European Journal of Pain

ORIGINAL ARTICLE OPEN ACCESS

Painful Mondays: Exploring Weekly Sleep Variations and Pain Perception in Healthy Women—An Experimental Study

Shima Rouhi | Natalia Egorova-Brumley | Amy S. Jordan

The University of Melbourne, Parkville, Australia

Correspondence: Natalia Egorova-Brumley (natalia.brumley@unimelb.edu.au)

Received: 16 August 2024 | **Revised:** 5 February 2025 | **Accepted:** 10 February 2025

Funding: S.R. was funded by the University of Melbourne (Melbourne Research Scholarship) doctoral studentship. N.E.-B. was supported by the Australian Research Council (FT230100235).

Keywords: conditioned pain modulation | experimental pain | pain summation | sleep fluctuation

ABSTRACT

Background: Acute experimental sleep deprivation induces pain hypersensitivity, particularly in females. While the impact of extreme sleep loss on pain perception has been largely studied, how subtle sleep fluctuations, for example, sleep variations across the week, affect pain perception remains unclear. This study investigated how weekly sleep variations affect pain perception in young healthy women.

Methods: A sleep-monitoring headband and self-reported questionnaire were used to assess sleep. Quantitative sensory testing was conducted on Monday and Friday, including heat, cold, pressure pain thresholds, tonic pain summation and conditioned pain modulation.

Results: A total of 26 healthy young (23.9 ± 0.9 years) women were included. Repeated measures ANOVAs revealed significant sleep variation across the week, including differences in N3 sleep stage duration ($M = 89.2 \pm 5.42$ min; $p = 0.022$, lowest on Friday and Sunday nights), bedtime ($M = 00{:}56\,AM \pm 0.29$; $p = 0.038$, latest on Friday vs. Sunday night) and wake-up time ($M = 07{:}04\,AM \pm 0.30$; $p = 0.007$ latest on Saturday vs. Monday morning). With most changes affecting Sunday night and Monday morning, pain sensitivity was higher on Monday compared to Friday, with a lower heat pain threshold ($B = -11.89$; $p = 0.002$) and increased heat pain summation ($B = 1.65$; $p < 0.001$).

Conclusions: The results showed higher heat pain hyperalgesia on Mondays due to weekly sleep variation. Since sleep is a modifiable factor, maintaining a consistent sleep schedule throughout the week could benefit pain management, particularly in chronic pain patients with less effective pain modulatory pathways.

Statement of Significance: How weekly sleep variations in real life between weekends and weekdays affect pain perception has not been studied before. This paper provides the first evidence that natural weekend–weekday sleep alterations, including shifts in bedtime and wake-up time over the weekend and the transition back on Sunday night, heighten pain sensitivity on Monday—known as the 'Monday effect'. The compromised pain pathways on Monday underscore the importance of maintaining a consistent sleep schedule throughout the week, potentially benefiting patients with chronic pain.

Study Preregistration Statement: The authors have nothing to report.

Natalia Egorova-Brumley and Amy S. Jordan Equal contribution senior author.

If the components of the research methodology needed to reproduce the reported procedure(s) and analyses will be publicly available.

1 | Introduction

Two-thirds of young adults experience weekday versus weekend sleep variations (Valdez et al. 1996), averaging 6.7 h on weekdays and 7.4 h on weekends (Breslau et al. 1997). Inconsistent sleep patterns, such as delayed bedtimes and sleeping in late on weekends, are commonly reported (Crowley and Carskadon 2010; Taylor et al. 2008; Valdez et al. 1996). Greater sleep variability on the Sleep Regularity Index (SRI) has been found to be associated with a higher all-cause mortality risk (Windred et al. 2023) and is a predictor of pain-related diseases (Chakradeo et al. 2018; Finan et al. 2013; Rouhi et al. 2024). This may be due to circadian misalignment, which can disrupt endogenous pain modulatory circuits that regulate pain processing and shape pain perception (Bumgarner et al. 2021; Nahman-Averbuch and King 2022), or be due to insufficient sleep, as evidence shows that even slight reductions in sleep can heighten pain sensitivity in pain-free individuals (Haack et al. 2023).

Two systematic reviews have highlighted the hyperalgesic effects associated with various forms of experimental sleep loss in healthy individuals (Chang et al. 2022; Rouhi et al. 2023). In our own work, total sleep deprivation had the greatest effect in lowering pain thresholds (heightened pain sensitivity), followed by sleep restriction and fragmented sleep. Furthermore, the impact of experimental sleep loss measured in healthy individuals with experimental measurement of pain sensitivity extends across different pain outcomes, with the heat pain threshold being the most prominently affected, followed by the pressure and then cold pain thresholds (Rouhi et al. 2023). Additionally, experimental sleep loss appears to affect pain-modulating circuits within the central nervous system, as indicated by proxy measurements such as conditioned pain modulation (Ramaswamy and Wodehouse 2021) (pain inhibition) and temporal summation (pain facilitation). Notably, these impairments were observed mostly in experimentally sleep-deprived females, with no such effects observed in sleep-deprived males (Rouhi et al. 2023).

Despite the clear detrimental hyperalgesic effect of experimental sleep loss in females, it is unclear whether natural sleep fluctuations across the week affect pain perception. Considering the common trend of increased sleep amounts on the weekend, an investigation into whether weekend–weekday sleep inconsistency would affect pain sensitivity is warranted. Only a few studies on this topic have been conducted. One study of 40 healthy individuals (20 females) compared two nights of natural sleep and found no significant differences in pain assessments despite significant differences in total sleep and rapid eye movement (REM) duration between the nights (Karmann et al. 2018). Similarly, a study (Stroemel-Scheder et al. 2019) in 20 chronic musculoskeletal pain patients and 20 healthy controls (14 females) found no significant sleep-pain relationship. However, the lack of significant findings in these studies may be related to the inclusion of both men and women, while, as noted above, the effect of experimental sleep loss on pain seems to differ between sexes (Rouhi et al. 2023). As previously reported, experimental sleep loss has a sex-dependent effect on descending pain pathways, with increased vulnerability in females (Eichhorn et al. 2018; Rouhi et al. 2023; Smith Jr et al. 2018).

This study aimed to investigate the impact of sleep variability, particularly the difference between weekday and weekend sleep, on pain perception in women, who prior studies have shown have a higher prevalence of chronic pain conditions (Petrov et al. 2015). Specifically, we sought to understand the role of weekday–weekend sleep fluctuations on pain responses measured on Friday and Monday, respectively. We hypothesised that reduced weekday sleep will be associated with increased pain sensitivity on Friday compared to Monday.

2 | Methods

2.1 | Study Design and Recruitment Procedure

The study, approved by the University of Melbourne's Human Research Ethics Committee (ref number: 2023-25,168-43,267-6), recruited healthy, pain-free participants through flyers placed across the University of Melbourne main campus and on university platforms. Participants received an information sheet and consent form online, and upon providing written consent, they completed baseline questionnaires on general health (inquiring into drug, alcohol consumption, caffeine and smoking status), demographics and Pittsburgh Sleep Quality Index (PSQI) to assess eligibility. The PSQI was not part of the exclusion criteria; it was included solely to track self-reported sleep quality and provide additional descriptive information. After the initial screening, participants had their pain testing sessions scheduled and received the sleep headband. Upon completing the study, participants were reimbursed $120 AUD.

2.1.1 | Inclusion and Exclusion Criteria

Please refer to the CONSORT diagram for more information (Figure S1). All inclusion and exclusion criteria for this study were determined through self-report screening conducted prior to the study.

2.1.1.1 | Inclusion Criteria.

- Pain-free females aged between 18 and 35 years. The age restriction was applied to minimise age-related changes in pain sensitivity (Eltumi and Tashani 2017), specifically in thermal pain threshold (Edwards and Fillingim 2001).
- Normal sleepers (habitual 6–8 h of sleep), with no severe medical or psychiatric illnesses
- Free from any sleep disorders (self-reported)
- Nonsmokers and non-nicotine users.
- Individuals with low caffeine intake consume less than two cups of caffeine or equivalent per day.

2.1.1.2 | Exclusion Criteria. Individuals with the following conditions were excluded:

- Sleep disorders, ongoing chronic or acute pain
- Significant cardiorespiratory, neurological, psychiatric or critical medical conditions,
- A lifetime history of alcohol or substance abuse,

- Antidepressants or other medications that might affect sleep or pain over the last 6 months.

2.1.2 | Sample Size Estimation

We used G*Power 3.1.33 to conduct an a priori power analysis to estimate the sample needed for assessing sleep and pain differences over 2 days (Monday and Friday) as a within-subject analysis using repeated measures ANOVA. A small effect size ($f = 0.29$) was selected to estimate the sample size, yielding a critical F-value of 4.2. This calculation indicated that $N = 26$ participants would be required to detect a significant effect at $\alpha = 0.05$ with 80% power. This sample size is comparable to prior studies with 24 (Staffe et al. 2019) and 32 (Julien et al. 2005), and thus was deemed sufficient.

2.1.3 | Procedure

Nocturnal sleep variations were assessed over a week at the participants' homes using DREEM headbands (DREEM 2 or DREEM 3, Beacon Biosignals, Boston MA) (Arnal et al. 2020) and sleep diaries. Two near-identical pain sensitivity testing sessions were conducted in the University of Melbourne's pain laboratory on Mondays (representing weekend sleep) and Fridays (representing weekday sleep) between 9:00 AM and 11:00 AM. Consistency was ensured by using the same assessor, conducting sessions at the same time of day and following the same testing order. Sleep monitoring began either on Friday or Tuesday evening (counterbalanced to reduce order effects), as shown in Figure 1 and Table S1.

2.2 | Questionnaires and Surveys

2.2.1 | Pittsburgh Sleep Quality Index (PSQI)

This validated sleep questionnaire (Buysse et al. 1989) with 19 questions assesses sleep habits, duration and overall quality. An overall score of ≤ 5 indicates good sleep quality, and all subdimensions were used for further evaluation.

2.2.2 | Epworth Sleepiness Scale (ESS)

The ESS is an 8-item questionnaire (Johns 1991) that measures daytime sleepiness. It asks individuals to rate their likelihood of dozing off or falling asleep in different situations, using a scale from 0 (would never doze) to 3 (high chance of dozing).

2.2.3 | Beck Depression Inventory (BDI)

The 21-item self-report questionnaire (Jackson-Koku 2016) evaluates depressive symptoms using a 4-point scale from 0 (no symptom) to 3 (severe symptoms). Total scores range from 0 to 63, with 0–13 indicating minimal, 14–19 indicating mild, 20–28 indicating moderate and 29+ indicating severe depression.

2.3 | Evaluation of Pain

In each testing session, the pain assessments occurred in the following order: heat pain threshold, cold pain threshold, pressure pain threshold, tonic heat pain summation and conditioned pain modulation. To mitigate the order effects of prior pain (Karmann et al. 2018), we started with pain threshold assessments (slightly painful stimuli), followed by more long-lasting painful stimuli (heat pain summation) and conditioned pain modulation. A 2-min interval was implemented between each measurement to minimise carry-over effects. Each session took up to 1 h to complete.

2.3.1 | Equipment

The thermal pain thresholds (heat and cold) were assessed using the Medoc Pathway (MEDOC, Israel) (Eichhorn et al. 2018; Rolke et al. 2006; Schuh-Hofer et al. 2013). The device is equipped with a contact probe that has a surface area of 9 cm^2 and a cut-off temperature range of 0°C–50°C. To measure the pressure pain threshold, a Medoc pressure algometer with a probe diameter of 1.0 cm^2 was used.

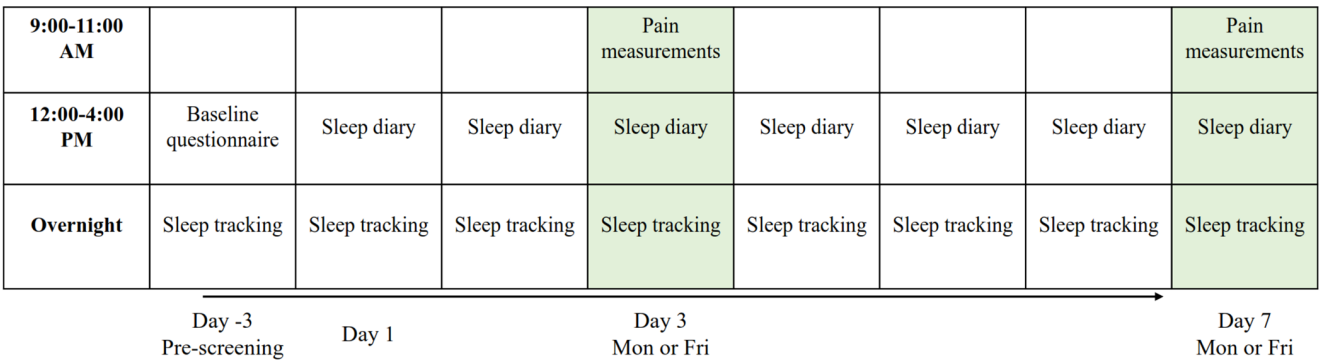

FIGURE 1 | Study design and flow. Participants were prescreened for up to three nights to adjust to the headband. Sleep tracking: Sleep data were collected using the Dreem headband. Pain measurements: quantitative sensory testing: heat, cold and pressure thresholds, conditioned pain modulation and tonic pain summation; Day 3 and Day 7 varied based on participant intake, either on Monday or Friday, to maintain order balance. Sleep diaries: Self-reported sleep questionnaires. Acronyms: BL: Baseline.

2.3.2 | Thermal Pain Thresholds (Heat and Cold)

The contact probe was placed on the dorsal section of the forearm, 10 cm from the elbow crest, on the nondominant hand. The temperature of the thermode started from a baseline of 32°C and increased/decreased at a rate of 0.5°C per second until the participant perceived the stimuli as painful and pressed the response button (Rødsjø 2020). This procedure was repeated three times for both heat and cold stimuli. The arithmetical mean of the three measurements was calculated to determine the heat pain threshold and cold pain threshold.

2.3.3 | Pressure Pain Threshold (PPT)

Pressure pain threshold was measured using a ramp-up rate of 50 kPa/s (starting at 0), with the probe placed on the thenar of the nondominant hand (Eichhorn et al. 2018). Participants were instructed to press a response button once the sensation turned painful. This procedure was repeated three times. The average of the three measurements was calculated to measure the pressure pain threshold.

2.3.4 | Tonic Pain Summation (TS) of Heat Pain

The procedure for assessing tonic suprathreshold hyperalgesia as described above for heat pain threshold was used to measure tonic pain summation (Granot et al. 2006; Matre et al. 2016; Ødegård et al. 2015). TS was quantified as the temperature that induced a pain intensity of 6 (pain6) on a 0–10 verbal NRS, where 0 represents 'no pain' and 10 represents the 'most imaginable pain', following the same paradigm used in previous studies (Granot et al. 2008; Matre et al. 2016). To briefly describe, it involved applying five different temperatures (43°C, 44°C, 45°C, 46°C and 47°C) for 7 s each. Participants were then asked to rate their pain on the NRS from 0 to 10. The temperature that corresponded to a pain rating of six on the NRS was selected and applied for 60 s, during which participants rated their pain levels every 10 s starting at 15 s. Participants who found the heat pain at 43°C intolerable were intended to be excluded from the study (Matre et al. 2016). However, no participants were excluded, as all participants tolerated temperatures above 43°C. Tonic suprathreshold hyperalgesia was calculated as the difference between pain ratings at 55 s compared to 15 s. Larger tonic pain summation values indicate more pronounced pain facilitation or intensification (i.e., Pain rating at 55 s minus Pain rating at 15 s > 0), while negative values suggest reduced pain perception or desensitisation (i.e., Pain rating at 55 s minus Pain rating at 15 s < 0).

2.3.5 | Conditioned Pain Modulation (CPM)

After assessing tonic pain summation, participants submerged their dominant hand in an ice water tank (0°C–2°C). (Eichhorn et al. 2018) Temperature checks were conducted before and during the test. Participants kept their hands in the cold water with fingers spread for as long as they could, up to 2 min as the conditioning stimulus. Upon removal, they rated the pain on a scale from 0 to 10. The duration of cold pain tolerance was recorded in seconds. After removing their hand from the ice water, the pressure pain threshold was remeasured (PPTpost). The difference between the pressure pain threshold before and after the conditioning stimulus was calculated to quantify CPM. Positive values for CPM indicate effective pain modulation (i.e., PPTpost—PPTpre > 0), whereas negative values suggest impaired CPM (i.e., PPTpost—PPTpre < 0).

2.4 | Evaluation of Sleep-Related Metrics

2.4.1 | Procedure

The procedure is outlined in Figure 1. During the prescreening phase, which occurred up to three nights before the study began, participants received the headband and had their pain testing sessions booked. Once they received the headband, participants were asked to wear the DREEM headband for up to 12 nights, with a minimum of three nights of wear before the first pain testing session. Most participants wore the headband for a total of seven nights, including three nights before each pain testing session. Due to scheduling changes; however, four participants wore the headband for more than seven nights to meet the requirements, with two participants wearing it for 12 nights. Participants also completed daily sleep diaries throughout the study.

The sleep diary questionnaires were administered via the SEMA application, which participants downloaded onto their smartphones. Participants received the questionnaire at the same time each day, starting at 12:00 PM, and the link was valid until 4:00 PM to encourage completion of the survey daily and to minimise the likelihood of retrospective reporting. Detailed information is in the Supplementary File (S.1.1).

2.4.1.1 | Sleep Diary. Participants completed a daily sleep diary to track subjective sleep patterns and experiences. Questions pertained to daytime naps (e.g., duration in minutes), bedtime, sleep onset latency (time to fall asleep in minutes), number and duration of awakenings and wake-up time. Participants also reported any daily pain experience (e.g., headaches and muscle pain) and rated their overall sleep quality on a 0–10 scale, with lower scores indicating poorer sleep quality.

2.4.1.2 | DREEM Headband. A sleep monitoring headband was used to monitor sleep, including both macro- and microstructural sleep parameters. Macrostructural parameters included total sleep time, sleep onset latency (SOL), wake after sleep onset (WASO), bedtime and wake-up time. Microstructural parameters included sleep stages, both nonrapid eye movement (NREM) stages (N1, N2 and N3) and rapid eye movement (REM) sleep. The DREEM headband has been validated against polysomnography with an 83.5% overall sleep staging accuracy compared to 86.4%, making it a reliable alternative for sleep monitoring (Arnal et al. 2020). All bedtimes and sleep data are reported based on the preceding day, even if sleep onset occurred after midnight (e.g., a bedtime of 2:00 AM on Saturday is reported as 'Friday night') throughout the manuscript.

2.5 | Statistical Analysis

Statistical analyses were performed using SPSS version 22 (IBM Corporation, Armonk, NY, USA), with a significance level

set at an alpha value of <0.05. Continuous variables are reported as mean ± SD, and graphical data as estimated marginal means ± SEM.

Repeated measures ANOVA measured the effect of the day of the week (7 days) on sleep within subjects. Pairwise comparisons used the Bonferroni correction to control for multiple testing. A general linear model assessed differences in pain outcomes over 2 days (Monday vs. Friday) with a within-subject effect. The Greenhouse–Geisser correction was applied for violations of sphericity (Mauchly's test, $p<0.05$).

A mixed-effects model using restricted maximum-likelihood estimation was employed with a within-participant design (Monday vs. Friday), incorporating sleep parameters as predictors and pain outcomes as the dependent variable. All models controlled for BMI, PSQI, age, BDI, race and menstrual cycle (Appelhans et al. 2013; Sherman and LeResche 2006; Song et al. 2011). Nonsignificant covariates were removed. Due to the small sample size, sleep parameters were analysed separately as predictors in the regression analysis, as previously reported (Karmann et al. 2018; Stroemel-Scheder et al. 2019).

3 | Results

3.1 | Participants Characteristics

All demographic and descriptive information is presented in Table 1. The study included 26 female participants, with an average age of 23.9 years (SD = 0.96) and a mean BMI of 22.5 (SD = 0.69), mostly within the normal BMI range, except for two participants with higher values (BMI 30.12 and 28.83). The sample's racial background was primarily Asian (65.4%), followed by White (26.9%) and Black (7.7%).

TABLE 1 | Descriptive data ($n=26$).

Measures	Baseline
Age	23.92 (0.96)
BMI	22.50 (0.69)
Racial background	
Asian	17 (65.4%)
White	7 (26.9%)
Black	2 (7.7%)
ESS	9.88 (1.08)
PSQI	
Subjective sleep quality	1.54 (0.159)
Sleep latency	1.21 (0.170)
Sleep duration	1.08 (0.190)
Habitual sleep efficiency	0.79 (0.199)
Sleep disturbance	1.83 (0.544)
Sleeping medication	0.38 (0.168)
Daytime dysfunction	1.96 (0.29)
Overall	7.58 (0.68)
BDI	11.81 (1.80)

Note: Results are presented as *M* (SD).
Abbreviations: BDI, Beck Depression Inventory; BMI, Body mass index; ESS, Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index.

Participants showed mild daytime sleepiness ($M=9.88$, SD = 1.08) and poor sleep quality (PSQI total score $M=7.58$, SD = 0.68). Additionally, the Beck Depression Inventory (BDI) score averaged 11.81, indicating minimal depressive symptoms within the sample.

Regarding menstrual cycle phases, the sample included participants in various stages: menstruation (7.7%), follicular (23.1%), ovulation (3.8%) and luteal (65.4%) on Monday and comparable proportions on Friday (Table S1). Health-related symptoms reported across the week included low incidences of restless legs, headaches, joint pain and muscle pain, with most participants not experiencing these symptoms consistently (Table S2).

The first pain testing session was conducted on Monday in 12 participants, comprising 46.2% of the group, while 14 participants (53.8%) had their first pain testing session on Friday (Table S1). The starting day (Monday vs. Friday) was included as a dummy variable in the model for all pain parameters and did not significantly affect the results (all $p>0.05$). The data supporting the findings of this study are available upon request from the corresponding author.

3.2 | Sleep Variability Across the Week

3.2.1 | Objective Sleep Measurements

Descriptive data of all sleep parameters are displayed in Figure 2. A repeated measures ANOVA, with each day of the week as a factor (7 days), was conducted using Pillai's trace to detect variations across the week. Pairwise comparisons based on mean differences were then used to identify significant differences between each pair of days. ANOVA revealed significant differences in N3 sleep stage (Figure 2), wake duration, bedtime and wake-up time (Figure 3) across the week. No other changes were found to be significant ($p>0.1$).

The duration of the N3 sleep stage significantly varied across the week ($M=89.2\pm5.42$ min; $F=3.24$; 95% CI: 78.08 to 100.48; $p=0.022$; power = 0.82; Figure 2) with the lowest values observed on Friday ($M=82.60\pm6.2$ min) and Sunday nights ($M=82.70\pm7.4$ min) compared to Saturday night ($M=96.3\pm6.9$ min). These differences remained statistically significant after adjusting for multiple comparisons using the Bonferroni method ($F=3.2$; $p=0.022$; power = 0.82). Wake duration varied across the week ($M=37.83\pm3$ min; $F=2.88$; 95% CI: 31.04–44.20; $p=0.048$; power = 0.71). However, the results did not withstand Bonferroni correction ($F=0.85$; $p=0.55$; power = 0.23).

Additionally, bedtime significantly varied across the week ($M=00{:}56\,AM\pm0.29$ min; $F=3.03$; 95% CI: 00:06–01:17; $p=0.038$; power = 0.75; see Figure 3). Pairwise comparisons indicated that this variation was primarily driven by differences between Friday and Sunday night bedtimes (mean difference $=0.76\pm0.2$ h; 95% CI: 0.051–1.48; $p=0.028$). The observed differences remained statistically significant following Bonferroni adjustment ($F=2.80$; $p=0.049$; power = 0.71; see

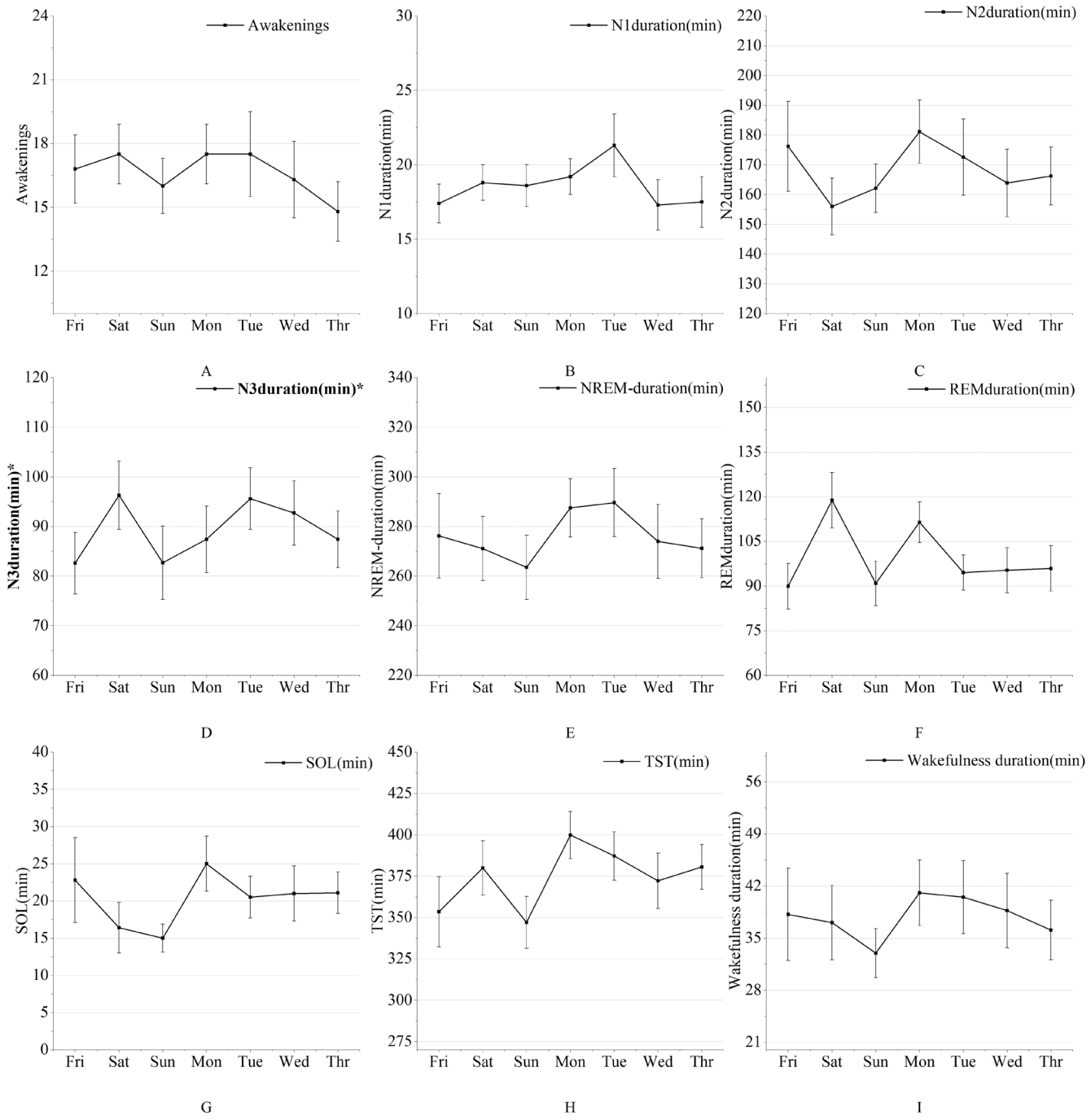

FIGURE 2 | Objective sleep measurements across the week. Estimated marginal means and standard errors for sleep parameters across the week. All sleep data were reported according to the preceding day, even if sleep onset occurred after midnight (e.g., a bedtime of 2:00 AM on Saturday was reported as 'Friday night'). Panel A represents the number of awakenings; panels B to F represent the duration of each sleep stage in minutes. Panel G represents SOL (sleep onset latency) in minutes. Panel H represents TST (total sleep time) in minutes. Panel I represents the duration of wakefulness over the night in minutes. Noted. Only N3 sleep stage duration (panel D) significantly differed across the week (F = 3.24; CI: 74.46 to 99.284; $p = 0.030$; power = 0.78) which has been bolded in the figure. Asterisks (*) indicate statistically significant.

Figure 3). Similarly, wake-up time significantly varied across the week ($M = 07{:}04 \pm 0.30$ min; $F = 4.48$; 95% CI: 06:40–08:03; $p = 0.007$; power = 0.92; see Figure 3). Pairwise comparisons indicated that this variation was primarily driven by differences in wake-up times between Saturday and Monday morning (mean difference = 0.99 ± 0.44 h; 95% CI: 0.067–1.92; $p = 0.037$). These results remained significant after Bonferroni correction ($F = 4.4$; $p = 0.007$; power = 0.92).

3.2.2 | Subjective Sleep Measurements

Descriptive data of all sleep parameters can be found in Table 2. ANOVA for repeated measures revealed significant differences in bedtime and wake-up time across the week. No other changes were found to be significant ($p > 0.1$). Bedtime significantly varied across the week ($M = 00{:}40$ AM ± 0.26 min; F = 3.02; 95% CI: 00:28–01:16; p = 0.038; power = 0.74; Figure S2) and BMI was found to be a

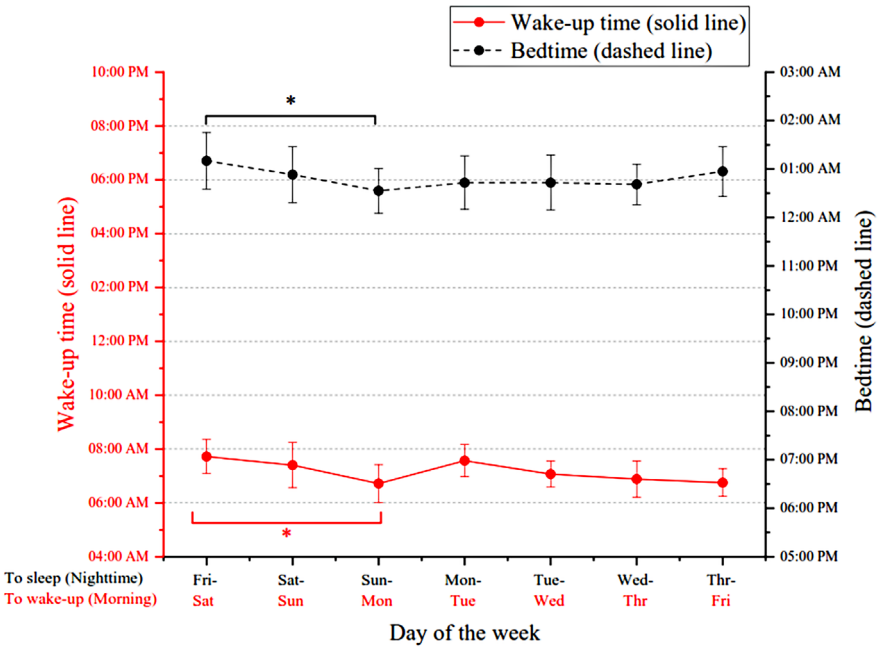

FIGURE 3 | Objective bedtime and wake-up time across the week. Mean values and standard error for bedtimes and wake-up time across the week, dashed and solid lines respectively. Bedtimes were reported according to the preceding day, even if sleep onset occurred after midnight (e.g., a bedtime of 2:00 AM on Saturday was reported as 'Friday night'). Asterisks (*) indicate statistically significant pairwise comparisons. Across all nights, the only two pairs showing statistically significant differences in bedtime and wake-up time were from Friday to Saturday and Sunday to Monday.

significant covariate of bedtime across the week ($F=4.9$, $p=0.006$, power = 0.93). Pairwise comparisons did not reveal any significant differences between any 2 days driving these results. However, the observed differences did not withstand Bonferroni adjustment ($F=0.52$; $p=0.77$; power = 0.15). Similarly, wake-up time significantly varied across the week (M = 07:21 ± 0.30 min; $F=3.08$; 95% CI: 06:18–07:44; $p=0.027$; power = 0.80; see Figure S2). Pairwise comparisons did not reveal any significant differences between any 2 days that could account for these results. However, the observed differences remained significant after Bonferroni adjustment ($F=3.08$; $p=0.02$; power = 0.80).

Sleep quality significantly varied across the week (M = 5.86 ± 0.35 min; $F=2.96$; 95% CI: 5.12–6.5; $p=0.031$; power = 0.78; Table 2). Pairwise comparisons revealed that significant differences were detected between Thursday night and Friday night, which may primarily contribute to the observed variations (mean difference = 1.53 ± 0.43; 95% CI: 0.71–3; $p=0.033$). The differences in sleep quality over the week remained statistically significant after Bonferroni adjustment ($F=2.96$; $p=0.031$; power = 0.78). The results indicated lower sleep quality over the weekend and on Sunday night. Towards the end of the week, there was an increase in sleep quality observed on Wednesday and Thursday, but it decreased again on Friday.

3.2.3 | Comparisons Between Objective Versus Subjective Sleep Metrics

A detailed comparison of sleep metrics reported by the DREEM headband and subjective sleep diary is provided in Table S3. A significant discrepancy was observed between individuals' subjective perception of overnight wake duration, as reported in sleep diaries, and the objective measurements obtained using the DREEM headband (mean difference obj-sub = 30.94 ± 3.61 min; $F=70.69$; 95% CI: 23.5–38.34; $p<0.001$; power = 1; Table S3).

3.3 | Fatigue, Alertness and Mood Level

No statistically significant differences were observed in self-reported mood, alertness and fatigue levels across the week (Table 2). Mood and alertness levels remained relatively stable throughout the week, with only minor fluctuations. Fatigue levels varied more noticeably, being higher on weekends and Monday, decreasing mid-week and rising again on Friday. For more details, please refer to supplementary S.1.2.

3.4 | Comparing Sleep the Night Before Pain Testing Sessions (Monday Versus Friday)

No sleep parameter was found to be statistically different between Sunday night and Thursday night. While differences were not statistically significant (lowest $p=0.06$), total sleep time (TST) tended to be shorter and the percentage of N1 sleep tended to be longer on Sunday night compared to Thursday night. All objective sleep data are displayed in Figure 2.

3.5 | Comparing Pain Changes: Monday Versus Friday

Descriptive data on all pain outcomes can be found in Table 3. How covariates affect all pain parameters is provided in Table S4, with further details in the supplementary file (S.1.3).

TABLE 2 | Subjective sleep parameters across the week.

Measures	To sleep (night time) To wake-up (morning)	Fri–Sat	Sat–Sun	Sun–Mon	Mon–Tue	Tue–Wed	Wed–Thu	Thu–Fri
Time awake[a]		17.61 (3.7)	17.95 (5.04)	19.19 (5.52)	18.81 (3.14)	18.61 (1.9)	14.14 (2.1)	13.38 (1.75)
Nb. of awakening		1.22 (1.02)	1.09 (1.15)	1 (1.2)	1.5 (1.5)	1.31 (1.21)	1.04 (1.21)	1.5 (1.6)
Wake-up time[a]		07:21 (2)	07:31 (1.64)	06:42 (1.6)	07:07 (1.5)	07:2 (1.42)	07:09 (1.65)	06:58 (1.9)
Bedtime[a]		00:56 (1.6)	00:09 (1.7)	00:10 (1.6)	00:10 (1.39)	00:09 (1.5)	00:09 (1.2)	00:27 (1.3)
Mood		6.15 (2.01)	6.57 (2.23)	5.88 (2.14)	6.23 (1.79)	6.34 (1.93)	6.30 (1.80)	6.00 (2.31)
Alert		5.80 (2.15)	5.88 (2.30)	5.76 (2.19)	6.11 (1.88)	6.30 (2.09)	6.03 (2.08)	5.46 (2.37)
Fatigue		9.88 (7.57)	7.61 (6.51)	8.76 (7.56)	8.42 (7.53)	7.5 (7.5)	7.84 (7.99)	9.11 (7.22)
Sleep quality		5.76 (2.08)	5.84 (2.37)	5.65 (2.36)	5.65 (2.48)	6.34 (2.15)	6.65 (1.76)	5.11 (2.56)

Note: Results are presented as *M* (SD); All bedtimes are reported based on the preceding day, even if sleep onset occurred after midnight (e.g., a bedtime of 2:00 AM on Saturday is reported as 'Friday night').
[a]Indicates a significant difference between Monday and Friday ($p < 0.05$); mood, alertness and sleep quality ranging from 0 to 10, and a higher score means better.

TABLE 3 | Descriptive data for pain outcomes ($n = 26$).

Pain measurements	Monday	Friday
Heat pain threshold (°C)[a]	43.66 (0.64)	44.33 (0.53)
Pressure pain thresholds post (Kpa)[b]	337.8 (24.2)	378.5 (30.8)
Pressure pain threshold (Kpa)	359 (32.2)	380.2 (30.2)
Tonic pain summation[a]	0.38 (0.37)	−0.88 (0.41)
Conditioned pain modulation (Kpa)	−23.5 (19.1)	3.55 (14.5)
Cold pain threshold (°C)	12.19 (1.7)	11.56 (1.5)
Cold pain tolerance (sec)	37.8 (6.5)	31.1 (3.8)
Cold pain rating (NRS)	7.8 (0.20)	7.74 (0.26)

Note: Results are presented as M (SD). Tonic pain summation > 0 indicates pain facilitation (intensified pain perception) and < 0 indicates decreased pain summation (dampened pain perception). CPM negative values mean dysfunctional pain inhibition, while positive values mean effective pain inhibition.
[a]Indicates a significant difference between Monday and Friday ($p < 0.05$).
[b]PPT post involves re-evaluating the pressure pain threshold after inducing conditioning stimuli (ice water tank).

3.5.1 | Pain Thresholds

Heat pain threshold was significantly lower on Monday compared to Friday ($B = -11.89$; SE = 3.89; $p = 0.002$), but no effect was observed for cold ($B = -0.71$; SE = 2.57; $p = 0.781$) or pressure pain threshold ($B = 0.319$; SE = 28.9; $p = 0.99$). Menstrual cycle phases were included as covariates in the pain models to account for potential effects on pain outcomes, though hormonal levels were not measured to confirm specific phases (Table S4). Notably, the follicular and ovulation phases showed associations with certain pain thresholds: the follicular phase was linked to a lower cold pain threshold, while the ovulation phase was associated with an increased pressure pain threshold. By including them in the respective model analyses, we only control for the potential phase-related effects on pain sensitivity, and no conclusion on how each phase affects pain can be made. For further information, please refer to the supplementary file (S1).

3.5.2 | Dynamic Pain Measurements

3.5.2.1 | Tonic Pain Summation. A significant day effect was observed on tonic pain summation between Monday and Friday ($B = 1.65$, SE = 0.49, $p < 0.001$; Figure 4). On average on Monday, pain summation occurred, where the perception of a given painful stimulus consistently intensified, while on average on Friday, perceived pain perception decreased gradually over the 1 min of pain testing.

3.5.2.2 | Conditioned Pain Modulation. No significant day effect was observed on conditioned pain modulation between Monday and Friday ($B = -26.02$, SE = 22.8, $p = 0.25$).

3.5.2.2.1 | Prediction of Pain Outcomes by Sleep Parameters. Descriptive data for all sleep parameters as predictors of pain outcomes are provided in Table 4.

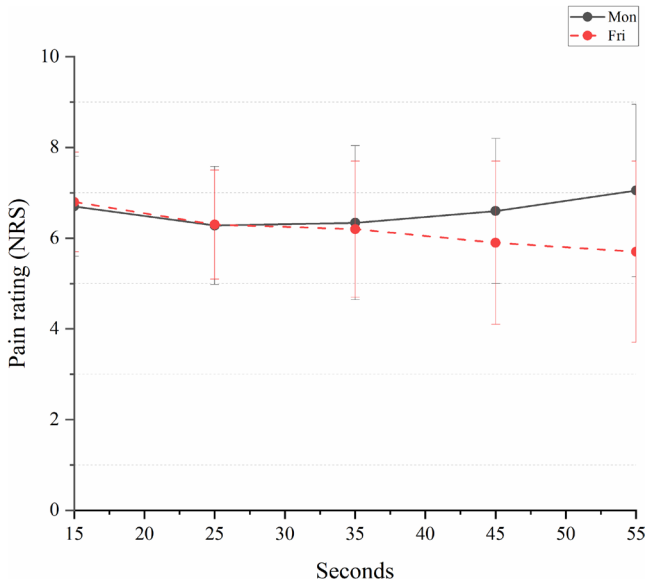

FIGURE 4 | Tonic pain summation on Monday versus Friday. Means and standard errors for tonic summation on Monday and Friday (sold and dashed lines respectively). *Note:* In the figure, the y-axis for pain rating given to painful stimuli is on a scale from 0 to 10, with the x-axis representing the initial (15 s) and final (55 s) pain ratings during heat painful stimuli. Both testing sessions displayed consistent initial heat pain thresholds (HPT), but a noticeable difference between the 2 days emerged over time. Furthermore, it is noteworthy that each 1-unit increase in pain rating corresponds to a 1° decrease in pain threshold.

3.5.2.3 | **Heat Pain Threshold.** An increase in the proportion of N3 sleep, as well as REM sleep, and shorter time to enter N2, N3 and REM sleep stages were associated with increased pain thresholds, while decreased sleep efficiency was a predictor of lower heat pain threshold.

3.5.2.4 | **Tonic Pain Summation.** Longer sleep duration, increased N3 sleep duration and more NREM sleep were associated with less pain facilitation, while lower sleep efficiency was linked to greater tonic pain summation.

4 | Discussion

This study examined the impact of weekly sleep fluctuations on pain perception in healthy females. Contrary to our expectation of reduced sleep on weekdays compared to weekends, we found minimal differences in sleep metrics across the week, with sleep duration remaining largely constant. Participants averaged only 6 h and 23 min of sleep per night, with a mean bedtime of approximately 20 min past midnight. Sleep patterns were consistent during weekdays, but notable variations occurred over the weekend. On Friday nights, participants went to bed later and slept in for an additional hour. However, on Sunday nights, they went to bed earlier and woke up earlier on Monday morning, resulting in the shortest sleep duration of the week. This shift was accompanied by a reduction in N3 sleep on both Friday and Sunday nights.

This pattern aligns with previous studies that report similar weekday–weekend sleep fluctuations among university students (Steptoe et al. 2006). According to a self-report study, undergraduate students had an average sleep duration of 6.9 h on weekdays (Steptoe et al. 2006), increasing to 8 h over weekends, with delayed bedtime and sleeping in over the weekend (Crowley and Carskadon 2010; Taylor et al. 2008; Valdez et al. 1996). However, our study demonstrated a smaller variation, with only a 1-h difference compared to weekdays. It is unclear why the increase in total sleep time over the weekend was less pronounced in our population, but it may be at least in part due to a high percentage of international students who might have part-time jobs on weekends. Notably, this weekend-to-weekday transition, commonly referred to as the 'Monday effect', was associated with heightened pain sensitivity, characterised by decreased heat pain thresholds and increased pain summation on Monday. Regression analyses indicated that NREM Stage 3 and sleep efficiency were associated with these changes. While these findings highlight a unique interaction between weekend-to-weekday sleep transitions and pain perception, other factors unrelated to these fluctuations may also influence the observed Monday pain sensitivity, as discussed below.

It is noteworthy that the current study revealed elevated PSQI scores across the sample, with a mean of 7.5 (SD = 0.68), exceeding the standard threshold of 5, which indicates poor sleep quality (Buysse et al. 1989). However, these findings align with previous research on university students, which reported a mean PSQI score of 10.41 (SD = 3.51) (Schlarb et al. 2017) highlighting that university students generally experience poorer sleep quality compared to the general population.

4.1 | Monday: Special Considerations for Sleep and Heat Pain

While we did not observe significant differences between Sunday night and Thursday nights with regard to sleep patterns, the observed sleep fluctuations across the week were primarily driven by differences over the weekend (Friday to Saturday night) and the start of the work week (Sunday to Monday night). Pairwise comparisons showed significant differences between Friday night and Sunday night regarding bedtime, wake-up time and, notably, a substantial reduction in N3 sleep stage duration on Sunday night compared to other weekdays. Despite participants reverting their sleep schedule to align with weekday demands on Sunday nights, with an earlier bedtime compared to other nights, their earlier wake-up time on Monday morning resulted in the shortest total sleep duration of the week, accompanied by a reduction in N3 sleep duration. Following the distinctive sleep pattern noticed on Sunday night, we observed reduced heat pain threshold and heat pain facilitation on Monday as compared to Friday.

The distinct 'Monday effect' on pain may be influenced by a shift in circadian rhythm. As previously reported, delayed bedtimes and wake-up times over the weekend resulted in a delayed circadian rhythm for the following week (Taylor et al. 2008). Another study also reported that a 2-h delay in bedtime and wake-up time over the weekend affected Sunday night's sleep pattern due to a 31.6-min delay in the circadian phase (Crowley and Carskadon 2010).

TABLE 4 | Prediction of heat pain threshold and tonic pain summation by sleep parameters.

	Heat pain threshold				Tonic pain summation			
Predictors	**B**	**Std Err**	**Wald chi-square**	***p***	**B**	**Std Err**	**Wald chi-square**	***p***
TST[a]	0.001	0.000	0.024	0.87	0.005	0.000	4.6	0.032
SOL[a]	0.036	0.03	1.3	0.24	0.001	0.032	000	0.98
WASO	0.037	0.012	8.1	0.004	0.013	0.013	0.88	0.34
Wakefulness[a]	0.03	0.017	2.9	0.08	0.015	0.012	1.63	0.19
Bedtime	0.23	0.29	0.63	0.42	0.31	0.10	9.42	0.002
Wake-up time	0.16	0.25	0.417	0.51	0.086	0.114	0.56	0.45
N1 duration[a]	0.01	0.05	0.040	0.84	0.003	0.038	0.008	0.92
N2 duration[a]	0.006	0.012	0.22	0.63	0.006	0.0047	1.85	0.17
N3 duration[a]	0.018	0.012	2.05	0.15	0.021	0.010	3.861	0.049
NREM[a]	0.003	0.008	0.08	0.76	0.009	0.003	9.07	0.003
REM duration[a]	0.017	0.011	1.9	0.15	0.002	0.006	0.078	0.78
N1 stage[b]	0.089	0.27	0.10	0.74	2.00	0.15	1.69	0.19
N2 stage[b]	0.007	0.02	0.07	0.78	0.008	0.015	0.30	0.58
N3 stage[b]	0.09	0.046	3.83	0.05	0.039	0.034	1.29	0.25
REM[b]	0.084	0.039	4.50	0.034	0.017	0.02	0.52	0.46
NREM[b]	0.06	0.033	3.77	0.05	0.013	0.017	0.56	0.45
N2 latency[a]	0.33	0.10	9.5	0.002	0.035	0.052	0.43	0.50
N3 latency[a]	0.064	0.02	5.66	0.017	0.019	0.018	1.007	0.31
REM latency[a]	0.009	0.01	0.77	0.37.	0.008	0.0054	2.20	0.13
Sleep efficiency[b]	−0.15	0.063	5.71	0.017	−0.09	0.042	5.28	0.021

Note: Prediction of heat pain threshold and tonic pain summation by sleep parameters. TST, total sleep time; SOL, sleep onset latency; WASO, wakefulness after sleep onset.
[a]Indicates the duration of each sleep parameter in minutes.
[b]Represents the values in percentage.

Another study (Elfering et al. 2020) has shown that, even when individuals go to bed at their usual bedtime on Sunday night, the shift in circadian rhythm caused by later wake-up times over the weekend (Saturday and Sunday morning) resulted in shorter sleep duration on Sunday night and increased fatigue on Monday morning (Elfering et al. 2020). A recent systematic review highlighted the direct effect of a shifted sleep–wake cycle on heightened pain sensitivity by influencing the periaqueductal grey, rostral ventromedial medulla, locus coeruleus and the endogenous opioid system in both humans and mice (Bumgarner et al. 2021). Although the shifted circadian rhythm likely contributed to the heightened pain sensitivity observed on Monday, we did not investigate circadian rhythm shifts in the current study and so cannot be sure but believe the possibility warrants further research.

Another factor that may contribute to the distinct 'Monday effect' is a forced early awakening required by work or university commitments, which restricts sleep more than any other night of the week and may not reflect the true effects of weekend sleep. Thus, the observed 'Monday effect' cannot be solely attributed to the 'unrestricted' weekend sleep pattern and may be confounded by the return to a weekday schedule. Future studies measuring pain on Saturday/Sunday would better capture the effects of 'unrestricted' weekend sleep on pain sensitivity.

It is noteworthy that the heightened pain sensitivity observed on Monday was evident only for heat pain, while other pain types, including pressure pain, cold pain and pressure-based conditioned pain modulation, showed no significant day effect.

4.2 | Specificity of Heat Pain Sensitivity to Sleep Changes

Drastic experimental sleep loss, such as total sleep deprivation, has been shown to affect multiple pain modalities (Kourbanova et al. 2022; Rouhi et al. 2023); however, results from the current study indicate that heat pain appears to be more sensitive and responsive to minor forms of sleep variation, such as weekday vs. weekend sleep alterations. Our previous systematic review (Rouhi et al. 2023) found that heat pain was significantly more

affected by experimental sleep loss than other pain modalities. Total sleep deprivation resulted in a large decrease in heat pain threshold, followed by smaller effects on mechanical pain thresholds and increased cold pain sensitivity, with no effect on pressure pain thresholds (Rouhi et al. 2023). The reviews concluded that experimental sleep loss has a dose-dependent effect (Chang et al. 2022; Rouhi et al. 2023) on pain hyperalgesia, with heat pain being the only modality significantly affected across all sleep loss conditions. Consistent with the current study, showing no day-of-the-week effect on pressure pain threshold and pressure-based conditioned pain modulation, two previous studies (Karmann et al. 2018; Stroemel-Scheder et al. 2019) also found that natural sleep fluctuations over two nonconsecutive nights did not predict pain changes. Their (Karmann et al. 2018) analyses indicated only a weak association between night-to-night sleep variations and experimentally measured pain. Our results further suggest that pressure and cold pain modalities are less sensitive to small, nonpathological nocturnal sleep changes compared to heat pain (Karmann et al. 2018; Stroemel-Scheder et al. 2019). Irwin et al. demonstrated that only the heat pain threshold was affected by experimental sleep disturbance (Irwin et al. 2023) but no other pain modalities were influenced. Consistent with the current study, they demonstrated that the reduction in N3 sleep is associated with increased heat pain sensitivity (Irwin et al. 2023). Reduction in the N3 sleep stage resulted in increased cellular inflammation, including elevated levels of interleukin 6 and tumour necrosis factor, which in turn may have induced heat pain hyperalgesia (Irwin et al. 2023).

Another potential mechanism for the greater sensitivity of heat pain might be due to the molecular classification of nociceptors, which detect different pain modalities. Nociceptors express specific channels, such as TRPV1 for heat, TRPM8 for cold, ASICs for acidic environments and TRPA1 for various chemical irritants (Basbaum et al. 2009; Julius and Basbaum 2001). This specificity may explain why heat pain, primarily mediated by TRPV1 channels, is more responsive to sleep changes compared to other types of pain (Ozathaley et al. 2023). A study on patients with gastro-oesophageal reflux disease, a chronic gastrointestinal condition, showed that TRPV1 expression is significantly higher in patients with sleep disturbance (Liu et al. 2023). Moreover, a study in mice (Murillo-Rodríguez et al. 2017) demonstrated that administration of AA-5-HT, a TRPV1 blocker, increased NREMS and REM sleep and altered spectral power, supporting a potential role of TRPV1 in the sleep modulation of heat pain sensitivity (Murillo-Rodríguez et al. 2017). However, this study did not control for body temperature—a factor that may contribute to the observed effects, as TRPV1 antagonists are known to impact thermoregulation (Murillo-Rodríguez et al. 2017). Nonetheless, these findings might be an indicator of a possible role for TRPV1 in sleep-related modulation of heat pain sensitivity, potentially mediated through both direct and/or influenced by thermoregulatory mechanisms.

Notably, in the current study, the mean heat pain threshold was 43.61°C (SD = 3.21) and tonic heat pain summation was 44.30°C (SD = 2.64). These values align with the activation threshold of the TRPV1 channel, triggered around 43°C (Basbaum et al. 2009; Caterina et al. 1997). This suggests that the observed heat pain sensitivity might be linked to TRPV1 activation, though this was not investigated in the current study. Future studies should focus on the underlying mechanisms of heat pain hyperalgesia and how they are linked to sleep changes.

4.3 | Nonsleep–Related Factors Contributing to the 'Monday Effect' on Pain

Prior studies have identified a 'Monday effect' on pain conditions, with occupational injuries peaking on Mondays and decreasing throughout the week (Fontaneda et al. 2024). This pattern was consistent across various injuries and persisted after controlling for sex and age (Fontaneda et al. 2024; Johnson et al. 1998), with the most significant peak for back injuries (Fontaneda et al. 2024). Building upon previous studies, results from the current study suggest that the 'Monday effect' might also affect tonic pain summation and heat pain sensitivity.

The heat pain sensitivity observed on Monday could be attributed to various contributing factors beyond sleep patterns (Geva et al. 2023). For example, stress influences pain perception by reducing pain thresholds and increasing tonic pain summation (Geva et al. 2023; Jennings et al. 2014), intensifying the overall pain experience. Stress elevates cortisol levels, disrupts the HPA axis and induces hypoglycaemia, which can trigger cytokine release and heighten pain sensitivity, potentially contributing to stress-related pain disorders (Jennings et al. 2014). The 'Monday effect' in pain summation might be associated with the start of the week, classes or part-time jobs (Geva et al. 2023).

Other factors, like a decline in cognitive function, reduced performance, low mood and heightened feelings of drowsiness and fatigue which have been reported on Monday mornings (Taylor et al. 2008; Valdez et al. 1996), might have also contributed to the observed pain modulation impairment. However, in the current study, mood and fatigue did not differ between Monday and Friday. Therefore, we cannot draw a firm conclusion about the exact reasons for the observed pain impairment on Monday. These factors should be studied further to understand their individual and combined impact on pain.

4.4 | Limitations and Future Direction

Several limitations may affect the generalisability of our findings. The small sample size of 26 participants limited the statistical power for regression analysis, and future studies should increase the sample size (Karmann et al. 2018). Second, the exclusive recruitment of females means the results are not generalisable to males; future research should include males to explore sex differences in sleep patterns and pain outcomes (Khan et al. 2018; Rouhi et al. 2023). The predominantly young adult university student sample (aged 18–35) limits generalisability to older adults, whose pain modulation responses differ (Grashorn et al. 2013). Fourth, pain testing was conducted between 9:00 and 11:00 AM to minimise circadian rhythm influences (Hackett et al. 2020; Hagenauer et al. 2017; Ramaswamy and Wodehouse 2021) with the order of sessions counterbalanced. Fifth, the included participants had a high mean PSQI score, which deviates from the classification of 'normal sleepers'. This elevated score is likely attributable to the short sleep duration observed across the study sample. Future studies should consider adopting stricter PSQI cut-off criteria in

the inclusion process to reduce variability and enhance the generalisability of findings. Finally, the use of two devices, DREEM 2 and DREEM 3, might affect the results. In addition to addressing limitations, other future directions are: given the sleep pattern on Sunday night resembles that of working days (as participants transition back to the workweek), it may not optimally represent the weekend's sleep pattern. Friday and Saturday night sleep monitoring and measuring pain on Saturday or Sunday morning would better reflect weekend fluctuations compared to the Monday morning that was conducted here. To further understand the underlying mechanisms linking sleep patterns and pain processing to unravel the direct causes of the observed pain impairment on Monday, factors beyond sleep will need to be considered, such as the potential impact of stress associated with the first working day.

5 | Conclusion

This study provides novel insights into the effects of weekly sleep variability on pain perception in young, healthy women. The results highlight how weekday versus weekend sleep variations, particularly delayed bedtimes and later wake-ups over the weekend, influence pain sensitivity, with heightened heat pain sensitivity observed on Mondays compared to Fridays. Despite reverting to their weekday bedtime schedule on Sunday night, participants still showed modified pain processing on Monday. These findings suggest that minor shifts in sleep timing, especially over weekends, may impact pain processing, resulting in a distinct 'Monday effect' that challenges the typical notion of weekend recovery sleep. Since sleep is a modifiable factor, maintaining a consistent sleep schedule throughout the week may help reduce pain sensitivity, especially for those with impaired pain modulation responses, such as patients with chronic pain.

Author Contributions

Shima Rouhi: Contributed to conceptualisation, investigation, methodology, validation, visualisation and formal analysis; wrote the original draft; participated in review and editing; managed the project; and curated data. Natalia Egorova-Brumley: Contributed to conceptualisation, investigation and methodology; acquired funding; provided supervision and resources; managed the project; and participated in validation, visualisation and review/editing. Amy S. Jordan: Contributed to conceptualisation, investigation and methodology; acquired funding; provided supervision and resources; managed the project; and participated in validation, visualisation and review/editing.

Acknowledgements

S.R. was funded by the University of Melbourne (Melbourne Research Scholarship) doctoral studentship. N.E.B. was supported by the Australian Research Council FT230100235. Open access publishing facilitated by The University of Melbourne, as part of the Wiley - The University of Melbourne agreement via the Council of Australian University Librarians.

Conflicts of Interest

The authors declare no Conflicts of Interest.

Data Availability Statement

The original datasets generated over the course of this study are available upon request from the corresponding author.

References

Appelhans, B. M., I. Janssen, J. F. Cursio, et al. 2013. "Sleep Duration and Weight Change in Midlife Women: The SWAN Sleep Study." *Obesity (Silver Spring)* 21, no. 1: 77–84. https://doi.org/10.1002/oby.20251.

Arnal, P. J., V. Thorey, E. Debellemaniere, et al. 2020. "The Dreem Headband Compared to Polysomnography for Electroencephalographic Signal Acquisition and Sleep Staging." *Sleep* 43, no. 11: 1–13. https://doi.org/10.1093/sleep/zsaa097.

Basbaum, A. I., D. M. Bautista, G. Scherrer, and D. Julius. 2009. "Cellular and Molecular Mechanisms of Pain." *Cell* 139, no. 2: 267–284. https://doi.org/10.1016/j.cell.2009.09.028.

Breslau, N., T. Roth, L. Rosenthal, and P. Andreski. 1997. "Daytime Sleepiness: An Epidemiological Study of Young Adults." *American Journal of Public Health* 87, no. 10: 1649–1653. https://doi.org/10.2105/ajph.87.10.1649.

Bumgarner, J. R., W. H. Walker, and R. J. Nelson. 2021. "Circadian Rhythms and Pain." *Neuroscience and Biobehavioral Reviews* 129: 296–306. https://doi.org/10.1016/j.neubiorev.2021.08.004.

Buysse, D. J., C. F. Reynolds, T. H. Monk, S. R. Berman, and D. J. Kupfer. 1989. "The Pittsburgh Sleep Quality Index: A New Instrument for Psychiatric Practice and Research." *Psychiatry Research* 28, no. 2: 193–213. https://doi.org/10.1016/0165-1781(89)90047-4.

Caterina, M. J., M. A. Schumacher, M. Tominaga, T. A. Rosen, J. D. Levine, and D. Julius. 1997. "The Capsaicin Receptor: A Heat-Activated Ion Channel in the Pain Pathway." *Nature* 389, no. 6653: 816–824. https://doi.org/10.1038/39807.

Chakradeo, P. S., A. Keshavarzian, S. Singh, et al. 2018. "Chronotype, Social Jet Lag, Sleep Debt and Food Timing in Inflammatory Bowel Disease." *Sleep Medicine* 52: 188–195. https://doi.org/10.1016/j.sleep.2018.08.002.

Chang, J. R., S.-N. Fu, X. Li, et al. 2022. "The Differential Effects of Sleep Deprivation on Pain Perception in Individuals with or Without Chronic Pain: A Systematic Review and Meta-Analysis." *Sleep Medicine Reviews* 101695: 101695. https://doi.org/10.1016/j.smrv.2022.101695.

Crowley, S. J., and M. A. Carskadon. 2010. "Modifications to Weekend Recovery Sleep Delay Circadian Phase in Older Adolescents." *Chronobiology International* 27, no. 7: 1469–1492. https://doi.org/10.3109/07420528.2010.503293.

Edwards, R. R., and R. B. Fillingim. 2001. "Effects of Age on Temporal Summation and Habituation of Thermal Pain: Clinical Relevance in Healthy Older and Younger Adults." *Journal of Pain* 2, no. 6: 307–317.

Eichhorn, N., R.-D. Treede, and S. Schuh-Hofer. 2018. "The Role of Sex in Sleep Deprivation Related Changes of Nociception and Conditioned Pain Modulation." *Neuroscience* 387: 191–200.

Elfering, A., C. Gerhardt, D. Pereira, A. Schenker, and M. U. Kottwitz. 2020. "The Monday Effect Revisited: A Diary and Sleep Actigraphy Study." *Sleep and Vigilance* 4, no. 2: 167–176. https://doi.org/10.1007/s41782-020-00105-5.

Eltumi, H. G., and O. A. Tashani. 2017. "Effect of Age, Sex and Gender on Pain Sensitivity: A Narrative Review." *Open Pain Journal* 10, no. 1: 44–55.

Finan, P. H., B. R. Goodin, and M. T. Smith. 2013. "The Association of Sleep and Pain: An Update and a Path Forward." *Journal of Pain* 14, no. 12: 1539–1552. https://doi.org/10.1016/j.jpain.2013.08.007.

Fontaneda, I., M. A. Camino López, O. J. González Alcántara, and B. A. Greiner. 2024. "The "Weekday Effect": A Decrease in Occupational Accidents from Monday to Friday—An Extension of the "Monday Effect"." *BioMed Research International* 2024: 4792012–4792081. https://doi.org/10.1155/2024/4792081.

Geva, N., S. Golan, L. Pinchas, and R. Defrin. 2023. "Sex Effects in the Interaction of Acute Stress and Pain Perception." *Pain* 164, no. 3: 587–597. https://doi.org/10.1097/j.pain.0000000000002743.

Granot, M., Y. Granovsky, E. Sprecher, R.-R. Nir, and D. Yarnitsky. 2006. "Contact Heat-Evoked Temporal Summation: Tonic Versus Repetitive-Phasic Stimulation." *Pain* 122, no. 3: 295–305. https://doi.org/10.1016/j.pain.2006.02.003.

Granot, M., I. Weissman-Fogel, Y. Crispel, et al. 2008. "Determinants of Endogenous Analgesia Magnitude in a Diffuse Noxious Inhibitory Control (DNIC) Paradigm: Do Conditioning Stimulus Painfulness, Gender and Personality Variables Matter?" *Pain* 136, no. 1–2: 142–149. https://doi.org/10.1016/j.pain.2007.06.029.

Grashorn, W., C. Sprenger, K. Forkmann, N. Wrobel, and U. Bingel. 2013. "Age-Dependent Decline of Endogenous Pain Control: Exploring the Effect of Expectation and Depression." *PLoS One* 8, no. 9: e75629. https://doi.org/10.1371/journal.pone.0075629.

Haack, M., L. C. Engert, L. Besedovsky, et al. 2023. "Alterations of Pain Pathways by Experimental Sleep Disturbances in Humans: Central Pain-Inhibitory, Cyclooxygenase, and Endocannabinoid Pathways." *Sleep* 46, no. 6: 1–12. https://doi.org/10.1093/sleep/zsad061.

Hackett, J., K. E. Naugle, and K. M. Naugle. 2020. "The Decline of Endogenous Pain Modulation with Aging: A Meta-Analysis of Temporal Summation and Conditioned Pain Modulation." *Journal of Pain* 21, no. 5–6: 514–528. https://doi.org/10.1016/j.jpain.2019.09.005.

Hagenauer, M. H., J. A. Crodelle, S. H. Piltz, N. Toporikova, P. Ferguson, and V. Booth. 2017. "The Modulation of Pain by Circadian and Sleep-Dependent Processes: A Review of the Experimental Evidence." In *Women in Mathematical Biology*, 1–21. Springer International Publishing.

Irwin, M. R., R. Olmstead, M. F. Bjurstrom, P. H. Finan, and M. T. Smith. 2023. "Sleep Disruption and Activation of Cellular Inflammation Mediate Heightened Pain Sensitivity: A Randomized Clinical Trial." *Pain* 164, no. 5: 1128–1137. https://doi.org/10.1097/j.pain.0000000000002811.

Jackson-Koku, G. 2016. "Beck Depression Inventory." *Occupational Medicine* 66, no. 2: 174–175.

Jennings, E. M., B. N. Okine, M. Roche, and D. P. Finn. 2014. "Stress-Induced Hyperalgesia." *Progress in Neurobiology* 121: 1–18. https://doi.org/10.1016/j.pneurobio.2014.06.003.

Johns, M. W. 1991. "A New Method for Measuring Daytime Sleepiness: The Epworth Sleepiness Scale." *Sleep* 14, no. 6: 540–545. https://doi.org/10.1093/sleep/14.6.540.

Johnson, W. G., M. L. Baldwin, and R. J. Butler. 1998. "Back Pain and Work Disability: The Need for a New Paradigm." *Industrial Relations: A Journal of Economy and Society* 37, no. 1: 9–34. https://doi.org/10.1111/0019-8676.691998032.

Julien, N., P. Goffaux, P. Arsenault, and S. Marchand. 2005. "Widespread Pain in Fibromyalgia Is Related to a Deficit of Endogenous Pain Inhibition." *Pain* 114, no. 1–2: 295–302. http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=med6&NEWS=N&AN=15733656.

Julius, D., and A. I. Basbaum. 2001. "Molecular Mechanisms of Nociception." *Nature* 413, no. 6852: 203–210. https://doi.org/10.1038/35093019.

Karmann, A. J., C. Lauer, E. Ziegler, L. Killian, C. Horn-Hofmann, and S. Lautenbacher. 2018. "Associations of Nocturnal Sleep with Experimental Pain and Pain Catastrophizing in Healthy Volunteers." *Biological Psychology* 135: 1–7. https://doi.org/10.1016/j.biopsycho.2018.02.015.

Khan, J., O. Korczeniewska, R. Benoliel, M. Kalladka, E. Eliav, and C. Nasri-Heir. 2018. "Age and Gender Differences in Mechanically Induced Intraoral Temporal Summation and Conditioned Pain Modulation in Healthy Subjects." *Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology* 126, no. 2: 134–141. https://doi.org/10.1016/j.oooo.2018.03.021.

Kourbanova, K., C. Alexandre, and A. Latremoliere. 2022. "Effect of Sleep Loss on Pain-New Conceptual and Mechanistic Avenues." *Frontiers in Neuroscience* 16: 1009902. https://doi.org/10.3389/fnins.2022.1009902.

Liu, D., L. Xu, L. Chen, et al. 2023. "TRPV1 Is a Risk Factor for Sleep Disturbance in Patients with Gastro-Oesophageal Reflux Disease: A Case Control Study." *Scandinavian Journal of Gastroenterology* 58, no. 8: 844–855. https://doi.org/10.1080/00365521.2023.2190438.

Matre, D., M. R. Andersen, S. Knardahl, and K. B. Nilsen. 2016. "Conditioned Pain Modulation Is Not Decreased After Partial Sleep Restriction." *European Journal of Pain* 20, no. 3: 408–416. https://doi.org/10.1002/ejp.741.

Murillo-Rodríguez, E., V. Di Marzo, S. Machado, et al. 2017. "Role of N-Arachidonoyl-Serotonin (AA-5-HT) in Sleep-Wake Cycle Architecture, Sleep Homeostasis, and Neurotransmitters Regulation." *Frontiers in Molecular Neuroscience* 10: 152. https://doi.org/10.3389/fnmol.2017.00152.

Nahman-Averbuch, H., and C. D. King. 2022. "Disentangling the Roles of Circadian Rhythms and Sleep Drive in Experimental Pain Sensitivity." *Trends in Neurosciences* 45, no. 11: 796–797. https://doi.org/10.1016/j.tins.2022.09.003.

Ødegård, S. S., P. M. Omland, K. B. Nilsen, M. Stjern, G. B. Gravdahl, and T. Sand. 2015. "The Effect of Sleep Restriction on Laser Evoked Potentials, Thermal Sensory and Pain Thresholds and Suprathreshold Pain in Healthy Subjects." *Clinical Neurophysiology* 126, no. 10: 1979–1987. https://doi.org/10.1016/j.clinph.2014.12.011.

Ozathaley, A., Z. Kou, Y. Ma, et al. 2023. "NLRP3 Upregulation Related to Sleep Deprivation-Induced Memory and Emotional Behavior Changes in TRPV1−/− Mice." *Behavioural Brain Research* 440: 114255. https://doi.org/10.1016/j.bbr.2022.114255.

Petrov, M. E., B. R. Goodin, Y. Cruz-Almeida, et al. 2015. "Disrupted Sleep Is Associated with Altered Pain Processing by Sex and Ethnicity in Knee Osteoarthritis." *Journal of Pain* 16, no. 5: 478–490. https://doi.org/10.1016/j.jpain.2015.02.004.

Ramaswamy, S., and T. Wodehouse. 2021. "Conditioned Pain Modulation—A Comprehensive Review." *Neurophysiologie Clinique* 51, no. 3: 197–208.

Rødsjø, E. 2020. *Sleep and Pain: An EEG Study of How Sleep Affects Pain Perception [NTNU]*. Master's thesis in Neuroscience. https://ntnuopen.ntnu.no/ntnu-xmlui/bitstream/handle/11250/2669182/no.ntnu%3Ainspera%3A58261154%3A9250054.pdf?sequence=1&isAllowed=y.

Rolke, R., R. Baron, C. A. Maier, et al. 2006. "Quantitative Sensory Testing in the German Research Network on Neuropathic Pain (DFNS): Standardized Protocol and Reference Values." *Pain* 123, no. 3: 231–243.

Rouhi, S., N. Egorova-Brumley, and A. S. Jordan. 2024. "Chronic Sleep Deficiency and Its Impact on Pain Perception in Healthy Females." *Journal of Sleep Research* 34, no. 1: e14284. https://doi.org/10.1111/jsr.14284.

Rouhi, S., J. Topcu, N. Egorova-Brumley, and A. S. Jordan. 2023. "The Impact of Sleep Disturbance on Pain Perception: A Systematic Review Examining the Moderating Effect of Sex and Age." *Sleep Medicine Reviews* 71: 101835. https://doi.org/10.1016/j.smrv.2023.101835.

Schlarb, A. A., A. Friedrich, and M. Claßen. 2017. "Sleep Problems in University Students – An Intervention." *Neuropsychiatric Disease and Treatment* 13: 1989–2001. https://doi.org/10.2147/NDT.S142067.

Schuh-Hofer, S., R. Wodarski, D. B. Pfau, et al. 2013. "One Night of Total Sleep Deprivation Promotes a State of Generalized Hyperalgesia: A Surrogate Pain Model to Study the Relationship of Insomnia and Pain." *Pain* 154, no. 9: 1613–1621. https://doi.org/10.1016/j.pain.2013.04.046.

Sherman, J. J., and L. LeResche. 2006. "Does Experimental Pain Response Vary Across the Menstrual Cycle? A Methodological Review." *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 291, no. 2: R245–R256.

Smith, M. T., Jr., B. Remeniuk, P. H. Finan, et al. 2018. "Sex Differences in Measures of Central Sensitization and Pain Sensitivity to Experimental Sleep Disruption: Implications for Sex Differences in Chronic Pain." *Sleep* 42, no. 2: 1–15. https://doi.org/10.1093/sleep/zsy209.

Song, Y., S. Ancoli-Israel, C. E. Lewis, S. Redline, S. L. Harrison, and K. L. Stone. 2011. "The Association of Race/Ethnicity with Objectively Measured Sleep Characteristics in Older Men." *Behavioral Sleep Medicine* 10, no. 1: 54–69. https://doi.org/10.1080/15402002.2012.636276.

Staffe, A. T., M. W. Bech, S. L. K. Clemmensen, H. T. Nielsen, D. B. Larsen, and K. K. Petersen. 2019. "Total Sleep Deprivation Increases Pain Sensitivity, Impairs Conditioned Pain Modulation and Facilitates Temporal Summation of Pain in Healthy Participants." *PLoS One* 14, no. 12: e0225849. https://doi.org/10.1371/journal.pone.0225849.

Steptoe, A., V. Peacey, and J. Wardle. 2006. "Sleep Duration and Health in Young Adults." *Archives of Internal Medicine* 166, no. 16: 1689–1692. https://doi.org/10.1001/archinte.166.16.1689.

Stroemel-Scheder, C., A. J. Karmann, E. Ziegler, et al. 2019. "Sleep, Experimental Pain and Clinical Pain in Patients with Chronic Musculoskeletal Pain and Healthy Controls." *Journal of Pain Research* 12: 3381–3393. https://doi.org/10.2147/jpr.S211574.

Taylor, A., H. R. Wright, and L. C. Lack. 2008. "Sleeping-In on the Weekend Delays Circadian Phase and Increases Sleepiness the Following Week." *Sleep and Biological Rhythms* 6: 172–179.

Valdez, P., C. Ramírez, and A. García. 1996. "Delaying and Extending Sleep During Weekends: Sleep Recovery or Circadian Effect?" *Chronobiology International* 13, no. 3: 191–198. https://doi.org/10.3109/07420529609012652.

Windred, D. P., A. C. Burns, J. M. Lane, et al. 2023. "Sleep Regularity Is a Stronger Predictor of Mortality Risk Than Sleep Duration: A Prospective Cohort Study." *Sleep* 47, no. 1: 1–11. https://doi.org/10.1093/sleep/zsad253.

Supporting Information

Additional supporting information can be found online in the Supporting Information section.