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Evaluating sleep's role in type 2 diabetes mellitus: Evidence from NHANES

Jijun Zhang [a,1], Chuanli Yang [b,c,1], Jie An [c], Yunhe Fan [c], Xiushan Dong [a,*]

[a] Third Hospital of Shanxi Medical University, Shanxi Bethune Hospital, Shanxi Academy of Medical Sciences, Tongji Shanxi Hospital, Taiyuan, 030032, China
[b] Key Laboratory of Environmental Medical Engineering and Education Ministry, School of Public Health, Southeast University, Nanjing, Jiangsu, China
[c] Department of General Surgery, Shanxi Bethune Hospital, Shanxi Academy of Medical Sciences, Tongji Shanxi Hospital, Third Hospital of Shanxi Medical University, Taiyuan, 030032, China

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ABSTRACT

Background: Evidence is limited regarding the relationship between sleep factors (self-reported sleep disorder diagnosis, subjective sleep difficulties, and sleep duration), sleep patterns, and risk of type 2 diabetes mellitus (T2D). Thus, this study aims to investigate the relationship between sleep factors, sleep patterns, and the risk of T2D using data from the National Health and Nutrition Examination Survey (NHANES).

Methods: A total of 14,652 individuals aged ≥18 years from the NHANES (2005–2014) were enrolled with complete data on sleep factors, T2D, and covariates. Information on self-reported sleep disorder diagnosis, subjective sleep difficulties, and sleep duration was collected during in-home visits by trained interviewers using the Computer-Assisted Personal Interviewing system. The sleep pattern was derived from scoring three mentioned factors: no self-reported sleep disorder diagnosis, no subjective sleep difficulties, and sleep duration of 7–9 h were classified as low-risk (score 0), while the presence of self-reported sleep disorder diagnosis, subjective sleep difficulties, or sleep duration <7 or >9 h were classified as high-risk (score 1). Cumulative scores range from 0 to 3, with 0 indicating a healthy sleep pattern, 1 an intermediate sleep pattern, and 2–3 a poor sleep pattern, respectively. Weighted logistic regression was conducted to assess the association of sleep factors and sleep patterns with the risk of T2D.

Results: Self-reported sleep disorder diagnosis (odds ratio (OR) = 1.32, $P = 0.01$), subjective sleep difficulties (OR = 1.29, $P = 0.001$), and sleep deprivation (<7 h; OR = 1.20, $P = 0.01$) were significantly positive with T2D. Poor sleep pattern also significantly increased T2D risk (OR = 1.52, $P < 0.0001$). Moreover, subgroup analyses stratified by age and BMI (body mass index) further confirmed that the positive association between sleep patterns and T2D was consistent and robust across groups.

Conclusion: Our findings indicate that poorer sleep patterns are associated with an increased risk of T2D. These results emphasize the importance of sleep management in T2D prevention. Further prospective studies are needed to investigate the causal or bidirectional relationship between sleep and T2D risk, as well as the underlying molecular mechanisms.

1. Introduction

Sleep is a highly complicated process occupying approximately one-third of the human lifespan (Regier et al., 2012). Although sleep is regarded as a passive state, it is a highly dynamic and active process (Besedovsky et al., 2019). According to the National Heart, Lung, and Blood Institute, an estimated 50 to 70 million U.S. adults suffer from a sleep disorder or reported sleep deprivation (St-Onge et al., 2016). Common sleep disorders, such as insomnia, sleep apnea, and restless legs syndrome, require professional diagnosis by a physician or healthcare provider. Beyond sleep disorders, self-reported trouble sleeping is also a critical indicator of sleep quality. A healthy sleep pattern is not only about having the appropriate sleep duration but also involves the entire sleep process, such as falling asleep within a proper time, without snoring, without obstructive sleep apnea, and restless legs syndrome (Petrov et al., 2014; Nelson et al., 2022). Sleep is vital for restoring brain function and growing evidence suggests that sleep is associated with type 2 diabetes mellitus (T2D)(Magnavita et al., 2017). However, previous studies have mainly focused on the relationship between sleep duration and T2D. There has yet to be a comprehensive evaluation of

* Corresponding author. Address: No.99 Longcheng Street, Shanxi Bethune Hospital, China.
E-mail address: dongxiushan2012@163.com (X. Dong).
[1] These authors have contributed equally to this work.

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how single sleep factors and sleep patterns (encompassing self-reported sleep disorder diagnosis, self-reported sleep difficulties, and sleep duration), relate to T2D.

T2D is a global health problem of great severity and rising prevalence. It is estimated that by 2045, approximately 700 million people will have diabetes (Wu et al., 2014). One study indicated that sleep insufficiency may contribute to the development of T2D (McNeil et al., 2013). In addition, a prospective study demonstrated significant associations between both excessive and insufficient sleep duration and T2D (Shan et al., 2015). Besides sleep duration, sleep disorders and trouble sleeping may also as risk factors for diabetes (Anothaisintawee et al., 2016). Mechanistically, insufficient sleep disrupts the normal secretion of various hormones, including leptin, ghrelin, and glucagon, among others. This disruption not only affects the secretion and function of insulin but also directly impairs insulin sensitivity, thereby exacerbating insulin resistance. Furthermore, sleep deprivation leads to elevated levels of inflammatory cytokines such as interleukin-6 (IL-6) and C-reactive protein (CRP), further increasing the risk of insulin resistance. (Reutrakul et al., 2018; Tang et al., 2014). However, most studies focus on single sleep factors (particularly sleep duration) rather than combined sleep behaviors, and the findings are often inconsistent due to differences in sample size and methodologies.

The present study investigated the association between single sleep factors (self-reported sleep disorder diagnosis, subjective sleep difficulties, and sleep duration), sleep patterns, and the risk of T2D in a representative sample of the US population. Additionally, we aimed to explore whether these relationships remain consistent across subgroups stratified by age and BMI.

2. Methods

2.1. Study subjects

The National Health and Nutrition Examination Survey (NHANES) is a complex multistage sample survey being conducted by the U.S. government. It determines the physical health status of the entire U.S. population by administering health questionnaires, physical examinations, and laboratory tests to a representative and specific group of people to guide people toward healthier lifestyles.

As illustrated in Fig. 1, we analyzed NHANES data from 2005 to 2014. We excluded 20,670 participants who were under 18 years old, as our study focused on adults. Additionally, 1067 participants without Mobile Examination Center (MEC) data were excluded due to incomplete health assessments. From the sleep questionnaire data, we excluded 43 participants missing self-reported sleep disorder diagnosis data, 7 participants missing subjective sleep difficulties data, and 50 participants missing sleep duration data. Furthermore, we excluded 2773 participants who lacked a confirmed T2D diagnosis. Finally, 11,703 participants with missing critical covariate data, such as BMI, dietary quality, or physical activity, were also excluded. After these exclusions, 14,652 participants were included in the final analysis.

2.2. Sleep factors and sleep patterns

Self-reported sleep disorder diagnosis, subjective sleep difficulties, and sleep duration were evaluated based on responses to the questions on the sleep questionnaire (SLQ). In the current study, to identify

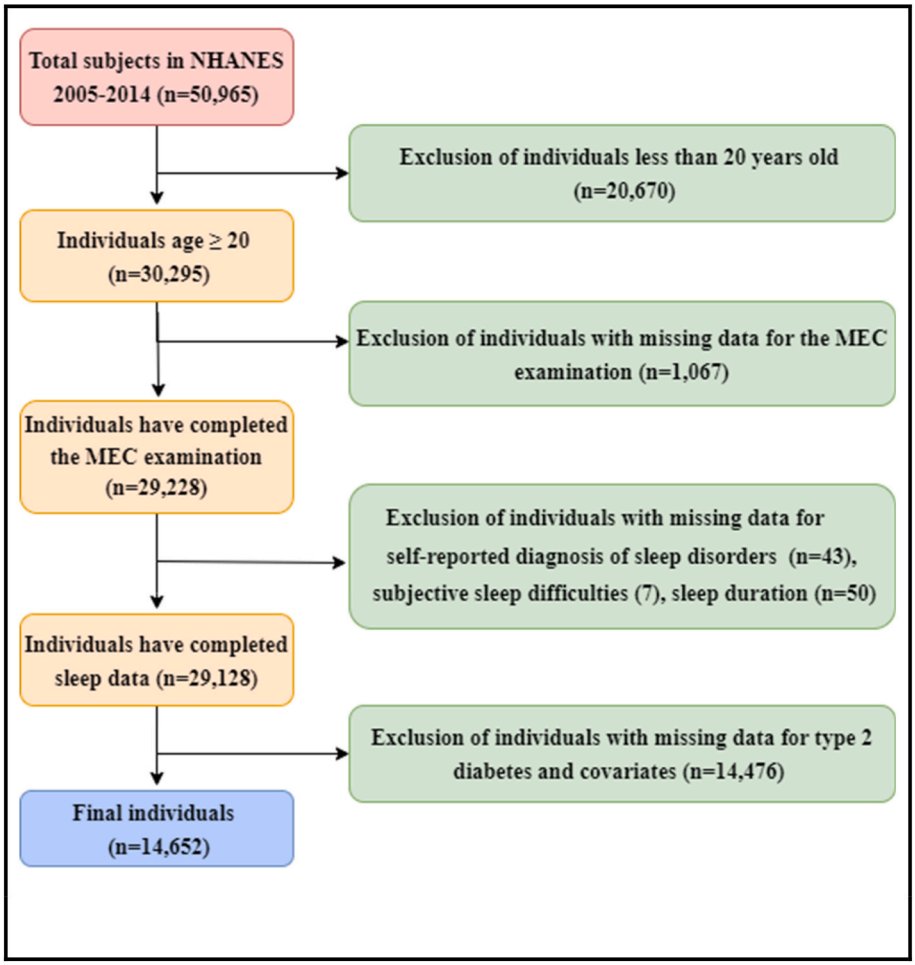

Fig. 1. Participants selection flowchart.

whether participants have self-reported sleep disorder diagnosis, whether they have subjective sleep difficulties, and their sleep duration, we used the NHANES variables SLQ060 ("Have you ever been told by a doctor or other health professional that you have a sleep disorder?"), SLQ050 ("Have you ever told a doctor or other health professional that you have trouble sleeping?"), and SLD010h ("How much sleep do you usually get at night on weekdays or workdays?"), respectively. Sleep duration was divided into three groups: <7, 7–9, and >9 h of sleep. No self-reported sleep disorder diagnosis, no subjective sleep difficulties, and sleep duration of 7–9 h are considered low-risk sleep factors and assigned a score of 0; the rest are considered high-risk sleep factors and assigned a score of 1. The three cumulative scores generate a total score ranging from 0 to 3. The sleep patterns can be determined based on this total score: a score of 2–3 indicates a poor sleep pattern, 1 indicates an intermediate sleep pattern, and 0 indicates a healthy sleep pattern.

2.3. T2D

T2D was diagnosed by a doctor. Medications to reduce blood glucose levels were provided. Fasting blood glucose was equal to or greater than 7.0 mmol/L, glycosylated hemoglobin was equal to or greater than 6.5%, and a 2-h blood glucose level was equal to or greater than 11.1 mmol/L from an oral glucose tolerance test.

2.4. Covariates

The covariates in this study were age, gender, ethnicity, educational level, marital status, Healthy Eating Index (HEI) 2015, poverty-to-income ratio (PIR), physical activity, body mass index (BMI), energy intake, smoking status, alcohol drinking status, hypertension, immune inflammation index (SII), and Patient Health Questionnaire (PHQ-9). Alcohol drinking status was classified into four groups: no, mild, moderate, and heavy. Smoking status was categorized as either smoking or nonsmoking. PIR was categorized with a cutoff of 2. BMI was classified into three groups: <25.0, 25.0–29.9, and ≥30.0 kg/m^2. Marital status was categorized as living with a partner or married, separated or divorced or widowed, and never married. Education was categorized as less than high school, high school or General Education Development (GED), and more than high school. Ethnicity was classified as non-Hispanic Black, non-Hispanic White, Mexican Americans, and other races. Age was divided into three groups: 20–39, 40–59, and ≥60 years. Physical activity was measured using metabolic equivalent and classified into four groups. SII is a novel marker derived from lymphocyte, neutrophil, and platelet counts, and is commonly used to assess systemic inflammation. Due to the SII distribution not conforming to the normal distribution we have log-transformed. In addition, we included the PHQ-9 as a covariate to assess depressive symptoms.

2.5. Statistical analysis

In the current study, NHANES survey weights, strata, and primary sampling units were considered to ensure that nationally representative estimates were calculated. Sample weights for the mobile examination center were used. Continuous variables such as age, HEI-2015 score, energy intake, and SII were presented as mean ± standard error (SE). Prior to statistical analysis, tests for normality and homogeneity of variance were conducted. If these assumptions were met, *t*-tests (for comparisons between groups with and without T2D) or one-way ANOVA (for comparisons among the three sleep patterns) were performed. Categorical variables were expressed as N (%) and assessed for statistical differences using the χ^2 test. To assess associations between variables and T2D, multivariate logistic regression was performed, and results were reported as odds ratios (OR) with 95% confidence intervals (CI). Before the regression analysis, the linear relationship between the logit-transformed continuous independent variables and T2D was confirmed, and multicollinearity among predictors was assessed. Three models were constructed for the logistic regression analysis: Model 1 was the unadjusted model. Model 2 was adjusted for gender, age, and ethnicity. Model 3 was adjusted for gender, age, ethnicity, marital status, BMI, PIR, smoking, alcohol consumption, physical activity, HEI-2015 index, hypertension, SII, PHQ-9. P value < 0.05 was considered statistically significant. All analyses in this study were performed using R software (version 4.2.2).

3. Results

3.1. Demographics of the study population

The characteristics of the participants, classified according to T2D status, are presented in Table 1. Of the total 14,652 participants, 6895 (47.06%) were female and 7757 (52.94%) were male, with a mean age of 44.95 ± 0.31 years. The overall weighted prevalence of T2D was 15.86%. Age, BMI, physical activity, energy intake, ethnicity, education, marital status, smoking, alcohol consumption, hypertension, PIR, SII, PHQ-9, self-reported sleep disorder diagnosis, subjective sleep difficulties, and sleep duration were significantly statistically different ($P <$ 0.0001) between those with and without T2D.

To examine the demographic characteristics of participants with different sleep patterns, we categorized them into three groups: healthy sleep, intermediate sleep, and poor sleep. Individuals with poor sleep patterns appeared to be older, more likely to be female, non-Hispanic White, have lower educational levels, live alone, and have poorer dietary quality, lower incomes, trends of obesity, and hypertension (Table S1). Furthermore, T2D prevalence was significantly higher in participants with poor sleep pattern (19.93%) compared to intermediate (10.75%) and healthy sleep patterns (9.31%) ($P < 0.0001$) (Table S1).

3.2. Link between sleep factors and risk of T2D

As presented in Table 2, in the unadjusted model (Model 1), participants with self-reported sleep disorder diagnosis, subjective sleep difficulties, and sleep duration <7 h had 2.51 (95% CI: 2.12–2.98), 1.73 (95% CI: 1.53–1.96) and 1.34-fold (95% CI: 1.20–1.50) more likely to have T2D, respectively. In the fully adjusted model (Model 3), self-reported sleep disorder diagnosis (OR = 1.32, $P = 0.01$) and subjective sleep difficulties (OR = 1.29, $P = 0.001$) remained significantly associated with the risk of T2D compared with the corresponding reference (Table 2). Compared to a sleep duration of 7–9 h, sleeping <7 h (OR = 1.20, P = 0.01) was associated with a higher risk of T2D, while sleeping ≥9 h (OR = 1.47, P = 0.13) showed no statistically significant association (fully adjusted model). These findings suggest that sleep deprivation may pose a greater risk for T2D and should be given more attention.

3.3. Association between sleep patterns and the risk of T2D

As demonstrated in Table 3, in the crude model (Model 1), a poor sleep pattern (OR = 2.42, $P < 0.0001$) and a intermediate sleep pattern (OR = 1.17, $P = 0.02$) were positively associated with the risk of T2D compared with a healthy sleep pattern. In the fully adjusted model (Model 3), this relationship between a poor sleep pattern and T2D remained robust (OR = 1.52, $P < 0.0001$), but no significant differences were observed between a intermediate sleep pattern and T2D.

3.4. Link between sleep patterns with risk of T2D by age and BMI

When categorized by age, poor sleep pattern were consistently associated with a significantly increased risk of constipation. Among individuals aged 40–59 years, the risk was highest (OR = 1.852, $P <$ 0.0001). The *P*-trend analysis indicated that the risk of constipation increased significantly with worsening sleep patterns in both the 40–59 years and ≥60 years age groups (*P*-trend <0.0001) (Table 4). Stratified

Table 1
Demographics of participants aged ≥20 years according to T2D.

Characteristics	Total (N = 14,652)	Without T2D (N = 12,328)	With T2D (N = 2324)	*P*-value
Age, (years)	44.95 ± 0.31	43.34 ± 0.32	57.29 ± 0.36	<0.0001
Gender, N (%)				0.05
Female	6895 (47.06)	5862 (48.73)	1033 (45.83)	
male	7757 (52.94)	6466 (51.27)	1291 (54.17)	
Ethnicity, N (%)				<0.0001
Non-Hispanic Black	3004 (20.5)	2411 (9.56)	593 (14.15)	
Non-Hispanic White	7178 (48.99)	6248 (73.21)	930 (65.00)	
Mexican American	2042 (13.94)	1642 (6.95)	400 (8.77)	
Others	2428 (16.57)	2027 (10.28)	401 (12.07)	
Education, N (%)				<0.0001
Less than high school	2994 (20.43)	2323(12.82)	671 (18.74)	
High school or GED	3323 (22.68)	2750 (21.54)	573 (26.63)	
More than high school	8335 (56.89)	7255 (65.64)	1080 (54.63)	
Marital status, N (%)				<0.0001
Living with partner or Married	8791 (60)	7352 (63.53)	1439 (65.95)	
Separated or Divorced or Widowed	2889 (19.72)	2216 (15.52)	673 (25.87)	
Never married	2972 (20.28)	2760 (20.95)	212 (8.18)	
HEI-2015 score	51.23 ± 0.23	51.16 ± 0.25	51.72 ± 0.41	0.17
PIR, N (%)				<0.0001
< 2	6460 (44.09)	5311 (30.96)	1149 (36.30)	
≥ 2	8192 (55.91)	7017 (69.04)	1175 (63.70)	
Physical activity (MET-h/week)				<0.0001
Q1 (≤560)	371 5 (25.35)	2997 (24.39)	718 (30.38)	
Q2 (560, 1680]	3875 (26.45)	3179 (26.17)	696 (31.33)	
Q3 (1680, 4800]	3552 (24.24)	3041 (25.47)	511 (21.72)	
Q4 (>4800)	3510 (23.96)	3111 (23.97)	399 (16.57)	
BMI, N (%), (kg/m^2)				<0.0001
< 25	4666 (31.85)	4346 (36.22)	320 (12.69)	
25–29.9	4973 (33.94)	4266 (34.98)	707 (27.93)	
≥ 30	5013 (34.21)	3716 (28.80)	1297 (59.38)	
Energy intake, (kcal)	2254.37 ± 12.32	2279.53 ± 12.57	2061.12 ± 27.40	<0.0001
Smoke, N (%)				<0.0001
No	11,446 (78.12)	9501 (77.98)	1945 (84.85)	
Yes	3206 (21.88)	2827 (22.02)	379 (15.15)	
Alcohol consumption, N (%)				<0.0001
No	4073 (27.8)	3076 (20.88)	997 (36.92)	
Mild	4961 (33.86)	4202 (36.15)	759 (37.10)	
Hypertension, N (%)				<0.0001
No	9207 (62.84)	8472 (71.69)	735 (32.88)	
Yes	5445 (37.16)	3856 (28.31)	1589 (67.12)	
SII	6.17 ± 0.01	6.16 ± 0.01	6.22 ± 0.01	<0.0001
PHQ-9, N (%)				<0.0001
[0,9]	13550 (93.71)	11482 (94.25)	2068 (90.75)	
[10,27]	1068 (6.14)	818 (5.75)	250 (9.25)	
Self-reported sleep disorder diagnosis, N (%)				<0.0001
No	13,509 (92.2)	11,507 (93.31)	2002 (84.72)	
Yes	1143 (7.8)	821(6.69)	322 (15.28)	
Subjective sleep difficulties, N (%)				<0.0001
No	11,133 (75.98)	9577 (75.92)	1556 (64.57)	
Yes	3519 (24.02)	2751 (24.08)	768 (35.43)	
Sleep time, hours N (%)				<0.0001
7-9	8634 (58.93)	7382 (63.39)	1252 (56.19)	
< 7	5741 (39.18)	4721 (35.02)	1020 (41.72)	
≥ 10	2,77 (1.89)	225 (1.59)	52 (2.08)	

Continuous variables were presented as mean ± SE; Categorical variables expressed as N (%); *t*-test and χ^2 test were performed to assess statistical differences between participants without T2D and with T2D.
BMI: body mass index; PIR: poverty income ratio, HEI-2015: Healthy Eating Index-2015; MET: metabolic equivalent; SII: immune inflammation index; PHQ-9: Patient Health Questionnaire; Q: quartile.

by BMI, poor sleep pattern were associated with a significantly increased risk of constipation only in individuals with BMI of 25–29.9 (OR = 1.475, $P = 0.040$) and BMI ≥30 kg/m2 (OR = 1.659, $P < 0.0001$). The *P*-trend analysis further revealed that the risk of constipation increased significantly with worsening sleep patterns in individuals with a BMI ≥30 (*P*-trend <0.0001) (Table 5).

4. Discussion

The present study investigated the correlation between sleep factors, sleep patterns, and T2D in a nationally representative U.S. population aged 20 years or older. We demonstrated that self-reported sleep disorder diagnosis, subjective sleep difficulties, sleep duration <7 h, and poor sleep patterns were associated with an increased risk of T2D. Then we performed subgroup analyses and observed that poor sleep pattern was associated with a significantly higher risk of T2D in all age groups and in individuals with BMI <25 and BMI≥ 30 kg/m^2.

T2D presents with impaired glucose metabolism in the liver and functional defects in the number and function of β-cells, resulting in inadequate insulin secretion and insulin resistance (Butler et al., 2003; Skyler et al., 2017). We can improve individuals' quality of life by addressing the risk factors for T2D to prevent its onset and reduce its effects. Sleep plays a crucial role in restoring endocrine, immune, and neurological functions and largely reflects overall human health (Chaput et al., 2023; Krueger et al., 2016; Irwin, 2019). We observed that individuals who slept <7 h exhibited a significantly higher risk of developing T2D compared to those who slept 7–9 h, which is consistent with previous findings. Holliday EG et al. reported that individuals sleeping <6 h per night had a 30% increased risk of T2D compared to those sleeping 7 h per night (Holliday et al., 2013). A meta-analysis of 447,124 participants also confirmed the association between short sleep duration and T2D incidence (RR = 1.33, 95% CI: 1.20–1.48) (Cappuccio et al., 2010). However, the relationship between long sleep duration and T2D risk remains controversial. In our study, no significant association

Table 2
Logistic regression analysis of sleep factors and T2D.

Model	Sleep factors	OR (95% CI)	*P*-value
Model 1	Self-reported sleep disorder diagnosis		
	No	ref	ref
	Yes	2.51 (2.12, 2.98)	<0.0001
	Subjective sleep difficulties		
	No	ref	ref
	Yes	1.73 (1.53, 1.96)	<0.0001
	Sleep time		
	7–9	ref	ref
	<7	1.34 (1.20, 1.50)	<0.0001
	≥10	1.48 (0.99, 2.21)	0.05
Model 2	Self-reported sleep disorder diagnosis		
	No	ref	ref
	Yes	2.32 (1.92, 2.80)	<0.0001
	Subjective sleep difficulties		
	No	ref	ref
	Yes	1.66 (1.45, 1.90)	<0.0001
	Sleep time		
	7–9	ref	ref
	<7	1.40 (1.24, 1.59)	<0.0001
	≥10	1.51 (0.94, 2.42)	0.09
Model 3	Self-reported sleep disorder diagnosis		
	No	ref	ref
	Yes	1.32 (1.06, 1.63)	0.01
	Subjective sleep difficulties		
	No	ref	ref
	Yes	1.29 (1.11, 1.49)	0.001
	Sleep time		
	7–9	ref	ref
	<7	1.20 (1.05, 1.38)	0.01
	≥10	1.47 (0.89, 2.43)	0.13

Model 1: crude model.
Model 2: adjusted for gender, age and ethnicity.
Model 3: adjusted for Mode 2 plus education, marital status, BMI, PIR, smoke, alcohol consumption, physical activity, Hei-2015 index, hypertension, SII, PHQ-9.
Ref: references.

Table 3
Logistic regression analysis of sleep patterns and T2D.

Model	Sleep patterns	OR (95% CI)	*P*-value
Model 1	Healthy sleep pattern	ref	ref
	Intermediate sleep pattern	1.17 (1.02, 1.35)	0.02
	Poor sleep pattern	2.42 (2.07, 2.84)	<0.0001
Model 2	Healthy sleep pattern	ref	ref
	Intermediate sleep pattern	1.20 (1.04, 1.38)	0.01
	Poor sleep pattern	2.31 (1.97, 2.71)	<0.0001
Model 3	Healthy sleep pattern	ref	ref
	Intermediate sleep pattern	1.13 (0.97, 1.32)	0.10
	Poor sleep pattern	1.52 (1.27, 1.82)	<0.0001

Healthy sleep pattern: score 0, no high-risk sleep factors. Intermediate sleep pattern: score 1, presence of 1 high-risk sleep factor. Poor sleep pattern: score 2–3, presence of 2–3 high-risk sleep factors.
Model 1: crude model.
Model 2: adjusted for gender, age and ethnicity.
Model 3: adjusted for Mode 2 plus education, marital status, BMI, PIR, smoke, alcohol consumption, physical activity, HEI-2015, hypertension, SII, PHQ-9.
Ref: reference.

was observed between long sleep duration and T2D risk (OR = 1.47, 95% CI: 0.89–2.43). Conversely, other studies have suggested that long sleep duration may increase T2D risk. For instance, a Finnish population-based cross-sectional study found that ≥8 h of sleep was associated with increased T2D risk in middle-aged women (Tuomilehto et al., 2008). Additionally, a recent meta-analysis indicated that long sleep duration (≥9 h) was associated with an elevated T2D risk (RR = 1.26, 95% CI: 1.15–1.39, $P < 0.001$) (Lu et al., 2021). Interestingly, a study of UK children reported a negative correlation between sleep

Table 4
Logistic regression analysis of sleep patterns and constipation by age.

Age, years	Healthy sleep pattern	Intermediate sleep pattern		Poor sleep pattern		*P*-trend
	OR (95% CI)	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	
Model 1						
20-39	ref	1.071 (0.715, 1.602)	0.737	2.759 (1.699, 4.452)	<0.0001	0.002
40-59	ref	1.391 (1.113, 1.737)	0.004	2.296 (1.819, 2.898)	<0.0001	<0.0001
≥60	ref	1.112 (0.882, 1.401)	0.365	2.044 (1.606, 2.600)	<0.0001	<0.0001
Model 2						
20-39	ref	1.060 (0.706, 1.592)	0.776	2.837 (1.755, 4.588)	<0.0001	0.001
40-59	ref	1.331 (1.064, 1.665)	0.013	2.364 (1.873, 2.983)	<0.0001	<0.0001
≥60	ref	1.083 (0.846, 1.386)	0.521	2.038 (1.597, 2.600)	<0.0001	<0.0001
Model 3						
20-39	ref	0.951 (0.626, 1.444)	0.810	1.797 (1.036, 3.118)	0.037	0.110
40-59	ref	1.296 (1.012, 1.634)	0.040	1.852 (1.433, 2.394)	<0.0001	<0.0001
≥60	ref	1.076 (0.822, 1.379)	0.577	1.802 (1.386, 2.342)	<0.0001	<0.001

Healthy sleep pattern: score 0, no high-risk sleep factors. Intermediate sleep pattern: score 1, presence of 1 high-risk sleep factor. Poor sleep pattern: score 2–3, presence of 2–3 high-risk sleep factors.
Model 1: without excluding any confounders.
Model 2: adjusted for gender, age and ethnicity.
Model 3: adjusted for Mode 2 plus education, marital status, BMI, PIR, smoke, alcohol consumption, physical activity, HEI-2015, hypertension, SII, PHQ-9.
Ref: reference.

duration and T2D (Rudnicka et al., 2017). Furthermore, a recent study found that sleep efficiency (<80%) was associated with T2D prevalence in individuals without sleep-disordered breathing (OR = 1.894, 95% CI: 1.187–3.022, P = 0.007) (Yan et al., 2020). These inconsistencies may be due to differences in sleep assessment criteria, and variations in study populations regarding age, ethnicity, and environmental factors. Self-reported sleep duration may also be influenced by demographic characteristics, socioeconomic status, employment conditions, and health behaviors. Therefore, further research is needed to elucidate the relationship between long sleep duration and T2D.

T2D is often comorbid with hypertension, obesity, cardiovascular disease and psychiatric disorders. Studies have shown that sleep deprivation is associated with obesity, potentially mediated by disruptions in leptin and ghrelin regulation (Spiegel et al., 2004). Obstructive sleep apnoea, a common sleep disorder, is particularly prevalent among obese individuals with T2D and its treatment has shown significant benefits in improving T2D symptoms (Kurnool et al., 2023). In our subgroup analysis, individuals with a BMI ≥30 and poor sleep patterns had a higher risk of T2D compared to those with healthy sleep patterns. In addition, maintaining a lifestyle that includes healthy sleep patterns may reduce the risk of T2D in hypertensive patients (Song et al., 2021). Depression is the most common mental disorder among individuals with T2D, with a prevalence of approximately 10%–15%, which is about twice that observed in non-diabetic individuals (Sartorius, 2018). Depression can lead to impaired self-management of the disease,

Table 5
Logistic regression analysis of sleep patterns and constipation by BMI.

BMI, Kg/m2	Healthy sleep	Intermediate sleep		Poor sleep		*P*-trend
	OR (95% CI)	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	
Model 1						
< 25	ref	1.248 (0.908, 1.714)	0.169	1.935 (1.198, 3.125)	0.008	0.006
25–29.9	ref	1.002 (0.796, 1.261)	0.986	1.715 (1.253, 2.346)	<0.001	0.006
≥ 30	ref	1.104 (0.889, 1.371)	0.368	2.133 (1.777, 2.560)	<0.0001	<0.0001
Model 2						
< 25	ref	1.198 (0.865, 1.658)	0.272	2.042 (1.263, 3.302)	0.004	0.006
25–29.9	ref	0.968 (0.768, 1.219)	0.779	1.745 (1.266, 2.405)	<0.001	0.008
≥ 30	ref	1.099 (0.883, 1.369)	0.394	2.154 (1.792, 2.588)	<0.0001	<0.0001
Model 3						
< 25	ref	1.187 (0.855, 1.649)	0.300	1.701 (0.967, 2.992)	0.065	0.053
25–29.9	ref	0.959 (0.743, 1.239)	0.747	1.475 (1.019, 2.134)	0.040	0.100
≥ 30	ref	1.152 (0.912, 1.456)	0.231	1.659 (1.349, 2.041)	<0.0001	<0.0001

Healthy sleep pattern: score 0, no high-risk sleep factors. Intermediate sleep pattern: score 1, presence of 1 high-risk sleep factor. Poor sleep pattern: score 2–3, presence of 2–3 high-risk sleep factors.
Model 1: without excluding any confounders.
Model 2: adjusted for gender, age and ethnicity.
Model 3: adjusted for Mode 2 plus education, marital status, BMI, PIR, smoke, alcohol consumption, physical activity, HEI-2015, hypertension, SII, PHQ-9.
Ref: reference.

worsened glycemic control, and increased complications and mortality (Holt et al., 2014). Furthermore, individuals with depression often experience poor sleep quality. In the current study, after adjusting for depressive symptoms as a covariate in multivariable logistic regression and subgroup analyses, we still observed an increased risk of T2D for self-reported sleep disorder diagnoses, subjective sleep difficulties, short sleep duration, and poor sleep patterns, suggesting these factors are independent risk factors for T2D. A NHANES-based study identified a nomogram model combining nine predictors, including sleep duration and sleep disorders, that accurately predicted the risk of depression in T2D patients with strong predictive performance (AUC >0.75) (Yu et al., 2024). However, in a small clinical trial, Safari M et al. found that treatment with Melissa officinalis extract improved symptoms of depression and anxiety in T2D patients with depression but had no significant impact on sleep quality after 12 weeks of intervention (Safari et al., 2023). Thus, further research is needed to explore the interrelationships between sleep factors, depression, and T2D.

Mechanistically, sleep restriction may increase T2D risk through multiple pathways. Studies have shown that sleep restriction significantly increases insulin resistance, reduces insulin sensitivity, and impairs glucose tolerance (Broussard et al., 2012), (Dracup et al., 1990). In a randomized crossover trial, restricting sleep to <5 h for 5 consecutive nights resulted in over-activation of the sympathetic nervous system and elevated serum norepinephrine levels (Dettoni et al., 2012). Sleep deprivation also elevates pro-inflammatory markers, such as IL-1β, IL-6, and C-reactive protein (Frey et al., 2007). Furthermore, in healthy individuals, restricting sleep to 4 h for 5 consecutive nights significantly increased the expression of IL-6, IL-1β, and IL-17 (van Leeuwen et al., 2009).

Based on these findings, we recommend strengthening the focus on sleep health in public health. First, health organizations should include evidence-based sleep recommendations in their guidelines to promote health. Second, public health promotion should focus on the importance of sleep duration and screening for sleep disorders. Finally, healthcare providers should regularly screen T2D patients for sleep disorders and implement targeted interventions to manage sleep disorders.

Our study utilized a robust and representative database, employing multiple models to minimize confounding factors. However, several limitations merit consideration. Firstly, reliance on self-reported sleep measures introduces potential recall and reporting biases, compromising the accuracy of sleep factor assessment. Objective methods, such as actigraphy or polysomnography, are necessary to validate our findings. Secondly, the cross-sectional design of our study prevents causal inferences between sleep factors and T2D risk, highlighting the need for longitudinal studies. Finally, this study did not explore potential molecular mechanisms underlying the association between sleep factors, sleep patterns, and T2D.

5. Conclusions

This nationally representative study emphasises self-reported diagnoses of sleep disorders, subjective sleep difficulties, sleep duration of <7 h, poor sleep patterns and T2D risk. Further prospective studies should be performed to investigate the causal relationship between sleep and T2D risk. In particular, exploring the underlying molecular mechanisms between sleep and T2D is also essential for the effective management of T2D.

CRediT authorship contribution statement

Jijun Zhang: Writing – original draft, Formal analysis, Data curation. **Chuanli Yang:** Writing – original draft, Formal analysis, Data curation. **Jie An:** Writing – review & editing. **Yunhe Fan:** Writing – review & editing. **Xiushan Dong:** Methodology.

Ethics approval and consent to participate

All NHANES surveys were approved by the National Centre for Health Statistics Research Ethics.

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Declaration of competing interest

The 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.

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Abbreviations:

BMI body mass index

T2D	Type 2 diabetes mellitus
MET	metabolic equivalent
HEI-2015	Healthy Eating Index-2015
NHANES	National Health and Nutrition Examination Survey
OR	odds ratio
SLQ	sleep questionnaire
PIR	Poverty-to-income ratio

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbih.2025.100953.

Data availability

The datasets used and analysed during the current study are available in the NHANES (http://www.cdc.gov/nchs/nhanes.htm).

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