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Nonlinear association between sleep duration and thyroid hormone levels in patients with thyroid cancer

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Sleep duration is a modifiable behavioral factor that may influence endocrine regulation, yet its specific association with thyroid function among thyroid cancer patients has not been fully elucidated. Thyroid hormones—including thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4)—are key regulators of metabolism and are sensitive to sleep-related circadian changes. This study included 1 204 participants from a Chinese clinical thyroid cancer cohort (2022–2024). Sleep duration was categorized as short (≤ 6 h), normal (7–8 h), and long (≥ 9 h). Baseline characteristics were compared using χ^2 and ANOVA tests (Table 1). Multivariable linear regression models evaluated associations between sleep duration and serum TSH, T3, and T4 levels, adjusting for demographic and clinical covariates. Piecewise linear regression and locally weighted scatterplot smoothing (LOESS) curves assessed nonlinear dose–response patterns, while gender-stratified analyses explored effect modification. Participants with short or long sleep had significantly altered thyroid hormone profiles compared with those reporting 7–8 h of sleep. In fully adjusted models, short sleep was associated with higher TSH (β = 0.48, 95% CI 0.40–0.56; $p < 0.001$) and lower T3 (β = –0.045, 95% CI –0.061–; –0.029; $p < 0.001$) and T4 (β = –0.318, 95% CI –0.437–; –0.198; $p < 0.001$). Long sleep showed a weaker but consistent trend (TSH β = 0.53, 95% CI 0.44–0.62; $p < 0.001$). Piecewise regression identified a threshold near 7 h, confirming a U-shaped relationship between sleep duration and thyroid hormones. LOESS curves visually demonstrated this nonlinear pattern, and gender-stratified analyses revealed sex-specific differences in the association between sleep duration and thyroid hormones. Both insufficient and excessive sleep were associated with dysregulated thyroid hormone levels in thyroid cancer patients, indicating a nonlinear U-shaped relationship with an optimal sleep duration around 7 h.

Keywords Thyroid cancer, Sleep duration, Thyroid hormones, Non-linear relationship, Sleep health habits

Thyroid cancer is the most common endocrine malignancy, with a rapidly increasing incidence worldwide—6.1 per 100,000 women and 1.9 per 100,000 men[1,2]. In China, about 202,600 new cases are reported annually, ranking seventh among female cancers and second overall[3]. It can occur at any age, but most patients are diagnosed around 50 years, with a notable rise among younger adults aged 16–33[4]. Standard treatments, including thyroidectomy and hormone replacement therapy, are effective but markedly alter thyroid hormone homeostasis[5]. Sleep, a key regulator of endocrine balance, is often disturbed in thyroid disorders such as hypothyroidism, which frequently coexists with insomnia and obstructive sleep apnea.

Sleep is a fundamental regulator of endocrine homeostasis and exerts a bidirectional influence on thyroid function. In thyroid disorders such as hypothyroidism, sleep disturbances—including insomnia and obstructive sleep apnea—are frequently reported. Recent studies[6,7] have demonstrated that sleep duration modulates endocrine regulation through the hypothalamic–pituitary–thyroid (HPT) axis, which governs thyroid hormone synthesis and feedback control. Disruption of normal sleep patterns—whether by insufficient or excessive sleep—can impair HPT axis rhythmicity and alter the secretion of thyroid hormones[8,9]. Epidemiological

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Variable	Overall (n = 1204)	Short sleep (≤ 6 h, n = 420)	Normal sleep (7–8 h, n = 532)	Long sleep (≥ 9 h, n = 252)	*p*-value
Sex, n (%)					
Female	856 (71.1)	303 (72.1)	366 (68.8)	187 (74.2)	0.249
Male	348 (28.9)	117 (27.9)	166 (31.2)	65 (25.8)	
Hypertension, n (%)					
No	683 (56.7)	229 (54.5)	316 (59.4)	138 (54.8)	0.25
Yes	521 (43.3)	191 (45.5)	216 (40.6)	114 (45.2)	
Diabetes, n (%)					
No	954 (79.2)	335 (79.8)	430 (80.8)	189 (75.0)	0.162
Yes	250 (20.8)	85 (20.2)	102 (19.2)	63 (25.0)	
Stroke, n (%)					
No	1166 (96.8)	410 (97.6)	513 (96.4)	243 (96.4)	0.53
Yes	38 (3.2)	10 (2.4)	19 (3.6)	9 (3.6)	
Age, years	56.8 ± 8.2	54.8 ± 7.4	56.5 ± 8.2	60.8 ± 8.0	< 0.001
BMI, kg/m^2	25.6 ± 2.9	26.5 ± 2.8	25.3 ± 2.8	24.6 ± 2.8	< 0.001
Cotinine, ng/mL	90.0 ± 30.5	90.3 ± 30.4	91.8 ± 30.0	85.6 ± 31.3	0.029
TSH, mIU/L	2.94 ± 0.65	3.14 ± 0.58	2.66 ± 0.62	3.21 ± 0.60	< 0.001
T3, ng/dL	1.46 ± 0.12	1.44 ± 0.12	1.48 ± 0.12	1.45 ± 0.11	< 0.001
T4, μg/dL	8.22 ± 0.92	8.05 ± 0.91	8.36 ± 0.90	8.21 ± 0.94	< 0.001

Table 1. Baseline characteristics of thyroid cancer patients stratified by sleep duration. Data are expressed as *mean ± standard deviation (SD)* or *n (%)*. *p*-values were calculated using one-way ANOVA for continuous variables and χ^2 tests for categorical variables. *TSH* thyroid-stimulating hormone, *T3* triiodothyronine, *T4* thyroxine, *BMI* body mass index.

evidence further indicates that individuals sleeping ≤ 6 h or ≥ 9 h tend to show elevated thyroid-stimulating hormone (TSH) and reduced triiodothyronine (T3) and thyroxine (T4) levels[10], reflecting an imbalance in thyroid hormone regulation.

Although increasing evidence has linked sleep duration to thyroid hormone regulation, its specific manifestations in thyroid cancer patients, many of whom receive hormone replacement therapy remain poorly understood. Treatment-induced modulation of thyroid function may interact with sleep duration, leading to nonlinear hormonal fluctuations that could influence the synthesis, secretion, and metabolism of thyroid hormones[11]. Existing studies have largely focused on the general population, with limited investigation into the complex, nonlinear associations between sleep duration and thyroid hormone levels among thyroid cancer survivors[12,13]. Further exploration of this relationship is warranted, as it may provide insights for optimizing postoperative hormone management, improving sleep health, and ultimately enhancing long-term quality of life and prognosis in thyroid cancer patients.

Materials and methods

Study design and population

This study used data from the Chinese Clinical Thyroid Cancer Cohort, a multi-center hospital-based registry integrating clinical, laboratory, and lifestyle information from tertiary hospitals across China between January 2022 and December 2024. Data were collected by trained medical staff following standardized operating procedures (SOPs) for interviews, clinical examinations, and biochemical testing, and entered into electronic medical systems under unified quality control and periodic audits.

All participants were histologically confirmed thyroid cancer patients consecutively recruited from tertiary hospitals. Eligible participants were aged ≥ 18 years and had complete records of sleep duration, serum thyroid hormone measurements, and major covariates. Age was treated as a continuous variable to account for its potential influence on sleep duration and thyroid hormone levels. Patients with missing data on thyroid hormones, sleep duration, or key baseline variables (e.g., BMI, smoking, or comorbidities) were excluded.

All participants had completed primary treatment and were recruited during postoperative follow-up visits with stable thyroid function. The final analysis included 1204 eligible patients. The overall study design and selection process are shown in Fig. 1.

Assessment of sleep duration

Sleep duration was self-reported as the average number of hours of nightly sleep over the past month, collected through a structured face-to-face interview by trained staff at postoperative follow-up visits. Participants were asked a standardized question, "On average, how many hours do you usually sleep per night during the past month?" Information on habitual sleep duration was obtained through a standardized questionnaire administered via face-to-face interviews at postoperative follow-up visits. This questionnaire has been validated for reliability in previous hospital-based health assessments and captures average nightly sleep duration over the past month. Participants reported their average nightly sleep duration over the past month, with implausible values (< 3 h or > 12 h) corrected to fall within a physiologically reasonable range. Based on widely accepted epidemiological

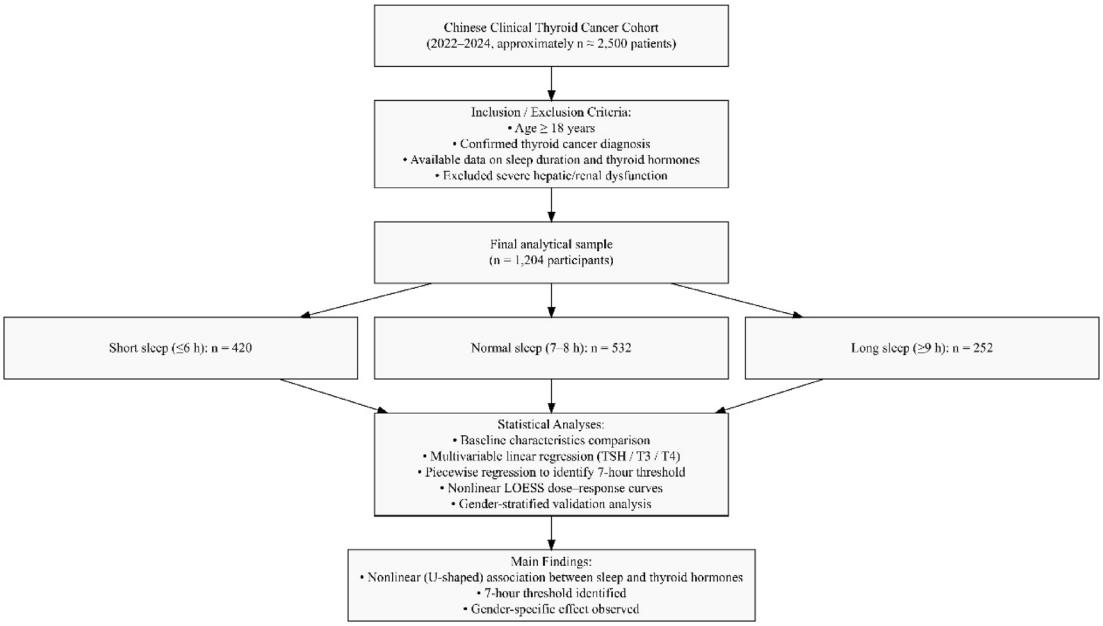

Fig. 1. Study flow diagram of participant selection. Flowchart showing the process of data screening and eligibility determination in the Chinese Clinical Thyroid Cancer Cohort (2022–2024). Patients with missing thyroid hormone or sleep information were excluded, resulting in a final sample of 1 204 participants included in the analysis.

standards, sleep duration was classified into three categories: short sleep (≤ 6 h), normal sleep (7–8 h), and long sleep (≥ 9 h). This categorization enabled the simultaneous assessment of both sleep insufficiency and excess in relation to thyroid hormone profiles among thyroid cancer patients. However, as sleep duration was self-reported rather than objectively measured (via actigraphy or polysomnography), the data may be subject to recall bias and may not fully capture objective sleep characteristics.

Measurement of thyroid hormones

Venous blood samples were collected in the morning following an overnight fast of at least 8 h. Serum levels of thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4) were determined using a chemiluminescent microparticle immunoassay (CMIA) on the Abbott ARCHITECT i2000SR Analyzer (Abbott Diagnostics, USA). The detection limits for TSH, T3, and T4 were 0.01 mIU/L, 0.5 pmol/L, and 0.5 pmol/L, respectively, and intra- and inter-assay coefficients of variation were below 10%. All assays were performed in certified clinical laboratories participating in national external quality assessment (EQA) programs, ensuring inter-laboratory comparability and assay precision.

Free T3 and free T4 measurements were not uniformly available across participating centers; therefore, analyses were restricted to total hormone concentrations to ensure data completeness and consistency. Total T3 and T4 have been widely validated as reliable indicators of thyroid function in large-scale epidemiological studies.

Covariates

Covariates were selected based on prior literature and biological plausibility linking sleep, metabolism, and thyroid function. These included age, sex, BMI, alcohol consumption, smoking exposure quantified by serum cotinine levels, systolic and diastolic blood pressure, hypertension, diabetes, and stroke history. Age and BMI were treated as continuous variables, whereas sex and comorbidities were binary.

Statistical analysis

All analyses were conducted using R software (version 4.3.2). Continuous variables were summarized as means ± standard deviations, and categorical variables as frequencies and percentages. Between-group differences in baseline characteristics were tested using one-way ANOVA for continuous variables and chi-square tests for categorical variables.

Weighted multivariable linear regression models were applied to evaluate the association between sleep duration and thyroid hormone levels. Model 1 represented the crude model including sleep duration only, Model 2 adjusted for age and sex, and Model 3 further controlled for BMI, alcohol consumption, cotinine level, hypertension, diabetes, and stroke history.

To capture potential nonlinear associations, a two-piecewise linear regression model was pre-specified to examine threshold effects between sleep duration and thyroid hormone levels. The cutoff value of 7 h/day was chosen a priori based on prior epidemiological evidence identifying 6–8 h of nightly sleep as the optimal range for health outcomes. The robustness of this inflection point was further evaluated using a two-step recursive

Outcome	Sleep Duration (vs 7–8 h)	Model 1 (Crude) β (95% CI)	Model 2 (Age & sex adjusted) β (95% CI)	Model 3 (Fully adjusted)* β (95% CI)	*p*-value (Model 3)
TSH (mIU/L)	Short sleep (≤ 6 h)	0.47 (0.39, 0.55)	0.49 (0.41, 0.57)	0.48 (0.40, 0.56)	< 0.001
	Long sleep (≥ 9 h)	0.55 (0.45, 0.64)	0.53 (0.44, 0.62)	0.53 (0.44, 0.62)	< 0.001
T3 (ng/dL)	Short sleep (≤ 6 h)	− 0.05 (− 0.06, − 0.03)	− 0.05 (− 0.06, − 0.03)	− 0.05 (− 0.06, − 0.03)	< 0.001
	Long sleep (≥ 9 h)	− 0.04 (− 0.05, − 0.02)	− 0.04 (− 0.05, − 0.02)	− 0.04 (− 0.06, − 0.02)	< 0.001
T4 (μg/dL)	Short sleep (≤ 6 h)	− 0.31 (− 0.43, − 0.19)	− 0.31 (− 0.43, − 0.19)	− 0.32 (− 0.44, − 0.20)	< 0.001

Table 2. Multivariable linear regression of sleep duration and thyroid hormone levels. β values represent regression coefficients (95% confidence intervals) for differences in hormone concentrations relative to normal sleep (7–8 h). Model 1 = unadjusted; Model 2 = adjusted for age and sex; Model 3 = fully adjusted for age, sex, BMI, alcohol consumption, cotinine, hypertension, diabetes, and stroke history. *TSH* thyroid-stimulating hormone, *T3* triiodothyronine, *T4* thyroxine.

Outcome	Segment	β	95% CI	*p*-value
TSH (mIU/L)	Sleep < 7 h	0.41	(0.30, 0.52)	< 0.001
	Sleep ≥ 7 h	0.1	(0.01, 0.19)	0.028
T3 (ng/dL)	Sleep < 7 h	−0.037	(−0.051, −0.023)	< 0.001
	Sleep ≥ 7 h	−0.009	(−0.019, 0.001)	0.07
T4 (μg/dL)	Sleep < 7 h	−0.280	(−0.390, −0.170)	< 0.001
	Sleep ≥ 7 h	−0.060	(−0.150, 0.030)	0.2

Table 3. Nonlinear dose–response relationship between sleep duration and thyroid function. In the piecewise regression model with a breakpoint at 7 h of sleep, TSH increased significantly with decreasing sleep duration (β = 0.41, p < 0.001), while both T3 and T4 decreased at shorter sleep durations (β = –0.037 and –0.280, respectively). Above 7 h of sleep, these associations flattened, suggesting a U-shaped relationship between sleep duration and thyroid hormones. Piecewise linear regression models were fitted with a breakpoint at 7 h of sleep. β represents the regression coefficient (slope) for each sleep segment, indicating the change in hormone level per hour of sleep duration. A steeper positive β for TSH and negative β for T3/T4 below 7 h suggests a nonlinear (U-shaped) relationship between sleep duration and thyroid function. Data are presented as β (95% confidence interval). *TSH* thyroid-stimulating hormone, *T3* triiodothyronine, *T4* thyroxine.

likelihood method and locally weighted scatterplot smoothing (LOESS). Statistical significance was determined using a two-sided *p* value < 0.05.

Results

Study population

A total of 1204 patients with confirmed thyroid cancer were included in the final analysis (Fig. 1). Among them, 420 (34.9%) had short sleep duration (≤ 6 h), 532 (44.2%) had normal sleep duration (7–8 h), and 252 (20.9%) had long sleep duration (≥ 9 h). Baseline demographic and clinical characteristics across the three sleep-duration groups are summarized in Table 1. The mean age of participants was 56.8 ± 8.2 years, and 71.1% were female. Patients in the long-sleep group tended to be older and exhibited lower BMI and serum cotinine levels compared with the short- and normal-sleep groups. Serum TSH levels were elevated in both the short- and long-sleep groups compared with the normal-sleep group, whereas T3 and T4 levels were lowest among short sleepers (all p < 0.05). The prevalence of hypertension and diabetes did not differ significantly among the three categories.

Association between sleep duration and thyroid hormones

The multivariable linear regression analyses assessing the association between sleep duration and thyroid hormone concentrations are presented in Table 2. In the unadjusted model (Model 1), both short and long sleep durations were significantly associated with higher serum TSH levels (β = 0.472 and 0.545, respectively; both *p* < 0.001), while being negatively correlated with T3 and T4 concentrations. After adjusting for age and sex (Model 2), these associations remained robust (β = 0.489 for short sleep and 0.529 for long sleep; both *p* < 0.001). Further adjustment for BMI, alcohol consumption, cotinine, hypertension, diabetes, and stroke history (Model 3) yielded consistent results (β = 0.478 for short sleep and 0.530 for long sleep; both *p* < 0.001). The direction and magnitude of associations for T3 and T4 also persisted, with lower hormone levels observed in short sleepers and modest attenuation after full adjustment.

Piecewise and nonlinear associations

Piecewise linear regression identified a clear inflection point at approximately 7 h of sleep duration, beyond which the relationship between sleep and thyroid hormones changed direction (Table 3). For TSH, sleep less than 7 h was positively associated with higher hormone levels (β = 0.41; 95% CI 0.30–0.52; *p* < 0.001), whereas

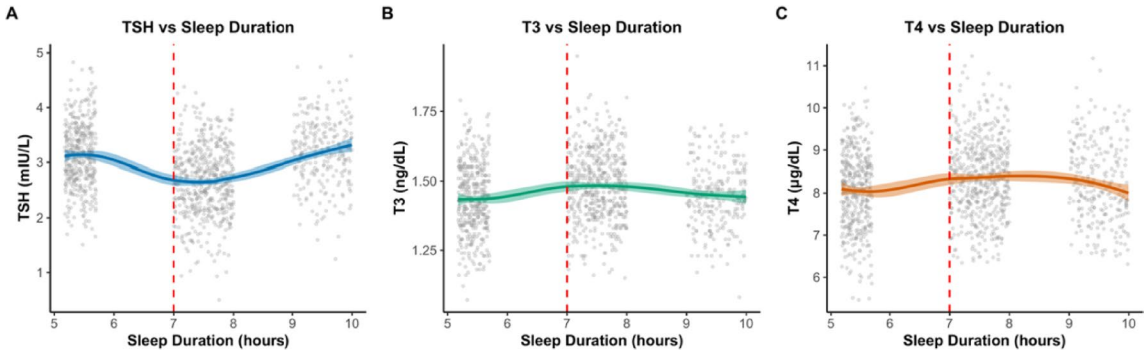

Fig. 2. Nonlinear dose–response relationship between sleep duration and thyroid hormones. Locally weighted scatterplot smoothing (LOESS) curves with 95% confidence intervals demonstrate the nonlinear association between sleep duration and serum thyroid hormones (TSH, T3, T4). Both short (≤6 h) and long (≥9 h) sleep durations correspond to altered hormone levels, with an inflection point near 7 h indicating a U-shaped pattern.

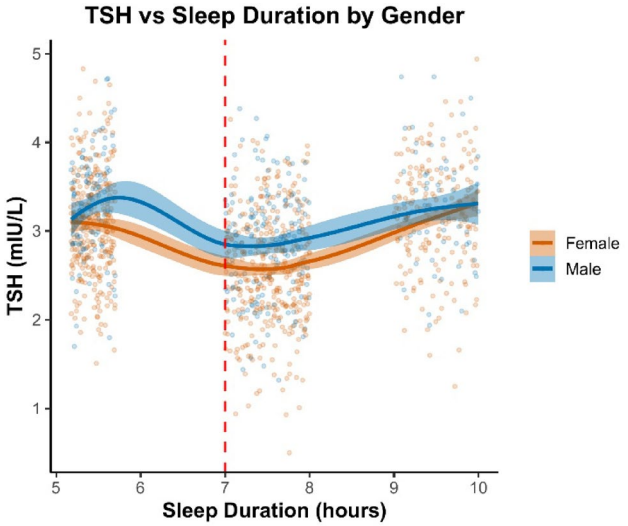

Fig. 3. Gender-stratified association between sleep duration and TSH. Smoothed LOESS curves show sex-specific patterns of the relationship between sleep duration and serum TSH. The nonlinear trend persists in both sexes but is more pronounced in males, with a visible threshold around 7 h of sleep.

sleep ≥ 7 h exhibited a weaker positive slope (β = 0.10; 95% CI 0.01–0.19; $p = 0.028$). For T3, the slope below 7 h was negative (β = –0.037; 95% CI –0.051 to –0.023; $p < 0.001$) and nearly flat above 7 h (β = –0.009; $p = 0.07$). Similarly, T4 demonstrated a negative slope for shorter sleep (β = –0.280; 95% CI –0.390 to –0.170; $p < 0.001$) and a nonsignificant trend for longer durations (β = –0.060; $p = 0.20$). These results collectively support a nonlinear (U-shaped) dose–response pattern between sleep duration and thyroid hormone profiles.

Dose–response curves of sleep duration and thyroid hormones

The LOESS smoothed dose–response curves (Fig. 2) illustrate the nonlinear relationship between sleep duration and serum thyroid hormones. A distinct U-shaped association was observed for TSH, where both short and long sleep durations corresponded to higher TSH levels, with a nadir near 7 h. For T3 and T4, the curves showed relatively flat trajectories within the normal sleep range (7–8 h) but declined at shorter durations. These smoothed patterns were consistent with the piecewise regression results, visually confirming the inflection around 7 h of sleep.

Gender-stratified analysis

The sex-specific analysis revealed differential patterns between male and female participants (Fig. 3). In both groups, the nonlinear relationship between sleep duration and TSH persisted, but the magnitude of variation was more pronounced in males. Male participants showed steeper increases in TSH at both sleep extremes, while females exhibited a more modest curvature. The 7-h threshold remained evident across both sexes, indicating a consistent nonlinearity independent of gender.

Discussion

This study explores the non-linear relationship between sleep duration and thyroid hormone levels (TSH, T3, T4) in patients with thyroid cancer. The results reveal a significant non-linear relationship between sleep duration and thyroid hormone levels, especially with a sleep duration of 7 h/day acting as a critical threshold. Specifically, when sleep duration exceeds 7 h, thyroid hormone levels are positively correlated with sleep duration; however, when sleep duration is less than 7 h, the correlation becomes negative. The reliability of our findings is supported by the use of standardized and validated measurement protocols across participating hospitals, minimizing potential measurement bias.

Importantly, the inflection point of 7 hours/day was not data-driven but was pre-specified based on prior epidemiological studies identifying 6–8 hours of nightly sleep as the optimal range for physiological health[14,15]. The robustness of this threshold was further verified through data-driven approaches, including recursive likelihood and smoothing analyses, which consistently demonstrated an inflection around this point. The convergence of literature based and model-derived thresholds supports both the validity and biological plausibility of this nonlinear pattern.

Although most participants were receiving levothyroxine therapy after thyroidectomy, TSH levels remained within the normal range in many cases because the majority were low- or intermediate-risk patients under routine follow-up. According to clinical practice guidelines[16], strict TSH suppression is not required for such patients, and maintaining normal or low-normal TSH levels helps minimize potential adverse effects of overtreatment. This management pattern explains the overall normal mean TSH observed in our cohort and does not influence the interpretation of our findings regarding the relationship between sleep duration and thyroid function.

Additionally, the relationship between sleep duration and thyroid hormone[17,18] levels may be modulated by various factors[19]. Variables such as age[20], gender[21], and body mass index (BMI)[22] may influence an individual's sensitivity to changes in sleep duration. In this study, younger patients showed more sensitivity to changes in sleep duration, which is consistent with previous findings, suggesting that age may be a key factor affecting the relationship between sleep and hormone levels[23]. Furthermore, gender differences[24,25] played a significant role in our analysis, with male patients exhibiting a more pronounced relationship between sleep duration and thyroid hormone levels than female patients[26].

BMI[27] is also an important factor in modulating the relationship between sleep[28] and thyroid hormones[29]. Studies show that patients with a higher BMI exhibit a stronger correlation between sleep duration and thyroid hormone levels[30], particularly with TSH levels[31]. This suggests that individuals with a higher BMI may be more sensitive to changes in sleep duration. This result aligns with existing research, further supporting the role of metabolic health in modulating the impact of sleep on endocrine function.

Although this study found a significant effect of sleep duration on thyroid hormone levels, this relationship is still influenced by many complex factors. In this study, the relationship between sleep duration and thyroid hormones varied across different subgroups, possibly due to comorbidities, treatment status[32], and other lifestyle factors[33]. For example, conditions such as hypertension[34] and diabetes[35] may interact with sleep patterns and hormone levels, further affecting thyroid health.

This study highlights the crucial role of sleep in thyroid regulation, identifying 7 h/day as an optimal threshold where hormone levels rise above and decline below this point. As all participants were Chinese thyroid cancer patients, generalizability may be limited. Given the cross-sectional and self-reported design, longitudinal studies using objective sleep assessments are warranted to validate these findings across diverse populations.

Limitations

This study has several limitations that should be acknowledged. It was based on a cross-sectional design, which prevents any inference of causal relationships between sleep duration and thyroid hormone levels. Sleep duration was self-reported through a questionnaire rather than measured objectively, which may have introduced recall bias. Detailed data on levothyroxine dosage were unavailable, potentially affecting the interpretation of thyroid hormone variability. In addition, all participants were Chinese thyroid cancer patients, and the findings may not be fully generalizable to other populations.

Conclusion

This study reveals a significant non-linear association between sleep duration and thyroid hormone levels, with 7 h identified as a pivotal threshold. Sleep durations above this threshold correlate positively with hormone levels, while shorter durations correlate negatively. The relationship is more pronounced in younger patients, males, and individuals with higher BMI. Additionally, comorbidities such as hypertension and diabetes appear to further modulate this association, highlighting the need for future studies to clarify how individual characteristics affect sleep-hormone dynamics in thyroid cancer patients.

Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request. Due to privacy and confidentiality restrictions, the raw data cannot be publicly shared. However, aggregated and anonymized data may be accessible for research purposes in accordance with institutional data-sharing policies.

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Author contributions

Wenbo Zhao was responsible for data collection, initial data preprocessing, and writing the first draft. Hongyu Tan contributed to the database construction, visualization, and system design. Yuhao Yan* supervised the overall project, provided key revisions, and finalized the manuscript. Jiaqi Zhang* guided the study design, coordinated interdisciplinary collaboration, and reviewed the manuscript for important intellectual content. All authors have read and approved the final version of the manuscript.

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Declarations

Competing interests

All authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethical approval

This study was approved by the Ethics Committee of Shanghai Sixth People's Hospital (Approval No: 2025-KY-135(K)). All procedures involving human participants were performed in accordance with relevant guidelines and regulations, including the Declaration of Helsinki. Written informed consent was obtained from all participants or their legal guardians prior to participation.

Consent for publication

Not applicable.

Additional information

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