**Annals Journal of Dental and Medical Assisting**
2025, Volume 5, Issue 1, Page No: 72-86

Available online at: www.tsdp.net

**ISSN: 3062-3510**

**Original Article**

# Polysomnographic Evaluation of Sleep Quality in Patients Experiencing Orofacial Pain and Headache

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**Received:** 21 December 2024; **Revised:** 28 March 2025; **Accepted:** 02 April 2025

## ABSTRACT

Sleep is an essential biological process that supports survival, tissue repair, cognitive functioning, and memory consolidation. Pain, an inherent part of human experience, often coexists with sleep disturbances. While subjective evaluations are common in sleep research, objective measures such as polysomnography provide critical insight into actual sleep quality. This study aimed to examine the interplay between orofacial pain (OFP), headache (HA), and sleep quality using both self-reported and instrument-based assessments. A secondary goal was to explore whether poor sleep results primarily from pain or is influenced by psychological factors like anxiety, stress, and depression. Participants with OFP and HA from the Outpatient Clinic for Temporomandibular Disorders at Wroclaw Medical University, Poland, underwent one night of video-polysomnography. Complementary questionnaires were used to evaluate sleep patterns, pain severity, headache characteristics, and psycho-emotional status. Objective sleep parameters measured through polysomnography did not show a significant relationship with the intensity of OFP or HA. In contrast, subjective sleep assessments demonstrated a clear association with reported pain levels. Pain severity was also closely linked to elevated depression, anxiety, and stress scores. Psychological factors significantly influence the perception of OFP and HA and may contribute to reduced subjective sleep quality, insomnia, and daytime drowsiness. Effective management of these conditions should include a thorough assessment of emotional well-being, as mood disturbances can profoundly affect symptom severity and response to treatment.

**Keywords:** Psycho-emotional state, Sleep quality, Polysomnography, Orofacial pain, Headache

**How to Cite This Article:** Gobbino M, Zani F, Galofaro V, Auh Q. Polysomnographic Evaluation of Sleep Quality in Patients Experiencing Orofacial Pain and Headache. Ann J Dent Med Assist. 2025;5(1):72-86. https://doi.org/10.51847/Ftdl7vjAVM

## Introduction

Sleep is a vital biological process that supports physical restoration, memory consolidation, and brain function, acting as a cornerstone of human health [1]. This complex neurobehavioral state relies on coordinated activity within the central nervous system (CNS) [1]. Pain, by contrast, serves as both a physiological and emotional alarm signaling bodily harm, strongly shaping human behavior [1]. Orofacial pain (OFP) represents a heterogeneous spectrum of conditions—including dental, musculoskeletal, neurovascular, neuropathic, and mucosal pain [1]—and can be classified based on duration and frequency. Conventional definitions of chronic pain, which describe it as persisting for more than three months, do not fully capture the patterns of OFP and headache (HA). For these conditions, chronicity is better defined as pain occurring on more than 15 days per month and lasting at least four hours per day over the previous three months [2-4].

Disruptions in the systems controlling pain and sleep can profoundly affect overall well-being. Studies

report that 67–88% of individuals with chronic pain experience sleep disturbances [5-7], while insomnia—one of the most prevalent sleep disorders—coexists with chronic pain in at least half of affected patients [8, 9]. Research indicates that poor sleep and pain severity are strongly linked, with psycho-emotional factors such as anxiety, depression, and somatization further influencing this relationship [10–15]. In particular, patients with OFP, including temporomandibular disorder (TMD)-related pain, and HA often exhibit impaired sleep quality alongside cognitive deficits, mood disturbances, immune changes, and additional somatic complaints [1, 2, 9, 16].

Evidence suggests that pain and sleep disturbances interact bidirectionally. Sleep disruption can impair recovery and homeostatic functions, promoting chronic pain development and reducing responsiveness to therapy [17, 18]. Conversely, pain can provoke cortical arousal, making it difficult to fall or stay asleep [19]. While acute pain usually produces immediate, reversible sleep impairment, chronic pain establishes a self-reinforcing cycle in which poor sleep and pain amplify each other over time [20]. Some studies further suggest that in OFP patients, sleep problems may stem less from pain itself and more from coexisting psycho-emotional challenges, such as high stress, anxiety, and depression, which are common among individuals with chronic pain.

Given the central role of emotional health in sleep quality [21, 22], objective measures are crucial. Most prior investigations relied heavily on subjective questionnaires, which may not fully capture true sleep architecture. Polysomnography offers an objective approach to assess sleep patterns accurately. The current study used single-night video-polysomnography alongside questionnaires to evaluate sleep quality in patients with OFP and HA, aiming to determine whether impaired sleep is primarily driven by pain or by accompanying psycho-emotional factors such as stress, anxiety, and depression.

## Material and Methods

*Participants*

This study recruited patients from the Outpatient Clinic for Temporomandibular Disorders at Wroclaw Medical University, Poland, who experienced orofacial pain (OFP), headaches (HA), or self-reported sleep disturbances. Eligible participants were referred for a single-night video-polysomnography at the Clinic of Internal Medicine, Occupational Diseases, Hypertension, and Clinical Oncology. Alongside the sleep assessment, participants completed standardized questionnaires to evaluate their pain, sleep quality, and psycho-emotional status. The cohort consisted of individuals with primary HA—defined as pain that appears without an identifiable underlying pathology, disease, or trauma, including migraine, tension-type headaches, and trigeminal autonomic cephalalgias [3]—and various forms of OFP. OFP categories included myofascial pain localized to masticatory muscles (with or without functional limitations), temporomandibular joint (TMJ) pain occurring either at rest, during jaw movement, or upon palpation, and orofacial pain mimicking primary HA in terms of intensity, duration, and quality, with or without associated HA symptoms but without concurrent headache [4]. The study received ethical approval from the Wroclaw Medical University Committee (approval No. KB-794/2019). All participants provided informed consent, and the study followed the Declaration of Helsinki. It is also registered on ClinicalTrials.gov (identifier NCT04214561).

*Inclusion and exclusion criteria*

Participants were adults (≥18 years) who reported OFP, HA, or sleep disturbances and were willing to participate. Individuals were excluded if they had substance or medication dependence, used drugs affecting neuromuscular function, had severe systemic or psychiatric conditions (including significant cognitive impairments), recorded less than four hours of sleep in polysomnography, or declined participation. Pregnant women, patients diagnosed with sleep apnea, and those using mandibular advancement devices (MAD) or continuous positive airway pressure (CPAP) were also excluded.

*Polysomnography procedure*

Polysomnographic recordings were performed in the Sleep Laboratory at Wroclaw Medical University using the Nox A1s™ system (Nox Medical, Reykjavík, Iceland) over one night, scheduled between 10:00 pm and 6:00 am according to the patient's usual sleep schedule. The study protocol included electroencephalography, electrocardiography, electrooculography, and electromyography from the chin and masseter muscles bilaterally, along with thoracic and abdominal respiratory monitoring, body position tracking, and synchronized audio-video recording. Oxygen saturation and pulse were continuously measured using a NONIN 3150 WristOx 2 pulse oximeter (Nonin Medical Inc., Plymouth, USA). Sleep data were processed and analyzed with Noxturnal™ software (Nox Medical), designed specifically for polysomnographic assessment.

*Pain assessment*

*McGill pain questionnaire*

The McGill Pain Questionnaire (MPQ) assesses pain using 78 descriptive terms, producing a total score between 0 (no pain) and 78 (severe pain). Scores above 5 are generally considered clinically meaningful [21]. The short-form MPQ (SF-MPQ) has been shown to reliably capture the multidimensional aspects of pain while being practical in research settings where time for patient evaluation is limited. It provides insights beyond simple pain intensity, making it suitable for studies requiring more nuanced pain characterization [23, 24].

*Graded chronic pain scale*

The Graded Chronic Pain Scale (GCPS) evaluates chronic pain by combining measures of intensity and functional impact. Pain is categorized into five grades: Grade 0 – no pain over the last six months; Grade I – low-intensity pain (<50) with minimal disability (<3 points); Grade II – high-intensity pain (≥50) with minimal disability (<3 points); Grade III – moderate disability (3–4 points) irrespective of pain intensity; and Grade IV – severe disability (5–6 points) regardless of pain intensity [25]. Research indicates that the one-month GCPS is as reliable, if not more so, than the six-month version for assessing pain intensity, interference, days of disability, and high-impact pain, supporting its validity for both clinical and research purposes [26].

*Headache impact test-6*

The HIT-6 measures the extent to which headaches disrupt daily functioning, with scores ranging from 36 to 78. Scores ≤49 indicate minimal impact, 50–55 slight impact, 56–59 moderate impact, and ≥60 severe impact. This tool has proven reliable and valid for assessing headache-related impairment in both episodic and chronic migraine populations, with strong internal consistency (0.82–0.90) [27].

*Migraine disability assessment*

The MIDAS questionnaire estimates migraine-related disability over the previous three months by summing days lost or impaired due to headache across work, school, household chores, and leisure activities. Disability is classified as: Grade I – minimal (0–5 days), Grade II – mild (6–10 days), Grade III – moderate (11–20 days), and Grade IV – severe (>21 days) [27]. MIDAS has shown good test-retest reliability and correlates well with clinical evaluations of the need for medical care. Its total score aligns closely with diary-based daily records (correlation = 0.63), supporting its validity as a practical measure of migraine-related functional impairment [28].

*Temporomandibular disorder pain screener*

The TMD pain screener identifies individuals likely to have painful temporomandibular disorders, with scores of 3 or higher (out of 7) indicating a positive result. This short assessment demonstrates high sensitivity (99%) and specificity (97%) for detecting pain-related TMD, while also accurately distinguishing non-painful TMJ conditions (specificity 95%) and headaches unrelated to TMD (specificity 96%) [29].

*Subjective assessment of sleep quality*

*Insomnia severity index*

The Insomnia Severity Index (ISI) evaluates the severity of insomnia with scores ranging from 0 to 28. Scores up to 10 are considered normal, 8–14 suggest subthreshold insomnia, 15–21 reflect moderate insomnia, and 22–28 indicate severe insomnia [30].

*Pittsburgh sleep quality index*

The Pittsburgh Sleep Quality Index (PSQI) assesses sleep quality over a one-month period. It consists of 19 items grouped into 7 components, which together produce a global score ranging from 0 to 21. Lower scores indicate better sleep quality, with a total score above 5 signifying poor sleep [31].



*Epworth sleepiness scale*

The Epworth Sleepiness Scale (ESS) is an eight-item questionnaire that measures subjective daytime sleepiness. Scores range from 0 (very unlikely to fall asleep in any situation) to 24 (high likelihood of dozing in all situations). Sleepiness levels are categorized as normal (1–10), mild (11–14), moderate (15–18), or severe (>18) [32].

*Psycho-emotional state assessment*

*Sense of stress questionnaire*

The Sense of Stress Questionnaire (KPS) evaluates different aspects of stress through 27 items, generating an overall stress score. It also provides sub-scores for emotional tension, intrapsychic stress (stress originating from self-reflection), and external stress (stress from social or environmental demands). The questionnaire includes a lie scale to detect response biases [33].

*Perceived stress Scale-10*

The Perceived Stress Scale-10 (PSS-10) quantifies perceived stress levels from 0 to 40. Scores of 0–13 indicate low stress, 14–26 indicate moderate stress, and 27–40 indicate high stress [34].

*Patient health Questionnaire-9*

The PHQ-9 assesses depressive symptoms over the preceding two weeks, with each item scored from 0 to 3. Total scores range from 0 to 27. Scores are interpreted as follows: <5 indicates no depression, 5–9 mild depression, 10–14 moderate depression, 15–19 moderately severe depression, and ≥20 severe depression. A score of 10 or higher signals elevated risk for a depressive episode [35].

*Beck depression inventory*

The Beck Depression Inventory (BDI) is a 21-item self-report instrument assessing key symptoms of depression, including mood, guilt, self-criticism, suicidal thoughts, irritability, social withdrawal, insomnia, fatigue, appetite and weight changes, somatic concerns, and loss of libido. Total scores are interpreted as minimal (0–13), mild (14–19), moderate (20–28), or severe (29–63) depression [36].

*Beck anxiety inventory*

The Beck Anxiety Inventory (BAI) measures anxiety across 21 items, with scores ranging from 0 to 63. Anxiety severity is classified as minimal (0–7), mild (8–15), moderate (16–25), and severe (26–63) [37,38].

*Generalized anxiety Disorder-7*

The GAD-7 questionnaire consists of 7 items assessing generalized anxiety, with total scores ranging from 0 to 21. Cut-off scores categorize anxiety as mild (5), moderate (10), or severe (15) [39].

*Statistical analysis*

Medical history data, questionnaire responses, and polysomnography results were compiled into a database and analyzed statistically. Because the data did not follow a normal distribution, the non-parametric Kendall's Tau correlation coefficient was used to examine relationships between variables. Correlations were considered statistically significant at a p-value below 0.05. Sample size estimation was performed using the power.cor function from the genefu package (https://rdrr.io/bioc/genefu/man/power.cor.html), based on an anticipated correlation coefficient of 0.7 and a desired confidence interval width of 0.05. This calculation indicated that 22 participants would be sufficient to detect significant correlations. Given that 114 patients were included in the study, the sample was considered adequate for identifying differences and relationships among the assessed parameters.

## Results

*Study sample*

The study enrolled 114 Caucasian adults, including 72 women and 42 men, ranging in age from 21 to 71 years, with a mean age of 37.67 years.

*Relationship between pain, headache, and subjective sleep quality*

Pain severity was evaluated using four distinct questionnaires **(Table 1)**. Based on the Graded Chronic Pain Scale (GCPS), 64 participants experienced low-intensity pain with minimal or no disability (Grade I). Two participants reported high-intensity pain without disability (Grade IIa), while 15 reported high-intensity pain with low disability (Grade IIb). Eleven participants described high-intensity pain causing moderately limiting disability (Grade III), and two reported severe pain with substantial disability (Grade IV). The remaining participants did not report any pain.



**Table 1.** Questionnaire results on pain severity, impact of pain on daily activities, perceived disability due to pain, sleep quality, insomnia, and daytime sleepiness reported by the participants

| Questionnaire | Result | Subjects n (%) |
|---|---|---|
| GCPS | Grade 0 | 20 (17.54) |
| | Grade I | 64 (56.14) |
| | Grade IIa | 2 (1.76) |
| | Grade IIb | 15 (13.16) |
| | Grade III | 11 (9.64) |
| | Grade IV | 2 (1.76) |
| HIT-6 | no or little impact | 48 (44.44) |
| | slight impact | 14 (12.96) |
| | significant impact | 11 (10.19) |
| | severe impact | 35 (32.41) |
| MIDAS | Grade I | 52 (52.00) |
| | Grade II | 10 (10.00) |
| | Grade III | 15 (15.00) |
| | Grade IV | 23 (23.00) |

| | | |
|---|---|---|
| SF-MPQ | ≤5 | 74 (64.91) |
| | >5 | 40 (35.09) |
| TMD pain screener | ≤3 | 47 (53.41) |
| | >3 | 41 (46.59) |
| PSQI | ≤5 | 36 (33.03) |
| | >5 | 73 (66.97) |
| ISI | normal | 29 (32.59) |
| | subthreshold insomnia | 33 (37.07) |
| | significant moderate insomnia | 21 (23.60) |
| | severe insomnia | 6 (6.74) |
| ESS | normal | 61 (69.31) |
| | mild daytime sleepiness | 18 (20.46) |
| | average daytime sleepiness | 7 (7.96) |

GCPS – Graded Chronic Pain Scale; HIT-6 – Headache Impact Test-6; MIDAS – Migraine Disability Assessment; SF-MPQ – short-form McGill Pain Questionnaire; TMD – temporomandibular disorders; PSQI – Pittsburgh Sleep Quality Index; ISI – Insomnia Severity Index; ESS – Epworth Sleepiness Scale.

The HIT-6 questionnaire evaluated how headaches (HA) affected participants' performance in daily contexts, including work, school, household duties, and social interactions. According to the results, 48 individuals reported that headaches had little or no effect on their routine activities, 14 noted a slight impact, 11 experienced a considerable impact, and 35 reported that headaches had a pronounced and severe influence on their everyday functioning.

Headache-related disability was further assessed using the MIDAS scale, which categorizes respondents into four groups according to the number of days their usual activities were restricted due to HA. Of the total sample, 52 participants were classified as having little or no disability (Grade I), 10 exhibited mild disability (Grade II), 15 reported moderate disability (Grade III), and 23 experienced severe disability (Grade IV). In the SF-MPQ assessment, 40 individuals reported clinically significant pain levels, while the remainder either experienced no pain or their complaints were deemed not clinically meaningful. Similarly, results from the TMD pain screener revealed that 41 participants demonstrated clinically significant temporomandibular disorder (TMD)-related pain.

In terms of sleep evaluation, several instruments were utilized. Based on the PSQI, 73 participants met the criteria indicating poor sleep quality, whereas 36 were categorized as having normal sleep. Findings from the ISI showed that 29 individuals exhibited normal sleep patterns, 33 presented with subthreshold insomnia, 21 experienced moderate insomnia, and 6 suffered from severe insomnia. According to the ESS, 61 respondents displayed no sleepiness, 18 showed mild daytime sleepiness, 7 reported moderate levels, and 2 experienced severe daytime drowsiness. These results are summarized in **Table 1**.

Statistical analysis using Kendall's Tau correlation revealed a significant link between increased self-reported pain severity and poorer subjective sleep quality. Higher pain intensity and pain-related disability, as determined by the GCPS, were associated with lower sleep quality according to the PSQI and ISI scores. The worsening of sleep quality measured by the ISI and PSQI corresponded significantly with greater pain intensity reported in the SF-MPQ. Participants with positive TMD pain screener scores also demonstrated markedly poorer sleep quality across both ISI and PSQI indices. Furthermore, higher headache impact scores from the HIT-6 questionnaire were significantly correlated with deteriorated sleep quality in the PSQI, ISI, and ESS assessments. Comparable associations were observed between MIDAS scores and all three sleep-related measures (PSQI, ISI, and ESS). Collectively, these results underscore that heightened pain severity and associated disability contribute substantially to diminished subjective sleep quality. **Table 2** presents an overview of these relationships between reported pain levels and perceived sleep disturbances.



**Table 2.** Presence of subjective sleep disturbances based on the increase in pain reported by the participants

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| GCPS & PSQI global score | 110 | 0.159381 | 2.468092 | 0.014* |
| GCPS & ISI | 87 | 0.204041 | 2.798379 | 0.005* |
| GCPS & ESS | 87 | −0.007137 | −0.097879 | 0.922 |
| HIT-6 & PSQI global score | 108 | 0.197245 | 3.025658 | 0.002* |
| HIT-6 & ISI | 88 | 0.343241 | 4.735509 | <0.001* |

| | | | | |
|---|---|---|---|---|
| HIT-6 & ESS | 85 | 0.254950 | 3.454555 | 0.001* |
| MIDAS & PSQI global score | 101 | 0.248177 | 3.677424 | <0.001* |
| MIDAS & ISI | 82 | 0.410572 | 5.460118 | <0.001* |
| MIDAS & ESS | 81 | 0.164313 | 2.171236 | 0.030* |
| SF-MPQ & PSQI global score | 114 | 0.290926 | 4.588840 | <0.001* |
| SF-MPQ & ISI | 89 | 0.359480 | 4.988756 | <0.001* |
| SF-MPQ & ESS | 88 | 0.138921 | 1.916616 | 0.055 |
| TMD pain screener & PSQI global score | 88 | 0.228828 | 3.157022 | 0.002* |
| TMD pain screener & ISI | 66 | 0.272170 | 3.230833 | 0.001* |
| TMD pain screener & ESS | 87 | 0.042816 | 0.587212 | 0.557 |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).

*Relationship between pain, headache, and polysomnographic sleep evaluation*

An objective analysis of sleep quality was carried out in a specialized sleep laboratory, where all participants underwent overnight polysomnography. This examination provided detailed recordings of physiological variables essential for assessing sleep structure and efficiency. The subsequent statistical evaluation included several core parameters: total sleep time (TST), denoting the overall duration of sleep in minutes; wake after sleep onset (WASO), reflecting the amount of time spent awake following the initial onset of sleep; sleep latency (SL), defined as the period between lights-out and the beginning of stable sleep—identified as the first instance of non-rapid eye movement (NREM) stage 2; and sleep efficiency (SE), determined by dividing TST by the total time spent in bed (TBT) and multiplying by 100%. The outcomes derived from the polysomnographic analyses are detailed in **Table 3**.

**Table 3.** Results of polysomnographic examination

| Parameter | M | Me | Minimum | Maximum | Lower quartile | Upper quartile | Quartile deviation | SD |
|---|---|---|---|---|---|---|---|---|
| TST | 434.0883 | 449.0000 | 145.4000 | 530.5000 | 399.5000 | 481.2000 | 81.70000 | 68.62323 |
| WASO | 43.6350 | 29.5000 | 0.5000 | 171.0000 | 17.5000 | 57.0000 | 39.50000 | 38.13132 |
| SL | 19.0922 | 14.9000 | 0.3000 | 68.1000 | 6.6000 | 25.4000 | 18.80000 | 16.06708 |
| SE | 86.2447 | 88.1000 | 59.1000 | 97.9000 | 80.6000 | 93.4000 | 12.80000 | 8.75870 |

TST – total sleep time; WASO – wake after sleep onset; SL – sleep latency; SE – sleep efficiency; M – mean; Me – median; SD – standard deviation.



The findings from the Kendall's Tau analysis were surprising, as polysomnography-based measures of sleep quality did not reveal a statistically significant association with higher levels of reported pain or pain-related disability. The only exception was participants with scores above the threshold on the TMD pain screener, who exhibited a decrease in the WASO parameter during polysomnography. **Table 4** summarizes the relationships between increased pain reported in questionnaires and the corresponding polysomnographic parameters.

**Table 4.** Relationship between pain complaints reported in questionnaire studies and selected parameters of sleep quality assessed in polysomnography

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| TMD pain screener & TST | 80 | −0.019701485 | −0.25865555 | 0.796 |
| TMD pain screener & WASO | 80 | −0.1873126 | −2.45917716 | 0.014* |
| TMD pain screener & SL | 80 | 0.0706424102 | 0.927445358 | 0.354 |
| TMD pain screener & SE | 80 | 0.0693498556 | 0.910475753 | 0.363 |
| HIT-6 & TST | 98 | 0.0455309984 | 0.664223607 | 0.507 |
| HIT-6 & WASO | 98 | −0.102724483 | −1.49858402 | 0.134 |
| HIT-6 & SL | 98 | −0.035229518 | −0.51394168 | 0.607 |
| HIT-6 & SE | 98 | 0.0281436189 | 0.41056987 | 0.681 |
| MIDAS & TST | 92 | 0.114810466 | 1.62095682 | 0.105 |
| MIDAS & WASO | 92 | −0.094433626 | −1.33326548 | 0.182 |
| MIDAS & SL | 92 | 0.0688945252 | 0.972690504 | 0.331 |
| MIDAS & SE | 92 | 0.0321546277 | 0.453976581 | 0.650 |
| GCPS & TST | 100 | 0.0637825268 | 0.940261478 | 0.347 |
| GCPS & WASO | 100 | −0.060060854 | −0.88539778 | 0.376 |

| | | | | |
|---|---|---|---|---|
| GCPS & SL | 100 | 0.052715005 | 0.777107635 | 0.437 |
| GCPS & SE | 100 | −0.016318200 | −0.24055766 | 0.810 |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).

A statistical evaluation was carried out to investigate the association between sleep latency (SL) and sleep efficiency (SE) obtained from polysomnography. In the PSQI questionnaire, these metrics corresponded to two components: C2, which tracks the time taken to fall asleep, including nights when SL exceeded 30 minutes, and C4, which reflects the proportion of total sleep time (TST) relative to total time in bed (TBT). **Table 5** presents the results of this comparison between SL and SE.

**Table 5.** Comparison of sleep latency and sleep efficiency in questionnaire studies and polysomnographic examination

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| TMD pain screener & SL | 80 | 0.070642 | 0.927445 | 0.354 |
| TMD pain screener & SE | 80 | 0.069349 | 0.910475 | 0.363 |
| TMD pain screener & PSQI SL | 85 | 0.183346 | 2.48432 | 0.013* |
| TMD pain screener & PSQI habitual SE | 85 | 0.164382 | 2.22736 | 0.026* |
| HIT-6 & SL | 98 | −0.035229 | −0.51394 | 0.607 |
| HIT-6 & SE | 98 | 0.0281436 | 0.410569 | 0.681 |
| HIT-6 & PSQI SL | 107 | 0.053866 | 0.82232 | 0.411 |
| HIT-6 & PSQI habitual SE | 107 | 0.173293 | 2.64551 | 0.008* |
| MIDAS & SL | 92 | 0.068894 | 0.97269 | 0.331 |
| MIDAS & SE | 92 | 0.032154 | 0.45397 | 0.650 |
| MIDAS & PSQI SL | 100 | 0.166755 | 2.45825 | 0.014* |
| MIDAS & PSQI habitual SE | 100 | 0.211255 | 3.11425 | 0.002* |
| GCPS & SL | 100 | 0.052715 | 0.77710 | 0.437 |
| GCPS & SE | 100 | −0.016318 | −0.24055 | 0.810 |
| GCPS & PSQI SL | 107 | 0.019255 | 0.29394 | 0.769 |
| GCPS & PSQI habitual SE | 107 | 0.109976 | 1.67891 | 0.093 |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).



**Table 6** summarizes the associations between polysomnography-derived sleep parameters and participants' self-reported sleep quality from questionnaire assessments. The absence of significant correlations indicates that subjective perceptions of sleep are affected by factors beyond pain, since pain levels alone did not correspond with the presence of sleep disturbances observed in the polysomnographic recordings.

**Table 6.** Comparison of sleep quality parameters derived from polysomnography with those obtained in questionnaire studies

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| TST & ISI | 81 | 0.091348 | 1.20708 | 0.227 |
| TST & ESS | 81 | 0.074740 | 0.98762 | 0.323 |
| TST & PSQI global score | 103 | 0.101410 | 1.51797 | 0.129 |
| WASO & ISI | 81 | 0.021504 | 0.28415 | 0.776 |
| WASO & ESS | 81 | −0.110765 | −1.46365 | 0.143 |
| WASO & PSQI global score | 103 | 0.008127 | 0.12165 | 0.903 |
| SL & ISI | 81 | 0.104323 | 1.37853 | 0.168 |
| SL & ESS | 81 | −0.068686 | −0.90762 | 0.364 |
| SL & PSQI global score | 103 | 0.0257558 | 0.38553 | 0.700 |
| SE & ISI | 81 | −0.039522 | −0.5222 | 0.601 |
| SE & ESS | 81 | 0.1059903 | 1.40056 | 0.161 |
| SE & PSQI global score | 103 | −0.052512 | −0.7860 | 0.432 |
| GCPS & SL | 100 | 0.052715 | 0.77710 | 0.437 |
| GCPS & SE | 100 | −0.016318 | −0.24055 | 0.810 |
| GCPS & PSQI SL | 107 | 0.019255 | 0.29394 | 0.769 |
| GCPS & PSQI habitual SE | 107 | 0.109976 | 1.67891 | 0.093 |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).

*Association between psycho-emotional factors and subjective sleep quality*

The participants' stress levels were assessed using the PSS-10 and KPS scales, depression was evaluated through the BDI and PHQ-9, and anxiety was measured with the GAD-7 and BAI questionnaires. **Table 7** summarizes the number of individuals considered at risk for psycho-emotional disturbances, indicated by heightened stress, anxiety, or depressive symptoms.

**Table 7.** Stress, anxiety and depression levels reported by the participants

| Questionnaire | Result | Subjects n (%) |
|---|---|---|
| PSS-10 | low level of stress | 30 (27.78) |
| | moderate level of stress | 65 (60.19) |
| | high level of stress | 13 (12.04) |
| KPS | STEN 1–4 | 74 (66.67) |
| | STEN 5–6 | 26 (23.42) |
| | STEN 7–10 | 11 (9.91) |
| BDI | no depression | 72 (65.45) |
| | mild depression | 22 (20.00) |
| | moderate depression | 11 (10.00) |
| | severe depression | 5 (4.55) |
| PHQ-9 | no depression | 33 (30.28) |
| | mild depression | 41 (37.61) |
| | moderate depression | 24 (22.02) |
| | moderately severe depression | 8 (7.34) |
| | severe depression | 3 (2.75) |
| GAD-7 | minimal anxiety | 55 (51.89) |
| | mild anxiety | 33 (31.13) |
| | moderate anxiety | 11 (10.38) |
| | severe anxiety | 7 (6.60) |
| BAI | minimal anxiety | 77 (73.33) |
| | moderate anxiety | 16 (15.24) |
| | severe anxiety | 12 (11.43) |

PSS-10 – Perceived Stress Scale-10; KPS – Sense of Stress Questionnaire;
BDI – Beck Depression Inventory; PHQ-9 – Patient Health Questionnaire-9; GAD-7 – Generalized Anxiety Disorder-7; BAI – Beck Anxiety Inventory.

The analysis showed a significant association between the presence and intensity of pain, the resulting disability that limits daily work, family, and social activities, and the participants' psycho-emotional well-being. These findings are summarized in **Table 8**.

**Table 8.** Relationship between the level of anxiety, depression and stress, and pain severity

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| TMD pain screener & PSS-10 | 85 | 0.271585 | 3.679949 | <0.001* |
| TMD pain screener & KPS | 87 | 0.245056 | 3.360890 | 0.001* |
| TMD pain screener & BDI | 86 | 0.280451 | 3.823269 | <0.001* |
| TMD pain screener & PHQ-9 | 86 | 0.327406 | 4.463393 | <0.001* |
| TMD pain screener & BAI | 83 | 0.330526 | 4.423432 | <0.001* |
| TMD pain screener & GAD-7 | 83 | 0.370594 | 4.959670 | <0.001* |
| GCPS & PSS-10 | 106 | 0.273809 | 4.159796 | <0.001* |
| GCPS & KPS | 109 | 0.193104 | 2.976261 | 0.003 |
| GCPS & BDI | 108 | 0.250197 | 3.837918 | <0.001* |
| GCPS & PHQ-9 | 108 | 0.273731 | 4.198926 | <0.001* |
| GCPS & BAI | 104 | 0.302987 | 4.558022 | <0.001* |
| GCPS & GAD-7 | 104 | 0.293885 | 4.421089 | <0.001* |
| HIT-6 & PSS-10 | 108 | 0.226315 | 3.471580 | 0.001* |
| HIT-6 & KPS | 107 | 0.300810 | 4.592199 | <0.001* |
| HIT-6 & BDI | 108 | 0.348665 | 5.348384 | <0.001* |
| HIT-6 & PHQ-9 | 107 | 0.330598 | 5.046953 | <0.001* |

| | | | | |
|---|---|---|---|---|
| HIT-6 & BAI | 104 | 0.319397 | 4.804873 | <0.001* |
| HIT-6 & GAD-7 | 105 | 0.310308 | 4.691280 | <0.001* |
| MIDAS & PSS-10 | 100 | 0.330638 | 4.874154 | <0.001* |
| MIDAS & KPS | 100 | 0.275152 | 4.056203 | <0.001* |
| MIDAS & BDI | 101 | 0.400234 | 5.930566 | <0.001* |
| MIDAS & PHQ-9 | 101 | 0.332793 | 4.931234 | <0.001* |
| MIDAS & BAI | 99 | 0.377552 | 5.536881 | <0.001* |
| MIDAS & GAD-7 | 99 | 0.328361 | 4.815483 | <0.001* |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).

A strong link was found between participants' psycho-emotional condition and their reported sleep problems. Elevated levels of stress, anxiety, and depression were associated with lower subjective sleep quality. Poorer mental-emotional health also corresponded with higher PSQI scores, greater daytime sleepiness on the ESS, and more severe insomnia according to the ISI. Notably, these associations were stronger than those observed between reported pain and perceived sleep quality, indicating that the psycho-emotional state may play a major role in how individuals experience and evaluate their sleep **(Table 9)**.

**Table 9.** Relationship between the level of anxiety, depression and stress, and the subjective assessment of sleep quality

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| PSS-10 & PSQI | 108 | 0.324845 | 4.982985 | <0.001* |
| PSS-10 & ISI | 88 | 0.394190 | 5.438427 | <0.001* |
| PSS-10 & ESS | 85 | 0.212189 | 2.875137 | 0.004* |
| KPS & PSQI | 112 | 0.302435 | 4.727067 | <0.001* |
| KPS & ISI | 88 | 0.339918 | 4.689672 | <0.001* |
| KPS & ESS | 87 | 0.200043 | 2.743542 | 0.006* |
| BDI & PSQI | 110 | 0.405912 | 6.285733 | <0.001* |
| BDI & ISI | 88 | 0.428127 | 5.906635 | <0.001* |
| BDI & ESS | 86 | 0.272479 | 3.714598 | <0.001* |
| PHQ-9 & PSQI | 109 | 0.437751 | 6.746935 | <0.001* |
| PHQ-9 & ISI | 87 | 0.480670 | 6.592274 | <0.001* |
| PHQ-9 & ESS | 86 | 0.341903 | 4.661024 | <0.001* |
| BAI & PSQI | 106 | 0.402993 | 6.122401 | <0.001* |
| BAI & ISI | 85 | 0.409755 | 5.552148 | <0.001* |
| BAI & ESS | 83 | 0.192642 | 2.578127 | 0.010* |
| GAD-7 & PSQI | 106 | 0.355888 | 5.406769 | <0.001* |
| GAD-7 & ISI | 86 | 0.428333 | 5.839290 | <0.001* |
| GAD-7 & ESS | 84 | 0.208219 | 2.804021 | 0.005* |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).



*Association between anxiety, depression, stress, and objective sleep measures*

The influence of psycho-emotional factors on sleep quality, as assessed via polysomnography, appeared to be complex and not consistently clear-cut. Individuals reporting higher stress levels on the KPS showed shorter total sleep time (TST, $p = 0.017$), reduced sleep efficiency (SE, $p = 0.031$), prolonged sleep latency (SL, $p = 0.030$), and increased wake after sleep onset (WASO, $p = 0.028$). However, when stress was measured with the PSS-10, only TST remained significantly affected ($p = 0.049$), and no meaningful associations were observed with WASO ($p = 0.115$), SL ($p = 0.615$), or SE ($p = 0.235$). Examination of the other polysomnographic parameters did not show abnormalities in participants with higher levels of stress, anxiety, or depression. The complete results are provided in **Table 10**.

**Table 10.** Relationship between the level of anxiety, depression and stress, and the objective assessment of sleep quality in polysomnography

| Pair of variables | n | Kendall's Tau | Z | p-value |
|---|---|---|---|---|
| PSS-10 & TST | 98 | 0.135076 | 1.97055 | 0.049* |

| | | | | |
|---|---|---|---|---|
| PSS-10 & WASO | 98 | −0.108172 | −1.57806 | 0.115 |
| PSS-10 & SL | 98 | −0.034472 | −0.50289 | 0.615 |
| PSS-10 & SE | 98 | 0.081371 | 1.18708 | 0.235 |
| KPS & TST | 102 | 0.159898 | 2.38142 | 0.017* |
| KPS & WASO | 102 | −0.147246 | −2.19299 | 0.028* |
| KPS & SL | 102 | −0.145709 | −2.17009 | 0.030* |
| KPS & SE | 102 | 0.1446899 | 2.1549 | 0.031* |
| BDI & TST | 100 | 0.074851 | 1.10343 | 0.270 |
| BDI & WASO | 100 | −0.080055 | −1.18014 | 0.238 |
| BDI & SL | 100 | −0.1006566 | −1.483847 | 0.138 |
| BDI & SE | 100 | 0.08106160 | 1.1949840 | 0.232 |
| PHQ-9 & TST | 99 | 0.072239 | 1.05941 | 0.289 |
| PHQ-9 & WASO | 99 | −0.098424 | −1.44341 | 0.149 |
| PHQ-9 & SL | 99 | 0.0114757 | 0.168293 | 0.866 |
| PHQ-9 & SE | 99 | 0.0490954 | 0.719996 | 0.472 |
| BAI & TST | 96 | 0.106368 | 1.53526 | 0.125 |
| BAI & WASO | 96 | −0.075987 | −1.09676 | 0.273 |
| BAI & SL | 96 | 0.01954793 | 0.282144 | 0.778 |
| BAI & SE | 96 | 0.02843335 | 0.410392 | 0.682 |
| GAD-7 & TST | 98 | 0.125856 | 1.83603 | 0.066 |
| GAD-7 & WASO | 98 | −0.077735 | −1.13403 | 0.257 |
| GAD-7 & SL | 98 | 0.078594 | 1.146563 | 0.252 |
| GAD-7 & SE | 98 | 0.028898 | 0.421578 | 0.673 |

*statistically significant ($p < 0.05$, Kendall's Tau correlation).

## Discussion

This study aimed to examine the association between self-reported orofacial pain (OFP), headache (HA), and sleep quality using both subjective and objective measures, while also investigating whether impaired sleep is solely related to OFP and HA or influenced by coexisting psycho-emotional factors such as stress, anxiety, and depression. Although patients reported poorer sleep quality in questionnaires, the polysomnographic recordings did not reveal significant deviations, despite these subjective complaints. Nevertheless, participants who indicated lower sleep quality also exhibited higher levels of stress, anxiety, and depression.

Previous research has shown that individuals experiencing chronic or severe pain and HA often present with elevated psycho-emotional distress, including stress, anxiety, and depression. These studies emphasize that psychosocial factors play a significant role in both the development and progression of severe pain or HA and can influence treatment outcomes [40-42]. The findings of the present study align with this evidence, underscoring the importance of evaluating the psycho-emotional state in patients with pain. Effective management should not only aim to reduce symptoms but also include psychological or, if necessary, psychiatric interventions to provide comprehensive, multidirectional care.

Yap *et al*. explored the influence of TMD pain severity on sleep quality and examined how different types of TMD diagnoses affected sleep disturbances [43]. Their results indicated that individuals with moderate to severe pain experienced significantly poorer sleep compared with those reporting mild pain. Participants with pain-related and/or intra-articular TMD demonstrated worse sleep than controls without TMD, and those with muscular pain, alone or combined with joint pain, reported significantly worse sleep than individuals with non-painful joint disorders. However, sleep quality in their study was assessed solely using the PSQI [43]. Similarly, Kim and Kim found notable differences in global PSQI scores across three TMD pain diagnosis groups among Korean patients [44]. Other studies have also suggested that diminished sleep quality in TMD patients may negatively affect both treatment outcomes and overall quality of life [45, 46]. These findings differ from the current study, which suggests that pain alone may not fully explain the sleep disturbances reported by patients. Supporting this, Yatani *et al.* showed that reduced sleep quality could also be driven by psychological distress and a low sense of control over life circumstances [47], a conclusion consistent with the present observations.

From a neurobiological perspective, it is essential to consider the interplay between OFP and the neural mechanisms regulating sleep, particularly regarding how pain and sleep disturbances influence each other. Chronic pain is frequently linked to impaired sleep quality, which can, in turn, exacerbate pain, creating a

bidirectional relationship. Consequently, effective management should target both pain reduction and sleep improvement simultaneously [20]. Patients with chronic pain often experience abnormal sleep durations—either too short or too long—alongside depressive symptoms [48, 49]. Other studies have noted that chronic pain may increase susceptibility to insomnia [50], which itself can lower pain tolerance and reduce sleep efficiency [51].

Lavigne and Sessle note that in healthy adults, nociceptive transmission is partially inhibited during normal sleep to preserve sleep continuity, resulting in a higher threshold for responding to painful stimuli during light sleep (N1 and N2) and even more so during deep sleep (N3) [20]. In REM sleep, however, this relationship is less consistent. These mechanisms help ensure that low-intensity stimuli minimally disrupt sleep when sleep occurs under favorable conditions [20]. Thus, while the connection between pain—particularly chronic pain—and sleep quality is evident, protective processes exist that prevent minor ailments from significantly affecting sleep.

Dubrovsky *et al.* conducted a double-night polysomnography study to evaluate sleep quality, using the Symptom Checklist-90 to measure depressive symptoms and the PSQI for subjective sleep assessment [52]. Their results were similar to those observed in the present study: women with myofascial pain who reported poor sleep were more influenced by depressive symptoms than by pain intensity or objective polysomnography measures. The authors cautioned against assuming that myofascial pain alone causes poor sleep quality and emphasized the value of questionnaires like the PSQI for detecting sleep disturbances. They also highlighted the importance of objective assessments via polysomnography, which often do not show deviations from normal patterns, suggesting that reported poor sleep may be driven more by psycho-emotional factors than by pain itself [52]. Conversely, Smith *et al.* found that approximately 36% of patients with TMD pain experienced insomnia, 28% had sleep apnea, and some exhibited mild sleep-disordered breathing, specifically respiratory effort-related arousals [53].

Understanding the interplay between pain, sleep quality, and psycho-emotional state also requires consideration of the hypothalamic–pituitary–adrenal (HPA) axis. Cortisol secretion follows a relatively stable daily rhythm, primarily governed by the circadian clock, and is modestly influenced by sleep. Sleep onset suppresses cortisol release, whereas awakenings and sleep offset stimulate it. During wakefulness, bursts of cortisol secretion are linked to central arousal indices. Sleep deprivation or reduced sleep quality can slightly activate the HPA axis, while abrupt changes in sleep duration can disrupt cortisol's circadian rhythm [54]. The HPA axis is essential for adapting to stress through glucocorticoid release, which is mainly regulated by neural mechanisms involving corticotropin-releasing hormone (CRH) from hypothalamic paraventricular neurons (PVN). CRH activation pathways vary depending on the type of stressor: homeostatic disruptions often trigger direct noradrenergic or peptidergic PVN input via sensory relays, whereas anticipatory responses engage oligosynaptic circuits from limbic structures. These intricate mechanisms require further investigation [54, 55].

Although numerous studies have examined the link between pain and sleep disturbances, only a few have incorporated objective sleep assessments such as polysomnography. In the present study, all participants underwent polysomnography, allowing for a direct comparison between subjective reports of poor sleep and objective sleep parameters. An added advantage of this research was the ability to relate both self-reported pain levels and sleep quality to psycho-emotional measures, offering a comprehensive view of the interconnections among pain, sleep, and emotional state.



## Limitations

This study had several limitations. First, sleep was assessed using a single-night video-polysomnography, which may not fully reflect participants' typical sleep patterns due to the unfamiliar environment of the sleep laboratory, particularly during the first night. Additionally, pain and headache (HA) were evaluated using questionnaires without the inclusion of objective tools to quantify pain severity. Another limitation was that the analysis did not account for potential gender-related differences in the examined relationships.

## Conclusions

Orofacial pain (including myofascial OFP, temporomandibular joint (TMJ) pain, and OFP resembling primary HA) and HA are prevalent and significant health issues. Sleep disturbances are frequently reported among individuals experiencing these conditions, highlighting the importance of considering sleep-related problems, such as insomnia, sleep apnea, and sleep-related hypoxia, in patients with OFP or HA. However, the findings of this study suggest that subjective reports of poor sleep quality may not always correspond with objective

abnormalities detected through polysomnography. Chronic pain is often accompanied by psycho-emotional challenges, including heightened anxiety, depression, and stress, which may underlie perceived sleep disruptions. Thus, evaluating the psycho-emotional state of patients with persistent pain is essential, and professional care should be offered when needed.

Incorporating polysomnography into the diagnostic process can strengthen the accuracy of sleep assessments, as it remains the gold standard for diagnosing sleep disorders. Effective management of patients with OFP or HA should therefore include the identification of comorbidities, careful evaluation of psycho-emotional health, and the use of validated sleep questionnaires. The current study demonstrated that while patient-reported OFP and HA reduced perceived sleep quality, objective measures did not reflect these disturbances, and psycho-emotional factors played a mediating role. Further research is required to clarify the mechanisms underlying the complex interactions between OFP, HA, and sleep.

**Acknowledgments:** None

**Conflict of Interest:** None

**Financial Support:** None

**Ethics Statement:** None

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