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Daily Variations in Objective Nighttime Sleep and Subjective Morning Pain in Older Adults with Insomnia: Evidence of Covariation Over Time

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Abstract

Objectives—To examine the relationship between objectively measured nocturnal sleep and subjective report of morning pain in older adults with insomnia. The goal of the paper was to not only examine the sleep-pain association between-persons (mean-level over 14 days), but also to investigate the within-person, day-to-day association.

Design—Cross-sectional.

Setting—North-Central Florida.

Participants—Fifty community-dwelling older adults (M_{age} = 69.10 years, SD_{age} = 7.02 years, range = 60 – 90 years) with insomnia participated in the study.

Measurements—This study employed daily home-based assessment utilizing nightly actigraphic measurement of sleep and daily self-report of pain. Measures were completed over fourteen consecutive days.

Results—Between persons, average sleep over 14 days was not associated with average levels of rated pain. However, following a night in which an older adult with insomnia experienced above-average total sleep time s/he subsequently reported below-average pain ratings. The model explained approximately 24% of the within-person and 8% of the between-person variance in pain ratings.

Conclusions—Sleep and pain show day-to-day associations (i.e., covary over time) in older adults with insomnia. Such associations may suggest that common physiological systems underlie both the experience of insomnia and pain. Future research should examine the crossover effects of sleep treatment on pain and of pain treatment on sleep.

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Keywords

Sleep; Pain; Older Adults; Multilevel Modeling; Daily Associations

INTRODUCTION

Daily variations in pain[1] and sleep[2] have been documented in isolation. However, the link between these two conditions on a day-to-day basis is not well understood.

Impact of Pain

Pain can be classified by site of injury, type of injury, and duration of the pain[3]. Chronic pain is generally defined as pain persisting beyond the expected healing phase[3]. Acute pain likely transitions to chronic pain during the subacute phase, often marked by an unexplained and unexpected spread of pain to other body areas not initially affected[4].

Pain is the most common reason for presentation in hospital or clinic settings. It has been estimated that pain is implicated in 80% of physician visits[4]. Furthermore, 38% of patients presenting to a primary care physician are suffering from chronic pain[4]. Chronic pain has been recognized as the leading cause of disability in the working-age population[4]. Pain affects individuals at all stages of life. However, it is particularly prevalent among older adults, affecting 40% of independently living older adults and 27-83% of older adults living in an institutional setting[5]. In this population, pain is commonly a symptom of one or more existing health conditions.

Impact of Chronic Insomnia

Chronic insomnia is defined as a predominant complaint of difficulty initiating or maintaining sleep, or non-restorative sleep, for at least 1 month which causes clinically significant distress or impairment in social, occupational, or other important areas of functioning[6]. Chronic insomnia has been linked to significant social and monetary costs due to lost productivity, work absenteeism, and greater utilization of the health-care system[7]. Insomnia has also been linked to higher prevalence rates and higher likelihood of developing depression and anxiety. The risk for developing depression is particularly increased among older adults[8].

Co-morbidity of Pain and Insomnia

The interaction between pain and insomnia has been well documented, as illustrated by rates of comorbidity that approach 70%[9]. Often, insomnia is considered secondary to pain. However, this assumes that insomnia is due solely to pain. In reality, it is probable that insomnia takes on a semi-independent course but remains linked to pain through a third variable. The insomnia may then be perpetuated via acquired cognitions and behaviors directly affecting patients' sleep. Behaviors like daytime naps, excessive time in bed, utilization of medications affecting sleep propensity and patterns, and little exercise/activity may be particularly relevant to the development of insomnia among individuals with pain[10]. Additionally, different degrees of sleep deprivation have been linked to reduced pain thresholds among healthy adults, whereas, subsequent sleep recovery has had an analgesic effect[10].

Research has confirmed that the relationship between pain and sleep is reciprocal. Laboratory-induced pain has been found to disturb sleep in healthy participants without history of sleep problems[11]. Likewise, researchers have found that repeated disruption and

deprivation of sleep can intensify pain sensitivity (with mood correlates) in chronic pain conditions[11].

Daily Variability in Sleep

Intra-individual variability, or heightened inconsistency/fluctuations, in sleep patterns is a fundamental component of disordered sleep. Research in this area is essential to broadening our understanding of sleep as a phenomenon. However, there have been relatively few studies directly examining within-person fluctuations in sleep. Current research suggests that individuals with insomnia exhibit highly variable sleep patterns[2], whereas normal sleepers tend to exhibit less variable sleep patterns[12].

Daily Variability in Pain

Day-to-day variability in pain perception has been well-documented throughout pain research[13], and suggests that single-item/time point measurement of pain may result in inaccurate portrayal of pain[1]. Pain threshold levels can vary on a day-to-day basis, and even within a given day. In addition, pain ratings at any point in time may be affected by a number of environmental and cognitive factors including depression, anxiety, and distress.

Covariation Over Time (i.e., Within-person Coupling)

The study of dynamic covariation is primarily concerned with how two or more variables covary across multiple occasions. Previous literature has revealed: (1) aberrant night's sleep is associated with fluctuations in affect in community-dwelling elders[14], (2) fluctuations in affect are significantly associated with variability in pain in older adults[15], and (3) daily subjective sleep quality is related to daily attention to pain in women with fibromyalgia[16]. However, to our knowledge, no study has examined the dynamic association between objective sleep and self-report pain in older adults with insomnia.

The Current Study

Rather than averaging measurements to generate aggregate estimates, the current study assumes the variations in sleep and pain ratings represent natural fluctuations in the individual's physiological/psychological condition. This study sought to address two main questions: (1) Between-persons, is average level of sleep associated with self-reported pain in older adults with insomnia? (2) Within-persons, does prior night's sleep affect subsequent morning's self-report of pain? It was hypothesized that poorer sleep, on average, would be associated with the experience of increased pain. It was also hypothesized that following a better than average night of sleep, individuals would report below average levels of pain.

METHODS

Participants

Recruitment—Older adults with insomnia (60 years or older) were recruited from North Central Florida via newspaper, radio, and television advertisements to participate in a randomized, controlled trial for insomnia in late-life. This study reports on baseline measures from that study. Criteria for chronic insomnia were consistent with the Diagnostic and Statistical Manual of Mental Disorders[6]. Inclusionary criteria were: (a) individual reported insomnia (sleep onset or awake time during night > 30 minutes); (b) insomnia present at least 3 nights per week for more than 6 months; (c) daytime dysfunction due to insomnia (mood, cognitive, social or occupational impairment); (d) no prescribed or over-the-counter sleep medication for at least 1 month, or stabilized on medication for 6+ weeks. Exclusionary criteria were: (a) significant medical (e.g., cancer) or neurological disorder (e.g., dementia); (b) major psychopathology (e.g., psychotic disorders, substance abuse); (c)

other sleep disorders [e.g., sleep apnea, periodic limb movements—assessed through single-night ambulatory monitoring (see 'f' below) and structured interview]; (d) cognitive impairment based on Mini-Mental State Exam (MMSE[17]) score lower than 23 (>9th grade education) or 19 (<9th grade education)[18]; (e) severe depressive symptomatology based on Beck Depression Inventory –2nd Edition (BDI-II [19]) score of 24 or higher or Geriatric Depression Scale (GDS [20]) score of 13 or higher; (f) suspected sleep disordered breathing based on single-night ambulatory monitoring (Compass F10; Embla) of blood-oxygen saturation and respiration indicating an apnea-hypopnea index (AHI) of >15.1 and minimum O_2 desaturation <93% .

The Institutional Review Board (IRB) approved the study. All participants signed an informed consent form prior to participation.

Measures

Objective Sleep—Participants wore an actigraph, the Actiwatch-L, on their nondominant wrist for 14 consecutive days. The Actiwatch-L monitors ambient light exposure and gross motor activity and contains an omni-directional, piezoelectric accelerometer with sensitivity of ≥ 0.01 g-force. The sensors of the Actiwatch-L are sampled 32 times/second and record peak values for each second. These peak values are then summed into 30-second "activity" counts. These activity counts are downloaded to a PC and analyzed using Actiware-Sleep v. 3.3, which uses a validated algorithm to identify each epoch as sleep or wake. Bedtime and time out of bed in the morning were based on sleep diary entries as recommended in the software manual. Actiware-Sleep determined sleep start automatically by searching for the first 10 min during which no more than one epoch was scored as wake. Likewise, sleep end was the last 10 min during which no more than one epoch was scored as wake. When measured objectively by actigraphy, total wake time (TWT_o) represents the sum of all wake epochs within the sleep period and total sleep time (TST_o) represents the sum of all sleep epochs between bedtime and time out of bed in the morning.

Subjective Pain—Pain was subjectively evaluated daily through participants' response to the question, "What is your current pain level?" Participants rated their pain on a 0 (No Pain) to 10 (Worst Possible Pain). This item corresponds to criteria recommended in a consensus statement by chronic pain researchers[21] but is still subject to the day-to-day and within day variability that all single time point measures are subject to. A change of 1.0 point on this type of scale has been associated with minimally important changes in pain intensity[21].

Analysis

The aim of the current study is to examine the predictive power of within-person and between-person objectively measured sleep variables on self-reported pain. To accomplish this, daily data from the objective sleep measures (TST_o and TWT_o) were used to predict pain level applying a multilevel model (MLM) approach. This provided the opportunity to examine how well sleep predicts pain both within- (level 1: across days) and between- (level 2: across persons) persons. Level 1 submodels addressed questions such as: "On days in which a person reports above-average total sleep time, does s/he also experience subsequent lower levels of pain?" This is accomplished through calculation of person-centered sleep variables (individual day-to-day fluctuations in amount of sleep around an individual's intrapersonal mean-level of sleep). Level 2 submodels examined questions like: "Do people who are generally poorer sleepers report higher levels of pain?". This is accomplished through calculation of mean-level sleep variables. Mean-level sleep variables represent average-level of sleep across the 14-day study period. The final model predicted daily pain with: average level of pain, linear time, demographic variables (age, gender, and total

number of medications taken), mean-level objective sleep scores, daily-centered objective sleep scores, random error term, and random residual component.

All variables were standardized into Z-score metrics between- and within-participants (thus, the average participant on the average day would have a mean of 0.0, standard deviation of 1.0) prior to parameterization of the MLM. This approach preserved both between- and within-person differences, while facilitating interpretation of model parameters. As such, the model-produced coefficients are similar to traditional standardized regression coefficients in ordinary least squares regressions. The specifics of MLM are beyond the scope of this paper. Interested readers are referred to other sources (e.g., [14]).a

RESULTS

Sample Characteristics

A total of 484 individuals initially responded to advertisements for participants. 328 individuals declined participation after receiving further information over the phone. Of the 156 persons who attended the screening appointment, 55 dropped out of the study for personal reasons [i.e., 5 = illness/health issues, 27 = study inconvenience (e.g., study length, study intensity, distance needed to travel), 12 = too busy, 4 = miscellaneous issues, 7 = reason not reported/missing], 12 were ruled out because they did not meet other criteria, 11 were ruled out for possible apnea/hypopnea, and one did not have insomnia. Thus, 77 individuals participated in baseline assessment. However, an additional 27 individuals had substantial missing data (24 had missing AHI data which precluded ruling out an apnea diagnosis, and 3 had missing demographic data) and were thus excluded from the present analyses.

The final sample included fifty older adults with insomnia (M_{age} = 69.10 years, SD_{age} = 7.02 years). Specific sample descriptive characteristics (including demographics, sleep, and pain information) can be found in Table 1. Self report of pain conditions revealed the following: 18 with arthritis, 5 with lower back pain, 1 with osteoporosis, 1 with fibromyalgia, and 25 without a specific pain condition. Frequently reported health conditions in participants' medical histories included: heart disease, cancer, high blood pressure, breathing problems, diabetes, and urinary tract infections. In general, the sample was comprised of young-old, highly educated, mostly healthy, and predominantly female Caucasians with chronic insomnia.

Multilevel Model

Prior to the parameterization of the MLM, multicollinearity between the predictor variables of TST_o and TWT_o was examined through estimation of a multivariate mixed-effects null model. The analysis revealed that TST_o and TWT_o were not significantly correlated at the between-person level ($p = 0.25$). However, TST_o and TWT_o were significantly correlated at the within-person level ($r = -0.24$, $p < 0.01$). Given the relatively small, yet significant, correlation between these two predictor variables we further examined potential multicollinearity by running all MLMs twice, once using raw variables and once using residualized variables to account for potential multicollinearity. Results indicated no substantial changes in the pattern or significance of results. Thus, multicollinearity among predictor variables does not appear to be problematic and all presented results are based on model parameterization using raw values.

The intraclass correlation coefficient (ICC), which serves as an index of within and between-person variability to be explained, was 0.64. Thus, the ICC indicates that 36% of the overall variability in pain ratings is a within-person phenomenon and 64% is a between-

person phenomenon. Thus, a MLM analytical framework, which separates within- and between-person variance components, is warranted.

In the final MLM predicting pain, there were no significant between-person (level 2) predictors. At the within-person level (level 1) both the predictors of Time, β = -0.09, $t(30.15) = -2.14$, $p < .05$, and TST_o, β = -0.10, $t(29.44) = -2.12$, $p < .05$ were significant, suggesting that individuals' pain ratings decreased over time (potentially a reaction to measurement) and that following a night of above/below average TST_o individuals reported below/above average pain. Based on Robinson et al. (2005), on 23.4% of days patients met criteria for clinically meaningful improvement in pain[22]; just over 50.0% (i.e., 50.7%) of these days were preceded by a prior night of above-average sleep. The model explained approximately 24% of the within-person variance and 8% of the between-person variance in pain ratings. See Table 2 for a total listing of predictor estimates, significance levels, variances explained, and model parameters for the final MLM.

DISCUSSION

The current literature posits that insomnia does not result in consistently worse objective daytime impairment assessed as sleepiness, physiological arousal (pupillometry, oral temperature, and pulse rates), cognitive performance, or psychopathology (specifically depression and anxiety)[23]. The one domain of daytime impairment that was routinely found among individuals with insomnia is fatigue (feeling physically or mentally tired). In contrast, results of the current study reveal that following a night of above or below average TST_o older adults with insomnia experience subsequent below or above average pain intensity. These results suggest that although insomnia may not be associated with consistent daytime impairments, deviations in sleep commonly observed in insomnia may be related to deviations in daytime consequences (i.e., pain).

The connection between daily sleep variability and daily pain variability may be moderated by a number of different processes, likely due to changes in biological mechanisms and cognitive processes. The effects of the hypothalamic-pituitary-adrenal (HPA) axis may lead to hyperarousal. Another possible biological explanation is related to the theory of central sensitization which emphasizes the role of hyperactivation of nociceptive transmitters in the spinal cord and brain[24]. These two theories are not contradictory but instead simply emphasize different communication systems within the body. Sleep difficulties and pain may feed both of these systems information, conjointly or in isolation, signaling environmental threat and resulting in a heightened sympathetic response. These processes may facilitate a reciprocal relationship between sleep and pain, as both conditions revolve around heightened sensitivity to environmental stimuli and heightened activation. Cognitive-behavioral theories link chronic pain conditions and insomnia through avoidant safety behaviors and catastrophizing thoughts. Together, these may lead to a reciprocal loop of emotional and cognitive distress[25]. There is also the possibility that there is no reciprocal association between pain and sleep systems and that awareness of pain is simply more likely among individuals who are awake to perceive it, or who are awakened by it.

To explore the possibility that sleep and pain share a dynamic reciprocal relationship in older adults with insomnia, a subsequent exploratory MLM was modeled (between-person [mean-level over 14 days] and within-person [day-to-day fluctuation] pain ratings predicting TST_o and TWT_o). Prior to running the MLM, all data were restructured such that prior day's pain rating would precede subsequent night's sleep. The model was parameterized similarly to those previously described. Results indicated within-person fluctuations in pain rating did not predict subsequent night's TST_o or TWT_o, $p = 0.21$ and 0.13, respectively. Similarly,

individuals who experienced higher levels of pain on average did not also experience worse average TST_o or TWT_o, $p = 0.72$ and 0.11, respectively.

This study may have limited generalizability to the general population due to the selective sample (limited demographic variability), small sample size, and high rates of drop out, non-consent, and missing AHI data. Given that the sample was largely female, the pain reports may be biased, as past research suggests that men and women experience and report pain differently[26]. Further, our sample was largely healthy, young-old, and highly educated—all of which may affect the report of pain. The sleep-pain relationships in the study may be influenced by the timing and frequency of the measurement of each. In the MLM where sleep predicts subsequent morning's pain, there was a relatively short time lag between sleep measurements and morning pain ratings. However, the data was restructured so that morning pain predicted subsequent night's sleep, had longer lag between morning pain ratings and measurements of subsequent night's sleep. Pain was not measured in proximity to sleep onset or during the night. As previously mentioned, pain varies throughout the day and morning pain ratings may be very different than evening pain rating due to activity, analgesic use, and other factors. The significant findings in the MLM indicating sleep predicts subsequent pain and not vice-versa may be a function of timing differences in the measurement of pain and sleep. Future studies should include additional recordings of pain that are proximal to sleep measurements. Of note, the present study included limited clinical information on the sample and their comorbidities resulting in the inclusion of few independent variables in the model. Future studies may consider examining possible relationship between comorbid conditions and nighttime sleep and morning pain ratings.

Cognitive-Behavioral Therapy for insomnia is effective in alleviating insomnia-related complaints in older adults[27] and preliminary research suggests improvements in pain perception may follow psychological treatment of insomnia[28]. Sleep restriction[29] actively aims at eliminating much of the inherent fluctuation in insomnia patients' sleep but the effects of this treatment on subsequent daily variation of pain perception has yet to be examined. Cognitive-Behavioral Treatment for pain has also demonstrated promise in the treatment of various pain conditions[30]. Subsequent research should examine cross-over effects of treatment on comorbid sleep disturbances. Future research might also benefit from the addition daily measures of affect along with physiological measures of diurnal and circadian variation, such as cortisol and melatonin. All of these could add significant insight into the shared variability of sleep and pain.

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APPENDIX

Briefly, objective sleep measures were used to predict pain ratings using a seven-step MLM approach (i.e.,14). In general, hierarchical model building approach was adopted. Step 1, the null (baseline) model, estimated only fixed and random intercept for pain rating and served as a comparison for later models. In step 2, time functions (linear) were added as covariates to the null model to control for any within-person inflations that may be caused by a

systematic change in the data. Next, demographic variables were added as covariates to the model influences as a result of age, gender, or medication use. In steps 4-7, the estimates of the fixed and random intercepts and fixed linear slopes for TST and TWT were added one variable per step. Thus, the daily pain ratings ($Pain_{ij}$) for each person were predicted by: average level of pain (γ_{00}), linear time (β_{1j}), between-person effects of demographic variables, between-person effects of mean-level objective sleep scores, within-person effects of daily-centered objective sleep scores, a between-person random error term (u_{0j}), and a within-person random residual component (e_{ij}). Random effects test whether there are significant individual differences in the size of a parameter. Thus, random between-person intercepts examine whether the intercept is the same for all participants; the random within-person slopes examine whether the association between a predictor and an outcome from day-to-day is the same for all persons.

The model was estimated under the repeated error assumptions of homogeneous variance and diminishing correlations over time (i.e., *first-order autoregressive*) and under the random error assumptions of homoscedasticity and independence of errors (i.e., *diagonal*). The model also employed the Maximum Likelihood (ML) method of estimation, because this provides the most accurate estimates of random effects and allows for the calculation of Deviance statistics. The ability of the model to predict pain better than the baseline model (i.e., Deviance, expressed as -2 Log Likelihood difference between models, which is distributed as a chi-squared statistic) was used as an index of Goodness of Fit. Improvements in prediction were determined by the amount of reduction of within-person residual variances and between-person intercept variances compared to the baseline model. Decreases in residual and intercept variances represent a proportional reduction of the prediction error, which is analogous to R^2, and were used as an estimate of within-person and between-person effect sizes.

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Table 1

Participant Descriptive Statistics (N=50).

	Minimum	Maximum	Mean	Std. Deviation
Participant Demographics				
Age[a]	60.0	90.0	69.10	7.02
Gender[b]	1.0	2.0	1.66	--
Education[a]	12.0	22.0	16.26	2.73
Insomnia Duration[a]	.5	50.0	12.23	14.53
Medications	1.0	13.0	5.26	2.86
Sleep Characteristics[c]				
Total Wake Time	0.0	362.0	63.52	44.58
Total Sleep Time	81.5	629.0	392.45	88.60
Pain Characteristics				
Pain Rating	0.0	9.0	1.67	1.85
Pain Conditions (# reporting)				
Arthritis	18			
Back Pain	5			
Osteoporosis	1			
Fibromyalgia	1			
No Specific Condition	25			

Notes:

[a] units of measurement in years

[b] gender measured 1=male, 2=female

[c] all sleep variables measured in minutes.

Table 2

Multilevel Model Predicting Daily Pain.

	Fixed Effects	
Predictor Variable	***B* (SE)**	***t* (*df*)**
Within-person		
Time	-0.09 (0.04)	-2.14 (30.15)*
$TWT_{centered}$	-0.0002 (0.03)	-0.006 (428.88)
$TST_{centered}$	-0.10 (0.05)	-2.12 (29.44)*
Between-person		
Age	-0.13 (13)	-1.01 (40.79)
Gender	0.08 (0.14)	0.57 (39.74)
Medication	0.02 (0.14)	0.16 (39.26)
TWT_{mean}	0.23 (0.21)	1.09 (38.72)
TST_{mean}	-0.05 (0.18)	-0.26 (39.68)
	Random Effects	
Covariance parameter estimate	*Variance* (SE)	*Z*
Time	0.03 (0.02)	1.77
$TWT_{centered}$	0.000[a]	0.000[a]
$TST_{centered}$	0.03 (0.02)	1.56
	Within Pseudo R^2	0.24
	Between Pseudo R^2	0.08

Notes:

** p <0.01.

* $p < 0.05$

[a] Variance too small to be estimated- The final Hessian matrix was not positive definite although all convergence criteria were satisfied.