Disability and Health Journal 13 (2020) 100908

Contents lists available at ScienceDirect

Disability and Health Journal

journal homepage: www.disabilityandhealthjnl.com

Original Article

Cross-lagged longitudinal analysis of pain intensity and sleep disturbance

Dagmar Amtmann, PhD [*], Alyssa M. Bamer, MPH, Robert Askew, PhD [1], Mark P. Jensen, PhD

University of Washington, Rehabilitation Medicine, Box 354237, 4907 25th Ave NE, Seattle, WA, 98105, USA

ARTICLE INFO

Article history:
Received 15 February 2019
Received in revised form
23 January 2020
Accepted 8 February 2020

Keywords:
Disability
Insomnia
Sleep
Cross-lagged panel design
Pain

ABSTRACT

Background: There is substantial evidence that pain intensity and sleep are related, with findings generally suggesting more support for the influence of sleep on pain intensity than vice versa. However, the strength and direction of the relationship has been found to vary among different populations, with few studies in individuals with chronic physical disabilities.
Objective: Examine the directionality of the sleep and pain relationship in individuals with chronic physical disabilities.
Methods: Cross-lagged effects models were generated using data from a longitudinal observational survey study of individuals (N = 1660) with multiple sclerosis (MS), muscular dystrophy (MD), post-polio syndrome (PPS), and spinal cord injury (SCI). Models evaluated the correlational effects of sleep disturbance and pain intensity, as well as the cross-lagged effects of sleep disturbance to pain intensity and vice versa.
Results: The effects of pain on sleep were stronger than sleep on pain, although the magnitude of the effects were both relatively weak. Analyses within individual samples were consistent with the overall sample results for MS, MD, and PPS. In the SCI sample the magnitude and direction of the cross-lagged model paths were more variable than in the other samples.
Conclusions: The relationship between pain intensity and sleep disturbance appears bi-directional, but the effects are small in a sample of individuals with long-term disabilities. The temporal effects of pain on sleep disturbance appear stronger than the effects of sleep disturbance on pain intensity. Future research is needed to better understand this relationship in the context of pain and/or sleep disturbance treatments.

Introduction

Pain and sleep dysfunction commonly co-occur. For example, 40% of individuals with chronic pain were found to meet criteria for poor sleep quality, compared to 22% of those without chronic pain in a large (N = 2635) population based sample.[1] Published studies in individuals with chronic disabilities indicate that pain is a common problem, including people with multiple sclerosis (MS),[2] spinal cord injury (SCI),[3] post-polio syndrome (PPS),[4] and muscular dystrophy (MD).[5] People living with these conditions also report elevated levels of sleep dysfunction (MS,[6] SCI,[7] PPS,[8] MD[9]), although sleep research in people with these conditions is limited.

Evidence also indicates that there is a relationship between sleep and pain intensity, although the direction of the relationship is not completely clear and the moderates and mediators of the relationship are not yet well understood. A review by Finan et al. (2013)[10] concluded that for a variety of disorders, the impact of sleep on pain tends to be stronger than the impact of pain on sleep. For example, six out of nine recent prospective studies testing both directional effects found evidence that sleep problems are more commonly associated with greater pain later on than more pain is associated with more sleep problems at subsequent time points.[10] However, three studies[11–13] also found support for a bi-directional effect that is equal or may even be in the opposite

* Corresponding author.
E-mail addresses: dagmara@uw.edu (D. Amtmann), adigiaco@uw.edu (A.M. Bamer), raskew@stetson.edu (R. Askew), mjensen@uw.edu (M.P. Jensen).
[1] Present address: Stetson University, 421 N. Woodland Blvd., Unit 8281, DeLand, Florida 32723, USA.

https://doi.org/10.1016/j.dhjo.2020.100908
1936-6574/

direction. Thus, the magnitude of the effects of sleep on pain and of pain on sleep may vary across different conditions associated with chronic pain.

There are a number of psychological and biological mechanisms that may explain the relationships found between sleep and pain. For example, researchers have found that psychological factors, such as depression, are not only associated with both sleep problems and pain in individuals with chronic pain[14–16], but may also mediate some of the associations found between sleep and pain.[17,18] In addition, many of the neurophysiological structures and processes that underlie sleep and pain are shared [see reviews[19,20]], and many treatments that influence one symptom are also found to influence the other, in both humans [see reviews[19,20]] and animals.[21] This significant overlap in both psychological and biological mechanisms that contribute to sleep problems may explain how sleep problems can influence subsequent pain and vice versa. However, as noted previously, the relative strength of the association between sleep problems and pain at a subsequent time point, and between pain and sleep problems reported at the next measurement, has been found to vary between pain populations, perhaps due to between-population differences in how the psychological and physiological factors contribute to these symptoms. Given the frequency and severity of both pain and sleep problems in individuals with long-term disabilities, it would be important to understand how sleep problems and pain relate to each other over time in order to determine when and if clinicians might treat one problem in order to influence the other.

Given these considerations, in the current project we sought to increase our understanding of the role of sleep on pain and vice versa in a large sample of individuals with long-term physical disabilities. Because of the potentially bi-directional associations between pain intensity and sleep disturbance, we used a longitudinal cross-lagged path model to estimate the direction and magnitude of the associations. This modeling technique is commonly used to address reciprocal influences (or feedback relationships) between constructs simultaneously.[22] Based on prior research and theory supporting mutual causation, we hypothesized that the association between sleep and pain would be positive in our disability sample (i.e., higher pain would be associated with more sleep problems at the subsequent time point and vice versa). In addition, given prior research showing a tendency for sleep problems to have a larger effect on pain intensity than the effects of pain intensity on sleep problems, we also hypothesized that this same pattern of associations would emerge in our sample.

Methods

Participants and procedures

Data for this study came from a large survey study that included four measurement time points (Time 1-Time 4) of individuals with conditions commonly associated with chronic physical disabilities. Eligible individuals were required to be able to read and write English, be at least 18 years of age when enrolled, and have a self-reported diagnosis of MS, SCI, MD, or PPS. Participants were recruited between July 2009 and March 2010 through a registry of previous research participants (68%) or via advertisements or word of mouth (32%). Paper surveys were sent to all eligible participants ($N = 2041$). Reminder letters were sent to non-responders after 4 weeks and an additional reminder call was made to non-responders after 6 weeks. The surveys included questions regarding symptoms (e.g., pain and fatigue) and psychosocial well-being. Each completed survey was checked for missing data, and participants were called by phone up to three times to collect missing data. The average time between surveys was 10 months (Time 1 to Time 2), 18 months (Time 2 to Time 3), and 9 months (Time 3 to Time 4). Of the 1862 individuals who completed the first (Time 1) survey, only those between 45 and 65 years (n = 1043) were invited to complete Time 2 and 3 surveys. All individuals who completed Time 1 were invited to complete Time 4 ($n = 1814$; $n = 48$ were not invited due to death or study withdrawal). Response rates were 91%, 95%, 91%, and 88% for Time 1–4 surveys respectively.

Individuals provided informed consent and were paid $25 per completed survey. The Human Subjects Division of the University of Washington approved all study procedures, and the research was conducted in accordance with the Declaration of the World Medical Association (www.wma.net). More information about the research participants and procedures can be found in previously published manuscripts.[23,24]

Measures

Symptoms and health indicators

The PROMIS® sleep disturbance v.1.0 short form (Time 1 & 2) and profile (Time 3 & 4)[25] were utilized as the primary sleep related outcome measures in this study (www.nihpromis.org). PROMIS measures were developed as banks of items calibrated to an item response theory (IRT) model and centered on a normative sample representative of the 2000 U.S. Census on age, gender, race, and ethnicity.[26] Scores are reported on a T-score metric with a mean of 50 and a standard deviation (SD) of 10 with higher sleep disturbance scores indicating worse sleep.

Pain intensity was measured using a numerical rating scale for pain intensity (NRS-I), with response options ranging from 0 indicating "*None*" to 10 indicating "*Very severe*." The NRS-I has been widely used in pain research. Review articles[27,28] have provided strong evidence for criterion-related validity and sensitivity to change in a variety of settings and populations.

Covariates

Model covariates include age, sex, the 9-item Patient Health Questionnaire (PHQ-9)[29] and PROMIS® fatigue[30] short form v.1.0. Fatigue and depression have been shown to be associated with both sleep and pain[31–33]and were therefore considered cofounders in this study. The PHQ-9 is a widely used measure of depression, with higher scores indicating more depressive symptoms. As with the PROMIS® Sleep measure, PROMIS® fatigue is on a T-score metric with higher scores indicating more fatigue.

Analyses

We used longitudinal cross-lagged path models[22] to investigate the reciprocal relationship sleep and pain have on each other. Pain intensity at Time 2, 3, and 4 was model by the previous score of sleep disturbance (e.g. sleep Time 1 onto pain Time 2) as well as its own score at the previous time point using first-order auto-regressive effects (e.g. pain Time 1 onto pain Time 2). To further account for large residual covariances between non-adjacent time points, the second-order auto-regressive effects were also included (e.g. pain Time 1 onto pain Time 3). Similar models were created for sleep, in which sleep at Time 2, 3, and 4 was modeled by the previous pain score as well as first and second order auto-regressive effects of prior sleep scores. In addition, age, sex, fatigue, and depression were entered in all models as time-invariant covariates measured at baseline, as they were not available at all timepoints, to control for possible confounding.

The analyses were conducted in two stages. First, a cross-lagged model was fit and evaluated for each diagnosis group separately to examine potential differences in the associations between pain and

sleep by diagnostic group. Second, in order to determine if an overall model for the combined sample was appropriate, a model was fit which included an interaction term between diagnosis group and either pain or sleep at the previous timepoint of the opposite outcome (e.g. pain at Time 3 was modeled by sleep Time 2, diagnosis group, sleep*diagnosis, pain Time 1, and pain Time 2). A lack of significance ($p > 0.05$) in the interaction terms was used as evidence to support fitting one combined model for all diagnostic groups.

Using Mplus Software 7.4,[34] we analyzed the data for descriptive statistics, and overall goodness of model fit of the cross-lagged models. Model fit was evaluated using χ^2, the comparative fit index (CFI),[35] the Tucker-Lewis index (TLI),[36] and the root mean square error of approximation (RMSEA). CFI and TLI values above 0.95[35] indicate a close fit, and an RMSEA value of less than 0.08 indicates a fair fit.[37] Standardized beta coefficients were examined across models and were interpreted similar to correlations with $\beta < 0.2$ considered a weak effect, $0.2 < \beta < 0.5$ a moderate effect, and $\beta > 0.5$ a strong effect (p.272 in Acock, 2014[38]).

Results

Participants

Mplus software[34] does not allow missing values of covariates (PROMIS fatigue, PHQ-9, sex and age) in the cross-lagged effect analysis. Thus 41 (2%) of the 1701 participants at time 1 were excluded due to missing data. The remaining sample was majority female (n = 1062; 64%), non-Hispanic white (n = 1519; 92%), married (n = 974; 59%), and had at least some college education (n = 1,333, 80%).

On average, all participants reported worse symptoms and quality of life indicators than the general U.S. population (e.g., more fatigue, sleep disturbance, and pain intensity). Individuals with MS and SCI reported the most sleep disturbance (MS: Mean = 53.01; SCI: Mean = 52.35) among the four diagnostic groups. Individuals with PPS and MS reported the greatest fatigue (MS: Mean = 58.66; SCI: Mean = 8.23); and individuals with MS reported the highest depression scores (Mean = 7.11; SD = 5.30), compared to the group average of 6.38 (SD = 5.08). Further, participants with SCI reported the worst pain intensity (M = 4.43; SD = 2.67) of all of the groups. Descriptive statistics are presented in Table 1.

Analyses

Subgroups

Table 2 shows the fit and magnitude of standardized coefficients for the cross lagged models for MS, SCI, MD, and PPS individually. Fit indices from the four diagnostic group models were acceptable; CFIs across the four groups ranged from 0.94 to 0.97; TLIs for all groups ranged from 0.88 to 0.95. Most RMSEAs meet the recommended level of <0.08, and ranged from 0.06 to 0.09. Model parameters were similar across all diagnostic groups. For example, the cross-lagged effects of pain intensity at Time 1 predicting sleep disturbance at Time 2 showed positive weak relationships across the four groups (standardized coefficients ranged from 0.10 to 0.24). The standardized cross-lagged regression paths from sleep disturbance at Time 1 to pain intensity at Time 2 also showed positive weak relationships across the four groups, and ranged from <0.01 to 0.15, though only MD and MS were statistically significant. The cross-lagged effects for subsequent time points (i.e., Time 2 to Time 3 and Time 3 to Time 4) had similar but weaker relationships, with many coefficients not statistically significant.

Combined sample

The model which included the interaction terms supported fitting one combined model as only one of the six interaction terms was statistically significant ($p = 0.014$ for pain Time 3 onto sleep at Time 4). The model fit for the combined cross-lagged model was acceptable, $\chi^2_{(32)}$ (n = 1660) = 292.9, $p < 0.001$, CFI = 0.95,

Table 1
Demographic and clinical characteristics at time 1.

	Overall (n = 1660)		MD (n = 299)		MS (n = 542)		PPS (n = 392)		SCI (n = 427)	
	n	Mean (SD)	*n*	Mean (SD)	*n*	Mean (SD)	*n*	Mean (SD)	*n*	Mean (SD)
Age (years)	1660	55.88 (12.53)	299	52.92 (12.25)	542	54.21 (10.31)	392	66.39 (8.00)	427	50.44 (13.11)
Duration (years)	1593	15.33 (10.57)	293	14.38 (11.09)	542	14.97 (9.78)	337	15.59 (10.22)	421	16.23 (11.37)
Pain NRS (0–10)	1655	3.80 (2.77)	298	3.49 (2.75)	542	3.12 (2.73)	389	4.31 (2.71)	426	4.43 (2.67)
PROMIS Sleep disturbance	1656	52.38 (9.62)	297	51.79 (9.18)	542	53.01 (10.00)	391	51.99 (9.25)	426	52.35 (9.74)
PROMIS Fatigue	1660	56.35 (8.24)	299	56.17 (8.20)	542	58.01 (8.23)	392	58.66 (7.21)	427	52.23 (7.64)
Depression (PHQ-9)	1660	6.38 (5.08)	299	6.69 (5.48)	542	7.11 (5.30)	392	5.89 (4.40)	427	5.69 (4.96)
	n	%	*n*	%	*n*	%	*n*	%	*n*	%
Gender										
Male	598	36.02%	119	39.80%	97	17.90%	96	24.49%	286	66.98%
Female	1062	63.98%	180	60.20%	445	82.10%	296	75.51%	141	33.02%
Ethnicity										
White (non-Hispanic)	1519	91.51%	284	95.00%	507	93.54%	365	93.11%	363	85.01%
Non-white	140	8.43%	15	5.00%	34	6.27%	27	6.89%	64	14.99%
Missing	1	0.06%	–	–	1	0.18%	–	–	–	–
Marital Status										
Married/Living with Significant Other	1062	63.98%	223	74.58%	390	71.96%	231	58.93%	218	51.05%
Other	597	35.96%	76	25.42%	152	28.04%	160	40.82%	209	48.95%
Missing	1	0.06%	–	–	–	–	1	0.25%	–	–
Education										
Less than high school	25	1.50%	4	1.33%	5	0.92%	5	1.28%	11	2.58%
High school graduate/GED	195	11.75%	43	14.38%	51	9.41%	36	9.18%	65	15.22%
Vocational or technical school	107	6.45%	18	6.02%	35	6.46%	12	3.06%	42	9.84%
Some college	402	24.22%	63	21.07%	148	27.31%	91	23.21%	100	23.42%
College graduate	514	30.96%	86	28.76%	181	33.39%	114	29.08%	133	31.15%
Graduate or professional school	417	25.12%	85	28.43%	122	22.51%	134	34.18%	76	17.80%

Abbreviations: Muscular dystrophy (MD), multiple sclerosis (MS), post-polio syndrome (PPS), spinal cord injury (SCI), standard deviation (SD), numerical rating scale (NRS), patient health questionnaire (PHQ), General Educational Development (GED).

Table 2
Fit indices, standardized betas, and R^2 values for all five models fit to the overall sample and each diagnostic group.

N	Overall	MD	MS	PPS	SCI
	1660	299	542	392	427
Fit indices					
CFI	0.95	0.94	0.94	0.95	0.97
TLI	0.92	0.88	0.88	0.9	0.95
RMSEA	0.07	0.09	0.08	0.07	0.06
RMSEA 90% CI	0.06; 0.08	0.07; 0.11	0.07; 0.09	0.05; 0.08	0.05; 0.08
χ^2(DF) - test of model fit	**292.86**[32]	**102.91**[32]	**140.31**[32]	**84.94**[32]	**82.93**[32]
BIC	55416.27	10127.46	19532.55	12303.03	13780.77
Standardized Betas					
Depression to Pain T1	**0.26**	**0.19**	**0.37**	**0.21**	**0.23**
Depression to Sleep T1	**0.47**	**0.44**	**0.41**	**0.43**	**0.57**
Fatigue to Pain T1	**0.18**	**0.3**	**0.16**	**0.21**	**0.23**
Fatigue to Sleep T1	**0.13**	**0.21**	**0.13**	**0.1**	**0.13**
Age to Pain T1	0.01	−0.02	**0.1**	−0.18	0.01
Age to Sleep T1	**−0.08**	−0.01	−0.05	**−0.11**	**−0.11**
Sex to Pain T1	**−0.06**	−0.01	0.02	0.09	0.04
Sex to Sleep T1	0.03	**0.11**	**0.09**	0.02	−0.03
Pain T1 to T2	**0.69**	**0.64**	**0.66**	**0.66**	**0.74**
Pain T2 to T3	**0.33**	**0.39**	**0.37**	**0.18**	**0.36**
Pain T3 to T4	**0.52**	**0.57**	**0.49**	**0.48**	**0.55**
Pain T1 to T3	**0.45**	**0.41**	**0.42**	**0.59**	**0.36**
Pain T2 to T4	**0.28**	**0.21**	**0.28**	**0.3**	**0.3**
Sleep T1 to T2	**0.66**	**0.61**	**0.66**	**0.61**	**0.72**
Sleep T2 to T3	**0.41**	**0.41**	**0.39**	**0.44**	**0.44**
Sleep T3 to T4	**0.44**	**0.52**	**0.44**	**0.44**	**0.42**
Sleep T1 to T3	**0.33**	**0.32**	**0.27**	**0.36**	**0.36**
Sleep T2 to T4	**0.33**	**0.33**	**0.33**	**0.3**	**0.34**
Pain T1 to Sleep T2	**0.16**	**0.24**	**0.13**	**0.22**	**0.1**
Pain T2 to Sleep T3	**0.06**	**0.16**	**0.13**	−0.08	0.03
Pain T3 to Sleep T4	0.02	−0.06	−0.03	0.05	**0.09**
Sleep T1 to Pain T2	**0.09**	**0.15**	**0.13**	0	0.04
Sleep T2 to Pain T3	**0.04**	0.09	−0.01	0.02	**0.11**
Sleep T3 to Pain T4	**0.08**	0.06	0.1	0.07	**0.1**
Sleep T1 w/Pain T1	**0.11**	0.01	0.07	**0.1**	**0.16**
Sleep T2 w/Pain T2	**0.16**	0.14	**0.13**	**0.23**	**0.23**
Sleep T3 w/Pain T3	**0.14**	0.1	0.07	**0.26**	**0.18**
Sleep T4 w/Pain T4	**0.11**	0.1	0.08	0.04	**0.2**
R^2					
Pain T1	**0.15**	**0.2**	**0.24**	**0.19**	**0.17**
Pain T2	**0.52**	**0.49**	**0.51**	**0.43**	**0.58**
Pain T3	**0.55**	**0.6**	**0.53**	**0.53**	**0.53**
Pain T4	**0.59**	**0.59**	**0.56**	**0.52**	**0.68**
Sleep T1	**0.31**	**0.37**	**0.27**	**0.27**	**0.44**
Sleep T2	**0.52**	**0.51**	**0.5**	**0.5**	**0.59**
Sleep T3	**0.5**	**0.54**	**0.46**	**0.5**	**0.59**
Sleep T4	**0.51**	**0.58**	**0.46**	**0.48**	**0.56**
Covariates not significant – Sleep T1	Sex	Age	Age	Sex	Sex
Covariates not significant – Pain T1	Age	Age, Sex	Sex	Sex	Age, Sex

Note: Numbers in Bold represent significant values; $p < 0.05$.
Abbreviations: Comparative fit index (CFI), Tucker-Lewis index (TLI), root mean square error of approximation (RMSEA), Bayesian information criterion (BIC), degrees of freedom (DF), Time (T).

TLI = 0.92, and RMSEA = 0.07 (from 0.063 to 0.078) (see Fig. 1). As expected, pain intensity was predictive of itself at a subsequent time point (standardized coefficients ranged from 0.28 to 0.69). Likewise, sleep disturbance was also predictive of subsequent sleep disturbance scores (standardized coefficients ranged from 0.33 to 0.66). The percent of variance explained in pain intensity ranged from 15% to 59%, and from 31% to 52% for sleep disturbance (Table 2). Higher sleep disturbance at earlier time points was a significant predictor of higher pain intensity at the following time point (standardized coefficients ranged from 0.04 to 0.09), and higher pain intensity in early time points predicted higher sleep disturbance at the following time point (except in the path from pain intensity at Time 3 to sleep disturbance at Time 4; standardized coefficients ranged from 0.02 to 0.16). The cross-lagged effects between pain intensity and sleep disturbance were weak and positive, and generally decreased across the four time points. Most effects from pain intensity on sleep disturbance were of greater magnitude than those of sleep on pain intensity. The correlations between pain intensity and sleep disturbance within each time point were statistically significant and showed positive weak relationships (higher pain intensity related to higher sleep disturbance) across the four time points (standardized coefficients ranged from 0.11 to 0.16). The covariates, depression and fatigue, were statistically significant predictors of both sleep and pain at Time 1. Increases in both depression and pain were related to increases in pain intensity and sleep disturbance. Age was a significant predictor of sleep disturbance only at time 1, with older individuals reporting less sleep disturbance. Sex was a significant predictor of pain intensity, with men reporting lower pain intensity.

Discussion

This study evaluated the direction and magnitude of the association between sleep disturbance and pain intensity in a large

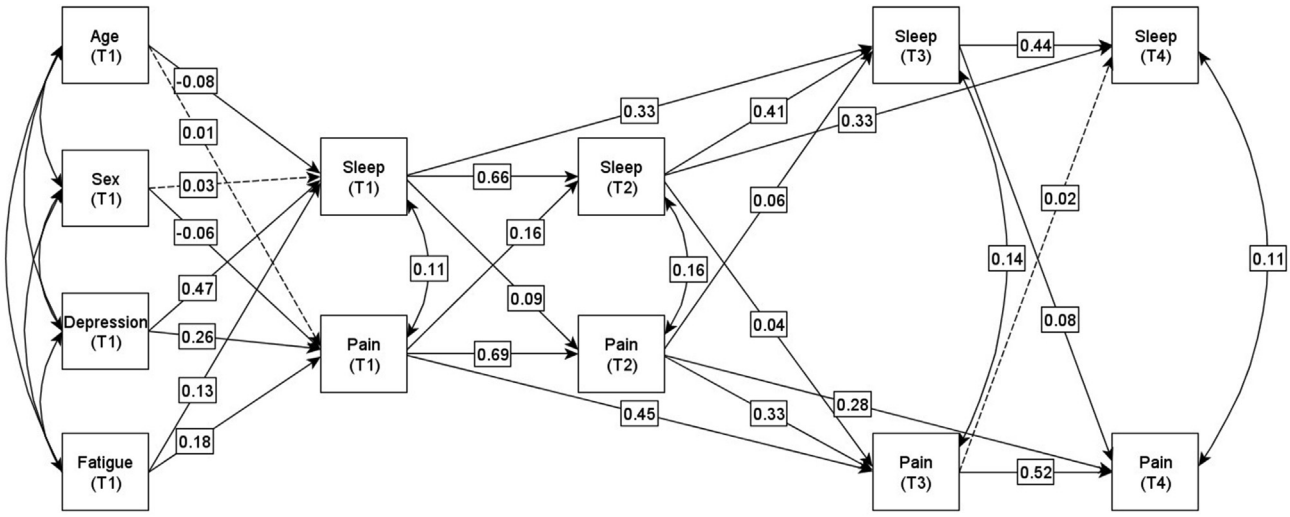

Fig. 1. Cross-lagged effect model between sleep disturbance and pain interference. Path coefficients are standardized values and solid lines represent significant paths, $p < 0.05$. Dashed lines represent non-significant paths.

sample of individuals with long-term physical disability. The results supported our hypothesis that pain and sleep are positively associated in individuals with chronic physical disability when modeled in the four diagnostic groups separately, as well as together. With the exception of one cross-lagged path (pain at time 3 to sleep at time 4), all paths between pain and sleep were positive and statistically significant within the overall sample, however the effects were relatively small as would be expected between measurement one year apart. The results did not support the second hypothesis that the effect of sleep on pain would be larger than pain on sleep. In the overall sample, the cross-lagged coefficients from time 1 to time 2 and from time 2 to time 3 are larger from pain to sleep, with only the time 3 to time 4 path larger from sleep to pain, providing evidence for the effect of pain on sleep to be somewhat stronger than the reverse.

This result was also true in three of the four diagnostic groups. For individuals with MD, MS, and PPS the significant cross-lagged path coefficients from pain to sleep are larger than those from sleep to pain, without exception. However, for individuals with SCI, the direction of the relationship between sleep and pain is less clear, and is not as consistent across timepoints.

The results of this study are only somewhat consistent with the results of two review articles[10,39] that examined the direction of the relationship between pain and sleep. The most recent review,[10] which included longitudinal and experimental studies, concluded that sleep impairments were a more reliable and stronger predictor of subsequent pain than the reverse across most groups. However, of the 17 longitudinal or micro longitudinal studies included in the review, none included any of the four populations included in this study, and nearly all of the studies were completed in either the general population or in chronic pain samples (exceptions being burn injury and depression samples). In addition, the review authors also concluded that the association between pain and sleep may vary for different conditions associated with chronic pain.[10]

Studies published since the most recent review[40–43] have also generally supported the directional relationship of sleep to pain, although again, none of the studies were conducted in persons with chronic physical disabilities. Thus, the findings of the current study suggest that the relationship between pain and sleep may be different in individuals with chronic disabilities than in the general population or other chronic pain samples. In short, and in individuals with chronic physical disabilities, pain may play a greater role in sleep disturbance than in individuals who report chronic pain as a primary problem.

The findings from this study can be used to inform and direct clinical practice for addressing pain and sleep problems of people with chronic physical disabilities, as well as health promotion and rehabilitation program content. Health promotion interventions and rehabilitation programs for people who experience both pain and sleep disturbance should include promoting sleep hygiene, relaxation methods, and environment management[44] because addressing sleep issues may have a beneficial impact on pain for individuals with MS, MD, PPS, and SCI. Data from these programs can also be used to study how changes in sleep quality and duration impact pain and health-related quality of life among individuals aging with disability. Future studies could expand on the current study by including other groups that experience disability-related pain, such as individuals with acquired amputations, ALS, Parkinson's, and traumatic brain injury, and by analyzing data from intervention studies aimed at improving pain and sleep.

This study has a number of limitations that should be considered when interpreting the results. First, the study participants had lived with their disability for an average of 15 years at the initial assessment point; the results may not necessarily hold for people newly diagnosed with a chronic condition and who may have difference experiences with sleep and pain. Second, this study only recruited participants from four disability groups (MS, MD, PPS, and SCI); the findings may or may not generalize to other groups of individuals with congenital or acquired disabilities and conditions. Third, the study sample primarily identified as non-Hispanic white. Because evidence suggests there are differences in pain beliefs and cognitions by race and ethnicity, the study findings may not be generalizable to samples consisting of other racial and ethnic backgrounds. Finally, the longitudinal survey from which data for the current analyses were drawn was designed to study the natural course of aging with disability with measurements spaced about a year apart. It did not include any interventions or control for treatment sought by participants for their pain and/or sleep problems. Data from a treatment study that aimed to manage/reduce pain or sleep problems and with more frequent measurements would provide more information about the causal relationships between pain and sleep and would be more likely to find greater effects. Conclusions regarding the impact of treatments which improve sleep quality on pain, and the impact of treatments which

reduce pain on sleep quality, will require clinical trials evaluating these potential effects. The current study indicates that such trials are warranted.

Summary and conclusions

Despite the study's limitations, the findings provide new information regarding the temporal precedence of sleep disturbance as a predictor of subsequent pain, and of pain as a predictor of subsequent sleep disturbance in a large sample of individuals with diagnoses associated with physical disability. The results indicate that in the samples studied, more pain intensity was related to more subsequent sleep disturbance and vice versa in the samples studied. Although the former relationship was stronger than the latter, the correlations tended to be rather weak, and generally consistent across the four time points for three of the conditions studied; the relationships between sleep disturbance and pain were more complicated and less consistent across the time points in participants with SCI. Future research is needed to better understand this relationship in people with SCI, and other chronic conditions, and to study these relationships in the context of pain and/or sleep disturbance treatments.

Funding

The contents of this publication were developed under grants from National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR grant numbers H133B080024 & 90RT5023-01-00). NIDILRR is a Center within the Administration for Community Living (ACL), Department of Health and Human Services (HHS). The contents of this publication do not necessarily represent the policy of NIDILRR, ACL, HHS, and you should not assume endorsement by the Federal Government. NIDILRR did not participate in decisions related to study design, the collection, analysis and interpretation of data, article preparation, or in the decision to submit the article for publication.

Declaration of competing interest

The authors have no other conflicts of interest to report.

Acknowledgments

The authors would like to acknowledge Jiseon Kim, PhD and Hyewon Chung, PhD for their assistance with some of the data analyses.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dhjo.2020.100908.

References

1. Karaman S, Karaman T, Dogru S, et al. Prevalence of sleep disturbance in chronic pain. *Eur Rev Med Pharmacol Sci*. 2014;18(17):2475–2481.
2. Foley PL, Vesterinen HM, Laird BJ, et al. Prevalence and natural history of pain in adults with multiple sclerosis: systematic review and meta-analysis. *Pain*. 2013;154(5):632–642.
3.. van Gorp S, Kessels AG, Joosten EA, van Kleef M, Patijn J. Pain prevalence and its determinants after spinal cord injury: a systematic review. *Eur J Pain*. 2015;19(1):5–14.
4. Trojan DA, Cashman NR. Post-poliomyelitis syndrome. *Muscle Nerve*. 2005;31(1):6–19.
5. Jensen MP, Abresch RT, Carter GT, McDonald CM. Chronic pain in persons with neuromuscular disease. *Arch Phys Med Rehabil*. 2005;86(6):1155–1163.
6. Bamer AM, Johnson KL, Amtmann D, Kraft GH. Prevalence of sleep problems in individuals with multiple sclerosis. *Mult Scler*. 2008;14(8):1127–1130.
7. Giannoccaro MP, Moghadam KK, Pizza F, et al. Sleep disorders in patients with spinal cord injury. *Sleep Med Rev*. 2013;17(6):399–409.
8. Jubelt B. Post-polio syndrome. *Curr Treat Options Neurol*. 2004;6(2):87–93.
9. Watson NF, Viola-Saltzman M. Sleep and comorbid neurologic disorders. *Continuum*. 2013;19:148–169, 1 Sleep Disorders.
10. Finan PH, Goodin BR, Smith MT. The association of sleep and pain: an update and a path forward. *J Pain*. 2013;14(12):1539–1552.
11. Jansson-Frojmark M, Boersma K. Bidirectionality between pain and insomnia symptoms: a prospective study. *Br J Health Psychol*. 2012;17(2):420–431.
12. O'Brien EM, Waxenberg LB, Atchison JW, et al. Intraindividual variability in daily sleep and pain ratings among chronic pain patients: bidirectional association and the role of negative mood. *Clin J Pain*. 2011;27(5):425–433.
13. Smith MT, Klick B, Kozachik S, et al. Sleep onset insomnia symptoms during hospitalization for major burn injury predict chronic pain. *Pain*. 2008;138(3):497–506.
14. Naranjo C, Del Reguero L, Moratalla G, Hercberg M, Valenzuela M, Failde I. Anxiety, depression and sleep disorders in patients with diabetic neuropathic pain: a systematic review. *Expert Rev Neurother*. 2019:1–9.
15. Stubbs B, Koyanagi A, Thompson T, et al. The epidemiology of back pain and its relationship with depression, psychosis, anxiety, sleep disturbances, and stress sensitivity: data from 43 low- and middle-income countries. *Gen Hosp Psychiatr*. 2016;43:63–70.
16. Wallen GR, Minniti CP, Krumlauf M, et al. Sleep disturbance, depression and pain in adults with sickle cell disease. *BMC Psychiatr*. 2014;14:207.
17. Ravyts SG, Dzierzewski JM, Raldiris T, Perez E. Sleep and pain interference in individuals with chronic pain in mid- to late-life: the influence of negative and positive affect. *J Sleep Res*. 2019;28(4), e12807.
18. O'Brien EM, Waxenberg LB, Atchison JW, et al. Negative mood mediates the effect of poor sleep on pain among chronic pain patients. *Clin J Pain*. 2010;26(4):310–319.
19. Eller-Smith OC, Nicol AL, Christianson JA. Potential mechanisms underlying centralized pain and emerging therapeutic interventions. *Front Cell Neurosci*. 2018;12:35.
20.. Haack M, Simpson N, Sethna N, Kaur S, Mullington J. Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications. *Neuropsychopharmacology*. 2020;45(1):205–216.
21. Wu YE, Li YD, Luo YJ, et al. Gelsemine alleviates both neuropathic pain and sleep disturbance in partial sciatic nerve ligation mice. *Acta Pharmacol Sin*. 2015;36(11):1308–1317.
22. Finkel SE. *Causal Analysis with Panel Data*. London: Sage; 1995.
23. Molton IR, Hirsh AT, Smith AE, Jensen MP. Age and the role of restricted activities in adjustment to disability-related pain. *J Health Psychol*. 2014;19(8):1025–1034.
24.. Smith AE, Molton IR, Jensen MP. Self-reported incidence and age of onset of chronic comorbid medical conditions in adults aging with long-term physical disability. *Disabil Health J*. 2016;9(3):533–538.
25. Yu L, Buysse DJ, Germain A, et al. Development of short forms from the PROMIS sleep disturbance and Sleep-Related Impairment item banks. *Behav Sleep Med*. 2011;10(1):6–24.
26. Cella D, Riley W, Stone A, et al. The Patient-Reported Outcomes Measurement Information System (PROMIS) developed and tested its first wave of adult self-reported health outcome item banks: 2005-2008. *J Clin Epidemiol*. 2010;63(11):1179–1194.
27. Jensen MP. The validity and reliability of pain measures in adults with cancer. *J Pain*. 2003;4(1):2–21.
28. Hjermstad MJ, Fayers PM, Haugen DF, et al. Studies comparing numerical rating scales, verbal rating scales, and visual analogue scales for assessment of pain intensity in adults: a systematic literature review. *J Pain Symptom Manag*. 2011;41(6):1073–1093.
29. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. *J Gen Intern Med*. 2001;16(9):606–613.
30. Lai JS, Cella D, Choi S, et al. How item banks and their application can influence measurement practice in rehabilitation medicine: a PROMIS fatigue item bank example. *Arch Phys Med Rehabil*. 2011;92(10 Suppl):S20–S27.
31. Vitkova M, Gdovinova Z, Rosenberger J, et al. Factors associated with poor sleep quality in patients with multiple sclerosis differ by disease duration. *Disabil Health J*. 2014;7(4):466–471.
32. Marck CH, De Livera AM, Weiland TJ, et al. Pain in people with multiple sclerosis: associations with modifiable lifestyle factors, fatigue, depression, anxiety, and mental health quality of life. *Front Neurol*. 2017;8:461.
33. Loh KP, Zittel J, Kadambi S, et al. Elucidating the associations between sleep disturbance and depression, fatigue, and pain in older adults with cancer. *J Geriatr Oncol*. 2018;9(5):464–468.
34. Muthén LK, Muthén B. *Mplus User's Guide*. sixth ed. Los Angeles, CA: Muthén & Muthén; 1998-2011.
35.. Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct Equ Modeling*. 1999;6(1):1–55. https://doi.org/10.1080/10705519909540118.
36. Tucker LR, Lewis C. A reliability coefficient for maximum likelihood factor analysis. *Psychometrika*. 1973;38:1–10.
37. Browne MW, Cudeck R. Alternative ways of assessing model fit. In: Bollen KA, Long JS, eds. *Testing Structural Equation Models*. Beverly Hills, CA: Sage; 1993:136–162.
38. Acock AC. *A Gentle Introduction to Stata*. fourth ed. College Station, Texas: Stata Press; 2014.

39. Smith MT, Haythornthwaite JA. How do sleep disturbance and chronic pain inter-relate? Insights from the longitudinal and cognitive-behavioral clinical trials literature. *Sleep Med Rev*. 2004;8(2):119–132.
40. Harrison L, Wilson S, Heron J, Stannard C, Munafo MR. Exploring the associations shared by mood, pain-related attention and pain outcomes related to sleep disturbance in a chronic pain sample. *Psychol Health*. 2016;31(5):565–577.
41. Sanders AE, Akinkugbe AA, Bair E, et al. Subjective sleep quality deteriorates before development of painful temporomandibular disorder. *J Pain*. 2016;17(6):669–677.
42. Koffel E, Kroenke K, Bair MJ, Leverty D, Polusny MA, Krebs EE. The bidirectional relationship between sleep complaints and pain: analysis of data from a randomized trial. *Health Psychol*. 2016;35(1):41–49.
43. Koffel E, Krebs EE, Arbisi PA, Erbes CR, Polusny MA. The unhappy triad: pain, sleep complaints, and internalizing symptoms. *Clin Psychol Sci*. 2016;4(1):96–106.
44. Floyd JA, Falahee ML, Fhobir RH. Creation and analysis of a computerized database of interventions to facilitate adult sleep. *Nurs Res*. 2000;49(4):236–241.