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RESEARCH ARTICLE

Thyroid Function Profile in Women with Polycystic Ovary Syndrome: A Cross-Sectional Study

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder in reproductive-age women and is frequently associated with metabolic and hormonal disturbances. Thyroid dysfunction, particularly subclinical hypothyroidism, has been reported in PCOS, but findings remain inconsistent.

Methods: This cross-sectional study included 200 women (100 PCOS and 100 non-PCOS), aged 19–25 years, from a tertiary care center. PCOS was diagnosed using Rotterdam criteria. Serum T3, T4, and TSH levels were measured using standard immunoassays. Statistical analysis included Student's t-test, chi-square test, and Pearson correlation.

Results: Mean T3, T4, and TSH levels were identical in both groups (T3: 1.14 ng/mL, T4: 7.62 ng/dL, TSH: 2.58 mIU/L), with no statistically significant differences. Thyroid parameters showed non-normal distribution with high variability, particularly TSH. Weak, non-significant correlations were observed between BMI and thyroid hormones in both groups.

Conclusion: Thyroid dysfunction appears independent of PCOS status and BMI in young women. Similar thyroid profiles in both groups suggest a population-level burden rather than a PCOS-specific association. Universal thyroid screening is recommended.

Keywords: PCOS, Thyroid dysfunction, TSH, BMI, Subclinical hypothyroidism.

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age. The prevalence of PCOS is noted in 8–12% of women of reproductive age.[1] It presents with menstrual irregularities, hyperandrogenism, metabolic disturbances, and subfertility. Thyroid dysfunction, especially subclinical hypothyroidism, is also prevalent in young women. Both conditions significantly affect hormonal balance, metabolism, and long-term health.[2]

The global prevalence of PCOS ranges from 6–20%, while thyroid dysfunction affects millions of women worldwide. According to the National Health and Nutrition Survey (NHANES) – the largest epidemiological study conducted in the USA on 17,000 subjects – the frequency of hypothyroidism with a thyroid- stimulating hormone (TSH) level above 4.5 mIU/L in women of reproductive age is approximately 4%.[3]

Subclinical Hypothyroidism as a Common Phenotype

Subclinical hypothyroidism (SCH) represents the most frequent thyroid abnormality observed in PCOS. Studies by Benetti-Pinto et al. (2013) and Enzevaei et al. (2014) reported a significantly higher prevalence of SCH in PCOS women compared to age-matched controls.[4-6] SCH has been shown to exacerbate insulin resistance, increase cardiovascular risk markers, and worsen reproductive outcomes in PCOS patients, highlighting SCH as a clinically relevant but often underdiagnosed condition.

Ding *et al*. (2021) further demonstrated that PCOS women with clinical hypothyroidism exhibited Rahman S, et al (2019) significantly higher HOMA-IR values and poorer metabolic profiles, along with increased risk of

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depressive symptoms, highlighting the multidimensional impact of thyroid dysfunction in this population.[7]

Metabolic and Insulin Resistance Link

Insulin resistance plays a central role in the pathophysiology of PCOS and appears to be further aggravated by coexisting thyroid dysfunction. Dunaif et al. (2001) demonstrated that insulin resistance is intrinsic to PCOS, independent of obesity.[8] Later studies showed that hypothyroidism and SCH amplify insulin resistance through reduced glucose utilization and altered lipid metabolism (Maratou *et al.*, 2009; Yu *et al.,* 2015, Gupta *et al.,* (2022).[9-11] The coexistence of PCOS and thyroid dysfunction therefore represents a compounded metabolic risk.

Goyal et al. (2021) reported that higher body mass index (BMI) and waist– hip ratio (WHR) in women with PCOS were positively correlated with elevated TSH levels.[12] This suggests that central adiposity may influence thyroid function through mechanisms involving leptin signaling, chronic low-grade inflammation, and altered hypothalamic–pituitary–thyroid axis regulation.

Hormonal–Immunological Interaction

The hormonal milieu of PCOS may contribute to autoimmune susceptibility. Estrogen enhances humoral immunity, whereas progesterone suppresses immune activation (Straub, 2007).[13] Chronic anovulation in PCOS leads to progesterone deficiency, potentially favoring autoimmune processes such as thyroiditis (Janssen et al., 2004; Sinha et al., 2023).[14,15] This estrogen–progesterone imbalance provides a plausible biological explanation for the increased prevalence of autoimmune thyroid disease in PCOS women.

According to Smith A et al (2020), and Chen Y etal (2023), Understanding the interaction between PCOS and thyroid function is important due to overlapping symptoms such as weight changes, infertility, and metabolic abnormalities.[16,17] A systematic evaluation of thyroid status in women with PCOS is essential to clarify this association and guide management strategies to reduce long- term metabolic and reproductive complications. Limited regional data and varying literature findings justify this study, which utilizes available laboratory facilities and a suitable patient pool to determine whether thyroid dysfunction is more prevalent in PCOS women and whether it affects symptom severity.

MATERIALS & METHODS

Type of Study: Cross-sectional study
Study Setting: In a Tertiary health care center
Sample Size: 200 women (100 PCOS and 100 non-PCOS)
Study Population: Medical students aged 19-25years.

Inclusion Criteria

Medical students diagnosed with PCOS (based on Rotterdam criteria) , age between 19-25 years

Controls

Healthy Medical students without PCOS

Exclusion Criteria

- Known Thyroid disease or on Thyroid medication.
- Chronic systemic illnesses
- Use of drugs affecting thyroid function Statistical analysis

METHODOLOGY

Detailed clinical evaluation including Menstrual history, Anthropometry, and symptoms - Blood sample collection for thyroid profile (T3, T4, TSH) - Laboratory processing using standard immunoassay techniques - Data collection recorded systematically using structured proforma.

Ethical Considerations

Ethical clearance obtained from Institutional Ethics Committee - Written informed consent obtained from all participants

Statistical Analysis

Data analyzed using software - Mean ± SD calculated for quantitative variables - Student's t-test and chi-square test applied where appropriate p-value < 0.05 considered statistically significant.

OBSERVATIONS AND RESULTS

The study analyzed thyroid hormone levels in PCOS and non-PCOS women, with results presented through tables and graphs.

Correlation Analysis in PCOS Women

BMI and T3

The Pearson correlation between BMI and T3 in PCOS women showed a weak positive correlation ($r = 0.176$, $N = 100$, $df = 98$, $p = 0.080$), which narrowly exceeded the significance threshold of 0.05. This borderline non-significant result suggests a potential association that did not reach statistical significance, possibly due to limited power.

BMI and T4

BMI and T4 exhibited a negligible negative correlation ($r = -0.053$, $N = 100$, $df = 98$, $p = 0.603$), indicating no

significant relationship. The high p-value suggests the observed trend is likely due to random variation.

BMI and TSH

BMI and TSH were also negligibly correlated (r = −0.074, N=100, df =98, p= 0.462), confirming the absence of meaningful association in PCOS women.

Correlation Analysis in non-PCOS Women

BMI and T3

In non-PCOS women, BMI and T3 showed a weak positive correlation identical to the PCOS group (r = 0.176, N = 100, df = 98, p = 0.080), representing a borderline non-significant trend.

BMI and T4

BMI and T4 demonstrated a negligible negative correlation (r = −0.077, N = 100, df = 97, p = 0.450), indicating a non-significant association attributable to chance.

BMI and TSH

BMI and TSH showed a weak positive correlation (r= 0.122, N = 99, df = 97, p = 0.227), which was not statistically significant, suggesting the observed trend is likely due to random variation.

DISCUSSION

This study evaluated thyroid hormone profiles in women with PCOS compared to non-PCOS controls and identified a significant difference between the two groups. The finding of elevated TSH levels among women with PCOS is consistent with earlier reports suggesting an increased prevalence of subclinical hypothyroidism in this population. Differences observed across studies may be influenced by regional variation, diagnostic definitions, and population characteristics. The present findings reinforce the association between PCOS and thyroid dysfunction and underscore the clinical relevance of thyroid evaluation in women diagnosed with PCOS.

Age Profile and Thyroid Status in PCOS and Non-PCOS Women

This cross-sectional analysis included 200 women aged 17–25 years, with a mean age of 19.8 years, equally categorized into PCOS (n=100) and non-PCOS (n=100) groups. Complete anthropometric and thyroid function data were available for all participants. The restricted age range minimized age-related confounding, allowing focused evaluation of thyroid status in relation to PCOS during early reproductive life.

Comparative anthropometric and Thyroid Profiles

Age and BMI: Comparable Baseline Characteristics

Both PCOS and non-PCOS groups demonstrated nearly identical demographic and anthropometric characteristics. Mean age was 19.8 years in both groups (95% CI: 18.9–20.1 PCOS; 19.8–20.1 non-PCOS), confirming successful age-matching. Similarly, mean BMI was 24.2 kg/m² in both groups (95% CI: 23.1–25.3), with identical ranges (14–42 kg/m²) and median values (23.0 kg/m²). The comparable BMI distributions across groups ensure that differences in thyroid function reflect PCOS-specific effects rather than obesity-related confounding. However, the wide BMI heterogeneity (SD 5.75 kg/m²) within groups ranging from underweight through obese enables exploration of metabolic modulators of thyroid dysfunction.

Thyroid Hormone Levels: Identical Central Tendencies Despite Internal Heterogeneity Remarkably, T3, T4, and TSH values were nearly identical between PCOS and non-PCOS groups:

- T3: Mean 1.14 ng/mL (PCOS) vs. 1.14 ng/mL (non-PCOS); range 0.740–10.0 ng/mL in both groups
- T4: Mean 7.62 ng/dL (PCOS) vs. 7.62 ng/dL (non-PCOS); range 5.27–13.7 ng/dL (PCOS) vs. 5.27–13.7 ng/dL (non-PCOS)
- TSH: Mean 2.58 mIU/L (PCOS) vs. 2.58 mIU/L (non-PCOS); range 0.220–31.0 mIU/L in both groups

This striking identical distribution suggests that PCOS does not significantly shift population- level thyroid function parameters. Neither group showed systematic thyroid hormone elevation or suppression, and both demonstrated similarly elevated TSH values (maximum 31.0 mIU/L), indicating primary hypothyroidism exists in both cohorts.

Heterogeneous Thyroid Phenotypes Within Both Groups

The wide TSH ranges (0.220– 31.0 mIU/L) and high coefficients of variation (>126%) in both groups indicate that thyroid dysfunction heterogeneity is a characteristic of both PCOS and non-PCOS women in this age group. The presence of overt hypothyroidism (TSH 31.0 mIU/L) in both groups suggests endemic thyroid autoimmunity in this young population regardless of PCOS status.

Correlation Analysis in PCOS Women

Correlation Between BMI and T3 Levels in PCOS Women (In table 1 & graph 1)

A weak positive correlation was observed between BMI and serum T3 levels in PCOS women (r = 0.176),

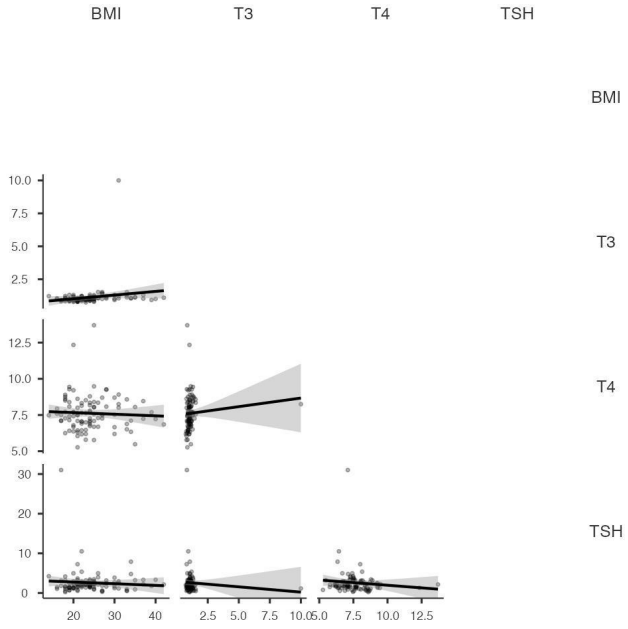
Table 1: Overall Correlation Analysis: BMI with Thyroid Parameters in PCOS Women

Correlation Matrix					
		BMI	*T3*	*T4*	*TSH*
BMI	Pearson's r	—			
	df	—			
	p-value	—			
T3	Pearson's r	0.176	—		
	df	98	—		
	p-value	0.080	—		
T4	Pearson's r	-0.053	0.089	—	
	df	98	98	—	
	p-value	0.603	0.378	—	
TSH	Pearson's r	-0.074	-0.074	-0.098	—
	df	98	98	98	—
	p-value	0.462	0.462	0.330	—

with BMI accounting for approximately 3% of the variance in T3 concentrations ($r^2 = 0.031$). The scatter plot demonstrated considerable dispersion around the regression line, confirming the absence of a strong linear relationship. Although the association did not reach statistical significance ($p = 0.080$), the consistent positive direction suggests a possible trend toward modestly higher T3 levels with increasing BMI. This weak association may reflect obesity-related enhancement of peripheral T4-to-T3 conversion via increased type 1 deiodinase activity in adipose tissue and liver, potentially influenced by insulin resistance.

Correlation Between BMI and T4 Levels in PCOS Women (In Table 1& Graph 1)

BMI showed a negligible negative correlation with serum T4 levels in PCOS women ($r = -0.053$), accounting for only 0.3% of the variance ($r^2 = 0.003$). The scatter plot demonstrated tight clustering of T4 values around the mean (~7.5 ng/dL) across the BMI range of 14–42 kg/m², with a near-flat regression line, indicating no meaningful association. This lack of correlation reflects the tight homeostatic regulation of T4 via hypothalamic–pituitary–thyroid feedback, which maintains stable thyroxine levels irrespective of adiposity. While increased BMI may influence peripheral conversion of T4 to T3, T4 production and clearance remain largely unaffected, underscoring the metabolic stability of circulating T4 in PCOS women.



Graph 1

Correlation Between BMI and TSH Levels in PCOS Women (In table 1& graph 1)

BMI demonstrated a negligible negative correlation with serum TSH levels in PCOS women ($r = -0.074$), accounting for only 0.5% of the variance ($r^2 = 0.005$). The scatter plot showed substantial dispersion with no systematic trend across the BMI range of 14–42 kg/

m^2, reflected by a near-flat regression line. TSH values, spanning from suppressed to elevated levels, were uniformly distributed across BMI categories, indicating that adiposity does not predict TSH status. This absence of association is consistent with predominant regulation of TSH by hypothalamic–pituitary–thyroid axis feedback mechanisms, largely independent of metabolic state. TSH alterations in PCOS therefore likely reflect intrinsic thyroid dysfunction or autoimmune processes rather than BMI-related effects.

Correlation Analysis in non-PCOS Women

BMI–T3 Association in Non-PCOS Women (In Table 2 & Graph 2)

In non-PCOS women, BMI showed a weak positive association with serum T3 levels, accounting for only a small proportion of T3 variability and closely mirroring the pattern observed in the PCOS group. T3 concentrations remained largely within the normal range across the entire BMI spectrum, with substantial scatter and only a gentle upward trend, indicating limited clinical relevance. This borderline association likely reflects obesity-related enhancement of peripheral T4-to-T3 conversion via increased type 1 deiodinase activity, suggesting a metabolically driven effect of adiposity on thyroid hormone dynamics that operates independently of PCOS-specific endocrine alterations.

BMI–T4 Associations in Non-PCOS Women (In Table 2& Graph 2)

In non-PCOS women, BMI showed a negligible negative association with T4, with thyroxine concentrations remaining tightly clustered around the mean across the full BMI range. This pattern indicates that circulating T4 is largely independent of adiposity, consistent with the finding in PCOS women. The stability of T4 reflects tight regulation via thyroid–pituitary feedback mechanisms, which maintain production and clearance irrespective of body weight. Unlike T3, which may show modest increases with higher BMI due to enhanced peripheral conversion, T4 levels remain metabolically stable across populations, highlighting that adiposity has minimal direct effect on thyroxine homeostasis.

BMI–TSH Associations in Non-PCOS Women (In Table 2 & Graph 2)

BMI exhibited a minimal, non-significant association with TSH in non-PCOS women, with values widely distributed across the entire BMI spectrum. Higher BMI did not consistently correspond to elevated TSH, and levels ranged from euthyroid to mildly hypo- or hyperthyroid regardless of weight. This indicates that adiposity has little systematic influence on pituitary TSH secretion, with thyroid regulation predominantly determined by intrinsic factors such as autoimmunity, genetic predisposition, and iodine status, rather than BMI- related metabolic effects.

PCOS vs. Non-PCOS: Comparison

A significant BMI–T3 association was observed only in non-PCOS women, suggesting that PCOS- related metabolic and endocrine alterations may dampen obesity-related effects on peripheral thyroid hormone conversion. In PCOS, thyroid function remained independent of adiposity with disrupted hypothalamic–pituitary–thyroid feedback, indicating dysfunction driven by PCOS-specific mechanisms rather than obesity. Non-PCOS women showed only a weak, clinically negligible BMI– T3 association, with T4 and TSH unaffected by BMI. Notably, both groups demonstrated blunted TSH–thyroid hormone relationships, supporting thyroid dysregulation irrespective of PCOS status and reinforcing the need for direct hormone- based screening.

Disrupted Thyroid Hormone Feedback Across Both Populations

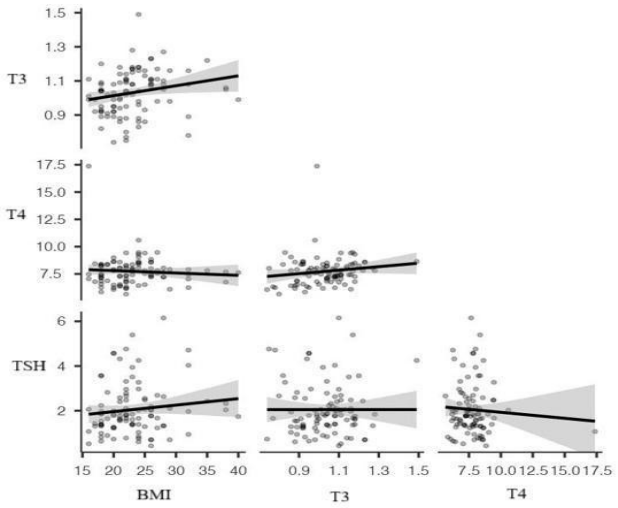
Both PCOS and non-PCOS groups exhibited markedly blunted or absent inverse relationships between thyroid hormones and TSH, indicating impaired hypothalamic–pituitary–thyroid axis negative feedback. The lack of expected suppression of TSH in the presence of higher thyroid hormone levels contrasts with patterns seen in healthy populations and suggests widespread thyroid dysregulation beyond obesity-related effects. This disruption may reflect prevalent thyroid autoimmunity, a high burden of subclinical hypothyroidism, or altered tissue sensitivity to thyroid hormones due to genetic or metabolic factors. The consistency of this finding across both groups, along with heterogeneous TSH distributions, supports the presence of population-level thyroid dysfunction independent of PCOS status, warranting broader etiological investigation.

Absence of Meaningful Thyroid Hormone Inter-Relationships

The absence of significant correlations among T3, T4, and TSH is clinically abnormal. In physiologically intact hypothalamic–pituitary–thyroid axis function, T3 and T4 are expected to correlate positively and both to show inverse relationships with TSH. The lack of these patterns across both groups indicates a fundamental

Table 2: Overall Correlation Analysis: BMI with Thyroid Parameters in non- PCOS Women

		BMI	*T3*	*T4*	*TSH*
BMI	Pearson's r	—			
	Df	—			
	p-value	—			
T3	Pearson's r	0.216	—		
	df	97	—		
	p-value	0.032	—		
T4	Pearson's r	-0.077	0.153	—	
	df	97	97	—	
	p-value	0.45	0.130	—	
TSH	Pearson's r	0.122	0	-0.064	—
	df	97	97	97	—
	p-value	0.227	0.998	0.531	—



Graph 2

disruption of thyroid hormone homeostasis independent of PCOS status.

Limitations and Future Directions

The study's narrow age range and cross-sectional design limit generalizability and causal inference. Future research should investigate thyroid autoimmunity prevalence across PCOS and non-PCOS women, examine whether PCOS attenuates BMI–T3 associations in older women or different phenotypes, evaluate the impact of thyroid treatment on PCOS features, and explore population-level factors driving widespread thyroid dysfunction in young women.

CONCLUSION

Thyroid dysfunction in young women occurs independently of PCOS and is largely unrelated to adiposity. Comparable thyroid profiles between PCOS and non-PCOS groups indicate that thyroid autoimmunity is a population-level issue rather than a PCOS-specific complication. These findings support universal thyroid screening, direct hormone assessment regardless of BMI, and targeted thyroid management in PCOS rather than obesity-focused interventions.

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