RESEARCH ARTICLE

Relationship between weekends catch-up sleep and risk of aging

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Abstract

Background

Sleep has been proven to be associated with various chronic diseases and aging. However, many individuals fail to achieve recommended sleep durations on weekdays and opt for compensatory sleep during weekends. This study aims to investigate the relationship between weekend catch-up sleep (CUS) and aging.

Methods

All participants were sourced from NHANES 2017–2018. Using the sleep questionnaire, we obtained participants' sleep timings and durations on weekdays and weekends. Weekend CUS was identified as an extension in average weekend sleep duration. Biological age is a biomarker for evaluating biological aging, and its difference from actual age is used to determine aging. Weighted logistic regression analysis was employed to explore the relationship between CUS and aging.

Results

A total of 4,713 participants were included in this study, with an average age of 47.54 ± 16.94 years. 50.6% of individuals experienced CUS. Compared to individuals without CUS, participants with CUS had a 20% lower risk of aging (OR = 0.80, 95% CI: 0.63−1). Specifically, participants who engaged in CUS for 0−1 hour showed a 23% lower risk of aging (OR = 0.77, 95% CI: 0.61–0.96), and those with CUS for 1−2 hours had a 20% lower risk of aging (OR = 0.80, 95% CI: 0.65–0.98). Stratifying by bedtime, the relationship between CUS and reduced aging risk is only observed in individuals who usually go to sleep before midnight and have CUS less than 2 hours.

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Abbreviations: CRP, C-reactive protein; CUS, catch-up sleep; NCHS, National Center for Health Statistics; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio; PIR, ratio of family income to poverty; RCS, restricted cubic spline.

Conclusion

The 0–2 hour CUS is associated with a reduced risk of aging, and this relationship is more significant in participants who go to bed early and have healthy sleep patterns.

Introduction

Sleep is closely related to health, and maintaining a regular sleep pattern, stable sleep duration, and high-quality sleep is crucial for overall well-being and disease resistance, which includes the impact on cardiovascular diseases, cognition, metabolism, and various other conditions such as tumors [1,2]. Numerous studies have recommended optimal sleep duration and patterns. A study by Li et al. from the UK Biobank, based on the non-linear association between sleep duration and genetic, cognitive, brain structure, and mental health factors, found that the optimal sleep duration was approximately 7 hours [3]. Furthermore, another study, analyzing two independent cohorts, demonstrated an association between a healthy sleep pattern including timing, duration, snoring, and excessive daytime sleepiness and a lower risk of heart failure, independent of traditional risk factors [4]. Some prospective studies also indicated a close association between excessively long or short sleep duration and an increased risk of cancer [5,6].

However, in recent years, the rapid development of society, coupled with the demands of work and irregular lifestyles, has led to an increase in staying up late and a reduction in sleep duration [7,8]. As a result, many people have resorted to opt for Catch-Up Sleep (CUS) during the weekends to compensate for the lack of sleep on weekdays. This concept is similar to the "weekend warrior" idea proposed by Dos et al., where many adults, due to factors such as work or overtime, are unable to meet the daily requirements for physical activity [9]. Instead, they opt to engage in equivalent intensity physical activity for one or two days each week to mitigate the harms associated with prolonged sitting and low physical activity [10]. This approach aims to achieve benefits and lower mortality rates similar to those who engage in regular physical exercise. Recent studies have defined various criteria for measuring catch-up sleep (CUS). A study based on the National Health and Nutrition Examination Survey (NHANES) defined CUS by the difference in sleep duration between workdays and weekends, categorizing it into no CUS, mild CUS (0-1h), moderate CUS (1-2h), and long CUS (>2h) [11,12]. CUS appears to be associated with certain diseases; for instance, Liu et al. demonstrated that individuals with 1–2 hours of CUS had a lower risk of depression compared to those who did not have CUS [12]. Chen et al. showed that compared with those without CUS, 2–3 hours of CUS was closely associated with a lower prevalence of chronic kidney disease [13]. However, its relationship with other diseases or aging remains unclear.

Aging is a collective term for the decline in physiological functions during adulthood, characterized by features such as increased susceptibility to diseases, genomic instability, epigenetic changes, and chronic inflammation [14,15]. Despite clear molecular-level characteristics of aging, it is challenging to precisely define

whether an individual has truly undergone aging at the population level. In fact, recent research encourages evaluating whether aging has occurred at the population level by examining the difference between physiological age and chronological age [14,16]. Coincidentally, Liu et al. developed a widely validated and used phenotypic age calculation formula based on NHANES data [17]. Up to now, although no studies have explored the relationship between the increasingly common weekend catch-up sleep (CUS) habit and aging, research has investigated the association between sleep duration patterns and aging. The results showed that compared with participants whose sleep duration was in a normal and stable trajectory, trajectories of increased sleep duration and short-term stable sleep duration were associated with a lower likelihood of successful aging. Worse sleep patterns were also closely related to cognitive decline, yet the relationship between catch-up sleep and aging remains unaddressed [18,19]. To fill this gap, this study aims to investigate the association between CUS and aging using the NHANES database, and further explore the anti-aging effects of weekend CUS among individuals with different sleep times, sleep durations, and sleep disorders.

Methods

Study population

As reported in our previous research, NHANES is a large-scale cross-sectional study conducted by the National Center for Health Statistics (NCHS). It aims to investigate the health and nutritional status of adults and children in the United States [20].

In this study, data from the year 2017–2018 were utilized, as during this survey cycle, the majority of participants reported their average sleep duration on workdays and weekends, as well as issues like snoring and sleep disorders. Among these patients, 4,771 individuals had information on variables used to calculate phenotypic age. Out of these, 11 participants lacked information on educational level, 4 lacked information on marital status, 36 were missing sleep duration data, and 7 were missing sleep timing information. Ultimately, 4,713 participants were included in this study.

Exposure assessment

The main exposure factor is CUS, derived from participants' responses to the NHANES questionnaire regarding average sleep duration on weekdays and weekends. An increase in average weekend sleep duration is considered as CUS. Additionally, the study distinguishes the extent of CUS based on the difference values, we classified CUS duration as≤0 hours (i.e., no CUS), 0 < CUS duration≤1 hour, 1 hour<CUS duration≤2 hour and >2 hour [8,9]. This information will be utilized to assess the relationship between weekend CUS and other factors such as phenotypic age and anti-aging effects [7].

Outcome

This study's primary outcome is aging. Initially, phenotypic age is calculated based on previous research. Factors within the equation include albumin, alkaline phosphatase, C-reactive protein, total cholesterol, blood creatinine, glycated hemoglobin, systolic blood pressure, blood urea nitrogen, uric acid, white blood cell count, lymphocyte percentage, mean cell volume, and red blood cell distribution width [21]. The specific calculation methods are detailed in Supplementary Method 1 [22]. Subsequently, the calculated phenotypic age is subtracted from the actual age. If the difference is greater than 0, it is categorized as aging; otherwise, it is classified as non-aging.

Covariates

Sleep-related content has been adapted from the Munich ChronoType Questionnaire, covering sleep habits, sleep disorders, sleep duration, and wake-up times [23]. Participants are typically asked, "What time do you usually fall asleep/wake up on weekdays or weekends?" Based on the sleep time and using midnight (00:00) as the reference point, participants are categorized as either early sleepers or late sleepers. Sleep duration is categorized based on previous research.

Participants are divided into three groups: < 7 hours, 7–8 hours, and >8 hours, and are respectively considered to have short, normal, and long sleep durations [24]. Sleep disorders are recognized by healthcare professionals, and participants are asked, "Have you ever told a doctor or other health professional that you have trouble sleeping?". Covariates in the study include age, gender, marital status (Married, Separated, Never married), BMI (considered obese if greater than or equal to 30 kg/m2), Ratio of family income to poverty (PIR, < 1.30, 1.30–3.49, ≥ 3.50) [25], education level, smoking, alcohol consumption, and physical activity. Data on physical activity are derived from the Global Physical Activity Questionnaire, and all adults provided interview data on physical activity, including vigorous activities, moderate-intensity exercise, and more [26].

Statistical analyses

All statistical analyses were conducted using R 4.2.3. A two-sided P-value of <0.05 was considered statistically significant.

Weighted analysis was employed in the analyses, implemented using the "survey" package in R [27]. Continuous variables were presented as mean ± standard deviation (SD) or median (P25, P75), and between-group differences were assessed using t-tests or Mann-Whitney U tests depending on the data distribution (normal or non-normal). Categorical variables were expressed as N (%), and between-group differences were compared using the chi-square test. Restricted cubic spline (RCS) plots were used to illustrate the nonlinear relationship between aging risk and sleep duration or weekend CUS duration. Weighted logistic regression analysis was employed to investigate the relationship between aging risk and weekend CUS, with results described in terms of odds ratios (OR) and 95% confidence intervals (CI). We conducted three models in the weighted logistic regression analysis. Model 1 represents the crude model, Model 2 adjusted for age, sex, marital status, PIR (Ratio of family income to poverty), educational level, and obesity. Model 3 adjusted for age and sex, marital status, PIR, educational level, obesity, smoke, alcohol use, sleep trouble, PA, sleep duration in work day (Supplementary Method 2). Considering that the timing and duration of sleep may also be related to aging, we further investigated the interaction between the timing and duration of sleep in the context of CUS and aging. CUS duration could act as a mediator between weekday sleep patterns (e.g., total weekday sleep, bedtime) and aging risk. Therefore, mediational analysis was used to clarify the role of CUS in these relationships.

Interaction analysis was used to explore whether CUS is a moderator of the relationship between weekday sleep duration or weekday bedtime and aging. Additionally, we conducted subgroup analyses and sensitivity analyses, including exploring the effects of CUS in participants with and without sleep disorders, as well as in those who were retired. Sensitivity analysis involved excluding patients taking sleep medications and antipsychotic medications (psychotherapeutic agents, stimulants, glucocorticoid, benzodiazepines, non – benzodiazepine hypnotics, miscellaneous anxiolytics, sedatives and hypnotics, benzodiazepine anticonvulsants, phenothiazine antipsychotics,ssri antidepressants and hypnotics, antiemetics), participants with night shifts or participation without a work schedule provided, participants with CUS less than −30 minutes.

Results

Baseline characteristics

A total of 4,713 participants (weighted: 102,908,622) were included in this study, with an average age of 47.54 ± 16.94 years. Among them, 2,267 were men, and 2,446 were women. 50.6% of individuals experienced CUS. Compared to those who did not catch up on sleep, participants with CUS were younger and more likely to have higher income or consume alcohol (Table 1). Regarding sleep duration, age showed a U-shaped correlation with weekday sleep duration and a negative relationship with weekend sleep duration (S1 Fig in S1 File). As for CUS duration, the proportion of individuals engaging in CUS increased initially with age, peaking in the 30–40 age group, and then declined. However, the proportion of individuals with CUS greater than 2 hours increased with age, corresponding to a decrease in this category (Fig 1).

Table 1. Baseline characteristics.

	Overall (Weighted)	Without CUS (Weighted)	With CUS (Weighted)	p
N (Weighted)	4713	2613 (49.4)	2100 (50.6)	
Age (mean±sd)#	47.54 (16.94)	50.92 (19.23)	43.36 (14.54)	<0.001*
Phenoage (mean±sd)#	46.19 (17.99)	51.82 (18.12)	41.57 (15.36)	<0.001*
Sex (%)				0.023*
Men	2267 (49.7)	1290 (52.6)	977 (46.9)	
Women	2446 (50.3)	1323 (47.4)	1123 (53.1)	
BMI (mean±sd)#	29.58 (6.83)	29.21 (6.38)	29.94 (7.23)	0.159
Obesity (%)				0.117
No	2761 (58.1)	1585 (60.9)	1176 (55.4)	
Yes	1952 (41.9)	1028 (39.1)	924 (44.6)	
Marital status (%)				0.023*
Married	2809 (64.4)	1504 (62.4)	1305 (66.4)	
Separated	1062 (16.5)	683 (19.8)	379 (13.3)	
Never married	842 (19.1)	426 (17.8)	416 (20.3)	
PIR (%)				0.030*
<1.30	1149 (14.8)	696 (17.9)	453 (11.8)	
1.30-3.49	1703 (30.3)	962 (30.6)	741 (30.0)	
≥3.50	1861 (54.9)	955 (51.4)	906 (58.2)	
Educational level (%)				0.127
Below college	3593 (75.7)	1974 (74.3)	1619 (77.2)	
College or above	1114 (24.3)	634 (25.7)	480 (22.8)	
Alcohol (%)	3053 (75.1)	1597 (71.8)	644 (78.3)	0.014*
Smoke (%)	1132 (25.1)	706 (26.2)	426 (23.9)	0.531
PA (%)	3509 (81.4)	1877 (80.9)	1632 (82.0)	0.511
Sleep trouble (%)	1347 (28.6)	806 (28.3)	541 (28.9)	0.831
CUS time (median [IQR])§	0.50 [0.00, 1.00]	0.00 [−0.50, 0.00]	1.00 [1.00, 2.00]	<0.001*
Sleep duration in workdays (median [IQR])§	7.50 [7.00, 8.00]	8.00 [7.00, 8.00]	7.50 [7.00, 8.00]	<0.001*
Sleep duration in weekends (median [IQR])§	8.00 [7.50, 9.00]	7.50 [7.00, 8.00]	9.00 [8.00, 9.50]	<0.001*

CUS, catch-up sleep, BMI, body mass index, PIR, poverty income ratio, PA, physical activity.

*Indicates statistical significance.

# Indicates that the inter-group difference was tested using the t-test.

§ Indicates that the inter-group difference was tested using the Mann-Whitney U test.

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The relationship between CUS and aging

RCS analysis reveals a U-shaped nonlinear relationship between the duration of CUS and the risk of aging, indicating that moderate CUS duration may be more beneficial for reducing aging risk (S2 Fig in S1 File). The subsequent logistic regression results (Table 2) showed that the OR values between CUS and aging risk were 0.79 (95% CI: 0.64–0.97), 0.77 (95% CI: 0.62–0.99), 0.80 (95% CI: 0.63–1) in Model 1, Model 2, and Model 3, respectively. In detail, when subdividing the duration of CUS and comparing it with participants who did not engage in CUS, those with CUS durations of 0–1 hour (OR=0.77, 95% CI: 0.61–0.96) and 1–2 hours (OR=0.80, 95% CI: 0.65–0.98) had a 23% and 20% reduced risk of aging, respectively. However, participants with excessively prolonged CUS (>2 hours) did not exhibit a significant reduction in the risk of aging (OR=1.06, 95% CI: 0.66–1.70), indicating that the protective effect of CUS on aging may disappear when the duration exceeds 2 hours.

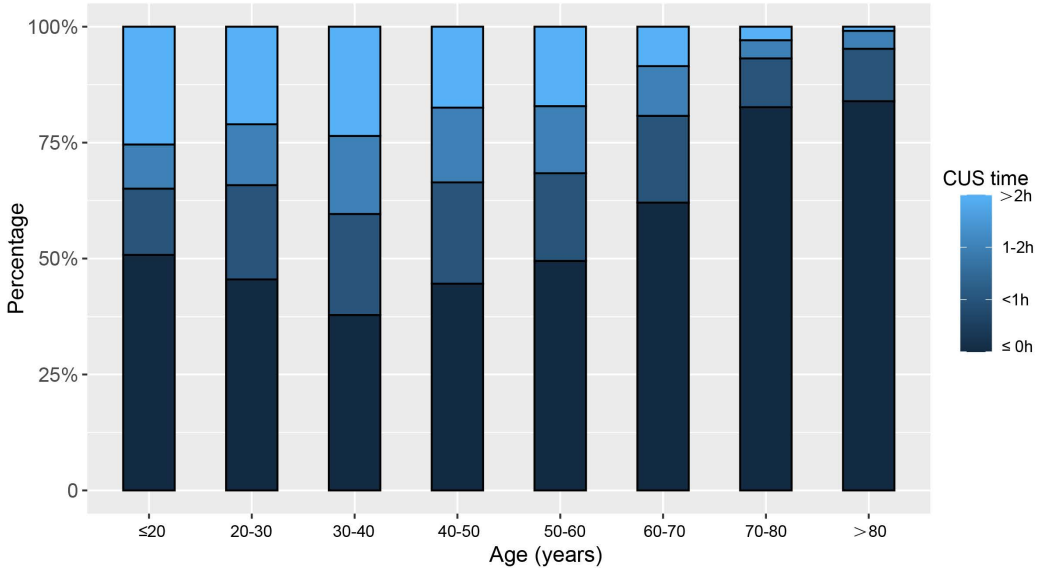


Fig 1. Percentages of CUS between different ages.

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Table 2. The relationship between weekend CUS and aging risk.

	Model 1		Model 2		Model 3	
Weekends CUS	**OR (95% CI)**	**P**	**OR (95% CI)**	**P**	**OR (95% CI)**	**P**
No	Ref.		Ref.		Ref.	
Yes	**0.79 (0.64, 0.97)**	**0.026***	**0.77 (0.62, 0.97)**	**0.032***	0.80 (0.63, 1)	0.051
Weekends CUS duration (h)						
≤0	Ref.		Ref.		Ref.	
0-1*	**0.71 (0.58, 0.88)**	**0.004***	**0.76 (0.60, 0.96)**	**0.016***	**0.77 (0.61, 0.96)**	**0.031***
1-2*	**0.72 (0.61, 0.85)**	**0.001***	**0.74 (0.60, 0.90)**	**0.004***	**0.80 (0.65, 0.98)**	**0.045***
>2	1 (0.67, 1.48)	0.990	0.99 (0.63, 1.56)	0.946	1.06 (0.66, 1.70)	0.751

*0h<Weekends CUS duration≤1h, 1h<Weekends CUS duration≤2h.

Model 1 was crude model.

Model 2 was adjusted age, sex, marital status, PIR, educational level, obesity.

Model 3 was adjusted age, sex, marital status, PIR, educational level, obesity, smoke, alcohol use, sleep trouble, PA, sleep duration in work day.

*indicates statistical significance.

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In addition to phenotypic age, we also explored the relationships between CUS and several indicators previously associated with cognitive aging (fasting blood glucose, HbA1c, Homeostatic model assessment of insulin resistance, insulin levels, low-density lipoprotein cholesterol, and CRP). CUS was negatively correlated with fasting blood glucose, HbA1c, and, but showed no association with insulin levels,low-density lipoprotein cholesterol, or CRP (S3 Fig in S1 File).

Bedtime and sleep duration in relation to CUS

We first investigated the relationship between bedtime and aging (S1 Table in S1 File). Compared to participants who sleep earlier than 00:00, participants who sleep later face a significantly increased risk of aging, both on weekdays (OR=1.41, 95% CI: 1.11–1.79) and on weekends (OR=1.21, 95% CI: 1.01–1.45). This suggests that delayed

bedtime—regardless of whether it occurs on weekdays or weekends—is associated with a higher risk of aging, with a more pronounced effect observed on weekdays (41% increased risk) compared to weekends (21% increased risk).

After stratifying by bedtime (Table 3), individuals who sleep early on weekdays and engage in CUS on weekends had a decreasing trend in aging risk (OR=0.73, 95% CI: 0.52–1.02). However, when further subdividing by duration, only those who both sleep early and have a CUS duration of 0–1 hours (OR=0.68, 95% CI: 0.48–0.96) and 1–2 hours (OR=0.62, 95% CI: 0.48–0.81) exhibited a reduced risk of aging, not those who sleep late ($OR_{0\text{-}1h}$ = 0.95, 95%CI: 0.55–1.62, $OR_{1\text{-}2h}$ = 1.01, 95%CI: 0.54–1.92, $OR_{>2h}$ = 0.81, 95%CI: 0.55–1.20). The protective effect of CUS on aging risk is conditional on a regular early bedtime; CUS does not appear to mitigate aging risk in individuals with delayed bedtime habits. Additionally, we investigated differences in weekend bedtime (S2 Table in S1 File). Similar to previous findings, only participants who sleep early on weekends and have a CUS duration of 0–1 hour were associated with a reduced risk of aging (OR=0.65, 95% CI: 0.45–0.95), corresponding to a 35% lower risk. This reinforces that early bedtime—whether on weekdays or weekends—may be a prerequisite for CUS to exert beneficial effects on aging. Interaction analysis showed a significant interaction between CUS and weekday sleep duration (p for interaction = 0.026). Furthermore, we combined bedtime and CUS duration to explore their relationship with aging (Fig 2). Compared to individuals who sleep early without CUS, only those who sleep early with a moderate amount of CUS (0–2 hours) showed a reduced risk of aging, while individuals with prolonged early sleep CUS (>2 hours) or late sleep did not exhibit a lower risk of aging.

Subsequently, we also observed the relationship between sleep duration and aging. RCS analysis revealed a U-shaped nonlinear relationship between sleep duration and the risk of aging (S4 Fig in S1 File). Compared to participants who sleep 7–8 hours, those who sleep more than 8 hours have a higher risk of aging (S3 Table in S1 File). The combined analysis shows that, compared to participants who sleep 7–8 hours and engage in catch-up sleep, those who sleep too little or too much without catch-up sleep have a significantly increased risk of aging (S4 Table in S1 File).

Compared to individuals without CUS, only those who usually sleep 7−8 hours exhibited a reduced risk of aging with CUS (OR=0.78, 95% CI: 0.58–0.96). However, for individuals with either excessively long sleep durations (OR=0.79, 95% CI: 0.48–1.30) or too short sleep durations (OR=1.23, 95% CI: 0.85–1.77), CUS did not improve their risk of aging. After further subdividing CUS duration, only individuals with usual sleep durations of 7−8 hours and CUS durations of 1−2 hours showed a reduced risk of aging (OR=0.47, 95% CI: 0.36–0.76) (Table 4). Mediation analysis showed that CUS mediated 14.5% of the relationship between sleep duration and aging, and 1.99% of the relationship between sleep timing and aging (S5 Fig in S1 File), so CUS may partially explain how sleep duration influences aging risk.

Table 3. The relationship between CUS and aging risk at different bedtime in workdays.

	Before 0:00 at workdays		After 0:00 at workdays	
Weekend CUS		**P**		**P**
No	Ref.		Ref.	
Yes	0.73 (0.52, 1.02)	0.060	0.93 (0.57, 1.52)	0.694
Weekend CUS duration (h)				
≤0	Ref.		Ref.	
0-1*	**0.68(0.48, 0.96)**	**0.036***	0.95(0.55, 1.62)	0.805
1-2*	**0.62(0.48, 0.81)**	**0.004***	1.01(0.54, 1.92)	0.958
>2	0.95(0.49, 1.87)	0.863	0.81(0.55, 1.20)	0.236

*0h<Weekend CUS duration≤1h, 1h<Weekend CUS duration≤2h.

Model was adjusted age, sex, marital status, PIR, educational level, obesity, smoke, alcohol use, sleep trouble, PA, sleep duration in work day.

*Indicates statistical significance.

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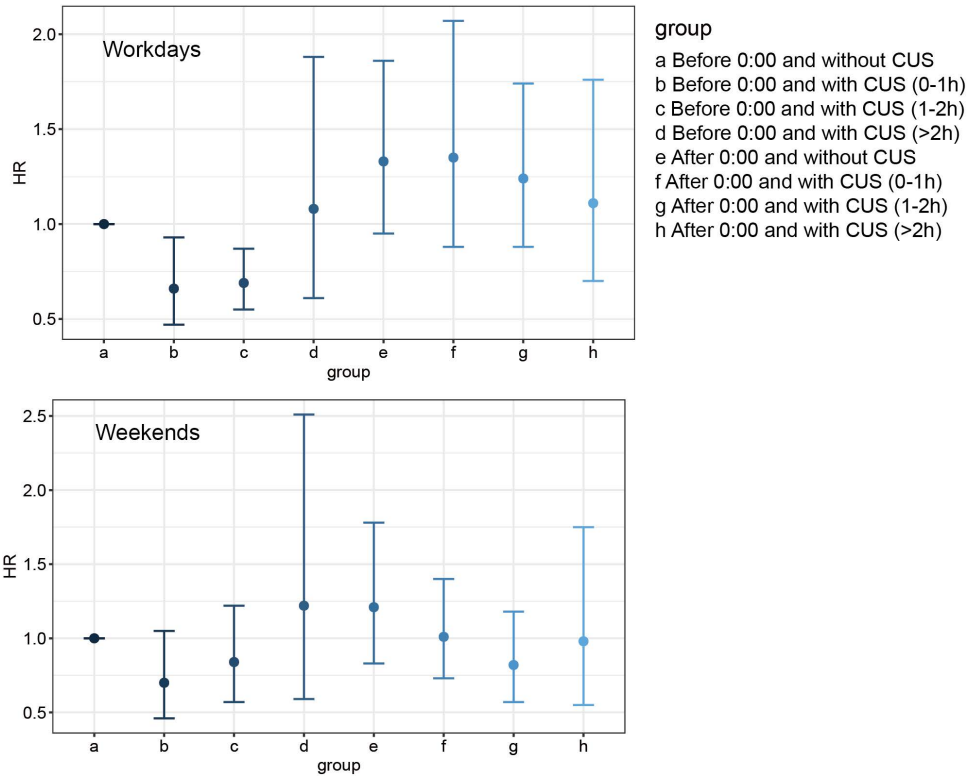


Fig 2. Joint analysis of CUS and different bedtime regarding the aging risk.

https://doi.org/10.1371/journal.pone.0332584.g002

Table 4. The relationship between CUS and aging risk at different sleep duration in workdays.

Sleep duration in workdays (h)	**<7**		**7-8**		**>8**	
Weekend CUS		**P**		**P**		**P**
No	Ref.		Ref.		Ref.	
Yes	0.81 (0.50, 1.32)	0.349	**0.75 (0.58, 0.96)**	**0.030***	0.79 (0.48, 1.30)	0.304
Weekend CUS duration (h)						
≤0	Ref.		Ref.			
0-1*	0.52 (0.27,1)	0.050	0.83 (0.63, 1.09)	0.144	0.72 (0.38, 1.37)	0.260
1-2*	0.99 (0.65, 1.51)	0.972	**0.47 (0.36, 0.76)**	**0.009***	0.81 (0.32, 2.06)	0.606
>2	0.73 (0.39, 1.38)	0.272	1.09 (0.53, 2.24)	0.778	1.20 (0.49, 2.95)	0.643

*0h<Weekends CUS duration≤1h, 1h<Weekends CUS duration≤2h.

Model was adjusted age, sex, marital status, PIR, educational level, obesity, smoke, alcohol use, sleep trouble, PA, sleep duration in work day.

*Indicates statistical significance.

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Subgroup analysis and sensitivity analysis

We conducted subgroup analyses based on the presence of sleep disorders and retirement status (S5 Table in S1 File). The results indicate that among individuals without sleep disorders, CUS reduced the risk of aging (OR=0.75, 95% CI: 0.56−1), and a CUS duration of 1−2 hours (OR=0.69, 95% CI: 0.49–0.96) was associated with the optimal reduction in the

risk of aging. In individuals who were not retired, a CUS duration of 1−2 hours (OR=0.71, 95% CI: 0.51–0.98) was also effective in lowering their risk of aging.

Considering significant differences in compensatory sleep patterns across different age groups, we further supplemented an analysis of age subgroups (S6 Table in S1 File). The results showed that compensatory sleep was associated with a reduced risk of aging in the subgroups of age < 30 (OR=0.63, 95%CI: 0.54–0.74), 30 ≤ Age < 40 (OR=0.67, 95%CI: 0.53–0.84), and 40 ≤ Age < 50 (OR=0.78, 95%CI: 0.62–0.97).

Sensitivity analysis indicated that after excluding participants taking sleep and psychiatric medications, the results remained robust (S7 Table in S1 File). Similar to the main findings, CUS continued to reduce the risk of aging in these participants (OR=0.77, 95% CI: 0.59–0.98), with a 23% lower risk, especially in those with CUS durations of 0−1 hour (OR=0.73, 95% CI: 0.55–0.95; 27% lower risk) and 1−2 hours (OR=0.69, 95% CI: 0.54–0.88; 31% lower risk). This confirms that the association between CUS and reduced aging risk is not confounded by sleep or psychiatric medications. Considering that some participants provided work schedules, we adjusted their work schedules and re-analyzed, or excluded some participants who worked night shifts. The results still showed that getting CUS within 2 hours had an effect on reducing the risk of aging, further supporting the stability of our findings.

Discussion

In this large cross-sectional study, we found that weekend CUS, especially when done in a reasonable manner (0–1 hour and 1–2 hours), can reduce the risk of aging in participants. However, excessively long CUS durations were not associated with improved aging. Further investigations revealed that CUS can improve the risk of aging in participants who usually have good sleep habits, including early bedtime (before 0:00) and normal sleep duration (7–8 hours). On the other hand, if participants usually experience sleep disorders, sleep late, or have abnormal sleep durations, weekend CUS was not associated with an improvement in their aging risk.

Although there is no direct evidence on the relationship between CUS and aging, some studies have investigated the relationship between sleep patterns and aging. A sleep index composed of sleep type, snoring, daytime sleepiness, sleep duration, insomnia, and difficulty waking up is negatively correlated with accelerated biological aging. This implies that the better the sleep quality, the lower the risk of aging for participants. The authors also found that good sleep quality can even offset the aging risk associated with air pollution [28]. Wang et al. also investigated the relationship between sleep parameters and biological age using the NHANES database. They found that greater sleep variability, more frequent irregular sleep patterns, and increased social jet lag were associated with an older biological age [29]. In addition, many studies have explored the relationship between Weekend CUS and components used to calculate biological age. Jang et al, after analyzing over 4,000 Koreans, reported that males with CUS of less than two hours on the weekends had a lower risk of abnormal blood lipid levels [30]. This is consistent with the findings of this study, suggesting that short periods of CUS may be more beneficial for the recovery of lipid metabolism, the authors also pointed out that normal sleep duration could reduce the risk of abnormal blood lipid levels. For another component, CRP, similar results were found. Han et al indicated a significant association between Weekend CUS and low-sensitivity CRP. Both longer and shorter durations of CUS were found to be unrelated to elevated CRP levels [31]. Another study specifically investigated the relationship between Weekend CUS and metabolic disorders, in addition to hypertension, Weekend CUS was found to be independently associated with obesity, type 2 diabetes, and high cholesterol levels [32]. In summary, these diseases are closely related to aging, and to some extent, they align with our study findings. However, some results differ from previous research. A study systematically investigated the relationship between CUS patterns and factors such as energy intake and insulin. They found that while there are benefits to Weekend CUS, such as restoring insulin sensitivity in the liver and muscles, this effect is short-lived, and irregular sleep following CUS can lead to delayed energy intake and weight gain [33]. The reasons for these differences may be attributed to variations in the definition of the reference group and differences in sample size and observation time in the study populations.

Interestingly, CUS exhibits the strongest protective effect against aging in participants who sleep 7–8 hours on workdays, suggesting that a regular lifestyle and periodic relief from work stress may contribute to a lower risk of aging. Sleep homeostasis may merely be a reflection of social stress [34]. Pan et al. demonstrated that stressful life events are significant factors leading to poor sleep quality, and interventions to reduce stress can effectively address sleep disorders [35]. This implies that maintaining a regular lifestyle and managing social stress may be the fundamental reasons for resisting aging.

From a mechanistic perspective, the relationship between irregular sleep, CUS, and aging is traceable. Sleep deprivation activates inflammatory signaling pathways, involving nuclear factor-κB, activating protein 1, and the signal transducer and activator of transcription family of proteins, which increases mRNA levels encoding pro-inflammatory cytokines [36,37]. Inflammation exacerbates cellular senescence, mitochondrial dysfunction, and DNA damage, consequently accelerating the aging process and increasing the phenotypic age [38]. On the other hand, circadian rhythm changes are also an important factor in explaining the relationship between CUS and aging. A regular circadian rhythm is a crucial condition for maintaining biological adaptation and adapting to the environment. Animal experiments indicate that mismatch between the internal biological clock and daily environmental changes is detrimental to survival [39]. The decline in aging and circadian rhythm function is mutually associated and mutually reinforcing. Normal aging is also accompanied by the decline in the function of the suprachiasmatic nucleus, DNA replication and hormonal changes, leading to disturbances in circadian rhythm [40,41]. This is because the peak of oxidation occurs during the day, followed by an increase in reactive oxygen species levels, while the peak of DNA replication happens at night. Normal circadian rhythm temporarily isolates these two processes to ensure that DNA replication occurs when ROS levels are at their lowest [42,43]. This also explains the reasons for the increased risk of aging in individuals with excessively long or short sleep durations in this study, and why the risk of aging does not continuously decrease in individuals with excessively long CUS durations.

Certainly, there are some limitations to our study. Firstly, it is a cross-sectional study, the uncertainty of causation is inevitable [44,45]. We cannot accurately determine whether CUS leads to aging or whether the biomarkers used in the definition of biological age affect sleep quality, forcing participants to get extra sleep. Secondly, participants' sleep information relies on self-reports rather than electronic device collection. However, self-reporting has the advantages of convenience and wide applicability, making it suitable for large-scale population studies like ours. NHANES lacks information on participants living with children or elderly people, and taking care of children or elderly people with limited mobility often leads to irregular sleep patterns [46,47]. Additionally, sleep disorders in the sensitivity analysis were only based on physician diagnosis, and no specific types of sleep disorders (such as OSA) were provided. Finally, the impact of some potential confounding factors, such as dietary patterns and exposure to toxins, should also be considered.

Conclusion

This large cross-sectional study suggests that, compared to participants without weekend CUS, having 0–2 hours of CUS on weekends contributes to a reduced risk of aging. Interestingly, the anti-aging benefits of CUS are more pronounced in individuals who habitually go to bed early (before 0:00) and have a normal sleep duration (7–8 hours). However, for those with irregular sleep patterns during weekdays, the anti-aging effects of CUS appear to be minimal. These findings provide new insights into anti-aging strategies and sleep management.

Supporting information

S1 File. Supplementary method, figures and tables.
(DOCX)

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References

1. Di H, Guo Y, Daghlas I. Evaluation of sleep habits and disturbances among US adults, 2017-2020. JAMA Netw Open. 2022;5:e2240788. https://doi.org/10.1001/jamanetworkopen.2022.40788
2. Li J, Cao D, Huang Y, Chen Z, Wang R, Dong Q. Sleep duration and health outcomes: an umbrella review. Sleep Breath. 2022;26(3):1479–501. https://doi.org/10.1007/s11325-021-02458-1
3. Li Y, Sahakian BJ, Kang J, Langley C, Zhang W, Xie C, et al. The brain structure and genetic mechanisms underlying the nonlinear association between sleep duration, cognition and mental health. Nat Aging. 2022;2(5):425–37. https://doi.org/10.1038/s43587-022-00210-2 PMID: 37118065
4. Li X, Xue Q, Wang M, et al. Adherence to a healthy sleep pattern and incident heart failure: a prospective study of 408 802 UK Biobank participants. Circulation. 2021;143:97–9. https://doi.org/10.1161/CIRCULATIONAHA.120.050792
5. Li W, Li C, Liu T, Wang Y, Ma X, Xiao X, et al. Self-reported sleep disorders and the risk of all cancer types: evidence from the Kailuan Cohort study. Public Health. 2023;223:209–16. https://doi.org/10.1016/j.puhe.2023.08.007 PMID: 37677850
6. Cai Y, Zhaoxiong Y, Zhu W, Wang H. Association between sleep duration, depression and breast cancer in the United States: a national health and nutrition examination survey analysis 2009-2018. Ann Med. 2024;56(1):2314235. https://doi.org/10.1080/07853890.2024.2314235 PMID: 38329808
7. Kim KM, Han SM, Min IK, Heo K, Kim W-J, Chu MK. Weekend catch-up sleep and depression: results from a nationally representative sample in Korea. Sleep Med. 2021;87:62–8. https://doi.org/10.1016/j.sleep.2021.02.058 PMID: 34520972
8. Meng R, Cao Y, Kong Y, Wang K, Yang Z, Jia Y, et al. Effects of circadian rhythm disorder on body composition in women aged 31-40 years. Ann Palliat Med. 2021;10(1):340–9. https://doi.org/10.21037/apm-20-2267 PMID: 33545769
9. Dos Santos M, Ferrari G, Lee DH, Rey-López JP, Aune D, Liao B, et al. Association of the "weekend warrior" and other leisure-time physical activity patterns with all-cause and cause-specific mortality: a nationwide cohort study. JAMA Intern Med. 2022;182(8):840–8. https://doi.org/10.1001/jamainternmed.2022.2488 PMID: 35788615
10. Liang J-H, Huang S, Pu Y-Q, Zhao Y, Chen Y-C, Jiang N, et al. Whether weekend warrior activity and other leisure-time physical activity pattern reduce the risk of depression symptom in the representative adults? A population-based analysis of NHANES 2007-2020. J Affect Disord. 2023;340:329–39. https://doi.org/10.1016/j.jad.2023.07.113 PMID: 37543116
11. Oh J, Kim E, Huh I. Associations between weekend catch-up sleep and health-related quality of life with focusing on gender differences. Sci Rep. 2023;13(1):20280. https://doi.org/10.1038/s41598-023-47244-z PMID: 37985799
12. 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. https://doi.org/10.1016/j.jad.2024.05.022
13. Chen S, Zhang T, Gao H, Zhang J. Association between weekend catch-up sleep and chronic kidney disease: insights from NHANES 2017-2020. Ren Fail. 2025;47(1):2461682. https://doi.org/10.1080/0886022X.2025.2461682 PMID: 39910840
14. López-Otín C, Blasco MA, Partridge L, Serrano M, Kroemer G. Hallmarks of aging: an expanding universe. Cell. 2023;186(2):243–78. https://doi.org/10.1016/j.cell.2022.11.001 PMID: 36599349
15. Cai Y, Song W, Li J, Jing Y, Liang C, Zhang L, et al. The landscape of aging. Sci China Life Sci. 2022;65(12):2354–454. https://doi.org/10.1007/s11427-022-2161-3 PMID: 36066811

16. Smagula SF, Zhang G, Gujral S, Covassin N, Li J, Taylor WD, et al. Association of 24-hour activity pattern phenotypes with depression symptoms and cognitive performance in aging. JAMA Psychiatry. 2022;79(10):1023–31. https://doi.org/10.1001/jamapsychiatry.2022.2573 PMID: 36044201
17. Liu Z, Kuo P-L, Horvath S, Crimmins E, Ferrucci L, Levine M. A new aging measure captures morbidity and mortality risk across diverse subpopulations from NHANES IV: a cohort study. PLoS Med. 2018;15(12):e1002718. https://doi.org/10.1371/journal.pmed.1002718 PMID: 30596641
18. Tian L, Ding P, Kuang X, Ai W, Shi H. The association between sleep duration trajectories and successful aging: a population-based cohort study. BMC Public Health. 2024;24(1):3029. https://doi.org/10.1186/s12889-024-20524-7 PMID: 39482676
19. Winer JR, Deters KD, Kennedy G, Jin M, Goldstein-Piekarski A, Poston KL, et al. Association of short and long sleep duration with amyloid-β burden and cognition in aging. JAMA Neurol. 2021;78(10):1187–96. https://doi.org/10.1001/jamaneurol.2021.2876 PMID: 34459862
20. Liu C-A, Liu T, Ge Y-Z, Song M-M, Ruan G-T, Lin S-Q, et al. Muscle distribution in relation to all-cause and cause-specific mortality in young and middle-aged adults. J Transl Med. 2023;21(1):154. https://doi.org/10.1186/s12967-023-04008-7 PMID: 36841788
21. Heymsfield SB, Smith B, Chung EA, Watts KL, Gonzalez MC, Yang S, et al. Phenotypic differences between people varying in muscularity. J Cachexia Sarcopenia Muscle. 2022;13(2):1100–12. https://doi.org/10.1002/jcsm.12959 PMID: 35170220
22. Kwon D, Belsky DW. A toolkit for quantification of biological age from blood chemistry and organ function test data: BioAge. Geroscience. 2021;43(6):2795–808. https://doi.org/10.1007/s11357-021-00480-5 PMID: 34725754
23. Roenneberg T, Wirz-Justice A, Merrow M. Life between clocks: daily temporal patterns of human chronotypes. J Biol Rhythms. 2003;18(1):80–90. https://doi.org/10.1177/0748730402239679 PMID: 12568247
24. Chen Y-H, Lyu Z-Y, Wang G, Feng X-S, Xie S-H, Chen S-H, et al. Relationship of sleep duration and annual changes in sleep duration with the incidence of gastrointestinal cancers: a prospective cohort study. Chin Med J (Engl). 2021;134(24):2976–84. https://doi.org/10.1097/CM9.0000000000001770 PMID: 34839316
25. Shan Z, Rehm CD, Rogers G. Trends in dietary carbohydrate, protein, and fat intake and diet quality among US adults, 1999-2016. JAMA. 2019;322:1178–87. https://doi.org/10.1001/jama.2019.13771
26. Liang J, Huang S, Jiang N, Kakaer A, Chen Y, Liu M, et al. Association between joint physical activity and dietary quality and lower risk of depression symptoms in US adults: cross-sectional NHANES study. JMIR Public Health Surveill. 2023;9:e45776. https://doi.org/10.2196/45776 PMID: 37163324
27. Liu C-A, Liu T, Ruan G-T, Ge Y-Z, Song M-M, Xie H-L, et al. The relationship between fat distribution in central region and comorbidities in obese people: Based on NHANES 2011-2018. Front Endocrinol (Lausanne). 2023;14:1114963. https://doi.org/10.3389/fendo.2023.1114963 PMID: 36843589
28. Gao X, Huang N, Guo X, Huang T. Role of sleep quality in the acceleration of biological aging and its potential for preventive interaction on air pollution insults: Findings from the UK Biobank cohort. Aging Cell. 2022;21(5):e13610. https://doi.org/10.1111/acel.13610 PMID: 35421261
29. Wang X, Xu Y, Li X, Mansuri A, McCall WV, Liu Y, et al. Day-to-day deviations in sleep parameters and biological aging: Findings from the NHANES 2011-2014. Sleep Health. 2023;9(6):940–6. https://doi.org/10.1016/j.sleh.2023.07.018 PMID: 37648648
30. Jang YS, Park YS, Hurh K, Park E-C, Jang S-I. Association between weekend catch-up sleep and dyslipidemia among Korean workers. Sci Rep. 2023;13(1):925. https://doi.org/10.1038/s41598-023-28142-w PMID: 36650276
31. Han K-M, Lee H-J, Kim L, Yoon H-K. Association between weekend catch-up sleep and high-sensitivity C-reactive protein levels in adults: a population-based study. Sleep. 2020;43(8):zsaa010. https://doi.org/10.1093/sleep/zsaa010 PMID: 32006432
32. Kim JJ, Hwang IC. Weekend catch-up sleep is associated with reduced metabolic derangements in Korean adults. Neurol Sci. 2021;42(2):735–7. https://doi.org/10.1007/s10072-020-04805-8 PMID: 33044669
33. 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.e4. https://doi.org/10.1016/j.cub.2019.01.069 PMID: 30827911
34. Loftus JC, Harel R, Núñez CL, Crofoot MC. Ecological and social pressures interfere with homeostatic sleep regulation in the wild. Elife. 2022;11:e73695. https://doi.org/10.7554/eLife.73695 PMID: 35229719
35. Pan Z, Zhang D. Relationship between stressful life events and sleep quality: the mediating and moderating role of psychological suzhi. Sleep Med. 2022;96:28–34. https://doi.org/10.1016/j.sleep.2022.04.026 PMID: 35576831
36. Irwin MR, Wang M, Ribeiro D, Cho HJ, Olmstead R, Breen EC, et al. Sleep loss activates cellular inflammatory signaling. Biol Psychiatry. 2008;64(6):538–40. https://doi.org/10.1016/j.biopsych.2008.05.004 PMID: 18561896
37. Irwin MR. Sleep and inflammation: partners in sickness and in health. Nat Rev Immunol. 2019;19(11):702–15. https://doi.org/10.1038/s41577-019-0190-z PMID: 31289370
38. Walker KA, Basisty N, Wilson DM, Ferrucci L. Connecting aging biology and inflammation in the omics era. J Clin Invest. 2022;132(14):e158448. https://doi.org/10.1172/JCI158448
39. Acosta-Rodríguez VA, Rijo-Ferreira F, Green CB, Takahashi JS. Importance of circadian timing for aging and longevity. Nat Commun. 2021;12(1):2862. https://doi.org/10.1038/s41467-021-22922-6 PMID: 34001884
40. Blacher E, Tsai C, Litichevskiy L. Aging disrupts circadian gene regulation and function in macrophages. Nat Immunol. 2022;23:229–36.

41. Amorim JA, Coppotelli G, Rolo AP, Palmeira CM, Ross JM, Sinclair DA. Mitochondrial and metabolic dysfunction in ageing and age-related diseases. Nat Rev Endocrinol. 2022;18(4):243–58. https://doi.org/10.1038/s41574-021-00626-7 PMID: 35145250

42. Welz P-S, Benitah SA. Molecular connections between circadian clocks and aging. J Mol Biol. 2020;432(12):3661–79. https://doi.org/10.1016/j.jmb.2019.12.036 PMID: 31887285

43. Fagiani F, Di Marino D, Romagnoli A, Travelli C, Voltan D, Di Cesare Mannelli L, et al. Molecular regulations of circadian rhythm and implications for physiology and diseases. Signal Transduct Target Ther. 2022;7(1):41. https://doi.org/10.1038/s41392-022-00899-y PMID: 35136018

44. Moskalev A, Guvatova Z, Lopes IDA, Beckett CW, Kennedy BK, De Magalhaes JP, et al. Targeting aging mechanisms: pharmacological perspectives. Trends Endocrinol Metab. 2022;33(4):266–80. https://doi.org/10.1016/j.tem.2022.01.007 PMID: 35183431

45. Xu F, Earp JE, Adami A, Weidauer L, Greene GW. The relationship of physical activity and dietary quality and diabetes prevalence in US adults: findings from NHANES 2011-2018. Nutrients. 2022;14(16):3324. https://doi.org/10.3390/nu14163324 PMID: 36014830

46. Bourke-Taylor H, Pallant JF, Law M, Howie L. Relationships between sleep disruptions, health and care responsibilities among mothers of school-aged children with disabilities. J Paediatr Child Health. 2013;49(9):775–82. https://doi.org/10.1111/jpc.12254 PMID: 23745960

47. Byars KC, Chini B, Hente E, Amin R, Boat T. Sleep disturbance and sleep insufficiency in primary caregivers and their children with cystic fibrosis. J Cyst Fibros. 2020;19(5):777–82. https://doi.org/10.1016/j.jcf.2020.04.003 PMID: 32461045