Sleep Characteristics in Adults with and without Chronic Musculoskeletal Pain: The Role of Mental Distress and Pain Catastrophizing

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Abstract

Objectives: Sleep disturbance is associated with persistence and exacerbation of chronic pain. As this relationship seems to be bidirectional, factors underpinning sleep disturbance may prove valuable in multimodal rehabilitation approaches. The aim of this cross-sectional study was to examine the impact of psychological symptoms on subjective and objective sleep measures in patients with chronic musculoskeletal pain (CMP) as compared to healthy controls (HC).

Methods: Sleep was assessed by self-report questionnaires, actigraphy and polysomnography recordings in 56 patients (75.0% female; M_{age} = 41.7 years, SD = 10.8 years) with CMP and compared to 53 matched HC (71.7% female; M_{age} = 41.8 years, SD = 10.7). Mental distress (HSCL) and pain catastrophizing (PCS) were tested as predictors of objective and subjective sleep measures in multiple regression models, and their indirect effects were tested in bootstrapped mediation models.

Results: The sleep data revealed substantially more subjective sleep disturbance (Hedge's g: 1.32-1.47, $p < .001$), moderately worse sleep efficiency in the actigraphy measures (Hedges g: 0.5-0.6, $p < .01$) and less polysomnography measured slow wave sleep (SWS) (Hedges g: 0.43, $p < .05$) in patients as compared to controls. HSCL was strongly associated with the self-reported measures Insomnia Severity Index (ISI) and Pittsburgh Sleep Quality Index (PSQI). HSCL also partially explained the pain (CMP / HC) to sleep association, but HSCL was not associated with any of the objective sleep measures. More pain catastrophizing was related to less SWS.

Discussion: The differences in subjective and objective sleep measures indicate that they probe different aspects of sleep functioning in patients with musculoskeletal pain, and their combined application may be valuable in clinical practice. Self-reported sleep disturbance seems to overlap with affective dimensions reflected by the HSCL- questionnaire.

Keywords: Chronic pain, polysomnography, sleep quality, sleep architecture, mental distress, pain catastrophizing, mediation.

1 Introduction

Insomnia afflicts about 2 of 3 patients with chronic pain, and this association seems to be bidirectional.[1,2] Those suffering from chronic musculoskeletal pain tend to have sleep characteristics comparable to insomnia, with both self-reported and objectively measured impairments.[3-5] Comorbid sleep disturbance most likely contribute to the persistence and exacerbation of chronic pain,[6] whereas non-disturbed sleep may alleviate pain.[7] According to a meta-analysis by Tang, the expected palliation of pain following sleep interventions seems to be modest,[8] still it may be of clinical interest to gain knowledge about factors contributing to sleep impairment in chronic pain conditions.

Self-reported and objective sleep measures seem to be differentially related to pain.[9-12] Self-report measures generally yield larger group differences than actigraphy and PSG measures in case-controlled chronic pain studies, and may be more strongly associated with reported levels of pain in experimental studies.[9,13] Polysomnography (PSG) is considered the gold standard of objective sleep recording, enabling assessment of both sleep continuity and sleep architecture, as well as detection of abnormal sleep related respiration and movement. However, the equipment may be perceived as intrusive, and is not suitable for long term monitoring. An actigraph, a wristwatch-like device measuring movement, is more convenient for longer recordings, and is regarded as valid proxy measure of sleep continuity in pain patients.[14]

Meta-analyses of controlled PSG studies in pain conditions suggest impaired sleep continuity with reduced sleep duration (total sleep time, TST), increased wake time during the night (wake after sleep onset, WASO) and reduced sleep time/ time-in-bed ratio (sleep efficiency, SE), whereas increased interval from bed-time to sleep onset (sleep onset latency, SOL) does not seem to be as consistently reported.[13,15] Among controlled actigraphy studies, several do not observe group differences in sleep continuity measures.[9,16,17] Others report

group differences, most consistently for WASO and SE.[18-20] In addition to differentiating groups, SE and WASO have been associated with reported pain,[18,21] and SE was related to pain inhibition in an experimental study among pain patients.[22]

Changes in sleep architecture, including amounts of slow wave sleep (SWS), are not uniform in studies of pain patients.[3,13,15] Yet, reduced SWS was observed among men who reported higher pain intensity in a large population study, and may thus be of particular interest.[23] In healthy persons, reduced SWS typically impairs subjective sleep quality, and increases the propensity to sleep at daytime.[24] However, how psychological factors like mental distress and pain catastrophizing associate with the possible loss of SWS in pain patients is less well studied.

Negative affect and mental distress is generally common in chronic pain,[25] and several pain comorbidity studies examining self-reported sleep measures in chronic pain conditions, have pointed to depressive symptoms, pre-sleep worry and arousal as consistent risk factors of impaired sleep quality and insomnia.[1,11,12,26-30] Cognitive responses to pain, as assessed by the Pain Catastrophizing Scale (PCS), may additionally contribute to arousal and reduced sleep quality.[31] On the other hand, the associations between psychological symptoms and PSG or actigraphy indices of sleep continuity are less uniformly described, as several studies report significant associations, whereas others do not.[12,18,19]

Mediation studies, recently reviewed by Whibley, suggest that affective and cognitive factors are involved as mediators in the reciprocal sleep-pain relationship.[32] However, these studies lack objective sleep measures.[32] In order to clarify the role of psychological symptoms for the pain-sleep association, it would accordingly be useful to study sleep both with subjective and objective measures. Such data would also be useful in statistical/mediation model building, aiming to clarify if a common underlying pathophysiology could explain these relationships.[33] It would be of particular interest to study how both objective and

subjective sleep measures can be explained by psychological variables among healthy subjects and patients with chronic pain.

The aim of this study was to estimate the influence of potentially modifiable psychological factors on sleep quality and insomnia-symptoms as well as PSG and actigraphy measured sleep disturbance, in patients with chronic musculoskeletal pain, as compared to healthy controls. The specific objectives were 1) to estimate polysomnographic and actigraphic sleep in pain patients compared to healthy controls using a blinded design, 2) to examine how mental distress and pain catastrophizing are associated with self-reported, actigraphy and PSG measures of sleep in pain patients and healthy controls and 3) to examine to what extent an association between pain (group affiliation) and sleep outcomes was explained by mental distress or pain catastrophizing as mediating variables.

2 Materials and Method

2.1 Participants and Procedure

Patients attending the outpatient clinics at the Rehabilitation Department and the Pain Clinic, both at the University Hospital of North Norway (UNN), were invited by mail. Criteria for inclusion were visits at the respective clinics during the last 18 months, age 18-65 years and having chronic musculoskeletal pain, defined by selected ICD-10 codes (Table 1). Patients were excluded if they had a major medical condition (cancer, inflammatory, symptomatic heart or lung, metabolic or endocrine disease), neurologic condition, psychiatric illness, drug abuse, were pregnant or participated in ongoing intervention studies. Patients previously diagnosed with sleep disorder, other than insomnia, were excluded. A group of pain free controls, matched one to one by age (+/- 5 years), sex and season of investigation, were recruited by poster advertisements among hospital and university employees and blood

donors. The same exclusion criteria were assessed based on medical records and interview for both patients and controls.

The study is a cross-sectional case-controlled observation study. Due to the purpose of another study, investigating seasonal variations in symptoms (paper submitted, in review), participants were enrolled either during summer or winter season. The study entailed one week of continuous actigraphy with accompanying sleep diary, and one night (first) of unattended polysomnography (PSG). The first appointment took place at the Department of Clinical Neurophysiology, UNN, where participants received detailed written and oral information, completed questionnaires, and had the devices for actigraphy and unattended home PSG attached. They returned the next morning to have the PSG unit disconnected, and finally returned after 7 days with the actigraphy device and completed sleep diary. Participants were instructed to conduct their daily life as usual during the study period. There were no restrictions to sleep schedule, habitual medication or daily activities, but periods of night shifts were avoided for participants with shift work.

2.2 Self-report Measures

Age, gender, educational level (high school vs higher education), marital status (single vs married/cohabiting), employment (no, yes), receiving social benefit (no, yes) and self-rated economic situation (poor/medium vs good) were registered. Medication was registered based on participants own reports at first visit. Regular medications were categorized into opioids, hypnotics, antidepressants, antiepileptic (taken as analgesic), NSAID/ paracetamol and "other". The category "other" included sporadic use of the aforementioned drugs as well as other medications (antihistamine, antiasthmatic, thyroxine etc.).

Pain severity items of a validated Norwegian version of the Brief Pain Inventory (BPI) short form were applied.[34,35] Participants estimated their worst, least and average pain during

the last week, as well as their current pain. Each of the four items were rated on an 11-point numeric rating scale (from 0, no pain, to 10, worst imaginable pain). We used the mean severity score of these four items in the analyses of pain severity.

The 25 item version of the Hopkins Symptoms Checklist (HSCL 25) is a self-report inventory assessing symptoms of depression and anxiety indicative of mental distress.[36] Items are rated from 1-not at all to 4-very much, from which a global average score is calculated (range: 1-4).

The validated Norwegian version of the Pain Catastrophizing Scale (PCS) was applied.[37,38] Thirteen items covering rumination, magnification and helplessness, are rated on a five-point Likert scale (0=not at all, 1=to a slight degree, 2=to a moderate degree, 3=to a great degree, 4=all the time). The sum score (range 0-52) was used for analyses.

The Insomnia Severity Index (ISI) encompasses seven items rated on a five-point Likert scale (0–4, total range 0-28).[39] An ISI cut-off >14 indicates clinical insomnia,[40] and the ISI is a recommended research measure of insomnia symptoms.[41]

The Pittsburgh Sleep Quality Index (PSQI) comprises 19 items probing sleep quality and disturbance during the previous month, across seven components: 1) Subjective sleep quality, 2) sleep latency 3) sleep duration 4) habitual sleep efficiency 5) sleep disturbance, 6) sleep medication and 7) daytime dysfunction. Each component is scored 0 (no difficulty) to 3 (severe difficulty), yielding a global score with a range of 0-21. A cut off score > 5 is recommended to distinguish good from poor sleepers.[42] The PSQI is a recommended research measure of global sleep symptoms.[41] The Norwegian translation has shown acceptable reliability and validity.[43]

2.3 Sleep Recording

The Actiwatch Spectrum Plus device (Phillips Respironics, Inc., Murrysville, PA) was applied as a proxy-measure of sleep. Good agreement with PSG for TST, SE and WASO has been reported in low back pain, whereas validity for SOL was poor.[14] Stability of sleep variables over time (year) seem reasonable for TST, SE and SOL, but larger night-to-night variability (particularly SOL) requires 5-6 days of recording for reliable measures.[44] The Actiwatch was applied on the non-dominant wrist, and was only to be removed shortly during shower or if required at work (e.g. due to hygiene or safety considerations). The participants registered their first sleep attempt and final morning awakening by pushing the event button as well as completing a sleep diary, reporting the hour of going to bed, attempting to sleep, waking up and getting out of bed as well as estimating sleep latency, and nightly awakenings. Actiware version 6.0.9 software was used for post-processing (30 second epochs, medium sensitivity for activity detection and an immobility threshold of 10 minutes for sleep onset). If necessary, information based on the event marker, sleep diary and light intensity information were consulted, in line with a published guideline.[45] Rest periods were scored by a trained research assistant (psychology student) supervised by a specialist in clinical neurophysiology (first author). Both were blinded to participant identity and group affiliation. The variables associated with insomnia; total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO) and sleep efficiency (SE) were averaged across the recorded 7 days.

SOMNOscreen equipment and Domino version 2.7.0 software (Somnomedics, Randersacker, Germany) were applied for PSG, and the recording and scoring were performed in accordance with The American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events, version 2.4.[46] The scoring was performed by the first author who was blinded to participant identity and group affiliation. Six EEG leads (F3/F4, C3/C4, O1/O2), right and left EOG and submental EMG were used for sleep scoring.

Pressure flow nasal cannula, inductive thoracic and abdominal belts (effort) and oximetry were used for respiratory assessment. The AASM hypopnea scoring rule 1A was applied (≥ 10 seconds duration of ≥ 30% of air flow reduction associated with a ≥ 3% decrease in oxygen saturation and/ or an EEG arousal). Bilateral pretibial EMG recordings were used for assessment of periodic limb movements (PLM). The participants used a marker button to indicate their first attempt to sleep. The variables TST, SOL, WASO, SE, distribution of sleep stages as proportion of TST (N1, N2, N3 (also termed slow wave sleep, SWS, in this paper) and REM), indexes of sleep stage shifts, wake bouts, arousals, limb movements (LM) in periodic LM sequences (PLM) and apneas/ hypopneas (AH) were obtained for the recording night. All indexes are denoted as number of events per hour of sleep (apnea hypopnea index, AHI, and periodic limb movement index, PLMI).

2.4 Statistical Analyses

The IBM SPSS 25 was used for general analyses, including the SPSS plugin PROCESS version 3 macro by Hayes for mediation analyses.[47]

Due to highly skewed distributions, bootstrapped 95% confidence intervals based on 5000 re-samplings were constructed for all analyses to derive empirically unbiased estimates of the sampling error.

Crude group differences were assessed by independent Student's t-tests and chi-square tests for continuous and dichotomous variables, respectively. In case of unequal variances (i.e., significant Levene's test), the degrees of freedom were appropriately adjusted. Between-group effect sizes are reported as Hedge's *g*.

Bivariate correlations, with estimation of Pearson correlation coefficients were assessed between the most important variables, separately in the CMP and HC groups.

Four multiple linear regression models with ISI, PSQI, SE (actigraphy) and SWS as dependent variables were assessed in each of the CMP and HC sub-groups. The 7-day actigraphy derived SE was preferred to the corresponding 1-day PSG measure as it was presumed superior as a measure of habitual sleep. The independent variables of interest, HSCL and PCS were entered in the regression model together with the covariates gender, age, education and AHI. The models were bootstrapped to provide unbiased confidence intervals. Standardized residuals were inspected for normality and heteroscedasticity.

The regression analyses were replicated (without AHI as a covariate) after having participants with AHI>15 removed from the sample, as a sensitivity test examining the robustness of the results.

Four mediation models were specified to investigate whether group differences (CMP vs HC) in the sleep measures ISI, PSQI, SE (actigraphy) and SWS could be explained by indirect effects through mental distress and pain catastrophizing. In situations considering multiple mediators, fitting a multiple mediation model may be preferable to several simple mediation models, as a) including multiple mediators enable estimation of the effect of a mediator conditional on the presence of other mediators, b) including multiple mediators in a single model allows the simultaneous estimation of their specific magnitudes, c) sequential simple mediation testing may suffer from an omission variable bias.[48] Including correlated mediators are acceptable, however with possible drawbacks related to reduced statistical power due to increased parameter variance that yield wider confidence intervals.[47,48] Mental distress and pain catastrophizing were thus entered simultaneously as mediators, but either were removed if non-significant (p>0.05). Standard errors and confidence intervals were bootstrapped.

The mediation Model 4 (applied for simple and multiple mediation) of the PROCESS macro partitions the total effect (Y=icept+cX) in two underlying components: the indirect or

mediating effect (*M*=icept+*a*X) and the adjusted direct effect (Y'=icept+*c'*X+*bM*). Here, the indirect effect runs from group (X) through mental distress / pain catastrophizing (M: path *a*) to sleep as outcome (Y: path *b*), and is thus estimated as the product of *a* and *b*. If the indirect path (*a*b*) explains all variability in the outcome measure, the adjusted direct effect (*c'*) will turn non-significant. The size of the mediation effect is represented by the ratio between the indirect (a*b) to the direct or total effect (*c*).

2.5 Ethical Approval

The study was approved by the Regional Committee for Medical and Health Research Ethics, Office North (reference number 2015/2473). Written informed consent was obtained from all participants.

3 Results:

3.1 Sample Characteristics

A total of 401 patients were invited to participate, of whom 91 responded. Based on criteria, 28 patients were excluded and 7 patients either moved or withdrew. The final sample consisted of 56 patients and 53 controls. The distribution of ICD-10 diagnoses is given in Table 1. Three participants in the pain group were not available for PSG recording and the PSGs of two control participants were technically unsuccessful. There were artifacts in respiratory leads in four participants (one control, three pain patients) as well as in the leg-movement leads in six participants (four controls, two pain patients), and they were excluded from analyses using these variables.

3.2 Group Differences

The numbers being unemployed, receiving social benefits and having poor economy were larger for patients compared to controls. Of the six patients using opioid medication, five

used tramadol (up to 200 mg daily dose) and one used a combination of paracetamol and codeine. Three patients used hypnotics (benzodiazepine-like drug), and five patients used the antidepressant amitriptyline in dosages intended for analgesia and sleep. One patient used escitalopram. Three patients used antiepileptic drugs for pain (Table 2). Due to small number of participants using diverse drugs with varying potential for affecting sleep, medication was not found useful as a covariate in further analyses.

As expected, patients reported significantly more pain symptoms (BPI), pain catastrophizing (PCS) and mental distress (HSCL) than control cases (Table 3). Patients also reported significantly more insomnia (ISI) and reduced sleep quality (PSQI) compared to controls (Table 4), and with strong effect sizes. Based on the ISI cut off score (> 14), the prevalence of insomnia was 30.4% and 3.8% ($p < .001$) in the patient and control groups, respectively. A corresponding analysis of the PSQI (cut off > 5) indicated that 76.8% and 26.4% of the patients and controls, respectively, had impaired sleep quality ($p < .001$).

The actigraphy data showed significant group differences of medium effect sizes including increased SOL and WASO, and reduced SE among CMP compared to HC. No significant group difference was observed for TST (Table 4). PSG showed no significant group differences in the sleep continuity variables, but the sleep stage distribution in patients showed less SWS (N3) and more light sleep (N2) compared to controls. There were no significant group differences in arousal index, sleep stage shift index, AHI or PLMI. (Table 4). The number of participants displaying AHI above 15 was 14 (28.6%) and 9 (18%) among CMP and HC respectively, and for AHI above 30 the corresponding numbers were 4 (8.2%) and 1 (2%).

3.3 Correlation and Regression Analyses

The bivariate correlations are available in Supplement Table 1. The regression analyses are presented separately for CMP and HC in Table 5. Mental distress, but not pain catastrophizing, was a significant predictor for both self-reported sleep outcomes, ISI and PSQI, among CMP and HC alike. Increased mental distress was associated with increased symptoms of insomnia and reduced sleep quality. Neither HSCL nor PCS significantly predicted SE (actigraphy), whereas PCS was significantly associated with SWS among patients with chronic pain solely.

A sensitivity test was performed by repeating the regression analyses while excluding participants with AHI> 15. Although PCS was no longer significantly associated with SWS (may be due to lack of power in a smaller sample, as regression coefficient is similar), this procedure did not substantially change the results in the other models, indicating robustness of the observed associations (Supplement Table 2).

3.4 Analyses of Indirect Effects

Mental distress (HSCL) partially and significantly explained variability in the relationship between the grouping variable and ISI and PSQI, respectively (Figure 1 and 2), whereas pain catastrophizing (PCS) was nonsignificant, and thus removed from the model. For insomnia (ISI) and global sleep quality (PSQI) as outcomes, the indirect pathways explained two thirds and half of the total effect, respectively. These findings suggest that the differences between the patient and the control sample with regard to self-reported insomnia and sleep quality is associated with levels of mental distress. Mental distress and pain catastrophizing did not significantly explain any other effects.

4 Discussion

The present cross-sectional case-control study reports substantial group differences between patients with chronic musculoskeletal pain (CMP) and healthy controls (HC) in self-reported sleep disturbance. However, the group differences in sleep continuity (actigraphy) and sleep architecture (PSG), were minor. Moreover, mental distress was strongly associated with insomnia and reduced subjective sleep quality in both groups, as well as explained a proportion of the group (case-control) difference in these sleep measures. Mental distress was not related to the sleep efficiency (SE) or slow wave sleep (SWS), whereas pain catastrophizing was associated with less SWS in CMP solely.

Our observations of large group differences in sleep quality and insomnia and less group differences in actigraphy or PSG measured sleep continuity align with previous studies of chronic pain conditions.[13,19] We also found a smaller group difference in sleep architecture with less SWS in the pain group. As reviewed by Bjurstrom,[3] controlled clinical studies including PSG are generally of small sample size, and there are somewhat diverging PSG findings regarding sleep architecture in patients with fibromyalgia and chronic widespread pain compared to healthy controls. Findings also diverged in two recent meta-analyses, one in patients with fibromyalgia and one in chronic pain associated with various conditions (however not including cancer and spinal cord injury), where only the former reported reduced SWS.[13,15] Thus, the role of SWS in pain does not seem to be firmly established, and may be dependent on the pain condition. The group differences in objective sleep parameters are generally smaller, and one may question whether a group difference in SE of less than three percent and in SWS of approximately 15 minutes, as in this study, are of significant clinical importance.

In the current study, mental distress statistically explained a substantial proportion of the variation in reported insomnia and sleep quality, as well as partially explained the CMP-

HC difference in these self-report measures. The strong association between affective measures and self-report sleep measures in pain patients is well documented,[1,11,26,28,29] and two previous studies of sickle cell disease and rheumatoid arthritis have reported negative mood/depression as a mediator of the relationship between pain intensity and sleep quality.[26,28] We did not observe any associations between mental distress and actigraphy-recorded SE, which also converges with previous studies.[12,18,19] As an extension of previous studies, we included actigraphy and PSG sleep measures in the mediation analyses, which disconfirmed any role of mental distress as a mediator of SE or SWS. These findings indicate that self-reported sleep measures, but not actigraphy or PSG measures, overlap with affective dimensions. Potential reasons for this may include measurement modality (self-reported vs. clinician evaluated insomnia and depression), diagnostic overlap between depression and sleep disturbance as these phenomena share clinical features such as fatigue and insomnia, or that actigraphy and PSG measures may reflect some homeostatic or circadian dimensions undetected by self-report measures. Apparently, sleep measures obtained by different modalities seem to probe different aspects of sleep functioning and its relation to pain. Future studies should examine whether these differences are complementary or clinically qualitatively different.

A cross-sectional design precludes any causal inferences from mediation modeling, as such models basically presume that the exposure occurs before the outcome and preferably also before the mediator. The proposed causal order tested in the present cross-sectional study is therefore tentative rather than conclusive. Although the grouping variable in our mediation analysis would not be meaningful to specify as a mediator or as an outcome, sleep quality and insomnia might instead be specified as a mediator of the pain-mental distress relationship. Indeed, previous mediation studies have reported mood and depressive symptoms as mediators of the sleep-pain relationship, as well as the reverse, i.e., pain-sleep relationship, as

our study represents.[32] Additionally, studies have tested sleep as a mediator of the pain-anxiety/depression relationship.[49-51] and of the pain catastrophizing-pain persistence relationship.[31] In one of these studies, PSG measured SE was a mediator of the pain-anxiety/depression relationship,[49] whereas the other studies solely applied self-reported sleep measures. Taken together, the findings of these mediation studies suggest that common neurobiological pathways may underpin symptoms of pain, depression and sleep disturbance.[33] To permit causal inferences to be drawn about mediation and mechanism between pain and sleep more reliably, longitudinal studies that ensure the temporal ordering of the variables are needed.

Polysomnography recordings allowed estimation of factors associated with changes in sleep architecture. The shift from SWS to more light sleep in this study was associated with the level of pain catastrophizing. This is a novel finding indicating a possible association between pain related cognitive processes and SWS in patients with chronic musculoskeletal pain. The Pain Catastrophizing Scale taps helplessness, magnification and rumination with negative pain-related thoughts and is associated with increased pain sensitivity and persistent pain.[37,52] When combined with sleep disturbances it may also be associated with central sensitization and clinical pain, where the rumination component in particular seems to be involved.[31,53]

In good sleepers, homeostatic factors will typically ensure compensatory increase in SWS following SWS deprivation, whereas in insomnia there is a lack of such compensation.[54] The models of psychophysiological hyperarousal and dysregulation of the sleep homeostasis have been proposed as potential mechanisms.[54,55] Presleep cognitive arousal, including rumination, seems to be associated with reduced sleep quality, whereas mixed results are reported for associations with actigraphy measured SE in pain patients.[12,19,30] Our findings of the association between pain catastrophizing and SWS may thus indicate that pain-specific

cognitive arousal is involved in the loss of SWS in chronic pain patients. This may be a pain-specific version of the hyperarousal of insomnia.

In the current study we excluded participants with previously diagnosed sleep disordered breathing (SDB). Over recent years PSG scoring criteria with varying sensitivity to detect respiratory events have been recommended, resulting in large variations in AHI and prevalence estimates of sleep disordered breathing (SDB).[56,57] Considering this source of variation in AHI, one may argue that the diagnosis of SDB in asymptomatic persons should not solely be based on the generally accepted cut-off score of AHI> 15.[57,58] Due to these considerations we chose not to exclude participants on the basis of the apnea-hypopnea index (AHI) emerging from the PSG in the study, but rather controlled for AHI as a continuous variable in the statistical models. As removing participants with AHI> 15, as a sensitivity test of the regression analyses, did not substantially change our results this seems like a reasonable approach.

In the current study, there was no group difference in AHI, yet sleep disordered breathing (SDB) has previously been proposed to be associated with pain.[15,59] It remains to be established how SDB contributes to pain, but treatment of comorbid obstructive sleep apnea should always be considered, and it should be further studied if such treatment could possibly also have an effect on pain in patients with comorbid SDB and chronic pain.

There are several limitations to this study. The cross-sectional design prevents any causal inference of associations between sleep, pain and mental distress. For such inferences one must rely on longitudinal studies. The sample size, with 109 subjects, was sufficient to detect clinically meaningful group differences, yet restricting the number of covariates to be included in the regression model. We chose to perform one night of PSG in order to prevent attrition, since most persons find the PSG equipment somewhat uncomfortable to wear. We may have introduced some first night effects,[60] yet presumably proportionate in both groups

that were compared,[15] and minimized by performing the PSG in the patients' home environment. In this study patients and controls were instructed to go about their lives as usual. This increases the ecological validity of the results, but also adds some diversity of uncontrolled factors, such as sleep schedule, diet, and activity. Several relevant confounders could be controlled for in the regression models (age, gender, education, AHI). Yet, there are possible confounders, such as activation of stress responses, and possibly also chronotypes, that were not measured. These are factors that may be associated with both sleep problems, mental distress and pain, and may thus possibly explain some of the associations observed in this study.[33,61]

Strengths of the study are the comprehensive recordings of sleep across multiple modalities, allowing assessment of various aspects of sleep. Since the association between sleep and pain may vary between pain conditions,[6] the fairly homogenous sample of pain patients may be an advantage. Another advantage is the strictly blinded evaluation of actigraphy and PSG recordings.

In conclusion, the study underlines the close relation between affective processes and sleep quality/insomnia, reflecting the commonly reported comorbidity of insomnia and depression, and where targeting both conditions simultaneously seem to yield superior treatment effects.[62] Whereas mental distress in this study was related to sleep quality and insomnia, such relation was not found for measures of SE and SWS. On the other hand, pain catastrophizing, possibly reflecting pain related cognitive hyperarousal, was significantly associated with reduced SWS. Hence it is possible that interventions directed towards such cognitive processes may also affect sleep architecture, which remains a future perspective. The study illustrates the complementary properties of self-reported, actigraphic and polysomnographic sleep measurements. In a clinical setting, adding actigraphy and/or PSG to diagnose sleep disturbance, at least in selected patients, may add valuable information as these

modalities seem to reflect different aspects of sleep functioning, which may in turn be addressed in a multidisciplinary rehabilitation context.

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6 References

1. Alfoldi P, Wiklund T, Gerdle B. Comorbid insomnia in patients with chronic pain: a study based on the Swedish quality registry for pain rehabilitation (SQRP). *Disability and rehabilitation* 2014;36:1661-9.
2. Finan PH, Goodin BR, Smith MT. The association of sleep and pain: an update and a path forward. *The journal of pain : official journal of the American Pain Society* 2013;14:1539-52.
3. Bjurstrom MF, Irwin MR. Polysomnographic characteristics in nonmalignant chronic pain populations: A review of controlled studies. *Sleep medicine reviews* 2015;26:74-86.
4. Tang NK, Goodchild CE, Hester J, et al. Pain-related insomnia versus primary insomnia: a comparison study of sleep pattern, psychological characteristics, and cognitive-behavioral processes. *Clin J Pain* 2012;28:428-36.
5. Roth T, Bhadra-Brown P, Pitman VW, et al. Characteristics of Disturbed Sleep in Patients With Fibromyalgia Compared With Insomnia or With Pain-Free Volunteers. *Clin J Pain* 2016;32:302-7.
6. Bonvanie IJ, Oldehinkel AJ, Rosmalen JG, et al. Sleep problems and pain: a longitudinal cohort study in emerging adults. *Pain* 2016;157:957-63.
7. Aili K, Nyman T, Svartengren M, et al. Sleep as a predictive factor for the onset and resolution of multi-site pain: a 5-year prospective study. *European journal of pain (London, England)* 2015;19:341-9.
8. Tang NK, Lereya ST, Boulton H, et al. Nonpharmacological Treatments of Insomnia for Long-Term Painful Conditions: A Systematic Review and Meta-analysis of Patient-Reported Outcomes in Randomized Controlled Trials. *Sleep* 2015;38:1751-64.

9. Boggero IA, Schneider VJ, 2nd, Thomas PL, et al. Associations of self-report and actigraphy sleep measures with experimental pain outcomes in patients with temporomandibular disorder and healthy controls. *J Psychosom Res* 2019;123:109730.

10. Whibley D, Braley TJ, Kratz AL, et al. Transient Effects of Sleep on Next-Day Pain and Fatigue in Older Adults With Symptomatic Osteoarthritis. *The journal of pain : official journal of the American Pain Society* 2019;20:1373-1382.

11. 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;27:425-33.

12. Tang NK, Goodchild CE, Sanborn AN, et al. Deciphering the temporal link between pain and sleep in a heterogeneous chronic pain patient sample: a multilevel daily process study. *Sleep* 2012;35:675-87A.

13. Wu YL, Chang LY, Lee HC, et al. Sleep disturbances in fibromyalgia: A meta-analysis of case-control studies. *J Psychosom Res* 2017;96:89-97.

14. Alsaadi SM, McAuley JH, Hush JM, et al. Assessing sleep disturbance in low back pain: the validity of portable instruments. *PloS one* 2014;9:e95824.

15. Mathias JL, Cant M, Burke ALJ. Sleep disturbances and sleep disorders in adults living with chronic pain: a meta-analysis. *Sleep Med* 2018;52:198-210.

16. van de Water AT, Eadie J, Hurley DA. Investigation of sleep disturbance in chronic low back pain: an age- and gender-matched case-control study over a 7-night period. *Man Ther* 2011;16:550-6.

17. Lunde LH, Pallesen S, Krangnes L et al. Characteristics of sleep in older persons with chronic pain: a study based on actigraphy and self-reporting. *Clin J Pain* 2010;26:132-7.

18. Lewandowski AS, Palermo TM, De la Motte S et al. Temporal daily associations between pain and sleep in adolescents with chronic pain versus healthy adolescents. *Pain* 2010;151:220-5.

19. Palermo TM, Toliver-Sokol M, Fonareva I et al. Objective and subjective assessment of sleep in adolescents with chronic pain compared to healthy adolescents. *Clin J Pain* 2007;23:812-20.

20. Murray CB, Murphy LK, Palermo TM et al. Pain and sleep-wake disturbances in adolescents with depressive disorders. *J Clin Child Adolesc Psychol* 2012;41:482-90.

21. Kravitz HM, Zheng H, Bromberger JT, et al. An actigraphy study of sleep and pain in midlife women: the Study of Women's Health Across the Nation Sleep Study. *Menopause* 2015;22:710-8.

22. Edwards RR, Grace E, Peterson S, et al. Sleep continuity and architecture: associations with pain-inhibitory processes in patients with temporomandibular joint disorder. *European journal of pain (London, England)* 2009;13:1043-7.

23. Weingarten JA, Dubrovsky B, Basner RC, et al. Polysomnographic Measurement of Sleep Duration and Bodily Pain Perception in the Sleep Heart Health Study. *Sleep* 2016;39:1583-9.

24. Dijk DJ, Groeger JA, Stanley N et al. Age-related reduction in daytime sleep propensity and nocturnal slow wave sleep. *Sleep* 2010;33:211-23.

25. Bair MJ, Robinson RL, Katon W et al. Depression and pain comorbidity: a literature review. *Archives of internal medicine* 2003;163:2433-45.

26. Nicassio PM, Ormseth SR, Kay M, et al. The contribution of pain and depression to self-reported sleep disturbance in patients with rheumatoid arthritis. *Pain* 2012;153:107-12.

27. Palermo TM, Wilson AC, Lewandowski AS, et al. Behavioral and psychosocial factors associated with insomnia in adolescents with chronic pain. *Pain* 2011;152:89-94.

28. Valrie CR, Gil KM, Redding-Lallinger R, et al. Daily mood as a mediator or moderator of the pain-sleep relationship in children with sickle cell disease. *J Pediatr Psychol* 2008;33:317-22.

29. Diaz-Piedra C, Catena A, Sanchez AI, et al. Sleep disturbances in fibromyalgia syndrome: the role of clinical and polysomnographic variables explaining poor sleep quality in patients. *Sleep Med* 2015;16:917-25.

30. Smith MT, Perlis ML, Smith MS, et al. Sleep quality and presleep arousal in chronic pain. *J Behav Med* 2000;23:1-13.

31. Buenaver LF, Quartana PJ, Grace EG, et al. Evidence for indirect effects of pain catastrophizing on clinical pain among myofascial temporomandibular disorder participants: the mediating role of sleep disturbance. *Pain* 2012;153:1159-66.

32. Whibley D, AlKandari N, Kristensen K, et al. Sleep and Pain: A Systematic Review of Studies of Mediation. *Clin J Pain* 2019;35:544-558.

33. Boakye PA, Olechowski C, Rashiq S, et al. A Critical Review of Neurobiological Factors Involved in the Interactions Between Chronic Pain, Depression, and Sleep Disruption. *Clin J Pain* 2016;32:327-36.

34. Cleeland CS Pain assessment in cancer. In: Osoba D ed. *Effect of cancer on quality of life*. Boca Raton, FL: CRC Press, 1991:293-305.

35. Klepstad P, Loge JH, Borchgrevink PC, et al. The Norwegian Brief Pain Inventory Questionnaire: Translation and Validation in Cancer Pain Patients. *Journal of pain and symptom management* 2002;24:517-525.

36. Derogatis LR, Lipman RS, Rickels K, et al. The Hopkins Symptom Checklist (HSCL): a self-report symptom inventory. *Behav Sci* 1974;19:1-15.

37. Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and validation. *Psychological Assessment* 1995;7:524-532.

38. Fernandes L, Storheim K, Lochting I et al. Cross-cultural adaptation and validation of the Norwegian pain catastrophizing scale in patients with low back pain. *BMC musculoskeletal disorders* 2012;13:111.

39. Morin CM. *Insomnia: Psychological assessment and management*. New York: The Guilford Press, 1993.

40. Morin CM, Belleville G, Belanger L et al. The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep* 2011;34:601-8.

41. Buysse DJ, Ancoli-Israel S, Edinger JD, et al. Recommendations for a standard research assessment of insomnia. *Sleep* 2006;29:1155-73.

42. Buysse DJ, Reynolds CF, 3rd, Monk TH, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry research* 1989;28:193-213.

43. Pallesen S. Pittsburgh sleep Quality Index. *Tidsskrift for Norsk Psykologforening* 2005:4.

44. Knutson KL, Rathouz PJ, Yan LL, et al. Intra-individual daily and yearly variability in actigraphically recorded sleep measures: the CARDIA study. *Sleep* 2007;30:793-6.

45. Ancoli-Israel S, Martin JL, Blackwell T, et al. The SBSM Guide to Actigraphy Monitoring: Clinical and Research Applications. *Behavioral sleep medicine* 2015;13 Suppl 1:S4-s38.

46. American Academy of Sleep Medicine. *The AASM Manual for the Scoring of Sleep and Associated Events, version 2.4*. Darien, IL: American Academy of Sleep Medicine, 2017.

47. Hayes AF. *Introduction to mediation, moderation and conditional analysis: a regression-based approach*. New York: The Guilford Press, 2018.

48. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behav Res Methods* 2008;40:879-91.

49. Diaz-Piedra C, Catena A, Miro E, et al. The impact of pain on anxiety and depression is mediated by objective and subjective sleep characteristics in fibromyalgia patients. *Clin J Pain* 2014;30:852-9.

50. Miro E, Martinez MP, Sanchez AI, et al. When is pain related to emotional distress and daily functioning in fibromyalgia syndrome? The mediating roles of self-efficacy and sleep quality. *Br J Health Psychol* 2011;16:799-814.

51. Campbell P, Tang N, McBeth J, et al. The role of sleep problems in the development of depression in those with persistent pain: a prospective cohort study. *Sleep* 2013;36:1693-8.

52. Edwards RR, Bingham CO, 3rd, Bathon J et al. Catastrophizing and pain in arthritis, fibromyalgia, and other rheumatic diseases. *Arthritis Rheum* 2006;55:325-32.

53. Campbell CM, Buenaver LF, Finan P, et al. Sleep, Pain Catastrophizing, and Central Sensitization in Knee Osteoarthritis Patients With and Without Insomnia. *Arthritis Care Res (Hoboken)* 2015;67:1387-96.

54. Pigeon WR, Perlis ML. Sleep homeostasis in primary insomnia. *Sleep medicine reviews* 2006;10:247-54.

55. Riemann D, Spiegelhalder K, Feige B, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep medicine reviews* 2010;14:19-31.

56. BaHammam AS, Obeidat A, Barataman K, et al. A comparison between the AASM 2012 and 2007 definitions for detecting hypopnea. *Sleep Breath* 2014;18:767-73.

57. Heinzer R, Vat S, Marques-Vidal P, et al. Prevalence of sleep-disordered breathing in the general population: the HypnoLaus study. *Lancet Respir Med* 2015;3:310-8.

58. Epstein LJ, Kristo D, Strollo PJ, Jr., et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 2009;5:263-76.

59. Nadeem R, Ghadai A, Iftikhar S, et al. Prevalence of clinically probable obstructive sleep apnea in American veterans with chronic musculoskeletal pain. *Healthy Aging Res* 2016;5.

60. Blackwell T, Paudel M, Redline S, et al. A novel approach using actigraphy to quantify the level of disruption of sleep by in-home polysomnography: the MrOS Sleep Study: Sleep disruption by polysomnography. *Sleep Med* 2017;32:97-104.

61. Knutson KL, von Schantz M. Associations between chronotype, morbidity and mortality in the UK Biobank cohort. *Chronobiol Int* 2018;35:1045-1053.

62. Manber R, Buysse DJ, Edinger J, et al. Efficacy of Cognitive-Behavioral Therapy for Insomnia Combined With Antidepressant Pharmacotherapy in Patients With Comorbid Depression and Insomnia: A Randomized Controlled Trial. *J Clin Psychiat* 2016;77:E1316-+.

Table 1. Distribution of ICD-10 diagnoses in pain sample

	n
M54.2 Cervicalgia	12
M54.5 Low back pain	11
M54.6 Pain in thoracic spine	1
M54.8 Other dorsalgia	2
M54.9 Dorsalgia, unspecified	11
M79.1 Myalgia	10
M79.6 Pain in limb	3
M79.7 Fibromyalgia	6

Table 2. Group Differences in Demographic Characteristics

	Patients (*n*=56)	**Controls (*n*=53)**	***p***
Age, *M* (*SD*)	41.7 (10.8)	41.8 (10.7)	*ns*
Female, n (%)	42 (75.0)	38 (71.7)	*ns*
Cohabitation, n (%)	35 (62.5)	40 (75.5)	*ns*
Higher education, n (%)	35 (62.5)	51 (96.2)	< .001
Employment, n (%)	42 (75.0)	52 (98.1)	< .001
Social benefit, n (%)	28 (50.0)	2 (3.8)	< .001
Good economy, n (%)	16 (28.6)	36 (67.9)	< .001
Medication			
Opioids, n (%)	6 (10.7)	0	.027
Hypnotics, n (%)	3 (5.4)	0	ns
Antidepressants, n (%)	6 (10.7)	0	.027
Antiepileptic drugs, n (%)	3 (5.4)	0	ns
NSAID, paracetamol, n (%)	18 (32.1)	0	<.001
Other, n (%)	34 (60.7)	7 (13.2)	<.001
	Patients (*n*=53)	Controls (*n*=53)	
BMI, *M* (*SD*)	27.0 (4.5)	25.4 (3.9)	*ns*

Note: BMI = body mass index, *ns* = p > .05

Table 3. Group Differences in Self-report Questionnaire Measures

	Patients (*n*=56) ***M* (*SD*)**	**Controls (*n*=53)** ***M* (*SD*)**	***CI* 95%**	***g***
BPI	4.12 (1.46)	0.81 (0.75)	-3.76 to -2.87	2.81***
HSCL	1.73 (0.52)	1.24 (0.28)	-0.64 to -0.33	1.16***
PCS	12.32 (10.69)	3.58 (3.44)	-11.79 to -5.89	1.08***

Note: *** $p < .001$, *CI* 95% = bootstrapped 95% confidence interval of mean difference, *g* = Hedge's standardized mean difference, BPI = Brief Pain Inventory, HSCL = Hopkins Symptom Checklist, PCS = Pain Catastrophizing Scale

Table 4. Group Differences in Sleep Measures

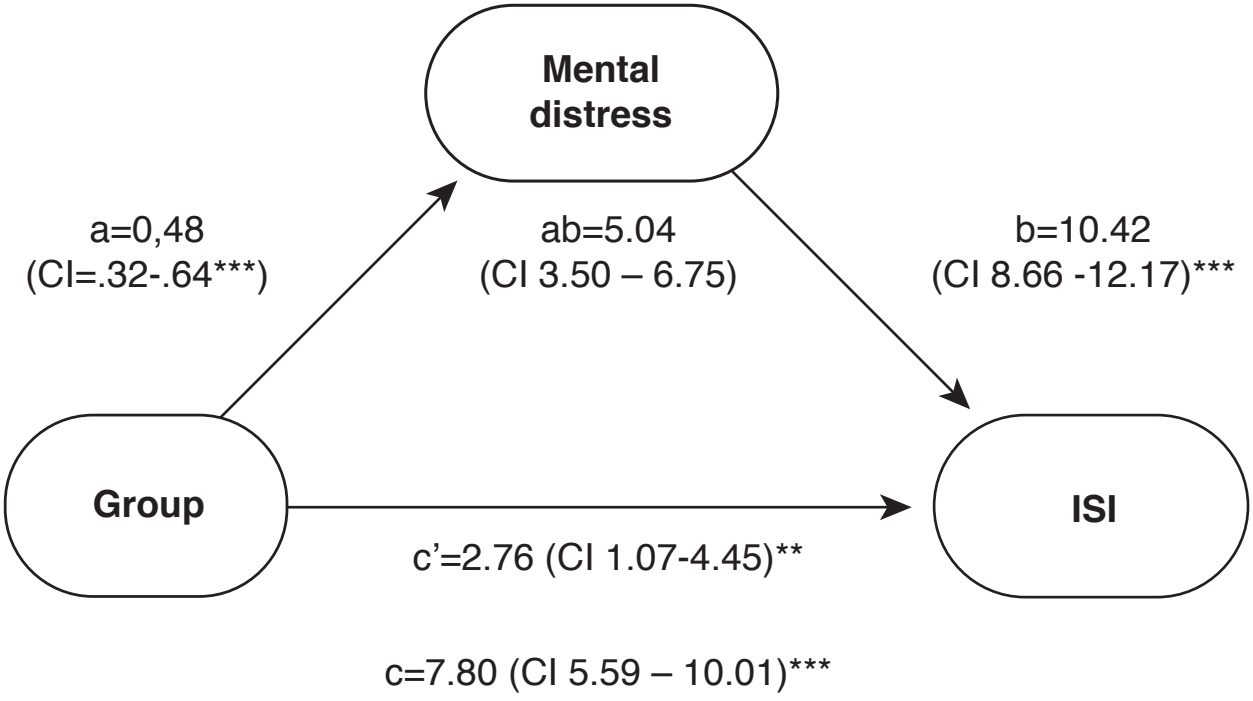
	Patients (*n*=56) ***M* (*SD*)**	**Controls (*n*=53)** ***M* (*SD*)**	***CI* 95%**	***g***
Self-report instruments				
ISI sum	11.9 (7.0)	4.1 (4.3)	-9.93 to -5.66	1.32***
PSQI global score	9.7 (4.3)	4.5 (2.4)	-6.55 to -3.97	1.47***
Actigraphy				
SOL, minutes	16.4 (18.1)	9.4 (8.0)	-12.56 to -2.22	0.49*
WASO, minutes	36.1 (15.5)	29.0 (10.4)	-12.22 to-2.20	0.53**
TST, minutes	398.7 (48.9)	399.8 (39.7)	-15.47 to 17.65	-0.02
SE, %	86.7 (5.0)	89.5 (4.0)	1.09 to 4.45	-0.61**
PSG	Patients (*n*=53)	Controls (*n*=51)		
SOL, minutes	23.0 (32.0)	16.9 (18.3)	-16.56 to 3.29	0.23
WASO, minutes	29.3 (30.6)	22.3 (18.6)	-16.85 to 2.20	0.27
TST, minutes	395.8 (75.3)	384.6 (54.9)	-36.22 to 14.61	0.17
SE, %	88.3 (9.5)	90.7 (5.9)	-0.55 to 5.46	-0.30
N1, % of TST	10.8 (5.7)	10.5 (5.2)	-2.36 to 1.88	0.05
N2, % of TST	47.0 (6.5)	43.5 (8.4)	-6.47 to -0.62	0.46*
N3, % of TST	22.7 (7.9)	26.7 (10.6)	0.57 to 7.66	-0.43*
REM, % of TST	19.5 (5.4)	19.3 (4.8)	-2.20 to 1.78	0.04
N3 latency, minutes	53.9 (56.9)	41.8 (29.6)	-30.79 to 3.83	0.26
Arousal index	9.6 (6.8)	10.6 (6.3)	-1.74 to 3.55	-0.15
Sleep stage change index	13.0 (3.9)	12.9 (3.2)	-1.52 to 1.19	0.03
Wake bout index	1.83 (1.21)	1.74 (0.99)	-0.50 to 0.33	
	Patients (*n*=49)	Controls (*n*=50)		
AHI	11.5 (11.9)	8.6 (8.5)	-7.03 to 1.15	0.28
AHI > 15, n (%)	14 (28.6)	9 (18)	*ns*	
	Patients (*n*=51)	Controls (*n*=47)		
PLMI	10.0 (11.8)	9.3 (16.9)	-6.33 to 5.39	0.05

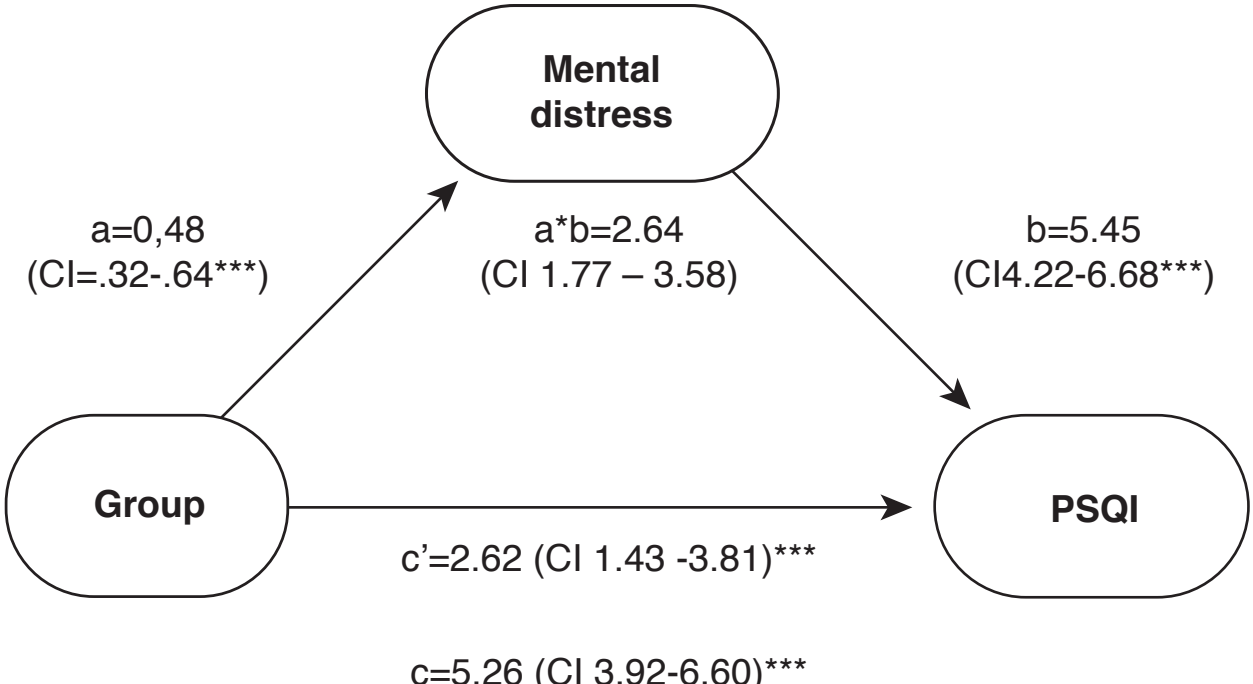
Note: * $p < .05$, ** $p < .01$, *** $p < 0.001$, *CI* 95% = bootstrapped 95% confidence intervals of mean difference, *g* = Hedge's standardized mean difference, ISI = Insomnia Severity Index, PSQI = Pittsburgh Sleep Quality Index, SOL = sleep onset latency, WASO= wake after sleep onset, TST = total sleep time, SE = sleep efficiency, AHI = apnea hypopnea index, PLMI= periodic limb movement index.

Table 5. Mental Distress (HSCL) and Pain Catastrophizing (PCS) as predictors of sleep measures, separately in chronic musculoskeletal pain (CMP) and healthy control (HC) group

Sleep outcome		**CMP**			**HC**	
Predictor	***Adj R^2***	***B (CI 95%)***	***t***	***Adj R^2***	***B (CI 95%)***	***t***
ISI	.62			.54		
HSCL		9.40 (6.30 to13.64)	5.02 ***		11.11 (6.46 to 15.69)	4.84 ***
PCS		-.04 (-.24 to .14)	-.39 ns		.10 (-.18 to .40)	.65 ns
Gender		-1.37 (-4.35 to 2.37)	-.80 ns		.48 (-1.73 to 2.91)	.40 ns
Age		.07 (-.08 to .19)	.93 ns		.08 (-.06 to .20)	1.18 ns
Education		-.19 (-3.19 to 2.51)	-.13 ns		4.75 (.83 to 8.08)	2.69 *
AHI		.16 (-.00 to .30)	2.11 *		-.06 (-.22 to .23)	-.55 ns
PSQI	.37			.52		
HSCL		5.43 (2.64 to 8.29)	3.76 **		5.97 (3.24 to 9.29)	4.02 ***
PCS		-.05 (-.19 to .13)	-.56 ns		.15 (-.06 to .33)	1.49 ns
Gender		-.60 (-3.15 to 2.19)	-.44 ns		-54 (-.70 to 1.76)	.87 ns
Age		-.01 (-.15 to .11)	-.22 ns		.04 (-.05 to .10)	.92 ns
Education		-.73 (-2.94 to 1.90)	-.59 ns		2.28 (.69 to 4.21)	2.60 *
AHI		.10 (-.04 to .20)	1.72 ns		-.05 (-14 to .13)	-.69 ns
$SE_{actigraphy}$	.05			.03		
HSCL		-2.74 (-7.21 to 1.93)	-1.19 ns		-4.14 (-11.05 to 1.06)	-1.33 ns
PCS		.14 (-.06 to .34)	1.38 ns		-.22 (-.63 to .25)	-.98 ns
Gender		-2.31 (-6.64 to 1.61)	-1.10 ns		.47 (-1.63 to 3.01)	.39 ns
Age		.15 (.03 to .28)	2.33 *		-.09 (-.24 to .05)	-1.19 ns
Education		1.04 (-2.51 to 4.62)	.58 ns		-4.63 (-8.63 to -1.01)	-2.51 *
AHI		-.09 (-.26 to .07)	-1.10 ns		.13 (-13 to .32)	1.17 ns
SWS	.04			.07		
HSCL		6.41 (-.08 to 12.88)	1.93 ns		-.11 (-9.15 to 14.11)	-.02 ns
PCS		-.37 (-.67 to -.05)	-2.34 *		-.07 (-1.13 to .80)	-.13 ns
Gender		3.16 (-3.18 to 9.07)	1.00 ns		-3.81 (-9.51 to 3.28)	-1.20 ns
Age		-.03 (-.39 to .25)	-.21 ns		-.50 (-.94 to -.15)	-2.47 *
Education		1.13 (-3.98 to 6.16)	.44 ns		-.03 (-18.51 to 11.97)	-.00 ns
AHI		-.09 (-.35 to .29)	-.53 ns		.31 (-.13 to .71)	1.53 ns

Note: * p< .05, ** p< .01, ***p< .001, ISI: Insomnia Severity index, PSQI: Pittsburgh Sleep Quality index, SE: Sleep Efficiency, SWS: Slow Wave Sleep, HSCL: Hopkin Symptom Checklist, AHI: Apnea Hypopnea Index


Mental distress
a=0,48
(CI=.32-.64***)
ab=5.04
(CI 3.50 – 6.75)
b=10.42
(CI 8.66 -12.17)***
Group
ISI
c'=2.76 (CI 1.07-4.45)**
c=7.80 (CI 5.59 – 10.01)***


Mental distress
a=0,48
(CI=.32-.64***)
a*b=2.64
(CI 1.77 – 3.58)
b=5.45
(CI4.22-6.68***)
Group
PSQI
c'=2.62 (CI 1.43 -3.81)***
c=5.26 (CI 3.92-6.60)***

Supplementary table 1. Bivariate Correlation. Healthy Controls Top Right, Chronic Musculoskeletal Pain Bottom Left																
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 BPI		.23	.37**	.32*	.29*	.34*	.01	-.27*	-.24	.05	-.08	.06	-.05	.17	.31*	.11
2 PCS	.53**		.36**	.29*	.40**	.31*	.02	.10	-.20	-.02	.01	-.00	-02	.09	.12	.14
3 HSCL	.42**	.80**		.74**	.71**	.20	.10	-.03	-.22	.27	-.11	.06	-.24	.49**	-.06	.19
4 ISI	.37**	.62**	.76**		.88**	.25	-.01	-.04	-.10	.09	-.12	.10	-.11	.36*	-.08	.14
5 PSQI	.37**	.50**	.63**	.75**		.35**	-.07	.04	-.09	.06	-.12	.10	-.09	.28	-.08	.06
6 $SOL_{actigraphy}$	.19	.17	.13	.09	.17		.13	-.26	-.60**	-.12	-.08	-.01	.29*	-.04	-.11	.02
7 $WASO_{actigraphy}$	.18	.12	.17	.08	.18	.09		.01	-.76**	.35*	.10	-.32*	.15	-.06	-.09	.32*
8 $TST_{actigraphy}$	.16	.09	.07	.02	-.08	.10	-.18		.41**	.10	-.09	.02	.02	-.12	-.08	-.06
9 $SE_{actigraphy}$	-.17	-.09	-.12	-.05	-.18	-.62**	-.69**	.41**		-.13	-.08	.21	-.19	-.01	.07	-.26
10 N1	.20	.06	-.09	.12	.04	.09	.17	-.10	-.18		-.07	-.41**	-.07	.31*	.11	.18
11 N2	.13	.39**	.35	.29	.25	.05	.15	.16	-.04	.08		-.76**	.00	.02	.34*	.07
12 N3	-.26	-.23	-.06	-.21	-.09	-.10	-.09	-.04	.09	-.57**	-.61**		-.42**	-.09	-.26	-.19
13 REM	.02	-.20	-.23	-.16	-.21	-.01	-.24	-.03	.11	-.33*	-.39	-.12		-.17	-.13	.10
14 AHI	.17	.23	.08	.35*	.28	-.13	.11	-.38**	-.12	.46**	-.05	-.17	-.20		.27	.28
15 PLMI	-.08	-.13	-.14	-.06	-.01	-.12	-.11	-.40**	-.04	.05	-.23	.26	-.16	.23		.03
16 ARI	.09	.10	.07	.17	.10	-.23	.16	-.22	-.07	.09	-.17	.08	-.01	.50**	.19	
Note: * p<.05, ** p<.01, BPI: Brief Pain Inventory, PCS: Pain Catastrophizing Scale, HSCL: Hopkin Symptom Checklist, ISI: Insomnia Severity Index, PSQI: Pittsburgh Sleep Quality Index, SOL: Sleep Onset Latency, WASO: Wake after Sleep Onset, TST: Total Sleep Time, SE: Sleep Efficiency, N1/N2/N3/REM: Sleep Stages as Proportion of TST, AHI: Apnea Hypopnea Index, PLMI: Periodic Limb Movement Index, ARI: Arousal Index. Indexes Specified as Event/Hour																

Supplementary table 2 Mental Distress (HSCL) and Pain Catastrophizing (PCS) as predictors of self-report and objective sleep measures in pain and healthy control samples separately, excluded participants with AHI > 15.						
Sleep outcome	**CMP**			**HC**		
Predictor	*Adj R^2*	*B (CI 95%)*	*t*	*Adj R^2*	*B (CI 95%)*	*t*
ISI	.57			.45		
HSCL		11.40 (6.10 to 17.90)	3.82 **		13.41 (6.49 to 18.49)	4.39 ***
PCS		-.04 (-.33 to .19)	-.31 ns		.10 (-.20 to .47)	.59 ns
Gender		-1.02 (-5.49 to 3.33)	-.44 ns		-.21 (-2.57 to 2.19)	-.17 ns
Age		.03 (-.13 to .18)	.36 ns		.04 (-.06 to .14)	.82 ns
Education		.33 (-3.11 to 3.81)	.19 ns		5.24 (2.98 to 7.94)	4.16 **
PSQI	.31			.53		
HSCL		6.26 (2.28 to 10.25)	3.01 **		8.70 (4.95 to 11.32)	5.43***
PCS		-.09 (-.30 to .15)	-.76 ns		.11 (-.10 to .31)	1.06 ns
Gender		-.51 (-4.02 to 3.49)	-.26 ns		.60 (-.55 to 1.81)	.99 ns
Age		.01 (-.12 to .13)	.10 ns		.01 (-.04 to .07)	.32 ns
Education		.02 (-2.72 to 3.11)	.01 ns		2.42 (1.12 to 3.68)	3.81 **
$SE_{actigraphy}$	.07					
HSCL		-.24 (-5.69 to 5.09)	-.09 ns		-5.16 (-14.29 to 3.95)	-1.13 ns
PCS		-.02 (-.31 to .25)	-.14 ns		-.18 (-.68 to .29)	.75 ns
Gender		-1.53 (-8.38 to 3.80)	-.49 ns		.79 (-1.68 to 3.54)	.59 ns
Age		.13 (-.01 to .28)	1.78 ns		-.08 (-.22 to .04)	-1.23 ns
Education		2.48 (-1.52 to 6.53)	1.24 ns		-5.01 (-8.18 to -1.80)	-3.04 *
SWS	-.07			.07		
HSCL		5.25 (-4.37 to 12.76)	1.22 ns		8.45 (-13.37 to 31 01)	.77 ns
PCS		-.35 (-.75 to .12)	-1.57 ns		-.47 (-1.67 to .72)	-.79 ns
Gender		2.40 (-6.48 to 10.12)	.56 ns		-.90 (-9.27 to 6.82)	-.22 ns
Age		-.11 (-.48 to .27)	-.56 ns		-.42 (-.83 to - .03)	-2.07 *
Education		.03 (-6.46 to 6.37)	.01 ns		10.00 (1.61 to 19.44)	2.24 ns
Note: * p< .05, ** p< .01, ***p< .001, CMP: Chronic Musculoskeletal Pain, HC: Healthy Control, ISI: Insomnia Severity index, PSQI: Pittsburgh Sleep Quality index, SE: Sleep Efficiency, SWS: Slow Wave Sleep, HSCL: Hopkin Symptom Checklist, AHI: Apnea Hypopnea Index						