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Relationship between sleep abnormalities and hypothyroidism: results from the National Health and Nutrition Examination Survey 2007–2012

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Abstract

Background Studies have emphasised the adverse effects of poor sleep on human health, however, the correlation between sleep abnormalities and hypothyroidism remains unclear. This study evaluated whether sleep abnormalities may be related to increased prevalence of hypothyroidism in general US adults. Methods: In total, 9016 adults who participated in the National Health and Nutrition Examination Survey from 2007 to 2012 were analysed. A standardised questionnaire was used to collect data regarding sleep duration, self-reported trouble sleeping and sleep disorders. Values were assigned to the three aforementioned sleep factors, resulting in an overall sleep score of 0–3. The sleep patterns were divided into healthy sleep pattern (overall sleep score = 3), intermediate sleep pattern (overall sleep score = 2) and poor sleep pattern (overall sleep score = 0 or 1) according to a former study. Hypothyroidism was defined as thyroid-stimulating hormone (TSH) levels > 5.6 mIU/mL or the need to take thyroid hormones. Multivariable logistic regression analysis was performed to assess the relationship between sleep abnormalities and hypothyroidism. Results: The overall prevalence of hypothyroidism was 8.0% among the 9016 participants. Self-reported trouble sleeping (odds ratio [OR] = 1.38, 95% CI: 1.14–1.68, $p = 0.001$) and sleep disorders (OR = 1.40, 95% CI: 1.06–1.86, $p = 0.0196$) were associated with increased prevalence of hypothyroidism. Neither short sleep duration (OR = 0.99, 95% CI: 0.82–1.19) nor long sleep duration (OR = 0.98, 95% CI: 0.61–1.58) was significantly associated with hypothyroidism. Moreover, poor sleep pattern was significantly associated with increased prevalence of hypothyroidism (OR = 1.30, 95% CI: 1.03–1.66, $p = 0.0301$). Conclusions: Both trouble sleeping and sleep disorders were associated with increased prevalence of hypothyroidism.

Keywords Sleep duration, Sleep patterns, Hypothyroidism, Epidemiology

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Introduction

Hypothyroidism is a disease caused by decreased synthesis and secretion of thyroid hormones or insufficient physiological effects, resulting in decreased body metabolism, and almost all patients with confirmed hypothyroidism need thyroid hormone replacement therapy. According to data from the National Health and Nutrition Examination Survey (NHANES) of individuals recruited from 1999 to 2002, the incidence of hypothyroidism was reported to be 3.7% in the US population [1]. The aetiology of hypothyroidism is complex. Most cases of hypothyroidism are caused by iodine deficiency and autoimmune thyroiditis, such as Hashimoto's thyroiditis [2]. However, other factors can also affect the epidemiology of hypothyroidism. For instance, aging, smoking habit, genetic susceptibility, racial differences and psychological status may all be associated with the occurrence of hypothyroidism, and the physiological phenomenon of sleep has also been paid attention to.

Sleep is a basic physiological requirement and is indispensable for the recovery of daily physiological activity. According to statistics, nearly half of people in the U.S. have trouble sleeping, also, an estimated 50 to 70 million Americans have chronic, or ongoing, sleep disorders [3]. Poor sleep quality may also have adverse effects on endocrine, immune, cardiovascular, neurological and cognitive functions and may result in the occurrence and development of chronic diseases [4–6]. Currently, studies have shown that hypothyroidism can lead to sleep disorders such as obstructive sleep apnoea (OSA), restless legs syndrome (RLS), snoring, trouble falling asleep and daytime sleepiness [7–9]. Meanwhile, the impact of sleep abnormalities on thyroid function has also been noted. For example, Bozkurt et al. [10] found that the prevalence of Hashimoto's disease was increased in patients with OSA and was associated with the severity of OSA. An analysis [11] from the Korea National Health and Nutrition Examination Survey (KNHANES) suggested that shorter and longer sleep durations were both associated with an increase in the risk of subclinical thyroid dysfunction. Wang et al. [12] believed that at short sleep durations, the increased sleep duration was significantly related to decreased FT3 levels. Thus, the impact of sleep abnormalities on thyroid function cannot be ignored. However, these studies have problems such as investigating a single exposure factor and small sample size. In addition, there is no reliable evidence to prove the impact of sleep abnormalities on hypothyroidism, and large-scale clinical studies are still lacking. Therefore, based on a large population of NHANES (a nationally representative survey), we first assessed the relationship between sleep abnormalities, including short or long sleep duration, trouble sleeping and sleep disorders, and the increased odds of hypothyroidism, and aims to provide new insights into the prevention and treatment of hypothyroidism.

Materials and methods

Study population

The NHANES is a nationwide survey conducted by the National Center for Health Statistics (NCHS) and the Centers for Disease Control and Prevention (CDC) to assess the health and nutritional status of the US population [13]. In this survey, a complicated multi-stage probability sampling method was used to obtain nationally representative samples. The NHANES programme was authorised by the Ethics Review Committee of the NCHS in the US, and all participants voluntarily participated and provided informed consent [14].

To assess the correlation between hypothyroidism and sleep, we selected data from three cycles from 2007 to 2012, as only these three cycles included complete data related to thyroid function tests. Our study included all participants who were ≥18 years old, had laboratory TSH data available and had completed a sleep survey questionnaire. We excluded participants who had a history of thyroid cancer or hyperthyroidism (i.e. TSH levels less than 0.34 mIU/L) or who were taking methimazole or propylthiouracil. A flow diagram of the subject selection process is presented in Fig. 1. Details regarding the methods and protocols for questionnaire surveys, laboratory tests and inspections can be acquired from the NHANES website (https://www.cdc.gov/nchs/nhanes/index.htm).

Definition of hypothyroidism

Hypothyroidism was the outcome variable. Thyroid function is mainly assessed based on TSH levels. The TSH level normally ranges from 0.34 to 5.6 mIU/L [15]. We excluded patients with hyperthyroidism who were currently taking methimazole or propylthiouracil, regardless of their TSH levels. We also excluded patients who had TSH levels less than 0.34 mIU/L and were not taking thyroid drugs. If the participants reported that they were currently taking levothyroxine, regardless of their TSH levels, or if their TSH levels were greater than 5.6 mIU/L, they were identified as having hypothyroidism [15]. Participants whose TSH levels were between 0.34 and 5.60 mIU/L and who had never taken any thyroid medication were included in the normal control group.

Evaluation of sleep factors and definition of sleep patterns

Sleep duration was obtained through a questionnaire survey asking 'How much sleep do you usually get at night on weekdays or workdays'? It was divided into short sleep duration (<7 h per night), normal sleep duration (7–9 h per night) and long sleep duration (>9 h per night) [16], using 7–9 h of sleep per night as the reference group. Trouble sleeping was diagnosed by asking

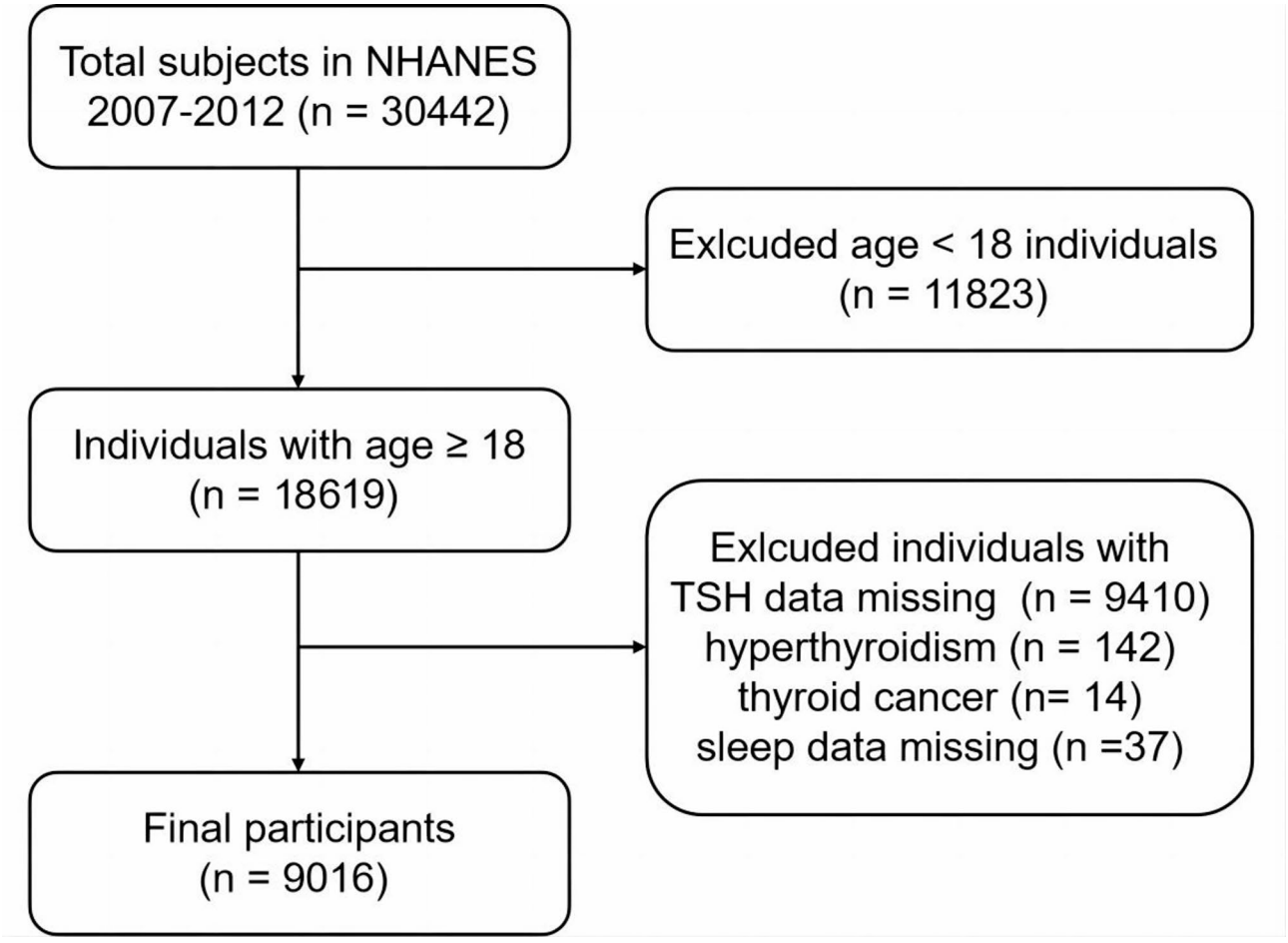

Fig. 1 Participant inclusion flowchart (National Health and Nutrition Examination Survey [NHANES])

'Have you ever told a doctor or other health professional that you have trouble sleeping? (yes or no)'. Sleep disorder was evaluated by asking 'Have you ever been told by a doctor or other health professional that you have a sleep disorder? (yes or no)'. Sleep patterns were determined based on a previous study [17]. Specifically, values were assigned to each of the three sleep characteristics (sleep duration, trouble sleeping and sleep disorders). For each sleep factor, low-risk behaviour was assigned a score of 1 and high-risk behaviour was assigned a score of 0 (e.g. sleeping for less than 7 h per night was considered high risk, whereas sleeping for 7–9 h or more was considered low risk). Based on this, a total score ranging from 0 to 3 was obtained. The overall sleep patterns were accordingly categorised into poor sleep pattern (overall sleep score=0 or 1), intermediate sleep pattern (overall sleep score=2) and healthy sleep pattern (overall sleep score=3) [17].

Other covariates

Numerous covariates, including age, sex (male or female), race (non-Hispanic White, non-Hispanic Black, Mexican American or other Hispanic), education level (less than high school, high school or more than high school), marital status (married/living with a partner, divorced/separated or never married), family poverty income ratio (PIR), body mass index (BMI) (<18.5, 18.5–24.9, 25.0–29.9 or ≥30.0 kg/m^2), smoking habit (yes or no), alcohol use (current, former or never), hypertension (yes or no), diabetes (yes or no), sedative/hypnotic medication (yes or no), thyroid peroxidase antibody (TPOAb), thyroglobulin antibody (TgAb), triglyceride, free triiodothyronine (fT3), free thyroxine (fT4), cholesterol, high-density lipoprotein-cholesterol (HDL-C), uric acid, creatinine, serum glucose and serum iron levels were selected based on literature review and clinical experience. Detailed information on the covariates can be obtained from the NHANES database (https://wwwn.cdc.gov/nchs/nhanes/Default.aspx).

Statistical analysis

Continuous variables are expressed as means±standard deviations (SDs), whereas categorical variables are expressed as percentages. For baseline data, statistical differences between the control group and hypothyroidism

group were analysed using one-way ANOVA for continuous variables and the chi-square test for categorical variables. The three NHANES cohorts were pooled together, and logistic regression analysis was performed to obtain the odds ratios (ORs) and 95% confidence intervals (95% CIs) in order to assess the association between each sleep factor and hypothyroidism. The same analyses were performed to investigate the association between sleep patterns and hypothyroidism. The model had three parts. Of these, model 1 was an unadjusted model. Model 2 was adjusted for age, sex, race, education level, marital status and family PIR. Model 3 was adjusted for the variables in model 2 plus BMI, smoking habit, alcohol use, hypertension, diabetes, sedative/hypnotic medication and TPOAb, TgAb, fT3, fT4, triglyceride, cholesterol, HDL-C, uric acid, creatinine, serum glucose and serum iron levels.

Next, interaction and stratified analyses were performed by age, sex, race/ethnicity, BMI, hypertension and diabetes, to identify potential effect modifiers.

All statistical analyses were performed using R software (http://www.R-project.org, The R Foundation) and EmpowerStats software (http://www.empowerstats.com, X&Y Solutions, Inc. Boston, MA, USA).

Results

Baseline characteristics of participants

In total, 9016 participants were eligible in this study. Of these, 718 (8.0%) participants had hypothyroidism. Descriptive statistics are provided in Table 1. Participants with hypothyroidism were more likely to be female, older, white, more educated and obese. A higher proportion of participants in the hypothyroidism group had hypertension, diabetes, and the use of sedative or hypnotic medications than in the control group. Significant differences were noted in some blood biochemical indices, such as fT4, fT3, TgAb, TPOAb, triglyceride, cholesterol, albumin, HDL-C, creatinine, serum glucose and serum iron levels, between the hypothyroidism group and control group. In addition, the proportion of participants who had trouble sleeping, sleep disorders and poor sleep pattern was higher in the hypothyroidism group than in the control group.

Associations of sleep abnormalities with increased odds of hypothyroidism

Figure 2 presents the association of sleep duration, trouble sleeping and sleep disorders with increased odds of hypothyroidism. In the unadjusted model (model 1), short sleep duration (OR=0.78, 95% CI: 0.67–0.92, $p=0.003$), trouble sleeping (OR=1.88, 95% CI: 1.60–2.21, $p<0.001$) and sleep disorders (OR=1.80, 95% CI: 1.42–2.28, $p<0.001$) were associated with hypothyroidism. After adjusting for age, sex, race, marital status, education level and PIR (model 2), trouble sleeping and sleep disorders remained statistically significant factors, whereas sleep duration did not show any statistical association with hypothyroidism. After additionally adjusting for BMI, smoking habit, alcohol use, hypertension, diabetes, sedative or hypnotic medications, TgAb, TPOAb, fT3, fT4, triglyceride, cholesterol, HDL-C, uric acid, creatinine, serum glucose and serum iron levels (model 3), trouble sleeping and sleep disorders were still significantly associated with increased odds of hypothyroidism.

The combined effect of the three factors is shown in Fig. 3. After adjusting for all covariates, poor sleep pattern (OR=1.30, 95% CI: 1.03–1.66, $p=0.0301$) remained significantly associated with increased prevalence of hypothyroidism, whereas the association of intermediate sleep pattern (OR=1.21, 95% CI: 0.99–1.47, $p=0.0596$) with increased prevalence of hypothyroidism was not statistically significant.

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Stratifed associations between sleep patterns and increased odds of hypothyroidism

We additionally investigated the relationship between sleep patterns and hypothyroidism after stratifying the participants by age (<50 or ≥50 years), sex (male and female), race/ ethnicity (non-Hispanic White, non-Hispanic Black, Mexican American or other Hispanic), BMI (<25, 25–29.9 or ≥30 kg/m^2), hypertension (yes or no), diabetes (yes or no), and their interactions were tested (Table 2). When these three sleep factors were considered together by forming the participants' sleep patterns, none of the selected variables significantly altered the relationship between hypothyroidism and sleep patterns (age [<50 or ≥50 years; P-interaction=0.8852], sex [male, female; P-interaction=0.6470], race/ethnicity [Non-Hispanic White, Non-Hispanic Black, Mexican American, other race; P-interaction=0.5120], BMI [<25, 25–29.9 and ≥30 kg/m^2; P-interaction=0.9152], hypertension [yes, no; P-interaction=0.6705], diabetes [yes, no; P-interaction=0.3066]). Which indicated that the results of the relationship between sleep patterns and hypothyroidism are consistent across different factor stratification.

Discussion

In this analysis of cross-sectional study based on a nationally representative population sample, we assessed the correlation between three sleep factors and hypothyroidism. Of the three sleep factors, trouble sleeping and sleep disorders were both associated with an increased prevalence of hypothyroidism. Participants who had trouble sleeping or sleep disorders had an approximately 1.38-fold and 1.4-fold higher prevalence of hypothyroidism, respectively, than those without these sleep

Table 1 Baseline characteristics of participants based on hypothyroidism status

Variable	Total ($n=9016$)	Non- Hypothyroidism ($n=8298$)	Hypothyroidism ($n=718$)	*P*-value
Age (years)	48.5 ± 18.6	47.4 ± 18.4	61.3 ± 16.3	< 0.001
Gender, *n* (%)				< 0.001
Male	4493 (49.8%)	4293 (51.7%)	200 (27.9%)	
Female	4523 (50.2%)	4005 (48.3%)	518 (72.1%)	
Race, *n* (%)				< 0.001
Non-Hispanic White	4108 (45.6%)	3622 (43.6%)	486 (67.7%)	
Non-Hispanic Black	1769 (19.6%)	1713 (20.6%)	56 (7.8%)	
Mexican American	1493 (16.6%)	1402 (16.9%)	91 (12.7%)	
Other	1646 (18.3%)	1561 (18.8%)	85 (11.8%)	
Education level, *n* (%)				
Less than high school	2651 (29.4%)	2466 (29.7%)	185 (25.8%)	0.133
High school	2133 (23.7%)	1962 (23.6%)	171 (23.8%)	
More than high school	4217 (46.8%)	3856 (46.5%)	361 (50.3%)	
Missing	15 (0.2%)	14 (0.2%)	1 (0.1%)	
Marital status, *n* (%)				< 0.001
Married or living with partner	5153 (57.2%)	4728 (57.0%)	425 (59.2%)	
Divorced or separated	1963 (21.8%)	1729 (20.8%)	234 (32.6%)	
Never married	1474 (16.3%)	1426 (17.2%)	48 (6.7%)	
Missing	426 (4.7%)	415 (5.0%)	11 (1.5%)	
Family PIR	2.5 ± 1.9	2.5 ± 1.9	2.7 ± 1.8	0.005
BMI (kg/m^2)				0.006
< 18.5	166 (1.8%)	159 (1.9%)	7 (1.0%)	
18.5–24.9	2597 (28.8%)	2419 (29.2%)	178 (24.8%)	
25.0–29.9	3033 (33.6%)	2790 (33.6%)	243 (33.8%)	
≥ 30.0	3220(35.7%)	2930 (35.3%)	290 (40.4%)	
Smoking, *n* (%)				0.366
Yes	4615 (51.2%)	4242 (51.1%)	373 (51.9%)	
No	4339 (48.1%)	3996 (48.2%)	343 (47.8%)	
Missing	62 (0.7%)	60 (0.7%)	2 (0.3%)	
Alcohol use, *n* (%)				< 0.001
Former	1553 (17.2%)	1405 (16.9%)	148 (20.6%)	
Current	5304 (58.8%)	4929 (59.4%)	375 (52.2%)	
Never	1185 (13.1%)	1052 (12.7%)	133 (18.5%)	
Missing	974 (10.8%)	912 (11.0%)	62 (8.6%)	
Hypertension, *n* (%)				< 0.001
Yes	3045 (33.8%)	2687 (32.4%)	358 (49.9%)	
No	5971 (66.2%)	5611 (67.6%)	360 (50.1%)	
Diabetes, *n* (%)				< 0.001
Yes	1085 (12.0%)	950 (11.4%)	135 (18.8%)	
No	7931 (88.0%)	7348 (88.6%)	583 (81.2%)	
Sedative or hypnotic medications				< 0.001
Yes	517 (5.7%)	430 (5.2%)	87 (12.1%)	
No	8499 (94.3%)	7868 (94.8%)	631 (87.9%)	
Sleep duration (hours), *n* (%)				< 0.001
< 7	3557 (39.5%)	3314 (39.9%)	243 (33.8%)	
7–9	5220 (57.9%)	4774 (57.5%)	446 (62.1%)	
> 9	239 (2.7%)	210 (2.5%)	29 (4.0%)	
Self-reported trouble sleeping, *n* (%)				< 0.001
Yes	2079 (23.1%)	1830 (22.1%)	249 (34.7%)	
No	6937 (76.9%)	6468 (77.9%)	469 (65.3%)	
Self-reported sleep disorder, *n* (%)				< 0.001
Yes	687 (7.6%)	599 (7.2%)	88 (12.3%)	
No	8329 (92.4%)	7699(92.8%)	630 (87.7%)	

Table 1 (continued)

Variable	Total ($n=9016$)	Non- Hypothyroidism ($n=8298$)	Hypothyroidism ($n=718$)	*P*-value
Sleep patterns, *n* (%)				< 0.001
Healthy sleep	4456 (49.4%)	4133 (49.8%)	323 (45.0%)	
Intermediate sleep	3107 (34.5%)	2865 (34.5%)	242 (33.7%)	
Poor sleep	1453 (16.1%)	1300 (15.7%)	153 (21.3%)	
TSH (mIU/mL)	2.1 ± 4.2	1.8 ± 1.0	5.6 ± 13.9	< 0.001
fT4 (ng/dL)	0.8 ± 0.2	0.8 ± 0.1	0.9 ± 0.3	< 0.001
fT3 (pg/mL)	3.2 ± 0.5	3.2 ± 0.5	2.9 ± 0.4	< 0.001
TPOAb (IU/mL)	22.5 ± 208.0	14.4 ± 74.1	115.4 ± 686.4	< 0.001
TgAb (IU/mL)	11.6 ± 101.3	6.5 ± 67.4	70.9 ± 269.4	< 0.001
Thyroglobulin	16.7 ± 60.5	16.7 ± 61.3	16.4 ± 50.7	0.885
Triglycerides (mg/dL)	157.1 ± 132.2	155.9 ± 133.6	171.1 ± 114.0	0.003
Cholesterol (mg/dL)	194.6 ± 41.9	194.3 ± 42.0	197.9 ± 41.7	0.028
HDL-C (mg/dL)	52.1 ± 15.7	51.9 ± 15.6	53.7 ± 16.3	0.003
Albumin (g/L)	4.2 ± 0.3	4.3 ± 0.3	4.2 ± 0.3	< 0.001
Uric acid (mg/dL)	5.5 ± 1.5	5.5 ± 1.5	5.6 ± 1.5	0.195
Creatinine (mg/dL)	0.9 ± 0.4	0.9 ± 0.4	0.9 ± 0.5	0.008
Serum glucose (mmol/L)	5.7 ± 2.1	5.6 ± 2.1	6.0 ± 2.2	< 0.001
Serum iron (μg/dL)	85.4 ± 36.3	85.8 ± 36.5	80.5 ± 32.6	< 0.001

Abbreviations: PIR, poverty income ratio; BMI, body mass index; TSH, thyroid-stimulating hormone; fT4, free thyroxine; fT3, free triiodothyronine; TPOAb, thyroid peroxidase antibody; TgAb, thyroglobulin antibody; HDL-C, high density lipoprotein-cholesterol

abnormalities. Sleep disorders, including insomnia, OSA, RLS, narcolepsy et al., are a group of conditions that disturb normal sleep patterns [18], and trouble sleeping is more likely to represent participants' subjective assessment of their own sleep status, both of which mean the loss of normal sleep patterns. However, sleep duration seemed to be unrelated to the increased odds of hypothyroidism. Neither short nor long sleep duration increased the prevalence of hypothyroidism. In addition, among sleep patterns that accounted for the combined effects of the three sleep factors, poor sleep pattern was still associated with increased prevalence of hypothyroidism after adjusting for relevant confounding variables.

At present, many epidemiological studies have investigated risk factors related to hypothyroidism, including genetic and ethnic susceptibilities [19], ageing [20] and sex [21]. Recently, the association between sleep and hypothyroidism has also been investigated [9, 22–24]. Based on our results, we demonstrated significant association between trouble sleeping, sleep disorders, and an increased prevalence of hypothyroidism. Although we cannot easily infer the potential mechanism underlying the association between sleep abnormalities and hypothyroidism, we can propose explanations for this phenomenon.

Firstly, sleep may strongly affects the production of thyroid hormones [12]. TSH is significantly influenced by circadian rhythms [25, 26], and the regulation of this rhythm is also influenced by sleep, including sleep quality and sleep duration [12, 26]. Recent studies have revealed that sleep deprivation could affect TSH secretion [27, 28]. For example, night shifts may cause acute sleep deprivation, and there is research supporting that night shift work increases the risk of hypothyroidism [29]. Kim et al. [11] suggested that compared to people with normal sleep duration (7–8 h/day), those with longer sleep (>8 h/day) have an increased risk of subclinical hyperthyroidism and hypothyroidism. Although this is inconsistent with our findings, it may be due to differences in sample selection, adjustment covariates, and definitions of normal sleep duration. In addition, Wang et al. [12] suggested that when sleep duration was less than or equal to 7 h, FT3 levels were significantly negatively correlated with sleep duration. The above studies focused on the effect of sleep duration on thyroid hormones or thyroid function. Although few studies have investigated the pathophysiological mechanism linking sleep quality and thyroid hormone release rhythm, based on the existing research results, we speculate that trouble sleeping and sleep disorders may interfere with the normal release of TSH or FT3, and thereby increasing the odds of hypothyroidism.

In addition, based on reported research, we speculate that immunity [10, 30, 31], mood changes [32–35], stress [36–38] and metabolism [33, 39–41] related to poor sleep may provide insights into the potential mechanisms by which sleep abnormalities are associated with the development of hypothyroidism. However, we are currently unable to provide more evidence to confirm, and further experiments and observations are needed for verification.

Previous research mainly focused on sleep disorders caused by hypothyroidism, in this analysis of cross-sectional data based on NHANES, we have paid attention to the role of sleep duration, trouble sleeping and sleep

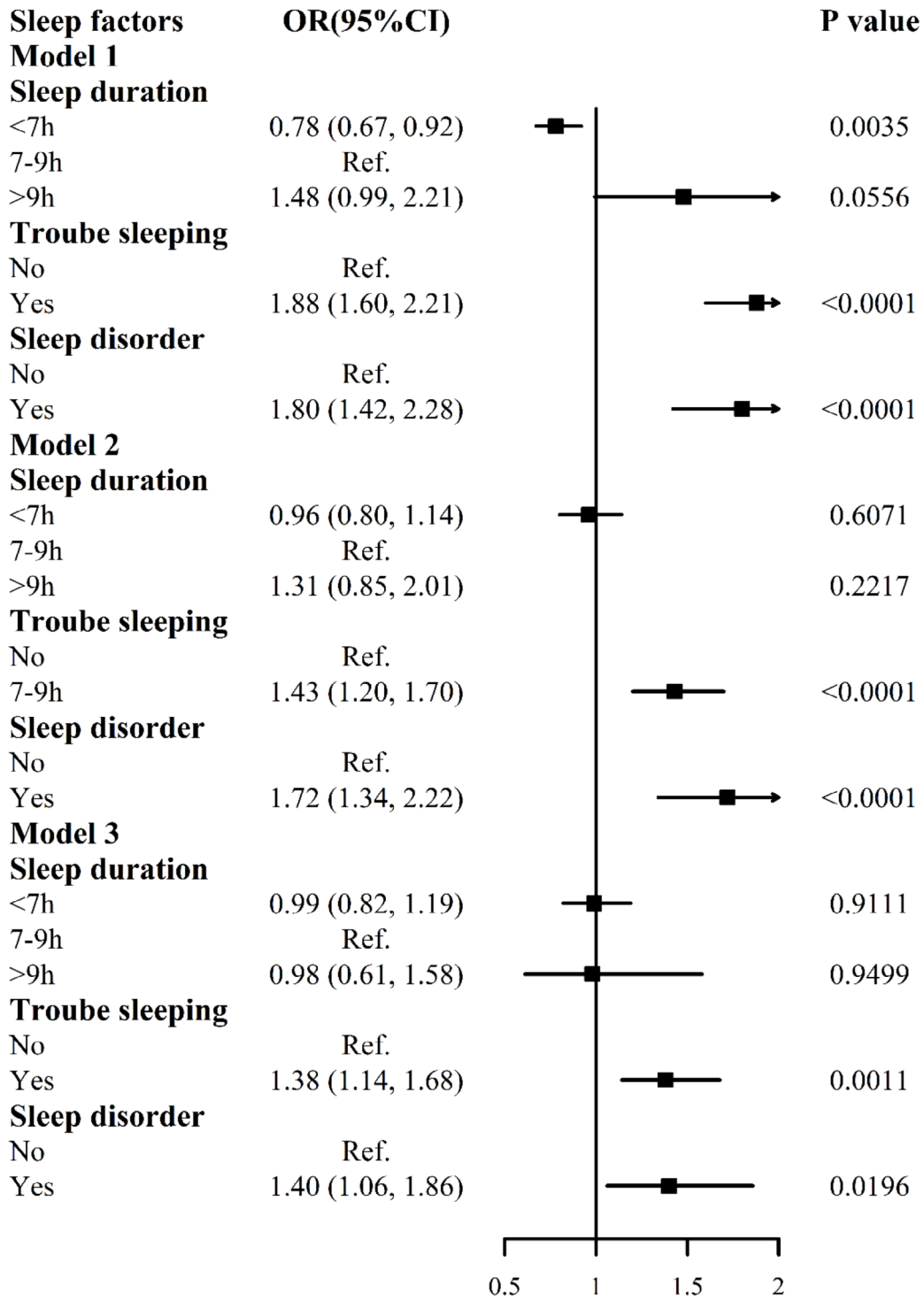

Fig. 2 Logistic regression analyses of the association between sleep factors and hypothyroidism. Model 1 was an unadjusted model. Model 2 was adjusted for age, sex, race, education level, marital status and family PIR. Model 3 was adjusted for the variables in model 2 plus body mass index (BMI), smoking habit; alcohol use, hypertension, diabetes, sedative or hypnotic medications, thyroid peroxidase antibody (TPOAb), thyroglobulin antibody (TgAb), free triiodothyronine (fT3), free thyroxine (fT4), triglyceride, cholesterol, high density lipoprotein-cholesterol (HDL-C), uric acid, creatinine, serum glucose and serum iron levels

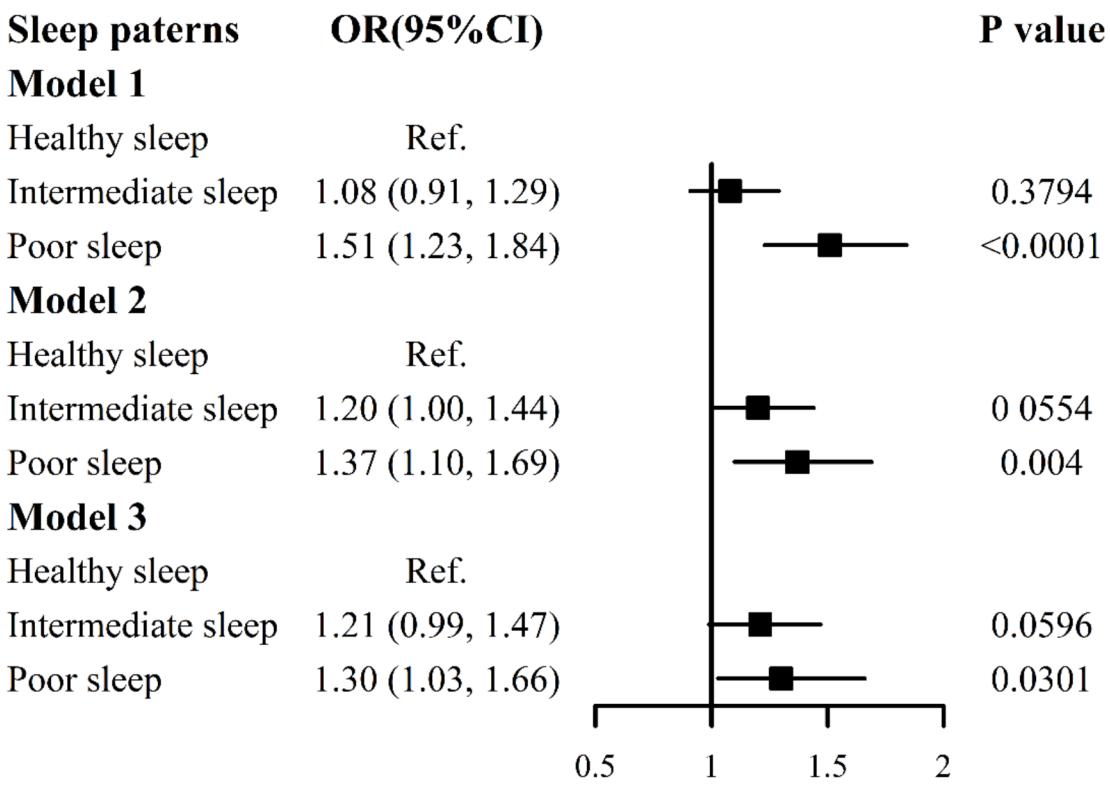

Fig. 3 Logistic regression analyses of the association between sleep patterns and hypothyroidism. Model 1 was an unadjusted model. Model 2 was adjusted for age, sex, race, education level, marital status and family PIR. Model 3 was adjusted for the variables in model 2 plus BMI, smoking habit, alcohol use, hypertension, diabetes, sedative or hypnotic medications, TPOAb, TgAb, fT3, fT4, triglyceride, cholesterol, HDL-C, uric acid, creatinine, serum glucose and serum iron levels

disorders in the hypothyroidism. In addition, we consider the role of overall sleep quality—sleep patterns that integrate sleep duration, trouble sleeping, and sleep disorders—in hypothyroidism for the first time. We believe that people who have sleep abnormalities such as trouble sleeping, sleep disorders, or overall poor sleep quality, need to pay attention to their thyroid function status. Meanwhile, our research findings may provide a new approach for the prevention and treatment of hypothyroidism. Our study also has several limitations. First, this was a cross-sectional analysis based on the NHANES database, and the results do not determine the causal relationship between sleep abnormalities and hypothyroidism. Because the association between sleep and hypothyroidism seems to be bidirectional, more in-depth research is required to better understand the extent to which sleep affects or is affected by hypothyroidism. Second, sleep-related data were collected through questionnaire surveys. Compared with sleep monitoring, this is a subjective method and may have memory bias, and any observed association may be due to participants' biased memories. Limited data were collected on sleep duration and referred only to workdays; data on the specific time to fall asleep was not provided, which may have affected the accuracy of the results of this study. Third, we did not examine the relationship between specific types of sleep disorders and hypothyroidism because detailed information cannot be obtained from the questionnaire. Therefore, the results of our analysis may attenuate or exaggerate the association between specific types of sleep disorders and hypothyroidism. Finally, although the sample size itself is not small, the number of participants in some different exposure categories is relatively low, which leads to a wide confidence interval for risk estimation and may have a certain impact on the authenticity of the results. Future research on this topic should ensure adequate numbers of cases and controls, objective monitoring of sleep, and long-term follow-up of participants.

In conclusion, in this analysis of cross-sectional data based on the NHANES from 2007 to 2012, trouble sleeping, sleep disorders, and poor sleep pattern were association with increased odds of hypothyroidism. More prospective cohort research should be conducted to investigate the causal or bidirectional association

Table 2 Multivariate association between hypothyroidism and sleep patterns – Interaction and stratified analysis

Subgroup	OR (95%CI)			*P* for interaction
	Healthy sleep pattern	Intermedia sleep pattern	Poor sleep pattern	
Age (years)				0.8852
< 50	Ref.	1.16 (0.92, 1.47)	1.25 (0.95, 1.65)	
≥ 50	Ref.	1.13 (0.77, 1.64)	1.19 (0.72, 1.98)	
Sex				0.6470
Male	Ref.	1.28 (0.90, 1.82)	1.17 (0.75, 1.81)	
Female	Ref.	1.18 (0.92, 1.50)	1.41 (1.05, 1.89)	
Race/Ethnicity				0.5120
Non-Hispanic White	Ref.	1.22 (0.95,1.57)	1.28 (0.94,1.74)	
Non-Hispanic Black	Ref.	1.11 (0.56,2.20)	1.27 (0.59,2.72)	
Mexican American	Ref.	0.77 (0.43,1.35)	1.04 (0.51,2.13)	
Other	Ref.	1.96 (1.13,3.40)	2.02 (1.01,4.06)	
BMI (kg/m^2)				0.9152
< 25	Ref.	1.25 (0.86, 1.83)	1.35 (0.79, 2.31)	
25–29.9	Ref.	1.10 (0.78, 1.55)	1.39 (0.90, 2.12)	
≥ 30	Ref.	1.27 (0.92, 1.76)	1.28 (0.90, 1.83)	
Hypertension				0.6705
Yes	Ref.	1.32 (1.01, 1.72)	1.25 (0.87,1.82)	
No	Ref.	1.06 (0.78, 1.43)	1.30 (0.94, 1.81)	
Diabetes				0.3066
Yes	Ref.	1.17 (0.94, 1.46)	1.44 (1.10, 1.87)	
No	Ref.	1.29 (0.77, 2.15)	0.84 (0.47, 1.50)	

Each stratification adjusted for all covariates (sex, race, education level, marital status, family PIR, BMI, smoking habit, alcohol use, hypertension, diabetes, Sedative or hypnotic medications, TPOAb, TgAb, fT3, fT4, triglyceride, cholesterol, HDL-C, uric acid, creatinine, serum glucose and serum iron levels) except the stratification variable itself

between sleep abnormalities and the risk of hypothyroidism. In addition, studying the potential mechanisms associated with sleep and hypothyroidism is essential for the effective prevention and treatment of hypothyroidism.

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Author contributions
Y.D. wrote the main manuscript text, Y.P. and J.Z. designed research methods, X.P. prepared research materials, X.H. conducted statistical analysis, C.Z. reviewed the manuscript text and supervised this study.

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Data availability
The datasets generated and/or analysed during the current study are available in the NHANES repository, https://www.cdc.gov/nchs/nhanes/index.htm.

Declarations

Ethics approval and consent to participate
Not applicable. The NHANES programme was authorised by the Ethics Review Committee of the NCHS in the US, and all participants voluntarily participated and provided informed consent. Our secondary analysis did not require additional ethics approval.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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