Article

Not peer-reviewed version

Association of Sleep Duration Ratio with a Reduced Risk of Heart Failure: Analysis of the 2017-2023 National Health and Nutrition Examination Survey

Narathorn Kulthamrongsri *, Thanathip Suenghataiporn, Adivitch Sripusanapan, Smuch Siramongkholkarn, Thitiphan Srikulmontri, Chanokporn Puchongmart, Thanaboon Yinadsawaphan, Ben Thiravetyan, Kridhitach Ngarmukos, Koravich Lorlowhakarn, Nathasith Tangprasittichok, Ekamol Tantisattamo, Abdulelah Nuqali

Posted Date: 22 May 2026

doi: 10.20944/preprints202605.1518.v1

Keywords: heart failure; sleep duration; weekend sleep recovery; NHANES; cardiovascular disease; public health

 doi:10.20944/preprints202605.1518.v1

Article

Association of Sleep Duration Ratio with a Reduced Risk of Heart Failure: Analysis of the 2017-2023 National Health and Nutrition Examination Survey

Narathorn Kulthamrongsri [1,2,*], Thanathip Suenghataiporn [3], Adivitch Sripusanapan [1,2], Smuch Siramongkholkarn [4], Thitiphan Srikulmontri [5], Chanokporn Puchongmart [6], Thanaboon Yinadsawaphan [1,2], Ben Thiravetyan [6], Kridhitach Ngarmukos [2], Koravich Lorlowhakarn [4], Nathasith Tangprasittichok [7], Ekamol Tantisattamo [8] and Abdulelah Nuqali [2,9]

[1] Department of Cardiovascular Medicine, Mayo Clinic, Phoenix, Arizona, USA
[2] Internal Medicine Residency Program, University of Hawaii, Honolulu, Hawaii, USA
[3] Department of Internal Medicine, Griffin Hospital, Derby, Connecticut, USA
[4] Department of Medicine, Boston Medical Center Brighton, Boston, Massachusetts, USA
[5] Albert Einstein Medical Center, Philadelphia, Pennsylvania, USA
[6] Department of Internal Medicine, Texas Tech University Health Sciences Center, Lubbock, Texas, USA
[7] Phramongkutklao College of Medicine, Mahidol University, Bangkok, Thailand
[8] American Heart Association Comprehensive Hypertension Center at the University of California Irvine Medical Center, Division of Nephrology, Hypertension and Kidney Transplantation, Department of Medicine, University of California Irvine School of Medicine, Orange, California, USA
[9] Queen's Heart Institute, The Queen's Medical Center, Honolulu, Hawaii, USA
* Correspondence: thames.kulthamrongsri@gmail.com; Tel No.: 347-248-4674

Address(es) of the author(s)
NK: 1356 Lusitana St #510, Honolulu, HI 96813
TSu: 130 Division St, Derby, CT 06418
AS: 1356 Lusitana St #510, Honolulu, HI 96813
SSi: 736 Cambridge St. Brighton, MA 02135
TSr: 5501 Old York Rd, Philadelphia, PA 19141
CP: 3601 4th St STOP 6238, Lubbock, TX 79430
TY: 1356 Lusitana St #510, Honolulu, HI 96813
BT: 3601 4th St STOP 6238, Lubbock, TX 79430
KN: 1356 Lusitana St #510, Honolulu, HI 96813
KL: 736 Cambridge St. Brighton, MA 02135
NT: 317 Ratchawithi Road, Thung Phaya Thai Sub-district, Ratchathewi District, Bangkok 10400, Thailand
ET: 101 The City Drive South, Pavilion 4, Building 25, Orange, CA 92868
AN: 1356 Lusitana St #510, Honolulu, HI 96813

Abstract

Background/Objectives: In the United States, HF prevalence is projected to progressively rise by 2030. Prior research suggests a strong association between reduced sleep duration and increased cardiovascular disease and HF risk. This study introduces an alternative parameter, the Weekend Sleep Recovery (WSR), measured by the weekend-to-weekday Sleep Duration Ratio (SDR), to evaluate its association with HF risk. **Methods:** We conducted a cross-sectional analysis of NHANES (National Health and Nutrition Examination Survey) 2017–2023 to examine self-reported sleep patterns. Participants were classified as WSR (SDR > 1) or non-WSR (SDR ≤ 1). Multivariate logistic regression assessed the association between WSR and HF, adjusting for demographics and comorbidities. **Results:** Among 14,311 participants, WSR was associated with 23% lower odds of HF (adjusted OR 0.77; 95% CI 0.62–0.94; P = 0.012) versus non-WSR. This inverse association persisted in

 doi:10.20944/preprints202605.1518.v1

hypertensive, non-dyslipidemic, smoking, and Class III obese individuals, but was not modified by diabetes, coronary disease, or prior myocardial infarction. **Conclusions:** Although adequate sleep duration is crucial, weekend sleep duration reflecting WSR was associated with lower odds of HF, particularly in those with risk factors. Further studies are needed to explore these associations and potential implications for high-risk populations.

Keywords: heart failure; sleep duration; weekend sleep recovery; NHANES; cardiovascular disease; public health

1. Introduction

In the United States, data from the National Health and Nutrition Examination Survey (NHANES) estimate the prevalence of heart failure (HF) to range from 1.9% to 2.6%, with projections indicating that the number of individuals affected may reach 8.5 million by 2030 [1–3].

Previous studies have shown that shorter sleep duration is associated with increased risk of cardiovascular disease (CVD) and HF. Sleeping fewer than 6 hours per night has been associated with a 1.24-fold higher risk HF. Compared with a sleep duration of 7.0–7.9 hours per night, individuals who sleep 6.0–6.9 hours have a 1.29-fold higher risk. In contrast, long sleep duration has not been consistently associated with HF risk [4–6].

Weekend catch-up sleep (WCS), characterized by sleeping an additional two or more hours on weekends compared to weekdays, was associated with a reduced prevalence of CVD among individuals who usually sleep fewer than six hours on weekdays [7]. However, the association between variations in weekend and weekday sleep patterns and the risk of HF remains poorly understood.

Our study presents Weekend Sleep Recovery (WSR) as an alternative parameter, defined using the weekend-to-weekday Sleep Duration Ratio (SDR) instead of the absolute difference in sleep duration between weekdays and weekends. This approach aims to investigate the association between WSR and HF risk in a nationally representative sample, leveraging data from NHANES.

2. Methods

NHANES is a comprehensive and diverse series of surveys that assess general health, employment characteristics, nutritional status, lifestyle factors, and laboratory findings. Conducted by the Centers for Disease Control and Prevention (CDC) in collaboration with the National Center for Health Statistics (NCHS), this survey provides valuable insights into the health of the U.S. population. For this study, data from 27,493 adults aged 20 to 80 years, representing the civilian, non-institutionalized population between 2017 and 2023, were analyzed. Participants were included if they reported their sleep duration on both weekdays and weekends, along with their HF status. Individuals with missing data, unsure responses, or refusals were excluded, resulting in a final study population of 14,311 participants. (**Figure 1**)

 doi:10.20944/preprints202605.1518.v1

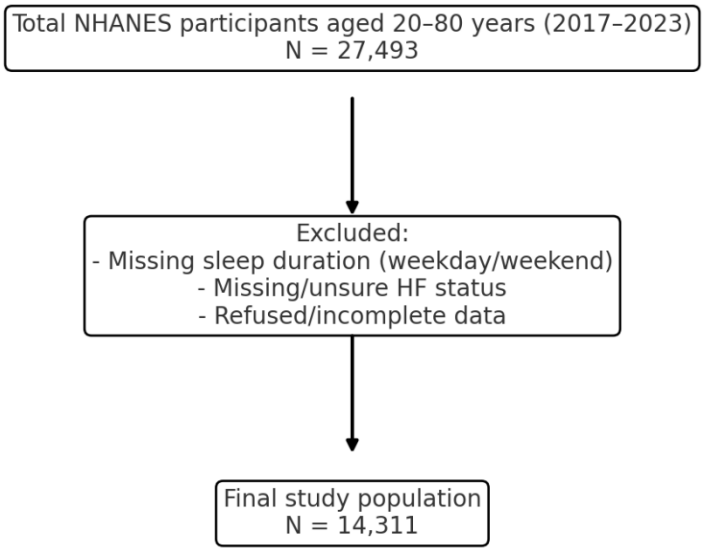

Figure 1. Flowchart of study participant selection from NHANES 2017–2023, showing inclusion and exclusion criteria leading to a final analytic sample of 14,311 adults aged 20–80 years.

This study did not necessitate consent or Institutional Review Board approval, as NHANES is a secondary dataset containing de-identified data that is publicly accessible.

2.1. Definition

Sleep Duration

Participants reported their average nightly sleep duration for both weekdays and weekends. Sleep duration data were collected in NHANES through two questions: “How much sleep do you usually get at night on weekdays or workdays?” and “How much sleep do you usually get at night on weekends or non-workdays?” Responses ranged from 1 to 12, with 12 representing 12 or more hours of sleep. Participants who selected "don't know" or "refused" to answer were excluded from the analysis.

Weekend Sleep Recovery and Weekend-to-Weekday Sleep Duration Ratio

The "SDR" was calculated by dividing the reported sleep duration on weekends by the reported sleep duration on weekdays. Participants were classified into two groups based on their SDR: WSR and non-WSR, represented individuals with an SDR >1 and ≤ 1, respectively.

Cardiovascular Condition and Other Confounders

NHANES collects data on various self-reported health conditions through the general question, “Has a doctor or other health professional ever told you that you had [name of medical condition]?” Participants who reported a diagnosis of HF were classified as having the primary outcome of interest.

Potential confounders, including demographic variables (race, gender, and age at screening), social factors (marital status and education level), and medical comorbidities (obesity status based on body mass index (BMI), smoking status, diabetes mellitus, hypertension, dyslipidemia, coronary artery disease (CAD) and myocardial infarction (MI), are detailed in Table 1.

2.2. Outcomes

The primary outcome was the association between WSR and self-reported HF, as defined by participants’ responses to the NHANES question asking whether a doctor or other health

 doi:10.20944/preprints202605.1518.v1

professional had ever told them they had heart failure. The analysis compared the odds of HF among individuals with WSR (sleep duration ratio [SDR] >1) versus those without WSR (SDR ≤1). Multivariable logistic regression models (Models 1–5) were used to estimate these associations, with sequential adjustment for potential confounding variables as described in the Statistical Analysis section.

Secondary analyses evaluated the association between WSR and HF across subgroups stratified as described in the Statistical Analysis section. These subgroup analyses were exploratory and hypothesis-generating.

2.3. Statistical Analysis.

Categorical data were presented as proportion, while continuous variables were expressed as means with standard deviations or median and interquartile range (IQR). Baseline differences for categorical variables were evaluated using the Chi-square test, and continuous variables were compared using the student's t-test. Logistic regression models were applied to evaluate the associations between WSR and HF as follows:

Model 1 (Unadjusted): Univariate logistic regression

Model 2 (Demographic-adjusted): Model 1 + demographic variables

Model 3 (Social factor–adjusted): Model 2 + social factor variables

Model 4 (Medical comorbidity–adjusted) (Primary adjusted model): Model 3 + medical comorbidity variables

Model 5 (CAD/MI-adjusted): Model 4 + CAD and MI

CAD and MI were not included in the primary adjusted model (Model 4) because they are highly correlated with HF and several other covariates, raising concerns about multicollinearity. Additionally, CAD and MI may lie on the causal pathway between sleep pattern variability and HF, functioning partly as mediators rather than pure confounders. Therefore, they were incorporated as additional covariates only in Model 5 and in sensitivity analyses to assess the robustness of the associations.

Sensitivity analyses examined the association between WSR and HF across subgroups defined by weekday sleep duration (≤3, >3–≤4, >4–≤5, >5–≤6, >6–≤7, >7–≤8, >8–≤9, >9–≤10, >10–≤11, >11–≤12, and ≥12 hours), age (<65 vs. ≥65 years), race, and common HF comorbidities, including diabetes mellitus, hypertension, dyslipidemia, smoking status, obesity categories (underweight [<18.5], normal [18.5–24.9], overweight [25.0–29.9], Class I [30.0–34.9], Class II [35.0–39.9], Class III [≥40]), and CAD and MI [8–10]. Only Black and White participants were included in race-stratified analyses because sample sizes for other racial groups were insufficient for meaningful comparisons.

Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). All p-values were two-sided and are reported as nominal, as no adjustments were made for multiple comparisons. A p-value threshold of <0.05 is presented for reference in both primary and sensitivity analyses. All statistical analyses were performed using Stata version 15.2.

3. Results

3.1. Baseline Characteristics of the Study Population

A total of 16,985 NHANES participants provided responses regarding their HF status, while 17,519 and 17,510 completed the sleep survey for weekdays and weekends, respectively. After applying the exclusion criteria, the final study cohort included 14,311 adults with SDR along with available HF status information.

Of 14,311 participants, the mean age was 43.9 ± 23.4 years, and 52.5% were female. Up to 4.13% (n = 702) reported a history of HF. The mean sleep duration was 7.7±1.7 hours on weekdays and 8.3±1.8 hours on weekends. Significant differences were observed between the two groups in weekday sleep and sleep duration ratios, but not in weekend sleep durations. Weekday sleep

 doi:10.20944/preprints202605.1518.v1

duration was significantly shorter in participants with HF compared to non-HF counterparts (mean difference: -0.26, 95% CI: -0.43 to -0.10, P = 0.002). (**Table 1B)**

Table 1B. *Baseline sleep characteristics* (mean ± SD (standard deviation) or median +/- Interquartile range (IQR) or n [%]) *of individuals categorized as heart failure (HF)* vs. *non-HF.*

Table 1B Baseline characteristics of individuals categorized as HF vs non-HF.

Variables	non-HF	HF	Mean difference	p-value
How much sleep do you get during weekdays? (hours)	7.6 ±1.6 (8 +/- 1.5) (n = 16,111)	7.9 ± 2.2 (8 +/- 2) (n = 681)	-0.3 ± 0.1 (-0.43, -0.10)	<0.01
How much sleep do you get during weekends? (hours)	8.3 ± 1.7 (8 +/- 2) (n = 16,104)	8.2 ± 2.2 (8 +/- 2.5) (n = 679)	0.0 ± 0.1 (-0.12,0.21)	0.62
Weekday to weekend Sleep duration ratio	1.1 ± 0.3 (1 +/- 0.2) (n = 16,052)	1.1 ± 0.3 (1 +/- 0.1) (n = 676)	0.04 ± 0.0 (0.02,0.07)	<0.01

This table summarizes weekday sleep duration, weekend sleep duration, and the weekday-to-weekend sleep duration ratio for individuals with and without HF. Significant group differences were observed in weekday sleep duration and sleep duration ratios, whereas weekend sleep duration did not differ meaningfully between groups

Participants in the non-WSR group (SDR ≤ 1) were older (mean age 55.5 ± 18.6 years) compared to those in the WSR group (SDR > 1; mean age 34.1 ± 22.5 years). The non-WSR group also had higher proportions of individuals with hypertension, diabetes mellitus, and dyslipidemia, reflecting important clinical differences between the groups (**Table 1A**).

Table 1A. Baseline characteristics (mean ± SD (standard deviation) or median ± Interquartile range (IQR) or n [%]) of individuals categorized as Non-WSR (Weekday to weekend sleep duration ratio, SDR ≤ 1) and WSR (SDR > 1). Significant differences were observed between the two groups in terms of age, race, BMI, smoking status, marital status, and several health conditions, including heart failure, coronary artery disease, myocardial infarction, hypertension, diabetes mellitus, and dyslipidemia. Sleep duration patterns, including weekday and weekend sleep durations and sleep duration ratio, also differed significantly between the groups.

Table 1A Baseline characteristics of individuals categorized as Non-WSR (SDR ≤ 1) vs WSR (SDR>1)

Variables	Non-WSR (SDR ≤ 1) (n = 9,691)	WSR (SDR>1) (n = 17,802)	p-value*
12,Age (years)	55.5 ± 18.6	34.1 ± 22.5	<0.0001
Women (%)	5,045 (52.1%)	6,059 (52.9%)	0.233
White (%)	4,869 (50.2%)	4,555 (39.8%)	<0.0001
Body mass index (BMI)	29.7 ± 7.3	30.3 ± 8.5	<0.0001
Overweight status (%)	1,938 (38.7%)	1,679 (39.9%)	0.356
Active smoker (%)	4,290 (44.7%)	2,796 (36.0%)	<0.0001
Marital Status (married) (%)	5,093 (54.0%)	4,313 (57.0%)	<0.0001
Education: above college (%)	2,640 (28.0%)	2,240 (29.6%)	0.098
Heart failure (%)	504 (5.4%)	198 (2.6%)	<0.0001
Coronary artery disease (%)	632 (6.7%)	190 (2.5%)	<0.0001

 doi:10.20944/preprints202605.1518.v1

Myocardial Infarction (%)	554 (5.9)	206 (2.7)	<0.0001
Hypertension (%)	4,111 (42.4%)	2,412 (30.1%)	<0.0001
Diabetes mellitus (%)	1,581 (16.3%)	914 (8.1%)	<0.0001
Dyslipidemia (%)	3,953 (40.8%)	2,397 (29.9%)	<0.0001
How much sleep do you get during weekdays? (hours)	8.1± 1.7	7.2 ± 1.5	<0.0001
How much sleep do you get during weekends? (hours)	7.7± 1.7	9.0 ± 1.6	<0.0001
Weekday to weekend Sleep duration ratio	1.0 ± 0.1	1.3 ± 0.3	<0.0001

The sleep duration patterns of the study population exhibited a normal distribution for both weekday sleep duration in hours and the weekend-to-weekday SDR, as illustrated in the histograms **(Figures 2A and 2B).** The line plot **(Figure 2C)** demonstrates considerable variability in sleep durations, ranging from 0 to approximately 15 hours for both weekdays and weekends. The sleep patterns of individuals with and without HF were compared using boxplots, which illustrate weekday and weekend sleep duration, as well as the weekend-to-weekday sleep ratio, showing the distribution, median, interquartile range, and outliers for each group **(Figure 3).**

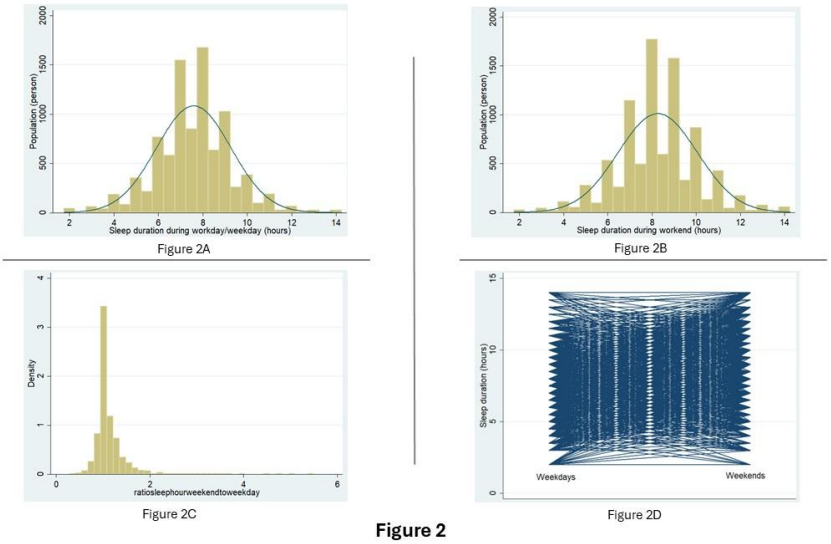

Figure 2. A-B. Histogram showing the distribution of sleep duration among a population during workdays or weekdays and weekend, measured in hours. The data is presented in intervals (bins) that show how many people fall into each range of sleep duration. **Figure 2C** Histogram showing the distribution of the ratio of sleep hours on weekends to weekdays. The *x*-axis represents the ratio of weekend to weekday sleep hours, and the *y*-axis represents the density, indicating how frequently each ratio occurs in the data. **Figure 2D** Line plot showing sleep duration (in hours) on the *y*-axis, separated by weekdays and weekends on the *x*-axis. Each point represents an individual data entry for sleep duration.

 doi:10.20944/preprints202605.1518.v1

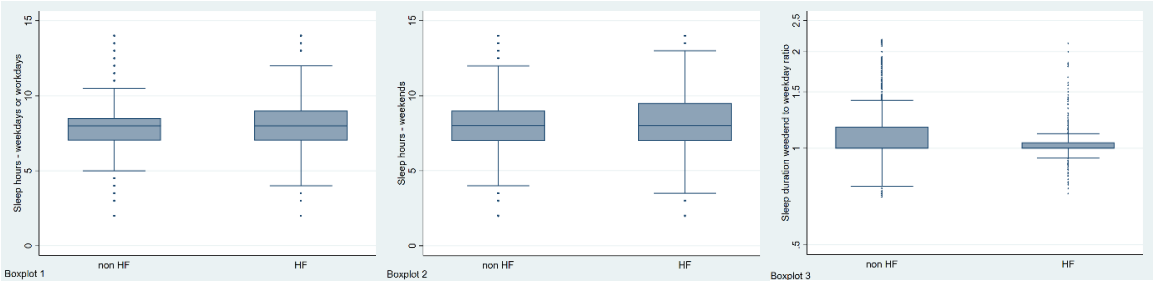

Figure 3. Boxplots comparing sleep patterns between individuals with heart failure (HF) and without heart failure (non-HF). Boxplot 1 shows weekday/workday sleep hours, Boxplot 2 shows weekend sleep hours, and Boxplot 3 shows the ratio of weekend to weekday sleep duration, after trimming the upper and lower 1% of the SDR distribution and applying a logarithmic scale to the *Y*-axis. Each plot displays the distribution, median, interquartile range, and outliers for each group.

3.2. Primary and Secondary Outcome

Our analysis revealed a consistent trend indicating that WSR was inversely associated with HF in all models of the overall population. In unadjusted analyses, individuals in the WSR group had lower odds of HF (OR: 0.47, 95% CI: 0.40–0.56; nominal p < 0.001). After adjustment for demographic variables (Model 2), the magnitude of the association was reduced (OR: 0.76, 95% CI: 0.62–0.93; nominal p = 0.008).Further adjustments for social factors (Model 3), medical comorbidities (Model 4) and CAD/MI (model 5) yielded similar results, with ORs of 0.75 (95% CI: 0.61–0.92, p = 0.005),0.77 (95% CI: 0.62–0.94, p = 0.012), and 0.79 (95% CI: 0.64–0.97, p = 0.023) respectively. These findings suggest that WSR is independently associated with lower odds of HF, even after accounting for key confounding variables. **(Table 2A)**

Table 2A. *Association between weekend sleep recovery (WSR; sleep duration ratio >1) and heart failure (HF) across unadjusted and adjusted models.*

Table 2A: Adjusted* association of WSR (SDR >1) and heart failure (HF)					
Population	**n**	**OR**	**p-value**	**95%CI**	
Unadjusted model 1	16,985	0.47	0.000	0.40	0.56
Adjusted model 2	14,312	0.76	0.008	0.62	0.93
Adjusted model 3	14,311	0.75	0.005	0.61	0.92
Adjusted model 4	14,311	0.77	0.012	0.62	0.94
Adjusted model 5	14,311	0.79	0.023	0.64	0.97

Model 1 presents the unadjusted association.

Model 2 adjusts for demographic variables (age, race, and gender).

Model 3 further adjusts for social factors (marital status and education level).

Model 4 additionally adjusts for medical comorbidities (body mass index, smoking status, hypertension status, diabetes mellitus status, and dyslipidemia status).

Model 5 additionally adjusts for concurrent heart diseases (coronary artery disease and myocardial infarction)

3.3. Sensitivity Analyses

The inverse association was most pronounced among individuals with 6–7 hours of weekday sleep (OR = 0.43, 95% CI: 0.23–0.79; nominal p = 0.006) and among those with 10–11 hours of weekday sleep (OR = 0.08, 95% CI: 0.01–0.79; nominal p = 0.03). Estimates in other weekday sleep categories were closer to the null, and confidence intervals included the null value. In analyses stratified by comorbid conditions, associations between WSR and HF varied across subgroups (Table 2B, Figure

 doi:10.20944/preprints202605.1518.v1

4). Some cardiometabolic strata showed lower odds estimates, whereas others were closer to the null. However, several estimates were imprecise, and these findings should be interpreted cautiously given the exploratory nature of the analyses.

Table 2B. (Adjusted Model 4), adjusted for age, race, gender, BMI, marital status, educational level, smoking status, hypertension status, diabetes mellitus status, and dyslipidemia status, presents the associations between weekend-to-weekday SDR (indicating WSR) and HF status. The analysis was stratified by key population characteristics, including weekday sleep duration, age categories, race, diabetes mellitus status, hypertension status, dyslipidemia status, coronary artery disease (CAD) status, myocardial infarction (MI) status, smoking status, and BMI categories.

Table 2B: Adjusted model 4 association of WSR (SDR >1) and heart failure (HF)

Population		n	OR	Nominal p-value	95%CI	
Stratified by weekday sleep duration	≤ 3 hours	150	0.59	0.498	0.13	2.72
	>3-≤4 hours	129	1.25	0.735	0.35	4.52
	>4-≤5 hours	597	0.90	0.788	0.41	1.97
	>5-≤6 hours	1,432	0.80	0.465	0.45	1.45
	>6-≤7 hours	2,799	0.43	0.006	0.23	0.79
	>7-≤8 hours	4,173	1.02	0.937	0.66	1.56
	>8-≤9 hours	2,723	0.88	0.616	0.53	1.46
	>9-≤10 hours	902	0.74	0.465	0.32	1.67
	>10-≤11 hours	334	0.08	0.030	0.01	0.79
	>11-≤12 hours	89	NA	NA	NA	NA
	> 12 hours	70	NA	NA	NA	NA
Stratified by age	< 65-year-old	10,261	0.69	0.017	0.51	0.94
	≥ 65-year-old	4,050	0.84	0.231	0.64	1.12
Stratified by race	White	6,398	0.59	0.002	0.43	0.82
	Black	2,963	0.81	0.269	0.55	1.18
Stratified by diabetes mellitus status	Diabetes mellitus	2110	0.80	0.161	0.59	1.09
	Non- diabetes mellitus	11739	0.76	0.066	0.57	1.02
Stratified by hypertension status	Hypertension	5447	0.72	0.006	0.58	0.91
	Non-hypertension	8848	1.05	0.838	0.66	1.67
Stratified by dyslipidemia status	Dyslipidemia	5424	0.90	0.413	0.69	1.16
	Non-dyslipidemia	8792	0.56	0.001	0.39	0.79
Stratified by CAD status	CAD	670	0.97	0.884	0.64	1.47
	Non-CAD	13,595	0.78	0.071	0.60	1.02
Stratified by MI status	MI	617	0.95	0.781	0.64	1.40
	Non-MI	13,674	0.77	0.056	0.59	1.01
	Smoking	5937	0.71	0.013	0.54	0.93

 doi:10.20944/preprints202605.1518.v1

Stratified by Smoking status	Non-Smoking	8363	0.87	0.404	0.63	1.20
Stratified by BMI	BMI <18.5 (Underweight)	157	1.24	0.867	0.10	15.2
	BMI 18.5-24.9 (Normal weight)	3432	0.76	0.333	0.44	1.32
	BMI 25.0-29.9 (Overweight)	4581	0.72	0.150	0.47	1.12
	BMI 30.0-34.9 (Class I obesity)	3060	1.04	0.838	0.70	1.56
	BMI 35.0-39.9 (Class II obesity)	1591	0.71	0.215	0.41	1.22
	BMI ≥40 (Class III obesity)	1366	0.55	0.012	0.35	0.88

SDR >1 means Sleep duration on weekend is more than Sleep duration on weekday
SDR ≤1 means Sleep duration on weekend is less than/equal Sleep duration on weekday (as reference)
NA, not available

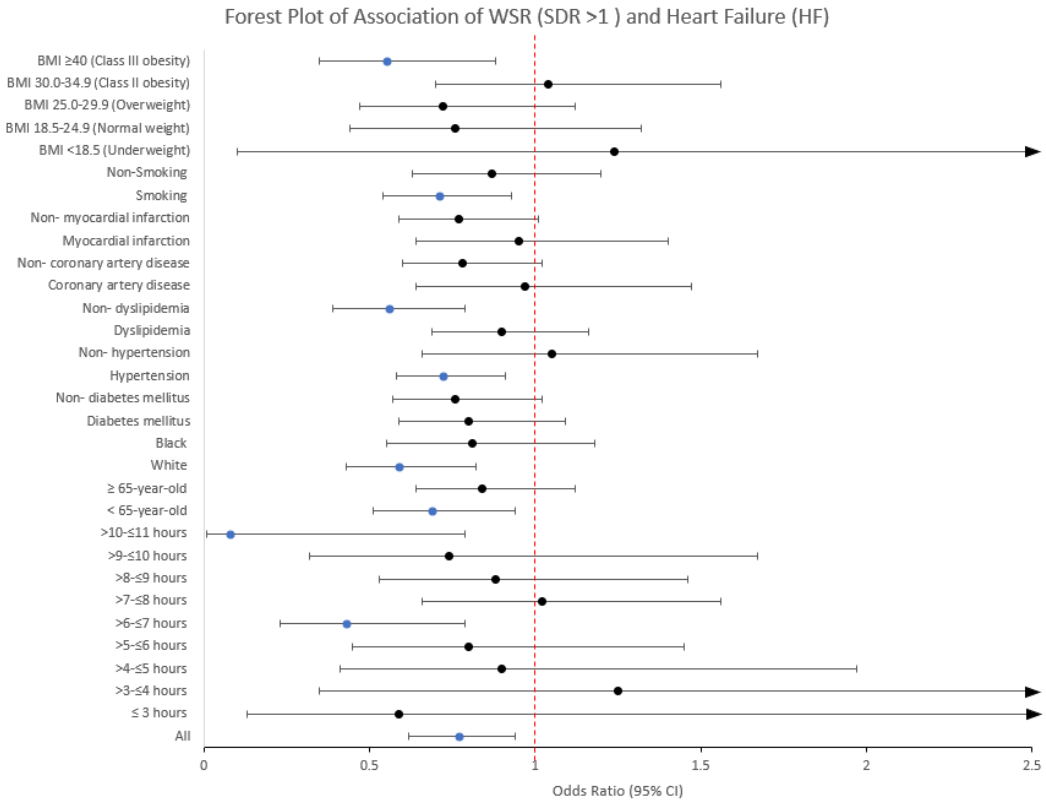

Figure 4. Forest plot showing the association between weekend-to-weekday sleep ratio (WSR; sleep duration ratio [SDR] >1) and heart failure (HF) across various subgroups. Odds ratios (ORs) and 95% confidence intervals (CIs) are displayed for subgroups based on BMI categories, smoking status, dyslipidemia, hypertension, diabetes mellitus, coronary artery disease, myocardial infarction, race, age, and sleep duration categories. Blue dots represent associations with lower odds of HF (nominal $p < 0.05$), while black dots represent associations with nominal $p \geq 0.05$.

 doi:10.20944/preprints202605.1518.v1

4. Discussion

This study observed an inverse association between WSR and HF, with individuals reporting weekend sleep recovery having lower odds of HF. Given the cross-sectional design, these findings should be interpreted as associative rather than indicative of a protective effect. The direction and magnitude of the association were generally similar across regression models.

In subgroup analyses, the inverse association between WSR and HF appeared more pronounced among individuals with 6–7 hours of weekday sleep, those younger than 65 years, and certain cardiometabolic subgroups. However, estimates across many strata were imprecise, and confidence intervals frequently included the null. Racially stratified analyses showed broadly similar directional estimates between White and Black participants (Table 2C), although precision was limited in several subgroups.

Table 2C. Adjusted Model 4 Association of Weekday-to-Weekend Sleep Ratio (WSR; SDR >1) With Heart Failure (HF) Stratified by Race and Weekday Sleep Duration.

Table 2C: Adjusted model 4 association of WSR (SDR >1) and heart failure (HF) comparing White and Black individuals											
Population		**White**					**Black**				
		n	**OR**	**nominal p-value**	**95%Cl**		**n**	**OR**	**nominal p-value**	**95%CI**	
All		6,398	0.59	0.002	0.43	0.82	2,963	0.81	0.269	0.55	1.18
Stratified by weekday sleep duration	≤ 3 hours	32	NA	NA	NA	NA	13	NA	NA	NA	NA
	>3-≤4 hours	36	0.50	0.678	0.02	12.93	64	5.42	0.234	0.34	87.28
	>4-≤5 hours	214	0.27	0.166	0.04	1.71	208	1.45	0.556	0.42	5.04
	>5-≤6 hours	511	0.50	0.185	0.18	1.39	189	1.09	0.885	0.35	3.39
	>6-≤7 hours	1,339	0.27	0.012	0.10	0.75	617	0.35	0.097	0.10	1.21
	>7-≤8 hours	2,089	0.66	0.201	0.35	1.25	652	1.47	0.423	0.57	3.74
	>8-≤9 hours	1,390	1.32	0.404	0.69	2.53	455	0.61	0.382	0.20	1.84
	>9-≤10 hours	455	0.70	0.55	0.22	2.26	124	0.51	0.424	0.10	2.66
	>10-≤11 hours	114	NA	NA	NA	NA	NA	NA	NA	NA	NA
	>11-≤12 hours	29	NA	NA	NA	NA	8	NA	NA	NA	NA
	> 12 hours	14	NA	NA	NA	NA	32	NA	NA	NA	NA

Table 2C presents the adjusted associations between WSR (SDR >1) and heart failure among White and Black individuals using Model 4, which adjusts for age, sex, race, BMI, marital status, education, smoking status, hypertension, diabetes, and dyslipidemia. Results are shown for the overall population as well as stratified by weekday sleep duration categories. Odds ratios (ORs), 95% confidence intervals (CIs), and nominal p-values are displayed for each subgroup. “NA” indicates insufficient sample size to produce stable model estimates.Figure/legend

 doi:10.20944/preprints202605.1518.v1

Given the number of subgroup comparisons and the absence of adjustment for multiple testing, these analyses should be considered exploratory. All p-values are nominal, and the subgroup findings are hypothesis-generating rather than confirmatory. Prospective studies are needed to determine whether these observed differences reflect true effect modification or statistical variability.

Our study builds on prior work introducing WSR as a relative measure of compensatory sleep. Traditional WCS defines recovery as an absolute ≥2-hour difference between weekday and weekend sleep, which may overlook smaller but proportionally meaningful changes (e.g., 4 vs. 5 hours). By using SDR, we capture proportional sleep compensation, allowing assessment of associations even when weekend extension is less than two hours. This approach may better reflect real-world sleep patterns, particularly for individuals unable to achieve large absolute increases in weekend sleep.

Our results aligned with previous research show that both short and long sleep durations are linked to an increased risk of HF and other cardiovascular conditions [6,8]. Prior studies have demonstrated U-shaped associations between sleep duration and HF risk, emphasizing the importance of optimal sleep for cardiovascular health [6]. Evidence also suggests that insufficient sleep is associated with increased inflammation, dysregulated appetite hormones, and higher BMI, which are well-established risk factors for HF [11]. Moreover, research on WCS highlights its potential to reduce CVD prevalence, although its direct impact on HF remains uncertain [7]. Notably, a recent NHANES 2017–2023 analysis by Zhang et al. [11] reported that WCS was associated with lower odds of hypertension, which is one of the HF-related comorbidities [8], supporting the concept that recovery sleep may mitigate cardiometabolic risk. Moreover, by using a relative measure like SDR, rather than the traditional ≥2-hour absolute difference, we capture proportional sleep compensation even with modest weekend extension, suggesting that recovery sleep patterns may influence cardiovascular risk across progressive stages of disease These findings highlight the need for further research into recovery sleep patterns and their role in reducing HF risk across diverse populations.

Limitation

This study has several limitations. First, we could not account for potential residual confounders, such as underreported alcohol consumption, SDOH which influences both sleep and HF. Second, while SDR has its advantages, it also has a key limitation: it does not distinguish between individuals with different sleep patterns but identical SDR values. For instance, someone sleeping 3 hours on weekdays and 4.5 hours on weekends has the same SDR of 1.5 as someone sleeping 6 hours on weekdays and 9 hours on weekends. Third, the cross-sectional design of NHANES precludes causal inference. Missing data and reliance on self-reported measures may also introduce recall, misclassification or social desirability bias. Furthermore, there were significant baseline differences between the non-WSR and WSR groups in terms of cardiovascular risk profiles; individuals in the non-WSR group were generally older, more likely to smoke, and had more comorbidities, which could have influenced the observed associations despite statistical adjustments. Finally, the findings are based on U.S. population data, which may limit generalizability to other populations.

5. Conclusion

Our findings suggest that WSR is associated with lower odds of HF, particularly among individuals with intermediate weekday sleep durations and certain cardiometabolic subgroups. These observations may indicate a relationship between recovery sleep patterns and cardiovascular risk; however, prospective and mechanistic studies are required before drawing conclusions regarding causality, clinical implications, or sleep recommendations. Reverse causality has not been excluded.

Author Contributions: Conceptualization, NK; Methodology, NK; Validation, TSu, ET and AN; Formal Analysis, NK; Writing – Original Draft Preparation, NK, AS, TSu and SSi; Writing – Review & Editing, NK, TSr, CP, TY, BT, KN, KL, NT, ET and AN; Visualization, ET and AN. All authors have read and agreed to the

 doi:10.20944/preprints202605.1518.v1

published version of the manuscript. NK: Conceptualization, Methodology, Formal Analysis, Writing – Original Draft, Review & Editing AS: Writing – Original Draft TSu: Validation, Writing – Original Draft SSi: Writing – Original Draft TSr: Writing – Review & EditingCP: Writing – Review & Editing TY: Writing – Review & Editing BT: Writing – Review & Editing KN: Writing – Review & Editing KL: Writing – Review & EditingNT: Writing – Review & Editing. ET: Visualization, Validation, Writing – Review & Editing AN: Visualization, Validation, Writing – Review & Editing.

Funding: This research received no external funding. The APC was funded by the authors.

Institutional Review Board Statement: Not applicable. The study used de-identified secondary data from the publicly available National Health and Nutrition Examination Survey (NHANES) database (https://www.cdc.gov/nchs/nhanes/), which is not subject to additional Institutional Review Board approval.

Informed Consent Statement: Not applicable. NHANES data are de-identified and publicly available; written informed consent was obtained by the National Center for Health Statistics at the time of original data collection.

Data Availability Statement: The data analyzed in this study were obtained from the publicly available National Health and Nutrition Examination Survey (NHANES) database (https://www.cdc.gov/nchs/nhanes/). Additional processed data and analysis outputs generated during the current study are not publicly available but are available from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Khan, M.S., et al., *Global epidemiology of heart failure.* Nat Rev Cardiol, 2024. **21**(10): p. 717-734.
2. Bozkurt, B., et al., *Heart Failure Epidemiology and Outcomes Statistics: A Report of the Heart Failure Society of America.* J Card Fail, 2023. **29**(10): p. 1412-1451.
3. Savarese, G., et al., *Global burden of heart failure: a comprehensive and updated review of epidemiology.* Cardiovasc Res, 2023. **118**(17): p. 3272-3287.
4. Zhuang, S., et al., *Prospective study of sleep duration, snoring and risk of heart failure.* Heart, 2023.
5. Wang, S., et al., *Associations between sleep duration and cardiovascular diseases: A meta-review and meta-analysis of observational and Mendelian randomization studies.* Front Cardiovasc Med, 2022. **9**: p. 930000.
6. Wannamethee, S.G., et al., *Self-Reported Sleep Duration, Napping, and Incident Heart Failure: Prospective Associations in the British Regional Heart Study.* Journal of the American Geriatrics Society, 2016. **64**(9): p. 1845-1850.
7. Zhu, H., S. Qin, and M. Wu, *Association between weekend catch-up sleep and cardiovascular disease: Evidence from the National Health and Nutrition Examination Surveys 2017-2018.* Sleep Health, 2024. **10**(1): p. 98-103.
8. Gallo, G. and C. Savoia, *Hypertension and Heart Failure: From Pathophysiology to Treatment.* Int J Mol Sci, 2024. **25**(12).
9. Aune, D., et al., *Tobacco smoking and the risk of heart failure: A systematic review and meta-analysis of prospective studies.* Eur J Prev Cardiol, 2019. **26**(3): p. 279-288.
10. Ebong, I.A., et al., *Mechanisms of heart failure in obesity.* Obes Res Clin Pract, 2014. **8**(6): p. e540-8.
11. Zhang, Y., et al., *Association between weekend catch-up sleep and hypertension in U.S. middle-aged and older adults: Insights from NHANES 2017-2023.* Medicine (Baltimore), 2025. **104**(40): p. e44858.