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Mapping the associations of daily pain, sleep, and psychological distress in a U.S. sample

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Abstract

Chronic pain, sleep problems, and psychological distress (PD) can be disabling conditions and previous research has shown that they are associated. The nuances of the comorbid nature of these conditions may be important to understand for those who treat these conditions. This study examined the bidirectional associations of these health factors concurrently and over time in a sample of U.S. adults (N = 1,008, M_{age} = 57.68) from the Midlife in the United States (MIDUS) study. Participants reported on their daily pain, sleep quantity, and psychological distress over eight days. A modified Random Intercept Cross-lagged Panel Model was used to analyze the relations, starting with the whole sample and then a comparison of those with and without chronic pain. Results indicated that nightly variation in sleep quantity predicted next day psychological distress for both groups. Sleep quantity also predicted next-day pain, but only for individuals with chronic pain. Associations between pain and psychological distress were found both at the daily level and individual (between-person) level. This between-person association was stronger for those with chronic pain. The lagged associations between sleep, and both pain and psychological distress for the chronic pain group indicate that, increased quantity of sleep predicts decreased next-day pain and psychological distress. Providers could consider this unidirectional lagged relationship when prioritizing treatment for patients with these comorbid conditions. Future research may examine whether responsive, just-in-time treatments might intervene after participants wake from a poor night's sleep to counteract the negative effects of reduced sleep on PD and pain.

Keywords Chronic Pain · Sleep duration · RI-CLPM · Mental Health

Physical pain, sleep problems, and psychological distress are three factors that contribute to human suffering and disability. Each of these factors alone is complex, and yet, as reviewed below, previous theoretical and empirical research indicates that they can influence each other. Research has shown that behavioral interventions on these factors can be effective, but not overwhelmingly so (Edinger et al., 2021; Feliu-Soler et al., 2018; Khoo et al., 2019; Wampold et al., 2011). Part of the lack of impact of treatment may be due to a lack of understanding about how these three factors, which are often comorbid, might have causal, maintaining, and exacerbating associations with each other (Boakye et al., 2016), including at the daily level. This study makes use of daily reports of physical pain, sleep, and psychological distress (PD; symptoms and impairment related to mental and emotional functioning), within a cross-lagged framework to examine how day-to-day variations in these factors might influence each other over time.

Pain

Pain is "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" (International Association for the Study of Pain, 2021). Dahlhamer and colleagues (2018) found that approximately 20% of adults reported chronic pain in a national survey in the United States (n = 33,028),, where chronic pain is often defined as pain that persists or recurs for more than 3 months (Treede et al., 2019). Interventions such as Cognitive Behavioral Therapy for Chronic Pain and Acceptance and Commitment Therapy have been

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used to treat pain and its related factors with some success (Feliu-Soler et al., 2018; Khoo et al., 2019).

Sleep

Sleep is an important biological process as it contributes to the repair of the brain and body (Eugene & Masiak, 2015). Ideally, people experience sleep that is of an appropriate duration and is high quality. It is generally recommended for adults to get 7–9 h of sleep (Ashbrook et al., 2020) and sleep quality is associated with various health outcomes (Hale et al., 2013). Ongoing deficits in sleep quantity and quality can lead to a diagnosis of insomnia, which has been associated with a wide range of negative outcomes (Natsky et al., 2020). Prevalence rates of acute insomnia vary widely by study, but is likely between 20 and 30% overall (Bhaskar et al., 2016; Zeng et al., 2020). Behavioral interventions can improve sleep quality and quantity in people seeking treatment, with the widely studied cognitive behavioral therapy for insomnia (CBT-I) resulting in significant improvements in sleep quality (0.66 standardized mean difference (SMD) and reductions in insomnia (0.95 SMD; Edinger et al., 2021).

Psychological distress

In line with a dimensional perspective of mental illness (Kotov et al., 2017), greater psychological distress (PD) can be understood as more mental health symptoms and greater impairment due to cognitive, physical, and emotional factors (Umucu et al., 2022).Based on the Global Burden of Disease study, depression is a leading cause of disability around the world (5.0% of the world adult population), while anxiety disorders also cause significant disability (4.8%; Friedrich, 2017; Institute of Health Metrics and Evaluation, 2023). In epidemiological studies, PD is often used to screen for the presence of mental illness, using a set of non-disorder-specific items. Interventions ranging from psychopharmacological treatment, to physical activity, to psychotherapy have shown promise with treating PD and mental illness (Ashdown-Franks et al., 2020; Huhn et al., 2014; Wampold et al., 2011).

Associations between Pain and Sleep

Pain and sleep, both foundational physiological processes, are associated in important ways. Over half, and as many as 88% of individuals with chronic pain complain of sleep problems (Smith & Haythornthwaite, 2004), while about half of those with chronic pain report insomnia (Mathias et al., 2018). Finan et al.'s (2013) review of the literature indicates that sleep problems often predict later pain, while the reciprocal effect was less commonly found. However, one large (n = 971) daily diary study revealed a bidirectional relationship between the two variables; sleep predicting pain and pain predicting sleep quality (Edwards et al., 2008). Experimental research has found that increased pain sensitivity follows sleep deprivation (e.g. Schrimpf et al., 2015), which may be due to reductions in the capacity to modulate pain signals (Tiede et al., 2010). It is interesting to note that pain, even acute pain, does not normally cause wakings (Foo & Mason, 2003). Despite the lack of awakening, the body still has physiological reactions to the pain. Chronic pain's impact on the serotonergic raphe cells, which are involved in modulating alertness, may impact continuity of sleep stages (Foo & Mason, 2003, Parades, 2019), while Finan and Smith (2013) also argue that impacts on dopaminergic signaling in that part of the brain simultaneously impact sleep and pain (and depression). Sleep deprivation also impacts the endogenous opioid systems, which play an important role in the modulation of pain (Ballantyne & Sullivan, 2017; Finan et al., 2013; Haack et al., 2020).

Associations between Pain and Psychological Distress

Substantial research has linked pain to PD (Li, 2015, Evans, 2017). In one study anger expression/negative affect was significantly higher for patients suffering from lower back pain relative to healthy controls (Bruehl et al., 2012; See also Sommer, 2019). Daily diary studies indicate that daily fluctuations in pain are often significantly associated with concurrent fluctuations in mood (e.g. Kothari & Davis, 2015). Potential mechanisms connecting these conditions include inflammatory cytokines associated both with depression and pain (Lees et al., 2015), stress hormone reactions (Thornton et al., 2010), and the dopaminergic system (Finan & Smith, 2013). Pain and affective processing happen in similar areas of the brain, share neurotransmitters, and can have similar medicinal treatments (Yang & Chang, 2019). From a cognitive and behavioral perspective, both pain and PD are associated with negative distortions in beliefs and with a reduction in engagement in positive activities (Asarnow & Manber, 2019). Problematic thinking may, for example, manifest as catastrophizing where those with chronic pain overestimate the intensity, impact, and unremitting nature of chronic pain, while those with PD may over overestimate the impact of a negative life event, leading them to experience substantial distress (Turner et al., 2000; Vîslă et al., 2016).

Associations between Sleep and Psychological Distress

The relationship between sleep and PD has been well studied. Substantial research has found that daily sleep quality, duration, and disturbance have reciprocal relationships with mood and PD (Konjarski et al., 2018; Tomaso et al., 2021). Objective measurements using actigraphy support this conclusion as well (Hickman & D'Oliveira, 2021). However, there are empirical exceptions to the expected bidirectional effects, where two daily diary studies found that sleep quality predicted next-day psychological distress, while the reverse association was not found (Harris et al., 2022; Wen et al., 2021). Various mechanisms have been forwarded to connect PD and sleep. Repetitive, ruminative thinking, which is characteristic of mental health disorders, predicts worse sleep (Takano et al., 2014). Impairments in the hypothalamic-pituitary-adrenal (HPA) axis and in neurotransmitter functioning are characteristic of problematic sleep and mood disturbance (Bao et al., 2017; Wulff et al., 2010). Further, Finan and Smith's (2013) dopaminergic model ties PD to sleep via alterations in the dopaminergic system due to PD, which in turn impacts sleep (See also Haack et al., 2020). Dysregulation in levels of arousal are another factor that can impact both sleep and PD (Kalmbach et al., 2021; Staner, 2010).

Associations between Pain, Sleep, and psychological distress

Previous studies have also attempted to investigate the relationships between pain, sleep and PD simultaneously, with various conceptualizations of mediation relationships. These have included sleep mediating pain and PD (Miró et al., 2011; Juan et al., 2020), PD mediating sleep and pain (Whibley et al., 2019), and pain mediating sleep and PD (Hamilton et al., 2012). Common neurobiological underpinnings related to changes in the HPA axis, serotonin, and brain-derived neurotrophic factor link these factors together (Boakye et al., 2016). The seemingly complex, bi-directional relationships among all of these variables call for further investigation. Understanding which factors have the strongest longitudinal associations, might inform the treatment of these conditions – revealing which one(s) should be prioritized.

The goal of the present study is to simultaneously analyze the concurrent and longitudinal relationships of these three variables – made possible via a version of a Random-Intercept Cross-Lagged Panel Models. The benefit of this approach is that it can help clarify whether variation in a variable at one time point is associated with variation in a separate variable at the next time point. While doing so, the model can control for the association of one variable (e.g. sleep on night one) with the same variable at the next time point (sleep on night two). The model is also able to control for individual differences in mean levels of each variable. Based on the research previously reviewed, cross-lagged associations (e.g. variable x at time 1 predicting variable y and time 2) for each pair of variables were expected (e.g. sleep quantity and pain; sleep quantity and PD; pain and PD). However, we were particularly interested in the association of these variables for individuals who had chronic pain. The expectation was that individuals with chronic pain would have stronger associations among the variables because individuals with chronic pain are sensitized to pain and may be more impacted by fluctuations in pain (van Wilget & Keizer, 2012), their brains may be conditioned to send increased signals of emotional distress to avoid potential pain (Ploghaus et al., 2001), and daily fluctuations in their already distressed sleep patterns may have a greater impact on other variables.

Methods

Design

Participants

The participants from this secondary analysis study include a sample of adults who were recruited as part of the Midlife in the United States (MIDUS) study (Radler, 2014). The MIDUS study was designed as a nationally representative longitudinal panel study, with the three waves of data collection starting in 1995–1996. During the second wave in 2004–2006 participants were invited to engage in an 8-day daily diary study. The sample from the present study relied on participants who were in the main Random Digit Dial sample (N = 1,079). Seventy-one participants did not report on their chronic pain from the MIDUS II survey. These participants were excluded from the analyses, resulting in a final sample size of N = 1,008. The original data collection was approved by IRBs at the University of Wisconsin-Madison and due to the de-identified and publicly available nature of the data, the IRB at the University of Southern Mississippi determined that IRB oversight was not needed for this study.

Measures

The measures included in this secondary analysis were those selected and developed by the developers of the MIDUS study.

Pain. Participants were asked to report on the presence of four types of pain. If the pain was present, they reported severity on a scale from 1 ("mild") to 10 ("very severe") on each day. The types of pain included backache, muscle soreness, joint pain, and headache. An initial factor analysis using the first day of data revealed that that standardized loading of headache for a latent pain variable had a lower loading (b = 0.24) than the others (> 0.61). After excluding headache, subsequent measurement invariance testing revealed up to strong invariance across the last 7 days for a latent pain variable with backache, muscle soreness, and joint pain as indicators[1]. In an effort to make a composite variable, the severity ratings related to backache, muscle soreness and joint pain were summed, such that the possible total pain score ranged from 0 to 30 on a given day. A Chronbach reliability coefficient using first day data was α = 0.64, and multilevel reliability resulted in a $\omega_{between}$ of 0.68 and an ω_{within} of 0.42 (See Lai, 2020). The within-person reliability appears low, but as Nezlek (2017) suggested, traditional standards of reliability may need to be relaxed when dealing with the realities of multilevel, daily diary type data. Participants were classified as having chronic pain based on their response to a question in the second wave of the MIDUS II survey, asking them whether they have chronic pain "that persists beyond the time of normal healing and has lasted from anywhere from a few months to many years."[2]

Psychological distress. PD was assessed with the Kessler six-item distress scale (Kessler et al., 2002). Participants were asked to report on how much of the day they felt restless or fidgety, nervous, worthless, so sad that nothing could cheer them up, that everything was an effort, and hopeless. Responses were on a continuum ranging from 0 = "none of the time" to 4 = "all of the time." The Kessler-6 scale has been widely used to assess non-specific PD, with a cutoff value of 5 indicating moderate mental distress (Prochaska et al., 2012). The items were summed to create a total PD score ranging from 0 to 24. The reliability for the first day of data was 0.71, while $\omega_{between}$ was 0.74 and an ω_{within} was 0.48.

[1] Headache pain was used by itself as an outcome in supplementary analyses. Of note however, there were not enough participants with chronic headache pain to conduct group comparisons.

[2] As a sensitivity analysis, an alternative way of determining of chronic pain was also modeled. It was based on whether the participant reported a four or greater on any form of pain on a majority of the days in which they responded to the daily diary survey. Values of four to five are often identified as the cutoff between mild and moderate pain (Boonstra et al., 2016; Oldenmenger et al., 2013). This approach recognizes that individuals with chronic pain can have fluctuations in the presence and severity of their pain (Mun et al., 2019). Because the findings from these alternative models were similar to the ones presented in the results below, they are not be discussed further, but the findings are available as supplemental material.

Sleep quantity. Participants reported the amount they slept since the previous day's interview, not including naps. Specifically, the participants were asked, "Since we spoke yesterday, how much time did you spend sleeping, not including time you may have spent napping." Interviewers recorded the hours and minutes that the participant provided.

Control variables. A series of variables were entered into the model to control for the impact of demographic and weekly variables on the relevant associations. Self-reported age and sex were included as well as the total number of stressors reported by the participant over the study. Each day the participants were asked by the interviewers whether they experienced any number of six stressful experiences (e.g. an argument, discrimination, a stressful work/school event) and one other open-ended experience. Example interview questions included "Did you have an argument or disagreement with anyone since we spoke yesterday?" and "Did anything else happen to you since we spoke yesterday that people would consider stressful?" The number of these stressful experiences they reported were summed to create a count of stressful experiences from that day.

Procedure

The participants were interviewed via telephone each day for 8 days by trained interviewers. Paid interviewers underwent about 30 h of training, including ongoing monitoring throughout the data collection process, and used a computer assisted telephone interviewing system to administer the standardized surveys. The timing of the interview varied, with about 33% of the interviews occurring in the "Evening" and the rest occurring in the "Daytime" as recorded in the codebook. The response rate to the surveys was generally high, with a retention rate being over 92%. Data collection occurred throughout the year to obtain variation in season. Additional information about the data collection process can be found at the Inter-university Consortium for Political and Social Research site (https://www.icpsr.umich.edu/web/ICPSR/studies/26841).

Analysis

All analyses were carried out in R (R Core Team, 2021) and RStudio software (RStudio Team, 2021). Particularly important packages included *tidyverse* (Wickham et al., 2019) and *lavaan* (Rosseel, 2012). In order to assess the lagged, concurrent, and between-person associations between sleep quantity, pain and PD, a random-intercept cross-lagged panel model was implemented (Hamaker et al., 2015). Having many similarities to cross-lagged panel models, the lagged associations between variables are assessed (i.e. sleep at time 1 predicting PD at time 2), controlling for the

lagged autocorrelations of each variable (i.e. sleep at time 1 predicting sleep at time 2). The additional control over between-person differences in the levels of these variables (i.e. random intercepts) in a RI–CLPM allows for an isolation of the within-person associations, such that fluctuations in one variable can be accurately examined as a predictor of fluctuations in another variable at a subsequent time. Thus, these models improve upon the interpretability problems of traditional cross-lagged panel models by parsing apart within-person variance and between-person variance in the relevant variables (Mulder & Hamaker, 2021). The RI-CLPM can support the exploration of interesting questions such as, controlling for person-averages of distress, pain and sleep, does higher than average daily pain impact subsequent PD and sleep? It also allows for the examination of associations among within-person variation between concurrently measured variales (i.e. pain and PD) and the associations between person averages at the between-person level (i.e. "are participant's average levels of PD, pain, and sleep duration correlated?").

Although reported at the same time by the participants, last night's sleep occurred temporally before today's pain. As such, a somewhat modified version of the RI-CLPM had to be used, with last-night sleep predicting, rather than covarying with, today's pain and PD. This was done despite all three variables being measured at the same time. Then, in turn, today's pain and today's PD predicted tonight's sleep (which was measured the next day). Daily pain and PD were allowed to covary with each other to see within-person associations between those variables. These temporal relationships among the within-person parts of the models can be viewed in Figs. 1 and 2. The between-person associations among pain, PD and sleep quantity were modeled as well.

To aid interpretability, the lagged and cross-lagged coefficients and residual variances were constrained, within each group, to be equal over time (Mulder & Hamaker, 2021). A scaled Chi-Square test statistic was used for comparing the fit of models with and without the constraints (Yuan & Bentler, 2000). A significant test would indicate worse fit in the constrained model, indicating that the constraints were not appropriate. A specific interest in this study was examining the associations of these variables in individuals with chronic pain relative to those without chronic pain. As such, a multiple-group RI-CLPM model was run, where coefficients were compared across groups to see if there were group differences. Scaled Chi-Square difference tests were used for comparing the coefficients across groups, with a significant test indicating that there are group differences. The expectation was that daily the associations of the variables would not change over the period of the seven-day study.

Data from the last seven days of the eight-day study were used, as the first day was used for the exploratory factor analysis of the pain variables (Fokkema & Greiff, 2017). Missing data were managed with Full Information Maximum Likelihood estimation and deviations from normality were addressed with Maximum Likelihood estimation with Robust Standard errors (Lee & Shi, 2021). The covariates of

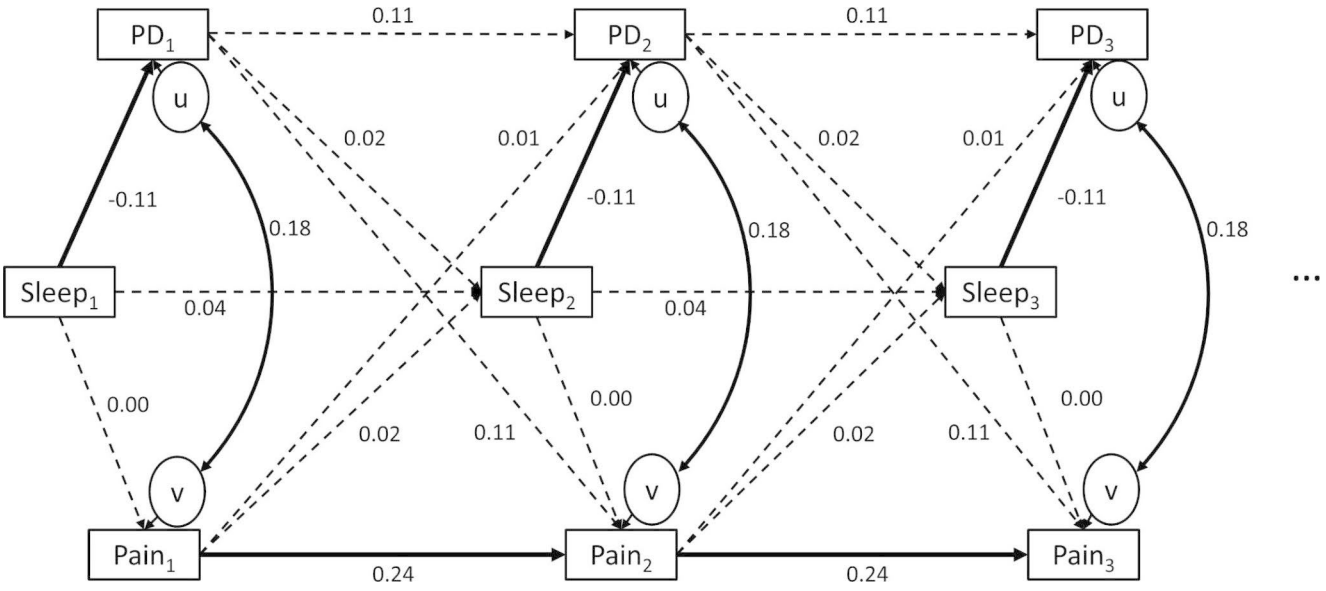

Fig. 1 Title: Within-person Associations for the Non-Chronic Pain Group

Description: Due to space constraints, this figure does not include representations of the between-person variables and their associations. PD = Psychological Distress. u and v represent residual variances of PD and pain respectively, which are correlated. Solid lines represent statistically significant effects, while dashed lines are not statistically significant

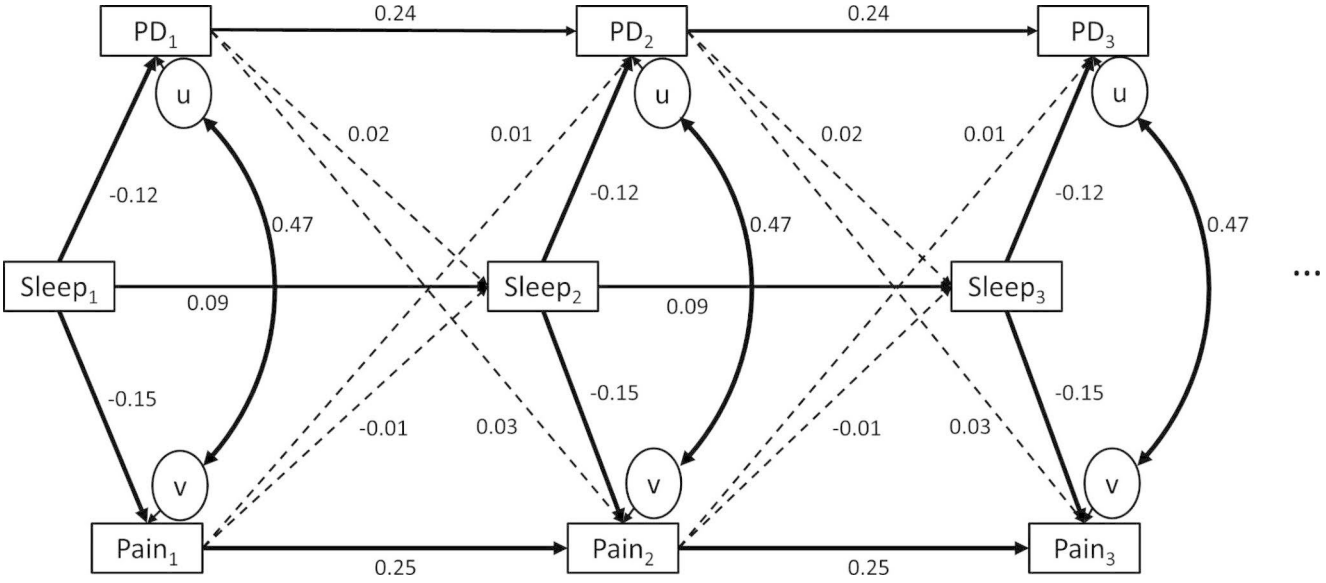

Fig. 2 Title: Within-person Associations for the Chronic Pain Group Description: Due to space constraints, this figure does not include representations of the between-person variables and their associations. PD = Psychological Distress. u and v represent residual variances of PD and pain respectively, which are correlated. Solid lines represent statistically significant effects, while dashed lines are not statistically significant

age, sex, and number of stressors were entered as predictors of each daily value of pain, sleep, and PD.

Results

Participant characteristics are available in Table 1. For each of the two models, we began by testing the RI-CLPM without any constraints, then constraints were added in a subsequent model to test changes in model fit. The full fit indices and model coefficients are available in the supplementary materials.

Whole sample model

The initial unconstrained model fit the data well ($X^2(141) = 219.27$, CFI = 0.99, RMSEA = 0.03 [0.02, 0.04], SRMR = 0.03), although when model constraints were implemented on the within-person lagged estimates, cross-lagged estimates, covariance estimates between pain and distress, and residual variance estimates, the fit decreased according to a significant Chi-square difference test ($\Delta X^2(70) = 102.63$, p = .007). However, when the residuals were allowed to vary by day, while retaining the other constraints, the fit of this partially constrained model was not significantly different from the baseline model ($\Delta X^2(53) = 54.711$, p = .409; see Table 2 for full fit indices).

The only significant cross-lagged effect was the negative association between sleep quantity and next day PD (B = -0.12, 95% CI [-0.17, -0.08], p < .001), indicating that on nights when the person slept more than their average, they experienced less PD than their average that next day. The autocorrelated within-person coefficients were significant for pain (B = 0.25, 95% CI [0.19, 0.31], p < .001), sleep (B = 0.06, 95% CI [0.03, 0.11], p = .013), and PD (B = 0.17, 95% CI [0.09, 0.25], p < .001). Also of note, the same-day within-person associations of PD and pain were significant (B = 0.28, 95% CI [0.14, 0.42], p < .001), indicating that on days when the person was exhibiting greater than average pain, they were also experiencing greater than average PD. At the between person level, pain was negatively associated with sleep quantity (B = -0.46, 95% CI [-0.78, -0.14], p = .005) and positively associated with PD (B = 1.815, 95% CI [1.00, 2.63], p < .001), while PD and sleep were not associated (B = -0.12, 95% CI [-0.26, 0.02], p = .105).

Multigroup model

The initial, fully constrained multigroup model fit the data poorly ($X^2(441) = 928.11$, CFI = 0.89, RMSEA = 0.07 [0.06, 0.07], SRMR = 0.24). Modification indices indicated that the following parameters should be allowed to vary across groups: sleep predicting next day pain, sleep predicting next day PD, sleep predicting next day sleep, pain predicting next day sleep, and PD predicting next day sleep, as well as same-day covariance between pain and PD. The variance of each random intercept and the covariance between the PD and sleep and PD and pain random intercepts were also allowed to vary across groups, along with the residual variances of PD, pain and sleep. This partially constrained multiple group

Table 1 Participant Characteristics

Variable	Whole Sample (N = 1,008)	Chronic pain group (N = 411)	Non-chronic pain group (N = 597)
Sex (Female)	56.75%	60.10%	54.67%
Race			
White	91.3% (n = 920)	91.5% (n = 376)	91.1% (n = 544)
Black and/or African American	3.5% (n = 35)	4.1% (n = 17)	3.0% (n = 18)
Native American/Alaskan Native	1.6% (n = 16)	1.2% (n = 5)	1.8% (n = 11)
Asian	0.6% (n = 6)	0.2% (n = 1)	0.8% (n = 5)
Other	2.8% (n = 28)	2.9% (n = 12)	2.7% (n = 16)
Missing	0.3% (n = 3)	0.0% (n = 0)	0.5% (n = 3)
	Mean (SD)	Mean (SD)	Mean (SD
Age	57.68 (12.44)	58.69 (12.36)	56.99 (12.46)
Household income	$51,062 ($53,111)[a]	$40,705 ($47,011)[b]	$57,833 (55,754)[c]
Number of stressors per week	3.75 (3.13)	3.93 (3.02)	3.63 (3.20)
Daily backache severity	0.77 (1.61)	1.35 (2.09)	0.36 (0.99)
Daily joint pain severity	1.03 (1.81)	1.70 (2.32)	0.57 (1.14)
Daily muscle soreness severity	0.80 (1.40)	1.17 (1.77)	0.54 (1.01)
Daily pain severity composite	2.60 (3.90)	4.22 (4.96)	1.47 (2.36)
Proportion of days with > 3 pain	0.22 (0.33)	0.34 (0.38)	0.13 (0.25)
Daily PD	0.98 (1.56)	1.26 (1.88)	0.78 (1.27)
Daily sleep hours	7.09 (1.03)	7.02 (1.16)	7.13 (0.92)

(a) Due to missing data, this is based on 941 participants; (b) based on 372 participants; (c) based on 569 participants

Table 2 Fit indices (robust versions)

Model	X^2 (DF); scaling factor	CFI	RMSEA [95% CI]	SRMR
Whole sample baseline	219.27 (141); 1.63	0.99	0.03 [0.02, 0.04]	0.03
Whole sample full constraints	319.63 (211); 2.00	0.98	0.03 [0.02, 0.04]	0.04
Whole sample partial constraints	268.67 (194); 1.45	0.99	0.03 [0.02, 0.03]	0.03
Multigroup full constraint	928.11 (441); 1.93	0.89	0.07 [0.06, 0.07]	0.24
Multigroup partial constraints	541.30 (424); 1.82	0.98	0.03 [0.02, 0.04]	0.05

DF = Degrees of freedom; CFI = Comparative fit index; RMSEA = Root mean square error of approximation; SRMR = Standardized root mean squared residual

model fit the data significantly better, ($\Delta X^2(17) = 177.33$, $p < .001$) and well in general ($X^2(424) = 541.30$, CFI = 0.98, RMSEA = 0.03 [0.02, 0.04], SRMR = 0.05).

For the non-chronic pain group, the only cross-lagged associations was for sleep predicting next day PD (B = -0.11, 95% CI [-0.17, -0.5], $p < .001$; see Table 3 for a full listing of relevant coefficients). The same day within-person association between pain and PD was significant (B = 0.18, 95% CI [0.03, 0.34], p = .02) for this group as well. At the between-person level, pain and sleep were negatively associated (B = -0.24, 95% CI [-0.45, -0.04], p = .02), while significant associations were not found for pain and PD (B = 0.67, 95% CI [-0.10, 1.45], p = .09), or PD and sleep (B = 0.02, 95% CI [-0.06, 0.11], p = .60).

For the chronic pain group, two of the cross-lagged parameters were significant: sleep negatively predicted both next day pain (B = -0.15, 95% CI [-0.26, -0.04], p = .01) and sleep predicted next day negative affect (B = -0.12, 95% CI [-0.19, -0.04], p = .002). This means that for individuals with chronic pain, experiencing more than their average amount of sleep on a given night, is associated with less than their average pain and negative affect on the next day. Using cross-group constraints to compare the size of the coefficients, the sleep-PD association was not significantly different between the two groups ($\Delta X^2(1) = 0.01$, p = .93), while the sleep-pain association was significantly greater for the chronic pain group ($\Delta X^2(1) = 4.61$, p = .04). There was a significant positive within-wave association between pain and PD for the chronic pain group (B = 0.47, 95% CI [0.19, 0.74], p = .001), which was not significantly greater than the non-chronic pain group ($\Delta X^2(1) = 3.66$, p = .06). At the between-person level, there was a significant positive association between Pain and PD (B = 3.03; 95% CI [1.56, 4.52], $p < .001$) and a significant negative association between sleep and pain (B = -0.24; 95% CI [-0.45, -0.04], p = .02). Difference tests indicate that the between-person Pain and PD relationship was significantly stronger for the chronic pain group ($\Delta X^2(1) = 13.41$, $p < .001$).

Table 3 Model coefficients

	Chronic pain group Coefficient (95% CI)	Non-chronic pain group Coefficient (95% CI)
Within-person		
Sleep → pain	**-0.15 (-0.26, -0.03)**	0.00 (-0.08, 0.08)
Sleep → PD	**-0.12 (-0.19,-0.04)**	**-0.11 (-0.17, -0.05)**
Sleep → Sleep	**0.09 (0.01, 0.19)**	0.04 (-0.02, 0.10)
Pain → Sleep	0.00 (-0.03, 0.01)	0.02 (-0.01, 0.04)
Pain → PD	0.01 (-0.01, 0.03)	0.01 (-0.01, 0.03)
Pain → Pain	**0.24 (0.18, 0.30)**	**0.24 (0.18, 0.30)**
PD → Sleep	0.02 (-0.03, 0.06)	0.02 (-0.02, 0.06)
PD → Pain	0.03 (-0.04, 0.09)	0.03 (-0.04, 0.09)
PD → PD	**0.24 (0.12, 0.36)**	**0.11 (0.01, 0.22)**
PD–Pain Covariance	**0.47 (0.19, 0.74)**	**0.18 (0.03, 0.34)**
Between-person		
Sleep-Pain covariance	**-0.24 (-0.45, -0.04)**	**-0.24 (-0.45, 0.04)**
Sleep-PD covariance	-0.24 (-0.50, 0.01)	0.02 (-0.06, 0.11)
Pain-PD covariance	**3.04 (1.56, 4.52)**	0.67 (-0.10, 1.45)

PD = Psychological distress. Bolded coefficients are statistically significant

Discussion

The purpose of this project was to examine the complex associations among three important health variables: pain, sleep quantity, and PD, especially for those with chronic pain. The initial model revealed that for the whole sample, amount of sleep negatively predicted PD. This finding held for both the chronic and non-chronic pain groups separately as well. This indicates that higher than average amounts of sleep, predicts less PD on the following day. The reverse association of daily PD predicting the following night's sleep was not found, which as reported by Wen and colleagues (2021), aligns with previous research showing that sleep tends to better predict next-day emotions, rather than vice versa.

The cross-lagged finding of sleep predicting next-day pain was found for those with chronic pain. If someone with chronic pain has a greater amount of sleep on a given night, they on average, also had less pain the next day, controlling for the previous day's pain and PD. The deep or slow-wave stages of sleep, which are understood to be the most important for physical and mental recovery, occur mostly in the early part of the sleep session (Dijk, 2009; Léger et al., 2018). Those with chronic pain may, due to heightened arousal reactions during sleep (Foo & Mason, 2003), have fragmented slow wave sleep (Drewes et al., 1998; Mathias et al., 2018), including during the first hours after sleep onset. For those without chronic pain, after having obtained restful sleep for the first 3–5 h (when most slow wave sleep occurs), fluctuations in wake time may not have a major impact. This has some peripheral support in sleep deprivation experiments where individuals who, via acoustic or electric stimulation, are inhibited from achieving slow wave sleep in the first part of the night experience a rebound effect where their body continues to transition into slow wave sleep to make up for the lack of it in the early hours of the sleep session (Cheng et al., 2021; Dijk et al., 1987). Thus, fluctuations in sleep duration for individuals with chronic pain may be more strongly associated with the amount of slow wave sleep obtained due to their fragmented slow-wave sleep. Further, experimental research indicates that slow wave sleep deprivation leads to reduced pain thresholds and increased pain symptoms (Lautenbacher et al., 2006; Irwin et al., 2022), while one study found that total sleep deprivation resulted in more pain in those with arthritis relative to those without arthritis (Finan et al., 2013). While observational research cannot firmly claim causal relations, this analysis, based on the RI-CLPM approach can provide some evidence that fluctuations in sleep quantity do lead to next-day pain for those who report chronic pain. Further, this daily diary approach adds ecological validity to previous experimental research linking sleep duration to pain (e.g. Schrimpf et al., 2015; Tomaso et al., 2021).

As with sleep-PD relationship, the reverse direction of pain predicting sleep was not found. As such, sleep quantity may be the more independent factor, while influencing daily fluctuations in PD and pain. These findings were somewhat similar to Lücke et al. (2022) who found that pain did not predict later sleep quantity, but in contrast to this study, they did not find sleep quantity predicting pain. However, in their study, they also had a sleep quality variable which did predict pain but was also not predicted by pain. Gerhart et al. (2017), also found that poor sleep quality predicted next day pain, but that pain did not predict next-night sleep quality. To restate their conclusions about the null findings, although it may seem intuitive that pain would impact next-night sleep, the evidence is generally weaker for that pathway (Finan et al., 2013; See also Arnison et al., 2022). The basic research conducted by Foo and Mason (2003) indicates that the impact of pain on sleep may not be consciously available and thus not adequately measured, at least by self-reports of sleep *quality*. As for sleep quantity, it appears that nighttime pain, rather than daytime pain may have associations with sleep (Kravitz et al., 2015; see also Raymond et al., 2001), potentially explaining the null findings because pain in this study was from "today". However, based on previous research, even the direction of this association is unclear and deserves further study.

The cross-lagged associations between PD and pain were not significant, while the concurrent, within-person associations were significant for both groups. In other words, for both groups, variations around individual averages in

pain or PD were not associated with next day variation in PD or pain, but same-day variations in pain and PD were positively associated. This indicated that on days in which a person experiences greater than average pain, they also tended to experience greater than average PD. The relatively larger association for the chronic pain groups speaks to how chronic pain may sensitize individuals to pain, such that fluctuations in their pain cause them more distress (Clark et al., 2019; van Wilget & Keizer, 2012), and at the same time, those with chronic pain may engage in more negative patterns of cognitive-emotional appraisal, that could increase attention to pain (Crombez et al., 2013).

At the between-person level, pain and PD were associated for both groups, but the effect was significantly larger for the chronic pain group. As such, within those who report chronic pain, the average amount of pain is more significantly associated with PD relative to the same relationship in the non-chronic pain group. As described above, trait-like tendencies towards negatively biased thinking in combination with an increased sensitivity to pain may contribute to a stronger association between these variables for the chronic pain group (Neblett et al., 2017). For example, those who tend to use catastrophizing (or interpreting something as much worse than it is) may have a negative reaction following a pain flare-up ("This pain is too much!"), which may in turn be followed up by an additional distress-inducing reaction ("I will never live a normal life again.").

From a clinical perspective, imagine a client arrives to treatment with comorbid pain, insomnia, and depression. There are CBT approaches for each of these conditions but based off of the data from this study alone, emphasizing the insomnia early *may* have a more meaningful impact on the client's overall profile of daily suffering as improved sleep may improve pain and depression, which are in turn associated strongly with each other. Further, The impact of improvements in sleep on pain and PD may be reinforced by the association that those two factors have with each other. Cognitive behavioral therapy (CBT) for insomnia alone has shown some improvements on pain (Selvanathan et al., 2021), and may in fact be more effective than CBT-pain or combined CBT-I and CBT-pain for PD, sleep, and pain outcomes (Enomoto et al., 2022).

These findings, in conjunction with previous literature have some implications for the practice of behavioral medicine. The comorbidity of pain, psychological distress and sleep disturbance highlights the importance of proper assessment, yet the daily diary data from this study indicate that measurement may extend beyond simple single-time assessments of past-week or past-month symptoms but could involve more regular assessment to examine how these factors covary day-to-day (Suso-Ribera et al., 2018). Behavioral health practitioners may need to be particularly sensitive to the ways in which these factors relate to each other and the apparent preeminence of sleep when creating treatment plans and prioritizing intervention targets (Enomoto et al., 2022). Guidance from professional organizations on the importance of considering the ways these factors correlate and the need for comprehensive assessment and prioritized treatment could be delivered as best practices or care guidelines (e.g. Department of Health & Human Services, 2019).

The process-focused, observational, and ecologically valid nature of the present daily diary study's findings compliments previous the findings from randomized trials, which utilize large-interval measurement designs. If future research examines more granular, just-in-time interventions for the management of these three important factors (Nahum-Shani et al., 2018), an important "state of vulnerability/opportunity" may be just after waking from poorer night sleep. Intervening at these points might provide the opportunity counteract the potential detriments of decreased sleep on the PD and pain outcomes. Pharmacological treatments may be of use as well. Based on some early-stage research, Gabapentin, which is often used to treat chronic pain (Fan et al., 2021; Wiffen et al., 2017), can increase slow wave sleep (Rosenberg et al., 2014), help improve insomnia (Furey et al., 2014), and reduce anxiety (Ahmed et al., 2019).

Limitations

This study has various limitations that should be noted. The major ones surround the measurement of the relevant variables – large longitudinal studies with many hundreds of measured variables often sacrifice measurement depth for breadth, as is the case in this study. Sleep quantity *and* quality could have been measured with a validated scale and/or with biometric devices, especially as sleep quality seems to have stronger associations with other factors (e.g. Lücke et al., 2022). Other unmeasured factors such as obstructive sleep apnea, perceived stress, or ongoing treatment may also be playing an important and potentially confoundingrole (Babiloni et al., 2021; Kramer et al., 2019).

Total pain was assessed with three self-report items, which were in-turn summed as a composite. This was, to our understanding, the first time these items had been used like this. Also of note, chronic pain, which is often conceptualized in terms of duration (e.g. "persistent or recurrent pain lasting longer than 3 months", Treede et al., 2015), was, due to the study design, measured in the main second wave of the MIDUS study, rather than during this daily diary study. The variability in when the interviews took place (daytime vs. evening), may have impacted participants responses to each of the central variables, especially pain. Further, due

to the logic of the interview, responding to some questions positively (i.e. having had pain) led to additional follow up questions (rate the severity of pain), which may have been perceived as burdensome and led to underreporting. Another potential source of bias that could have impacted the data is impression management as the participants were speaking with an interviewer rather than responding to self-report surveys (McGrath et al., 2010). Lastly, with the sample being mainly of White racial background, the generalizability of the findings is somewhat limited.

Conclusion

The purpose of this project was to examine the daily associations among three important health-related variables: pain, sleep quantity, and PD. This included the goal of comparing the associations between these variables among those with and without chronic pain. The findings of this study indicate that sleep may play an especially important role in the daily life of those with chronic pain as daily fluctuations in sleep duration were negatively associated with next day pain and PD, while pain and PD were associated with each other within the same day. The reverse associations of PD and pain predicting sleep duration were not found. In light of other research indicating the importance of sleep relative to the other behaviors, practitioners may prioritize intervening on sleep difficulties first. Other same-day and between-person associations among these variables were found and as such, treating any of these presenting problems must involve a comprehensive assessment. Future research may examine whether intervening after a poor night's sleep may benefit those suffering from these three comorbid conditions to a greater degree than prioritizing pain and PD.

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Data Availability The Midlife in the United States (MIDUS) study data are available at the Inter-university Consortium for Political and Social Research website: https://www.icpsr.umich.edu/web/ICPSR/studies/26841. The R code and model output files are made available at https://osf.io/m65nr/?view_only=c8dc47179a52447d9d68b75d127e311d.

Declarations

Conflicts of interest/Competing interests Not applicable, no conflicts of interest to report.

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