BMC Medicine

RESEARCH

Open Access

Investigating the associations between weekend catch-up sleep and insulin resistance: NHANES cross-sectional study

Xianling Liu[1], Aihui Chu[2] and Xiahao Ding[3*]

Abstract

Background Insulin resistance (IR) is a precursor to metabolic syndrome. Weekend catch-up sleep (WCS) is practiced to compensate for insufficient weekday sleep, but its impact on IR remains unclear. This study investigated associations between WCS and severe IR risk.

Methods Data from 1,903 adults participating in the National Health and Nutrition Examination Survey 2017–2020 were analyzed. IR was assessed using the Homeostatic Model Assessment for IR (HOMA-IR) and Metabolic Score for IR (METS-IR), with severe IR defined as the highest quartile. WCS was calculated by subtracting weekday sleep duration from weekend sleep duration and was categorized into five groups. Weighted logistic regression and restricted cubic spline analyses were performed to examine associations between WCS patterns and severe IR risk. Percentages reported were weighted to account for sampling design and population distribution.

Results The majority of participants were under 60 yrs (75.2%, $n=1,344$) and had a body mass index below 30 kg/m^2 (59.2%, $n=1,082$). Slightly more than half of the participants were female (51.3%, $n=990$). A U-shaped relationship between WCS duration and severe IR risk was observed, with the lowest risk at approximately 0.7–1.0 h of WCS. Short WCS durations ($0<\text{WCS}\leq 1$ h) were associated with a significantly reduced risk of severe IR as defined by HOMA-IR (OR = 0.63, 95% CI: 0.41–0.97, $P=0.037$) compared to stable sleep pattern (WCS = 0). Long WCS durations (WCS ≥ 2 h) were associated with an increased risk of severe IR as defined by METS-IR (OR = 1.88, 95% CI: 1.13–3.14, $P=0.018$). Sensitivity analyses showed that the reduction in severe IR risk associated with short WCS durations was more significant in individuals with weekday sleep durations of seven hours or less.

Conclusions WCS duration exhibits a U-shaped association with severe IR risk, with approximately 0.7–1.0 h of WCS linked to the lowest risk. Both insufficient and excessive WCS are associated with increased severe IR risk, emphasizing the importance of optimal sleep patterns for metabolic health.

Keywords Weekend catch-up sleep, Insulin resistance, National Health and Nutrition Examination Survey, Homeostatic Model Assessment for Insulin Resistance, Metabolic Score for Insulin Resistance

*Correspondence:
Xiahao Ding
mzkdxh@hotmail.com
Full list of author information is available at the end of the article

Background

Metabolic syndrome (MetS) is a cluster of metabolic abnormalities, including abdominal obesity, elevated blood pressure, impaired glucose tolerance, and dyslipidemia, which is prevalent in approximately 34 percent of Americans and 25 percent of the world populace [1, 2]. As an increasing global public health challenge, MetS has entailed an enormous financial burden and medical care policy issues [3]. Insulin resistance (IR) has been proven to be a common pathophysiological mechanism and a precursor for MetS, which is defined physiologically as a state of reduced responsiveness in insulin-targeting tissues to high physiological insulin levels [4, 5]. Therefore, screening and early warning for populations at high risk for IR may be the basis and key step for preventive treatment and management of MetS.

A growing number of people have experienced declined sleep duration and poor sleep quality during recent years [6–8]. More than one-third of U.S. adults report sleeping less than the recommended seven hours per night and 30% report sleeping less than six hours per night [9]. These trends may be strongly associated with MetS prevalence, such as type 2 diabetes, hypertension, hyperlipidemia, and cardiovascular diseases [10–12]. Epidemiologic evidence has accumulated to support the role of long-term insufficient sleep as an independent risk factor for the development and exacerbation of IR [13, 14]. Experimental studies have also shown that sleep deprivation is associated with changes in IR and β-cell function [15, 16]. In this situation, one common strategy to recover from insufficient sleep during work or school days is to catch up or recover on sleep during weekends or free days [17, 18]. Previous studies [19–21] examining the relationship between weekend catch-up sleep (WCS) and MetS have produced inconsistent results. Observational studies have suggested that WCS may reduce the risk of MetS in middle-aged individuals with chronic short sleep [19, 20], while a randomized controlled trial found no significant protective effects of WCS in preventing MetS associated with recurrent insufficient sleep [21]. These mixed findings highlight the need for further investigation into the role of WCS in metabolic health. Although WCS has been studied in the context of MetS, limited research has specifically focused on its potential impact on IR, a key precursor to diabetes and cardiovascular diseases.

Furthermore, previous studies indicate that the relationship between sleep behavior and metabolic health can differ by demographic characteristics [22, 23]. Sex differences in the link between sleep and insulin sensitivity may stem from biological variations in hormone regulation or sleep architecture [22]. Additionally, married individuals—who often have more regular sleep patterns and lower psychological stress—may derive greater benefits from sleep supplementation, whereas single or divorced individuals may experience diminished protective effects due to social isolation or disrupted sleep patterns [23–25]. However, it remains unclear whether the associations between WCS and IR exhibits similar heterogeneity across different populations. Therefore, this study seeks to fill these gaps by investigating the association between WCS and IR and exploring whether these associations vary among diverse demographic groups.

This study leveraged data from the National Health and Nutrition Examination Survey (NHANES) to investigate the associations between the pattern and time of WCS and IR among U.S. adults. To assess IR, we utilized two complementary methods: Homeostatic Model Assessment for IR (HOMA-IR) and Metabolic Score for IR (METS-IR). HOMA-IR, a well-established clinical marker, is calculated from fasting glucose and insulin levels, providing insights into hepatic insulin resistance [26, 27]. On the other hand, METS-IR includes additional metabolic variables, such as lipid profiles, that contribute to a broader assessment of IR, encompassing peripheral insulin sensitivity as well [28]. By employing both HOMA-IR and METS-IR, this study captures distinct dimensions of IR, allowing for a comprehensive evaluation of the potential relationship between WCS and IR. Understanding the potential relationship between WCS and IR can provide information for public health recommendations and personal behavioral strategies, potentially refining guidelines and interventions aimed at reducing the burden of IR and related metabolic disorders in the general population.

Methods

Data source and study population

NHANES is a continuous and population-based cross-sectional survey designed to assess the nutrition and health status of the general population in the U.S. The survey employs a complex, multistage probability sampling design to provide information on demographic characteristics, socioeconomic status, physiological measurements, biochemical indicators, and various health-related aspects, and to ensure that participants in each two-year cycle are representative of the national population. Meanwhile, participants in NHANES provided written informed consent, and the study protocol was approved by the Research Ethics Review Board of the National Center for Health Statistics and the U.S. Army Research Institute of Environmental Medicine Human Use Review Committee. Detailed information can be achieved at (https://www.cdc.gov/nchs/nhanes/index.htm).

A total of 15,560 individuals from NHANES 2017–2020 were initially considered for inclusion in this study. Participants were excluded if they had missing key data, including information on sleep-related variables (n = 6,567), metabolism-related parameters required to calculate IR indices, such as fasting plasma glucose (FPG), fasting serum insulin (FINS), triglyceride (TG), high-density lipoprotein cholesterol (HDL-c), or body mass index (BMI) (n = 5,265), socioeconomic variables such as education level, marital status, or family income measured as the ratio of family income to poverty (FPIR) (n = 771), and lifestyle factors such as smoking status, alcohol consumption, or dietary recall information (n = 1,054). After applying these exclusion criteria, a total of 1,903 individuals were included in the final analysis. A detailed overview of the participant selection process and exclusion criteria was presented in Fig. 1. Since all participants in NHANES provided written informed consent and only publicly available data were used in this analysis, no ethical approval was required.

Definitions of IR

In this study, HOMA-IR and METS-IR were calculated to provide parameters of whole-body IR [26, 29]. For subsequent weighted logistic regression models, the entire sample was divided into quartiles based on the two IR indicators mentioned above, with the highest quartile representing the severe IR [30]. According to the NHANES protocol, FINS levels were determined by the AIA-PACK IRI, a two-site immunoenzymometric assay performed on a Tosoh AIA Chemistry Analyzer. FPG, TG, and HDL-c levels were measured by an automated biochemical analysis instrument. The formulas for the two indices were presented as follows: HOMA-IR = FINS (μU/ml) × FPG (mmol/L)/22.5 [26]; METS-IR = Ln [(2 × FPG (mg/dL) + fasting TG (mg/dL)] × BMI (kg/m^2)/Ln [HDL-c (mg/dL)] [29].

Assessments of sleep parameters

In the NHANES surveys conducted between 2017 and 2020, participants reacted to the following sleep-related doubts to find out their sleep duration, quality, and regularity: (1)"the number of hours usually sleep on weekdays or workdays", (2)"the number of hours usually sleep on weekends or non-workdays", (3)"what time do you usually fall asleep on weekdays or workdays?", (4)"what time do you usually wake up on weekdays or workdays?", (5)"what time do you usually fall asleep on weekends or non-workdays?", (6)"what time do you usually wake up on weekends or non-workdays?", (7)"in the past 12 months, how often did you snore while sleeping?", (8)"how often did you snort, gasp, or stop breathing?", (9)"ever told a doctor or other health professional that you have trouble sleeping?", (10)"how often feel excessively or overly sleepy during day?".

The average sleep duration was calculated using the following formula [31, 32]: Average sleep duration = (5 × weekday sleep duration + 2 × weekend sleep duration)/7.

Social jetlag status was assessed based on responses regarding typical bedtimes, and wake times on weekdays and weekends. The midpoint of sleep was assessed by the sleep onset time and wake time. This midpoint was computed separately for weekdays (MSW) and weekends (MSF). Social jetlag was defined as the absolute difference between these two sleep midpoints, expressed in hours (|MSF-MSW|) [33]. In our study population, 98% of participants exhibited social jetlag ranging between 0

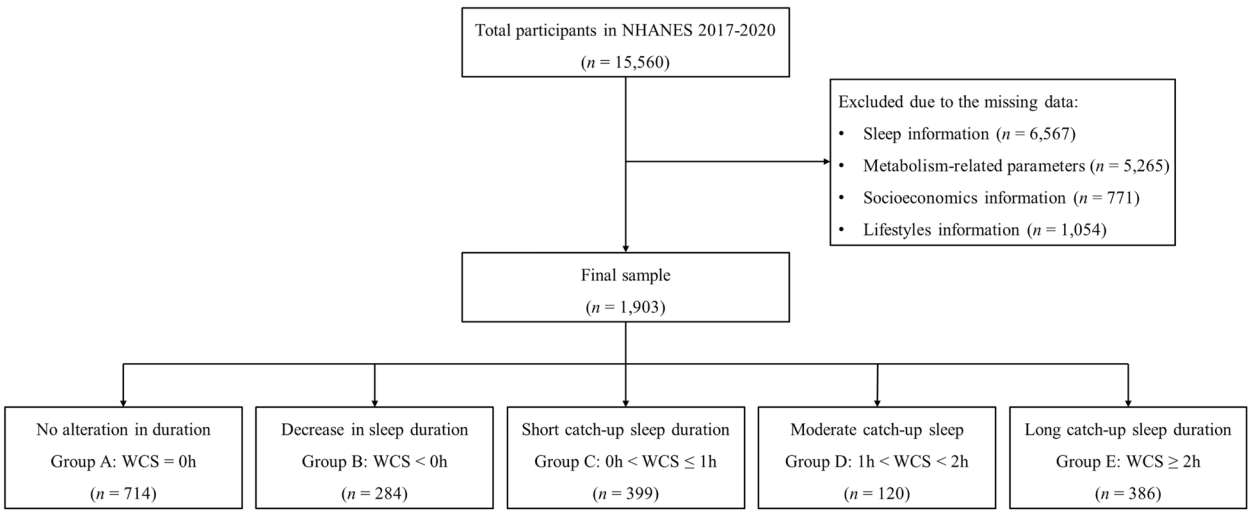

Fig. 1 Flow chart of the study population. Abbreviation: NHANES, National Health and Nutrition Examination Survey; WCS, weekend catch-up sleep

and 4.75 h. To facilitate analysis, participants were categorized into three groups: Less than one hour of social jetlag, one to less than two hours of social jetlag, and two or more hours of social jetlag. Consistent with previous studies, we used participants with less than 1 h of social jetlag as the reference group [34, 35].

The time of WCS was defined as the time of sleep on weekends minus the time of sleep on weekdays according to the patient's self-report. It was then classified into five groups [36]: stable sleep pattern (Group A, WCS = 0), decrease in sleep duration (Group B, WCS < 0), short catch-up sleep duration (Group C, 0 < WCS ≤ 1 h), moderate catch-up sleep duration (Group D, 1 < WCS < 2 h), and long catch-up sleep duration (Group E, WCS ≥ 2 h).

Assessments of covariates

The selection of covariates adhered to biological considerations and previously published literature [19, 32, 37]. Demographic characteristics were elucidated in the demographic questionnaire, encompassing age (< 60, ≥ 60), sex (male, female), race (Mexican American, non-Hispanic black, non-Hispanic white, other races), education level (< high school, high school, and > high school), marital status (never married, married/living with a partner, and widowed/divorced/separated), and FPIR [low income (≤ 1.3), middle income (> 1.3 and ≤ 3.5), and high income (> 3.5)] [38].

Furthermore, information regarding smoking and alcohol intake status was derived from the cigarette use and alcohol use questionnaires. Smoking status was categorized into non-smokers, former smokers, and current smokers (non-smokers: smoked less than 100 cigarettes in life; former smokers: smoked more than 100 cigarettes in life and smoke not at all now; current smokers: smoked more than 100 cigarettes in life and smoke some days or every day) [39]. Alcohol intake status encompassed never drinking, former drinking, and current drinking, further classified into mild, moderate, or heavy drinking based on the frequency and amount of consumption (current heavy drinking: ≥ 3 drinks per day for females, ≥ 4 drinks per day for males, or binge drinking on 5 or more days per month; current moderate drinking: ≥ 2 drinks per day for female, ≥ 3 drinks per day for males, or binge drinking ≥ 2 days per month; current mild drinking: not meet the above) [40].

In addition, daily energy intake (kcal/d) was obtained from two 24-h dietary recall interviews, calculated by averaging the total energy intake on Day 1 and Day 2. The physical activity (PA) data in the NHANES survey were collected using the Global Physical Activity Questionnaire, developed by the World Health Organization [41]. This questionnaire assesses PA across three domains: occupational (work-related activities), transportation (such as walking or cycling), and recreational (leisure-time exercise). Participants reported the frequency, duration, and intensity of activities within each domain. For ease of analysis, the duration of vigorous activities was converted to an equivalent amount of moderate activity using a standard conversion: one minute of vigorous PA is equivalent to two minutes of moderate PA [42, 43]. The calculation formula used is as follows: total PA time per week = moderate activity frequency per week × duration per session + 2 × vigorous activity frequency per week × duration per session. According to the World Health Organization guidelines [44], participants who engage in moderate intensity aerobic physical activity for ≥ 150 min per week are classified as high levels of PA. People who do not meet these thresholds are classified as having low PA levels, while participants who report no PA are defined as inactive.

Comorbidities considered in the study include hypertension and obesity. Hypertension was defined by the following criteria: (1) average systolic blood pressure ≥ 140 mmHg, (2) average diastolic blood pressure ≥ 90 mmHg, or (3) the use of antihypertensive medication, or (4) subjects with a self-reported hypertension diagnosis [45]. In our study, height and weight were directly measured by trained NHANES personnel as part of the physical examination. BMI is calculated by dividing an individual's weight (in kilograms) by the square of their height (in meters). Obesity was defined as BMI ≥ 30 kg/m^2 for the need for subgroup analysis [46].

Statistical analysis

We adopted NHANES-recommended sample weights to account for the survey's complex sampling design. We conducted descriptive statistics on population characteristics based on the categories of WCS. Use the Shapiro–Wilk test to assess the normality of the data. Variables that conform to normal distribution are described using mean and standard deviation (SD), and intergroup differences are analyzed using one-way analysis of variance. Variables that deviate from normality are characterized by median and interquartile range (IQR), and we use Kruskal–Wallis rank-sum test to conduct groups comparisons. Categorical variables are expressed in sample counts and weighted percentages, and Rao-Scott chi-square test is used to analyze the population characteristics of different sleep patterns.

Using weighted logistic regression to explore the influencing factors of severe IR. We also used weighted logistic regression models to investigate the relationship between WCS and severe IR risk in the general population. Model 1 was adjusted based on demographic data, while Model 2 was adjusted based on demographic data and lifestyle factors; Model 3 was also adjusted based on

comorbidities; Model 4 was further adjusted for sleep related factors (such as average sleep time, social jetlag, snore, stop breathing, trouble sleeping, and overly sleepy during day) based on Model 3.

A logistic regression model with weighted restricted cubic spline (RCS) was used to investigate the potential nonlinear relationship between WCS duration and severe IR risk. We tested nodes between three and seven and selected the model with the lowest Akaike information standard value for RCS. If RCS analysis shows that U-shaped, inverted U-shaped, or L-shaped curves have identifiable inflection points, the data is divided into two different parts based on the inflection points and segmented logistic regression is performed to gain a more detailed understanding of the relationship between the duration of WCS and IR.

Subgroup analyses were conducted to investigate whether this association varies due to demographic variables (age, sex, race, education level, and marital status), and likelihood tests were used to test the interaction effects. Sensitivity analyses were performed to explore associations between WCS and severe IR risk at different weekday sleep durations.

All analyses in this research were conducted using *R* software (version 4.4.0). All statistical tests were two-sided, with the significant threshold considered at $\alpha = 0.05$.

Results

Baseline characteristics of participants stratified by patterns of WCS

Table 1 summarizes the baseline characteristics of participants according to WCS patterns. Key demographic, lifestyle, and clinical variables showed statistically significant differences across groups. Younger participants (< 60 years) predominantly belonged to Groups B through E, while elderly participants (≥ 60 years) were more commonly observed in Group A ($P < 0.001$). Non-Hispanic Whites were most prevalent in Groups A and D ($P = 0.004$), whereas Groups C and D had more individuals with higher educational attainment ($P < 0.001$). Social jetlag followed a gradient pattern, increasing progressively from Group A to Group E ($P < 0.001$). Groups D and E exhibited longer weekend sleep times (median 9.0–9.5 h) ($P < 0.001$). Regarding metabolic parameters, glucose levels differed significantly across groups ($P = 0.046$), with Group B displaying lower median glucose levels. However, no significant differences were noted for HOMA-IR or METS-IR metrics of IR, nor for their associated severe IR prevalence across the groups.

Associations between patterns of WCS and severe IR risk defined by HOMA-IR and METS-IR indices

We first explored the associations between WCS patterns and severe IR using univariate and multivariable weighted logistic regression (Table 2). For severe IR defined by HOMA-IR, participants in Group C (0 < WCS ≤ 1 h) had a significantly lower risk of severe IR compared to Group A (WCS = 0) (OR = 0.63, 95% CI: 0.41–0.97, $P = 0.037$), while defined by METS-IR, those in Group E (WCS ≥ 2 h) showed a notably higher risk (OR = 1.88, 95% CI: 1.13–3.14, $P = 0.018$). To further examine these relationships, we applied stepwise-adjusted weighted logistic regression models in the general population (Table 3). Consistent with the findings in Table 2, short durations of WCS (Group C) were associated with reduced severe IR risk under HOMA-IR (OR = 0.63, 95% CI: 0.41–0.97, $P = 0.037$), whereas long durations of WCS (Group E) significantly increased severe IR risk under METS-IR (OR = 1.88, 95% CI: 1.13–3.14, $P = 0.018$).

RCS analysis investigating nonlinear associations between the time of WCS and severe IR risk

As shown in Fig. 2, a U-shaped association between WCS time and the risk of severe IR was observed in both analyses after adjusting for demographics characteristics (age, sex, race, education level, marital status, and FPIR), lifestyle factors (alcohol consumption, smoke, energy intake, and physical activity), comorbities (BMI ≥ 30 kg/m^2 and hypertension), and sleep-related factors (average sleep time, social jetlag, snore, stop breathing, trouble sleeping and overly sleepy during day) (P for nonlinearity < 0.01; P for overall < 0.01). For HOMA-IR (Fig. 2A), at around 0.7 h of WCS, the severe IR risk reached its minimum value (OR = 0.82, 95% CI: 0.69–0.99, $P = 0.036$), and then gradually increased, although there was no statistical significance at approximately four hours (OR = 1.28, 95% CI: 0.96–1.72, $P = 0.091$). A similar pattern was observed for METS-IR (Fig. 2B), where the risk of severe IR was lowest near one hour of WCS, and significantly increased with prolonged WCS (OR = 1.72, 95% CI: 1.22–2.44, $P = 0.004$).

Subgroup and sensitivity analyses of associations between patterns of WCS and severe IR risk

As shown in Tables 4 and 5, the association between patterns of WCS and severe IR risk, as defined by HOMA-IR and METS-IR, was examined through various sociodemographic subpopulations. Generally, the associations between WCS patterns and severe IR risk were consistent among different subgroups. However, for severe IR

Table 1 Baseline characteristics of participants stratified by the patterns of WCS

Characteristics[b]	Patterns of WCS[a]						*P*-value[c]
	Total (*n* = 1,903)	Group A (*n* = 714)	Group B (*n* = 284)	Group C (*n* = 399)	Group D (*n* = 120)	Group E (*n* = 386)	
Age, *n* (weighted %)							< 0.001
< 60	1,344 (75.2%)	382 (58.4%)	221 (82.7%)	303 (80.5%)	101 (81.1%)	337 (92.0%)	
≥ 60	559 (24.8%)	332 (41.6%)	63 (17.3%)	96 (19.5%)	19 (18.9%)	49 (8.0%)	
Sex, *n* (weighted %)							0.360
Female	990 (51.3%)	367 (52.0%)	145 (49.7%)	216 (53.3%)	73 (60.9%)	189 (45.4%)	
Male	913 (48.7%)	347 (48.0%)	139 (50.3%)	183 (46.7%)	47 (39.1%)	197 (54.6%)	
Race, *n* (weighted %)							0.004
Non-Hispanic Black	489 (11.1%)	162 (9.8%)	98 (18.2%)	99 (9.8%)	20 (5.9%)	110 (12.3%)	
Non-Hispanic White	693 (65.5%)	323 (71.0%)	91 (58.9%)	141 (67.3%)	50 (70.0%)	88 (56.3%)	
Mexican Americans	246 (8.6%)	71 (6.5%)	36 (9.9%)	43 (6.2%)	21 (11.1%)	75 (13.5%)	
Other races	475 (14.8%)	158 (12.8%)	59 (13.0%)	116 (16.7%)	29 (13.0%)	113 (17.9%)	
Education level, *n* (weighted %)							< 0.001
< High school	259 (7.8%)	90 (7.1%)	46 (11.0%)	36 (4.3%)	13 (7.5%)	74 (11.2%)	
High school	409 (23.8%)	165 (26.5%)	71 (28.8%)	69 (15.2%)	19 (14.5%)	85 (29.1%)	
> High school	1,235 (68.4%)	459 (66.4%)	167 (60.2%)	294 (80.5%)	88 (78.0%)	227 (59.7%)	
Marital status, *n* (weighted %)							0.064
Never married	361 (19.1%)	113 (14.8%)	65 (25.9%)	74 (20.2%)	17 (10.1%)	92 (24.4%)	
Married/living with partner	1,177 (64.2%)	425 (65.0%)	162 (60.8%)	260 (65.7%)	86 (70.6%)	244 (60.7%)	
Widowed/Divorced/Separated	365 (16.7%)	176 (20.2%)	57 (13.3%)	65 (14.0%)	17 (19.3%)	50 (15.0%)	
FPIR, *n* (weighted %)							0.001
≤ 1.3	451 (14.7%)	176 (18.6%)	86 (21.4%)	72 (7.8%)	23 (8.0%)	94 (14.2%)	
(1.3, 3.5]	737 (33.4%)	277 (30.9%)	118 (39.1%)	133 (27.8%)	45 (32.2%)	164 (41.5%)	
> 3.5	715 (51.8%)	261 (50.6%)	80 (39.6%)	194 (64.4%)	52 (59.8%)	128 (44.3%)	
BMI (kg/m^2), *n* (weighted %)							0.494
< 30	1,082 (59.2%)	421 (62.1%)	167 (62.0%)	219 (58.4%)	67 (51.4%)	208 (55.8%)	
≥ 30	821 (40.8%)	293 (37.9%)	117 (38.0%)	180 (41.6%)	53 (48.6%)	178 (44.2%)	
Alcohol consumption, *n* (weighted %)							0.007
Never	180 (7.2%)	72 (8.8%)	31 (6.7%)	37 (6.3%)	12 (10.7%)	28 (4.5%)	
Mild	876 (48.0%)	370 (54.9%)	110 (35.4%)	190 (52.1%)	50 (36.8%)	156 (43.2%)	
Moderate	412 (23.2%)	131 (19.1%)	67 (27.0%)	89 (23.0%)	36 (35.7%)	89 (23.8%)	
Heavy	435 (21.5%)	141 (17.2%)	76 (30.9%)	83 (18.6%)	22 (16.8%)	113 (28.5%)	
Smoke, *n* (weighted %)							0.405
Never	1,106 (59.1%)	380 (55.5%)	160 (60.6%)	239 (60.8%)	83 (69.3%)	244 (59.0%)	
Former	452 (26.7%)	208 (30.2%)	59 (20.9%)	86 (26.4%)	24 (23.0%)	75 (26.0%)	
Now	345 (14.2%)	126 (14.3%)	65 (18.4%)	74 (12.8%)	13 (7.7%)	67 (15.0%)	

Table 1 (continued)

Characteristics[b]	Patterns of WCS[a]						*P*-value[c]
	Total (*n* = 1,903)	Group A (*n* = 714)	Group B (*n* = 284)	Group C (*n* = 399)	Group D (*n* = 120)	Group E (*n* = 386)	
Hypertension, *n* (weighted %)	790 (34.5%)	358 (42.5%)	112 (30.1%)	148 (30.3%)	52 (44.4%)	120 (24.5%)	0.004
PA, *n* (weighted %)							0.101
Inactive	375 (16.5%)	153 (16.9%)	58 (17.4%)	76 (17.4%)	23 (21.5%)	65 (12.3%)	
Low levels of PA	260 (12.6%)	112 (15.7%)	25 (5.7%)	52 (11.7%)	16 (14.3%)	55 (11.9%)	
High levels of PA	1,268 (70.9%)	449 (67.4%)	201 (76.9%)	271 (70.9%)	81 (64.2%)	266 (75.8%)	
Energy intake (kcal/d)	2,006 (1,547, 2,540)	1,963 (1,504, 2,465)	2,120 (1,582, 2,715)	1,890 (1,476, 2,414)	1,935 (1,527, 2,543)	2,079 (1,633, 2,684)	0.004
Weekday sleep time (h/d)	7.50 (7.00, 8.50)	8.00 (7.00, 8.50)	8.50 (8.00, 9.00)	7.50 (7.00, 8.00)	7.50 (6.50, 8.00)	7.00 (6.00, 7.50)	< 0.001
Weekend sleep time (h/d)	8.00 (7.50, 9.00)	8.00 (7.00, 8.50)	7.00 (6.00, 8.00)	8.00 (8.00, 9.00)	9.00 (8.00, 9.50)	9.50 (8.91, 10.00)	< 0.001
Average sleep time (h/d)	7.79 (7.00, 8.50)	8.00 (7.00, 8.50)	8.07 (7.36, 8.71)	7.64 (7.29, 8.29)	7.93 (6.93, 8.43)	7.53 (6.57, 8.21)	< 0.001
Social jetlag (h)	0.75 (0.00, 1.50)	0.00 (0.00, 0.00)	1.00 (0.50, 1.50)	1.16 (0.50, 1.50)	1.25 (0.75, 1.75)	1.75 (1.00, 2.50)	< 0.001
Social jetlag, *n* (weighted %)							< 0.001
< 1 h	1,019 (53.2%)	599 (83.0%)	132 (47.2%)	191 (47.4%)	51 (45.7%)	46 (12.9%)	
[1 h, 2 h)	474 (28.3%)	71 (12.9%)	100 (34.2%)	125 (35.7%)	48 (38.1%)	130 (39.7%)	
≥ 2 h	410 (18.5%)	44 (4.1%)	52 (18.6%)	83 (16.9%)	21 (16.3%)	210 (47.4%)	
Snore, *n* (weighted %)							0.487
Never	444 (25.5%)	155 (23.8%)	77 (27.8%)	97 (30.8%)	28 (16.2%)	87 (23.9%)	
Rarely	544 (31.3%)	202 (30.8%)	83 (32.3%)	105 (27.1%)	44 (37.1%)	110 (34.4%)	
Occasionally	375 (17.6%)	144 (17.2%)	52 (15.1%)	90 (20.1%)	23 (20.7%)	66 (15.8%)	
Frequently	540 (25.6%)	213 (28.2%)	72 (24.7%)	107 (22.0%)	25 (26.0%)	123 (25.9%)	
Stop breathing, *n* (weighted %)							0.510
Never	1,381 (75.3%)	495 (72.1%)	213 (78.6%)	296 (76.7%)	92 (77.3%)	285 (76.6%)	
Rarely	267 (13.6%)	111 (15.2%)	37 (12.0%)	52 (12.9%)	15 (9.3%)	52 (14.1%)	
Occasionally	129 (6.3%)	61 (7.6%)	16 (5.8%)	29 (6.7%)	6 (4.3%)	17 (4.3%)	
Frequently	126 (4.8%)	47 (5.0%)	18 (3.6%)	22 (3.7%)	7 (9.0%)	32 (5.0%)	
Trouble sleeping, *n* (weighted %)	558 (32.5%)	250 (37.0%)	79 (27.4%)	110 (31.9%)	31 (30.2%)	88 (29.0%)	0.264
Overly sleepy during day, *n* (weighted %)							0.191
Never	270 (12.8%)	109 (14.7%)	38 (10.7%)	58 (11.7%)	20 (18.6%)	45 (10.0%)	
Rarely	449 (22.7%)	153 (22.0%)	69 (28.6%)	106 (23.1%)	33 (20.3%)	88 (20.4%)	
Sometimes	695 (37.4%)	269 (38.4%)	113 (35.8%)	134 (39.3%)	40 (38.0%)	139 (34.3%)	
Often	337 (18.8%)	123 (18.7%)	47 (16.4%)	76 (19.9%)	22 (17.8%)	69 (19.6%)	
Almost always	152 (8.3%)	60 (6.2%)	17 (8.4%)	25 (6.0%)	5 (5.4%)	45 (15.7%)	
Glucose (mg/mL)	102 (95, 110)	103 (97, 113)	100 (93, 107)	101 (95, 109)	102 (95, 111)	102 (96, 109)	0.046
Glucose (mmol/L)	5.66 (5.27, 6.11)	5.72 (5.38, 6.27)	5.55 (5.16, 5.94)	5.61 (5.27, 6.05)	5.66 (5.27, 6.15)	5.66 (5.33, 6.05)	0.046
Insulin (μU/mL)	9 (6, 15)	9 (6, 15)	10 (6, 16)	8 (5, 13)	9 (6, 15)	10 (6, 18)	0.072
TG (mg/dL)	89 (61, 134)	94 (62, 140)	84 (61, 125)	91 (58, 132)	85 (67, 131)	87 (57, 138)	0.634
HDL-c (mg/dL)	52 (43, 63)	53 (44, 67)	51 (41, 60)	53 (45, 61)	52 (41, 65)	49 (42, 60)	0.159
HOMA-IR	2.28 (1.45, 4.20)	2.41 (1.45, 4.19)	2.31 (1.44, 4.66)	1.99 (1.26, 3.67)	2.58 (1.56, 4.24)	2.48 (1.53, 4.96)	0.074

Table 1 (continued)

Characteristics[b]	Patterns of WCS[a]						*P*-value[c]
	Total (*n* = 1,903)	Group A (*n* = 714)	Group B (*n* = 284)	Group C (*n* = 399)	Group D (*n* = 120)	Group E (*n* = 386)	
Severe IR defined by HOMA-IR, *n* (weighted %)	475 (21.8%)	173 (21.2%)	70 (25.9%)	86 (15.4%)	34 (22.7%)	112 (27.4%)	0.072
METS-IR	42 (34, 51)	41 (34, 49)	41 (33, 50)	42 (34, 49)	43 (33, 52)	43 (35, 53)	0.497
Severe IR defined by METS-IR, *n* (weighted %)	475 (23.6%)	165 (21.8%)	73 (23.9%)	91 (20.5%)	30 (25.4%)	116 (29.9%)	0.294

Abbreviations: *BMI* body mass index, *FPIR* ratio of family income to poverty, *HDL-c* high-density lipoprotein cholesterol, *HOMA* homeostatic model assessment, *IR* insulin resistance, *METS* metabolic syndrome, *PA* physical activity, *TG* triglyceride, *WCS* weekend catch-up sleep

[a] Group A: stable sleep pattern (WCS = 0), weighted *N* = 48,209,019; Group B: decrease in sleep duration (WCS < 0), weighted *N* = 17,664,346; Group C: Short catch-up sleep duration (0 < WCS ≤ 1 h), weighted *N* = 32,039,017; Group D: moderate catch-up sleep duration (1 < WCS < 2 h), weighted *N* = 9,825,264; Group E: long catch-up sleep duration (WCS ≥ 2 h), weighted *N* = 26,633,495; Total participants: weighted *N* = 134,371,140

[b] Continuous variables are presented as medians and interquartile ranges, and categorical variables are expressed as weighted percentages

[c] Chi-squared test with Rao & Scott's second-order correction. Kruskal–Wallis rank-sum test for complex survey samples. *P*-values < 0.05 are considered statistically significant

defined by METS-IR, significantly elevated IR risk was observed among never-married (OR = 5.05, 95% CI: 1.32–19.21, *P* = 0.02) and widowed/divorced/separated individuals (OR = 7.87, 95% CI: 1.58–39.27, *P* = 0.01) in Group E (WCS ≥ 2 h). Similar results were also observed in widowed/divorced/separated individuals of Group B (WCS < 0) (OR = 9.70, 95% CI: 2.07–45.57, *P* = 0.01).

In sensitivity analyses stratified by weekday sleep duration (Table 6), distinct associations between WCS patterns and severe IR risk were observed, varying by weekday sleep duration and IR indicators. Among participants sleeping less than six hours on weekdays, a short WCS duration (0 < WCS ≤ 1 h) was significantly associated with a lower risk of severe IR as defined by METS-IR (OR = 0.05, 95% CI: 0.00–0.71, *P* = 0.029), while no significant associations were observed for HOMA-IR (OR = 0.86, 95% CI: 0.27–2.75, *P* = 0.791). In the 6–7 h group, short WCS (0 < WCS ≤ 1 h) was linked to reduced severe IR risk as defined by HOMA-IR (OR = 0.20, 95% CI: 0.05–0.77, *P* = 0.021). For participants with weekday sleep durations of 7–8 h, there was no significant statistical difference between short WCS (0 < WCS ≤ 1 h) and severe IR risk reduction, whether assessed by HOMA-IR (OR = 0.46, 95% CI: 0.14–1.54, *P* = 0.198) or METS-IR (OR = 0.34, 95% CI: 0.10–1.11, *P* = 0.073). Similarly, no significant associations was observed for individuals sleeping more than 8 h on weekdays, regardless of the IR assessment method used.

Discussion

In this study, data from a survey on a representative population of the U.S. was used to investigate the associations between WCS patterns and the risk of severe IR among adults. Our findings reveal a U-shaped relationship between WCS duration and severe IR risk, with the lowest risk observed at approximately 0.7–1.0 h of WCS. Both insufficient and excessive WCS were associated with increased severe IR risk, highlighting the importance of optimal sleep patterns for metabolic health.

Research has shown that sleep is not only essential for physiological rest but also plays a critical role in maintaining metabolic homeostasis and health through complex and dynamic processes [19, 20, 37]. In the present study, we observed that participants with short durations of WCS (0 < WCS ≤ 1 h) exhibited a significantly lower risk of severe IR, as defined by HOMA-IR. Specifically, their risk of severe IR was reduced by 37% compared to those with a stable sleep pattern (WCS = 0). This association remained robust after adjusting for potential confounders, including demographic, lifestyle, and clinical factors. Our findings align with previous research, demonstrating a significant association between WCS and metabolic health, reflecting the nuanced role of WCS in influencing IR risk [19, 20]. The observed association between short WCS durations and reduced IR risk may be attributed to several interconnected mechanisms involving hormonal regulation, energy balance, and autonomic nervous system activity. First, insufficient sleep is known to decrease leptin (a satiety hormone) and increase ghrelin (an appetite-stimulating hormone), resulting in an elevated ghrelin-to-leptin ratio that promotes appetite and caloric intake [10, 47]. In our study, participants with short WCS durations (0 < WCS ≤ 1 h) took in the least energy among all groups, suggesting that short WCS may help maintain a more balanced ghrelin-to-leptin ratio, potentially mitigating appetite dysregulation and lowering IR risk.

Table 2 Associations between patterns of WCS and severe IR risk by univariate and multivariable weighted logistic regression analyses

Characteristics	Severe IR defined by HOMA-IR						Severe IR defined by METS-IR					
	Univariable analysis			Multivariable analysis			Univariable analysis			Multivariable analysis		
	OR	95% CI	*P*-value[a]	OR	95% CI	*P*-value[a]	OR	95% CI	*P*-value[a]	OR	95% CI	*P*-value[a]
Age, ≥ 60 yrs	1.09	0.73, 1.65	0.655	0.86	0.41, 1.81	0.685	0.79	0.52, 1.19	0.247	0.46	0.22, 0.96	0.039
Sex, male	1.23	0.79, 1.90	0.352	1.40	0.88, 2.24	0.152	0.87	0.60, 1.27	0.465	0.89	0.46, 1.73	0.717
Race												
Non-Hispanic Black	Ref			Ref			Ref			Ref		
Non-Hispanic White	0.74	0.46, 1.20	0.208	0.96	0.52, 1.77	0.891	0.73	0.51, 1.03	0.071	1.22	0.83, 1.81	0.300
Mexican Americans	2.16	1.26, 3.71	0.007	2.67	1.32, 5.41	0.008	1.48	1.03, 2.14	0.036	1.59	0.93, 2.72	0.088
Other races	1.00	0.70, 1.44	0.984	1.63	1.00, 2.65	0.049	0.73	0.51, 1.05	0.088	1.20	0.61, 2.37	0.589
Education level												
< High school	Ref			Ref			Ref			Ref		
High school	1.15	0.71, 1.87	0.549	1.66	0.90, 3.06	0.104	1.14	0.72, 1.79	0.566	1.40	0.62, 3.18	0.403
> High school	0.54	0.34, 0.87	0.013	0.90	0.47, 1.73	0.735	0.75	0.49, 1.15	0.182	1.00	0.45, 2.25	0.995
Marital status												
Never married	Ref			Ref			Ref			Ref		
Married/living with partner	0.93	0.66, 1.33	0.691	0.66	0.40, 1.08	0.092	1.06	0.56, 1.99	0.851	0.70	0.30, 1.64	0.400
Widowed/Divorced/Separated	0.59	0.39, 0.88	0.013	0.36	0.19, 0.70	0.004	0.61	0.34, 1.09	0.091	0.29	0.13, 0.65	0.004
FPIR												
≤ 1.3	Ref			Ref			Ref			Ref		
(1.3, 3.5]	0.96	0.66, 1.40	0.842	1.15	0.69, 1.92	0.575	1.13	0.78, 1.65	0.491	1.78	1.02, 3.12	0.044
> 3.5	0.59	0.38, 0.91	0.020	0.98	0.55, 1.75	0.941	0.61	0.39, 0.97	0.036	1.01	0.54, 1.90	0.968
BMI, ≥ 30 kg/m^2	8.43	6.02, 11.81	< 0.001	7.61	5.49, 10.54	< 0.001	165.40	78.51, 348.45	< 0.001	185.45	84.08, 409.02	< 0.001
Alcohol consumption												
Never	Ref			Ref			Ref			Ref		
Mild	1.44	0.92, 2.25	0.108	1.78	0.87, 3.67	0.111	1.21	0.71, 2.06	0.473	1.56	0.78, 3.14	0.198
Moderate	1.09	0.62, 1.94	0.755	1.18	0.51, 2.70	0.687	1.04	0.65, 1.67	0.864	0.87	0.45, 1.69	0.681
Heavy	1.57	0.90, 2.72	0.106	1.61	0.72, 3.62	0.233	1.26	0.89, 1.78	0.176	1.07	0.48, 2.37	0.870
Smoke												
Never	Ref			Ref			Ref			Ref		
Former	1.25	0.89, 1.76	0.194	1.19	0.74, 1.94	0.457	1.02	0.71, 1.47	0.909	1.13	0.71, 1.79	0.587
Now	0.84	0.58, 1.21	0.325	0.79	0.51, 1.20	0.255	0.88	0.64, 1.22	0.426	1.47	0.81, 2.65	0.191
Hypertension	2.83	1.89, 4.23	< 0.001	2.66	1.86, 3.81	< 0.001	2.29	1.84, 2.84	< 0.001	2.13	1.22, 3.71	0.010
PA												
Inactive	Ref			Ref			Ref			Ref		
Low levels of PA	1.04	0.55, 1.94	0.908	1.47	0.76, 2.84	0.240	0.97	0.56, 1.70	0.923	1.19	0.50, 2.86	0.686

Table 2 (continued)

Characteristics	Severe IR defined by HOMA-IR						Severe IR defined by METS-IR					
	Univariable analysis			Multivariable analysis			Univariable analysis			Multivariable analysis		
	OR	95% CI	*P*-value[a]	OR	95% CI	*P*-value[a]	OR	95% CI	*P*-value[a]	OR	95% CI	*P*-value[a]
High levels of PA	0.69	0.49, 0.97	0.035	0.85	0.59, 1.22	0.368	0.60	0.45, 0.81	0.002	0.66	0.41, 1.07	0.089
Energy intake (kcal/d)	1.01	0.81, 1.26	0.922	0.93	0.75, 1.17	0.525	1.01	0.90, 1.14	0.813	1.06	0.83, 1.34	0.647
Average sleep time (h/d)	0.99	0.77, 1.27	0.932	1.14	0.93, 1.38	0.194	1.05	0.84, 1.30	0.682	1.20	0.98, 1.48	0.072
Social jetlag (h)	1.02	0.86, 1.22	0.792	0.91	0.72, 1.15	0.416	1.06	0.92, 1.21	0.428	0.88	0.66, 1.16	0.346
Snore												
Never	Ref			Ref			Ref			Ref		
Rarely	1.42	0.91, 2.21	0.118	1.14	0.56, 2.33	0.709	1.30	0.90, 1.87	0.159	0.93	0.51, 1.68	0.793
Occasionally	1.62	0.90, 2.92	0.101	1.07	0.50, 2.29	0.861	1.80	1.14, 2.84	0.014	0.87	0.46, 1.64	0.645
Frequently	3.69	2.49, 5.47	< 0.001	1.87	1.11, 3.16	0.021	3.44	2.32, 5.12	< 0.001	1.05	0.62, 1.78	0.841
Stop breathing												
Never	Ref			Ref			Ref			Ref		
Rarely	1.29	0.68, 2.43	0.414	0.69	0.36, 1.33	0.256	1.36	0.79, 2.34	0.255	0.94	0.49, 1.79	0.842
Occasionally	2.11	1.02, 4.39	0.046	1.24	0.49, 3.15	0.632	2.73	1.57, 4.74	0.001	2.52	1.38, 4.62	0.004
Frequently	2.88	1.47, 5.67	0.004	0.91	0.40, 2.06	0.815	5.05	2.85, 8.96	< 0.001	3.66	1.30, 10.30	0.016
Trouble sleeping	1.53	1.15, 2.05	0.006	0.98	0.71, 1.36	0.905	1.86	1.28, 2.72	0.002	1.15	0.68, 1.94	0.589
Overly sleepy during day												
Never	Ref			Ref			Ref			Ref		
Rarely	1.94	0.99, 3.83	0.055	1.96	0.90, 4.27	0.086	1.83	1.01, 3.33	0.048	2.09	1.00, 4.38	0.050
Sometimes	1.95	1.03, 3.68	0.041	1.96	0.94, 4.08	0.070	1.57	0.93, 2.65	0.086	1.26	0.57, 2.82	0.551
Often	3.17	1.60, 6.29	0.002	2.61	1.29, 5.28	0.009	3.13	1.70, 5.75	< 0.001	1.77	0.84, 3.76	0.129
Almost always	3.42	1.26, 9.28	0.018	3.99	1.25, 12.81	0.022	2.08	0.92, 4.73	0.077	1.43	0.35, 5.86	0.602
Patterns of WCS												
Group A (WCS = 0)	Ref			Ref			Ref			Ref		
Group B (WCS < 0)	1.30	0.83, 2.03	0.242	1.54	0.93, 2.57	0.093	1.13	0.68, 1.89	0.630	1.34	0.71, 2.52	0.355
Group C (0 < WCS ≤ 1 h)	0.68	0.45, 1.02	0.059	0.63	0.41, 0.97	0.037	0.93	0.55, 1.57	0.775	0.74	0.41, 1.35	0.316
Group D (1 < WCS < 2 h)	1.09	0.51, 2.33	0.820	1.08	0.45, 2.59	0.859	1.22	0.59, 2.52	0.568	0.88	0.34, 2.25	0.784
Group E (WCS ≥ 2 h)	1.40	0.92, 2.12	0.110	1.49	0.79, 2.79	0.207	1.54	1.00, 2.37	0.052	1.88	1.13, 3.14	0.018

Abbreviations: *BMI* body mass index, *CI* confidence interval, *FPIR* ratio of family income to poverty, *HOMA* homeostatic model assessment, *IR* insulin resistance, *METS* metabolic syndrome, *OR* odds ratio, *PA* physical activity, *WCS* weekend catch-up sleep

[a] *P*-values < 0.05 are considered statistically significant

Table 3 Associations between the patterns of WCS and severe IR risk

Characteristics	Unadjusted Model		Model 1[a]		Model 2[b]		Model 3[c]		Model 4[d]	
	OR (95%CI)	*P*-value[e]	OR (95%CI)	*P*-value[e]	OR (95%CI)	*P*-value[e]	OR (95%CI)	*P*-value[e]	OR (95%CI)	*P*-value[e]
Patterns of WCS (Severe IR defined by HOMA-IR)										
Group A (WCS = 0)	Ref		Ref		Ref		Ref		Ref	
Group B (WCS < 0)	1.30 (0.83, 2.02)	0.240	1.23 (0.78, 1.92)	0.357	1.31 (0.85, 2.03)	0.208	1.48 (0.91, 2.41)	0.107	1.54 (0.93, 2.57)	0.093
Group C (0 < WCS ≤ 1 h)	0.68 (0.45, 1.01)	0.057	0.76 (0.50, 1.17)	0.204	0.77 (0.50, 1.18)	0.214	0.63 (0.42, 0.95)	0.028	0.63 (0.41, 0.97)	0.037
Group D (1 < WCS < 2 h)	1.09 (0.51, 2.31)	0.819	1.24 (0.54, 2.88)	0.598	1.31 (0.58, 2.93)	0.501	1.00 (0.42, 2.40)	0.998	1.08 (0.45, 2.59)	0.859
Group E (WCS ≥ 2 h)	1.40 (0.92, 2.11)	0.108	1.33 (0.83, 2.14)	0.227	1.31 (0.81, 2.14)	0.259	1.31 (0.75, 2.28)	0.335	1.49 (0.79, 2.79)	0.207
Patterns of WCS (Severe IR defined by METS-IR)										
Group A (WCS = 0)	Ref		Ref		Ref		Ref		Ref	
Group B (WCS < 0)	1.13 (0.68, 1.88)	0.629	0.99 (0.57, 1.70)	0.955	1.04 (0.60, 1.82)	0.881	1.24 (0.60, 2.54)	0.550	1.34 (0.71, 2.52)	0.355
Group C (0 < WCS ≤ 1 h)	0.93 (0.55, 1.57)	0.775	0.96 (0.58, 1.61)	0.887	0.96 (0.58, 1.61)	0.875	0.70 (0.42, 1.16)	0.156	0.74 (0.41, 1.35)	0.316
Group D (1 < WCS < 2 h)	1.22 (0.60, 2.50)	0.567	1.23 (0.56, 2.67)	0.595	1.25 (0.57, 2.72)	0.560	0.87 (0.33, 2.30)	0.768	0.88 (0.34, 2.25)	0.784
Group E (WCS ≥ 2 h)	1.54 (1.00, 2.36)	0.050	1.40 (0.90, 2.17)	0.128	1.39 (0.89, 2.18)	0.136	1.35 (0.87, 2.11)	0.175	1.88 (1.13, 3.14)	0.018

Abbreviations: *BMI* body mass index, *CI* confidence interval, *FPIR* ratio of family income to poverty, *HOMA* homeostatic model assessment, *IR* insulin resistance, *METS* metabolic syndrome, *OR* odds ratio, *WCS* weekend catch-up sleep

[a] Model 1 was adjusted for demographic characteristics (age, sex, race, education level, marital status, and FPIR)

[b] Model 2 was additionally adjusted for lifestyle factors (alcohol consumption, smoke, energy intake, and physical activity) based on Model 1

[c] Model 3 was additionally adjusted for comorbities (BMI ≥ 30 kg/m^2 and hypertension) based on Model 2

[d] Model 4 was additionally adjusted for sleep-related factors (average sleep time, social jetlag, snore, stop breathing, trouble sleeping and overly sleepy during day) based on Model 3

[e] *P*-values < 0.05 are considered statistically significant

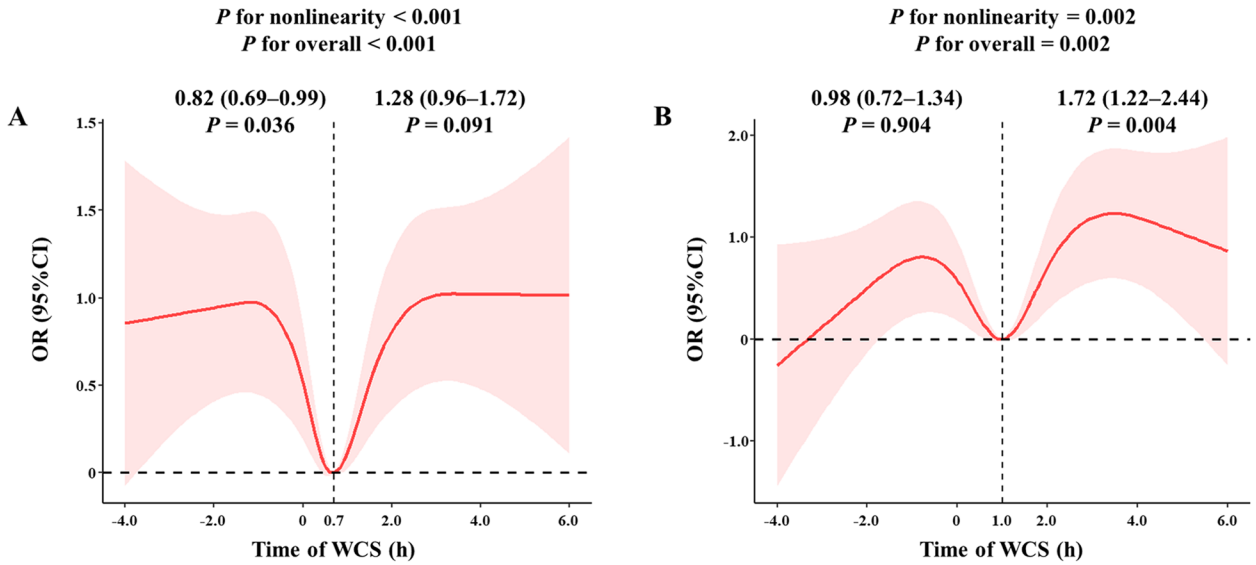

Fig. 2 Weighted RCS of the association between the time of WCS and the risk of severe IR defined as severe IR by HOMA-IR (**A**) and METS-IR (**B**). The association was adjusted for age, sex, race, education level, marital status, FPIR, BMI, alcohol consumption, smoke, hypertension, energy intake, physical activity, average sleep time, social jetlag, snore, stop breathing, trouble sleeping, and overly sleepy during day. Abbreviations: BMI, body mass index; CI, confidence interval; FPIR, ratio of family income to poverty; HOMA, homeostatic model assessment; IR, insulin resistance; METS, metabolic syndrome; OR, odds ratio; RCS, restricted cubic spline; WCS, weekend catch-up sleep

Second, insufficient sleep is associated with heightened sympathetic nervous system (SNS) activity and suppressed parasympathetic nervous system (PNS) function, impairing pancreatic beta-cell function, reducing insulin secretion, and promoting visceral fat accumulation, which collectively exacerbate IR risk [48, 49]. Short WCS durations may help restore the balance between SNS and PNS activity, thereby improving insulin sensitivity. Finally, chronic sleep deprivation is linked to persistently elevated cortisol levels, which antagonize insulin action, reduce insulin sensitivity, and increase IR risk [50, 51]. However, short WCS durations may mitigate these cortisol dysregulation effects typically observed with insufficient sleep, preserving normal insulin signaling pathways and metabolic stability.

We also found that participants with long WCS (WCS ≥ 2 h) had a higher risk of severe IR when defined by METS-IR. Like the findings of our study, other previous research has also indicated that the health effects of WCS vary depending on its duration [31, 52]. A cross-sectional study suggested that compensating for excessive weekday sleep deprivation with WCS of 2 h or more may be associated with a negative impact on blood lipid regulation [53]. Based on these findings, it can be expected that WCS of 0–1 h during weekends to achieve the optimal average sleep duration may be beneficial for metabolic homeostasis. However, trying to compensate for excessive weekday sleep deprivation with WCS of 2 h or more to achieve the optimal average sleep duration may not have metabolic benefits. The specific mechanism by which excessive WCS time is associated with IR may be related to sleep fragmentation, fatigue, and immune function [54–56], leading to increased levels of inflammation in the body and affecting metabolic status [57–59]. In addition, prolonged WCS exceeding two hours may also reduce the time of physical activities and have a higher tendency to unhealthy behaviors [60], which may affect the overall health of the body, lead to IR, further develop into obesity, hypertension, and diabetes, and promote the occurrence and development of MetS [61, 62]. Our study provides a theoretical basis for considering individual sleep patterns in the management of metabolic health.

Our results show that the definition of severe IR critically affects the observed associations between WCS and severe IR risk, underscoring the importance of study-specific IR metrics. HOMA-IR, which is derived from fasting insulin and glucose concentrations, primarily reflects hepatic insulin sensitivity [26]. Notably, short WCS exhibited a protective effect on severe IR defined by HOMA-IR, possibly through partial restoration of hepatic metabolic homeostasis via brief sleep recovery. In contrast, METS-IR encompasses a broader spectrum of systemic metabolic dysfunction, including dyslipidemia [29]. Our findings indicated that long WCS heightened the risk of severe IR as defined by METS-IR, particularly after adjusting for sleep-related factors. This suggests that extended irregular sleep patterns may exacerbate circadian misalignment or lead to fragmented

Table 4 Subgroup analysis of associations between patterns of WCS and severe IR risk defined by HOMA-IR

Characteristics	Group A (WCS = 0)	Group B (WCS < 0)		Group C (0 < WCS ≤ 1 h)		Group D (1 < WCS < 2 h)		Group E (WCS ≥ 2 h)		*P* for interaction[a]
		OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]	
Age										0.51
< 60	Ref	1.86 (1.01, 3.42)	0.05	0.79 (0.43, 1.46)	0.44	1.02 (0.38, 2.77)	0.96	1.64 (0.74, 3.61)	0.21	
≥ 60		1.95 (0.60, 6.30)	0.25	0.64 (0.20, 1.98)	0.42	2.55 (0.75, 8.66)	0.13	3.24 (0.75, 13.94)	0.11	
Sex										0.56
Female	Ref	1.76 (1.05, 2.97)	0.03	0.57 (0.29, 1.13)	0.10	1.01 (0.36, 2.82)	0.98	1.53 (0.91, 2.59)	0.11	
Male		1.43 (0.62, 3.32)	0.38	0.87 (0.50, 1.50)	0.60	0.85 (0.20, 3.61)	0.82	2.23 (0.82, 6.09)	0.11	
Race										0.25
Non-Hispanic Black	Ref	0.80 (0.30, 2.12)	0.63	0.96 (0.51, 1.81)	0.89	0.55 (0.14, 2.16)	0.38	1.49 (0.63, 3.54)	0.35	
Non-Hispanic White		2.06 (0.87, 4.89)	0.10	0.37 (0.15, 0.87)	0.02	1.02 (0.34, 3.06)	0.97	1.68 (0.38, 7.48)	0.48	
Mexican Americans		5.46 (1.22, 24.38)	0.03	3.32 (0.90, 12.24)	0.07	0.52 (0.07, 3.56)	0.48	2.39 (0.62, 9.20)	0.19	
Other races		0.93 (0.37, 2.38)	0.88	0.93 (0.36, 2.38)	0.87	2.72 (1.16, 6.36)	0.02	1.59 (0.64, 3.91)	0.30	
Education level										0.32
< high school	Ref	0.90 (0.17, 4.80)	0.89	1.12 (0.17, 7.28)	0.90	5.43 (1.58, 18.69)	0.01	3.27 (1.03, 10.39)	0.05	
high school		2.51 (0.88, 7.18)	0.08	1.37 (0.35, 5.34)	0.64	1.39 (0.36, 5.47)	0.62	2.09 (0.42, 10.44)	0.35	
> high school		1.97 (1.01, 3.85)	0.05	0.58 (0.34, 0.99)	0.05	1.06 (0.40, 2.80)	0.91	1.42 (0.61, 3.32)	0.40	
Marital status										0.34
Never married	Ref	1.70 (0.34, 8.52)	0.51	0.79 (0.21, 2.99)	0.72	7.52 (0.80, 70.74)	0.08	6.01 (1.62, 22.28)	0.01	
Married/living with partner		1.97 (1.07, 3.62)	0.03	0.69 (0.41, 1.16)	0.15	0.94 (0.32, 2.77)	0.91	1.57 (0.68, 3.62)	0.28	
Widowed/Divorced/Separated		0.52 (0.09, 2.96)	0.45	0.48 (0.13, 1.78)	0.26	0.66 (0.16, 2.73)	0.55	0.58 (0.16, 2.12)	0.39	

The analysis was adjusted for FPIR, alcohol assumption, smoke, energy intake, physical activity, BMI, hypertension, average sleep time, social jetlag, snore, stop breathing, trouble sleeping, overly sleepy during day

Abbreviations: *BMI* body mass index, *CI* confidence interval, *FPIR* ratio of family income to poverty, *HOMA* homeostatic model assessment, *IR* insulin resistance, *OR* odds ratio, *WCS* weekend catch-up sleep

[a] *P*-values < 0.05 are considered statistically significant

Table 5 Subgroup analysis of associations between patterns of WCS and severe IR risk defined by METS-IR

Characteristics	Group A (WCS = 0)	Group B (WCS < 0)		Group C (0 < WCS ≤ 1 h)		Group D (1 < WCS < 2 h)		Group E (WCS ≥ 2 h)		*P* for interaction[a]
		OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]	
Age										0.64
< 60	Ref	1.87 (0.72, 4.82)	0.19	0.82 (0.39, 1.70)	0.58	0.83 (0.24, 2.88)	0.76	1.97 (1.01, 3.82)	0.05	
≥ 60		0.56 (0.20, 1.51)	0.24	0.66 (0.17, 2.61)	0.54	0.74 (0.10, 5.66)	0.76	1.02 (0.18, 5.78)	0.98	
Sex										0.84
Female	Ref	1.77 (0.66, 4.75)	0.24	0.74 (0.34, 1.63)	0.44	0.76 (0.24, 2.42)	0.64	2.49 (1.15, 5.42)	0.02	
Male		1.22 (0.47, 3.16)	0.67	0.92 (0.34, 2.47)	0.86	0.74 (0.19, 2.88)	0.65	1.96 (0.63, 6.10)	0.24	
Race										0.54
Non-Hispanic Black	Ref	1.83 (0.76, 4.37)	0.17	0.57 (0.19, 1.70)	0.30	0.99 (0.18, 5.48)	0.99	1.89 (0.63, 5.69)	0.24	
Non-Hispanic White		1.06 (0.30, 3.70)	0.92	0.90 (0.40, 2.03)	0.80	1.17 (0.33, 4.07)	0.80	3.12 (1.09, 8.87)	0.03	
Mexican Americans		19.22 (2.58, 143.46)	0.01	1.55 (0.33, 7.42)	0.56	0.25 (0.03, 1.74)	0.15	0.74 (0.14, 3.85)	0.71	
Other races		0.79 (0.07, 8.43)	0.84	0.59 (0.19, 1.86)	0.35	1.70 (0.36, 8.04)	0.49	1.84 (0.43, 7.84)	0.39	
Education level										0.44
< high school	Ref	0.76 (0.13, 4.63)	0.76	1.18 (0.13, 10.94)	0.88	1.43 (0.21, 9.58)	0.70	0.24 (0.04, 1.38)	0.11	
high school		3.46 (0.70, 17.22)	0.12	0.95 (0.23, 3.91)	0.94	0.31 (0.02, 3.96)	0.36	10.69 (1.85, 61.76)	0.01	
> high school		1.91 (0.80, 4.56)	0.14	0.86 (0.45, 1.62)	0.62	1.25 (0.32, 4.83)	0.74	2.03 (1.08, 3.83)	0.03	
Marital status										0.06
Never married	Ref	3.27 (0.74, 14.45)	0.11	0.33 (0.07, 1.53)	0.15	0.95 (0.05, 17.15)	0.97	5.05 (1.32, 19.21)	0.02	
Married/living with partner		0.92 (0.42, 2.04)	0.84	0.78 (0.42, 1.44)	0.41	1.06 (0.34, 3.34)	0.92	1.47 (0.73, 2.95)	0.27	
Widowed/Divorced/Separated		9.70 (2.07, 45.57)	0.01	0.99 (0.20, 4.93)	0.99	0.18 (0.03, 1.28)	0.08	7.87 (1.58, 39.27)	0.01	

The analysis was adjusted for FPIR, alcohol assumption, smoke, energy intake, physical activity, BMI, hypertension, average sleep time, social jetlag, snore, stop breathing, trouble sleeping, overly sleepy during day

Abbreviations: *BMI* body mass index, *CI* confidence interval, *FPIR* ratio of family income to poverty, *METS* metabolic syndrome, *IR* insulin resistance, *OR* odds ratio, *WCS* weekend catch-up sleep

[a] *P*-values < 0.05 are considered statistically significant

Table 6 Sensitivity analyses of associations between the patterns of WCS and severe IR stratified by different weekday sleep time

Patterns of WCS	HOMA-IR		METS-IR	
	OR (95%CI)	*P*-value[a]	OR (95%CI)	*P*-value[a]
Weekday sleep time ≤ 6 h				
Group A (WCS = 0)	Ref		Ref	
Group B (WCS < 0)	1.08 (0.13, 9.06)	0.941	11.12 (0.45, 276.28)	0.135
Group C (0 < WCS ≤ 1 h)	0.86 (0.27, 2.75)	0.791	0.05 (0.00, 0.71)	0.029
Group D (1 < WCS < 2 h)	3.60 (0.42, 30.81)	0.230	2.53 (0.15, 41.71)	0.501
Group E (WCS ≥ 2 h)	1.38 (0.45, 4.21)	0.563	3.47 (0.65, 18.51)	0.138
6 < Weekday sleep time ≤ 7 h				
Group A (WCS = 0)	Ref		Ref	
Group B (WCS < 0)	1.48 (0.30, 7.17)	0.616	2.32 (0.55, 9.85)	0.242
Group C (0 < WCS ≤ 1 h)	0.20 (0.05, 0.77)	0.021	1.09 (0.18, 6.42)	0.924
Group D (1 < WCS < 2 h)	0.32 (0.07, 1.57)	0.153	1.45 (0.27, 7.82)	0.653
Group E (WCS ≥ 2 h)	0.24 (0.03, 1.86)	0.164	11.14 (0.67, 186.17)	0.090
7 < Weekday sleep time ≤ 8 h				
Group A (WCS = 0)	Ref		Ref	
Group B (WCS < 0)	0.94 (0.34, 2.63)	0.901	0.36 (0.09, 1.43)	0.140
Group C (0 < WCS ≤ 1 h)	0.46 (0.14, 1.54)	0.198	0.34 (0.10, 1.11)	0.073
Group D (1 < WCS < 2 h)	1.77 (0.52, 6.00)	0.348	0.77 (0.13, 4.47)	0.759
Group E (WCS ≥ 2 h)	2.29 (0.46, 11.50)	0.300	0.74 (0.12, 4.42)	0.729
Weekday sleep time > 8 h				
Group A (WCS = 0)	Ref		Ref	
Group B (WCS < 0)	1.78 (0.70, 4.54)	0.215	1.78 (0.74, 4.29)	0.190
Group C (0 < WCS ≤ 1 h)	0.85 (0.47, 1.52)	0.563	0.87 (0.28, 2.67)	0.796
Group D (1 < WCS < 2 h)	0.57 (0.05, 6.10)	0.632	0.10 (0.01, 0.90)	0.041
Group E (WCS ≥ 2 h)	1.41 (0.28, 7.06)	0.663	2.36 (0.28, 20.09)	0.418

The analysis was adjusted for demographics characteristics (age, sex, race, education level, marital status, and FPIR), lifestyle factors (alcohol consumption, smoke, energy intake, and physical activity), comorbities (BMI ≥ 30 kg/m^2 and hypertension), and sleep-related factors (average sleep time, social jetlag, snore, stop breathing, trouble sleeping and overly sleepy during day)

Abbreviations: *BMI* body mass index, *CI* confidence interval, *FPIR* ratio of family income to poverty, *HOMA* homeostatic model assessment, *IR* insulin resistance, *METS* metabolic syndrome, *OR* odds ratio, *WCS* weekend catch-up sleep

[a] *P*-values < 0.05 are considered statistically significant

sleep, ultimately promoting visceral adiposity and dysregulated lipid metabolism—key components of metabolic syndrome. This pattern aligns with evidence linking social jetlag to an increased risk of metabolic syndrome, especially in individuals with preexisting obesity [63]. By elucidating these mechanistic pathways, our work underscores the value of a dual-metric approach to IR, offering a more nuanced perspective on how weekend sleep patterns interact with specific metabolic phenotypes.

Although the overall testing of the interaction did not reach statistical significance, there was a significant association between WCS patterns and severe IR in specific subgroups. The elevated risk of severe IR among never-married and widowed/divorced/separated individuals with long WCS durations might reflect the impact of psychosocial stressors and social isolation on sleep patterns and metabolic regulation. Moreover, social isolation may reduce the regulation of sleep–wake cycles, resulting in irregular sleep patterns and a greater need for sleep compensation on weekends [64, 65]. This misalignment can adversely affect glucose metabolism and lipid profiles, contributing to an increased risk of severe IR [66].

We further analyzed our data by incorporating average weekday sleep duration to eliminate potential confounding effects on the risk of severe IR. Among chronic short sleepers (sleeping less than six hours on weekdays), we found that a WCS duration of approximately one hour was significantly associated with a lower risk of severe IR as defined by METS-IR. This suggests that even minimal catch-up sleep on weekends can partially mitigate the adverse metabolic effects of severe weekday sleep deprivation by reducing accumulated sleep debt and improving insulin sensitivity, consistent with the findings of Kim et al. [19]. Among participants with weekday sleep durations of 6–7 h, short WCS durations (0 < WCS ≤ 1 h) were associated with a reduced risk of severe IR.

According to HOMA-IR, the risk decreased by 80%. This indicates that slight extensions of sleep duration on weekends might enhance metabolic health by providing additional restorative sleep without causing significant circadian misalignment. In contrast, for participants sleeping more than seven hours on weekdays, WCS patterns showed limited associations with severe IR, possibly because these individuals are already obtaining sufficient sleep during weekdays, and additional weekend sleep does not confer extra metabolic benefits. Overall, these results highlight the importance of individualized sleep recommendations, considering both weekday sleep duration and the potential metabolic impacts of weekend sleep patterns.

A major strength of this study is that we used nationally representative data from NHANES, enabling us to generalize our findings to a broader population. Additionally, the study also controlled for a wide range of covariates, including demographic variables, lifestyle factors, and comorbidities, further increasing the statistical power of our findings. Despite the substantial strengths of this study, there are certain limitations in our study. In this study, sleep characteristics, physical activity, and dietary intake were estimated using self-reported survey data, which are subject to limitations such as recall bias, social desirability bias, and measurement inaccuracies [67]. These factors may lead to misclassification or inaccuracies in the recorded data, potentially influencing our findings. Furthermore, due to the cross-sectional design of NHANES, we are unable to assess the longitudinal association between the pattern or time of WCS and IR risk. In the future, we need to conduct objective measurements in more prospective studies to confirm these associations and explore the effects of WCS time or different patterns of WCS on metabolic health.

Conclusions

Our study found a U-shaped relationship between WCS duration and severe IR risk. Approximately 0.7–1.0 h of WCS was linked to the lowest IR risk. Short WCS durations ($0 < \text{WCS} \leq 1$ h) were associated with reduced severe IR risk, especially in individuals with weekday sleep durations of seven hours or less. Both insufficient and excessive WCS increased IR risk, emphasizing the importance of optimal sleep patterns for metabolic health. These findings highlight the need for personalized sleep recommendations to reduce metabolic risks.

Abbreviations

BMI	Body mass index
CI	Confidence interval
FINS	Fasting serum insulin
FPG	Fasting plasma glucose
FPIR	Ratio of family income to poverty
HDL-c	High-density lipoprotein cholesterol
HOMA	Homeostatic model assessment
IQR	Interquartile range
IR	Insulin resistance
MetS	Metabolic syndrome
METS	Metabolic score
NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio
PA	Physical activity
PNS	Parasympathetic nervous system
RCS	Restricted cubic spline
SD	Standard deviation
SNS	Sympathetic nervous system
TG	Triglyceride
WCS	Weekend catch-up sleep
WTSAF2YR	Fasting Subsample 2 Year Mobile Examination Center Weight

Acknowledgements

All authors thank the NHANES 2017-2020 participants for their invaluable contributions.

Authors' contributions

X.L. conceived the idea. A.C. participated in data collection and statistical analysis. X.L. drafted the manuscript. X.D. edited the paper and gave many valuable comments on the draft and polished it. All authors have read and approved the manuscript.

Funding

The study received no funding.

Data availability

The data were obtained from publicly available sources, and detailed information could be achieved at (https://www.cdc.gov/nchs/nhanes/index.htm).

Declarations

Ethics approval and consent to participate

Participants in NHANES provided written informed consent, and the study protocol was approved by the Research Ethics Review Board of the National Center for Health Statistics and the US Army Research Institute of Environmental Medicine Human Use Review Committee (Protocol Number: Protocol #2011–17, Protocol #2018–01) (https://www.cdc.gov/nchs/nhanes/about/erb.html). The present study is a secondary analysis of publicly available and de-identified data and was therefore exempt from further institutional review.

Consent for publication

All authors have reviewed and approved the final version of the manuscript.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Cardiology, The First Affiliated Hospital of Nanjing Medical University, No. 300 Guangzhou Road, Nanjing 210029, Jiangsu, China. [2]Department of Nursing, The First Affiliated Hospital of Nanjing Medical University, No. 300 Guangzhou Road, Nanjing 210029, Jiangsu, China. [3]Department of Anesthesiology, Nanjing Drum Tower Hospital, Medical School of Nanjing University, No. 321 Zhongshan Road, Nanjing 210008, Jiangsu, China.

Received: 24 September 2024 Accepted: 20 May 2025
Published online: 28 May 2025

References

1. Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, et al. Heart Disease and Stroke Statistics-2019 Update: A Report From the American Heart Association. Circulation. 2019;139(10):e56–528.
2. Alemany M. The Metabolic Syndrome, a Human Disease. Int J Mol Sci. 2024;25(4):2251.

3. Madan K, Paliwal S, Sharma S, Kesar S, Chauhan N, Madan M. Metabolic Syndrome: The Constellation of Co-morbidities, A Global Threat. Endocr Metab Immune Disord Drug Targets. 2023;23(12):1491–504.
4. Lee SH, Park SY, Choi CS. Insulin Resistance: From Mechanisms to Therapeutic Strategies. Diabetes Metab J. 2022;46(1):15–37.
5. Fahed G, Aoun L, Bou Zerdan M, Allam S, Bou Zerdan M, Bouferraa Y, et al. Metabolic Syndrome: Updates on Pathophysiology and Management in 2021. Int J Mol Sci. 2022;23(2):786.
6. Wang X, Ma H, Gupta S, Heianza Y, Fonseca V, Qi L. The Joint Secular Trends of Sleep Quality and Diabetes Among US Adults, 2005–2018. J Clin Endocrinol Metab. 2022;107(11):3152–61.
7. Di H, Guo Y, Daghlas I, Wang L, Liu G, Pan A, et al. Evaluation of Sleep Habits and Disturbances Among US Adults, 2017–2020. JAMA Netw Open. 2022;5(11):e2240788.
8. Xu J, Luo L, Gamaldo A, Verdery A, Hardy M, Buxton OM, et al. Trends in sleep duration in the U.S. from 2004 to 2018: A decomposition analysis. SSM Popul Health. 2024;25:101562.
9. Ford ES, Cunningham TJ, Croft JB. Trends in Self-Reported Sleep Duration among US Adults from 1985 to 2012. Sleep. 2015;38(5):829–32.
10. Chaput JP, McHill AW, Cox RC, Broussard JL, Dutil C, da Costa BGG, et al. The role of insufficient sleep and circadian misalignment in obesity. Nat Rev Endocrinol. 2023;19(2):82–97.
11. Xie J, Li Y, Zhang Y, Vgontzas AN, Basta M, Chen B, et al. Sleep duration and metabolic syndrome: An updated systematic review and meta-analysis. Sleep Med Rev. 2021;59:101451.
12. Jin Q, Yang N, Dai J, Zhao Y, Zhang X, Yin J, et al. Association of Sleep Duration With All-Cause and Cardiovascular Mortality: A Prospective Cohort Study. Front Public Health. 2022;10:880276.
13. Sondrup N, Termannsen AD, Eriksen JN, Hjorth MF, Færch K, Klingenberg L, et al. Effects of sleep manipulation on markers of insulin sensitivity: A systematic review and meta-analysis of randomized controlled trials. Sleep Med Rev. 2022;62:101594.
14. Zuraikat FM, Laferrère B, Cheng B, Scaccia SE, Cui Z, Aggarwal B, et al. Chronic Insufficient Sleep in Women Impairs Insulin Sensitivity Independent of Adiposity Changes: Results of a Randomized Trial. Diabetes Care. 2024;47(1):117–25.
15. Antza C, Kostopoulos G, Mostafa S, Nirantharakumar K, Tahrani A. The links between sleep duration, obesity and type 2 diabetes mellitus. J Endocrinol. 2021;252(2):125–41.
16. Hong SH, Lee DB, Yoon DW, Kim J. Melatonin Improves Glucose Homeostasis and Insulin Sensitivity by Mitigating Inflammation and Activating AMPK Signaling in a Mouse Model of Sleep Fragmentation. Cells. 2024;13(6):470.
17. Gombert M, Reisdorph N, Morton SJ, Wright KP Jr, Depner CM. Insufficient sleep and weekend recovery sleep: classification by a metabolomics-based machine learning ensemble. Sci Rep. 2023;13(1):21123.
18. Leger D, Richard JB, Collin O, Sauvet F, Faraut B. Napping and weekend catchup sleep do not fully compensate for high rates of sleep debt and short sleep at a population level (in a representative nationwide sample of 12,637 adults). Sleep Med. 2020;74:278–88.
19. Kim DJ, Mun SJ, Choi JS, Kim J, Lee GH, Kim HW, et al. Beneficial effects of weekend catch-up sleep on metabolic syndrome in chronic short sleepers. Sleep Med. 2020;76:26–32.
20. Lee K. Evaluation of weekend catch-up sleep and weekday sleep duration in relation to metabolic syndrome in Korean adults. Sleep Breath. 2023;27(6):2199–207.
21. Depner CM, Melanson EL, Eckel RH, Snell-Bergeon JK, Perreault L, Bergman BC, et al. Ad libitum Weekend Recovery Sleep Fails to Prevent Metabolic Dysregulation during a Repeating Pattern of Insufficient Sleep and Weekend Recovery Sleep. Curr Biol. 2019;29(6):957–967.e954.
22. Broussard JL, Ehrmann DA, Van Cauter E, Tasali E, Brady MJ. Impaired insulin signaling in human adipocytes after experimental sleep restriction: a randomized, crossover study. Ann Intern Med. 2012;157(8):549–57.
23. Kiecolt-Glaser JK, Newton TL. Marriage and health: his and hers. Psychol Bull. 2001;127(4):472–503.
24. Umberson D, Williams K, Powers DA, Liu H, Needham B. You make me sick: marital quality and health over the life course. J Health Soc Behav. 2006;47(1):1–16.
25. Bulanda JR, Brown JS, Yamashita T. Marital quality, marital dissolution, and mortality risk during the later life course. Soc Sci Med. 2016;165:119–27.
26. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412–9.
27. Mousa N, Abdel-Razik A, Sheta T, Shabana W, Zakaria S, Awad M, et al. Serum leptin and homeostasis model assessment-IR as novel predictors of early liver fibrosis in chronic hepatitis B virus infection. Br J Biomed Sci. 2018;75(4):192–6.
28. Duan M, Zhao X, Li S, Miao G, Bai L, Zhang Q, et al. Metabolic score for insulin resistance (METS-IR) predicts all-cause and cardiovascular mortality in the general population: evidence from NHANES 2001–2018. Cardiovasc Diabetol. 2024;23(1):243.
29. Bello-Chavolla OY, Almeda-Valdes P, Gomez-Velasco D, Viveros-Ruiz T, Cruz-Bautista I, Romo-Romo A, et al. METS-IR, a novel score to evaluate insulin sensitivity, is predictive of visceral adiposity and incident type 2 diabetes. Eur J Endocrinol. 2018;178(5):533–44.
30. Epstein EJ, Osman JL, Cohen HW, Rajpathak SN, Lewis O, Crandall JP. Use of the estimated glucose disposal rate as a measure of insulin resistance in an urban multiethnic population with type 1 diabetes. Diabetes Care. 2013;36(8):2280–5.
31. Han KM, Lee HJ, Kim L, Yoon HK. Association between weekend catch-up sleep and high-sensitivity C-reactive protein levels in adults: a population-based study. Sleep. 2020;43(8):zsaa010.
32. 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(1):98–103.
33. Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. Chronobiol Int. 2006;23(1–2):497–509.
34. Islam Z, Hu H, Akter S, Kuwahara K, Kochi T, Eguchi M, et al. Social jetlag is associated with an increased likelihood of having depressive symptoms among the Japanese working population: the Furukawa Nutrition and Health Study. Sleep. 2020;43(1):zsz204.
35. Jung EJ, Cho SS, Lee HE, Min J, Jang TW, Kang MY. Association between social jetlag and self-rated health: Evidence from Korean representative working population. Sleep Med. 2024;114:86–91.
36. Liu Y, Yin J, Li X, Yang J, Liu Y. Examining the connection between weekend catch-up sleep and depression: Insights from 2017 to 2020 NHANES information. J Affect Disord. 2024;358:61–9.
37. Son SM, Park EJ, Cho YH, Lee SY, Choi JI, Lee YI, et al. Association Between Weekend Catch-Up Sleep and Metabolic Syndrome with Sleep Restriction in Korean Adults: A Cross-Sectional Study Using KNHANES. Diabetes Metab Syndr Obes. 2020;13:1465–71.
38. Liu X, Liu X, Wang Y, Zeng B, Zhu B, Dai F. Association between depression and oxidative balance score: National Health and Nutrition Examination Survey (NHANES) 2005–2018. J Affect Disord. 2023;337:57–65.
39. Zhu S, Ji L, He Z, Zhang W, Tong Y, Luo J, et al. Association of smoking and osteoarthritis in US (NHANES 1999–2018). Sci Rep. 2023;13(1):3911.
40. Rattan P, Penrice DD, Ahn JC, Ferrer A, Patnaik M, Shah VH, et al. Inverse Association of Telomere Length With Liver Disease and Mortality in the US Population. Hepatol Commun. 2022;6(2):399–410.
41. Yang Y, Wang Y, Yang L. Association between physical activity and sedentary behavior and depression in US adults with cardiovascular disease: NHANES 2007–2016. J Affect Disord. 2024;367:342–9.
42. Wu D, Jia Y, Liu Y, Pan X, Li P, Shang M. Dose response of leisure time physical activity and biological aging in type 2 diabetes: a cross sectional study. Sci Rep. 2024;14(1):26253.
43. Liao J, Hu M, Imm K, Holmes CJ, Zhu J, Cao C, et al. Association of daily sitting time and leisure-time physical activity with body fat among U.S. adults. J Sport Health Sci. 2024;13(2):195–203.
44. Bull FC, Al-Ansari SS, Biddle S, Borodulin K, Buman MP, Cardon G, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. Br J Sports Med. 2020;54(24):1451–62.
45. Hua Y, Liu HL, Sun JY, Kong XQ, Sun W, Xiong YQ. Association Between Serum Calcium and the Prevalence of Hypertension Among US Adults. Front Cardiovasc Med. 2021;8:719165.
46. Liu B, Du Y, Wu Y, Snetselaar LG, Wallace RB, Bao W. Trends in obesity and adiposity measures by race or ethnicity among adults in the United States 2011–18: population based study. BMJ. 2021;372:n365.
47. Zhu B, Shi C, Park CG, Zhao X, Reutrakul S. Effects of sleep restriction on metabolism-related parameters in healthy adults: A comprehensive

review and meta-analysis of randomized controlled trials. Sleep Med Rev. 2019;45:18–30.
48. Cao W, Shi M, Wu L, Li J, Yang Z, Liu Y, et al. Adipocytes initiate an adipose-cerebral-peripheral sympathetic reflex to induce insulin resistance during high-fat feeding. Clin Sci (Lond). 2019;133(17):1883–99.
49. Tobaldini E, Costantino G, Solbiati M, Cogliati C, Kara T, Nobili L, et al. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. Neurosci Biobehav Rev. 2017;74(Pt B):321–9.
50. Liu PY, Lawrence-Sidebottom D, Piotrowska K, Zhang W, Iranmanesh A, Auchus RJ, et al. Clamping Cortisol and Testosterone Mitigates the Development of Insulin Resistance during Sleep Restriction in Men. J Clin Endocrinol Metab. 2021;106(9):e3436–48.
51. Rao R, Somvanshi P, Klerman EB, Marmar C, Doyle FJ, 3rd. Modeling the Influence of Chronic Sleep Restriction on Cortisol Circadian Rhythms, with Implications for Metabolic Disorders. Metabolites. 2021;11(8):483.
52. Kim YC, Um YJ, Yoon SH, Kim TW, Seo HJ, Jeong JH, et al. Association between weekend catch-up sleep and the risk of prediabetes and diabetes: A cross-sectional study using KNHANES. J Psychosom Res. 2024;179:111618.
53. Jang YS, Park YS, Hurh K, Park EC, Jang SI. Association between weekend catch-up sleep and dyslipidemia among Korean workers. Sci Rep. 2023;13(1):925.
54. Chen N, Guo L, Wang L, Dai S, Zhu X, Wang E. Sleep fragmentation exacerbates myocardial ischemia-reperfusion injury by promoting copper overload in cardiomyocytes. Nat Commun. 2024;15(1):3834.
55. Min A, Seo J, Kang M, Hong HC. Sleep Deprivation and Fatigue among Nurses Working Consecutive Night Shifts: A Prospective Observational Study. J Korean Acad Nurs. 2024;54(2):139–50.
56. Garbarino S, Lanteri P, Bragazzi NL, Magnavita N, Scoditti E. Role of sleep deprivation in immune-related disease risk and outcomes. Commun Biol. 2021;4(1):1304.
57. Sang D, Lin K, Yang Y, Ran G, Li B, Chen C, et al. Prolonged sleep deprivation induces a cytokine-storm-like syndrome in mammals. Cell. 2023;186(25):5500–5516.e5521.
58. Li X, Cao Y, Xu X, Wang C, Ni Q, Lv X, et al. Sleep Deprivation Promotes Endothelial Inflammation and Atherogenesis by Reducing Exosomal miR-182-5p. Arterioscler Thromb Vasc Biol. 2023;43(6):995–1014.
59. Zheng Y, Zhang L, Bonfili L, de Vivo L, Eleuteri AM, Bellesi M. Probiotics Supplementation Attenuates Inflammation and Oxidative Stress Induced by Chronic Sleep Restriction. Nutrients. 2023;15(6):1518.
60. Beaman A, Bhide MC, McHill AW, Thosar SS. Biological pathways underlying the association between habitual long-sleep and elevated cardiovascular risk in adults. Sleep Med. 2021;78:135–40.
61. Qiu Y, Fernández-García B, Lehmann HI, Li G, Kroemer G, López-Otín C, et al. Exercise sustains the hallmarks of health. J Sport Health Sci. 2023;12(1):8–35.
62. Ozemek C, Lavie CJ, Rognmo Ø. Global physical activity levels - Need for intervention. Prog Cardiovasc Dis. 2019;62(2):102–7.
63. Roenneberg T, Allebrandt KV, Merrow M, Vetter C. Social jetlag and obesity. Curr Biol. 2012;22(10):939–43.
64. Benson JA, McSorley VE, Hawkley LC, Lauderdale DS. Associations of loneliness and social isolation with actigraph and self-reported sleep quality in a national sample of older adults. Sleep. 2021;44(1):zsaa140.
65. Leigh-Hunt N, Bagguley D, Bash K, Turner V, Turnbull S, Valtorta N, et al. An overview of systematic reviews on the public health consequences of social isolation and loneliness. Public Health. 2017;152:157–71.
66. Duan D, Kim LJ, Jun JC, Polotsky VY. Connecting insufficient sleep and insomnia with metabolic dysfunction. Ann N Y Acad Sci. 2023;1519(1):94–117.
67. Anaya G, Pettee Gabriel K, St-Onge MP, van Horn LV, Alfini A, Badon SE, et al. Optimal Instruments for Measurement of Dietary Intake, Physical Activity, and Sleep Among Adults in Population-Based Studies: Report of a National Heart, Lung, and Blood Institute Workshop. J Am Heart Assoc. 2024;13(21):e035818.

Publisher's Note