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

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OBJECTIVES: To examine the relationship between objectively measured nocturnal sleep and subjective report of morning pain in older adults with insomnia; to examine not only the difference between persons in the association between sleep and pain (mean level over 14 days), but also the within-person, day-to-day association.

DESIGN: Cross-sectional.

SETTING: North-central Florida.

PARTICIPANTS: Fifty community-dwelling older adults (mean age ± standard deviation 69.1 ± 7.0, range 60–90) with insomnia.

MEASUREMENTS: Daily home-based assessment using nightly actigraphic measurement of sleep and daily self-report of pain over 14 consecutive days.

RESULTS: Between persons, average sleep over 14 days was not associated with average levels of rated pain, but after a night in which an older adult with insomnia experienced above-average total sleep time he or she 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 the experience of insomnia and pain. Future research should examine the crossover effects of sleep treatment on pain and of pain treatment on sleep. **J Am Geriatr Soc 58:925–930, 2010.**

Key words: sleep; pain; older adults; multilevel modeling; daily associations

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

EFFECT OF PAIN

Pain can be classified according to 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 probably transitions to chronic pain during the subacute phase and is 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, although it is particularly prevalent in older adults, affecting 40% of independently living older adults and 27% to 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.

EFFECT OF CHRONIC INSOMNIA

Chronic insomnia is defined as a predominant complaint of difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month and that 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

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DOI: 10.1111/j.1532-5415.2010.02803.x

0002-8614/10/$15.00

lost productivity, work absenteeism, and greater use of the healthcare 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 high in older adults.[8]

COMORBIDITY OF PAIN AND INSOMNIA

The interaction between pain and insomnia has been well documented, as illustrated by rates of comorbidity that approach 70%.[9] Insomnia is often considered secondary to pain, but 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 through acquired cognitions and behaviors directly affecting patients' sleep. Behaviors like daytime naps, excessive time in bed, use of medications affecting sleep propensity and patterns, and little exercise or activity may be particularly relevant to the development of insomnia in individuals with pain.[10] Additionally, different degrees of sleep deprivation have been linked to lower pain thresholds in 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

Intraindividual variability, or heightened inconsistency or fluctuations, in sleep patterns is a fundamental component of disordered sleep. Research in this area is essential to broadening understanding of sleep as a phenomenon, but there have been 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 in pain research[13] and suggests that single-item or time point measurement of pain may result in inaccurate portrayal of pain.[1] Pain threshold levels can vary day to day and even within a given day. In addition, a number of environmental and cognitive factors including depression, anxiety, and distress may affect pain ratings at any point in time.

COVARIATION OVER TIME (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 that aberrant night's sleep is associated with fluctuations in affect in older community-dwelling adults,[14] that fluctuations in affect are significantly associated with variability in pain in older adults,[15] and that daily subjective sleep quality is related to daily attention to pain in women with fibromyalgia,[16] but no study was found that 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 assumed that the variations in sleep and pain ratings represent natural fluctuations in the individual's physiological and psychological condition. This study sought to address two main questions: Between persons, is average level of sleep associated with self-reported pain in older adults with insomnia? 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 greater pain. It was also hypothesized that, after a better-than-average night of sleep, individuals would report below-average levels of pain.

METHODS

Participants

Recruitment

Older adults with insomnia (aged $\geq$60) were recruited from north-central Florida through 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 $>$30 minutes), (b) insomnia present at least 3 nights per week for more than 6 months, (c) daytime dysfunction due to insomnia (mood, cognitive, social, or occupational impairment), and (d) no prescribed or over-the-counter sleep medication for at least 1 month or stabilized on medication for 6 or more weeks. Exclusionary criteria were (a) significant medical (e.g., cancer) or neurological (e.g., dementia) disorder, (b) major psychopathology (e.g., psychotic disorders, substance abuse), (c) other sleep disorders (e.g., sleep apnea, periodic limb movements—assessed through single-night ambulatory monitoring (see "f" below) and structured interview), (d) cognitive impairment based on Mini-Mental State Examination[17] score lower than 23 ($>$9th grade education) or 19 ($<$9th grade education),[18] (e) severe depressive symptomatology based on Beck Depression Inventory, Second Edition,[19] score of 24 or higher or Geriatric Depression Scale[20] score of 13 or higher, and (f) suspected sleep-disordered breathing based on single-night ambulatory monitoring (Compass F10; Embla, Broomfield, CO) of blood-oxygen saturation and respiration indicating an apnea–hypopnea index (AHI) of greater than 15.1 and minimum oxygen desaturation less than 93%.

The institutional review board at the University of Florida approved the study. All participants signed informed consent form before participation.

Measures

Objective Sleep

Participants wore an actigraph, the Actiwatch-L (Mini Mitter, a Respironics Company, Bend, OR) on their nondominant wrist for 14 consecutive days. The Actiwatch-L monitors ambient light exposure and gross motor activity and contains an omnidirectional piezoelectric accelerometer with sensitivity of 0.01 g-force or greater. The sensors of the Actiwatch-L are sampled 32 times per 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 personal computer 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 minutes during which no more than one epoch was scored as wake. Likewise, sleep end was the last 10 minutes during which no more than one epoch was scored as wake. When measured objectively using 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 scale of 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 was 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 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 he or she 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 such as: "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, sex, 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) before parameterization of the MLM. This approach preserved 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 article. Interested readers are referred to other sources (e.g.,[14]). For a brief review of MLM, please see Appendix A.

RESULTS

Sample Characteristics

Four hundred eighty-four individuals initially responded to advertisements for participants; 328 declined participation after receiving further information over the telephone. Of the 156 persons who attended the screening appointment, 55 dropped out of the study for personal reasons (27 for study inconvenience (e.g., study length, study intensity, distance needed to travel), 12 for too busy, 7 for reason not reported or missing, 5 for illness or health problems, 4 for miscellaneous issues), 12 were excluded because they did not meet other criteria, 11 were excluded for possible apnea or hypopnea, and one did not have insomnia. Thus, 77 individuals participated in baseline assessment, although an additional 27 individuals had substantial missing data (24 had missing AHI data, which precluded excluding an apnea diagnosis, and 3 had missing demographic data) and were thus excluded from the analyses.

The final sample included 50 older adults with insomnia (mean age 69.1 ± 7.0). Specific sample descriptive characteristics (including demographics, sleep, and pain information) can be found in Table 1. Self-report of pain conditions revealed 18 with arthritis, five with lower back pain, one with osteoporosis, one with fibromyalgia, and 25 without a specific pain condition. Frequently reported health conditions in participants' medical histories were heart disease, cancer, high blood pressure, breathing problems, diabetes mellitus, and urinary tract infections. In general, the sample consisted of young-old, highly educated, mostly healthy, and predominantly female Caucasians with chronic insomnia.

Multilevel Model

Before 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 ($P = .25$), although they were significantly correlated at the within-person level ($r = -0.24$, $P < .001$). Given the small, yet significant, correlation between these two predictor variables, potential multicollinearity was further examined by running all MLMs twice, once using raw variables and once using residualized variables to

Table 1. Participant Descriptive Statistics (N = 50)

Variable	Value
Participant demographics	
Age, mean ± SD (range)	69.1 ± 7.0 (60–90)
Sex	
Male	17
Female	33
Education, years	16.3 ± 2.7 (12–22)
Insomnia duration, years	12.2 ± 14.5 (0.5–50)
Medications	5.3 ± 2.9 (1–13)
Sleep characteristics, minutes, mean ± SD (range)	
Total wake time	63.5 ± 44.6 (0–362)
Total sleep time	392.4 ± 88.6 (81.5–629)
Pain rating, mean ± SD (range)	1.7 ± 1.8 (0–9)
Pain conditions (n reporting)	
Arthritis	18
Back pain	5
Osteoporosis	1
Fibromyalgia	1
No specific condition	25

SD = standard deviation.

Table 2. Multilevel Model Predicting Daily Pain

	Fixed Effects	
Predictor Variable	*B* (Standard Error)	*t* (Degrees of Freedom)
Predictor variable		
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)
Sex	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 (Standard Error)	*Z*
Time	0.03 (0.02)	1.77
$TWT_{centered}$†	0.00	0.00
$TST_{centered}$	0.03 (0.02)	1.56

$P < .05$.

† Variance too small to be estimated; the final Hessian matrix was not positive definite, although all convergence criteria were satisfied.

Within pseudo coefficient of determination (R^2) = 0.24; Between pseudo R^2 = 0.08.

TWT = total wake time; TST = total sleep time.

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) the predictors of time ($\beta = -0.09$, $t(30.15) = -2.14$, $P = .04$) and TST_o ($\beta = -0.10$, $t(29.44) = -2.12$, $P = .04$) were significant, suggesting that individuals' pain ratings decreased over time (potentially a reaction to measurement) and that, after a night of above- or below-average TST_o, individuals reported below- or above-average pain. Based on previous research, on 23.4% of days, patients met criteria for clinically meaningful improvement in pain;[22] a night of above-average sleep preceded 50.7% of these days. 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 in individuals with insomnia was fatigue (feeling physically or mentally tired). In contrast, results of the current study reveal that, after 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 (pain).

A number of different processes, probably due to changes in biological mechanisms and cognitive processes, may moderate the connection between daily sleep variability and daily pain variability. The effects of the hypothalamic-pituitary-adrenal 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, because 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 in 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). Before 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 that within-person fluctuations in pain rating did not predict subsequent night's TST_o ($P = .21$) or TWT_o ($P = .13$). Similarly, individuals who experienced higher levels of pain on average did not also experience worse average TST_o ($P = .72$) or TWT_o and ($P = .11$).

This study may have limited generalizability to the general population because of the selective sample (limited demographic variability); small sample size; and high rates of dropout, nonconsent, and missing AHI data. Given that the sample was largely female, the pain reports may be biased, because past research suggests that men and women experience and report pain differently.[26] Furthermore, the sample was largely healthy, young-old, and highly educated—all of which may affect the report of pain. The timing and frequency of the measurement of the sleep–pain relationships in the study may have influenced those relationships. In the MLM, where sleep predicts subsequent morning's pain, there was a short time lag between sleep measurements and morning pain ratings. Although the data were restructured so that morning pain predicted subsequent night's sleep, there was a long 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 different from evening pain rating because of activity, analgesic use, and other factors. The significant findings in the MLM indicating that 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. 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 a 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 that 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 crossover effects of treatment on comorbid sleep disturbances. Future research might also benefit from the addition of 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.

ACKNOWLEDGMENTS

Conflict of Interest: The editor in chief has reviewed the conflict of interest checklist provided by the authors and has determined that the authors have no financial or any other kind of personal conflicts with this paper.

This study was supported by the National Institutes of Health, National Institute on Aging (AG0244591; McCrae-PI). Joseph M. Dzierzewski was supported by an Institutional Training Grant (T32-AG-020499) awarded to the University of Florida and by an Individual Training Grant (F31-AG-032802), both awarded by the National Institute on Aging.

Author Contributions: Joseph M. Dzierzewski: conceptualization, acquisition of subjects and data, analysis and interpretation of data, and preparation of manuscript. Jacob M. Williams and Daniela Roditi: conceptualization, interpretation of data, and preparation of manuscript. Michael Marsiske: study design, acquisition of subjects, critical review and feedback, and interpretation of data. Karin McCoy: study design, critical review and feedback. Joseph McNamara and Natalie Dautovich: acquisition of subjects and data, critical review and feedback. Michael E. Robinson: interpretation of data, critical review and feedback. Christina S. McCrae: study design, acquisition of subjects and data, interpretation of data, critical review and feedback.

Sponsor's Role: The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute on Aging or the National Institutes of Health, nor was either funding institution responsible for the design, methods, subject recruitment, data collection, analysis, or preparation of manuscript.

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APPENDIX A

Briefly, objective sleep measures were used to predict pain ratings using a seven-step multilevel model approach (i.e.,[14]). In general, a 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 a systematic change in the data may cause. Next, demographic variables were added as covariates to the model influences as a result of age, sex, or medication use. In Steps 4 through 7, the estimates of the fixed and random intercepts and fixed linear slopes for total sleep time and total wake time were added one variable per step. Thus, the daily pain ratings ($Pain_{ij}$) for each person were predicted according to 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, and 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 (first-order autoregressive) and under the random error assumptions of homoscedasticity and independence of errors (diagonal). The model also employed the maximum likelihood 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-square statistic) was used as an index of goodness of fit. Improvements in prediction were determined according to the amount of reduction of within-person residual variances and between-person intercept variances from the baseline model. Decreases in residual and intercept variances represent a proportional reduction of the prediction error, which is analogous to coefficient of determination, and were used as an estimate of within-person and between-person effect sizes.