Original Paper

Observational Assessment of the Effect of Sleep Bruxism on Oxyhaemoglobin Saturation: A Preliminary Study

MARCELO PALINKAS[1,2], SELMA SIÉSSERE[1,2], SIMONE CECILIO HALLAK REGALO[1,2]

[1]Department of Basic and Oral Biology, Ribeirão Preto School of Dentistry, University of São Paulo, Brazil
[2]National Institute for Science and Technology in Translational Medicine (INCT-TM)

ABSTRACT: Objectives: Sleep bruxism, characterized by involuntary muscle activity and classified into phasic and tonic patterns, warrants analysis with respect to physiological parameters, as this could aid in understanding its broader health implications. This observational study aimed to compare oxyhaemoglobin saturation between subjects with and without sleep bruxism. Material and Methods: Ninety subjects aged between 18 and 45 years, with natural permanent dentition, normal occlusion, no temporomandibular dysfunction, and no obstructive sleep apnoea, were selected and divided into two homogeneous groups regarding age, sex, weight, and height. The sleep bruxism group comprised 29 women and 16 men (mean age 30.5±6.8), as did the control group (mean age 29.4±7.9). All subjects underwent an overnight PSG session, with the diagnosis of sleep bruxism established according to the criteria of the American Academy of Sleep Medicine. Minimum, mean, and maximum oxyhaemoglobin saturation levels were evaluated using pulse oximetry. Data were analyzed using Student's t-test ($p<0.05$). Results: Comparisons between the groups revealed significant differences in the maximum oxyhaemoglobin saturation index ($p=0.04$). The sleep bruxism group showed a lower maximum oxyhaemoglobin saturation index compared to the control group. No significant differences were observed for minimum or mean oxyhaemoglobin saturation. Conclusion: Sleep bruxism was associated with a lower maximum oxyhaemoglobin saturation compared with controls. Although the observed difference was small and remained within normal physiological limits, these findings suggest a possible relationship between sleep bruxism and oxygenation dynamics during sleep that warrants further investigation.

KEYWORDS: *Sleep bruxism, polysomnography, oxyhaemoglobin saturation.*

Introduction

According to the latest international consensus, sleep bruxism is a rhythmic (phasic) or non-rhythmic (tonic) masticatory muscle activity occurring during sleep [1].

Rather than a disorder, it is considered a motor behaviour that may act as a risk, protective, or neutral factor depending on the health outcome involved [2].

Sleep bruxism is common in adults, although prevalence estimates vary considerably according to the diagnostic criteria and assessment methods used [3,4].

To facilitate its assessment, sleep bruxism can be evaluated using non-instrumental (self-report and clinical examination) and instrumental methods, primarily electromyographic recordings and polysomnography (PSG).

Based on these approaches, sleep bruxism may be classified as possible, probable, or definite, with definite cases requiring instrumental confirmation [5].

Among the available instrumental methods, PSG with audio-video recording is considered the gold standard for sleep bruxism assessment, as it provides the most accurate characterization of sleep-related masticatory muscle activity.

Instrumental recordings allow objective quantification of rhythmic and non-rhythmic jaw-muscle activity during sleep and remain the reference method against which other assessment approaches are validated [6-8].

Beyond its relevance for the assessment of masticatory muscle activity, sleep bruxism may have implications for systemic health in addition to causing discomfort and damage to the stomatognathic system [9].

Given this, it is essential to investigate not only the clinical consequences within the oral cavity, such as tooth wear and fractures, dental sensitivity, non-carious cervical lesions, pain or discomfort in the temporomandibular joint, and headaches, but also its influence on physiological parameters [10].

Changes in tissue oxygenation may be clinically relevant because they can contribute to muscle fatigue and ischemic stress, potentially helping to explain why some individuals exhibit bruxism without adverse consequences (normobruxism), whereas others develop pain-related conditions (pathobruxism).

Experimental evidence has shown that sustained clenching induces a transient ischemic

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state in the masseter muscles, whereas grinding has a less pronounced effect on tissue oxygenation [11].

One physiological parameter that has attracted increasing attention is blood oxygenation during sleep.

Previous studies suggest that sleep bruxism may be associated with sleep fragmentation events and oxygen desaturation [12], such as microarousals [13] and obstructive sleep apnoea [14].

These conditions can lead to episodes of hypoxemia, suggesting a link between motor activities during sleep and blood oxygenation in the human body [15,16].

Moreover, subjects with sleep bruxism may experience changes in blood oxygen saturation even in the absence of obstructive sleep apnoea.

It is common for this motor activity during sleep to be accompanied by an increase in heart and respiratory rates, peripheral vasoconstriction, and heightened muscle activity due to modulation by neurotransmitters such as noradrenaline, dopamine, serotonin, acetylcholine, and glutamate [17,18] which may promote local hypoxia, affecting oxygen saturation.

Taken together, these findings suggest a possible interaction between sleep bruxism and oxyhaemoglobin saturation, although the nature and extent of this relationship remain unclear.

Therefore, understanding this potential connection between sleep bruxism and oxyhaemoglobin saturation is important for expanding knowledge about the systemic implications of sleep bruxism and its consequences for human health.

Thus, the objective of this observational study was to investigate the association between sleep bruxism and oxyhaemoglobin saturation in adult subjects.

The null hypothesis formulated was that there is no association between sleep bruxism and oxyhaemoglobin saturation.

Material and Methods

Study design

This cross-sectional observational study with a control group was developed at the Ribeirão Preto School of Dentistry, University of São Paulo, Brazil.

A convenience sample was drawn from an initial population of 580 subjects of both sexes.

After applying the inclusion and exclusion criteria, 90 subjects were selected and allocated to two homogeneous groups: a sleep bruxism group and a control group.

Each group comprised 29 women and 16 men and was matched for age, sex, weight, and height (Table 1) [19].

Table 1. Mean and standard deviation (±) of the demographic data for the sleep bruxism and control groups.

Demographic data	Groups		p-value
	Control	Sleep Bruxism	
Age	29.4±7.8	30.5±6.7	0.46
Weight	72.76±15.76	73.09±15.24	0.91
Height	1.70±0.09	1.69±0.10	0.85

A significant difference was observed, as indicated by the t-test ($p<0.05$).

Eligibility Criteria

The inclusion criteria for subjects consisted of adults aged between 18 and 45 years, who possessed all their natural permanent teeth and exhibited normal occlusion.

Conversely, exclusion criteria were established to include subjects with neurological movement disorders, temporomandibular disorders, obstructive sleep apnoea, and those who withdrew from participating in the conventional PSG.

Sleep bruxism

The subjects underwent a PSG that took place overnight under the supervision of a specialized technician using a digital system (Sonolab Polysomnograph 632 030003).

The PSG, considered the gold standard, aimed to diagnose sleep bruxism in accordance with the criteria established by the American Academy of Sleep Medicine, ensuring that the technician had no prior knowledge of the subjects' clinical diagnoses.

The study was conducted in a sleep laboratory located in the city of Ribeirão Preto, São Paulo, Brazil.

The PSG was carried out in a controlled environment, climate-controlled and with reduced external noise [20].

The PSG system simultaneously recorded electroencephalographic, electrooculographic, electromyographic, electrocardiographic, respiratory, and pulse oximetry signals throughout the sleep period.

These recordings were used to characterize sleep architecture, identify sleep-related events, and detect rhythmic masticatory muscle activity compatible with sleep bruxism.

Episodes of sleep bruxism were identified based on the rhythmic activity of the masseter muscle, bilaterally, as observed through electromyographic examination during the PSG.

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For events to be considered episodes of sleep bruxism, rhythmic masticatory activity with intervals of up to 3 seconds between them had to meet one of the following criteria: tonic, with at least one electromyographic activation of the masseter muscles lasting over 2 seconds; phasic, with three or more peaks of electromyographic activity, each lasting between 0.25 and 2 seconds; or mixed, combining both patterns.

Subjects were classified as having no sleep bruxism when exhibiting fewer than 2 episodes per hour of sleep [21,22].

According to the international graded diagnostic system for bruxism, the diagnosis established by PSG combined with electromyographic recordings classified subjects in the sleep bruxism group as having definite sleep bruxism [5].

Oxyhaemoglobin saturation

The minimum, average, and maximum oxyhaemoglobin saturation (SpO_2) was assessed using pulse oximetry during the PSG [16].

Pulse oximetry provides a non-invasive estimate of arterial oxyhaemoglobin saturation based on photoplethysmographic detection of oxygenated and deoxygenated haemoglobin.

In the present study, the pulse oximeter continuously recorded SpO_2 values throughout the sleep period, from which the minimum, mean, and maximum oxyhaemoglobin saturation indices were obtained.

The SpO_2 was used to determine the subjects' blood oxygenation index.

For adults, an oxyhaemoglobin saturation between 95% and 100% was considered normal.

Statistical analysis

The normal distribution of the variables was verified using the Kolmogorov-Smirnov test.

The data were processed with SPSS software (SPSS for Windows, version 20.0, Armonk, NY, USA).

To compare the sleep bruxism and control groups, the Student's t-test was employed, with a probability (p) of less than 0.05 considered significant.

Results

Table 2 presents the data regarding the percentage of minimum, average, and maximum oxyhaemoglobin saturation in the sleep bruxism and control groups.

Comparisons between the groups revealed significant differences in the maximum oxyhaemoglobin saturation index (p=0.04).

The sleep bruxism group exhibited a lower maximum oxyhaemoglobin saturation index than that observed in the control group.

Table 2. Mean and standard deviation (±) of oxyhemoglobin saturation for the sleep bruxism and control groups.

Oxyhemoglobin Saturation (%)	Groups		p-value
	Control	Sleep Bruxism	
Minimum	83.61±15.17	83.67±10.98	0.98
Mean	95.80±1.89	95.79±1.31	0.97
Maximum	99.02±0.81	98.69±0.65	0.04

A significant difference was observed, as indicated by the t-test ($p<0.05$).

Discussion

The null hypothesis was rejected, as subjects with sleep bruxism exhibited lower maximum oxyhaemoglobin saturation than controls.

This parameter, oxyhaemoglobin saturation, represents the percentage of haemoglobin in the blood that is bound to oxygen and serves as a relevant indicator of respiratory function.

Although a maximum saturation of 98% is still considered within normal ranges, the 1% difference between the sleep bruxism and control groups, albeit small, was statistically significant.

This suggests possible variations in physiological aspects between the groups that may reflect an association between sleep bruxism and blood oxygenation.

Although the observed difference was limited to maximum oxyhaemoglobin saturation, this finding suggests that sleep bruxism may be associated with subtle alterations in oxygenation dynamics during sleep.

Considering the current concept of sleep bruxism as a behaviour that may have both physiological and pathological implications, the identification of changes in oxygenation parameters may contribute to a better understanding of its potential systemic effects [2,23].

Studies on sleep and awake bruxism have indicated that variations in muscle oxygen recovery may be linked to changes in saturation.

Oxygen recovery is slower following light and prolonged clenching compared to strong and brief clenching.

This reinforces the importance of assessing oxygen saturation in subjects with bruxism [24].

These findings support the hypothesis that bruxism-related muscle activity may influence oxygenation parameters, although the available evidence remains limited [7].

A recent study analyzed the relationship between sleep bruxism, microarousals, and oxyhaemoglobin desaturations through PSG.

The findings revealed that, although sleep bruxism is significantly associated with an increase in microarousals, the relationship with oxyhaemoglobin desaturations was unclear, as no significant differences were observed between the sleep bruxism and control groups.

This indicates that the implications for oxyhaemoglobin desaturation require further investigation [25].

These findings differ from the results of the present study, which demonstrated a significant difference between the groups regarding maximum oxyhaemoglobin saturation.

Differences in study design, sample characteristics, exclusion criteria, and the oxygenation parameters analyzed may partially explain these divergent findings.

Notably, the present study excluded subjects with obstructive sleep apnoea, reducing the influence of a major confounding factor known to affect oxyhaemoglobin saturation during sleep [16].

A possible explanation for the lower maximum oxyhaemoglobin saturation in the sleep bruxism group may be related to respiratory interference during sleep, as bruxism is associated with an increase in microarousals accompanied by variations in respiratory patterns.

This can result in a reduction in blood oxygenation, even in the absence of obstructive sleep apnoea [14,26].

Furthermore, sleep bruxism activates the neurons of the trigeminal mesencephalic nucleus, which could interfere with cardiac activation, respiratory function, and brain activity [18].

This activation may lead to an increase in heart rate and blood pressure, resulting in a stress response that affects the cardiovascular system, which, in turn, may impact oxyhaemoglobin saturation [27].

This increase in sympathetic activity, resulting from the physical and emotional stress associated with sleep bruxism, may trigger vasoconstriction, leading to increased resistance to blood flow to the tissues, including the brain, thereby reducing blood oxygenation [28].

While these mechanisms provide a plausible physiological explanation for the findings, their clinical significance should be interpreted with caution.

Although a 1% difference may seem small, in studies with rigorous measurements, even minimal variations can be significant, reflecting real physiological differences [29,30].

Therefore, the lower oxyhaemoglobin saturation in the sleep bruxism group may be explained by an association of subtle respiratory difficulties and physiological stress.

From a clinical perspective, the magnitude of the observed difference should be interpreted with caution, as both groups remained within normal oxyhaemoglobin saturation ranges.

Nevertheless, even small variations may indicate underlying physiological adaptations associated with sleep bruxism and warrant further investigation, particularly in subjects presenting additional systemic risk factors [31].

Physiological stress may contribute to muscle fatigue, intensifying the sensation of discomfort and leading to a decrease in respiratory efficiency [32].

The constant activation of the masticatory muscles during episodes of sleep bruxism may promote muscle tension that radiates to the cervical and thoracic regions.

This tension not only affects respiratory mechanics but may also reduce the functional capacity of the muscles involved in chewing and breathing, potentially hindering blood oxygenation [33].

The analysis of the impact of sleep bruxism on oxyhaemoglobin saturation may reveal significant consequences of this condition.

An international publication in the field suggests that the relationship between sleep bruxism and systemic health is significant.

Studies indicate an important correlation between oxygen desaturation and the frequency of bruxism episodes in hypertensive subjects, revealing a lower average oxygen saturation. Conversely, this relationship is not observed in normotensive subjects [27].

Understanding this influence not only enriches scientific knowledge but also contributes to the development of more effective interventions.

The results may provide important insights for healthcare professionals, facilitating the identification of complications associated with sleep bruxism and promoting more integrated treatment with possible relationships with quality of life [34].

This study has limitations that should be considered.

Firstly, the small sample size, which consists of only 90 subjects, may limit the generalisability of the obtained results.

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The exclusion of subjects with obstructive sleep apnoea may have influenced the homogeneity of the analyzed groups.

Furthermore, although PSG allows the simultaneous assessment of cerebral, cardiac, respiratory, and masticatory muscle activity, the analyses were restricted to oxyhaemoglobin saturation and did not investigate its temporal relationship with individual sleep bruxism episodes.

Consequently, no causal or temporal inferences regarding the association between sleep bruxism and oxyhaemoglobin saturation can be drawn.

Future studies with larger samples and more comprehensive physiological analyses are needed to validate and expand the present findings.

Conclusion

Within the limitations of this study, a significant difference in maximum oxyhaemoglobin saturation was observed between subjects with sleep bruxism and controls.

These findings support a possible association between sleep bruxism and oxyhaemoglobin saturation during sleep, although the nature of this relationship remains unclear and requires further investigation.

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None to declare.

Author Contributions

Conceptualization: M.P., S.S. and S.C.H.R.; Methodology: M.P., S.S. and S.C.H.R.; Data analysis: M.P.; Manuscript writing and initial draft preparation: M.P.; Manuscript review and editing: S.S. and S.C.H.R.; Supervision: S.C.H.R. All authors read and approved the final manuscript.

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Conflict of interest

None to declare.

Institutional Review Board

The study was approved by the Research Ethics Committee of the Ribeirão Preto School of Dentistry, University of São Paulo, Brazil (protocol No: 02735812.9.0000.5419).

Consent Statement

Written informed consent was obtained from all subjects prior to enrolment.

Data availability

All data are available upon request to the corresponding author.

References

1. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med, 2003,14(1):30-46.
2. Verhoeff MC, Lobbezoo F, Ahlberg J, Bender S, Bracci A, Colonna A, Dal Fabbro C, Durham J, Glaros AG, Häggman-Henrikson B, Kato T, Koutris M, Lavigne GJ, Nykänen L, Raphael KG, Svensson P, Wieckiewicz M, Manfredini D. Updating the bruxism definitions: report of an international consensus meeting. J Oral Rehabil, 2025, 52(9):1335-1342.
3. Manfredini D, Winocur E, Guarda-Nardini L, Paesani D, Lobbezoo F. Epidemiology of bruxism in adults: a systematic review of the literature. J Orofac Pain, 2013, 27(2):99-110.
4. Zieliński G, Pająk A, Wójcicki M. Global prevalence of sleep bruxism and awake bruxism in pediatric and adult populations: a systematic review and meta-analysis. J Clin Med, 2024, 13(14):4259.
5. Lobbezoo F, Ahlberg J, Raphael KG, Wetselaar P, Glaros AG, Kato T, Santiago V, Winocur E, De Laat A, De Leeuw R, Koyano K, Lavigne GJ, Svensson P, Manfredini D. International consensus on the assessment of bruxism: Report of a work in progress. J Oral Rehabil, 2018, 45(11):837-844.
6. Balasubramaniam R, Manfredini D, Guan G. Sleep bruxism: a narrative review of current concepts, mechanisms, and clinical implications. J R Soc N Z, 2026, 56(2):e70046.
7. Li C, Yap S, Loh A, Yap YJ, Kujan O, Balasubramaniam R. Ambulatory devices to detect sleep bruxism: a narrative review. Aust Dent J, 2024, 69 Suppl 1(Suppl 1):S53-S62.
8. Cid-Verdejo R, Chávez Farías C, Martínez-Pozas O, Meléndez Oliva E, Cuenca-Zaldívar JN, Ardizone García I, Martínez Orozco FJ, Sánchez Romero EA. Instrumental assessment of sleep bruxism: A systematic review and meta-analysis. Sleep Med Rev, 2024, 74:101906.
9. Polmann H, Domingos FL, Melo G, Stuginski-Barbosa J, Guerra ENDS, Porporatti AL, Dick BD, Flores-Mir C, De Luca Canto G. Association between sleep bruxism and anxiety symptoms in adults: A systematic review. J Oral Rehabil, 2019, 46(5):482-491.
10. Ainoosah S, Farghal AE, Alzemei MS, Saini RS, Gurumurthy V, Quadri SA, Okshah A, Mosaddad SA, Heboyan A. Comparative analysis of different types of occlusal splints for the management of sleep bruxism: a systematic review. BMC Oral Health, 2024, 24(1):29.
11. Ohta Y, Noguchi T, Ohtawa Y, Takeda T, Fukuda KI. Effects of experimental clenching and grinding on the masseter muscles and the autonomic nervous system: a preliminary study. J Dent Anesth Pain Med, 2026, 26(2):143-153.

12. Kostrzewa-Janicka J, Jurkowski P, Zycinska K, Przybyłowska D, Mierzwińska-Nastalska E. Sleep-related breathing disorders and bruxism. Adv Exp Med Biol, 2015, 873:9-14.
13. Palinkas M, Semprini M, Filho JE, de Luca Canto G, Regalo IH, Bataglion C, Rodrigues LAM, Siéssere S, Regalo SCH. Nocturnal sleep architecture is altered by sleep bruxism. Arch Oral Biol, 2017, 81(5):56-60.
14. Romero-Corral A, Caples SM, Lopez-Jimenez F, Somers VK. Interactions between obesity and obstructive sleep apnea: implications for treatment. Chest, 2010, 137(3):711-719.
15. Martin SE, Engleman HM, Kingshott RN, Douglas NJ. Microarousals in patients with sleep apnoea/hypopnoea syndrome. J Sleep Res, 1997, 6(4):276-280.
16. Thorisdottir K, Hrubos-Strøm H, Karhu T, Nikkonen S, Dammen T, Nordhus IH, Leppänen T, Jónsdóttir MK, Arnardottir ES. Verbal memory is linked to average oxygen saturation during sleep, not the apnea-hypopnea index nor novel hypoxic load variables. Sleep Med, 2024, 123:29-36.
17. Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain, 2009, 23(2):153-166.
18. Giovanni A, Giorgia A. The neurophysiological basis of bruxism. Heliyon, 2021, 7(7):e07477.
19. Palinkas M, Bataglion C, de Luca Canto G, Machado Camolezi N, Theodoro GT, Siéssere S, Semprini M, Regalo SC. Impact of sleep bruxism on masseter and temporalis muscles and bite force. Cranio, 2016, 34(5):309-315.
20. Palinkas M, De Luca Canto G, Rodrigues LA, Bataglion C, Siéssere S, Semprini M, Regalo SC. Comparative Capabilities of clinical assessment, diagnostic criteria, and polysomnography in detecting sleep bruxism. J Clin Sleep Med, 2015,11(11):1319-1325.
21. Rompré PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res, 2007, 86(9):837-842.
22. Polmann H, Pauletto P, Duarte J, Réus JC, Maia I, De Luca Canto G. Association between sleep bruxism and snoring in adults: An observational study. Sleep Med, 2024,124:276-281.
23. Sambale J, Koehler U, Conradt R, Kesper K, Cassel W, Degerli M, Viniol C, Korbmacher-Steiner HM. Is sleep bruxism in obstructive sleep apnea only an oral health related problem? BMC Oral Health, 2024, 24(1):565.
24. Satokawa C, Nishiyama A, Suzuki K, Uesugi S, Kokai S, Ono T. Evaluation of tissue oxygen saturation of the masseter muscle during standardised teeth clenching. J Oral Rehabil, 2020, 47(1):19-26.
25. de Miranda Diniz SA, de Magalhães Lopes R, Guedes LM, Bruzinga FFB, de Aguilar Seraidarian KK, de Magalhães Barros V, de Barros Massahud ML, Seraidarian PI. Sleep-related bruxism, microarousals and oxyhaemoglobin desaturations in sleep stages: A cross-sectional study in a large apnoeic population. J Oral Rehabil, 2024, 51(10):2140-2149.
26. Restrepo C, Lobbezoo F, Castrillon E, Svensson P, Santamaria A, Manfredini D. Correlations between sleep architecture and sleep-related masseter muscle activity in children with sleep bruxism. J Oral Rehabil, 2024, 51(1):110-116.
27. Kanclerska J, Wieckiewicz M, Poreba R, Szymanska-Chabowska A, Gac P, Wojakowska A, Frosztega W, Michalek-Zrabkowska M, Mazur G, Martynowicz H. Polysomnographic evaluation of sleep bruxism intensity and sleep architecture in nonapneic hypertensives: a prospective, observational study. J Clin Med, 2022, 11(11):3113
28. Michalek-Zrabkowska M, Martynowicz H, Wieckiewicz M, Smardz J, Poreba R, Mazur G. Cardiovascular implications of sleep bruxism-a systematic review with narrative summary and future perspectives. J Clin Med, 2021, 10(11): 2245
29. Knudsen N, Laurberg P, Rasmussen LB, Bülow I, Perrild H, Ovesen L, Jørgensen T. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. J Clin Endocrinol Metab, 2005, 90(7):4019-4024.
30. Patel R, Ucer C, Wright S, Khan RS. Differences in dental implant survival between immediate vs. delayed placement: a systematic review and meta-analysis. Dent J (Basel), 2023,11(9):218.
31. Fulek M, Frosztega W, Wieckiewicz M, Szymanska-Chabowska A, Gac P, Poreba R, Mazur G, Sciskalska M, Kepinska M, Martuszewski A, Martynowicz H. The link between sleep bruxism and oxidative stress based on a polysomnographic study. Sci Rep, 2025, 15(1):3567.
32. Gruet M. Fatigue in chronic respiratory diseases: theoretical framework and implications for real-life performance and rehabilitation. Front Physiol, 2018, 9:1285.
33. Uchima Koecklin KH, Aliaga-Del Castillo A, Li P. The neural substrates of bruxism: current knowledge and clinical implications. Front Neurol, 2024, 15:1451183.
34. Sin EC, Raftu G, Bușțiuc SG, Caraiane A, Briceag R. The relationship between sleep bruxism index and quality of life. Curr Health Sci J, 2024, 50(5):428-435.

Corresponding Author: Marcelo Palinkas, Department of Basic and Oral Biology, Ribeirão Preto School of Dentistry, University of São Paulo, Avenida do Café, s/n, Bairro, Monte Alegre, Ribeirão Preto, SP 14040-904, Brazil, e-mail: palinkas@usp.br

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