Association between weekend catch-up sleeping and BMI of the United States population from 2017 to 2018

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Research Article

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Abstract

Background

Obesity represents a major public health issue worldwide. In this study, we aimed to investigate the association between weekend catch-up sleep (CUS) and BMI.

Method

Our data were derived from the National Health and Nutrition Examination Survey (NHANES) database. We applied the linear regression model and the multiple logistic regression model to examine the correlation between weekend CUS and BMI adjusting for confounders, and stratified regression analysis was conducted to identify specific populations.

Results

A total of 5,434 adult participants with complete data were selected for the study. Participants with CUS were more likely to have obesity status (OR = 1.17, P = 0.03). The correlation did have a difference in the population with normal BMI or obesity groups (P for interaction = 0.02). Furthermore, in enough weekday sleep group, CUS is related to higher BMI (β: 0.70, 95%CI: 0.02 to 1.38, P = 0.04). The sensitivity analyses in patients with a restricted range of BMI were consistent with our main findings.

Conclusion

Our study confirmed that weekend CUS is associated with an increased BMI, with different odds ratio values among different subgroups, which also provides evidence for exploring underlying mechanisms and potential clinical applications in obesity treatment.

Introduction

With the prevalence increasing globally, obesity has been considered a pandemic over the years, representing a major public health concern\[1\]. According to World Obesity Atlas 2023 published by the World Obesity Federation (WOF), over 4 billion people may be affected by overweight and obesity by 2035, compared with over 2.6 billion in 2020. Body mass index (BMI), a numerical value calculated as the weight in kilograms divided by the square of the height in meters, is most commonly used as the measure of obesity because of its convenience and high efficiency\[2\]. Many contributors to obesity have been reported, such as heredity, unhealthy lifestyle, and excessive calorie intake\[3−5\]. These days, the impact of sleep behavior on obesity has drawn more and more attention.
In modern society, people always get insufficient sleep on account of their work or lifestyle, especially on weekdays\[^{6,7}\]. As a result of this, many people choose some other measures to counteract the deleterious effects of insufficient sleep. Weekend catch-up sleep (CUS) refers to a lifestyle in that people usually sleep more on weekends, especially for those sleeping insufficiently on weekdays\[^{8}\]. Several previous studies indicated that weekend CUS seems to play a mitigating role in the diseases caused by lack of sleep\[^{9−11}\]. However, on the contrary, CUS may prolong the sleep duration of the weekend, which can also affect physical health. On the other hand, this change in sleep duration between weekdays and weekends will not be beneficial to the stabilization of energy balance. And it has been proved that poor sleep duration (short or long sleep length) can increase the risk of obesity\[^{12,13}\]. As the research about the association of weekend CUS and obesity or BMI is still limited, more nationwide population-based studies are quite necessary to evaluate this influence.

The present study investigated the relationship between weekend CUS and body mass index using data from the National Health and Nutrition Examination Survey (NHANES) database by regression analysis.

**Materials and methods**

**Study design and participants**

NHANES is a research project that provides a wide range of health and nutrition status of adults and children across the United States, including demographics, dietary, examination, laboratory, and questionnaire data. All the participants provided written informed consent.

In the present study, we performed statistical analysis on NHANES 2017−2018 data, which had an enrollment of 9254 participants. Our analysis restricted study inclusion to adults (age ≥ 18) with complete data on BMI and sleep duration on both weekdays and weekends. The final sample size of the study comprised 5434 patients. Detailed exclusion criteria were shown in the flow chart. The study protocol was reviewed and approved by the NCHS Research Ethics Review Board.

**Definition of CUS**

The exposure variable of the study was weekend CUS. NHANES obtained the sleep time (time of falling asleep) and waking-up time on weekdays through questionnaire surveys and added two additional questions regarding sleep time and waking-up time on weekends or non-working days. The research group then calculated the sleep duration within the main sleep period of weekdays and weekends for patients (in hours, excluding sleep time in non-main sleep periods such as napping). The weekend CUS time was calculated by subtracting the weekday sleep duration from the weekend sleep duration. Results greater than 0 were defined as having the habit of catching up on sleep on weekends.

**Assessment of outcome variable**

The outcome variable of the study was the BMI status. The calculation method for the participants' BMI provided by NHANES was to divide the weight in kilograms by the square of height in meters, and then
round to one decimal place. The weight and height data were obtained from physical measurements taken by trained health technicians at the Mobile Examination Center.

**Assessment of other covariates**

The covariate variables included age, gender, race, education level, marital status, family income, hypertension, high cholesterol, diabetes, alcohol use, income-to-poverty ratio, enough sleep on weekdays, and metabolic equivalent (MET) level. Age = 80 included those at or above 80 years old in NHANES. Alcohol intake was defined as having consumed any type of alcoholic beverage in the past. MET level was the total metabolic equivalent of tasks in a typical day, obtained through a self-reported questionnaire, calculated as the sum of the product of activity time and corresponding MET score for each kind of activity, and then categorized into three groups (≤ 499, > 499 and ≤ 999, >999)[14]. Medical history, including hypertension, high cholesterol, and diabetes, was self-reported by patients regarding whether they had been informed by a doctor or healthcare professional of having such conditions or not. Income to poverty ratio was calculated by dividing family (or individual) income by the poverty guidelines specific to the survey year. The value was not computed if the respondent only reported income as <$20,000 or ≥ $20,000. If family income was reported as a more detailed category, the midpoint of the range was used to compute the ratio. Values at or above 5.00 were coded as 5.00 or more because of disclosure concerns. We divided the ratio into three groups as previously reported[15].

**Statistical analysis**

The basic characteristics comparison between the BMI ≥ 25 (high BMI) group, BMI < 25 and ≥ 18.5 (moderate BMI) group, and BMI < 18.5 (low BMI) group was evaluated using weighted chi-square test (for categorical variables) and weighted linear regression model (for continuous variables).

Two approaches were applied to discuss the association between weekend CUS and BMI. First, we used the linear regression model to describe the correlation between weekend CUS and continuous BMI. Second, we categorized BMI data into two groups (BMI ≥ 25 was defined as obesity, while BMI < 25 was defined as non-obesity) and performed analysis under a multiple logistic regression model. In addition, to identify specific populations, stratified regression analysis was conducted regarding the risk factors for high BMI. The stratification variables included gender, age, marital status, alcohol intake, hypertension, high cholesterol, smoking, MET level, and sleep duration on weekdays.

Women's BMI tends to undergo certain changes after menopause, so we took 45 years old as the start of menopause and analyzed the relationship between weekend CUS and BMI in different age groups of women. The present study further explored the correlation between weekend CUS and BMI in two groups (obesity and non-obesity).

To eliminate the potential effect of the low BMI group (BMI < 18.5) on the overall distribution of BMI in the present study, we restricted our participants to the moderate and high BMI groups in sensitivity analysis. We evaluated the association between CUS and BMI and repeated the subgroup analysis in patients from the two groups.
Results

The present study had an inclusion of 5434 adult participants in the analysis and documented 3963 high BMI and 99 low BMI. The characteristics of the participants according to BMI are presented in Table 1. The proportion of patients from four main ethnic groups had increased in the high BMI group. Alcohol use and lower metabolic levels were correlated with higher BMI. The high BMI group had a significantly higher possibility of hypertension, high cholesterol, and diabetes, in contrast with the moderate and low BMI groups, and was more likely to have received under-university education. Individuals who have never been married and those with higher income levels tend to have a moderate or lower BMI.
Table 1
The characteristics of the study participants (CUS and BMI).

<table>
<thead>
<tr>
<th></th>
<th>BMI &lt; 18.5 (N = 99)</th>
<th>BMI ≥ 18.5, &lt; 25 (N = 1372)</th>
<th>BMI ≥ 25 (N = 3963)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>39.56 ± 19.98</td>
<td>46.72 ± 20.29</td>
<td>50.96 ± 17.68</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td>0.113</td>
</tr>
<tr>
<td>male</td>
<td>43 (43.43%)</td>
<td>634 (46.21%)</td>
<td>1946 (49.10%)</td>
<td></td>
</tr>
<tr>
<td>female</td>
<td>56 (56.57%)</td>
<td>738 (53.79%)</td>
<td>2017 (50.90%)</td>
<td></td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Mexican American</td>
<td>6 (6.06%)</td>
<td>113 (8.24%)</td>
<td>619 (15.62%)</td>
<td></td>
</tr>
<tr>
<td>Other Hispanic</td>
<td>5 (5.05%)</td>
<td>94 (6.85%)</td>
<td>406 (10.24%)</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic White</td>
<td>40 (40.40%)</td>
<td>469 (34.18%)</td>
<td>1356 (34.22%)</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic Black</td>
<td>26 (26.26%)</td>
<td>278 (20.26%)</td>
<td>956 (24.12%)</td>
<td></td>
</tr>
<tr>
<td>Other race</td>
<td>22 (22.23%)</td>
<td>418 (30.47%)</td>
<td>626 (15.80%)</td>
<td></td>
</tr>
<tr>
<td><strong>Education level</strong></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Less than high school degree</td>
<td>30 (30.30%)</td>
<td>287 (20.94%)</td>
<td>837 (21.16%)</td>
<td></td>
</tr>
<tr>
<td>High school grad or some college degree</td>
<td>54 (54.55%)</td>
<td>705 (51.42%)</td>
<td>2267 (57.32%)</td>
<td></td>
</tr>
<tr>
<td>College graduate or above</td>
<td>15 (15.15%)</td>
<td>379 (27.64%)</td>
<td>851 (21.52%)</td>
<td></td>
</tr>
<tr>
<td><strong>High blood pressure</strong></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Yes</td>
<td>13 (13.13%)</td>
<td>304 (22.22%)</td>
<td>1655 (41.82%)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>86 (86.87%)</td>
<td>1064 (77.78%)</td>
<td>2302 (58.18%)</td>
<td></td>
</tr>
<tr>
<td><strong>High blood cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Mean ± SD for continuous variables; % for categorical variables
<table>
<thead>
<tr>
<th>Metric</th>
<th>BMI &lt; 18.5 (N = 99)</th>
<th>BMI ≥ 18.5, &lt; 25 (N = 1372)</th>
<th>BMI ≥ 25 (N = 3963)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>9 (9.09%)</td>
<td>339 (24.85%)</td>
<td>1479 (37.59%)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>90 (90.91%)</td>
<td>1025 (75.15%)</td>
<td>2456 (62.41%)</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Yes</td>
<td>2 (2.02%)</td>
<td>144 (10.50%)</td>
<td>834 (21.07%)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>97 (97.98%)</td>
<td>1228 (89.50%)</td>
<td>3125 (78.93%)</td>
<td></td>
</tr>
<tr>
<td>Income to poverty ratio</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>&lt; 1</td>
<td>28 (33.73%)</td>
<td>233 (19.56%)</td>
<td>608 (17.90%)</td>
<td></td>
</tr>
<tr>
<td>&gt;=1, ≤3</td>
<td>39 (46.99%)</td>
<td>512 (42.99%)</td>
<td>1554 (45.75%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 3</td>
<td>16 (19.28%)</td>
<td>446 (37.45%)</td>
<td>1235 (36.35%)</td>
<td></td>
</tr>
<tr>
<td>MET level</td>
<td></td>
<td></td>
<td></td>
<td>0.004</td>
</tr>
<tr>
<td>≤ 499</td>
<td>53 (53.54%)</td>
<td>740 (53.94%)</td>
<td>2244 (56.62%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 499, ≤ 999</td>
<td>25 (25.25%)</td>
<td>215 (15.67%)</td>
<td>539 (13.60%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 999</td>
<td>21 (21.21%)</td>
<td>417 (30.39%)</td>
<td>1180 (29.78%)</td>
<td></td>
</tr>
<tr>
<td>Alcohol use</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Yes</td>
<td>66 (76.74%)</td>
<td>1095 (86.49%)</td>
<td>3329 (89.73%)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>20 (23.26%)</td>
<td>171 (13.51%)</td>
<td>381 (10.27%)</td>
<td></td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Married/ living with partner</td>
<td>37 (45.12%)</td>
<td>703 (56.10%)</td>
<td>2322 (60.55%)</td>
<td></td>
</tr>
<tr>
<td>Widowed/ Divorced/ Separated</td>
<td>20 (24.39%)</td>
<td>269 (21.47%)</td>
<td>890 (23.20%)</td>
<td></td>
</tr>
</tbody>
</table>

Mean ± SD for continuous variables; % for categorical variables.
The association between CUS and continuous BMI is discussed in Table 2. Three models with progressively greater degrees of adjustment had shown a positive correlation between CUS and BMI (Model 1: $\beta = 0.41, P = 0.04$; Model 2: $\beta = 0.52, P = 0.01$; Model 3: $\beta = 0.61, P = 0.009$). The results of the logistic regression model strengthened the evidence as the participants with CUS were more likely to have obesity status (OR = 1.17, $P = 0.03$) (Table S1).

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>BMI $&lt; 18.5$ (N = 99)</th>
<th>BMI $\geq 18.5, &lt; 25$ (N = 1372)</th>
<th>BMI $\geq 25$ (N = 3963)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never married</td>
<td>25 (30.49%)</td>
<td>281 (22.43%)</td>
<td>623 (16.25%)</td>
<td></td>
</tr>
</tbody>
</table>

Mean ± SD for continuous variables; % for categorical variables

In subgroup analyses, results of multivariate linear regression analysis stratified by selected factors are presented in Fig. 1. Under different subgroups, the positive significance was observed only in females ($\beta$: 0.76, 95% CI: 0.12 to 1.40, $P = 0.02$), middle age ($\geq 44, < 60$) ($\beta$: 1.06, 95% CI: 0.24 to 1.89, $P = 0.01$), being ($\beta$: 0.71, 95% CI: 0.18 to 1.24, $P = 0.008$) or have been ($\beta$: 1.82, 95% CI: 0.88 to 2.75, $P = 0.0001$) married, have high cholesterol ($\beta$: 0.99, 95% CI: 0.27 to 1.71, $P = 0.007$), smoke ($\beta$: 1.04, 95% CI: 0.34 to 1.73, $P = 0.004$), have alcohol use ($\beta$: 0.69, 95% CI: 0.22 to 1.15, $P = 0.004$) and low MET level ($\beta$: 0.83, 95% CI: 0.24 to 1.42, $P = 0.006$). Interestingly, both hypertension ($\beta$: 1.19, 95% CI: 0.46 to 1.92, $P = 0.001$) and non-hypertension group ($\beta$: 0.59, 95% CI: 0.07 to 1.11, $P = 0.03$) had seen an increase in BMI when having CUS. The correlation did have a difference in population in two groups categorized by BMI (P for interaction = 0.02) (Fig. 2). BMI dramatically rose in the obese patients with CUS ($\beta = 0.93, P < 0.0001$), while the result
in patients without obesity is neither obvious ($\beta = 0.013$) nor significant ($P = 0.91$). CUS was associated with higher BMI elevation in the obesity group compared with the non-obesity group.

Analysis found that in enough weekday sleep group (6 hours $\leq$ sleep duration $< 8$ hours), CUS is related to higher BMI ($\beta: 0.70$, 95%CI: 0.02 to 1.38, $P = 0.04$). No significance was observed in two other groups (Table 3). When restricted population to females only, the association between CUS and BMI varied before and after menopause ($P$ for interaction $= 0.0145$). Results didn't indicate the significance in females under 45 years old ($P = 0.40$) (Fig. 3).

Table 3
Association between CUS and BMI in population with different sleep duration in weekdays.

<table>
<thead>
<tr>
<th>Sleep duration in weekdays</th>
<th>N</th>
<th>$\beta$ (reference = 0)</th>
<th>(95%CI)</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td>$&lt; 6$</td>
<td>598</td>
<td>-0.91</td>
<td>(-2.58, 0.76)</td>
<td>0.29</td>
</tr>
<tr>
<td>$\geq 6, &lt;8$</td>
<td>2175</td>
<td>0.70</td>
<td>(0.02, 1.38)</td>
<td>0.04</td>
</tr>
<tr>
<td>$\geq 8$</td>
<td>2661</td>
<td>0.32</td>
<td>(-0.34, 0.99)</td>
<td>0.34</td>
</tr>
</tbody>
</table>

Adjusted for: race; education level; marital status; high blood pressure; high cholesterol; diabetes; income to poverty ratio; MET level; smoke; age; gender.

The results of sensitivity analyses in patients with a restricted range of BMI were consistent with our main findings (Table S2). However, in subgroup analysis after excluding patients with low BMI, we found a significant positive association in the non-smoke group, moderate MET level group, and patients without high cholesterol, which was not observed in the main study (Table S3).

Discussion

Retrospect of our study

In this study, we utilized a nationally representative sample and demonstrated a clear positive correlation between CUS and elevated BMI risk, which further implies the increased risk of obesity. Compared to individuals without CUS, the group with CUS had a 1.24 times higher likelihood of obesity. Furthermore, our study performed stratified analyses and indicated that this correlation is more pronounced among females, middle-aged individuals (aged 40–60), those who are divorced, widowed or separated, individuals with high blood pressure, elevated cholesterol levels, alcohol consumers, and those with a lower metabolic equivalent.

Besides, there are also other circumstances characterized by increased BMI index in which CUS plays an important role. For individuals with habitual sleep deprivation (weekday sleep duration $< 6$ hours), we believe that catching up on sleep may not alleviate the changes in BMI caused by sleep deprivation, and finally lead to the same ending. Additionally, for those with sufficient sleep during weekdays (weekday sleep duration ranged from 6h to 8h), catching up on sleep could potentially directly contribute to an
elevated BMI (Table 3). In the regression model analysis, participant grouping analysis, and study feature analysis, the outcomes of different groups all exhibit statistical significance.

**Comparison between our study and previous investigations**

There are similarities in results to mechanisms between our study and existing research. One randomized, three-group, in-laboratory Clinical Translational Research Center (CTRC) protocol study including 36 people found that in repeated instances of inadequate sleep, weekend recovery sleep temporarily and partially improves metabolic imbalances\(^{[16]}\). Another study by the same group showed that inadequate sleep, with or without weekend recovery sleep, had similar effects on 24-hour energy balance\(^{[17]}\). Wang et al.'s randomized study enlisted 62 volunteers from Columbia, South Carolina, and surrounding areas, discovered that sleep restriction (SR) hindered fat mass reduction despite similar weight loss\(^{[18]}\), supporting the idea that weekend sleep catch-up may not fully counteract SR-induced body composition changes\(^{[19]}\), which agrees with other research. Considering these studies and our research, we conclude that unhealthy behaviors like insufficient weekday sleep and subsequent weekend catch-up sleep likely lead to obesity. Therefore, we discourage "sleep compensation" to counter weekday sleep debt, as this might not yield positive results. A healthier lifestyle involves evenly distributing sleep throughout each day.

More importantly, for the group of individuals who typically have normal sleep patterns, CUS during the weekends also increases the risk of obesity (SD > 6, < 8; β, 0.6989; 95% CI, 0.0164–1.3813 and SD > 8; β, 0.3239; 95% CI, -0.3378, 0.9856). A large cross-sectional analysis including 2848 participants indicates that obesity seems to be linked with both short and long sleep patterns, and individuals who slept for 9 hours or more exhibited the highest levels of fasting insulin and insulin resistance, whereas those who slept between 7 to 7.5 hours displayed the lowest levels, in which a pattern resembling a U-shaped relationship can be observed\(^{[21]}\). These findings suggest that longer sleep duration may more significantly affect insulin secretion than shorter sleep, and sleep may directly impact adipose tissue function, exacerbating metabolic dysfunction in obesity. Besides, when considering energy balance, excessive sleep might lead to fatigue and drowsiness, increasing sedentary time and causing weight gain. Meanwhile, an excessively long duration of sleep will correspondingly reduce the time available for other activities such as physical exercise. In turn, as obesity is closely associated with the existence of sleep-disordered breathing\(^{[22]}\), long-sleepers may extend their sleep duration due to conditions like obstructive sleep apnea, which disrupts sleep quality and can be associated with obesity. This may cause individuals to seek longer sleep to attain a refreshing sensation similar to that of a ‘typical sleeper’\(^{[21]}\).

However, what is noteworthy is that conflicting results have already been presented by others. For example, a Hong Kong cross-sectional study contains 5159 elementary students\(^{[23]}\), and a South Korean cohort study including 936 children aged 10 ~ 11 years\(^{[24]}\) suggests that extending weekend catch-up sleep might guard against weight gain due to ongoing sleep shortage\(^{[25]}\). The results of the aforementioned study are inconsistent with our findings, possibly due to differences in the racial and age demographics of the study population. To ensure the rigor and reliability of our results, we have
incorporated a larger sample size (with 5,434 participants in our study). Additionally, we have taken into account potential covariates that may influence the outcomes, aiming to minimize the occurrence of errors.

**Possible mechanisms**

Given the previous research findings, here are the potential mechanisms through which CUS may lead to obesity.

A review concerning sleep duration and quality suggests that the association between sleep duration, sleep disorders, and hypertension remains inconclusive\[26\], as various meta-analyses yield contradictory conclusions\[27–29\]. There’s a potential explanation that short sleep duration may lead to the overproduction of proinflammatory cytokines and inflammatory markers\[30\], and previous reviews have shown that inflammation progress can be activated during chronic obesity and revealed the mutual effect of inflammation and obesity\[31\]. What’s more, A consistent correlation exists between short sleep duration and elevated overall energy and fat intake\[32,33\], which could potentially mediate the accumulation of obesity during workdays. Due to variability/regularity also stands as a significant facet of sleep health and CUS could also be considered as a form of sleep variability, we can presume that cardiometabolic outcomes like obesity\[34–37\], metabolic syndrome\[33,37,38\], glucose control\[37,39\] and hypertension\[37,40,41\], which have been proven to be connected to sleep variability. Potential mechanisms involve alterations in hormones related to appetite regulation, such as hunger hormone ghrelin and leptin, especially in women\[42\]; social jet lag(SJL) inducing changes in brain regions associated with hedonic eating, resulting in increased appetite and hunger\[43\]. Besides, under conditions of sufficient sleep, there is also a certain association between social jet lag (SJL) and a less favorable gut microbiome\[44\], which also contributes to obesity. Disruptions in sleep and circadian rhythms can lead to metabolic disorders, including disruptions in energy metabolism and bile acid metabolism, which in turn affect cholesterol breakdown metabolism, promoting the occurrence of both cholesterol-related issues and obesity\[45\].

**Limitations**

Our study also has certain limitations. Firstly, as a cross-sectional study based on national databases, we can only establish a correlation between CUS and obesity, causal relationships cannot be confirmed. However, based on our understanding of other relevant research, our study leans towards that CUS increases the risk of obesity. Secondly, we employed the BMI index to define obesity, which introduces some bias as it does not consider the possibility that individuals with high muscle mass and low body fat may be categorized as obese in our study. Future improvements involving metrics such as body fat percentage and lean body mass can help us define obesity more accurately. Additionally, NHANES lacks some data that could potentially impact the results, such as the use of sleeping pills or weight-loss medications, which somewhat limits the scope of our statistical analysis. Lastly, we did not correct for mid-sleep time on free days (MSFsc), and variations in different sleep periods may introduce deviations.
Despite these points, our study still possesses advantages such as a large sample size, rigorous categorization, and comprehensive inclusion of covariates to minimize potential confounding factors.

**Conclusion**

In summary, our study confirms CUS leads to an increase in BMI. For individuals with insufficient sleep (SD < 6 hours), CUS doesn't fully counteract the BMI increase resulting from sleep deprivation. Conversely, for individuals with adequate sleep (SD > 6 hours), CUS raises the risk of obesity. Therefore, we recommend that individuals who have a habit of CUS should increase their weekday sleep moderately and avoid excessive weekend sleep, which can contribute to obesity prevention and better overall health. Our study also provides evidence for exploring underlying mechanisms and potential clinical applications in obesity treatment.

**Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviations</th>
<th>Full name</th>
</tr>
</thead>
<tbody>
<tr>
<td>NHANES</td>
<td>National Health and Nutrition Examination Survey</td>
</tr>
<tr>
<td>CUS</td>
<td>catch-up sleep</td>
</tr>
<tr>
<td>BMI</td>
<td>body mass index</td>
</tr>
<tr>
<td>MET</td>
<td>metabolic equivalent task</td>
</tr>
<tr>
<td>OR</td>
<td>Odds ratio</td>
</tr>
<tr>
<td>P</td>
<td>P-value</td>
</tr>
</tbody>
</table>

**Declarations**

**Ethics approval and consent to participate**

All of the 9254 participants of National Examination Survey (NHANES) 2017-2018 provided informed consent. National Center For Health Statistics (NCHS) Research Ethics Review Board (ERB) provided approval of the survey protocol.

**Consent for publication**

Not applicable

**Availability of data and materials**

The datasets analysed during the current study are available on the National Health and Nutrition Examination Survey (NHANES) database. [https://www.cdc.gov/nchs/index.htm]
Competing interests

The authors declare no competing interests.

Funding

None

Authors’ contributions

Y.Z.: Conceptualization, Formal analysis, Writing - review & editing, Project administration, Supervision.

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none

Disclosure: The authors declare no conflict of interest

References


**Figures**
Figure 1

Title: Association between CUS and BMI, stratified by gender, age, marital status, high blood pressure, high cholesterol, alcohol use, smoke and MET level.

Legends: Adjusted for: family income; race; education level; diabetes; income to poverty ratio.

Figure 2

Title: Association between CUS and BMI in population with or without obesity.
Legends: Adjusted for: race; education level; diabetes; income to poverty ratio; family income.

**Figure 3**

Title: Association between CUS and BMI in females before and after menopause.

Legends: Adjusted for: race; education level; marital status; high blood pressure; high cholesterol; diabetes; income to poverty ratio.

**Supplementary Files**

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- supplementarymaterialonline.docx