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Regulating Effect of Weekend Catch-up Sleep on Association of Hepatic Steatosis with Atherosclerotic Cardiovascular Disease

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Background: Both insufficient and excessive catch-up sleep durations have been implicated in influencing the risk of cardiovascular diseases (CVDs) and metabolic disorders. However, the specific impact of weekend catch-up sleep (WCS) on the relationship between hepatic steatosis (HS) and atherosclerotic cardiovascular disease (ASCVD) remains unclear.

Aims: This cross-sectional study aims to examine the potential regulatory effect of WCS on the association between controlled attenuation parameter (CAP) and ASCVD.

Methods: Weighted logistic regression analyses were employed to evaluate the associations of CAP and WCS with ASCVD, expressed in terms of odds ratios (ORs) and 95% confidence intervals (CIs). The study also explored the effect of WCS on the CAP-ASCVD relationship and assessed the potential regulatory role of WCS in subgroups based on age, gender, body mass index, and obesity status.

Results: Eligible participants were categorized into two groups: those with an ASCVD risk < 7.5% (n = 1536) and those with an ASCVD risk ≥ 7.5% (n = 1612). After adjusting for covariates, CAP ≥ 274 dB/m was associated with a higher likelihood of ASCVD compared to the CAP < 274 dB/m (OR, 1.84, 95% CI, 1.24-2.73). When compared to a WCS duration of 0-1 hour, WCS ≥ 1 hour was found to increase the potential ASCVD risk associated with CAP (OR, 3.29, 95% CI, 1.41-7.68). Furthermore, among individuals with WCS ≥ 1 hour, CAP ≥ 274 dB/m was linked to a higher ASCVD risk than among those with CAP < 274 dB/m (OR, 3.72, 95% CI, 1.99-6.93). Additionally, in subgroups of participants aged ≥ 60 years, females, non-obese and obese individuals, WCS ≥ 1 hour was associated with an increased ASCVD risk related to CAP (all, $p < 0.05$).

Conclusion: A WCS duration of ≥ 1 hour may be associated with an increased ASCVD risk in adults with HS aged ≥ 40 years. However, further research is necessary to clarify the causal relationships between WCS, HS, and ASCVD.

INTRODUCTION

Hepatic steatosis (HS) continues to be highly prevalent globally, imposing a significant disease burden.[1,2] In addition to hepatic complications like cirrhosis and hepatocellular carcinoma, cardiovascular diseases (CVDs), particularly atherosclerotic cardiovascular disease (ASCVD), represent critical adverse outcomes in HS patients, potentially linked to metabolic aberrations associated with HS.[3-6] Recent evidence indicates that both shortened and prolonged sleep durations may influence CVD risk and associated risk factors, including metabolic syndrome and arterial stiffness.[7-10] Specifically, in patients with non-alcoholic fatty liver disease (NAFLD), reduced sleep duration and compromised sleep quality have been correlated with increased arterial stiffness.[11] Additionally, the duration of catch-up sleep has garnered increasing research attention. Data suggest that extending sleep, termed catch-up sleep, among sleep-deprived individuals may mitigate CVD and metabolic disease risk.[12] For instance, moderate durations of catch-up sleep (e.g., ≤ 2 hours) have been associated with decreased risks of metabolic syndrome, hypertension, and dyslipidemia.[13-16] Conversely, a recent study highlighted that individuals with catch-up sleep durations of less than 1 hour exhibited higher all-cause mortality risks related to hypertension compared to those with durations exceeding 1 hour.[17] However, excessive catch-up sleep, also referred to as sleep insufficiency, may detrimentally impact cardiovascular health by increasing the risk of obesity and hypertension, and it correlates with reduced overall cardiovascular health metrics.[18] Furthermore, in patients with diabetes mellitus (DM), extended catch-up sleep has been linked to heightened risks of obesity and insulin resistance.[19] To date, the impact of catch-up sleep duration on ASCVD risk in the context of HS remains unexplored. This study aims to examine the influence of weekend catch-up sleep (WCS) on the association between HS and ASCVD, offering insights for the prevention of ASCVD related to HS.

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MATERIALS AND METHODS

Study population

This cross-sectional study utilized data from the National Health and Nutrition Examination Survey (NHANES) database spanning 2017-2020. NHANES is a nationally representative program designed to assess the health and nutritional status of the U.S. population through a complex, multistage stratified probability sampling method. The survey collects data from approximately 5,000 individuals biennially, covering 15 geographic regions. Detailed methodology is available at: NHANES Documentation. Ethical approval and informed consent requirements for this study were waived by the Institutional Review Board (IRB) due to the public availability of de-identified NHANES data.

Adults aged ≥ 40 years, with available data on liver ultrasound transient elastography, weekday/weekend sleep duration, and ASCVD risk calculation were included. Exclusion criteria included: (1) history of CVD, (2) hepatitis B or C virus infection, and (3) ≥ 8 drinks per week for women and ≥ 15 for men, per NHANES guidelines. The NHANES database has been ethically approved by relevant IRBs, and ethical approval for this secondary analysis was waived by our institution's IRB.

Definition of hepatic steatosis

HS was defined using the controlled attenuation parameter (CAP), with a cutoff value of ≥ 274 dB/m, as established in prior studies.[20]

Calculation of weekend catch-up sleep

WCS duration was calculated using NHANES questionnaire responses to two questions: (1) "Number of hours usually slept on weekdays/workdays" and (2) "Number of hours usually slept on weekends/non-workdays." WCS was defined as the difference between weekend and weekday sleep durations. Normal WCS was categorized as ≤ 1 hour, and participants were stratified into three groups: < 0 hours, 0-1 hour, and ≥ 1 hour.

Assessment of 10-year ASCVD risk

ASCVD risk was calculated using the 2013 American College of Cardiology/American Heart Association Pooled Cohort Equations,[24] which predict the 10-year risk of stroke, coronary artery disease, or non-fatal myocardial infarction in adults aged 40-79 years with low-density lipoprotein < 190 mg/dl and no prior ASCVD events. The equation incorporates age, sex, race, high-density lipoprotein cholesterol, total cholesterol, systolic blood pressure, antihypertensive medication use, smoking status, and DM. ASCVD risk was dichotomized into < 7.5% and ≥ 7.5% based on prior studies.[25]

Variables selection

Potential confounding variables extracted from the NHANES database include variables such as age, sex, race, education level, marital status, poverty-to-income ratio (PIR), smoking, alcohol consumption, physical activity, antidiabetic medication, dyslipidemia treatment, chronic kidney disease (CKD), family history of myocardial infarction, body mass index (BMI), total energy intake, liver stiffness measurement, alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), C-reactive protein, and uric acid (UA).

Physical activity data were converted to weekly energy expenditure (MET·min/week) using standardized formulas and categorized by a 450 MET·min/week threshold. BMI was classified as non-obesity (< 30 kg/m^2) or obesity (≥ 30 kg/m^2) per World Health Organization criteria. Smoking status was categorized as non-smoker, current smoker, or former smoker based on self-reported data. Alcohol consumption was defined as < 1 time/week or ≥ 1 time/week. CKD was diagnosed as estimated glomerular filtration rate < 60 ml/min/1.73 m^2 or UACR ≥ 30 mg/g.[27] Dietary intake was assessed via two 24-hour recalls, and total energy intake was calculated from food and supplement consumption. Median liver stiffness was dichotomized at 8.0 kPa.

Statistical analysis

Continuous variables were expressed as mean ± standard error, while categorical variables were described using frequencies and percentages (n, %). Comparisons between the low and high ASCVD risk groups were conducted using weighted t-tests for continuous variables and chi-square tests (χ^2) for categorical variables, adhering to NHANES guidelines for incorporating the full-sample MEC examination weights.

Weighted univariate logistic regression analyses were employed to identify covariates associated with ASCVD. Subsequent weighted multivariate logistic regression models were developed to evaluate the associations between CAP, WCS, and ASCVD. Model 1 included adjustments for demographic variables (age, sex, education level, PIR, and marital status), whereas model 2 incorporated all selected covariates, including smoking, antidiabetic medication, dyslipidemia treatment, CKD, BMI, median liver stiffness, ALP, GGT, and UA. The results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs), considering a two-sided p value < 0.05 as statistically significant. Additionally, the study examined the effect of WCS on the CAP-ASCVD relationship and assessed the potential moderating role of WCS in age, gender, and BMI obesity subgroups. All analyses were performed using R version 4.2.3 (Institute for Statistics and Mathematics, Vienna, Austria). Missing data were addressed through random forest interpolation, with sensitivity analyses conducted to compare variables before and after imputation (Supplementary Table 1).

RESULTS

Participant characteristics

The study flowchart (Figure 1) illustrates the participant selection process. Initially, 5,135 adults aged ≥ 40 years with liver ultrasound transient elastography data were included. After excluding those lacking sleep duration information (n = 69), ASCVD risk data (n = 686), or with a history of CVD (n = 646), hepatitis B/C infection (n = 123), or significant alcohol use (n = 463), a final sample of 3,148 participants was obtained.

TABLE 1. Characteristics of Participants Between low ASCVD Risk Group and High ASCVD Risk Group.

Variables	Total, (n = 3148)	ASCVD risk < 7.5%, (n = 1536)	ASCVD risk ≥ 7.5%, (n = 1612)	Statistics	*p*
Age, years, mean (S.E.)	57.57 (0.42)	50.70 (0.37)	65.87 (0.42)	t = -28.56	**< 0.001**
Gender, n (%)				χ^2 = 65.95	**< 0.001**
Male	1385 (43.15)	491 (33.80)	894 (54.44)		
Female	1763 (56.85)	1045 (66.20)	718 (45.56)		
Race, n (%)				χ^2 = 1.58	0.454
Non-hispanic white	1091 (67.32)	487 (66.16)	604 (68.72)		
Non-hispanic black	807 (9.73)	393 (9.83)	414 (9.62)		
Others	1250 (22.95)	656 (24.01)	594 (21.66)		
Education level, n (%)				χ^2 = 29.96	**<0.001**
Under high school	534 (9.57)	214 (7.91)	320 (11.57)		
High school	703 (24.90)	305 (20.97)	398 (29.64)		
Graduate and above	1911 (65.54)	1017 (71.12)	894 (58.80)		
PIR, n (%)				χ^2 = 18.29	**< 0.001**
≤ 1.3	690 (12.40)	314 (11.51)	376 (13.47)		
1.3-3.5	1204 (31.77)	547 (27.43)	657 (37.01)		
> 3.5	1254 (55.83)	675 (61.05)	579 (49.51)		
Marital status, n (%)				χ^2 = 9.52	**0.002**
Married	2032 (71.24)	1050 (74.94)	982 (66.76)		
Others	1116 (28.76)	486 (25.06)	630 (33.24)		
Smoking, n (%)				χ^2 = 56.43	**< 0.001**
Non-smoking	2009 (62.45)	1174 (72.19)	835 (50.68)		
Quit smoking	763 (27.24)	202 (17.93)	561 (38.49)		
Current smoking	376 (10.30)	160 (9.87)	216 (10.83)		
Drinking, time/week, n (%)				χ^2 = 1.03	0.309
< 1	2303 (67.88)	1136 (66.35)	1167 (69.73)		
≥ 1	845 (32.12)	400 (33.65)	445 (30.27)		
Physical activity, MET·min/week, n (%)				χ^2 = 3.30	0.192
< 450	321 (9.82)	144 (9.25)	177 (10.50)		
≥ 450	2002 (69.55)	1033 (71.88)	969 (66.74)		
Unknown	825 (20.63)	359 (18.87)	466 (22.76)		
Antidiabetic drug, n (%)				χ^2 = 71.94	**< 0.001**
No	2610 (86.33)	1419 (93.74)	1191 (77.39)		
Yes	538 (13.67)	117 (6.26)	421 (22.61)		
Drug for dyslipidemia, n (%)				χ^2 = 146.27	**< 0.001**
No	1936 (64.14)	1210 (80.54)	726 (44.34)		
Yes	1212 (35.86)	326 (19.46)	886 (55.66)		
CKD, n (%)				χ^2 = 31.27	**< 0.001**
No	2634 (86.92)	1403 (92.73)	1231 (79.89)		
Yes	514 (13.08)	133 (7.27)	381 (20.11)		
Family history of myocardial infarction, n (%)				χ^2 = 1.77	0.184
No	2777 (85.73)	1352 (86.91)	1425 (84.30)		

TABLE 1. Continued

Variables	Total, (n=3148)	ASCVD risk < 7.5%, (n=1536)	ASCVD risk ≥ 7.5%, (n=1612)	Statistics	*p*
Yes	371 (14.27)	184 (13.09)	187 (15.70)		
BMI, kg/m², Mean (S.E.)	29.72 (0.19)	29.49 (0.30)	29.99 (0.20)	t = -1.46	0.156
BMI obesity, kg/m², n (%)				χ^2 = 4.17	**0.041**
< 30	1814 (59.08)	884 (61.07)	930 (56.68)		
≥ 30	1334 (40.92)	652 (38.93)	682 (43.32)		
Total energy intake, kcal, mean (S.E.)	2098.12 (21.67)	2104.40 (38.70)	2090.53 (29.59)	t = 0.25	0.803
Median liver stiffness, kPa, n (%)				χ^2 = 12.41	**< 0.001**
< 8.0	2822 (91.13)	1421 (93.75)	1401 (87.97)		
≥ 8.0	326 (8.87)	115 (6.25)	211 (12.03)		
ALT, U/l, mean (S.E.)	21.97 (0.22)	22.48 (0.38)	21.35 (0.41)	t = 1.74	0.094
AST, U/l, mean (S.E.)	21.15 (0.22)	21.25 (0.37)	21.03 (0.30)	t = 0.44	0.661
ALP, IU/l, mean (S.E.)	75.41 (0.62)	73.56 (0.75)	77.65 (0.89)	t = -3.77	**< 0.001**
GGT, IU/l, mean (S.E.)	28.13 (0.61)	26.82 (0.81)	29.70 (0.93)	t = -2.33	**0.028**
CRP, mg/l, mean (S.E.)	3.61 (0.20)	3.41 (0.17)	3.86 (0.36)	t = -1.17	0.255
UA, mg/dl, mean (S.E.)	5.31 (0.04)	5.06 (0.05)	5.61 (0.05)	t = -7.67	**< 0.001**
CAP, dB/m, mean (S.E.)	271.06 (1.71)	262.31 (2.17)	281.64 (2.16)	t = -7.09	**< 0.001**
CAP, dB/m, n (%)				χ^2 = 29.86	**< 0.001**
< 274	1615 (52.53)	859 (58.52)	756 (45.30)		
≥ 274	1533 (47.47)	677 (41.48)	856 (54.70)		
WCS, hours, mean (S.E.)	0.68 (0.04)	0.86 (0.06)	0.47 (0.04)	t = 5.79	**< 0.001**
WCS duration, hours, n (%)				χ^2 = 51.52	**< 0.001**
(0, 1)	1529 (49.17)	586 (40.91)	943 (59.14)		
< 0	421 (11.84)	202 (11.45)	219 (12.32)		
≥ 1	1198 (38.99)	748 (47.65)	450 (28.53)		

t, t-test; χ^2, chi-square test; ASCVD, atherosclerotic cardiovascular disease, S.E., standard error; PIR, poverty-to-income ratio; CKD, chronic kidney disease; BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; CRP: C-reactive protein; UA, uric acid; CAP, controlled attenuation parameter; WCS, weekend catch-up sleep.

Table 1 presents the baseline characteristics of participants stratified by ASCVD risk (< 7.5% vs. ≥ 7.5%). The mean age of the cohort was 57.57 years, with a majority being female (56.85%). Most participants exhibited median liver stiffness < 8.0 kPa (91.13%). Significant differences were observed between the two groups, with the low ASCVD risk group having lower CAP values (262.31 dB/m vs. 281.64 dB/m) and higher WCS durations (0.86 hours vs. 0.47 hours) compared to the high ASCVD risk group.

Associations of CAP and WCS with ASCVD

Prior to analyzing the associations between CAP, WCS, and ASCVD, covariates related to ASCVD risk were identified through weighted univariate logistic regression (Supplementary Table 2). Variables such as age, gender, education level, PIR, marital status, smoking, antidiabetic medication, dyslipidemia treatment, CKD, median liver stiffness, ALP, GGT, and UA were significantly associated with ASCVD (all, $p < 0.05$). After adjusting for these covariates, individuals with CAP ≥ 274 dB/m demonstrated a higher likelihood of ASCVD (OR, 1.84, 95% CI, 1.24-2.73) compared to those with CAP < 274 dB/m. However, no significant association was found between WCS duration and ASCVD risk (all, $p > 0.05$) (Table 2).

Moderating effect of WCS on CAP-ASCVD association

Table 3 highlights the moderating effect of WCS on the CAP-ASCVD relationship. After adjusting for covariates, individuals with WCS ≥ 1 hour exhibited a stronger association between CAP and ASCVD risk compared to those with WCS durations of 0-1 hour (OR, 3.29, 95% CI, 1.41-7.68). This interaction was visually represented in Figure 2. Further analysis under varying WCS conditions (Table 4) confirmed that CAP ≥ 274 dB/m was associated with higher ASCVD odds in individuals with WCS ≥ 1 hour (OR, 3.72, 95% CI, 1.99-6.93).

Subgroup analyses

Subgroup analyses (Table 5) revealed that individuals aged ≥ 60 years or females who had WCS ≥ 1 hour had lower ASCVD odds compared to those with WCS durations of 0-1 hour. Conversely, in

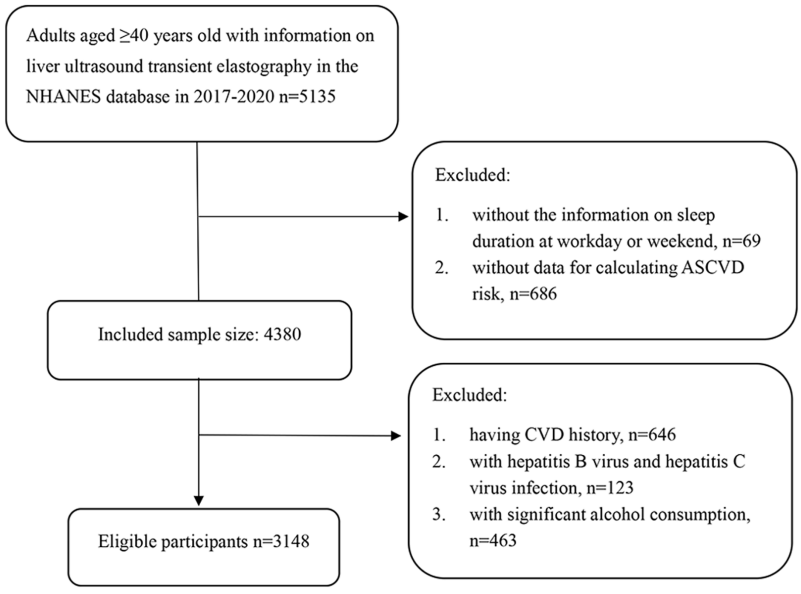

FIG. 1. Flowchart of study process.
NHANES, National Health and Nutrition Examination Survey; ASCVD, atherosclerotic cardiovascular disease; CVD, cardiovascular diseases.

TABLE 2. Associations of CAP and WCS with ASCVD.

	Model 1		Model 2	
Variables	**OR (95% CI)**	***p***	**OR (95% CI)**	***p***
CAP				
< 274	Reference		Reference	
≥ 274	2.40 (1.61-3.56)	< 0.001	1.84 (1.24-2.73)	**0.004**
WCS duration				
(0, 1)	Reference		Reference	
< 0	1.86 (1.00-3.45)	0.051	1.79 (0.88-3.62)	0.103
≥ 1	1.23 (0.84-1.81)	0.272	1.23 (0.76-1.99)	0.373

Model 1, adjusted for age, gender, education level, poverty-to-income ratio (PIR) and marital status; Model 2, adjusted for age, gender, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, chronic kidney disease, body mass index, median liver stiffness, alkaline phosphatase, gamma-glutamyl transferase and uric acid; CAP, controlled attenuation parameter; WCS, weekend catch-up sleep; ASCVD, atherosclerotic cardiovascular disease; OR, odds ratio; CI, confidence interval.

subgroups of individuals aged ≥ 60 years, females, and both obese and non-obese individuals, WCS ≥ 1 hour intensified the CAP-ASCVD association. Notably, in participants with WCS ≥ 1 hour, CAP was linked to elevated ASCVD odds, particularly in females (OR, 7.86, 95% CI, 2.24-27.56), non-obese (OR, 3.58, 95% CI, 1.14-11.27), and obese individuals (OR, 3.04, 95% CI, 1.10-8.37) (Table 6).

DISCUSSION

The present study examined the associations between WCS and CAP with ASCVD in adults aged ≥ 40 years and evaluated the potential moderating effect of WCS on the relationship between CAP and ASCVD. The findings revealed that individuals with CAP ≥ 274 dB/m exhibited higher odds of ASCVD compared to those with CAP < 274 dB/m. Notably, WCS durations of ≥ 1 hour were associated with an increased likelihood of ASCVD compared to durations of 0-1 hour. Furthermore, this moderating effect of WCS on the CAP-ASCVD relationship was observed across various subgroups, including individuals aged ≥ 60 years, females, and both non-obese and obese populations.

This study is the first to investigate the moderating role of WCS in the association between CAP and ASCVD. Prior research has explored the relationship between WCS and cardiovascular/metabolic diseases, but none has specifically addressed its interaction with CAP. For instance, Jang et al.[16] reported a negative association between WCS and dyslipidemia in male workers using data from the Korea National Health and Nutrition Examination Survey (KNHANES). Other cross-sectional studies based on KNHANES have linked WCS to NAFLD[28] and demonstrated that optimal WCS durations of 1-2 hours improved blood glucose regulation, whereas durations exceeding 3 hours were associated with impaired glucose metabolism.[29]

TABLE 3. Regulating Effect of WCS on Association of CAP with ASCVD.

	Model 1		Model 2	
Variables	**OR (95% CI)**	***p***	**OR (95% CI)**	***p***
WCS < 0 (reference: 0 ≤ WCS < 1)				
CAP	1.55 (0.89-2.71)	0.119	1.00 (0.52-1.93)	0.993
WCS	1.62 (0.76-3.44)	0.202	1.50 (0.76-2.98)	0.232
CAP*WCS	1.99 (0.65-6.06)	0.214	1.95 (0.58-6.54)	0.267
WCS ≥ 1 (reference: 0 ≤ WCS < 1)				
CAP	1.54 (0.93-2.55)	0.092	1.03 (0.56-1.88)	0.920
Catch-up time	0.75 (0.44-1.27)	0.269	0.66 (0.37-1.20)	0.165
CAP*WCS	2.68 (1.24-5.79)	0.014	3.29 (1.41-7.68)	**0.008**

*Multiplicative interaction, and the *p* values were interaction *p;* Model 1, adjusted for age, gender, education level, poverty-to-income ratio (PIR) and marital status; Model 2, adjusted for age, gender, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, chronic kidney disease, body mass index, median liver stiffness, alkaline phosphatase, gamma-glutamyl transferase and uric acid; WCS, weekend catch-up sleep; CAP, controlled attenuation parameter; ASCVD, atherosclerotic cardiovascular disease; OR, odds ratio; CI, confidence interval.

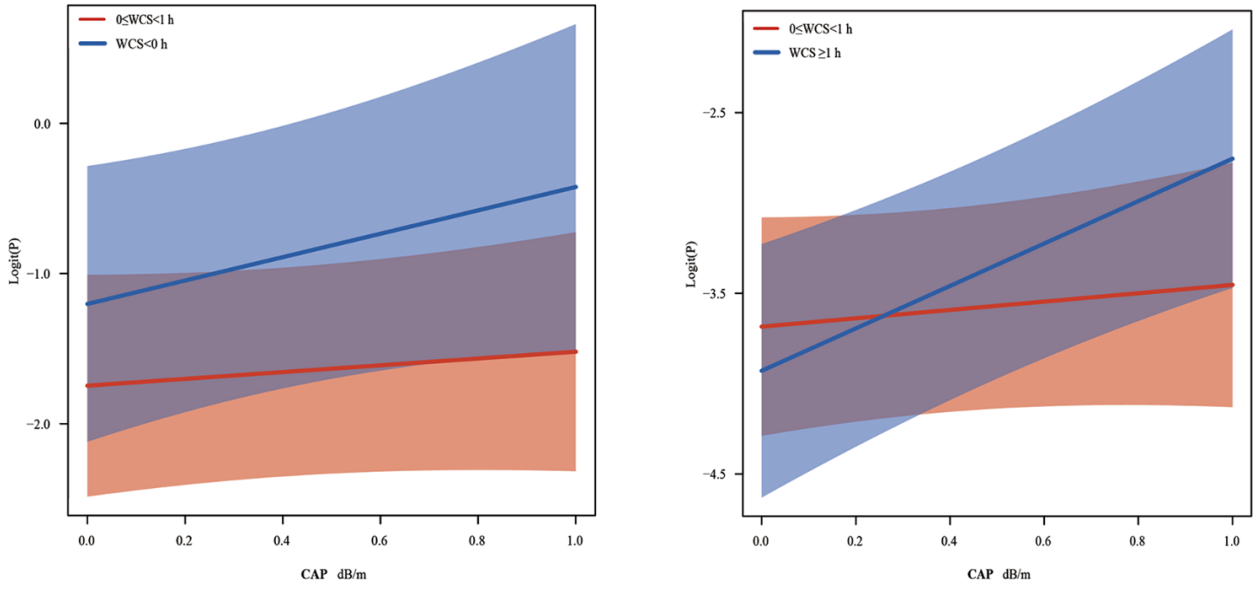

FIG. 2. Interaction plots of association between CAP and ASCVD in 0 ≤ WCS < 1 hour group and WCS ≥ 1 hour group.
CAP, controlled attenuation parameter; ASCVD, atherosclerotic cardiovascular disease; WCS, weekend catch-up sleep.

A recent analysis of the 2017-2018 NHANES data further highlighted that WCS durations > 2 hours were strongly associated with reduced CVD prevalence when weekday sleep durations were < 6 hours.[21] Consistent with Zhu et al.'s[21] approach, our study utilized NHANES data to investigate the moderating effect of WCS on CAP-related ASCVD risk, leveraging its large, representative U.S. population sample. However, our analysis of 2017-2020 data (two 2-year cycles) revealed that WCS durations ≥ 1 hour may elevate ASCVD risk in individuals with CAP ≥ 274 dB/m. These discrepancies may stem from racial and lifestyle differences between Asian and European populations. Nonetheless, the causal relationships between CAP, WCS, and ASCVD warrant further investigation.

CAP serves as a non-invasive tool for early NAFLD detection, with CAP-defined NAFLD (≥ 222 dB/m) independently associated with coronary plaques, particularly non-calcified plaques.[30] NAFLD is a leading global cause of liver disease and is linked to heightened cardiovascular risk.[31] Our results align with prior evidence, demonstrating that CAP ≥ 274 dB/m is associated with higher ASCVD odds in adults aged ≥ 40 years. While the mechanisms underlying the relationship between WCS and ASCVD risk remain unclear, several plausible pathways exist. The hypothalamic-pituitary-adrenal axis and autonomic nervous system play critical roles in regulating physical and mental health. Dysregulation of these systems, influenced by sleep parameters such as duration, quality, and circadian alignment, may increase NAFLD risk.[32,33] NAFLD-induced low-grade systemic inflammation promotes the release of pro-inflammatory cytokines, which may drive ASCVD progression via endothelial dysfunction and plaque formation.[34] Given the interplay between sleep and immune function,[35] moderate WCS may mitigate NAFLD-related inflammation, reducing cytokine release and ASCVD

TABLE 4. Association of CAP with ASCVD Under Different WCS Duration Conditions.

	Model 1		Mode 2	
Variables	**OR (95% CI)**	***p***	**OR (95% CI)**	***p***
0 ≤ WCS < 1 vs. WCS < 0				
0 ≤ WCS < 1				
CAP < 274	Reference		Reference	
CAP ≥ 274	1.57 (0.91-2.69)	0.099	0.96 (0.52-1.76)	0.891
WCS < 0				
CAP < 274	Reference		Reference	
CAP ≥ 274	3.13 (0.94-10.43)	0.062	1.66 (0.36-7.72)	0.505
0 ≤ WCS < 1 vs. WCS ≥ 1				
0 ≤ WCS < 1				
CAP < 274	Reference		Reference	
CAP ≥ 274	1.57 (0.91-2.69)	0.099	0.96 (0.52-1.76)	0.891
WCS ≥ 1				
CAP < 274	Reference		Reference	
CAP ≥ 274	3.81 (2.22-6.55)	< 0.001	3.72 (1.99-6.93)	**< 0.001**

Model 1, adjusted for age, gender, education level, poverty-to-income ratio (PIR) and marital status; Model 2, adjusted for age, gender, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, chronic kidney disease, body mass index, median liver stiffness, alkaline phosphatase, gamma-glutamyl transferase and uric acid; WCS, weekend catch-up sleep; CAP, controlled attenuation parameter; ASCVD, atherosclerotic cardiovascular disease; OR, odds ratio; CI, confidence interval.

TABLE 5. Regulating Effect of WCS on Association of CAP with ASCVD.

	WCS < 0 (reference: 0 ≤ WCS < 1), (n = 1950)					WCS ≥ 1 (reference: 0 ≤ WCS < 1), (n = 2727)			
Variables	**OR (95% CI)**	***p***	**OR (95% CI)**	***p***	**Variables**	**OR (95% CI)**	***p***	**OR (95% CI)**	***p***
	Age < 60 (n = 828)		**Age ≥ 60 (n = 1122)**			**Age < 60 (n = 1438)**		**Age ≥ 60 (n = 1289)**	
CAP	1.85 (0.88-3.89)	0.101	0.82 (0.50-1.35)	0.422	CAP	1.44 (0.58-3.57)	0.417	0.78 (0.48-1.25)	0.280
WCS	1.73 (0.64-4.69)	0.270	0.86 (0.31-2.40)	0.758	WCS	0.69 (0.32-1.52)	0.349	0.33 (0.18-0.58)	**< 0.001**
CAP*WCS	1.18 (0.30-4.65)	0.811	2.65 (0.41-17.25)	0.293	CAP*WCS	1.75 (0.66-4.67)	0.250	4.06 (1.11-14.80)	**0.035**
	Male (n = 866)		**Female (n = 1084)**			**Male (n = 1199)**		**Female (n = 1528)**	
CAP	0.80 (0.34-1.89)	0.604	1.40 (0.57-3.43)	0.447	CAP	0.85 (0.36-1.98)	0.690	1.28 (0.60-2.72)	0.509
WCS	1.47 (0.52-4.17)	0.451	1.77 (0.77-4.10)	0.171	WCS	1.02 (0.50-2.08)	0.959	0.41 (0.16-0.99)	**0.049**
CAP*WCS	4.10 (0.66-25.40)	0.124	0.57 (0.11-2.97)	0.493	CAP*WCS	2.38 (0.82-6.90)	0.107	5.91 (1.66-21.04)	**0.008**
	Non-obesity (n = 1158)		**Obesity (n = 792)**			**Non-obesity (n = 1584)**		**Obesity (n = 1143)**	
CAP	1.12 (0.44-2.85)	0.798	0.72 (0.25-2.03)	0.514	CAP	1.16 (0.49-2.78)	0.721	0.69 (0.26-1.82)	0.436
WCS	1.92 (0.80-4.64)	0.139	0.60 (0.13-2.77)	0.498	WCS	0.71 (0.36-1.38)	0.294	0.56 (0.26-1.19)	0.125
CAP*WCS	2.16 (0.37-12.51)	0.375	3.54 (0.42-30.00)	0.235	CAP*WCS	3.40 (1.07-10.80)	0.039	3.96 (1.37-11.49)	**0.013**

*Multiplicative interaction, and the *p* values were interaction *p*. **Age subgroups:** adjusted for gender, education level, poverty-to-income ratio (PIR), marital status, smoking, antidiabetic drug, drug for dyslipidemia, chronic kidney disease (CKD), body mass index (BMI), median liver stiffness, alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT) and uric acid (UA); **Gender subgroups:** adjusted for age, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, CKD, BMI, median liver stiffness, ALP, GGT and UA; **BMI obesity subgroups:** adjusted for age, gender, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, CKD, median liver stiffness, ALP, GGT and UA. WCS, weekend catch-up sleep; CAP, controlled attenuation parameter; ASCVD, atherosclerotic cardiovascular disease; OR, odds ratio; CI, confidence interval; BMI, body mass index.

risk. However, excessive WCS durations may negate these benefits and increase mortality risk.[36,37] Our findings suggest that limiting WCS to 0-1 hours may be optimal for minimizing ASCVD risk.

Age, DM, and hypertension are well-established risk factors for coronary heart disease in NAFLD patients.[38] Additional factors include dyslipidemia, insulin resistance, metabolic syndrome, dietary habits, smoking, and physical inactivity.[39] After adjusting for

TABLE 6. Association Between CAP and ASCVD Under Different WCS Conditions in Subgroups.

	0 ≤ WCS < 1		WCS ≥ 1	
Subgroups	**OR (95% CI**	***p***	**OR (95% CI)**	***p***
Age ≥ 60 years old	0.80 (0.48-1.35)	0.386	3.01 (0.94-9.67)	0.064
Female	1.22 (0.53-2.79)	0.624	7.86 (2.24-27.56)	**0.002**
Non-obesity	1.13 (0.47-2.75)	0.772	3.58 (1.14-11.27)	**0.031**
Obesity	0.73 (0.26-2.07)	0.539	3.04 (1.10-8.37)	**0.033**

Age subgroup: adjusted for gender, education level, poverty-to-income ratio (PIR), marital status, smoking, antidiabetic drug, drug for dyslipidemia, chronic kidney disease (CKD), body mass index (BMI), median liver stiffness, alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT) and uric acid (UA); **Gender subgroup:** adjusted for age, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, CKD, BMI, median liver stiffness, ALP, GGT and UA; **BMI obesity subgroups:** adjusted for age, gender, education level, PIR, marital status, smoking, antidiabetic drug, drug for dyslipidemia, CKD, median liver stiffness, ALP, GGT and UA; CAP, controlled attenuation parameter; ASCVD, atherosclerotic cardiovascular disease; WCS, weekend catch-up sleep; OR, odds ratio; CI, confidence interval.

covariates significantly associated with ASCVD, subgroup analyses revealed that WCS durations ≥ 1 hour were associated with elevated ASCVD risk in older adults (≥ 60 years), females, and both non-obese and obese individuals. These findings contrast with Son et al.'s[40] observation that WCS ≥ 1 hour reduced metabolic syndrome risk in younger populations, underscoring the need for age-specific considerations in WCS recommendations. Additionally, gender-specific differences in sleep patterns, such as women's higher insomnia prevalence,[42] may influence WCS effects. Dysregulated glucose metabolism and insulin resistance, common to both NAFLD and CVD, further highlight the importance of optimizing sleep patterns. While WCS may offer protective effects against obesity and metabolic syndrome in chronic short sleepers,[14,45] our results emphasize that appropriate WCS durations are critical for ASCVD risk reduction, regardless of obesity status.

This study is the first to explore the moderating effect of WCS on the CAP-ASCVD relationship, offering novel insights for ASCVD prevention. HS and hepatic fibrosis were assessed using transient elastography, a highly accurate method, and potential confounders comprehensively addressed using the representative NHANES database. However, limitations exist. The cross-sectional design precludes causal inferences, and NHANES WCS data are only available for 2017-2020. Future prospective studies should clarify the dose-response relationship between WCS and ASCVD risk.

In conclusion, WCS durations ≥ 1 hour may heighten ASCVD risk in adults with HS. Further research should focus on refining WCS duration recommendations to guide ASCVD prevention strategies.

Ethics Committee Approval: Not applicable.

Informed Consent: Not applicable.

Data Sharing Statement: The datasets analyzed during the current study are available from the corresponding author upon reasonable request.

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Supplementary Table 1: balkanmedicaljournal.org/img/files/87-SUPPLEMENTARY%20TABLE%201.%281%29.pdf

Supplementary Table 2: https://balkanmedicaljournal.org/img/files/87-SUPPLEMENTARY%20TABLE%202.%281%29.pdf

REFERENCES

1. Perumpail BJ, Khan MA, Yoo ER, Cholankeril G, Kim D, Ahmed A. Clinical epidemiology and disease burden of nonalcoholic fatty liver disease. *World J Gastroenterol*. 2017;23:8263-8276. [CrossRef]
2. Choi YS, Beltran TA, Calder SA, et al. Prevalence of hepatic steatosis and fibrosis in the United States. *Metab Syndr Relat Disord*. 2022;20:141-147. [CrossRef]
3. Duell PB, Welty FK, Miller M, et al. Nonalcoholic fatty liver disease and cardiovascular risk: a scientific statement from the American Heart Association. *Arterioscler Thromb Vasc Biol*. 2022;42:e168-e185. [CrossRef]
4. Kasper P, Martin A, Lang S, et al. NAFLD and cardiovascular diseases: a clinical review. *Clin Res Cardiol*. 2021;110:921-937. [CrossRef]
5. Munteanu MA, Mircea PA. From NAFLD to cardiovascular disease. Is it (still) the metabolic syndrome? *Clujul Med*. 2014;87:80-86. [CrossRef]
6. Deprince A, Haas JT, Staels B. Dysregulated lipid metabolism links NAFLD to cardiovascular disease. *Mol Metab*. 2020;42:101092. [CrossRef]
7. Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur Heart J*. 2011;32:1484-1492. [CrossRef]
8. Xie J, Li Y, Zhang Y, et al. Sleep duration and metabolic syndrome: an updated systematic review and meta-analysis. *Sleep Med Rev*. 2021;59:101451. [CrossRef]
9. Matsubayashi H, Nagai M, Dote K, et al. Long sleep duration and cardiovascular disease: associations with arterial stiffness and blood pressure variability. *J Clin Hypertens (Greenwich)*. 2021;23:496-503. [CrossRef]
10. Saz-Lara A, Lucerón-Lucas-Torres M, Mesas AE, Notario-Pacheco B, López-Gil JF, Cavero-Redondo I. Association between sleep duration and sleep quality with arterial stiffness:a systematic review and meta-analysis. *Sleep Health*. 2022;8:663-670. [CrossRef]
11. Cao X, Zhou J, Yuan H, Chen Z. Association between sleep condition and arterial stiffness in Chinese adult with nonalcoholic fatty liver disease. *J Thromb Thrombolysis*. 2016;42:127-134. [CrossRef]
12. Pizinger TM, Aggarwal B, St-Onge MP. Sleep extension in short sleepers: an evaluation of feasibility and effectiveness for weight management and cardiometabolic disease prevention. *Front Endocrinol (Lausanne)*. 2018;9:392. [CrossRef]
13. Kim JJ, Hwang IC. Weekend catch-up sleep is associated with reduced metabolic derangements in Korean adults. *Neurol Sci*. 2021;42:735-737. [CrossRef]
14. Kim DJ, Mun SJ, Choi JS, et al. Beneficial effects of weekend catch-up sleep on metabolic syndrome in chronic short sleepers. *Sleep Med*. 2020;76:2-32. [CrossRef]
15. Hwangbo Y, Kim WJ, Chu MK, Yun CH, Yang KI. Association between weekend catch-up sleep duration and hypertension in Korean adults. *Sleep Med*. 2013;14:549-554. [CrossRef]

16. Jang YS, Park YS, Hurh K, Park EC, Jang SI. Association between weekend catch-up sleep and dyslipidemia among Korean workers. *Sci Rep*. 2023;13:925. [CrossRef]
17. Li H, Yang M, Bao Y. Intelligent medicine on the regulation effect of weekend catch-up sleep on the relationship between hypertension and all-cause mortality. *Int J Comput Intell Syst*. 2023;16:59. [CrossRef]
18. Cabeza de Baca T, Chayama KL, Redline S, et al. Sleep debt: the impact of weekday sleep deprivation on cardiovascular health in older women. *Sleep*. 2019;42:zsz149. [CrossRef]
19. Arora T, Chen MZ, Cooper AR, Andrews RC, Taheri S. The impact of sleep debt on excess adiposity and insulin sensitivity in patients with early type 2 diabetes mellitus. *J Clin Sleep Med*. 2016;12:673-680. [CrossRef]
20. Ciardullo S, Carbone M, Invernizzi P, Perseghin G. Exploring the landscape of steatotic liver disease in the general US population. *Liver Int*. 2023;43:2425-2433. [CrossRef]
21. Zhu H, Qin S, Wu M. Association between weekend catch-up sleep and cardiovascular disease: evidence from the National Health and Nutrition Examination Surveys 2017-2018. *Sleep Health*. 2024;10:98-103. http://doi.org/10.1016/j.sleh.2023.09.006
22. Cash RE, Crowe RP, Goldberg SA, et al. Association between sleep characteristics, ideal cardiovascular health, and systemic inflammation, NHANES 2017-2018. J Sleep Res. 2022;31:e13497. [CrossRef]
23. Stone NJ, Robinson JG, Lichtenstein AH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;63:2889-2934. [CrossRef]
24. Goff DC Jr, Lloyd-Jones DM, Bennett G, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(25 Suppl 2):49-73. [CrossRef]
25. Grundy SM, Stone NJ, Bailey AL, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2019;139:e1082-e1143. Erratum in: *Circulation*. 2019;139:e1182-e1186. Erratum in: *Circulation*. 2023;148:e5. [CrossRef]
26. ALHarthi SSY, Natto ZS, Midle JB, et al. Association between time since quitting smoking and periodontitis in former smokers in the National Health and Nutrition Examination Surveys (NHANES) 2009 to 2012. *J Periodontol*. 2019;90:16-25. [CrossRef]
27. Jespersen T, Kruse N, Mehta T, Kuwabara M, Noureddine L, Jalal D. Light wine consumption is associated with a lower odd for cardiovascular disease in chronic kidney disease. *Nutr Metab Cardiovasc Dis*. 2018;28:1133-1139. [CrossRef]
28. Seo Y, Sung GH, Lee S, Han KJ. Weekend catch-up sleep is associated with the alleviation of non-alcoholic fatty liver disease. *Ann Hepatol*. 2022;27:100690. [CrossRef]
29. Kim YC, Um YJ, Yoon SH, 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. [CrossRef]
30. Park HE, Lee H, Choi SY, et al. Clinical significance of hepatic steatosis according to coronary plaque morphology: assessment using controlled attenuation parameter. *J Gastroenterol*. 2019;54:271-280. [CrossRef]
31. Pennisi G, Di Marco V, Buscemi C, et al. Interplay between non-alcoholic fatty liver disease and cardiovascular risk in an asymptomatic general population. *J Gastroenterol Hepatol*. 2021;36:2389-2396. [CrossRef]
32. Slavish DC, Taylor DJ, Lichstein KL. Intraindividual variability in sleep and comorbid medical and mental health conditions. *Sleep*. 2019;42:zsz052. [CrossRef]
33. Kim CW, Yun KE, Jung HS, et al. Sleep duration and quality in relation to non-alcoholic fatty liver disease in middle-aged workers and their spouses. *J Hepatol*. 2013;59:351-357. [CrossRef]
34. Tang WHW, Bäckhed F, Landmesser U, Hazen SL. Intestinal microbiota in cardiovascular health and disease: JACC state-of-the-art review. *J Am Coll Cardiol*. 2019;73:2089-2105. [CrossRef]
35. Besedovsky L, Lange T, Haack M. The sleep-immune crosstalk in health and disease. *Physiol Rev*. 2019;99:1325-1380. [CrossRef]
36. Lee H, Kim YJ, Jeon YH, Kim SH, Park EC. Association of weekend catch-up sleep ratio and subjective sleep quality with depressive symptoms and suicidal ideation among Korean adolescents. *Sci Rep*. 2022;12:10235. [CrossRef]
37. Grandner MA, Drummond SP. Who are the long sleepers? Towards an understanding of the mortality relationship. *Sleep Med Rev*. 2007;11:341-360. [CrossRef]
38. Toh JZK, Pan XH, Tay PWL, et al. A meta-analysis on the global prevalence, risk factors and screening of coronary heart disease in nonalcoholic fatty liver disease. *Clin Gastroenterol Hepatol*. 2022;20:2462-2473. [CrossRef]
39. Huang DQ, Downes M, Evans RM, et al. Shared mechanisms between cardiovascular disease and NAFLD. *Semin Liver Dis*. 2022;42:455-464. [CrossRef]
40. Son SM, Park EJ, Cho YH, 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;13:1465-1471. [CrossRef]
41. 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;13:20280. [CrossRef]
42. Zhang B, Wing YK. Sex differences in insomnia: a meta-analysis. *Sleep*. 2006;29:85-93. [CrossRef]
43. Mong JA, Cusmano DM. Sex differences in sleep: impact of biological sex and sex steroids. *Philos Trans R Soc Lond B Biol Sci*. 2016;371:20150110. [CrossRef]
44. Petersen KF, Dufour S, Savage DB, et al. The role of skeletal muscle insulin resistance in the pathogenesis of the metabolic syndrome. *Proc Natl Acad Sci U S A*. 2007;104:12587-12594. [CrossRef]
45. Im HJ, Baek SH, Chu MK, et al. Association between weekend catch-up sleep and lower body mass: population-based study. *Sleep*. 2017;40. [CrossRef]