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## Temporal daily associations between pain and sleep in adolescents with chronic pain versus healthy adolescents

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### Abstract

Adolescents with chronic pain frequently report sleep disturbances, particularly short sleep duration, night wakings, and poor sleep quality. Prior research has been limited by assessment of subjectively reported sleep only and lack of data on daily relationships between sleep and pain. The current study utilized multilevel modeling to compare daily associations between sleep and pain in adolescents with chronic pain and healthy adolescents. Ninety-seven adolescents (n=39 chronic pain; n=58 healthy) aged 12–18, 70.1% female participated. Adolescents completed pain diary ratings (0–10 NRS) and actigraphic sleep monitoring for 10 days. Actigraphic sleep variables (duration, efficiency, WASO) and self-reported sleep quality were tested as predictors of next-day pain, and daytime pain was tested as a predictor of sleep that night. Effects of age, gender, study group, and depressive symptoms on daily associations between sleep and pain were also tested. Multivariate analyses revealed that nighttime sleep ( $p<.001$ ) and minutes awake after sleep onset (WASO) ( $p<.05$ ) predicted next-day pain, with longer sleep duration and higher WASO associated with higher pain. Contrary to hypotheses, neither nighttime sleep quality nor sleep efficiency predicted pain the following day. The interaction between nighttime sleep efficiency and study group was significant, with adolescents with pain showing stronger associations between sleep efficiency and next day pain than healthy participants ( $p=.05$ ). Contrary to hypotheses, daytime pain did not predict nighttime sleep. Daily associations between pain and sleep suggest that further work is needed to identify specific adolescent sleep behaviors (e.g., compensatory sleep behaviors) that may be targeted in interventions.

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#### Conflict of interests

The present manuscript is submitted exclusively to *Pain* and is not under consideration in any other journal. There are no financial relationships that might lead to a conflict of interest.

#### \*Summary

The current study utilized multilevel modeling to reveal daily associations between sleep and pain in adolescents with chronic pain and healthy adolescents.

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## Keywords

pain; chronic pain; actigraphy; adolescents; sleep; multilevel-modeling

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## 1. Introduction

Chronic pain is reported in 20–40% of youth<sup>22</sup>, with the majority reporting sleep disturbances<sup>9, 25</sup> including shorter sleep duration<sup>21, 28</sup>, poorer sleep quality<sup>8, 20</sup>, and more night wakings<sup>13, 36</sup>. Disturbed sleep in youth with chronic pain is associated with limitations in social functioning<sup>9</sup>, lower quality of life, and greater disability<sup>12, 18</sup>. Previous research on youth with chronic pain has been limited by subjective sleep assessment and small samples<sup>17, 31</sup>. These studies have consistently shown perception of greater sleep disturbances in adolescents with chronic pain versus healthy adolescents. Findings on objectively measured sleep are equivocal. While some research shows adolescents with chronic pain have lower sleep efficiency compared to healthy adolescents<sup>20</sup>, other findings indicate similar sleep latency, efficiency, and duration<sup>8</sup>.

While subjective reports are useful for assessing perceptions of sleep quality, they may not accurately measure sleep patterns. Measures of sleep efficiency (percentage of time asleep accounting for night wakings), and minutes awake after sleep onset (WASO) provide estimates of sleep fragmentation. Objective assessment of sleep-wake patterns can be accomplished using actigraphy, a small watch-like device that records movement<sup>1</sup>. Actigraphy is unobtrusive and sleep patterns are monitored at home over several days rather than a single night<sup>15</sup>.

A theoretical framework developed by Lewin and Dahl proposes that relationships between pediatric pain and sleep are bidirectional<sup>11</sup>. Pain sensations can increase sleep latency and night wakings, and poor sleep can increase pain sensitivity and lower pain tolerance. Adult chronic pain studies have found that pain predicted nighttime sleep disturbances that night, and sleep disturbances predicted pain the following day<sup>2, 24</sup>. Investigators posit that this bidirectional relationship leads to a sleep-pain cycle that worsens pain and increases functional limitations<sup>16</sup>. Research examining daily relationships between pain and sleep in youth with chronic pain is limited to a study of children with sickle cell disease. Negative mood partially mediated the relationship between daytime pain and poor sleep that night and between poor sleep and pain the following day<sup>30</sup>. Expanding this research to other pediatric clinical and community populations is important.

The current study used actigraphy and diary reports to examine daily temporal relationships between sleep and pain to: 1) compare sleep patterns in adolescents with chronic pain to a healthy age and sex-matched comparison group, and 2) examine the daily temporal relationships between sleep and pain.

It was hypothesized that adolescents with chronic pain would have shorter sleep duration, lower sleep efficiency, higher WASO, and poorer self-reported sleep quality than healthy youth. Significant bi-directional associations between pain and sleep were also hypothesized: 1) greater sleep fragmentation (longer sleep duration, poorer sleep quality and efficiency, and higher WASO) would be associated with more next-day pain, and 2) daytime pain would predict greater nighttime sleep fragmentation (longer sleep duration, poorer sleep quality and efficiency, higher WASO). Effects of age, gender, study group, and depressive symptoms on relationships between sleep and pain were also examined given previous research finding significant associations<sup>19</sup>.

## 2. Methods

### 2.1 Participants and setting

Participants included 97 adolescents, ages 12–18 years (39 with chronic pain and 58 healthy participants). Adolescents with chronic pain were recruited from a multidisciplinary pain clinic at a tertiary care hospital in the Pacific Northwest. IRB approval was obtained from the institution prior to data collection. Over a two-year time period, all new patients and their parents who presented to the pain clinic and met inclusion criteria were contacted by a research assistant via telephone following their appointment and invited to participate in the study. Parents gave informed consent and children gave assent prior to study participation.

### 2.2. Inclusion/exclusion criteria

Adolescents with chronic pain were eligible for participation if they were between 12 and 18 years old and reported having pain at once/week for a minimum of three months. Healthy adolescents were recruited from advertisements posted in the local community. Participants seeking to take part in the study contacted the study coordinator via phone and answered questions to determine their eligibility.

Participants were excluded from the study if they were not between 12–18 years of age, did not speak English, or had a history of a chronic medical condition or developmental disability. Healthy adolescents were excluded if they reported having chronic pain. Participants with chronic pain were excluded from participation if the pain related to a chronic medical condition (e.g. arthritis, cancer) because of potential effects of disease factors on sleep. The rate of participation in the study was 87.2%, and the primary reason for refusal was the time commitment involved.

### 2.3 Assessment procedures

After determining eligibility and obtaining informed consent, self-report retrospective questionnaires were administered by a trained research assistant either during a home visit or via mail. Participants were also given instructions on wearing the Actiwatch and for completing electronic diaries that assessed daily sleep and pain. A research assistant administered questionnaires and provided participants with study materials either in the clinic or the home setting. Adolescents completed the 10-day daily diary using handheld computers (Palm® PDA) running custom software. Participants were instructed to complete the diary two times each day, morning and evening. Participants were asked not to make any changes to their treatment regimen or medications during the 10-day monitoring period. Each morning participants reported on the quality of their sleep the previous night. Each evening participants rated the intensity of their pain on an 11-point numerical rating scale (NRS), ranging from 0= no pain to 10= worst pain. The participants' caregivers completed a basic demographic form, which provided information on the adolescent's age, race, gender, and family income level. All materials were returned via mail and families received a \$30 gift card as compensation for their participation.

### 2.4. Descriptive, predictor and outcome variables

**2.4.1. Objective sleep patterns**—Objective sleep parameters were measured with wrist actigraphy (Actiwatch 64, Phillips Respironics/MiniMitter Company Inc., Bend, OR), a watch-like device worn at home by each adolescent on their non-dominant wrist for 10 consecutive days. Actigraphy has previously been validated in the assessment of pediatric sleep patterns<sup>26</sup> and the Actiware software has been previously validated<sup>34</sup>. Previous studies have shown that 5 or more nights of actigraphic monitoring are optimal for obtaining reliable assessments of sleep patterns in adolescents<sup>1</sup>. Study participants were instructed to depress a button (event

marker) on the Actiwatch at bedtime and upon waking in the morning. These events were also compared to the corresponding sleep diaries adolescents completed daily. With the aid of these diaries and event markers, researchers used a protocol for actigraphy scoring and set sleep periods. Sleep-wake patterns were extracted from the actigraphy data using the Actiware Sleep version 3.4, which bases its algorithm on the amplitude and frequency of detected movements, which were scored in 1-minute epochs.

Three actigraphic sleep variables were computed and used in analyses: total sleep time, sleep efficiency, and minutes awake after sleep onset (WASO). Total sleep time was scored as sleep in minutes from sleep onset to sleep offset. Sleep efficiency was calculated as the ratio of estimated total sleep time divided by total time spent in bed as a percentage times 100, with values closer to 100 meaning the most efficient sleep. WASO was calculated by adding the number of minutes in which youth were awake from sleep onset to final awakening. For ease of interpretation total sleep time (amount in minutes) is referred to as sleep duration in the analyses.

**2.4.2. Sleep Quality**—Each participant also provided a subjective rating of self-reported sleep quality using the electronic diary. Sleep quality was reported each morning using an 11-point NRS, with lower ratings associated with poorer sleep quality (0 = extremely poor sleep, 10 = extremely good sleep). Subjective global sleep quality ratings have been used in other studies of youth with chronic pain<sup>29</sup>.

**2.4.3. Pain Intensity**—Participants reported on their daily pain intensity each evening using an 11-point numerical rating scale, with higher ratings associated with more pain (0 = no pain, 10 = worst pain). Numerical rating scales have been recommended for assessment of pain intensity in children and adolescents with chronic pain conditions<sup>33</sup>.

**2.4.4. Depressive Symptoms**—To assess baseline depressive symptoms, adolescents completed the Center for Epidemiological Studies Depression Scale (CES-D)<sup>23</sup>. On the CES-D, scores are calculated by summing all items to yield a total score (range 0 – 60) with higher scores indicating greater depressive symptoms. The cutoff for clinically significant depression is a raw score of 16 in youth. The CES-D has demonstrated adequate one-week test-retest reliability, and validity has previously been established through relationships with other anxiety and depression measures<sup>5, 23</sup>. For the current sample, the internal consistency of the CES-D was moderate ( $\alpha = 0.80$ ).

### 3. Statistical Analyses

Summary statistics were used to describe the demographic characteristics of the sample. Means and standard deviations were used for continuous data, and categorical items were described using frequency statistics. T-tests and chi-square analyses were conducted to test equivalencies between groups on age, gender, racial background and income level. Pearson product moment correlations were used to assess the relationship between continuous predictor and outcome variables. MANOVAs and t-tests were used to compare the two groups (adolescents with chronic pain and healthy participants) on predictor and outcome variables. MANOVAs, correlations and t-tests were calculated using individual averages of sleep and pain data collected during the 10-day study period. On average, participants completed 8.91 days (range 4–11) of ratings/recordings. In the event that participants were missing data, averages were calculated with data available.

From data available from our pilot study<sup>20</sup> using actigraphy measures of sleep in adolescents with chronic pain we conducted a power calculation for group comparison analyses using Power Analysis and Sample Size (PASS) software. Findings revealed that sample size of 80

would achieve 90% power to detect a 6.8 point difference between the two groups on the sleep efficiency variable.

The SAS Proc Mixed procedure for multilevel modeling was used to produce unstandardized maximum likelihood estimates ( $\beta$  coefficients) which are partial correlations that estimate the magnitude and direction of association in changes in the dependent variable with changes in the independent variable. The linear mixed model also allows for the examination of temporal relationships between variables and utilizes a better mechanism for handling missing values<sup>35</sup>, which includes different numbers of observations (diary days) from participants. All diary data points from each participant were assessed as a single trajectory over the 10 day recording period.

A series of linear mixed models were used to analyze the diary data to assess the associations between sleep and pain, while accounting for correlations among repeated measures among daily records, and adjusting for potential confounding variables. Four models were tested to assess whether adolescents' nighttime sleep (total sleep duration, self-reported sleep quality, sleep efficiency, WASO) were associated with pain intensity the following day. Four additional models testing whether adolescent's daytime pain scores predicted nighttime sleep (total sleep duration, self-reported sleep quality, sleep efficiency, WASO) were also examined. Effects of age, gender, group status (chronic pain versus healthy) and depressive symptoms were tested in each of the models to determine how these factors impacted associations between adolescents' pain and sleep. Models included random-effects variables, fixed effects variables, covariates and interactions among them. Data analyses were conducted using SAS Version 9.2 (SAS 9) and the Statistical Package for the Social Sciences Version 17.0 (SPSS 17.0). Significance levels were set at  $P < .05$ .

## 4. Results

### 4.1 Descriptive Data

Adolescents had a mean age of 14.93 years ( $SD = 1.71$ ), with 70.1% of participants being female. There were no significant differences in age, gender, income or racial background between healthy adolescents and those with chronic pain (see Table 1). On average participants reported low to moderate levels of depressive symptoms (CES-D raw score  $M = 11.01$ ,  $SD = 8.69$ ) with participants with chronic pain reporting higher levels of depressive symptoms ( $t(96) = 7.01$ ,  $P < .01$ ) than healthy youth. Thirteen participants (13.4%) met the clinical cut-off for depression, nine being participants with chronic pain.

For adolescents with chronic pain, average pain intensity was in the moderate range ( $M = 6.18$ ,  $SD = 1.73$ ) with 82.1% reporting daily pain occurrence. Primary pain type and location included headache (48.7%), abdominal pain (20.5%), back pain (15.4%), and other musculoskeletal pain (15.4%). In contrast, healthy adolescents tended to report low pain intensity ( $M = 3.23$ ,  $SD = 2.14$ ) and less frequently occurring pain (on average a few times per month). When pain was reported, the most common type and location for healthy youth was musculoskeletal pain (32.6%), followed by back pain (22.4%), headache (13.8%), and abdominal pain (12.1%).

Average sleep duration (mean over the 10 day monitoring period) for all participants was 6.97 hours ( $SD = 1.04$ ), with 31% of youth experiencing extremely short sleep duration (defined as less than 6.5 hours of sleep). Mean sleep efficiency for the total sample was 84.16 percent ( $SD = 4.19$ ) with 48.4% of youth having low sleep efficiency (defined as less than 85%). In the combined sample, higher levels of depressive symptoms were correlated with higher daily pain intensity ( $r = .32$ ,  $P < .001$ ) and poorer self-reported sleep quality ( $r = -.25$ ,  $P < .001$ ). Small but significant correlations between daily pain intensity and both nightly sleep duration ( $r = .17$ ,  $P < .01$ ) and self-reported sleep quality ( $r = -.18$ ,  $P < .01$ ) also emerged; with higher pain

intensity associated with longer sleep duration and poorer self-reported sleep quality. There were no correlations among self-reported sleep quality and any of the actigraphic sleep variables. Strong correlations among WASO and sleep efficiency emerged ( $r = -.76, P < .001$ ) with more minutes awake after sleep onset associated with poorer nighttime sleep efficiency. There were no correlations among sleep duration and either WASO or sleep efficiency.

Data on prescription medication use in the sample is presented in Table 1. The majority of participants with pain were taking at least one type of medication (e.g. antidepressant, anticonvulsant), only a single participant in the healthy group reported taking any prescription medication (antidepressant).

Raw sleep data organized by study group are located in Table 2. Data from a total of 864 diary days were used in the analyses. Participants completed an average of 8.91 days (range 4–11) of actigraphic monitoring during the data collection period. This table reflects complete sleep data (864 diary days) for all 97 participants. About 30% of adolescents with chronic pain (30.1%) and healthy adolescents (31.7%) had extremely short sleep duration (< 6.5 hrs sleep) on approximately 1/3 of nights. Healthy participants and participants with chronic pain had similar numbers of nights achieving 6.5 – 8 hours of sleep (48.4% versus 42.9%) and nights with greater than 8 hours (27.0% versus 20.0%) ( $\chi^2=5.78, P=.056$ ) of sleep. In terms of self-reported sleep quality, significant group differences emerged at both low and high levels of sleep quality. Adolescents with chronic pain reported a sleep quality of <6 (reflecting lower or poorer sleep quality) on significantly more observations than healthy participants (52.1% versus 24.8%). In contrast, healthy participants reported a sleep quality of >8 (reflecting higher or good sleep quality) on 35.1% of observations compared to only 12.8% by the group with chronic pain ( $\chi^2=70.18, P < .001$ ). There were also significant group differences in nightly WASO ( $P=.05$ ). Specifically, youth with chronic pain had more nights with WASO of >70 minutes than healthy participants (38.6% versus 30.5%). Comparing adolescents with pain and healthy adolescents, observations of percent sleep efficiency were largely equivalent and not significantly different ( $\chi^2=4.87, P = .09$ ). See Table 2.

#### 4.2 Group differences on average sleep quality, sleep efficiency, WASO, and sleep duration during the 10-day assessment period

As hypothesized, when examining individual averages of sleep data obtained during the recording period, participants with chronic pain reported significantly poorer sleep quality ( $F = 21.4, P < .001$ ) than healthy participants. However, contrary to hypotheses, groups did not differ on averages of actigraphic sleep variables (sleep duration, sleep efficiency, or WASO). See Table 3.

#### 4.3 Multilevel Random Effects Models with Diary Data

Multilevel analyses examined if: 1) adolescents' nighttime sleep duration, WASO, self-reported sleep quality, and sleep efficiency were associated with pain intensity the following day, and 2) adolescent's daytime pain scores were associated with nighttime sleep (duration, quality, efficiency, WASO). Interactions among predictor variables and age, gender, group status, and depressive symptoms were assessed to determine if these factors impacted the daily associations (slope of the relationship) between pain and sleep.

**4.3.1 Nighttime sleep as a predictor of pain intensity the following day**—Four separate models evaluated nighttime sleep efficiency, self-reported sleep quality, WASO, and sleep duration as predictors of pain the next day, testing depressive symptoms and study group as covariates. As hypothesized, nighttime sleep duration predicted pain intensity the next day ( $t(311) = 3.47, P < .001$ ) with longer sleep duration associated with higher pain reports. Similarly, as hypothesized WASO predicted pain intensity the next day ( $t(311) = 2.24, P = .$



03, with greater number of minutes awake following sleep onset associated with higher pain the following day. Contrary to hypotheses, neither nighttime sleep efficiency nor self-reported sleep quality predicted pain the next day. See Table 4.

Two covariates, depressive symptoms and study group, emerged as significant predictors of pain in all four models, so interactions with sleep duration, self-reported sleep quality, sleep efficiency, and WASO were tested. While there was no main effect for nighttime sleep efficiency on pain, the interaction between sleep efficiency and study group in predicting pain was significant ( $t(310) = 1.94, P = .05$ ). In probing the interaction, the coefficients for adolescents with pain and healthy adolescents were in opposite directions, indicating that associations between sleep efficiency and pain the following day were different for participants with chronic pain and healthy participants (see Table 4). Interactions among depressive symptoms and sleep duration, self-reported sleep quality and WASO were not significant.

**4.3.2 Daytime pain as a predictor of nighttime sleep disturbance**—To test the hypothesis that daytime pain would predict nighttime sleep, daytime pain intensity was tested as a predictor of nighttime sleep efficiency, self-reported sleep quality, WASO, and sleep duration. Contrary to hypotheses, daytime pain did not predict nighttime sleep duration, self-reported sleep quality, WASO or sleep efficiency (see Table 5). While there were no main effects for daytime pain on nighttime sleep, interactions among daytime pain and age, gender, depressive symptoms and study group were tested. Interactions were not significant in predicting nighttime sleep.

## 5. Discussion

Findings from the current study revealed important differences in sequential daily associations between sleep and pain in adolescents receiving treatment for chronic pain versus healthy adolescents with intermittent pain complaints. Nighttime sleep duration predicted pain the following day for both groups, showing that sleep can impact pain for both adolescents with chronic pain and adolescents in the community. The finding that longer nighttime sleep duration was associated with higher daytime pain intensity is similar to results from a recent adult population-based study<sup>7</sup> showing similar associations between nighttime sleep and next day pain (longer sleep duration associated with more pain) although in that study pain frequency rather than pain intensity was examined.

Given substantial literature showing associations between poor sleep and pain<sup>14, 16</sup>, the directionality of the relationship between sleep duration and pain in the current study may seem counter intuitive. However in this sample, longer sleep was not associated with better, more efficient or more restorative sleep. Self-reported sleep quality was not associated with sleep duration, sleep efficiency, and WASO variables. This is also demonstrated by the significant association between greater minutes awake after sleep onset (WASO; a measure of sleep fragmentation) and higher levels of next day pain. In fact, adolescents in the study may have attempted longer sleep periods in an effort to compensate for sleep difficulties. Compensatory sleep has been found in adults with rheumatoid arthritis where higher daytime pain was associated with increased rather than decreased slow wave nighttime sleep<sup>6</sup>.

Findings revealed that changes in nighttime sleep efficiency had a differing impact on next day pain for adolescents with chronic pain than for healthy participants. These differences in daily associations among sleep efficiency and pain in clinical versus community samples may be due to the presence of persistent pain in the clinical sample while pain reported in the community sample was milder and related to short-term experience of illness or injury. Chronic and acute pain have differing pathophysiological mechanisms<sup>3</sup> and persistent pain has been

linked to sleep disturbances which can increase pain sensitivity and hyperalgesic pain response<sup>10, 16</sup>.

While our study findings showed a temporal sequential relationships between both nighttime sleep duration and fragmentation (WASO) and pain the following day, the hypothesis that nighttime self-reported sleep quality would predict next day pain was not supported. Previous studies in adult populations have found daily associations between poorer sleep quality and higher pain the following day<sup>2, 29</sup>. Future work is needed to understand the potential differences found in the adolescent population. Furthermore contrary to hypotheses, daytime pain did not predict nighttime sleep. Several factors may explain these findings. First, it is possible that sleep is a stronger predictor of pain than pain is a predictor of sleep. Research showing that sleep deprivation can significantly increase pain symptoms and lead to hyperalgesic responses (in both healthy and chronic pain samples)<sup>10</sup> supports this explanation. It is also possible that factors not assessed in the current study (e.g. stress, mood, activity level) predict sleep, and rather than being a direct predictor, pain acts as a moderator. For example, pain intensity may impact associations between daytime activity level and nighttime sleep with increased activity associated with higher pain which in turn predicts sleep that night.

It is also possible that factors such as daily medication use or daytime napping may have impacted associations among sleep and pain. While data on whether or not participants were prescribed medication were available, medication was not included in multilevel analyses because we did not have data on actual daily medication usage. Recent research using retrospective reports has shown no differences in pain, perception of sleep quality, or actigraphic sleep variables between adolescents prescribed sleep-promoting medication and those not taking such medications<sup>13, 20</sup>. Studies assessing medication use each day will further clarify how prescription medications may impact daily temporal associations among sleep and pain. Furthermore, we did not ask participants to document daily naps. One study examining daytime napping in adolescents with musculoskeletal pain found that higher frequency of daytime napping was associated with lower sleep efficiency and total nighttime sleep<sup>27</sup>. Future studies should collect daily diary report daytime napping to determine if either impact the daily relationship between sleep and pain.

Results from the study also provide important information on general adolescent sleep patterns. First, average sleep duration of these adolescents was less than seven hours for adolescents with chronic pain and healthy adolescents, which is significantly below recommended guidelines. Furthermore, on 1/3 of all observations both participants with pain and healthy participants had extremely short sleep duration (less than 6.5 hours of sleep). Studies have reported that 9.2 hours of sleep is optimal for development, physical health, and cognitive functioning in adolescents<sup>4</sup> indicating sleep deficits in our sample. Second, while adolescents with chronic pain conditions reported significantly more frequent and intense pain than healthy participants, healthy participants reported experiencing some pain. Because sleep duration and sleep fragmentation were associated with pain for both groups, interventions targeted at improving sleep would likely be beneficial in reducing pain for clinical and community samples of adolescents.

The current study has several strengths, including the use of both objective and subjective measures of sleep, a healthy comparison group, and a larger sample size than previous studies examining associations between sleep and pain in pediatric populations. The present study also used daily assessment of four sleep parameters. Prior studies examining associations between sleep and pain have been limited by use of a single average score of sleep quality or disturbance rather than looking at daily associations<sup>20</sup> or studies did not utilize actigraphic assessment of sleep making it impossible to examine temporal relationships between variables. Using mixed



modeling allows for examining daily variation in pain reports and sleep within individuals that cannot be captured with standard regression analyses.

Several limitations should be considered in interpreting the findings. First, participants in the study completed a single pain rating each day that may not have captured fluctuations in pain throughout the day. Future studies should utilize techniques such as electronic momentary assessment to capture within-day variation in pain ratings. Second, study participants completed a single 10-day assessment period of diary reports and actigraphic monitoring so variation across weeks and months was not captured in the current study. Longitudinal studies capturing variation in sleep and pain over longer timer periods will be critical for future research. Finally, clinical pain variables and treatment variables (e.g., medication-type/dosage, participation in physical therapy) could not be examined due to relatively small sample sizes and cross-sectional study design. Studies that include larger samples may allow for increased understanding of the role of specific pain diagnoses on daily sleep-pain associations.

The results of this study have potential clinical implications for assessment and management of sleep in youth with chronic pain. Comprehensive assessment of sleep patterns including asking questions about sleep times, sleep interference, and sleep quality is recommended. Developing interventions to enhance sleep may be important additions to pain management programs to reduce pain, particularly given the significant associations between nighttime sleep fragmentation and next-day pain. Cognitive behavioral interventions targeted at sleep have been effective for adults with osteoarthritis in reducing sleep disturbances (e.g. decreasing sleep latency and night wakings, increasing sleep efficiency) while also leading to significant reductions in pain intensity both immediately post-treatment and at one-year follow-up<sup>32</sup>. Similar efforts will be important to direct to the pediatric population.

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**Table 1**

Demographic characteristics of the sample.

Characteristic	Pain (n=39)	Healthy (n=58)	Total (n=97)
<u>Age in years: M (SD)</u>	15.32 (1.54)	14.66 (1.78)	14.93 (1.71)
<u>Gender: n (%)</u>			
Male	11 (28.2%)	17 (29.3%)	28 (28.9%)
Female	28 (71.8%)	41(70.3%)	69 (71.1%)
<u>Prescription Medications</u>			
Sleep	3 (7.7%)	0 (0.0%)	3(3.1%)
Analgesic	7 (17.9%)	0 (0.0%)	7 (7.2%)
Antidepressants	13 (33.3%)	1 (1.0%)	14 (14.4%)
Anticonvulsants	19 (48.7%)	0 (0.0%)	19 (19.6%)
<u>Child Ethnicity</u>			
Caucasian	34 (87.2%)	46 (79.3%)	80 (82.5%)
African American	0 (0.0%)	7 (12.1%)	7 (7.2%)
American Indian	1 (2.6%)	1 (1.7%)	2 (2.1%)
Asian	2 (5.1%)	1 (1.7%)	3 (3.1%)
Other	2 (2.6%)	3 (5.2%)	5 (5.2%)
<u>Family Income</u>			
< \$29,000	4 (10.3%)	7 (12.1%)	11 (11.3%)
\$30,000 – \$49,000	7 (17.9%)	7 (12.1%)	14 (14.4%)
\$50,000 – \$69,000	5 (12.8%)	10 (17.2%)	15 (15.4%)
> \$70,000	22 (12.4%)	34 (58.6%)	56 (57.7%)
Missing	1 (2.6%)	0 (0.0%)	1 (1.0%)

**Table 2**

Complete data for observations (n=864) of nightly sleep time, sleep duration, WASO, and sleep efficiency by study group.

	<b>Pain</b>	<b>Healthy</b>	<b>Chi-Square</b>	<b><i>p</i></b>
<b>Sleep Duration (hours)</b>				
< 6.5	30.1%	31.7%		
>6.5 – 8	42.9%	48.4%		
> 8	27.0%	20.0%	5.78	.06
<b>Sleep Quality (0 – 11 NRS)</b>				
< 6	52.1%	24.8%		
6 – 8	35.1%	40.1%		
8 – 10	12.8%	35.1%	70.18	<.001
<b>Sleep Efficiency (0–100%)</b>				
< 80	21.2%	16.9%		
80–85	31.6%	28.4%		
>85	47.2%	54.7%	4.87	.09
<b>WASO (2–205 min)</b>				
< 45	24.6%	28.0%		
45–70	36.8%	41.6%		
>70	38.6%	30.5%	5.91	.05

**Table 3**

Means and standard deviations and group differences on depression and sleep variables.

Variable	Mean (SD)		t or F	p	$\eta^2$
	Pain Group	Healthy			
Pain Intensity	6.14 (1.70)	3.63 (1.41)	7.12	.00	.63
Depressive Symptoms (raw score)	13.4 (9.89)	8.84 (6.54)	7.01	.01	.07
Sleep Quality (0–10 NRS)	5.27 (1.37)	6.69 (1.49)	21.4	.00	.19
Total Sleep Time (minutes)	428.7 (45.9)	411.6 (71.0)	1.69	.20	.02
Sleep Efficiency	83.4 (4.84)	84.7 (3.68)	2.01	.16	.02
WASO (minutes)	67.08 (19.09)	61.65 (17.0)	2.08	.15	.02



**Table 4**

Summary of multivariate random effects analyses of associations between nighttime sleep next-day pain.

	Next day Pain		
	$\beta$	t	p
Sleep time	.01	3.47	<.001
Depression	.07	3.69	<.001
Group <sup>a</sup>	2.31	6.66	<.001
Gender <sup>b</sup>	.61	1.58	.11
Age	-.06	-.58	.57
Sleep quality	-.06	-1.24	.21
Depression	.06	3.12	<.01
Group	2.27	6.26	<.001
Gender	.40	.98	.33
Age	-.12	-1.04	.30
Sleep efficiency	.01	.18	.86
Depression	.07	3.45	<.001
Group	2.39	6.77	<.001
Gender	.51	1.28	.21
Age	-.10	-.87	.39
Sleep efficiency*group	.07	1.94	.05
WASO	.01	2.24	.03
Depression	.07	3.47	<.001
Group	2.35	6.63	<.001
Gender	.42	1.07	.29
Age	-.10	-.90	.37

<sup>a</sup>Group: pain=0, healthy=1<sup>b</sup>Gender: pain=0, healthy=1

**Table 5**  
 Summary of multivariate random associations between daytime pain intensity and nighttime sleep.

	Sleep Duration			Sleep Quality			Sleep Efficiency			WASO		
	$\beta$	t	p	$\beta$	t	p	$\beta$	t	p	$\beta$	t	p
Daytime pain	3.74	1.57	.11	.022	.34	.73	.17	1.05	.29	.79	.92	.36
Depression	-1.79	-2.84	.01	.034	.28	.78	-.09	-1.69	.09	.003	.01	.99
Group	5.62	.44	.66	1.25	1.76	.08	-1.94	-1.99	.05	5.68	1.03	.30
Gender	-33.2	-2.54	.01	6.03	2.40	.02	-4.12	-4.04	.00	10.84	1.89	.06
Age	-9.61	-2.65	.01	1.40	2.03	.05	-1.17	-.60	.55	-.79	-2.65	.62