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Disruption of sleep by one night of in-home polysomnographic recording: a longitudinal actigraphy study of patients with chronic musculoskeletal pain and pain-free controls

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Abstract

Background Patients with chronic pain frequently have comorbid sleep disturbances. Since improvement of sleep may alleviate both sleep problems and to some extent pain, sleep studies in this group becomes relevant. Polysomnography (PSG) is considered the gold standard for characterizing sleep; however, it is resource-demanding and may itself disrupt sleep due to the use of inconvenient equipment in unfamiliar sleep environments. To circumvent disruptive first night effect that may occur, sleep protocols may prescribe several nights of PSG to facilitate adaptation despite the equipment may still influence sleep on all nights. Moreover, the disruptive effects of polysomnography may vary between patient groups and healthy persons, yet have not previously been studied in patients with chronic pain. The present study aimed to assess whether sleep disruption during one night of in-home PSG was more severe in patients with chronic musculoskeletal pain compared to pain-free controls.

Method Sleep was assessed by self-reported sleep quality and actigraphy measured sleep onset latency, sleep duration, wake after sleep onset and sleep efficiency during one night of in-home PSG and the following six nights in 56 patients and 53 pain-free participants. Additionally, sleep schedule was assessed by sleep onset time, wake up time and time in bed. The repeated sleep measures were analysed with mixed model regressions, comparing mean score changes between and within groups.

Results A disruptive effect of PSG was evident for self-reported sleep quality and actigraphy measured sleep onset latency in both groups. These effects were however not significantly different between the groups, indicating comparable sensitivity to a single night of PSG between pain patients and pain-free controls.

Conclusion These findings suggest that a singlenight in-home PSG protocol may be considered for case–control studies of patients with chronic pain.

Keywords Chronic musculoskeletal pain, Actigraphy, Polysomnography, Sleep disturbance, First-night effect

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Background

Comorbid sleep disturbances are frequently reported among patients with chronic pain (Alfoldi et al. 2014), and the relationship between sleep and pain seems to be of a bidirectional character (Finan et al. 2013). In multidisciplinary pain treatment settings, sleep behaviour may be addressed therapeutically to help alleviate pain and/or improve sleep (Tang et al. 2015), thus motivating studies of sleep in this patient group. Polysomnography (PSG) is considered the gold standard method for assessing sleepfeatures, as it provides a comprehensive assessment of sleep continuity, sleep architecture and physiological parameters related to breathing, heart rate, and movement (Academy and of Sleep Medicine 2017). However, the PSG equipment may itself generate sleep disruption as it may feel uncomfortable to wear, and interfere with sleeping positions. Subjects may also be required to sleep in unfamiliar environments (e.g., a sleep lab). The combined effect of these factors tends to be relatively more pronounced during the first night of PSG. Adaptation to the PSG is observed as improvement of sleep over 2–3 nights of repeated recording, and has been termed "first night effect" (FNE) (Coates et al. 1981; Edinger et al. 1991; Agnew et al. 1966). This effect has led to research protocols including one or several adaptation nights.

FNEs typically present with sleep characteristics consistent with sleep disruption, such as increased sleep onset latency (SOL), increased wake after sleep onset (WASO), longer latency to rapid eye movement sleep (REM sleep), and reduced total sleep time (TST), sleep efficiency (SE) slow wave sleep (SWS) and REM sleep (Coates et al. 1981; Agnew et al. 1966; Bon et al. 2003; Saletu et al. 1996). Studies have described FNEs, or even reversed FNEs, in various patient groups (Coates et al. 1981; Edinger et al. 1991, 1997; Bon et al. 2003; Saletu et al. 1996; Mendels and Hawkins 1967), but to our knowledge FNE has not yet been described in patients with chronic pain.

Since repeated PSG recordings describe the propensity for adaptation and not merely discrepancy in sleep between the first night of PSG and habitual nights (without PSG), which have been reported to last up to four nights (Bon et al. 2001), other methods for bypassing this potential bias are worth considering. In the present study we compared actigraphy measured sleep parameters of a single PSG-night with the subsequent nights, which represents an understudied methodological approach to understanding the disruptive effects of PSG (Blackwell et al. 2017; McCall and McCall 2012b; Withrow et al. 2019).

PSG is demanding in terms of economic and technical resources which may restrict its implementation, particularly within clinical or research settings assessing large numbers of participants. A more parsimonious protocol would thus be preferable, given satisfactory validity.

The aim of the current study was to examine the disruptive effect of a single night of PSG, as well as differences in such effects between patients with chronic musculoskeletal pain and pain-free control subjects, using a controlled sleep study protocol. We examined disruption in sleep continuity by actigraphy-recording and self-reported sleep quality (SQ) during one night of PSG-recording compared to the subsequent six home-nights without PSG in both groups. In the patient group we also conducted an additional data collection without PSG separated by about six months, thus enabling assessment of any effect of entering the study itself. We hypothesized that sleep would be worse during the night of PSG compared to the subsequent nights with respect to self-reported SQ and actigraphy measured SOL, WASO, TST and SE, and that patients with chronic pain would be more susceptible to such effects and evidence more pronounced sleep disruption than healthy controls.

Methods

Procedure

Fifty-six patients with chronic musculoskeletal pain, who had attended an interdisciplinary out-patient pain clinic, and 53 pain free controls matched for age, sex and season of PSG were recruited by procedures previously reported (Abeler et al. 2020). Details regarding demographic characteristics, pain medication, psychological measures and sleep recordings (including PSG) have been reported previously (Abeler et al. 2020). Participants underwent 7 days of actigraphy recording and sleep quality scoring, and during the first night a PSG-recording was conducted concomitantly. Sleep quality scoring referred to the preceding night, thereby the night before PSG was included for this measure. Due to the purpose of another study assessing seasonal variations, patients were examined at two occasions; during summer and winter, and PSG was only performed at one of these two occasions, evenly distributed between seasons. Assessment of the pain free controls was only performed on one occasion, either during summer or winter. Thus, analyses in the present study included three groups: Patients without PSG (Group 1), patients with PSG (Group 2), and pain-free controls with PSG (Group 3). Five healthy controls were excluded as PSG was not performed on their first night. Three patients did not undergo PSG-recording, and one patient did not undergo data-collection without PSG. For one patient and one control the PSG was technically unsuccessful, but as the complete PSG equipment was attached during the night, these data were included in analyses of the concomitant actigraphy.

SOMNOscreen equipment (Somnomedics, Randersacker, Germany) was applied for PSG, and the recording was performed in accordance with The American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events, version 2.4 (American Academy of Sleep Medicine 2017). Six electroencephalographic leads (F3/F4, C3/C4, O1/O2), right and left electrooculogram and submental electromyogram were used for sleep scoring. Pressure flow nasal cannula, inductive thoracic and abdominal belts (effort) and oximetry were used for respiratory assessment. One-channel ECG was applied for heart rhythm, and bilateral pretibial electromyogram recordings for assessment of periodic limb movements. PSG data have been previously reported (Abeler et al. 2020).

Actigraphy

The Actiwatch Spectrum Plus device (Phillips Respironics, Inc., Murrysville, PA) was applied as a proxy-measure of sleep. The agreement with PSG is good for TST, SE and WASO based on low back pain patients, whereas concordance with SOL is poor (Alsaadi et al. 2014). These measures were collected across seven days in a week to obtain more stable sleep estimates, particularly for SOL which may have considerable night-to-night variability (Knutson et al. 2007). Since wearing the PSG equipment could potentially also affect sleep behavior as reflected in sleep schedule, the variables sleep onset time (SOT), wake up time (WUT) and time in bed (TIB) were added in ancillary analyses. 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 an event button. They also completed a seven-day sleep diary, recording the time going to bed, attempting to sleep, nightly awakenings, time out of bed, early morning wake up, as well as sleep latency. Actiware version 6.0.9 software was used for post-processing data (30 s epochs, medium sensitivity for activity detection and an immobility threshold of 10 min for sleep onset). When necessary, information based on the event marker, sleep diary and light intensity information were consulted, in line with a published guideline (Ancoli-Israel et al. 2015). Resting 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.

Questionnaire assessments

Demographics: Age, sex, and educational level – dichotomized as including high school (0) and education beyond high school (1) – were included as demographic variables.

Sleep quality -daily: One single question of daily overall sleep quality; "was the last night a good night (0)—bad night (100)?" was scored on a visual analogue scale each morning.

Insomnia: The Insomnia Severity Index (ISI) is a recommended research measure of insomnia symptoms (Buysse et al. 2006). It encompasses seven items scored on a Likert scale from 0 to 4 (total range 0–28) (Morin 1993). The ISI is a valid and reliable tool for insomnia, and a cut-off > 14 indicates clinical insomnia (Morin et al. 2011).

Sleep quality: The Pittsburgh Sleep Quality Index (PSQI) is a recommended research measure of global sleep symptoms (Buysse et al. 2006). It comprises 19 items probing sleep quality and disturbance during the last month, which are used to calculate seven sleep 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 from 0 (no difficulty) to 3 (severe difficulty), yielding a global score range of 0–21, with a higher score indicating worse sleep quality. A cut off score > 5 is recommended to distinguish poor sleepers from good sleepers (Buysse et al. 1989). The Norwegian translation has shown acceptable reliability and validity (Pallesen 2005).

Mental distress: 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 (Derogatis et al. 1974). Items are rated from 1-not at all to 4-very much, from which a global average score is calculated (range: 1–4). Scores above 1.75 indicate a treatment need (Sandanger et al. 1998).

Pain: Pain severity items of a validated Norwegian version of the Brief Pain Inventory (BPI) short form were applied (Cleeland and Osoba 1991; Klepstad et al. 2002). 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 (0-no pain to 10-worst imaginable pain). We used a mean severity score of these four items in the analyses of pain severity.

Statistical methods

The IBM SPSS 28 was used for all analyses. Descriptive statistics are presented for demographic and baseline sleep characteristics of the sample. The self-reported sleep quality (SQ) and the actigraphy-measured variables, i.e., sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST) and sleep efficiency (SE) were dependent variables. The ancillary

variables sleep onset time (SOT), wake up time (WUT) and time in bed (TIB) were added to reflect sleep schedule. Some of the dependent sleep variables were extremely skewed and kurtotic, which in most cases could be remedied by a natural logarithm or square root transformation, or a combination. In one instance (with SE as outcome) high kurtosis was still present after transformation, which were remedied by centering the scores and squaring the values on each side of the center by a factor of 0.90. The alpha level was set to 0.01. For interpretation purposes, the *p*-values obtained based on the transformed data were used to construct 99% normally distributed confidence intervals for the estimated mean scores of the original scale using the formula by Altman and Bland (2011).

The dependent variables were examined using the linear mixed model regression module for the three groups G1- patients without PSG, G2- patients with PSG and G3- healthy controls with PSG. Correlations in the repeated sleep data were accounted for by specifying a compound symmetry model for the residual covariance matrix, as other models (e.g., auto-regressive) did not fit better according to the Bayesian information criteria. All models specified three fixed factors: Time (estimating daily changes during the 7-day recording period), Group (estimating mean differences between the three comparison groups), and Time*Group (estimating different day-specific changes across the groups). In addition, sex, age, education level and week (0-workday, 1-weekend) were added as covariates.

A disruptive effect of PSG would be present if recovery in sleep occurred during the first night (+1 night) following the PSG night (tested by a simple comparison). We also examined if sleep recovery was maintained throughout the week (+2–6 nights) as compared with the PSG night (tested by defining contrasts for time $= -1(PSG\ night)\ vs \frac{1}{6}, \frac{1}{6}, \frac{1}{6}, \frac{1}{6}, \frac{1}{6}, \frac{1}{6}$). These contrast comparisons were examined for each of the three groups. To examine if the sleep data were stable or changed during the recording period (+2–6 nights), linear and quadratic contrasts were additional tested and described given significance. Finally, we examined if any FNEs, as examined above, were significantly different between the three groups by specifying additional *group* comparison contrasts.

Results

Demographic and baseline measures of sleep, pain and mental distress are reported in Table 1. The crude measures of sleep indices are presented in Fig. 1. The results of the effect of a single in-home night of PSG for each sleep outcome variable are presented in Table 2.

Table 1 Sample characteristics

	Group 1 *n* = 55	Group 2 *N* = 53	Group 3 *n* = 48
Females, n (%)	41 (74.5%)	39 (73.6%)	34 (70.8%)
Age, mean (95% CI)	41.7 (38.7, 44.5)	42.2 (39.3, 45.0)	41.8 (38.9, 44.7)
Education beyond high school, n (%)	34 (61.8%)	33 (62.2%)	46 (95.8%)
ISI, mean (95% CI)	11.2 (9.4, 12.8)	11.9 (10.1, 13.8)	4.0 (2.9, 5.4)
PSQI, mean (95% CI)	9.3 (8.3, 10.5)	9.9 (8.7, 11.1)	4.5 (3.8, 5.2)
HSCL, mean (95% CI)	1.75 (1.61, 1.91)	1.74 (1.60, 1.89)	1.23 (1.17, 1.30)
BPI, mean (95% CI)	4.2 (3.9, 4.6)	4.1 (3.8, 4.5)	0.8 (0.7, 1.1)

Group 1 Patients without PSG, *Group 2* Patients with PSG, *Group 3* Controls with PSG, *95% CI* Bootstrapped 95% confidence intervals, *ISI* Insomnia severity index, *PSQI* Pittsburgh sleep quality index, *HSCL* Hopkins symptoms checklist, *BPI* Brief pain inventory

Sleep Quality (SQ)

Both groups performing a PSG recording (G2/G3) rated their SQ significantly poorer (higher SQ-scores indicates worse sleep quality) during the PSG night compared to the next night (+1 night), the subsequent nights (+2–5 night) as well as the night preceding the PSG (-1 night). Similar changes were not observed in the no-PSG group (G1). The between-group contrasts showed that these change scores were significantly different between the no-PSG group (G1) and the two PSG groups (G2 and G3) for the recovery night (+1) and the subsequent nights (+2–5 nights).

Main effect analyses showed that healthy controls reported better overall sleep quality (all nights) than both patient groups (G3 vs. G1/G2: $M_{diff} = 10.1$, $p = 0.005$ / $M_{diff} = 13.5$, $p < 0.001$), whereas overall SQ was not different between the two patient groups.

Sleep Onset Latency (SOL)

Comparably as for SQ, actigraphy measured SOL was longer during the PSG-night compared to the recovery night (+1 day), and the subsequent nights (+2–6 days) in both PSG-groups (G2 and G3). Similar changes were not observed in the no-PSG group (G1). The between-group contrast tests showed significant differences between G1 (no-PSG) and G2 (PSG) on the recovery night, and between G1 and the G2 and G3 groups on the subsequent nights. A significant linear trend was present in G1 (no-PSG) as SOL increased towards the end of the 7-day period.

Main effect group comparisons showed significantly higher overall SOL (all nights) in G2 than in G3 ($M_{diff} = 6.0$, $p = 0.009$), thus PSG-patients had overall more difficulty falling asleep than PSG-control cases.

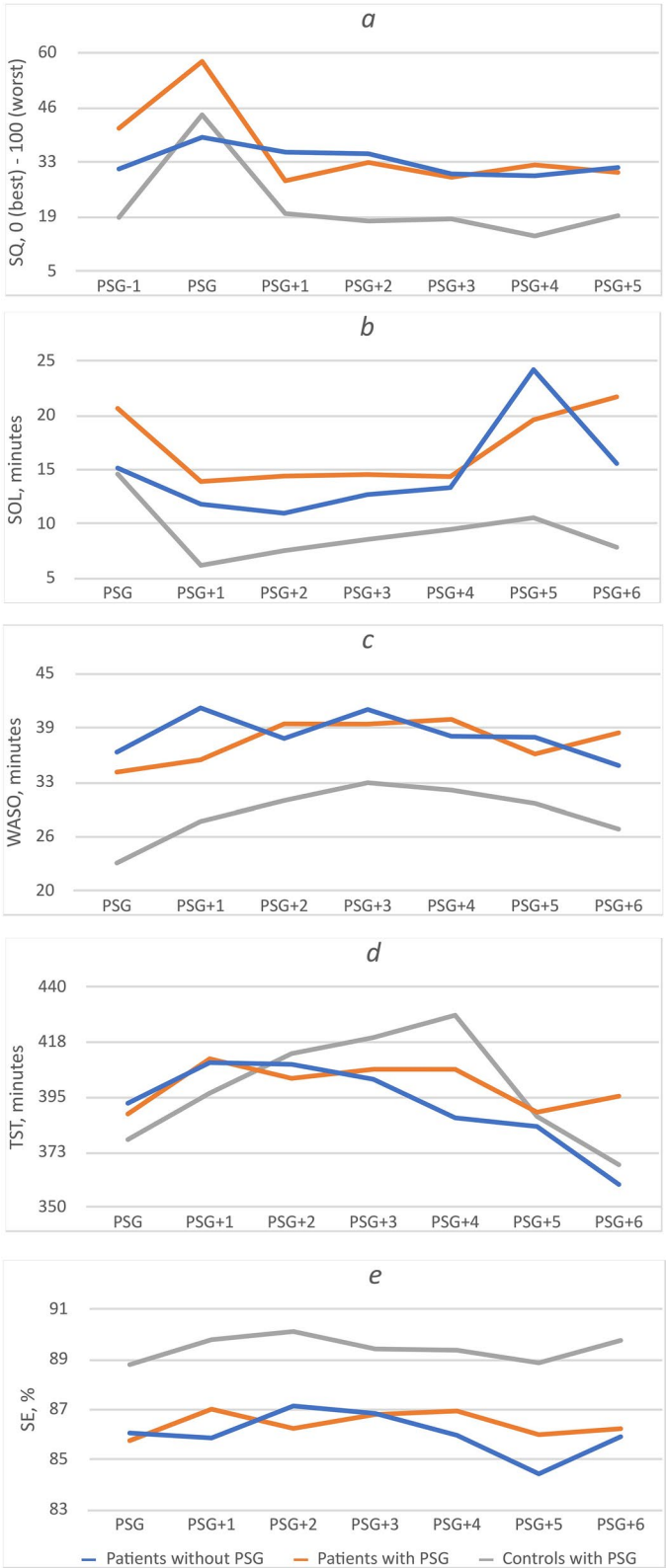

Fig. 1 Crude sleep indices during PSG and following nights. For sleep quality the night preceding PSG is included. Notes: SQ: sleep quality, SOL: sleep onset latency, WASO: wake after sleep onset, TST: total sleep time, SE: sleep efficiency, PSG: polysomnography

Table 2 Model Estimated Effects of Polysomnography in Actigraphy Measured Sleep

	-1 night ***M* diff** $_{99\%\ CI}$	**PSG night** ***M* (*SD*)**	**+1 night** ***M* diff** $_{99\%\ CI}$	**remaining nights** ***M* diff** $_{99\%\ CI}$	**trend**
Sleep Quality (SQ)	*Z-skew/kurt* (10.8 / -2.8). Transformation did not change the findings				
G1: No PSG (patients)	-7.4 $_{-18.1 \mid 3.3}$	38.1 (28.5)	- 3.7 $_{-14.4 \mid 7.9\backslash 0}$	- 6.4 $_{-14.7 \mid 1.9}$	
G2: PSG (patients)	-16.6*** $_{-27.2 \mid -5.9}$	57.9 (28.3)	- 30.6*** $_{-41.3 \mid -1.9}$	- 28.0*** $_{-38.3 \mid -19.7}$	
G3: PSG (controls)	-25.9*** $_{-37.3 \mid -14.6}$	45.4 (22.8)	- 25.8*** $_{-37.2 \mid -14.5}$	- 27.4*** $_{-36.1 \mid -18.8}$	
Between G1-G2-G3 contrasts ^{BC}	G3 < G1**	G2 > G1*** G2 > G3*	G2 < G1*** G3 < G1***	G2 < G1*** G3 < G1***	
Sleep Onset Latency (SOL)	*Z-skew/kurt* (78.4 / 366.5), which a *ln*(√SOL) transformation remedied (1.1 / -2.5)				
G1: No PSG (patients)		14.7 (29.1)	- 3.6 $_{-20.9 \mid 13.4}^{CI}$	- 0.4 $_{-2.2 \mid 2.0}^{CI}$	6,7 *
G2: PSG (patients)		20.3 (28.8)	- 6.7*** $_{-11.4 \mid -2.0}^{CI}$	- 4.1** $_{-7.6 \mid -0.6}^{CI}$	
G3: PSG (controls)		16.0 (13.6)	- 8.9* $_{-17.9 \mid 0.1}^{CI}$	- 6.7** $_{-12.4 \mid -1.0}^{CI}$	
Between G1-G2-G3 contrasts ^{BC}		G2 > G1*	G2 < G1*	G2 < G1* G3 < G1*	
Wake After Sleep Onset (WASO)	*Z-skew/kurt* (13.0 / 15.0), which a *ln*(*WASO*) transformation remedied (-0.4 / -0.2)				
G1: No PSG (patients)		36.5 (23.3)	3.9 $_{-5.9 \mid 13.7}^{CI}$	1.4 $_{-3.9 \mid 6.8}^{CI}$	
G2: PSG (patients)		34.6 (23.4)	0.6 $_{-8.2 \mid 9.4}^{CI}$	2.8 $_{-3.7 \mid 9.3}^{CI}$	
G3: PSG (controls)		25.1 (14.3)	3.6* $_{-0.8 \mid 7.9}^{CI}$	5.1*** $_{1.3 \mid 8.9}^{CI}$	
Between G1-G2-G3 contrasts ^{BC}		G1 > G3** G2 > G3**		G3 > G1*	
Total Sleep Time (TST)	*Z-skew/kurt* (2.2 / 15.7). Transformation did not change the findings				
G1: No PSG (patients)		6h 40m (88m)	6m $_{-40 \mid 29}$	-8m $_{-35 \mid 18}$	-26m**
G2: PSG (patients)		6h 40m (85m)	15m $_{-20 \mid 50}$	2m $_{-25 \mid 29}$	
G3: PSG (controls)		6h 27m (73m)	9m $_{-28 \mid 46}$	11m $_{-17 \mid 40}$	-19m^{**qt}
Between G1-G2-G3 contrasts ^{BC}					
Sleep Efficiency (SE %)	*Z-skew/kurt* (-26.1 / 80.0). Due to the severy skew, a *ln(SE)* transformation with additional kurtosis adjustment was performed even though it did not change the findings (1.2 / 2.3)				
G1: No PSG (patients)		86.3 (8.8)	-0.3 $_{-2.5 \mid 3.1}$	-0.1 $_{-2.2 \mid 2.1}$	
G2: PSG (patients)		86.0 (7.5)	1.2 $_{-1.7 \mid 4.0}$	0.7 $_{-1.5 \mid 2.9}$	
G3: PSG (controls)		88.5 (5.7)	1.0 $_{-2.0 \mid 4.1}$	0.8 $_{-1.6 \mid 3.1}$	
Between G1-G2-G3 contrasts ^{BC}		G3 > G1* G3 > G2*			

* $p < .05$, $^{**}p < .01$, $^{***}p < .001$. *Z-skew* / *Z-kurt* = Z-test skewness and kurtosis values based on fitted residual scores. ^{BC} Between group contrasts examine if change scores in sleep data from PSG to off-PSG nights are significantly different between two specified groups, or for the PSG night column, if the group mean levels are significantly different. ^{CI} 99% approximate confidence intervals based on *p*-values using transformed data. Trend = Significance test of a linear trend in sleep data during the remaining nights. ^{qt} quadratic trend during the remaining nights

Wake After Sleep Onset (WASO)

The WASO data did not indicate any unfavourable effects of PSG. Instead, a minor favourable effect was observed in G3 (PSG-controls) only, as WASO increased with 3.6 and 5.1 min during the next and subsequent nights, respectively. Furthermore, this WASO change was significantly different between the G1 (no-PSG patients) and the G3 group.

The main effect group comparisons indicated tentatively worse overall WASO (all nights) in the two patient groups (G1 and G2) as compared to the G3 (control) group ($M_{\text{diff}} = 8.6$, $p = 0.02$; $M_{\text{diff}} = 7.3$, $p = 0.04$, respectively).

Total Sleep Time (TST)

None of the tests showed any significant differences neither within nor between groups, nor in the main effect group comparisons regarding total sleep time. A negative linear trend was observed in G1 (no-PSG patients) indicating significantly less total sleep time towards the last days. Within G3 (PSG-controls), a significant quadratic effect emerged as TST increased from the first PSG night (6h 27min) beyond the middle period (+4 day: 7h 0min), followed by an abrupt reduction towards the two last nights (6h 16min).

Sleep Efficiency (SE)

None of the tests showed any significant differences neither within nor between the groups. The main effect group comparisons showed that healthy controls had better SE than both patient groups (G3 vs. G1/G2: $M_{diff}=3.1\%$, $p=0.005$ / $M_{diff}=2.4\%$, $p=0.011$).

Sleep timing and Time in Bed

Corresponding analyses of the ancillary variables sleep onset time (SOT), wake up time (WUT) as well as total Time In Bed (TIB) showed no significant differences between the G1, G2 and G3 groups neither for the fixed main group effects nor the contrast difference tests. The main effect of Time was however significant for SOT and WUT (both p's < 0.001), but the interaction effects (Group*Time) were not. The time effect for SOT indicated later times for sleep onset on the first night post-PSG (13, 17 and 10 min later) or all nights post-PSG (23, 39 and 28 min) as compared to the PSG night for the G1, G2 and G3 groups, respectively. The average sleep onset time was 0:15 o'clock. The corresponding delays for WUT were 23, 33 and 22 min and 16, 44 and 44 min. The average wake up time was 7:44 o'clock. Any further detailing is not laid out due to the lack of any relevant significant effects.

Discussion

In this study we assessed whether sleep during one night wearing full PSG equipment differed from subsequent nights without PSG, in patients with chronic musculoskeletal pain compared to pain-free controls. We observed reduced self-reported SQ, and prolonged actigraphy measured SOL during the PSG night among both patients and controls. In all groups, also in patients without PSG, earlier bedtimes were observed during the first night of data collection. No significant effects were observed for WASO, TST, SE or TIB during PSG, and there were no significant differences between patients and controls in any of the outcome measures.

PSG is considered a gold standard for sleep assessment, yet it is a concern that wearing the PSG-equipment and sleeping in a sleep-laboratory environment may introduce sleep disruption the first night— often characterized as the FNE. By repeated nights of PSG recordings, adaptation is assumed to take place resulting in less sleep disruption. Some studies have indicated that the adaptation to PSG equipment may be related to a more general capacity for adaptation, as FNE seems to be related to certain personality traits and sociodemographic factors (Edinger et al. 1991; Zheng et al. 2012). Additionally, REM-sleep may be particularly vulnerable for FNE, which may have implications for conditions where REM sleep is of particular importance, for example psychiatric conditions (Bon et al. 2001; Lorenzo and Barbanoj 2002). FNE may thus be expected to differ between patient groups. The disruptive effects of PSG – in the context of FNE or otherwise has, to our knowledge, not previously been studied among patients with chronic pain.

Among healthy volunteers, in-home PSG-assessment seems to minimize the multiple sleep disruptive effects reported in laboratory settings regarding sleep continuity and sleep architecture parameters, such as increased wake and drowsy states, increased latencies to SWS and REM-sleep as well as decreased TST, SE and amounts of SWS and REM-sleep (Coates et al. 1981; Agnew et al. 1966; Edinger et al. 1997; Mendels and Hawkins 1967; Bon et al. 2001). In-home PSG assessment studies of large population samples indicate minor disruption of sleep continuity and architecture parameters in females (Zheng et al. 2012) and older males (Blackwell et al. 2017). Among the 48 healthy controls in the current study, actigraphy data did not detect any substantial sleep disruption, except less than 9 min increased SOL, during one night of home-recording, and thus confirming findings of the majority of in-home PSG studies in healthy persons.

Previous studies of various other patient groups such as insomnia (Coates et al. 1981; Edinger et al. 1991, 1997), insomnia combined with general anxiety disorder (Saletu et al. 1996), depression (Mendels and Hawkins 1967), and chronic fatigue syndrome (Bon et al. 2003) have observed variable FNEs in sleep continuity and sleep architecture. Other studies have reported an absence of FNE in patients with arrythmia (Abumuamar et al. 2018), and even improved sleep — so-called reversed FNE — in a study of insomnia with depression (McCall and McCall 2012a) and in a subsample of patients with chronic fatigue (Bon et al. 2003). Reversed FNEs may occur if the PSG or lab-environment disrupts the maladaptive associations activated in the normal sleep environment (Riemann et al. 2010) or may reflect intraindividual variation (Buysse et al. 2010). Of notice, McCall and McCall observed reduced self-reported SQ in combination with the actigraphy-measured reversed FNEs (McCall and McCall 2012a, b). In the same vein, in the current study we observed a significant FNE in SQ, which was poorly reflected in actigraphy measures, thus illustrating the well-known weak correlation between subjective and objective sleep measures (Morin et al. 2011; Buysse et al. 1989; Wilson et al. 1998).

Fewer studies have compared FNE between groups, one such study did not identify significant differences in FNEs between insomniacs and controls (Edinger et al. 1997), in concurrence with the findings of the present study.

In a systematic review and two meta-analyses of controlled chronic pain studies, more than half of the included studies applied adaptation nights (Bjurstrom and Irwin 2015; Mathias et al. 2018; Wu et al. 2017). A potential bias of FNE may be suspected in studies lacking adaptation nights (Bjurstrom and Irwin 2015). Yet, the meta-analyses did not identify group effects of any sleep continuity or architecture parameter to be associated with protocols including adaptation night(s). Rather, group differences of comparable effect size between patients and controls were generally observed, irrespective of adaptation nights. The exception was an increase among patients´ sleep stage N1 only evident in the studies applying adaptation nights (Mathias et al. 2018; Wu et al. 2017). These findings are in agreement with the current study.

Most studies exploring FNEs have used repeated PSG recordings to assess adaptation, thereby introducing PSG itself as a potential confounding factor, as pointed out by Blackwell et al. (Blackwell et al. 2017). A few studies have applied actigraphy during and/or after PSG recording in order to bypass this potential problem, as we did in the current study. These studies have reported disruptive effects of PSG and recovery night phenomena regarding TST, SE and SOL in a population sample (Blackwell et al. 2017), recovery night in insomnia (Withrow et al. 2019), and reversed effects in combined insomnia and depression (McCall and McCall 2012a).

For TST we observed a non-linear trend with the longest TST during the middle of the data collection period and an abrupt reduction in TST during the last two nights, yet no significant group differences were observed. Due to the specific data collection procedure in the current study, with PSG performed from Monday through Thursday and actigraphy assessment continuing the following six nights, weekends frequently occur at day three to five of the data collection. Due to misalignment of biological and social circadian rhythms, sleep in weekends may be associated with later bedtimes and longer recovery sleep duration (Wittmann et al. 2006), which may have influenced our findings. Another explanation may be slow recovery over several days after PSG, which seems less likely. Although we included weekend as a covariate, it may not have fully controlled for all weekend and social jetlag effects. Nevertheless, weekend remains an important control variable in studies recording sleep both during weekdays and weekends.

There are several limitations to this study. Ideally, actigraphy-recorded sleep should have been measured for some nights prior to the PSG recording. This was however less convenient for the participants in the present study as most were fully employed. It would also imply more visits to the lab during the data collection period. Also, PSG could not be performed during weekends, thereby potentially introducing a systematic bias as discussed for TST. The approach of assessing sleep disruptive effects of PSG by means of actigraphy has some advantages as discussed above, but at the cost of missing measures of sleep architecture which may be more compromised by PSG than observed for the sleep continuity measures in the present study.

Conclusion

PSG is considered the gold standard of sleep recording, but the method demands considerable technical, medical, and economic resources potentially restricting its use. In the present study we detected no statistical difference in FNEs between healthy controls and patients with chronic musculoskeletal pain. Additionally, meta-analyses of controlled studies of sleep in chronic pain conditions have observed no or minimal effects of adaptation nights on group differences. These findings indicate that simplified PSG-protocols, without adaptation nights, may be considered in controlled sleep studies of chronic pain populations, particularly when performed in a home-setting.

Abbreviations

BPI	Brief Pain Inventory
FNE	First night effect
HSCL 25	Hopkins Symptoms Checklist 25
ISI	Insomnia Severity Index
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
REM	Rapid eye movement
SE	Sleep efficiency
SOL	Sleep onset latency
SQ	Sleep quality
SWS	Slow wave sleep
TST	Total sleep time
WASO	Wake after sleep onset

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Authors' contributions

KA performed data collection, scoring of sleep studies and writing the manuscript. OF performed the statistical analyses. OF and SB contributed actively in the writing process, and all authors read and approved the final manuscript.

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Availability of data and materials

The dataset generated during the current study are not publicly available since consent to publicly archive data was not included in the ethical approval at the time of data collection. An anonymized dataset may be prepared by the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
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.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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