# The correlation of thyroid function with obesity

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## Abstract

The thyroid is an endocrine gland. It controls the body's iodine balance as well as the creation and release of thyroid hormones from its location in the inferior, anterior neck. The thyroid produces both triiodothyronine, which is mostly active, and thyroxine (T4), which is primarily dormant (T3). Thyroid hormone that has undergone peripheral conversion is either activated thyroid hormone or another inactive thyroid hormone. Body composition and thyroid hormones appear to be closely related. Thyroid hormones regulate basal metabolism, thermogenesis and play an important role in lipid and glucose metabolism, food intake and fat oxidation. Thyroid dysfunction is associated with changes in body weight and composition, body temperature and total and resting energy expenditure (REE) independent of physical activity. The hypothalamic-pituitary-thyroid axis is more likely to be activated in obese patients with healthy thyroid function and greater blood levels of thyroid hormones. On the other hand, slight variations in thyroid function are linked to weight changes of up to 5 kg. An online survey was conducted in Al_Nahrain university college of pharmacy. The aim of this survey is to find the correlation between thyroid function and obesity in the Iraqi population who are diagnosed and treated of hypothyroidism. Current study outcomes is to find the correlation between thyroid function and obesity in the Iraqi population who are diagnosed and treated of hypothyroidism. The correlation between BMI and TSH showed elevattion 4.5-10 mIU/mL, 36 had severely Elevated > 10 mIU/mL while 9 are normal 0.5-4.5 mIU/mL.

**Aim:** The aim of this survey is to find the correlation between thyroid function and obesity in the Iraqi population who are diagnosed and treated of hypothyroidism.

**Keywords:** Thyroid , obesity

## Introduction

The thyroid gland is considered the largest endocrinal gland in the human body, located in the anterior aspect of the neck. It synthesizes and releases thyroid hormones that considerably influence the basal metabolic rate (BMR) and protein synthesis [1]. Furthermore, these hormones are also critical for children and adolescents' neurocognitive development and maintaining normal physiological functioning in adults [2]. Thyroid disorders are one of the most common medical conditions worldwide [3]. Iodine deficiency is a major cause of thyroid disorders. It is estimated that about one-third of the world population lives in an iodine-deficient area, and over 190 million suffer from iodine deficiency disorders [4]. If left untreated, thyroid disorders may lead to complications that may impact the patients' quality of life [5]. Thyroid disorders are conditions that result from either over-secretion (hyperthyroidism)/under-secretion (hypothyroidism) of thyroid hormones as well as thyroid gland enlargement (goiter). Thyroid disorders can be primary (directly related to the gland itself) or secondary (thyroid dysfunction due to other factors). These disorders were reported in more than 110 countries, with 1.6 billion people at risk. The clinical symptoms of a thyroid disorder mainly depend on the type of the disorder and may affect different systems of the body. Furthermore, since most of the symptoms are not specific, thyroid disorders can be easily missed or confused with other medical conditions [6]. Hypothyroidism refers to the common pathological condition of thyroid hormone deficiency. If untreated, it can lead to serious adverse health effects and ultimately death. Because of the large variation in

clinical presentation and general absence of symptom specificity, the definition of hypothyroidism is predominantly biochemical [7].
Hypothyroidism can be classified as primary (due to thyroid hormone deficiency), secondary (due to TSH deficiency), tertiary (due to thyrotropin releasing hormone deficiency), and peripheral (extra-thyroidal; panel). Central hypothyroidism (including both secondary and tertiary) and peripheral hypothyroidism are rare and account for less than 1% of cases [8]. Whether the existing reference ranges of TSH and free thyroxine should be used to define thyroid dysfunction is a matter of debate. This issue is of clinical importance because the reference ranges are generally used as a threshold for treatment [9]. The heritability of TSH and free thyroxine concentrations in serum is estimated to be 65% and 23–65%, respectively [10]. Hypothyroidism is more common in patients with autoimmune diseases, such as type 1 diabetes, autoimmune gastric atrophy, and coeliac disease, and can occur as part of multiple autoimmune endocrinopathies. Individuals with Downs' syndrome or Turners' syndrome have an increased risk of hypothyroidism. By contrast, tobacco smoking and moderate alcohol intake are associated with a reduced risk of hypothyroidism [11]. The clinical manifestations of hypothyroidism range from life threatening—in the case of myxedema coma—to no signs or symptoms [12]. Myxedema coma leads to an altered mental status, hypothermia, progressive lethargy, and bradycardia and can eventually result in multiple organ dysfunction syndrome and death. Therefore, early initiation of thyroid hormone therapy and other supportive measures is crucial [13]. The most common symptoms of hypothyroidism in adults are fatigue, lethargy, cold intolerance, weight gain, constipation, change in voice, and dry skin, but the clinical presentation can include a wide variety of symptoms that differ with age, sex, and time between onset and diagnosis [14].

The symptoms for the diagnosis of hypothyroidism are non-specific, especially in elderly patients who present with fewer and less classic signs and symptoms than younger individuals [15]. An increase in the severity of symptoms might predict hypothyroidism, since a change in seven or more symptoms in the past year increases the likelihood of hypothyroidism [16]. Treatment targets include normalization of TSH concentrations and resolution of physical and mental complaints, while avoiding under treatment or overtreatment. Levothyroxine monotherapy in solid formulation, taken on an empty stomach, is the treatment of choice. The presence of clinical features of hypothyroidism, with biochemical confirmation of overt hypothyroidism, is the indication for treatment initiation. No rationale exists for avoiding the prescription of generic preparations, but switches between levothyroxine products in patients who are stable are not recommended [17]. The optimal daily dose in overt hypothyroidism is 1·5—1·8 μg per kg of bodyweight. In patients with coronary artery disease, the starting dose is generally 12·5—25·0 μg per day and should be gradually increased on the basis of symptoms and TSH concentrations [18]. This regimen is often preferred in the elderly, especially in patients with many co-morbidities [19]. In younger patients without comorbidities, the full dose can usually be given from the start with adequate monitoring to avoid overtreatment. After the initiation of therapy, TSH measurement is repeated after 4–12

weeks and then every 6 months and, once stabilized, annually. Adjustments should be made according to laboratory findings, keeping in mind that in some patients (ie, those with low bodyweight or older patients) small changes in dose can have substantial effects on serum TSH concentrations. The clinical significance of low tri-iodothyronine concentrations in some patients despite reaching normal TSH concentrations is unknown. Routine measurement of triiodothyronine should not be used to assess treatment effectiveness [20]. Because of physiological changes during pregnancy, an increase in levothyroxine dose is required to maintain euthyroidism [21]. Therefore, women of childbearing age with levothyroxine-treated hypothyroidism should be informed to increase their dose by 30% once pregnant and directly contact their physician for further guidance [22]. Body composition and thyroid hormones appear to be closely related. Thyroid hormones regulate basal metabolism, thermogenesis and play an important role in lipid and glucose metabolism, food intake and fat oxidation. Thyroid dysfunction is associated with changes in body weight and composition, body temperature and total and resting energy expenditure (REE) independent of physical activity [23]. Hypothyroidism is associated with decreased thermogenesis, decreased metabolic rate, and has also been shown to correlate with a higher body mass index (BMI) and a higher prevalence of obesity. There is clinical evidence suggesting that even mild thyroid dysfunction in the form of subclinical hypothyroidism is linked to significant changes in body weight and represents a risk factor for overweight and obesity [24]. It has been further noted that small variations in serum TSH caused by minimal changes in L-T4 dosage during replacement therapy are associated with significantly altered REE in hypothyroid patients [25]. Evidence suggests that slight variations in thyroid function that are within laboratory reference ranges, also contribute to the tendency to gain weight; although this has not been confirmed by all studies. An inverse correlation between free T4 (fT4) and BMI, even when fT4 remains in the normal range has been reported; fat accumulation has been associated with lower fT4 and higher TSH levels among slightly overweight euthyroid individuals, thereby resulting in a positive correlation between TSH and the progressive increase in weight with time [26]. Altered thyroid function with normal feedback regulation may be the primary event that induces alterations in energy expenditure with subsequent increases in BMI and weight [27].

**Materials and Methods**

An online survey was conducted in Al_Nahrain university college of pharmacy. The aim of this survey is to find the correlation between thyroid function and obesity in the Iraqi population who are diagnosed and treated of hypothyroidism. A Google Forms questionnaire was sent in medical field groups on Facebook from 12th Dec 2022 to 10th Mar 2023. The questionnaire was also conducted in the field in the hospital, specifically Al-Kadhimiya Teaching Hospital. The survey included 100 participants (32 males and 68 females) aged from pediatric to geriatric years. We administered an online survey with several questions that allowed us to collect specific data, to assess two main clinical states thyroid hormone and obesity and how are they are correlated to each other. The questionnaire was constructed on the basis of what we intended to report.

**Questionnaire**

- ➢ Age
- ➢ Gender
- ➢ Euthyrox dose
- ➢ Weight
- ➢ Height
- ➢ BMI
- ➢ TSH
- ➢ T4
- ➢ T3
- ➢ Anti TPO
- ➢ Weight post treatment
- ➢ Average weight loss

**Results**

**1-Distribution of the sample according to gender:**

The study sample consisted of 100 patients. Distribution of the sample subjects by gender is shown in figure (1) and table (1), the percentage of males is 32% while the percentage of females is 68%.

**Table1**: distribution of the sample according to gender.

| Male | Female |
|---|---|
| 32% | 68% |


**Figure1**: distribution of the sample according to gender.

**2-Distribution of the sample according to age:**

90% of the patients were adults, 7% were geriatric and only 3% were pediatric figure (2) and table (2).

**Table2**: distribution of the sample according to age.

| Pediatric | Adult | Geriatric |
|---|---|---|
| 3% | 90% | 7% |


**Figure 2:** distribution of the sample according to age.

**3-Distribution of the sample according to BMI categories:**

Approximately half of participants (55%) are in obese state as shown in figure (3) and table (3). The other half is divided between overweight (32%) and (13%) normal patients.

**Table3**: distribution of the sample according to BMI categories.

| Obese | Overweight | Normal |
|---|---|---|
| 55% | 32% | 13% |

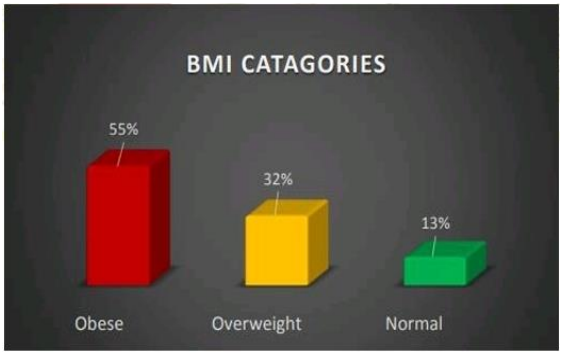



**Figure3**: distribution of the sample according to BMI categories.

**4-Distribution of the sample according to correlation between BMI and TSH**

Figure (4) and table (4) show 55 had mildly elevated 4.5-10 mIU/mL, 36 had severely Elevated > 10 mIU/mL while 9 are normal 0.5-4.5 mIU/mL.

**Table4**: distribution of the sample according to correlation between BMI and TSH

| TSH | Normal 0.5-4.5 mIU/mL | Mildly Elevated 4.5-10 mIU/mL | Severly Elevated > 10 mIU/mL |
|---|---|---|---|
| percentage | 9% | 55% | 36% |
| average BMI | 27.9 | 29.4 | 30.9 |

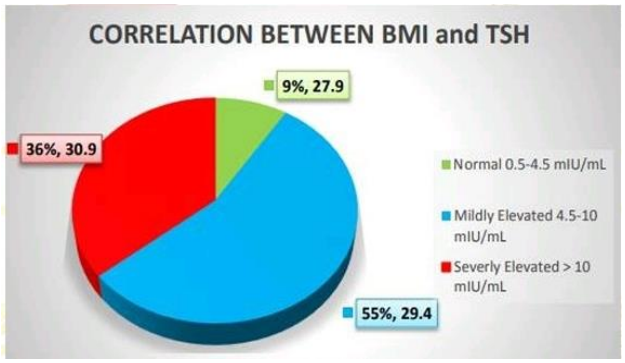



**Figure4:** distribution of the sample according to correlation between BMI and TSH

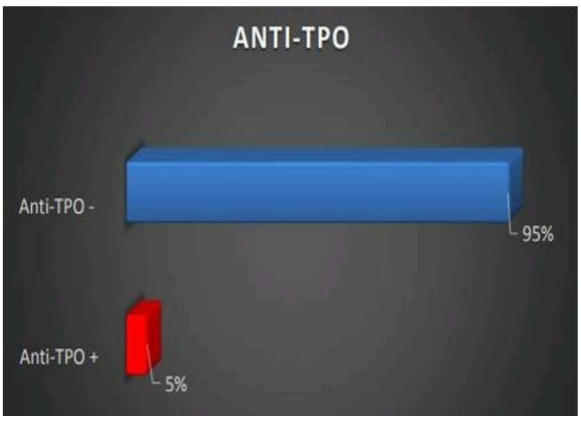
**5-Distribution of the sample according to ANTI-TPO :**

Majority of patients (95%) got Anti-TPO – and only 5% got Anti-TPO + as illustrated in figure (5) and table (5).

**Table5**: distribution of the sample according to ANTI-TPO

| Anti-TPO + | Anti-TPO - |
|---|---|
| 5% | 95% |

**Figure5**: distribution of the sample according to ANTI-TPO




**Discussion**

Thyroid gland is an endocrine gland. It is located in the inferior, anterior neck and is in charge of thyroid hormone production and secretion, as well as iodine balance in the human body. The thyroid gland generates around 90% inactive thyroid hormone, or thyroxine (T4), and 10% active thyroid hormone, or triiodothyronine (T3). Inactive thyroid hormone is peripherally transformed to either activated thyroid hormone or another inactive thyroid hormone [28]. The thyroid gland is in charge of producing three different types of iodothyronines. The predominant secretory product is inactive thyroxine, or T4, a triiodothyronine, or T3 prohormone. Type 1 deiodinase converts T4 to T3 in peripheral organs with high blood flow, such as the liver and kidneys. T4 is converted to active T3 in the brain by type 2 deiodinase, which is generated by glial cells. Reverse T3, or rT3, is the name given to the third iodothyronine. rT3 is inactive and is formed by T4 type 3 deiodinase activity [29]. Iodothyronines are made up of thyroglobulin and iodine. Thyroglobulin is synthesized inside the thyroid cells from basal to apical amino acids. Thyroglobulin is subsequently released into the follicular lumen, where it combines with iodine enzymatically to generate iodinated thyroglobulin. Endosomes holding this iodinated thyroglobulin subsequently merge with lysosomes, releasing the thyroglobulin enzymatically from the resulting thyroid hormone [30]. The cell then releases the thyroid hormones, while the leftover thyroglobulin is deiodinated and recycled for future use. Several studies [31-38] investigated changes in thyroid function related with variations in body mass index (BMI) in euthyroid people. However, the outcomes have been uneven.

The outcome of the research is to find the correlation between thyroid function and obesity in the Iraqi population who are diagnosed and treated of hypothyroidism. In comparison to another research, 1564 people aged 18 to 70 years were recruited at Beijing Chaoyang Hospital, Capital Medical University, between November 2018 and November 2019. They were all quizzed about their medical history and given physical exams and laboratory testing. Thyroid dysfunction, use of hormone replacement therapy such as thyroid or sex hormones; use of antithyroid treatment; antithyroid peroxidase antibody (TPOab) titers 60; severe hepatic or renal insufficiency; use of weight loss medications, antihypertensive or lipid-lowering drugs, glucocorticoids or amiodarone; and moderate-to-severe anemia, cancer, acute infection, and pregnancy were excluded. In all, 1564 people were included in the final study, with 468 of them being women. In the normal weight, overweight, and obese categories, BMI was 22.771.65, 27.011.34, and 32.071.98 kg/m2 (P0.001). The three groups' mean ages were 47.1812.68, 46.979.03, and 46.4010.87 years, respectively. Age or gender variations between the three groups were not statistically significant. The serum lipid profile changed significantly as BMI climbed. The levels of TC, TG, and LDL-C likewise increased in the three groups in accordance with BMI (P0.001). As BMI rose, HDL-C levels declined considerably (P0.001). The glucose metabolism indicators differed significantly across the three groups. FBG levels were 5.420.52, 5.600.54, and 5.670.56 mmol/L in the normal weight, overweight, and obese groups, respectively (P0.001). Fasting insulin, HOMA-, and HOMA-IR readings all rose as BMI increased. There was a similar trend in the numbers of WBCs, neutrophils, lymphocytes, and platelets; however, there were no statistically significant variations in NLR between the three groups. Tukey post hoc tests indicated statistically significant differences in all pairwise comparisons of BMI, HDL-C, TG, FINS, HOMA-IR, HOMA-, WBCs, neutrophils, and lymphocytes across the three groups. Thyroid function varied amongst the three groups. FT3 levels were 5.280.54, 5.360.56, and 5.460.52 pmol/L in the normal weight, overweight, and obese groups, respectively (P0.001). Their FT3/FT4 ratios were correspondingly 0.320.04, 0.330.04, and 0.340.04 (P0.001). All pairwise comparisons of FT3 and FT3/FT4 levels were statistically significant, according to post hoc testing. TPOab levels were lower in the normal weight group compared with the other two groups. TSH levels tended to rise when BMI rose, albeit the differences were not statistically significant. The three groups had no statistically significant variations in FT4 or TgAb levels [39,40]. Previous research has highlighted the significance of the link between obesity and thyroid dysfunction. Reports on the precise association between obesity and thyroid function, on the other hand, are scarce and contradictory. Fontenelle et al [41] collected several research on the state of thyroid hormones in euthyroid obese persons published between 2005 and 2015. Half of the studies (9/19) found a statistically significant positive association between BMI and TSH, whereas the other half did not. Similarly, half of the studies (6/11) discovered a link between BMI and FT3 or total triiodothyronine. Only three of the 16 studies found a negative relationship between BMI and FT4. The purpose of this research was to look at the relationship between BMI and thyroid function in euthyroid Chinese people. The current study findings

revealed a link between BMI and FT3 levels, as well as BMI and FT3/FT4 ratios. However, there were no statistically significant variations in FT4 levels across BMI groups. The cause and clinical consequences of alterations in thyroid function linked with obesity are yet unknown. Obesity is thought to impact thyroid function via a variety of pathways, including adipose tissue malfunction, the effects of hyperinsulinemia, and changes in thyroid gland form and function. Several investigations have shown that adipose tissue malfunction is the primary cause of changes in thyroid hormone homeostasis [41]. Obesity is understood to be a chronic, low-grade inflammatory disorder. Obese persons had higher levels of inflammatory indicators such as WBC, neutrophil, and lymphocyte counts [42]. Although the exact method by which WBCs and neutrophils influence thyroid function is unknown, a recent investigation discovered that these inflammatory indicators have a role in the development of nodular thyroid disease [43]. The current research included a limited number of participants, notably in the obese group. One probable explanation is that obese people pay less attention to their health and are less likely to undergo regular check-ups than their nonobese counterparts. As a result, despite the relatively small sample size, we did not observe a linear relationship between BMI and TSH levels. Nonetheless, using logistic regression analysis, we discovered that obese persons were more likely to have higher TSH levels than normal weight individuals, suggesting that BMI may have an influence on TSH levels mainly in obese patients. Another study was conducted in the internal medicine consultation department of Rizgary Teaching Hospital Erbil, Iraq, from May 2017 to May 2018. Sixty-eight consecutive women with the metabolic syndrome as per the Harmonizing definition were compared to 95 age matched women with no metabolic syndrome as a control group. Detailed history and physical examination with blood pressure and waist circumferences recorded and appropriate fasting blood samples were tested for plasma glucose, lipid profile, TSH, free T3, and free T4.

Subclinical hypothyroidism was present in 10 (14.7%) of the 68 women with metabolic syndrome and four women (4.2%) of the 95-control group with a significant difference (P = 0.018). Central obesity, hypertriglyceridemia, and low levels of high-density lipoprotein were present in 9 (17.3%), 9 (23.1%), and 10 (16.4%) women with subclinical hypothyroidism with a significant difference for each association (P = 0.013, 0.001 and 0.006, respectively).

## Conclusions

1.In the near future, the increasing prevalence of obesity may confound the definition of normal TSH range in population studies.

2. We did not observe a linear relationship between BMI and TSH levels.

3. Obesity is generally regarded by patients as being secondary to thyroid dysfunction.

4. Majority of patients (95%) got Anti-TPO – and only 5% got Anti-TPO +.

5. The correlation between BMI and TSH showed mildly elevated 4.5-10 mIU/mL, 36 had severely Elevated > 10 mIU/mL while 9 are normal 0.5-4.5 mIU/mL.

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## Conflict of Interest

The authors have no conflict of interest.

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