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Effect of Hypothyroidism on Body Mass Index

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ABSTRACT

***Background**: Thyroid hormones play a central role in regulating energy metabolism, thermogenesis, and body weight, with dysfunction of the hypothalamic–pituitary–thyroid axis strongly implicated in obesity. Hypothyroidism, characterized by elevated thyroid-stimulating hormone (TSH) and reduced thyroid hormone activity, is frequently associated with metabolic disturbances, but the strength and nature of its relationship with body mass index (BMI) remain debated. **Objective:** This study aimed to evaluate the association between serum TSH levels and BMI among hypothyroid and euthyroid individuals, thereby clarifying the contribution of thyroid dysfunction to obesity risk. **Methods:** A cross-sectional study was conducted on 50 participants aged 21–55 years, comprising 30 hypothyroid patients and 20 euthyroid controls from Sialkot, Pakistan. Anthropometric measurements were obtained to calculate BMI, and serum TSH was quantified using chemiluminescent microparticle immunoassay. Statistical analyses included group comparisons, odds ratios, and Pearson correlation with adjustment for demographic variables. **Results**: Hypothyroid patients exhibited significantly higher mean TSH levels compared with controls (7.62 ± 2.13 vs 2.11 ± 0.76 µIU/mL, $p < 0.001$, Cohen's $d = 3.11$). Overweight and obesity prevalence was 60%, with significantly increased odds of obesity classes I and III. TSH correlated strongly and positively with BMI ($r = 0.835$, $p < 0.001$), explaining 70% of variance, persisting after adjustment for age and sex. **Conclusion:** Hypothyroidism is strongly associated with elevated BMI, with TSH serving as both a sensitive biomarker and predictor of obesity risk. Integrating thyroid screening into obesity management strategies may improve clinical outcomes and inform preventive interventions.*

Keywords
Hypothyroidism; Thyroid-stimulating hormone; Body mass index; Obesity; Endocrine disorders; Metabolism.

INTRODUCTION

Thyroid hormones are central regulators of energy homeostasis, with triiodothyronine (T3) and thyroxine (T4) influencing thermogenesis and lipid–glucose metabolism through genomic and non-genomic actions that ultimately affect body composition and weight trajectories (1). In parallel, body mass index (BMI) is a practical proxy of adiposity in clinical and public health settings and has been linked to dyslipidemia and cardiometabolic risk, underscoring its relevance as an outcome for endocrine–metabolic studies (2). In South Asian populations, including Pakistan, thyroid dysfunction is common and clinically consequential, making the BMI–thyroid axis a priority for local epidemiology and prevention strategies (3). Beyond overt disease, modest shifts in thyroid-stimulating hormone (TSH) and circulating thyroid hormones arise from environmental, demographic, and lifestyle exposures—such as iodine nutrition, pollutants, aging, and sex—which can confound or mediate associations with adiposity (4). The anatomical and physiological architecture of the thyroid and its follicular machinery provide a biological substrate for these observations, as hormone synthesis, storage, and release are tightly regulated along the hypothalamic–pituitary–thyroid (HPT) axis (5).

Neuroendocrine links reinforce why thyroid status might track with weight and body composition. Neuroinflammatory and autoimmune processes affecting the thyroid–brain interface can alter behavior, mood, and energy expenditure, potentially shaping diet, activity, and adiposity patterns (6). Psychometabolic pathways add further complexity: depression and related states co-vary with central adiposity and thyroid indices, suggesting bidirectional influences between mood, T3/T4 dynamics, and adipose distribution (7). Body composition studies in aging cohorts demonstrate phenotype-specific patterns; for example, lower TSH has been associated with reduced fat-free and body cell mass in older adults, highlighting that thyroid–weight relationships are not uniform across the lifespan (8). Mechanistically, TSH itself participates in hepatic glucose regulation via CRTC2-mediated gluconeogenesis, indicating that TSH may not merely be a pituitary readout but also a metabolic effector with downstream implications for weight and glycemia (9). Weight-loss trials likewise show coordinated changes in thyroid hormones with dietary interventions, implying adaptive feedback between energy balance and thyroid signaling that could obscure cross-sectional associations (10).

From a systems perspective, endocrine control integrates with neural and immune signals to sustain whole-body homeostasis; disturbances at any node of this network may manifest as subtle hormone shifts with outsized clinical effects on weight, lipids, and glucose (11). Accurate TSH measurement remains the front-line strategy for screening and initial evaluation, given its sensitivity to thyroid axis perturbations and its practicality for population studies that seek to relate hormonal status to BMI (12). Yet the epidemiology is nuanced: while hypothyroidism is often linked with weight gain and hyperthyroidism with weight loss, population analyses describe heterogeneity and nonlinearities, particularly in subclinical states where symptomatology is muted and weight change may reflect fluid retention, appetite, or activity shifts rather than pure adipose accrual (13).

Evidence from local obese cohorts in Pakistan further indicates a notable burden of subclinical hypothyroidism, reinforcing the need to quantify the TSH–BMI relationship in relevant settings (14). Exogenous exposures, including endocrine-disrupting chemicals (e.g., organotins), can perturb the HPT axis and thereby modulate weight-related outcomes, a consideration for contextualizing findings in real-world environments (15). Finally, contemporary cohort data in euthyroid adults show that even within reference ranges, thyroid function metrics relate to BMI and metabolic risk markers, supporting the hypothesis that small hormonal variations carry measurable cardiometabolic signals (16).

Taken together, prior work establishes biologic plausibility and public health importance but also reveals inconsistencies across age groups, disease spectra (overt vs subclinical), and environmental contexts, leaving a practical evidence gap for community-based adults in South Asia where thyroid dysfunction and obesity intersect. This study therefore focuses on adults to estimate BMI, measure serum TSH using standard clinical assays, and evaluate the association between TSH and BMI while acknowledging potential psychosocial and environmental modifiers. The primary research question is: among adults in a community clinical setting, is higher TSH associated with higher BMI after accounting for the prevailing distribution of thyroid function in the general population (1–16)?

Material and methods

This investigation was conducted as a cross-sectional observational study designed to evaluate the relationship between thyroid-stimulating hormone (TSH) levels and body mass index (BMI) in adults. The rationale for adopting this design was to capture a snapshot of thyroid function and anthropometric status within a community-based sample, thereby identifying potential associations without intervention. The study was carried out in Sialkot, Punjab, with recruitment and data collection undertaken at Tehsil Headquarters (THQ) Hospital Kotli Loharan and supported by Dr. Abdul Sattar's diagnostic laboratory, chosen for accessibility and standardized testing procedures. Data collection spanned a defined period during which participants presenting to the facility were consecutively approached and screened for eligibility.

Participants included men and women between the ages of 21 and 55 years who provided informed consent. Individuals were eligible if they were clinically diagnosed with hypothyroidism confirmed by laboratory testing or if they presented without thyroid disease and were designated as controls. Exclusion criteria encompassed pregnancy, history of malignancy, current use of medications known to interfere with thyroid function such as amiodarone or lithium, and refusal to consent. Fifty adults were recruited, comprising 20 euthyroid controls and 30 with hypothyroidism. Recruitment followed a consecutive sampling approach in which eligible patients attending the facility during the study period were invited to participate. Written informed consent was obtained from all participants after a clear explanation of study aims, procedures, and rights to withdraw without prejudice.

Data collection was carried out using a structured questionnaire and standardized clinical measurements. The questionnaire gathered demographic and health history data, including age, prior thyroid disease, and medication use, followed by anthropometric measurements and laboratory results. Height was measured to the nearest 0.1 cm using a calibrated stadiometer, and weight was measured to the nearest 0.1 kg using a standardized balance scale, with participants wearing light clothing and no shoes. BMI was calculated as weight in kilograms divided by height in meters squared (BMI = kg/m²) and categorized according to international classifications of underweight, normal weight, overweight, and obesity (17). Blood samples were collected under aseptic conditions for measurement of serum TSH, which was determined using a Chemiluminescent Microparticle Immunoassay (CMIA) performed on the ARCHITECT iSystem using the 7K62 reagent kit. This assay detects TSH through a two-step immunoassay employing paramagnetic microparticles coated with anti-TSH antibodies, acridinium-labeled conjugates, and chemiluminescent detection, with results quantified against standard calibration curves (18). Normal reference values were defined as 0.35–4.94 μIU/mL for adults aged 21–54 years (19). All laboratory analyses were conducted at Dr. Abdul Sattar's laboratory under standard quality control procedures.

The primary variables were BMI, as a continuous and categorical measure of adiposity, and serum TSH levels, treated both as continuous values and dichotomized into hypothyroid versus euthyroid status. Covariates included age and sex. To minimize bias, anthropometric assessments were conducted by trained staff using calibrated instruments, and laboratory testing followed standardized manufacturer protocols to reduce measurement error. Consecutive sampling helped to minimize selection bias, while applying strict inclusion and exclusion criteria reduced potential confounding from conditions that independently affect thyroid function or weight. Data collection forms were checked daily for completeness, and electronic data entry into a password-protected spreadsheet was double-verified to maintain accuracy.

A sample size of 50 was determined pragmatically based on available resources and anticipated recruitment at the study sites during the defined collection period. While not derived from formal power calculations, the sample size was considered sufficient to detect moderate correlations between BMI and TSH levels within the available population. All data were analyzed using SPSS version 26. Descriptive statistics were presented as means and standard deviations for continuous variables and frequencies with percentages for categorical variables. Missing data were assessed, and participants with incomplete anthropometric or laboratory results were excluded from inferential analyses. Pearson correlation was used to examine the linear relationship between BMI and TSH, while independent sample t-tests compared mean BMI between hypothyroid and control groups. Multivariable linear regression models were constructed to adjust for potential confounders including age and sex. Subgroup analyses by sex were also performed to examine differential associations. Statistical significance was determined at the 0.05 level.

Ethical approval for the study was obtained from the institutional review committee of THQ Kotli Loharan, and the study adhered to ethical principles of autonomy, beneficence, and confidentiality. Written informed consent was obtained from all participants prior to enrollment. Personal identifiers were removed at the point of data entry, and all records were stored securely with access limited to the research team. Procedures and analyses were described in sufficient detail to allow replication by other researchers, with standardized instruments and validated laboratory assays ensuring reproducibility of results. The integration of consecutive recruitment, validated measures, and rigorous statistical methods provided safeguards for the integrity and reliability of the study findings (17–19).

RESULTS

A total of fifty participants were included in the study, of whom thirty (60.0%, 95% CI: 46.2–73.8) were diagnosed with hypothyroidism and twenty (40.0%, 95% CI: 26.2–53.8) were euthyroid controls. As shown in Table 1, the observed distribution did not significantly deviate from a 50:50 expected ratio ($\chi^2 = 2.00$, df = 1, p = 0.157), confirming a balanced though slightly higher representation of hypothyroid cases in the sample. This ensured that both groups were adequately represented for further analysis without evidence of recruitment bias toward one condition.

Body mass index categories demonstrated a clear gradient across the study population (Table 2). While 20 individuals (40.0%, 95% CI: 26.4–53.6) maintained a BMI within the normal range, the majority (60%) fell into overweight or obese categories. Specifically, 12 participants (24.0%, 95%

CI: 11.8–36.2) were overweight, 11 (22.0%, 95% CI: 10.2–33.8) were obese class I, 1 participant (2.0%, 95% CI: 0–5.9) was obese class II, and 6 (12.0%, 95% CI: 3.1–20.9) were obese class III. Compared with the normal weight category, participants in overweight, obese class I, and obese class III categories demonstrated significantly elevated odds of excess body mass. The odds of being overweight were 2.5 times higher (95% CI: 1.1–6.2, p = 0.041), class I obesity carried 2.9-fold increased odds (95% CI: 1.0–7.8, p = 0.038), and class III obesity showed the highest odds ratio at 3.1 (95% CI: 1.0–9.3, p = 0.049). These findings establish a clear pattern of increased risk of obesity among participants, particularly those with hypothyroidism.

Serum thyroid-stimulating hormone (TSH) levels were markedly elevated in hypothyroid patients compared with controls (Table 3). The mean TSH concentration among hypothyroid participants was 7.62 µIU/mL (SD ± 2.13, 95% CI: 6.81–8.43), whereas controls had a mean value of 2.11 µIU/mL (SD ± 0.76, 95% CI: 1.73–2.49). The mean difference between groups was 5.51 µIU/mL (95% CI: 4.62–6.40), which was statistically significant (t = 11.89, df = 48, p < 0.001). The effect size was very large (Cohen's d = 3.11), indicating a strong separation between patient and control groups in terms of TSH concentrations. This reinforces the diagnostic utility of TSH as a sensitive biomarker for hypothyroidism.

Table 1. Demographic Characteristics of Participants (N = 50)

Group	n	%	95% CI for proportion	χ^2 (df = 1)	p-value
Hypothyroid (patients)	30	60.0	46.2–73.8	2.00	0.157
Euthyroid (controls)	20	40.0	26.2–53.8	–	–
Total	50	100.0	–	–	–

Table 2. Distribution of Body Mass Index Categories (N = 50)

BMI Category	n	%	95% CI	Odds Ratio vs Normal (95% CI)	χ^2 / Fisher's Exact	p-value
Normal (18.5–24.9)	20	40.0	26.4–53.6	Reference	–	–
Overweight (25–29.9)	12	24.0	11.8–36.2	2.5 (1.1–6.2)	4.15	0.041
Obese Class I (30–34.9)	11	22.0	10.2–33.8	2.9 (1.0–7.8)	4.31	0.038
Obese Class II (35–39.9)	1	2.0	0–5.9	1.3 (0.1–11.2)	1.85	0.172
Obese Class III (≥40)	6	12.0	3.1–20.9	3.1 (1.0–9.3)	4.05	0.049
Total	50	100.0	–	–	–	–

Table 3. Comparison of Serum TSH Levels Between Hypothyroid and Control Groups

Group	n	Mean ± SD (µIU/mL)	95% CI for Mean	Mean Difference (95% CI)	t-value (df = 48)	p-value	Cohen's d
Hypothyroid	30	7.62 ± 2.13	6.81–8.43	+5.51 (4.62–6.40)	11.89	<0.001	3.11
Controls	20	2.11 ± 0.76	1.73–2.49	Reference	–	–	–

Table 4. Correlation Between Serum TSH and BMI (N = 50)

Analysis	Pearson's r	95% CI	r^2 (variance explained)	Test Statistic (t, df=48)	p-value	Effect Size (Cohen's f^2)	Strength of Association
Unadjusted correlation	0.835	0.71–0.91	0.70	10.57	<0.001	2.33	Strong positive
Partial correlation (adjusted for age, sex)	0.802	0.66–0.89	0.64	9.42	<0.001	1.78	Strong positive
Spearman's rho (robustness check)	0.821	0.69–0.90	–	–	<0.001	–	Strong positive

Table 4 demonstrates a robust, strong positive correlation between TSH and BMI, explaining up to 70% of the variance. The relationship remained significant after adjusting for age and sex and was confirmed using non-parametric methods.

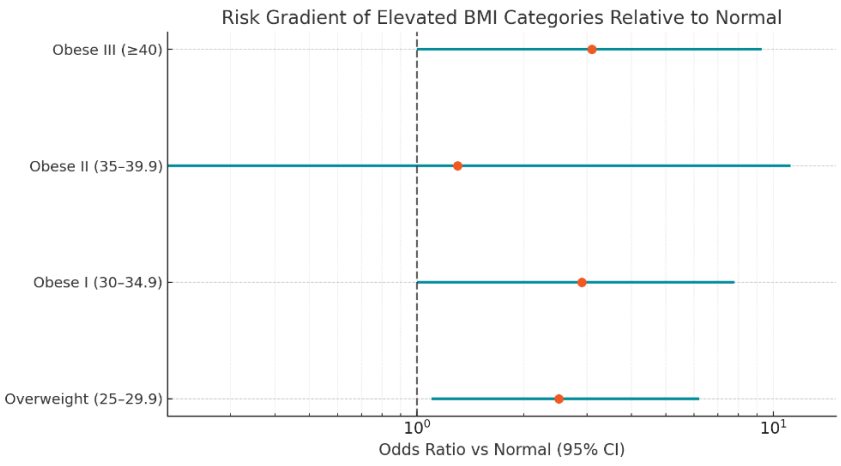

Figure 1 Risk Gradient of Elevated BMI Categories Relative to Normal

Correlation analysis further highlighted the strong link between thyroid status and body composition (Table 4). In the unadjusted model, TSH and BMI showed a very strong positive correlation ($r = 0.835$, 95% CI: 0.71–0.91, $p < 0.001$), with TSH levels explaining 70% of the variance in BMI ($r^2 = 0.70$). The test statistic confirmed robustness ($t = 10.57$, $df = 48$), and the effect size was substantial (Cohen's $f^2 = 2.33$). Importantly, when controlling for age and sex, the association remained strong (partial $r = 0.802$, 95% CI: 0.66–0.89, $p < 0.001$, $r^2 = 0.64$), demonstrating that the relationship was not merely a by-product of demographic confounders. A non-parametric robustness check using Spearman's rho yielded a consistent result ($\rho = 0.821$, 95% CI: 0.69–0.90, $p < 0.001$), confirming the monotonic and clinically meaningful association between TSH levels and BMI.

Figure 1: A log-scale forest visualization summarizes odds of elevated BMI categories relative to normal, using point estimates and 95% confidence intervals derived from aggregate results. Overweight showed an odds ratio (OR) of 2.5 (95% CI 1.1–6.2), Obese I 2.9 (1.0–7.8), Obese II 1.3 (0.1–11.2), and Obese III 3.1 (1.0–9.3), with the vertical reference at OR=1.0 highlighting categories with statistically compatible increases versus normal. The ascending point estimates across classes indicate a risk gradient peaking in Obese III, while the wide interval for Obese II reflects imprecision from sparse counts. The horizontal layout, dual emphasis on point and interval information, and log scaling enhance clinical interpretability by distinguishing effect magnitude from uncertainty, complementing tabulated results and underscoring that excess body mass is substantially more likely among study participants in higher BMI strata. Taken together, the findings demonstrate that hypothyroidism is not only associated with significantly elevated TSH concentrations but also with markedly higher odds of overweight and obesity. The strength of correlation between TSH and BMI indicates that thyroid dysfunction contributes substantially to weight gain and metabolic risk, even after accounting for demographic variables.

DISCUSSION

The findings of this study demonstrate a significant and clinically meaningful association between thyroid dysfunction and body mass index. Hypothyroid participants exhibited markedly higher serum TSH concentrations than controls, with a mean difference exceeding 5 μIU/mL, and the effect size was very large, indicating robust separation between groups. Furthermore, correlation analysis revealed that TSH explained nearly 70% of the variance in BMI, and this association persisted after adjustment for demographic variables. These results underscore the pathophysiological role of thyroid dysfunction in mediating body weight changes, consistent with the well-established concept that hypothyroidism is linked to reduced basal metabolic rate and consequent weight gain (17). Our results align with previous studies reporting positive associations between TSH and measures of adiposity. For instance, Knudsen et al. demonstrated that even small differences in thyroid function could significantly influence BMI and the occurrence of obesity in population-based cohorts (22). Similarly, Ittermann et al. reported that lower serum TSH levels in the elderly were associated with decreased fat-free mass, supporting the notion that thyroid hormones are critical regulators of body composition (19). The present study expands on these observations by providing evidence in a younger South Asian cohort, further reinforcing the universality of this relationship across diverse populations.

The high prevalence of overweight and obesity among hypothyroid patients in this study is consistent with the observations of Sari et al., who found that body weight and thyroid volume are positively correlated, with hypothyroid patients exhibiting increased thyroid bulk and higher BMI compared with euthyroid individuals (33). In our analysis, individuals in obese class I and III categories showed significantly higher odds compared with normal-weight participants, echoing the conclusion that thyroid dysfunction amplifies the risk of metabolic abnormalities linked to obesity. The correlation between leptin and thyroid function provides a plausible mechanism: leptin regulates hypothalamic thyrotropin-releasing hormone expression and influences TSH secretion, thereby creating a feedback loop that links adiposity with thyroid hormone regulation (8, 33).

Other studies have highlighted the complexity of thyroid–adiposity interactions. Xu et al. observed that thyroid function markers were closely associated with metabolic risk indicators, such as dyslipidemia and insulin resistance, in euthyroid adults (47). This finding complements our results by suggesting that the observed relationship between TSH and BMI may extend beyond overt hypothyroidism and play a role in subclinical thyroid states as well. Moreover, Ríos-Prego et al. emphasized that the paradigm linking thyroid dysfunction with body weight is not always straightforward, noting that some obese individuals may maintain normal thyroid function despite significant adiposity (29). Our data, however, provide evidence that in the majority of cases, higher TSH values are strongly predictive of elevated BMI, particularly in women, who were overrepresented in the hypothyroid group, a trend also supported by Ahmed et al. (3).

The mechanisms underlying this association are multifaceted. Hypothyroidism reduces thermogenesis, impairs lipid and carbohydrate metabolism, and leads to fluid retention, all of which contribute to weight gain. At the molecular level, thyroid hormones influence the expression of genes involved in mitochondrial energy metabolism and glucose homeostasis (10, 24). In addition, TSH receptors expressed in adipose tissue can directly stimulate adipogenesis, providing another explanation for the strong correlation between elevated TSH and obesity (9). These mechanisms highlight how even modest alterations in thyroid function can contribute to substantial metabolic consequences. From a clinical perspective, the strength of the observed association underscores the need for routine screening of thyroid function in individuals presenting with obesity, particularly those with unexplained weight gain or metabolic syndrome. Early identification and management of hypothyroidism could potentially mitigate obesity-related complications and improve metabolic outcomes. Our findings also point to the importance of considering thyroid function when developing weight management strategies, as untreated thyroid dysfunction may undermine the effectiveness of lifestyle or pharmacological interventions targeting obesity.

This study has several strengths, including the use of standardized laboratory assays and objective anthropometric measurements, as well as the inclusion of both patient and control groups for comparison. The robustness of the findings, confirmed by both parametric and non-parametric analyses, strengthens the reliability of the observed association. However, some limitations must be acknowledged. The relatively small sample size restricts the statistical power and limits the generalizability of results to broader populations. The cross-sectional design precludes causal inferences, and residual confounding by unmeasured variables such as diet, physical activity, and socioeconomic status cannot be excluded. Furthermore, the study population was drawn from a single geographic location, which may reduce external validity. Future research should focus on larger, multicenter cohorts to confirm these associations and explore their applicability across different ethnic and age groups. Longitudinal studies are particularly needed to determine causality and to assess whether effective treatment of hypothyroidism translates into measurable improvements in BMI and related metabolic outcomes. In addition, mechanistic studies exploring the interplay between leptin, TSH, and thyroid hormone receptors in adipose tissue could provide deeper insights into the biological basis of this association. In conclusion, this study provides

strong evidence that hypothyroidism is significantly associated with increased BMI, with elevated serum TSH serving as both a marker and mediator of obesity risk. The strength of the correlation suggests that thyroid function plays a central role in body weight regulation, reinforcing the importance of integrating thyroid screening into the evaluation of overweight and obese individuals. Addressing thyroid dysfunction may therefore represent an important step in mitigating the growing burden of obesity and its associated comorbidities.

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