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Review

REVIEW

# Sleep bruxism and obstructive sleep apnea: association, causality or spurious finding? A scoping review

Patrícia Pauletto[1,2,*], Helena Polmann[1,2], Jéssica Conti Réus[1,2], Carla Massignan[2,3], Beatriz Dulcinéia Mendes de Souza[1], David Gozal[4], Gilles Lavigne[5], Carlos Flores-Mir[6] and Graziela De Luca Canto[1,2]

[1]Department of Dentistry, Federal University of Santa Catarina, Florianópolis, Brazil, [2]Brazilian Centre for Evidence-Based Research (COBE), Federal University of Santa Catarina, Florianópolis, Brazil, [3]Department of Dentistry, University of Brasília, Brasília, Brazil, [4]Department of Child Health, University of Missouri, Columbia, Missouri, United States, [5]Department of Dentistry, Faculty of Dental Medicine, Université de Montreal, Montréal, Canada and [6]Department of Dentistry, University of Alberta, Edmonton, Canada

*Corresponding author. Patrícia Pauletto, The Federal University of Santa Catarina, Department of Dentistry, Health Sciences Center Delfino Conti, S/N, Trindade, Florianópolis, Santa Catarina 88040-900, Brazil. Email: patricia.pauletto.p@gmail.com.

## Abstract

**Study Objectives:** To evaluate the available evidence on the putative relationships between sleep bruxism (SB) and, obstructive sleep apnea (OSA) to assess the extent of research on this topic, and to formulate suggestions for future research.

**Methods:** A scoping review including studies examining temporal and overall association and prevalence of SB and OSA was performed. Six main databases and gray literature were searched. The studies selection was conducted by three independent reviewers. A narrative synthesis of the results was carried out.

**Results:** Thirteen studies in adults and eight studies in children were finally included. The median of concomitant conditions prevalence was 39.3% in adults and 26.1% in children. Marked methodological variability was identified among studies in adults and even more when we compared detection methods in children. No significant association between OSA and SB emerged in most studies in adults, while an association may be possible in children.

**Conclusions:** Based on the current literature, it is not possible to confirm that there is a relationship between SB and OSA in adults. In patients under pediatric care, although this association seems plausible, there is currently insufficient supportive evidence. Standardized validated methodologies for identifying SB should be consistently used in both populations before reaching any conclusion regarding such association. Furthermore, assessment of shared phenotypes between patients with SB and patients with OSA may reveal new insights that will contribute to personalized approaches aiming to optimize the management of such comorbidities.

### Statement of Significance

Studying a possible relationship between sleep bruxism (SB) and obstructive sleep apnea (OSA) is of interest to physicians and dentists as it will allow improved comprehensive management in presence of either comorbidity. The present synthesis reveals that it is not possible to confirm a relation between SB and OSA in adult patients due to the large variability and lack of standardized methods. In patients under pediatric care, although this association might be possible, it is not conclusive from the current literature. Phenotyping of the overlap between these comorbidities may reveal specificity in a subgroup that will contribute to personalized optimal management of the complex interactions between these conditions.

**Key words:** sleep bruxism; obstructive sleep apnea; scoping review; association; prevalence

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## Introduction

Sleep bruxism (SB) has been defined as a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) activity, and it is not a movement disorder or a sleep disorder in otherwise healthy individuals [1]. The prevalence of SB in adults has been reported to be 12.8 ± 3.1% [2], while the prevalence of SB assessed by polysomnography (PSG) alone was found to be at 7.4% and PSG with a questionnaire at 5.5% [3]. In children, the prevalence of SB has varied widely and is estimated at around 3.5% to 46.0% [4].

Sleep bruxism has long been viewed as a villain in dentistry due to its detrimental effects on the stomatognathic system. Tooth wear and damage [5], morning fatigue in the masticatory muscles [6], reduction in bite force [7], technical and biological complications in dental implants [8,9], and higher failure in prostheses [8,10] are among some problems that have been related to SB. Furthermore, the presence of SB seems to negatively influence sleep quality and quality of life [11–13].

On the other hand, SB has been evoked as a possible "good guy" in specific situations. For example, studies that evaluated gastroesophageal reflux disease have raised the possibility of SB serving as a protective factor [14,15]. Some authors suggested that SB might exert a protective role to maintain breathing patency and attenuate the severity and occurrence of obstructive sleep apnea (OSA) [16–20]. Nonetheless, such hypotheses are not yet supported by conclusive evidence.

Nowadays, the literature has avoided using the expression "diagnosis of bruxism", preferring the use of the terms "assessment" or "detection" since in otherwise healthy individuals, SB is considered a physiological motor behavior [1,6]. SB detection remains a challenge in clinical practice. In 2018, the International Consensus for the Assessment of Bruxism proposed the following classification: (1) possible SB, exclusively based on self-report; (2) probable SB, based on a positive clinical inspection result with or without a positive self-report; and (3) definitive SB, based on a positive measurement tool assessment with or without positive self-report and/or positive clinical inspection [1]. Accordingly, definitive SB can be confirmed with the use of electromyography (EMG) recording masticatory muscle during sleep, although alternative methodologies have been recently proposed [21]. When the presence of comorbidities is suspected, EMG activity recordings of the masticatory muscles should ideally be part of a more comprehensive examination, i.e. PSG that includes respiratory variables and, when possible, audio and video recordings [3,22–24].

OSA is a highly prevalent sleep disorder that involves either cessation or significant decreases in airflow in the presence of augmented breathing efforts in the context of increased upper airway resistance. OSA is the most common type of sleep-disordered breathing. The recurrent upper airway collapse episodes during sleep are associated with recurrent oxyhemoglobin desaturations and arousals from sleep [25]. A systematic review (SR) suggested that the overall OSA prevalence ranged from 9 to 38% and was higher in adult men [26]. In children, the prevalence estimates vary depending on the populations studied and the stringency of the diagnostic criteria being considered, but estimates are traditionally reported to range between 1 to 5% [27]. OSA is not adequately managed in the general population and remains undiagnosed and non-treated in a substantial portion of the population [28]. Furthermore, unrecognized OSA has a substantial economic impact on healthcare systems [29,30].

Several authors [1,19,31] have suggested a possible relationship between SB and OSA; however, this association was only explored by detecting the presence of the two conditions [16,32–34]. Moreover, two studies challenged the suggested cause and effect temporal relationship between SB and OSA, whether the SB episodes happen before or after the OSA episode [35,36].

Due to the importance of this topic for clinical practice, three SRs focused on adult patients were published, and the general conclusion is that there was not enough conclusive evidence to support such an association [37–39]. For this reason, we elected to conduct a scoping review to provide a more comprehensive perspective of related knowledge, map existing literature, to examine how research is conducted in this field, analyze knowledge gaps and suggest future research [40]. Thus, the objective of this scoping review is to assess the relationship between OSA and SB in both adults and children, to evaluate the research conducted to date in this area and formulate suggestions for future investigation.

## Methods

### Protocol and registration

This scoping review was reported according to the Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) Checklist [41] (Supplementary Appendix S1). The protocol of this study was registered on the Open Science Framework registration platform (https://osf.io/) under the identification code DOI:10.17605/OSF.IO/CZWJ4.

### Eligibility criteria

We included observational studies that assessed the concurrent prevalence, association between SB and OSA, or temporal association (if the SB episode happens before or after the apneic event) and respiratory variables, without a limited period of publication dates or any language restrictions.

Any kind of "association", that can occur by coincidence or chance, happening at the same time or in sequence, one resulting from the other, as a cause, or due to shared risk factors were considered.

Two types of studies were included:

(1) Studies in adults (>18 years), in which SB and OSA were both assessed by using PSG.
(2) Studies in children (<12 years), in which any detection method (PSG, questionnaire, and clinical evaluation) were used.

### Exclusion criteria

1. Studies that did not evaluate the relationship between SB and OSA;
2. Studies in adults that did not detect the SB and OSA by PSG;
3. Reviews, letters, books, conference abstracts, case reports, case series, opinion articles, technique articles, posters,

guidelines, short paper, pilot studies, mechanism evaluation studies;
4. Full-text or data not available, even after three attempts to contact the corresponding authors over three weeks.

### Information sources

A search strategy developed with the help of an experienced health science librarian was applied on six databases: Embase, LILACS, Livivo, PubMed, Web of Science, and Scopus. Gray literature was searched on Proquest Dissertation and Theses, OpenGrey, and Google Scholar. Additionally, experts were contacted for the additional indication of studies for inclusion. Hand searches of bibliographies from included studies were also conducted. The search was carried out on March 17, 2021, and it was updated on August 9, 2021. The references were imported into a reference software manager (EndNote X9 ®; Thomson Reuters, Philadelphia, PA, United States), and the duplicate documents were excluded.

### Search

The electronic search strategy on the PubMed database is presented in Table 1. The search strategies applied in other databases can be found in Supplementary Appendix S2.

### Selection of sources of evidence

The selection of the studies was performed in two phases by three independent reviewers (P.P., H.P., and J.C.R.). In phase-1, titles and abstracts were screened using online software Rayyan® (Qatar Computing Research Institute, Qatar). Next, in phase-2, the same reviewers applied the eligibility criteria to the full-text studies. A fourth author (C.M.) was consulted in both phases if any disagreement arose.

### Data charting process

The charting process was done by an independent reviewer (P.P.) and subsequently independently checked by two other reviewers (H.P. and J.C.R.). Disagreements were resolved at a consensus meeting. The collected data were inserted in a form previously prepared using Microsoft® Excel 16.29.1 (Microsoft Office 2019, Microsoft, Redmond, United States).

Extracted data comprised: Study Characteristics: author, publication year, country, and study design; Objective of study; Population Characteristics: Sample, sex, mean age, inclusion criteria/setting; Methods: OSA diagnoses criteria and SB detection criteria; Results: Findings/main conclusions, statistical analysis, prevalence of the SB and OSA conditions concomitant; and Additional Information: Report on the sources of funding and conflicts of interest.

### Data items

1. Frequency of concomitant SB and OSA: proportion based on dividing the number of patients affected by SB and concomitant OSA by the number of patients evaluated.
2. Sleep Efficiency (SE): the number of total sleep time/ total time in bed * 100%.
3. Arousal Index (AI): the total number of arousals/ total sleep time * 60.
4. Minimum oxygen saturation (minOSAT): the number of events of 3% drops in oxygen saturation per hour of sleep.
5. Oxygen desaturation index (ODI): the number of times per hour of sleep that the blood's oxygen level drops by a certain degree from baseline.
6. Apnea-Hypopnea Index (AHI): a reduction in breathing amplitude by ≥30% for ≥10 s with a ≥3% decline in blood oxygen saturation or arousal.

### Synthesis of results

The results were presented in two tabular summaries according to study characteristics, the study's objective, population characteristics, methods, results, and additional information in adult and child populations. Two figures regarding adult and children's studies were generated to highlight the associated findings between SB and OSA.

A narrative summary was drafted to synthesize the findings and describe the evidence identified concerning the review objective.

## Results

### Selection of sources of evidence

A total of 1,161 articles were identified in the databases and 42 in the gray literature repositories, as shown in the PRISMA flow chart (Figure 1). After the removal of duplicates, 700 studies were screened by title and abstracts. A complete reading of 41 articles was performed, and 21 studies were included in this scoping review. Supplementary Appendix S3 displays references of excluded articles alongside reasons for exclusion.

### Characteristics of sources of evidence

Twenty-one studies were included and all of them were published in English. The studies in adults ($n$ = 13) were published between 1986 [42] and 2020 [33,34,43,46]. These studies were carried out in Brazil [33,34,44], Canada [35], Japan [17,32,36], Poland [43,45], Singapore [16], South Korea [46], and United States [42,47].

The studies in children ($n$ = 8) were conducted in Brazil [48–50], India [51], Italy [52], Japan [53], and United States [54,55]. They were published between 2008 [50] and 2020 [48,52].

**Table 1.** Search strategy in PubMed database

| |
|---|
| ("bruxism"[MeSH Terms] OR "bruxism"[All Fields]) AND ("sleep apnea, obstructive"[MeSH Terms] OR "obstructive sleep apnea"[All Fields] OR ("OSA"[All Fields] OR "SDB"[All Fields] OR "sleep disordered breathing"[All Fields]) |

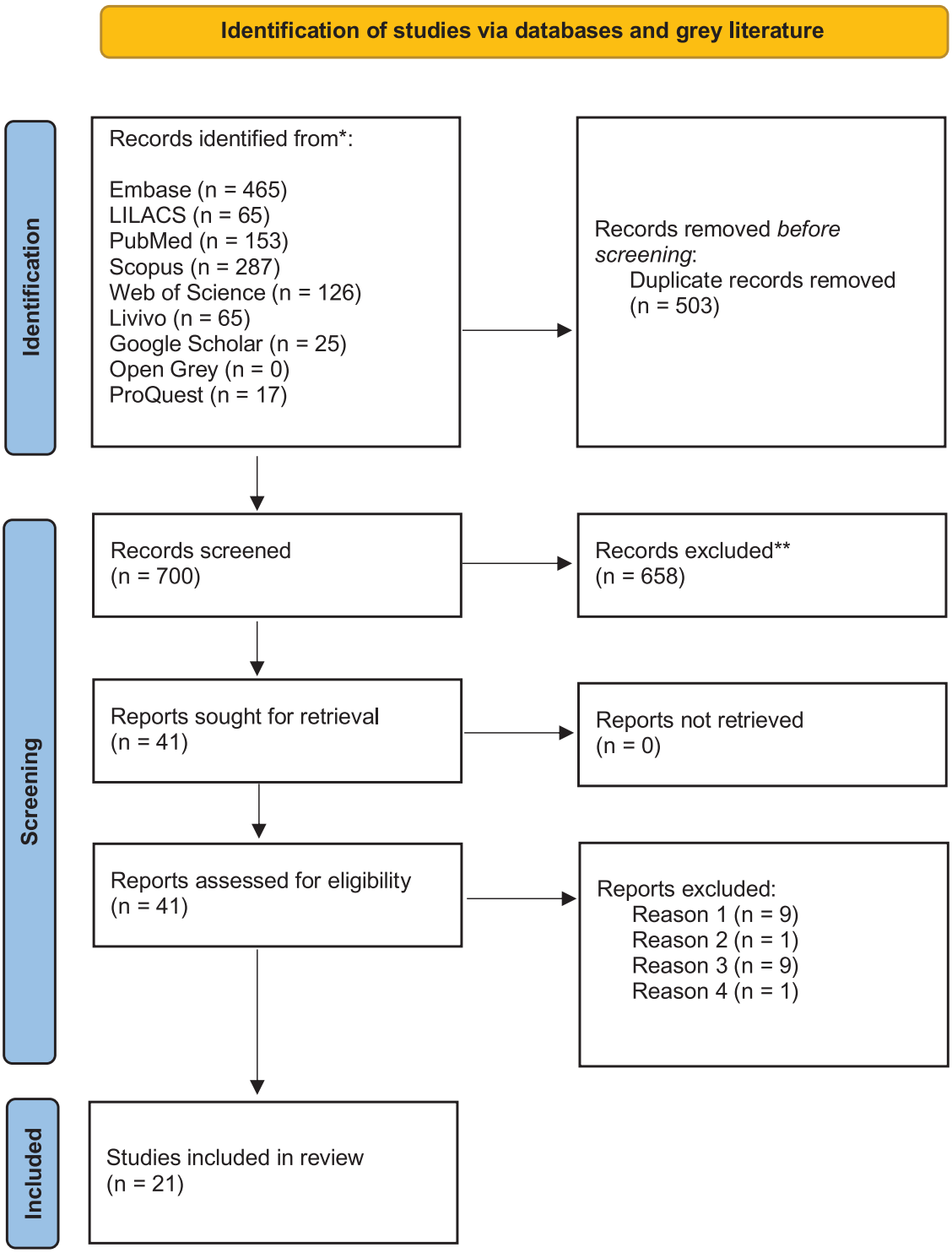


**Figure 1.** Flow diagram of literature search and selection criteria.

## Results of individual sources of evidence

Complete data referring to the individual characteristics of the studies in adults and children are shown in Tables 2 and 3, respectively.

## Sleep bruxism and obstructive sleep apnea assessment

### *Adults*

SB and OSA conditions were assessed by using PSG.

### Children

#### *Sleep bruxism detection*

One study used clinical evaluation only [55], three studies used clinical evaluation plus questionnaire [48,49,52], three studies only questionnaire [50,51,53], and only one PSG [54].

#### *Obstructive Sleep Apnea diagnosis*

Three studies used PSG [50,54,55], three studies used only questionnaires answered by the caretakers [48,51,53], and two studies used

**Table 2.** Characteristics of the included studies in adults ($n$ = 13)

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample (N), Sex (M/F)<br>Mean age (years) | Inclusion Criteria/Setting | OSA Diagnoses Criteria | SB Criteria | Findings/ Main Conclusion<br>Statistical Analysis | Prevalence SB/OSA concomitant | Report on the sources of funding<br>Conflict of Interest |
| Coelho et al., 2012 [44]<br>Brazil<br>Descriptive | To investigate the prevalence of comorbidity between the SB and OSA in the polysomnographic findings of patients with sleep disturbance. | N = 909 (NR)<br>NR | Medical records of patients of both genders, with suspected sleep disorders, in the period from March 2007 to June 2011. | Apnea plus hypopnea index per hour of sleep, with the occurrence of at least five apneas plus hypopnea per hour of sleep, added to clinical symptoms, the most important of which are loud snoring and excessive daytime sleepiness. | At least two episodes of rhythmic activity of masticatory muscles (ARMM) associated with the sound of "gnashing of teeth"; more than four ARMM episodes per hour of sleep, with no "gritting your teeth" sound; more than five electromyographic bursts per AMMR episodes; or more than 25 EMG bursts per hour of sleep. | The SB/OSA comorbidity was prevalent in the surveyed population.<br>Descriptive | 5.28% (48/909) | NR<br>NR |
| Holanda et al., 2020 [34]<br>Brazil<br>Cross-Sectional | To evaluate the association between the diagnosis SB scored by PSG recordings, clinical conditions, and sleep architecture. | N = 116<br>SB: 58 (M = 25, F = 33)<br>Non-SB: 58 (M = 25, F: 33)<br>SB: 42.20 ± 14.52<br>Non-SB: 42.55 ± 14.78 | All PSG recordings and self-reported data obtained from patients (20 years or older) who underwent PSG at the Pelotas Sleep Institute, a private medical outpatient clinic, from January 2015 to December 2017. | NR | RMMA index was greater than 2 episodes per hour of sleep. | SB was diagnosed more frequently in subjects who had fewer OSA events ($p$ = .005). OSA decreased the chances (OR 0.55; 95 % CI: 0.23–1.30; $p$ = .173) of an SB diagnosis.The AHI ($p$ = .002) was all lower in bruxers than in nonbruxers.<br>Logistic Regression test. | 15.5% (58/116) | This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.<br>None declared. |
| Hosoya et al., 2014 [17]<br>Japan<br>Cross-Sectional | To examine the relationships between SB and sleep respiratory events in patients with OSA and healthy volunteers. | N = 83<br>OSA:67 Non-OSA:16<br>OSA: 54.3 ± 13.2 Non-OSA: 23.9 ± 5.5 | Patients with suspected OSA who consulted the Respiratory Medicine Department of Tohoku University Hospital between May 2010 and August 2011. | **Apnea:** cessation of airflow lasting 10 s or more.**Hypopnea:** a greater than 50% decrease in the thoracoabdominal amplitude associated with a greater than 3% decline in the oxygen saturation from the preceding value. | Subjects were diagnosed with SB when they had more than four bruxism events per hour of sleep. | **Comparison:** Apnea/hypopnea and desaturation events occurred significantly more frequently in subjects with than without SB. There were no significant differences in sleep efficiency, micro-arousal event, between subjects with and without SB.**Correlation:** The frequency of SB events was positively correlated with frequencies of each of the following: apnea/hypopnea, OSA, micro-arousal and oxygen desaturation.<br>Mann-Whitney U test and Spearman correlation test | 47.8% (32/67) | NR<br>None to declare. |

**Table 2.** Continued

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample (N), Sex (M/F)<br>Mean age (years) | Inclusion Criteria/Setting | OSA Diagnoses Criteria | SB Criteria | Findings/ Main Conclusion<br>Statistical Analysis | Prevalence SB/OSA concomitant | Report on the sources of funding<br>Conflict of Interest |
| Kim et al., 2020 [46]<br>South Korea<br>Cross-Sectional | To identify the factors associated with SB episodes in patients with OSA using in-laboratory PSG records. | N = 100<br>SB:10 (M)<br>Non-SB: 90 (M = 80, F = 10)<br>SB: 43.4 ± 16.1<br>Non-SB: 48.5 ± 13.9) | Patients who visited the hospital for snoring, OSA, or drowsiness during the day and underwent in-laboratory full-night PSG between March 2017 and February 2019. | **Apnea:** a drop in the peak signal excursion by ≥ 90% of the pre-event baseline using a thermistor or as complete airflow cessation for at least 10 s. **Hypopnea:** a peak signal excursion drops by ≥ 30% of the pre-event baseline using a pressure cannula and a ≥ 3% oxygen desaturation from the pre-event baseline, whether or not it was associated with arousal.**AHI:** was defined as the total number of apnea and hypopnea events within 1 h of sleep. | A constant burst episode lasting longer than 2 s or three or more bursts that were 0.25–2 s long was a SB episode. Simultaneously, tooth grinding sounds and typical jaw movements were required to consider these EMG activities or bursts as SB episodes. | Lower AHI, and higher oxygen saturation were associated with SB episodes in patients with OSA.<br>Mann-Whitney U | 10 % (10/100) | This research received no external funding. The authors declare no conflict of interest. |
| Maluly et al., 2020 [33]<br>Brazil<br>Cross-Sectional | To assess the strength of the associations between SB, insomnia and OSA in a general population. | N = 620<br>SB:56 (M = 22, F = 34).<br>Non-SB: 564 (M = 247, F = 317).<br>NR | General representative population of São Paulo (EPISONO study. | The apnea and AHI were classified as events per hour: normal from 0 to 5, mild from 5 to 15, moderate from 15 to 30, and severe if greater than 30. | Participants who responded positively to the questionnaire in respect of SB symptoms and had ≥ 2 RMMA episodes per hour in PSG recordings. | No statistical difference was found.<br>Logistic regression | 39.3% (22/56) | NR<br>None |
| Martynowicz et al., 2019 [45]<br>Poland<br>Cross-Sectional | To assess the relationship between SB and OSA. | N = 110 (M = 66, F = 44)<br>51.02 ± 14.19 | Patients with age between 18 and 90 years with clinical suspicion of OSA who went toDepartment and Clinic of Internal Diseases, Occupational Diseases, Hypertension, and Clinical Oncology at the Wroclaw Medical University betweenMarch 2017 and March 2019. | **Apnea:** the absence of airflow for ≥ 10 s. **Hypopnea**: was defined as a reduction in the amplitude of breathing by ≥ 30% for ≥ 10 s with a ≥ 3% decline in blood oxygen saturation or arousal. | For the consideration of SB, EMG bursts should not be separated by > 3 s to be considered part of the same episode, and EMG activity had to be at least twice the amplitude of the background EMG. | The relationship between OSA and SB depends on the degree of severity of OSA. From the results of the present study, mild-to-moderate OSA is associated with SB in the group of patients with increased risk of OSA.<br>Mann-Whitney U test, chi-square test and correlation. | 45.4% (50/110)<br>Mild OSA: 61.6% (20/33)<br>Moderate: 64.3% (18/28)<br>Severe: 35.3% (12/34) | This research received no external funding. The authors declare no conflict of interest. |
| Okeson et al., 1991 [47]<br>United States<br>Cross-Sectional | To determine if patients with OSA experience morebruxing events than patients without OSA. | N = 24 (male)<br>SDB = 57 ± 11.5 years<br>Non-SDB = 57 ± 11.7 years | Subjects who were referred to the University of Kentucky sleep laboratory for evaluation of OSA. | **Apnea:** was defined as cessation of air flow at the nose and mouth for 10 s or longer during sleep. **Hypopnea:** was defined as a 50% reduction in the thermocouple signal amplitude associated with a 4% fall in oxygen saturation. The number of apneas and hypopneas were combined and recorded as SDB events. | A clench was defined as activity of the masseter muscle exceeding 40% of the maximum clench of the muscle and lasting for 2 seconds or longer. | The SDB group had a total of 193 bruxing events, an average of 16.1 per subject (range 0 to 43, SD 11.6). The control group had a total of 314 bruxing events. There was an average of 26.2 bruxing events per subject (range 0 to 57, SD 23.9). There were no statistical differences.<br>Student's t-test | NR | NR<br>NR |

**Table 2.** Continued

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample (N), Sex (M/F)<br>Mean age (years) | Inclusion Criteria/Setting | OSA Diagnoses Criteria | SB Criteria | Findings/ Main Conclusion<br>Statistical Analysis | Prevalence SB/OSA concomitant | Report on the sources of funding<br>Conflict of Interest |
| Phillips et al., 1986 [42]<br>United States<br>Cross-Sectional | To determine the relationship-between OSA and parafunctional activity. | N = 24 (M = 21, F = 3).<br>OSA:14 Non-OSA:10<br>OSA: 52 ± 15.9 years<br>Non- OSA: 50.2 ± 16.4 years | Patients referred to the University of Kentucky Sleep Apnea Laboratory during the four-month period between July and October 1985 for evaluation of possible OSA. | **Apnea:** was defined as cessation of airflow at the nose and mouth for 10 s or longer during sleep.**Hypopnea:** was defined as a reduction in airflow associated with a 4% fall in oxygen saturation. | A clench was defined as activity of the masseter muscle exceeding 40% of the maximum clench of that muscle and lasting for two seconds or longer. | Nocturnal clenching was slightly higher in patients with OSA than those without (12.2 vs 7.6 $p = .18$), and there was a correlation between the clench index and AHI by linear regression ($r = 0.49$, $p < .05$). There were significant falls in both the AHI (64.4 ± 28.8 vs 36.5 ± 36.7, $p = .02$) and clench index (12.5 ± 12.1 vs 7.0 ± 8.6, $p = .04$) in the lateral decubitus vs supine sleeping positions.<br>NR | NR<br>NR | NR<br>NR |
| Saito et al., 2013 [36]<br>Japan<br>Descriptive | To investigate the temporal association between SB events and OSA events. | N = 10 (male)<br>46.7 ± 11.5 years | Subjects with confirmed OSA and SB. | **Apnea-hypopnea event:** American Academy of Sleep Medicine | **SB:**Events were identified as RMMA. The amplitude was set twice the baseline activity. | In patients with concomitant OSA and SB, most SB events occurred after OSA events, suggesting that SB events occurring close to OSA events is a secondary form of SB. | NR | NR<br>No conflict of interest declared. |
| Saito et al., 2016 [32]<br>Japan<br>Cross-Sectional | To investigate, in a population reporting awareness of both OSA and SB, the associations between each specific breathing and jaw muscle event. | N = 59 (M = 47, F = 12)<br>44.8 ± 10.8 years | Japanese patients reporting awareness of breathing cessation and tooth grinding as well as signs and symptoms of OSA and SB. | Apnea and hypopnea events were scored according to standard criteria, with an AHI threshold of 5 events/h or more. | RMMA/SB Episode/h > 2 and/ or RMMA/SB bursts/h > 25. | AHI did not show a significant correlation with RMMA/SB episodes nor with RMMA/SB bursts. Sleep arousals in patients with concomitant SB and OSA are not strongly associated with onset of RMMA/SB.<br>Spearman correlation test. | 50.84% (30/59) | No<br>No |
| Sjoholm et al., 2000 [35]<br>Canada<br>Cross-Sectional | To test the hypothesis of a direct association between SDB and SB. | N = 21 (M = 19, F = 2)<br>40.0 ± 29.2 years | Patients with OSA. | **Apnea:** was defined as the cessation of airflow for at least 10 s. **Hypopneas:** were defined as a decrease of more than 50% in thoracoabdominal amplitude for at least 10 s. | (1) Subjective estimation: teeth grinding, or clenching was reported by the patient one to two nights or more per week.<br>(2) Clinical: the number and extent of visible wear facets on tooth enamel (attrition), the presence of masticatory muscle fatigue, and/or discomfort of the temporomandibular joint.<br>(3) Masseter EMG: If the arbitrary cut-off point of 2.5 rhythmic jaw-movement episodes per hour was exceeded, the participant was as a bruxer during that night. | Masseter contraction episodes were associated with the termination of apnea or hypopnea episodes in 3.5% of the mild group and 14.4% of the moderate group ($p < 0.05$). It appears that SB is rarely directly associated with apneic events.<br>Student's t-test | 47.61% (10/21)<br>Mild OSA 54% (6/11)<br>Moderate OSA 40% (4/10) | NR<br>NR |

**Table 2.** Continued

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample (N), Sex (M/F)<br>Mean age (years) | Inclusion Criteria/Setting | OSA Diagnoses Criteria | SB Criteria | Findings/ Main Conclusion<br>Statistical Analysis | Prevalence SB/OSA concomitant | Report on the sources of funding<br>Conflict of Interest |
| Smardz et al., 2020 [43]<br>Poland<br>Cross-Sectional | To evaluate the relationship between SDB and SB. | N = 77 (M = 21, F = 56)<br>SB:58<br>Non-SB:19<br>34.8 ± 10.8 years | Patients above 18 years age, with a positive diagnosis of probable SB of the Clinic of Prosthetic Dentistry operating in the Department of Prosthetic Dentistry at Wroclaw Medical University. | **Apnea:** defined as the absence of airflow through the airway for more than 10 s. **Hypopnea:** has been defined as a decrease in respiratory amplitude by more than 30% for more than 10 s, followed by subsequent blood desaturation of more than 3% or subsequent arousal. | Episodes qualified as bruxism if there was a RMMA, often accompanied by grinding sounds and characteristic movements in the mandible occurring after a minimum of 3s break from the last muscle activity. Episodes were classified as phasic (lasting 0.25 to 2s), tonic (lasting more than 2s), or mixed. | Both groups (SB and Non-SB) not differ statistically significantly in terms of oxygen desaturation index (U = 540.0, $p$ = .90) and AHI (U = 531.5, $p$ = .82). Quantitative analysis showed a lack of a statistically significant relationship between AHI and ODI in the patients with and without SB.<br>Mann-Whitney U test | NR | Authors have no competing interests to declare.<br>This study was co-financed by financial resources for Young Researchers of Wroclaw Medical University, Poland (STM. B022.17.011). The funding source was not involved in the study design, in the collection, analysis and interpretation of data, in the writing of the report, and in the decision to submit the article for publication. |
| Tan et al., 2019 [16]<br>Singapore<br>Cross-Sectional | To determine the prevalence of SB in adult patients with OSA, to assess the association between SB and OSA in terms of sleep macrostructure and respiratory parameters and to determine possible OSA risk factors for SB. | N = 147 (M = 100, F = 47)<br>SB: 49 (M = 37, F = 12)<br>Non-SB: 98 (M = 63, F = 35) | Patients diagnosed with mild, moderate, or severe OSA and aged 25 years and above who underwent a PSG at the Ng Teng Fong General Hospital sleep clinic from July 2015 to February 2016. | **Apnea:** was determined as a cessation in airflow of 90% for a minimum period of 10 s. **Hypopnea:** was identified when the airflow dropped by 30% for a period of 10 s, accompanied by an oxygen desaturation of 3%. **AHI**: indicated the severity of OSA through the representation of number of apnea/hypopnea events per hour of sleep. Mild OSA was defined as having an AHI of 5 or more and less than 14 respiratory events per hour, moderate OSA between 15 or more and less than 30 events per hour, and severe OSA as 30 or more events per hour. | SB episodes were established when masseter RMMA exhibited twice the background EMG amplitude and were proceed by a period of > 3 second of stable background EMG. | Of the 147 patients with OSA, 49 (33.3%; 95% CI: 25.7 to 40.9%) were diagnosed with SB, while 98 (66.7; 95% CI: 59.1% to 74.3%) had no SB (descriptive analysis). An association was found between AHI and SB group. Patients with SB had significantly higher Respiratory *Arousal Index* and Oxygen Desaturation Index than patients without SB. This study demonstrated that SB occurs in about one-third of patients with OSA.<br>Mann-Whitney U test. | 33.33 % (49/147) | NR<br>No Conflict of interest. |

**Legend:** AHI: Apnea-Hypopnea Index; CI: Confidence Interval; EMG: Electromyography; F: Female; M: Male; NR: Not Reported; OSA: Obstructive Sleep Apnea; PSG: Polysomnography; RMMA: Rhythmic Masticatory Muscle Activity; SB: Sleep Bruxism; SAHOS: Obstructive sleep apnea-hypopnea syndrome.

**Table 3.** Characteristic of the included studies in children ($n$ = 8).

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample<br>Sex (M/F)<br>Mean age or range | Inclusion Criteria<br>OSA and SB Assessment | OSA Diagnoses Criteria | SB Criteria | Findings<br>Statistical Analysis | Prevalence SB/OSA con-comitant | Report on the sources of funding<br>Conflict of Interest |
| Castilho et al., 2020 [48]<br>Brazil<br>Cross-Sectional | To study the incidence of mouth breathing and its association with sleep disorders, dental caries, malocclusion, and deleterious oral habits, in children. | 152 (M = 80, F = 72)<br>6–9 years | Children of three schools with different socioeconomic indicators, chosen by the Education Department of the Municipality of Botucatu/SP, Brazil during the period of May 2016 to November 2017.<br>**OSA:**OSA-18<br>**SB:** Questionnaire and clinical evaluation | The result of the OSA-18 questionnaire is given by the sum of the values chosen by the person interviewed for the frequency with which the cited events occur. The numerical value can be translated as low (<60 points), moderate (≥60 points and < 80 points) or high (≥80 points) risk for OSAS by the child. | NR | Children who had a low and moderate risk for OSAS (OSA-1 and OSA-2) had 39% of bruxism. In 29 patients with severe risk for OSAS (OSA-3), the proportions were 67%.<br>A descriptive statistical analysis of the data with frequency and percentage for the qualitative variables. Chi-square orFisher's exact test was performed when necessary | 40.78 % (62/152)<br>OSA mild and moderate: 39% (54/138)<br>OSA severe 67% (8/14) | NR<br>NR |
| Ferreira et al., 2015 [49]<br>Brazil<br>Cross-Sectional | To evaluate the prevalence and to testfor possible associations between SB and OSA. | 496 (M = 249, F = 247)<br>4.49 ± 1.04 years | Children of preschool age (3–6 years old) who live in the city of Taubate.<br>**OSA:** clinical examinations and application of a questionnaire to be answered by the parents, based on a modified version of the Mallampati questionnaire.<br>**SB:** clinical examinations and questionnaire to be answered by the parents. | Those participants who had a Mallampati score of III or IV and whose parents answered "Yes" for all the questions on the OSA questionnaire were diagnosed with sleep apnea. | Clinical examinations evaluated the presence of wear facets on canines and incisors where the worn borders of the teeth fit the wear facet of the antagonist tooth during excursive movements. | An association was found between the presence of SB and OSA, where 11.03% of subjects with SB also exhibit OSA, and 97% of subjects without SB did not present OSA $p < .001$. SB was associated with OSA. Descriptive statistics, and possible associations were tested using a Chi-square test. ANOVA was used to compare the average age among children who were and were not diagnosed with SB and OSA. | 2.82 % (14/496) | The authors would like to thank CNPq (a Brazilian governmental research agency) and the University of Taubate´ for the grant received by the first author (NMRF) to develop this study (PIBIC #072/12).<br>The authors deny any conflict of interest. |
| Goyal et al., 2018 [51]<br>India<br>Cross-Sectional | To estimate the prevalence of OSA in school children aged 5–10 years and its association with academic performance. | 1346 (M = 836, F = 510)<br>NR<br>5–10 years | Children aged between 5 and 10 years at three purposively selected schools of Bhopal, India, from July 2015 to November 2015.<br>**OSA:** Validated 22-item pediatrics sleep-related breathing disorder (SRBD).<br>**SB:** Questionnaire. | OSA: SRBD > 33%. | NR | Students with positive SRBD had higher chances of having bruxism (29% vs. 15.4%; $p < .0001$, adjusted OR: 1.7; 95% CI: 1.1–2.6).<br>Logistic regression analysis | NR | No<br>There are no conflicts of interest. |
| Gregório et al., 2008 [50]<br>Brazil<br>Cross-Sectional | To investigate the symptoms most frequently found in children with a PSG diagnosis of OSA. | 38 (M = 19, F = 19) ** 3 children who were nonapneic.<br>8.4 ± 3.99 years | Children consecutively referred to the sleep laboratory with suspicion of OSAHS between June of 2003 and December of 2004.<br>**OSA:** PSG<br>**SB:**Pre-sleep questionnaire | **OSA:** was defined as a decrease of at least 50% of the baseline flow associated with the desaturation of 4% or more and/or micro-arousals.**AHI:** was calculated based on the number of obstructive apneas and hypopnea events occurring during one hour of sleep. The adopted classification were: normal with an AHI < 1; mild with an AHI between 1 and 5; moderate when the AHI is between 5 and 10 and serious when the AHI is > 10. | NR | In children with OSA, bruxism was seen in 31.3%. All the children diagnosed with severe OSA also presented bruxism.<br>Student's t-test or Mann-Whitney test | 31.3 % (11/35) | NR<br>NR |

**Table 3.** Continued

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample<br>Sex (M/F)<br>Mean age or range | Inclusion Criteria<br>OSA and SB Assessment | OSA Diagnoses Criteria | SB Criteria | Findings<br>Statistical Analysis | Prevalence SB/OSA concomitant | Report on the sources of funding<br>Conflict of Interest |
| Segu et al., 2020 [52]<br>Italy<br>Cross-Sectional | To assess such correlations in a large sample of school children between SB and sleep disorders. | 741 (M = 409, F = 332)<br>11.26 ± 4.05 years | A group of 741 consecutive children of a private orthodontic practice between January 2016 and May 2019.<br>**OSA and SB:**Clinical evaluation and questionnaire Sleep Disturbance Scale for Children. | NR | NR | The Spearman test reported a significant correlation between parental-reported tooth grinding and sleep apnea (r = 0.092). There is a significant correlation between parental-reported tooth grinding and OSA.<br>Spearman test | NR | NR<br>The authors declare that there are no conflicts of interest. |
| Sheldon SH, 2010 [54]<br>United States<br>Cross-Sectional | To evaluate the presence of SB noted on comprehensive polysomnography in 119 consecutive patients with possible OSA. | 119 (NR)<br>7.0 ± 4.0 years | Patients between ages 3 and 16 years, referred to the Pediatric Sleep Medicine Center with symptoms of snoring.<br>**OSA and SB:** PSG | **OSA:** was scored when a greater than 90% decrease was present in the signal amplitude for 90% or greater of the entire respiratory event compared with pre-event baseline amplitude.<br>**Hypopnea:** was scored if the event was associated with a 50% or greater decline in the amplitude of the nasal pressure for at least two respiratory efforts, the fall in nasal pressure lasted 90% or more of the entire respiratory event compared with the amplitude preceding the event, and the event was associated with an arousal, awakening, or 3% or greater oxygen desaturation. | Bruxism was defined as three or more rhythmic contractions of the temporalis muscles, as measured with temporalis muscle electromyogram (EMG), occurring during NREM or REM sleep lasting more than 3 seconds, but less than 15 seconds. | Sleep-related rhythmic temporalis muscle activity associated with arousalis significantly associated with indices of respiratory disturbance, particular the Arousal Index, and AHI, as measured using standard pediatric polysomnographic techniques.<br>Mann-Whitney. | NR | NR<br>NR |
| Singh N, 2011 [55]<br>United States<br>Cross-Sectional | To investigate whether sleep bruxism-related tooth wear could be a clinical marker for pediatric OSA. | 50 (M = 25, F = 25)<br>No OSA (14)<br>Mild OSA (21)<br>Moderate OSA (7)<br>Severe OSA (8)<br>7.6 ± 1.5 years | The subjects were recruited from a pediatric sleep disorder center and a private dental practice.<br>**OSA:** PSG<br>**SB:** Dental wear score. | The AHI scored the OSA events and was used to classify the subjects into those with OSA and those with no OSA (controls; AHI < 1). | Score 1 = no dental wear, or obvious wear of enamel or wear through the enamel to the dentin in single spots.<br>Score 2 = wear of the dentin up to one-third of the crown height<br>Score 3 = wear of the dentin more than one-third of the crown height. | The results revealed no statistically significant association between both the presence and severity of OSA and the presence and severity of sleep bruxism-related dental wear. SB related dental wear is not a clinical indicator of pediatric OSA.<br>Descriptive | NR | Dr. Nischal Singh is NOT receiving any financial sponsorship or remuneration to conduct this study.<br>There is no conflict of interest that would compromise his position or this research study. |

**Table 3.** Continued

| Study | Objective | Population | | Methods | | Results | | Additional Information |
|---|---|---|---|---|---|---|---|---|
| Author, Year<br>Country<br>Study Design | Objective | Sample<br>Sex (M/F)<br>Mean age or range | Inclusion Criteria<br>OSA and SB Assessment | OSA Diagnoses Criteria | SB Criteria | Findings<br>Statistical Analysis | Prevalence SB/OSA concomitant | Report on the sources of funding<br>Conflict of Interest |
| Tachibana et al., 2016 [53]<br>Japan<br>Cross-Sectional | To investigate the prevalence of sleep bruxism in children in Japan, and its relationships with sleep-related factors and daytime problematic behavior. | 6023 (M = 2975, F = 3048)<br>NR | 2191 preschoolers and 3832 elementary school students from Japan were subject to analysis.<br>**OSA and SB:** Japanese Sleep Questionnaire | Higher scores indicated greater signs of sleep disorders or deleterious sleep habits. | SB Does he/she grind his/her teeth during sleep by rating on a 6-point intensity Likert scale. | Sleep bruxism significantly correlated with OSA.<br>Logistic regression | 21% (1263/6023) | Fundings from the Challenge to Intractable Oral Diseases and the Grantin-Aid for Scientific Research and from the Center of Innovation Science and Technology based Radical Innovation and Entrepreneurship Program.<br>None to declare. |

**Legend:** AHI: Apnea-Hypopnea Index; F: female; M: male; NR: Not Reported; OSA: Obstructive Sleep Apnea; PSG: Polysomnography; SB: Sleep Bruxism; SDB: Sleep-disordered breathing.

a questionnaire plus clinical evaluation to detect OSA [49,52]. More details are presented in Table 3.

### Obstructive sleep apnea diagnostic criteria

The studies conducted in adults [16,17,32–36,42–47] adopted the American Academy of Sleep Medicine (AASM) [56] recommended or acceptable diagnostic criteria for OSA in the PSG analysis (Table 2).

In children, there was significant variation in the diagnostic criteria for OSA due to the different types of questionnaires used for detection (Table 3).

### Sleep bruxism detection criteria

In general terms, the studies in adults considered SB events as increases in the masseter EMG activity of at least twice the amplitude of the background EMG [16,17,32,33,36,45,46]. Two studies [42,47] considered an event when the activity of the masseter muscle exceeded 40% of the maximum clench of the muscle. However, other studies did not report the criterion for SB events [34,35,43,44]. The number of episodes of SB used as a classification score is described in Table 2.

The criteria used by studies in children to detect bruxism are shown in Table 3.

## Synthesis of Results

### Prevalence of concurrent sleep bruxism and obstructive sleep apnea

#### *Adults*

The prevalence of co-occurrence of the two conditions among studies that used PSG to detect both conditions ranged from 5.28% [44] to 50.84% [32] (median 39.3%; interquartile range 34.9%).

#### *Children*

The prevalence of the two concurrent conditions ranged from 2.82% [49] to 40.78% [48] (median 26.1%; interquartile range 31.0%).

### Association between sleep bruxism and obstructive sleep apnea

#### *Adults*

Ten studies [16,17,32–34,42,43,45–47] evaluated the association between SB and OSA, and four studies showed a positive association [16,17,42,45]. Martynowicz et al. [45] reported that this association only occurred in patients with mild and moderate OSA. Two studies revealed a negative association [34,46]; that is, fewer episodes of OSA occurred in patients with detected SB. Four studies found no association [32,33,43,47] (Figure 2).

#### *Children*

Most studies demonstrated a positive association; however, as mentioned, there was no homogeneity in the detection methods [49,51–54] (Figure 3). Although an association has been shown to be present in studies involving children, it is worth mentioning that most studies used questionnaires and clinical evaluation as the method of detection and not the standard reference exam, the PSG.

### Temporal relationship

Two studies evaluated the temporal relationship between SB and OSA. The study by Sjoholm et al., 2000 [35] showed that events between the two conditions seem to be rarely associated. Saito et al. (2013) [36] indicated that most bruxism events occurred

after sleep apnea-hypopnea events and that some occurred before, reducing the strength of a causal, temporal sequence.

### Respiratory variables in adult studies

Findings related to the respiratory variables analyzed (Oxygen Desaturation Index, Sleep Efficiency, Arousal Index, AHI, and Minimum Oxygen Saturation) are shown in Table 4. There were no statistical differences in sleep efficiency between the groups with and without SB [16,17,46]. Oxygen Desaturation Index was associated with patients with SB in two studies [16,17]. Higher Arousal Index and lower Minimum Oxygen Saturation were associated with patients having SB in one study [16], but with a small statistically significant difference, albeit not clinically relevant. Regarding the AHI, there were contradictory results, and one study showed higher AHI in SB participants [17], another in non-SB participants [46] and another study reported that no differences were found [43].

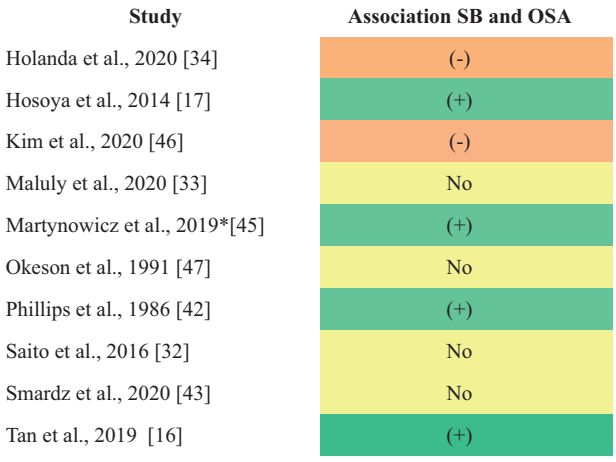


**Figure 2.** Association between SB and OSA in adults, where (+) means positive association, (–) negative association and No (no association). *In the Martynowicz et al., 2019 study, this association was found in mild and moderate OSA.

## Discussion

This scoping review aimed to map the available scientific evidence on the relationship between OSA and SB. A relationship between them cannot be supported in adults at this time. In children, there may be a possible association; however, the evidence is limited since most of the SB diagnostic methods used are heterogeneous. Furthermore, based on the current evidence, studies that did not use PSG data could not be considered reliable [57].

Although OSA is linked to serious morbidities, it is still underdiagnosed and consequently not adequately or timely managed in the general population [45]. This fact emphasizes the importance of identifying factors that could

| Study | Association SB and OSA | SB Detection | OSA Detection |
|---|---|---|---|
| Ferreira et al., 2015 [49] | (+) | Clinical examination and questionnaire. | Clinical examination and questionnaire (Modified version of the Mallampati) |
| Goyal et al., 2018 [51] | (+) | Questionnaire | Validated 22-item pediatrics sleep-related breathing disorder. |
| Segu et al., 2020 [52] | (+) | Clinical evaluation and questionnaire Sleep Disturbance Scale for Children | Clinical evaluation and questionnaire Sleep Disturbance Scale for Children |
| Sheldon SH, 2010 [54] | (+) | PSG | PSG |
| Singh N, 2011 [55] | No | Dental Wear Score | PSG |
| Tachibana et al., 2016 [53] | (+) | Japanese Sleep Questionnaire | Japanese Sleep Questionnaire |

**Figure 3.** Association between SB and OSA in children, where (+) means positive association, and No (no association).

**Table 4.** Results related to the respiratory variables analyzed in adults (ODI, SE, AI, AHI, and minOSAT)

| | Study | SB Mean±SD | Non-SB Mean±SD | *p* value |
|---|---|---|---|---|
| Oxygen Desaturation Index (ODI) | Hosoya et al., 2014 [17] | 33.7 ± 25.0 | 22.4 ± 19.3 | <.05* |
| | Smardz et al., 2020 [43] | 5.09 ± 8.24 | 3.28 ± 3.22 | .90 |
| | Tan et al., 2019 [16] | 35.05 ± 24.75 | 26.1 ± 28.65 | .005* |
| Sleep Efficiency (SE) | Kim et al., 2020 [46] | 86.2 ± 14.1 | 81.5 ± 14.7 | .14 |
| | Hosoya et al., 2014 [17] | 16.7 ± 8.6 | 17.6 ± 8.6 | >.05 |
| | Tan et al., 2019 [16] | 85.20 ± 12.40 | 85.49 ± 13.01 | .83 |
| Arousal Index (AI) | Kim et al., 2020 [46] | 6.9 ± 9.3 | 21.0 ± 26.3 | .13 |
| | Hosoya et al., 2014 [17] | 40.0 ± 18.8 | 31.3 ± 15.9 | >.05 |
| | Tan et al., 2019 [16] | 49.92 ± 18.05 | 43.44 ± 21.07 | .03* |
| Minimum Oxygen Saturation (minOSAT) | Kim et al., 2020 [46] | 83.8 ± 8.5 | 80.4 ± 9.6 | .26 |
| | Smardz et al., 2020 [43] | 88.90 ± 6.34 | 88.16 ± 9.39 | .93 |
| | Tan et al., 2019 [16] | 78.47 ± 10.21 | 81.47 ± 10.76 | .04* |
| Apnea-Hypopnea Index (AHI) | Kim et al., 2020 [46] | 25.5 ± 16.6 | 45.1 ± 28.2 | .03* |
| | Hosoya et al., 2014 [17] | 37.2 ± 22.9 | 27.0 ± 19.7 | <.05* |
| | Smardz et al., 2020 [43] | 5.52 ± 9.40 | 3.49 ± 3.55 | .82 |

indicate an increased probability of having OSA. In addition to physicians, the dentist could serve as one of the primary care professionals to screen patients for risk factors for OSA such as age, gender, body mass index, poor sleep, and especially, orofacial pain or morning headache, retrognathia, high palate, and enlarged tonsils or tongue due to their field of practice [58]. Furthermore, physicians and dentists can carry out the detection of possible SB by interviewing the patients on awareness of tooth grinding or clenching in relation to sleep, and to a probable status through clinical examination of jaw muscle pain or tenderness, and tooth-related signals. In this regard, it is essential to emphasize that the clinical evaluation should not be done in isolation and be based solely on tooth wear, as the latter is not the most reliable sign [1]. The assessment of tooth wear provides information on the cumulative amount of tooth surface loss, but does not provide information on the timing of the loss, i.e. whether the process is ongoing or is a result of a previous loss due to grinding *vs*. exacerbation by gastric reflux or acidic diet [5].

The studies conducted to assess the association between SB and OSA showed significant variability in their findings and conclusions. The studies were based on very different populations (i.e. SB only for which respiratory variables were analyzed, preferentially OSA with some SB overlap up to general population) and were designed with *a priori* intent to explore other objectives. It is also possible to observe gender and age differences in the test and control groups among the studies. It is known that the incidence of OSA is higher in men than in women, and there is a higher prevalence of OSA with high body mass index (BMI) and aging [26]. On the other hand, the incidence of self-reported SB decreases with age, and there appears to be no gender-related differences, although no difference was observed using PSG data [2,3]. The included studies do not describe the population in detail, particularly concerning the presence of comorbidities such as gastroesophageal reflux disease, habits such as alcohol and coffee intake, smoking, and use of antidepressant drugs, all of which are factors known to be associated with SB [59]. Due to their cross-sectional and descriptive study design, it is therefore impossible to establish a causal relationship even when an association is found [60]. Furthermore, only two studies evaluated the temporal relationship between OSA and SB events, which could be a better strategy for potentially explaining the putative causal relationship between the two conditions.

Variability was also noted in the metrics used to detect OSA and SB. For the diagnosis of OSA, the metrics used have improved or changed over time. In the earliest literature [42,47], a hypopnea event was defined as a 50% reduction in the thermocouple signal amplitude associated with a 4% fall in oxygen saturation. Nowadays, the scoring rules from the AASM [56] are quite different, with a hypopnea event being defined as a reduction in airflow of ≥ 30% for ≥ 10 s with a ≥ 3% decline in blood oxygen saturation or arousal [56]. Thus, depending on the metrics used, it is possible to expect differences in the association of SB with respiratory events. In addition, the evidence we have is likely to change, considering the need to refine the OSA metrics by including additional phenotypic parameters [61].

Along the same lines, different metrics have recently been reported on the cut-off values used to define sleep bruxers. There is no standardization regarding this cut-off point among studies that assess the association of SB and OSA controlled for gender and age and BMI and other putative factors such as OSA-related phenotype, anatomical obstruction, muscle tone, loop gain reactivity, and arousal threshold [62,63]. Also, the traditional standard approach consists of counting SB episodes recorded by electromyography during a PSG or with a portable recording device [64]. The identification of such events is made visually or with an algorithm detector, and training is required to obtain accuracy and precision. Such assessment is time-consuming, should be done blind to patient status or study objective, and is subject to inaccuracies since, similarly to other PSG scoring approaches, it may be sensitive to fatigue or distraction by the observer, leading to a risk of high inter- and intraindividual variability [65,66]. The development of more homogenized metrics will contribute to improve the accuracy and refinement of the SB assessment while moving towards a comprehensive approach, with ideally sleep recording done in a natural environment over more than one night to take into account the night-to-night variability of both SB and OSA metrics. [24,61]. Alternatively, should we have to rethink the methods considered as a reference standard to detect SB when

a study design challenges causality? Whether the temporality of SB episodes is protective or not was also not clarified since only two studies evaluated the temporal association, and even such studies used different methodologies [35,36].

Among the respiratory variables analyzed in the adult studies, not statistically or clinically significant correlations emerged in most of them. It is possible, although not proven, as observed in about 20% of otherwise healthy individuals with SB, that minor and transient fluctuations in the oxygen saturation levels might contribute to the genesis of rhythmic muscular masticatory activity during sleep [67] by promoting the occurrence of micro-arousals [68]; a hypothesis not supported by recent analyses in patients with comorbid SB and OSA [69]. Furthermore, the average arousal index values of the papers analyzed in the present scoping review were not different across studies, thereby refuting these assumptions, or at least suggesting that more in-depth assessments of these joint events are needed to identify if subgroups of patients may have more specific characteristics, based on anatomical or non-anatomical phenotype [62,63].

The challenge of studying the association between OSA and SB in children is even more problematic. Although not ideal, due to its complexity, costs, and the need to sleep in a sleep laboratory, the gold standard for reaching the diagnosis of OSA in children remains nocturnal PSG [57]. Due to the difficulties in performing this exam in children, a large proportion of the research studies related to OSA and SB implemented alternatives to PSG, such as sleep-related questionnaires [70–72] and symptoms-based scores [73]. A SR that evaluated the prevalence of SB in children pointed out that a major limitation of the existing studies was that PSG was not performed [74]. Although most of the included studies [49,51–53] showed an association between the SB and OSA, which is also in agreement with the literature [75], we must be careful when adjudicating this association as factual, considering the highly variable and relatively inaccurate methodology implemented in such studies, mainly the fact that the most studies not used the standard reference (PSG) to detect SB and some studies do not report the score to SB detection.

## Strengths, limitations

The extensive search in the literature for articles on the proposed topic that encompassed international and multidisciplinary databases, the meticulous subscription to scoping review guidelines and data summary, and consulting of the gray literature are obvious strengths of the present study, along with the inclusion of studies involving both adults and children. Furthermore, we are unaware of another knowledge synthesis article involving this topic in children.

As limitations, the most evident is the variability of the findings found along with the impossibility of interventional studies that could allow for more robust derivation of a potential causal relationship. It also became apparent that new metrics and technologies are needed to promote improved delineation of the criteria for SB and OSA detection.

Scoping reviews do not require a quality assessment of the reviewed studies, as is the case for SRs [76]. Therefore, such limitations in the interpretation of results must be acknowledged. Another limitation that should be highlighted was the attempt to investigate confounding factors. However, studies underreport this information, presenting simple statistics without adjustment for possible confounding factors.

## Suggestions for future research

In view of the mapped literature, it is possible to begin to understand why the systematic reviews on the subject reached the same conclusion as the current one, namely that there is not enough evidence to confirm or refute an association between SB and OSA [37–39]. If the primary studies continue to be developed in the same way, a definitive answer will remain elusive. Thus, based on our findings, we propose the following suggestions for future studies and a reflection on this field.

Future studies should focus their analysis not only on identifying a relationship between SB and OSA, but to investigate the temporal association of the episodes (order of occurrence, spacing between episodes, and duration) [35,36]. Furthermore, studies with larger population-based samples, participants who present the condition and do not present the condition (not just people with suspected OSA) [33] will be necessary. Population matching, based on sex, age, and BMI [16,77], should also be needed which can act as important confounders [78]. Along with the inclusion of accurate information on issues that can induce or attenuate SB such as tobacco, alcohol, caffeine consumption [59,79], use of medications and addictive substances [80], or previous treatment with positive airway pressure [81]. Information should be collected based on the medical history and behavior of the patient like hypertension, orofacial pain, tooth grinding, clenching [82], sleepiness, insomnia, fatigue, snoring choking, with validated questionnaire-based tools (e.g. Stop Bang or similar) [83].

PSG remains the standard reference method for the detection of SB and OSA, although home sleep testing offers other advantages, such as multiple night recordings to take into account intrinsic night-to-night variability [23,84]. However, despite using the criteria for diagnosing OSA established by the AASM that are primarily based on AHI, overreliance on AHI needs to be monitored, as suggested by a critical appraisal of the extensive literature on this subject [61]. Regarding SB detection, the AASM recommendations also seem to reflect the current state of the field. Based on these recommendations, an episode is considered if it happens at twice the basal amplitude and the events display the following characteristics: (1) tonic (at least one masseteric EMG shot greater than 2 seconds), (2) phasic (three or more shots of masseteric EMG lasting between 0.25 and 2 seconds), or (3) mixed (both types). The cut-off for defining SB should be more than 2 episodes per hour and/or > 25 bursts per hour, although their validity in general population studies when comorbidities are present still needs to be confirmed [85].

Again, keeping an eye on future developments is a must, as suggested by a recent review about research routes on improved SB metrics considering technological innovation for accurate assessments [24] and differences that may be explained by non-anatomical phenotype for OSA (e.g. muscle tone, loop gain, arousal threshold) [86] and SB (e.g. arousal index, heart rate variability, presence of big breath, other body movements) [69]. The summary of this information is shown in Table 5.

Considering the current findings, the following question arises: should financial and intellectual resources be invested in the search

**Table 5.** Summary of suggestions for future studies

- Investigate the temporal relationship of OSA and SB episodes
- The aim and hypotheses should be specific to challenge the association of OSA and SB
- Populations with sufficient sample size to support the statistical comparison
- Control for the influence or moderation of the following: age, gender, body mass index, anatomical variables, Mallampati and Freidman scores, use of medication, alcohol, cannabis, previous treatment (Continuous Positive Airway Pressure, oral device)
- Plan specific causality challenge: risk factor exposure, using medication or device to test if a causality can be reversed
- Use data collection method and scoring according to recognized standards
- Assess if frequency-severity is correlated (number of rhythmic masticatory muscle activity and AHI)
- Collect data in sleep environment if medical risk for patient, otherwise favor home sleep testing with oromotor, cardio-respiratory outcomes and this over 3-4 nights
- Collect questionnaire-based information on medical history of the patients and, when possible, using validated questionnaires
- If possible, to assess role of non- anatomical phenotype for OSA and SB.

for an association between OSA and SB? The answer is definitely yes, since so far, we do not have clear evidence on the matter, and acquiring such evidence would allow us to make better decisions in patient management. The limitations listed above, however, need to be overcome. In addition, it may also be necessary to strive for truly plausible metrics and more effective and cheaper detection methods.

For adults, it doesn't seem to be possible to confirm a relationship between OSA and SB, nor confirm a protective effect of bruxism in patients with OSA based on current literature. Although a relationship with OSA seems plausible for SB children, the identified scientific evidence is scarce and present limitations on the SB detection methods. At this point, it is important to study appropriate metrics, taking into account the possible existence of a subgroup for whom an association of SB and OSA may be present, i.e. a distinct phenotype that still needs to be identified.

## Supplementary material

Supplementary material is available at *SLEEP* online.

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## References

1. Lobbezoo F, *et al*. International consensus on the assessment of bruxism: report of a work in progress. *J Oral Rehabil*. 2018;**45**(11):837–844.
2. Manfredini D, *et al*. Epidemiology of bruxism in adults: a systematic review of the literature. *J Orofac Pain*. 2013;**27**(2):99–110.
3. Maluly M, *et al*. Polysomnographic study of the prevalence of sleep bruxism in a population sample. *J Dent Res*. 2013;**92**(7 Suppl):97S–103S.
4. Manfredini D, *et al*.. Prevalence of sleep bruxism in children: a systematic review of the literature. *J Oral Rehabil*. 2013;**40**(8):631–642.
5. Wetselaar P, *et al*. Associations between tooth wear and dental sleep disorders: a narrative overview. *J Oral Rehabil*. 2019;**46**(8):765–775.
6. Stuginski-Barbosa J, *et al*. Agreement of the international classification of sleep disorders criteria with polysomnography for sleep bruxism diagnosis: a preliminary study. *J Prosthet Dent*. 2017;**117**(1):61–66.
7. Palinkas M, *et al*. Impact of sleep bruxism on masseter and temporalis muscles and bite force. *Cranio*. 2016;**34**(5):309–315.
8. Zhou Y, *et al*. Does bruxism contribute to dental implant failure? a systematic review and meta-analysis. *Clin Implant Dent Relat Res*. 2016;**18**(2):410–420.
9. Chrcanovic BR, *et al*. Factors influencing the fracture of dental implants. *Clin Implant Dent Relat Res*. 2018;**20**(1):58–67.
10. Chrcanovic BR, *et al*. Analysis of technical complications and risk factors for failure of combined tooth-implant-supported fixed dental prostheses. *Clin Implant Dent Relat Res*. 2020;**22**(4):523–532.
11. Neu D, *et al*. Effect of sleep bruxism duration on perceived sleep quality in middle-aged subjects. *Eur J Oral Sci*. 2018;**126**(5):411–416.
12. Camara-Souza MB, *et al*. Association of sleep bruxism with oral health-related quality of life and sleep quality. *Clin Oral Investig*. 2019;**23**(1):245–251.
13. Duarte J, *et al*. Association between sleep bruxism and quality of life: a systematic review. *J Oral Facial Pain Headache*. 2020;**34**(4):341–352.
14. Feu D, *et al*. A systematic review of etiological and risk factors associated with bruxism. *J Orthod*. 2013;**40**(2):163–171.
15. Castroflorio T, *et al*. Sleep bruxism and related risk factors in adults: A systematic literature review. *Arch Oral Biol*. 2017;**83**:25–32.
16. Tan MWY, *et al*. Prevalence of sleep bruxism and its association with obstructive Sleep Apnea in adult patients: a retrospective polysomnographic investigation. *J Oral Facial Pain Headache*. 2019;**33**(3):269–77.
17. Hosoya H, *et al*. Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea syndrome. *Sleep Breath*. 2014;**18**(4):837–844.
18. Hollowell DE. Mandible position and activation of submental and masseter muscles during sleep. *J Appl Physiol*. 1991;**71**(6):2267–2273.
19. Lavigne GJ, *et al*. Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med*. 2013;**14**(1):30–46.

20. Hollowell DE, *et al*. Activation of masseter muscles with inspiratory resistance loading. *J Appl Physiol*. 1989;**67**(1):270–275.
21. Martinot J-B, *et al*. Artificial intelligence analysis of mandibular movements enables accurate detection of phasic sleep bruxism in OSA patients: a pilot study. *Nat and Sci Sleep*. 2021;**13**:1449–1459.
22. Lavigne GJ, *et al*. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res*. 1996;**75**(1):546–552.
23. Casett E, *et al*. Validity of different tools to assess sleep bruxism: a meta-analysis. *J Oral Rehabil*. 2017;**44**(9):722–734.
24. Lavigne G, *et al*. Research routes on improved sleep bruxism metrics: Toward a standardised approach. *J Sleep Res*. 2021;**30**(5):e13320.
25. Guilleminault C, *et al*. The sleep apnea syndromes. *Annu Rev Med*. 1976;**27**(1):465–484.
26. Senaratna CV, *et al*. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev*. 2017;**34**:70–81.
27. Tan HL, *et al*. Obstructive sleep apnea in children: a critical update. *Nat Sci Sleep*. 2013;**5**:109–123.
28. Park JG, *et al*. Updates on definition, consequences, and management of obstructive sleep apnea. *Mayo Clin Proc*. 2011;**86**(6):549–554.
29. Leger D, *et al*. Impact of sleep apnea on economics. *Sleep Med Rev*. 2012;**16**(5):455–462.
30. AlGhanim N, *et al*. The economic impact of obstructive sleep apnea. *Lung*. 2008;**186**(1):7–12.
31. Manfredini D, *et al*. Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. an expert opinion. *Sleep Breath*. 2015;**19**(4):1459–1465.
32. Saito M, *et al*. Weak association between sleep bruxism and obstructive sleep apnea. A sleep laboratory study. *Sleep Breath*. 2016;**20**(2):703–709.
33. Maluly M, *et al*. Sleep bruxism and its associations with insomnia and OSA in the general population of Sao Paulo. *Sleep Med*. 2020;**75**:141–148.
34. de Holanda TA, *et al*. Sleep architecture and factors associated with sleep bruxism diagnosis scored by polysomnography recordings: a case-control study. *Arch Oral Biol*. 2020;**112**:104685.
35. Sjoholm TT, *et al*. Sleep bruxism in patients with sleep-disordered breathing. *Arch Oral Biol*. 2000;**45**:889–896.
36. Saito M, *et al*. Temporal association between sleep apnea-hypopnea and sleep bruxism events. *J Sleep Res*. 2013;**23**:196–203.
37. De Luca Canto G, *et al*. Sleep bruxism and sleep-disordered breathing: a systematic review. *J Oral Facial Pain Headache*. 2014;**28**(4):299–305.
38. da Costa Lopes AJ, *et al*. Is there an association between sleep bruxism and obstructive sleep apnea syndrome? A systematic review. *Sleep Breath*. 2020;**24**(3):913–921.
39. Jokubauskas L, *et al*. Relationship between obstructive sleep apnoea syndrome and sleep bruxism: a systematic review. *J Oral Rehabil*. 2017;**44**(2):144–153.
40. Munn Z, *et al*. Systematic review or scoping review? Guidance for authors when choosing between a systematic or scoping review approach. *BMC Med Res Methodol*. 2018;**18**(1):143.
41. Tricco AC, *et al*. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. *Ann Intern Med*. 2018;**169**(7):467–473.
42. Phillips BA, *et al*. Effect of sleep position on sleep apnea and parafunctional activity. *Chest*. 1986;**90**(3):424–429.
43. Smardz J, *et al*. The meaning of the masticatory muscle tonic-type electromyographic pathway correlated with sleep bruxism and sleep-related breathing disorders—a polysomnographic study. *Sleep Med*. 2020;**68**:131–137.
44. Coelho PR, *et al*. Prevalência da Comorbidade entre o Bruxismo do Sono e a Síndrome de Apneia - Hipopneia Obstrutiva do Sono: Um Estudo Polissonográfico. *Pesq Brasil Odontopediatr Clín Integr*. 2012;**12**(4):491–496.
45. Martynowicz H, *et al*. The relationship between sleep bruxism and obstructive sleep apnea based on polysomnographic findings. *J Clin Med*. 2019;**8**(10):1–10.
46. Kim DH, *et al*. Sleep Bruxism episodes in patients with obstructive sleep apnea syndrome determined by in-laboratory polysomnography. *Appl Sci*. 2020;**10**(23).
47. Okeson JP, *et al*. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. *J Craniomand Disord*. 1991;**5**:258–264.
48. Castilho RL, *et al*. The interface between dentistry and respiratory sleep disorders in children. *Sleep Sci*. 2020;**13**(4):220–223.
49. Ferreira NMR, *et al*. Sleep bruxism associated with obstructive sleep apnea syndrome in children. *Cranio®*. 2016;**33**(4):251–255.
50. Gregório PB, *et al*. Symptoms of obstructive sleep apnea-hypopnea syndrome in children. *J Bras Pneumol*. 2008;**34**(6):356–361.
51. Goyal A, *et al*. Association of pediatric obstructive sleep apnea with poor academic performance: a school-based study from India. *Lung*. 2018;**35**(2):5.
52. Segu M, *et al*. Correlation between parental-reported tooth grinding and sleep disorders: investigation in a cohort of 741 consecutive children. *Pain Res Manag*. 2020;**2020**:3408928.
53. Tachibana M, *et al*. Associations of sleep bruxism with age, sleep apnea, and daytime problematic behaviors in children. *Oral Dis*. 2016;**22**(6):557–565.
54. Sheldon SH. Obstructive sleep apnea and bruxism in children. *Sleep Medicine Clinics*. 2010;**5**(1):163–168.
55. Singh N. *Sleep Bruxism Related Tooth Wear as a Clinical Marker for Obstructive Sleep Apnea/Hypopnea Syndrome in Children: Tufts University School of Dental Medicine*; 2011.
56. Berry RB, *et al*. AASM Scoring Manual Version 2.2 updates: new chapters for scoring infant sleep staging and home sleep apnea testing. *J Clin Sleep Med* 2015;**11**(11):1253–1254.
57. Tan HL, *et al*. Pediatric home sleep apnea testing: slowly getting there!. *Chest*. 2015;**148**(6):1382–1395.
58. Lavigne GJ, *et al*. Critical issues in dental and medical management of obstructive sleep apnea. *Crit Rev Oral Biol Med*. 2020;**99**:26–35.
59. Melo G, *et al*. Bruxism: an umbrella review of systematic reviews. *J Oral Rehabil*. 2019;**46**(7):666–690.
60. Grimes DA, *et al*. Descriptive studies: what they can and cannot do. *The Lancet*. 2002;**359**(9301):145–149.
61. Pevernagie DA, *et al*. On the rise and fall of the apnea-hypopnea index: a historical review and critical appraisal. *J Sleep Res*. 2020;**29**(4):e13066.
62. Osman AM, *et al*. Obstructive sleep apnea: current perspectives. *Nat Sci Sleep* 2018;**10**:21–34.
63. Carberry JC, *et al*. Personalized management approach for OSA. *Chest* 2018;**153**(3):744–755.
64. Thymi M, *et al*. Signal acquisition and analysis of ambulatory electromyographic recordings for the assessment of sleep bruxism: a scoping review. *J Oral Rehabil*. 2021;**48**(7):846–871.
65. Penzel T, *et al*. Computer based sleep recording and analysis. *Sleep Med Rev*. 2000;**4**(2):131–148.

66. Estevez PA, *et al*. Polysomnographic pattern recognition for automated classification of sleep-waking states in infants. *Med Biol Eng Comput*. 2002;**40**(1):105–113.
67. Dumais I, *et al*. Could transient hypoxia be associated with rhythmic masticatory muscle activity in sleep bruxism in the absence of sleep-disordered breathing? A preliminary report. *J Oral Rehabil*. 2015;**42**(11):810–818.
68. Chowdhury T, *et al*. Sleep disorders: is the trigemino-cardiac reflex a missing link? *Front Neurol*. 2017;**8**:63.
69. Suzuki Y, *et al*. Changes in oxygen and carbon dioxide in the genesis of sleep bruxism: a mechanism study. *J Prosthodont Res*. 2020;**64**(1):43–47.
70. Kadmon G, *et al*. Validation of a pediatric obstructive sleep apnea screening tool. *Int J Pediatr Otorhinolaryngol*. 2013;**77**(9):1461–1464.
71. Hasukic B. OSA-18 survey in evaluation of sleep-disordered breathing in children with adenotonsillar hypertrophy. *Med Arch*. 2013;**67**(2):111–114.
72. Kuwada A, *et al*. Japanese Sleep Questionnaire for Elementary Schoolers (JSQ-ES): validation and population-based score distribution. *Sleep Med*. 2018;**41**:69–77.
73. Chang L, *et al*. Combination of symptoms and oxygen desaturation index in predicting childhood obstructive sleep apnea. *Int J Pediatr Otorhinolaryngol*. 2013;**77**(3):365–371.
74. Machado E, *et al*. Prevalence of sleep bruxism in children: a systematic review. *Dental Press J Orthod*. 2014;**19**(6):54–61.
75. Castroflorio T, *et al*. Risk factors related to sleep bruxism in children: a systematic literature review. *Arch Oral Biol*. 2015;**60**(11):1618–1624.
76. Arksey H, *et al*. Scoping studies: towards a methodological framework. *International Journal of Social Research Methodology*.2005;**8**(1):19–32.
77. Kuang B, *et al*. Associations between sleep bruxism and other sleep-related disorders in adults: a systematic review. *Sleep Med*. 2022;**89**:31–47.
78. Pauletto P, *et al*. Association between obstructive sleep apnea and health-related quality of life in untreated adults: a systematic review. *Sleep Breath*. 2021;**25**(4):1773–1789. doi:10.1007/s11325-021-02323-1.
79. Bertazzo-Silveira E, *et al*. Association between sleep bruxism and alcohol, caffeine, tobacco, and drug abuse: a systematic review. *J Am Dent Assoc*. 2016;**147**(11):859–866 e4.
80. de Baat C, *et al*. Medications and addictive substances potentially inducing or attenuating sleep bruxism and/or awake bruxism. *J Oral Rehabil*. 2021;**48**(3):343–354.
81. Oksenberg A, *et al*. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. *Sleep Med*. 2002;**3**:513–515.
82. Carra MC, *et al*. Overview on sleep bruxism for sleep medicine clinicians. *Sleep Medicine Clin*. 2015;**10**(3):375–384.
83. Kline LR, *et al*. Clinical presentation and diagnosis of obstructive sleep apnea in adults. 2017. UpToDate [https://www.uptodate.com/contents/clinical-presentation-and-diagnosis-of-obstructive-sleep-apnea-in-adults?topicRef=7695&source=related_link] Accessed Feb 06 2022.
84. Rundo JV, *et al*. Polysomnography. *Handb Clinical Neurol*. 2019;**160**:381–392.
85. Rompré P, *et al*. Identification of a sleep bruxism subgroup with a higher risk of pain. *J Dent Res*. 2007;**86**(9):837–842.
86. Eckert DJ. Phenotypic approaches to obstructive sleep apnoea—new pathways for targeted therapy. *Sleep Med Rev*. 2018;**37**:45–59.