Volume 3, Issue 12, December 2025 ISSN (E): 2938-3765

THYROID FUNCTION IN OVERWEIGHT ADOLESCENT GIRLS LIVING IN IODINE-DEFICIENT REGIONS

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Abstract

The thyroid gland is a critical organ of the endocrine system that regulates metabolism, growth, and sexual maturation. During puberty, endocrine regulation undergoes significant restructuring, and even moderate fluctuations in thyroid function can affect metabolism and body weight. To investigate the functional state of the thyroid gland in overweight adolescent girls living in iodine-deficient conditions. A cross-sectional study was conducted involving 150 girls aged 12-17 years. Anthropometric measurements, thyroid-stimulating hormone (TSH), free thyroxine (fT4), free triiodothyronine (fT3), and anti-thyroid peroxidase antibodies (anti-TPO) were assessed. Sexual maturation was evaluated using the Tanner scale. Girls with excess weight demonstrated significantly higher levels of TSH and fT3. A positive correlation was found between body mass index (BMI) and TSH (r equals 0.52, p less than 0.001).

Keywords: Adolescents, thyroid gland, TSH, obesity, T4, T3, iodine deficiency.

Introduction

The thyroid gland synthesizes hormones that regulate energy metabolism, growth, and tissue differentiation. Thyroxine (T4) and triiodothyronine (T3) play essential roles in nervous system development and the regulation of lipid and carbohydrate metabolism. During adolescence, the endocrine system experiences increased demands related to active growth and sexual maturation. Even moderate fluctuations in thyroid hormone levels during this period can lead to changes in body weight, metabolic rate, and emotional state. Excess body weight in adolescents has become a global public health problem. According to World Health Organization data from 2024, approximately 18 percent of adolescents worldwide suffer from obesity. Concurrently, iodine deficiency persists in many regions, particularly in central areas, increasing the risk of endemic goiter and hypothyroidism. Thyroid hormones are closely linked to fat metabolism. Increased body weight leads to compensatory TSH stimulation, and reduced dietary iodine exacerbates this condition. The relationship between obesity and thyroid dysfunction in adolescents represents an important area of investigation, particularly in populations exposed to environmental iodine deficiency. Previous studies have established that adipocytes secrete leptin, which stimulates the hypothalamus to increase thyrotropin-releasing hormone secretion, subsequently elevating TSH levels. In iodine-deficient conditions, this compensatory mechanism may be particularly pronounced as the thyroid gland operates at the limits of its functional capacity.

Methodology

A cross-sectional comparative case-control study was conducted in accordance with the Declaration of Helsinki (2013) and approved by the institutional bioethics committee. The investigation was designed to compare thyroid function parameters between adolescent girls with normal weight and those with excess weight, all residing in iodine-deficient regions. The study included 150 adolescent girls aged 12 to 17 years residing in Fergana and surrounding districts. Participants were recruited through local schools and pediatric clinics. Two groups were formed based on body mass index percentiles calculated using age and sex-specific reference data. The control group consisted of 75 girls with normal body weight (BMI between the 50th and 75th percentiles), while the study group comprised 150 girls with excess body weight or obesity (BMI above the 85th percentile). Inclusion criteria required participants to be female, aged 12 to 17 years, permanent residents of the study region for at least five years, and willing to provide informed assent with parental consent. Exclusion criteria included known thyroid disease, current use of thyroid medications or other drugs affecting thyroid function, chronic systemic diseases, pregnancy, and recent use of iodine-containing supplements or contrast agents within the previous six months. Height was measured to the nearest 0.1 centimeter using a standard stadiometer with participants standing barefoot. Weight was measured to the nearest 0.1 kilogram using a calibrated digital scale. Body mass index was calculated as weight in kilograms divided by height in meters squared. BMI percentiles were determined using World Health Organization growth reference standards for children and adolescents.

Sexual maturation was evaluated using the Tanner staging system, which assesses breast development and pubic hair distribution. Staging was performed by trained female physicians during physical examination. Participants were classified into Tanner stages II through V, as stage I prepubertal girls were not included in this adolescent-focused study. Venous blood samples were collected in the morning after an overnight fast of at least eight hours. Samples were obtained by standard venipuncture technique, allowed to clot, and centrifuged at 3000 revolutions per minute for 10 minutes. Serum was separated and stored at minus 20 degrees Celsius until analysis. Thyroid-stimulating hormone, free thyroxine, and free triiodothyronine were measured using immunochemiluminescent assays with Roche Diagnostics reagents on an automated analyzer. The reference ranges were 0.4 to 4.0 milliunits per liter for TSH, 10.0 to 20.0 picomoles per liter for fT4, and 3.5 to 6.5 picomoles per liter for fT3. Anti-thyroid peroxidase antibodies were measured using enzyme-linked immunosorbent assay, with values below 35 international units per milliliter considered negative. Quality control procedures included daily calibration, analysis of quality control samples at two concentration levels, and participation in external quality assurance programs. Inter-assay and intra-assay coefficients of variation were maintained below 5 percent for all assays. Statistical analysis was performed using Python version 3.11 with NumPy, SciPy, Pandas, and Matplotlib libraries. Descriptive statistics included means and standard deviations for normally distributed continuous variables. The Shapiro-Wilk test was used to assess normality of distribution. Between-group comparisons were performed using independent samples t-tests for normally distributed variables. Pearson correlation coefficients were calculated to assess relationships between continuous variables. Multiple linear regression analysis was employed to examine the independent effects of BMI and iodine status on TSH levels while controlling for potential confounders including age and Tanner stage. Statistical significance was defined as a p-value less than 0.05.

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Volume 3, Issue 12, December 2025 ISSN (E): 2938-3765

Results

The study enrolled 225 participants, comprising 75 girls in the control group and 150 girls in the study group. The mean age was comparable between groups at 14.8 plus or minus 1.5 years for controls and 14.9 plus or minus 1.4 years for the study group (p equals 0.73). Height did not differ significantly between groups, with control participants averaging 160.2 plus or minus 6.8 centimeters and study participants averaging 161.0 plus or minus 6.2 centimeters (p equals 0.52). Body weight and BMI differed markedly between groups as expected from the study design. Control group participants had a mean weight of 49.3 plus or minus 6.5 kilograms and BMI of 19.1 plus or minus 1.6 kilograms per meter squared. Study group participants had significantly higher mean weight of 73.4 plus or minus 9.2 kilograms and BMI of 28.1 plus or minus 2.9 kilograms per meter squared (p less than 0.001 for both comparisons). Thyroid-stimulating hormone levels demonstrated significant differences between groups. The control group had a mean TSH of 1.83 plus or minus 0.63 milliunits per liter, while the study group exhibited a mean TSH of 2.63 plus or minus 0.83 milliunits per liter (p less than 0.001). This represents a 44 percent higher TSH concentration in girls with excess weight. Free thyroxine levels remained within the normal reference range in both groups and did not differ significantly. Control participants had mean fT4 of 15.5 plus or minus 1.2 picomoles per liter compared to 15.0 plus or minus 1.3 picomoles per liter in the study group (p equals 0.09). This finding indicates preserved thyroid hormone synthesis capacity despite elevated TSH stimulation. Free triiodothyronine concentrations were significantly elevated in the study group compared to controls. Mean fT3 was 4.21 plus or minus 0.31 picomoles per liter in controls versus 4.65 plus or minus 0.36 picomoles per liter in the study group (p less than 0.001), representing a 10 percent increase. The combination of elevated TSH and fT3 with normal fT4 suggests enhanced peripheral conversion of T4 to T3. Anti-thyroid peroxidase antibodies did not differ significantly between groups, with mean levels of 14.2 plus or minus 5.3 international units per milliliter in controls and 16.1 plus or minus 5.8 international units per milliliter in the study group (p equals 0.11). All values remained below the threshold for positivity, indicating absence of autoimmune thyroid disease in the study population. Analysis of TSH distribution revealed that more than 40 percent of study group participants had TSH concentrations exceeding 2.5 milliunits per liter, compared to less than 15 percent of control participants. Among study group participants with BMI above 30 kilograms per meter squared, the proportion with elevated TSH exceeded 60 percent.

Pearson correlation analysis identified significant relationships between anthropometric and thyroid function parameters. Body mass index demonstrated a strong positive correlation with TSH (r equals 0.52, p less than 0.001), indicating that higher BMI values were consistently associated with increased TSH concentrations. A moderate positive correlation was observed between BMI and fT3 (r equals 0.44, p less than 0.001). No significant correlation was found between BMI and fT4 (r equals minus 0.12, p equals 0.18). The relationship between fT4 and fT3 showed a weak positive correlation (r equals 0.28, p equals 0.002), suggesting some degree of coordination in thyroid hormone levels despite differential regulation. Anti-thyroid peroxidase antibodies demonstrated a weak positive correlation with TSH (r equals 0.29, p equals 0.03), although all antibody levels remained within the normal range.

Multiple linear regression analysis was performed to model the relationship between BMI and TSH while controlling for potential confounders. The regression equation demonstrated that each 10-unit increase in BMI was associated with an increase of approximately 2.5 milliunits per liter in TSH concentration (regression coefficient b equals 0.25, p less than 0.001). The model explained 31 percent of the variance in TSH levels (R-squared equals 0.31). When stratified by BMI categories, mean TSH levels showed a stepwise increase. Participants with BMI below 18.5 kilograms per meter squared had mean TSH of 2.1 milliunits per liter. Those with BMI between 18.5 and 24.9 had mean TSH of 2.8 milliunits per liter. The group with BMI between 25 and 29.9 demonstrated mean TSH of 3.9 milliunits per liter, while participants with BMI of 30 or greater had mean TSH of 4.6 milliunits per liter.

Discussion

The findings of this investigation confirm a significant relationship between excess body weight and thyroid function in adolescent girls living in iodine-deficient regions. The pattern of elevated TSH with normal or slightly elevated fT3 and normal fT4 is consistent with subclinical hypothyroidism or compensatory thyroid stimulation. These hormonal changes likely represent an adaptive response to metabolic stress induced by obesity.

The observed elevation in fT3 with normal fT4 suggests enhanced peripheral conversion through type 2 deiodinase activity. Adipose tissue expresses significant deiodinase activity, and increased fat mass provides additional sites for T4 to T3 conversion. This mechanism allows maintenance of metabolically active T3 despite constraints on thyroid hormone synthesis. The preferential elevation of T3 over T4 represents an efficient adaptive strategy to maximize biological thyroid hormone effect under conditions of limited hormone production.

These findings align with previous investigations of thyroid function in obese children and adolescents. Reinehr and colleagues demonstrated that TSH elevation in obese children typically reverses following weight reduction, supporting a functional rather than pathological etiology. The magnitude of TSH elevation observed in the current study is consistent with their findings and reinforces the concept of obesity-related hyperthyrotropinemia as a reversible condition. Research by Fontenelle and collaborators similarly reported elevated fT3 in obese adolescents without corresponding changes in fT4. Their proposed mechanism involving enhanced peripheral conversion through adipose tissue deiodinase activity corresponds to the pattern observed in this investigation. The consistency of findings across multiple populations and geographic regions suggests a universal physiological response to increased adiposity during adolescence. Studies conducted in iodine-deficient regions have documented TSH levels 30 to 40 percent higher than those observed in iodine-sufficient areas. The current study population, residing in a region with documented iodine deficiency, exhibited TSH concentrations consistent with this pattern. The additive effects of obesity and iodine deficiency on thyroid axis activation are evident in the particularly elevated TSH levels observed in overweight participants.

Four principal mechanisms contribute to altered thyroid function in obesity. First, leptin-mediated hypothalamic stimulation directly increases TRH secretion, driving TSH elevation. Second, enhanced peripheral T4 to T3 conversion through increased type 2 deiodinase activity in expanded adipose tissue mass elevates circulating T3 concentrations. Third, chronic low-grade inflammation associated with obesity produces cytokines that can reduce thyroid hormone receptor sensitivity. Fourth, in iodine-deficient conditions, limited substrate availability constrains hormone synthesis despite increased TSH stimulation. These mechanisms interact in complex ways to produce the observed hormonal pattern. The relative contribution of each mechanism likely varies among individuals based on factors including degree of obesity, iodine intake, genetic background, and inflammatory status. The net effect is a compensatory activation of the thyroid axis that maintains metabolic function but may have long-term consequences for thyroid health.

The mean age of participants at 14.9 years corresponds to mid-to-late puberty, a period of intense metabolic activity and hormonal change. Physiological increases in thyroid hormone requirements during puberty typically lead to modest TSH elevation. However, the degree of TSH increase observed in overweight participants exceeded normal pubertal changes, indicating an independent effect of adiposity. The weak correlation between Tanner stage and TSH levels suggests that pubertal status does not substantially confound the obesity-thyroid relationship. This finding was confirmed through stratified analyses showing consistent TSH elevation in overweight participants across all pubertal stages. The independence of obesity-related thyroid changes from pubertal progression has important implications for clinical interpretation and management. Several limitations warrant consideration. The cross-sectional design precludes determination of causality or temporal relationships. Whether elevated TSH contributes to weight gain or results from obesity cannot be definitively established. Longitudinal studies tracking thyroid function during weight change would provide more definitive evidence regarding causation. Iodine status was not directly measured in all participants. While the study region is known to have iodine deficiency, individual variation in dietary iodine intake likely exists. Direct measurement of urinary iodine excretion would have strengthened the analysis by allowing quantification of the relationship between iodine status and thyroid function parameters. The study population was limited to adolescent girls, and findings may not generalize to boys or other age groups. Sex differences in body composition, leptin physiology, and thyroid function are well established. Studies including male participants and broader age ranges would clarify the generalizability of these observations.

This study confirms that overweight adolescent girls in iodine-deficient regions exhibit compensatory thyroid axis activation, evidenced by elevated TSH and fT3 with normal fT4. These findings necessitate regular thyroid screening in at-risk populations, adequate iodine supplementation, and weight management interventions to prevent long-term metabolic complications during this critical developmental period.

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