

Etiology of sleep bruxism - a review of the literature

Pola Malinowska¹, Antonina Feikis², Bartłomiej Gałuszka³, Dominika Pietrzyk⁴, Magdalena Kalucka - Janik⁵, Katarzyna Zajac⁶, Patryk Król⁷, Bartosz Oder⁸, Michał Pogoda⁹, Krzysztof Kościański¹⁰

¹ *Medical University in Katowice, Poniatowskiego 15, Katowice, 40-055, Poland*
ORCID: <https://orcid.org/0009-0000-0477-3906>

² *Medical University in Katowice, Poniatowskiego 15, Katowice, 40-055, Poland*
ORCID: <https://orcid.org/0009-0007-7644-052X>

³ *Independent Public Health Care Facility, Municipal Hospitals Complex in Chorzów, Chorzów, Poland*
ORCID: <https://orcid.org/0009-0006-1426-6709>

⁴ *Specialist Hospital No. 2 in Bytom, Bytom, Poland*
ORCID: <https://orcid.org/0000-0002-8114-3622>

⁵ *Independent Public Health Care Facility, Municipal Hospitals Complex in Chorzów, Chorzów, Poland*
ORCID: <https://orcid.org/0009-0006-5389-0993>

⁶ *Specialist Hospital No. 2 in Bytom, Bytom, Poland*
ORCID: <https://orcid.org/0009-0000-2056-8128>

⁷ *University Clinical Hospital No. 2 of Pomeranian Medical University in Szczecin, Szczecin, Poland*
ORCID: <https://orcid.org/0009-0003-3753-3978>

⁸ *Pomeranian Medical University in Szczecin, Rybacka 1, 70-204, Szczecin, Poland*
ORCID: <https://orcid.org/0009-0008-2599-9339>

⁹ *Pomeranian Medical University in Szczecin, Rybacka 1, 70-204, Szczecin, Poland*
ORCID: <https://orcid.org/0009-0000-2936-4367>

¹⁰ *University Clinical Hospital No. 2 of Pomeranian Medical University in Szczecin, Szczecin, Poland*
ORCID: <https://orcid.org/0009-0005-7566-5810>

Corresponding author: Bartłomiej Gałuszka,

ABSTRACT

Sleep bruxism, through its unexplained etiology, has become the subject of much research and discussion. The multidimensionality and complexity of the process makes it impossible to identify a single source of the problem. Over the years, new theories have emerged and others have been debunked. Reviewing the available literature, the plausible sources of bruxism have been collected and divided into three groups. It is not possible to determine which group is dominant, as the risk factors for bruxism are sometimes correlated. The essence of bruxism requires further consideration and research.

KEY WORDS

bruxism; etiology of bruxism; risk factors of bruxism; sleep bruxism

INTRODUCTION

According to the generally accepted definition from 2018, bruxism can be defined as a repetitive action of the masticatory muscles, characterised by grinding or clenching of the teeth and pushing or thrusting of the mandible. It also includes jaw stiffening [1,2]. This activity is regulated by the central nervous system and may not only affect occlusal contacts [2]. Parafunctional activities associated with bruxism also include habits performed during the day, such as biting the cheeks and tongue [3]. Symptoms observed during clinical examination include tongue biting and linea alba on the cheek mucosa [4].

The former classification of bruxism was based on the time of day when it occurred. Scientists divided it into daytime bruxism, where episodes of clenching and grinding of the teeth occurred during the day, and night-time bruxism, where pathophysiological activity occurred during the night. Currently, this classification has been changed – we divide bruxism according to the state of the body – into bruxism during wakefulness and during sleep [2]. Sleep bruxism, which occurs during sleep, is

a relatively common phenomenon and can take the form of single episodes or rhythmic contractions. On the other hand, bruxism during wakefulness refers to all habits related to clenching and grinding teeth during daily activities, which may be related to stress, but also to one's profession. Most often, this parafunctional activity is performed unconsciously by patients. Neither form is a movement disorder in healthy individuals [4]. Regardless of the type of activity, the factors that influence the occurrence of bruxism are similar. Bruxism is a multifactorial disease and, despite many different concepts, it is currently mainly determined by three groups of factors. These are biological, psychological and exogenous factors. However, it should be added that episodes of clenching and grinding teeth do not always immediately indicate parafunction. In childhood, these activities occur as adaptive processes, where the child gets used to the presence of teeth in the mouth. Later on, however, grinding and rubbing are associated with a physiological process aimed at wearing down the cusps of the milk teeth. This process leads to the proper preparation for the eruption of the first permanent molars and, consequently, enables the teeth to be positioned in the correct Angle Class I [5]. In 2023, a diagnostic tool was created to diagnose patients experiencing symptoms that may indicate bruxism. The form consists of two parts, which focus on the likely conditions and triggers of bruxism episodes. This allows us to conduct a thorough interview and diagnose bruxism, and thus manage it more effectively [1].

METHODS

This article is a narrative review of the literature on the etiology of sleep bruxism. A comprehensive search was conducted in the PubMed, Scopus, and Google Scholar databases up to October 2025, using combinations of the following keywords: “*sleep bruxism*,” “*etiology*,” “*pathophysiology*,” “*risk factors*,” “*neurotransmitters*,” “*stress*,” and “*sleep disorders*.” Original research articles, systematic reviews, and meta-analyses written in English were included.

ETIOLOGY

Disputes regarding the aetiology of bruxism have been ongoing for years. Progress has been made among clinicians and researchers, but no clear cause or mechanism for episodes of bruxism has been proven. Many risk factors have been discovered [6, 7]. It has been found that their accumulation increases the likelihood of this parafunction occurring [8].

The phenomenon of teeth grinding or abrasion has always accompanied humanity. The first mentions of bruxism can be found in the Bible. Due to the fact that it was associated with pain, it was called ‘the first punishment from God’ [9]. Not all cases of teeth grinding have a positive correlation with bruxism. During the development of the stomatognathic system, after the age of 3, physiological abrasion of the cusps of deciduous teeth occurs. This is a preparatory process for the replacement of teeth with permanent teeth and is associated with the necessary laterotrusive displacement of the mandible [10].

According to Lobbezoo, bruxism has a multifactorial origin [2]. It is currently believed that episodes of bruxism can be caused by three groups of factors. The first group includes biological factors, which include cortical stimulation, neurotransmitter imbalances in neurological diseases such as Parkinson's disease or depression; genetic factors; sleep problems; and malocclusion. The second group includes psychological factors such as exposure and susceptibility to stress, psychological personality, and social factors such as place and working conditions. The Covid-19 pandemic has had a major impact on this group of factors. The third group includes exogenous factors: caffeine, alcohol, nicotine, drugs, and certain medications [11, 12, 13].

FIRST GROUP: BIOLOGICAL FACTORS

1. NEUROTRANSMITTER DISORDERS

An imbalance between the two neurotransmitter pathways is considered to be responsible for muscle hyperactivity during nocturnal dyskinesia, i.e. bruxism. It is likely that the indirect and direct pathways of the basal ganglia, which is a group of five

subcortical nuclei, are involved in the coordination of movements. In the indirect pathway, before the signal reaches the thalamus, it must pass through several intermediate nuclei. In the second case, afferent signals running from the striatum to the thalamus directly project onto the cerebral cortex. An imbalance between these pathways causes disturbances in the transmission of action potentials involving neurotransmitters such as dopamine [14].

Although the link between Parkinson's disease (PD) and bruxism has not yet been fully discovered, there are some suggestions that point to this.

In PD, in addition to the motor disorders that occur in bruxism, the dopaminergic network in the central nervous system plays an important role. Due to reduced dopamine levels, the likelihood of uncontrolled movements in the stomatognathic system increases, which can ultimately lead to bruxism [15]. An analogy can be found in other diseases associated with dysfunction of the dopaminergic or serotonergic pathways, such as depression [16].

2. GENETIC FACTORS

In studies conducted by Więckiewicz M. et al., researchers sought to discover the genetic basis of sleep bruxism (SB). SB is likely a genetically complex condition.

The findings suggested possible genetic variability in the genes encoding dopamine receptors (DRD1) and serotonin receptors (HTR2A). The DRD1 rs686 polymorphism appeared to correlate with the risk of developing sleep bruxism. The HTR2A gene polymorphism is associated with the mental disorders mentioned above [17].

According to another polysomnographic study, 37% of patients with diagnosed SB had at least one first-degree relative suffering from SB. Data from a population sample conducted by Kohury also confirmed this positive correlation with SB. However, as their other results showed, no difference was found between the sexes [18].

3. SLEEP DISORDERS

One of the parafunctional activities is nocturnal activity. It occurs during sleep. It is a relatively common phenomenon. It can take the form of single episodes or rhythmic contractions – bruxism. A single sleep cycle can be divided into four stages. These consist of the NREM (non-rapid eye movement) phase and the REM phase. The first and second stages consist of many series of rapid alpha waves, beta waves and ‘sleep spindles’. Stages three and four are deep sleep stages. Slower beta waves dominate there. During NREM, regeneration takes place, which is important in terms of physical rest. Disorders of this phase are associated with pain and increased sensitivity of musculoskeletal tissues. The REM phase is important for restoring the proper functioning of the brain stem and cerebral cortex. Scientists believe that emotional tensions are ‘released’ during this last phase. REM sleep is also very important for mental rest. There is much controversy regarding the correlation between sleep bruxism and a specific phase of sleep. Some researchers argue that SB occurs mainly during the REM phase of sleep. Others argue that it never occurs during this phase or that it occurs during both the NREM and REM phases. These and other studies have shown that episodes of bruxism may be closely related to the phases of sleep that are responsible for waking up.

So-called dental sleep disorders, such as gastroesophageal reflux disease, sleep apnoea and periodic limb movements, are associated as possible causes of SB. Evidence is currently being gathered on the link between obstructive sleep apnoea and jaw-closing muscle activity [6]. SB rarely occurs on its own [19]. Many of the sleep-related conditions listed above have been linked together in a complex network of comorbidities [6].

Poor sleep quality is also reflected in excessive daytime sleepiness (EDS). People with EDS have difficulty staying alert and attentive during the day. EDS is associated with insufficient sleep, diseases (COPD, sleep apnoea, stroke, narcolepsy, asthma, circadian rhythm disorders) and poor sleep habits [11].

Studies have been conducted in which the main finding was poorer sleep quality in volunteers with SB, which was reflected in their excessive daytime sleepiness. Based on the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale, it was concluded that 80% of SB patients showed a decrease in sleep quality [20].

The aforementioned Kohury conducted his research on many levels. One of them concerned the relationship between episodes of bruxism and sleep disorders. SB-positive subjects did not show any problems with falling asleep compared to SB-negative subjects. However, SB-positive subjects had slightly more difficulty staying asleep. In some of them, this was associated with the occurrence of pain symptoms [18].

4. MALOCCLUSION

In the past, the prevailing theories were those claiming that bruxism could be closely related to malocclusion. However, the inability to define ‘perfect’ occlusion became a problem in confirming the validity of this theory. None of the control studies have been able to demonstrate the possibility of eliminating the symptoms of bruxism by correcting occlusal obstacles [14]. More recent studies show no or little association between malocclusion and the occurrence of bruxism [21]. No association was found between malocclusion according to Angle's I, II and III classifications and bruxism. However, people with bruxism are more likely to develop crowding [22].

GROUP 2. PSYCHOLOGICAL FACTORS

Later on, bruxism began to be increasingly associated with high emotional tension, i.e. stress – increased CNS impulsivity causes increased muscle tension. In addition, studies have been conducted which confirm that patients suffering from bruxism have higher levels of catecholamines in their urine than patients who do not suffer from this condition [21].

1. STRESS

Stress itself, also known as mental tension, is a physiological adaptive response in every human being. Although there is no clear definition, it can be described as “a state or feeling experienced when a person perceives that the demands placed on them exceed their available resources”. It occurs when a threat, difficulty, challenge or inability to perform daily tasks arises. It is also associated with the stress hormone cortisol, which is released during stressful situations. Cortisol is responsible for adapting the human body to tackle a given difficulty and thus restore physical and mental balance [23]. Although stress is an essential part of every human being's life, its long-term effects and exceeding the body's adaptive capacity cause various dysfunctions, including those related to the stomatognathic system.

Stress and the stimuli that cause it, i.e. stressors, do not always have to be negative. Therefore, we can distinguish between eustress, sustress and distress. Eustress is so-called positive stress, motivating action, which can occur before important events in life. In addition, it causes a mild stress response and allows the body to increase its ability to buffer homeostasis. Sustress, on the other hand, is a response in which the body does not respond properly to the stress response and thus reduces its ability to buffer homeostasis. In other words, sustress is “inappropriate stress or even a lack of stress”. Distress is “bad stress” - which causes the body to overreact to stress.

According to the latest research, sustress and distress are responsible for the impairment of physiological functions, including the development of bruxism. Eustress itself has a positive effect and is usually an adaptive response to a given situation, allowing a return to mental and physical balance [24].

Emotional tension, or stress, affects the body through increased nerve impulses and activation of the hypothalamus, which, thanks to the autonomic nervous system, prepares the entire body for an appropriate response. This preparation consists of an increase in the activity of gamma-type efferent fibres, which, via intrafusal fibres, can lead to muscle spindle contraction. This puts them in a state of readiness for activation, resulting in increased muscle tone. Even the slightest stretching of the fibre can lead to

a reflex contraction. Therefore, in a state of increased emotional tension, there is also increased muscle tension, which can lead to the development of bruxism.

So why is bruxism not observed in all people?

According to various authors, bruxism is described as a stress-relieving activity and even a way to get rid of stress completely. However, there are many ways to cope with stress, and they do not always accumulate in the masticatory organ [7].

According to Okeson, stress is compared to energy that is accumulated in the body and must be dealt with appropriately. We distinguish between two ways of coping with stressful situations: the external mechanism and the internal mechanism.

The external mechanism consists of physical activity, shouting and all activities that lead to the “release” of energy outside the body.

The internal mechanism, on the other hand, involves accumulating stress, not getting rid of it externally, but releasing it internally. It has been proven that internal release most often leads to increased muscle tension, and thus, people who prefer this type are at risk of bruxism.

2. SOCIAL FACTORS – Occupation or work-related habits

Bruxism is not always directly related to emotional tension. It can affect people who perform specific occupations that require non-physiological activities, leading to the development of this parafunction. These include intellectual workers, whose mental effort manifests itself in teeth clenching, but also manual workers and anyone who performs activities that require holding an object between their head and shoulder. Musicians and divers, who hold mouthpieces directly in their mouths, are also at risk of bruxism episodes.

3. COVID-19 and bruxism

The Covid-19 pandemic has also contributed to the development and exacerbation of bruxism episodes in many people. According to research, the new living conditions imposed by the pandemic have significantly increased levels of stress and anxiety. Fear of isolation, forced distancing, concern for the health and lives of loved ones, and uncertainty about the development of the situation caused psychological distress, which had a negative impact on the entire body. In addition, it has been proven that people suffering from temporomandibular joint disorders and bruxism experienced higher levels of stress during this time than those who did not have these conditions [25].

GROUP 3. EXOGENOUS FACTORS/LIFESTYLE

1. STIMULANTS (caffeine, alcohol, nicotine, drugs)

The third group of possible causes of sleep bruxism includes drugs and addictive substances.

Studies have been conducted, based on the available literature, which have presented indications or evidence of the adverse effects of certain addictive substances, as well as several (classes of) drugs. The authors of the study identified three groups: drugs that potentially cause bruxism; individual drugs; and addictive substances that potentially cause bruxism.

The authors included anticonvulsants containing barbiturates, phenylethylamines and selective serotonin reuptake inhibitors (SSRIs) in the first group. Patients treated with compounds containing barbiturates showed an increased incidence of unspecified bruxism. In individuals with ADHD treated with high doses of phenylethylamine, one of the side effects was bruxism occurring during sleep [26]. Medications containing SSRIs have an inhibitory effect on specific dopaminergic neurons, which may consequently cause episodes of bruxism [15]. In the second group, the authors found no evidence or only isolated clinical cases of bruxism occurring during treatment with aripiprazole, atomoxetine, flecainide or ketotifen. In the case of

atomoxetine, the researchers cited the case of a 7-year-old boy with ADHD, in whom the presence and severity of sleep bruxism was associated with discontinuation and dosage of the drug. Duloxetine and venlafaxine, which belong to the serotonin and norepinephrine reuptake inhibitors, were positively associated with sleep bruxism. In the case of methadone, they referred to studies conducted in Israeli prisons on heroin addicts. Although the studies showed a higher incidence of both forms of bruxism in methadone users, unfortunately there were too many confounding factors (alcohol, stress, psychotropic drugs, opioids).

The third group includes popular stimulants such as alcohol, nicotine, caffeine and drugs [26]. Ethanol contained in alcohol has a depressant effect on the central nervous system. It acts as an agonist for the GABA receptor and an antagonist for the N-methyl-D-aspartate receptor [27]. People who consume alcohol frequently may experience reduced sleep quality due to disruption of the normal sleep cycle [28, 29]. Nicotine, depending on the amount consumed, can stimulate or inhibit the CNS. This is because it is an acetylcholine agonist. According to recent findings, smokers have shown increased sleep fragmentation, which is associated with increased bruxism intensity [27]. Narcotic drugs have a proven effect on the occurrence of bruxism episodes. In people addicted to opioids (e.g. heroin), it is a dominant symptom. Also, in chronic methamphetamine users, as a result of increased noradrenergic neurotransmission and, consequently, increased neuromuscular activity of the stomatognathic system, bruxism and jaw clenching occur [30]. Caffeine, through its known properties, can affect sleep architecture, which is reflected in sleep bruxism [31].

CONCLUSIONS

This article summarises the probable aetiological factors of sleep bruxism. It has been defined as a multifactorial condition in which all groups of factors intertwine. Clinically, all of the above-mentioned sources may play a significant role, as they are classified between potentially harmful and non-threatening bruxistic behaviours. It is impossible to determine unequivocally which one has the greatest advantage, but this

role is increasingly attributed to psychological tension. Much research is still needed to properly and effectively manage sleep bruxism.

REFERENCES

1. Manfredini D, Ahlberg J, Aarab G, Bender S, Bracci A, Cistulli PA, Conti PC, De Leeuw R, Durham J, Emodi-Perlman A, Ettlin D, Gallo LM, Häggman-Henrikson B, Hublin C, Kato T, Klasser G, Koutris M, Lavigne GJ, Paesani D, Peroz I, Svensson P, Wetselaar P, Lobbezoo F. Standardised Tool for the Assessment of Bruxism. *J Oral Rehabil.* 2023;00;1-6.
2. 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: 837– 844.
3. Piquero K, Ando T, Sakurai K. Buccal mucosa ridging and tongue indentation: incidence and associated factors. *Bull Tokyo Dent Coll.* 1999;40(2):71-8.
4. Ahlberg J, Piirtola M, Lobbezoo F, Manfredini D, Korhonen T, Aarab G, Hublin C, Kaprio J. Correlates and genetics of self-reported sleep and awake bruxism in a nationwide twin cohort. *J Oral Rehabil.* 2020 Sep;47(9):1110-1119.
5. Irena Karłowska, Zarys Współczesnej Ortodoncji, Warszawa 2016, str. 36-37
6. Manfredini D, Ahlberg J, Aarab G, Bracci A, Durham J, Emodi-Perlman A, Ettlin D, Gallo LM, Häggman-Henrikson B, Koutris M, Peroz I, Svensson P, Wetselaar P, Lobbezoo F: The development of the Standardised Tool for the Assessment of Bruxism (STAB): An international road map. *J Oral Rehabil.* 2022; 00: 1- 14.
7. Sato S, Sasaguri K, Ootsuka T, Saruta J, Miyake S, Okamura M, Sato C, Hori N, Kimoto K, Tsukinoki K, Watanabe K, Onozuka M: Bruxism and Stress Relief. *Novel Trends in Brain Science.* Springer, Tokyo. 2008, 183-200.
8. Maciejewska-Szaniec Z, Kaczmarek-Ryś M, Hryhorowicz S, Przysańska A, Gredes T, Maciejewska B, Hoppe-Gołębiewska J, Słomski R, Pławski A, Czajka-Jakubowska A. Polymorphic variants in genes related to stress coping are associated with the awake bruxism. *BMC Oral Health.* 2021 Oct 5;21(1):496.
9. Veiga N, Ângelo T, Ribeiro O, Baptista A: Bruxism – Literature review. *Int J Dent Oral Health* 2015;1,5
10. Dorota Olczak-Kowalczyk, Joanna Szczepańska, Urszula Kaczmarek, *Współczesna Stomatologia Wieków Rozwojowego*, Warszawa 2017, str. 79
11. Turcio KH, de Moraes-Melo-Neto CL, de Caxias FP, Goiato MC, Dos Santos DM, Januzzi MS, Guiotti AM, Bertoz AM, Brandini DA. Relationship of excessive daytime sleepiness with bruxism, depression, anxiety, stress, and sex in odontology students - A cross sectional study. *J Clin Exp Dent.* 2022 Jun 1;14(6):e464-e470.
12. Chattratrai T, Blanken TF, Lobbezoo F, Su N, Aarab G, Van Someren EJW. A network analysis of self-reported sleep bruxism in the Netherlands sleep registry: its associations with insomnia and several demographic, psychological, and life-style factors. *Sleep Med.* 2022 May;93:63-70.
13. Pop-Jordanova N., Loleska S.. On Psychosomatic Problems in Dentistry. *PRILOZI.* 3920;41(1): 57-63.
14. Behr M, Hahnel S, Faltermeier A, Bürgers R, Kolbeck C, Handel G, Proff P. The two main theories on dental bruxism. *Ann Anat.* 2012 Mar 20;194(2):216-9.

15. Verhoeff MC, Lobbezoo F, Wetselaar P, Aarab G, Koutris M. Parkinson's disease, temporomandibular disorders and bruxism: A pilot study. *J Oral Rehabil.* 2018 Nov;45(11):854-863.
16. Liu, X, Liu, J. Biosensors and sensors for dopamine detection. *VIEW.* 2021; 2:20200102.
17. Wieckiewicz M, Bogunia-Kubik K, Mazur G, Danel D, Smardz J, Wojakowska A, Poreba R, Dratwa M, Chaszczewska-Markowska M, Winocur E, Emodi-Perlman A, Martynowicz H. Genetic basis of sleep bruxism and sleep apnea-response to a medical puzzle. *Sci Rep.* 2020 May 4;10(1):74-97.
18. Khoury S, Carra MC, Huynh N, Montplaisir J, Lavigne GJ. Sleep Bruxism-Tooth Grinding Prevalence, Characteristics and Familial Aggregation: A Large Cross-Sectional Survey and Polysomnographic Validation. *Sleep.* 2016 Nov;39(11):2049-2056.
19. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. *Chest.* 2001 Jan;119(1):53-61.
20. Câmara-Souza MB, de Figueredo OMC, Rodrigues Garcia RCM. Association of sleep bruxism with oral health-related quality of life and sleep quality. *Clin Oral Investig.* 2019 Jan;23(1):245-251.
21. Saczuk K, Wilmont P, Pawlak Ł, Łukomska-Szymańska M. Bruxism: Aetiology and diagnostics. A literature review. *Prosthodontics.* 2018;68(4):456-463.
22. Ribeiro-Lages MB, Martins ML, Magno MB, Masterson Ferreira D, Tavares-Silva CM, Fonseca-Gonçalves A, Serra-Negra JM, Maia LC. Is there association between dental malocclusion and bruxism? A systematic review and meta-analysis. *J Oral Rehabil.* 2020 Oct;47(10):1304-1318.
23. Yaribeygi H, Panahi Y, Sahraei H, Johnston TP, Sahebkar A. The impact of stress on body function: A review. *EXCLI J.* 2017 Jul 21;16:1057-1072.
24. Lu S, Wei F, Li G. The evolution of the concept of stress and the framework of the stress system. *Cell Stress.* 2021 Apr 26;5(6):76-85
25. Emodi-Perlman A, Eli I. One year into the COVID-19 pandemic - temporomandibular disorders and bruxism: What we have learned and what we can do to improve our manner of treatment. *Dent Med Probl.* 2021 Apr-Jun;58(2):215-218.
26. de Baat C, Verhoeff MC, Ahlberg J, Manfredini D, Winocur E, Zeevers P, Rozema F, Vissink A, Lobbezoo F. Medications and addictive substances potentially inducing or attenuating sleep bruxism and/or awake bruxism. *J Oral Rehabil.* 2021 Mar;48(3):343-354.
27. Frosztega W, Wieckiewicz M, Nowacki D, Michalek-Zrabkowska M, Poreba R, Wojakowska A, Kancłerska J, Mazur G, Martynowicz H. Polysomnographic Assessment of Effects of Tobacco Smoking and Alcohol Consumption on Sleep Bruxism Intensity. *J Clin Med.* 2022 Dec 15;11(24):74-53.
28. Du C, Zan MCH, Cho MJ, Fenton JI, Hsiao PY, Hsiao R, Keaver L, Lai CC, Lee H, Ludy MJ, Shen W, Swee WCS, Thrivikraman J, Tseng KW, Tseng WC, Doak S, Folk SYL, Tucker RM. The Effects of Sleep Quality and Resilience on Perceived Stress, Dietary Behaviors, and Alcohol Misuse: A Mediation-Moderation Analysis of Higher Education Students from Asia, Europe, and North America during the COVID-19 Pandemic. *Nutrients.* 2021 Jan 29;13(2):442.
29. Purani H, Friedrichsen S, Allen AM. Sleep quality in cigarette smokers: Associations with smoking-related outcomes and exercise. *Addict Behav.* 2019 Mar;90:71-76.
30. Teoh L, Moses G, McCullough MJ. Oral manifestations of illicit drug use. *Aust Dent J.* 2019 Sep;64(3):213-222.

31. Smardz J, Martynowicz H, Wojakowska A, Michalek-Zrabkowska M, Mazur G, Wieckiewicz M.
Correlation between Sleep Bruxism, Stress, and Depression-A Polysomnographic Study. J Clin Med. 2019 Aug
29;8(9):1344