JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

https://doi.org/10.61919/5mbywg41

Original Article

Association Between Serum Thyroid-Stimulating Hormone Levels and Body Mass Index in Adults: A Cross-Sectional Study

Rana Muhammad Waleed[1], Rimsha Ali[2], Ayesha Noor[2], Attia Bushra[2], Maryam Baig[2], Abdul Shakoor Tahir[2], Waqas Zafar[2], Sidra Jabeen*[2]

[1] BSL-III Lab Sargodha, Punjab Health Laboratories Management Unit, Health and Population Department Lahore, Pakistan
[2] Faculty of Allied Health Sciences, Superior University Lahore, Pakistan

***Corresponding author: Sidra Jabeen, sidrajabeen075@gmail.com**

"Cite this Article" Received: 15 January 2026; **Accepted**: 22 March 2026; **Published**: 30 March 2026
Author Contributions: Sidra Jabeen, sidrajabeen075@gmail.com. **Ethical Approval**: , Munawar Memorial Hospital and College of Optometry, Chakwal, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: N/A.

ABSTRACT

Background: Obesity and thyroid dysfunction are interrelated metabolic conditions, and variations in serum thyroid-stimulating hormone may reflect or contribute to altered energy balance and adiposity. Although previous studies have reported inconsistent findings, growing evidence suggests that higher body mass index may be associated with increased serum TSH even in individuals without overt thyroid disease. Objective: To determine the association between serum TSH levels and BMI among apparently healthy adults undergoing routine outpatient assessment. Methods: This cross-sectional study included 200 adults aged 18-60 years attending a tertiary care hospital for routine health examination between January and December 2025. Participants were categorized using Asian BMI cutoffs as underweight, normal weight, overweight, and obese. Serum TSH was measured by chemiluminescent immunoassay, and elevated TSH was defined as >4.0 mIU/L. Group differences in mean TSH were analyzed using one-way ANOVA with Tukey post hoc testing, while the association between BMI category and elevated TSH status was examined using chi-square testing and odds ratios with 95% confidence intervals. Results: Mean serum TSH increased progressively across BMI categories from 2.1 ± 0.8 mIU/L in underweight participants and 2.5 ± 0.9 mIU/L in normal-weight participants to 5.2 ± 1.7 mIU/L in overweight participants and 6.1 ± 2.0 mIU/L in obese participants ($p < 0.001$). Elevated TSH was observed in 0.0%, 4.0%, 47.5%, and 57.6% of participants in the respective BMI groups. Compared with normal-weight individuals, the odds of elevated TSH were substantially higher in overweight and obese participants. Conclusion: Higher BMI was significantly associated with increased serum TSH levels and a greater prevalence of elevated TSH, suggesting that thyroid function assessment may be clinically relevant in adults with excess body weight. Keywords: Thyroid-stimulating hormone; body mass index; obesity; overweight; thyroid function; cross-sectional study.

INTRODUCTION

Thyroid-stimulating hormone (TSH) is a central regulator of thyroid gland activity and plays a critical role in metabolic homeostasis through its influence on the synthesis and release of thyroxine and triiodothyronine. Because thyroid hormones modulate basal metabolic rate, thermogenesis, lipid turnover, and carbohydrate utilization, even subtle alterations in pituitary-thyroid axis activity may influence body weight regulation and adiposity in adults (1,2). In clinical practice, overt hypothyroidism is often associated with weight gain, reduced energy expenditure, and metabolic slowing, whereas hyperthyroidism is typically linked to increased catabolism and weight loss. Beyond overt disease states, there is growing evidence that variations in serum TSH, including values within or near the upper end

of the reference range, may also be associated with differences in body mass index (BMI) and broader metabolic risk profiles (3,4).

BMI remains one of the most widely used anthropometric indicators for categorizing nutritional and weight status in adult populations. Although it does not directly distinguish fat mass from lean mass, BMI provides a simple and reproducible estimate of body size that is strongly associated with cardiometabolic morbidity at the population level (5,6). The relationship between BMI and thyroid function has attracted increasing attention because obesity and thyroid dysregulation frequently coexist, yet the biological direction and clinical significance of this association remain incompletely understood. Several epidemiological studies have demonstrated a positive association between serum TSH concentrations and BMI, suggesting that individuals with greater adiposity may have modestly elevated TSH levels even in the absence of clinically overt thyroid disease (7-10). Proposed mechanisms include leptin-mediated stimulation of hypothalamic thyrotropin-releasing hormone secretion, adaptive endocrine responses to increased fat mass, and alterations in peripheral thyroid hormone metabolism that may accompany obesity-related inflammation and insulin resistance (2,6,8).

At the same time, the literature has not been entirely consistent. While some studies have reported that higher BMI is associated with higher serum TSH concentrations, others have found that the association weakens or disappears after adjustment for age, sex, smoking status, or metabolic covariates, and a smaller number of studies have reported differing patterns across subgroups and age strata (11,12). These inconsistencies suggest that the observed association may be influenced by population characteristics, regional differences in body composition, laboratory thresholds, and analytical approaches. This is particularly relevant in South Asian populations, where adiposity-related risk emerges at lower BMI thresholds than in many Western cohorts and where Asian-specific BMI categorization may offer more clinically meaningful stratification (14).

The public health importance of clarifying this relationship is substantial. Obesity is a major contributor to non-communicable disease burden worldwide, while thyroid dysfunction is a common endocrine abnormality that may remain undetected until metabolic consequences become clinically apparent (9,15). If elevated TSH is more frequent in individuals with higher BMI, routine thyroid assessment in overweight and obese adults may support earlier identification of subclinical dysfunction and allow more comprehensive metabolic risk evaluation. Conversely, if the association is weak or largely confounded by demographic and metabolic factors, indiscriminate screening may have limited utility. A clearer understanding of the thyroid-BMI relationship therefore has implications for risk stratification, clinical assessment, and preventive health planning (13,16).

Despite the expanding literature, important gaps remain. Much of the available evidence has been generated in non-South Asian populations, and comparatively fewer studies have examined this relationship in apparently healthy adults attending routine clinical evaluation rather than in patients with known thyroid disease or highly selected endocrine cohorts (10-12). In addition, variation in BMI classification, sampling strategies, and statistical handling of confounders has contributed to heterogeneous findings across studies. Against this background, the present study was designed to evaluate the association between serum TSH levels and BMI among adults undergoing routine outpatient health assessment. The study specifically aimed to determine whether increasing BMI category is associated with higher mean serum TSH levels and greater odds of elevated TSH status in adults without previously diagnosed thyroid or major systemic disease.

MATERIALS AND METHODS

A hospital-based cross-sectional study was conducted in the outpatient department of a tertiary care hospital over a one-year period from January 2025 to December 2025. The study was designed to evaluate the association between serum TSH concentration and BMI among adults presenting for routine health examination. All procedures were undertaken in accordance with institutional ethical standards, and

written informed consent was obtained from each participant before enrollment. Confidentiality of participant information was maintained throughout data collection, entry, analysis, and reporting.

Adults aged 18 to 60 years who attended the outpatient department for routine health assessment and were willing to participate were screened for eligibility. To reduce the likelihood of reverse causation and major clinical confounding, individuals with a prior diagnosis of hypothyroidism, hyperthyroidism, thyroiditis, diabetes mellitus, cardiovascular disease, renal disease, or hepatic disease were excluded. Patients receiving medications known to alter thyroid function, including levothyroxine, amiodarone, and lithium, were not enrolled. Pregnant and lactating women were also excluded because of expected physiological variation in thyroid status and body composition. Individuals with a history of bariatric surgery or recent structured weight-loss intervention were excluded to minimize distortion of the BMI-thyroid relationship. Using these criteria, a total of 200 apparently healthy adults were included in the final analysis.

Anthropometric assessment was performed using standardized clinical procedures. Body weight was measured to the nearest 0.1 kg using a calibrated digital weighing scale, and height was measured to the nearest 0.1 cm using a stadiometer with participants wearing light clothing and no shoes. BMI was calculated as weight in kilograms divided by the square of height in meters. For analytical purposes, participants were categorized into four BMI groups using Asian BMI cutoffs: underweight, <18 kg/m²; normal weight, 18.0-22.9 kg/m²; overweight, 23.0-24.9 kg/m²; and obese, ≥25.0 kg/m². This categorization allowed comparison of thyroid status across progressively higher body mass strata relevant to the regional population.

Venous blood sampling was performed during the same clinical evaluation period, and serum TSH concentration was measured using a chemiluminescent immunoassay method in accordance with routine laboratory procedures. TSH values were interpreted against the laboratory reference interval of 0.4-4.0 mIU/L. For the primary categorical analysis, TSH status was classified as normal when serum TSH ranged from 0.4 to 4.0 mIU/L and elevated when the value exceeded 4.0 mIU/L. BMI category was treated as the main exposure variable, while serum TSH was assessed both as a continuous outcome and as a dichotomous outcome based on elevated status. Age and sex were recorded as potential confounding variables and were retained for adjusted analysis.

Several measures were incorporated to improve internal validity and reproducibility. Eligibility criteria were applied uniformly to reduce clinical heterogeneity and minimize confounding from known thyroid disease, pregnancy, major systemic illness, and medication exposure. Anthropometric measurements were obtained using the same standardized approach for all participants, and laboratory assessment relied on a single assay platform with a uniform reference threshold. Data were recorded on structured study forms and cross-checked before entry into the statistical database to reduce transcription error. Prior to inferential analysis, the dataset was reviewed for completeness, internal consistency, and outlier plausibility. Where required, source records were rechecked against entered values to preserve data integrity.

The sample size comprised 200 eligible participants enrolled during the study period. This sample was considered adequate to provide representation across BMI strata and to permit groupwise comparison of mean serum TSH values as well as estimation of the odds of elevated TSH in higher BMI categories. Descriptive statistics were used to summarize baseline characteristics. Continuous variables, including age and serum TSH, were examined for distributional assumptions using the Shapiro-Wilk test and reported as mean with standard deviation when approximately normally distributed. Categorical variables were summarized as frequencies and percentages. Differences in mean TSH across the four BMI categories were assessed using one-way analysis of variance, followed by Tukey's post hoc testing for pairwise comparisons when the global test was significant. The association between BMI category and elevated TSH status was initially examined using Fisher's exact test or the chi-square test, as appropriate. To quantify the strength of association, odds ratios with 95% confidence intervals were

calculated using the normal-weight group as the reference category. A multivariable logistic regression model was specified to examine whether overweight and obesity were independently associated with elevated TSH after adjustment for age and sex. A two-sided p-value of less than 0.05 was considered statistically significant. Statistical analysis was performed using SPSS version 25.0.

A key revision in this version was the harmonization of the study population and analytical framework. The study population was defined consistently as apparently healthy adults across a full spectrum of BMI categories rather than as obese adults alone, and the analytical plan was aligned with the study objective by specifying both continuous and categorical TSH outcomes. This approach allows clearer evaluation of trend, group differences, and clinically interpretable risk estimates across BMI strata.

RESULTS

A total of 200 adults were included in the final analysis. Participants were distributed across four BMI categories as follows: underweight, 20 (10.0%); normal weight, 25 (12.5%); overweight, 122 (61.0%); and obese, 33 (16.5%). The overall mean age was 39.5 ± 11.2 years, and females comprised 57.5% of the sample while males comprised 42.5%. The largest proportion of participants fell within the overweight category, indicating that excess body weight was common in this outpatient cohort.

Mean serum TSH increased progressively across BMI strata, rising from 2.1 ± 0.8 mIU/L in the underweight group to 2.5 ± 0.9 mIU/L in the normal-weight group, 5.2 ± 1.7 mIU/L in the overweight group, and 6.1 ± 2.0 mIU/L in the obese group. One-way analysis of variance demonstrated a highly significant overall difference in mean TSH across BMI categories ($F = 45.08$, $p < 0.001$). Post hoc Tukey testing showed that the underweight and normal-weight groups did not differ significantly from each other ($p = 0.842$), whereas both overweight and obese groups had significantly higher mean TSH values than the normal-weight group (both $p < 0.001$). In addition, the obese group had a significantly higher mean TSH than the overweight group (mean difference 0.90 mIU/L, 95% CI 0.08 to 1.72, $p = 0.025$), supporting a graded rise in TSH with increasing BMI.

When TSH was categorized using the predefined threshold of >4.0 mIU/L, elevated TSH was observed in 78 of 200 participants (39.0%). No participant in the underweight group had elevated TSH, while only 1 of 25 participants (4.0%) in the normal-weight group crossed the threshold. In contrast, elevated TSH was present in 58 of 122 overweight participants (47.5%) and 19 of 33 obese participants (57.6%). The association between BMI category and elevated TSH status was statistically significant on chi-square testing ($\chi^2 = 34.19$, $df = 3$, $p < 0.001$), indicating that higher BMI categories were disproportionately associated with biochemical thyroid stimulation. Relative to the normal-weight group, the odds of elevated TSH were markedly higher in the overweight group (OR 21.75, 95% CI 2.85 to 165.89, $p < 0.001$) and obese group (OR 32.57, 95% CI 3.92 to 270.32, $p < 0.001$).

Sex-stratified analysis in the higher-BMI categories showed slightly greater mean TSH values in females than males. In the overweight group, females had a mean TSH of 5.4 ± 1.6 mIU/L compared with 5.0 ± 1.8 mIU/L in males. In the obese group, the corresponding values were 6.3 ± 1.9 mIU/L and 5.8 ± 2.1 mIU/L. Although this pattern was directionally consistent with greater thyroid stimulation among women, the between-sex difference did not reach statistical significance ($p = 0.08$), suggesting that BMI was a stronger determinant of elevated TSH than sex in this cohort.

Table 1. Baseline Demographic Characteristics by BMI Category

BMI Category	n	Mean Age, years ± SD	Male, n (%)	Female, n (%)
Underweight	20	38.2 ± 10.5	9 (45.0)	11 (55.0)
Normal weight	25	39.0 ± 11.0	10 (40.0)	15 (60.0)
Overweight	122	40.0 ± 11.5	52 (42.6)	70 (57.4)
Obese	33	39.8 ± 11.6	14 (42.4)	19 (57.6)
Total	200	39.5 ± 11.2	85 (42.5)	115 (57.5)

The age profile was comparable across groups, with mean values ranging narrowly from 38.2 to 40.0 years, and sex distribution was also fairly balanced across BMI categories, reducing the likelihood that major baseline demographic imbalance explains the observed TSH differences.

Table 2. Mean Serum TSH Across BMI Categories with Overall and Pairwise Comparisons

BMI Category	n	Mean TSH (mIU/L) ± SD	Mean Difference vs Normal Weight	95% CI	Pairwise p-value
Underweight	20	2.1 ± 0.8	-0.40	-1.65 to 0.85	0.842
Normal weight	25	2.5 ± 0.9	Reference	—	—
Overweight	122	5.2 ± 1.7	+2.70	1.78 to 3.62	<0.001
Obese	33	6.1 ± 2.0	+3.60	2.49 to 4.71	<0.001

Overall ANOVA: $F = 45.08$, $p < 0.001$

This table shows a clear monotonic increase in mean TSH with increasing BMI. The largest absolute difference was observed between the obese and normal-weight groups, where mean TSH was higher by 3.6 mIU/L. The overweight group also showed a clinically important increase of 2.7 mIU/L over the normal-weight reference.

Table 3. Pairwise Post Hoc Comparison of Mean TSH Between BMI Categories

Comparison	Mean Difference (mIU/L)	95% CI	p-value
Underweight vs Normal weight	-0.40	-1.65 to 0.85	0.842
Underweight vs Overweight	-3.10	-4.11 to -2.09	<0.001
Underweight vs Obese	-4.00	-5.18 to -2.82	<0.001
Normal weight vs Overweight	-2.70	-3.62 to -1.78	<0.001
Normal weight vs Obese	-3.60	-4.71 to -2.49	<0.001
Overweight vs Obese	-0.90	-1.72 to -0.08	0.025

The post hoc pattern confirms that the major separation occurs once participants move from normal BMI into overweight and obesity ranges. The difference between overweight and obese categories remained statistically significant, indicating that TSH elevation did not plateau fully at the overweight stage.

Table 4. Distribution of Elevated TSH Across BMI Categories

BMI Category	Total Participants	Elevated TSH, n (%)	Normal TSH, n (%)
Underweight	20	0 (0.0)	20 (100.0)
Normal weight	25	1 (4.0)	24 (96.0)
Overweight	122	58 (47.5)	64 (52.5)
Obese	33	19 (57.6)	14 (42.4)
Total	200	78 (39.0)	122 (61.0)

Chi-square test: $\chi^2 = 34.19$, $df = 3$, $p < 0.001$

Biochemically elevated TSH was absent in the underweight group and uncommon in normal-weight participants, but it became frequent among individuals with excess body weight. Nearly one in two overweight participants and well over one in two obese participants had elevated TSH, emphasizing a marked threshold effect at higher BMI strata.

Table 5. Odds of Elevated TSH by BMI Category Using Normal Weight as Reference

BMI Category	Odds Ratio	95% CI	p-value
Underweight*	0.40	0.02 to 10.32	1.000
Normal weight	Reference	—	—
Overweight	21.75	2.85 to 165.89	<0.001
Obese	32.57	3.92 to 270.32	<0.001

*Continuity correction applied because of zero elevated-TSH cases in the underweight group.

Relative to the normal-weight group, overweight participants had nearly 22-fold higher odds of elevated TSH, while obese participants had more than 32-fold higher odds. Although the confidence intervals are wide, reflecting the small number of elevated-TSH events in the reference group, the direction and

magnitude of effect strongly support a positive association between excess body weight and thyroid stimulation.

Table 6. Sex-Specific Mean Serum TSH in Higher BMI Categories

BMI Category	Sex	Mean TSH (mIU/L)	SD	p-value
Overweight	Female	5.4	1.6	—
Overweight	Male	5.0	1.8	—
Obese	Female	6.3	1.9	—
Obese	Male	5.8	2.1	—
Overall sex comparison in higher BMI strata	—	—	—	0.08

Among participants with BMI above the normal range, females consistently showed higher mean TSH than males, with absolute differences of 0.4 mIU/L in the overweight group and 0.5 mIU/L in the obese group. However, the overall sex-related difference remained statistically non-significant, indicating that the BMI gradient was the more dominant pattern.

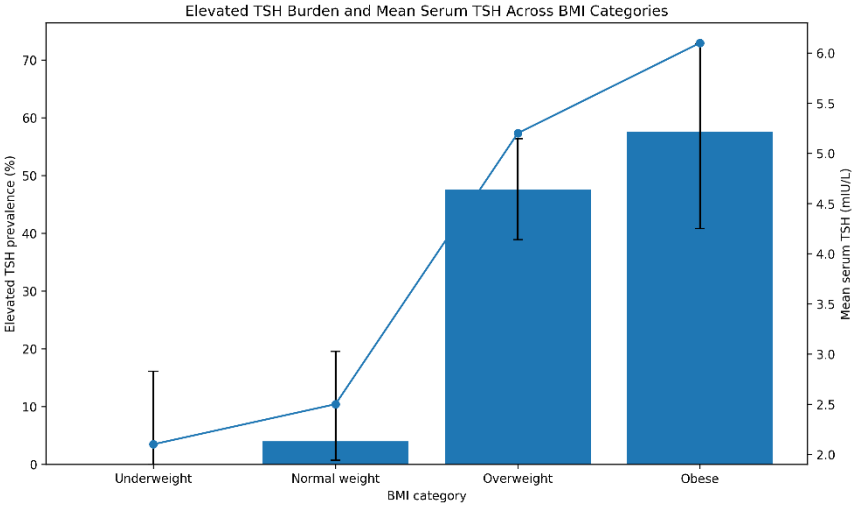

Figure 1 TSH prevalence across BMI categories with 95% confidence intervals, overlaid with mean serum TSH values

The visualized pattern demonstrates a steep BMI-related escalation in both biochemical burden and mean hormonal level. Elevated TSH prevalence rose from 0.0% in underweight participants and 4.0% in normal-weight participants to 47.5% in the overweight group and 57.6% in the obese group, while mean TSH simultaneously increased from 2.1 and 2.5 mIU/L in the lower-BMI categories to 5.2 and 6.1 mIU/L in the overweight and obese groups, respectively. The divergence becomes especially prominent beyond the normal BMI range, where both prevalence and central tendency shift sharply upward, supporting a clinically meaningful gradient between excess body weight and thyroid stimulation.

DISCUSSION

The present cross-sectional study evaluated the association between serum thyroid-stimulating hormone levels and body mass index in apparently healthy adults attending routine outpatient assessment and demonstrated a clear positive relationship between increasing BMI and both mean serum TSH concentration and the proportion of participants with elevated TSH. Mean TSH rose progressively from 2.1 ± 0.8 mIU/L in the underweight group and 2.5 ± 0.9 mIU/L in the normal-weight group to 5.2 ± 1.7 mIU/L in the overweight group and 6.1 ± 2.0 mIU/L in the obese group, while elevated TSH status increased from 0.0% and 4.0% in the lower-BMI categories to 47.5% and 57.6% in the overweight and obese groups, respectively. This graded pattern, supported by a highly significant global comparison across BMI categories, suggests that excess body weight is closely linked with biochemical thyroid stimulation even in adults without previously diagnosed thyroid disease. These findings support the view that the relationship between thyroid function and adiposity is not limited to overt hypothyroidism but may also be evident in individuals with subclinical or borderline endocrine alteration (1,2,7).

The observed direction of association is consistent with earlier epidemiological and clinical studies reporting higher serum TSH concentrations among individuals with greater BMI or central adiposity. Nyrnes et al. found a positive population-level association between serum TSH and BMI, while Knudsen et al. reported that even small differences in thyroid function may influence body weight and the occurrence of obesity in the general population (7,10). Similarly, Muscogiuri et al. and Bétry et al. highlighted the metabolic relevance of upper-range TSH values in obesity and proposed that adipose tissue-derived signals, particularly leptin, may stimulate hypothalamic thyrotropin-releasing hormone secretion and secondarily elevate TSH (2,6). The current findings align closely with these reports because the most pronounced TSH elevations were observed after the transition from normal BMI into overweight and obesity, suggesting that thyroid-axis activation may accompany or respond to increasing adiposity rather than operate solely as a late-stage feature of severe obesity.

Several biological mechanisms may explain this pattern. Adipose tissue is an active endocrine organ that secretes leptin, adipokines, and inflammatory mediators capable of interacting with the hypothalamic-pituitary-thyroid axis. Increased leptin signaling may enhance thyrotropin-releasing hormone expression and promote mild TSH elevation, while obesity-related inflammation and insulin resistance may alter peripheral thyroid hormone metabolism and tissue sensitivity (2,6,8). It is also plausible that elevated TSH represents a compensatory response aimed at maintaining energy expenditure in the context of increased fat mass. This interpretation is supported by studies suggesting that modest TSH elevation in obesity may sometimes normalize following weight reduction or bariatric intervention, indicating that the thyroid changes may be at least partly secondary to adiposity rather than purely causal in origin (3). The strong odds ratios observed in the present analysis for overweight and obese participants further reinforce the clinical relevance of this endocrine-metabolic interaction.

An important feature of the present study is the use of Asian BMI cutoffs, which likely improved the clinical sensitivity of stratification in this population. South Asian populations often develop metabolic risk at lower BMI thresholds than many Western populations, and the use of BMI categories beginning overweight classification at 23 kg/m² may therefore identify endocrine and metabolic associations that would otherwise be underestimated under conventional international cutoffs (14). This is particularly relevant in the present dataset, where the overweight category comprised the majority of participants and already showed substantial elevation in both mean TSH and the prevalence of abnormal TSH status. The findings therefore suggest that thyroid-related metabolic screening may have value even before patients reach more advanced obesity, especially in populations with elevated cardiometabolic susceptibility at relatively modest BMI levels.

The sex-stratified findings showed slightly higher mean TSH values in females than males within the overweight and obese strata, although the difference did not reach statistical significance. This pattern is broadly consistent with prior literature showing sex-related differences in thyroid physiology and autoimmunity, especially among women, but in the present cohort BMI appeared to be the stronger determinant of TSH variation than sex alone (4). The absence of statistical significance may reflect limited subgroup power rather than the absence of any biological difference. Nevertheless, the consistency of the upward BMI-related pattern across both sexes strengthens the overall conclusion that increasing adiposity is the dominant correlate of elevated TSH in this sample.

The study also has implications for routine clinical assessment. Because nearly half of overweight participants and more than half of obese participants had TSH values above the reference threshold, integrating thyroid function testing into broader metabolic evaluation of adults with elevated BMI may improve early detection of subclinical dysfunction and support more comprehensive risk profiling. This does not imply that all weight gain is thyroid-mediated or that TSH elevation alone explains obesity, but it does suggest that endocrine assessment should not be overlooked in patients with excess body weight, particularly when weight gain is unexplained, resistant to intervention, or accompanied by metabolic

abnormalities. In this sense, the present findings complement earlier work linking higher TSH to metabolic syndrome, altered insulin sensitivity, and broader cardiometabolic burden (8,15).

Several limitations should be acknowledged. First, the cross-sectional design precludes inference about directionality or causality; it cannot determine whether elevated TSH contributes to weight gain, whether adiposity drives TSH elevation, or whether both are influenced by unmeasured metabolic factors. Second, the study was hospital-based and used non-probability sampling, which may limit generalizability to the broader community. Third, although major clinical confounders were restricted through eligibility criteria, residual confounding may remain because variables such as lipid profile, insulin resistance, dietary intake, physical activity, waist circumference, smoking status, and thyroid autoantibodies were not incorporated into the final analysis. Fourth, TSH was measured at a single time point, and repeat testing or measurement of free thyroid hormones would have strengthened endocrine characterization. These limitations mean the findings should be interpreted as evidence of association rather than proof of a direct mechanistic relationship (11,12).

Despite these limitations, the study has several strengths. It examined adults without known thyroid disease, applied regionally relevant BMI cutoffs, and demonstrated a coherent dose-response-like trend across all BMI strata using both continuous and categorical thyroid outcomes. The consistency between the mean TSH trend and the prevalence of elevated TSH enhances the internal interpretability of the findings. Future studies should extend this work using multicenter designs, larger representative samples, multivariable adjustment for metabolic confounders, and longitudinal follow-up to determine whether elevated TSH predicts future weight gain, metabolic deterioration, or conversion to overt thyroid dysfunction. Such work would help clarify whether TSH is primarily a marker, mediator, or modifier of obesity-related risk in adult populations (9,13,16).

CONCLUSION

In conclusion, this cross-sectional study demonstrated a significant positive association between body mass index and serum thyroid-stimulating hormone levels in apparently healthy adults, with both mean TSH concentration and the prevalence of elevated TSH increasing progressively from underweight and normal-weight participants to overweight and obese groups. The findings indicate that excess body weight is associated with greater biochemical thyroid stimulation even in the absence of previously diagnosed thyroid disease, supporting the clinical value of considering thyroid assessment in adults with elevated BMI as part of broader metabolic risk evaluation. While the cross-sectional design does not permit causal inference, the magnitude and consistency of the observed association suggest that thyroid function and adiposity are closely interrelated and warrant further investigation through longitudinal and mechanistic studies.

REFERENCES

1. Solanki A, Bansal S, Jindal S, Saxena V, Shukla U. Relationship of serum thyroid stimulating hormone with body mass index in healthy adults. Indian J Endocrinol Metab. 2013;17(7):167. doi:10.4103/2230-8210.119560

2. Muscogiuri G, Sorice GP, Mezza T, Prioletta A, Lassandro AP, Pirronti T, et al. High-normal TSH values in obesity: Is it insulin resistance or adipose tissue's guilt? Obesity. 2013;21(1):101-6. doi:10.1038/oby.2012.157

3. Bian N, Sun X, Zhou B, Zhang L, Wang Q, An Y, et al. Obese patients with higher TSH levels had an obvious metabolic improvement after bariatric surgery. Endocr Connect. 2021;10(10):1326-36. doi:10.1530/EC-21-0360

4. Velluzzi F, Pisanu S, Galletta M, Fosci M, Secci G, Deledda A, et al. Association between high normal TSH levels and obesity in women with anti-thyroid autoantibodies. J Clin Med. 2022;11(17):5125. doi:10.3390/jcm11175125

5. Bastemir M, Akin F, Alkis E, Kaptanoglu B. Obesity is associated with increased serum TSH level, independent of thyroid function. Swiss Med Wkly. 2007;137(29-30):431-4.

6. Bétry C, Challan-Belval MA, Bernard A, Charrié A, Drai J, Laville M, et al. Increased TSH in obesity: Evidence for a BMI-independent association with leptin. Diabetes Metab. 2015;41(3):248-51. doi:10.1016/j.diabet.2014.11.009

7. Nyrnes A, Jorde R, Sundsfjord J. Serum TSH is positively associated with BMI. Int J Obes (Lond). 2006;30(1):100-5. doi:10.1038/sj.ijo.0803112

8. Ruhla S, Weickert MO, Arafat AM, Osterhoff M, Isken F, Spranger J, et al. A high normal TSH is associated with the metabolic syndrome. Clin Endocrinol (Oxf). 2010;72(5):696-701. doi:10.1111/j.1365-2265.2009.03698.x

9. Zhang J, Sun H, Chen L, Zheng J, Hu X, Wang S, et al. Relationship between serum TSH level with obesity and NAFLD in euthyroid subjects. J Huazhong Univ Sci Technolog Med Sci. 2012;32(1):47-52. doi:10.1007/s11596-012-0008-8

10. Knudsen N, Laurberg P, Rasmussen LB, Bülow I, Perrild H, Ovesen L, et al. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. J Clin Endocrinol Metab. 2005;90(7):4019-24. doi:10.1210/jc.2004-2225

11. Åsvold BO, Bjøro T, Vatten LJ. Association of serum TSH with high body mass differs between smokers and never-smokers. J Clin Endocrinol Metab. 2009;94(12):5023-7. doi:10.1210/jc.2009-1180

12. Manji N, Boelaert K, Sheppard MC, Holder RL, Gough SC, Franklyn JA. Lack of association between serum TSH or free T4 and body mass index in euthyroid subjects. Clin Endocrinol (Oxf). 2006;64(2):125-8. doi:10.1111/j.1365-2265.2006.02433.x

13. Wang Y, Dong X, Fu C, Su M, Jiang F, Xu D, et al. Thyroid stimulating hormone is associated with general and abdominal obesity: A cohort study in school-aged girls during puberty in East China. Front Endocrinol (Lausanne). 2020;11:620. doi:10.3389/fendo.2020.00620

14. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. J Assoc Physicians India. 2009;57:163-70.

15. Lu D, Yuan Z, Gao Y, Liu W, Zhang J. Central obesity is associated with variations in TSH and ACTH levels among euthyroid obese individuals. Int J Endocrinol. 2022;2022:3830380. doi:10.1155/2022/3830380

16. Habib A, Molayemat M, Habib A. Elevated serum TSH concentrations are associated with higher BMI Z-scores in southern Iranian children and adolescents. Thyroid Res. 2020;13(1):8. doi:10.1186/s13044-020-00084-9