The Impact of Cigarette Smoking on Sleep Quality of Patients with Masticatory Myofascial Pain

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Aims: To evaluate the impact of cigarette smoking on the sleep quality of patients with masticatory myofascial pain and to determine whether the association between smoking and impaired sleep is influenced by other factors such as demographic, psychological, and behavioral variables. **Methods:** Data from a retrospective case series of 529 patients diagnosed with masticatory myofascial pain according to group I of the Research Diagnostic Criteria for Temporomandibular Disorders were obtained. Patients completed a standardized pain questionnaire and psychometric tests. Differences between smokers and nonsmokers were evaluated using independent samples *t* tests and chi-square tests. Hierarchical linear multiple regression models were used to examine predictors of sleep disturbances. **Results:** Of the entire sample, 170 (32%) reported that they were smokers. Smokers reported higher pain severity and more sleep disturbances and psychological distress than nonsmokers. Cigarette smoking significantly predicted sleep disturbance ($\beta = 0.229$, $P < .001$), but this relationship was attenuated after controlling for pain severity and psychological distress ($\beta = 0.122$, $P < .001$). **Conclusion:** Cigarette smoking is associated with numerous adverse health outcomes, including pain severity, alterations in mood, and disrupted sleep, and seems to be a significant predictor of sleep quality in patients with masticatory myofascial pain. *J Oral Facial Pain Headache 2015;29:15–23. doi: 10.11607/ofph.1266*

Key words: *masticatory myofascial pain, psychological distress, sleep quality, smoking, temporomandibular disorders*

Cigarette smoking is among the most prevalent causes of morbidity and mortality in the United States. The prevalence of adult cigarette smokers in the United States in 2011 was estimated to be 19.0%.[1] The detrimental effects of cigarette smoking are well documented. It is acknowledged that tobacco use increases the risk for cardiovascular problems, peripheral vascular disease, oral cancer, throat cancer, lung cancer, stroke, and gastrointestinal disorders.[2] Cigarette smokers present a higher incidence of chronic pain, sleep disturbance, anxiety, and depression than nonsmokers.[3–6] On the other hand, cigarette smoking has been documented as a mechanism of coping with pain, reducing emotional distress, and elevating mood.[7,8] It is also recognized that current smokers are often engaged in other negative health behaviors, such as high caffeine and alcohol consumption, which in turn may affect sleep quality.[9–11]

Cigarette smoking has been linked to a wide spectrum of sleep disorders, including insomnia, snoring, obstructive sleep apnea, sleep fragmentation, decreased sleep duration, and daytime sleepiness.[12–16] The mechanisms by which cigarette smoking negatively affects sleep are not fully understood. It has been postulated that nicotine acts centrally, promoting the release of multiple neurotransmitters that are involved in the arousal process responsible for wakefulness.[17,18] Hence, it is suggested that these stimulatory effects of nicotine might contribute to increased sleep latency. Alternatively, nightly nicotine withdrawal experienced by

habitual smokers might explain the fragmented sleep over the course of the night.[16,19] Yet, whether the frequency and duration of cigarette smoking and levels of nicotine dependence influence the magnitude of sleep disturbance is unclear,[20,21] but there is a consensus in the literature that cigarette smoking adversely affects sleep quality and sleep architecture.

Sleep disturbances also can have a negative impact on quality of life and may reduce the sense of well-being, cause alterations in mood, and generate psychological distress. In addition, sleep disturbances have been associated with chronic painful conditions such as fibromyalgia and temporomandibular disorders (TMD).[22–27] It has been demonstrated that smokers among chronic pain patients report greater pain intensity and are more likely to have unfavorable treatment outcomes than nonsmokers who are chronic pain patients.[28–31] Current cigarette smoking is also highly correlated with widespread pain (fibromyalgia),[24,32,33] TMD,[34–39] and other musculoskeletal conditions, functional impairment, and symptom severity.[29,40] Overall, the literature supports a strong negative relationship between chronic pain, sleep disturbances, and current cigarette smoking.

Only a limited number of studies have specifically assessed the relationship between cigarette smoking and TMD.[34–39] Collectively, these studies showed that cigarette smoking is adversely related to pain intensity, psychological function, and sleep quality of TMD patients regardless of the TMD subcategory.

Several studies have explored the relationship between TMD, sleep quality, and sleep architecture.[23,26,39,41–44] These studies showed that the majority of TMD patients presented with sleep disorders. They also showed that the pain inhibitory system measured by changes in diffuse noxious inhibitory control, currently better known as conditioned pain modulation in humans,[45] was less responsive in TMD patients when associated with poor sleep quality.[26] Furthermore, pain severity and psychological factors independently predicted sleep disturbance.[41] In summary, although a mutual association between pain and sleep quality has been demonstrated, there is a paucity of research evaluating the relationship between sleep, cigarette smoking, and TMD.

While cigarette smoking has been indicated as a causative and perpetuating risk factor for sleep disturbances and pain, the question of whether cigarette smoking independently influences the sleep quality of TMD patients remains unanswered. Thus, the purpose of this study was to evaluate the impact of cigarette smoking on the sleep quality of patients with masticatory myofascial pain and to determine whether the association between smoking and impaired sleep is influenced by other factors such as demographic, psychological, and behavioral variables.

Material and Methods

Participants

A retrospective case series based on chart reviews was conducted in the Orofacial Pain Clinic of the College of Dentistry of the University of Kentucky (UK). The study was approved by the UK Institutional Review Board for the Protection of Human Subjects. Informed consent was obtained from all participants on the day of their initial visit to the UK College of Dentistry. Data were collected from 5,494 patients referred to the clinic during the period of 2000 to 2010. Patients with a primary diagnosis of masticatory myofascial pain were included in the analyses. The classification of masticatory myofascial pain was based on the Research Diagnostic Criteria for TMD Axis I (RDC/TMD) group I.[46] Patients with other orofacial pain subcategories as primary or secondary diagnoses were excluded. Participants also met the following criteria: *(1)* pain level at least 3 out of 10 on a numerical rating scale, *(2)* pain duration of at least 3 months, *(3)* at least 18 years of age, and *(4)* absence of other chronic pain conditions such as neurovascular and neuropathic pain. The study excluded any patients who had a history of severe psychiatric disorders, such as schizophrenia and dementia, as well as those who were unable to answer the questionnaires due to cognitive impairment caused by other factors.

The final study sample consisted of 529 myofascial pain patients aged between 18 and 84 years. No data with regard to body mass index (BMI) or medical comorbidities, such as fibromyalgia and sleep apnea, were available. The use of medications that might have positively affected the sleep quality and architecture was reported by 57% of participants: 7% reported use of sleep medication less than once a week, 10% reported use of sleep medication once or twice per week, and 39% reported use of sleep medication three or more times per week. Information about other medication classes that might also have affected sleep quality was not available. Hence, some of these potential contributors masking or accenting sleep disturbance were not accounted for in this study.

Measures

Prior to the clinical examination, each participant completed a battery of psychometric tests that assessed various dimensions of sleep quality as well as psychological characteristics. Patients also completed a general pain questionnaire that asked for demographic variables such as age, gender, occupation, and marital status. This latter questionnaire also encompassed items regarding their habits such as caffeine and alcohol consumption, smoking status, and quantity and duration of tobacco use. Caffeine

and alcohol intake was determined by the number of cups of caffeine-containing beverages and alcoholic drinks consumed per day, respectively. Smoking status was assessed using a self-report question (yes/no). The participants were dichotomized into smokers and nonsmokers. The nonsmokers category included participants who had never smoked or were former smokers.

Sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI), a 19-item self-completion questionnaire using a four-point scale and assessing sleep quality and disturbances for the preceding month. This instrument assesses seven different domains: sleep disturbances, habitual sleep efficiency, sleep latency, sleep duration, subjective sleep quality, use of sleep medications, and daytime dysfunction. Respondents rate each component from "no difficulty" (0) to "severe difficulty" (3) along with five questions rated by a partner or roommate, generating a global score range from 0 to 21, with a higher score indicative of greater sleep impairment. A global score greater than 5 indicates significant sleep disturbance. Therefore, the PSQI total score cutoff of 5 is used to discriminate good from poor sleepers, based on a sensitivity of 89.6% and a specificity of 86.5%. This scale has good internal consistency (alpha = 0.83) and overall test-retest reliability (r = 0.85). The developers reported test-retest reliability ranging from 0.65 (medication use) to 0.84 (sleep latency) among the different components of sleep quality.[47]

Current psychological status was evaluated with the Global Severity Index (GSI) of the Revised Symptom Checklist-90 (SCL-90R), a self-rated multidimensional inventory, assessing the presence and severity of 90 symptoms in the past 7 days.[48] Respondents were asked to indicate how much they were distressed by each issue on a five-point Likert scale, from "not at all" (0) to "extremely" (4). The SCL-90R contains nine subscale scores along primary symptoms dimensions (somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism) and three global indices of distress (GSI, positive symptom distress index, and positive symptom total). In the present study, the only psychological parameter evaluated was the GSI. The GSI represents the average score of the 90 items of the inventory, and it is suggested to be the best single indicator of the current level of overall psychological distress. To compare an individual status with the population norm, raw scores of all dimensions are converted to normalized T scores. An extensive research base supports the reliability and validity of the SCL-90R.[49] A recent study[50] evaluating 118 psychiatric outpatients demonstrated high internal consistency (r = 0.97) of the GSI. Moreover, studies evaluating psychological distress in chronic pain patients have indicated that the GSI is the most reliable composite measure of the SCL-90R when compared to the more specific domains.[51–53]

The TMD symptoms were assessed by means of the aforementioned comprehensive pain questionnaire containing questions about the presence, duration, and severity of joint and/or muscle pain, functional limitation, temporomandibular joint (TMJ) sounds, history of oral parafunctional habits, and history of TMJ locking. The pain severity was assessed through a numerical rating scale from 0 to 10, where 0 represents no pain and 10 the worst pain imaginable. TMD signs were investigated using the RDC/TMD axis I criteria. The clinical examinations were standardized and performed by calibrated examiners.

Statistical Analyses

Chi-square tests and independent sample t tests were used to compare sociodemographic and clinical features of smokers and nonsmokers in the sample. Categorical variables (eg, gender, employment, and marital status) were summarized using frequency and percentages (n, %), whereas continuous variables (eg, age and sleep characteristics) were summarized as mean ± SD values, unless stated differently. Differences between smokers and nonsmokers on the GSI score, PSQI, and other sleep dimensions were also evaluated by means of independent sample t tests. GSI values are represented in standardized normalizing transformations.

Subsequently, multiple linear regression models were applied to assess whether smoking status influenced sleep quality independently of other variables, including age, caffeine use and alcohol intake, and overall psychological distress (GSI). Sleep quality (measured by PSQI total score) was selected as the primary dependent variable. Smoking status was evaluated as the primary predictor of the first regression analysis, followed by the inclusion of age in the second model, pain severity in the third model, psychological characteristics (GSI) in the fourth model, and caffeine and alcohol intake in the fifth model. Beta-weights (β standardized) were examined to determine how much each variable contributed as a predictor of sleep quality. The R-squared (R^2) represents the percentage of variance in the dependent variable accounted for in each model, and ΔR^2 represents the change in R^2 from one model to the next. Due to the higher prevalence of females, all data were computed for females only (n = 472); however, no significant changes in the results were observed (data not shown).

The criterion for level of significance was set at $P < .01$, due to the extensive number of tests that were conducted. P values less than .05 were considered to be statistical trends that could be important

Table 1 Comparison of Self-Report Sociodemographic and Sleep Characteristics Based on Smoking Status

Variables	Smokers (n = 170)	Nonsmokers (n = 359)	Statistic	*df*	*P*
Age (y), mean ± SD	35.25 ± 9.77	38.78 ± 13.62	$t = 3.39$	525	< .01
Females	35.55 ± 9.96	38.20 ± 13.57	$t = 2.37$	468	
Males	33.30 ± 8.35	44.26 ± 13.05	$t = 3.86$	55	
Gender, n (%)			$\chi^2 = 1.97$	1	.43
Females	147 (86.5)	325 (90.5)			
Males	23 (13.5)	34 (9.5)			
Marital status, n (%)			$\chi^2 = 21.74$	3	< .01
Single	24 (14.1)	65 (18.1)			
Married	70 (41.2)	202 (56.3)			
Divorced	35 (20.6)	29 (8.1)			
Widowed	2 (1.2)	8 (2.2)			
Missing	39 (22.9)	55 (15.3)			
Employment status, n (%)			$\chi^2 = 32.67$	6	< .01
Full-time	43 (25.3)	145 (40.4)			
Part-time	12 (7.1)	26 (7.2)			
Unemployed	31 (18.2)	68 (18.9)			
Disabled	39 (22.9)	31 (8.6)			
Retired	3 (1.8)	20 (5.6)			
Student	1 (0.6)	6 (1.7)			
Missing	41 (24.1)	63 (17.5)			
PSQI cutoff, n (%)			$\chi^2 = .43$	1	.51
PSQI < 5	8 (4.7)	22 (6.1)			
PSQI ≥ 5	168 (95.3)	337 (93.9)			
Pain severity, mean ± SD	7.77 ± 1. 80	7.02 ± 1. 91	$t = -4.20$	527	< .01
Pain duration, mean ± SD	57.89 ± 78.12	62.77 ± 79.72	$t = .66$	527	.50
GSI, mean ± SD	65.89 ± 10.14	61.80 ± 9.80	$t = -4.38$	520	< .01
Alcohol intake, mean ± SD	0.13 ± 0.57	0.19 ± 0.79	$t = -.75$	436	.45
Daily caffeine intake, mean ± SD	3.45 ± 2.07	1.71 ± 1.41	$t = -9.88$	522	< .01

Independent sample *t* test (*t*) and chi-square tests (χ^2) were used, $P \leq .01$ was considered statistically significant. PSQI, Pittsburgh Sleep Quality Index; GSI, Global Severity Index.

to consider in future studies but did not meet the established criteria for significance within this particular study. All data analyses were performed using SPSS software, version 20.

Results

Sample Characteristics

The 529 subjects had a mean (± SD) age of 37.63 ± 12.59. Among the participants, 472 (89.2%) were females and 57 (10.8%) were males, and 170 (32.1%) reported to be smokers and 359 (67.9%) reported to be nonsmokers (Table 1). The overall mean pain severity was 7.2 ± 1.93 and the mean pain duration was 61.4 ± 79.3 months.

Smokers were significantly more likely to be younger, disabled, or divorced, and they were less frequently married than nonsmokers (Table 1). Nonsmokers were older and more likely to be employed full time or retired (Table 1). Compared to nonsmokers, smokers exhibited higher pain severity (7.77 ± 1.80 vs 7.02 ± 1.91, $P < .01$), had more disrupted sleep (PSQI total = 12.58 ± 4.48 vs 10.58 ± 4.19, $P < .001$), and consumed more caffeinated products (3.45 ± 2.07 vs 1.71 ± 1.41, $P < .01$; Tables 1 and 2).

With respect to psychological characteristics, 284 (53.6%) participants presented psychological distress based on the GSI of the SCL-90-R (GSI normalized T scores above 63 are considered clinically elevated). The average level of overall psychological distress was significantly higher among smokers (65.9 ± 10.14, $P < .01$) than nonsmokers (61.8 ± 9.80; Table 1).

Associations Between Smoking Status and Sleep Quality

The results of independent *t* tests of different sleep dimensions in smokers versus nonsmokers are presented in Table 2. The PSQI test revealed significant

Table 2 Comparison of Sleep Quality Scores According to Smoking Status

Variables	Smokers (n = 170)	Nonsmokers (n = 359)	*t*	*df*	*P*
Subjective sleep quality	2.11 ± 0.80	1.69 ± 0.84	-5.33	511	< .001*
Sleep latency	2.07 ± 0.96	1.68 ± 1.07	-4.18	514	< .001*
Sleep duration	1.90 ± 1.02	1.35 ± 0.94	-5.84	500	< .001*
Habitual sleep efficiency	1.50 ± 1.25	1.11 ± 1.16	-3.37	481	.01*
Sleep disturbances	2.09 ± 0.65	1.86 ± 0.69	-3.58	512	< .001*
Use of sleep medication	1.56 ± 1.38	1.55 ± 1.38	0.48	503	.96
Daytime dysfunction	1.65 ± 0.83	1.51 ± 0.82	-1.80	508	.07
PSQI total score	12.58 ± 4.48	10.58 ± 4.19	-4.96	516	< .001*

*$P < .01$ was considered statistically significant based on 2-tailed tests. Independent sample *t* test (*t*) and chi-square tests (χ^2) were used. PSQI, Pittsburgh Sleep Quality Index.

Table 3 Hierarchical Regression Models Examining Prediction of Sleep Disturbances

Predicting variable	β (standardized)	*P*	*R*	R^2	Δ R^2
Cigarette smoking	0.229	< .001*			
Model 1			0.23	0.05	–
Cigarette smoking	0.242	< .001*			
Age	0.126	< .001*			
Model 2			0.26	0.07	0.02
Cigarette smoking	0.193	< .001*			
Age	0.127	< .001*			
Pain severity	0.247	< .001*			
Model 3			0.36	0.13	0.06
Cigarette smoking	0.108	.01*			
Age	0.103	.01*			
Pain severity	0.151	< .001*			
GSI	0.436	< .001*			
Model 4			0.54	0.30	0.17
Cigarette smoking	0.122	.01*			
Age	0.107	.01*			
Pain severity	0.148	< .001*			
GSI	0.438	< .001*			
Caffeine	-0.028	.55			
Alcoholic drinks/day	-0.019	.65			
Model 5			0.54	0.30	0.00

*Indicates statistical significance ($P < .01$). GSI, Global Severity Index.

differences for most of the sleep dimensions between the two groups, with the exception of use of sleep medication and daytime dysfunction.

Hierarchical regression models were used to identify the strongest predictors of sleep disturbances, including subclinical disturbances suggested by clinical cutoff scores (Table 3). Cigarette smoking was included as the primary predictor of sleep disturbance (Model 1), (β = 0.229, $P < .001$) and accounted for 5% of variance ($R^2 = 0.05$). Age was entered into the second model, and it demonstrated a small but significant β-weight (β = 0.126, $P < .001$), adding an additional 2% of the variance (total $R^2 = 0.07$). Subsequently, pain severity was included into the third model (β = 0.247, $P < .001$) and was found to explain an additional 6% of the variance (total $R^2 = 0.13$). In the next model, the GSI was included, and it demonstrated a significant β-weight (β = 0.436, $P < .001$), adding an additional 17% of variance (total $R^2 = 0.30$) to the model. In the last model (Model 5) alcohol and caffeine were entered, but neither variable added to the prediction of sleep disturbance. This suggests that alcohol and caffeine are not independent contributors to sleep disturbances after controlling for the other variables. Overall, the final model explained 30% of variance ($R^2 = 0.30$, $P < .001$) associated with sleep disturbance.

Discussion

The purpose of this study was to evaluate whether cigarette smoking status adversely influences the sleep quality of patients with masticatory myofascial pain. The results demonstrated that cigarette smoking positively predicts sleep impairment, but the relationship between smoking and sleep disturbance was markedly overshadowed by the contributions of pain severity and psychological distress in the regression model. Psychological distress and pain severity appear to be better predictors of sleep disturbance, followed by smoking status. Yet, due to the exploratory and cross-sectional nature of the study, the cause-effect direction of this association could not be determined. One interpretation of these results could be that smoking status and pain severity are related, such that smoking might increase pain severity, which in turn increases sleep disturbance. An alternative explanation is that insufficient sleep can increase pain perception,[22,23,26,27] generate fatigue, and precipitate alterations in mood that motivate smoking.[54] Thus, it is possible that pain severity and psychological distress might at least partially mediate the association between cigarette smoking and sleep disturbance. Future studies evaluating this potential mediation would shed light on the association between cigarette smoking and sleep disturbance.

Psychological distress accounted for the most variance in the present model predicting sleep disturbance; in fact, it was the strongest predictor of sleep disturbance in this study. These findings are consistent with a previous study demonstrating that psychological distress independently predicted sleep disturbance, and to a lesser extent than pain severity in orofacial pain patients.[41] The present data corroborate the association between psychological distress and sleep disturbance found in other studies that used single subscales of the SCL-90 such as depression and anxiety.[6,20,41,55] The use of more broad variables to assess psychological functioning on chronic pain populations is supported by previous data that have demonstrated that GSI is the most reliable measure of psychological distress for these patients.[51,52]

Several studies[23,26,39,41–44] have investigated the association between pain and sleep disturbance, demonstrating that nonrestorative sleep is a common complaint among patients with musculoskeletal pain, including TMD. The directionality and underlying mechanisms of the association between pain and sleep are still uncertain and are possibly multifactorial.[22] It has been posited that disruption of the descending pain inhibitory pathways, particularly the serotonergic system, would lead to increased pain perception, which in turn leads to increased arousal during the night that can affect the restorative function of sleep.[27,56] In the present study, most of the masticatory myofascial pain patients (94%) endorsed poor sleep quality, and pain severity was found to be a robust predictor of sleep disturbance. These findings are consistent with previous investigations that have demonstrated a positive relationship between pain severity and sleep disturbance in TMD patients[39,41,43,44] and patients with other medical conditions.[22,24,29]

This study demonstrated a positive relationship between cigarette smoking and sleep disturbance in patients with masticatory myofascial pain. Consistent with previous studies,[21,39,57] smokers reported poorer sleep quality and exhibited significantly higher scores on PSQI total and most of the other sleep domains when compared to nonsmokers. Moreover, the study indicated that smokers presented longer sleep latency, reduced sleep quality, and shorter sleep duration, corroborating findings in other studies using polysomnography.[5,13,16] Cigarette smoking also correlated with increased pain severity and with poor psychological functioning in patients with masticatory myofascial pain. These findings agree with the results of a previous study suggesting that smokers experience significantly more negative psychological outcomes than nonsmokers.[39]

A possible biologic explanation for the undesirable effects of cigarette smoking includes the direct effect of nicotine on the central nervous system (CNS). Nicotine commonly delivered by cigarette smoking is a well-known stimulant with a direct agonist effect on pre- and postsynaptic α_7 and $\alpha_4\beta_2$ nicotine-cholinergic receptors. Activation of cholinergic neurotransmission potentiates the release of several neurotransmitters, such as acetylcholine, serotonin, norepinephrine, glutamate, γ-amino butyric acid (GABA), and dopamine, that are involved in numerous neuronal pathways. These neurotransmitter systems are vital in the regulation of the sleep-wake cycle, as well as in pain and mood modulation.[58–60] Therefore, smoking likely activates neurobiologic systems involved in the arousal process and the transitions between sleep stages.[16]

Unfortunately, a dose-response relationship between the quantity and duration of cigarette smoking and the degree of sleep disturbance could not be explored in the present study. Numerous studies have tried to evaluate a dose-response relationship between cigarette smoking and different outcomes (eg, pain severity, sleep disturbance) based on the number of cigarettes, or packs of cigarettes, smoked per day.[13,21,31,32,36,40] However, the lack of a universally accepted classification of cigarette-smoking habits, individual differences in cigarette-smoking behavior, and variation in the type of products smoked (filter, size, brands, low/high nicotine) and components (menthol) of cigarettes limit the assessment of nic-

otine levels in the present sample. The classification of smoking status in the literature varies in definition and number of categories. Future studies using specific subgroups based on their cigarette-smoking behavior or levels of nicotine dependence would enable more accurate measurement of smoking status and exposure to nicotine. Moreover, real-time measurement of nicotine levels in TMD patients would provide important information that would further understanding of the role of nicotine in influencing sleep and pain behavior.

As expected, age predicted sleep disturbance and accounted for 2% of the measured variance. The effects of aging on sleep pattern are well known. A meta-analysis evaluating sleep architecture through the adult lifespan suggested that changes in sleep pattern begin at a young age. Aging is associated with increased awakenings, increased sleep latency, lighter stages of sleep, and reduction of rapid eye movement (REM), deep and slow wave sleep, reducing total sleep time and sleep efficiency.[61] Sleep disruption is more pronounced in elderly people, in part due to numerous medical comorbidities, including neurologic disorders, and also due to use of more medications that can affect sleep quality.[62] In this study, TMD patients were mostly individuals in young adulthood and middle-age, which might further account for the low variance added by age in the present regression model.

Interestingly, alcohol use and caffeine intake did not independently contribute to sleep disturbances. Even if added as the first predictors into the model, only caffeine predicted sleep disturbance, and this association was no longer significant after controlling for smoking. It is well established that nicotine and caffeine generate insomnia-like symptoms.[10] However, an antagonistic effect on subjective arousal has been reported when these stimulants are administered together.[63] The weak association between caffeine consumption and sleep disturbance in the current study might be due to the fact that only chronic pain patients who generally use a variety of medications that might override the effects of caffeine were evaluated in this study. Caffeine intake and alcohol use are common modifiable risk factors that might affect sleep quality; therefore, interventions such as sleep hygiene techniques might positively impact the sleep quality of chronic pain patients.

The linear model used in this study accounted only for 30% of the variance. This suggests that other factors not accounted for in the current study might be mediating the association between cigarette smoking and sleep in TMD patients.[64] Levels of nicotine dependence, other demographic characteristics and lifestyle indicators (eg, shiftwork exposure[65] and difficulties in interpersonal relationships[66]), medical comorbidities, and sleep-related problems might represent some of these factors. Likewise, other sleep disorders, including restless legs syndrome and obstructive sleep apnea, are associated with nonrestorative sleep and with excessive daytime sleepiness due to an increased number of arousals and awakening episodes.[21] These medical conditions and sleep-related disorders are common comorbidities in chronic pain patients with sleep disturbances[67] that were not accounted for in the present study.

Study Limitations

Several limitations of this study merit discussion. The retrospective fashion of the current study prevents drawing any conclusions about causality but does generate directions for future research based on the observed associations. Caution should be taken when interpreting the results, since this study relied only on self-report questionnaires to assess sleep quality; no polysomnographic recordings were used to confirm sleep disturbance. However, the PSQI has demonstrated to be a reliable measure of subjective sleep quality.[47] Similarly, smoking status was assessed based on patient self-report; no measures of nicotine dependence or duration of tobacco or biomarkers of nicotine use were obtained, which may underestimate the prevalence of cigarette smoking or result in recall bias.[68] Future longitudinal studies using biochemical markers or levels of nicotine dependence to assess smoking status and polysomnography to assess sleep quality would improve understanding of the relationship between sleep, cigarette smoking, and pain.

Another potential limitation of the present study is that information about former smokers was not available, and as such, former smokers were included in the group of nonsmokers. However, since several studies have shown no differences in terms of pain or sleep quality when former smokers were compared to nonsmokers,[29,35,69] the consequences of including former smokers in the nonsmokers group are deemed little to none. Also, information about medical comorbidities, medication use, and body mass index was not available for this study. The presence of medical comorbidities such as fibromyalgia and respiratory diseases such as sleep apnea could represent some of the unaccounted variance that may contribute to sleep disturbance. The sociodemographic characteristics of the smokers in this study were comparable to smokers' features in other studies[35,36,39] evaluating TMD patients. However, the number of smokers (32.1%) in this study was higher than that in other studies (10.8%,[36] 15.1%[35]) and higher than the overall national (19.0%)[1] and overall regional (24.6%) prevalence of cigarette smoking.[70] These higher rates might represent selection bias in this study's sample.

Conclusions

In summary, cigarette smoking is associated with numerous adverse health outcomes, including pain severity, alterations in mood, and disrupted sleep. The current findings suggest that cigarette smoking is found to be a contributor (5%) to subjectively measured sleep disturbances in patients with masticatory muscle pain. This association is attenuated when including other variables in the model. Neither caffeine and alcohol intake nor demographic factors appear to predict sleep quality over and above cigarette smoking. This study suggests that smoking-cessation interventions may be appropriate for patients with masticatory muscle pain and may improve sleep quality. The findings also highlight the importance of implementing multifaceted pain-management protocols that incorporate strategies to reduce psychological distress and improve sleep quality. However, future studies using mediation analyses to clarify the interdependent relationship between psychological distress, pain severity, and sleep disturbances are warranted.

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