The Relationship Between Sleep Bruxism Behavior and Salivary Stress Biomarker Level

Michiko Makino, DDS[a]/Chihiro Masaki, DDS, PhD[b]/Kei Tomoeda, DDS[a]/Elyes Kharouf, DDS, PhD[c]/
Tetsuji Nakamoto, DDS, PhD[b]/ Ryuji Hosokawa, DDS, PhD[d]

Purpose: Bruxism and parafunctions are potential risk factors for implant and prosthodontic treatment failure. However, the etiology of bruxism remains unknown. This study sought to clarify the relationship between bruxism behavior and a salivary stress biomarker level. ***Materials and Methods:*** Forty-six volunteers (23 men, 23 women) participated in this study. Bruxism behavior was assessed using a self-administered questionnaire, study casts, and BiteStrip, a miniature electronic screener used to detect sleep bruxism. The questionnaire consisted of six items asking about bruxism, occlusion, jaw functional limitation, and dentition. Occlusal wear was assessed from dental casts and calculated as the sum of the facets in the maxillary arch segment. Participants used BiteStrip at home for one night and the score was evaluated. Two-minute stimulated whole saliva samples were collected from participants to measure daytime levels of chromogranin A (CgA), a major salivary stress biomarker. Nonparametric tests of the relationship between variables were performed using the Spearman *R* and Kendall τ statistical correlation tests. ***Results:*** There was a positive correlation between self-reported bruxism and self-reported jaw functional limitation. Occlusal wear did not significantly correlate with occlusion, jaw functional limitation, or dentition, but it did significantly correlate with self-reported bruxism behavior, as well as the BiteStrip score. It was interesting to find that the CgA level was significantly negative in correlation with the BiteStrip score. ***Conclusion:*** Sleep bruxism is believed to be a stress-related sleep disorder. The results suggest that daytime psychological stress level is significantly negative in correlation with sleep bruxism behavior. *Int J Prosthodont 2009;22:43–48.*

Bruxism is a very common parafunction of the masticatory system and is defined as unconscious gnashing, grinding, or clenching of the teeth.[1] Although often described together, it has been acknowledged that 2 components of occlusal parafunction, namely diurnal and nocturnal bruxism, may be distinguished from one another, probably with different etiologies.[2]

[a]*PhD Candidate, Department of Oral Reconstruction and Rehabilitation, Kyushu Dental College, Graduate School, Fukuoka, Japan.*
[b]*Assistant Professor, Department of Oral Reconstruction and Rehabilitation, Kyushu Dental College, Fukuoka, Japan.*
[c]*Visiting Research Fellow, Department of Oral Reconstruction and Rehabilitation, Kyushu Dental College, Fukuoka, Japan.*
[d]*Professor, Department of Oral Reconstruction and Rehabilitation, Kyushu Dental College, Fukuoka, Japan.*

Correspondence to: *Dr Ryuji Hosokawa, 2-6-1 Manazuru, Kokurakita, Kitakyushu 803-8580, Japan. Fax: +81-93-592-3230. E-mail: hosokawa@kyu-dent.ac.jp*

Examples of problems associated with bruxism often discussed in the literature[2–4] are tooth wear, muscular pain, temporomandibular joint pain, toothache, mobile teeth, headaches, tooth survival in periodontitis,[5] cracks in posterior teeth,[6] implant failure,[7] and complications with fixed partial dentures on implants.[8]

Currently, there is a general consensus regarding the multifactorial nature of the etiology of bruxism. It is thought to be a central nervous system phenomenon related to stress and pain behavior rather than structural components.[9,10] There have been many experimental studies attempting to better understand this parafunction. For example, according to Huynh et al,[11] clonidine, a selective alpha2-adrenergic agonist, could reduce sleep bruxism by preventing the sequence of autonomic to motor activation of sleep bruxism. Landry et al reported that short-term use of a temporary, custom-fit mandibular advancement device was associated with a remarkable reduction in sleep bruxism motor activity.[12] In addition, Huynh et al also showed that a temporary

Table 1 Self-Administered Questionnaire Examining Sleep Bruxism

	Answer (%)	
Question	Yes	No
1. Do you feel any instability when you bite?	13	87
2. Do you feel any difficulty when opening your mouth or moving your jaw?	22	78
3. Do you feel any discomfort about the height of your bite?	15	85
4. Do you feel any dissatisfaction about your alignment of teeth?	61	39
5. Do you have a habit of grinding or strongly biting your teeth?	43	57
6. Do you have a habit of chewing on one side of your mouth?	61	39

Table 2 Ordinal Scale Used for Grading Severity of Occlusal Wear

Scale	Description
0	No visible facets in enamel. Occlusal/incisal morphology intact.
1	Marked wear facets in the enamel. Occlusal/incisal morphology altered.
2	Wear into the dentin. The dentin exposed occlusally/incisally or adjacent tooth surface. Occlusal/incisal morphology changed in shape with height reduction of the crown.
3	Extensive wear into the dentin. Larger dentin area (> 2 mm^2) exposed occlusally/ incisally or adjacent tooth surface. Occlusal/incisal morphology totally lost locally or generally. Substantial loss of crown height.
4	Wear into secondary dentine (verified by photographs).

custom-fit mandibular advancement device and clonidine together were even more promising experimental treatments for sleep bruxism, but further longitudinal trials were needed.[13] Thus, findings on the etiology and treatment of sleep bruxism are inconclusive.

For decades, it has been claimed that occlusal disturbances provoke bruxism. Recently, some reviews have suggested that the hypothesis claiming that the presence of some occlusal characteristics can trigger parafunctional activities seems to have no biological plausibility. Others have suggested that the etiology of bruxism might shift from occlusal to psychological-based theories.[14] Some authors suggest that sleep bruxism functions as a form of stress management.[15] However, the relationship between bruxism and subjective stress has remained unclear in clinical studies.

Recently, salivary biomarkers such as chromogranin A (CgA), cortisol, and secretory immunoglobulin A (sIgA) have been used to assess stress reactions. CgA is an acidic glycoprotein located in the secretory granules of a wide variety of endocrine and neuronal tissues.[16–18] Moreover, CgA is co-released with catecholamines during exocytosis from sympathetic nerve terminals and chromaffin cells. Nakane et al demonstrated that salivary CgA can be a quantitative index for monitoring the activity of the sympathetic nervous system. This constitutes the initial alarm reaction phase in stress response. In particular, the level of salivary CgA provides a sensitive and reliable index for evaluating psychological stress.[19–21]

If psychological stress promotes bruxism, CgA could be a suitable marker for clinical examination for parafunction. The objective of this study is to clarify the relationship between bruxism behavior and CgA level in saliva.

Materials and Methods

Forty-six subjects (ages 21 to 45, mean: 23.9 years) randomly selected from the third-year undergraduates of Kyushu Dental College participated in this study. No subject wearing a conventional full or partial denture was excluded. Instead, subjects were excluded if they had gross malocclusion on the basis of the study cast examination. There was no regard for any history of bruxism.

The self-administered questionnaire was independently completed by the subjects and was based on a previous study by Agerberg et al.[22] The questionnaire consisted of 6 items asking about bruxism, occlusion, jaw functional limitation, and dentition (Table 1).

In order to determine the presence of any indicators of bruxism, occlusal wear was assessed from dental casts and calculated as the sum of the facets in the maxillary arch segment. All scorings were performed by the same examiner. The Carlsson ordinal scale was used to survey occlusal tooth wear from each dental cast (Table 2).[23] Teeth yielding a score higher than 1 were counted and considered to be attrition scores.

BiteStrip was used to measure the severity of sleep bruxism.[24] BiteStrip is a miniature single-use electronic screener for sleep bruxism. It is composed of electromyogram (EMG) electrodes and an amplifier to acquire muscle signals and a CPU with real-time software, which detects and analyzes EMG patterns. All subjects were instructed to perform 4 to 5 maximum voluntary

clenches (MVCs) to establish an individual threshold for bruxing. Bruxism events were scored as such that a phasic and/or tonic increase of EMG tonus exceeded 30% of the MCV and lasted longer than 0.25 seconds. Lavigne et al suggested the polysomnographic diagnostic cut-off criteria that were used in this study: *(1)* more than 4 bruxing episodes per hour, *(2)* more than 25 bruxism bursts per hour of sleep, and *(3)* at least 2 episodes with grinding sounds.[25] The BiteStrip cut-off criterion is considered to be 25 bursts per hour (equivalent to 125 bursts per 5 hours of sleep) to establish severe bruxism. In the morning, after removal of the device, the total number of bruxing events throughout the night was displayed (Table 3).

After subjects measured the severity of their bruxism by using BiteStrip, 2-minute stimulated whole saliva samples were collected from them. Subjects chewed on a small piece of cotton for 2 minutes at their own rhythm, after which the cotton piece was placed into a tube. After the collection, the samples were kept in an icebox and immediately transported to the laboratory and centrifuged, where they were stored at -30°C until analysis. The concentration of CgA (pmol/mL) was measured using the YK070: CgA (Human) EIA kit (Yanaihara Institute). The Bio-Rad Protein assay kit was used to determine the protein concentration (mg/mL) in the saliva samples. CgA levels were corrected by protein concentration and expressed as pmol/mg protein.

Data analysis was performed using SPSS 13.0 for Windows. Paired sample *t* tests and non-parametric tests of the relationship between variables were performed using the Spearman *R* and Kendall τ tests, and the level of statistical significance was set at $P < .05$.

Results

Questionnaire

Results of the self-administered questionnaire are shown in Table 1. According to this questionnaire, 13% of those examined reported that they felt some discomfort during occlusion (Q1), 22% reported jaw functional limitation (Q2), 15% reported dissatisfaction about the height of their teeth (Q3), 61% reported dissatisfaction about their dentition (Q4), 43% reported that they had a habit of grinding or strongly biting their teeth (Q5), and 61% reported that they had a habit of chewing on one side of their mouth (Q6).

Evaluation of Attrition

The mean value (± SD) for occlusal wear was 2.6 ± 0.295 (range: 0 to 7) (Fig 1).

Table 3 BiteStrip Scores of Subjects

BiteStrip score	Severity of bruxism	No. of bruxism events over 5 h	No. of subjects (n = 46)
0	No bruxism	< 39	18
1	Mild	40–47	13
2	Moderate	75–124	11
3	Severe	> 125	4

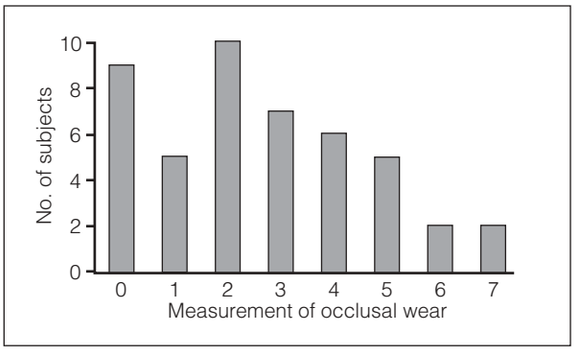

Fig 1 Distribution of attrition. Two thirds of the subjects showed tooth wear ranging from 0 to 3. One third of subjects' scores were from 4 to 7.

EMG BiteStrip

The mean BiteStrip score was 1.0 ± 0.147. The score ranged from 0 to 3 based on the severity of the bruxism. About 40% of the subjects' scores were 0, and they were therefore considered to be nonbruxers. The maximum score was 3. Subjects with this score were considered to be extremely severe bruxers (9%) (Table 3).

Stress Marker Level

The minimum CgA level found was 0.947 pmol/mg protein, and the maximum was 143.6 pmol/mg protein. The value of CgA concentration per total protein was 18.9 ± 0.370 pmol/mg protein (Table 4).

Correlation Among Variables

Occlusal wear did not significantly correlate with most of the items on the self-administered questionnaire (ie, occlusion, jaw functional limitation, or dentition). However, self-reported bruxism behavior was the only item found to significantly correlate with occlusal wear (Q5) (Kendall $\tau_b = 0.309$, $P = .041$) (Fig 2). The BiteStrip score also significantly correlated with occlusal wear (Kendall $\tau_b = 0.313$, $P = .0099$) (Fig 3). The CgA level was significantly negative in correlation with the

Table 4 CgA Level Concentrations of Subjects

	BiteStrip score			
	0	1	2	3
Mean CgA level ± SD	20.79 ± 21. 914	24. 67 ± 38.141	12.15 ± 11.788	10.46 ± 14.667

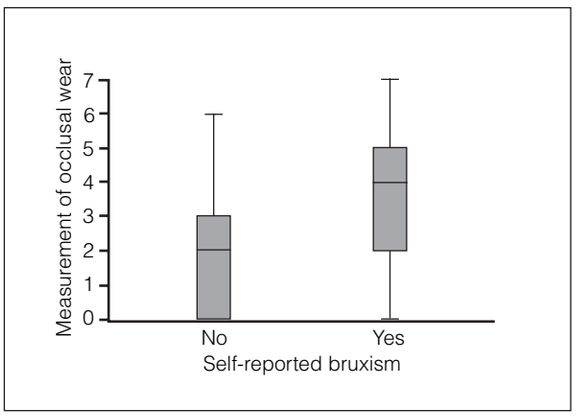

Fig 2 Relationship between occlusal wear and self-reported bruxism. The measurement of occlusal wear was significantly correlated to self-reported bruxism behavior ($P < .05$).

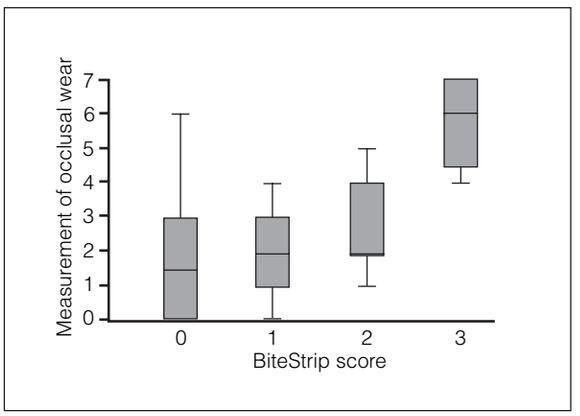

Fig 3 Relationship between measurement of occlusal wear and BiteStrip score. The measurement of occlusal wear was significantly correlated to the BiteStrip score ($P < .01$).

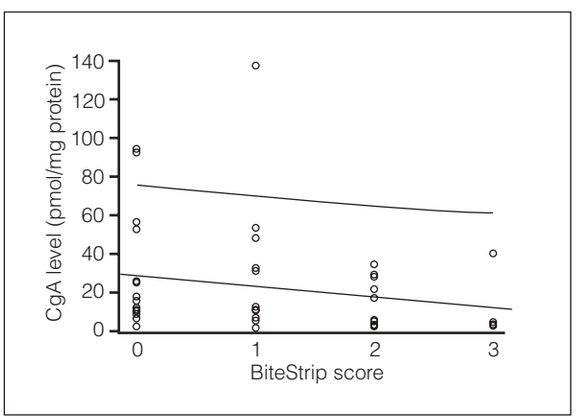

Fig 4 Correlation between CgA level and BiteStrip score. The CgA level was negatively correlated with the BiteStrip score ($P < .05$).

Fig 5 Correlation between CgA level and self-reported bruxism behavior. The CgA level was negatively correlated with bruxism behavior (Q5) ($P < .05$).

BiteStrip score (Kendall $\tau_b = -0.234$, $P = .041$) (Fig 4), as well as self-reported bruxism behavior (Q5) (Kendall $\tau_b = -0.251$, $P = .041$) (Fig 5). Correlations of variables used in this study are summarized in Fig 6.

Discussion

Self-reported grinding or clenching of the teeth combined with the clinical observation of tooth wear are considered valuable means to diagnose bruxism.[25] In particular, tooth wear was caused by contact between opposing and adjacent teeth occurring during function and parafunction (clenching and grinding). Sleep bruxism is characterized by a combination of clenching and grinding-type activity.[26] However, tooth wear by itself is not a definitive marker to diagnose whether subjects have the habit of sleep bruxism.[25] Using self-reported questionnaires completed by the study subjects or bed partner alone to diagnose sleep bruxism would be less reliable. Thus we examined the presence of sleep bruxism through both self-reported questionnaire as well as tooth wear. There was a

positive correlation between tooth wear and self-reported bruxism (Fig 2, Table 1). Those who had a high degree of tooth wear were aware that they bruxed more frequently during sleep. We suggest that the combination of self-reported questionnaires and occlusal tooth wear is a useful parameter to assess a history of bruxism events.

Regarding other questionnaires, comparisons are always difficult due to differences in the methods used. The frequency of symptoms of parafunctions (Q5), as judged from the present study, is similar to a study by Agerberg and Carlsson that showed that 50% of those who answered were aware of some form of parafunction.[22] However, the frequency of symptoms of functional disorders of the masticatory system (Q2) is high compared with their study that showed that 10% of those who answered were aware of the parafunction (Table 1).

It is believed that the gold-standard diagnostic method for bruxism is the use of polysomnographic recordings in a sleep laboratory. However, this has some faults as subjects need to sleep in a specialized sleep laboratory. This might create more stress than sleeping in their normal environments. Also, it limits the number of people able to record bruxism at one time, thus the cost is higher. Accordingly, we used the BiteStrip to make it easier and lower the cost to record bruxism events at home and found that there was a positive correlation between the BiteStrip score and tooth wear (Fig 3, Table 2). Therefore, we concluded that BiteStrip is a useful device to judge the existence and severity of bruxism.

Recently, some biomarkers, such as cortisol and CgA are also regarded as being stress markers, though it has been difficult to objectively evaluate psychological stress. Cortisol is a major glucocorticoid found in the human body that reflects adrenocortical activity. Activation of the HPA (hypothalamic-pituitary-adrenocortical) axis and a subsequent release of cortisol are major components of the physiological stress response.[27] In this study, we used CgA as a stress marker since CgA is the most sensitive biological marker for psychological stress and is not affected by any physiological factors.[19] Also, we chose a saliva sample instead of a blood sample because most methods of saliva collection are easy to perform, noninvasive, rapid, and generally require no special equipment or expertise. We tried to avoid creating any additional stress in this study because we were investgating the state of our subjects' stress levels.

In the relationship between bruxism behavior and CgA level in saliva, we found that the CgA level was significantly negative in correlation with the BiteStrip score. The results indicated that bruxers had less psychological stress than nonbruxers.

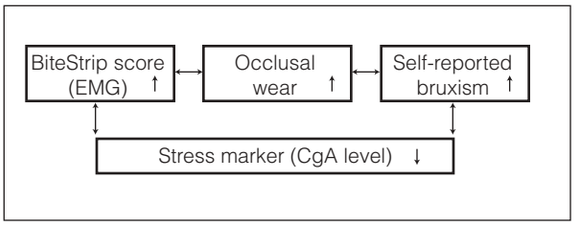

Fig 6 Correlation among variables in this study. The results show that Bruxism factors (BiteStrip and FC and self-reported bruxism (Q5)) are negatively correlated with psychological stress marker (CgA level).

A recent literature review shows a clear transition from a mechanistic attitude to psychological and biopsychosocial concepts in the development of ideas in the etiology, pathogenesis, and therapy of bruxism.[28] Ahlberg et al stated that frequent bruxism may link to ongoing multifactorial stress in normal life and work.[29] The clenching and bruxing function of the masticatory organ might be an emergency exit during periods of psychic overloading. Therefore, occlusion of the masticatory organ might contribute significantly to an individual's ability to manage stress. A growing body of knowledge supports the contention that bruxism is a sleep disorder related to waking emotional states.[30] It may be daytime stress,[31] but it is more probable that bruxism is associated with stress anticipation.[31,32] While reasonable evidence exists that emotional states such as anxiety or stress can elicit muscle tension,[31,33] an absolute relationship between sleep bruxism and stress or any other emotional state has not yet been demonstrated.[33]

Others proposed that sleep bruxism functions as a form of stress management.[15] However, there is little scientific and objective evidence to support this proposition. In this study, there was a negative correlation between the BiteStrip score measuring the severity of bruxism and CgA concentration per total protein in higher states of stress, as well as in cases of self-reported bruxism (Fig 6). The more the factors of bruxism increased, the less the CgA stress marker decreased. In other words, bruxers were under less stress. However, in this research, both the saliva collection and measure of the BiteStrip was carried out only one time in a cross-sectional study. Research needs to be completed in a longitudinal study (ie, before and after sleep) to further these results.

Conclusion

Sleep bruxism is believed to be a stress-related sleep disorder. However, the results showed that daytime psychological stress level is significantly negative in correlation with sleep bruxism behavior.

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