

TSH LEVELS AND BODY COMPOSITION IN OBESITY

Obezitede TSH düzeyleri ve Vücut Kompozisyonu

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ABSTRACT

ÖZ

Objective: This study aimed to compare serum thyroid-stimulating hormone (TSH) levels across body mass index (BMI) categories in euthyroid adults and to investigate the associations between TSH and detailed body composition parameters, including visceral fat rating, metabolic age, fluid percentage, and muscle mass.

Material and Methods: This retrospective cross-sectional study included 119 euthyroid adults who attended the internal medicine outpatient clinic of a university hospital in 2024. BMI was calculated using measured height and weight and categorized as underweight (<18.5 kg/m²), normal weight (18.5 – 24.9 kg/m²), overweight (25 – 29.9 kg/m²), and obese (≥ 30 kg/m²). Body composition parameters: visceral fat rating, metabolic age, fluid percentage, and muscle mass; were assessed using a Tanita BC-418 bioelectrical impedance analyzer. Group comparisons were performed using one-way ANOVA with post-hoc Tukey tests. Associations between TSH and body composition were analyzed using Spearman rank correlation.

Results: The mean age of participants was 32.5 ± 13.7 years, and 63.9% were female. Mean BMI was 25.5 ± 5.6 kg/m², and mean TSH was 1.71 ± 0.99 μ IU/mL. TSH increased across BMI groups: 1.25 ± 0.48 μ IU/mL in underweight, 1.49 ± 0.70 μ IU/mL in normal weight, 1.91 ± 1.11 μ IU/mL in overweight, and 2.21 ± 1.29 μ IU/mL in obese individuals (ANOVA $F(3,115)=4.20$, $p=0.007$). Post-hoc analyses showed higher TSH in obese individuals than underweight ($p=0.033$) and normal-weight participants ($p=0.027$). Spearman analyses showed positive correlations between TSH and visceral fat rating ($\rho=0.24$, $p=0.008$), metabolic age ($\rho=0.24$, $p=0.011$), muscle mass ($\rho=0.18$, $p=0.047$), and a negative correlation with fluid percentage ($\rho=-0.23$, $p=0.011$).

Conclusion: Higher TSH levels within the reference range are associated with obesity and unfavourable body composition parameters, including increased visceral adiposity, higher metabolic age, lower fluid percentage, and reduced muscle mass. These findings suggest that body composition may provide insight beyond BMI when interpreting TSH levels in euthyroid adults and highlight the clinical relevance of incorporating detailed composition metrics into metabolic and endocrine assessments.

Amaç: Bu çalışma, eutiroid yetişkinlerde vücut kitle indeksi (VKİ) kategorileri arasında serum tiroid uyarıcı hormon (TSH) düzeylerini karşılaştırmayı ve TSH ile visceral yağ derecesi, metabolik yaş, sıvı yüzdesi ve kas kütlesi gibi ayrıntılı vücut kompozisyonu parametreleri arasındaki ilişkileri araştırmayı amaçlamıştır.

Gereç ve Yöntem: Bütrospektif kesitsel çalışma, 2024 yılında bir üniversite hastanesinin iç hastalıkları polikliniğine başvuran 119 ötiroid yetişkini içermektedir. BMI ölçülen boy ve kilo kullanılarak hesaplandı ve zayıf ($<18,5$ kg/m²), normal kilo ($18,5$ – $24,9$ kg/m²), fazla kilo (25 – $29,9$ kg/m²) ve obez (≥ 30 kg/m²) olarak sınıflandırıldı. Vücut kompozisyonu parametreleri; visceral yağ derecesi, metabolik yaş, sıvı yüzdesi ve kas kütlesi; Tanita BC-418 biyoelektrik empedans analizörü kullanılarak değerlendirildi. Serum TSH ve laboratuvar parametreleri hastane elektronik kayıtlarından alındı. Grup karşılaştırmaları tek yönlü ANOVA ve post-hoc Tukey testleri kullanılarak yapıldı. TSH ve vücut kompozisyonu arasındaki ilişkiler Spearman sıralama korelasyonu kullanılarak analiz edildi.

Bulgular: Katılımcıların ortalama yaşı $32,5 \pm 13,7$ idi ve %63,9'u kadındı. Genel ortalama BMI $25,5 \pm 5,6$ kg/m² ve ortalama TSH $1,71 \pm 0,99$ μ IU/mL idi. TSH düzeyleri BMI grupları arasında kademeli olarak artmıştır: zayıf grupta $1,25 \pm 0,48$ μ IU/mL, normal kilo grubunda $1,49 \pm 0,70$ μ IU/mL, $1,91 \pm 1,11$ μ IU/mL, obez grupta $2,21 \pm 1,29$ μ IU/mL (ANOVA $F(3,115)=4,20$, $p=0,007$). Post-hoc analizler, obez bireylerde zayıf ($p=0,033$) ve normal kilolu katılımcılara ($p=0,027$) kıyasla anlamlı olarak daha yüksek TSH düzeyleri olduğunu göstermiştir. Spearman korelasyon analizleri, TSH ile visceral yağ derecesi ($\rho=0,24$, $p=0,008$), metabolik yaş ($\rho=0,24$, $p=0,011$) ve kas kütlesi ($\rho=0,18$, $p=0,047$) ile pozitif bir ilişki olduğunu ve sıvı yüzdesi ($\rho=-0,23$, $p=0,011$) ile negatif bir ilişki olduğunu ortaya koydu.

Sonuç: Referans aralığı içindeki yüksek TSH düzeyleri, obezite ve artmış visceral yağlanma, yüksek metabolik yaş, düşük sıvı yüzdesi ve azalmış kas kütlesi gibi olumsuz vücut kompozisyonu parametreleri ile ilişkilidir. Bu bulgular, ötiroid yetişkinlerde TSH düzeylerini yorumlarken vücut kompozisyonunun BMI'nin ötesinde ek bilgiler sağlayabileceğini göstermekte ve ayrıntılı kompozisyon ölçütlerinin metabolik ve endokrin değerlendirmelere dahil edilmesinin potansiyel klinik önemini vurgulamaktadır.

Keywords: Obesity, thyroid hormones, metabolic age

Anahtar Kelimeler: Obezite, tiroid hormonları, metabolik yaş



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INTRODUCTION

Obesity is a rapidly growing public health problem associated with numerous complications, including type 2 diabetes, cardiovascular disease, osteoarthritis, obstructive sleep apnoea, and various cancers (endometrial, colorectal, postmenopausal breast cancer, etc.).¹ Body mass index (BMI) is widely used to assess obesity; however, it may not provide an accurate estimate of metabolic risks, particularly due to its failure to account for visceral fat accumulation and fat distribution. Recent studies have highlighted that BMI is insufficient for determining cardiovascular and metabolic risks; instead, imaging techniques or anthropometric measurements indicating fat/muscle ratio should be used in conjunction with assessments for more accurate results.²⁻⁴ This approach reflects the fact that obesity increases disease risks not only based on body weight but also on fat distribution. Thyroid function, particularly thyroid-stimulating hormone (TSH) levels, plays a central role in energy metabolism. Mild elevations in TSH can lead to a decrease in basal metabolic rate and mild weight gain; in fact, even in euthyroid individuals, a 5-10% change in TSH can result in a few kilograms of weight change per year.⁵⁻⁷ The levels of TSH have been examined in obese individuals in relation to metabolic syndrome components (insulin resistance, dyslipidemia and hypertension), and it has been suggested that high-normal TSH levels may increase the prevalence of metabolic syndrome. Similarly, a similar relationship between BMI and TSH has been observed in individuals who are not obese but have subclinical hypothyroidism.⁸ Additionally, low-grade chronic inflammation and adipocyte-derived cytokines (IL-6, TNF- α) in obesity may affect thyroid cell function. Following weight loss achieved through bariatric surgery or calorie-restricted diets, significant decreases in TSH and FT3 levels were observed; it was noted that these changes were largely reversible.⁹ This study aims to compare serum TSH levels according to BMI groups in euthyroid adults. Our study is planned to contribute to filling the knowledge gap in this field by supporting the data in the literature on the relationship between obesity and TSH. Additionally, it is aimed that the data obtained will serve as a guide for physicians who evaluate TSH in clinical practice.

Previous studies have mostly focused on the relationship between BMI and TSH, while the relationship between TSH and detailed body composition parameters (e.g., visceral fat, muscle mass, metabolic age, fluid percentage) has been investigated in a limited number of studies. The originality of this study lies in its examination of the connection between TSH levels and body composition in euthyroid individuals, going beyond the classic BMI criteria. Therefore, this study

aimed to compare serum thyroid-stimulating hormone (TSH) levels across World Health Organization BMI categories in euthyroid adults and to examine the associations between TSH and detailed body composition parameters, including visceral fat rating, metabolic age, fluid percentage, and muscle mass. We hypothesized that individuals with higher BMI and unfavourable body composition profiles; characterized by greater visceral adiposity, higher metabolic age, lower fluid percentage, and reduced muscle mass, would exhibit higher TSH levels within the reference range.

MATERIALS AND METHODS

Study Design and Population

This retrospective cross-sectional study was conducted at the Internal Medicine outpatient clinic of Harran University Hospital in 2024. The study protocol was approved by the Harran University Clinical Research Ethics Committee (Date: 01.09.2025, No: 25.14.12). As a retrospective study using anonymised clinical data, informed consent was waived according to institutional policy.

A total of 119 adults were included in the analysis. The inclusion criteria were: (1) age ≥ 18 years, (2) availability of serum TSH, FT3 and FT4 measurements, (3) euthyroid status (FT3 and FT4 within reference ranges), and (4) completion of a body composition assessment on the same day as the laboratory tests. Exclusion criteria included: known thyroid disease (hypothyroidism, hyperthyroidism, thyroid nodules), use of thyroid hormone or antithyroid medications, pregnancy, malignancy, chronic systemic disease, acute infection, and a history of bariatric surgery.

Anthropometric Measurements and BMI Classification

Height (cm) and weight (kg) were measured using standard clinical procedures, and body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Participants were categorized into four groups according to World Health Organization criteria: underweight ($<18.5 \text{ kg/m}^2$), normal weight ($18.5\text{-}24.9 \text{ kg/m}^2$), overweight ($25\text{-}29.9 \text{ kg/m}^2$), and obese ($\geq 30 \text{ kg/m}^2$).

Body Composition Analysis

Body composition parameters were assessed using a Tanita BC418 segmental bioelectrical impedance analyzer (BIA). All measurements were performed in the outpatient clinic with participants barefoot, wearing light clothing and free of metal accessories. Measurements were obtained after bladder emptying, at similar daytime hours, and in a resting, non-exercised state in accordance with manufacturer recommendations. The device provided the following parameters: visceral fat rating, metabolic age, fluid percentage, and muscle mass (kg). These measurements

were recorded on the same day as venous blood sampling.

Laboratory Measurements

Venous blood samples were analyzed in the hospital's central laboratory. Serum thyroid-stimulating hormone (TSH), free triiodothyronine (FT3), and free thyroxine (FT4) were measured using chemiluminescent immunoassay methods. Additional laboratory parameters included C-reactive protein (CRP), glucose, creatinine, liver enzymes (ALT, AST), lipid profile (HDL, LDL, triglycerides), and complete blood count indices. The reference range for TSH was accepted as 0.2-4.5 mIU/L.

Statistical Analysis

Data analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean \pm standard deviation (SD) or median (interquartile range) depending on distribution, and categorical variables as numbers and percentages. Normality of numerical variables was assessed using the Shapiro-Wilk test. Comparisons of TSH levels among BMI categories were performed using one-way analysis of variance (ANOVA), followed by post-hoc Tukey HSD tests for pairwise comparisons. Because TSH, visceral fat rating, metabolic age, and muscle mass were not normally distributed, the associations between TSH and body composition parameters were analyzed using Spearman rank correlation (ρ). Correlation coefficients (ρ) and p-values were reported. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

A total of 119 individuals were included in the study. The mean age of the participants was 32.55 ± 13.65 years, with 63.9% being female ($n=76$) and 36.1% being male ($n=43$). Participants were divided into four groups based on BMI: low BMI 10.1% ($n=12$), normal weight 42% ($n=50$), overweight 31.9% ($n=38$), and obese 16% ($n=19$).

Laboratory findings for the patients were as follows: white blood cell count (WBC) $7.17 \pm 2.24 \times 10^3/\mu\text{L}$, haemoglobin (Hgb) $14.3 \pm 1.9 \text{ g/dL}$, glucose $91.7 \pm 19.2 \text{ mg/dL}$, creatinine $0.79 \pm 0.1 \text{ mg/dL}$, alanine aminotransferase (ALT) $21.65 \pm 35.74 \text{ U/L}$, aspartate aminotransferase (AST) $21.3 \pm 11.86 \text{ U/L}$, sodium (Na) $140.65 \pm 1.9 \text{ mmol/L}$, potassium (K) $4.3 \pm 0.3 \text{ mmol/L}$, C-reactive protein (CRP) $9.7 \pm 0.4 \text{ mg/L}$, TSH $1.71 \pm 0.98 \mu\text{IU/mL}$, T3 $3.4 \pm 0.5 \text{ pg/mL}$, T4 $1.22 \pm 0.1 \text{ ng/dL}$, vitamin D $11.02 \pm 8.1 \text{ ng/mL}$, LDL cholesterol $108.67 \pm 36.38 \text{ mg/dL}$, and HDL cholesterol $50.7 \pm 13.5 \text{ mg/dL}$ were determined (Table 1).

Table 1: Demographic and laboratory characteristics of the study population

Variable	Mean \pm SD	n (%)
Age (years)	32.5 ± 13.7	119
Female sex	—	76 (63.9)
Male sex	—	43 (36.1)
BMI (kg/m^2)	25.5 ± 5.6	119
Underweight (<18.5)	18.13 ± 2.27	12 (10.1)
Normal weight (18.5 - 24.9)	21.95 ± 2.01	50 (42.0)
Overweight (25 - 29.9)	28.35 ± 2.91	38 (31.9)
Obese (≥ 30)	33.86 ± 3.41	19 (16.0)
WBC ($10^3/\mu\text{L}$)	7.18 ± 2.24	119
Hemoglobin (g/dL)	14.39 ± 1.95	119
Glucose (mg/dL)	91.75 ± 19.21	119
Creatinine (mg/dL)	0.80 ± 0.17	119
ALT (U/L)	21.65 ± 35.74	119
AST (U/L)	21.30 ± 11.86	119
Sodium (mmol/L)	140.65 ± 1.96	119
Potassium (mmol/L)	4.39 ± 0.30	119
CRP (mg/L)	0.43 ± 0.49	117
TSH ($\mu\text{IU/mL}$)	1.71 ± 0.98	119
T3 (pg/mL)	3.46 ± 0.54	113
T4 (ng/dL)	1.23 ± 0.13	119
Vitamin D (ng/mL)	11.02 ± 8.19	119
LDL cholesterol (mg/dL)	108.68 ± 36.38	110
HDL cholesterol (mg/dL)	50.78 ± 13.55	110

BMI: Body mass index, WBC: White blood cell, Na: Sodium, K: Potassium, CRP: C-reactive protein, TSH: Thyroid stimulating hormone, LDL: Low-density lipoprotein, HDL: High-density lipoprotein

When TSH levels were examined according to BMI groups; the mean TSH level was $1.25 \pm 0.4 \mu\text{IU/mL}$ in the low BMI group, $1.48 \pm 0.69 \mu\text{IU/mL}$ in the normal weight group, $1.95 \pm 1.1 \mu\text{IU/mL}$ in the overweight group, and $2.2 \pm 1.2 \mu\text{IU/mL}$ in the obese group. In the one-way ANOVA analysis, a statistically significant difference was found between BMI groups in terms of TSH levels ($F(3,115) = 4.202$, $p = 0.007$). Post hoc (Tukey) tests revealed that TSH levels in obese individuals were significantly higher than those in the low BMI group ($p = 0.033$) and the normal BMI group ($p = 0.027$) (Table 2).

Table 2: TSH levels according to BMI groups

BMI Group	TSH ($\mu\text{IU/mL}$) Mean \pm SD	p-value
Low BMI	1.25 ± 0.4	0.008
Normal	1.48 ± 0.69	0.011
Overweight	1.95 ± 1.1	0.011
Obese	2.2 ± 1.2	0.047
ANOVA (F/p)	$F(3,115) = 4.202$ / $p = 0.007$	

BMI: Body mass index, TSH: Thyroid-stimulating hormone

When correlation analyses were performed on all participants together, a positive and significant relationship was found between TSH levels and visceral fat percentage ($p=0.007$). In addition, a significant correlation was found between metabolic age and TSH levels ($p=0.01$). A negative correlation was observed between fluid percentage and TSH ($p=0.007$). A statistically borderline significant correlation was detected between TSH levels and muscle mass ($p=0.05$) (Table 3).

Table 3: Spearman correlations between TSH and body composition

Variable	Spearman ρ	p-value
Visceral fat rating	0.24	0.008
Metabolic age (years)	0.24	0.011
Fluid percentage (%)	-0.23	0.011
Muscle mass (kg)	0.18	0.047

The fact that slight increases in TSH levels are more frequently observed in obese individuals suggests that there may be a complex relationship between these two conditions. Various studies have reported that serum TSH levels tend to be higher in individuals with increased body mass index, which may be related to metabolic adaptations or leptin-mediated central mechanisms.¹¹⁻¹³ Furthermore, it has been proposed that obesity might influence thyroid function, and that mild elevations in TSH could be associated with alterations in energy metabolism observed in obesity.¹⁴ However, the direction and causality of this relationship have not yet been clearly established, and there are conflicting findings in the literature on this subject. In this study, the relationship between BMI and serum TSH levels in euthyroid individuals was investigated; it was found that TSH levels were statistically significantly higher in the obese group. Additionally, a positive correlation was found between visceral fat percentage and metabolic age and TSH levels, while a negative correlation was found with fluid percentage. These findings suggest that obesity is associated with thyroid function not only through direct hormonal effects but also through indirect metabolic and inflammatory effects.

While some studies have described the increase in TSH levels as a phenomenon that may contribute to reduced energy metabolism and subsequent weight gain, other researchers have suggested that it may represent an adaptive physiological response to obesity.¹¹ In obese individuals, proinflammatory cytokines such as leptin, IL-6, and TNF- α have been reported to influence TSH levels by affecting the hypothalamic-pituitary-thyroid axis in association with increased adipose tissue.¹⁵ Pleić et al. reported that TSH levels are associated with metabolic syndrome components even when within the reference range.¹⁶ This finding suggests that TSH levels

may be considered not only as an indicator of thyroid function but also as a potential marker of metabolic balance.

Another noteworthy finding in our study is the positive correlation between TSH levels and visceral adiposity. Visceral fat has been associated with insulin resistance, dyslipidemia, and inflammation, and it may also be linked to thyroid function.¹⁷ Additionally, the observed relationship between metabolic age and TSH levels may reflect changes in thyroid function that occur alongside the age-related decrease in basal metabolic rate.^{18,19} The negative correlation between fluid ratio and TSH levels is another notable finding, although this has been addressed in only a limited number of studies. Research on the effects of cellular hydration on endocrine responses is still scarce. Mohamed and colleagues have suggested that intracellular fluid status might influence hormonal secretion, particularly in relation to pituitary hormones.²⁰ Alterations in endocrine parameters associated with intracellular fluid changes may be related to subclinical volume variations commonly observed in obese individuals. This finding suggests that fluid balance may be associated with the thyroid axis, indicating that assessment of hydration status may be considered a relevant parameter that could provide additional insights into thyroid function analysis.^{6,21} Although the relationship between TSH levels and BMI has been demonstrated in numerous studies, data on the relationship between TSH and metabolic age and fluid ratio remain limited. Our study contributes new observations to the literature in this regard. In particular, the negative correlation between fluid ratio and TSH may reflect a potential link between hydration status and the thyroid axis, which may offer additional perspectives for clinical practice. The observed relationship between muscle mass and TSH may be relevant in the context of thyroid hormones and their potential role in protein metabolism and muscle function. The literature reports that thyroid hormones are involved in protein synthesis and protein breakdown in muscle tissue.²² The association between muscle mass and TSH levels represents one of the relatively few pieces of evidence suggesting a link between thyroid hormones and protein metabolism. This finding may provide a basis for future larger-scale studies. In this study, TSH levels were observed to increase with higher BMI, particularly reaching relatively higher levels in obese individuals, and these changes may be associated with variations in body composition. In clinical practice, not only TSH but also FT3, FT4, visceral fat ratio, and other body composition parameters may be considered. Individuals with high-normal TSH levels may be monitored for potential metabolic risks, and lifestyle modifications may be suggested as appropriate. Future larger, multicentre, and longitudinal studies could

provide further insights into the relationship between TSH and obesity. Additionally, hormone-level-based intervention studies are warranted.

This study has several limitations. First, its retrospective, cross-sectional and single-centre design limits the ability to infer causality and may reduce generalisability to broader populations. Second, although the sample size was adequate for primary analyses, subgroup comparisons—particularly across BMI categories—may be underpowered. Third, body composition was assessed using a bioelectrical impedance analyzer rather than gold-standard imaging methods such as DEXA or MRI, which may introduce measurement variability. Fourth, potential confounding factors, including insulin resistance, dietary intake, physical activity level, and detailed inflammatory markers, were not assessed and may influence both body composition and thyroid function. Fifth, hydration status was evaluated using BIA-derived fluid percentage rather than direct physiological measurements, which may lead to limited precision. Finally, the study included only euthyroid individuals, which improves internal validity but restricts generalisability to populations with thyroid dysfunction. Future multicentre, prospective, and mechanistic studies incorporating comprehensive metabolic and hormonal profiling are needed to validate and expand upon these findings.

In conclusion, this study observed that TSH levels tend to increase with higher BMI and that these changes may be associated with body composition parameters such as visceral fat mass, metabolic age, and body fluid ratio. These findings suggest that TSH may have potential as a biomarker reflecting metabolic load and energy balance beyond classical thyroid assessment. This study contributes to the literature by examining the relationship between TSH levels and various components of body composition, not solely BMI. In particular, the associations between TSH and metabolic age and fluid ratio are parameters that have been relatively underexplored in previous studies. In this context, our findings indicate that TSH may provide additional insights into metabolic status beyond traditional thyroid evaluation.

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Ethics Committee Approval: The study protocol was approved by the Harran University Clinical Research

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REFERENCES

1. Perdomo CM, Cohen RV, Sumithran P, Clément K, Frühbeck G. Contemporary medical, device, and surgical therapies for obesity in adults. *Lancet*. 2023;401(10382):1116-1130.
2. Powell-Wiley TM, Poirier P, Burke LE, et al. Obesity and cardiovascular disease: A scientific statement from the American Heart Association. *Circulation*. 2021;143(21):e984-e1010.
3. Omma A, Erden F, Çolak SY, et al. Is the visceral adiposity index associated with the presence of cardiovascular risk scores and comorbidity in psoriatic disease? *J Turk Soc Rheumatol*. 2024;16(3):90-96.
4. Pandey A, Patel KV, Segar MW, et al. Effect of liraglutide on thigh muscle fat and muscle composition in adults with overweight or obesity: Results from a randomized clinical trial. *J Cachexia Sarcopenia Muscle*. 2024;15(3):1072-1083.
5. Walczak K, Sieminska L. Obesity and thyroid axis. *Int J Environ Res Public Health*. 2021;18(18):9434.
6. Biondi B. Subclinical hypothyroidism in patients with obesity and metabolic syndrome: A narrative review. *Nutrients*. 2023;16(1):87.
7. Song RH, Wang B, Yao QM, Li Q, Jia X, Zhang JA. The impact of obesity on thyroid autoimmunity and dysfunction: A systematic review and meta-analysis. *Front Immunol*. 2019;10:2349.
8. Teixeira PFDS, Dos-Santos PB, Pazos-Moura CC. The role of thyroid hormone in metabolism and metabolic syndrome. *Ther Adv Endocrinol Metab*. 2020;11:2042018820917869.
9. Cordido M, Juiz-Valiña P, Urones P, Sangiao-Alvarellos S, Cordido F. Thyroid function alteration in obesity and the effect of bariatric surgery. *J Clin Med*. 2022;11(5):1340.
10. Salmón-Gómez L, Catalán V, Frühbeck G, Gómez-Ambrosi J. Relevance of body composition in phenotyping the obesities. *Rev Endocr Metab Disord*. 2023;24(5):809-823.
11. Reinehr T. Obesity and thyroid function. *Mol Cell Endocrinol*. 2010;316(2):165-171.
12. Rotondi M, Loporati P, La Manna A, et al. Raised serum TSH levels in patients with morbid obesity: Is it enough to diagnose subclinical hypothyroidism? *Eur J Endocrinol*. 2009;160(3):403-408.
13. Tian Z, Nie Y, Li Z, et al. Total weight loss induces the alteration in thyroid function after bariatric surgery. *Front Endocrinol (Lausanne)*. 2024;15:1333033.
14. Nannipieri M, Cecchetti F, Anselmino M, et al. Expression of thyrotropin and thyroid hormone receptors in adipose tissue of patients with morbid obesity and/or type 2 diabetes: Effects of weight loss. *Int J Obes (Lond)*. 2009;33(9):1001-1006.
15. Le Moli R, Vella V, Tumino D, et al. Inflammasome activation as a link between obesity and thyroid disorders: Implications for an integrated clinical management. *Front Endocrinol (Lausanne)*. 2022;13:959276.
16. Pleić N, Gunjača I, Babić Leko M, Zemunik T. Thyroid function and metabolic syndrome: A two-sample bidirectional mendelian randomization study. *J Clin Endocrinol Metab*. 2023;108(12):3190-3200.
17. Wei Y, Yang M, Liu J, Wang Y, Wang G. Associations between sensitivity to thyroid hormones and visceral adiposity in euthyroid adults. *J Clin Endocrinol Metab*. 2025;110(8):e2744-e2753.

18. Le Moli R, Malandrino P, Russo M, et al. Levothyroxine therapy, calculated deiodinases activity and basal metabolic rate in obese or nonobese patients after total thyroidectomy for differentiated thyroid cancer, results of a retrospective observational study. *Endocrinol Diabetes Metab.* 2023;6(2):e406.
19. Walsh JP. Thyroid function across the lifespan: Do age-related changes matter? *Endocrinol Metab (Seoul).* 2022;37(2):208-219.
20. Mohamed E, Nashed A, Abo Elainin M, Osman D. Effect of body composition on thyroid hormones in euthyroid post-pubertal females: An observational study. *Egyptian J Phys Ther.* 2022;9(1):9-14.
21. Armstrong LE, Muñoz CX, Armstrong EM. Distinguishing low and high water consumers-a paradigm of disease risk. *Nutrients.* 2020;12(3):858.
22. Nappi A, Moriello C, Morgante M, Fusco F, Crocetto F, Miro C. Effects of thyroid hormones in skeletal muscle protein turnover. *J Basic Clin Physiol Pharmacol.* 2024;35(4-5):253-264.