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

Daniel R. Reissmann, DDS, Dr Med Dent, PhD
Associate Professor
Department of Prosthetic Dentistry, Center for Dental and Oral Medicine
University Medical Center Hamburg-Eppendorf
Hamburg, Germany

Mike T. John, DDS, MPH, PhD
Associate Professor
Division of Oral Medicine, Diagnosis and Radiology
Department of Diagnostic and Biological Sciences, School of Dentistry
University of Minnesota;
Adjunct Associate Professor
Division of Epidemiology and Community Health, School of Public Health
University of Minnesota
Minneapolis, Minnesota, USA

Annette Aigner, MA, MSc
Research Associate

Gerhard Schön, Dipl-Soz
Research Associate

Institute of Medical Biometry and Epidemiology
University Medical Center Hamburg-Eppendorf
Hamburg, Germany

Ira Sierwald, DDS, Dr Med Dent
Assistant Professor
Department of Orthodontics, Dentofacial Orthopedics, and Pedodontics
Charité-Universitätsmedizin Berlin
Berlin, Germany

Eric L. Schiffman, DDS, MSc
Professor
Division of TMD and Orofacial Pain
Department of Diagnostic and Biological Sciences, School of Dentistry
University of Minnesota
Minneapolis, Minnesota, USA

Correspondence to:
Dr Daniel R. Reissmann
Department of Prosthetic Dentistry
Center for Dental and Oral Medicine
University Medical Center Hamburg-Eppendorf
Martinistrasse 52, 20246 Hamburg, Germany
Fax: +49-40-7410 57077
Email: d.reissmann@uke.de

Aims: To explore whether awake and sleep bruxism interact in their associations with painful temporomandibular disorders (TMD) and whether the interaction is multiplicative or additive. **Methods:** In this case-control study, all participants (n = 705) were part of the 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 between self-reported bruxism (sleep and/or awake) and the presence of painful TMD, and odds ratios (ORs) with 95% confidence intervals (95% CIs) were computed. Regression models included an interaction term to test for multiplicative interaction, and 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 to 12.9) and sleep (OR = 5.1; 95% CI: 3.1 to 8.3) bruxism were significant. While the multiplicative interaction (OR = 0.57; 95% CI: 0.24 to 1.4) was not significant, the results indicated a significant positive additive interaction (RERI = 8.6; 95% CI: 1.0 to 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. *J Oral Facial Pain Headache 2017;31:299–305. doi: 10.11607/ofph.1885*

Keywords: *awake bruxism, interaction effect, pain, sleep bruxism, temporomandibular disorders*

Painful temporomandibular disorders (TMD) have a prevalence of about 10% in the general population[1] and a substantial negative impact on quality of life (QoL).[2] There is a need to investigate risk factors for painful TMD to improve the understanding of their etiology so that clinically relevant risk factors can be addressed for its prevention. Bruxism has been recognized as an important risk factor for TMD,[3–5] with bruxism patients having an eight-fold increased risk for TMD pain.[3,6–8]

Although bruxism is often considered one entity, a distinction must be made between awake and sleep bruxism due to their differences 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 that differ from those of chewing.[12,13] In contrast, awake bruxism is a nonfunctional behavior.

While current evidence suggests that the risk of painful TMD differs only slightly with respect to the type of bruxism,[3] it is not clear if these two forms of bruxism act as two independent risk factors or if they affect each other in the risk of developing painful TMD; ie, 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 masticatory muscles.[12–14] Hence, the effects of both forms of bruxism are unlikely to be independent. It is plausible that there is instead a biologic interaction, whereby the effect of one form of bruxism is changed

when the other form is present.[15] Interactions between risk factors for pain are well known[16–18]; however, while most studies assume a multiplicative interaction, additive interactions are often more likely for risk factors having a similar mode of action in the multifactorial pathogenesis of a disease or disorder.[19] A recent study suggests the presence of a multiplicative interaction for painful TMD,[3] but no study has differentiated between multiplicative and additive interactions of awake and sleep bruxism. Therefore, the aims of this study were to explore whether awake and sleep bruxism interact with each other regarding their associations with painful TMD and whether the interaction is multiplicative or additive.

Materials and Methods

Participants, Study Design, and Setting

In this case-control study, all participants were part of the multicenter Validation Project[20] that assessed the reliability and validity of the Research 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 systemic pain conditions (chondromatosis, fibromyalgia, or rheumatoid arthritis) were excluded from the analyses, resulting in a final sample size of 705 participants. For more details regarding study design and 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 or 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 with a semi-structured interview and a review of their responses to questionnaires and diagnostic tests, which were considerably more comprehensive than those specified by the RDC/TMD protocol.[20] Since the study aimed to explore the association between awake and sleep bruxism and painful TMD, participants with a painful TMD diagnosis were considered cases (n = 500). Controls for this study had either a pain-free TMD diagnosis (n = 114) or were without signs and symptoms of TMD (ie, with a negative current history, examination, and imaging [panoramic radiograph, magnetic resonance imaging (MRI), and computed tomography (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. Self-reports of bruxism were classified as possible bruxism, according to an international consensus.[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] (ie, 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, including perceived stress (assessed with the 10-item Perceived Stress Scale [PSS-10]),[25] oral behaviors (assessed with the 21-item Oral Behaviors Checklist [OBC]),[26] and beliefs regarding explanatory pain factors (assessed with the Explanatory Model Scale [EMS]).[27,28]

Data Analyses

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

There are two ways of statistically modeling the biologic interaction between awake 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, and 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 be more consistent with the concept of biologic interaction than the multiplicative scale.[29] In the present study, both scales are presented and discussed.

Due to the 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 . . .

$$\ln(Odds[TMD\ pain]) = \beta_0 + \beta_1\ awake\ bruxism + \beta_2\ sleep\ bruxism + \beta_3\ awake\ bruxism \times sleep\ bruxim$$

. . . 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 (95% CIs) 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, which were adjusted for all potential confounders but based only on the subset of controls with no signs or symptoms of TMD.

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

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

Results

Participant Characteristics

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 the lowest in controls without signs or symptoms of TMD. The vast majority of participants had at least 1 year of college education, with nonsignificant 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, with osteoarthrosis showing the lowest prevalence (11%). By definition, no pain-related TMD diagnoses were present in the TMD controls, but 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 on 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). On the EMS, behavioral factors were rated highest.

Frequency of Awake and Sleep Bruxism

The minority of controls indicated that they were aware of awake or sleep bruxism (controls without TMD = 30%; controls with pain-free TMD = 39%). 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 (5.1) and sleep bruxism (4.2) 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 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

Table 1 Sociodemographic, Clinical, Psychosocial, and Behavioral Characteristics of Participants

	Controls W/o TMD n = 91	Cases Pain-free TMD n = 114	Cases Painful TMD n = 500	Significance[a] *P* value
Demographic				
Age (y), mean (SD)	35.8 (12.7)	39.3 (13.1)	36.4 (13.1)	.076
Gender (female), n (%)	57 (62.6)	88 (77.2)	434 (86.8)	< .001
Education, n (%)				.954
No college	15 (16.5)	17 (14.9)	78 (15.6)	
≥ 1 year of college	76 (83.5)	97 (85.1)	421 (84.4)	
Household income per year, n (%)				.008
< $50,000	63 (70.0)	68 (60.2)	268 (54.4)	
$50,000–$79,999	19 (21.1)	25 (22.1)	121 (24.5)	
≥ $80,000	8 (8.9)	20 (17.7)	104 (21.1)	
TMD diagnoses, n (%)				NA
Myofascial pain w/o limited opening	NA	NA	210 (42.0)	
Myofascial pain with limited opening	NA	NA	285 (57.0)	
Disc displacement with reduction	NA	82 (71.9)	280 (56.0)	
Disc displacement w/o reduction with limited opening	NA	2 (1.8)	66 (13.2)	
Disc displacement w/o reduction w/o limited opening	NA	31 (27.2)	162 (32.4)	
Arthralgia	NA	NA	302 (60.4)	
Osteoarthritis	NA	NA	169 (33.8)	
Osteoarthrosis	NA	39 (34.2)	54 (10.8)	
Nonspecific physical symptoms (somatization), n (%)				< .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, n (%)				< .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 (%)				NA
Grade 1 (low disability, low-intensity pain)	NA	NA	208 (42.4)	
Grade 2 (low disability, high-intensity pain)	NA	NA	221 (45.0)	
Grade 3 (high disability, moderately limiting)	NA	NA	36 (7.3)	
Grade 4 (high disability, severely limiting)	NA	NA	26 (5.3)	
Oral behaviors, mean (SD)				
OBC sum score	15.3 (6.3)	17.5 (7.4)	25.8 (8.9)	< .001
Perceived stress, mean (SD)				
PSS-10 sum score	10.6 (5.6)	10.4 (5.7)	13.6 (7.0)	< .001
Explanatory model scale, mean (SD)				
Physical factors	NA	NA	1.4 (1.4)	NA
Behavioral factors	NA	NA	2.8 (1.2)	NA
Stress or emotional upset	NA	NA	1.9 (1.3)	NA

OBC = Oral Behaviors Checklist; PSS = Perceived Stress Scale. [a]Differences between groups were calculated using analysis of variance (ANOVA) for age, OBC summary score, and PSS-10 summary score; Kruskal Wallis rank test for annual income, nonspecific physical symptoms, and depression and vegetative symptoms; and chi-square test for gender and level of education. NA = not applicable.

Table 2 Crude Odds and Odds Ratios (OR) for TMD Pain

	Sleep bruxism	
Awake 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)

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 to 1.6), but was no longer significant on the additive scale (RERI: 9.0; 95% CI: –10.4 to 36.3).

Discussion

This is the first study to test for an additive interaction in the associations between both self-reported awake and sleep bruxism and painful TMD. The findings of this study indicate that both awake 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 and sleep bruxism was substantially larger than the sum of the individual crude ORs for both types of bruxism. Therefore, awake and sleep bruxism are not just two representations of the same risk factor; rather, 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 with a similar mode of action in the multifactorial pathogenesis of a disease or disorder.[19]

The increased presence of painful TMD due to awake and sleep bruxism observed 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 controls 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 between clenching and myofascial pain plus arthralgia compared to subjects without clenching.[31] Although the actual ORs were somewhat higher in the present study than presented elsewhere,[32] this was potentially only due to methodologic reasons.

The authors had previously investigated a potential interaction between awake 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 study[3] 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 to 8.6), supporting the validity of the present findings.

The strengths of the present study included the large sample sizes for cases and controls, which 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' interpretations 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 Project[20] to develop reliable and valid revised RDC/TMD Axis I diagnostic algorithms,[21] the present 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 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 the present study were related to the nature of case-control studies. In these settings, as the prevalence of the disease is fixed by design, only the OR can be used as an effect measure. 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. As cases search for an explanation of their disease, they may be more aware of risk factors; therefore, the prevalence of the risk factors in cases is

Table 3 Bruxism and Painful TMD: Results of Multiple Logistic Regression with Awake and Sleep Bruxism as Independent Predictors (Model 1), Interaction Between Awake and Sleep Bruxism (Model 2), and Adjusted for Sociodemographic, Socioeconomic, and Psychosocial Characteristics (Model 3)

	OR/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	3.5–2.8
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 y)[a]	–	–
Second quartile (26–35 y)	0.80	0.46–1.4
Third quartile (36–48 y)	0.64	0.37–1.1
Fourth quartile (49–67 y)	0.63	0.37–1.1
Gender (female)	2.1	1.3–3.3

OR = odds ratio; RERI = relative excess risk due to interaction; 95% CI = 95% confidence interval. Awake bruxism/sleep bruxism indicates the interaction term. [a]Reference category.

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%) were 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 were collected years in advance of the present study's analyses, there seems to be no reason that the study's findings regarding the additive interaction should change if the data had been analyzed sooner. Furthermore, if new data were collected now 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 and awake bruxism and painful TMD.

Study participants were recruited as a convenience sample from both clinical and community sources. Participants were selected based on methodologic 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 the generalizability of results).[20] Therefore, the sample is not representative of 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 and sleep bruxism were assessed based on participant self-reports. While bruxism can be diagnosed based on self-reports (possible bruxism), clinical examinations (probable bruxism), and electromyographic and polysomnographic recordings (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 (eg, extensive tooth attrition or muscle hypertrophy). Such findings occur variably, even in subjects with persistent chronic bruxism, and are not indicative of 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.[34,37] Interestingly, cut-off points for a polysomnographic assessment of sleep bruxism were developed 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 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 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; ie, the effect of one factor depends on the presence of the other.

Acknowledgments

Research reported in this publication was supported by the National Institute of Dental and Craniofacial Research of the National Institutes of Health under Award Number U01DE013331 and by the German Research Foundation under Award Number RE 3289/2-1. The authors report no conflict of interest. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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