No Dose-Response Association Between Self-Reported Bruxism and Pain-Related Temporomandibular Disorders: A Retrospective Study

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Aims: To investigate whether a dose-response relationship exists between the intensity of pain-related temporomandibular disorders (TMDs) and the amount of self-reported bruxism activities in a group of TMD pain patients. Methods: A total of 768 patients referred to a specialized clinic for complaints of orofacial pain and dysfunction were initially enrolled in the study. Of these patients, 293 who were diagnosed with at least one type of pain-related TMD according to the Diagnostic Criteria for Temporomandibular Disorders were selected. The questionnaire-based reports of TMD pain intensity, as assessed by an 11-point numeric rating scale (NRS), were subsequently compared to the reports of sleep bruxism (single question; 5-point Likert scale) and awake bruxism (mean score of six questions; 5-point Likert scale). Spearman correlations were used to assess associations, and possible confounding effects of depression, somatic symptoms, and anxiety were taken into account. Results: Spearman correlation tests provided no significant correlation between the amount of self-reported sleep bruxism and TMD pain intensity. On the other hand, the amount of awake bruxism was positively correlated with the intensity of TMD pain; however, the latter correlation was lost when the model was controlled for the effects of depression. **Conclusion:** The assumption that there is a dose-response gradient association between bruxism and TMD pain, reflected in more bruxism leading to more overloading and thus to more pain, could not be justified. J Oral Facial Pain Headache 2018;32:375-380. doi: 10.11607/ofph.2090

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omplaints of pain in the orofacial region are a common reason patients seek help in a dental practice. Once the cause(s) and contributing factors of this pain are identified, a treatment plan can be designed to eliminate the pain. In most cases, orofacial pain is related to dental diseases, often expressed by identifiable tissue damage. In contrast, pain-related temporomandibular disorders (TMD), the second most prevalent orofacial pain, have a chronic nature and are usually not directly associated with actual tissue damage.¹ Since TMD pain shares many features with other common chronic pain conditions, it is placed within the same biopsychosocial model currently used to study and manage such conditions.^{2,3} Pain-related TMD occur in approximately 10% of the population over age 18.^{1,4,5} The origin of this pain most often lies in the masticatory muscles, and the pain is increased during function.¹

Several factors have been suggested to play a role in the predisposition, onset, and maintenance of TMD pain. Among them is bruxism, a repetitive jaw muscle activity that is characterized by clenching or grinding and/or bracing or thrusting of the mandible, occurring either during wakefulness (awake bruxism) or sleep (sleep bruxism).⁶ A number of studies on self-reported or clinical diagnosis of bruxism have shown a positive association between bruxism and TMD pain.⁷ Moreover, a commonly held view in the literature and in clinical practice is that there is a dose-response relationship between TMD pain and bruxism; ie, more bruxism (of higher intensity and/or duration) leads to more overloading

© 2018 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITHOUT WRITTEN PERMISSION FROM THE PUBLISHER. and therefore to more pain.8 However, one should keep in mind that most studies on this topic used binary data (no/yes) to assess the presence of bruxism, the presence of TMD pain, or both. This precludes a conclusion as to whether a dose-response relationship exists between self-reported bruxism and TMD pain. In an attempt to determine whether a dose-response relationship does exist between the amount of self-reported bruxism activities and the intensity of self-reported TMD pain, van der Meulen et al conducted two studies.9,10 The outcomes of these studies suggested no clinically relevant relationships; however, patients with other forms of orofacial pain were included in these studies. Therefore, the aim of the present study was to investigate whether a dose-response relationship exists between the intensity of pain-related TMD and the amount of self-reported bruxism activities in a group of TMD pain patients. To that end, a clinical diagnosis of TMD pain was set according to the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD),¹¹ and a distinction was made between sleep bruxism and awake bruxism. The hypothesis was that a positive dose-response association would be found between both types of bruxism (awake and sleep) and the intensity of TMD pain. Since psychologic factors are generally believed to play a role in TMD pain experience as well,^{12,13} the possible confounding effects of depression, somatic symptoms, and anxiety were also taken into account.

Materials and Methods

Participants

Participants were selected from among patients who attended the Clinic for Orofacial Pain and Dysfunction of the Department of Oral Kinesiology at the Academic Centre for Dentistry Amsterdam (ACTA), the Netherlands. Reasons for referral to this clinic were complaints in the orofacial area (including TMD pain), bruxism, tooth wear, and/or sleep apnea. The recruitment period was between September 2013 and June 2015.

Data Collection

Prior to the first clinical visit, all patients completed a digital diagnostic questionnaire. This questionnaire contained various instruments derived from the Axis II protocol of the DC/TMD,¹¹ including screening tools for pain intensity, pain-related disability, psychologic distress, jaw functional limitations, parafunctional activities (including bruxism), and the presence of comorbid pain conditions. At the time of data collection, all patients were informed through the ACTA website that their data could be used anonymously for research not regulated by the Dutch law for Medical Research Involving Human Subjects Act (ie, medical interventions for research purposes). If patients did not want their data to be used for research purposes, it was clearly indicated that they could inform their dentist, and this would not influence their care in any way. Furthermore, the internal ethical committee pronounced that the study complied with the ethical research code of conduct at ACTA and that the patient data could be used in this retrospective medical file study.

As part of the clinical examination, palpation of the masticatory muscles and the temporomandibular joints (TMJs) was performed by dentists extensively trained in the DC/TMD Axis I protocol. A clinical diagnosis of TMD pain was based on information derived from both the screening instruments included in the DC/TMD Axis II protocol and the clinical examination implemented in the DC/TMD Axis I protocol.¹¹ TMD pain was considered present when at least one of the possible TMD pain diagnoses according to the DC/TMD protocol was set: (local) myalgia, myofascial pain, myofascial pain with referral, headache attributed to TMD, or arthralgia.¹¹ All patients without any pain in the orofacial area as reported in the digital questionnaire were excluded from the analyses. This was also done for all patients who initially reported orofacial pain in the questionnaire but for whom a clinical diagnosis of TMD pain could not be established. No exclusion criteria were applied other than that all patients had to be at least 18 years of age.

Outcome Variable

The outcome variable consisted of the intensity of clinically diagnosed TMD pain derived from the three questions that assessed orofacial pain intensity (pain right now, worst pain, and average pain; Graded Chronic Pain Scale).^{11,14} The average of the three numeric rating scale (NRS) scores (0–10) was calculated and multiplied by 10 in order to give a 0–100 score.

Independent Variable

For measuring bruxism, the Dutch version of the Oral Behaviours Checklist (OBC) was used.¹⁰ The OBC is a 21-item scale for identifying and quantifying the frequency of jaw overuse behaviors¹⁵ and is implemented in the DC/TMD Axis II protocol.¹¹

The following item on the OBC was used to record the intensity of sleep bruxism: "Clench or grind teeth when asleep, based on any information you may have." The possible response options ranged between 0 (none of the time) and 4 (4–7 nights per week). To assess any awake bruxism behavior, the following six items were used: (1) "Grind teeth together during waking hours"; (2) "Clench teeth together during waking hours"; (3) "Press, touch, or hold teeth together other than while eating (that is, contact between upper and lower teeth)"; (4) "Hold, tighten, or tense muscles without clenching or bringing teeth together"; (5) "Hold or jut jaw forward or to the side"; and (6) "Hold jaw in rigid or tense position, such as to brace or protect the jaw." The possible response options ranged between 0 (none of the time) and 4 (all of the time). The average of all six 5-point (0-4)Likert scale scores was calculated.

Potential Confounding Variables

Since it is generally acknowledged that psychologic maladjustment plays an important role in both the first onset and the chronicity of TMD pain,^{12,13} it was decided to adjust for the potential confounding effects of psychologic factors. To that end, three Axis II instruments were used in order to obtain a more comprehensive assessment of psychologic functioning of the participants.¹¹ This assessment uses the Patient Health Questionnaire (PHQ)-9 to screen for depression. As a measure of the severity of depression, the PHQ-9 score was calculated by assigning scores from 0 (not at all) to 3 (nearly every day) to the nine items (total scores could range from 0 to 27). To assess the severity of somatic symptoms, the PHQ-15 was used. The PHQ-15 consists of a list of 15 somatic symptom clusters that account for more than 90% of all physical complaints. Each symptom is scored from 0 (not bothered at all) to 2 (bothered a lot), and the total score can range

Table 1 Mean and Standard Deviation (SD) Scores for Sleep Bruxism,Awake Bruxism, Pain Intensity, Somatic Symptoms,Depression, and Anxiety

	Mean	SD
Sleep bruxism (0–4)	2.53	1.71
Awake bruxism (0–4)	1.45	0.89
Pain intensity (0–100)	57.95	20.45
Somatic symptoms (0–30)	9.56	5.06
Depression (0–27)	6.20	5.30
Anxiety (0–21)	4.67	4.63

Table 2 Spearman Correlations Between Sleep Bruxism,Awake Bruxism, Pain Intensity, Somatic Symptoms,Depression, and Anxiety

	Sleep bruxism		Awake bruxism		Pain intensity	
	r	P value	r	P value	r	P value
Sleep bruxism	-	-	0.529	< .001	0.055	.350
Awake bruxism	0.529	< .001	-	-	0.183	.002
Pain intensity	0.055	.350	0.183	.002	-	-
Somatic symptoms	0.142	.015	0.220	< .001	0.328	< .001
Depression	0.153	.009	0.276	< .001	0.319	< .001
Anxiety	0.186	.001	0.284	< .001	0.262	< .001

from 0 to 30. Finally, for anxiety screening, the Generalized Anxiety Disorder-7 (GAD-7) was used. The total anxiety score (range 0-21) was calculated by summing the scores of all seven items (0 = not at all to 3 = nearly every day).

Statistical Analyses

Summary statistics were performed to examine the mean and standard deviation (SD) for each of the ordinal variables; for the dichotomous variable gender, the frequency counts are given. First, the interrelations between sleep bruxism, awake bruxism, pain intensity, and the DC/TMD Axis II variables depression, somatic symptoms, and anxiety were assessed using Spearman correlation coefficients. Subsequently, partial Spearman correlations were calculated in order to test the hypothesis that an association exists between the intensity of clinically diagnosed TMD pain and the amount of self-reported sleep and awake bruxism, and these correlations were controlled for the potential confounding effects of depression, somatic symptoms, and anxiety. All analyses were conducted using the IBM SPSS Statistics 24 software package (IBM Corp). *P* values < .05 were considered statistically significant.

Results

Initially, 768 patients were included in the study. After removal of 326 patients without any pain in the orofacial area and 149 patients who reported orofacial pain in the questionnaire but in whom no clinical TMD pain diagnosis was established, the final sample consisted of 293 patients who were all diagnosed with at least one type of pain-related TMD. Women constituted 86% of these patients, and the mean \pm SD age of the participants was 40.3 \pm 14.7 (range 18–76) years. The descriptive statistics for sleep bruxism, awake bruxism, pain intensity, somatic symptoms, depression, and anxiety are shown in Table 1.

Spearman correlation tests provided no significant correlation between the amount of self-reported sleep bruxism and TMD pain intensity (Table 2).

Table 3	Partial Spearman Correlations Between Bruxism
	and Temporomandibular Disorder Pain when
	Controlled for Potential Confounders

	Sleep bruxism-pain intensity		Awake bruxism-pain intensity	
Controlled for:	r	Р	r	Р
Somatic symptoms	-0.015	.795	0.124	.036
Depression	-0.011	.857	0.093	.113
Anxiety	-0.001	.987	0.130	.027

On the other hand, the amount of awake bruxism was positively correlated with the intensity of TMD pain. Both types of bruxism were positively correlated with somatic symptoms, depression, anxiety, and with each other.

Even though the relationship between sleep bruxism and TMD pain intensity turned out to be nonsignificant, the partial Spearman correlations were calculated, taking into account any confounding effects of the three psychologic factors. Again, no relationship was found between the factors, as shown in Table 3. For awake bruxism, the initial significant correlation with pain intensity was lost when the model was controlled for the effects of depression. On the other hand, the correlation between awake bruxism and intensity of TMD pain remained significant after controlling for somatic symptoms and anxiety.

Discussion

This study aimed to examine whether there was a dose-response relationship between the amount of self-reported bruxism and intensity of self-reported TMD pain in a group of patients clinically diagnosed with TMD pain. To that end, the questionnaire-based reports of TMD pain intensity, as assessed by an 11-point NRS scale (0-10), were compared to the reports of sleep bruxism and awake bruxism. A positive correlation was found between the amount of awake bruxism activities and intensity of self-reported TMD pain. This correlation remained significant when the effects of somatic symptoms and anxiety were taken into account; however, depression had a confounding effect on this correlation, because significance was lost after depression was included as a factor in the model. Spearman correlation test indicated that the amount of self-reported sleep bruxism was not correlated with intensity of TMD pain.

Bruxism is assumed to have destructive effects on oral structures, such as tooth wear and dental restoration/implant fracture/failure.^{6,16,17} At the same time, overloading of the masticatory structures due to clenching and grinding of the teeth is frequently suggested as a causative factor of pain-related TMD.^{7,18} Surprisingly, even though it is clinically plausible that these phenomena are causally related, the assumed cause-effect relationship between bruxism and TMD pain is still controversial in the literature.^{8,19} So far, most studies that used polysomnography to diagnose sleep bruxism have not shown

an association between this behavior and TMD pain.^{20,21} In fact, the evidence regarding an association between sleep bruxism and TMD pain mainly comes from questionnaire studies.²²⁻²⁴ However, studies using self-report or even a clinician report of bruxism activities are potentially biased. The patient's desire to explain the presence of their jaw muscle pain as resulting from the simple-and therefore very clear and attractive-etiologic model of loading damage causing muscle pain could influence self-reported bruxism behavior. This was demonstrated in the study by van der Meulen et al, in which the majority of patients thought that their bruxism was a factor causing their facial pain.¹⁰ Additionally, a treating clinician's etiologic model has been shown to influence the patient's reports of tooth grinding.²⁵ These beliefs might have played a role in the positive dose-response correlation between self-reported awake bruxism and TMD pain found in the present study. The fact that intensity of sleep bruxism was not correlated with intensity of self-reported TMD pain could, in turn, be due to the lack of awareness about the tooth-grinding behavior that happens when patients are asleep. It has been shown that awareness of sleep-related tooth grinding is highly unlikely to be a valid indicator of bruxism behavior when compared to laboratory-based polysomnographic recording.²⁶

The fact that the initial significant correlation between awake bruxism and TMD pain intensity disappeared after controlling for depression highlights the importance of psychologic factors on the presence and intensity of TMD pain. Psychologic factors play a profound role in the presence of pain-related TMD,13,27 and considerable research has also indicated that they are associated with bruxism activities as well.28,29 From this point of view, erroneous relationships between bruxism and TMD pain may occur due to confounding of psychologic comorbidity, meaning that the obtained results do not reflect the actual relationship between the variables under study. Indeed, the observed relationship between awake bruxism and TMD pain turned out to be false in the present study since a third confounding variable (ie, depression) explained a large part of the correlation between the first two variables. Depression is a powerful emotional condition that affects the patient's

pain experience.³⁰ At the same time, patients with chronic pain are at increased risk for emotional disorders such as anxiety and depression.³¹ Therefore, it is highly recommended to include such disorders as possible confounders in future studies on the association between bruxism and TMD pain.

In future research, it would be interesting to examine how medication use affects the dose-response association under study. A significant amount of medication types are prescribed nowadays, and some have a possible effect on pain perception, psychologic status, or both. This can potentially modify the outcome of the study.

It would also be interesting to study the potential dose-response associations between intensity of self-reported bruxism activities and intensity of TMD pain, taking into account specific TMD diagnoses. It can be speculated that potential overloading due to parafunctional activities would have different effects on the different anatomical structures (ie, pain originating from muscle tissues, TMJ cartilage, or TMJ capsule).

Conclusions

This study found no significant correlation between the amount of self-reported sleep bruxism and intensity of pain-related TMD. Even though the results of the study initially suggested that there was a positive dose-response association between the amount of self-reported awake bruxism and the intensity of TMD pain, this association disappeared when controlled for the confounding effect of depression. Thus, the purported assumption that there is a dose-response gradient between bruxism and TMD pain, such that more bruxism leads to more overloading and thus to more pain, could not be justified.

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The authors report no conflicts of interest.

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