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Interaction Between Awake and Sleep Bruxism Is Associated with Increased Presence of Painful Temporomandibular Disorder

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Abstract

Aims: To explore whether awake bruxism and sleep bruxism interact with each other in their association with painful temporomandibular disorders (TMD).

Methods: In this case-control study, all participants (n=705) were part of a multicenter Validation Project and were recruited as a convenience sample of community cases and controls, and clinic cases. Logistic regression analyses were applied to test for the association of self-reported awake and sleep bruxism with the presence of painful TMD, and odds ratios (OR) with 95% confidence intervals (CI) were computed. Regression models included an interaction term to test for

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Conflict of interest statement

The authors report no conflict of interest.

multiplicative interaction. Additive interaction was calculated as the relative excess risk due to interaction (RERI).

Results: Based on logistic regression analyses adjusted for age and gender, the main effects for both awake (OR = 6.7; 95% CI: 3.4 – 12.9) and sleep (OR = 5.1; 95% CI: 3.1 – 8.3) bruxism were significant. While the multiplicative interaction (OR = 0.57; 95% CI: 0.24 – 1.4) was not significant, the results indicated a significant positive additive interaction (RERI = 8.6; 95% CI: 1.0 – 19.7) on the OR scale.

Conclusion: This study has demonstrated that awake and sleep bruxism are associated with an increased presence of painful TMD, and that both types of bruxism are not independently associated, but interact additively. As such, the presence of each factor amplifies the effect of the other.

Keywords

Temporomandibular disorders; pain; awake bruxism; sleep bruxism; interaction effect

Painful temporomandibular disorders (TMD) have a prevalence of about 10% in the general population¹ and have a substantial negative impact on patients' quality of life.² There is a need to investigate risk factors for painful TMD so as to improve the understanding of TMD etiology and to improve prevention by efficiently addressing clinically relevant risk factors. In the multifactorial etiology of painful TMD, bruxism has been recognized as an important risk factor,^{3–5} with an eight-fold increased risk of patients having TMD pain.^{3, 6–8}

Although bruxism is often considered as one entity, a distinction has to be made between sleep bruxism and awake bruxism due to their difference in etiology and pathogenesis.^{9, 10} Sleep bruxism is now considered a sleep-related movement disorder and part of a sleep arousal response,¹¹ characterized by typical patterns of rhythmic masticatory muscle activity which differ from those of chewing.^{12, 13} In contrast, awake bruxism is a non-functional behavior.

While current evidence suggests that the risk of painful TMD only slightly differs with respect to the type of bruxism,³ it is not clear whether they act as two independent risk factors or whether they affect each other in the risk of developing painful TMD. This means that it is not clear whether the risk due to one type of bruxism is modified by the presence of the other. Such an interaction is likely, since both forms of bruxism are characterized by repeated muscle activity that might lead to potential overload and micro-injuries to the temporomandibular joint (TMJ) and the masticatory muscles.^{12–14} Hence, the effects of both forms of bruxism are unlikely to be independent. Instead, a biological interaction is plausible, whereby the effect of one form of bruxism is changed when the other form is present.¹⁵ Interactions of risk factors for pain are well known.^{16–18} However, most studies assume a multiplicative interaction, while for risk factors having a similar mode of action in the multifactorial pathogenesis of a disease or disorder, additive interactions are often more likely.¹⁹ For painful TMD, a recent study suggests the presence of a multiplicative interaction.³ So far, no study has differentiated between multiplicative and additive interactions of awake and sleep bruxism. Therefore, the aim of this study was to explore

whether awake and sleep bruxism interact with each other regarding the association with painful TMD while differentiating between multiplicative and additive interactions.

Materials and Methods

Participants, study design and setting

In this case-control study, all participants were part of a multicenter Validation Project²⁰ which assessed the reliability and validity of the Research for Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) Axis I and II assessment protocol, and subsequently developed a reliable and valid revised Axis I and II protocol.²¹ Participants (n=724) were recruited as a convenience sample from community cases and controls, and clinic cases between August 2003 and September 2006 at the University of Minnesota, the University of Washington, and the University at Buffalo. Five participants who could not be classified as cases or controls, and 14 participants with comorbid systematic pain conditions (chondromatosis, fibromyalgia, or rheumatoid arthritis) were excluded from analyses, resulting in a final sample size for this study of 705 participants. For more details regarding study design, participant recruitment, and examination see Schiffman *et al.*²⁰

This research was conducted in accordance with accepted ethical standards for research practice, and underwent review and approval by the Institutional Review Board at each of the three study sites.²⁰ Written informed consent was obtained from all participants prior to their enrollment.

Assessment of TMD and allocation of case-control status

In the Validation Project, six experts in TMD and orofacial pain served as criterion examiners to establish the reference standard diagnoses based on the consensus of two examiners at each study site. Participants were assessed by using a semi-structured interview, review of responses to questionnaires and responses to diagnostic tests that were considerably more comprehensive than those specified by the RDC/TMD protocol.²⁰ Since the study aimed at exploring the association of awake and sleep bruxism with painful TMD, cases were enrolled as participants with a painful TMD diagnosis (n=500). Controls for this study had either pain-free TMD diagnoses only (n=114), or were without signs or symptoms of TMD, i.e. with a negative current history, examination, and imaging (panoramic radiograph, MRI, and CT findings; n=91).

Assessment of bruxism

Bruxism was assessed by participants' self-report on two items of the standard RDC/TMD history questionnaire.²² Participants completed the questionnaire prior to the physical examination. Awake bruxism was assessed by asking the question: 'During the day, do you grind your teeth or clench your jaw?' Sleep bruxism was assessed by asking the question: 'Have you been told, or do you notice, that you grind your teeth or clench your jaw while sleeping in the night?' Both questions had 'yes' and 'no' response options. According to an international consensus, self-reports of bruxism are classified as 'possible' bruxism.¹⁴

Assessment of psychosocial characteristics

Psychosocial characteristics of study participants were assessed with Axis II measures contained in the Validation Project,²⁰ consisting of those described in the original RDC/TMD,²² i.e. measures for depression (Depression and Vegetative Symptoms) and somatization (Nonspecific Physical Symptoms) from the revised version of the Symptom Checklist 90 (SCL-90-R),²³ and severity of chronic pain from the 7-item Graded Chronic Pain Scale (GCPS).²⁴

Additional psychosocial and behavioral characteristics were assessed, and included perceived stress that was assessed with the 10-item Perceived Stress Scale (PSS-10),²⁵ oral behaviors that were assessed with the 21-item Oral Behaviors Checklist (OBC),²⁶ and beliefs regarding explanatory pain factors that was assessed with the Explanatory Model Scale (EMS).^{27, 28}

Data analyses

For the description of the sample, sociodemographic, clinical, psychosocial, and behavioral characteristics were presented as means and standard deviations (SD) or frequencies and percentages for all three groups separately. Differences between groups were tested using ANOVA for continuous data, Kruskal-Wallis rank test for ordered data, and Chi-squared test for categorical data.

There are two ways of statistically modeling biologic interaction between awake bruxism and sleep bruxism: as a multiplicative or an additive interaction. The multiplicative scale models whether the total effect of two variables exceeds the multiplication of the two individual effects, whereas the additive scale models whether the total effect exceeds the sum of the individual effects. In epidemiologic research, the additive scale is considered to have more public health relevance and is more consistent with the concept of biologic interaction compared to the multiplicative scale.²⁹ For completeness, both scales are presented and discussed.

Due to the study design of a case-control study, unconditional logistic regression was applied to formally test the interaction. The inclusion of the interaction term in the logistic regression is equivalent to testing for a multiplicative interaction on the odds ratio (OR) scale. Given the following logistic regression model

$$\ln(\text{Odds}[TMD \text{ pain}]) = \beta_0 + \beta_1 \text{ awake bruxism} + \beta_2 \text{ sleep bruxism} + \beta_3 \text{ awake bruxism} * \text{sleep bruxism} ,$$

the additive interaction on the same scale can be calculated via the relative excess risk due to interaction (RERI)^{29, 30}:

$$RERI = OR_{11} - OR_{10} - OR_{01} + 1 = e^{\beta_1 + \beta_2 + \beta_3} - e^{\beta_1} - e^{\beta_2} + 1.$$

The 95% confidence intervals (CI) of the RERI were calculated by using the bootstrap percentile method. For that reason, 10,000 samples were drawn with replacement from the original data, each of the same size as the original sample. Models are presented with and without adjustment for age quartiles and gender.

Additionally, sensitivity analyses were conducted, by applying the same models, adjusted for all potential confounders but based only on the subset of controls with no sign or symptoms of TMD.

Only 3 (0.4%) participants had missing information for awake bruxism and sleep bruxism and were therefore excluded from analyses modelling the risk of TMD pain, resulting in a total of 702 participants with complete data (controls without TMD: n=91, controls with pain-free TMD: n=113, cases with painful TMD: n=498).

All analyses were performed using the statistical software STATA/MP (Stata Statistical Software: Release 13.1, StataCorp LP, College Station, TX, USA), with the probability of a type I error set at the .05 level.

Results

Characteristics of participants

Mean age was 35.8 years in controls without signs and symptoms of TMD, 39.3 years in controls with pain-free TMD, and 36.4 years in painful TMD cases (Table 1). While the mean age of participants was not statistically significantly different between the groups, the proportion of women significantly differed ($P<.001$), with the highest proportion in cases and lowest in those controls without signs or symptoms of TMD. The vast majority of participants had at least one year of college education with non-significant differences between case and control groups ($P=.954$).

Almost all cases (99%) were diagnosed with muscle pain (myofascial pain with or without limited mouth opening), and the majority (87%) also had joint pain (arthralgia or osteoarthritis). However, all other possible TMD diagnoses were also observed in this group; osteoarthritis had the lowest prevalence (11%). In controls with TMD, by definition, no pain-related TMD diagnoses were present, and slightly more than two-thirds of these participants were diagnosed with disc displacement with reduction.

Cases differed significantly from both control groups for psychosocial and behavioral characteristics including higher levels of somatization ($P<.001$), higher levels of depression ($P<.001$), and higher scores of the OBC ($P<.001$) and the PSS-10 ($P<.001$). About 13% of the cases (but none of the controls) exhibited signs of dysfunctional chronic pain (GCPS Grade 3 or 4). For the EMS, behavioral factors were rated highest.

Frequency of awake and sleep bruxism

The minority of controls indicated that they were aware of awake bruxism or sleep bruxism (controls without TMD: 30%; controls with pain-free TMD: 39%). In contrast, this proportion was substantially larger in cases (84%). Slightly more than half of the cases

(54%) reported having knowledge of both awake and sleep bruxism, while only 10% of controls without TMD and 12% of controls with pain-free TMD indicated having both forms.

Association between bruxism and TMD pain

Crude OR estimates are displayed in Table 2. Based on logistic regression without potential confounders and interaction term, the ORs for both awake bruxism (5.1) and sleep (4.2) bruxism were significant, indicating that both are considerably associated with painful TMD (Model 1, Table 3). The additional interaction term was below 1, suggesting a negative multiplicative interaction on the OR scale, however this effect was not significant (Model 2, Table 3). The RERI on the other hand was significantly above 0, indicating a positive additive interaction on the OR scale. Adjusting for all potential confounders did not substantially change the ORs of the main effects of awake bruxism and sleep bruxism, nor the interaction effects (Model 3, Table 3).

The sensitivity analysis through exclusion of controls with pain-free TMD did not lead to substantially different results. The ORs for the main effects were somewhat higher (awake bruxism: 7.9; sleep bruxism: 6.2). The interaction on a multiplicative scale remained insignificant (OR: 0.42; 95% CI: 0.11 – 1.6), but was no longer significant on the additive scale (RERI: 9.0; 95% CI: –10.4 – 36.3).

Discussion

This is the first study to test for an additive interaction in the association of self-reports of awake bruxism and sleep bruxism with painful TMD. The findings of this study indicate that both awake bruxism and sleep bruxism are considerably associated with painful TMD, and that their total effect exceeds the sum of the individual effects.

The crude OR for the presence of painful TMD in the case of the co-occurrence of awake bruxism and sleep bruxism was substantially larger than the sum of the individual crude ORs for both types of bruxism. Therefore, awake bruxism and sleep bruxism are not just two representations of the same risk factor, but each type comprises a specific risk of having painful TMD. Concurrently, the two types are not independent but interact in that the risk of one type of bruxism is modified by the presence of the other type. Such an additive interaction is very likely for risk factors having a similar mode of action in the multifactorial pathogenesis of a disease or disorder.¹⁹

The observed increased presence of painful TMD due to awake bruxism and sleep bruxism was essentially in accordance with those observed in other studies. Fernandes *et al.* reported an increased presence of myofascial pain (OR = 5.9) and arthralgia (OR = 2.3) in TMD patients with sleep bruxism compared to those without.⁶ Michelotti *et al.* observed an increased presence of myofascial pain (OR = 4.9) in TMD patients with awake bruxism compared to controls without TMD and without awake bruxism.⁸ Huang *et al.* reported an OR of 4.8 for the association between clenching and myofascial pain and 3.3 for the association of clenching and myofascial pain plus arthralgia compared to subjects without

clenching.³¹ Although the actual OR were somewhat higher in the present study than presented elsewhere,³² this was potentially only due to methodological reasons.

The authors had previously investigated a potential interaction between awake bruxism and sleep bruxism,³ and found evidence for an interaction on the multiplicative scale. However, this is not contradictory to the present findings since the primary analytical approach in the previous study³ did not involve testing for an additive interaction. When using the original data of the previous study and applying the methodology of the present study, a significant additive interaction was observed (RERI = 5.8; 95% CI: 3.8 – 8.6), supporting the validity of the present findings.

The strengths of the present study included the large samples sizes for cases and controls that allowed the identification of significant effects. Also, all study participants were examined twice by experienced and reliable examiners according to the Validation Project assessment protocol. The final TMD diagnoses were based on the consensus between the two examiners and the radiologists' interpretation at each site,³³ ensuring high validity. Furthermore, since the TMD diagnoses in the present study served as the reference standard diagnoses in the Validation Project²⁰ to develop reliable and valid revised DC/TMD Axis I diagnostic algorithms,²¹ the study's diagnoses are more accurate than diagnosis rendered from any resulting diagnostic algorithm. A sensitivity analysis was performed to test whether the findings were related to the definition of the case-control status. The effect for the additive interaction (RERI) changed only slightly, but the 95% CI included 0. Thus, the additive interaction in the sensitivity analysis became statistically insignificant. However, this does not contradict the present findings since it is probably the result of the reduced statistical power due to the exclusion of a substantial number of subjects (number of controls decreased from 200 to 89).

The major limitations of this study related to the nature of case-control studies. In these settings, only the OR can be used as an effect measure, as the prevalence of the disease is fixed by design. Subsequently, the OR is interpreted as a relative risk, which is an approximation based on the assumption that the investigated disease has a low prevalence. Furthermore, case-control studies are retrospective and as cases search for an explanation of their disease, they may be more aware of risk factors and therefore the prevalence of the risk factor in cases is sometimes overestimated due to recall bias. Similarly, health professionals want to provide their patients with a reason for their disease and may therefore inform them that bruxism is a risk factor for the onset and perpetuation of painful TMD. The potential bias due to these limitations is not known. However, in this study, the questions regarding bruxism were a small part of the psychosocial and behavioral assessments and were not the only factor focused on by participants. Most cases (76%) came from the community and were respondents to study flyers and advertisements.²⁰ Only a small proportion (24%) was referred to the university-based TMD clinics from local health professionals, limiting the potential of recall bias since most cases had probably not seen a doctor for their painful TMD before. While the data for this study was collected years in advance of the present study's analyses, there seems no reason why the study's findings of the presence of the additive interaction should change if the data had been analyzed sooner. Furthermore, if new data were collected now by using a different sample, then it would be expected that the

strength of the association would change somewhat but it would not change the main finding of an additive interaction. A final limitation of a case-control study is the lack of ability to establish a causal relationship between sleep bruxism and awake bruxism with painful TMD.

Study participants were recruited as a convenience sample from both clinical and community sources. Participants were selected based on methodological considerations of the Validation Project, that is, to ensure a sufficient number of participants for each of the TMD diagnoses and to have a full spectrum of cases to improve generalizability of results.²⁰ Therefore, the sample is not representative for TMD patients or the general population but is representative of the full spectrum of cases with painful TMD. However, it is not expected that findings would substantially differ if random samples had been included. This assumption is supported by the accordance of the present findings with those of the study by Sierwald *et al.*,³ which included a random sample of the general population and a sample of TMD patients.

Awake bruxism and sleep bruxism were assessed as participants' self-reports. While bruxism can be diagnosed based on self-reports ('possible' bruxism), clinical examination ('probable' bruxism), electromyography and polysomnographic recording ('definite' bruxism),^{14, 34} most studies use self-reports assessed by means of questionnaires only. Even though the two items used to assess bruxism in the present study are part of the RDC/TMD,²² and are therefore highly standardized, validity is limited.^{14, 35, 36} However, bruxism assessment based on clinical examinations depends on the presence of clinical findings that are considered to be associated with bruxism, e.g. extensive tooth attrition or muscle hypertrophy. Such findings occur variably even in subjects with persistent, chronic bruxism, and are not indicative of the current status. Electromyography and polysomnography require extensive equipment, and are therefore only suitable for a small subject group. Furthermore, such a gold standard assessment is currently only available for sleep bruxism but not for awake bruxism.^{34, 37} Interestingly, cut-off points for a polysomnographic assessment of sleep bruxism were developed by using a case definition requiring a history of frequent tooth grinding occurring at least 3 nights per week for the preceding 6 months, as confirmed by a sleep partner, clinical presence of tooth wear, masseter muscle hypertrophy, and report of jaw muscle fatigue or tenderness in the morning.³⁷ As mentioned above, not all subjects with bruxism fulfill all these criteria simultaneously, which might at least in part explain the insufficient concordance between self-report and polysomnographic assessment of sleep bruxism.³⁶ Accordingly, based on current knowledge regarding advantages and drawbacks of the methods described above, participants' self-reports are probably the most feasible approach for the assessment of sleep bruxism and awake bruxism in a study setting with a large population such as that used in the present study. Furthermore, the applied items are commonly used, thus ensuring comparability of study findings. This will also help to transfer findings from scientific research into settings in which oral health care is actually delivered to the patient.

Conclusions

This study has demonstrated that awake bruxism and sleep bruxism are both associated with an increased presence of painful TMD and that both types of bruxism are not independent, but interact additively, i.e. the effect of one factor depends on the presence of the other.

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Table 1 –
Participant Sociodemographic, Clinical, Psychosocial, and Behavioral Characteristics

	Controls		Cases		
	w/o TMD	pain-free TMD	painful TMD		
	n=91	n=114	n=500		Significance [†]
	Mean (SD) or N (%)				P-value
Demography					
Age [years]	35.8 (12.7)	39.3 (13.1)	36.4 (13.1)		.076
Gender [female]	57 (62.6)	88 (77.2)	434 (86.8)		<.001
Education					
No college	15 (16.5)	17 (14.9)	78 (15.6)		.954
1 year of college	76 (83.5)	97 (85.1)	421 (84.4)		
Household income per year					
Less than \$50,000	63 (70.0)	68 (60.2)	268 (54.4)		.008
\$50,000 – \$79,999	19 (21.1)	25 (22.1)	121 (24.5)		
\$80,000 or more	8 (8.9)	20 (17.7)	104 (21.1)		
TMD diagnoses					
					n/a
Myofascial pain w/o limited opening	n/a	n/a	210 (42.0)		
Myofascial pain with limited opening	n/a	n/a	285 (57.0)		
Disc displacement with reduction	n/a	82 (71.9)	280 (56.0)		
Disc displacement w/o reduction with limited opening	n/a	2 (1.8)	66 (13.2)		
Disc displacement w/o reduction w/o limited opening	n/a	31 (27.2)	162 (32.4)		
Arthralgia	n/a	n/a	302 (60.4)		
Osteoarthritis	n/a	n/a	169 (33.8)		
Osteoarthritis	n/a	39 (34.2)	54 (10.8)		
Nonspecific physical symptoms (somatization)					
					<.001
Low	83 (92.2)	100 (87.7)	238 (47.8)		
Moderate	6 (6.7)	12 (10.5)	167 (33.5)		
Severe	1 (1.1)	2 (1.8)	93 (18.7)		
Depression and vegetative symptoms					
					<.001
Low	73 (81.1)	95 (84.1)	300 (60.2)		
Moderate	13 (14.4)	15 (13.3)	134 (26.9)		
Severe	4 (4.4)	3 (2.7)	64 (12.9)		
Graded chronic pain					
					n/a
Grade 1 (low disability – low intensity pain)	n/a	n/a	208 (42.4)		
Grade 2 (low disability – high intensity pain)	n/a	n/a	221 (45.0)		
Grade 3 (high disability – moderately limiting)	n/a	n/a	36 (7.3)		
Grade 4 (high disability – severely limiting)	n/a	n/a	26 (5.3)		
Oral behaviors					
OBC sum score	15.3 (6.3)	17.5 (7.4)	25.8 (8.9)		<.001
Perceived stress					
PSS–10 sum score	10.6 (5.6)	10.4 (5.7)	13.6 (7.0)		<.001

	Controls		Cases	
	w/o TMD	pain-free TMD	painful TMD	
	n=91	n=114	n=500	Significance [†]
	Mean (SD) or N (%)			P-value
Explanatory model scale				
Physical factors	n/a	n/a	1.4 (1.4)	n/a
Behavioral factors	n/a	n/a	2.8 (1.2)	n/a
Stress or emotional upset	n/a	n/a	1.9 (1.3)	n/a

w/o – without, n/a – not applicable,

[†] ANOVA for age, OBC summary score, PSS-10 summary score; Kruskal-Wallis rank test for annual income, nonspecific physical symptoms, depression and vegetative symptoms; Chi-squared test for gender, level of education

Table 2 –

Crude Odds and Odds Ratios for TMD pain

Awake bruxism	Sleep bruxism	
	No	Yes
No	Odds ₀₀ = 0.59 (OR ₀₀ = 1)	Odds ₀₁ = 2.88 (OR ₀₁ = 4.91)
Yes	Odds ₁₀ = 3.93 (OR ₁₀ = 6.70)	Odds ₁₁ = 11.61 (OR ₁₁ = 19.79)

OR – odds ratio

Table 3 –

Bruxism and painful TMD: Results of Multiple Logistic Regression with Awake Bruxism and Sleep Bruxism as Independent Predictors (Model 1), with Interaction between Awake Bruxism and Sleep Bruxism (Model 2), and Adjusted for Sociodemographic, Socioeconomic, and Psychosocial Characteristics (Model 3) Awake bruxism#sleep bruxism indicates the interaction term

	Odds ratio / RERI	95% CI
Model 1		
Awake bruxism	5.1	3.3 – 7.8
Sleep bruxism	4.2	2.8 – 6.2
Model 2		
Awake bruxism	6.7	3.5 – 12.8
Sleep bruxism	4.9	3.0 – 7.9
Awake bruxism#sleep bruxism:		
Multiplicative Interaction	0.60	0.25 – 1.4
Additive Interaction (RERI)	9.5	1.3 – 19.7
Model 3		
Awake bruxism	6.7	3.4 – 12.9
Sleep bruxism	5.1	3.1 – 8.3
Awake bruxism#sleep bruxism:		
Multiplicative Interaction	0.57	0.24 – 1.4
Additive Interaction (RERI)	8.6	1.0 – 19.7
Age		
First quartile (18 – 25 yrs) *	–	–
Second quartile (26 – 35 yrs)	0.80	0.46 – 1.4
Third quartile (36 – 48 yrs)	0.64	0.37 – 1.1
Fourth quartile (49 – 67 yrs)	0.63	0.37 – 1.1
Gender [female]	2.1	1.3 – 3.3

* reference category

OR – odds ratio; RERI – relative excess risk due to interaction, CI – confidence interval