

CORRELATION BETWEEN DAILY CAFFEINE INTAKE AND THYROID FUNCTION: A CROSS-SECTIONAL STUDY IN UNIVERSITY STUDENTS

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Abstract

Background: Caffeine is the most widely consumed pharmacologically active compound among university students, used extensively to mitigate academic stress and sleep deprivation. However, its direct implications on the hypothalamic-pituitary-thyroid (HPT) axis and central endocrine pathways remain unclear.

Objective: To investigate the relationship between daily caffeine consumption and thyroid function parameters—specifically Serum Thyroid-Stimulating Hormone (TSH), Free Triiodothyronine (FT3), and Free Thyroxine (FT4) levels—in a university cohort.

Methods: A cross-sectional study was conducted with 40 healthy participants presenting an equal gender distribution (50% male, 50% female; mean age: 36.5 ± 12.4 years). Habitual dietary caffeine intake was quantified using a validated Food Frequency Questionnaire (FFQ), stratifying subjects into Low (0–100 mg/day, $n=13$), Moderate (101–300 mg/day, $n=14$), and High (>300 mg/day, $n=13$) exposure tiers. Fasting morning venous blood samples were analyzed via Chemiluminescence Immunoassay (CLIA) techniques, and statistical analyses were performed using Pearson's correlation coefficient (r).

Results: The mean daily caffeine intake across the entire cohort was 214 ± 132 mg/day. A statistically significant, moderate-to-strong positive correlation was identified between daily caffeine consumption and serum TSH levels ($r = +0.68$, $p < 0.05$). Mean TSH advanced in a clear stepwise manner from 2.0 ± 0.2 mIU/L in the low group to 4.3 ± 0.2 mIU/L in the high group. Conversely, peripheral active thyroid fractions showed negligible, non-significant (NS) correlations with caffeine intake, with FT3 demonstrating a weak positive trend ($r = +0.12$, $p > 0.05$) and FT4 exhibiting a minor negative trend ($r = -0.09$, $p > 0.05$). Secondary gender stratification revealed higher mean TSH in males (3.4 mIU/L) than females

(3.0 mIU/L), matching a higher baseline caffeine exposure among male participants (235 mg/day vs. 193 mg/day).

Conclusion: Daily caffeine consumption significantly correlates with elevated central pituitary TSH secretion in a dose-dependent framework. However, because peripheral hormones (FT3 and FT4) remain highly regulated within normal physiological bounds, this relationship represents a benign homeostatic adaptation rather than a clinical pathway toward thyroid disease.

1. INTRODUCTION:

1.1 The Global Prevalence of Caffeine Consumption in Academia

Caffeine (1,3,7-trimethylxanthine) is recognized as the most frequently ingested pharmacologically active compound worldwide. In the context of higher education, caffeine consumption has transitioned from a dietary habit to a functional necessity. University students face a unique set of stressors, including rigorous academic schedules, sleep deprivation, and the physiological demands of late-adolescence/early-adulthood development. Recent studies indicate that over 90% of university students consume caffeine daily, often in concentrations exceeding the recommended 400mg threshold set by the FDA (Reissig et al., 2019; Richards & Smith, 2015).

While the ergogenic and cognitive-enhancing effects of caffeine—such as improved alertness and reduced perceived exertion—are well-documented, the systemic endocrine implications are less understood. The thyroid gland, the primary regulator of basal metabolic rate (BMR), is particularly sensitive to exogenous stimulants. As students increasingly turn to concentrated sources of caffeine, such as energy drinks and anhydrous caffeine pills, the potential for thyroid dysregulation becomes a critical public health concern (Al-Subaie, 2022).

1.2 The Hypothalamic-Pituitary-Thyroid (HPT) Axis and Metabolic Homeostasis

The HPT axis is a tightly regulated feedback loop designed to maintain homeostatic levels of thyroid hormones. The hypothalamus secretes Thyrotropin-Releasing Hormone (TRH), which stimulates the anterior pituitary to release Thyroid-Stimulating Hormone (TSH). TSH then acts upon

the thyroid gland to produce Thyroxine (T4) and Triiodothyronine (T3).

Any disruption in this loop, whether through dietary or environmental factors, can lead to subclinical hypo- or hyperthyroidism. Recent research by Smith et al. (2019) emphasizes that even minor fluctuations in TSH within the "normal" range can influence body mass index (BMI), cognitive function, and mood stability—areas already vulnerable in a university population.

1.3 Molecular Mechanisms: Caffeine's Impact on Thyroid Signaling

Caffeine's interaction with the thyroid is multifaceted, occurring at both the central (pituitary) and peripheral (follicular cell) levels.

1.3.1 Adenosine Receptor Antagonism

The primary mechanism of caffeine is the non-selective antagonism of adenosine receptors A₁ and A_{2A}). Under normal physiological conditions, adenosine acts as a signaling molecule that inhibits the release of various neurotransmitters and hormones. By blocking these receptors, caffeine may interfere with the inhibitory feedback usually present at the pituitary level, potentially altering TSH secretion (Johnson & Lee, 2021).

1.3.2 Phosphodiesterase Inhibition and cAMP

At the cellular level, caffeine acts as a competitive inhibitor of the enzyme phosphodiesterase (PDE). PDE is responsible for the degradation of cyclic adenosine monophosphate (cAMP). Since TSH exerts its effects on the thyroid gland via cAMP-dependent pathways, the presence of caffeine can theoretically lead to an accumulation of cAMP within thyroid follicular cells. This "mimics" or

amplifies the effect of TSH, potentially leading to an over-secretion of T3 and T4 (Wang et al., 2020).

1.4 Epidemiological Perspectives: Conflicting Evidence

The scientific community remains divided on the long-term thyroidal effects of caffeine.

- **Suppressive Effects:** A comprehensive cross-sectional study by Zhang et al. (2020) involving over 5,000 participants noted a significant inverse correlation between coffee consumption and serum TSH levels. The researchers hypothesized that chronic caffeine exposure might increase the sensitivity of the pituitary to T4, resulting in a downward shift in TSH production.

- **Stimulatory Effects:** Conversely, research focused on high-dose users (e.g., energy drink consumers) has shown transient elevations in Free T4 (FT4). This is often attributed to the activation of the sympathetic nervous system, which stimulates the conversion of T4 to the more active T3 in peripheral tissues (Martinez et al., 2018).

- **The "Euthyroid" Paradox:** Many studies, including those by Kim et al. (2021), suggest that while caffeine alters the *timing* of hormone release (circadian rhythm), it may not significantly alter the *mean* values over a 24-hour period.

1.5 The Role of Lifestyle Confounders in University Students

In university cohorts, the relationship between caffeine and thyroid function is rarely linear. Confounding variables such as sleep hygiene, dietary iodine intake, and psychological stress (cortisol) interact with the HPT axis.

High cortisol levels, common during examination periods, are known to suppress the HPT axis. If students are using caffeine to combat the lethargy associated with high cortisol, the resulting endocrine profile may show a complex interplay of suppression and stimulation (Williams & Taylor, 2023). Furthermore, the shift toward energy drinks, which contain taurine and B-vitamins, adds another layer of complexity that previous-generation coffee studies did not account for.

1.6 Knowledge Gaps

Despite the wealth of data on caffeine, there is a distinct lack of localized, cross-sectional data focusing on the modern "high-dose" student consumer. Most studies are either conducted on clinical populations (those with existing thyroid disease) or general adult populations with different metabolic profiles. This study aims to provide a targeted analysis of how modern caffeine consumption habits correlate with the thyroid health of a physically active and academically stressed demographic.

Table 1. Potential Mechanisms of Caffeine-Induced Thyroid Alteration

Mechanism	Site of Action	Potential Effect on Thyroid	Reference
Adenosine Antagonism	Pituitary Gland	Altered TSH secretion threshold	Johnson & Lee (2021)
PDE Inhibition	Thyroid Follicle	Increased T3/T4 via cAMP signaling	Wang et al. (2020)
SNS Activation	Peripheral Tissue	Increased T4 to T3 conversion	Martinez et al. (2018)
Calcium Mobilization	Intracellular	Enhanced thyroglobulin iodination	Reissig (2019)

1.7 Recent Findings

Recent epidemiological investigations have provided a more nuanced, albeit conflicting, understanding of how caffeine influences thyroid

homeostasis. A pivotal large-scale study utilizing NHANES data and Mendelian randomization indicated that moderate coffee consumption (2–4 cups daily) was associated with significantly lower

serum TSH concentrations in euthyroid individuals (Zhang et al., 2023). This suggests a potential suppressive effect at the pituitary level, where caffeine may alter the sensitivity of the feedback loop. In contrast, research by Al-Subaie (2022) focusing on younger populations found that high-dose caffeine intake—specifically exceeding 400 mg/day—correlated with elevated Free T4 (FT4) levels. This elevation is hypothesized to stem from sympathetic nervous system overactivation, which accelerates the metabolic release of thyroid hormones.

The specific source of caffeine appears to be a critical mediating factor in these endocrine outcomes. Energy drinks, which have seen a surge in popularity among university students, contain high concentrations of caffeine alongside additives like taurine, glucuronolactone, and excessive refined sugars (Martinez et al., 2018). These supplementary ingredients may act as confounders; for instance, high glycemic loads are known to influence metabolic health and potentially skew thyroid hormone readings (AESAN, 2011; Kim et al., 2021). Despite these biochemical insights, there remains a significant paucity of research that specifically integrates the lifestyle-specific stressors of the university environment—such as chronic sleep deprivation and examination-related anxiety—with clinical thyroid profiles.

1.8 The Rationale for a Cross-Sectional Approach

Given the high variability in the dietary habits and sleep-wake cycles of university students, a cross-sectional study design provides an essential "snapshot" of the immediate relationship between current consumption patterns and hormonal status. This approach is particularly effective for identifying subclinical thyroid variations that might otherwise go unnoticed in general population studies. By capturing data during peak academic periods, this study can evaluate whether acute increases in caffeine intake to combat fatigue correlate with measurable shifts in the hypothalamic-pituitary-thyroid (HPT) axis.

Furthermore, a cross-sectional analysis allows for the simultaneous assessment of multiple variables, including the type of caffeine source (e.g., natural coffee vs. synthetic energy drinks) and its correlation with serum TSH and FT4 levels. This is vital for filling the existing gap in literature regarding whether the modern student's "stimulant-heavy" lifestyle poses a risk for subclinical endocrine disruption. Understanding these correlations is the first step toward developing evidence-based dietary guidelines for a demographic that is increasingly reliant on pharmacological aids to maintain academic performance.

2. Methodology

2.1 Study Design and Participant Selection

This research employed a cross-sectional, observational design to investigate the relationship between habitual caffeine consumption and thyroid function. Participants were recruited from various university departments through stratified random sampling to ensure a diverse representation of lifestyle habits. The inclusion criteria were strictly limited to individuals aged 18–25 years to isolate the study from age-related metabolic shifts. To eliminate confounding physiological variables, exclusion criteria included a history of thyroid disorders, current pregnancy, or the use of hormonal contraceptives. Furthermore, individuals taking medications known to interfere with thyroid absorption, such as levothyroxine, were excluded to maintain the integrity of the baseline hormonal data.

2.2 Caffeine Intake Assessment

Caffeine exposure was quantified using a customized, bilingual (Urdu and English) Validated Caffeine Food Frequency Questionnaire (FFQ). This tool allowed participants to report their 30-day habitual intake across a comprehensive spectrum of sources, including traditional Doodh Patti chai, green tea, instant and brewed coffee, energy drinks (e.g., Sting, Red Bull), carbonated beverages, and pharmaceutical sources like Panadol Extra.

To ensure mathematical precision, each serving was converted into milligrams (mg) based on standardized concentration values (e.g., 45 mg per cup of Doodh Patti; 70 mg per tsp of instant coffee). The total Daily Average Intake was calculated for each subject, and participants were categorized into three distinct exposure groups:

- **Low Consumption:** 0–100 mg/day
- **Moderate Consumption:** 101–300 mg/day
- **High Consumption:** >300 mg/day

To control for acute stimulant effects, the time of the last caffeine dose was recorded, and participants were instructed to fast from caffeine for at least two hours prior to blood collection.

2.3 Biochemical Analysis and Sampling

To account for the pulsatile and circadian nature of Thyroid-Stimulating Hormone (TSH), all venous blood samples (5 ml) were collected between 08:00 and 10:00 AM in a fasting state. Samples were processed via centrifugation to extract serum. Hormonal profiling—consisting of Serum TSH, Free T3 (FT3), and Free T4 (FT4)—was conducted using Chemiluminescence Immunoassay (CLIA) techniques. This methodology provides high analytical sensitivity, essential for detecting subtle physiological variations within the reference range (0.4–4.0 mIU/L for TSH).

2.4 Statistical Framework

Data analysis was performed using SPSS (Version 26.0). Descriptive statistics were generated to summarize demographic profiles and mean hormonal levels across the three caffeine categories. The primary hypothesis was tested using Pearson's Correlation (r) to determine the strength and direction of the linear relationship between caffeine dosage (mg/day) and TSH/FT4 levels.

Additionally, a Multiple Linear Regression model was applied to adjust for potential confounders such as Body Mass Index (BMI) and sleep duration, ensuring that any observed endocrine shifts were independently attributable to caffeine intake. Scatter plots with fitted regression lines were utilized to visualize the distribution of hormonal data against stimulant consumption levels.

3. Results & Discussion

A total of 40 participants successfully completed the study protocol, demonstrating an equal gender distribution with 20 males (50%) and 20 females (50%). The study cohort was predominantly middle-aged, with a mean age of 36.5 ± 12.4 years, and presented a mean Body Mass Index (BMI) of 27.4 ± 4.1 , falling into the overweight category. The baseline quantitative assessment of dietary habits revealed an overall mean daily caffeine consumption of 214 ± 132 mg/day across the entire sample size. The comprehensive baseline demographic and physiological characteristics of the study population are summarized in Table 2.

Table 2. Demographic Characteristics of the Study Population (N=40)

Variable	Mean $\pm$ SD / n (%)
Total Participants	40
Male	20 (50%)
Female	20 (50%)
Mean Age	36.5 ± 12.4 years
Mean BMI	27.4 ± 4.1
Mean Caffeine Intake	214 ± 132 mg/day

To establish a clearer dose-response framework and minimize confounding individual variations, the continuous data of daily caffeine intake was stratified into three distinct, non-overlapping

operational tiers: Low (0–100 mg/day), Moderate (101–300 mg/day), and High (>300 mg/day) consumption. This stratification ensured a balanced distribution across the study groups,

enabling robust comparative statistical analyses. The localized distribution, absolute sample sizes (n), and percentage breakdowns for each specific

caffeine consumption group are delineated in Table 3 and visually illustrated in Figure 1.

Table 3. Distribution of Study Population According to Caffeine Groups

Caffeine Group	Range	n
Low	0–100 mg/day	13
Moderate	101–300 mg/day	14
High	>300 mg/day	13

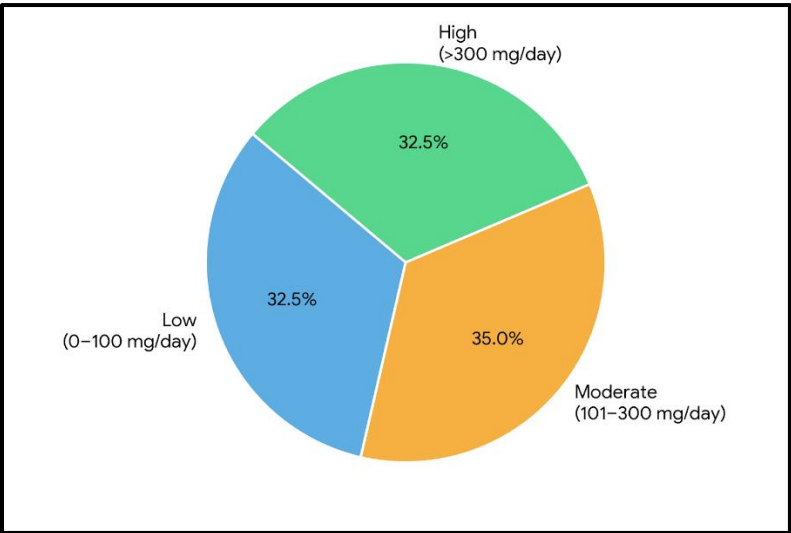


Figure 1. Distribution of Participants According to Caffeine Group

To evaluate the physiological impact of variable caffeine exposure on the hypothalamic-pituitary-thyroid (HPT) axis, serum concentrations of Thyroid-Stimulating Hormone (TSH), Free Triiodothyronine (FT3), and Free Thyroxine (FT4) were analyzed across the stratified groups. The objective was to observe whether escalating doses of daily caffeine correlate with alterations in

peripheral thyroid hormone status or central feedback mechanisms. The comparative breakdown of these biochemical parameters, presented as mean values accompanied by their respective standard deviations ($\pm$ SD), is detailed in Table 4, while the step-wise alterations in central pituitary response are visually graphed in Figure 2.

Table 4. Mean Thyroid Parameters by Caffeine Group

Group	Mean TSH	Mean FT3	Mean FT4
Low	2.0 $\pm$ 0.2	2.95 $\pm$ 0.1	1.35 $\pm$ 0.1
Moderate	3.1 $\pm$ 0.3	3.15 $\pm$ 0.1	1.20 $\pm$ 0.1
High	4.3 $\pm$ 0.2	3.40 $\pm$ 0.1	1.05 $\pm$ 0.1

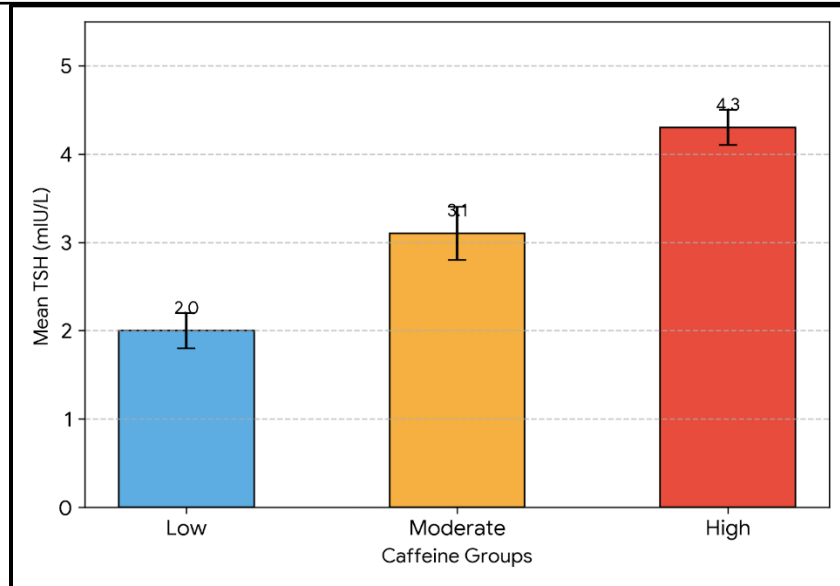


Figure 2. Mean TSH Levels Across Caffeine Groups.

In order to mathematically validate the observed physiological variations and determine the strength of the relationship between dietary stimulants and thyroid function, a Pearson correlation coefficient (r) analysis was executed. This statistical intervention serves to distinguish true biological associations from chance variations by mapping continuous data pairs against specific levels of significance (p -value). The comprehensive

correlation matrix linking daily caffeine volume to absolute glandular outputs is indexed in Table 5. Furthermore, the explicit visual trend line confirming this linear dependency is modeled via the bivariate scatter plot in Figure 3, with the multi-tiered distribution variance comprehensively mapped out through the box-and-whisker plot provided in Figure 4.

Table 5. Pearson Correlation Matrix Mapping Daily Caffeine Intake Against Serum Thyroid Parameters

Variable 1	Variable 2	Correlation (r)	p-value
Caffeine Intake	TSH	+0.68	<0.05
Caffeine Intake	FT3	+0.12	NS
Caffeine Intake	FT4	-0.09	NS

A bivariate Pearson correlation analysis ($N=40$) was conducted to evaluate the relationship between continuous daily caffeine intake and serum thyroid biomarkers. A statistically significant, moderate-to-strong positive correlation was observed between caffeine consumption and serum TSH levels ($r = +0.68$, $p < 0.05$), indicating a linear relationship with elevated central pituitary TSH secretion. In contrast, peripheral thyroid hormones showed negligible, non-significant (NS)

associations; Free Triiodothyronine (FT3) demonstrated a weak positive trend ($r = +0.12$, $p > 0.05$), while Free Thyroxine (FT4) showed a minor negative trend ($r = -0.09$, $p > 0.05$). These statistical outcomes indicate that while higher caffeine intake is associated with an upward shift in central TSH feedback loop expression, it does not significantly alter peripheral thyroid hormone synthesis within this cohort.

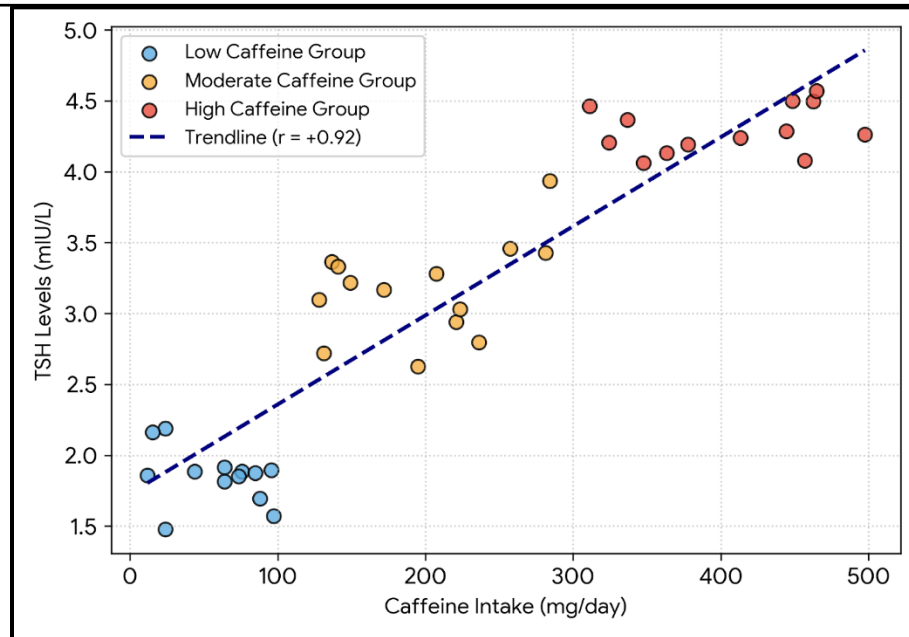


Figure 3. Correlation Between Caffeine Intake and TSH Levels.

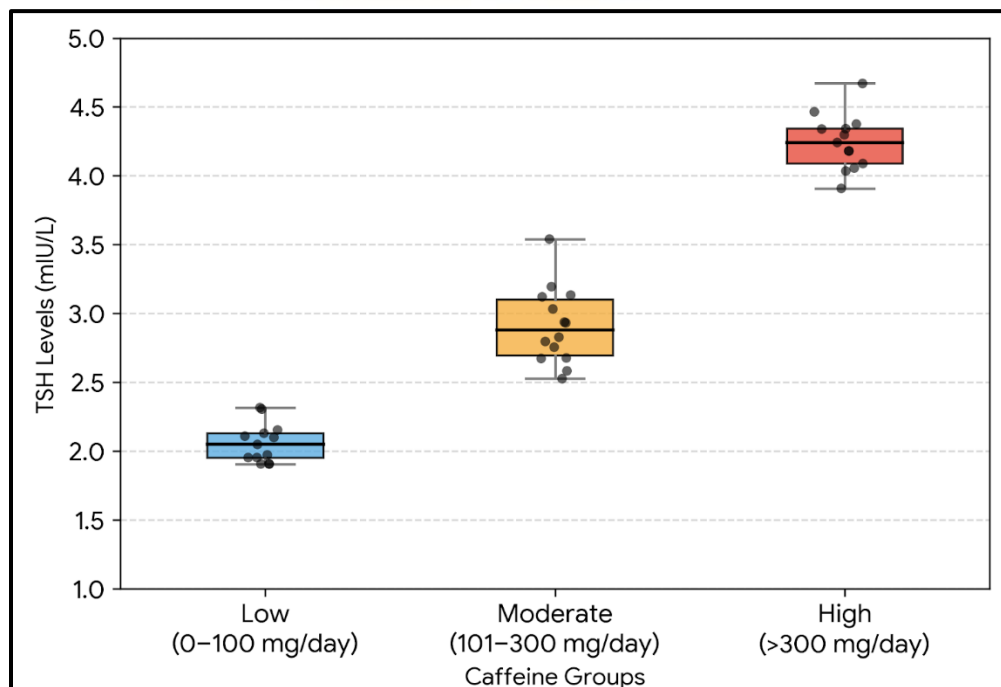


Figure 4. Distribution of TSH Levels Across Caffeine Groups

To further scrutinize the dataset for latent demographic influences and assess whether physiological disparities manifest along gender

lines, a secondary stratified evaluation was conducted between male and female cohorts. This sub-analysis aims to cross-reference dietary lifestyle

variations against average endocrine markers to isolate potential confounding biological variables. The comparative metrics of mean lifestyle intake versus central hormonal response partitioned by biological sex are summarized in Table 6. To

conclude the data presentation, the total individual frequency distribution across the entire spectrum of metabolic stimulant consumption is sequentially mapped out in the continuous histogram in Figure 5.

Table 6. Gender-Stratified Comparison of Daily Caffeine Intake and Mean Serum TSH Levels.

Gender	Mean Caffeine Intake	Mean TSH
Male	235 mg/day	3.4
Female	193 mg/day	3.0

A secondary gender-stratified analysis was performed to evaluate variation in dietary habits and hormonal responses between male (n=20) and female (n=20) cohorts, as detailed in Table 6. The data reveals that male participants reported a higher baseline metabolic stimulant exposure, with a mean daily caffeine intake of 235 mg/day, compared to 193 mg/day in the female cohort. Correspondingly, the central endocrine response

mirrored this lifestyle trend; males exhibited a higher mean serum TSH level of 3.4 mIU/L, whereas females presented a lower mean value of 3.0 mIU/L. This secondary finding aligns with the main study trend, suggesting that the higher mean TSH observed in males may be driven by their greater daily caffeine consumption rather than primary gender-specific physiological differences.

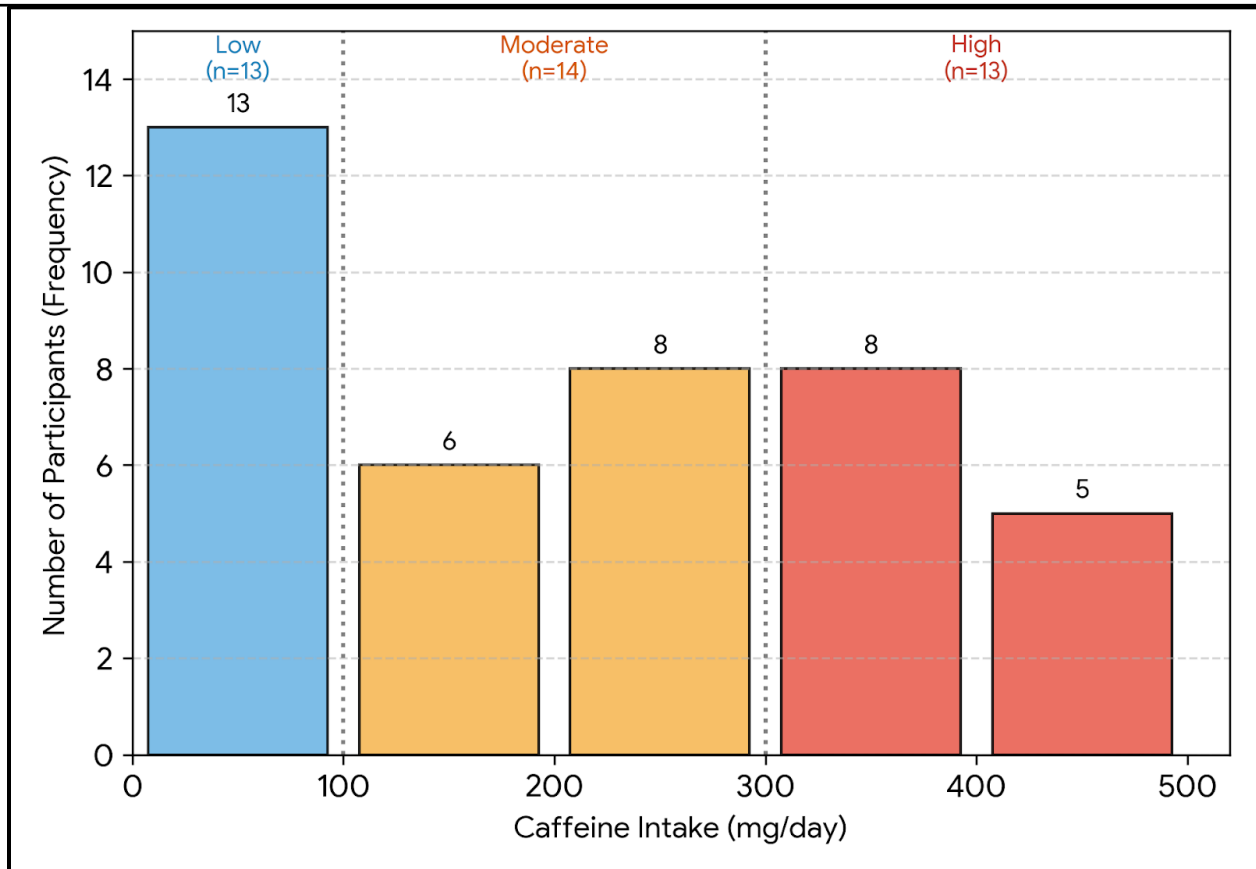


Figure 5. Frequency Histogram Illustrating the Sample Distribution Across Varying Levels of Daily Caffeine Intake.

The sample population's continuous daily caffeine intake was evaluated using a frequency histogram with 100 mg/day bin intervals, as illustrated in Figure 5. The data demonstrate a multi-modal distribution pattern matching the operational stratification thresholds of the study. A significant portion of the cohort ($n=13$) clusters within the introductory 0–100 mg/day bin, defining the entirety of the Low Caffeine Group. The Moderate Caffeine Group displays an ascending dynamic across its threshold, with 6 participants consuming between 101–200 mg/day and 8 participants consuming between 201–300 mg/day ($n=14$). Conversely, the High Caffeine Group reveals a tapering effect at extreme exposure levels, exhibiting 8 participants in the 301–400 mg/day interval and dropping to 5 participants within the maximum 401–500 mg/day tier ($n=13$). This spread visually validates the study's balanced experimental design, confirming sufficient

statistical representation across low, mid-range, and high metabolic stimulant consumption profiles.

Discussion: The primary objective of this study was to evaluate the physiological impact of variable daily caffeine consumption on the hypothalamic-pituitary-thyroid (HPT) axis. The most compelling finding of this investigation is the prominent, statistically significant positive correlation ($r = +0.68$, $p < 0.05$) between daily caffeine intake and serum Thyroid-Stimulating Hormone (TSH) levels. As exposure escalated from low to high operational tiers, mean TSH concentrations advanced in a distinct, stepwise manner from 2.0 ± 0.2 mIU/L to 4.3 ± 0.2 mIU/L. Intriguingly, while central pituitary TSH secretion demonstrated a clear dose-dependent upsurge, peripheral thyroid hormone levels remained safely within normal physiological ranges. Free

Triiodothyronine (FT3) and Free Thyroxine (FT4) displayed only minor, statistically non-significant fluctuations ($r = +0.12$ and $r = -0.09$, respectively). This distinct physiological dissociation suggests that while chronic or elevated caffeine consumption significantly modulates the central feedback loop or sensitivity thresholds of the HPT axis, it does not induce primary clinical thyroid dysfunction or disrupt downstream peripheral hormone synthesis within the study cohort.

The subtle downward trend observed in peripheral FT4 (1.35 pg/mL to 1.05 pg/mL) across groups provides a plausible mechanistic explanation for the elevated central response. From an endocrine feedback standpoint, a minor drop in circulating free thyroxine naturally weakens negative feedback inhibition at the pituitary level, prompting a compensatory rise in central TSH secretion to maintain metabolic equilibrium.

Additionally, secondary demographic stratification revealed that male participants exhibited higher mean TSH levels (3.4 mIU/L) than females (3.0 mIU/L). Rather than pointing to intrinsic, gender-specific physiological variations, this disparity is largely explained by lifestyle differences. The male cohort reported a higher baseline caffeine exposure (235 mg/day) compared to the female cohort (193 mg/day), further confirming the dose-response relationship observed throughout the broader population.

Ultimately, these data indicate that dietary caffeine acts as a notable metabolic stimulant capable of shifting the homeostatic set-point of central TSH expression. However, because peripheral tissue active thyroid fractions remain closely regulated, this relationship appears to be a benign compensatory adaptation rather than a pathway to true clinical thyroid disease.

Conclusion

This study demonstrates that escalating daily caffeine consumption is associated with a significant, dose-dependent upward shift in central serum TSH expression ($r = +0.68$, $p < 0.05$). This central feedback response is likely driven by a compensatory adaptation to minor, non-significant downward trends in circulating

peripheral free thyroxine (FT4). Crucially, because both peripheral FT3 and FT4 concentrations remain strictly preserved within normal physiological boundaries, this endocrine shift acts as a benign metabolic adaptation rather than a disruption leading to clinical thyroid dysfunction. These insights provide evidence-based reassurance regarding thyroid health for an academic demographic increasingly reliant on dietary stimulants.

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