyroid symptoms. Gluten presence in the diet shows an inflammatory state (Soares et al., 2013). Inflammation, obesity, and insulin resistance are all reduced by a gluten-free diet, which is connected to the expression of Peroxisome proliferator-activated receptors (PPAR alpha and PPAR gamma) (Soares et al., 2013). This condition weakens the immune system and further reduces thyroid function (Mancini et al., 2016). Inflammation in the gut reduces T4 to T3 conversion, reduces nutrient absorption, and predisposes to the condition that triggers increased intestinal penetrability (AKA leaky gut). If any of the symptoms are spotted, it may be due to the development of autoimmune

thyroiditis and high levels of antibodies. Irritable bowel syndrome, constipation, inflammatory bowel disease, low stomach acid, gas or bloating, intestinal dysbiosis, acid reflux, diarrhea, and functional abdominal pain. Increasing vitamin D is one of the ways to increase thyroid functioning, and it also reduces the autoimmunity and inflammation in autoimmune thyroiditis. According to these studies, it is shown that lower vitamin D worsens Hashimoto's symptoms and even worsens thyroid function. As a result, there is a rise in antibodies (Aranow, 2011). One of the most important factors in the development of autoimmune illnesses is a high level of stress. It is shown that about 80% of patients suffer due to high levels of emotional stress before setting to the autoimmune disease (Yadid et al., 2000), especially Hashimoto's. In most cases, Hashimoto's and autoimmune thyroiditis trigger stressful situations. The stress can be emotional, traumatic, and physical, the stress triggering events include the death of a loved one, divorce, a medical illness in loved and divorce. Stress also influences other factors that further influence the body to develop the autoimmune disease; therefore, eliminating stress levels is very important to lower the antibody levels. High thyroid peroxidase antibodies have corresponding low testosterone levels because of thyroid hormone and its testosterone levels effects. Gluten impacts immunity and dairy products are removed because most people cross react to both gluten and dairy. Carbohydrate intake is mandatory, and several steps should be taken to reduce the chemical impact. Drink filtered water in the glass containers and avoid plastic containers (Vojdani, 2014). Touching receipts and usage of plastic toys should be completely reduced, organic foods, grass fed meats should be consumed, cosmetics made of chemicals should be completely avoided. In some cases, fast or active detoxification is recommended because these chemicals deposit in the body fat. The presence of anti TPO antibodies is completely unsafe, and when it is combined with hypothyroid symptoms, it results in autoimmune thyroiditis. When the subject is experiencing hypothyroid symptoms, it must undergo a complete thyroid lab panel check that includes antithyroid antibodies. The basic understanding of diagnosing and managing the condition is fundamental because various thyroid abnormalities are experienced. The autoimmune thyroiditis treatment is completely different because it is a combination of both inflammatory and autoimmune conditions. The subject needs to focus on the combination supplements, stress reduction, diet, and hormone replacement to control or reduce inflammation. Early diagnosis of this condition preserves the thyroid function for a long time. Though hypothyroidism results from low thyroid functioning, there may be lots of reasons and different conditions. Sometimes thyroid function reduces because of damage in the thyroid itself, or it loses the capacity of producing

enough thyroid hormones; this condition happens in Hashimoto's (Sheehan, 2016). Low thyroid function can also be caused by the pituitary gland not sending the correct signals to the thyroid gland, which can occur when the hypothalamus or pituitary are dysfunctional. If the hormones are not activated in the peripheral tissues or the cells are impervious to the hormone, it results in low thyroid function. All these above conditions results reduce the level of circulating thyroid hormone and results in hypothyroid symptoms. All these conditions are completely different in one way, whether or not treated naturally and cured. The thyroid gland does not produce the hormone independently because other organs should be stimulated before producing hormones (Shahid et al., 2021). The organs responsible for stimulating the thyroid gland are the pituitary gland and hypothalamus. Some possibilities are having a normal functioning thyroid gland capable of producing enough thyroid hormones. Still, the brain's right stimulation is not achieved, then it results in hypothyroidism, cure of this condition depends on the amount of damage that occurred to the pituitary or hypothalamus glands. The brain is a slow healer, some of the nerves are generated at a slow rate, and they heal quickly as the skin cells or intestinal tract. Subjects with hypothyroidism suffer brain related injuries on the pituitary gland or hypothalamus, then the condition is curable (Shahid et al., 2021) and is no hope of curing the condition after removing the thyroid gland or with radioactive iodine (RAI). Once the thyroid gland is completely removed or destroyed, nothing can be done. The body becomes 100% reliant on thyroid medication for survival. When the body fails to produce the thyroid hormone naturally, it must follow the prescribed medications such as levothyroxine, armour thyroid, and synthyroid. Obliterating the thyroid is the result of the out of control stage. Thyroid removal is suggested when there is a requirement for thyroid cancer treatment, extremely large goiters, hyperthyroidism and thyroid nodules. When the doctor recommends thyroid removal, it is best to opt for natural options before undergoing the procedure. If there is thyroid cancer, the natural options are not applicable, but there are effective natural options for thyroid nodules and hyperthyroidism (Mumtaz et al., 2009).

3.3 CURE FOR HYPOTHYROIDISM

It is essential to consider the primary treatment options to treat thyroid issues naturally.

3.3.1 Diet: It is not good to include processed, refined foods, gluten food products, dairy products , soy as it influences the hormones and negatively impacts the thyroid (Messina & Redmond, 2006). Organic foods, drink adequate water, in taking healthy fats, a balanced

ratio of proteins and carbohydrates should be included. Food intake should suit the metabolism.

3.3.2 Supplements: Thyroid improving nutrients such as iodine, selenium, zinc, tyrosine, and vitamin A supplements are helpful in improving the thyroid health. Usage of adrenal adaptogens helps in stress control or cortisol dysfunction as it improves the thyroid function, balances the cortisol, and helps the body tolerate stress. Supplements with adaptogens and adrenal glandular give better results. Other essential nutrients such as or multi vitamin, iron and vitamin D, B vitamin with activated B complex gives better results. Activated B vitamins help the body to utilize and absorb nutrients and increases energy immediately (Triggiani et al., 2009).

3.3.3 Exercise: The optimal amount of exercise is required for perfect body as well as to balance the hormonal level. It is imperative to avoid over exercising or to put too much strain on the adrenals. Exercising regularly, just from moderate to high intensity exercises, will help the body to tolerate. It is good to avoid over exercising and restrict calories as it will strain both the thyroid and cortisol systems. Healthy eating, sleep, and exercising are equally important (Booth et al., 2012).

3.4 THYROID STIMULATING HORMONES

Once the TSH level decreases, the thyroid will lose the ability to produce sufficient T4 and T3 levels (Pirahanchi et al., 2021). Hypothyroidism symptoms, as well as those linked with low testosterone, low progesterone, low estrogen, and low growth hormones, are experienced when the free thyroid hormone level lowers. Trauma to the head causes damage to the pituitary gland. It's caused by a lack of blood flow during pregnancy. Hypothyroidism symptoms, hair loss, weariness, lower body temperature, constipation, weight gain, and cold sensitivity are all indications of low TSH caused by pituitary gland malfunction. Reduced libido, vaginal dryness, irregular menstrual cycles, changes in breast size or volume, hunger changes, and hot flashes are some of the additional signs of low sex hormones. Another important reason for low TSH is lab anomaly because not all the lab tests are accurate all the time. It is estimated that three to five percent of laboratory examination are less accurate at the specified time (Ottomano, 2014). Because low TSH symptoms do not occur in this group, lab abnormalities are easier to spot. However, ordering TSH might lead to a misleading conclusion when there is an overabundance of thyroid hormone in the body. The

TSH is a broad technique to assess thyroid hormones, however there are other different tests that may be performed to assess the thyroid. TSH testing provides a specific set of data regarding the pituitary gland. It still doesn't tell you how much free thyroid hormone is circulating or how much hormone is being used in the body. All of these criteria are promptly assessed using additional testing. (Dunlap, 1990).

3.5 THE LIST OF TESTS THAT SHOULD BE ORDERED BEYOND TSH

Beyond TSH, T3, T4, Free T3, Free T4, Total T3, TPO antibodies, and Tg antibodies, the following assays should be investigated (Alevizaki et al., 2005; Sheehan, 2016), Cortisol (Walter et al., 2012), CRP (Nagasaki et al., 2007), SHBG (Alevizaki et al., 2005) . The aforementioned lab tests aid in determining the various reasons of low TSH and provide a better understanding of what is going on in the body. The following is a list of lab patterns and their corresponding conditions:

In euthyroid ill syndrome, low TSH, high reverse T3, low free T3, and low free T4 were evaluated as patterns (Vexiau et al., 1993).

In situations of endogenous hyperthyroidism, such as Graves' disease, low TSH, extremely high Free T3, and low Free T4 are investigated. (Kamal & Binod, 2021).

Low Free T3, high Free T4, and low TSH patterns are found with thyroid medication, however this does not always imply that the person is hyperthyroid (Koulouri et al., 2013).

With the lab abnormality, low TSH, standard Free T4, and normal Free T3 patterns were seen.

When pituitary dysfunction develops, low TSH, low Free T3, low Free T4, and low reverse T3 patterns are evaluated. (Gurnell, 2013).

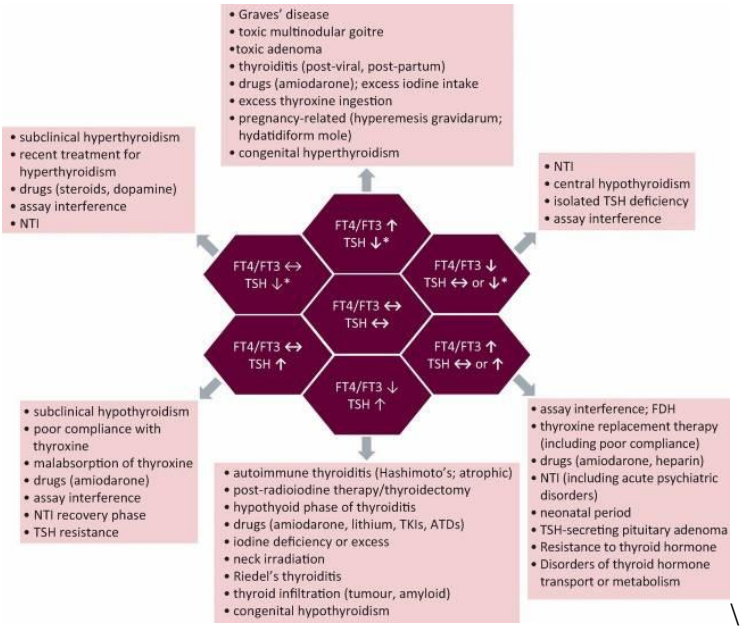

Figure 7: Diagrammatic interpretation of various thyroid tests and their causes.
Source: Click Here

TSH is a game maker in assessing thyroid function. Unless TSH is compared to other lab tests, it falls short and can be deceptive in a variety of ways. Low TSH is the result of five main conditions that are associated with other symptoms. All the symptoms are differentiated from one another in further evaluations. It is easy to identify these conditions with the help of different thyroid lab patterns and where the subjects fit in. If the TSH level is average, then the thyroid function is normal and reasonable. However, depending on TSH is not an accurate way to analyze thyroid functioning. This is the reason why the highest number of patients are misdiagnosed. Therefore, according to many recent studies, a great understanding of TSH suggests tighter references (Wartofsky & Dickey, 2005). Even evaluating the only TSH is not the best way to assess thyroid function (Samuels et al., 2016).

3.6 ANTI TPO ANTIBODY

One of the study shows 59.52% (n=50) of anti TPO antibodies are spotted in hypothyroidism subjects' blood circulation. Autoimmunity plays a critical role in the disease's aetiology. Autoimmune hypothyroidism subjects are spotted with an increased level of anti TPO antibody ($p<0.0001$) (Patel et al., 2016). According to the one of the study, hypothyroidism in India is not only due to intake of low iodine; the anti TPO antibodies are spotted in half of the hypothyroidism subjects. The same study was conducted on thyroid disease occurrence in eight Indian cities. The anti TPO antibodies presence is one of the primary

reasons for the disease (Unnikrishnan et al., 2013). The incidence of autoimmune hypothyroidism, in particular, has not been documented. At the onset of thyroid dysfunction, the anti TPO patients have a larger percentage of subclinical hypothyroidism, overt hypothyroidism, and hyperthyroidism than the control group. Before the control group, there was no significant change in the participants with anti TG. Therefore, anti Tg is considered a successful marker in Differentiated Thyroid Cancer (DTC) diagnosis (Asik et al., 2014). Antibodies development may be due to different thyroid antigens, and antithyroid peroxidase is a common antigen. The formation of anti Tg is due to TSH receptor blocking antibodies thyrotropin binding inhibiting immunoglobulins (TBII), TSH antibodies attack the thyroid tissues to restrict thyroid hormone production. Only 10-15% of the population has clinically obvious illness despite the presence of negative blood antibodies. Antibodies that are positive indicate a clinical condition (Leung & Leung, 2019; Yuan et al., 2019). The thyroid function is interrupted by antithyroid antibodies, which stimulate the thyroid profile testing (Tipu et al., 2018). The anti TPO antibodies are statistically higher in female subjects, and TSH and anti Tg antibodies are also high, but the differences are not statistically significant (Tipu et al., 2018). Thus, 68% of the women population are traced with hypothyroidism, and women over 60 years are prone to this disease. Only 3% of men are affected due to hypothyroidism. There is about a 4% annual risk of developing subclinical hypothyroidism associated with positive TPO antibodies (Díez & Iglesias, 2004). Subjects with autoimmune thyroiditis respond to an ongoing inflammation with specific antibodies in the serum, and inflammation plays an indirect role in the pathogenesis disorder. Antithyroid peroxidase and antithyroglobulin autoantibodies are linked to disease activities. Willms et al. reported that decreased echogenicity, multifocal pseudonodular hypoechoic, and heterogeneity infiltration indicate inflammation activity, and the sonographic findings were associated with significantly higher TPO Ab activity. The presence of TPO Ab has been associated to a higher incidence of overt hypothyroidism. (Okuroglu et al., 2017). The American Association of Clinical Endocrinologists (AACE) clinical practice guidelines for assessing and treating hypothyroidism and hyperthyroidism (2002 update) suggest that measuring the thyroid antibodies in subclinical hypothyroidism patients is mandatory to decide the treatment. The AACE recommends treating patients with TSH> 5mIU/L if there are thyroid antibodies or the patient has a goiter. According to two randomized controlled trials, the patient's TSH value was less than 10mIU/L, and there was no symptomatic development through thyroxine treatment (Meier, 2001 and Kong, 2002). Patients were advised to exercise caution since overt hypothyroidism is a possibility. A 20-

year follow-up research found a link between TSH levels and anti-TPO antibodies (Bijay 2008).

3.6.1 Anti TPO Antibody Levels of the Hypothyroid Patient

Autoimmunity appears to play a key role in the aetiology of the illness, according to earlier study. In contrast, when autoimmune hypothyroidism patients are compared to controls, they have higher anti TPO antibody levels (Patel et al., 2016). Insufficient iodine consumption is not entirely responsible for hypothyroidism disease in Indian patients, according to one study, Anti-TPO antibodies were found in more than half of the patients investigated in this study. The similar pattern was discovered in a recent study on the frequency of thyroid problems in India's eight major cities. The existence of anti-TPO antibodies is one of the main causes of the illness (Unnikrishnan et al., 2013). The prevalence of autoimmune hypothyroidism has not been recorded in the study. In comparison to the collective control group, both subclinical hypothyroidism and hyperthyroidism are present in a significant percentage of patients with anti TPO before the development of thyroid dysfunction. There was no significant difference between the participants who had the anti-TG antibody and the control group. This is one of the predicted outcomes, as anti-Tg antibodies are a well-known sign in the diagnosis of differentiated thyroid carcinoma (DTC). This is also a less specific thyroid disease sign than anti TPO antibodies (Leung & Leung, 2019). Antibodies to several thyroid antigens develop in the majority of individuals, with antithyroid peroxidase being one of the most prevalent antigens. Apart from anti-TPO antibodies, the majority of antigens come from anti-thyroglobulin and TSH receptor blocking antibodies, as well as immunoglobulins that prevent thyrotropin binding (TBII). All of these antibodies wreak havoc on thyroid tissues, reducing thyroid hormone synthesis. Only a small percentage of the population, perhaps 10 to 15%, is found to have a clinically obvious disease but no blood antibodies. TPO antibodies that are positive are a red flag for the clinical condition (Leung & Leung, 2019; Yup et al., 2015) known for affecting thyroid function and influence thyroid profile testing. Females have a larger number of anti-TPO antibodies than men. Anti-Tg antibodies and TSH are discovered at higher amounts in females; however, these differences are not statistically significant. Women have a 4% chance of acquiring clinical hypothyroidism each year, and subclinical hypothyroidism is connected to positive TPO antibodies. Spontaneous subclinical hypothyroidism in people over 55 years old: the study's findings are based on an examination of the natural course and risk variables associated with overt

thyroid failure (Díez & Iglesias, 2004). The typical autoantibodies in the serum of the subjects with autoimmune thyroiditis are indirectly responsible for the ongoing inflammatory reaction. However, they play a slight role in the pathogenesis of the disorder, antiperoxidase and the antithyroglobulin auto antibodies are frequently connected with the disease activity (Fröhlich & Wahl, 2017). According to the researcher, there is strong evidence of decreased echogenicity, heterogeneity, and multifocal pseudo nodular hypoechoic infiltration, indicating the inflammatory condition at a higher level. The sonographic findings are associated with the higher TPO antibody activity, and further, this is associated with the higher risk of hypothyroidism (Anil et al., 2011).

3.7 SEX HORMONE BINDING GLOBULIN

The Sex Hormone Binding Globulin blood test and the SHBG serum blood test provide important information about the hormone. Low SHBG levels indicate poor thyroid function as well as low estrogen levels. When SHBG levels are high, it can bind up testosterone, causing weight gain, sadness, and other sex hormone bind-up symptoms. There are two types of hormones that float about in the blood. 1. Protein-binding (Hobbs et al., 1992), If the hormones are linked to protein, they are unavailable to utilise (Azami et al., 2019e). It's like keeping food in the deep freezer for later use if these hormones are kept in the body. 2. Free and active hormones: if a hormone is not attached to a protein, it is free to enter cells and execute its function. The total T4 and T3 hormones have less therapeutic usefulness than the free hormones. As a result, free hormones are active hormones that aid in the measurement of all hormones. In comparison to the bound hormones in the blood, the number of free hormones is insignificant. The binding system is used by the body to control hormones and maintain equilibrium. As a result, the SHBG serves as a supportive hormone, which is advantageous given the presence of inactive hormones in the serum. This is why SHBG is known as a goldilocks protein, meaning it doesn't require too much or too little, only a small bit is necessary. Hormonal imbalance is one of the most prevalent causes of subclinical or overt hypothyroidism, which affects 20% of the population.

Thyroid hormone (Pasquali et al., 1997) and estrogen levels are two key hormone systems that regulate SHBG (Maggio et al., 2008). Both hormones increase the quantity of sex hormone binding globulin in the bloodstream. A rise in SHBG affects the quantity of free testosterone in the blood, and it's one of the primary reasons why women on oral contraceptives (OCP) gain weight and have mood swings. Excessive thyroid hormone

replacement treatment is another cause of elevated SHBG. It is commonly noticed in persons who use T3-containing drugs since it has a direct effect on hepatic SHBG production. It happens in persons who use T4 medications such as levothyroxine, just as natural desiccated thyroid hormone, liothyronine, or cytomel. Because women have high estrogen and hypothyroidism, examining SHBG during the menstrual month is difficult. While SHBG levels and thyroid hormone replacement are high, estrogen levels and the present thyroid dosage must be considered. Low SHBG levels are linked to an increased risk of type 2 diabetes (Yup et al., 2015). This is one of the most important reasons to increase your SHBG level if it is low. Estrogen hormone, too much or too little can lead to serious problems. Because estrogen has a big impact on thyroid hormones, it's important to get the right amount. Estrogen is a very sensitive hormone that has direct and indirect effects on thyroid function. Even a small change in estrogen and thyroid gland interaction can have a big influence (Santin & Furlanetto, 2011). Too much estrogen hinders the thyroid gland's health as estrogens excessive blood levels increase the thyroid binding globulin production through the liver. The T3 and T4 amount is decreased due to protein that binds the thyroid hormones, and the thyroid gland production is cranked. An excess amount of estrogen enlarges the thyroid, and little estrogen amount results in inadequate thyroid tissue. The thyroid hormone in the blood stream is controlled or normalized by estrogen through protein binds. Thyroglobulin is also stimulated by estrogen. Goiter is caused by an overabundance of estrogen, whereas a lack of estrogen produces a decrease in thyroglobulin, which reduces thyroid function. Thyroid hormones and progesterone have a reciprocal relationship: the ovaries require a sufficient amount of thyroid hormones to generate progesterone, and progesterone aids the thyroid. The majority of studies have indicated that progesterone raises thyroid hormone levels in the blood (Marqusee et al., 2000). Estrogen serves a variety of functions in the body. Estrogen helps to grow and maintain the reproductive system as well as feminine characteristics like pubic hair and breasts. It's critical to have the correct quantity of estrogen in your body. Too much or too little estrogen can create serious difficulties, and the quantity of estrogen in the body has a big impact on the thyroid. Estrogen stimulates thyroid tissue growth, whereas too little estrogen results in insufficient thyroid tissue production. On the other hand, too much estrogen enlarges the thyroid gland. Estrogen stimulates the thyroid glands. As result, thyroid hormone precursor and thyroglobulin are produced. Fewer estrogen results in insufficient thyroid hormone precursors, while too many results in enlarged thyroid glands. Enlarged thyroid glands are the result of the high thyroglobulin. Estrogen raises the protein that transmits the thyroid

hormones to the blood. Too much protein and insufficient thyroid hormones will be accessible and efficiently transmitted to the blood cells, resulting in hypothyroidism (Gillies & McArthur, 2010). There is a mutual relationship between progesterone and thyroid. Therefore, it is imperative to have adequate thyroid hormone counts for the female ovaries to produce progesterone. According to the research, progesterone helps increase the thyroid hormone levels by decreasing the protein amount which transmits the thyroid in the blood. Therefore, if the thyroid hormones are higher, they are free and quickly get into the blood cells (Sathi et al., 2013).

3.7.1 SHBG and Hypothyroidism

According to another study the higher SHBG levels are related with hypothyroidism. According to this study, there is a noteworthy reduction in SHBG after normalizing the thyroid hormones in the subjects (Selva & Hammond, 2009). Hepatocyte nuclear factor 4 alpha, or HNF4A, monitors SHBG function, which begins in the liver. Thyroid hormones are thought to raise SHBG levels through increasing HNF4A gene expression, which in turn upregulates SHBG levels indirectly. High SHBG levels have negative consequences, and the primary cause of high SHBG is a reduction in the circulation of free and unbound sex hormones in the bloodstream. According to the study, a drop in estimated free testosterone is the primary cause of excessive SHBG levels in men with normal testosterone levels (Antonio et al., 2016). Therefore, it is imperative to know a significant connection between hypogonadism symptoms and high SHBG levels in males. So, to know about the imbalance, it is imperative to assess the SHBG levels and thyroid hormones. Boron supplements are suggested to reduce the higher SHBG levels (Naghii et al., 2011). It is also essential to evaluate the sex hormones, adrenal hormone level, thyroid and neurotransmitters because they all function to offer optimum health and throughout body balance. SHBG symptoms are linked to thyroid function and low estrogen levels, and women with low SHBG may suffer the negative consequences of high free testosterone levels. The symptoms of SHBG, on the other hand, vary from person to person. This is why it's important to look at SHBG with other hormones like progesterone or estrogen, testosterone, and thyroid hormone (Hammond, 2016). The ability to measure thyroid function is one of SHBG's most important clinical features. Testing SHBG at baseline is critical in the case of low SHBG and hypothyroidism to determine if the correct type and amount of thyroid hormone is being administered. T4 alone thyroid medication, such as levothyroxine or synthroid, may not enhance SHBG in certain circumstances (Humberto et al., 1988). This suggests that the body

is having trouble converting T4 to T3 in peripheral tissue. T3 raises SHBG levels more than T4 does. The SHBG level may also be used to determine the thyroid dose level, which should be lowered if it is too high. The SHBG binding protein collects data about the body's different hormones. When there is too much estrogen, the SHBG rises, and when the SHBG falls, hypothyroidism develops. It's critical to figure out what's causing the anomalies in order to treat SHBG. This will assist normalize SHBG levels in the blood and alleviate symptoms (Alberti et al., 2002).

3.8 CORTISOL

The stress hormone cortisol is generated by the adrenal glands (Yup et al., 2015). It affects the heart, blood vessels, bones, muscles, lungs, brain, and hormone-producing glands (Habib et al., 2017). Cortisol functions include stress controlling, infection fighting, blood sugar level adjusting, and regulating blood sugar level (Habib et al., 2017; Hakamata et al., 2017). Cortisol, an adrenal hormone, is released in reaction to stress, and cortisol also stimulates the flight response. Thyroid hormone synthesis is reduced as a result of cortisol's reduction in TSH levels. The T4 to T3 conversion is blocked by cortisol, which causes T4 conversion to reverse T3. The significant effects of adrenals on thyroid functions are carried out through blood sugar. Higher or lower cortisol results in hyperglycemia or hypoglycemia, or both blood sugar imbalance may result in hypothyroid symptoms in many different ways (Kalra et al., 2014). An underactive thyroid increases the cortisol levels in blood (Iranmanesh et al., 1990). According to the cross-sectional study, subclinical hypothyroidism subjects have an intensification C-Reactive Protein value. On the other hand, the subclinical hypothyroidism subjects are also connected with a high risk of atherosclerosis (Rodondi et al., 2006) and the risk of coronary heart disease. If their TSH is greater than 10 mIU/L or if they have a positive thyroid peroxidase antibodies, they may be at a higher risk. The recent studies conducted by Gerold Huber and the team on the spontaneous course of the subclinical hypothyroidism subjects showed that risk factors for developing overt hypothyroidism are in the baseline TSH>12mIU/L (Huber et al., 2002). The essential intention of the study is to focus on the susceptible C-Reactive Protein of the subclinical hypothyroidism in the hospital (Panchal & Gondaliya, 2019). According to the study, HsCRP was linked with patients with subclinical hypothyroidism suffering from metabolic syndrome. According to the previous studies, there is no evidence of conflicting reflection on the connotation between HsCRP and hypothyroidism and showed clear association hypothyroidism and HsCRP (Alpaslan et al., 2005; Christ-Crain et al., 2003;

Pearce et al., 2003). Few studies tried to link endocrine and immune function to *yoga* practice as some of the *haṭa yoga* postures enhances the immune system or treatment (Iyengar, 2005). There are more possibilities that drug usage may confound cortisol response. Intake of some of the antidepression medications increases the cortisol levels upon acute administration (Hinkelmann et al., 2012). Regular *yoga* practise lowers depression, raises serotonin levels, and lowers monoamine oxidase, an enzyme that breaks down neurotransmitters and cortisol. Stress aggravates the underlying autoimmunity, *yoga* reduces sympathetic arousal, and hypo-thalamus-pituitary-adrenal axis activity further reduces stress and anxiety. Gamma-aminobutyric acid (GABA) and Brain Derived Neurotrophic Hormone (BDNF) levels are also improved by *yoga,* and both of these hormones play a role in reducing depression (Cahn et al., 2017; Streeter et al., 2012). Depression is linked with hypercortisolemia and results in hyper functioning of the hypo-thalamic-pituitary-adrenal axis (Christensen & Kessing, 2001; Gillespie & Nemeroff, 2005) and the increased cortisol responses to stress, acute or chronic (Raison & Miller, 2003). Stress is due to negative life events that are known to precipitate depression (Raison & Miller, 2003). *Yoga* is an effective intervention to decrease stress and depressive illness (Janakiramaiah et al., 2000) and lowers cortisol levels (Vedamurthachar et al., 2006).

3.9 C-REACTIVE PROTEIN

Patients with subclinical hypothyroidism have higher C-Reactive Protein levels, according to a cross-sectional research. The condition of subclinical hypothyroidism is also linked to an increased risk of atherosclerosis. One of the most serious issues is that subclinical hypothyroidism might progress to overt hypothyroidism over natural history. If the TSH reaches 10mIU/L or if the thyroid peroxidase antibody is positive, the people may be at a higher risk. The risk factors for overt hypothyroidism may grow, according to a new study by Gerold Huber and colleagues on the spontaneous course of subclinical hypothyroidism individuals. TSH > 12mlU/L is the normal range (Huber et al., 2002) and decreased thyroid reserve and the presence of the thyroid peroxidase antibody (Panchal & Gondaliya, 2019). HsCRP is linked to subclinical hypothyroidism in metabolic syndrome patients, according to the study. There is a contradicting remark between HsCRP and hypothyroidism in previously published study (Alpaslan et al., 2005; Crain et al., 2003). The authors have described a clear association between hypothyroidism and the progressed HsCRP. By

opposing this study (Pearce et al., 2003) suggested that Hashimoto's thyroiditis subjects, postpartum thyroiditis, and temporary hypo thyroiditis resulted with similar HsCRP compared to euthyroid controls. This is why the HsCRP levels play only a partial role in diagnosing thyroid disorder (Pearce et al., 2003).

3.10 WEIGHT AND HYPOTHYROIDISM

Hypothyroidism and obesity are closely related, and the link between both is applicable in the situation of an extraordinary increase in the commonness of obesity throughout the world. Thyroid dysfunction patients' second most significant symptom is obesity. Thyroid stimulating hormone is next to obesity, according to a fresh viewpoint. According to current research, obesity and thyroid autoimmunity with the adipocyte hormone leptin are the major elements that link these conditions (Sanyal & Raychaudhuri, 2016). Hypothyroidism is associated with a reduction in metabolic rate and thermogenesis, as well as a higher BMI and obesity prevalence (Danforth et al., 1979). According to clinical evidence, even thyroid dysfunction in mild form in subclinical hypothyroidism results in substantial changes in body Weight, and there are risk factors of over Weight and obesity. A slight variation within the laboratory reference contributes to Weight gain (Yuan et al., 2019); however, all studies have not been established. It has been observed that there is a negative relationship between Free T4 and BMI, even when FT4 is in the normal range (Knudsen et al., 2005). According to the clinical perspective, minor thyroid failure and obesity are common in hypothyroidism, and they coexist.

3.11 HYPOTHYROID AND BMI

Clinical evidence demonstrates that even modest thyroid problems, such as subclinical hypothyroidism, are linked to significant weight fluctuations and are a risk factor for obesity (Danforth et al., 1979; Sanyal & Raychaudhuri, 2016). Despite the fact that no studies have proven it, evidence suggests that slight variations in thyroid function within the laboratory reference range contribute to the tendency to gain weight (Yuan et al., 2019). Even when FT4 is within normal ranges, there is an inverse association between Free T4 and BMI (Knudsen et al., 2005; Portmann & Giusti, 2007). Obesity and moderate thyroid dysfunction are prevalent disorders in clinical practice, and they commonly coexist. Obesity was also more prevalent (46 percent vs. 34 percent) in overt hypothyroidism than in subclinical hypothyroidism (Sanyal & Raychaudhuri, 2016). When thyroid cells are lost as a result of autoimmune hypothyroidism, the thyroid gland is gradually destroyed, resulting in thyroid

hormone insufficiency. Antithyroid peroxidase antibodies and antibodies, as well as, less typically, antithyroglobulin antibodies, abnormalities in the circulating T cell population, and goitre with lymphocytic infiltration, are all immunological characteristics of this thyroid condition (Eguchi, 2001; Kwanhoon & Lim, 2018; Stassi & Maria, 2002).

3.12 QUALITY OF LIFE AND HYPOTHYROIDISM

Although there is no relationship between thyroid hormone blood levels and Quality of Life, in untreated hypothyroid people with hypothyroidism, a lower Quality of Life leads to tiredness. Patients with low quality of life perceive an improvement in their quality of life after therapy (Cooper & Biondi, 2012; Naghii et al., 2011). A study by Oken (Oken, Zajdel, Kishiyama, Flegal, Haas, et al., 2006) comparing the yoga group's Quality of Life and physical measurements to the exercise and waiting control groups, there was a significant improvement in illness specific impairments in Quality of Life throughout long-term follow-up of patients with various pituitary adenomas. According to Mischalsen's research, sadness, emotional state wellness, joint pain, anxiety, physiological complaints, and psychological Quality of Life all decreased significantly. All these aspects are well among the distressed woman who was following *yoga* intervention for three months (Burns et al., 2001; Klaauw et al., 2008). Antithyroid peroxidase antibody titers can aid in decision-making. It is simple to anticipate the risks of progressing to overt hypothyroidism if the results are good. It's critical to think about LT4 treatment, especially for young individuals, pregnant women, and pregnant women with positive antibodies. The usual treatment for hypothyroidism is levothyroxine. Thyroid function is disrupted and thyroid conversion is blocked by either mental or physical stress. Medication does not always have a favourable outcome. Some drugs can interfere with thyroid function. Seizure drugs, blood pressure meds, diabetic medications, iodine steroids, heart medications, and PTU/Methimazole, for example, can all interfere with thyroid function. Stopping this drug is tough for anybody, but it is important to seek out the best option. The influence of *yoga* on female patients suffering from hypothyroidism. The WHO Quality of Life Scale evaluated 20 females' Quality of Life with hypothyroidism disorder and noted noteworthy development in overall health and Quality of Life, in post *yoga* intervention (Kumar et al., 2018; Singh et al., 2011). Most people are completely reliant on medicines for the rest of their lives; however, prior studies have shown that individuals with hypothyroidism who followed an integrated strategy with *yoga* treatment for six months had favourable improvements. Hypothyroidism

patients' TSH levels were lowered from 36.6 to 5.82, and the best part is that no medical therapy was required (Kumar et al., 2018).

3.13 *YOGA*

Definitions of *yoga*

सम दोश समाग्नि समधतु मलक्रियह् प्रसन्नत्म इन्द्रिय

मनह् स्वस्थ इत्यभिधियते (षुश्रुत षुत्र || १५| ३)

sama dośa samagni samadhatu mala kriyāha prasannatma
indriya manah svastha ityabhidhiyate (Ṣusruta Ṣutra 15/3)

Perfect health is achieved only when there is a balanced state of bio chemicals, body constituents, metabolism, proper elimination of waste, and the mind, senses, and soul are blissful.

योगश्चित्तवृत्तिनिरोधः ॥ प यो सु १.२ ॥

yogaścittavṛttinirodhaḥ || PYS 1.2||

Sage *Patañjali* defines *yoga* as a state achieved through cessation of all mental modifications.

समत्वं योग उच्यते ॥ भ गि २ / ४८

samatvaṁ yoga ucyate || (*Bhagavatgītā*: 2 / 48)

In *Bhagavatgītā, yoga* is defined as a state of equanimity as well as dexterity in action. Ancient Indian science *yoga* is the first to distinguish the strong relationship between mind and body. This mind and body relationship restores and promotes health with the help of various effective postures, breathing exercises, and meditation. *Yoga* means unite in *Saṁskṛita* which explains about the unity of mind, and body artistically. The ancient sage *Patañjali* described *yoga* as '*citta vṛitti nirodha'*. *Citta* denotes mind which is always busy in constant thinking, which revolves around uncontrolled emotions which leads to *vritti* and that needs be controlled known as *nirodha'* through practice or *abhyāsa* and *vairāgya* (Bhugra & Kalra, 2010). *Yoga* in the form of physical exercises helps in gaining body balancing, strength and flexibility physically and helps in muscle relaxation. *Yoga* in the

form of spiritual exercise trains the mind to maintain peace and happiness. *Yoga* practice includes combination of *āsana*, *prāṇayāma, pratyahāra, dhāraṇa*, *dhyāna* and *samādhi*. All the *yoga āsanās* helps in building physical strength and mental exercises build concentration (Kjellgren et al., 2007). The main intention of *yoga* practice is to attain the state of control through physically, breathing, mentally or emotionally (Saraswati, 1976) or by silencing the mind and to create the internal awareness that helps the mind to slowdown the conscious and offers physiological rest. Sage *Gaudāpada*, in *Maṇdukya Kārika* proposed a concept that deepens the rest level with the help of alternative stimulation and which are followed by relaxation techniques (Atreya, 1993; Mashyal et al., 2014; Nagendra & Raghuram, 1977).

3.14 *PAÑCA KOŚA* CONCEPT, IAYT AND HEALTH

Yoga is an ancient Indian discipline that combines mental, spiritual, and physical activities. Deep relaxation methods, including meditation and therapeutic *āsana*, are essential for a proper *yoga* plan. *Yoga* practices strengthen the body and create awareness in body, mind, breath, engaging *prāṇayāma*, and *mudrās* (Sengupta, 2012). *Yoga* exercise improves body function, including cardiovascular, metabolic control mechanisms, and respiratory. Practicing *yoga* restores balance in both body and mind and helps to regain focus and self confidence. *Yoga* is an excellent and complete package for beginners as well as regular practitioners and it benefits all age groups (Singh et al., 2011). Better health and an extreme sense of happiness are wholly achieved through *yoga*, and it is an excellent complementary therapy when combined with medical therapy in treating thyroid disorders. Through *yoga* it is easy to achieve proper awareness of deep physiological therapies. One can quickly relieve from diseases, and even it is easy to reverse the diseases. It has been proved that *yoga* is valuable in improving overall health and thyroid health and related symptoms in thyroid patients (Singh et al., 2011). The Integrated Approach of *Yoga* Therapy (IAYT) is a complete approach to establish health and wellbeing. *Yoga's* concept of human existence is comprised of five sheaths (layers), they are the gross body (*annamaya kośa*), the energy body (*prāṇamaya kośa*), the emotional body

(*manomaya kośa*), the intellectual body (*vijñānamaya kośa*) and the bliss body (*ānandamaya kośa*). An integrated approach includes various *yogic* techniques that address each *kośa* to establish overall health (physical, psychological, social, and spiritual). The IAYT intervention (independent variable) was the customized IAYT based *yoga* program for this research, which addresses the issues at the *pañca kośa* level and includes practices such as *āsana, prāṇayāma*, meditation, relaxation, prayers/chanting, and knowledge point (Krishnamoorthy, 2007; Raghuram & Hongasndra, 2004; Raghuram & Hongasandra, 2008; Rahuhram & Hongasandra, 1985).

3.15 SUMMARY TABLE OF SCIENTIFIC LITERATURE

Author and Year	**Title of study**	**Design and Method**	**Results and Conclusions**	**Limitations**
Nilakanthan S, Metri K, et al. (2016).	Effect of six months intense *yoga* practice on lipid profile, thyroxine medication and serum TSH level in women suffering from hypothyroidism: A pilot study.	22 female subjects, 30-40 years 4±1.12year history of hypothyroidism. Six month *yoga* intervention one hour a day for four days a week	*Yoga* improved cholesterol levels, thyroxine and serum TSH, in subjects	To confirm the current conclusion, more randomised controlled research are needed.
Sridip Chatterjee and Samiran Mondal (2017).	Effect of a combined *yoga* program on blood levels of thyroid hormones: A quasiexperimental study.	Subject: 23 Experimental: male – fifteen, female – eight Waitlisted control group: male – fifteen, female – seven. *Yoga* practice six days per week, twelve	According to the baseline data comparison, twelve weeks of *yogic* training results in a substantial rise in serum TSH level for men and a drop in T3	To some degree, the study's weaknesses might be attributed to convenient sampling (rather than a randomised control trial) and the absence of a residential camp.

		weeks, for the control group, usual routine activity.	and T4 for both male and female subjects, no changes in control group after twelve weeks.	
Gaurav Swami, Savita Singh, et al. (2009).	Effect of *yoga* on pulmonary function tests of hypothyroid patients.	Subject: Twenty female study group and twenty female control group, healthy volunteers.	*Yoga* has a positive impact on subjects with pulmonary function tests. All subjects has been taking thyroxin for the previous two to three years, so *yoga* is responsible for the majority of the effects shown in the last six months.	No randomisation, significantly less sample, and no guided practice; after 21 days of learning, they have to practice independently.
Singh P, Singh B, et al. (2011)	The impact of *yoga* upon female patients suffering from hypothyroidism.	WHO QOL scale (22) one month daily one-hour *yoga,* twenty female, hypothyroid subjects	Patients' QOL scores were significantly greater after the *yoga* training than before ($p < 0.01$). Subjects were reported	

			improvement in overall QOL.	
Krishna Sharma and Mahaabala (2016).	The effect of *yogic* practices on thyroid functions.	Subjects: ten experimental and ten control, before and after the *yogic* practice. Three months. Parameters: triiodothyronine, tetraiodothyronine, and TSH.	*yogic* practices brought hormone balance to the body. *Yogic* practices could be used to improve the health of any individual.	Long term of such study with a good number of subjects could bring better significant results and helps in the improvement of general health economically.
Rawal SB, et.al. (1994).	Effect of *yogic* exercises on thyroid function in subject's resident at sea level upon exposure to high altitude.	Subjects: Ten male, five *yoga* groups, five control physical training. The thyroidal growth and radioactive iodine release have been noted.	The trans thyroidal availability of radioiodine was significantly reduced after one month of *yoga* activities. Thyroid activity has been raised as a result of traditional physical training.	–
AnandaBalayogiBhavanani, et.al. (2011)	Effect of *yoga* on subclinical hypothyroidism: A case report.	Subject: one female, 36 years, one year *yoga* therapy.	*Yoga* might be a helpful supplement to thyroid treatment. After *yoga* intervention TSH was	–

			changed to 9.39 IU/ml to 2.66 mIU/L, T4 changed to 12.57 pmol/L to 8.98 pmol/L.	
Minal S Pajai, et al. (2014).	Role of *yoga* in the prevention of hypothyroidism.	Review article One hour *yoga*	*Yoga* can assist hypothyroid individuals in coping with their disease-related symptoms.	*Yoga* is beneficial in supporting hypothyroid individuals in managing their disease-related signs and symptoms.
Ambika Gopalakrishnan et.al. (2011)	Thyroid disorders in India: An epidemiological perspective.	Review article	According to the review, exciting work in development to determine the normal reference range of thyroid hormones in India was covered especially in pregnancy and children.	_
Asima Kumar et.al. (2018)	*Yogic* management for hypothyroidism: a case study.	46 year, female Case study IAYT	Before *Yoga* TSH 37.53 mciu/ml interim reading TSH 15.67 mciu/ml	Hypothyroidism patients benefit from an Integrated Approach to *Yoga* Treatment

			After *yoga* TSH 5.82 mciu/ml.	(IAYT). This method might be used to treat hypothyroidism in the future.
S. Rani et.al. (2021)	Effect of *yoga* on depression in hypothyroidism: A pilot study	38 women in a single-arm pre–post design 3-IY (*āsanas*, *Prāṇayāma* and relaxation methods) for 60 minutes each day (5 days a week), At baseline and after 12 weeks, depression, sTSH, and lipid profile indices, BMI, tiredness, anxiety, and stress were measured. Data was analysed using R Studio software.	After three months, there was a substantial (P 0.05) reduction in depression (58 percent), sTSH (37 percent), BMI (6%), tiredness (64 percent), anxiety (57 percent), lipid profile indices (HLD rose significantly), and stress (55 percent) when compared to baseline values.	small sample size, lack of a control group, and lack of objective parameters of depression
Dr. Javed Akhtar 2019	Role of *yoga* in improving Quality of Life of hypothyroidism patients	Sixty audult subjects, WHO QOL BREF,	At the conclusion of six months, patients in the *yoga* group had a substantial improvement of 17.79 percent in	Shorter time

			the physical element of Quality of Life when compared to patients in the control group. In addition, there was a significant improvement of 18.38 percent in the psychological element of patients' Quality of Life.	
Rukamani Nair, et.al. (2021)	Efficacy of *yoga* in controlling subclinical-hypothyroidism: a randomized controlled trial	This is a single-blinded two-arm, parallel-group RCT age 21-59 years, TSH level 4.2 mIU/l >TSH level <10 mIU/l T3 and T4 with in normal range 100 participants are Intermediate follow up at 3	Subclinical hypothyroidism is more likely to progress to hypothyroidism (overt).The results of this study will show if *yoga* is useful in avoiding advancement from subclinical hypothyroid to over hypothyroid stage, as well as in reversing subclinical hypothyroid to euthyroid stage.	

		month and final follow-up will be at six month post intervention.		

3.16 NEED FOR THE CURRENT STUDY ON HYPOTHYROIDISM

Studies on *yoga* as a therapeutical application and as a complementary therapy for thyroid dysfunction population concluded benefits from *yoga*, but the evidence is low in methodological quality and quantity. Inconsistency and shorter duration, smaller sample size, lack of randomisation, follow up data, non specificity concerning the *yoga* content, significant methodological and designing weakness, need for more systematic data collection are some of the other conclusions of the studies. Research studies further recommend the following for future studies on hypothyroid dysfunction. More direct research with particular physical and emotional outcomes, larger sample numbers to improve statistical power, use of suitable control groups, and randomization. To conclude, based on the literary survey, it is understood that the study of the effect of *yoga* based IAYT practices on hypothyroidism is essential and maintaining a healthy thyroid is one of the body's primary functions in a significant population. No such study or survey was conducted earlier. *Yoga* appears to have a good influence on personality development, cognitive capacities, and the management of psychosomatic disorders in thyroid patients, according to early findings. However, no research on yoga as a thyroid control programme have been conducted on a major population in the Indian population. The root cause of thyroid dysfunction is a mainly irregular lifestyle. To overcome the irregular lifestyles IAYT and *astanga yogic* principles *yama, niyama*, *āsanās*, *prāṇayāma*, and meditation are very useful*, astanga yoga* is one means to overcome irregular lifestyle. Due to the severe side effects and drawbacks of antithyroid medication, this study is essential.

The current chapter covered critical view of scientific literature on hypothyroidism and its related tests ordered to conclude on the patient's hypothyroidism status. The upcoming study describes aim and objectives of design 1 and design 2 of the study.