The Association of Smoking Status with Sleep Disturbance, Psychological Functioning, and Pain Severity in Patients with Temporomandibular Disorders

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Aims: *To evaluate the impact of smoking on pain severity, psychosocial impairment, depression, anxiety, and sleep disturbances in a large sample of patients with temporomandibular disorders (TMD).* ***Methods:*** *A retrospective database review was performed on data from 3,251 patients with TMD, diagnosed according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD). Pain severity ratings and psychometric data regarding impairment, sleep disturbance, depression, and anxiety were obtained. Differences between smokers and nonsmokers were evaluated by means of chi-square tests and independent samples* t *tests. Logistic regression models were used to study the impact of smoking, pain severity, and psychometric variables.* ***Results:*** *Of the total population, 42.5% comprised RDC/TMD group I (muscle pain), 25.3% comprised RDC/TMD group III (joint pain), and 32.2% comprised a mixed RDC/TMD group consisting of patients with both a group I and a group III diagnosis. Of the entire population, 26.9% admitted they were smokers. Even after controlling for relevant covariates, smokers reported significantly higher pain severity, impairment, anxiety, depression, and sleep disturbances than nonsmokers.* ***Conclusion:*** *Smokers with TMD reported higher pain severity than nonsmokers with TMD. These patients are at higher risk for factors that may adversely affect treatment outcomes.* J OROFAC PAIN 2013;27:32–41. doi: 10.11607/jop.1040

Key words: anxiety, depression, nicotine, sleep disturbance, temporomandibular disorders

Smoking has been associated with chronic pain. Some studies have found that the percentage of smokers in chronic pain populations was remarkably higher than the national average. Smoking prevalence of 37% to 54% has been reported in patients with chronic pain and low-back pain, respectively, in the United States.[1,2] Recent surveys targeting large populations in Canada and Sweden revealed that smokers more often reported low-back pain than nonsmokers.[3,4] There are strong indications that smokers with chronic pain tend to report higher pain severity, greater functional disability, and more anxiety, depression, and sleep disturbances.[1,5–9]

Indeed, two retrospective chart reviews revealed that smokers with temporomandibular disorders (TMD) also reported higher pain intensity and life interference from pain than nonsmokers with TMD.[10,11] However, the impact of smoking on psychological functioning and sleep disturbances was not assessed in these two studies. Moreover, the number of smokers in these studies was relatively small (38 [11%] and 91 [15%], respectively). On the other hand, a prospective cohort study spanning 6 years evaluated whether smokers would be more

prone to having, maintaining, or developing signs and symptoms of TMD.[12] The study found no differences between the cohort of smokers and the cohort of nonsmokers with regard to the presence, development, or disappearance of signs and symptoms. Pain severity, psychological, and sleep variables were not assessed in the study, which comprised a community sample in contrast to the previously mentioned studies that comprised treatment-seeking TMD patients. The majority of the participants (over 90%) did not report tenderness to palpation or jaw movements at baseline or at 6 years, although some differences between smokers and nonsmokers were found with regard to the presence of temporomandibular joint (TMJ) load pain at baseline, and pain on movement at the 6-year time period, with smokers having less favorable outcomes.

Because smoking may impact a variety of important outcomes in chronic pain, smoking may be a key prognostic factor in chronic pain conditions. The aim of the present study was to evaluate the impact of smoking on pain severity, psychosocial impairment, depression, anxiety, and sleep disturbances in a large sample of patients with TMD.

Materials and Methods

A retrospective database review was performed. The patient population consisted of 3,263 consecutive eligible patients with painful TMD. Of this population, 12 persons were excluded because their smoking status could not be identified. Hence, the final sample consisted of 3,251 patients. The study was approved by the internal review board of the University of Kentucky. All patients gave informed consent on the day of the initial visit and allowed the use of their data for research purposes.

Patient Sample

Women comprised 85.4% of the sample. The mean (± SD) age for the women was 38.1 ± 13.8 years and the mean age for the men was 38.0 ± 14.0 years. Patients were examined by faculty or by residents who were trained by faculty to perform the examinations in a consistent manner. If the patient was examined by a resident, a faculty member routinely reexamined the patient to confirm the diagnosis. Patients were diagnosed according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)[13] and classified as having myofascial pain (RDC/TMD group I) if their primary and secondary (if present) diagnoses were of myogenous origin. They were classified as having arthralgia/osteoarthritis (RDC/TMD group III) if their primary and secondary (if present) diagnoses were of painful arthrogenous origin. Patients with osteoarthrosis (by definition, pain free) as a primary diagnosis were excluded from the study, whereas patients with osteoarthrosis as a secondary diagnosis were included if their primary diagnosis was arthralgia/osteoarthritis (note that this could be the contralateral joint). In this group, a secondary diagnosis of disc displacement (RDC/TMD group II) was also allowed. Finally, patients with a primary and secondary pain-related diagnosis, one from each RDC/TMD group I and RDC/TMD group III, comprised the mixed TMD group.

Instruments

Current smokers were identified from a pain questionnaire based on self-report of smoking. Patients were asked to identify themselves as smokers based on the question: "Do you smoke?" with the option to choose "yes" or "no." Patients who answered "yes" were also asked to indicate how many packs per day they smoked, assuming that a pack contained 20 cigarettes. Currently, there is no consensus with regard to the definition of intermittent, light, moderate, or heavy smokers.[14,15] Initial analyses showed that with regard to the main psychometric domains (see below) light smokers, defined as having less than 5 cigarettes per day, did not differ significantly from heavier smokers or nonsmokers. Since these light smokers (n = 15) represented a very small percentage of the larger sample, given the large sample size and given the questionable reliability of self-reported number of packs of cigarettes smoked per day, it was decided to divide the sample based on self-identified smoking status.

Pain severity was derived from a 100-mm visual analog scale (VAS) asking patients to report maximum, average, and minimum pain over the past month; in the present study, only average pain severity was used.

Depression, anxiety, and Global Symptom Index (GSI) scores were derived from the Symptom Check List 90 - Revised (SCL-90-R).[16] The SCL-90-R is a 90-item multidimensional self-report measure. It measures nine primary symptom dimensions of psychological functioning and calculates three global indices. Test-retest reliabilities range from r = 0.78 to 0.90 for nonpatient samples, and internal consistencies range from 0.77 to 0.90.

Life control, life interference, and affective distress measures were derived from the Multidimensional Pain Inventory (MPI).[17] The MPI is a 61-item self-report measure that assesses impairment due

Table 1 Comparison of Select Demographics with Regard to Sex

	Sex		
	Male 474 (14.6%)	Female 2,776 (85.4%)	Total 3,250* (100%)
Employment status, n (%)			
Unemployed	150 (32.3%)	1,062 (38.6%)	1,212 (37.7%)
Employed	315 (67.7%)	1,691 (61.4%)	2,006 (62.3%)
Marital status, n (%)			
Single	146 (34.0%)	638 (25.6%)	784 (26.9%)
Married	250 (58.3%)	1,485 (59.7%)	1,735 (59.5%)
Divorced	31 (7.2%)	304 (12.2%)	335 (11.5%)
Widowed	2 (0.5%)	62 (2.5%)	64 (2.2%)
Smoking status, n (%)			
Smoker	136 (28.7%)	738 (26.6%)	874 (26.9%)
Nonsmoker	338 (71.3%)	2,038 (73.4%)	2,376 (73.1%)
Mean age, years (± SD)	38.0 (± 14.0)	38.1 (± 13.8)	38.1 (± 13.8)

*Sex data missing for one person; marital status: (χ^2 = 24.497; P < .001); employment status (χ^2 = 6.764; P = .009); smoking status (χ^2 = .914; P = .339); age (t = -.267; P = .790).

Table 2 Comparison of Select Demographics with Regard to Smoking Status

	Smoking status		
	Smoker 874 (26.9%)	Nonsmoker 2,376 (73.1%)	Total 3,250* (100%)
Sex, n (%)			
Male	136 (15.6%)	338 (14.2%)	474 (14.6%)
Female	738 (84.4%)	2,038 (85.8%)	2,776 (85.4%)
RDC/TMD group, n (%)			
Group I (myogenous)	406 (46.4%)	977 (41.0%)	1,383 (42.5%)
Group III (arthrogenous)	181 (20.7%)	640 (26.9%)	821 (25.3%)
Mixed group	288 (32.9%)	759 (31.9%)	1,047 (32.2%)
Employment status, n (%)			
Unemployed	384 (44.3%)	828 (35.2%)	1,214 (37.7%)
Employed	482 (55.7%)	1,525 (64.8%)	2,011 (62.3%)
Marital status, n (%)			
Single	212 (26.8%)	573 (27.2%)	785 (26.9%)
Married	398 (62.5%)	1,337 (51.0%)	1,735 (59.4%)
Divorced	157 (20.1%)	178 (8.3%)	335 (11.5%)
Widowed	13 (1.7%)	51 (2.4%)	64 (2.2%)
Mean age, years (± SD)	35.4 (± 11.0)	39.1 (± 14.6)	38.1 (± 13.8)
Average pain intensity/VAS, mm (± SD)	59.1 (± 23.3)	45.6 (± 23.9)	49.3 (± 23.5)

*Sex data missing for one person; smoking status (χ^2 = 0.914; P = .339); marital status: (χ^2 = 83.472; P < .001); employment status (χ^2 = 22.590; P < .001); RDC/TMD group (χ^2 = 14.271; P = .001); age (t = 7.7; P < .001); pain intensity (t = 14.0; P < .001).

to pain, levels of social support, and activity. Test-retest reliabilities of scale scores range from r = 0.68 to 0.86, and internal consistencies range from 0.73 to 0.90.

The total score of the Pittsburgh Sleep Quality Index (PSQI) was used as a measure of sleep disturbance.[18,19] The PSQI is an 18-item self-report measure that assesses general sleep quality. It provides information on the number of hours spent in bed and asleep, number of sleep disturbances, sleep latency, sleep efficiency, and use of sleep medication. The PSQI has been shown to be a valid and reliable assessment for overall sleep quality and disturbance, with good test-retest reliability (r = 0.85) and internal consistency (α = 0.83).

Statistical Analyses

First, a set of χ^2 tests and independent samples *t* tests examined potential sex differences in diagnostic group, smoking status, and demographics (age, marital status, and employment status). Second, another set of χ^2 tests and *t* tests was used to identify differences between smokers and nonsmokers in diagnostic groups and demographics (age, marital status, and employment status). Hierarchical linear regressions were performed to evaluate the impact of smoking on depression, anxiety, and global symptoms (SCL-90 subscales); life interference, affective distress, and life control (MPI subscales); and average pain severity (VAS scale); controlling for covariates with robust relations with smoking. All continuous predictors were screened for normality and mean-centered prior to use in regression models. The criterion for statistical significance for all analyses was set at $\alpha = .05$. Normal hierarchical linear regression analyses were performed in Predictive Analytics Soft Ware (PASW, release 18; formerly SPSS); ZINB regression analyses were performed in SAS (version 9.2).

Results

A total of 3,251 patients with a TMD diagnosis were included in this study. Of this population, 42.5% comprised the RDC/TMD group I, 25.3% the RDC/TMD group III, and 32.2% the mixed RDC/TMD group. There were no differences in these diagnostic groups with regard to sex ($\chi^2 = 3.426$; $P = .180$). As shown in Table 1, men were more likely to be employed. In terms of marital status, a similar percentage of men and women were married, but men were more likely to be single, whereas women were more likely to be divorced. There was no difference in smoking status between men and women.

Of the entire population, 26.9% admitted that they were smokers. There was a significant difference in smoking status between the diagnostic groups ($\chi^2 = 14.271$; $P = .001$), with the RDC/TMD group I containing the highest number of smokers (29.4%) and the RDC/TMD group III containing the lowest number of smokers (22%); 27.5% in the mixed RDC/TMD group admitted being smokers. As shown in Table 2, smokers were more likely to be unemployed than nonsmokers. With regard to marital status, a similar percentage of smokers and nonsmokers were single, but smokers were more likely to be divorced whereas nonsmokers were more likely to be married. Smokers were significantly younger than nonsmokers and reported significantly higher pain intensity than nonsmokers (Table 2). Smokers also reported significantly more distress on all psychometric and sleep vairables (Fig 1).

Regressions Examining the Impact of Smoking on Sleep and Psychological Functioning

Based upon previously established robust relations with smoking, the following variables were included as covariates in all regression analyses: age, sex (coded as 0 for male and 1 for female), employment status (coded as 0 for unemployed and 1 for employed), marital status (coded as 0 for unmarried and 1 for married), and VAS average pain severity.[5–8] Results of the models predicting these variables are presented in Table 3. After controlling for covariates, smoking was associated with higher scores on the PSQI, greater anxiety, greater depression, greater global psychological distress (GSI), greater life interference, greater affective distress, and lower life control. Earlier models included caffeine and alcohol use, but these covariates did not change the beta weight of the model for smoking's effect on the psychosocial variables.

To determine whether the impact of smoking differed by diagnostic category, dummy-coded variables were created for each diagnostic category and interaction terms were created with smoking status (ie, diagnostic category × smoking status). For each dependent variable, two regression models were fitted that included covariates, smoking status, dummy-coded variables representing two of the three diagnostic categories, and two interaction terms representing the interactions between those two dummy-coded variables and smoking status in the fourth step. No significant differences were found, indicating that the effect of smoking on outcome variables was not moderated by diagnostic category.

Regressions Examining the Impact of Smoking on Average Pain Severity

Based upon previous findings that anxiety and depression are closely related with pain ratings,[20–22] both anxiety and depression (SCL subscales) were included as covariates in analyses predicting pain severity ratings. In addition, the covariates age, sex, marital status, and employment status were retained. Results of the hierarchical linear regression model predicting VAS average pain severity ratings are presented in Table 4. Controlling for covariates, smoking predicted higher average pain severity ratings. Moderation by pain category was

Table 3 Results of Hierarchical Linear Regression Models Regressing SCL Anxiety, SCL Depression, SCL Global Symptom Index, MPI Interference, MPI Affective Distress, and MPI Life Control on Smoking and the Covariates Age, Sex, Employment Status, Marital Status, and VAS Average Pain Severity

Variable	β	*P*	Total R^2	R^2 Δ	*P*
Sleep disturbance (PSQI total)					
Step 1			.20	.20	< .001
Age	.066	< .001			
Sex	.018	.26			
Employment status	–.094	< .001			
Marital status	–.012	.48			
Pain severity	.41	< .001			
Step 2			.22	.02	< .001
Smoking	.15	< .001			
Anxiety (SCL subscale)					
Step 1			.09	.09	< .001
Age	.048	.008			
Sex	–.09	< .001			
Employment status	–.093	< .001			
Marital status	–.015	.42			
Pain severity	.25	< .001			
Step 2			.10	.01	< .001
Smoking	.13	< .001			
Depression (SCL subscale)					
Step 1			.10	.10	< .001
Age	.082	< .001			
Sex	–.089	< .001			
Employment status	–.08	< .001			
Marital status	.003	.88			
Pain severity	.26	< .001			
Step 2			.11	.01	< .001
Smoking	.11	< .001			
Global psychological functioning (SCL GSI subscale)					
Step 1			.12	.12	< .001
Age	.052	< .01			
Sex	–.084	< .001			
Employment status	–.105	< .001			
Marital status	–.003	.85			
Pain severity	.31	< .001			
Step 2			.14	.02	< .001
Smoking	.13	< .001			
Life interference (MPI subscale)					
Step 1			.35	.35	< .001
Age	.084	< .001			
Sex	–.029	.05			
Employment status	–.10	< .001			
Marital status	–.001	.96			
Pain severity	.57	< .001			
Step 2			.36	.01	< .001
Smoking	.09	< .001			
Affective distress (MPI subscale)					
Step 1			.13	.13	< .001
Age	–.074	< .001			
Sex	.025	.14			
Employment status	–.039	.03			
Marital status	–.007	.69			
Pain severity	.34	< .001			

Table 3 (continued)

Variable	β	*P*	Total R^2	R^2 Δ	*P*
Step 2			.15	.02	< .001
Smoking	.13	< .001			
Life control (MPI subscale)					
Step 1			.10	.10	< .001
Age	.052	< .01			
Sex	–.009	.58			
Employment status	.11	< .001			
Marital status	.008	.66			
Pain severity	–.28	< .001			
Step 2			.11	.01	< .001
Smoking	–.085	< .001			

Sex was coded 0 = male and 1 = female. Employment status was coded 0 = unemployed and 1 = employed. Marital status was coded 0 = unmarried and 1= married. Smoking was coded 0 = nonsmoker and 1 = smoker.
SCL, Symptom Check List; GSI, Global Symptom Index; MPI, Multidimensional Pain Inventory; PSQI, Pittsburgh Sleep Quality Index; VAS, visual analog scale.

Table 4 Results of Hierarchical Linear Regression Models Regressing VAS Average Pain Severity on Smoking and the Covariates Age, Sex, Employment Status, Marital Status, Depression, and Anxiety

Variable	β	*P*	Total R^2	R^2 Δ	*P*
VAS average pain severity					
Step 1			.32	.32	< .001
Age	–.09	< .001			
Sex	.06	< .001			
Employment status	–.14	< .001			
Marital status	.005	.78			
Depression (SCL subscale)	.17	< .001			
Anxiety (SCL subscale)	.11	< .001			
Step 2			.37	.05	< .001
Smoking	.19	< .001			

Sex was coded 0 = male and 1 = female. Employment status was coded 0 = unemployed and 1 = employed. Marital status was coded 0 = unmarried and 1= married. Smoking was coded 0 = nonsmoker and 1 = smoker.
VAS, visual analog scale; SCL, Symptom Check List.

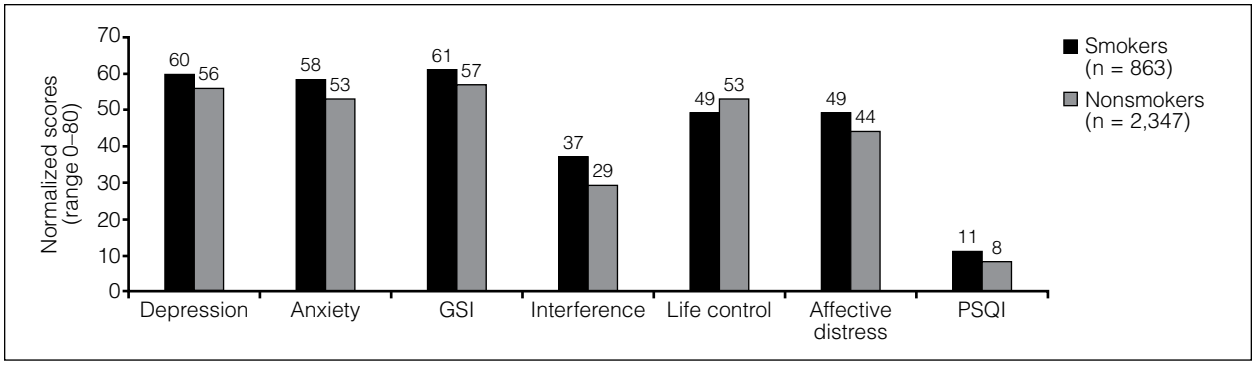

Fig 1 Differences between smokers and nonsmokers in psychometric variables and sleep quality. SCL (range 0–100 MPI); raw score PSQI (range 0–21). SCL, Symptom Check List; MPI, Multidimensional Pain Inventory; PSQI, Pittsburgh Sleep Quality Index; GSI, Global Symptom Index. $P < .05$ for all subscales.

again investigated as described above; no significant differences were found, indicating that the effect of smoking on average pain severity did not differ by diagnostic category.

Discussion

This study showed that smoking is associated with undesirable psychometric characteristics in patients

with TMD. Smokers reported more anxiety, depression, and sleep disturbances, after the analyses controlled for pain intensity and demographic variables. In addition, they reported having less life control, more life interference, and greater affective distress than nonsmokers. The associations of smoking with these outcomes did not appear to be influenced by diagnostic category. These findings build on those reported in smaller studies exploring the relationship between smoking and TMD,[10,11] and are consistent with previous findings in studies exploring the relationship between smoking and other types of chronic pain.[5,7–9] The present study improves upon previous work by replicating the association between smoking and negative outcomes in chronic pain in a much larger sample of patients with TMD, and increases confidence in the nature of these relationships by demonstrating unique effects of smoking after controlling for a variety of potential "nuisance" variables known to be robustly related to both smoking and negative outcomes.

Smoking and Pain Severity

Smokers reported significantly higher pain severity after controlling for demographic variables, anxiety, and depression. These findings corroborated those of other studies.[5,7–10] Only a few previous studies examining this link have controlled for demographic variables; none have controlled for psychometric variables. Yunus et al, who adjusted for education and age, found a positive relationship between smoking and pain in patients with fibromyalgia.[5] A study by Weingarten and colleagues found that pain intensity as measured by the graded chronic pain scale (GCPS), a scale combining pain intensity with interference, was no longer significantly different between smoking and nonsmoking TMD patients after adjusting for age, sex, marital status, education, and employment status.[11] Possible explanations for the lack of significance and conflicting results with the current study could be the smaller number of patients in their study (about 600 patients, 15% of whom were smokers), the use of a different pain intensity scale, and the fact that they did not control for psychometric variables. Because of the large number of patients and the inclusion of a considerable number of covariates in the current study, it seems reasonable to conclude that smoking has a significant impact on pain intensity.

Smoking and Sleep Disturbances

Nicotine activates the nicotinic acetylcholine receptors (nAChRs) in several brain areas, leading to increased cholinergic activity in pathways throughout the brain and release of several neurotransmitters, including dopamine, glutamate, norepinephrine, and γ-aminobutyric acid.[23] The ascending reticular arousal system, responsible for wakefulness, also heavily depends on acetylcholine, monoamines, and other neuropeptides.[24] Thus, the neurotransmitters released by nicotine may have a stimulating effect and impede sleep by enhancing the activity of the ascending reticular arousal system, interfering with the competing sleep-promoting system.

Zhang et al[25] confirmed the subjective sleep disturbances reported by smokers in earlier epidemiologic surveys with objective data obtained through home polysomnograms in a large community-based sample. They found that smokers had longer latency to sleep onset as well as to first rapid eye movement (REM) sleep, and less total sleep time. They also found that smokers spent more time in lighter sleep stages, and less time in slow-wave sleep stages than nonsmokers.

Spending more time in lighter sleep stages clearly could be associated with easy arousals, as less stimulation is required to wake one up from lighter stages than from slow-wave stages of sleep, and thus could lead to poor sleep quality.[26] Nicotine withdrawal during sleep may also be associated with arousal and could directly affect sleep quality. Smokers in general, and especially night smokers, report to be high caffeine consumers,[25,27] and therefore sleep disturbances could be related to the direct effect of caffeine on sleep. Zhang and colleagues adjusted for caffeine and alcohol intake, as well as for medical conditions associated with smoking such as pulmonary diseases, which may have an independent effect on sleep quality, and still showed an autonomous effect of nicotine on sleep architecture.[25] The initial adjustments in the present study for caffeine and alcohol use culminated in similar findings of an independent effect of smoking on sleep disturbances.

Smoking and Psychological Functioning

In the present study, smoking was associated with a variety of negative psychological outcomes. Even after controlling for age, sex, employment and marital status, and pain severity, smokers reported higher levels of anxiety, depression, overall psychological distress, affective distress, higher levels of pain-related life interference, and lower perceptions of control over one's life. These findings are consistent with previous work demonstrating that smoking is associated with poorer psychological functioning among both otherwise healthy individuals and chronic pain patients.[28–31] The present study is the first to

demonstrate that such links in chronic pain patients remain significant after controlling for pain severity, which may be associated with both higher levels of smoking and poorer psychological functioning.

A host of studies of otherwise healthy individuals have found that smokers exhibit higher levels of anxiety and depression than nonsmokers.[28–31] Further, several studies have documented higher levels of concurrent and prospective risk of meeting diagnostic criteria for anxiety and mood disorders among smokers, including increased risk of generalized anxiety disorder, social phobia, posttraumatic stress disorder, panic disorder, agoraphobia, major depressive disorder, and dysthymic disorder.[28,32–37] In particular, evidence points to a strong causal link between smoking and the onset of panic attacks and panic disorder that may be mediated by lung disease.[37–43] Furthermore, the link between smoking and anxiety or mood disorders appears to be amplified among nicotine-dependent individuals, with some studies finding that smoking only leads to increased risk of anxiety or mood disorders if one is currently nicotine-dependent[32,33,35] and that greater nicotine dependence is associated with higher levels of anxiety and depression.[33,44] Such evidence is consistent with recent work suggesting that neurotransmitter changes associated with smoking may be partially responsible for its detrimental effects on mental health.[45–47]

Only a few studies have examined the impact of smoking on psychological functioning in chronic pain patients. These studies demonstrated that smoking was associated with higher overall levels of affective distress, depression, anxiety, and pain catastrophizing.[48,49] The results of the present study replicate and extend these findings by demonstrating a virtually identical pattern of results in a sample of individuals with TMD, providing evidence that smoking may also be associated with lower levels of life control and higher levels of life interference, and by providing evidence that these associations remain significant after controlling for pain severity.

Clinicians are beginning to perceive that long-term pain improvement is difficult, if not impossible, to achieve if sleep dysfunction persists and patients continue the use of nicotine. Through its action on nAChRs, nicotine stimulates the release of numerous neurotransmitters that orchestrate aspects of arousal, sleep, anxiety, cognition, and pain modulation.[50,51] The powerful peripheral and central effects produced by nicotine can adversely influence the autonomic nervous system and endocrine, immune, and cytokine function[50,52,53]; alterations in central nervous system physiology endure even after smoking cessation.[54]

Although some data suggest that acute nicotine exposure can produce antinociception,[50,55,56] persistent nicotine exposure impairs the physiological capacity to control pain and distress. Smoking is associated with overexpression of nAChRs throughout the brain, persistent sympathetic arousal, sleep dysregulation, depletion of endogenous opioid capacity, increased cytokine production, and withdrawal symptoms—all processes that, when disturbed, may impair pain and mood modulation.[52,57–59] In addition, nicotine affects the liver cytochrome P450 enzymes and related transferases to increase morphine metabolism, which may contribute to enhanced hyperalgesic responses and increased pain perception.[60–62] Smokers use more opioids than nonsmokers,[3,63,64] and opioid use increases nicotine consumption in a dose-dependent response relationship, potentially exposing smokers to increased opioid-induced hyperalgesic responses.[65] Nicotine exposure in an animal neuropathic pain model increases mechanical hyperalgesia, while opioid use in humans can result in hyperalgesic responses to subsequent viscerosomatic stimuli.[66,67]

Chronic pain patients may use smoking as a coping mechanism,[68–70] but the perceived pain relief may actually be related to a decreased awareness of painful stimuli rather than the blunting of nociception and the need to satisfy nicotine craving as time after the last smoke lengthens.[52,56] Additionally, nicotine impairs peripheral perfusion, and carbon monoxide from smoking increases heme oxygenase associated with cellular processes such as inflammation, oxidative stress, and apoptosis.[71–73] Nicotine's pervasive effects are too extensive in scope to outline in this article, but its adverse physiological influences provide possible insight as to why chronic pain smokers and nonsmokers present different psychometric profiles, and why continued nicotine use may make treatment of chronic pain extremely difficult.

Study Limitations

A shortcoming of this study was that smoking was treated as a dichotomous variable. The questionnaire included an entry on the number of packs per day, which precluded analyses based on number of cigarettes per day. Future studies may investigate possible dose-dependent relationships by taking into consideration the number of cigarettes smoked or use measures of nicotine dependency. Another limitation of the study was that test-retest reliability data with regard to examination and diagnoses were not available. Residents were trained and calibrated by experienced clinicians. However, all diagnoses were either confirmed or contested and corrected by supervising faculty.

Conclusions

Smokers with TMD reported higher pain severity than nonsmokers with TMD. These patients may be at higher risk for factors that may adversely affect treatment outcomes. It is therefore important that healthcare professionals counsel smokers on the unfavorable impact of nicotine on their pain condition and prognosis for improvement, and recommend smoking cessation as an integral part of the treatment plan.

Disclaimer

The views expressed in this paper are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Army, Air Force, Department of Defense, nor the U.S. Government.

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