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Evaluating Thyroid Function in Obese Adults in Dhi Qar, Iraq

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Abstract. Obesity is a global public health challenge with profound endocrine and metabolic implications, particularly affecting the thyroid axis, which regulates basal metabolic rate and energy expenditure. Emerging evidence suggests that obese individuals may exhibit altered thyroid function even in the absence of overt thyroid disease. Despite existing studies, limited data are available from Iraq regarding the impact of obesity and aging on thyroid hormone profiles. This study aimed to evaluate thyroid hormone levels—TSH, free T4, and free T3—in obese adults across two age groups and compare them with non-obese healthy controls in Dhi Qar, Iraq. A cross-sectional study of 180 male participants revealed significant hormonal alterations. Elderly obese individuals (Group B, 60–75 years) had the highest TSH levels (4.16 ± 1.25 µIU/mL), followed by younger obese adults (Group A, 35–45 years, 3.45 ± 1.10 µIU/mL), compared with healthy controls (2.12 ± 0.80 µIU/mL). Both obese groups exhibited reduced fT4 and fT3 levels, most pronounced in elderly subjects (0.86 ± 0.14 ng/dL and 2.25 ± 0.30 pg/mL, respectively). This is one of the first studies in Iraq to demonstrate a recurrent association between obesity, age, and thyroid hormone dysregulation, pointing to subclinical hypothyroidism. The findings underscore the importance of routine thyroid function screening in obese individuals, particularly the elderly, for early detection and prevention of endocrine complications.

Highlights:

1. Pseudomonas aeruginosa effectively removed up to 70% of organic pollutants (COD) and over 60% of nitrogen compounds from wastewater.
2. The bacteria showed high heavy metal removal efficiency, with 87.2% for nickel and 77.5% for zinc.
3. Nitrate removal was the lowest (30.3%), highlighting the need for optimized treatment conditions to improve efficiency.

Keywords: Obesity, Thyroid hormones, TSH, Free T4, Free T3, Age groups, Subclinical hypothyroidism, Hormonal imbalance, Body mass index, Iraq.

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Introduction

Obesity is a complex, multifactorial chronic disease with an excess of body fat, which negatively affects health. Obesity has reached epidemic levels globally, with over 650 million adults being classified as obese in 2016 (WHO). Obesity is associated with an elevated risk of developing type 2 diabetes mellitus, cardiovascular disorders, musculoskeletal diseases, and cancers (1). The pathophysiology of obesity is multifactorial and involves genetic, environmental, metabolic, and hormonal factors. Among the key hormonal mechanisms involved in body weight regulation is the thyroid hormone axis. The thyroid gland plays a central function in basal metabolic rate (BMR) regulation, thermogenesis, lipid and glucose metabolism, and appetite. It secretes and produces the hormones thyroxine (T4) and triiodothyronine (T3), which control energy homeostasis and mitochondrial function in various tissues (2). Alterations in levels of thyroid hormone have been reported to profoundly influence body weight as well as pattern of fat.

There has been evidence in literature of an association between thyroid dysfunction, particularly subclinical hypothyroidism with increased thyroid-stimulating hormone (TSH) in the presence of normal free T4 levels, and obesity. In the obese, increased TSH levels have been reported even in the absence of frank thyroid disease, which could reflect a compensatory or adaptive response to increased body mass (3). Additionally, increased leptin levels, which are typical in obesity, can be used to modulate hypothalamic-pituitary-thyroid (HPT) axis activity by stimulating thyrotropin-releasing hormone (TRH) production, leading to the increase in TSH secretion (4).

Furthermore, obesity has also been seen to influence peripheral conversion of T4 into T3 and consequently modulate thyroid hormone function and metabolism. Increased activity of deiodinase type 2 (DIO2), the enzyme responsible for converting T4 to the more active T3, in adipose tissue has been observed in the obese, which may be a compensatory mechanism to increase energy expenditure (5). Conversely, chronic inflammation and cytokine dysregulation in obesity can impair the function of thyroid hormone receptors and lead to a state of tissue-level hypothyroidism in the presence of normal serum levels (6).

Conversely, thyroid disease may also be causally or contributively involved in the development or exacerbation of obesity. Hypothyroidism decreases metabolic rate, diminishes thermogenesis, and increases deposition of lipids, potentially leading to weight gain and to resistance to weight loss. In such cases, therapeutic replacement with thyroid hormones may induce partial reversal of these metabolic changes and minimal weight loss (7). It is not necessarily spectacular, however,

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suggesting other hormonal and lifestyle factors play a role too. The bidirectional interaction between thyroid hormones and obesity shows that there is an interaction of many folds. Although thyroid hormones influence body weight regulation, inflammatory mediators and adipokines, which are hormones from adipose tissue, may modify thyroid gland function and thyroid hormone metabolism. The above reciprocity favors the evaluation of thyroid function in obese patients and vice versa[8-11]. This study aimed to evaluate the profiles of the key thyroid hormones—TSH, free T4, and free T3—among obese patients of two age groups and compare them with profiles of non-obese healthy individuals.

Materials and Methods

This one-year cross-sectional observational study was carried out in Dhi Qar Governorate, Iraq, from January 2024 to December 2024. The objective was to compare thyroid hormone profiles of obese individuals from two age groups and a healthy, non-obese control group.

A total of 180 volunteers were recruited and divided into three groups:

Group A – Obese Individuals Below 45 Years (n = 60): Participants aged between 35 and 45 years with primary obesity (≥30 kg/m² BMI). All the participants in this group were Dhi Qar natives and had no known history of thyroid illness, diabetes, or chronic ailments.

Group B – Older Obese Patients (n = 60): Patients aged 60-75 years, with BMI ≥ 30 kg/m², and without any known thyroid disease or other endocrine disorders. They were selected from the local outpatient clinics and obesity consult clinics in Dhi Qar.

Group C – Healthy Control Group (n = 60): Sex- and age-matched healthy individuals with normal body weight (BMI 18.5–24.9 kg/m²) and lack of endocrine or metabolic disease history. Subjects were enrolled from the general population at regular health screening visits.

Sample Collection and Hormonal Testing

After informed consent, 5 mL of venous blood was obtained from each subject in a fasting condition (8–12 hours). The samples of blood were centrifuged to divide the serum and stored at –20°C until analysis.

Concentration of the following thyroid-related hormones was measured in serum using highly specific and sensitive commercial ELISA kits:

Thyroid Stimulating Hormone (TSH) – in units of μIU/mL

Thyroxine (T4) – in units of ng/dL

Triiodothyronine (T3) – in units of pg/mL

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All the assays were performed in the central diagnostic laboratory of College of Science, University of Thi-Qar, following standard operating procedure and quality control practice.

Statistical Analysis:

The data were expressed as Mean ± Standard Deviation (SD). Comparisons between groups were performed by one-way ANOVA followed by post hoc Tukey tests for multiple comparisons. A p-value of less than 0.05 was taken to be statistically significant.

Result

Thyroid hormone levels in the three study groups were compared, and relevant differences were noticed pointing towards the impact of obesity as well as age upon thyroid function. As can be seen from Table 1, the mean serum TSH levels of the obese groups were far greater than those of the healthy controls, with the highest among them being the elderly obese Group B at 4.16 ± 1.25 µIU/mL, followed by the young obese Group A at 3.45 ± 1.10 µIU/mL, whereas the healthy control Group C had a value significantly lower at 2.12 ± 0.80 µIU/mL. This trend is depicted graphically in Figure 1, a clear rising trend with increasing body weight and age for TSH. Elevated levels of TSH in obese subjects, particularly the elderly, may represent a compensatory response or subclinical hypothyroidism, possibly secondary to leptin-stimulated stimulation of the HPT axis or a difference in feedback sensitivity. By contrast, the concentration of free thyroxine (T4) was decreased in both obese groups, with the minimum mean being in Group B (0.86 ± 0.14 ng/dL), followed by Group A (0.90 ± 0.15 ng/dL) in comparison to a much increased level in the control group (1.23 ± 0.18 ng/dL), as depicted in Figure 2.

This inverse relationship indicates impaired thyroid hormone secretion or disrupted peripheral metabolism in the obese subjects, which may be exacerbated by age-related atrophy of the thyroid gland or concomitant chronic low-grade inflammation that disrupts hormone synthesis. Similarly, free triiodothyronine (T3) levels also showed a similar pattern, lower in the obese groups (2.55 ± 0.35 pg/mL in Group A and 2.25 ± 0.30 pg/mL in Group B) compared with the healthy controls (3.15 ± 0.40 pg/mL), as shown in Figure 3. More pronounced suppression of fT3 among the elderly obese subjects indicates reduced deiodinase activity or peripheral conversion of T4 to T3, as noted in metabolic syndrome and chronic inflammation. These findings collectively suggest obesity to be associated with mild but significant alterations in thyroid hormone balance, with aged subjects showing more inhibited thyroid profile than young obese adults. This supports the

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hypothesis that adiposity and age synergistically impair dysregulation of the thyroid axis, and suggests the value of routine thyroid screening among obese persons, especially in the elderly, to diagnose early dysfunction and prevent progression to overt hypothyroidism.

Table 1. The results of thyroid function tests (Mean **± Standard Deviation (SD))** for each group

Parameter	Group A (Young Obese, n=60)	Group B (Elderly Obese, n=60)	Group C (Healthy Controls, n=60)
TSH (µIU/mL)	3.45 ± 1.10	4.16 ± 1.25	2.12 ± 0.80
T4 (ng/dL)	0.90 ± 0.15	0.86 ± 0.14	1.23 ± 0.18
T3 (pg/mL)	2.55 ± 0.35	2.25 ± 0.30	3.15 ± 0.40

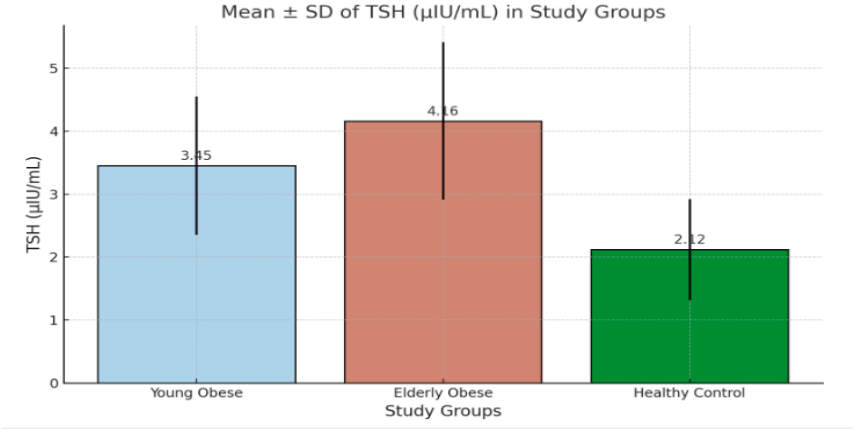

Fig. (1) Tsh Serum Level Of The Patient Groups And Control Group

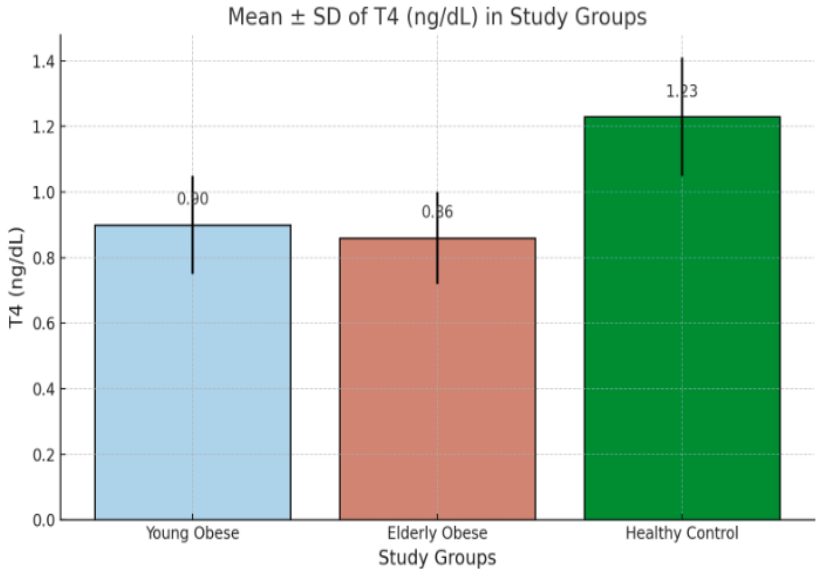

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Fig. (2) T4 Serum Level Of The Patient Groups And Control Group

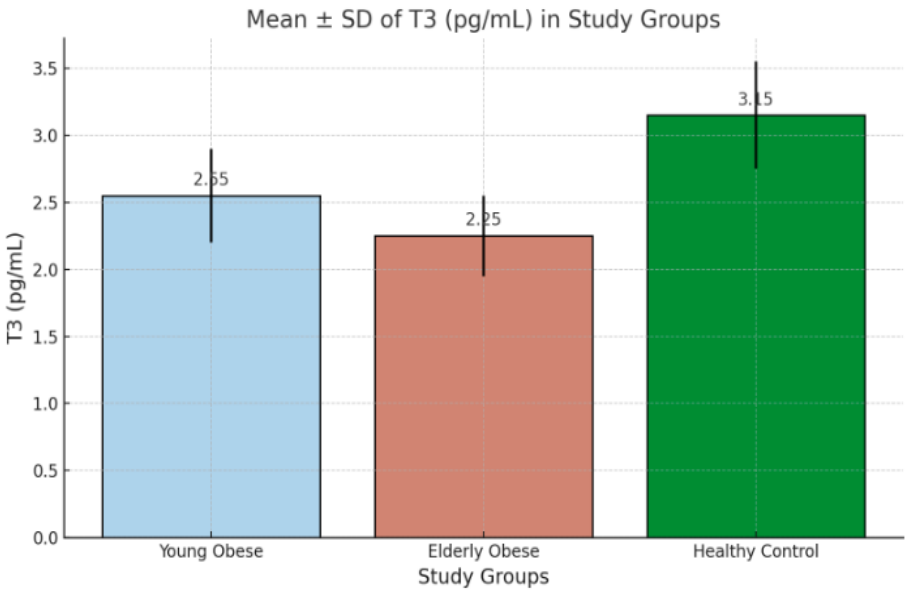

Fig. (3) T3 Serum Level Of The Patient Groups And Control Group

Discussion

It is important to combat this pandemic because obesity is the cause of all diseases, including those that lead to low life expectancy such as cardiovascular diseases [12]. While the impacts of obesity are well known, its etiology is less clear and complicated [13]. Understanding the cause of this rapid escalation in obesity levels is important for prevention and effective treatment. But in the overweight individuals, etiologic myths regarding obesity run rampant, and these create a paralysis or ineffective interventions. Despite the efforts expended in most countries, intervention against environmental causes has not led to the anticipated slowdown in obesity rates, though stabilization is observed in some regions. On a basic level, sedentarily and poor diet are uncontested risk factors [12-15].

Yet, obesity as a chronic disorder develops from the complicated interaction of many factors leading finally to energy imbalance and fat deposition. The resulting perception of ineffectiveness of severe diets or augmented physical activity leads patients to seek other possible etiologies for their disorder, such as hormonal disorders. Other conditions like insulin resistance and hypothyroidism, which usually co-exist with obesity, are by the patients usually regarded as causative factors rather

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than the effects of weight gain. This kind of thinking can have an effect on treatment and patient behavior [13-15].

Several studies agree with the findings of our current study, which demonstrated elevated TSH and lowered fT4 and fT3 levels in obesity, particularly in elderly individuals. Reinehr et al. demonstrated that obese children and adolescents exhibited significantly elevated levels of TSH and fT3 and lowered fT4 levels compared with their nonobese counterparts, substantiating the hypothesis of disturbed thyroid function in obesity [16-18]. Similarly, in a large cross-sectional study among obese adults, the authors found that up to 25% of the study subjects had subclinical hypothyroidism and elevated TSH levels that were significantly correlated with BMI and body fat [16,19].

Obese adults, in another comparative study, had high TSH and fT3 but low fT4 levels, which are like the hormonal patterns observed among our patient groups. A Korean pediatric cohort study also found that children who had abdominal obesity had significantly higher TSH and lower fT4 levels compared to non-obese children, indicating an earlier development of hormonal dysregulation. Aging also has an amplifying effect, as noted in a review that documented a spontaneous increase in TSH and decrease in fT3 in older individuals, in line with observations in Group B. Another study by the Pomerania cohort in Germany demonstrated that TSH was linearly related to fat mass in individuals over 60 years of age, further consolidating the synergistic effect of age and obesity on thyroid regulation. Finally, a refined statistical analysis using generalized additive models confirmed that TSH and fT3 increase, while fT4 decreases, with increased BMI even when age and sex were adjusted for. Such studies collectively add to the evidence that aging and obesity act synergistically to alter thyroid hormone profiles, supporting the findings of our study [19-25].

Conclusions

The current study identified significant differences in thyroid hormone profiles between the obese group and healthy controls when the two groups were compared. Both the young and old obese groups presented with an increased serum TSH level, culminating in an extreme value among the old group, pointing towards a trend towards subclinical hypothyroidism. Free T4 and free T3 levels were also significantly lower among the obese subjects, particularly among the old age group, suggesting faulty thyroid hormone production or defective peripheral conversion. These hormonal changes were not seen among the normal control group that had intact thyroid profiles. The

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investigation reveals a positive correlation between thyroid dysfunction and obesity, which appears to escalate with increasing age. This suggests the potential role of age-specific and obesity-related metabolic stress in thyroid deregulation. The research suggests that thyroid function should be screened for universally in obese patients, especially old patients, to allow early detection and management of hormonal imbalance that may result in further metabolic disturbances.

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