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Depressive Symptoms Account for Differences between Self-reported Versus Polysomnographic Assessment of Sleep Quality in Women with Myofascial TMD

Boris Dubrovsky^{1,2}, Malvin N. Janal³, Gilles J. Lavigne⁴, David A. Sirois¹, Pia E. Wigren^{1,5}, Lena Nemelivsky⁶, Ana C. Krieger⁷, and Karen G. Raphael¹

¹Department of Oral and Maxillofacial Pathology, Radiology and Medicine, NYU College of Dentistry, New York, USA

²Department of Medicine, Division of Pulmonary and Critical Care Medicine, Center for Sleep Disorders, New York-Presbyterian Brooklyn Methodist Hospital, Brooklyn, USA

³Department of Epidemiology and Health Promotion, NYU College of Dentistry, New York, USA

⁴Faculté de Médecine Dentaire, Université de Montréal, Montréal, Canada

⁵Private practice, Stockholm, Sweden

⁶Cancer Clinical Trials Office, Icahn School of Medicine at Mount Sinai, New York, USA

⁷Departments of Medicine, Neurology and Genetic Medicine, Weill Cornell Medical College, Cornell University, New York, USA

Abstract

Background—Temporomandibular disorder (TMD) patients report poor sleep quality on the Pittsburgh Sleep Quality Index (PSQI). However, polysomnographic (PSG) studies show meager evidence of sleep disturbance on standard physiological measures.

Objective—The present aim was to analyze self-reported sleep quality in TMD as a function of myofascial pain, PSG parameters, and depressive symptomatology.

Methods—PSQI scores from 124 women with myofascial TMD and 46 matched controls were hierarchically regressed onto TMD presence, ratings of pain intensity and pain-related disability, in-lab PSG variables, and depressive symptoms (Symptoms Checklist-90).

Results—Relative to controls, TMD cases had higher PSQI scores, representing poorer subjective sleep, and more depressive symptoms (both $P < 0.001$). Higher PSQI scores were strongly predicted by more depressive symptoms ($P < 0.001$, $R^2 = 26\%$). Of 19 PSG variables, two

Correspondence: Dr. B. Dubrovsky, Department of Medicine, Division of Pulmonary and Critical Care Medicine, Center for Sleep Disorders, New York-Presbyterian Brooklyn Methodist Hospital, 501 Sixth Street, Brooklyn, NY 11215-9008, USA, bod9006@nyp.org.

DR. BORIS DUBROVSKY (Orcid ID : 0000-0002-5384-4915)

Ethical approval

The study procedures and the consent form were approved by the Institutional Review Board at the NYU School of Medicine.

Conflict of interest

The authors have indicated no financial conflicts of interest.

had modest contributions to higher PSQI scores: longer REM latency in TMD cases ($P = 0.01$, $R^2 = 3\%$) and more awakenings in all participants ($P = 0.03$, $R^2 = 2\%$). After accounting for these factors, TMD presence and pain ratings were not significantly related to PSQI scores.

Conclusion—These results show that reported poor sleep quality in TMD is better explained by depressive symptoms than by PSG-assessed sleep disturbances or myofascial pain. As TMD cases lacked typical PSG features of clinical depression, the results suggest a negative cognitive bias in TMD and caution against interpreting self-report sleep measures as accurate indicators of PSG sleep disturbance. Future investigations should take account of depressive symptomatology when interpreting reports of poor sleep.

Keywords

temporomandibular disorders; myofascial pain; sleep; polysomnography; depression; women

Background

Patients with temporomandibular disorders (TMD) comprise approximately 10% of the population, are predominantly female and frequently present with chronic pain in the masticatory muscles and temporomandibular joint.¹

Numerous studies show poor self-reported sleep quality in patients with TMD using the Pittsburgh Sleep Quality Index (PSQI).^{2–11} The PSQI combines 18 sleep history items into a global score, which, in excess of 5, is indicative of clinically poor sleep.¹² In uncontrolled TMD samples, mean PSQI scores ranged approximately between 7 and 11, depending on the type of myofascial pain and psychosocial factors, such as exposure to stressors.^{2–6} Case-control studies with and without matching report significantly higher PSQI scores in TMD patients than controls,^{7–11} providing further evidence for self-reported, or subjective, sleep disturbance in TMD.

Some studies report higher levels of self-reported pain among TMD patients with elevated PSQI scores,^{2,2,6,9} suggesting that pain is a causal factor in sleep impairment. However, other studies employing the PSQI do not evidence a connection between poor sleep and pain intensity.^{4,5} This lack of consistency in the PSQI-pain relationship in the TMD literature suggests that factors other than pain may contribute to subjectively poor sleep; that is, the TMD diagnosis may be a marker for some correlated factor that better explains reports of impaired sleep.

Several studies indicate that TMD patients reporting poor sleep on the PSQI also report more symptoms of depression, anxiety and distress.^{2–4,6,7,9} Additionally, recent evaluations of non-clinical samples of participants without a history of pain or sleep disorders show an association between PSQI scores and reports of depressive symptoms and distress, but not between PSQI data and such objective sleep measures as polysomnography (PSG) and actigraphy.^{13,14} These data suggest that the psychological distress often associated with chronic pain may account for self-reported poor sleep in TMD patients.

While the literature is consistent in showing subjective reports of impaired sleep in TMD patients, PSG evaluations of physiological variables generally fail to provide objective evidence of sleep disturbance. Two early investigations that employed small samples and restrictive selection criteria found no differences in PSG parameters between TMD patients and controls.^{15,16} A larger, yet uncontrolled, study of TMD patients revealed an essentially normal sleep architecture, based on measures such as sleep onset latency, rapid eye movement (REM) sleep latency, sleep efficiency and sleep stage distribution^{17,18} A recent PSG study of a large TMD sample showed mild, albeit significant, increases in the percentage of stage N1 sleep (N1%) and in the frequency of respiratory effort related arousals (RERAs) relative to demographically matched controls of similar body mass index (BMI); TMD patients were within normal limits on all other PSG variables.¹⁹ Thus, standard objective measurements of sleep quality fail to support the conclusion of impaired sleep quality in TMD patients that is suggested by self-reports.

The present aim was to evaluate how self-report ratings of sleep quality vary as a function of pain intensity, PSG measures, and reports of depressive symptoms in a large sample of women with TMD and demographically matched controls. It was hypothesized that subjectively poor sleep in TMD cases would be better explained by depressive symptoms than by objective sleep measures or pain severity.

Materials and methods

Participants

Participants were recruited at the New York University College of Dentistry (NYUCD) based on the Research Diagnostic Criteria for TMD with myofascial pain (RDC/TMD Group I).²⁰ Only women participated in the study, as TMD patients are predominantly female.¹ Control participants without myofascial TMD, matched for age, socioeconomic status and self-identified race and ethnicity, were recruited from those seeking care at the NYUCD dental clinics and among acquaintances of participating cases. Participants with potential sources of facial pain other than TMD and with certain conditions concerning pain or sleep, e.g., diabetes or the use of an appliance for sleep disordered breathing, were excluded. More detailed descriptions of inclusion/exclusion criteria have been reported.^{19,21}

The final sample included 124 TMD cases and 46 controls. Approximately 63% self-identified as white, 14% as black, and 23% as “other;” 23% self-identified as Hispanic. The mean $\pm$ SD age was 39.2 ± 14.6 years, the mean duration of education was 15 ± 2.2 years, and the mean BMI was 25.0 ± 5.1 kg/m². Cases and controls were similar in both exclusion rates and demographic composition, as reported elsewhere.^{19,21}

Procedures

Participants were consented, examined and interviewed at the NYU School of Medicine. Included participants provided self-report measures of sleep and mood and were scheduled to undergo two consecutive nocturnal PSG recordings at an affiliated sleep laboratory.

Self-report myofascial pain variables—Control participants free from myofascial pain, as ascertained during the initial evaluation, did not provide pain ratings. For TMD

cases, a standardized RDC/TMD pain history questionnaire was used to ascertain the length and severity of pain, and pain-related functional impairment.

To measure pain severity, participants rated (i) pain at the time of the interview, (ii) their worst pain and (iii) their average pain in the preceding 6 months, using a numeric scale from 0 (“no pain”) to 10 (“pain as bad as it could be”). The mean of three ratings was the characteristic pain intensity (CPI).²²

For functional impairment, participants rated the degree of pain interference with regard to the ability to perform (i) daily activities, (ii) social activities and (iii) work in the preceding 6 months, using a scale from 0 (“no interference/no change”) to 10 (“unable to carry on activities/extreme change”). The mean of these three ratings was the functional pain-related disability (FPD).

Self-report depression—The Symptom Checklist 90 (SCL-90) was used to evaluate depressive symptoms in all participants during the preceding month. The SCL-90 encompasses 19 cognitive and behavioral symptoms associated with depression, with the severity of each rated on a 0–4 scale; the mean of these ratings is the depression score (SCL-90-D).²³

Self-report sleep quality—The PSQI was used to measure subjective sleep quality in all participants during the preceding month. The PSQI utilizes 18 items to obtain estimates of quality and duration of sleep, sleep onset, frequencies of various sleep-related disturbances and daytime sequelae. It yields 7 “components”, a sum of which is a global score ranging 0–21, where higher values represent more disturbed sleep.¹² A psychometric analysis of the PSQI in TMD patients revealed good inter-component and test-retest reliability, and convergent validity with two sleep-related items from the General Health Questionnaire.²⁴

PSG variables—Two consecutive nocturnal PSG recordings were conducted for each participant on SomnoStarPro acquisition system (Viasys Healthcare, San Diego, CA) using a standard clinical PSG montage with additional electromyography (EMG) leads, for a total of 22 channels. Detailed descriptions of the recording and scoring procedures are reported elsewhere.^{19,21} To minimize the first night effect, PSG data from the second night were used for analysis, except for 9 cases and 1 control whose second night data were not available due to technical failure or attrition.

Based on scored PSG data, the following objective measures of sleep architecture and continuity were used: total sleep time (TST, in minutes), wake after sleep onset (WASO, in minutes), sleep efficiency (SEF = total sleep time / time in bed * 100%), sleep onset latency (SOL, time from lights out to sleep onset, in minutes), number of awakenings (AWAKE), number of sleep stage shifts into stage N1 from any other stage of sleep (N1Shift), percentages of sleep stages N1, N2, N3, and REM sleep (N1%, N2%, N3% and REM%, respectively; time in a given sleep stage / TST * 100%), REM sleep latency (REMLat, time from sleep onset to REM sleep onset, in minutes), and index values (number of respective events per hour of sleep) for the following events: total arousals (TotAr, transient encephalographic changes towards wakefulness during sleep), spontaneous arousals

(SponAr), respiratory arousals (RespAr, combined arousals associated with apneas, hypopneas and RERAs), RERAs, apneas and hypopneas (AHI), combined apneas, hypopneas and RERAs (RDI), periodic limb movements (PLM) and PLM-related arousals (PLMAr).

Data analysis

Group differences on the PSQI and the SCL-90-D were evaluated with independent samples t-tests. The PSQI scores were modeled in a hierarchical linear regression that first entered case-control status, then a stepwise selection of significant PSG variables, followed by entry of the SCL-90-D. Another regression model was fit for TMD cases only, predicting the PSQI based on a stepwise selection of CPI and FPD, a stepwise entry of PSG variables, and the SCL90-D. The alpha level for all analyses was set at 0.05. All analysis used IBM SPSS (v.22; IBM Corp., Armonk, NY).

Results

On average, TMD participants suffered from pain for over 10 years. The mean pain intensity was moderate (CPI mean = 5.2 ± 1.7), while the functional effect of pain was mild, with a positive skew (FPD mean = 1.7 ± 2.2 , median = 0.7).

Compared to controls, TMD cases reported significant, albeit mild, elevations in subjective sleep disturbance (the PSQI: mean_{cases} = 6.0 ± 3.5 vs. mean_{controls} = 3.5 ± 2.5 , $df = 110$, $t = 5.2$, $P < .001$) and depressive symptomatology (the SCL-90-D: mean_{cases} = 1.00 ± 0.78 vs. mean_{controls} = 0.34 ± 0.33 , $df = 165$, $t = 7.7$, $P < .001$). Case-control comparisons on PSG measures have been reported earlier.²³

PSQI scores were modeled as a function of case-control status, characteristics of the PSG, and depressive symptomatology (Table 1). Zero-order correlations showed significant relationships between PSQ scores and case-control status, many parameters from the PSG study, and SCL-90-D scores. In Model 1, TMD status accounted for about 11% of variance in the PSQI, or average scores that were about 2.5 units higher among cases. In Model 2, TMD effect was retained when AWAKE, the strongest PSG predictor of PSQI scores, was entered. Model 2 shows that numerous zero-order PSG-PSQI relationships could be captured by the variance represented by AWAKE, and the contribution of awakenings to subjective sleep quality overlaps with other PSG measures in all participants. When SCL-90-D scores were entered in Model 3, case-control status effect, diminished to 0.7 units, was no longer significant. However, there was little change in the adjusted AWAKE-PSQI relationship, indicating that awakenings are related to the PSQI in a different way than depressive symptoms. Depressive symptoms accounted for 26% ($P < 0.001$), and AWAKE accounted for 2% ($P = 0.03$) of the variance in PSQI responses. This is further illustrated in Figure 1, which shows that PSQI scores remain associated with SCL-90-D scores after adjusting for the effects of the TMD status and AWAKE. Thus, the case-control difference in PSQI scores can be attributed to their different levels of depressive symptoms; the remaining explained variability in PSQI scores can be attributed to awakenings regardless of the case-control status.

Among the TMD cases (Table 2), Model 1 showed collinearity between the FPD and CPI scores; CPI was the stronger predictor, accounting for about 9% of the PSQI variance, and was associated with about a 0.6 point increase in PSQI scores for every 1 point increase in pain ratings. Several PSG measures had zero-order correlations with PSQI scores; however, in Model 2 they could all be captured by REM latency, where PSQI scores increased 0.1 units for every 10 min increase in REM latency, independently of the CPI effect. In Model 3, the SCL-90-D accounted for 24% ($P < 0.001$) of the PSQI variance, and REM latency, for 3% ($P = 0.01$), while the CPI effect was no longer significant. This is further illustrated in Figure 2, which shows that PSQI scores remain associated with SCL-90-D scores after adjusting for the effects of the CPI and REM latency. Identical results were obtained when using a modified SCL-90-D scoring without its three sleep-related items and a modified PSQI scoring without one pain-related item, eliminating “structural confounding” in the response scales. Together, these analyses suggest that depressive symptoms covary with pain ratings and provide better explanation than pain for reports of disturbed sleep.

Discussion

A sample of women with myofascial TMD was evaluated for pain intensity and functional effects, depressive symptomatology using the SCL-90, objective sleep parameters using 2-night PSG recordings, and subjective sleep quality using the PSQI. In this largest PSG-evaluated TMD sample to date, moderate levels of chronic myofascial pain intensity were consistent with levels reported in previous studies.^{2–8,10,11,15} Reports of sleep quality were significantly worse among cases than in demographically matched controls, and there was little concordance between PSQI scores and PSG indicators. As hypothesized, the results showed that reports of poor sleep quality were better accounted for by symptoms of depression than by pain or objective sleep measures.

Depressive symptoms were significantly higher in TMD cases than controls, and the relationship between the PSQI and TMD status disappeared when depressive symptomatology was taken into account. Similarly, among TMD cases the association between pain intensity and sleep ratings disappeared when depressive symptomatology was taken into account. This suggests that depressed mood, which contains effects related to pain intensity, provides the better explanation for subjective ratings of poor sleep. From multiple standard PSG parameters, only two measures accounted for a small portion of additional variance in the PSQI (Tables 1 and 2): awakenings in all participants, and REM latency among cases. As previously reported for this sample, the frequency of awakenings was not statistically different between TMD cases and control, and the mean REM sleep latency in TMD cases, albeit longer, remained within normal limits.¹⁹ In the present analysis, depressive symptomatology, elevated in TMD cases relative to controls, explained by far the largest portion of the variance in self-reported sleep quality, approximately 25% (Tables 1 and 2, Figures 1 and 2).

The PSQI, a psychometrically sound self-report sleep measure widely used in TMD population,²⁴ failed to show much of a relationship with PSG measures in the present study. The general paucity of PSQI-PSG relationships in all participants is consistent with studies examining PSQI scores in relation to objective sleep measures in non-pain samples.^{13,14}

Taken together, these data do not support inferences of disturbed polysomnographic sleep (e.g., altered sleep stage distribution or duration) on the basis of PSQI data alone, although wider-scale PSG evaluations of TMD patients would be needed to corroborate this conclusion.

The lack of PSG evidence of disturbed sleep notwithstanding, the pattern presented by TMD cases – poor subjective sleep largely explained by depressive symptomatology and accompanied by relatively unimpaired objective sleep – suggests a subtype of chronic insomnia. According to clinical guidelines, the diagnosis of primary insomnia is based mainly on subjective reports of poor sleep and adverse daytime consequences; objective PSG findings are not required, and the likelihood of psychological and psychosomatic symptoms is increased.²⁵ A meta-analysis of subjective and objective measures in primary insomnia revealed a disproportionate increase of PSQI-reported disturbance relative to PSG findings.²⁶ A previous evaluation of TMD patients reported largely normal PSG findings in the context of elevated prevalence of primary insomnia diagnosis.^{17,18} The present study provides further evidence for subjectively poor sleep without clear objective findings in TMD cases, a pattern consistent with chronic insomnia. Therefore, while subjective reports of sleep disturbance in TMD should be taken with caution, clinical attention to the possibility of insomnia may still be warranted.

A strong relationship of self-reported poor sleep with depressive symptomatology but not objective PSG findings in the present sample is consistent with previous studies linking psychological symptoms and self-reported sleep in participants with and without TMD^{2-4,6,7,9,13,14} However, the mean SCL-90 depression score was only slightly elevated in TMD cases from the clinical viewpoint. Moreover, major depressive disorder is characterized by key PSG features, including reduced sleep efficiency, reduced stage N3 sleep, and abbreviated REM sleep latency,²⁷ which were absent in the present TMD sample.¹⁹ In fact, TMD cases demonstrated a tendency towards longer REM latency,¹⁹ which presently was also associated with a modest increase in the PSQI. These findings suggest that self-reported depressive symptomatology in TMD may indicate a negative cognitive bias, perhaps related to chronic pain, rather than endogenous depression. Consistently, negative cognition may link depressive symptomatology with elevated PSQI scores in a non-pain population.¹⁴

A large longitudinal study assessing risk factors for the development of TMD revealed that the elevated PSQI and the subsequent reduction in subjective sleep quality predict future onset of TMD independently of stress or comorbid conditions.²⁸ However, perceived stress was shown to mediate the link between the PSQI and TMD onset, while the PSQI did not mediate the role of stress in TMD development.²⁹ Similarly, our findings underscore the importance of psychological variables in the PSQI-TMD relationship and suggest that differences in the baseline PSQI and subsequent sleep ratings preceding the TMD onset^{28,29} may not reflect differences in PSG-measured sleep parameters but represent negative cognitive bias and depressive symptomatology.

Limitations of the present study include selection of only women, the absence of clinical assessment for sleep and mood disorders, the absence of more subtle polysomnographic

measures of sleep disturbance, such as spectral power analysis of EEG, and the retrospective nature of subjective pain and sleep-related measures. A prospective study of TMD patients indicates that reported symptoms of disturbed sleep appear to be better predictors of later pain experience than vice versa.³⁰ Therefore, further research, with prospective measurements of pain, sleep, and psychological variables as well as more detailed analysis of physiological sleep data, may provide better understanding of the relationship and causal connections between sleep, pain, and depressive symptomatology in myofascial TMD patients. Employing multiple PSG assessments over time and matching for depressive symptomatology may be useful tools in future studies.

In sum, this paper demonstrates that the increase in self-reported sleep problems in women with myofascial TMD is better attributed to depressive symptom reporting than to pain intensity or objective measures of sleep. These results caution against the assumption that myofascial pain is associated with an important change in more objective quantitative sleep outcome, as well as against the conclusion that self-report sleep measures accurately estimate the degree of objective sleep disturbance in TMD patients.

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Figure 1.

The Pittsburgh Sleep Quality Index (PSQI) global scores, adjusted for the effects of the case-control status and the number of awakenings on PSG, as a function of the depression scores on The Symptom Checklist 90 (SCL-90 Depression) in all participants (n=170). PSQI scores range 0–21, with higher scores representing greater sleep disturbance. SCL-90 Depression scores range 0–4, with higher scored representing more symptoms of depression.

Figure 2.

The Pittsburgh Sleep Quality Index (PSQI) global scores, adjusted for the effects of the characteristic pain intensity (CPI) and REM sleep latency on PSG, as a function of the depression scores on The Symptom Checklist 90 (SCL-90 Depression) in TMD participants only (n=124). PSQI scores range 0–21, with higher scores representing greater sleep disturbance. SCL-90 Depression scores range 0–4, with higher scored representing more symptoms of depression.

Table 1

Results of a hierarchical regression predicting global PSQI based on TMD status (124 cases and 46 controls), PSG variables and SCL-90-D^a

Predictor Variables	Zero-order Correlations		Model 1 predictors: TMD			Model 2 predictors: TMD, PSG			Model 3 predictors: TMD, PSG, SCL-90-D		
	r	p ^c	β (SE) ^d	p ^c	R ^{2d}	β (SE) ^d	p ^c	R ^{2d}	β (SE) ^d	p ^c	R ^{2d}
TMD status	0.33	<0.001	2.53 (0.56)	<0.001	0.11	2.26 (0.55)	<0.001	0.08	0.68 (0.50)	0.17	0.007
PSG ^b											
TST	-0.02	0.41					0.45			0.58	
WASO	0.22	0.002					0.24			0.07	
SEF	-0.16	0.02					0.68			0.45	
SOL	-0.02	0.43					0.61			0.24	
N1Shift	0.16	0.02					0.54			0.15	
AWAKE	0.26	<0.001				0.07 (0.02)	0.004	0.04	0.04 (0.02)	0.03	0.02
N1%	0.19	0.006					0.80			0.28	
N2%	0.07	0.20					0.54			0.29	
N3%	-0.16	0.02					0.34			0.35	
REM%	-0.09	0.12					0.87			0.86	
REMLat	0.23	0.001					0.06			0.13	
TotAr	0.13	0.05					0.98			0.33	
SponAr	-0.02	0.40					0.20			0.09	
RespAr	0.18	0.008					0.32			0.61	
RERA	0.20	0.005					0.17			0.36	
AHI	0.14	0.04					0.39			0.44	
RDI	0.21	0.003					0.17			0.28	
PLM	0.08	0.16					0.31			0.95	
PLMAr	0.06	0.20					0.41			0.83	
SCL-90-D	0.62	<0.001							2.56 (0.30)	<0.001	0.26

^aTMD status only was entered first, all PSG variables were entered second in a stepwise fashion, SCL-90-D was entered last.

^bPSG variable abbreviations: TST – total sleep time; WASO – wake after sleep onset; SEF – sleep efficiency; SOL – sleep onset latency; N1Shift – # of sleep stage shifts into N1; AWAKE – # of awakenings; N1%, N2%, N3% and REM% – percentages of respective sleep stages in a total sleep time; REMLat – REM sleep latency; TotAr – total arousal index; SponAr – spontaneous arousal index;

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RespAr – respiratory arousal index (includes arousals associated with apneas, hypopneas and RERA events); RERA – respiratory effort related arousal index; AHI – apnea-hypopnea index; RDI – respiratory disturbance index (includes apneas, hypopneas and RERA events); PLM – periodic limb movement index; PLMAR – PLM-arousal index;

^cP values significant at $\alpha=0.05$ are in bold.

^dValues are shown only for variables included in the respective model.

Table 2

Results of a hierarchical regression predicting global PSQI in TMD cases (n=121^a) only based on pain variables, PSG variables and SCL-90-D^b

Predictor Variables	Zero-order Correlations		Model 1 predictors: Pain			Model 2 predictors: Pain, PSG			Model 3 predictors: Pain, PSG, SCL-90-D		
	r	p ^c	β (SE) ^d	p ^c	R ^{2d}	β (SE) ^d	p ^c	R ^{2d}	β (SE) ^d	p ^c	R ^{2d}
Pain											
CPI	0.29	0.001	0.59 (0.18)	0.001	0.09	0.63 (0.17)	< 0.001	0.10	0.30 (0.15)	0.06	0.02
FPD	0.22	0.007		0.26			0.20			0.26	
PSG ^b											
TST	0.07	0.22					0.22			0.43	
WASO	0.18	0.03					0.49			0.11	
SEF	-0.10	0.15					0.99			0.60	
SOL	-0.06	0.27					0.67			0.28	
N1Shift	0.11	0.12					0.74			0.73	
AWAKE	0.19	0.02					0.33			0.48	
N1%	0.17	0.03					0.48			0.95	
N2%	0.06	0.26					0.99			0.68	
N3%	-0.17	0.03					0.30			0.29	
REM%	-0.05	0.28					0.54			0.43	
REMLat	0.24	0.004				0.014 (0.005)	0.003	0.07	0.01 (0.004)	0.01	0.03
TotAr	0.09	0.15					0.93			0.51	
SponAr	0.10	0.46					0.79			0.78	
RespAr	0.11	0.12					0.71			0.74	
RERA	0.14	0.06					0.92			0.88	
AHI	0.09	0.17					0.88			0.80	
RDI	0.14	0.06					0.86			0.78	
PLM	0.07	0.22					0.93			0.27	
PLMar	0.07	0.24					0.79			0.75	
SCL-90-D	0.59	< 0.001							2.30 (0.34)	< 0.001	0.24

^a Only 121 cases were used for these analyses, as 3 TMD cases had FPD scores missing.

^bPain variables, CPI (characteristic pain intensity) and FPD (functional pain-related disability), were entered first in a stepwise fashion, all PSG variables were entered second in a stepwise fashion, SCL-90-R was entered last.

^cPSG variable abbreviations: TST – total sleep time; WASO – wake after sleep onset; SEF – sleep efficiency; SOL – sleep onset latency; N1Shift – # of sleep stage shifts into N1; AWAKE – # of awakenings; N1%, N2%, N3% and REM% – percentages of respective sleep stages in a total sleep time; REMLat – REM sleep latency; TotAr – total arousal index; SponAr – spontaneous arousal index; RespAr – respiratory arousal index (includes arousals associated with apneas, hypopneas and RERA events); RERA – respiratory effort related arousal index; AHI – apnea-hypopnea index; RDI – respiratory disturbance index (includes apneas, hypopneas and RERA events); PLM – periodic limb movement index; PLMAR – PLM-arousal index;

^dp values significant at $\alpha=0.05$ are in bold.

^eValues are shown only for variables included in the respective model.