BMC Psychiatry

RESEARCH

Open Access

Association of weekend catch-up sleep ratio with depressive risk: insights from NHANES 2021–2023

Shilin Sun[1†], Min Liu[2†], Han Liu[3†], Runzhou Li[4], Qun Liang[4*] and Weiwei Quan[1*]

Abstract

Background Depression is a common global mental health issue, affecting around 3.8% of the population. It significantly impacts quality of life and social functioning, posing a major public health challenge. Sleep is a key factor influencing depression, with both sleep quality and quantity linked to mental health. However, sleep deprivation is widespread, and many people compensate by "weekend sleep recovery." The effects of sleep deprivation and weekend recovery on depression risk are unclear, as irregular sleep patterns may worsen depressive symptoms. This study introduces the "Weekend Catch-up Sleep Ratio" (CUS ratio) to better understand the relationship between sleep patterns and depression.

Methods Cross-sectional data were obtained from individuals who participated in the 2021–2023 National Health and Nutrition Examination Survey (NHANES) and had complete data on CUS and the Patient Health Questionnaire (PHQ-9). Multivariable logistic regression was performed to assess the potential independent association between depression and the CUS ratio. Additionally, smoothing curve fitting, threshold effect analysis, subgroup analysis, and interaction tests were conducted.

Results A total of 4,656 individuals were analyzed, categorized by depression symptoms (PHQ-9 score of 10 or higher), with an overall depression risk of 12.4%. In the adjusted model, the CUS ratio was significantly positively associated with depression risk (AOR = 1.75, 95% CI: 1.25–2.45), exhibiting a nonlinear threshold effect (inflection point at 1.11). When the CUS ratio ≤ 1.11, an increase in the ratio was associated with a reduced depression risk (AOR = 0.34, 95% CI: 0.13–0.89), whereas when the CUS ratio > 1.11, each unit increase in the ratio significantly increased depression risk by 187% (AOR = 2.87, 95% CI: 1.84–4.50). Individuals with education levels of less than 9th grade, some college or an Associate of Arts (AA) degree, those who are overweight (25 ≤ BMI < 30), and those without diabetes appeared more sensitive to fluctuations in sleep patterns.

In the adjusted model for the severity of depressive symptoms, the CUS ratio was significantly positively associated with depression severity (Aβ = 0.19, 95% CI: 0.09–0.28), also exhibiting a nonlinear threshold effect (inflection point at 1.11). When the CUS ratio ≤ 1.11, an increase in the ratio was associated with a reduction in depression severity

†Shilin Sun, Min Liu and Han Liu contributed equally to this work and share first authorship.

*Correspondence:
Qun Liang
Qunliang1968@sina.com
Weiwei Quan
weiweiquan1980@126.com
Full list of author information is available at the end of the article

(Aβ = -0.35, 95% CI: -0.62 to -0.09), whereas when the CUS ratio > 1.11, each unit increase in the ratio significantly increased depression severity (Aβ = 0.36, 95% CI: 0.24–0.49). In particular, individuals without diabetes appeared more sensitive to fluctuations in sleep patterns.

Conclusions This study suggests that maintaining a balanced sleep pattern, with a CUS ratio between 1 and 1.11, may help reduce depression risk and promote better mental health.

Keywords Weekend catch-up sleep ratio, Sleep pattern fluctuations, Depressive, NHANES

Introduction

Depression is one of the most common mental health disorders worldwide, affecting individuals across all ages, genders, and social backgrounds [1–3]. Its main symptoms include persistent low mood, apathy, sadness, chronic fatigue, and reduced motivation, with severe cases potentially leading to suicidal thoughts [4, 5]. As societal pressures increase and lifestyles change, depression has become an increasingly serious public health issue, with an estimated 3.8% of the global population—approximately 280 million people—affected [6, 7]. Due to its profound impact on individual quality of life, work productivity, and social functioning, the prevention and treatment of depression have become significant challenges in the field of public health [8, 9].

In recent years, research on depression has gradually revealed various potential causes and risk factors, with sleep being recognized as one of the key influences on depression [10–12]. Both the quality and quantity of sleep are closely related to emotional well-being and play a crucial role in maintaining mental health [13, 14]. However, sleep deprivation is a widespread issue in modern society, particularly during weekdays, when adults and adolescents often fail to get adequate sleep due to the pressures of work and study. To compensate for this sleep deficit, many individuals choose to recover by weekend catch-up sleep [15]. Yet, the impact of sleep deprivation and weekend catch-up sleep on depression risk remains inconclusive. Studies have shown that sufficient sleep can alleviate depressive symptoms, but excessive sleep or irregular sleep patterns may also exacerbate the onset and progression of depression [16, 17]. Severe sleep problems, such as Co-Morbid Insomnia and Sleep Apnea (COMISA), can extend beyond depression and significantly impact suicidality [18–20]. Therefore, understanding the relationship between sleep patterns, sleep deprivation, and weekend catch-up sleep is critical for developing more effective sleep interventions and depression prevention strategies.

Currently, many studies exploring the relationship between sleep duration and depression often use the "weekend catch-up sleep" (CUS) indicator [21]. However, due to the inconsistency of sleep duration on weekdays, this method, which relies solely on the difference between weekend and weekday sleep durations, may not accurately reflect individual differences in sleep patterns. Therefore, this study uses the ratio of weekend sleep duration to weekday sleep duration, referred to as the "weekend catch-up sleep ratio" (CUS ratio) [22], to further investigate the relationship between sleep and depression. This ratio standardizes an individual's baseline sleep duration during weekdays, thereby quantifying the relative extent of weekend sleep compensation (e.g., a ratio >1 indicates extended weekend sleep). It more precisely reflects differences in individual sleep patterns. Compared to absolute differences, the ratio indicator eliminates the interference of baseline differences in weekday sleep (e.g., the same difference may have different physiological meanings in different baseline groups), allowing for a more accurate capture of variations in individual sleep duration. This improves the scientific rigor and comparability of the analysis of the relationship between sleep patterns and depression.

This study aims to systematically analyze the relationship between the volatility of sleep patterns and the risk of depression using the CUS ratio as an exposure indicator, based on data from the nationally representative sample of the United States (NHANES 2021–2023). We hope that this research will provide new insights and offer strong support for the future management of sleep patterns in clinical treatment and public health interventions.

Methods

Study population and research design

The National Center for Health Statistics (NCHS) conducted a nationwide, stratified, multi-stage probability sampling survey known as NHANES, aimed at evaluating the relationship between disease prevention and the promotion of nutritional health. This biennial survey covers demographic, nutrition, examination, laboratory, and questionnaire components, combining both interviews and physical exams [23]. From 2021 to 2023, a total of 11,933 individuals participated in the registration process. After applying a strict set of inclusion and exclusion criteria, the final sample consisted of 4,656 U.S. adults from the NHANES 2021–2023 cycle. Specifically, this study excluded 4,124 individuals under the age of 20.

Other exclusions included 140 participants with missing sleep data, 2,470 participants missing PHQ-9 data, and 543 participants with missing covariate data (Fig. 1).

This study was approved by the Institutional Review Board of the National Center for Health Statistics (NCHS) (https://www.cdc.gov/nchs/nhanes/about/erb.html#print). All participants provided written informed consent, agreeing to participate in the survey and to the use of their data for health-related statistical research. The full NHANES study methods and data can be accessed by the public at www.cdc.gov/nchs/nhanes/.

Definition of sleep duration and depression

Sleep duration was assessed using standardized NHANES survey questions, with participants reporting their average sleep duration on both weekdays and weekends [15].

Depression was evaluated using the nine-item Patient Health Questionnaire (PHQ-9) from NHANES [24], which assessed the severity of depression in participants over the past 2 weeks. The questionnaire consists of 9 questions: "Have little interest in doing things?"; "Feeling down, depressed, or hopeless?"; "Trouble sleeping or sleeping too much?"; "Feeling tired or having little energy?"; "Poor appetite or overeating?"; "Feeling bad about yourself?"; "Trouble concentrating on things?"; "Moving or speaking slowly or too fast?"; "Thought you would be better off dead?". Trained interviewers at the mobile exam center (MEC) administered these questions using a computer-assisted personal interview (CAPI) system as part of the process. The total score ranges from 0 to 27, with response options for each symptom including "Not at all," "Several days," "More than half the days," and "Nearly every day," corresponding to scores of 0 to 3. Participants with a total PHQ-9 score of 10 or higher were considered to have depressive symptoms, a threshold that demonstrates high sensitivity (88%) and specificity (88%) in detecting major depression [25]. The severity of depressive symptoms were further classified based on the total PHQ-9 score as follows: 0 to 4 points for no depressive symptoms, 5 to 9 points for mild depression, 10 to 14 points for moderate depression, 15 to 19 points for moderate-to-severe depression, and 20 points or higher for severe depression [26, 27]. To better understand the relationship between the CUS ratio and depression, this study conducted sensitivity analyses using two classifications: depression symptoms (PHQ-9 score of 10 or higher) and the severity of depressive symptoms (no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression).

In this study, the CUS ratio was used as the exposure factor, with depression as the outcome variable. The CUS ratio is calculated by dividing the average weekend sleep duration by the average weekday sleep duration, i.e., CUS ratio = average weekend sleep duration/average weekday sleep duration [22].

Covariates [28–33]

The covariates selected for this study include gender (male or female), age (years), race (including six categories such as Mexican American), education level (including five categories such as Less than 9th grade), marital status (including three categories such as Married/Living with partner), family monthly poverty level category (including three categories such as Monthly

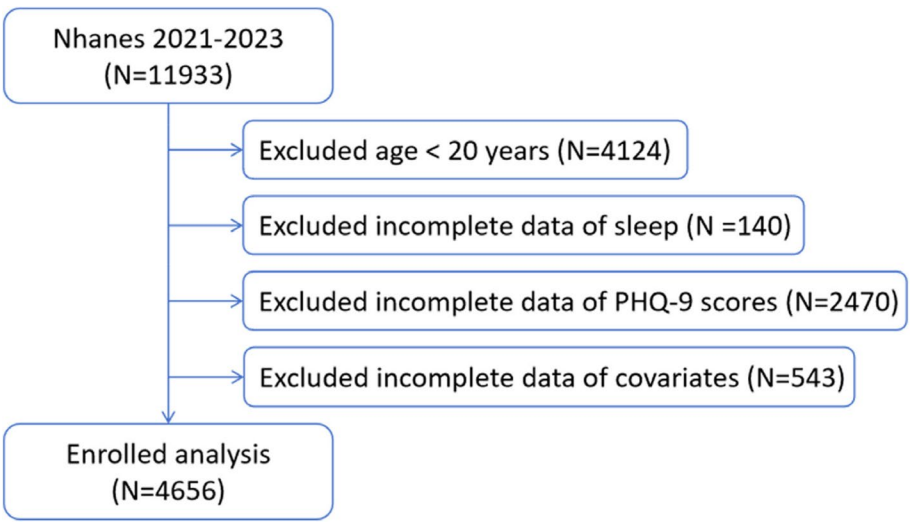

Fig. 1 Flow chart of study participants

poverty level index ≤ 1.30), and body mass index (BMI, kg/m^2), categorized as underweight (BMI < 18.5), normal weight (18.5 ≤ BMI < 25), overweight (25 ≤ BMI < 30), and obese (BMI ≥ 30). The participants' alcohol consumption status was categorized into six groups: never (< 12 drinks in a lifetime), former (≥ 12 drinks in a lifetime and no alcohol consumption in the past year), mild (≥ 1 drink/day for women and ≥ 2 drinks/day for men), moderate (≥ 2 drinks/day for women and ≥ 3 drinks/day for men, or binge drinking on ≥ 2 days/month), and heavy (≥ 3 drinks/day for women and ≥ 4 drinks/day for men, or binge drinking on ≥ 5 days/month). Smoking status and diabetes status were also included (both coded as yes or no), with diabetes defined by one or more of the following: (1) physician diagnosis; (2) HbA1c > 6.5%; (3) fasting blood glucose > 7.0 mmol/L; (4) random blood glucose > 11.1 mmol/L; or (5) use of insulin or anti-diabetic medications. Mental health history was determined based on participants' responses (yes or no) to the question: "During the past 12 months, have you seen or talked to a mental health professional such as a psychologist, psychiatrist, psychiatric nurse, or clinical social worker about your health?" Detailed measurement procedures for these variables can be accessed by the public at www.cdc.gov/nchs/nhanes/.

Statistical analysis

All data analyses were performed using EmpowerStats (http://www.empowerstats.net/cn/index.php) and R 3.4.3 (https://www.r-project.org/) statistical software. For continuous variables, baseline characteristics are presented as mean ± standard deviation (SD), while categorical variables are reported as proportions. Differences between groups for continuous variables were assessed using the Kruskal–Wallis rank sum test or analysis of variance (ANOVA), and differences for categorical variables were analyzed using the chi-square test. To evaluate the relationship between the exposure factor (CUS ratio) and the outcome variable (depression), three multivariable logistic regression models were applied: Model 1(an unadjusted model), Model 2 (adjusting for key demographic variables), and Model 3 (adjusting for all covariates). Additionally, subgroup analyses and interaction tests were conducted to further explore the relationship between the exposure factor and depression across different demographic characteristics. To assess the nonlinear relationship between the CUS ratio and depression, a smoothing curve fitting based on generalized linear models (GLM) was used. A p-value < 0.05 was considered statistically significant.

Results

Baseline characteristics of participants

Table 1 lists the baseline characteristics of participants stratified by the presence or absence of depressive symptoms (a total PHQ-9 score of 10 or higher) in the NHANES 2021–2023 dataset, including 579 participants with depression and 4,077 without, resulting in an overall depression prevalence of 12.4%. Compared to the non-depressed group, participants in the depressed group were younger (48.06 ± 17.26 years vs. 54.54 ± 16.78 years, $p < 0.001$), had a higher proportion of females (64.25% vs. 53.86%, $p < 0.001$), had a higher BMI (31.41 ± 8.72 vs. 29.61 ± 7.03, $p < 0.001$), reported shorter weekday sleep duration (7.47 ± 2.10 h vs. 7.71 ± 1.43 h, $p < 0.001$), had a higher diabetes prevalence (22.80% vs. 15.55%, $p < 0.001$), and had a higher smoking rate (48.70% vs. 40.59%, $p < 0.001$). Additionally, the depressed group had a lower proportion of individuals with mild alcohol consumption (24.35% vs. 38.63%, $p < 0.001$), and higher proportions in the "Never," "Former," "Moderate," and "Heavy" alcohol consumption categories. Furthermore, a significantly smaller proportion of individuals in the depressed group had no psychiatric history (62.00% vs. 87.42%, $p < 0.001$). There were no significant differences between the two groups in weekend sleep duration. These differences highlight the importance of considering metabolic factors and lifestyle behaviors in the assessment of depression risk.

Table 2 lists the baseline characteristics of participants further stratified by the severity of depressive symptoms based on the total PHQ-9 score. Except for weekend sleep duration, all other variables showed significant univariate associations with the classification of depressive symptoms ($p < 0.05$) and were therefore included in the multivariate model for further analysis.

To better illustrate the relationships between predictive factors, Supplemental Table 1 presents the associations between the CUP ratio and other variables, showing how these factors may independently or jointly contribute to the development of depression.

The association between CUS ratio and depression

Multivariable logistic regression models

We constructed multivariable logistic regression models to assess the association between the CUS ratio and depression symptoms (PHQ-9 score of 10 or higher), as shown in Table 3. In the unadjusted model, the CUS ratio was significantly positively associated with depression (OR = 1.92, 95% CI: 1.39, 2.64, $p < 0.0001$). After adjusting for demographic, clinical, and lifestyle factors, this association remained significant in the fully adjusted model (Model 3) (AOR = 1.75, 95% CI: 1.25, 2.45, $p =$

Table 1 Baseline characteristics of study participants stratified by depression symptoms (PHQ-9 score of 10 or higher)

Characteristics	Non-Depression group	Depression group	*P*-value
N	4077	579	
Age (years)	54.54 ± 16.78	48.06 ± 17.26	< 0.001
Sex (%)			< 0.001
Male	1881 (46.14%)	207 (35.75%)	
Female	2196 (53.86%)	372 (64.25%)	
Race/Ethnicity (%)			0.002
Mexican American	245 (6.01%)	42 (7.25%)	
Other Hispanic	375 (9.20%)	60 (10.36%)	
Non-Hispanic White	2580 (63.28%)	340 (58.72%)	
Non-Hispanic Black	437 (10.72%)	70 (12.09%)	
Non-Hispanic Asian	190 (4.66%)	13 (2.25%)	
Other Race—Including Multi-Racial	250 (6.13%)	54 (9.33%)	
Education level (%)			< 0.001
Less than 9th grade	120 (2.94%)	26 (4.49%)	
9-11th grade (Includes 12th grade with no diploma)	270 (6.62%)	55 (9.50%)	
High school graduate/GED or equivalent	799 (19.60%)	142 (24.53%)	
Some college or Associate of Arts (AA) degree	1231 (30.19%)	219 (37.82%)	
College graduate or above	1657 (40.64%)	137 (23.66%)	
Marital status (%)			< 0.001
Married/Living with partner	2320 (56.90%)	216 (37.31%)	
Widowed/Divorced/Separated	998 (24.48%)	172 (29.71%)	
Never married	759 (18.62%)	191 (32.99%)	
Family monthly poverty level category			< 0.001
Monthly poverty level index < = 1.30	807 (19.79%)	227 (39.21%)	
1.30 < Monthly poverty level index < = 1.85	533 (13.07%)	96 (16.58%)	
Monthly poverty level index > 1.85	2737 (67.13%)	256 (44.21%)	
BMI (kg/m2)	29.61 ± 7.03	31.41 ± 8.72	< 0.001
Underweight (BMI < 18.5)	45 (1.10%)	16 (2.76%)	
Normal weight (18.5 ≤ BMI < 25)	1054 (25.85%)	120 (20.73%)	
Overweight (25 ≤ BMI < 30)	1335 (32.74%)	161 (27.81%)	
Obese (BMI ≥ 30)	1643 (40.30%)	282 (48.70%)	
Weekday sleep duration(hours)	7.71 ± 1.43	7.47 ± 2.10	< 0.001
Weekend sleep duration(hours)	8.23 ± 1.56	8.13 ± 2.17	0.630
CUS ratio	1.08 ± 0.21	1.13 ± 0.39	< 0.001
Drinking (%)			< 0.001
Never	341 (8.36%)	52 (8.98%)	
Former	615 (15.08%)	104 (17.96%)	
Mild	1575 (38.63%)	141 (24.35%)	
Moderate	787 (19.30%)	116 (20.03%)	
Heavy	759 (18.62%)	166 (28.67%)	
Smoke (%)			< 0.001
No	2422 (59.41%)	297 (51.30%)	
Yes	1655 (40.59%)	282 (48.70%)	
Diabetes (%)			< 0.001
No	3443 (84.45%)	447 (77.20%)	
Yes	634 (15.55%)	132 (22.80%)	
PHQ-9 score	2.59 ± 2.55	13.94 ± 3.76	< 0.001
Mental health history (%)			< 0.001
No	3564 (87.42%)	359 (62.00%)	
Yes	513 (12.58%)	220 (38.00%)	

Monthly poverty level index: a ratio of monthly family income to the HHS poverty guidelines specific to family size; *BMI* Body Mass Index

Table 2 Baseline characteristics of study participants stratified by severity of depressive symptoms

Characteristics	No depressive symptoms	Mild depression	Moderate depression	Moderate-to-severe depression	Severe depression	*P*-value
N	3155	922	370	151	58	
Age (years)	55.69 ± 16.34	50.60 ± 17.69	47.48 ± 17.08	49.72 ± 18.09	47.45 ± 16.18	< 0.001
Sex (%)						< 0.001
Male	1529 (48.46%)	352 (38.18%)	127 (34.32%)	58 (38.41%)	22 (37.93%)	
Female	1626 (51.54%)	570 (61.82%)	243 (65.68%)	93 (61.59%)	36 (62.07%)	
Race/Ethnicity (%)						< 0.001
Mexican American	191 (6.05%)	54 (5.86%)	27 (7.30%)	10 (6.62%)	5 (8.62%)	
Other Hispanic	264 (8.37%)	111 (12.04%)	36 (9.73%)	18 (11.92%)	6 (10.34%)	
Non-Hispanic White	2032 (64.41%)	548 (59.44%)	222 (60.00%)	80 (52.98%)	38 (65.52%)	
Non-Hispanic Black	327 (10.36%)	110 (11.93%)	45 (12.16%)	22 (14.57%)	3 (5.17%)	
Non-Hispanic Asian	160 (5.07%)	30 (3.25%)	7 (1.89%)	6 (3.97%)	0 (0.00%)	
Other Race—Including Multi-Racial	181 (5.74%)	69 (7.48%)	33 (8.92%)	15 (9.93%)	6 (10.34%)	
Education level (%)						< 0.001
Less than 9th grade	88 (2.79%)	32 (3.47%)	13 (3.51%)	11 (7.28%)	2 (3.45%)	
9-11th grade (Includes 12th grade with no diploma)	202 (6.40%)	68 (7.38%)	24 (6.49%)	23 (15.23%)	8 (13.79%)	
High school graduate/GED or equivalent	616 (19.52%)	183 (19.85%)	95 (25.68%)	36 (23.84%)	11 (18.97%)	
Some college or AA degree	913 (28.94%)	318 (34.49%)	138 (37.30%)	56 (37.09%)	25 (43.10%)	
College graduate or above	1336 (42.35%)	321 (34.82%)	100 (27.03%)	25 (16.56%)	12 (20.69%)	
Marital status (%)						< 0.001
Married/Living with partner	1905 (60.38%)	415 (45.01%)	154 (41.62%)	50 (33.11%)	12 (20.69%)	
Widowed/Divorced/Separated	723 (22.92%)	275 (29.83%)	106 (28.65%)	41 (27.15%)	25 (43.10%)	
Never married	527 (16.70%)	232 (25.16%)	110 (29.73%)	60 (39.74%)	21 (36.21%)	
Family monthly poverty level category						< 0.001
Monthly poverty level index < = 1.30	570 (18.07%)	237 (25.70%)	141 (38.11%)	66 (43.71%)	20 (34.48%)	
1.30 < Monthly poverty level index < = 1.85	388 (12.30%)	145 (15.73%)	59 (15.95%)	29 (19.21%)	8 (13.79%)	
Monthly poverty level index > 1.85	2197 (69.64%)	540 (58.57%)	170 (45.95%)	56 (37.09%)	30 (51.72%)	
BMI (kg/m2)	29.24 ± 6.67	30.89 ± 8.03	31.31 ± 8.75	32.22 ± 8.90	29.92 ± 7.94	< 0.001
Underweight	33 (1.05%)	12 (1.30%)	10 (2.70%)	3 (1.99%)	3 (5.17%)	
Normal weight	827 (26.21%)	227 (24.62%)	82 (22.16%)	25 (16.56%)	13 (22.41%)	
Overweight	1097 (34.77%)	238 (25.81%)	98 (26.49%)	44 (29.14%)	19 (32.76%)	
Obese	1198 (37.97%)	445 (48.26%)	180 (48.65%)	79 (52.32%)	23 (39.66%)	
Weekday sleep duration(hours)	7.72 ± 1.34	7.67 ± 1.71	7.45 ± 1.94	7.64 ± 2.46	7.22 ± 2.13	0.004
Weekend sleep duration(hours)	8.22 ± 1.48	8.27 ± 1.81	8.12 ± 2.05	8.23 ± 2.50	7.95 ± 2.01	0.423
CUS ratio	1.08 ± 0.20	1.10 ± 0.24	1.13 ± 0.43	1.11 ± 0.28	1.15 ± 0.37	< 0.001
Drinking (%)						< 0.001
Never	283 (8.97%)	58 (6.29%)	26 (7.03%)	21 (13.91%)	5 (8.62%)	
Former	469 (14.87%)	146 (15.84%)	66 (17.84%)	30 (19.87%)	8 (13.79%)	
Mild	1299 (41.17%)	276 (29.93%)	92 (24.86%)	34 (22.52%)	15 (25.86%)	
Moderate	569 (18.03%)	218 (23.64%)	84 (22.70%)	21 (13.91%)	11 (18.97%)	
Heavy	535 (16.96%)	224 (24.30%)	102 (27.57%)	45 (29.80%)	19 (32.76%)	
Smoke (%)						< 0.001
No	1940 (61.49%)	482 (52.28%)	192 (51.89%)	80 (52.98%)	25 (43.10%)	
Yes	1215 (38.51%)	440 (47.72%)	178 (48.11%)	71 (47.02%)	33 (56.90%)	
Diabetes (%)						< 0.001
No	2678 (84.88%)	765 (82.97%)	289 (78.11%)	111 (73.51%)	47 (81.03%)	
Yes	477 (15.12%)	157 (17.03%)	81 (21.89%)	40 (26.49%)	11 (18.97%)	

Table 2 (continued)

Characteristics	No depressive symptoms	Mild depression	Moderate depression	Moderate-to-severe depression	Severe depression	*P*-value
PHQ-9 score	1.42 ± 1.37	6.58 ± 1.35	11.54 ± 1.36	16.76 ± 1.43	21.91 ± 1.68	< 0.001
Mental health history (%)						< 0.001
No	2860 (90.65%)	704 (76.36%)	244 (65.95%)	91 (60.26%)	24 (41.38%)	
Yes	295 (9.35%)	218 (23.64%)	126 (34.05%)	60 (39.74%)	34 (58.62%)	

Table 3 The association between CUS ratio and depression (PHQ-9 score of 10 or higher) in a multiple logistics regression model

Character	Model 1		Model 2		Model 3	
	OR(95%CI)	p	AOR(95%CI)	p	AOR(95%CI)	p
CUS ratio	1.92 (1.39, 2.64)	< 0.0001	1.51 (1.11, 2.06)	0.0096	1.75 (1.25, 2.45)	0.0011

Model 1 adjust for: None

Model 2 adjust for: Sex; Age; Ethnicity

Model 3 adjust for: Sex; Age; Ethnicity; Education level; Marital status; Family monthly poverty level category; BMI; Drinking; Diabetes; Smoke; Mental health history

0.0011), indicating a significant relationship between the CUS ratio and depression risk.

Table 4 presents the association between the CUS ratio and the severity of depressive symptoms (no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression). In the unadjusted model, the CUS ratio was significantly positively associated with depression severity (β = 0.25, 95% CI: 0.15, 0.36, $p < 0.0001$). After controlling for demographic, clinical, and lifestyle factors, this association remained significant in the fully adjusted model (Model 3) (Aβ = 0.19, 95% CI: 0.09, 0.28, $p = 0.0002$), indicating a significant relationship between the CUS ratio and depression severity.

Smoothing curve fitting and threshold effect analysis

Smoothing curve fitting based on Model 3 further revealed a nonlinear relationship between the CUS ratio and depression symptoms (PHQ-9 score of 10 or higher), as shown in Fig. 2a. To further elucidate this relationship, a threshold effect analysis was conducted (Table 5). The threshold analysis identified the inflection point of the CUS ratio as 1.11 (likelihood ratio test, $p < 0.001$). When the ratio was below 1.11, each unit increase in the ratio was associated with a 66% reduction in depression risk (AOR = 0.34, 95% CI: 0.13, 0.89, $p = 0.0237$). However, when the ratio exceeded 1.11, each unit increase was associated with a 187% increase in depression risk (AOR = 2.87, 95% CI: 1.84, 4.50, $p < 0.0001$).

Figure 2b shows the nonlinear relationship between the CUS ratio and the severity of depressive symptoms (no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression). The threshold effect analysis (Table 5) also identified the inflection point of the CUS ratio as 1.11 (likelihood ratio test, $p < 0.001$). When the ratio was below 1.11, each unit increase was associated with a 0.35 unit decrease in depression severity (Aβ = −0.35, 95% CI: −0.62, −0.09, $p = 0.0098$). However, when the ratio exceeded 1.11, each unit increase was associated with a 0.36 unit increase in depression severity (Aβ = 0.36, 95% CI: 0.24, 0.49, $p < 0.0001$).

These findings highlight the importance of considering threshold effects in the relationship between the CUS ratio and both depression risk and severity.

Table 4 The association between CUS ratio and the severity of depressive symptoms in a multiple logistics regression model

Character	Model 1		Model 2		Model 3	
	β(95%CI)	p	Aβ(95%CI)	p	Aβ(95%CI)	p
CUS ratio	0.25 (0.15, 0.36)	< 0.0001	0.15 (0.04, 0.25)	0.0053	0.19 (0.09, 0.28)	0.0002

Model 1 adjust for: None

Model 2 adjust for: Sex; Age; Ethnicity

Model 3 adjust for: Sex; Age; Ethnicity; Education level; Marital status; Family monthly poverty level category; BMI; Drinking; Diabetes; Smoke; Mental health history

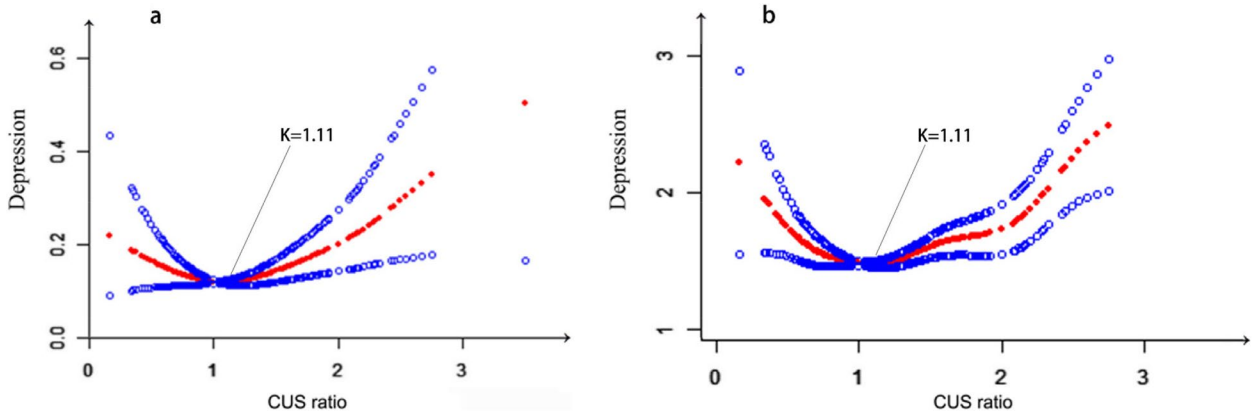

Fig. 2 The smoothing curve fitting results of CUS ratio and Depression. **a** A non-linear relationship between the CUS ratio and depression symptoms (PHQ-9 score of 10 or higher) was identified using the generalized additive model. **b** A non-linear relationship between the CUS ratio and the severity of depressive symptoms (no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression) was also identified. CUS ratio = average weekend sleep duration/average weekday sleep duration. The solid red line represents the smoothed curve fit, while the blue dotted lines indicate the 95% confidence interval

Table 5 Threshold effect analyses of CUS ratio on depression in adults using two-stage logistic regression models in NHANES, 2021–2023

Threshold effect analysis	Depression (PHQ-9 score of 10 or higher) AOR (95%CI), *p*-value	The severity of depressive symptoms Aβ (95%CI), *p*-value
Inflection point of LAP (K)	1.11	1.11
< K slope	0.34 (0.13, 0.89), 0.0273	−0.35 (−0.62, −0.09), 0.0098
> K slope	2.87 (1.84, 4.50), < 0.0001	0.36 (0.24, 0.49), < 0.0001
Log-likelihood ratio test	< 0.001	< 0.001

Sex; Age; Ethnicity; Education level; Marital status; Family monthly poverty level category; BMI; Drinking; Diabetes; Smoke; Mental health history were adjusted

Subgroup analysis

To assess the consistency of the relationship between the CUS ratio and depression across different population subgroups, we conducted a subgroup analysis (Fig. 3). For depression risk, defined as a PHQ-9 score of 10 or higher, the results showed no significant interactions in subgroups stratified by age, gender, race, marital status, household income, weekend sleep duration, alcohol consumption, and smoking history (P for interaction > 0.05). This suggests that the findings from the overall population are robust, with a significant association between the CUS ratio and depression. However, significant interactions were observed between education level, BMI category, diabetes history, and the CUS ratio (P for interaction < 0.05). Subgroup analysis identified specific groups—those with education levels of "Less than 9th grade" or "Some college or AA degree," individuals who are overweight (25 ≤ BMI < 30), and those without a history of diabetes—where a significant association between the CUS ratio and depression was found.

For the severity of depressive symptoms, defined as no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression, the results showed no significant interactions in subgroups stratified by age, gender, race, education level, marital status, household income, BMI category, weekday sleep duration, alcohol consumption, and smoking history (P for interaction > 0.05). This further supports the reliability of the overall findings. However, significant interactions were observed between diabetes history and the CUS ratio (P for interaction < 0.05). Subgroup analysis revealed that individuals without a history of diabetes had a significant association between the CUS ratio and depression.

Mental health history was not included in the subgroup analysis to avoid potential collinearity with depression status itself, ensuring that the focus remained on other socio-demographic and lifestyle factors that could influence depression risk.

Discussion

This study, based on a nationally representative sample from the NHANES 2021–2023, used the CUS ratio as the exposure factor to systematically explore the relationship

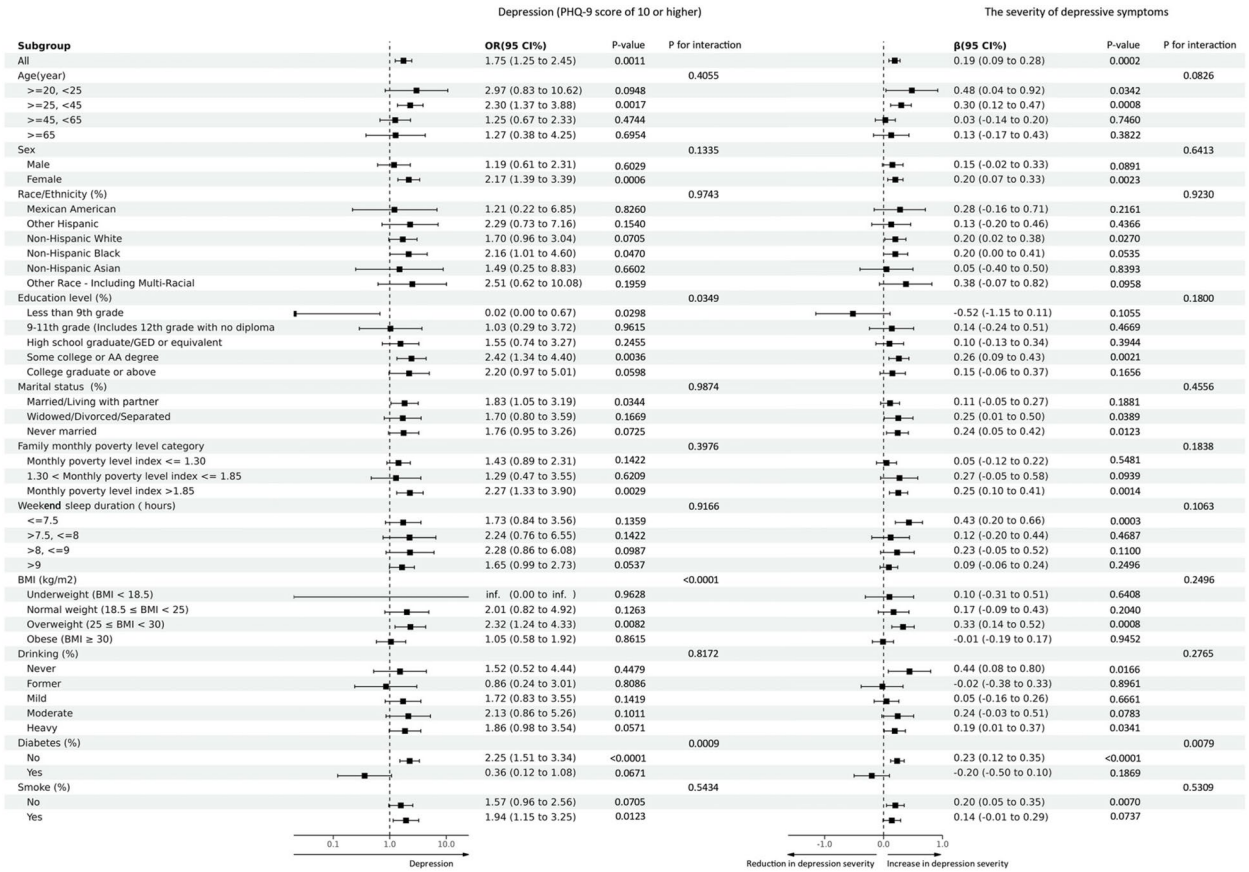

Fig. 3 Stratified analyses for the association between CUS ratio and depression. All models were adjusted for 10 risk factors, excluding stratification variables, and the interaction significance was assessed using the likelihood ratio test

between relative fluctuations in sleep patterns and the symptoms of depression, defined as a PHQ-9 score of 10 or higher. The results revealed a significant positive association between the CUS ratio and depression risk after adjusting for demographic, metabolic, and lifestyle confounders (AOR = 1.75, 95% CI: 1.25–2.45). Furthermore, a nonlinear threshold effect was observed, with an inflection point at a CUS ratio of 1.11. When the CUS ratio was ≤ 1.11, an increase in the CUS ratio was associated with a reduction in depression risk (AOR = 0.34, 95% CI: 0.13–0.89). However, when the CUS ratio exceeded 1.11, each unit increase was associated with a significant 187% increase in depression risk (AOR = 2.87, 95% CI: 1.84–4.50). These findings suggest that moderate sleep compensation may help with sleep recovery, while a CUS ratio above the threshold may reflect a disrupted sleep pattern, which could be a potential marker for increased depression risk. This highlights the importance of maintaining a balanced sleep duration and avoiding extreme fluctuations in sleep patterns.

To further validate the association between the CUS ratio and depression, we conducted a sensitivity analysis by exploring the relationship between sleep fluctuations and the severity of depressive symptoms, categorized as no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression. This analysis confirmed the findings observed for depression risk. Specifically, a significant positive association was found between the CUS ratio and depression severity (Aβ = 0.19, 95% CI: 0.09–0.28), with a similar nonlinear threshold effect at the inflection point of 1.11. When the CUS ratio was ≤ 1.11, each unit increase was associated with a 0.35 unit decrease in depression severity (Aβ = −0.35, 95% CI: −0.62, −0.09, $p = 0.0098$). However, when the ratio exceeded 1.11, each unit increase was associated with a 0.36 unit increase in depression severity (Aβ = 0.36, 95% CI: 0.24, 0.49, $p < 0.0001$). These results reinforce the notion that moderate sleep compensation may reduce depression severity, while excessive fluctuations in sleep duration may worsen symptoms.

In summary, the findings from both the primary analysis of depression risk and the sensitivity analysis of depression severity suggest that the CUS ratio plays a

significant role in influencing mental health outcomes. Moderate sleep compensation appears beneficial for reducing both the risk and severity of depression, while extreme fluctuations in sleep patterns may exacerbate depressive symptoms. These results underscore the importance of not only considering total sleep duration but also the balance of sleep patterns when managing mental health. Personalized sleep management interventions tailored to individual sleep behaviors, combined with enhanced sleep health education, are crucial for improving mental health outcomes and preventing depression.

In our subgroup analysis, we further validated the robustness of the results obtained from the overall population. When depression symptoms was defined as a PHQ-9 score of 10 or higher, we found that certain subgroups, including those with education levels of "Less than 9th grade" or "Some college or AA degree," individuals who are overweight ($25 \leq \text{BMI} < 30$), and those without a history of diabetes, may be more sensitive to fluctuations in sleep patterns.Research has shown that lower-education individuals are more likely to experience higher levels of work-related stress [34]. Moreover, the improvement in educational attainment is associated with a decrease in depressive symptoms [35], suggesting that low-education individuals may lack effective strategies to cope with stress. In this context, increasing weekend sleep duration may be a low-cost, simple, and effective way to help them recover energy and regulate mood, thereby significantly reducing the risk of depression. However, while higher education may act as a protective factor for depression by providing better access to healthcare and more stable employment opportunities, individuals with "Some college or AA degree" benefit less from this protection compared to those with a college degree or higher [36]. If they overcompensate with sleep, they are more likely to disrupt their biological clock, which can lead to imbalances in sleep rhythms and increase the risk of depression. In addition, overweight individuals often face issues like snoring and sleep apnea, which negatively impact their sleep quality [37, 38]. As a result, an increase in CUS ratio may not alleviate the fatigue caused by physiological factors and could instead disrupt their stable sleep patterns, thus increasing the risk of depression. Furthermore, the bidirectional relationship between obesity and depression has been well-documented, with these conditions interacting in a physiological vicious cycle. This cycle involves genetic factors, changes in systems regulating homeostasis, and brain circuits that integrate emotional regulation [39], meaning that the effects of sleep changes may be relatively weaker for this group. Lastly, diabetic patients typically experience metabolic dysfunction and are already at higher risk for sleep deprivation and depression [40]. Therefore, fluctuations in the CUS ratio may not have a noticeable effect on their depression risk. In contrast, non-diabetic individuals, whose metabolic functions are generally normal, show greater sensitivity to changes in the CUS ratio. This is consistent with the response observed in the overall population, and the underlying biological mechanisms are also similar.

For the severity of depressive symptoms, defined as no depressive symptoms, mild depression, moderate depression, moderate-to-severe depression, and severe depression, the subgroup analysis identified a significant interaction only in individuals without a history of diabetes. No significant interaction was observed between education level, BMI category, and the CUS ratio. This discrepancy may be explained by the fact that different grouping methods can lead to varying interactions. The simplified binary classification (depressed vs. non-depressed) is more likely to reveal significant interactions with the CUS ratio. However, when depressive symptoms are subdivided into multiple levels, increased sample heterogeneity, reduced statistical power, and the varying biological and psychological mechanisms underlying different levels of depression severity may influence the ability to detect significant interactions.

The subgroup analysis highlights the need for personalized sleep interventions based on factors such as education level, BMI, and diabetes status. For lower-education individuals, increasing weekend sleep duration may help reduce depression risk by improving mood and energy levels. However, for those with some college education, excessive sleep compensation may disrupt their biological clock, increasing depression risk. Overweight individuals, who often suffer from sleep disorders like snoring and sleep apnea, may not benefit from increased sleep duration and may experience worsened sleep quality and higher depression risk. Additionally, for diabetic patients, whose depression and sleep disturbances are more closely linked to metabolic dysfunction, fluctuations in sleep patterns may have a limited impact. Given these findings, personalized sleep management interventions are crucial. Furthermore, sleep health education should be strengthened to help these groups understand the importance of maintaining regular sleep schedules and moderate weekend sleep compensation for better mental health.

Previous research on weekend catch-up sleep aligns with the findings of this study, both suggesting that moderate weekend catch-up sleep is associated with a lower risk of depression. For example, studies by Le et al. [15] and Liu et al. [41], based on NHANES 2017–2020 data, found that 1 to 2 h of weekend catch-up sleep was significantly associated with a reduced incidence of depression (OR = 0.74, 95% CI: 0.55–0.99; OR = 0.22, 95% CI:

0.08–0.59). Similarly, Kim et al.'s [21] analysis of data from the 2016 Seventh Korean National Health and Nutrition Examination Survey concluded that individuals who had 1 to 2 h of weekend catch-up sleep experienced a significantly lower depression risk (OR = 0.517, 95% CI: 0.309–0.865). Likewise, Li et al. [42] conducted a study with 44,356 middle school students in a district of Shenzhen, China, and found that 0–2 h of weekend catch-up sleep was significantly associated with a reduced risk of mental health symptoms (weekday sleep duration < 8 h: OR = 0.74, 95% CI: 0.61–0.91; weekday sleep duration ≥ 8 h: OR = 0.69, 95% CI: 0.57–0.82). However, unlike previous studies based on CUS, this study standardized the weekday sleep baseline using the CUS ratio model, addressing the limitations of traditional difference indicators that overlook individual heterogeneity. For example, the physiological significance of the same sleep difference varies between short sleepers and long sleepers, meaning that models based solely on differences may not accurately reflect an individual's true situation. This provides a fresh perspective on understanding the potential protective effect of weekend catch-up sleep on depression and, through a more precise model, highlights the complex relationship between catch-up sleep duration and depression risk. These findings offer valuable insights for developing sleep management strategies in clinical and public health interventions in the future.

At the same time, some scholars have offered different perspectives on the extent of weekend catch-up sleep. Zheng et al. [43], in their analysis of NHANES 2017–2018 data, suggested that depression treatment should encourage patients to get adequate sleep on weekends. Similarly, Koo et al. [44], in a study of 8,655 high school students from 15 regions in South Korea, found that weekend catch-up sleep lasting 2 h or more could lower the risk of depression. However, in contrast to these "absolute sleep duration recommendations" based on earlier data (such as from 2016 or 2017–2018), this study utilized more recent data from 2021–2023. Given the fast-paced changes in society and sleep patterns in the post-pandemic era, this data is more in line with current realities.The results suggest that simply extending weekend sleep duration (e.g., ≥ 2 h) may mislead intervention strategies by neglecting individual baseline differences. Therefore, this study proposes that more effective interventions should focus on controlling the CUS ratio. Combining threshold effect analysis, the relationship between the CUS ratio and depression risk is not a linear increase. A CUS ratio between 1 and 1.11 may represent a more universal "safe window," which helps reduce depression risk. For instance, if weekday sleep is 7 h, weekend sleep should not exceed 7.8 h, keeping the ratio under 1.11. This phenomenon may be related to tolerance for circadian rhythm gene phase [45–47] shifts or the diminishing marginal returns of compensatory sleep [48]. It also suggests that while CUS can provide short-term relief, it cannot replace long-term, sufficient sleep [42].

In addition to the above, although increasing sleep duration has consistently been shown to improve mental health outcomes, extending sleep beyond habitual patterns may also lead to a decrease in steady-state sleep drive, resulting in reduced sleep efficiency [49]. This highlights the complexity of sleep interventions, as simply increasing sleep duration does not necessarily lead to improvements in sleep quality or mental health. Furthermore, the phenomenon of social jetlag, which arises from irregular weekend catch-up sleep, has become a key consideration in sleep and mental health research. Studies have shown that social jetlag can contribute to mental health problems, particularly depression, among young people. This effect is more pronounced in females and individuals living in high-latitude regions [50]. Given these factors, the current study focuses on fluctuations in sleep patterns throughout the week, rather than solely on total sleep duration. By considering both sleep consistency and timing, this study helps mitigate the potential risks of irregular sleep patterns while also emphasizing the potential benefits of weekend catch-up sleep within a more nuanced.

The interaction between sleep and depression involves various complex biological mechanisms, potentially linked to inflammation, biochemical pathways, genetic factors, circadian rhythms, and the regulation of nervous system functions. Research suggests that poor sleep quality may activate the inflammatory response, altering the levels of inflammatory markers such as baseline C-reactive protein (CRP), interleukin-6 (IL-6), and soluble tumor necrosis factor receptor 1 (sTNF-αR1). These changes could be key factors in increasing the risk of depression [51–53]. Sleep disorders are commonly associated with depression, and this disorder is closely related to an increase in the percentage of effector memory CD8 + T cells and a decrease in the percentage of CD56 + CD16-NKC cells. The dysregulation of immune markers is considered a potential mechanism by which insomnia triggers depression risk [54, 55]. Genome-wide association studies (GWAS) based on large datasets have deeply explored the genetic pathways, tissues, and cell types associated with insomnia, revealing a significant genetic correlation between depression and sleep duration [56, 57]. The circadian rhythm system plays a central role in regulating the duration, structure, and continuity of the sleep–wake cycle, and irregular sleep patterns have been clearly identified as a risk factor for depression. In animal experiments, subjects displayed hyperactive behavioral traits such as excessive movement, aggression,

and stereotypic behavior, accompanied by a decrease in mitochondrial enzyme activity, reduced serotonin levels, and increased oxidative stress and inflammation markers in the whole brain homogenate. These changes collectively contributed to the occurrence of depression-like behaviors [42, 58, 59]. Furthermore, insufficient sleep may also have potentially harmful long-term effects on white matter development and internalization issues, leading to activation of the prefrontal cortex and further increasing the risk of depression [60, 61]. As one of the most pressing issues facing people today, insufficient sleep has a broad impact on public health. Studies have confirmed that CUS positively affects health [43, 62–64], and also has a beneficial effect on depressive states [15, 21, 41, 42, 44]. This study standardized the workday sleep baseline using the CUS ratio model and found a nonlinear threshold effect (the turning point at 1.11), revealing a more universal "safe window" ($1 < \text{CUS ratio} < 1.11$). This finding more precisely elucidates the complex relationship between sleep duration and depression risk, providing new insights into sleep management and depression prevention.

Advantages and limitations

This study used the CUS ratio model to reveal a significant non-linear threshold effect (turning point at 1.11) between sleep patterns and depression risk. This new finding provides valuable insights into how fluctuations in sleep duration affect mental health, especially depression. The study used the most recent 2021–2023 NHANES data, ensuring the timeliness and real-world relevance of the results. Additionally, this study controlled for a range of demographic, metabolic, and lifestyle confounders and conducted sensitivity analyses using both depression symptoms and the severity of depressive symptoms, enhancing the reliability and robustness of the conclusions. Subgroup analyses also yielded consistent results, emphasizing the importance of tailoring sleep interventions based on factors such as education level, BMI, and diabetes status.

Regarding the public health application of the CUS ratio of 1.11, the findings suggest that this threshold can serve as an important reference for public health interventions, particularly when developing personalized sleep health strategies. Public health policies could consider incorporating this threshold into sleep health assessments and provide tailored interventions for different groups, such as those with education levels of "Less than 9th grade" or "Some college or AA degree," individuals who are overweight ($25 \leq \text{BMI} < 30$), and those without a history of diabetes. Clinicians can use this threshold to help identify patients at higher risk of depression and recommend that patients with irregular sleep patterns make moderate adjustments to their weekend sleep without overcompensating, thus reducing depression risk. Additionally, schools can design reasonable sleep management and health intervention programs, especially for younger or academically stressed students. Human resources departments can promote sleep health education among the workforce, reminding employees to maintain sleep regularity to reduce health problems and improve work efficiency.

Although this study provides important insights, several limitations should be considered. First, the cross-sectional design of the study prevents causal inference, meaning that the relationship between sleep patterns and depression risk cannot be definitively established as causal. However, previous research has identified a bidirectional relationship between sleep duration and depression, with short sleep duration being a risk factor for both the onset and recurrence of depression [65]. Furthermore, studies have indicated that sleep disorders and depression can predict each other, and emotion regulation deficits may play a key role in breaking the cycle between sleep disorders and depression [66]. Second, due to the inherent limitations of the database, this study relied on self-reported sleep duration, which may introduce recall bias and affect the accuracy of the data. However, steps were taken to mitigate this bias. Specifically, we removed missing data, excluded extreme outliers in self-reported sleep duration, and applied three multivariable logistic regression models. Despite these efforts, the limitations of self-reported data should still be acknowledged. Additionally, the NHANES data is collected year-round, making it difficult to determine whether the CUS ratio remains stable across different seasons, which is another limitation of the database. Third, the exclusion of other potential confounding factors, such as social stress, occupational type, and sleep disorders, limits the comprehensiveness of the conclusions. Caution should be exercised when generalizing these findings to different cultural contexts or high-pressure social groups. Fourth, due to the limitations of the database, important factors such as occupational stress and sleep disorders (e.g., insomnia, obstructive sleep apnea [OSA]) were not included in this study. We plan to explore these relationships more thoroughly in future research through prospective cohort studies to better understand the effects of these factors on depression and sleep patterns. Fifth, while this study focused on depression, it did not explore the significant comorbidity between depression and anxiety. Many of the observed effects may not be solely attributed to depression but could also be linked to common features of anxiety. Previous studies have shown that the unique component of depression (such

as anhedonia, i.e., low positive affect) may capture different emotional and cognitive outcomes compared to the overlapping symptoms of depression and anxiety (e.g., general distress, i.e., elevated negative affect) [67]. Furthermore, anxiety-specific symptoms are closely related to cognitive functions, such as memory impairments. Improving sleep quality and increasing sleep duration may help alleviate learning disabilities in individuals with more severe anxiety symptoms [68]. Future research will aim to integrate more objective sleep measurement methods, such as activity trackers, and explore biological mediators (e.g., melatonin, inflammatory markers) through prospective cohort studies to further investigate the mechanisms linking sleep duration and depression and to validate our findings.Additionally, future studies will explore the impact of the CUS ratio on both depression and anxiety symptoms to determine whether the effects are unique or similar.

Conclusion

This study reveals the non-linear relationship between the CUS ratio and depression risk, finding that when the CUS ratio exceeds 1.11, an imbalance in sleep patterns significantly increases the risk of depression. Moderate sleep supplementation is associated with a reduction in depression risk, while excessive sleep supplementation may be linked to an increased risk of depression (the "safe window": 1 < CUS ratio < 1.11). The study suggests that a reasonable balance in sleep duration is crucial for mental health and advocates for incorporating the CUS ratio threshold into depression prevention strategies, with a focus on maintaining circadian rhythm stability rather than a uniform sleep duration target. Future research should use longitudinal designs and circadian biomarkers to verify causal relationships, optimizing precise sleep interventions.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12888-025-07083-w.

Supplementary Material 1: Supplemental Table 1. Baseline characteristics of study participants stratified by CUS ratio.

Acknowledgements
We sincerely thank the NHANES for the data.

Clinical trial number
Not applicable.

Authors' contributions
Shilin Sun: Writing – review & editing, Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. Min Liu: Writing – editing, Writing – original draft, Software, Methodology, Data curation, Conceptualization. Han Liu: Writing – review, Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization.Runzhou Li: Software, Formal analysis, Data curation. Qun Liang: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Conceptualization. Weiwei Quan: Writing – review & editing, Writing – original draft, Visualization, Supervision, Resources, Conceptualization.

Funding
This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability
The data underlying this article are available in the National Health and Nutrition Examination Survey (NHANES) at https://wwwn.cdc.gov/nchs/nhanes/default.aspx.

Declarations

Ethics approval and consent to participate
This study adheres to the ethical guidelines outlined in the Declaration of Helsinki. The data utilized were sourced from the publicly available, de-identified National Health and Nutrition Examination Survey (NHANES). NHANES follows the principles of the Declaration of Helsinki, including obtaining informed consent from participants and ensuring the confidentiality of their data. Since this research involves secondary analysis of NHANES data, no additional ethical approval or consent was required. The study received approval from the Ethics Review Board (ERB) of the National Center for Health Statistics (NCHS).

Competing interests
The authors declare no competing interests.

Author details
[1]Guangzhou Medical University, Guangzhou 511436, China. [2]The Second Affiliated Hospital of Guangzhou Medical University, Guangzhou 510260, China. [3]UCL Medical School, University College London, London, UK. [4]Heilongjiang University of Chinese Medicine, Harbin 150040, China.

Received: 1 April 2025 Accepted: 4 June 2025
Published online: 01 July 2025

References

1. Li SQ, Nan WB, Peng ZY, Huang Q, Chen Q, He BM. Association between methylmalonic acid and prevalence of depression in US adults: evidence from NHANES 2011–2014. Eur J Psychotraumatol. 2025. https://doi.org/10.1080/20008066.2025.2450109.
2. Wang XR, Zhu BQ, Li JP, Li XY, Zhang LN, Wu YB, Ji LL. The moderating effect of frailty on the network of depression, anxiety, and loneliness in community-dwelling older adults. J Affect Disord. 2025;375:508–16.
3. Jeevarajan JR, Theodorou A, Nandy K, Guerra M, Madia ND, Claassen C, Goodman LC, Farmer D, Wakefield SM, Minhajuddin A, Slater H, Mayes T, Soutullo CA, Rosenberg A, Ladd S, Ahumada N, Jackson S, Rush AJ, Trivedi MH. Psychometric properties of the 10-item Connor-Davidson Resilience Scale (CD-RISC-10) in adolescent and young adult psychiatric outpatients in the Texas Youth Depression and Suicide Research Network (TX-YDSRN). J Affect Disord. 2025;375:155–64.
4. Zhang L, Yin J, Sun H, Dong W, Liu Z, Yang J, Liu Y. The relationship between body roundness index and depression: a cross-sectional study using data from the National Health and Nutrition Examination Survey (NHANES) 2011–2018. J Affect Disord. 2024;361:17–23.
5. Paykel ES. Basic concepts of depression. Dialogues Clin Neurosci. 2008;10:279–89.
6. Fusar-Poli P, Estradé A, Stanghellini G, Esposito CM, Rosfort R, Mancini M, Norman P, Cullen J, Adesina M, Jimenez GB, da Cunha Lewin C, Drah EA, Julien M, Lamba M, Mutura EM, Prawira B, Sugianto A, Teressa J, White LA, Damiani S, Vasconcelos C, Bonoldi I, Politi P, Vieta E, Radden J, Fuchs T, Ratcliffe M, Maj M. The lived experience of depression: a bottom-up review co-written by experts by experience and academics. World Psychiatry. 2023;22:352–65.

7. Limenih G, MacDougall A, Wedlake M, Nouvet E. Depression and global mental health in the global south: a critical analysis of policy and discourse. Int J Soc Determ Health Health Serv. 2024;54:95–107.
8. Luo L, Yuan J, Wu C, Wang Y, Zhu R, Xu H, Zhang L, Zhang Z. Predictors of depression among Chinese college students: a machine learning approach. BMC Public Health. 2025;25:470.
9. Zanardi R, Carminati M, Attanasio F, Fazio V, Maccario M, Colombo C. How different definition criteria may predict clinical outcome in treatment resistant depression: results from a prospective real-world study. Psychiatr Res. 2024;334:115818.
10. Minbay M, Khan A, Ghasemi AR, Ingram KK, Ay AA. Sex-specific associations between circadian-related genes and depression in UK Biobank participants highlight links to glucose metabolism, inflammation and neuroplasticity pathways. Psychiatr Res. 2024. https://doi.org/10.1016/j.psychres.2024.115948.
11. Harrington MO, Pennington K, Durrant SJ. The 'affect tagging and consolidation' (ATaC) model of depression vulnerability. Neurobiol Learn Memory. 2017;140:43–51.
12. Alhusaini M, Eissa N, Saad AK, Beiram R, Sadek B. Revisiting preclinical observations of several histamine H3 receptor antagonists/inverse agonists in cognitive impairment, anxiety, depression, and sleep-wake cycle disorder. Front Pharmacol. 2022. https://doi.org/10.3389/fphar.2022.861094.
13. Yang P, Tian LH, Xia Y, Hu MY, Xiao X, Leng YP, Gong LG. Association of sleep quality and its change with the risk of depression in middle-aged and elderly people: a 10-year cohort study from England. J Affect Disord. 2025;373:245–52.
14. Gravante F, Pucciarelli G, Sperati F, Cecere L, Anastasi G, Mancin S, Rea T, Latina R. Risk factors of anxiety, depression, stress, job burnout, and characteristics of sleep disorders in critical care nurses: an observational study. Aust Crit Care. 2025. https://doi.org/10.1016/j.aucc.2024.101165.
15. Le L, Lan Z, Chen C. Association between weekend catch-up sleep and depression in US adults. BMC Public Health. 2025;25:439.
16. Mirchandaney R, Asarnow LD, Kaplan KA. Recent advances in sleep and depression. Curr Opin Psychiatry. 2023;36:34–40.
17. Dong L, Xie YW, Zou XH. Association between sleep duration and depression in US adults: a cross-sectional study. J Affect Disord. 2022;296:183–8.
18. Sweetman A, Lack L, Bastien C. Co-morbid insomnia and sleep apnea (COMISA): prevalence, consequences, methodological considerations, and recent randomized controlled trials. Brain Sci. 2019. https://doi.org/10.3390/brainsci9120371.
19. Kalra S, Nagaraja N, Kalra D. Increased risk of suicidal ideation and/or self-harm in co-morbid insomnia and obstructive sleep apnea (COMISA). Sleep Epidemiol. 2024;4:100088.
20. Lang CJ, Appleton SL, Vakulin A, McEvoy RD, Wittert GA, Martin SA, Catcheside PG, Antic NA, Lack L, Adams RJ. Co-morbid OSA and insomnia increases depression prevalence and severity in men. Respirology. 2017;22:1407–15.
21. Kim KM, Han SM, Min IK, Heo K, Kim WJ, Chu MK. Weekend catch-up sleep and depression: results from a nationally representative sample in Korea. Sleep Med. 2021;87:62–8.
22. Lee H, Kim YJ, Jeon YH, Kim SH, Park EC. Association of weekend catch-up sleep ratio and subjective sleep quality with depressive symptoms and suicidal ideation among Korean adolescents. Sci Rep. 2022;12:10235.
23. Hou XZ, Lv YF, Li YS, Wu Q, Lv QY, Yang YT, Li LL, Ye XJ, Yang CY, Wang MS, Cao LL, Wang SH. Association between different insulin resistance surrogates and all-cause mortality in patients with coronary heart disease and hypertension: NHANES longitudinal cohort study. Cardiovasc Diabetol. 2024;23:86.
24. Qi X, Wang S, Huang Q, Chen X, Qiu L, Ouyang K, Chen Y. The association between non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR) and risk of depression among US adults: a cross-sectional NHANES study. J Affect Disord. 2024;344:451–7.
25. Gu W, Bao K, Li X, Xiang S, He J, He J, Ye L, Huang Z. Association between body fat percentage and depression: a cross-sectional study of NHANES. J Affect Disord. 2025;371:305–14.
26. Zhang Z, Ruan XY, Ma W. The association between a body shape index and depressive symptoms: a cross-sectional study using NHANES data (2011–2018). Front Nutr. 2024;11:1510218.
27. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16:606–13.
28. He J, Chen L. Perspective from NHANES data: synergistic effects of visceral adiposity index and lipid accumulation products on diabetes risk. Sci Rep. 2025;15:258.
29. Liu Y, Zhang L, Yang C, Zhi L, Xu XS, Yuan M. Oral microbiome diversity shapes the association between sleep duration and depression. Front Neurol. 2024. https://doi.org/10.3389/fneur.2024.1442557.
30. Zhang Z, Tang Z, Zhang Z-X. Association between recreational physical activity and depressive symptoms in adults with diagnosed sleep disorders: an analysis of NHANES 2007–2018 data. BMC Psychiatry. 2025;25:150.
31. Shi J, Liang Z, Zhang X, Ren S, Cheng Y, Liu Y, Zhang M. Association of physical activity and dietary inflammatory index with overweight/obesity in US adults: NHANES 2007–2018. Environ Health Prev Med. 2023;28:40.
32. Li M, Yu X, Zhang W, Yin J, Zhang L, Luo G, Liu Y, Yang J. The association between weight-adjusted-waist index and depression: results from NHANES 2005–2018. J Affect Disord. 2024;347:299–305.
33. Burruss NC, Girgis M, Green KE, Lu L, Palakshappa D. Association between food insecurity and access to a mental health professional: cross-sectional analysis of NHANES 2007–2014. BMC Public Health. 2021;21:754.
34. Lunau T, Siegrist J, Dragano N, Wahrendorf M. The association between education and work stress: does the policy context matter? PLoS ONE. 2015;10:e0121573.
35. Li L, Sun W, Luo J, Huang H. Associations between education levels and prevalence of depressive symptoms: NHANES (2005–2018). J Affect Disord. 2022;301:360–7.
36. Bauldry S. Variation in the protective effect of higher education against depression. Soc Ment Health. 2015;5:145–61.
37. Campos AI, García-Marín LM, Byrne EM, Martin NG, Cuéllar-Partida G, Rentería ME. Insights into the aetiology of snoring from observational and genetic investigations in the UK Biobank. Nat Commun. 2020;11:817.
38. Dong Z, Xu X, Wang C, Cartledge S, Maddison R, Shariful Islam SM. Association of overweight and obesity with obstructive sleep apnoea: a systematic review and meta-analysis. Obes Med. 2020;17:100185.
39. Milaneschi Y, Simmons WK, van Rossum EFC, Penninx BWJH. Depression and obesity: evidence of shared biological mechanisms. Mol Psychiatry. 2019;24:18–33.
40. Zhang P, Lou P, Chang G, Chen P, Zhang L, Li T, Qiao C. Combined effects of sleep quality and depression on quality of life in patients with type 2 diabetes. BMC Fam Pract. 2016;17:40.
41. Liu YC, Yin JH, Li XH, Yang JG, Liu YX. Examining the connection between weekend catch-up sleep and depression: insights from 2017 to 2020 NHANES information. J Affect Disord. 2024;358:61–9.
42. Li M, Zhang Y, Huang M, Ma Z, Wang D, Fan F. Associations of weekend compensatory sleep and weekday sleep duration with psychotic-like experiences among Chinese adolescents. J Affect Disord. 2025;372:627–34.
43. Zheng YX, Bao J, Tang L, Chen CH, Guo WQ, Li KC, Zhang RX, Wu JZ. Association between weekend catch-up sleep and depression of the United States population from 2017 to 2018: a cross-sectional study. Sleep Med. 2024;119:9–16.
44. Koo DL, Yang KI, Kim JH, Kim D, Sunwoo JS, Hwangbo Y, Lee HR, Hong SB. Association between morningness-eveningness, sleep duration, weekend catch-up sleep and depression among Korean high-school students. J Sleep Res. 2021. https://doi.org/10.1111/jsr.13063.
45. Brenna A, Borsa M, Saro G, Ripperger JA, Glauser DA, Yang Z, Adamantidis A, Albrecht U. Cyclin-dependent kinase 5 (Cdk5) activity is modulated by light and gates rapid phase shifts of the circadian clock. Elife. 2025;13:RP97029.
46. Chhe K, Hegde MS, Taylor SR, Farkas ME. Circadian effects of melatonin receptor-targeting molecules in vitro. Int J Mol Sci. 2024;25:13508.
47. de Assis LVM, Kramer A. Circadian de (regulation) in physiology: implications for disease and treatment. Genes Dev. 2024;38:933–51.
48. Liu Z, Song YJ. Weekend compensatory sleep is associated with reduced risk of heart disease: a prospective UK Biobank-based cohort study. Eur Heart J. 2024. https://doi.org/10.1093/eurheartj/ehae666.3582.
49. Niu X, Zhou S, Casement MD. The feasibility of at-home sleep extension in adolescents and young adults: a meta-analysis and systematic review. Sleep Med Rev. 2021;58:101443.
50. Henderson SEM, Brady EM, Robertson N. Associations between social jetlag and mental health in young people: a systematic review. Chronobiol Int. 2019;36:1316–33.

51. Huang MH, Chan YLE, Chen MH, Hsu JW, Huang KL, Li CT, Tsai SJ, Su TP, Bai YM. A longitudinal study of the association between pro-inflammatory cytokines and mood symptoms in bipolar disorder. Acta Psychiatr Scand. 2023;147:81–91.
52. Huang MH, Chan YLE, Chen MH, Hsu JW, Huang KL, Li CT, Tsai SJ, Bai YM, Su TP. Pro-inflammatory cytokines and cognitive dysfunction among patients with bipolar disorder and major depression. Psychiatry Clin Neurosci. 2022;76:450–8.
53. Gimeno D, Kivimäki M, Brunner EJ, Elovainio M, De Vogli R, Steptoe A, Kumari M, Lowe GDO, Rumley A, Marmot MG, Ferrie JE. Associations of C-reactive protein and interleukin-6 with cognitive symptoms of depression: 12-year follow-up of the Whitehall II study. Psychol Med. 2009;39:413–23.
54. Suzuki H, Savitz J, Teague TK, Gandhapudi SK, Tan C, Misaki M, McKinney BA, Irwin MR, Drevets WC, Bodurka J. Altered populations of natural killer cells, cytotoxic T lymphocytes, and regulatory T cells in major depressive disorder: association with sleep disturbance. Brain Behav Immun. 2017;66:193–200.
55. Besedovsky L, Lange T, Haack M. The sleep-immune crosstalk in health and disease. Physiol Rev. 2019;99:1325–80.
56. Jansen PR, Watanabe K, Stringer S, Skene N, Bryois J, Hammerschlag AR, de Leeuw CA, Benjamins JS, Muñoz-Manchado AB, Nagel M, Savage JE, Tiemeier H, White T, Agee M, Alipanahi B, Auton A, Bell RK, Bryc K, Elson SL, Fontanillas P, Furlotte NA, Hinds DA, Huber KE, Kleinman A, Litterman NK, McCreight JC, McIntyre MH, Mountain JL, Noblin ES, Northover CAM, Pitts SJ, Sathirapongsasuti JF, Sazonova OV, Shelton JF, Shringarpure S, Tian C, Wilson CH, Tung JY, Hinds DA, Vacic V, Wang X, Sullivan PF, van der Sluis S, Polderman TJC, Smit AB, Hjerling-Leffler J, Van Someren EJW, Posthuma D, T. The 23andMe Research. Genome-wide analysis of insomnia in 1,331,010 individuals identifies new risk loci and functional pathways. Nat Genet. 2019;51:394–403.
57. Lind MJ. Analysis of novel sleep variable highlights shared genetics of sleep and psychiatric disorders. Sleep. 2024;47:1-3. https://doi.org/10.1093/sleep/zsad311.
58. Arora S, Dharavath RN, Bansal Y, Bishnoi M, Kondepudi KK, Chopra K. Neurobehavioral alterations in a mouse model of chronic partial sleep deprivation. Metab Brain Dis. 2021;36:1315–30.
59. Agamme A, Tufik S, Torterolo P, D'Almeida V. Effects of paradoxical sleep deprivation on MCH and hypocretin systems. Sleep Sci. 2024;17:e392–400.
60. Yang S, Kong X, Zhang X, Zhang Y, Li X, Ge YJ. The impact of insomnia on prefrontal activation during a verbal fluency task in patients with major depressive disorder: a preliminary fNIRS study. Sleep Med. 2025;125:114–9.
61. Guldner S, Sarvasmaa AS, Lemaître H, Massicotte J, Vulser H, Miranda R, Bezivin – Frère P, Filippi I, Penttilä J, Banaschewski T, Barker GJ, Bokde ALW, Bromberg U, Büchel C, Conrod PJ, Desrivières S, Flor H, Frouin V, Gallinat J, Garavan H, Gowland P, Heinz A, Nees F, Papadopoulos-Orfanos D, Smolka MN, Schumann G, Artiges E, Martinot MLP, Martinot JL. Longitudinal associations between adolescent catch-up sleep, white-matter maturation and internalizing problems. Dev Cogn Neurosci. 2023;59:101193.
62. Oh J, Kim E, Huh I. Associations between weekend catch-up sleep and health-related quality of life with focusing on gender differences. Sci Rep. 2023. https://doi.org/10.1038/s41598-023-47244-z.
63. Zhu H, Qin S, Wu M. Association between weekend catch-up sleep and cardiovascular disease: evidence from the National Health and Nutrition Examination Surveys 2017–2018. Sleep Health. 2024;10:98–103.
64. Chen S, Zhang T, Gao H, Zhang J. Association between weekend catch-up sleep and chronic kidney disease: insights from NHANES 2017–2020. Ren Fail. 2025;47:2461682.
65. Sun Y, Shi L, Bao Y, Sun Y, Shi J, Lu L. The bidirectional relationship between sleep duration and depression in community-dwelling middle-aged and elderly individuals: evidence from a longitudinal study. Sleep Med. 2018;52:221–9.
66. Niu X, Snyder HR. The role of maladaptive emotion regulation in the bidirectional relation between sleep and depression in college students during the COVID-19 pandemic. Anxiety Stress Coping. 2023;36:83–96.
67. den Hollander-Gijsman ME, Wardenaar KJ, de Beurs E, van der Wee NJA, Mooijaart A, van Buuren S, Zitman FG. Distinguishing symptom dimensions of depression and anxiety: an integrative approach. J Affect Disord. 2012;136:693–701.
68. Niu X, Utayde MF, Sanders KEG, Cunningham TJ, Zhang G, Kensinger EA, Payne JD. The effects of shared, depression-specific, and anxiety-specific internalizing symptoms on negative and neutral episodic memories following post-learning sleep. Cogn Affect Behav Neurosci. 2025;25:114–34.

Publisher's Note